
Joseph Jones

University of Massachusetts - Amherst

Follow this and additional works at: https://scholarworks.umass.edu/dissertations_2

Part of the Biological and Physical Anthropology Commons

Recommended Citation


https://scholarworks.umass.edu/dissertations_2/307

This Open Access Dissertation is brought to you for free and open access by the Dissertations and Theses at ScholarWorks@UMass Amherst. It has been accepted for inclusion in Doctoral Dissertations by an authorized administrator of ScholarWorks@UMass Amherst. For more information, please contact scholarworks@library.umass.edu.
THE POLITICAL ECOLOGY OF EARLY CHILDHOOD LEAD EXPOSURE
AT THE NEW YORK AFRICAN BURIAL GROUND

A Dissertation Presented

by

JOSEPH L. JONES

Submitted to the Graduate School of the
University of Massachusetts Amherst in partial fulfillment
of the requirements for the degree of

DOCTOR OF PHILOSOPHY

February 2015

Anthropology
DEDICATION

To Ancestors and Family
ACKNOWLEDGMENTS

It is a good thing to be able to look back on a long journey with genuinely fond memories as I do now. I am fortunate to have had the best mentors and teachers imaginable and I thank them wholeheartedly for making my graduate experience so rich and worthwhile. I begin by acknowledging the patience, wit and wisdom of my advisor and friend, Dr. Alan Goodman. I thank him tremendously for his constant support. It has been an honor being his “gradual student.” Thanks also to Dr. Chaia Heller and Ruby Goodman for sharing him – and also their home during my visits from Maryland and Virginia.

I wish to thank the other members of my Dissertation Committee for their support and intellectual stimulation and guidance. They are: Drs. Dulasiri Amarasiriwardena, John Bracey, Robert Paynter and Alan Swedlund. Beyond and well before providing critical feedback and writing advice on dissertation chapters, each of these individuals has been instrumental to my professional development and vision of what a scholar should be and do. I gladly spent many days and evenings in Hampshire College’s chemistry laboratory, for example, inspired by Dr. Amarasiriwardena’s enthusiasm for research and teaching as he admonished me to “think analytically!” I am grateful to my Dissertation Committee, as well, for the flexibility and patience afforded me as I left Amherst to work at the American Anthropological Association’s (AAA) and gradually reoriented to dissertation writing.

I cannot thank Drs. Swedlund and R. Brooke Thomas enough for opening up their homes and providing me a place to stay as I commuted from Virginia to teach at Hampshire College. Dr. Thomas previously sat on my M.A. Committee (and practically served on my Dissertation Committee). The kindness and generosity shown by them and their wives, M.A. Swedlund and
Shirley Thomas, is rare. I cherish the encouragement, advice and friendship of these biocultural pioneers.

Others associated with UMass and/or Hampshire College whose knowledge and support, at some time or another, has proven important include Drs. Alexis Dolphin, Brigitte Holt, Steve King, Betsy Krause, Tom Leatherman, Lynette Leidy, Maddie Marquez, Ventura Perez, Rhan-ju Song, Pamela Stone, and Martin Wobst. Ms. Kristen Shrout provided necessary technical assistance at Hampshire College and I am supremely indebted to Ms. Shelley Silva for all of her logistical wizardry and institutional knowledge of the university.

It is a wonderful privilege to be a part of something in which you truly believe. I have been a part of two such projects. I thank Dr. Michael Blakey for his contributions to African diasporic bioarchaeology and for inviting me to join the New York African Burial Ground (NYABG) Project. Under his leadership, the project’s mission of sound scholarship and social justice resonated deeply with me as an undergraduate student at Howard University as it does today. Our work and my lessons continue. Likewise, my time at the AAA was spent working closely with Dr. Yolanda Moses on the RACE public education initiative. During this time, I came to value her warmth and insights immensely.

I would be remiss if I did not acknowledge at this point Mr. Mark Mack who taught my Introduction to Biological Anthropology course, all but sealing my fate as a future anthropologist. Mr. Mack was a truly gifted teacher and an even better friend and brother. Having joined the ancestors, he is missed but this study bears his influence as well. Dr. Rachel Watkins, another colleague whom I first met at Howard University, also warrants special mention for her longtime support, as do two other principal researchers on the NYABG Project, Drs. Fatimah Jackson and Warren Perry. I thank Drs. Christopher DeCorse and Peter Outridge and Mr. James Boachie-Ansah for facilitating the inclusion of African control samples and
calibration standards required for this study. I am grateful to the New York African American descendant community for allowing this research. May it prove useful.

Finally, it is a beautiful thing to have the love and support of family. Danielle, my wife, has shared this journey. Her patience and understanding have made this – our dissertation – possible. One day Nia, my daughter, will read this and know that she was my wonder and hope when the path was not always clear. Steven, David, Tracia and Stacia – my siblings – and parents, Rev. Robert and Mary Jones, have always been my steadfast supporters. Although dad, too, has joined the ancestors, I like to think he would be proud.
ABSTRACT

THE POLITICAL ECOLOGY OF EARLY CHILDHOOD LEAD EXPOSURE AT THE NEW YORK AFRICAN BURIAL GROUND

FEBRUARY 2015

JOSEPH L. JONES, B.A., HOWARD UNIVERSITY

M.A., UNIVERSITY OF MASSACHUSETTS AMHERST

Ph.D., UNIVERSITY OF MASSACHUSETTS AMHERST

Directed by: Professor Alan H. Goodman

Nearly 25 years ago federal officials unearthed over 400 skeletal remains in Lower Manhattan. The site of the excavation was the New York African Burial Ground (NYABG), a 17th- and 18th-century cemetery for the city’s mostly enslaved African population. Today, the burial ground serves as a reminder of New York’s 200-year experiment with slavery. It is the first National Monument to honor enslaved African New Yorkers. This recognition is a testament to the resolve of African American descendants and their allies who, through political activism, would see these ancestors afforded in death some of the respect denied them in life.

Descendant community activism also paved the way for the site’s interdisciplinary investigation, the NYABG Project. Recovering complex diasporic biohistories from the NYABG was a major scientific undertaking made more challenging and rewarding by the project’s high standards of public inclusion and accountability. Co-developed by community members and scholars, the NYABG Project now stands as a model of critically engaged biocultural anthropology.

This dissertation study draws upon and continues the work of NYABG researchers. It is a reconstruction of early life lead exposure via laser ablation-inductively coupled plasma-mass spectrometry (LA-ICP-MS) of dental enamel. With its high inorganic content, enamel provides a
stable chemical record of an individual’s diet, nutrition and pollution events, which in turn reflect political, economic and cultural factors. For this population, relatively high levels of skeletal lead suggest time spent in the Americas while low levels more likely indicate birth in Africa prior to forced migration. Here, lead concentrations in enamel that forms during the first several years of life are measured, mapped microspatially, and rendered as chronological age profiles. Mean differences and distribution/age profiles are compared for 44 NYABG children and adults in order to determine their African or American birthplaces and related health and cultural experiences (e.g., lead poisoning and dental modification). For some individuals, comparative analysis of later forming teeth was undertaken to explore the possibility of migration during childhood.

Enamel-lead concentrations range from 0.39 μg g\(^{-1}\) (i.e., the instrument limit of detection or LOD) to 14.7 μg g\(^{-1}\), suggesting a range of exposures in which some individuals spent their childhoods in high-lead environments. The most striking finding is that mean enamel-lead concentration for young children (5.88 μg g\(^{-1}\)) is over five times that of adults (1.11 μg g\(^{-1}\)), a significant difference reflecting these groups’ mostly American versus African geographic origins, respectively. Other findings raise questions at the intersections of natality, health and culture. For example, contra most reports, do relatively high lead concentrations for some individuals indicate that cultural dental modification persisted in the Americas?

This study is the first quantitative LA-ICP-MS analysis of human lead exposure in early America. LA-ICP-MS has proven critical for assessing overall lead burden as well as age-related changes in the sources and nature of exposure. The methodology developed for this study has enabled a rich assessment of African diasporic environmental biohistory, health and culture during slavery. As with the “rediscovery” of the NYABG, this is a moment and a tool for discovering new history and new dimensions of the human experience, then and now.
# TABLE OF CONTENTS

<table>
<thead>
<tr>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACKNOWLEDGMENTS</td>
</tr>
<tr>
<td>ABSTRACT</td>
</tr>
<tr>
<td>LIST OF TABLES</td>
</tr>
<tr>
<td>LIST OF FIGURES</td>
</tr>
<tr>
<td>CHAPTER</td>
</tr>
<tr>
<td>1. INTRODUCTION</td>
</tr>
<tr>
<td>2. BIOARCHAEOLOGY: RECONSTRUCTING LIFE HISTORY AND LIVED EXPERIENCE</td>
</tr>
<tr>
<td>Introduction</td>
</tr>
<tr>
<td>Life Stories from Skeletal Remains</td>
</tr>
<tr>
<td>Biohistory</td>
</tr>
<tr>
<td>Bioculture</td>
</tr>
<tr>
<td>Political Ecology</td>
</tr>
<tr>
<td>3. ANTHROPOLOGY OF THE HISTORICAL AFRICAN DIASPORA</td>
</tr>
<tr>
<td>Introduction</td>
</tr>
<tr>
<td>African Diaspora in History, Theory and Practice</td>
</tr>
<tr>
<td>Origins, Agency and Artifacts</td>
</tr>
<tr>
<td>Origins, Agency and Teeth</td>
</tr>
<tr>
<td>4. THE NEW YORK AFRICAN BURIAL GROUND</td>
</tr>
<tr>
<td>Introduction</td>
</tr>
<tr>
<td>Slavery in New York and the African Burial Ground</td>
</tr>
<tr>
<td>The NYABG Project</td>
</tr>
<tr>
<td>Background</td>
</tr>
<tr>
<td>Research Goals</td>
</tr>
<tr>
<td>Major Research Findings</td>
</tr>
<tr>
<td>5. POLITICAL ECOLOGY OF HUMAN LEAD EXPOSURE</td>
</tr>
<tr>
<td>Introduction</td>
</tr>
<tr>
<td>Environmental Lead and Human Biology</td>
</tr>
</tbody>
</table>
6. MATERIALS AND METHODS ............................................................. 118

Introduction ......................................................................................... 118
Laser Ablation-Inductively Coupled Plasma-Mass Spectrometry (LA-ICP-MS) .......... 118
Study Sample ....................................................................................... 125
Sample Preparation ........................................................................... 130
LA-ICP-MS Measurement .................................................................. 131
Data Processing and Analysis .............................................................. 135
Quantification ..................................................................................... 135
Distribution and Age Profiling ......................................................... 137
Analysis .............................................................................................. 140

7. RESULTS: MEAN ENAMEL-LEAD CONTENT ......................................... 143

Permanent First Molars/Incisors .......................................................... 143
Subadults and Adults ........................................................................... 146
Age ...................................................................................................... 147
Sex ...................................................................................................... 149
Temporal Groups ................................................................................ 152
Comparison of Permanent First Molars/Incisors and Third Molars .......... 154
Conclusions ....................................................................................... 158

8. RESULTS: ENAMEL-LEAD PROFILES ..................................................... 159

Microspatial Pb Distribution Profiles .................................................. 160
Chronological Age Profiles ............................................................... 164
Subadults ............................................................................................ 165
Modified Adults .................................................................................. 171
Non-modified Adults ......................................................................... 176
Early Versus Late Enamel-lead ......................................................... 182
Conclusions ....................................................................................... 186

9. DISCUSSION .......................................................................................... 188

Natality ............................................................................................... 193
Sources and pathways of exposure ................................................................. 202
  Alcohol ........................................................................................................... 204
  Water ............................................................................................................. 208
  Foods and other beverages ........................................................................... 210
  Dust and soil ................................................................................................. 212
Health consequences .......................................................................................... 221
Conclusions ....................................................................................................... 225

10. SUMMARY AND CONCLUDING REMARKS ..................................................... 226

APPENDICES ....................................................................................................... 232
A. SAMPLE DATA COLLECTION FORM ............................................................ 233
B. DATA PROCESSING IN EXCEL ....................................................................... 234
C. ENAMEL-LEAD MICROSpatial DISTRIBUTION GRAPHS: NYABG SUBADULTS .... 235
D. ENAMEL-LEAD MICROSpatial DISTRIBUTION GRAPHS: NYABG MODIFIED ADULTS ........................................................................................................ 236
E. ENAMEL-LEAD MICROSpatial DISTRIBUTION GRAPHS: NYABG NON-MODIFIED ADULTS ........................................................................................................ 237

BIBLIOGRAPHY .................................................................................................... 238
### LIST OF TABLES

<table>
<thead>
<tr>
<th>Table</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>3.1.</td>
<td>African dental modification patterns and contemporary national boundaries.</td>
</tr>
<tr>
<td>4.1.</td>
<td>Importations of captives to New York by region (via West Indies).</td>
</tr>
<tr>
<td>4.2.</td>
<td>Direct trade of captives from Africa to New York.</td>
</tr>
<tr>
<td>5.1.</td>
<td>Commonly used lead compounds.</td>
</tr>
<tr>
<td>5.2.</td>
<td>Biomarkers of human lead exposure and uptake.</td>
</tr>
<tr>
<td>6.1.</td>
<td>Study sample.</td>
</tr>
<tr>
<td>6.2.</td>
<td>Laser ablation and ICP-MS optimized operating conditions.</td>
</tr>
<tr>
<td>7.1.</td>
<td>Enamel-lead concentrations by individual/tooth.</td>
</tr>
<tr>
<td>7.2.</td>
<td>NYABG M1 or I1 enamel-lead concentrations by sex and analytical cohort.</td>
</tr>
<tr>
<td>7.3.</td>
<td>NYABG M1 or I1 enamel-lead concentrations by temporal group and analytical cohort.</td>
</tr>
<tr>
<td>7.4.</td>
<td>NYABG M1 or I1 enamel-lead concentrations by temporal group and sex.</td>
</tr>
<tr>
<td>8.1.</td>
<td>NYABG M1 or I1 enamel-lead microspatial distribution patterns by analytical cohort.</td>
</tr>
<tr>
<td>9.1.</td>
<td>Potential sources of lead exposure for the NYABG population.</td>
</tr>
<tr>
<td>9.2.</td>
<td>Distribution of Blacks in New York City Households in 1703, by Occupation of Household Head.</td>
</tr>
<tr>
<td>9.3.</td>
<td>Enamel-lead and derived blood-lead concentrations by individual/tooth (following Grobler et al. [2000]).</td>
</tr>
</tbody>
</table>
# LIST OF FIGURES

<table>
<thead>
<tr>
<th>Figure</th>
<th>Description</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.1</td>
<td>Location of the New York African Burial Ground</td>
<td>2</td>
</tr>
<tr>
<td>1.2</td>
<td>NYABG Burial 114, a 45-50-year-old man with culturally-modified dentition.</td>
<td>4</td>
</tr>
<tr>
<td>2.1</td>
<td>Nineteenth-century critiques of scientific racism by Frederick Douglass (left) and Anténor Firmin (right) foreshadowed contemporary critical biocultural perspectives on human biology, health and race.</td>
<td>16</td>
</tr>
<tr>
<td>2.2</td>
<td>Franz Boas (left), William Montague Cobb (center) and Ashley Montagu (right) emphasized the importance of human plasticity over racial and racist explanations of human biological variation.</td>
<td>17</td>
</tr>
<tr>
<td>2.3</td>
<td>Political economy model of stressor interactions that result in social inequalities and impaired adaptive capacity.</td>
<td>24</td>
</tr>
<tr>
<td>3.1</td>
<td>Cultural dental modification appears amongst historic populations from all parts of the world. These permanent central upper incisors from a Tamil man of uncertain have been sharpened and incised to create labial grooves.</td>
<td>52</td>
</tr>
<tr>
<td>3.2</td>
<td>Furrowed incisors from a Viking Age archaeological site in Sweden.</td>
<td>53</td>
</tr>
<tr>
<td>3.3</td>
<td>African dental modification.</td>
<td>54</td>
</tr>
<tr>
<td>4.1</td>
<td>Eight primary coastal regions that exported captive and enslaved Africans to the Americas during the Transatlantic Slave Trade.</td>
<td>70</td>
</tr>
<tr>
<td>4.2</td>
<td>Construction and partial excavation of the African Burial Ground at the 290 Broadway Block in Lower Manhattan.</td>
<td>79</td>
</tr>
<tr>
<td>4.3</td>
<td>Protesting the GSA's handling of the African Burial Ground.</td>
<td>81</td>
</tr>
<tr>
<td>4.4</td>
<td>Artifacts from the NYABG.</td>
<td>88</td>
</tr>
<tr>
<td>4.5</td>
<td>Lead musket ball found lodged in the ribs of Burial 25.</td>
<td>89</td>
</tr>
<tr>
<td>4.6</td>
<td>Strontium isotope ratios for NYABG children and adults.</td>
<td>93</td>
</tr>
<tr>
<td>5.1</td>
<td>Risk focusing model on the relationship of social class and toxic exposure.</td>
<td>98</td>
</tr>
<tr>
<td>5.2</td>
<td>Generalized model of elemental exposure, uptake and tissue deposition.</td>
<td>102</td>
</tr>
<tr>
<td>6.1</td>
<td>Laser ablation (A) and ICP-MS (B) instrumentation.</td>
<td>132</td>
</tr>
<tr>
<td>6.2</td>
<td>Triplicate scans of B-101 showing reproducible LA-ICP-MS results.</td>
<td>133</td>
</tr>
</tbody>
</table>
6.3. Epoxy-embedded and polished permanent upper left first molar in bucco-lingual cross-section showing dental tissues and the pulp cavity (Burial 35; 9x).

6.4. Plot of $^{208}\text{Pb}/^{43}\text{Ca}$ vs. Pb concentration (0.12, 2.09 and 18.45 μg g$^{-1}$) in calcium phosphate external calibration standards.

6.5. Diagram of a molar tooth in bucco-lingual cross-section.


6.7. Estimates for chronological ages of human enamel formation for permanent anterior teeth (incisors and canines) by decile of crown growth.

7.1. Box plot of M1 or I1 enamel-lead concentrations by analytical cohort for NYABG individuals.

7.2. Scatterplot of M1 or I1 enamel-lead concentrations by age-at-death for NYABG individuals.

7.3. Scatterplot of M1 or I1 enamel-lead concentrations by age-at-death for NYABG adults.

7.4. Box plot of M1 or I1 enamel-lead concentrations by sex for NYABG individuals.

7.5. Box plot of M1 or I1 enamel-lead concentrations by sex and analytical cohort for NYABG adults.

7.6. Box plot of M1 or I1 enamel-lead concentrations by temporal group for NYABG individuals showing variation from the mid-1730s through the post-American Revolutionary era.

7.7. Box plot comparison of M1 or I1 and M3 enamel-lead concentrations for NYABG adults.

7.8. Paired NYABG M1 or I1 and M3 enamel-lead concentrations.

8.1. Examples of NYABG M1 or I1 $^{208}\text{Pb}$ microspatial distribution pattern A, a steady lead signal.

8.2. Examples of NYABG M1 or I1 $^{208}\text{Pb}$ microspatial distribution pattern B, an increasing lead signal.

8.3. Examples of NYABG M1 or I1 $^{208}\text{Pb}$ microspatial distribution pattern C, a decreasing lead signal.

8.4. Examples of NYABG M1 or I1 $^{208}\text{Pb}$ microspatial distribution pattern D, a “mixed” or fluctuating lead signal.

8.5. Enamel-lead distribution graph for the Kasana control tooth sample.
8.6a. Enamel-Pb/age graph for Burial 7 (LRM1), a 3- to 5-year-old subadult of undetermined sex. ........................................................................................................165
8.6b. Enamel-Pb/age graph for Burial 22 (LRM1), a 2.5- to 4.5-year-old subadult of undetermined sex. ........................................................................................................166
8.6c. Enamel-Pb/age graph for Burial 35 (ULM1), an 8- to 10-year-old subadult of undetermined sex. ........................................................................................................166
8.6d. Enamel-Pb/age graph for Burial 39 (LRM1), a 5- to 7-year-old subadult of undetermined sex. ........................................................................................................167
8.6e. Enamel-Pb/age graph for Burial 43 (LRM1), a 2.5- to 4.5-year-old subadult of undetermined sex. ........................................................................................................167
8.6f. Enamel-Pb/age graph for Burial 126 (LLM1), a 3.5- to 5.5-year-old subadult of undetermined age. ........................................................................................................168
8.6g. Enamel-Pb/age graph for Burial 138 (URM1), a 3- to 5-year-old subadult of undetermined age. ........................................................................................................168
8.6h. Enamel-Pb/age graph for Burial 180 (ULM1), an 11- to 13-year-old female. ..........169
8.6i. Enamel-Pb/age graph for Burial 219 (LRM1), a 4- to 5-year-old subadult of undetermined age. ........................................................................................................169
8.6j. Enamel-Pb/age graph for Burial 244 (LLM1), a 5- to 9-year-old subadult of undetermined age. ........................................................................................................170
8.6k. Enamel-Pb/age graph for Burial 405 (URM1), a 6- to 10-year-old subadult of undetermined age. ........................................................................................................170
8.7a. Enamel-Pb/age graph for Burial 9 (LLM1), a 35- to 45-year-old female. .............171
8.7b. Enamel-Pb/age graph for Burial 9 (LRM3), a 35- to 45-year-old female. .............171
8.7c. Enamel-Pb/age graph for Burial 47 (LLM1), a 35- to 45-year-old male. ..........172
8.7d. Enamel-Pb/age graph for Burial 101 (LRI1), a 26- to 35-year-old male. ..........172
8.7e. Enamel-Pb/age graph for Burial 101 (LLM3), a 26- to 35-year-old male. ..........173
8.7f. Enamel-Pb/age graph for Burial 106 (LRM1), a 25- to 35-year-old female (probable) ........................................................................................................173
8.7g. Enamel-Pb/age graph for Burial 266 (URM1), a 25- to 35-year-old female. ..........174
8.7h. Enamel-Pb/age graph for Burial 366 (LLM1), a 34- to 62-year-old adult of undetermined sex. ........................................................................................................174
8.7i. Enamel-Pb/age graph for Burial 367 (ULM1), a 25- to 35-year-old female (probable) adult of undetermined sex. .................................................................175
8.7j. Enamel-Pb/age graph for Burial 377 (ULM1), a 33- to 58-year-old female........175
8.8a. Enamel-Pb/age graph for Burial 12 (ULM1), a 35- to 45-year old female...........176
8.8b. Enamel-Pb/age graph for Burial 25 (URI1), a 20- to 24-year-old female. ...............176
8.8c. Enamel-Pb/age graph for Burial 49 ULM1, a 40- to 50-year-old female.............177
8.8d. Enamel-Pb/age graph for Burial 63 (LRM3), a 35- to 45-year old male.. ............177
8.8e. Enamel-Pb/age graph for Burial 150 (LRM1), a 20- to 28-year-old female.............178
8.8f. Enamel-Pb/age graph for Burial 176 (LRM1), a 20- to 24-year-old male. .................178
8.8g. Enamel-Pb/age graph for Burial 179 (LRM1), a 25- to 30-year-old male. ...............179
8.8h. Enamel-Pb/age graph for Burial 323 (LRM1), a 19- to 30-year-old male. ...............179
8.8i. Enamel-Pb/age graph for Burial 324 ULM1, a 25- to 35-year-old female. ...............180
8.8j. Enamel-Pb/age graph for Burial 324 (LRM3), a 25- to 35-year-old female..............180
8.8k. Enamel-Pb/age graph for Burial 335 (LLLM1), a 25- to 35-year-old female.............181
A.1. Sample data and laser conditions record form. ....................................................233
B.1. Data processing in Excel (background subtraction and normalization steps). .............234
C.1. M1 or I1 enamel-lead microspatial distribution graphs (log scale) for NYABG subadults. Surface enamel peaks are indicated by a red arrow. .................................................................235
D.1. M1 or I1 enamel-lead microspatial distribution graphs (log scale) for NYABG modified adults. Surface enamel peaks are indicated by a red arrow.....................................................236
E.1. M1 or I1 enamel-lead microspatial distribution graphs (log scale) for NYABG non-modified adults. Surface enamel peaks are indicated by a red arrow. .................................237
CHAPTER 1

INTRODUCTION

In the spring of 1991 archaeologists unearthed human skeletal remains in Lower Manhattan while surveying land on behalf of the United States General Services Administration (GSA). The location was 290 Broadway, just north of City Hall, and the site of the planned 34-story Ted Weiss Federal Building. The remains were soon discovered to be amongst the city’s earliest inhabitants, those of New York’s “African Founders” (Blakey 2010) – mostly enslaved children, women and men. To the surprise of many, under some 25 feet of landfill and debris, the archaeologists had uncovered the 18th-century “Negros [sic] Burial Ground,” soon to be renamed the “African Burial Ground” (Figure 1.1).

The site’s excavation eventually yielded skeletal remains of over 400 individuals. While only a fraction of the 15,000 people estimated to have been buried in the full 5.5-acre cemetery, these remains currently constitute the largest colonial African and African-American bioarchaeological sample (Blakey 2009). This unique and unexpected find garnered considerable attention within and beyond academic circles. Curious onlookers included journalists, legislators, scholars, activists and a host of other community members from varied backgrounds. Their interest was understandable. The bones and teeth of these enslaved Africans belied easy, uncritical depictions of a “free North.” Michael Blakey and others have written extensively about the site’s historical significance and controversies surrounding its “rediscovery” (Blakey 2009, 2010; Epperson 2004; Harrington 1993). Such issues would inform the development of the New York African Burial Ground (NYABG) Project, a study groundbreaking in several respects including the leadership roles assumed by African diasporic scholars under Blakey’s direction. These issues and the project – its origins, primary goals and some major findings – are discussed in detail in chapter four.
The physical presence of ancestral skeletal remains affords the opportunity to assess collective and individual experiences or “biohistories” directly and in light of current biocultural theory and methods. This study is an attempt at recovering a part of these biohistories by reconstructing early life lead pollution. I hope to shed light on place of birth and early life migrations, as well as the sources and consequences of lead pollution. I have analyzed dental enamel, which develops incrementally as a near-permanent chronological archive of childhood elemental exposure. I collected enamel-lead data by laser ablation- (LA) inductively coupled plasma-mass spectrometry (ICP-MS), a versatile method for determining elemental concentrations in solid samples.

Following the convention established in the project’s scholarly reports, I refer to the full cemetery as the “African Burial Ground” and the excavated portion studied as the “New York African Burial Ground.”
Why focus on lead? Prior research suggests that, on average, NYABG children who likely grew up enslaved in the Americas experienced earlier and more chronic exposure to lead than did adults with and without culturally-modified teeth (CMT), a practice commonly associated with African-born individuals (Goodman et al. 2009). The main goal of this study is to further clarify these patterns while identifying sources, pathways, and biocultural consequences – i.e., the political ecology – of lead exposure and burden for those buried at the NYABG.

For this dissertation study I analyzed enamel-lead distributions in order to better understand group and individual differences in the following.

1) **Natal or geographic origin and associated skeletal material culture.** Skeletal-lead content potentially reflects different lead exposures during life and thus different pollutant environments. With the proper tooth, enamel-lead content in particular is useful for estimating natality or birthplace. Enamel-lead levels should reflect natural or background versus technological exposure. Lead use throughout the Americas was widespread during the 18th century and relatively lacking in western Africa where most enslaved people originated, making enamel-lead a potential biomarker of different pollutant and natal environments for enslaved Africans.

This distinction is of interest because it allows for an evaluation of the position that CMT indicates African natality because dental modification was counter-adaptive in the Americas (Handler 1994; Corruccini et al. 1987; Schroeder et al. 2012, 2013).

Following this logic and based on earlier NYABG studies, I hypothesized that average lead concentrations are higher for NYABG subadults than adults, reflecting the probable American geographic origins of most children. I also hypothesized greater variation in lead content for subadults than for adults with CMT given the likely
restricted, low-level range of non-technological exposure for the latter. An example of CMT from the burial ground appears in Figure 1.2.

2) **Sources and pathways of lead exposure.** Laser ablation-inductively coupled plasma-mass spectrometry (LA-ICP-MS) enables the charting of microspatial and temporal lead distributions across enamel layers. As these layers form within known age ranges, the distribution profiles thus produced were used to determine the timing of lead exposure as well as the patterns, from highly variable to constant over time, which in turn may suggest specific pathways if not specific sources of exposure (Warren 2000). I turned to historical and epidemiological literature to explore the most likely sources of early-life lead exposure in 18th-century New York.

3) **Health and developmental consequences of lead exposure.** An environmental poison, lead's toxicokinetic properties vary with age and physiological and health
status. In extreme cases, lead poisoning causes encephalopathy and death, but even low-level exposure can impair neurological development and cognitive function (Lanphear et al. 2005; CDC 2012). Without access to blood-lead data, it is difficult to determine who would have shown symptoms of clinical lead poisoning and what those specific symptoms might have been. The existing literature on enamel- and blood-lead relationships remains inconclusive. Nonetheless, I address this topic briefly in chapter nine. My primary focus, however, is on identifying health cohorts, i.e., groups of individuals consisting of those most and least likely to have suffered similar toxic effects of lead based on their overall early-life lead burden.

This dissertation is organized into ten chapters. Following this introduction, chapters two through five provide necessary anthropological, archaeological and historical context for the current study. In this first section, I introduce the major theoretical elements (e.g., political ecology and diaspora) that inform my interpretations of enamel-lead data. I also explain the important role that skeletal-lead analysis has played in African diasporic bioarchaeology and why teeth are especially important for piecing together past lives, including detecting diasporic movements across the life span. The remaining five chapters lay out the study’s experimental component, the results of analysis and interpretation of data, and offer next steps for honing our understanding of enslaved African origins, migrations and lifeways.

In chapters two and three I discuss theoretical developments that are enabling scholars to expand upon old and develop new interpretations of African diasporic biohistory. I begin in chapter two with an overview of biocultural anthropology from its emergence during the second half of the twentieth century through the current development of “social bioarchaeology.” I introduce political ecology as a synthetic approach to the study of human biology that combines adaptability, ecological and political-economic perspectives (Leatherman 2005; Leatherman and
Thomas 2001). Uniquely, this approach allows for integrative analysis of historical, evolutionary and political-economic forces that shape and alter skeletal biologies that comprise the bioarchaeological record (Thomas 1998; Armelagos et al. 2005). From this perspective, researchers are expanding the concept of human biology to include considerations of the “upstream” sociocultural conditions that promote or undermine optimal health. Often, these conditions – such as migration and poverty – result from structurally unequal access to necessary material resources and, therefore, constitute major risk factors for compromised health over the life span (Armelagos et al. 2009). My focus on early-life environmental health conditions for forced migrant and enslaved diasporic Africans is one example of what critical biological anthropologists have termed the “biology of poverty” (Thomas 1998).

There are multiple African diasporas occupying various, sometimes overlapping times and spaces (Dodson 2001), and multiple ways of discerning diaspora at the population and individual levels. This study is concerned with the historical African diaspora of the Americas associated with the Transatlantic Slave Trade as part of the formation of an “Atlantic world.” Through processes of forced migration and social displacement, millions of captive and enslaved individuals moved from Africa and throughout the “New World.” These same violent processes that created African diasporic communities also created a restorative mandate to engage the questions of enslaved Africans’ geographic and ethnic origins and how these relate to cultural formations; a mandate pursued by historians, anthropologists and others through their respective analytical means (Blakey 2009a; Walker 2001).

In chapter three, “Anthropology of the Historical African Diaspora,” I explain how historians, archaeologists and biological anthropologists have approached the diaspora concept and related issues of origins and identity over time. Included in this chapter is a discussion of cultural dental modification. As noted above, this practice is typically associated with African
birth in the bioarchaeological literature. Handler and co-workers (1986; Corruccini et al. 1987) argue this position most convincingly based on their study of skeletal lead variation at the Newton Plantation in Barbados and Handler’s (1994) subsequent analysis of runaway ads. Neonatal chemistry, especially as determined by LA-ICP-MS of teeth, may offer new perspectives on this topic.

Attention shifts squarely to the New York African Burial Ground and its analysis in chapter four. The chapter begins with an account of slavery in New York in order to establish the burial ground’s historical origins and unique significance. The African Burial Ground is the largest colonial-era cemetery analyzed to date. I then present key archaeological and skeletal biological findings of the NYABG Project. Through an innovative biocultural and publicly engaged research program, NYABG researchers have generated new knowledge about (1) geographic origins, (2) biocultural transformations, (3) physical quality of life, and (4) modes of “humanity maintenance” for colonial Africans in New York (Blakey 2009). This study continued research began as part of the NYABG Project and its findings are relevant to each of these major research topics.

In chapter five I detail some major sociocultural pathways of lead exposure and attempt to show that poverty in the present – like slavery in the past – increases the likelihood of childhood lead exposure and its ill effects. At its core, this study is about power and how its abuse rendered some people more vulnerable than others to this specific environmental stressor. In this respect, I join a growing number of anthropologists and other health researchers who view “vulnerability,” “disadvantage,” and “risk” as conceptual tools for understanding how power disparities reproduce inequality and patterns of morbidity and mortality (Adler and Stewart 2010; Leatherman 2005; deFur et al. 2007; Panter-Brick and Fuentes 2009; Swedlund
This chapter also includes an overview of skeletal biomarkers of lead exposure and an explanation of why teeth are so valuable for reconstructing early-life environments.

Today’s bioarchaeologists have at their disposal powerful tools for reading human bones and teeth as archives of “lives and life styles” (Larsen 2006). Among them, LA-ICP-MS is a recently developed method allowing for spatially- and time-resolved analysis of elemental and isotopic data (e.g., Dolphin and Goodman 2009; Farell et al. 2013). In chapter six, I describe this technique and how its use is expanding our knowledge of environmental chemical exposure, with a primary focus on nutritional and pollutant studies involving teeth. Following an overview of LA-ICP-MS and some examples of previous anthropological applications, I detail the study methodology: (1) the sampling strategy; (2) sample preparation; (3) LA-ICP-MS measurement; and (4) data processing and analysis. This study is the first quantitative LA-ICP-MS analysis of African diasporic natality and its methodology is applicable to other sites. As such, it is the foundation for future research into the comparative experiences of African- versus American-born individuals and collectives.

I report the results of analysis in two parts. Chapter seven focuses on the mean concentration of lead found in each individual. These data are used primarily to provide an independent test of place of birth and early-life lead exposure. Mean concentrations serve as the basis for statistical comparisons of NYABG groups. In chapter eight I present enamel-lead microspatial distribution patterns and chronological age profiles. These patterns and profiles illustrate graphically the extent, timing and nature of lead exposure for NYABG infants and young children. This chapter illustrates clearly how LA-ICP-MS of incrementally-developing tissues expands the analytical landscape with respect to issues of geographic origin and migration.
Chapter nine outlines the study’s major findings and includes a discussion of their biocultural implications. Drawing upon historical, bioarchaeological and cultural anthropological concepts and frameworks presented in earlier chapters, I interpret enamel-lead data in relation to patterns of natality, dental modification, potential sources of exposure, and possible health consequences for NYABG individuals and groups. Some findings were predicted. Others, particularly around the relationship of natality to cultural dental modification, suggest new and interesting scenarios that require further investigation.

I conclude the dissertation in chapter ten with a summary of the study’s findings in light of the study goals stated above and those of the NYABG Project, and suggestions for an expanded bioarchaeometry of this important site. These were the bones that the builders rejected – twice – and this study is rooted in biocultural anthropology and political struggle. In keeping with the spirit and principles of the NYABG Project, this study is an attempt at a new science of human dignity (Jones et al. 2007). It is my fervent hope that the new histories revealed from within these ancestors’ teeth continue to move us toward a more accurate depiction of our shared and living human past.
CHAPTER 2

BIOARCHAEOLOGY: RECONSTRUCTING LIFE HISTORY AND LIVED EXPERIENCE

Introduction

Human skeletons are reservoirs that record evolutionary and everyday events. One hallmark of biological anthropology has been the development and adoption of new methods providing ever-increasing access to these biohistories. Yet, the framing of human biology and variation is anything but straightforward and, often, contentious. Anthropology is an inherently political science. In this chapter I introduce bioculturalism as a general orientation toward accessing and interpreting the vital data archived within bones and teeth. I discuss its evolving goals and the developmental milestones that have led to a recent, critical re-synthesis of biological and socio-cultural analyses. Emphasis is placed on bioarchaeology and its increasingly relevant perspectives on human-environment interactions. Some of the themes introduced here are elaborated further in chapter five in the context of environmental lead exposure.

Life Stories from Skeletal Remains

Biohistory

The forces that shape human bones and teeth are, at once, evolutionary, cultural and individual. Within a range of developmental plasticity or potential, the diversity of forms, microstructural arrangements and mineral content observed for calcified tissues reveal the evolutionary life history of the species. Their morphologies and chemical make-up also reflect practices and experiences such as migration, diet, poverty and (voluntary and involuntary) body
modification. Sometimes these practices and experiences play out more or less uniformly for human collectives while others unfold in ways unique to individuals. Contemporary bioarchaeologists – with theories and methods developed from paleopathology, skeletal biology and archaeology – seek to retrieve this information from skeletal remains recovered from archaeological contexts (Armelagos 2011).

For my purposes, bioarchaeology is considered part of the larger academic endeavor of biohistory, defined here as an interdisciplinary area of research into biological, social and cultural factors that influence health, morbidity and mortality for a given population (after Rankin-Hill 1997). Within this broad goal, research methods vary and the specific aims of biohistorical research unite but are conceptualized differently across disciplines including history, anthropology and biology. For example, demographic and economic historians use documentary evidence (e.g., anthropometric, military and court records) to infer past health and nutritional conditions (Steckel et al. 2002) while skeletal biohistory remains primarily an anthropological undertaking – even though historians may well find skeletal research techniques and data useful. Overall, Smith (2002: 2) suggests that biology can enrich historical research in two ways:

by offering techniques and methods, a new set of practical tools and approaches for addressing existing historical questions, and second, more controversially, by offering hypotheses derived from biology to interpret existing historical data. In terms of the material to be analysed, we can distinguish between two kinds of data sources: the written records accumulated by literate societies, and the various kinds of human remains, ranging in scale from mass burials to molecules, which can be scrutinized to describe in life the people they represent.

---

2 I use the term **skeletal biohistory** in reference to studies of historic populations such as this one that involve direct analysis of bones and/or teeth. Although much of their research certainly is captured within this broad definition, biological anthropologists do not use the term **biohistory** regularly, perhaps to avoid terminological confusion since their studies of past peoples often take the form of bioarchaeology (Blakey 2001). A few exceptions are studies of African diasporic populations by Larsen et al. (2002) and Rankin-Hill (1997).
History can contribute to a richer understanding of human biology and biological outcomes as well. Biological anthropologists have for some time found historical and ethnohistorical literature to be fertile ground for expanding interpretations of skeletal data (e.g., Blakey 1988; Herring and Swedlund 2002; Rankin-Hill 1997). In 2002 collaboration between economic historian Richard Steckel and skeletal biologist Jerome Rose resulted in the publication of *The Backbone of History*, a study in “macrobioarchaeology.” The contributors to this edited volume bridged methodological divides between history and anthropology in order to establish and compare patterns of health and stress for geographically and temporally diverse historic African American, European American and Native American populations.

More recently, biological anthropologist Alan Swedlund (2010), a pioneer of critical bioculturalism, turned to historical archives as a resource for developing a rich biocultural history of illness, death and coping in New England during the mid-19th and early-20th centuries. These two examples illustrate the potential that disciplinary border-crossing holds for more biologically-informed history and more historically-informed biology.

The historical scale of environmental interactions considered by biological anthropologists can vary greatly, from macroevolutionary processes to more localized variability within and across specific populations. What links much contemporary bioanthropological research is an ecological orientation informed by adaptationist models. Increasingly, these models are framed by life-history theory, which “provides a comparative evolutionary framework for understanding reproductive and developmental strategies, both within and across species” (McDade 2003: 101) as well as energetic and functional trade-offs associated with those strategies (Leonard et al. 2010).

Central to evolutionary biology, life-history theory helps explain how the “evolved design for fitness optimization... generates the species-specific suite of features... that comprise
its distinctive life course” (Worthman and Kuzawa 2005: 96). From this perspective, researchers investigate the evolution and interaction of these features, sometimes called “life-history traits,” which include number, size, and sex of offspring; growth pattern; age and size at maturity; age-, stage- or size-specific reproductive effort; and lifespan (Fabian and Flatt 2012). Biological anthropologists have found a lifespan organizational framework to be quite useful for tracking health trajectories across the various stages of human growth and development, i.e., from the prenatal period through old age and senescence (Bogin 1999; Leidy 1996; Armelagos et al. 2009).

Recently, Roksandic and Armstrong (2011) have suggested that bioarchaeologists should adopt life-history-derived stages instead of the age groups currently in use. They argue that doing so will produce more predictable chronological age information and thereby bridge their research and that of paleodemographers, for a better understanding of health trends in the past. The life-history orientation has yielded powerful insights into menopause (Leidy Sievert 2006), immune development and function (McDade 2003), and the fetal origins of health disparities (Worthman and Kuzawa 2005; Ellison and Jasienska 2009) and various other aspects of human health and primate evolution (Godfrey et al. 2001).

**Bioculture**

Though critical for probing human bio-variability, evolutionist ecological models alone cannot explain its range and complexities. This is the case for past as well as contemporary populations (Marks 2012a). What links biological anthropology to other fields of anthropology and defines its uniqueness is the exploration of evolutionary dynamics alongside social and cultural forces for a more comprehensive understanding of the factors shape biologies both
visibly and “under the skin” (Goodman 2009). In this section, I provide an overview of the historical background, ongoing development and major research domains of contemporary biocultural anthropology, the overarching mission of which is to realize this particular aspect of anthropological holism. Ultimately, I focus on the development and growing influence of a specific line of critical biocultural research – political ecology – as currently realized in bioarchaeology and biological anthropology more broadly.

I begin this brief historical sketch by noting the nature and central role of bioculturalism within anthropology. The term biocultural does not refer to a methodology but, more broadly, to an entire analytical orientation for viewing and investigating the origins and politics of human variability. Thus, bioculturalism entails various foci and approaches that “explicitly recognize the dynamic interactions between humans as biological beings and the social, cultural, and physical environments they inhabit” (Dufour 2006: 1). Biocultural approaches are fundamentally alike, also, in that their implementation often invites certain challenges, which include: (1) defining key constructs (e.g., socioeconomic status and poverty), (2) operationalizing these variables such that they are measurable, replicable and valid ethnographically (or ethnohistorically), and (3) “defining and measuring multiple causal pathways” (Dufour 2006: 1). For those engaged in biocultural research, even defining core anthropological concepts such as culture and environment can be a process more complex than one might think (Armelagos 2003; Dressler 2005).

Despite these challenges, bioculturalism serves as the conceptual glue that links the fields of anthropology and, through critique of naturalized social orders, helps to redefine the discipline’s social relevancy beyond its colonialist roots. Traditionally considered the domain of biological anthropology perspectives, biocultural research is also a rich “borderland” for cultural anthropologists (Gravlee 2013), and particularly for “bridging subdisciplines” such as medical
anthropology and nutritional anthropology (Goodman and Leatherman 1998a). Indeed, intradisciplinary insights are likely to prove increasingly useful as biocultural anthropology expands and evolves new mechanisms for probing meanings and perceptions at the various poles and intersections of social construction and lived experience (e.g., health/wellness and nature/culture) where so much of the human condition is defined.

As Crooks (1996) and Armelagos (2003) observe, human biological research that does not account for relevant cultural and social processes, by definition, is not anthropology. Moreover, inattentiveness to human action or agency and ideology may render research prone to biodeterministic pitfalls that plagued the discipline’s beginnings and, thus, can actually hamper knowledge production (Blakey 1998; Armelagos 2011). The pre-World War II proliferation of studies producing racist depictions of superior and inferior human anatomical, psychological and intellectual types, for example, is a well-documented example of this problem (Blakey 1987, 1998; Barkan 1992; Harding 1993; Armelagos and Goodman 1998).³

Anthropologists continue to wrestle with this issue, now in the form of what might be called genetically-modified reductivism.⁴ In the current “age of genetics and genomics,” understandings and misunderstandings of human diversity and difference rest increasingly on interpretations of DNA sequences. While the tools have changed, for many, the search for race persists; a problem that Swedlund () identifies as “21st-century technology applied to 19th-century biology” (cited in Armelagos and Van Gerven 2003).

³ Marks (2012b) argues that the pitfall of scientific racism persists today in the form of twin crises of (social) morality and (scientific) authority that arise when scientists fail to acknowledge the inherently intertwined and political nature of research into human evolution and diversity. This issue is part of broader debates about the future of biological anthropology as social science, the cohesiveness of the discipline, and the nature of science itself (Smith and Thomas 1998).

⁴ See McKinnon and Silverman [2005] for an excellent collection of critical responses to the resurgence of biodeterminism in the current “age of genetics.”
Figure 2.1: Nineteenth-century critiques of scientific racism by Frederick Douglass (left) and Anténor Firmin (right) foreshadowed contemporary critical biocultural perspectives on human biology, health and race.

Also problematic is the misapplication of evolutionary principles to sociocultural phenomena. Throughout the late nineteenth and early twentieth centuries, as physical anthropologists created racial types, ethnologists and cultural anthropologists busied themselves constructing bounded and static culture groups. This period of rampant scientific ethnocentrism and racism was one in which interactions of biology and culture were viewed as mechanical and, therefore, little understood. By academic convention, races and their individual members “had” their proper, corresponding cultures, which so happened to reflect varying degrees of “civilization.” Thus characterized, different peoples were subject to ethnological ranking via the Eurocentric theory of unilineal cultural evolution (Blakey 1991, 1998; Baker 2010).

Valid alternative explanations of human variation existed. For example, Frederick Douglass (1950 [1854]; Figure 2.1) emphasized the role that social environments and poverty played in shaping bodies and producing the differences in health status and disease profiles
observed across race and class. Indeed, his refutation of craniometric arguments for white supremacy by Samuel Morton and other polygenists seeded the 20th-century biocultural critique of bioreductionism (See Blakey 1998). Forty years later, Haitian anthropologist and statesman Anténor Firmin (2002[1885]; Fluehr-Lobban 200) argued “the equality of human races” within the nascent anthropological establishment. Anthropologists such as Franz Boas (1911), William Montague Cobb (1936) and Ashley Montagu (1942) would follow in the early 20th century, eventually extending critique of bioreductionism to include the very notion of human biological races (see Figure 2.2). Prior to this development, Boas (1911, 1940), the founder of modern American anthropology, challenged the premise that biological race and culture are interdependent. In order uproot entrenched scientific racism, Boas sought to separate biology and culture as topics of scientific inquiry (Boas 1940). Boas collaborated at times with W. E. B. Du Bois, whose anthropological contributions to the study of race and African-American culture and health are often overlooked (Harrison 1992).
For decades, most American anthropologists would follow Boas’s lead and reintegration of biological and cultural analyses occurred slowly. Piece by piece, and aided at times by social forces outside of the academy, anthropologists acquired and articulated the theoretical lenses necessary to see beyond the racial worldview (Smedley 2007). This process began in earnest in the 1960s when some biological anthropologists turned to human ecology in order to frame their research on patterns of cultural and phenotypic variation. Ecology is the study of species-environmental relationships. Researchers often emphasize homeostatic mechanisms amongst these relationships that protect against environmental degradation and provide for the survival of species within a niche or ecosystem.

The 1960s was a period of growing environmentalist concern spurred by the publication of Rachel Carson’s *Silent Spring* (1962) in which she documented the harmful impact of synthetic pesticide use. Awareness and influence of ecology as a means of measuring and addressing such issues spread within the academy and well beyond. Originally associated with biology, ecological perspectives now inform geology, economy, history and various other disciplines within the natural and social sciences. Anthropologists of the time viewed ecological anthropology as the means to understand and bridge biological and behavioral adaptations to diverse environmental conditions (Moran 2006).

The spread of ecological approaches coincided with other events that would shape the future of biocultural anthropology. One such event was Washburn’s (1951) call for a “New Physical Anthropology” based upon hypothesis testing and theory-driven research. Washburn

---

5 Dufour (2006) observes that, as early as 1930, Raymond Pearl’s work – much of which centered on biostatistics – included analysis of “culturally defined variables.” Pearl’s research was atypical in this regard; a precursor to biocultural approaches developed decades later.

6 Throughout this section, I use the terms *physical anthropology* and *physical anthropologists* where appropriate for the time period under discussion. The term *biological anthropology* implies greater contemporary focus on evolutionary dynamics as opposed to physical structures (Fuentes 2012) and its use has largely supplanted that of *physical anthropology* in professional
sought to include physical anthropology within a broader scientific trending away from descriptive reporting and toward explanations of biological phenomena. Influenced by the “modern synthesis” of Darwinian evolution and Mendelian inheritance, he “explicitly emphasized a perspective wherein ‘process and the mechanism of evolutionary change’ would replace the archaic paradigm, the one concerned with ‘sorting the results of evolution,’ and followed by most” (Marks 1995:59). Eventually, this proposed emphasis on dynamic processes (mechanisms) instead of static typologies would provide a basis for exploring social and cultural factors that influenced patterns of human diversity and position biological anthropology as an ecological science (Goodman and Leatherman 1998a).

Research into the cultural-historical ecology of sickle cell by Frank Livingstone (1958) demonstrated the promise of this new approach. In an oft-cited study, Livingstone (1958) established sickle cell trait as a balanced polymorphism maintained by heterozygous advantage in the context of endemic malaria. He found that regions of equatorial Africa with high rates of malaria correlated with areas where millennia-old agricultural practices facilitated an increase in human population size and, with it, likelihood of contact with mosquitos that transmit malaria. Moreover, this pattern of co-variation of endemic malaria with high frequency of sickle cell is observed in parts of southern Europe and Asia as well; an interesting finding because, for decades, this trait had served as a medical marker for Negro/black racial classification (Tapper 1998; Wailoo 2001).

The timing of the study was critical. In the United States context, African Americans do exhibit relatively high rates of sickle cell. Yet, Livingstone demonstrated clearly how culture-environment-gene interactions explain the trait’s geographic distribution in Africa and

---

self-identification. Recently, Fuentes (2012) has called for a “New Biological Anthropology,” i.e., an update and expansion of Washburn’s challenge in light of the “explosion in methodological and theoretical innovations over the past three decades” (4).
elsewhere. His research findings undermined the tautological race argument by uncovering how an actual evolutionary mechanism (natural selection) produced this distribution (Armelagos and Goodman 1998). With the position of bio-racial hardliners already weakened by social condemnation of Nazi Germany’s eugenic atrocities during the Holocaust, Livingstone’s research added momentum to the paradigmatic shift from the task of racial characterization that defined the field of physical anthropology.

The 1960s and 1970s would prove a “golden age” of ecological bioculturalism in which researchers developed a deep understanding of how people adapt physiologically and behaviorally to extreme environmental conditions (Goodman and Leatherman 1998b). Unfortunately, however, Livingstone’s success in unlocking the biocultural history of sickle cell was not easily duplicated for other genetic traits. Nor, initially, did large numbers of physical anthropologists attempt to alter the typological course set by decades of racial anthropometry. Throughout the field, development was heavily weighted toward methodological advancement as researchers developed and adopted sophisticated new techniques for assessing and reconstructing diet, nutrition and health patterns. However, as Armelagos and Van Gerven (2003) observe, most maintained their descriptive foci on human differences with little apparent concern for their underlying mechanisms. Rarely did in-depth bio-profiling generate new questions or conceptual frameworks or lead researchers to question, critically, the nature or causes of human difference (see also Armelagos 2011).

One important exception to this trend was the integration of an epidemiological component within paleopathology, i.e., the skeletal study of past health, migration, morbidity and mortality. The development of “paleoepidemiology” expanded skeletal biology’s scope of purpose far beyond the documentation and description of bone and dental lesions and their proximate (or immediate) causes to incorporate the study of population-wide demographic and
health trends. Ortner (2003) traces the origins of paleoepidemiology to *The Indians of Pecos Pueblo*, in which Earnest Hooton (1930) attempts to link patterns of anatomical change, disease and culture in New Mexico over time (1100 to ca. 1800 AD). Although tethered to the racialist tradition of identifying craniometric and “morphological types,” Hooton’s (1930) use of historical and archaeological research to contextualize skeletal data broke new methodological ground. However, attempts at linking multiple lines of evidence of this sort were limited in number until the 1970s when the influences of the “new” physical anthropology and “processual” archaeology would converge with “anthropologized” ecology and paleopathology to give rise to the distinct subfield of bioarchaeology (Zuckerman and Armelagos 2011).

Biological anthropologists were not yet ready to reintegrate analyses of biology and culture, however. Early attempts often more closely resembled sociobiology than the “new biocultural synthesis” that would emerge toward the end of the century (Goodman and Leatherman 1998). Developed by evolutionary biologists, the overriding concern of sociobiology is in revealing how different behaviors evolved to maximize reproductive fitness. Unfortunately, for the anthropologist, this perspective allows for very little, if any, analysis of the ways that history and social power constrain and promote certain behaviors and thereby shape biological outcomes (Thomas 1998). From social Darwinism to evolutionary psychology, biobehavioral theories tend to explain sometimes important biological differences (e.g., health disparities) as expressions of “good” or “bad” genes and genomes (Gould 1996; Blakey 1998, 1999; Goodman 2000), and presumably related cultural practices and behaviors to biological impulses.

Hyper-functionalist adaptationism would come under fire from anthropologists and even evolutionary biologists (e.g., Levins and Lewontin 1985; Singer 1989; Goodman 1994). In a pointed critique, medical anthropologist Merrill Singer (1996) argued that, too often, studies of human adaptability were insensitive to the biological consequences of history and sociopolitical
marginalization, which exert a form of “unnatural selection” on human diversity. Meanwhile, cultural anthropologists and archaeologists were showing greater interest in issues of social power, human agency and political economy. In diverse settings, they increasingly turned to world systems, dependency/underdevelopment and other critical perspectives in order to explore particular global-local connections associated with the “modern world” as marked by global capitalist expansion (Goodman and Leatherman 1998b; Paynter 2000b; Roseberry 1988, 1998; Wolf 1982).

Importantly, some biological anthropologists agreed with the general assessment that their field had reached the explanatory limits of the adaptationist program – at least as applied independently to the study of human behavior and variation (Crooks 1996; Thomas 1998; Thomas 1997). In their view, creative response was necessary in order to address these criticisms and the larger, related problem of possible disciplinary fission. The Boasian biocultural separation, while necessary, had become a rift between biological and cultural anthropologists.

As more and more biological anthropologists adopted a biomedical orientation, their training and research interests often aligned more closely with those in the natural sciences than with their fellow anthropologists. At the same time, many biological anthropologists found less and less of value in cultural anthropology, especially late 20th-century postmodern and poststructuralist theories perceived as having little material grounding. Moreover, it had become clear to some that anthropologists, like other scholars, have an ethical responsibility to work towards solving and not simply understanding the issues and problems they study. As these problems are often biocultural in nature, their full engagement necessitates common conceptual ground and interests within the discipline (Smith and Thomas 1998). Anthropology had to evolve. At stake was its own identity and relevance as a way of knowing and engaging the human experience.
The response to these challenges came primarily in the form of efforts to expand the scope of bioculturalism by infusing it with critical – mostly political-economic – theory. During the 1980s and 1990s, biological anthropologists adopting ecologically-oriented and historically-informed population approaches systematically pushed the theoretical limits of ecological bioculturalism. Faculty and graduate students associated with University of Massachusetts Amherst led this new wave of conceptual modeling in which biology and culture were investigated as open and integrative systems responsive to social as well as biotic conditions. Their findings generally undermined traditionally progressive narratives of urbanity, industrialization, etc. by revealing how health often is compromised by the integration of local, small-scale economic units into larger sociopolitical entities.

In *Paleopathology at the Origins of Agriculture*, Cohen and Armelagos (1984) and many of their contributors established negative health impact of the Neolithic Revolution and associated population growth. Using the paleoepidemiological approach, they found this to be the case for globally diverse prehistoric populations. By measuring skeletal as well as physiological indicators, Goodman et al. (1988) developed a robust model for studying health and its political-economic contexts in past and contemporary populations. Using the Selyean stress concept and the notion of “multiple stressors,” the authors illustrated the cyclical nature of stress and adaptive responses that can lead to “adaptive disintegration” and, ultimately, to compromised immune and cardiovascular function and other biological insults (see Figure 2.3). Also adopting a political-economic perspective, Leatherman and colleagues (1986) documented household-level impacts of the shift from small-scale agro-pastoralism to wage labor in the Andes (southern Peruvian highlands). These impacts included loss of “flexibility and control” (in the form of predictable labor) for men and greater productive responsibilities for women even as their productive capacities were undermined increasingly by illness.
Figure 2.3: Political economy model of stressor interactions that result in social inequalities and impaired adaptive capacity. From Thomas (1998, Figure 5)

Swedlund and Armelagos’s (1990) edited volume, Disease in Human Populations in Transition: Anthropological and Epidemiological Perspectives highlighted or foreshadowed several approaches to the study of health and health disparities now prevalent or gaining currency in anthropology and public health (e.g., social epidemiology and an emphasis on the diaspora/migration studies). Meanwhile, Blakey (1994) expanded the biocultural domain to include an emphasis on psychophysiology and perceived stress alongside social and economic factors and launched the NYABG Project in which the current study is rooted. The model of engaged bioarchaeology developed by Blakey and colleagues for the NYABG Project (Blakey 2010; Blakey and Rankin-Hill 2009; see chapter 4) would break new theoretical ground in conceptualizing descendant community members as “ethical clients” of anthropology. These were novel contributions to the study of past and contemporary health contexts and conditions.
Political Ecology

The “UMass School” was instrumental in charting a path for biological anthropology that benefited from the extensive knowledge of adaptive responses developed during previous decades while avoiding adaptationist excesses. The goals and an immediate agenda for this project were defined in Goodman and Leatherman’s (1998a) important volume, *Building a New Biocultural Synthesis: Political-Economic Perspectives on Human Biology*. This book contains papers that were first presented at a Wenner-Gren Foundation-sponsored conference convened by the editors for the purpose of exploring the potential and potential directions of this research agenda. As defined by Goodman and Leatherman (1998b: 5), the contemporary biological anthropologist’s “ultimate concern is with understanding the roots of human biological conditions, which are traced to the interaction of political-economic processes and local conditions.” Accordingly,

a bioanthropological political economy seeks to understand how particular local histories shape everyday realities of anthropological subjects, and moreover, how separate communities are connected through larger historical political-economic processes that affect human biologies (Goodman and Leatherman 1998b: 20).

This is a particular vision of bioculturalism; a critical political ecological approach requiring the integration of political-economic, human adaptability, and ecological perspectives. Political ecology involves the study of social and cultural power relations (political economy) centered on human constructions of and interactions with various environments (ecology) (Hvalkof and Escobar 1998). Like biocultural anthropology, political ecology is an approach for addressing multiple sets of concerns and issues. Moreover, political ecology is not limited to any single discipline, but has emerged and matured over the past several decades into an interdisciplinary area of study that spans natural and social sciences. Thus, researchers are able to consider the consequences of human-environment interactions from various perspectives.
and at multiple levels, from the political construction and dichotomization of nature and culture to international and national environmental movements to local toxin exposure (Hvalkof and Escobar 1998; Leatherman and Thomas 2001; Schell 1997). Specifically, Goodman and Leatherman (1998b) suggest that this particular biocultural synthesis will help to clarify:

1) biological variation associated with social relations that form proximate environments by enabling or restricting access to needed resources and labor;

2) sociopolitical and economic links between local and global conditions;

3) history and historical contingency of social change and its biological consequences;

4) human agency in the construction of environments; and

5) the influence of ideology and knowledge (of subject and scientist) on human action including control of resources.

Influenced by the postprocessual critique within historical archaeology (McGuire and Paynter 1991; Paynter 2000a) and its explicit focus on power relations, the critical political ecology described here is concerned with identifying and addressing the ultimate or structural causes and biological consequences of social inequality. As another biocultural pioneer, R. Brooke Thomas (1998: 44), explains,

a complementary political-economy/human adaptability perspective that acknowledges the dialectic between adjustment and exploitation would seem to offer much in addressing a reality where hope and human action are entangled with oppression and marginality. The need to understand this dialectic seems to have intensified in recent decades as political-economic relationships constrain adaptive capacity, and people attempt to circumvent or counter the social conditions that cause them.

From this perspective, instances and patterns of well-being and dysfunction fundamentally reflect the actions of human agents as they attempt to construct, alter and navigate living conditions and life chances for themselves and those for whom they care.

Successfully implementing research of this nature requires a concept of environmental stressors not limited to biotic considerations. Rather, the total environment includes social and historical
processes and conditions of race/racism, class, etc. with which individuals and groups must contend. Likewise, adaptation entails more than favorable biological modification to environmental conditions, however defined, since physiological and genetic adjustments often serve as “backup responses” (Thomas 1998: 51) to behavioral strategies when social support systems fail (Singer 1998). Equally important is an understanding of how and why these strategies and systems break down, and how “disintegration” of adaptive measures leaves its biocultural mark on bodies, bones and teeth. Understanding these marks – their sociopolitical, economic and cultural roots; who is most likely to exhibit them; why and to what biological and social effect – is central to the line of research that Thomas (1998) has termed the “biology of poverty.” Current biocultural research has even begun to move beyond identification of political-economic forces necessary for framing health patterns or trends and is revealing how stressors affect specific physiological pathways (Panter-Brick and Fuentes 2009; Worthman and Costello 2009).

The influence of biocultural and social theory within bioarchaeology has grown considerably since the late 1990s, prompting several recent assessments of “new directions” and research foci for the subdiscipline (Knudson and Stojanowski 2008; Martin and Harrod 2012). According to Agarwal and Glencross (2011), this exciting period marks a third phase of theoretical innovation, the meaning of which is captured in the title of their recent book, Social Bioarchaeology. The “founding and first wave of theoretical engagement” saw widespread adoption of the population approach. The second wave was defined by critical assessment of sampling biases produced by “selective mortality and hidden heterogeneity” in the bioarchaeological record (e.g., Wood et al.’s [1992] “osteological paradox”) and accompanied by
development and adoption of important methodological (e.g., chemical, histological, DNA) techniques.\(^7\)

Zuckerman and Armelagos (2011), in their contributed chapter to *Social Bioarchaeology*, provide an historical overview of the emergence and development of biocultural bioarchaeology. They describe the growing acceptance and use of biocultural approaches as part of a maturation process that is enhancing the scope, social significance and ethical standing of bioarchaeological research. Thus, alongside established foci (e.g., the evolution of human diet; gender-based violence; studies of the African diaspora; and the emergence, ecology and evolution of disease), bioarchaeologists now engage a range of new topics. These include individual health and disease experiences in broad social contexts and relationships of biological and social identities (e.g., via critical theories of sexuality, gender, ethnicity, and disability). Bioarchaeological approaches to embodiment of sociocultural conditions (Joyce 2005; Sofaer 2006; Nystrom 2011) and childhood e.g., (Perry 2005; Halcrow and Tayles 2011) seem especially pertinent to this study, which considers early-life lead exposure in the context of forced migration and culturally-modified teeth as “political artifacts” of social control and, possibly, resistance (Schepurer-Hughes and Lock 1987). Importantly, biocultural approaches also enable (self-) critical appraisal of the ethical dilemmas and implications of bioarchaeological research in the United States and abroad as well as actions necessary to redress unequal power relations between academic and other stakeholders (e.g., Barrett and Blakey 2011; Blakey 2009, 2010; Turner and Andrushko 2011; Larsen and Walker 2004). I discuss the relevance of these particular theoretical developments further in the next two chapters.

To summarize, biocultural anthropology is an approach to the study of human biology across time and place that emphasizes the important influences of cultural, historical and social processes on well-being and health. Critical biocultural approaches in particular serve as correctives to hyper-functionalist interpretations of human variability, the latter of which are not synonymous with an evolutionist approach. Sometimes juxtaposed, biocultural and evolutionist perspectives actually complement one another and both are integral components of anthropological, including bioarchaeological, analysis (Armelagos et al. 2005). Combined, they produce a more nuanced and layered understanding of the structure of human biological variation than is possible via from any single theoretical perspective (Armelagos et al. 2005). Still, one must acknowledge that different analytical orientations offer different “big pictures.” I have tried to illustrate that biocultural approaches for investigating human-environment dynamics and dialectics are best suited for investigating and exposing the biological consequences of social injustice and vulnerability, or biologies of inequality.
CHAPTER 3
ANTHROPOLOGY OF THE HISTORICAL AFRICAN DIASPORA

Introduction

There is no single African diaspora. Various, often complex migrations gave rise to multiple African diasporic formations before, during and since the “long 18th century.” As Singleton (2010: 119) explains, the African diaspora concept “refers to worldwide dispersal of Africans and their descendants usually of the last two millennia, particularly those diasporas emanating from slave trading.” The clear focus of this study is the “classical” or early historic phase of diaspora formation in the Americas (Harris 2001). Africans in early New York represented one of many transatlantic African diasporic communities that overlapped physically and culturally. These connections fostered locally diverse as well as shared “Afro-Atlantic” sensibilities and identities, all within the broader context of an emerging Atlantic world (Dodson 2001; Harrison 2006). This modern, “new” world arose from networks of commercial, cultural and intellectual exchange intensified and forged by global processes of imperialism, colonialism and capitalist expansion from the 15th through the 19th centuries. Enslaved African labor fueled these processes which, in turn, transformed political and economic landscapes and reshaped social relations on four continents (Inikori 2001; Wolf 1982).

In the previous chapter I presented bioculturalism as a theoretical orientation uniquely suited for investigating how NYABG individuals experienced and ultimately embodied diasporic environments. This chapter entails discussion of anthropology’s role in defining the African diaspora as a unit of study. In the next section, I explain how historians and anthropologists engaged the diaspora concept from its early pan-Africanist focus rooted in understanding the consequences of transatlantic slavery and countering racism to current studies that tend to
privilege more recent dispersals and transnational identity formations (Harrison 2006). I describe the prominent role of cultural anthropologists in framing debates over origins and agency and discuss how archaeologists have taken up these debates over the past several decades. I then discuss cultural dental modification as a locus for research into early diasporic migrations and identities in bioarchaeology. I conclude the chapter with a brief survey of the state of African Diaspora Studies and anthropology within this field.

**African Diaspora in History, Theory and Practice**

Human diasporas reflect a range of conditions and activities leading to voluntary and other forms of migration. The term *diaspora* derives from the Greek for “dispersion” or “scattering,” as one does seeds across a field. Hence, its traditional academic usage implies social scattering, dislocation or displacement of a people and tends to emphasize forced migration from a point of geographic origin (Braziel and Mannur 2003). Historians were the first to apply a diasporic approach to the study of Africans and their descendants in the Americas, usually as a means of understanding and countering racial discrimination. Carter G. Woodson (2005 [1933]) and others saw racism as a system supported intellectually by formal “mis-education” through which people of African descent learned their history through the prism of slavery. Therefore, education about a deeper history of African and diasporic connections and contributions was necessary for undoing racist social relations and structures. Frequently, these early adopters referenced Greek and Jewish historical literatures wherein the idea of human diaspora was already established and well defined. They drew parallels between collective experiences of migration, exile and sociopolitical alienation in antiquity and the Americas (Skinner 1993).
George Shepperson is often credited with popularizing the term *African diaspora* in a paper at the 1965 meeting of the International Congress of African Historians in Tanzania. This meeting and that widely-circulated paper, “The African Abroad or the African Diaspora,” marked an important moment in the development of African Diaspora Studies as a mainstream field of study (Butler 2010), although the concept was hardly new. Since the late 19th century, professionally trained academics such as Alexander Crummell, George Washington Williams, W.E.B. Du Bois and Woodson emphasized the need to understand the “African background” of the black experience in the Americas. So, too, did activists and lay historians such as Marcus Garvey, Arturo Schomburg and John Edward Bruce (or “Bruce Grit”). Then, as now, philosophical outlooks and strategies for connecting Africans and African-descendants differed amongst scholars, activists and scholar-activists (Mullings 2009). Bracey et al. (1970) have detailed the various black nationalisms (cultural, economic, religious and so forth) that characterized this period, many of which persist today. Yet, theirs was the collective task of reclaiming “usable pasts” lost to the historical distortions necessitated by chattel slavery and its legacy, racism.

These historians of the African diaspora typically acknowledged and embraced the political dimensions of intellectual work. For them, African diasporic history was a “weapon” in the struggle for racial justice (Cabral 1966). “Vindicationist” scholars reframed historical problems and rewrote black histories in an effort to destabilize entrenched narratives of Negro or black inferiority that supported discriminatory policies and practices of the day (La Roche and Blakey 1997; Reed 1997). A strong revisionist tendency thus links their efforts, as exemplified in seminal works by Du Bois (1970 [1935]) on southern Reconstruction and C.L.R. James (1989 [1938]) on Saint Domingue’s revolutionary transformation into Haiti, the first black republic in the Americas. Taking on the then-dominant Dunning School, Du Bois was able to deconstruct claims that the black enfranchisement and corruption doomed Radical Reconstruction,
warranting “southern redemption.” In a similar vein, James reclaims the Haitian Revolution as the largest and perhaps only completely successful “slave” rebellion and the first revolution of the so-called Third World. Critical insights on racism, colonialism, and class inequality introduced by Du Bois, James and others remain central to diaspora, transnational and postcolonial studies. There are ongoing efforts to canonize these scholars’ neglected anthropological contributions (Harrison 1992; Paynter 1992; Paynter et al. 1994).

If a primary goal of these first- and second-generation scholars of the African diaspora was to challenge white supremacy by promoting “proper” knowledge of human history and equality, what distinguished the research agenda articulated by Shepperson and his colleagues? What were the important points of continuity and departure and how did these influence the growth and development of African diaspora theory? Butler (2010) observes that Shepperson and his colleagues set out to situate diasporic experiences squarely within African history. They did so by enlarging the field’s scope of inquiry to include more explicit focus on contemporary political conditions in Africa. For Shepperson (1968: 153), the goal of African Diaspora Studies was to clarify ‘a series of reactions to coercion, to the imposition of the economic and political rule of alien peoples in Africa, to slavery and imperialism’ (cited in Butler 2010). This definition maintained and expanded upon the field’s traditional focus on slavery in the context of global capitalism. It also signaled at least two theoretical adjustments with long-term implications for a more expansive and dynamic view of “the” African diaspora as both a unit and mode of analysis.

First, this conceptualization of the African diaspora concept was more integrative than previous incarnations. The modified approach of the mid-to-late 20th century attended to the “modernity struggles” of African and Afro-descendant peoples against the mechanisms of global white supremacy. This development marked a crucial step towards bridging substantively the efforts of Americanist and Africanist academics that generally did not reference each other’s
work. Outside of pan-Africanist circles, communication between these groups had been mostly one-sided and utilitarian in nature. Scholars working in the Americas sometimes attempted to establish African contexts and time-depth for diasporic cultural practices and traditions. Africanists saw little use for investigating diasporic dynamics outside the continent. Studies of slavery and imperialism unified Africans and their descendants as a single domain or unit of historical, political-economic and sociocultural analysis.

Secondly, diaspora had evolved from being simply a thing or entity to be known into a way of interpreting why and how certain migrations occur. In referencing “a series of reactions” to slavery and imperialism, Shepperson (1968) highlighted a growing awareness of the variety and complexity of these diaspora-forming processes. Increasingly, study of the African diaspora entailed analysis of its political structuring (i.e., homelands, destinations and branches) as well as underlying structures of meaning associated with diverse “homeland/hostland relationships” and transnational identities (Butler 2010; Falola 2013; Skinner 1993). The study of slavery, particularly American slavery, maintained its central importance but scholars began taking greater pains to detail the institution’s geographic and temporal diversity throughout and beyond the Americas. Many continued to probe agricultural plantation settings of the South where most enslaved Africans lived, labored and died. Some branched out to explore other, lesser-known enslavement experiences. Of particular relevance to this study, during this period Edgar McManus (1966) produced one of the earliest contemporary treatments of slavery in New York. Still others studied historic free black (Litwack 1970) and maroon (Price 1973) communities. Moreover, the field of study extended beyond the Black or Afro-Atlantic framework, as demonstrated by Harris (1971, 1993) who focused attention on the East African and trans-Saharan Slave Trades. Ultimately, merging diasporic and Africanist perspectives meant
acknowledging multiple African diasporas with unique touchstones and internal workings of identity formation.

This Afro-Atlantic framework emerged amid profound social and political changes as Africans and African Americans secured vital human and civil rights. The historians’ meeting in Tanzania took place only a few months after passage of the 1965 Voting Rights Act, a signature legislative achievement of the US Civil Rights Movement. As African Americans and their allies dismantled the legal basis of racial discrimination in the US, they watched with keen interest and pride and offered both material and moral support to Africans waging their own freedom struggles. Diasporic Africans celebrated as Ghanaians attained independence from British rule in 1957, and then as Congo (1960), Kenya (1963) and other countries followed suit in attaining national sovereignty. For many, the reform and revolutionary movements of the “turbulent sixties” served to bolster historical relationships and affinities strained by slavery and the Middle Passage (Bracey et al. 1970; Skinner 1992). As Africa took on new political and social meanings in diasporic settings, important changes followed within the academy. Chief among them, the social movements of the 1960s and 1970s fostered the development of Black or Afro-American Studies. Around this time, archaeological investigation of African diasporic communities emerged as a formal area of study that, over time, would prove important to the maturation of African Diaspora studies (Ogundiran and Falola 2007; Singleton 2010).

This also was a period in which scholars employed new theoretical and methodological tools to reconstruct demographic trends and cultural diversity within and across diasporic

---

8 Exceptions of course abounded. In the United States, some African Americans conformed to ethnocentric views of Africans that permeated society (and vice versa). All were well aware of the racial and cultural baggage that African descent represented in the politics of national reception and belonging. Constructing ancestral ties and collective identity is complex, unending work involving conflicting positions as seen in contemporary debates over the slave castle tourism in Ghana (Osei-Tutu 2007). Yet, successful freedom struggles of this period seemed to lighten this baggage and, to an unprecedented degree, diasporic peoples celebrated their African heritage openly.
communities. Some, like Philip Curtin, relied primarily upon cliometric approaches involving statistical and economic analyses of slave ship manifests and other documentary evidence to assess the numerical scope of transatlantic slavery. Over forty years ago, Curtin (1969) estimated that slavery in the Americas involved the forced migration of some 11 to 13 million enslaved and captive Africans, about 9 to 11 million of which actually made it to the Americas. Occasional updates have generally confirmed his findings. Recently, using archival sources for nearly 35,000 slave ship voyages between the 16th and 19th centuries, Eltis and Richardson (2010) derived that approximately 12.5 million enslaved and captive Africans left western African shores for the Americas. Among other things, Eltis and Richardson’s searchable Voyages Database (www.slavevoyages.org) allows users to reconstruct specific slave ships routes and to extrapolate geographic and temporal trends in the overall transatlantic trade.

Curtin’s (1969) book, *The Atlantic Slave Trade: A Census*, helped integrate Africa into the broader study of the “Atlantic World.” Falola and Roberts (2008) explain that international leaders originally developed this construct in the wake of World War II when the Marshall Plan and the creation of North American Treaty Organization (NATO) led to discussion of an “Atlantic community.” Scholars of colonial British North America appropriated the construct in the 1960s to connote shared senses of place, social identity and political connection reflecting processes of “migration, colonialism, trade, and intellectual exchange that came to dominate the Atlantic region starting in the mid-fifteenth century” (Falola and Roberts 2008: ix). In the colonial Americas, this presence was early and large. Eltis and Richardson (2010) observe that, until circa 1820, perhaps three times as many Africans as Europeans crossed the Atlantic. In recent years anthropologists and historians have employed the terms “Black Atlantic” (Gilroy 1993), “Atlantic Africa” (Ogundiran and Falola 2007) and “Afro-Atlantic” (Yelvington 2006) in reference to both
historic and contemporary diasporic formations. These terms also acknowledge the analytical tension between Atlantic and global or world history paradigms.

Two other key interventions in the evolution of African Diaspora Studies warrant mention here. The first was the development of social history. With its emphasis on “average people, interactions, and institutions,” social history emerged as a means of focusing attention on those forgotten or diminished in historical narratives (Butler 2010); in the words of Eric Wolf (1982), on “people without history.” Yet, like “conventional” history, this approach is sometimes limited by inherent biases of documentary evidence against those without means to produce official narratives. This “impasse” led a previous generation of social historians to cultural history as “a way to examine the life worlds of populations only barely discernable in the archives of states, churches, and commercial enterprises” (Brown 2010: 220). Oral history, folk songs and other sources once off-limit now became valuable tools for exploring complex relationships between beliefs and behaviors (e.g., see Levine 1977).

In the second edition of his influential book, *Global Dimensions of the African Diaspora*, Harris (1993: 3-4) defined the core elements of the African Diaspora as

the global dispersion (voluntary and involuntary) of Africans throughout history; the emergence of a cultural identity abroad based on origin and social conditions; and the psychological or physical return to the homeland, Africa. Thus viewed, the African diaspora assumes the character of a dynamic, continuous, and complex phenomenon stretching across time, geography, class, and gender.

Harris chaired the 1965 session in Tanzania at which George Shepperson spoke and later organized the First African Diaspora Studies Institute at Howard University in 1979. His definition references voluntary migrations that would begin to take center stage in the following decades but retained a primary focus on the homeland-exile dialectic associated with slavery and forced migration. Notable here is Harris’s explicit characterization of diasporic peoples and communities as cultural and not merely physical extensions of Africa, a clear reflection of the
important role of the social and cultural historiographical turns in African Diaspora Studies. At issue by this point are primary questions of cultural identity and agency that anthropologists have wrestled with for many decades.

**Origins, Agency and Artifacts**

The study of cultural transmission from Africa during slavery poses major challenges for scholars of the historical African diaspora in the Americas. Questions of cultural agency and identity have long garnered attention and generated strong debate amongst anthropologists and others as demonstrated perhaps most famously when Melville Herskovits, E. Franklin Frazier and other leading scholars clashed over the issue of “Africanisms” in the early-to-mid 20th century. The terms and intensity of debate have changed over the decades but issues of cultural agency in the African diaspora remain grist for anthropological engagement; often tied to questions of geographic and ethnic ancestry and always responsive to the “racial politics of culture” (Baker 2010; Perry and Paynter 1999).

The preeminent Africanist anthropologist of his generation, Herskovits (1930, 1941) was convinced that Afro-descendants in the Americas maintained direct cultural links to Africa – “survivals” or “retentions” evident across a wide range of behavioral domains including, for example, music, religion, and child-rearing practices. In the American context, Herskovits further developed his mentor Franz Boas’s acculturation thesis, based on the concept of intergroup “culture contact,” to account for processes of culture change. Focusing primarily on black culture in the Caribbean and South America, where he argued cultural survivals were most evident, Herskovits did much to establish “the New World Negro” as a “positive anthropological problem” (Scott 1991). Among others, his ideas were influenced by his work with Zora Neale
Hurston and the writings of Jean Price-Mars and Fernando Ortiz who studied Africanisms in Haiti and Cuba, respectively (Yelvington 2011).

A major critic of the “survivals” paradigm, the sociologist Frazier (1939) saw little value in the study of “Africanisms.” He argued that the disciplines of the Middle Passage and enslavement erased many transatlantic cultural connections. Moreover, he viewed the academic pursuit of Africanisms as somewhat misguided during a period when emphasizing African roots did little to advance blacks socially, but served to confirm suspicions of their racial and cultural inferiority (see Baker 2010). Ironically, Herskovits and others (e.g., Woodson 1936; Turner 1949) saw establishing an African background as necessary for overcoming arguments of diasporic cultural deficiency or what Herskovits (1941) referred to as “the myth of the Negro past.” The black family unit was a focal point of dispute. Where Herskovits saw a great deal of cultural continuity in African and diasporic African family forms, Frazier suggested scholars were better served studying the roles that American slavery, racism and poverty played in eroding positive familial structures and relations. Yelvington (2011) notes that scholarly sparring did not impinge upon their friendship. Frazier actually allowed for linguistic and religious survivals and the two men shared research informants in Central and South America. Eventually, few would seriously question an African cultural presence in the Americas (Walker 2001).


---

9 For an overview of scholarship on the Middle Passage, the literal, tortuous link between Africa and the Americas, see Lovejoy (2008). Early demographically-focused scholars considered questions related to the overall volume of the Transatlantic Slave Trade such as whether or not “tight-packing” ships was an efficient means of maximizing cargo yields and, thus, profits. In contrast to the cliometric approach, some recent studies emphasize the human histories that unfolded on slave ships (e.g., Rediker 2007; Smallwood 2008).
arguments. Although now commonly associated with cultural processes and theory, Price (2010: 56) notes that the concept of creolization – “the process by which people, flora and fauna, ideas, and institutions with roots in the Old World are born in the New where they develop and reproduce themselves” – originated in the field of natural history before “migrating” to linguistics and then anthropology. He identifies a 1928 letter from Jonkeer L. C. van Panhuys to Herskovits as containing the earliest English usage of the term creolization in reference to cultural instead of biological processes. van Panhuys used the term to describe cultural change amongst the Suriname Maroons. The term would not gain significant traction amongst anthropologists until a 1968 conference on linguistic pidginization and creolization at the University of the West Indies, after which it was adopted “as an analytical tool applied to the unusual processes of culture change that first took place in the violent colonial cauldron of the early New World” (Price 2010: 57).

Mintz and Price (1992) argued that the “‘miraculous’ contestational process” of creolization involved “cosmopolitan” Atlantic Creoles assuming primary responsibility for creating culture in the New World. These cultural innovators then transmitted important survival and other life lessons to recently imported African-born individuals. Price (2010: 57-58) explains that the essay’s “clarion call” was historicization and contextualization. To understand general processes of cultural change, he and Mintz raised a series of questions.

“For example, how ‘ethnically’ homogeneous (or heterogeneous” were the enslaved Africans arriving in a particular locality – in other words, to what extent was there a clearly dominant group – and what were the cultural consequences? What were the processes by which these imported Africans became African Americans? How quickly and in what ways did Africans transported to the Americas as slaves, and their American offspring, begin thinking and acting as members of new communities – that is, how rapid was creolization? In what ways did African arrivants choose to – and were they able to – continue particular ways of thinking and of doing things that came from the Old World? What did ‘Africa’ (or its subregions and peoples) mean at different times to African arrivants and their descendants? How did the various demographic profiles and social conditions of New World plantations in particular places and times encourage or inhibit these processes?
Price (2010) reminds his readers that scholars of the African diaspora initially employed the creolization model to explain Afro-Caribbean culture change. Labor demands and demographic trends associated with diverse enslavement regimes would have altered the pace and course of creolization elsewhere. For Price, the analytical challenge of understanding these differences across space and time underscores rather than undermines the value of the creolization concept. As proof, he points to the work of Ira Berlin and other historians who have used the concept to explain how early generations of African Americans “re-created lifeways” that sometimes quickly gave way to “fully formed” cultural institutions. For example, a strong Atlantic Creole cultural influence was established early in Florida. Meanwhile, the intensive tobacco regime of the early Chesapeake required constant importation or “re-Africanization” of the labor force which, in turn, guaranteed considerable inter-African syncretism. By about 1720, however, a greater proportion of enslaved individuals were American-born and they had begun crafting a racial identity and a distinctive African-American culture. In the rice plantations of the Carolina Lowcountry a third pattern of constant African cultural “re-grounding” emerged as the ranks of American-born blacks did not outnumber those of African-born “saltwater slaves” until the middle of the 18th century.¹⁰

*The Birth of African American Culture* was published in book format in 1992, the same year as the first edition of John Thornton’s *Africa and Africans in the Making of the Atlantic World, 1400-1680*. The two books represented divergent approaches to the subject of enslaved African cultural production. Whereas Mintz and Price emphasized the creation of new culture in new lands, Thornton stressed historical and cultural continuity with African ethnicities. By this time, historians had identified seven primary export regions for captive and enslaved Africans:

(1) Senegambia (Senegal and Gambia); (2) Sierra Leone (including Guinea-Bissau, Guinea, Liberia and the Ivory Coast); (3) Gold Coast (Ghana); (4) Bight of Benin (Togo, Benin and southwest Nigeria); (5) Bight of Biafra (southeast Nigeria, Cameroon and Gabon); (6) west Central Africa (Democratic Republic of the Congo and Angola); and (7) Mozambique-Madagascar (southeast Africa, including part of southern Tanzania) (Richardson 1989). Thornton identifies Upper Guinea, Lower Guinea and Angola as three major western African “cultural zones” from which the majority of captive and enslaved Africans were taken. He and other Africanist historians like Michael Gomez (1998, 2006) and Paul Lovejoy (1997) drew upon an increasingly detailed demography of the Transatlantic Slave Trade – importation rates and ratios and destinations – to reconstruct social dynamics in relevant African societies and to track transatlantic migration patterns for specific ethnic groups.

These scholars contend that relatively intact African ethnic groupings sometimes made it to the Americas where they utilized shared backgrounds and traditions to shape local and regional cultural development – religion, cuisine, song, dance, etc. Ethnicity also influenced the nature and outcomes of resistance to enslavement. Gomez begins his 1998 book *Exchanging Our Country Marks* with a narrative account of Denmark Vesey’s planned insurrection in 19th-century South Carolina, noting that it was organized and, ultimately, compromised along African ethnic lines. He goes on to explain the varied regional impacts of groups linked by geography, language and religion in Africa such as Muslims and the Akan and Igbo peoples throughout the colonial and antebellum South. For Gomez, ethnicity is a “reductionist” enterprise; a marker of difference based on contrasting “networks of sociocultural communication.” He describes a slow shift from ethnically-based identities emphasizing intra-African differences and uniqueness to an African American collective identity that emerges as enslaved Africans “learned the significance of race.” Influenced by religious diversity and emerging class divisions, this shift or
“translation” is complete by the early 19th century. A prominent and somewhat unique example of Americanist scholarship in this vein is found in the work of Gwendolyn Midlo Hall (2005) whose meticulous research of ship records, court records and other documents revealed that enslaved Africans from Senegambia and the Bight of Benin contributed heavily to the creation of a distinctive Creole culture in 18th-century Louisiana.

That African ethnicity was as an important consideration for enslaved and enslaver alike is undeniable. Slaver traders often targeted regions in Africa where they could acquire people known to possess knowledge and expertise necessary to meet regionally-specific American labor demands (e.g., see Carney [2001] on the transfer of African rice cultivation technology to South Carolina and Eltis et al. [2007] for a critical review of this “black rice” thesis). As historian Philip Morgan (2006: 53) explains, the Transatlantic Trade was no random, unsystematic business. In general, the slave trade of any African region was heavily centered at one or two places. About 80 percent of all slaves from the Bight of Biafra left from just two outlets, Bonny and Calabar. Ships leaving on a slave voyage would normally trade in only one African region, though occasionally at several locations in that region. Only about one in 10 slave vessels traded at two or more ports, and only one in 20 traded across regional boundaries. ... Similarly, most Trans-Atlantic ships disembarked their migrants at a single port in the Americas. Over 95 percent of slave ships landed all their slaves at one place. And usually one or perhaps two ports in an American territory garnered most arrivals.

As a creolist, however, Morgan finds that innovation rather than the staying power of African ethnicities best explains the general process of cultural production. He warns those who emphasize cultural continuity over discontinuity against projecting contemporary ethnic identifications onto the historical record. Moreover, like Price, he argues that these scholars fail to appreciate ethnicity as an inherently fluid component of identity, a condition intensified by the violence and social upheavals that accompanied forced migration on both sides of the Atlantic. Price (2010) in particular is skeptical of the “genealogical imperative” to seek out an
“authentic past” rooted in Africa, a practice he attributes to the influence of Herskovits and cultural nationalist identity politics within African Diaspora Studies.11

For their part, “Afrogenic” or “Africa-centric” scholars counter that creolists underestimate the cultural authority that Africa held for enslaved Africans (Walker 2001), and in so doing diminish the mental and intellectual agency of enslaved Africans. They view their perspective as necessary (1) for realizing Africa and Africans as more than backdrops in the study of her diasporas and (2) for expanding the study of historic African diasporic peoples beyond that of slavery and its consequences. Certainly chattel slavery engendered extreme conditions for cultural production and reproduction. Nonetheless, these scholars contend that Africa loomed large in real-time; in the collective thoughts, actions and imaginations of those who faced the difficult task of forging new community and culture. Prior to the Middle Passage, some of these individuals were enslaved in Africa and some were captives perhaps months or only weeks removed from freedom. Others knew only life in the Americas. Whatever their particular and diverse circumstances, the counterargument here is that diasporic Africans drew heavily upon specific knowledge and traditions from Africa as crucial and foundational reference points for understanding and influencing their current and future fates (Brown 2010).

Yet, ultimately, these perspectives do not so much compete as they emphasize different aspects of enslaved African agency in the overarching process of cultural identity reproduction. Creolists acknowledge the importance of probing African and American geo-cultural connections and Afrogenists recognize that demographic trends alone did not dictate cultural histories. Both cultural tenacity and creolization were likely operative in the production of diasporic cultural identities.

---

11 Ironically, Herskovits argued against the politicization of scholarship. However, many black scholars found solidarity and inspiration in his vision of diasporic cultural continuity.
How then to bridge these frameworks? Price (2010) suggests current ethnography might indicate common theoretical ground. As one example, he points to recent work by J. Lorand Matory on Yoruba identity in Nigeria and Brazil. Matory (2005, 2006) illustrates how individuals reproduce a particular form of black consciousness and distinctiveness through “live dialogue” and strategic deployment of traditions through transatlantic channels (Matory 2006). Although perceived as “authentically” African, these traditions are more accurately transnational in origin. One interesting and important implication of Matory’s study is that, even for those enslaved, transatlantic communication of culture would have been dynamic; a perpetual dialogue or conversation rather than a one-time, one-way transmission of ideas and behaviors from a static African background or past.\(^{12}\) Hence, as Brown (2009: 1245) suggests, the “meaning of the category ‘African’ was not merely a reflection of cultural tenacity but the consequence of repeated acts of political imagination.” Retrieving these acts and figuring out how and why these meanings shifted and overlapped locally and regionally seems a task for creolist and Afrogenic scholars alike.

Ethnography of course can be a powerful tool for developing emic insights. However, problems associated with the use of ethnographic sources for reconstructing past African lifeways – e.g., conflation of indigenous with later colonial political constructions – are well documented (e.g., DeCorse 2001; Stahl 2007). Fortunately, there are means available to anthropologists for investigating historical matters of identity and agency directly, i.e., through the material remains of past actions. Here, I briefly sketch key issues and themes in the

---

\(^{12}\) Matory (2006: 163) develops his dialogue metaphor as a critique of Paul Gilroy (1993) and others’ use of “collective memory,” a conceptual tool that has produced “several rich discussions,” but overall “hides rather than highlights the unending struggle over the meaning and usage of gestures, monuments, words and memories” that cultural reproduction entails. For exceptions, see David Scott (1991) on tradition as the theoretical bridge between memory and tradition and Fabre and O’Meally’s (1994) edited volume, *History and Memory in African-American Culture*. 
emergence, development and current practice of Africa diaspora archaeology. My discussion follows that of Theresa Singleton (2010: 128), in which she summarizes the field in the following manner.

Analyses of archaeological materials recovered from the places where Africans and their descendants of diasporas lived, worked, sought refuge, or died provide information on their materials world – housing, use of space, personal and household items, craft production, culinary practices, and so forth. Careful study of these material components of everyday life permits archaeologists to infer about nonmaterial aspects of diasporic peoples’ lives, including their agency, group formations, survival strategies, religious beliefs, cultural practices, power struggles, and interactions with other peoples.

Archaeological findings may “be complementary and interdependent or contradictory and independent” with respect to historical accounts. Whether by documenting “striking” homogeneity or “unexpected variation, as autonomous lines of evidence or “cables of inference,” archaeology has deepened understandings of past lifeways, real and imagined (Wylie 1993).

According to Singleton (2010: 128), archaeologists use the term *African diaspora* “primarily as a label for this research and not as an analytical, conceptual, or methodological tool as in some other disciplines to investigate experiences of displacement, trace specific groups from the homeland to the new settings, or compare linkages with other groups of the African diaspora.” Why does archaeological development of the African diaspora concept lag behind that observed for other fields? Late adoption of the diaspora concept probably explains some uncertainty and ambiguity around its use. Like other scholars, archaeologists studied Afro-descendant peoples for decades without explicitly diasporic frameworks. Indeed, the term *diaspora* does not appear in the archaeological literature until the 1990s (e.g., Singleton and Bograd 1995; La Roche and Blakey 1997; Orser 1998). Also, there is the issue of the field’s broad scope, which spans five continents and over 2,000 years of human migration. Given this breadth, Singleton (2010: 127) suggests that “[p]erhaps the best way to tie the disparate time
periods and regions of African diasporas together is through theoretical and methodological approaches related to common themes: labor, everyday life, resistance, master/slave relations, identify formation, religious practices, and so forth.” A third and likely major consideration (for anthropology as a whole) is limited input from African Diasporan scholars and subjects whose often-critical and sometimes “excavated” perspectives are beginning to exert influence from the discipline’s margins (Battle-Baptiste 2011; Harrison and Harrison 1999; Harrison 2008; LaRoche and Blakey 1997; Blakey 2001, 2009; Patterson 2013).

In response to these limitations, some scholars have undertaken to develop an integrative African/African diasporic research program. For example, inspired by early pan-Africanist scholarship and Paul Lovejoy’s “revisionist” or “continuous historical experience” thesis, Ogundiran and Falola (2007: 6) propose an archaeology premised on the assumption that “African history is incomplete without the history of its diaspora in the Americas, and that African history holds the key to the comprehension of the diaspora.” For African diaspora archaeology, this project holds great promise. Africanist perspectives provide important context as they can help to clarify cultural formations potentially misinterpreted through “generalized, ahistorical and presentist” misunderstandings of African practices (Ogundiran and Falola 2007; DeCorse 1999; Kelly 2001). Conversely, theory generated by Americanist archaeologists may prove useful for the study of internal diasporas in Africa (Haviser and MacDonald 2006).

Until recently, attempts to bridge African and African diasporic archaeologies have been hampered by the lack of a unifying research agenda and framework. In Africa, archaeologists initially explored the “origins of specific states, development of trade and ethnic group relationships, continuities and changes of indigenous African economic and social institutions, and other topics that could foster national pride for newly independent African nations” (Singleton 2010: 121). In the United States, African American archaeology outside of plantation
settings arose in response to the aforementioned socio-political and intellectual movements of the 1960s and 1970s. Singleton notes that black activists promoted archaeological studies as a method of documenting and preserving historic buildings and towns endangered by development and gentrification. Elsewhere in the Americas, African diaspora archaeology emerged and developed unevenly. For example, the tradition of plantation archaeology originated relatively early, by the 1950s, in Cuba where histories of slavery and resistance have long been recognized. Studies of Afro-descendants are less common in Argentina and other places where they have greater potential to unsettle normative myths of national whiteness.

By the 1980s some Africanist archaeologists had begun investigating landscapes associated with the Transatlantic Slave Trade (e.g., Posnansky and DeCorse 1986). This was a crucial initial step toward the development of an Afro-Atlantic archaeology for which Singleton (2010) identifies the major themes. On the African side, current archaeological research focuses on: (1) the rise of towns and polities whose emergence or growth are tied to transatlantic trade such as Elmina and Dahomey; (2) changes in trade, production and consumption of certain commodities (e.g., iron and pottery); and (3) the demographic and landscape effects of slave raiding and warfare (e.g., site abandonment, depopulation, or the presence of fortified settlements). Major research themes in the Americas include: (1) plantation slavery and the various dimensions of enslaved African community life and identity (e.g., daily life, family formation, gender, use of domestic space, religious beliefs, etc.), particularly within the United States; (2) runaways or Maroon communities, especially outside the United States in countries such as Brazil, Cuba, Jamaica and Suriname; (3) free blacks (who gained freedom legally during the era of slavery); and (4) post-emancipation African-American communities in varied settings (e.g., black towns, tenant labor plantations, and western frontier settlements).
According to Singleton (2010: 127), archaeologists exploring these themes on both sides of the Atlantic are contributing to “a post-colonial discourse that allows us to reevaluate colonialism and gain insights into the lives of those who suffered from it.” Just as the physical life stresses of enslavement represent a distinct dimension of the biology of poverty, African diasporic landscapes contain important insights concerning the ways in which historical inequalities of race, gender and class shaped the modern Atlantic world (Paynter and McGuire 1991; Paynter 2000b; Orser 2001; Battle-Baptiste 2011; Epperson 1999). From this perspective, African diaspora archaeology has a unique and important role in the larger, critical project of “decolonizing” or “reworking” anthropology (Harrison 1991, 2008); a project rooted in earlier attempts to “reinvent” the discipline beyond its colonial and imperial origins and legacy (e.g., Willis 1972).  

But critical insights do not result automatically from the study of subaltern subjects. Also needed is historically sensitive theory linking identity and agency under conditions of economic and political domination or exploitation. Warren Perry and Robert Paynter (1999) make this point in their enlightening commentary on ethnicity and agency in African-American archaeology. They illustrate that theoretical limitations sometimes stem from methodological entrapment into addressing “nonproblems” such as the search for cultural agency in African survivals. In the United States an early focus on African-American ethnicity gave way to explicit

---

13 For Harrison (2008), success in reworking of contemporary anthropology into a primarily critical endeavor rests on achieving nine interrelated objectives and outcomes: (1) reconstructing or “rehistoricizing” understandings of relationships between researchers and those researched; (2) reassessing what constitutes and who produces valid theory and theorizing; (3) rethinking the possibilities and implications of intra- and interdisciplinarity; (4) pursuit of a socially responsible ethics and politics of ethnography; (5) mapping of the “mediated connections” between the local and supralocal sphere; (6) interrogating the organization and practice of academic and nonacademic anthropology in the United States and abroad; (7) further democratization of anthropology as an intellectual community; (8); mobilizing knowledge and professional resources for democratic engagements that link academic pursuits and public interests; and (9) decentering Western dominance by leveling the “grossly asymmetrical” power relationships between Northern and Southern anthropologies.
consideration of identity over the past several decades. The early studies of ethnicity were problematic on several fronts. For example, researchers often were hampered by notions of ethnicity that were wither “primordial” and essentialist (i.e., either equated to or conflated with biological race) or too fluid (i.e., insufficiently attentive to matters of power) for investigating slavery in (mostly) plantation settings. Frequently, claims of African ethnic heritage and African-American cultural agency were subject to the litmus test of Africanisms.

Perry and Paynter (1999) make the case that debates over Africanisms fundamentally are not about establishing or contesting an African cultural presence in the post-Columbian Americas. Rather, they remind their peers that debates over survivals, retentions, transformations, etc. are part of a long and torturous discourse about the nature and meaning – the “character” – of that presence in American culture. It is precisely for this reason that these debates persist and resonate so profoundly within and outside the academy. From this perspective, questions about cultural continuity and change take on new meanings. Questions about cultural “retentions” or transformations remain relevant, but the primary focus more interestingly becomes that of why people, perhaps at great risk, did or did not perpetuate old practices in new environments. These are questions of motivation and meaning. Specifically, as Brown (2009) observes, the point is to understand how enslaved people forged meaning for their lives and those of future generations by attempting to impose cultural order onto social chaos.

Yet, identifying agency can be tricky. Its interpretation in the historical African diaspora presents the peculiar challenge of seeing “slavery as a condition…[while] viewing enslavement as a predicament, in which enslaved Africans and their descendants never ceased to pursue a politics of belonging, mourning, accounting , and regeneration” (Brown 2009: 1248). Hence, Perry and Paynter (1999) argue the need to interpret artifacts as potentially “multivalent”
expressions possibly reflecting multiple players and positions of power in varying contexts of racial slavery. Assuming and foregrounding agency frees one to pursue cultural logics rather than cultural provenience as an end in itself. In this way, archaeologists are better able to assess processes of structural power and dynamic ethnogenic and racial identity formations. Furthermore, as these processes and formations leave marks on bodies as well as landscapes, insights into the social origins and multivalent nature of artifacts have relevance beyond archaeology. Indeed, some bioarchaeologists have begun to conceptualize skeletal remains as a unique class of material culture (Sofaer 2006).

**Origins, Agency and Teeth**

In this section I present dental modification as a cultural practice through which bioarchaeologists also engage with complex interactions of origins, identity and agency. As discussed in chapter two, current biocultural theory addresses the question of how people mediate – and their bodies manifest – such interactions. In chapter nine I present a new biocultural analysis of dental modification amongst NYABG individuals based on findings of this study. Here, my goal is to provide an overview of this practice, which bioarchaeologists have come to accept as a means of distinguishing natal or “saltwater” Africans from those who were American- or “country-born.” For a comprehensive discussion, see Jones (2004) and Goodman et al. (2009).

Non-therapeutic alteration of the dentition toward a cultural ideal is an ancient and widespread practice (Milner and Larsen 1991). This practice is one of the more obvious ways that humans assert their likeness or distinctiveness to other animals, including other humans. As
Figure 3.1: Cultural dental modification appears amongst historic populations from all parts of the world. These permanent central upper incisors from a Tamil man of uncertain have been sharpened and incised to create labial grooves. From Gonzalez et al. (2010; Figure 3)

such, important similarities and variations exist with respect to associated methods, rationales and outcomes. For example, there are no reported cases of culturally-modified deciduous teeth. Modification is apparently restricted to the permanent dentition and, usually, to the most visibly prominent maxillary incisors and canines. Occasionally included are the anterior mandibular teeth and the premolars. Chipping, filing and staining are the most commonly reported methods for modifying teeth (Singer 1953; Gould et al. 1984; Alt and Pichler 1998; Milner and Larsen 1991; Ikehara-Quebral and Douglas 1997). Other methods include labial incising, inlaying of precious metals, and tooth extraction (van Rippen 1918; Gould et al. 1984). The most extensive bioarchaeological studies of culturally-modified teeth (CMT) have been conducted for pre-Hispanic South America where some of the most extensive modification patterns are found (Williams and White 2006; Mower 1999).
Modification styles and patterns often vary regionally and across ethnic and cultural groups. For example, Figure 3.1 is a photograph of sharpened incisors with labial grooves from a pre-19th-century Tamil man from India where reports of CMT are scarce (González et al. 2010). This modification differs significantly from the two examples of horizontal filing resulting in furrowed incisors seen in Figure 3.2. These teeth are from a Viking Age (800-1050 AD) archaeological site located in Europe (present-day Sweden) where reported cases of CMT are also rare. The Viking teeth also demonstrate a regular finding in studies of CMT, i.e., of style variation within populations. The reason for the differing depths and numbers of the furrows is unknown but Arcini (2005) suggests they may correspond to individual tolerance for pain or to differential levels of social status or achievement.

Interpretations of dental modification in the African diaspora quite naturally rest upon knowledge of African practices. Finucane et al. (2008) report what they believe to be the
Figure 3.3: African dental modification. These are the CMT of a “liberated” African excavated at St. Helena Island in the South Atlantic. The notches of the permanent upper central incisors were produced by filing. From Pearson et al. (2011, Figure 4.12)

region’s earliest finding of CMT. Four individuals with maxillary filing were recovered from a Late Stone Age (ca. 4500-4200 BP) site in contemporary eastern Mali. However, the authors note a general dearth of osteological data that complicates attempts to accurately assess the antiquity and prevalence of intentional dental modification. Unfortunately, skeletal preservation conditions are poor in tropical western Africa where most enslaved and captive individuals destined for the Americas originated.

An example of African dental modification is seen in Figure 3.3. This maxilla is from 1 of 325 articulated skeletal remains (and a “considerable volume” of disarticulated remains) unearthed during the recent excavation of a “Liberated African” graveyard on St. Helena Island in the South Atlantic (Pearson et al. 2011). British authorities used the island as a depot location for receiving and treating “Liberated” or “recaptive” Africans taken from slave ships intercepted after the abolition of transatlantic slavery in 1807. Interestingly, of 303 individuals with
assessable dentitions, 115 individuals (38%) had CMT; a high percentage relative to that found at the NYABG and in other diasporic settings.

While the skeletal record may be limited, there is ample ethnohistorical evidence of dental modification in African societies impacted by the Transatlantic Slave Trade. European travel literature offers some explanations as to the major motivations and methods for transforming teeth (e.g., Almada 1984 [1594]; Jones 1983). Later ethnography may also yield important details concerning the circumstances of modification. As in other parts of the world, frequently cited reasons for having one’s teeth “prepared” include ethnic affiliation, aesthetic enhancement, and age-related and other (e.g., occupational or clandestine societal) initiation rites (van Rippen 1918). Typically, dental modification in West Africa was conducted on adolescents or adults by a skilled individual such as a blacksmith or carpenter and involved chipping and/or filing the teeth with iron implements or sharpened stones (Whitridge 1913; Bohannan and Bohannan 1953; Singer 1953). In most instances, the practice does not appear to have been sex-linked, although its particulars might. For example, amongst the Igbo it once was customary that females could not bear children and males could not achieve their first titles before having teeth their filed (Whitridge 1913).

Ethnohistorical accounts also reveal some of the patterns one might have encountered during this period. For example, two commonly observed patterns were incisors filed to points and mesial filing or “notching” between the maxillary central incisors that resulted in an “inverted V” shape (Singer 1953; Gould et al. 1984; Jones 1992). Although ethnic affiliation clearly was one of the motivating factors behind dental modification, the ethnohistorical record does not reveal how patterns varied along ethnic lines. Gould et al.’s (1984) survey suggests considerable overlap occurred in the distribution of patterns across western Africa (see Table 3.1). One implication of this observation is a general inability to link individuals to specific
Table 3.1: African dental modification patterns and contemporary national boundaries. From Gould et al. (1984; Table 1, Figure 5)

<table>
<thead>
<tr>
<th>Dental Modification Patterns</th>
<th>Regions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mesial filing of maxillary central incisors</td>
<td>Guinea, Togo, Angola, Democratic Republic of the Congo, Uganda, Kenya and Tanzania</td>
</tr>
<tr>
<td>Mesial and distal filing of maxillary central incisors</td>
<td>Guinea, Central African Republic, Democratic Republic of the Congo, Angola</td>
</tr>
<tr>
<td>Filing six maxillary anterior teeth to pointed shape</td>
<td>Democratic Republic of the Congo, Zimbabwe</td>
</tr>
<tr>
<td>Filing four maxillary and four mandibular incisors to pointed shape</td>
<td>Guinea, Cameroon, Republic of the Congo</td>
</tr>
<tr>
<td>Horizontally filing maxillary central incisors</td>
<td>Guinea, Democratic Republic of the Congo</td>
</tr>
<tr>
<td>Central notching of incisors</td>
<td>Sierra Leone</td>
</tr>
<tr>
<td>Serrating incisors</td>
<td>Mozambique</td>
</tr>
<tr>
<td>Mesial triangular notching of the gingival one-third of central incisors</td>
<td>Republic of the Congo, Sudan</td>
</tr>
<tr>
<td>Concave filing of maxillary incisors with convex filing of mandibular incisors</td>
<td>Tanzania, Mozambique</td>
</tr>
<tr>
<td>Extracting maxillary central incisors</td>
<td>Zambia</td>
</tr>
<tr>
<td>Extracting mandibular central incisors</td>
<td>Uganda, Kenya</td>
</tr>
<tr>
<td>Extracting primary mandibular canines</td>
<td>Democratic Republic of the Congo, Sudan, Uganda</td>
</tr>
<tr>
<td>Extracting four maxillary incisors</td>
<td>South Africa</td>
</tr>
<tr>
<td>Extracting four mandibular incisors</td>
<td>Sudan</td>
</tr>
<tr>
<td>Extracting four maxillary and four mandibular incisors</td>
<td>Democratic Republic of the Congo, Uganda</td>
</tr>
<tr>
<td>Extracting single lateral incisor (note: maxillary in diagram)</td>
<td>South Africa</td>
</tr>
<tr>
<td>Artificial prognathism with facially flared maxillary central incisors</td>
<td>Senegal, Kenya</td>
</tr>
<tr>
<td>Red-staining teeth (from chewing “mkua” fruit or guru [kola] nuts)</td>
<td>Morocco, Niger</td>
</tr>
</tbody>
</table>
African locations via observed modification patterns. In other words, one cannot infer regionally specific or ethnic origins solely from CMT.

At a larger geographic scale, however, dental modification serves as a marker of African versus American birth. The presence of CMT suggests not only African ancestral or cultural ties, but also physical African origin. Stewart and Groome (1968) were perhaps the first to make this connection in a study of skeletal remains from Grenada and St. Croix (while entertaining the possibility that first-generation American-born individuals engaged in the practice). However, this position is most often associated with Jerome Handler and colleagues as first set forth in their study of the 17th- to 19th-century Newton Plantation in Barbados. Through archaeological, paleopathological, and chemical (bone-lead) analyses, Handler et al. (1986; Corruccini et al. 1987) determined that enslaved Barbadians with CMT were African-born. Based on these findings and a subsequent study of runaway ads from the American South, Handler (1994) reasons that enslaved Africans would have foregone dental modification as these indelible markings made them easily identifiable and, thus, undermined any attempts at escape.

Subsequent studies employing elemental and isotopic analyses of tooth enamel for individuals with CMT support the hypothesis that CMT indicate African birth (e.g., Price et al. 2006, 2012; Schroeder et al. 2012, 2013; Prevedorou et al. 2010). In a sense, then, dental modification, although likely traced to Africa, functions analytically more or less as the opposite of Africanism. It is treated as a practice that could not persist in the early Americas because it was “maladaptive” in the context of slavery. Interestingly, the only possible exception to date comes from the NYABG (Goodman et al. 2009). I revisit this issue in the next chapter on the NYABG Project, and the Newton Plantation study in chapter five, as part of a discussion of early African diasporic environmental lead exposure.
In concluding this chapter, I ask: what is the current state of African Diaspora Studies and of the anthropology of the historical African diaspora? Clearly, both have come a long way since the original Africanism debates of the mid-20th century. More integrative studies of the Afro-Atlantic and more nuanced analyses of diasporic identities have only bolstered the African diaspora’s academic appeal. A steady stream of scholarship attests to this. Yet, as always, challenges remain.

The past half-century has seen African Diaspora Studies evolve into a specialized field involving a range of humanistic and scientific approaches from various disciplines. Yet, questions, problems and solutions demand interdisciplinary attention. As a result, although the study of diasporic Africans is an academic field currently enjoying rapid growth, it does so at risk of developing with too little programmatic interdisciplinary collaboration. Olaniyan and Sweet (2010) address this issue in their co-edited book The African Diaspora and the Disciplines. This important volume brings together perspectives from disciplines with longstanding focus on African diasporic issues such as history, anthropology and ethnomusicology. Also included are perspectives from relatively new fields such as Cultural Studies and others logically connected to, but less known for their theoretical contributions like theater studies, philosophy and geography. Underscoring anthropology’s longstanding central importance to the field, the first section of the book, “Histories,” includes three chapters by anthropologists. Here, one will find the essays on creolization by Price and African diaspora archaeology by Singleton that inform this chapter. The third chapter by Fatimah Jackson and Latifah Borgelin is a discussion of “How Genetics Can Provide Detail to the Transatlantic African Diaspora.”

Olaniyan and Sweet (2010) offer four suggestion that, together, comprise a general framework for those invested in the interdisciplinary study of African diaspora peoples. First, they suggest that Africa as a homeland, whether “real or imagined,” should remain the
intellectual starting point of African Diaspora Studies. Perhaps once intuitive, such a position can no longer be assumed as one considers the increasing focus on transnational movements and internal diasporas that may not originate with or involve African locales. Secondly, because the African diaspora is “mutually constitutive of other political formations such as race, class, gender, sexuality” (4), it must be studied from an overlapping, comparative perspective. To these “axes of inequality” (Farmer 1999) one might add social age and disability, topics that bioarchaeologists now engage.

Thirdly, they call for a renewed emphasis on an African diaspora intellectual genealogy, with particular attention to the pioneering works of figures such as W.E.B. Du Bois, C.L.R. James, Frantz Fanon and Walter Rodney. This position echoes that of Faye Harrison (2008) and others (e.g., Baker 1998; Blakey 2001; Drake 1980, 1990; Watkins 2007) who have critiqued the omission of African diasporic scholars and their critical perspectives from anthropological cannon. Lastly, noting that “sources are inscribed in some of the most unexpected places – objects, memories, shrines, and even bodies,” Olaniyan and Sweet (2010: 5) urge scholars to remain “open to the multitude of nondiscursive expressions that constitute ‘sources’ for the study of the African diaspora.” Indeed, scholars of the African diaspora should take notice of the growing literature on embodiment of lived experiences. Such experiences range from performances of cultural knowledge (e.g., through dance [Daniel 2001]) to the physiological mediation and skeletal incorporation of environments and inequality (Krieger 2003, 2005; Nystrom 2011).

Finally, a recent shift in focus away from themes of trauma, exile, alienation and oppression has prompted anthropologist Faye Harrison (2006: 384-385) to ask: “Is this broadened mapping a consequence of diaspora studies being mainstreamed and losing the critical conceptual and political edge they once had? Or is it simply a reflection of how
heterogeneous the field of study is, with competing definitions and models of what constitutes a diaspora?" For Harrison, there is danger in conceptualizing diaspora too broadly and she cautions against uncritical application of the term to all forms of transnational migration. Price (2010), also, is troubled by some current uses of the creolization concept, especially outside of the Caribbean context. Specifically, he warns that scholars will misemploy this concept unless due consideration is given to location-specific political dimensions of culture change. These observations serve as reminders that African diaspora, like all analytical perspectives and frameworks, evolve and are open to redefinition – and that their reformulations are, likewise, subject to critique.

Popular in some academic circles since the 1960s, but in use for much longer, the African diaspora framework continues to provide fresh insights into cultural, national and transnational connections and conditions of Africans “at home and abroad.” Anthropologists have long been and remain central to the study of Afro-Atlantic peoples. In this chapter I have presented some of the main concepts and debates through which they do so, concentrating primarily on questions of origins, agency and identity. In the following chapter, I discuss how NYABG Project researchers addressed these issues by integrating diasporic and biocultural analysis, in ways that broke new ethical, theoretical and methodological ground in bioarchaeology.
CHAPTER 4
THE NEW YORK AFRICAN BURIAL GROUND

Introduction

The 17th- and 18th-century African Burial Ground was quite possibly New York's first African-American institution (Medford and Brown 2009). Estimates put the number of people buried in this approximately 6-acre Lower Manhattan cemetery as high as 15,000. Most of these would have been enslaved individuals although, quite possibly, free Africans and individuals from outside the African community were buried there as well. Virtually lost to public memory, the burial ground’s partial excavation in the early 1990s provided the opportunity to reconstruct these individuals’ lives and deaths through bioarchaeological analysis. The biocultural and interdisciplinary study of this site and population was the purpose of the NYABG Project.

In this chapter, I draw upon the work of NYABG Project researchers to discuss the burial ground’s historical and cultural significance and the material evidence of African life in early New York. The bulk of this chapter centers on the city’s history of slavery from the Dutch colonial period through the mid-19th century. Using the work of project historians and others, I reconstruct living conditions for those buried at the site. I then explain the project’s emergence as an early example of “social bioarchaeology,” outlining its major goals and some of its key research findings. I focus primarily on bioarchaeological findings of population and individual origins, as determined from analyses of mortuary practices, genetics and especially dental chemistry that preceded and informed this study.

Slavery in New York and the African Burial Ground
The first documented reference to the African Burial Ground appears in 1712. In “Proposals for Erecting a School, Library and Chapel at New York,” Chaplain John Sharpe (1881, cited in Howson et al. 2009) of Her Majesty’s Forces in the Province of New York observes that African New Yorkers conducted burials outside the city in the Common. A standard feature of European colonial settlements, the Common was an area where townspeople could freely access water, timber, and other collectively held resources and attend to tasks that required open space and distance from the city. Colonial New Yorkers used the Common – located just north of the city near the Collect (Kalkhook or Fresh Water) Pond – for grazing, livestock slaughtering and beer brewing among and other activities. As NYABG researchers observe, the Common was the “locus of the unwanted.” It was here that Africans, prisoners and the poor were buried and that public executions took place. For example, Africans allegedly involved in the 1741 “Negro Plot” or “conspiracy” to burn down the city were executed at the Common (Howson et al. 2009). The African Burial Ground was contained mostly within Calk Hook Farm and the Van Borsum patent, privately owned land located just north of the Common’s northern edge, but may have extended into the Common as well.

Sharpe’s (1881) document and 18th-century maps help to locate the burial ground spatially. Yet, the date of its earliest use is uncertain. Unfortunately, archaeological analysis has not resolved this matter as the excavated portion may not contain the earliest burials. NYABG researchers suggest a probable 17th-century origin. In 1697 the Anglican Trinity Church banned the burial of Africans, Jews and Catholics in its cemetery, possibly necessitating the establishment of an autonomous burial site for the growing African population prior to the turn of the 18th century (Medford and Brown 2009). Alternatively, the burial ground may have originated earlier, around the middle of the 17th century, on land owned by free and “half-free” Africans. Located at the northern edge of the Collect Pond, this land fell outside the commercial
designs of Dutch colonial administrators and would have afforded a degree of privacy for cultural expressions eventually outlawed within city limits (Howson et al. 2009a). Early use of the burial ground probably coincided with that of public and congregational cemeteries within the city as well as burial plots on farms where Africans labored (Medford and Brown, 2009).

Whether originating in the mid-17th or early 18th century or sometime in between, the site’s significance would have increased over time with the advent of racial slavery and as colonial officials restricted the burial of Africans at these other sites. The African Burial Ground remained in use until 1795 when the African Society—a mutual aid society—secured a new cemetery on Chrystie Street for “the interment of people of color.”

Thus, the African Burial Ground’s story is rooted in the history of slavery in New York, which spans the Dutch (1624-1664) and British (1664-1783) colonial periods and extends halfway through the 19th century. This long history begins shortly after the establishment of a permanent European presence in the region. This was a slow process begun by traders who, by 1614, referred to the southern tip of Manhattan Island as “New Netherland.” In 1615 Amsterdam merchants founded a trading company in the area for the primary purpose of facilitating the increasingly lucrative fur trade with Mohawk and other indigenous peoples. Six years later, in 1621, the newly-formed Dutch West India Company attained a trading monopoly and began the process of settling along the Hudson River.

Initial attempts to attract labor proceeded slowly at first. Prospective European migrants seeking greater economic freedom and opportunity in the New World instead found disappointment in the Company’s restrictive policies that ensured its tight control over land and livestock (Medford et al. 2009). In 1624 the Company’s efforts at transforming New Netherland from a small trading post or village into a permanent settlement were bolstered by the arrival of some 30 families of French-speaking Walloons (Protestants) (Blakely 2006). Two years later,
Director-general of the Company, Peter Minuit, “purchased” the island from indigenous (probably Lenape) peoples for trade items valuing 60 guilders. The European population of New Netherland became more diverse over the next few decades when the colony was “awash in the swirl of international economic competition” (Blakely 2006: 64). Although variously at war with other colonies such as New England and New Sweden, German, Norwegian and Swedish residents comprised as much as half of the New Netherland population by the middle of the 17th century.

Historians are uncertain as to precisely when, but the first enslaved Africans arrived at the island shortly after the wave of Walloon settlers. In 1625 or 1626 privateers traded eleven African men captured from Spanish vessels to the Company in exchange for provisions; the beginnings of a permanent African presence in the area. The Company would become the largest owner of enslaved labor. These men labored on farms and in various public works including construction of Fort Amsterdam, the colony’s administrative center. Eventually, these eleven men attained “half-free” status and, with their wives, helped to establish the aforementioned free /half-free African community that possibly initiated the African Burial Ground. Historian Christopher Moore (2005) contends this may have been the first community of legally emancipated African Diasporans in North America.

By 1630, Dutch colonists were importing African laborers for whom enslavement was a permanent condition. Enslaved Africans built much of the colony’s main city, New Amsterdam. They built forts and palisades, cleared and farmed land and were critical to the city’s upkeep and growth (Blakely 2006). Most enslaved Africans arrived from Curaçao and elsewhere in the Dutch Caribbean. Others came directly from western Africa; primarily West Central Africa as the New Netherland colony was restricted from trading in “Guinea” or West Africa until near the end of Dutch colonial rule. Indeed, Medford et al. (2009) report that 92.8 % of all Africans imported to
the Americas between 1601 and 1650 were West Central Africans. This pattern is reflected in surnames such as “Angola” and “Congo.”

Within this context of quickened migration and social change, slavery emerged in the North, initially, as a set of unregulated practices and relationships. The African experience in Dutch New Amsterdam has been characterized as one of “quasi-freedom” because enslaved people held legal rights and protections later done away with under British rule. For example, enslaved Africans testified in court, worked for wages, and bore arms in defense of the colony (Harris 2004; Medford et al. 2009). Some African men attained “half-freedom” for themselves and their wives by paying an annual tax (e.g., of maize or wheat and a hog) and providing a specified amount of labor for the Company (Higginbotham 1978). As noted above, among these rights was landownership, including the ability to purchase, sell and will land. However, failure to meet their obligations to the Company meant a return to “full” slavery. Worse, half-free status did not extend to children who remained “bound and obligated to serve the honorable West India Company as slaves” (Moore 2005: 45). With great difficulty, some free and half-free Africans purchased, successfully petitioned for, or otherwise negotiated freedom for their children and other relatives.

Heywood and Thornton (2009) note that the trade region in West Central Africa extended over 700 miles and was politically diverse, including the major states of Kongo and Ndongo as well as numerous small, “semautonomous” polities. Still, individuals from the region likely exhibited a degree of familiarity with one another that would have informed their cultural responses to life and slavery in colonial America. Those from major states may also have been familiar with Europeans as Dutch, English and Portuguese traders had established a strong presence in West Central Africa by this time. Initially limited to existing commercial networks and constrained by political alliances, with time Europeans exerted considerable influence over indigenous politics and economies. The Portuguese in particular had longstanding relations in the area, having launched the Atlantic trade in enslaved Africans in the mid-15th century. The nature of European influence and African agency in the promotion, growth and evolution of slavery is a major topic of debate in Afro-Atlantic historiography (Rodney 1966; Fage 1969; Inikori 2001).
Various factors likely contributed to Africans’ ambiguous legal standing in New Amsterdam. Hodges (1999) points to the fact that Holland did not recognize slavery and, therefore, provided colonial administrators with no legal model for its institution. Others note that granting half-freedom was a trade-off that allowed the Company to secure a portion of the labor of half-free Africans while absolving itself of any costs associated with their care (see, for example, Higginbotham 1978). Furthermore, by locating free and half-free Africans’ farmland north of the Common, the Company created a buffer zone between the city and hostile Indian country (Swan [1998] cited in Medford et al. 2009).

Whatever its basis, the relatively humane treatment of Africans in New Amsterdam was undermined by the turn to race and racism and the emergence of a racialized hierarchy of labor. Economic competition with slavery led European wage laborers to champion newly racial arguments of African inferiority for the specific purpose of deskill ing enslaved laborers. Harris (2004:341) explains how

[p]ractically from the arrival of the first slaves, European indentured servants and free laborers sought to distinguish themselves from African slaves because of competition with them in a tight labor market; one slave could be purchased for the same amount as a free laborer’s annual wages. In 1628, white workers requested that the Dutch West India Company not train slaves for skilled labor as it did in other American colonies. In appeasing white laborers by agreeing to exclude slaves from skilled occupations such as bricklayer and carpenter, the Dutch West India Company unwittingly encouraged settlers to use racial differences to determine who was suitable for certain occupations. By the 1650s, European settlers had begun to declare publicly that Africans were not as competent skilled laborers as Europeans.

Efforts at limiting the use of enslaved Africans were unsustainable, however, as their skilled and non-skilled labor proved essential for the colony’s prosperity. To secure this labor, Dutch colonists combined race and slavery as core aspects and markers of New World identity and social status.

In 1664 the colony’s Director-general, Peter Stuyvesant, ceded New Netherland to the British who renamed the New Netherland colony and the city of New Amsterdam in honor of
the Duke of York. Slavery flourished in New York. British officials sought to make Manhattan “a major North American slave port, and the New York colony a major market for slaves” (Harris 2004: 342). The next year, legislators began drafting the colony’s slave code. New laws established business incentives for promoting the growth of the slave market. Slavers imported Africans to work in local industries and domestic settings and to speculate on the buying and selling of enslaved Africans in trading with other colonies. At the same time, authorities set prohibitive costs and time constraints on the use of white indentured servitude (Higginbotham 1978). Harris (2004: 342) observes that the provincial assembly, fearing reprisal by local tribes, prohibited the enslavement of Native Americans in 1679 and that a 1706 British law stated explicitly that “Negroes only shall be slaves” and that “baptism shall not alter the condition of servitude of the Negro slave.” This legally severed the already tenuous connection between Christianity and freedom for African slaves. In the same law, the British ensured the hereditary nature of slavery by having children inherit the mother’s condition of slavery or freedom.

Thus, under British rule and a solidifying “racial worldview” (Smedley 2007), slavery expanded and came to be associated exclusively and permanently with Africans and African descent. By the end of the 17th century, New York’s African population was the largest of any North American city. By the middle of the 18th century, Africans accounted for just over 20 percent of the city’s population and trailed only Charleston and New Orleans amongst urban centers (Medford et al. 2009).

Who were these enslaved individuals whose labor enabled the transformation of a small Dutch trading post into an American metropolis? This study assumes a subpopulational approach or perspective at the group (e.g., children and adults with dental modification) and individual level. However, a deep, biohistorical understanding of who these people were begins with a broader assessment of patterns of forced migration. Analyses of ships’ logs and merchants’ records reveal different pathways by which enslaved and captive people arrived at
New York. By reconstructing these pathways – which included direct trade with Africa, the provisions trade with the West Indies and Chesapeake colonies, privateering and piracy – scholars are able to shed light on who these enslaved individuals were genetically, geographically and socio-culturally (Goodman 2007; Jackson et al. 2009).

Building on demographic studies by James Lydon (1978), David Eltis (1999) and others, NYABG Project historians have identified important patterns concerning the ethnic and geographic origins of this population. Medford et al. (2009:43) report that “[t]he period of greatest importation of enslaved laborers was between 1715 and 1774, when upward of 6,000 arrived” and go on to note that, primarily, “trade to the city consisted of two types: a direct trade with Africa... and smaller shipments (rarely more than 10 individuals) from the West Indies.” During the 18th century, most enslaved Africans (57%) arrived in New York via the provisions trade with the West Indies – primarily from the British colonies of Jamaica, Antigua, Barbados and Bermuda. In exchange for foodstuffs and other critical products such as lumber and value-added products such as beer and snuff, New Yorkers received sugar, rum, European goods and enslaved laborers. The Dutch West Indies continued to supply enslaved Africans to New York, albeit in a diminished capacity, as did some Danish and French colonies.

Many of those who reached New York City via the West Indies were African-born. The estimated ethnic origins of those imported via the West Indies are found in Table 4.1. West Central Africans from Angola remained a significant presence in the city as they had been under the Dutch, but were joined now by large numbers of captives from West Africa. Medford et al. (2009: 53) observe that some individuals procured as part of the provisions trade “would have been in the islands for mere days before being transshipped to New York, but others may have spent months—if not years—there, finally suffering sale as superannuated laborers unfit for plantation work.” Indeed, as one of the last American stops for slaving voyages, New Yorkers
Table 4.1: Importation of captives to New York by region (via West Indies). From Medford et al. (2009, Table 7)

<table>
<thead>
<tr>
<th>Region/Coast</th>
<th>Percentage</th>
<th>Estimated Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Senegambia/Sierra Leone</td>
<td>25</td>
<td>800</td>
</tr>
<tr>
<td>Gold Coast</td>
<td>14</td>
<td>448</td>
</tr>
<tr>
<td>Bight of Benin</td>
<td>9</td>
<td>288</td>
</tr>
<tr>
<td>Bight of Biafara</td>
<td>33</td>
<td>1,056</td>
</tr>
<tr>
<td>Angola</td>
<td>19</td>
<td>608</td>
</tr>
</tbody>
</table>

Note: Original source of data: Eltis et al. (1999)

Table 4.2: Direct trade of captives from Africa to New York. From Medford et al. (2009, Table 6)

<table>
<thead>
<tr>
<th>Region/Coast</th>
<th>Percent</th>
<th>Estimated Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Senegambia</td>
<td>77</td>
<td>2,156</td>
</tr>
<tr>
<td>Gold Coast</td>
<td>20</td>
<td>560</td>
</tr>
<tr>
<td>Angola</td>
<td>3</td>
<td>84</td>
</tr>
</tbody>
</table>

Note: Original source of data: Eltis et al. (1999)

Complained of slavetraders’ who sought to unload superannuated workers deemed undesirable in West Indian and southern American markets.  

Forty-three percent of enslaved laborers arrived in New York directly from Africa. In a shift from the Dutch colonial period, this direct trade drew most heavily from West Africa (see Table 4.2). British New Yorkers imported captives from five of the seven major regions of export of enslaved Africans: (1) the Senegambia, (2) the Sierra-Leone-Liberia region, (3) the Gold Coast, (4) the Bight of Benin, and (5) the Niger Delta (Figure 4.1). The vast majority of this group embarked from the Senegambia (77%) and Gold Coast (20%) regions. Thus, African New Yorkers

15 See chapter nine for a discussion of dumping in relation to dental modification.
were culturally diverse; a mix of Ashanti, Igbo, Gur, Mande and other peoples of primarily western African geographic and ethnic origin, reflecting different religious backgrounds and degrees of creolization. For example, a strong contingent of Mande and other Senegambian peoples would have ensured a strong Muslim presence. “Illegitimate” trade with privateers and pirates contributed even more regional diversity.\textsuperscript{16}

\textsuperscript{16} For example, Medford et al. (2009) report that Frederick and Adolph Philipse, members of a prominent merchant family, were “avid participants” in an illegal trade with pirates who
At any given time, the New York African demographic profile reflected shifting and interrelated social, cultural and economic dynamics. Consider, for example, some of the factors that would have influenced decisions to import people directly from Africa as opposed to the West Indies. Slaveowners’ generally preferred laborers “seasoned” by time spent in the West Indies who knew European languages, were familiar with work routines, and had acquired some resistance to New World diseases (Berlin 1998). This preference helps to explain why the majority of enslaved Africans arrived in New York from the West Indies during the 18th century.

Other factors favored direct trade from Africa. Cost was a primary consideration. Seasoned laborers typically were more expensive than those imported directly from Africa. Availability was another issue as slave ships usually arrived at New York after traveling to southern American markets where buyers had the first option to acquire these laborers. Enslaved individuals’ actions influenced these decisions as well. Seasoned laborers – and men in particular – were implicated in the planning of revolts and, thus, were sometimes viewed as “troublesome property.” In slaveowners’ calculations, this perception might offset any potential advantages associated with seasoned labor. Apparently, this was the case in the aftermath of a 1712 enslaved African uprising and the uncovering in 1741 of a “conspiracy” to burn down the city that involved a diverse network including enslaved Africans, Irish soldiers, and descendants of some of the original Dutch settlers. Direct importation from Africa increased following both events (Blakey 2010; Barrett and Blakey 2011).17

brought hundreds of captives to the city from Southeast Africa (Madagascar) in the late 17th century.

17 Recall that many who arrived in New York via the West Indies were African-born. So, the important question here is that of how seasoning promoted shared, creole identities or ethnic solidarity. In the case of the 1741 “plot,” at least some organization occurred along ethnic lines as a group of friends and alleged conspirators had Akan day names. See chapter three for a comparison of the creolization and ethnic tenacity models.
Attempts to characterize this population must also account for gendered and child labor practices and associated shifts in age and sex distributions. Medford et al. (2009) provide an overview of the range of labor required of African men, women and children during the 18th century. As the city urbanized and assumed a prominent role in the Atlantic shipping industry, demands on enslaved laborers naturally expanded and became more diverse. Enslaved men continued working on farms and in mines outside the city while being utilized increasingly for jobs related to maritime trade such as coopering, sailmaking, shipbuilding and sailing. African women served primarily as domestic laborers tasked with sewing, cooking, cleaning and caring for slaveowners’ children, but were employed for work in the field and other tasks as needed.

Of course women “bore the added burdens of childbirth and rearing, usually without a co-residential mate” (Medford et al. 2009: xix). Most slave owning households in the North included two or three enslaved individuals; typically, a mother and her child or children. With different owners, adult males usually lived in separate houses. Limited visiting rights with their families were a source of tension that may have contributed to the 1741 incident Medford et al. (2009).

Children also served primarily as domestic laborers. Most 18th-century censuses defined a “child” as an individual younger than 15 years. This legal classification was subject to change and, thus, of limited use for assessing the onset of “adult” work stresses. Indeed, children as young as age 6 were advertised for sale and it was not unusual for a child to begin learning a trade between the ages of 6 and 12 (Barrett and Blakey 2011). Like their counterparts throughout the colonial Americas, slaveowners in early New York exhibited a general preference for young adult male laborers. When importation rates for men declined, women and children found their labor roles expanded accordingly. Such would have been the case following the 1712 revolt and 1741 “conspiracy” to burn down the city (Blakey 2010). Women and children
enslaved to shopkeepers and merchants were especially vulnerable to conscription for labor typically assigned to men (Medford et al. 2009). Chapter nine provides a discussion of labor practices, particularly those that would have resulted in lead exposure.

While the factors driving the demographic formation and transformation of this population were dynamic and complex, the overarching narrative of 18th-century New York African life is rather straightforward. Returning once more to the 1712 uprising, one observes in its immediate aftermath the implementation of severe restrictions on the movements of enslaved Africans and their ability to congregate within the city. New laws limited the number of people who could attend funerals and proscribed heavy fines for free Africans who sold goods to enslaved individuals or those who entertained them in their homes or workplaces. Unfortunately, this period was defined by the “tightening vise” of racial slavery and greater psychosocial stresses for enslaved people.

Free Africans were also vulnerable in 18th-century New York. Representing perhaps only 5 percent of the growing African population at midcentury (Blakey 2010), members of this small community faced the loss of economic independence and property. All free workers suffered as enslaved Africans undertook a growing range of labor and became even more central to the city’s economy. Growth of the enslaved African population actually outpaced that of the European population between 1698 and 1738. In response, many Europeans opted to establish independent farms or businesses or relocated to places like Pennsylvania where conditions for free or indentured workers were more favorable (Harris 2004). However, those European workers who stayed benefited from legal protections against free black labor competition. Caught between New Yorkers’ preferences for enslaved labor on one hand and a racially segregated free workforce on the other, some free Africans were forced into indentured service.
in order to avoid being charged as indolent or in violation of vagrancy laws (Medford et al. 2009).

Another major issue facing free Africans at this time was the collective loss of their landholdings as the city expanded northward. Laws passed following the 1712 revolt forbade individuals freed after 1712 from purchasing land and stripped free African landowners of property, some of which had been accumulated since Dutch rule. By 1716, African landholdings in Lower Manhattan were a thing of the past. The loss of property had symbolic as well as material significance. The emerging American republican ideology stressed political independence and landownership as the basis of true freedom. Hence, these developments served the dual function of further positioning white freedom and black slavery as antithetical even as they blurred the line between free and enslaved status for Africans. Figuratively and sometimes literally, those who did not own property were at risk of becoming the property of another. Such was the case especially for free Africans.

Chattel slavery remained the lot of most African New Yorkers throughout the 18th and well into the 19th century when manumissions debates intensified. During the Revolutionary years, abolitionists portrayed as hypocrites those who championed freedom from British tyranny while maintaining a large enslaved labor force at home. Proponents of slavery countered that legislated emancipation was tantamount to robbing slaveowners of their Lockean natural rights of property. Members of both groups questioned whether blacks, the vast majority of whom owned no property (and, thus, lacked political independence) had the capacity for self-government. Eventually, some individuals earned their freedom by fighting alongside British soldiers during the American Revolution. When the British evacuated in 1783, some 4,000 black loyalists – many of them fugitives from slaveowners – emigrated to Nova Scotia, Sierra Leone and England where they faced harsh new realities of discrimination in
freedom (Medford and Brown 2009). Others escaped slavery through military service with American patriots from whom promises of emancipation came more begrudgingly.

For most enslaved New Yorkers, however, emancipation came decades later, the result of a gradual legislative process. Compared to other northern states, emancipation in New York came particularly slowly amid increased reliance on enslaved labor that fueled postwar economic growth.\(^\text{18}\) A measure for eventual emancipation proposed in 1777 began a “long death” for slavery in New York that proceeded over half a century of “cautious steps toward black freedom” (Rael 2005: 124). Another important step in this process was the founding of the New York Manumission Society in 1785. The Society pressed for abolition through the legislature, lobbied slaveowners to manumit enslaved Africans voluntarily, provided legal representation and protection against kidnappers for free and enslaved individuals, and was instrumental in establishing African Free Schools for black children (Rael 2005; Medford and Brown 2009). Nor can the significance of African-American efforts toward freeing themselves be overstated. Enslaved individuals escaped the countryside in the hope of blending into the “anonymous masses” of the city while free blacks stepped up efforts to purchase enslaved loved ones.

Following more than a decade of debate, the New York state legislature passed the Gradual Emancipation Act of 1799. This act did not provide for universal abolition. Rather, it freed only children born after July 4, 1799 and, even then, after a long period of indentured servitude or apprenticeship. Male and female children were indentured to their mothers’ enslavers for 28 and 25 years, respectively. A second emancipation law passed in 1817.

\(^{18}\) According to Rael (2005:126), “New York City became a haven for slavery. Of all the major northeastern cities, it alone remained committed to forced labor. In 1790, Philadelphia counted only 300 enslaved African Americans; even Baltimore, the rapidly expanding Southern port city, listed only 1,300. In contrast, over 2,300 resided in New York.” The free black population grew immensely as well, by over 200 percent during the 1790s.
According to this new law, all enslaved African Americans born before July 4, 1799 would become free on that date in 1827. Enslaved individuals born between 1817 and July 4, 1827 became free following indentured service of up to 21 years; their labor essentially providing slaveowners compensation for the loss of future profits. Thus, an African-American New Yorker born in 1827 could be bound until 1848. Indeed, Berlin and Harris (2005b) report that federal census enumerations included black men and women as “slaves” until 1850. The last piece of legislation addressing slavery in New York passed in 1841. This law prohibited non-New Yorkers from staying in the city with enslaved African Americans for more than 9 months.

New York City was an extremely important site with respect to the length of time and the scale at which its citizens participated in the institution of slavery. For 200 years, New Amsterdam and then New York maintained an enslaved African population through direct trade with Africa and a provisions trade with West Indian and Chesapeake colonies. Slavery began under the Dutch as a relatively informal set of practices and evolved under British rule into a racially codified system of labor, punishment and political-economic stratification. Enslaved New Yorkers built much of the city that came to symbolize American wealth. Yet, as Medford and Brown (2009: 102) observe, “[I]n time, the cemetery and the people interred there became a deeply buried memory, inaccessible to posterity and denied their rightful place in New York history.” Shortly after the cemetery’s closing in 1795, developers targeted the Common and Collect Pond as sites of new commercial and residential properties. With construction activity, the burial ground itself was buried under landfill, disappearing from sight as well as public awareness. Also fading was the memory of enslaved New Yorkers interred there, who became historical footnotes in the city’s official narrative. In the words of Wolf (1982), these were a
“people without history.” It would be almost 200 years before further construction efforts and subsequent activism and scholarship rectified this situation.

The NYABG Project

Background

Currently, the African Burial Ground is located in the Civic Center area of New York beneath various government facilities among other structures; its recent reappearance the result of a United States General Services Administration (GSA) building project. Having obtained Congressional approval in 1988, the GSA planned to construct a 34-story office tower, the Ted Weiss Federal Building, and a 4-story pavilion at 290 Broadway, one block north of City Hall. With the unearthing of hundreds of burials came awareness of the site’s unique cultural, spiritual and research significance and, today, its place in history seems secure.

A major research project culminated in 2003 with the Rites of Ancestral Return, the ceremonial reinterment of excavated remains at the original site. The excavated and analyzed portion of the burial ground was designated a National Monument in 2006. The site memorial was unveiled in 2007 and a permanent interpretive center opened in 2010. As of October of 2009, the site operates under the auspices of the National Park Service and plays host to regular educational forums and cultural celebrations.

And, yet, the journey from forgotten cemetery to National Monument was not smooth. The GSA building project was contentious from its start as public fascination with the cemetery and its hidden history morphed into anger and protest over what many came to see as its – and their – disrespectful treatment. Why and how did this happen? What factors drove this conflict?
Was conflict avoidable? Most importantly, how were the exposed issues addressed and what have scholars and others learned from attempts to resolve them?

A key question, often raised in public presentations on the NYABG Project, is that of whether or when the GSA knew that burials would be encountered at this site. As the burial ground appears on maps dating to the mid-18th century, federal officials acknowledged early on the possibility of uncovering skeletal remains. As dictated by the 1966 National Historic Preservation Act (NHPA), identification of a cultural heritage resource such as a historic cemetery on federal land requires efforts to protect those resources; in this case, to mitigate the potentially destructive impact of building activity. Specifically, amongst other responsibilities, Section 110 of the NHPA directs federal agencies to protect and preserve the integrity of “nationally significant historical properties.” The African Burial Ground met this criteria and in 1993 would be designated a New York City Landmark and a National Historic Landmark.

To develop an environmental impact statement, the GSA hired the firm Edwards and Kelcey who subcontracted Historic Conservation and Interpretation (HCI) for cultural resource management (CRM). HCI was tasked with determining those areas most likely to contain intact burials. In May of 1991, HCI confirmed the presence of skeletal remains approximately 15 feet below grade level. As anticipated by the environmental impact statement, these burials had been disturbed by prior construction. However, further testing revealed in situ burials; a finding that naturally stirred tremendous interest and curiosity when first reported the following month in The New York Times (Hays 1991). Under these circumstances, a prudent (and typical) next step would have entailed more testing to assess the full scope of the undisturbed cemetery context, towards the development of an appropriate archaeological excavation and research plan. Instead, the GSA acted upon HCI’s initial estimation of 50 in situ burials within the construction site and ordered the analysis of 10 burials. This, GSA officials reasoned, satisfied
legal obligations to “protect and preserve” the cultural heritage represented by the affected portion of the burial ground and construction was underway by October of 1991 (Figure 4.2).

It soon became apparent that HCI grossly underestimated the extent of recoverable burials when remains of several hundred people were unearthed over the course of the next few months. These burials had been protected from prior construction activity beneath 25-30-foot-thick 19th-century landfill deposits. Now, some were being damaged by the use of backhoes and other heavy equipment. Also lost or destroyed were original ground surface features and other archaeological context valuable for dating purposes. Under pressure from the GSA to speed up excavation and analysis of the site, HCI subcontracted with Metropolitan Forensic Area Team (MFAT) and Michael Parrington, the latter of whom had excavated the 19th-century First African Baptist Church site in Philadelphia.

By this point, several factors conspired to strain relations between the GSA and a growing, increasingly agitated segment of the public. While GSA officials may not have
anticipated unearthing hundreds of burials, critical observers concluded that actions following this important discovery fell short of meeting the agency’s legal responsibilities. For example, in violation of the NHPA, the GSA initiated excavation and construction without having in place an approved research design. Furthermore, Section 106 of the NHPA instructs federal agencies to involve the public and “other parties” or consultants during the early stages of project planning. While public hearings on the project occurred, many members of the African-American descendant community complained of their perfunctory nature. To them, the purpose of these meetings seemed to be their appeasement rather than constructive dialogue with potential for real influence on the site’s handling. Although Michael Parrington brought valuable experience from the First African Burial Ground archaeological site, this impression was reinforced by GSA officials’ apparent resistance to consult with African-American scholars. The critique was strengthened, also, when the President’s Advisory Council on Historic Preservation (ACHP) found that the research plan finally produced for the GSA inadequately addressed African diasporic perspectives. Meanwhile, Howard University Biological Anthropologist Michael Blakey inspected excavated remains at Lehman College and found poor preservation conditions including mold growth, lack of temperature control, and insufficient storage space. As a result, for many, interest and curiosity gave way to concern and contempt for GSA officials’ perceived intransigence and apparent insensitivity toward the descendant community, the broader public, and this sacred site (Figure 4.3).

Organized protest ensued. The voices of descendant community members and other concerned citizens were unified if not uniform. Some viewed excavation under any circumstances as an act of desecration and called for a permanent halt to all construction activity as well as immediate reburial at the site. Other protesters focused their outrage on federal officials’ lack of transparency and rushed, unsystematic excavation practices employed
Figure 4.3: Protesting the GSA’s handling of the African Burial Ground. Many community members viewed construction on the site and excavation without an accepted plan for scientific research plan as disrespectful to their ancestors and themselves.

by various CRM firms. Members of this latter group were more amenable to, and even advocated for, the site’s scientific analysis but expected from GSA an adequate research design and, if appropriate, a modified construction plan. Overall, most protestors agreed that the GSA’s treatment of the site seemed to mark yet another chapter of institutional insensitivity and racism for African-American New Yorkers; disrespect that mirrored the identification and treatment of their recovered ancestors as “slaves” in life.

For the GSA, financial considerations trumped community and scientific concerns; even those concerns largely shared by other governmental agencies. GSA officials saw as their first and main priority the expedient removal of skeletal remains from the work site. Accordingly, substantive responses to the requests and demands of other stakeholders were usually slow-coming and often negative. On several occasions, then Mayor David Dinkins, who had established a Task Force to oversee the site, requested an end to the excavation. In its official
“watchdog” capacity the ACHP recommended that the GSA secure additional consultants and perhaps work with the Army Corps of Engineers which was better equipped to address an urban project of this scope. The GSA hired John Milner Associates, Inc. (JMA) to oversee excavations and revise the research design. Under mounting public pressure over the exclusion of African-American personnel and perspectives, the GSA also hired Michael Blakey to collaborate with JMA on the new research plan – only a few weeks before the ACHP’s requested deadline.

The GSA’s reluctance to comply with its requests led the ACHP to recommend suspending all work at the site pending an approved research plan, lack of which constituted a violation of the GSA’s memorandum of agreement (MOA) for the project. In their advisory roles, however, entities such as the ACHP and the Mayor’s Task Force exerted limited influence on GSA officials’ decision-making. Michael Blakey has written extensively about the events surrounding the African Burial Ground’s “rediscovery,” analysis and memorialization (e.g., 1998, 2001, 2009, 2010; La Roche and Blakey 1997).

As Blakey (2010: 62) summarizes the situation,

> [t]he bureaucratic strategy was to plough forward with construction while holding required public meetings and expediting the archaeological excavation needed to mitigate the total destruction of cultural resources. The public strategy, consistent with the legacy of the Civil Rights Movement, was to organize mass public protests and to lobby legislators to end excavation and construction when meetings with the GSA were found to be without substance.

Calls to halt excavation and/or downscale construction were dismissed by GSA officials who estimated the cost of doing so in the tens of millions of dollars.

The combination of public protest and lobbying proved critical for ending the standoff between community activists and the GSA. This strategy succeeded in garnering vital Congressional support for those seeking to end the excavation. At the conclusion of a Congressional Subcommittee Hearing on the African Burial Ground held in July of 1992, U.S. Representative Gus Savage, the Chair, concurred with community members and the ACHP that
the GSA was in violation of Section 106 of the NHPA. Congressman Savage found that GSA officials had not acted in good faith in their dealings with public stakeholders and immediately convened a Federal Steering Committee to oversee proper compliance with the MOA. Ultimately, Congressional intervention resulted in: (1) an early end to excavation (which, unchecked, likely would have meant disturbing over 100 additional burials); (2) modification of construction plans to accommodate reburial of skeletal remains; (3) development of an Office of Public Education and Interpretation and, later, a memorial and visitor center; and (4) involvement of African-American scientists in future research. For members of the descendant community and their supporters, this was a victory for human dignity and heritage rights – for the living as well as the dead.

When excavation ceased and the African Burial Ground closed officially in October of 1992, workers had identified over 400 burials. The excavated portion of the cemetery, technically referred to as “The New York African Burial Ground,” is considered one of the most important archaeological discoveries of the 20th century (Cantwell and Wall 2001). In 1993, GSA contracted with Howard University to conduct the site’s scientific analysis – the New York African Burial Ground Project – as detailed in the research design developed by Blakey and JMA. Blakey, then curator of the university’s William Montague Cobb Skeletal Collection, would serve as the project’s Scientific Director.

The descendant community’s decision for African-American leadership and intellectual stewardship of the project was not an automatic or unthinking one. Rather, this was an organized attempt to make scholarship relevant by opening up new critical space in an academic field where African-Americanist theoretical influence has been traditionally marginalized (Blakey 2001; Watkins 2007). The descendant community’s position on this matter proved instrumental when the ACHP and some scholars voiced concern over some of the vindicationist aspects of the
Blakey/JMA research design. Critics questioned the objectivity of such an approach. Presented with alternative scientific viewpoints, community members in turn questioned the objectivity of scientists who favored bio-racial assessments traditionally linked to arguments of black inferiority – and that obscured important biohistorical information. In this light, many came to see the proposed research agenda as a necessary corrective to the types of Eurocentric historical distortions that rendered the African Burial Ground invisible in the first place.

As a colonial-era cemetery in New York, the African Burial Ground held unprecedented potential for exploring processes of ethnogenesis and early racial formation. Howard University received 419 individuals; a fraction of the estimated 15,000 individuals buried at the cemetery, yet still the largest African diasporic skeletal sample in the Americas.\(^19\) Moreover, the site dates to a foundational stage of African-American identity in the North, a region for which scholarship on slavery is relatively scarce. In light of the project’s contentious history, researchers sought to demonstrate that public and scholarly interests and goals concerning this sacred site could be compatible, even shared. Tapping the burial ground’s research potential would require a new model of bioarchaeology: one that was fully interdisciplinary, biocultural and publicly engaged.

### Research Goals

The NYABG Project differed from previous bioarchaeological studies of the African diaspora in several important ways. For example, under Blakey’s direction, the research team assembled was exceptionally diverse, drawing upon the expertise of over 30 scholars at 9 institutions. The large sample size and the site’s distinctive historical and geographic location required researchers, from art historians to geneticists, to approach the project from multiple “cross-fertilizing” perspectives. Technological advances also played an important role in defining

\(^{19}\) From 436 burials assigned in the field, 419 individuals were inventoried. Of these, 391 individuals were sufficiently preserved for laboratory analysis.
the project’s potential as the research design called for the use and development of sophisticated and emerging analytical techniques such as the laser sampling employed in this study. These divergences from prior practice were primarily of a technical nature or matters of scale.

A more fundamental challenge to bioarchaeology “as usual” came from project leaders’ efforts to redefine the power dynamics that govern interactions between scientist and subject. Blakey has written extensively about the racial politics of the NYABG Project that influenced this new model of engaged anthropology (e.g., see Blakey 1998, 2001, 2009, 2010; La Roche and Blakey 1997). What most distinguished the NYABG Project’s theory of public engagement was an insistence that the descendant community fulfilled the role of "ethical clientele." This framework pushed the envelope of engaged anthropology beyond attempts to identify community “partners.”

As with most academic projects, NYABG researchers reported to a funder. In this case, the “business client” was the GSA. Within this new model, the research team was also accountable to the descendant community, which received regular scholarly updates and had the power to propose and even deny specific research avenues (such as those involving invasive measures). Not surprisingly, this latter condition – i.e., community approval for research plans and methods – proved controversial within the discipline. On what grounds, some asked, should non-experts determine acceptable or proper scientific protocol? In response, project leaders argued that scientists should learn from GSA officials’ missteps, and not seek to exert their own authority over the skeletal remains. Indeed, they could not. When community members asserted their heritage rights and demanded an end to excavation and proper memorialization of the site, they also earned the right to help determine what information gleaned from these ancestral remains was most valuable and worth securing. Respecting this right was an important
step away from anthropology’s colonial and colonizing legacy, and in line with current efforts to move the discipline constructively beyond academic walls (Low and Merry 2010). 20

This theoretical approach combining community experience with scientific experience and technical expertise had major and overwhelmingly positive implications for the nature and scope of the project. In addition to determining that Howard University could undertake the project, community input was solicited to help determine the major research questions. Collaboration between community members and researchers produced 3 key areas of inquiry, which appear in the Blakey/JMA plan:

1) What were individuals' geographic origins and cultural backgrounds?
2) What cultural and biological processes were associated with African-American identity formations?
3) What was the physical quality of life wrought by enslavement?

Project leaders later added a fourth:

4) What modes of resistance or "humanity maintenance" did enslaved New Yorkers employ?

As we have seen, racial slavery influenced every aspect of black life in early New York. These questions acknowledge this reality while anticipating as well the facts of human agency and diaspora and experiences that crosscut varied political, economic and cultural settings, sometimes within a lifetime. Their primary purpose, therefore, was to establish a biocultural and diasporic framework that would enable a full exploration of lived experiences leading up to and including life and death in New York. Thus charged, NYABG researchers undertook to investigate the full humanity of this population.

---

20 Interestingly, the NYABG Project approach captured the full range of anthropological engagements identified by Low and Merry (2010): sharing and support; teaching and public education; social critique; collaboration; activism and advocacy.
Major Research Findings

The final research findings were published in 2009 as a series of edited volumes under the title *The New York African Burial Ground: Unearthing the African Presence in Colonial New York*. These technical skeletal biological (Blakey and Rankin-Hill, 2009), archaeological (Perry et al. 2009) and historical (Medford, 2009) reports represent over a decade of research into early African New Yorkers’ origins, migrations, lifeways and deaths, all in diasporic perspective. Many of the major historical findings appear above, in the first few sections of this chapter. Here, I summarize key archaeological and skeletal biological research findings, with emphasis on those relevant to this study’s focus on geographic origins and migration.

Construction activity destroyed valuable surface features typically associated with religious-based African diasporic mortuary practices before they could be recorded by the field archaeologists. This loss affected the interpretation of the social and ideological contexts surrounding the deaths of the overwhelming majority of the recovered individuals. It also destroyed information that could have informed an understanding of the chronological order of the burials. Despite this loss, the NYABG project archaeologists, using coffin characteristics and the very few temporally diagnostic artifacts recovered from the graves, were able to categorize burials within one of four temporal groupings: Early (pre-1735); Middle (circa 1735 to 1760); Late-Middle (1760-1776); and Late (post-1776). These temporal groupings are used throughout my study.

Most individuals, and all children, were buried in coffins. Coffin use was increasingly common on both sides of the Atlantic during the 18th century e.g., among the Akan whose burials required either coffins or shrouds. The west-headed orientation and supine extended burial position found for so many of the burials is a level of consistency very suggestive of the existence of a distinctive mortuary program being practiced by the New York African
descendant community to address the spiritual needs of people from such a wide variety of cultures throughout Africa (Perry et al. 2009). A few coffins were shared and were generally interpreted as evidence of familial relations. Researchers plan to test this hypothesis through genetic analysis.

The destructive recovery practices certainly help to explain why there are not many artifacts. This said, other studies of cemeteries and quarters of enslaved populations also report a limited number of artifacts (Handler et al. 1986; Parrington and Roberts 1984; Samford 2007). Among the artifacts that were recovered, most of them were copper shroud pins. Shrouding was associated primarily with infants, children and adult females – but also with nearly half of all males. Also recovered were beads, cowries, shells, and several types of adornments (e.g., buttons and cuff links) (Figure 4.4). Of particular interest for this study, Figure 4.5 is a photo of a lead musket ball found lodged in the rib cage of Burial 25, a 20 to 24-year-old woman.

Skeletal remains are an invaluable source of insight into processes of migration, health and disease that underlie demographic patterns. Project researchers employed historical
demography and paleodemography to establish patterns of migration, fertility, mortality, and population structure for enslaved African New Yorkers. Historical data affirmed the steady importation of enslaved people from the colonial South, the Caribbean, and directly from Africa. However, Rankin-Hill et al. (2009) note several inherent limitations of the documentary record. These factors include insufficient biographical details and intentional undercounting of enslaved Africans for purposes of smuggling and tax evasion. Given such limitations, the authors suggest that paleodemographic reconstructions based on skeletal age and sex determinations may be more appropriate in African diasporic bioarchaeology.21

The NYABG paleodemographic sample consisted of 301 individuals: 102 males and 69 females for which age and sex were assessed and 130 subadults for which age was determined. Rankin-Hill et al. (2009) stated 4 primary objectives of paleodemographic research:

21 Paleodemography is not without its own controversies and limitations. For example, in their influential critique, Wood et al. (1992) argued that paleodemographic interpretations too often failed to account for dynamic and changing migration patterns, the selective nature of mortality (for which rates may not be readily inferred from pathological observations), and “hidden heterogeneity” based on differential susceptibility to disease. Also, bioarchaeological analyses are also subject to the question of just how representative the skeletal sample is of the actual living population of the time.
1. establishing population profiles and demographic trends that integrate historical and paleodemographic evidence;

2. contextualizing the New York African population within its surrounding temporal, political, economic and sociocultural landscape;

3. placing the skeletal sample within an African diasporic biohistorical framework; and

4. providing a conceptual framework for archaeological research.

With these objectives in place, the authors were able to identify important trends with respect to mortality, fertility and population structure. Mortality rates were high for infants and young children, with approximately a third of all individuals dying before age 5. Among those less than 15 years of age, nearly a quarter died younger than 6 months. Another 17% died before age 1 and over half of all children died before reaching age 2. For adults, Rankin-Hill et al. (2009) report that more females (25%) died between the ages of 30 and 34 than any other age range, compared to a peak mortality rate (18%) for males between the ages of 40 and 44. For males, this rate is just slightly above that observed for age ranges 45-49 (17%) and 50-54 (15%).

Adult sex ratios seem to suggest that the NYABG population did not achieve natural increase through childbirth. Blakey (2010) observes that the NYABG adult sex ratio, while dynamically responsive to changing labor demands, generally resembled that of the Caribbean where men outnumbered women leading to low fertility rates and population growth. This finding of low fertility rates may seem to contradict that of high infant and young child mortality rates. However, Rankin-Hill et al. (2009) point out that overall population growth was slow for African New Yorkers during the 18th century, and most likely the result of increased childhood importation rates over time.

Demographic trends provide an overview of population health conditions or, more accurately, reflect their consequences. Paleopathological analysis, on the other hand, offers a direct and sometimes specific view of actual health and disease processes down to the
individual and even lifespan level. Here, 306 (86%) of 358 individuals for whom analysis was possible showed evidence of skeletal pathology. Some diagnostic markers point to specific conditions, such as porotic hyperostosis, generally considered an indicator of anemia. Others, like periostitis, are general or nonspecific indicators of disease. Periostitis is a nonspecific indicator of infectious disease. Both porotic hyperostosis and periostitis were frequently found amongst NYABG individuals.

Another class of skeletal pathological markers are enamel developmental defects. Enamel defects are extremely informative as they indicate metabolic stress from nutritional disorders or infectious diseases during specific periods in life (Goodman et al. 1980). As tooth crowns form during well-documented age ranges (Reid and Dean 2006), lesions occurring during enamel matrix formation (hypoplasias) or mineralization (hypocalcification) are nigh-permanent records of health disruptions during fetal, early childhood and adolescent periods of growth and development (see chapter 5). NYABG Project researchers found that, for the 65 individuals for whom teeth that formed between birth and age 6.5 years could be assessed, 71 percent (n=46) of the 65 individuals had hypoplasias (Blakey et al. 2009). The authors note that a peak hypoplasia frequency between 3.5 and 6.5 years of age suggests that this was a particularly stressful for enslaved African children, perhaps related to approaching and reaching the age at which one might be sold and separated from parents and other loved ones.

Skeletal research indicates that this population endured harsh work regimes as evidenced by advanced (early onset) osteoarthritis, fractures, and work stress lesions or markers. For example, excessive loading on top of the head (axial loading) may have resulted in the ring fracture of the base of the skull observed for Burial 107, a female aged 35-40 years. This was a perimortem fracture occurring at or near the time of death. Musculoskeletal stress markers (MSM) associated with intense physical labor include hypertrophic bone development
and furrows at tendinous attachment (enthesopathies) or ligamentous attachment sites (syndesmoses). Such indicators of work stress were observed for men, women and children.

Instances of interpersonal violence were also observed, and some may have reflected forms of punishment. Such cases included perimortem fractures possibly occurring at or near the time of death and highly polished bones consistent with burning. Among the more striking cases was that of Burial 25, the young woman noted above for the presence of a lead musket ball in her rib cage. This woman also had evidence of blunt-force trauma to her face and a “spiral” fracture just above the right wrist caused by twisting and pulling action. Possibly, this woman faced a violent response to her attempts at resistance (Wilczak et al. 2009). Skeletal analysis also reveals possible cases of grave-robbing, which Medford and Brown (2009) identify as a common practice in late-18th-century New York.

Finally, concerning ancestry, the primary focus of this study, Goodman (2007) identifies three often-entangled and yet distinct types: genetic, geographic and social-ethnic. NYABG researchers have explored all three forms of ancestry as each potentially plays an important role in the construction of human identities. Craniometric and mitochondrial DNA analyses suggest genetic affinities to contemporary Senegambia, Ghana and Benin for some individuals (Jackson et al. 2009). Overall, genetic research supports historians’ conclusions that African-born New Yorkers hailed primarily from West and West Central Africa.

Chemical analysis provides the most direct means of geographic sourcing at the individual level. Prior research suggests there was considerable variation in dental enamel lead levels among NYABG individuals (Goodman et al. 2009; Webb et al. 2005). This finding is consistent with widespread lead use documented during the colonial period for North America and the West Indies through which many New York-bound captives were routed (e.g., McCord, 1954; Handler et al. 1986; see chapters 5 and 9). An especially important and consistent finding
Figure 4.6: Strontium isotope ratios for NYABG children and adults. Values for adults with cultural dental modification were more variable and, generally, above the 0.711-0.712 “local” Manhattan range. Higher values suggest African birth. From Goodman et al. (2009, Figure 48)

of dental chemistry has been that young children exhibit higher average and more variable lead content than adults. One study found that children’s enamel had over four times the lead content of adults with culturally-modified teeth: 5.9 μg g^-1 versus 1.4 μg g^-1, respectively (Jones et al. 2007). While it is intriguing to find that lead is both present and variable in concentration in NYABG teeth, many questions remain unanswered such as the sources of lead and the consequences of high-level lead exposure.

In addition to enamel-lead analysis, NYABG researchers have conducted strontium isotopic (^{87}Sr/^{86}Sr) analysis of dental enamel (Goodman et al. 2009). ^{87}Sr/^{86}Sr analysis is a widely accepted method in bioarchaeological studies of geographic origin and migration (e.g., Price et al. 2012). This method is based on the principle that a landscape’s unique isotopic signature will be incorporated into forming skeletal structures. ^{87}Sr/^{86}Sr isotope analysis of early-forming
enamel and dentine revealed greater variation for adults with culturally-modified teeth (fig. 4.6). Values for most of these individuals were higher than the assigned “local” Manhattan range of 0.711-0.712 (Goodman et al. 2009), suggesting African birth. Interestingly, for nearly all individuals with non-local enamel values, the less-stable dentine $^{87}$Sr/$^{86}$Sr values appeared to equilibrate towards the Manhattan range. However, interpreting dentine chemistry is complicated for reasons noted in the next chapter and these values may reflect in vivo or diagenetic change.

This dissertation continues the work of reconstructing biohistories begun by the NYABG Project and addresses these questions. It pushes dental chemical methodologies in ways that are relevant to each of the four main research areas, albeit with a primary focus on geographic ancestry. The next chapter situates this study in a broader academic context of human environmental health research, i.e., the political ecology of lead exposure. Then, in the second half of the dissertation, I detail the current attempt to understand lead, literally, an “element of diaspora.”
CHAPTER 5

POLITICAL ECOLOGY OF HUMAN LEAD EXPOSURE

Introduction

In this chapter I seek to explain some of the major pathways of lead exposure and how lead, once in the body, can become a “legacy pollutant” and part of the “viciously biocultural cycle” (Goodman 2009: xv) that reproduces poverty today. Here, I pick up on threads from chapter 2, which introduced biocultural conceptions of “environment” and “political ecology,” and combine these with concepts such as biomonitoring and risk focusing from environmental science and public health.

This chapter also contains a discussion of bones, teeth and biomarkers. Tooth development is one of the best studied areas of human anatomy, resulting in a plethora of studies in the past decade. Most of these studies are of modern populations, and the teeth are not exposed to diagenesis and taphonomic issues associated with excavated human remains. I describe some notable studies in this vein. However, I focus on studies of past populations known through bioanthropological research that provide comparative skeletal biological findings for the NYABG population. This discussion is geared towards understanding how certain groups become more highly exposed (or at least more at-risk) than others to environmental stressors.

Environmental Lead and Human Biology

Lead (Pb; density: 11.3 g/cm³) is a metal, defined by certain physical and chemical properties including its luster, malleability, heat/electricity conductivity and tendency to form positively charged ions, or cations. Lead’s low boiling point (327°C) and the ease of smelting
galena (lead sulfide) help to explain the presence of lead artifacts from the late seventh millennium BC (Rapp and Hill 1998). All metals up to uranium occur as natural elements, but their roles in human health vary substantially. Some such as iron and zinc are essential to human life while others such as barium serve no known physiological role. Lead is among the latter group. Like other heavy metals such as arsenic, cadmium and mercury, lead is also xenobiotic; even trace-level exposure may adversely affect health (Hu 2002). Hence, the problem with lead: what industry has dubbed "the useful metal" is also a "versatile, subtle, and persistent poison" (Needleman 1998) that ultimately finds its way into bones and teeth.

Social and Cultural Pathways of Lead Exposure

Human lead exposure results from a variety of social, cultural and biological sources and pathways. At natural or background level, lead is introduced into human environments through atmospheric deposition of geological and anthropogenic emissions. Natural sources such as the erosion of the earth's crust, production of sea spray and volcanic eruptions account for earliest human lead exposure but only a small portion of contemporary environmental lead (Davidson and Rabinowitz 1992). Because human evolution has occurred primarily in this low-level exposure context, human biology is lead-intolerant.

Most current human lead exposure results from anthropogenic emissions due to industrial activity, especially combustion of oil and its derivatives and metal production and recycling (Davidson and Rabinowitz 1992). Lead, which occurs in various forms, is used extensively in the manufacture of batteries and the production of pigments, glazes, solder and cable sheathing among other products (Hu 2002). Globally, most lead emissions are due to use of leaded gasoline, which was introduced in 1923. The phase-out of leaded gasoline begun with federal regulatory legislation during the 1970s has contributed to a drastic decrease in overall
blood-lead levels in United States children, from 12.8 μg dL\(^{-1}\) to 2.3 μg dL\(^{-1}\) between the late 1970s and the early 1990s (Pirkle et al. 1994). Currently, a worldwide phase-out effort is underway, but leaded gasoline and other exposures are still prevalent in many parts of the world (e.g., Jain and Hu 2006; Adebamowo et al. 2006; Olivero-Verbel et al. 2007), and continued industrial lead use ensures that contemporary and future populations are at risk for environmental lead exposure. Table 5.1 lists some commonly used lead compounds.

Employing risk focusing modeling, biological anthropologist Lawrence Schell and co-workers' (e.g., Schell 1997; Schell and Denham 2003; Figure 5.1) have shown how so-called universal exposures may become unevenly distributed and subsequently reproduced due to political and economic factors. This is clearly illustrated by the current race-class profile of pediatric lead poisoning. The primary source of pediatric lead poisoning is deteriorating lead-based paint in the form of dust and chips ingested by infants and young children. Once considered a universal exposure source for all United States residents, this threat has subsided greatly since the 1978 federal ban on use of leaded paint on interior surfaces (Pirkle et al. 1994).

<table>
<thead>
<tr>
<th>Compound</th>
<th>Name</th>
<th>Use</th>
</tr>
</thead>
<tbody>
<tr>
<td>PbS</td>
<td>Lead sulfide, or galena</td>
<td>Ore</td>
</tr>
<tr>
<td>PbO</td>
<td>Lead monoxide</td>
<td>Glazed pottery</td>
</tr>
<tr>
<td>PbCrO(_4)</td>
<td>Lead chromate</td>
<td>Yellow pigment (road lines)</td>
</tr>
<tr>
<td>Pb(_3)O(_4)</td>
<td>Red lead</td>
<td>Paint (corrosion resistance)</td>
</tr>
<tr>
<td>Pb(_3)(CO(_3))(_2)OH(_2)</td>
<td>White lead, or lead carbonate</td>
<td>Paint (prior to 1971)</td>
</tr>
<tr>
<td>Pb(_3)(AsO(_4))(_2)</td>
<td>Lead arsenate</td>
<td>pesticides</td>
</tr>
<tr>
<td>Pb(C(_2)H(_3)O(_2))(_2)·3H(_2)O</td>
<td>Lead acetate (&quot;sugar of lead&quot;)</td>
<td>Paint and varnish drier</td>
</tr>
<tr>
<td>Pb(C(_2)H(_5))(_4)</td>
<td>Lead tetraethyl</td>
<td>Gasoline antiknock compound</td>
</tr>
</tbody>
</table>
Figure 5.1: Risk focusing model on the relationship of social class and toxic exposure. Poverty "recycles" lead exposure over a life span, undermining efforts at social mobility necessary for escaping high-lead environments. From Schell and Denham (2003, Figure 3; see also Crooks 1995).

However, leaded paint is still found in many urban environments, mainly in old housing where cases of elevated blood-lead levels and pediatric lead poisoning are most prevalent (American Academy of Pediatrics 2005; Lanphear and Roghmann 1997). In fact, 40% of the nation's housing stock may still contain leaded paint (Wakefield, 2002; cited in Barbosa et al. 2005). Soil contaminated by airborne lead is another major source of pediatric lead poisoning also concentrated in urban environments (American Academy of Pediatrics 2005). Families living in these areas and homes are mostly poor and disproportionately racial minorities (Lanphear
and Roghmann 1997; Weintraub 1997; Melwani 2006). According to 2007-2010 National Health and Nutrition Examination Surveys (NHANES) data, overall blood-lead levels for all children aged 1 to 5 years (1.3 μg dL⁻¹) continue to decrease, but remain significantly higher for blacks at 1.8 μg dL⁻¹ compared with 1.3 μg dL⁻¹ for both Mexican Americans and whites (Wheeler and Brown 2013).

Trotter (1990) suggests the following four cultural "parameters" or domains useful for investigating lead exposure and poisoning.

1) Subsistence-related exposures result from lead's many modern technological uses, which increase ambient lead levels and affect most people in industrialized and developing countries. The other three domains "are areas where lead poisoning occurs due to beliefs and behaviors outside the boundaries of technological exposure... [and] are extremely insidious because they are unexpected" (Trotter 1990: 79). They are:

2) Food habits (or foodways). This refers to the conceptualization as well as the consumption of food and beverages. Food growth, processing and preparation are potential sources of lead exposure through contaminated soil or, as in the well-known case of the Roman Empire, high lead content storage vessels and utensils. In contemporary societies, contamination is most likely to occur during food processing. Needleman and Bellinger (1991) note higher lead levels for food from soldered cans than for unprocessed food or food from seamless aluminum cans. Also, leaching from lead-glazed pottery remains an important dietary exposure source in Mexico and other places (Olaiz et al. 1996; Brown et al. 2000; Tunstall and Amarasiriwardena 2002).

3) Health practices; an exposure source since antiquity. Metallotherapy has long been a part of medical experimentation and practice. Lead and its compounds, due to their antiseptic properties, were among the first mineral drugs and still are used in herbal and "folk" medicinals throughout the world (Nriagu 1992).

4) Beauty or aesthetic practices involving leaded pigments or cosmetics are yet another longstanding exposure source. Elevated blood-lead levels in the Middle East, Asia and Nigeria have been linked to leaded eye applications as children may ingest the pigments after touching their mother's or their own faces (Needleman and Bellinger 1991).
These cultural pathways may also overlap, as seen with the practice of pica or geophagy in which potentially lead-contaminated non-food items such as soils and clays are consumed, sometimes as an attempt to address nutrient deficiencies.\(^{22}\) Whether or not pica is practiced for health purposes, materials deemed appropriate for consumption are often culturally defined (Vermeer 1970).

The exposure sources described above are all exogenous or external in nature. They are introduced into the body and the bloodstream via environmental and dietary sources. Once in the body, blood-lead is sequestered and stored in bone. Although highly mineralized, bone is dynamic. Throughout life, the skeleton is renewed through episodic resorption of old tissue that is replaced with newly-formed tissue (Scheuer and Black 2004). This process is known as remodeling. One of the functions of bone remodeling or turnover is to provide the body with stored calcium and phosphate (Tuross 2003). In the process, however, bone-lead may also be liberated and remobilized into the bloodstream (Mushak 1992; Hu and Hernandez-Avila, 2002; Gulson et al. 1996). In this way, bone-lead becomes a potential source of internal or endogenous exposure.

Lead workers subject to ongoing exposure and those who undergo rapid bone remodeling are particularly susceptible to endogenous contamination. Among the latter are individuals with certain (e.g., calcium, iron and zinc) nutrient deficiencies, pregnant and lactating women, and fetuses and young children. Gulson and co-workers (2003) found that greater bone turnover rates correlate with increased blood-lead levels during pregnancy. Lead concentrations were even higher for postpartum women.

Infants and children are vulnerable to their own as well as maternal endogenous lead. Schell et al. (2003: 95) report that “lead burden begins before birth with lead transferred from

---

\(^{22}\) See chapter 9 for a discussion of pica as a possible source of lead exposure for enslaved Africans.
maternal circulation and increases rapidly in the first few years of life, as exposure to environmental lead increases.” Despite its many health benefits, breastfeeding is energetically demanding and facilitates further mobilization of bone-lead in lactating women. Maternal lead is transferable via breast milk, and has been linked to increased blood-lead levels in infants (Chuang et al. 2001; Téllez-Rojo et al. 2002; Gulson et al. 2003, 2004; Manton et al. 2003). Thus, while Schell and Denham (2003) describe lead as a "legacy pollutant" due to its persistence in urban environments, the term also suggests the lifelong and intergenerational nature of exposure.

Lead Uptake, Metabolism and Tissue Deposition

Understanding lead's bodily distribution and toxicological properties, and charting the impacts of lead regulatory legislation, requires routine assessment of human exposure at two levels. **Environmental monitoring** refers to “quantitative measurement of lead levels in those environmental media which also serve as exposure routes for humans” such as air, leaded paint, dust and soil, while **biological monitoring** (or **biomonitoring**) “describes the quantitative assessment of lead in biological media from exposed individuals, the total body lead burden, and the toxicologically active lead burden” (Mushak 1992: 47). These biological media include blood, bones and teeth and are known as **biomarkers**. The public health significance of regular biomonitoring for exposure to lead and many other chemical pollutants is increasingly recognized. In 2001, the US Centers for Disease Control and Prevention (CDC), as part of NHANES, tested blood and urine for only 27 chemicals, but now tests for over 200 (Paustenbach

---

23 Other research suggests this effect is modified somewhat by calcium supplementation. Gulson et al. (2004) and Ettinger et al. (2004) observed low levels of lead in breast milk even for women with documented high lifetime exposures. For a good overview of this topic, see Bellinger (2005).
Figure 5.2: Generalized model of elemental exposure, uptake, tissue deposition, and biomonitoring. Postmortem chemical alteration and contamination from diagenesis and sample preparation are minimized with solid sample analysis of teeth (see chapter 6). Modified from Jones (2004) and Galbraith 2006). Figure 5.2 is a model of trace element flow from environmental sources to human biomarkers. Here, one sees that numerous sources potentially contribute to the skeletal deposition of environmental elemental exposures, which is mediated by various physiological factors.

Environmental monitoring may be preceded by emission and source distribution monitoring, and biomonitoring involves biological effect assessment (Mushak 1992). Barbosa et al. (2005) caution against conflating exposure and effect, or toxicity, biomarkers (see also Schmidt, 2006). Exposure is best measured as lead concentrations directly from tissues and fluids while biological effect is assessed via metabolite levels in fluids known to be influenced by
lead exposure. This distinction is important because associations between exposure, uptake and toxic effect are mediated through complex physiological processes that vary by age and sex among other factors. Depending upon these variables, similar exposures may produce dissimilar effects. The proposed study entails historical biomonitoring of lead exposure, although I will explore possible relationships between exposure and health trends for NYABG individuals.

Finally, an individual's body lead burden is a function of recent and past exposure and lead biokinetics, i.e., exchange or cycling among bodily fluids and tissues (Barbosa et al. 2005). Measuring variation in the sources, duration and intensity of exposure over time requires use of different biomarkers, which reflect different periods and/or rates of elemental exposure and uptake (Barbosa et al. 2005; Mushak 1992; Paustenbach and Galbraith, 2006; Sexton et al. 2004). Following is a brief overview of four primary biomarkers of lead exposure: blood, bone, tooth dentine and tooth enamel. Other biomarkers such as saliva, hair, nails, and urine are generally less temporally informative and, in most cases, not preserved in bioarchaeological populations.  

---

**Lead Biomonitoring**

**Blood**

Environmental lead enters the body primarily through ingestion into the gastrointestinal system, inhalation into the lungs, and dermal transfer (Ferguson 1990). Ingestion is the most common route, especially for children. Once absorbed, lead circulates via the bloodstream until its level plateaus, at which point it is excreted through the kidneys or deposited in soft tissues ("target organs"), bones and teeth (Baird 1999). Over 99% of circulating lead is located in the

---

24 Hair and nails are sometimes recovered, as some hair samples were at the NYABG.
whole blood compartment bound to erythrocytes (red blood cells) that have a lifespan of about 90-120 days (Paustenbach and Galbraith 2006). The remainder is found within plasma. The mean excretory half-life or "residence time" of whole blood-lead has been reported as approximately 35 days for healthy adult males, but longer for children and pregnant women due to bone remodeling (Rabinowitz et al. 1976). Blood-lead levels begin to rise within hours of increased uptake (Mushak 1992). Thus, blood-lead has long served as a biomarker of recent and short-term or transitory (e.g., acute) exposure. Indeed, as early as the 1890s, blood and urine of lead-exposed factory workers was screened as part of efforts to prevent acute lead poisoning (Sexton et al. 2004). Table 5.2 summarizes the benefits and limitations of blood and other biomarkers of lead exposure.

For about the last 50 years, whole blood-lead has been the most commonly used biomarker for exposure and toxicological research (Barbosa et al. 2005). During this time, advances in analytical chemistry included powerful new methods enabling reliable assessment of trace element concentrations and isotopic profiles directly from body tissues at extremely low detection limits (i.e., on the order of parts per billion [ppb] or lower). Among these, atomic absorption spectrometry (AAS) and ICP-MS are commonly used for lead research (ATSDR 2007). The interpretation of measured lead concentrations is complicated by several issues related to lead biokinetics. First, as noted in the previous section, lead deposited in bone may be remobilized into the bloodstream, and, thus, serves as a source of ongoing contamination. This is also the case for lead stored in soft tissues (Rabinowitz et al. 1976; O’Flaherty 1998). Hence, researchers today recognize blood-lead as an index of both recent and past exposure, and some attempt to assess long-term exposure (e.g., through serial sampling), as an elevated blood-lead value may reflect chronic high-level or a single acute exposure (Barbosa et al. 2005).
<table>
<thead>
<tr>
<th>Biomarker</th>
<th>Exposure</th>
<th>Benefits</th>
<th>Limitations</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood (whole)</td>
<td>Integrated recent and past/endogenous</td>
<td>Accurately, reliably measured; associated with toxic effects</td>
<td>Serial sampling required to discern chronic from acute exposure</td>
<td>Commonly used for screening, diagnosis and long-term biomonitoring</td>
</tr>
<tr>
<td>Blood (plasma and serum)</td>
<td>Integrated recent and past/endogenous</td>
<td>Exhibits rapid exchange with other fluids, tissues; increasingly reliable measurements</td>
<td>Difficult, costly and time-sensitive analysis; lack of certified reference materials</td>
<td>Possibly better index than whole blood-lead</td>
</tr>
<tr>
<td>Bones</td>
<td>Cumulative over years</td>
<td>Associated with toxic effects; in vivo analysis; preserves archaeologically</td>
<td>Turnover rates vary significantly by bone type, age and metabolic status; diagenesis</td>
<td>Increasingly used; more research required on remineralization (esp. for children)</td>
</tr>
<tr>
<td>Teeth</td>
<td>Cumulative during formation (enamel and primary dentine) and life of tooth (secondary dentine)</td>
<td>Easily collected, (exfoliated); little turnover/loss (esp. enamel); chronological data; preserves well archaeologically</td>
<td>Variation by tissue/region and type; age and/or sex influence not known; diagenesis (mostly dentine)</td>
<td>Probably best indicator of cumulative early life and childhood exposure; recent in vivo studies</td>
</tr>
<tr>
<td>Saliva</td>
<td>Ongoing, long-term</td>
<td>Easy, noninvasive collection; may correlate with blood- and urine-lead</td>
<td>Highly variable ion content over short periods; no reference range</td>
<td>Not widely accepted; further validation required</td>
</tr>
<tr>
<td>Hair</td>
<td>Cumulative during growth</td>
<td>Easy, noninvasive collection; low-cost analysis</td>
<td>Contamination; no reference range</td>
<td>Not yet widely used/accepted</td>
</tr>
<tr>
<td>Nails</td>
<td>Long-term</td>
<td>Easy, noninvasive collection</td>
<td>Age-related and intraindividual (including within-sample) variation</td>
<td>Not widely accepted; findings lack reproducibility</td>
</tr>
<tr>
<td>Urine</td>
<td>Ongoing, long-term</td>
<td>Easy, noninvasive collection; may correlate with plasma-lead</td>
<td>Highly variable over short periods, 24-hr sampling required</td>
<td>Commonly used for screening and long-term biomonitoring</td>
</tr>
<tr>
<td>Biomarker</td>
<td>Exposure</td>
<td>Benefits</td>
<td>Limitations</td>
<td>Comments</td>
</tr>
<tr>
<td>-----------</td>
<td>----------</td>
<td>----------</td>
<td>-------------</td>
<td>----------</td>
</tr>
<tr>
<td>Feces</td>
<td>Integrated dietary and environmental exposure <em>and</em> intake</td>
<td>Should indicate total body burden</td>
<td>Requires sampling over multiple days</td>
<td>May prove valuable; more info. on biliary physiology required</td>
</tr>
</tbody>
</table>


Second, in assessing current environmental conditions, it is important to establish the relative contributions of exogenous and endogenous exposures to current blood lead levels. This is a potentially difficult process as blood-lead exchange with other body compartments is continuous. The issue is further complicated by substantial variability of lead’s biokinetic properties with age, physiological and nutritional status, and possibly sex (Popovic et al. 2005). Fortunately, toxicokinetic studies involving stable isotope ratio analysis are helping to establish exchange rates between blood, bone and teeth. For example, Smith et al. (1996) determined that bone-lead contributed 40-70% of blood-lead for individuals without high-level exposure. Gulson et al. (1996) reported similar findings from a study of women who had recently emigrated from Eastern Europe to Australia. Gwiazda et al. (2005) found that bone-lead contributions to blood-lead may exceed 90% for children with elevated blood concentrations.

Lastly, current research takes into account specific kinetic properties of various blood subcompartments when monitoring for exposure, uptake or toxic effect. For example, although it accounts for less than 1% of total blood-lead, plasma/serum-lead may provide a "more relevant index of exposure to, distribution of, and health risks associated with lead than does BPb [or whole blood-lead]" (Barbosa et al. 2005: 1670). The plasma/serum component has the fastest exchange rate of all blood compartments and plasma-/serum-lead is toxicologically active while whole blood-lead is bound (Chuang et al. 2001). It is likely, then, that plasma/serum
concentrations are better indicators of lead levels in target organs such as brain and liver and, by extension, of toxic effect (Barbosa et al. 2005). Chuang and co-workers (2001) found that maternal plasma levels were more highly correlated with fetal lead exposure than whole blood-lead. The value of plasma-/serum-lead for biomonitoring is increasingly recognized, although its more involved, difficult analysis – and the need to further validate reported findings – has, thus far, served to limit its widespread use (Barbosa et al. 2005).

**Bone**

Vertebrate calcified tissues are "complex, composite structures" comprised of cellular, vascular, mineral, collagenous (except for enamel), and extracellular noncollagenous protein phases (Tuross 2003). Over the course of years, many elements are incorporated into the hydroxyapatite mineral component, which consists mostly of calcium phosphates and is usually represented in its simplified form as $\text{Ca}_5(\text{PO}_4)_{10}(\text{OH})_2$. These include major impurities such as carbonate (CO$_3$) as well as minor substitutions "from environmental contaminants, biological processes such as enzymatic reactions, and trace nutrient constituents" (Tuross 2003: 67).

Among the minor or trace substitutions are divalent cations such as lead (Pb$^{2+}$), strontium (Sr$^{2+}$), barium (Ba$^{2+}$) and zinc (Zn$^{2+}$), which may replace isovalent calcium sites through (1) adsorption onto crystal surfaces, (2) direct or coupled ion substitution, and (3) growth of discrete trace metal-phosphate phases (Webb et al. 2005; Trueman and Tuross 2002). Skeletal lead accounts for most of the body's lead burden; approximately 95% and 70% for adults and children, respectively (Barbosa et al. 2005). Losee et al. (1974; cited in Curzon 1983) reported that lead was one of 41 elements incorporated into dental enamel during development, and the only element with an atomic number greater than 60.
Lead and other "bone-seeking" elements are not evenly distributed throughout the skeleton (Wittmers et al. 1988), and bone and tooth levels may reflect exposure during different periods of an individual's lifespan. The specific information archived within a given tissue depends upon several related factors. For example, bone-lead accumulates with ongoing exposure and has a residence time of up to 30 years (Rabinowitz 1991), reflecting its unique crystalline and kinetic properties. Compared to those of dental tissues, bone apatite crystals are small (200-400 Å), less densely packed, and have large, highly reactive surface areas (Tuross 2003).

As noted above, bone remodels continuously, slowly releasing lead at a rate that varies by age and health and physiological status as well as bone type. Trabecular bone remodels five to ten times faster than cortical bone (Ortner and Turner-Walker 2003). Furthermore, bone apatite has a substantial organic component (i.e., about 22% by dry weight) (Hillson 1996), which serves as a ready source of biologically active lead for incorporation during remodeling. Thus, bone provides a cumulative and integrative record of lead exposure during the last approximately 10 years of life – roughly the time it takes to remodel a "new" skeleton – but is quite susceptible to foreign ion exchange. Tibia (cortical) and patella (trabecular) bone are commonly used in lead studies.

Bone-lead biomonitoring of contemporary populations has increased over the last decade with greater acceptance of X-ray fluorescence (XRF) for in vivo analysis (Barbosa et al. 2005). However, an important consideration in the use of archaeological remains is diagenesis, the postmortem physical and chemical alteration that occurs within burial contexts. Simply put, processes that introduce lead and other elements into living bone may continue postmortem via water and soil contact and microbial invasion, resulting in net loss or gain of skeletal lead (Trueman and Tuross 2002). Diagenesis is not easily distinguished from biogenic signals of
interest (Sillen et al. 1989), and some researchers who pioneered the anthropological study of bone-lead have cautioned strongly against its use (Wittmers et al. 2002, 2008).

**Teeth**

Dental chemistry offers a means of overcoming the potentially confounding effects of diagenesis and provides alternative chronologies of elemental exposure that complement the information recorded in bone. From a biomonitoring perspective, each tooth is an archive or a capsule of "snapshots" revealing elemental and isotopic exposure during early life development (Grupe 1998; Webb et al. 2005). Teeth consist of 3 incrementally-formed tissues that vary in the amount and composition of their mineral components. **Dentine** surrounds the innervated pulp cavity and comprises the majority of the tooth, serving as its main force-bearing structure. A thin layer of dental cement, or **cementum**, covers the dentine in the root portion of the tooth. The primary function of cementum is to attach the periodontal ligament to the root's surface.

Unlike bone, dentine and cementum are avascular and do not remodel continuously (Hillson 1996). The proportion of organic content in dentine and developing cementum is similar to that of bone; approximately 20% and 25% by dry weight, respectively. This amount varies substantially in mature cementum (Hillson 1996). Dentine and cementum undergo remineralization and post-formative (including diagenetic) lead incorporation, albeit at a slower rate than bone. Both tissues record integrative and cumulative exposure. Epidemiological studies have tended to focus on dentine in order to understand the consequences of ongoing exposure in contemporary populations (Grandjean et al. 1984; Rabinowitz et al. 1991; Needleman and Bellinger, 1991; Fergusson et al. 1997).²⁵

²⁵ A notable early exception is Purchase and Fergusson's (1986) study of variation in lead concentration within and between dentine and enamel, in which the authors found the enamel to dentine to secondary dentine ratio to be 1:2:6. Secondary (or circumpulpal) dentine is the
This study involves analysis of **enamel**, the visible outer layer of the tooth crown. Mature enamel is avascular, acellular, almost totally (>96%) inorganic, and basically does not remodel. Enamel is the hardest and most heavily mineralized tissue in the body. These properties confer resistance to *in vivo* chemical alteration upon ameloblast degeneration, as well as to the fungi and bacteria that transport soil cations to skeletal tissues in burial contexts, so that enamel resists diagenesis (Grupe 1998; Budd et al. 2000; Lee-Thorp and Sponheimer 2003). The exception to this generalization is that the outermost enamel is subject to some degree of remineralization as it is "regularly challenged by acid from plaque bacterial metabolism" in the oral environment (Curzon and Featherstone 1983: 131). Reitznerová et al. (2000) determined that this remineralization zone is limited, extending at most 150 μm from the enamel surface.

*Thus, dental enamel is a virtually permanent record of elemental exposure during crown formation, the timing of which varies by tooth and occurs in well-documented stages.* In humans, these stages extend from the prenatal period through adolescence, when third molar crowns form (Massler et al. 1941), and variation in the developmental timing of dental enamel across human populations is minimal (Goodman and Song 1999; Reid and Dean 2006). The stable biogenic signals found in enamel may be compared for different teeth to reconstruct dietary regimes and other aspects of trace metal exposure at different ages (Prowse et al. 2007). Combined with biomarkers of lifetime or more recent exposure, enamel is the basis for estimating natality and establishing elemental and isotopic "life history trajectories" (Bower et al. 2007; Cox and Sealy, 1997; Cucina et al. 2005; Price et al. 2006; Prowse et al. 2007). Enamel-lead has been analyzed to understand changes in atmospheric exposure over time (Budd et al. thin, innermost layer, which lines the pulp cavity and accumulates lead from the completion of root formation through the life of the tooth.
Consequences of Lead Burden

Lead is highly toxic to humans in all its chemical forms. Lead poisoning has been recognized since antiquity and the subject of medical inquiry since the 18th century (McCord 1953, 1954; Nriagu 1983; Aufderheide 1993; Hernberg 2000). Lead's toxic effects are dose-dependent and determined by its source and form as well as the age and health status of the affected individual. Modern standards recognize two "polarities": chronic versus acute, and clinical versus subclinical lead poisoning (Warren 2000).

Prior to the 1940s, occupational "plumbism" was the focus of nearly all research on lead toxicity, which was identified only by its wide-ranging clinical manifestations (Warren 2000). With low-level exposure, symptoms include headaches, constipation and slowed nerve conduction. At blood-Pb levels of 80 μg dL⁻¹ or greater, symptoms include lethargy, encephalopathy, convulsions, coma, peripheral neuropathy and even death. Within these extremes, lead impairs numerous aspects of growth, development and functioning such as hearing, vitamin D and zinc metabolism, erythrocyte production and hemoglobin synthesis (Hu 2002). According to Aufderheide (1993: 820), "The symptom of lead poisoning most commonly encountered in the historical literature is abdominal pain... usually attributed to intestinal spasm, though the abdominal muscles may participate in the painful, uncontrolled contractions usually termed 'colic'." Such acute lead poisoning is now relatively uncommon. When encountered, it is usually treated with chelation therapy. Chelation involves administration of compounds (e.g., ethylenediaminetetraacetic acid [EDTA]) that attract lead more strongly than do target enzymes, so that upon their excretion, lead is expelled as well (Baird 1999). Chelation
is often followed by an immediate drop in an individual's blood-lead concentration. However, levels tend to rise gradually with ongoing exposure or as stored lead is released from tissues back into the bloodstream (Warren 2000).

Most current research focuses on pediatric lead poisoning and, increasingly, on the cognitive effects of low-level exposure (Needleman 1998; Silbergeld 1997), although Spivey (2007) notes a reemerging concern with adult exposure. As explained above, children are most susceptible to lead poisoning due to their exploratory behavior and rapid physiologic activity, which increases the rate of absorption and metabolism of bioavailable lead. The fetus is especially vulnerable to neurological insult from transplacental lead transfer due to immaturity of its blood-brain barrier (Goyer 1990, 1996). In adults, chronically elevated lead levels have been positively associated with presence of dental caries (Moss et al. 1999); risk of hypertension (Cheng et al. 2001; Rothenberg et al. 2002; Vupputuri et al. 2003); male infertility (Hu 2002); and renal failure (Spivey 2007). High maternal lead levels may increase risk of preterm and low birth weight pregnancy (Schell and Denham 2003). As with other toxicants, males may be more sensitive to lead (Needleman and Bellinger 1991).

The underlying mechanisms of lead's toxic effects are not fully understood. Much of what is known regarding these mechanisms comes from experimental animal studies. Lead and other "calcium impostor" cations share a strong affinity for sulfur, competing with and replacing calcium at sulfhydryl sites on proteins, with the effect of inhibiting and retarding enzymatic processes that control the rate of critical metabolic reactions (Baird, 1999). Among its many other effects, lead inhibits heme synthesis and calcium entry into cells (contributing to apoptosis, or cell death) and distorts neurotransmission (Needleman, 2004). One recent experimental study suggests that lead impairs cognitive development by stifling neurogenesis in the hippocampus, a region of the brain important for learning and development. Verina et al.
(2007) found that hippocampal neurons were fewer and less likely to be replaced in lead-
exposed rats. Further, the lead-exposed rats' neuronal processes (dendrites) were short, twisted
and less densely connected when compared to untreated rats, which resulted in subsequent
communication and learning difficulty. In a recent study of non-occupationally exposed adults,
Weisskopf et al. (2007) found that hippocampal glial effects may be more sensitive than
neuronal effects to cumulative lead exposure as indicated from bone-lead concentrations.

Finally, lead's toxic effects are modified by dietary practice and nutritional status. These
relationships are influenced by nutrient interactions and other physiological factors, and thus
are not easily interpreted. For example, while lead level may be positively related to dietary
quantity (i.e., total caloric intake) due to lead ingestion (Schell et al. 2003), lead absorption and
tissue retention is enhanced with reduced caloric intake (Mahaffey 1990). Lead absorption and
retention also increase with poor dietary quality (i.e., caloric sources). Experimental and human
studies suggest that lead levels are inversely related to calcium, iron, phosphorus, zinc, vitamin
(C, D and E) and perhaps other nutrient intake (Mahaffey 1990; Yip 1990; Cheng et al. 1998;
Schell et al. 2003; Kemp et al. 2007). Maternal diet, nutrition and anthropometric attributes also
affect lead levels for very young children (Schell et al. 2004).

Today, lead poisoning is defined as the presence of elevated blood-lead levels. However,
establishing what constitutes an "elevated" level is a controversial and unresolved matter. The
framing of lead exposure and lead poisoning as public health issues has major social and
economic implications, as evidenced by resistance to regulatory efforts by the lead industry, real
estate and insurance interests, and others (Needleman 1998). Until 1970, the blood-lead level of
concern was 30 μg dL⁻¹ or greater. In 1991, the CDC action level for exposure among children
was set at 10 μg dL⁻¹. As evidence mounted for cognitive and behavioral impacts below this
level, the CDC in 2012 established the current “upper reference range value” of 5 μg dL⁻¹ (1). The
Occupational Safety and Health Administration (OSHA) threshold for exposed workers is 40 μg dL⁻¹. These action levels, representing the lowest clinically significant blood-lead levels, are contested. Quite possibly, there is no actual threshold at which absorbed lead does not result in some form of biological impairment (Spivey, 2007).

**Lead in African Diasporic Biohistory**

Sources of lead exposures have been well-documented only since the 1970s and must be inferred for historical populations from studies of past dietary, health and labor practices (Warren, personal communication). Much of what is known of lead’s role in African diasporic biohistory is derived from skeletal research, mainly at plantation sites (e.g., Handler et al. 1986; Corruccini et al. 1987; Aufderheide et al. 1988). The specific value of lead research in African diasporic bioarchaeology lies in the fact that lead use was much more pervasive in early New York and throughout the colonial Americas than in contemporaneous western Africa. Thus, studies of lead exposure may also provide information on geographic origins. Here, I summarize key anthropological findings.

Bioarchaeological research indicates that among enslaved and captive Africans’ new experiences in the Americas was increased lead exposure. Studies of lead and North American slavery have been conducted at historic plantation sites in the South. For example, Rathbun (1987) analyzed remains of enslaved individuals unearthed at a construction site near Charleston. Mean lead concentration was higher for males (102.7 μg g⁻¹ compared with 87.1 μg g⁻¹ for females). Overall, these values are higher than those reported for other African American skeletal populations and slightly lower than reported for the Newton plantation.
Aufderheide et al. (1981, 1985) measured bone-lead concentrations for remains of free and enslaved Africans and whites buried in 17th to 19th-century Virginia, Maryland and Georgia. Skeletal lead patterns proved useful for identifying subgroups within these populations. At the Clifts Plantation in Virginia, remains of wealthy slaveowners produced the highest mean values (185 μg g⁻¹) and those of enslaved Africans, the lowest (35 μg g⁻¹). At the College Landing site, also in Virginia, mean lead concentration for free blacks (41.9 μg g⁻¹) was comparable to that of enslaved individuals at the Clifts plantation, but more variable, ranging from 9.9 to 93.3 μg g⁻¹. Aufderheide et al. (1988) suggest that skeletal lead may be used (1) to assess the extent of lead technology in a cultural group; (2) to separate socioeconomic subgroup within a population; (3) identify specific individuals' social or occupational roles; (4) to assist in separating mixed skeletal tissues; (5) to predict health effects; and (6) to identify remains as ancient or modern.

In the most complete study of lead exposure and its health consequences for enslaved Africans, Jerome Handler and co-workers (1986; Corruccini et al. 1987) found evidence of a "previously unappreciated epidemic" at the mid-17th to early 19th-century Newton Plantation cemetery in Barbados. Atomic absorption spectrometry of cortical bone samples from 48 individuals yielded lead concentrations of 0 to 424 μg g⁻¹, the widest range of any population in the Americas. Mean lead concentration was 118 μg g⁻¹, comparable to values found for Romans and mainland North American slaveowners for whom high lead exposure is well-documented (Waldron et al. 1976), and considerably higher than those found for most other enslaved populations (Aufderheide et al. 1981). Sex differences were observed but not statistically significant. Female lead levels were generally higher and more variable, possibly due to their greater representation among domestic workers with easier access to pewter items.

Enslaved Barbadians' widely ranging bone-lead concentrations likely corresponded to diverse health effects. Handler et al. (1986) compared Newton bone values to those of living
individuals for whom bone- and blood-lead concentrations were correlated to lead poisoning symptoms. The authors concluded that most enslaved individuals experienced mild symptoms of lead toxicity while others would have suffered severe intestinal, nervous, and cognitive dysfunction. Lead accumulation may have reflected length of residence in Barbados as age-at-death correlated positively with lead content. The authors suggest that lead fumes from the refining of sugar and rum contaminated by lead-based distillation machinery were the major exposures for enslaved Africans and poor Europeans (Handler et al. 1986; Corruccini et al. 1987; Wittmers et al. 2002).

All Newton individuals determined to be African-born had low bone-lead concentrations. Corruccini et al. (1987: 237) suggested low lead content as one aspect of a "burial trait complex" indicating African natality. This complex also included "dental modification and tooth root hypercementosis... associated with north-headed burial orientation and/or absence of a coffin." Individuals with dental modifications had relatively low mean lead concentrations – 44.7 μg g⁻¹ versus 126.2 μg g⁻¹ for non-modified individuals – and none were particularly young at death (Corruccini et al. 1987).

Lead played important roles in industry and health elsewhere in the Caribbean. In his study of New Montpelier village, a Jamaican plantation site dating from 1739 to 1912, Higman (1998: 216) observes that

Lead was almost as common as iron.... It occurred in sheets, rings, cones, pipes, balls, and lumps. Most of this lead probably started its use-life in the estate' boiling house or distillery, where its malleability made it a preferred material for many purpose, particularly piping to carry liquids. In this way it contributed heavily to the mortality of slave and free alike by poisoning the water, rum and cane juice which they drank. The uses of lead in the village community included sinkers for fishing, musket ball, and personal adornment; the easy working characteristics of the metal and its ready availability must have made it an attractive material for kitchen and yard.
Lead 'bracelets,' possibly sawn from the ends of pipes, were also excavated. Jamaica and Barbados contributed considerably to New York's African population when both the Newton and New Montpelier village cemeteries were in use.

Thus, bioarchaeological evidence has proven critical for generating interest in lead as one of the many "life stresses of slavery" (Kelley and Angel 1987). As Rathbun (1987) observes, comparison of findings across sites should take into account differences in soil chemistry, sampling strategy, and analytical methods. One may safely conclude, however, that lead exposure and toxicity among enslaved individuals would have resulted from a wide range of practices and experiences. Some were common to the colonial or colonial African situation, while others were unique within the varied political economies and cultural geographies of American slavery. The remaining chapters begin to illuminate and situate the experiences of enslaved New Yorkers within this broader environmental biohistory of the African diaspora.
CHAPTER 6

MATERIALS AND METHODS

Introduction

This chapter begins with a brief introduction to laser ablation-inductively coupled plasma-mass spectrometry (LA-ICP-MS), the technique employed for chemical analysis in this study. I describe the basic underlying principles and key components of LA-ICP-MS and some of its recent anthropological and archaeological applications. In the next section, I explain the sample selection process including the rationale for comparing enamel-lead concentrations for subadults, adults with dental modification, and adults without modified teeth. I then detail the methodology developed for this study, the first to combine quantitative LA-ICP-MS measurement with recent developments in histology to produce age-resolved microspatial distribution profiles of early-life lead burden.

Laser Ablation-Inductively Coupled Plasma-Mass Spectrometry (LA-ICP-MS)

ICP-MS is a widely used tool for elemental and isotopic measurement. The basic mechanics of ICP-MS include three principal components: 1) sample introduction (as solution, vapor, solid or slurries); 2) sample atomization, ionization and excitation (usually by argon plasma); and 3) ionic separation by quadrupole or magnetic sector mass analyzer where analyte ions are separated according to their mass-to-charge ratios (m/z) (Günther and Hattendorf 2005). The analyte ion signal is plotted against the m/z ratio, and this plot is called mass spectrometry. Using a series of known analyte standards, the instrument can be calibrated. This calibration function is then used to determine the concentration of an unknown analyte (i.e., lead). Rapid sample throughput and high sensitivity (below parts per billion, or ppb) capabilities have made digestion or solution-based ICP-MS the analytical “gold standard” in the earth and
environmental sciences (Pollard et al. 2007). However, “bulk analysis” through acid digestion may entail loss of valuable sample and information. LA-ICP-MS provides a solution to this problem and other unique benefits and, since its introduction in 1985, has developed into a reliable method for analysis of diverse environmental and biological materials.

LA-ICP-MS began as a tool for "exploratory geochemistry" where in situ analytical capability proved ideal for identifying mineral inclusions useful in provenance studies employing elemental "fingerprinting" or signature analysis (ESA) (Outridge 1996; Gray 1985). Nearly a decade ago, Durrant and Ward (2005: 821) noted that laser ablation of solids had become “routine for many applications.” Like other ICP-MS techniques, LA-ICP-MS provides highly sensitive analysis, with lower limits of detection (LODs) reported in the parts per million (μg g⁻¹) to ppb range (Resano et al. 2010). LA-ICP-MS is distinguished, however, as a microprobe technique that enables both surface area and depth profiling of solid materials (Kang et al. 2004). With this method, sampling occurs when a high-energy pulsed laser interacts directly with a solid. Spatial distribution profiling is enabled by placing the sample within a cell on a movable, computer-controlled stage where minute amounts of the sample are vaporized along a pre-programmed track and introduced into the ICP-MS unit via an argon gas stream.

LA-ICP-MS analytical performance (e.g., LOD) can vary considerably depending upon various factors including sample matrix, analyte, ICP-MS type, laser characteristics and optimization, and desired spatial resolution (affecting the amount of sample ablated) (Durrant and Ward 2005). The major limitation of LA-ICP-MS relative to solution-based analysis is the difficulty of quantifying measurements. This process can be complicated by non-uniform ablations and, especially, the lack of solid, matrix-matched standard reference material (SRM) calibrants with certified values (Belloto and Miekeley 2000; Günther et al. 2000). Data initially collected in the form of ion intensity counts per second (cps) signals may be background-
subtracted and normalized to an internal standard (such as $^{43}$Ca in teeth), with accuracy of ± 30-50% (Amarasiriwardena et al. 1997). While this level of uncertainty may be acceptable for screening purposes in qualitative or semi-quantitative analysis (Habicht-Mauche et al. 2002), quantified concentrations must be obtained via element-for-element, response curve or internal calibration (Durrant and Ward 2005; Bellis et al. 2006, 2009).

Researchers have made significant progress in overcoming the analytical challenge of converting intensity counts to concentrations for lead measurement by LA-ICP-MS. In an early attempt at quantitative LA-ICP-MS measurement, Uryu et al. (2003) reported good agreement for human enamel-lead concentrations derived from LA-ICP-MS and digestion methods (ICP-MS and ICP-atomic emission spectrometry [AES]). However, this study involved one-point calibration with an "in-house" reference material (pelletized chicken bone). Bellis et al. (2006) achieved true quantitative measurement of lead in bone through calibration with multiple-level, candidate reference materials produced from physiologically-enriched, ground bone of lead acetate-dosed cows and goats. The caprine (goat) reference materials were produced as part of a controlled study by the New York State Department of Health.

Despite the challenge of quantifying intensity counts as concentrations, various unique capabilities make LA-ICP-MS an attractive alternative to destructive bulk analytical techniques. Compared to solution-based ICP-MS, for example, laser sampling enables higher sample throughput with less preparation and contamination risk, and is influenced by fewer polyatomic interferences that result from interaction of water and acid with the argon plasma (Belloto and Miekeley, 2000). The separation of the sampling and ionization steps with LA-ICP-MS grants independent control of each process such that laser operating parameters including the pulse mode and repetition rate, energy level and degree of focus may be optimized according to specific research goals (Denoyer et al. 1991). Advances in laser technology are allowing for
increasingly high-resolution spatial analysis and ablation craters of less than 10 μm in diameter are now attainable. Thus, laser ablation is minimally disruptive, often allowing repeat analyses even when sample integrity and preservation are primary concerns. Finally, laser ablation does not require loss of valuable chronological data stored in teeth and other incremental hard tissues as does sample digestion. Rather, LA-ICP-MS provides information on the microspatial distribution of elements and isotopes.

With these unique capabilities, LA-ICP-MS is perfectly suited for archaeological and biomonitoring research. Archaeologists have used this method to characterize and source precious metals, ceramics, obsidian flakes, coins and other heterogeneous matrices, including glass beads, which are difficult to analyze by traditional means such as XRF (Eastwood et al. 1998; Guerra et al. 1999; Habicht-Mauche et al. 2002; Speakman and Neff 2002, 2005). In a recent study, Speer (2014) combined LA-ICP-MS with multivariate statistical analysis to determine the local versus nonlocal sources of chert artifacts in a study of hunter-gatherer mobility during the Clovis period.

This method has been used to investigate the spatial distribution of various trace metals across different regions of incrementally-formed hard tissue structures including fish otoliths (Outridge et al. 2002), shells (Perkins et al. 1991; Fuge et al. 1993; Belloto and Miekeley 2000), hair (Steely et al. 2007; Byrne et al. 2010; Bartkus et al. 2011), and teeth (Budd et al. 1998; Farell et al. 2013; Humphrey et al. 2008; Kang et al. 2004; Dolphin et al. 2005; Arora et al. 2006; Bellis et al. 2009). In the first published LA-ICP-MS environmental study, Perkins et al. (1991) reported that lead levels increased when sampling inner to outer layers of marine bivalve shells, indicating increased pollutant exposure over time. Evans et al. (1995) and Outridge et al. (1995) found that arctic walrus and beluga whale cementum layers formed during early life had higher
lead, zinc and copper content than those formed later, possibly indicating decreased metal intake with the shift from reliance on maternal milk.

LA-ICP-MS studies of human trace metal exposure often focus on lead given the element’s longstanding and well-documented environmental health impact. Cox et al. (1996) first analyzed human teeth with LA-ICP-MS to reconstruct pollutant exposure, comparing teeth from contemporary Poland and 19th-century Spitzenberg (northern Scandinavia and Russia). The authors suggest that higher lead and tin levels in the older tooth may reflect the use of pewter utensils. In a study of lead, strontium, tin and zinc in incisors from lead acetate-dosed rats and deciduous human teeth, Lee et al. (1999: 182) conclude that "lead is incorporated into the hydroxyapatite (enamel) under formation at the time of injection and for the period of time afterwards when lead is still available in the body." They reported $^{43}$Ca normalized elemental intensities. Calcium is the major element in dental tissues (~37% enamel and ~27% dentine, dry weight) and $^{43}$Ca is free of isobaric and polyatomic interferences, making it an ideal internal standard for tooth (and bone) analysis. Enamel-lead was among the trace metals analyzed by Cucina et al. (2005) in their study of geographic origins of enslaved Africans in colonial Campeche, Yucatan.

Many early studies compared elemental compositions across tooth tissues and regions, especially pre- versus postnatal formations. For example, comparing intensity counts for 14 elements in human deciduous teeth, Lochner et al. (1999) observed increased levels of lead and most other elements in postnatal tissue formations. Kang et al. (2004) rastered 196x339 μm$^2$ areas including pre- and postnatal enamel, the neonatal line (enamel), enamel-dentine junction (EDJ), primary dentine, and the dentine-pulp junction. They found that lead and zinc levels were highest in the pulp region and that the order of magnitude for $^{43}$Ca normalized intensities followed a general pattern: Sr > Mg >> Zn > Pb > Fe > Cu. An earlier study by Budd and co-
workers (1998) identified significant and similar differences between enamel and dentine elemental intensities for modern and archaeological teeth including elevated Pb/Ca levels for the EDJ and surface enamel (SE). The observation of surface Pb/Ca peaks for modern teeth led them to conclude that these were not diagenetic in nature, but the result of lifetime effluvial interaction. Budd et al. (1998) also report good agreement between Pb/Ca findings of LA-ICP-MS and lead concentrations measured by isotope dilution-thermal ionization mass spectrometry (ID-TIMS).

Arora et al. (2006) measured pre- and postnatal enamel and dentine lead in deciduous teeth from children enrolled in the Broken Hill Lead Management Program in New South Wales, Australia. Enamel-lead values were consistently low, but the authors reported a significant increase in mean postnatal dentine lead levels, which they attribute to increases in blood-lead levels from birth to age 1. Interestingly, in light of dentine’s remineralization properties (see chapter 5), they suggest that dentine-lead is a valid biomarker for reconstructing pre- and neonatal lead exposure. Assuming the roles of historical detectives, Stadlbauer et al. (2007) quantitatively measured Pb, Cr, Hg, As and Sb concentrations from hair, bones and tooth enamel in an attempt to authenticate the "Mozart cranium." Trace elemental analysis proved insufficient for this task but revealed different patterns of heavy metal uptake reflecting changes in background environmental (Pb) and medical (As, Hg, Pb and Sb) exposures over the last several centuries.

I conclude this section with some examples of LA-ICP-MS application within biological anthropology. Such examples are limited in number, undoubtedly due in part to the limited availability of necessary equipment. As costs decrease and analysis becomes more automated, one would expect that LA-ICP-MS analysis will evolve into a regular, if not routine, feature of bioarchaeometric research, with implications for expanding the boundaries of knowledge.
concerning the recent human past and even early human evolution. For example, Sponheimer et al. (2006) used laser sampling to reconstruct seasonal dietary behavior and climatic conditions for South African australopithecines (*Paranthropus robustus*). Carbon ($\delta^{13}\text{C}$) and oxygen ($\delta^{18}\text{O}$) isotopic enamel profiles suggest *Paranthropus*, like early *Homo* species, employed flexible dietary strategies incorporating forest- and savanna-based foods. Their findings challenge the well-established hypothesis that *Paranthropus* overreliance upon savanna-based foods contributed to its extinction during the late-Pleistocene when savanna environments became increasingly arid and seasonal.

Several studies conducted at Hampshire College under the direction of Drs. Dula Amarasiriwardena and Alan Goodman illustrate the potential of LA-ICP-MS analysis for opening new avenues of biocultural inquiry into health conditions. In her doctoral dissertation, Song (2004) explored ancient Mayan infant weaning and dietary practices, finding cultural continuity with modification (i.e., extended breastfeeding duration) following contact with colonial Spaniards. In a longitudinal study of the functional consequences of mild-to-moderate malnutrition, Goodman et al. (2003) correlated lead intensities of prenatal enamel with reduced height and weight in the Sólis Valley (Mexico). In a follow-up paper, Dolphin et al. (2005) reported that intensities for lead and nutritionally significant elements (Fe, Zn and Ba) were generally higher and more variable in postnatal enamel. Magnesium values were lower for postnatal enamel and strontium values for the two regions did not vary significantly. Finding an inverse relationship between maternal consumption of foods with high zinc bioavailability and Zn/Ca ratios in prenatal enamel of their infants, Dolphin and Goodman (2009) conclude that zinc is not a reliable indicator for paleodiet reconstruction.

The growing body of work involving LA-ICP-MS analysis of human tissues reflects the method’s value for excavating lifespan experiences and life history events. Spatial elemental
analysis is especially important for uncovering complex experiences embedded and often hidden, until now, within incrementally-formed bones and teeth. I now refocus attention on the New York African Burial Ground. The remainder of this chapter details the methodology developed for quantitative measurement of lead and the construction of chronological age-exposure profiles from the teeth of children, women buried there.

**Study Sample**

The study sample includes NYABG teeth and an archaeological control from Kasana (northern Ghana). The NYABG sample set was selected from teeth located at Hampshire College, the site of ongoing chemical and histological analysis under the direction of Dr. Alan Goodman, Director for Chemical Studies of the African Burial Ground Project. Hampshire College currently houses samples of 456 teeth from 122 NYABG individuals. From these, initially 62 permanent teeth from 45 NYABG individuals were selected for analysis, including subadults, “modified” adults (i.e., with CMT) and “non-modified” adults (i.e., without CMT). The rationale for investigating these subsamples and tooth types is given below. Two teeth from Burial 281, a modified male of indeterminate age, showed evidence of diagenesis and were excluded from the study. Listed in Table 6.1, the complete study sample thus consists of 61 teeth from 45 individuals:

1) NYABG subadults (n=11 teeth/11 individuals);
2) NYABG adults with CMT (n=28 teeth/19 individuals);
3) NYABG adults without CMT (n=21 teeth/14 individuals); and
4) Kasana adult (control) (n=1 tooth).

NYABG subadults and modified adults are critical to this study as they potentially represent American and African birth cohorts, respectively. The ratio of African- to American-
Table 6.1: Study sample

<table>
<thead>
<tr>
<th>Burial</th>
<th>a Tooth/Teeth</th>
<th>b Age (y)</th>
<th>c Sex</th>
<th>d Cohort</th>
<th>e Temporal Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subadults</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>LRM1</td>
<td>4.0</td>
<td>u</td>
<td>SA</td>
<td>Late-Middle</td>
</tr>
<tr>
<td>22</td>
<td>LRM1</td>
<td>3.5</td>
<td>u</td>
<td>SA</td>
<td>Middle</td>
</tr>
<tr>
<td>35</td>
<td>ULM1</td>
<td>9.0</td>
<td>u</td>
<td>SA</td>
<td>Middle</td>
</tr>
<tr>
<td>39</td>
<td>LRM1</td>
<td>6.0</td>
<td>u</td>
<td>SA</td>
<td>Middle</td>
</tr>
<tr>
<td>43</td>
<td>LRM1</td>
<td>3.5</td>
<td>u</td>
<td>SA</td>
<td>Late-Middle</td>
</tr>
<tr>
<td>126</td>
<td>LLM1</td>
<td>4.5</td>
<td>u</td>
<td>SA</td>
<td>Middle</td>
</tr>
<tr>
<td>138</td>
<td>URM1</td>
<td>4.0</td>
<td>u</td>
<td>SA</td>
<td>Late</td>
</tr>
<tr>
<td>180</td>
<td>ULM1</td>
<td>12.0</td>
<td>f</td>
<td>SA</td>
<td>Late</td>
</tr>
<tr>
<td>219</td>
<td>LRM1</td>
<td>4.5</td>
<td>u</td>
<td>SA</td>
<td>Late-Middle</td>
</tr>
<tr>
<td>244</td>
<td>LLM1</td>
<td>7.0</td>
<td>u</td>
<td>SA</td>
<td>Late</td>
</tr>
<tr>
<td>405</td>
<td>URM1</td>
<td>8.0</td>
<td>u</td>
<td>SA</td>
<td>Middle</td>
</tr>
<tr>
<td>Modified adults</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>LLM1, ULM3</td>
<td>27.5</td>
<td>m</td>
<td>MA</td>
<td>Late</td>
</tr>
<tr>
<td>9</td>
<td>LLM1, LRM3</td>
<td>40.0</td>
<td>m</td>
<td>MA</td>
<td>Middle</td>
</tr>
<tr>
<td>23</td>
<td>URM1</td>
<td>40.0</td>
<td>m</td>
<td>MA</td>
<td>Early</td>
</tr>
<tr>
<td>47</td>
<td>LLM1</td>
<td>40.0</td>
<td>m</td>
<td>MA</td>
<td>Middle</td>
</tr>
<tr>
<td>68</td>
<td>LRM3</td>
<td>23.0</td>
<td>m</td>
<td>MA</td>
<td>Early</td>
</tr>
<tr>
<td>101</td>
<td>LRI1, LLM3</td>
<td>30.5</td>
<td>m</td>
<td>MA</td>
<td>Late-Middle</td>
</tr>
<tr>
<td>106</td>
<td>LRM1, LRM3</td>
<td>30.0</td>
<td>f</td>
<td>MA</td>
<td>Late-Middle</td>
</tr>
<tr>
<td>115</td>
<td>LLM1, LRM3</td>
<td>30.0</td>
<td>f</td>
<td>MA</td>
<td>Middle</td>
</tr>
<tr>
<td>151</td>
<td>LRM1</td>
<td>40.0</td>
<td>m</td>
<td>MA</td>
<td>Late</td>
</tr>
<tr>
<td>165</td>
<td>LLM1, LRM3</td>
<td>u</td>
<td>u</td>
<td>MA</td>
<td>Late</td>
</tr>
<tr>
<td>241</td>
<td>URI1</td>
<td>60</td>
<td>f</td>
<td>MA</td>
<td>Late</td>
</tr>
<tr>
<td>243</td>
<td>ULI1, URM3</td>
<td>45</td>
<td>m</td>
<td>MA</td>
<td>Late</td>
</tr>
<tr>
<td>266</td>
<td>URM1, URM3</td>
<td>30.0</td>
<td>f</td>
<td>MA</td>
<td>Late</td>
</tr>
<tr>
<td>270</td>
<td>LLM1, LLM3</td>
<td>u</td>
<td>m</td>
<td>MA</td>
<td>Middle</td>
</tr>
<tr>
<td>281</td>
<td>ULM1, ULM3</td>
<td>u</td>
<td>m</td>
<td>MA</td>
<td>Early</td>
</tr>
<tr>
<td>366</td>
<td>LLM1</td>
<td>u</td>
<td>u</td>
<td>MA</td>
<td>Middle</td>
</tr>
<tr>
<td>367</td>
<td>ULM1</td>
<td>30.0</td>
<td>f</td>
<td>MA</td>
<td>Middle</td>
</tr>
<tr>
<td>377</td>
<td>ULM1</td>
<td>45.2</td>
<td>f</td>
<td>MA</td>
<td>Late-Middle</td>
</tr>
<tr>
<td>384</td>
<td>ULM1</td>
<td>35.0</td>
<td>f</td>
<td>MA</td>
<td>Middle</td>
</tr>
<tr>
<td>397</td>
<td>ULM1</td>
<td>35.0</td>
<td>f</td>
<td>MA</td>
<td>Middle</td>
</tr>
<tr>
<td>Non-modified adults</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>ULM1</td>
<td>40.0</td>
<td>f</td>
<td>NMA</td>
<td>Late</td>
</tr>
<tr>
<td>25</td>
<td>URI1</td>
<td>22.0</td>
<td>f</td>
<td>NMA</td>
<td>Middle</td>
</tr>
<tr>
<td>25</td>
<td>LRM3</td>
<td>22.0</td>
<td>f</td>
<td>NMA</td>
<td>Middle</td>
</tr>
<tr>
<td>49</td>
<td>ULM1</td>
<td>45.0</td>
<td>f</td>
<td>NMA</td>
<td>Middle</td>
</tr>
<tr>
<td>63</td>
<td>LRM3</td>
<td>40.0</td>
<td>m</td>
<td>NMA</td>
<td>Late</td>
</tr>
<tr>
<td>135</td>
<td>LRM3</td>
<td>35.0</td>
<td>m</td>
<td>NMA</td>
<td>Late</td>
</tr>
<tr>
<td>150</td>
<td>LRM1</td>
<td>24.0</td>
<td>f</td>
<td>NMA</td>
<td>Late</td>
</tr>
<tr>
<td>172</td>
<td>LLM1, LLM3</td>
<td>30.0</td>
<td>f</td>
<td>NMA</td>
<td>Late</td>
</tr>
<tr>
<td>176</td>
<td>LRM1, LLM3</td>
<td>22.0</td>
<td>m</td>
<td>NMA</td>
<td>Late-Middle</td>
</tr>
<tr>
<td>Burial</td>
<td>Tooth/Teeth</td>
<td>Age (y)</td>
<td>Sex</td>
<td>Cohort</td>
<td>Temporal Group</td>
</tr>
<tr>
<td>--------</td>
<td>-------------------</td>
<td>---------</td>
<td>-----</td>
<td>--------</td>
<td>----------------</td>
</tr>
<tr>
<td>179</td>
<td>LRM1, LLM3</td>
<td>27.5</td>
<td>m</td>
<td>NMA</td>
<td>Late</td>
</tr>
<tr>
<td>196</td>
<td>LRM1</td>
<td>22.0</td>
<td>u</td>
<td>NMA</td>
<td>Late</td>
</tr>
<tr>
<td>262</td>
<td>LRM3</td>
<td>16.0</td>
<td>m</td>
<td>NMA</td>
<td>Late</td>
</tr>
<tr>
<td>323</td>
<td>LRM1, LRM3</td>
<td>24.5</td>
<td>m</td>
<td>NMA</td>
<td>Late</td>
</tr>
<tr>
<td>324</td>
<td>ULM1, LRM3</td>
<td>30.0</td>
<td>f</td>
<td>NMA</td>
<td>Middle</td>
</tr>
<tr>
<td>335</td>
<td>LLM1, URM3</td>
<td>30.0</td>
<td>f</td>
<td>NMA</td>
<td>Middle</td>
</tr>
<tr>
<td>KAS</td>
<td>ULM1</td>
<td>u</td>
<td>f</td>
<td>control</td>
<td>n/a</td>
</tr>
</tbody>
</table>

* Tooth type/position key: L or U = lower or upper; L or R = left or right; M or I = molar or incisor; 1 or 3 = first or third. All teeth are from the permanent dentition (e.g., LLM1 = permanent lower left first molar).
* Age in years = midpoint of the assessed osteological age range; u = undetermined
* m = male; f = female; u = undetermined
* SA = subadult; MA = modified adult (i.e., w/CMT); NMA = non-modified adult (i.e., without CMT)
* Early = pre-1735; Middle = ca. 1735-1760; Late-middle = ca. 1760-1776; Late = post-1776

Born enslaved individuals in early New York shifted temporally, increasing or decreasing in response to economic and social dynamics. As noted in chapter four, such factors included changing labor demands, bias against importation of African-born adults – especially males – in the aftermath of uprisings, and the eventual abolition of the Transatlantic Trade which took effect in 1808. Further complicating matters, the age parameters of “childhood” and labor expectations of children also shifted over time. Enslaved children in New York frequently were sold between the ages of 6 – the mean age of subadults included in this study – and 12 (Medford et al. 2009). However, overall, young adults capable of hard manual labor were the primary interests of transatlantic slave traders. As a practical economic matter, young children in particular were much less likely than adults to survive the rigors of the Middle Passage, contributing to their overall greater likelihood of being born in New York.

Conversely, as noted in chapter three, the bioarchaeological literature describes cultural dental modification as a rite of passage linked exclusively to individuals born in Africa. Historical and preliminary chemical research cautions against assuming these subsamples are homogenous with respect to geographic origin, as will be discussed in chapter nine. Yet, their
comparative analysis may help to inform estimations of geographic origin of individuals without
CMT or other possible indicators of natality. To assess this possibility, a third subsample
comprised of non-modified NYABG adults is included in the study.

All NYABG teeth initially were inventoried, measured and inspected for “morphological
traits, attrition rates, enamel defects, culturally induced alterations, and pathological
observations” (Blakey et al. 2009:60) under the supervision of Mr. Mark Mack, laboratory
director at Howard University. Teeth were included for analysis in this study barring the
presence of severe attrition and large caries. Ideally, all teeth sampled would be free of any
crown defects, but this criterion proved impractical given the overall poor dental health of the
NYABG population. Strict adherence to this criterion would have further restricted access to the
already limited sample of NYABG individuals with CMT. Fortunately, data collection problems
posed by sampling defective enamel were offset somewhat by the localized, high-resolution
sampling capabilities of laser ablation as well as data processing techniques discussed below.

Selected teeth included permanent first molars (M1s), first incisors (I1s) and third
molars (M3s). When available, M1s were analyzed to reconstruct early-life exposure. M1
enamel-lead is an ideal biomarker for this task as crown formation begins at birth (or in utero)
and usually completes circa three years of age. Four NYABG adults (Burials 25, 101, 241 and 243)
were selected although their M1s either were not recovered or were unsuitable for analysis.
These individuals warranted inclusion in the study because they exhibited dental modification
and/or were otherwise identified as distinctive with respect to pathological assessment, burial
orientation or associated material culture. For these individuals, I1s were analyzed in place of
M1s. I1s also capture early life exposures despite their slightly later developmental timing.
According to Reid and Dean (2006), upper I1 enamel forms from about 4 months to 4.6 years of
age and lower I1 crowns develop from approximately three months to 3.6 years of age.
Third molar enamel was analyzed for a subset of 20 modified and non-modified adults in order to assess early versus late childhood exposure. Reid and Dean (2006) estimate that M3 enamel formation occurs from about 8 to 11 years of age, but note that the timing of initial mineralization is highly variable for M3s relative to other teeth. Variation in first and third molar lead concentrations may result from a variety or combination of factors. Among such factors, migration between areas with substantially different environmental lead levels would be expected for some NYABG individuals.

Previous NYABG enamel-lead studies included control samples from Dominase and Eguaso, villages located in the hinterland adjacent to the coast in Ghana’s Central Region (Goodman et al. 2009). This study includes an M1 control sample from an adult female burial excavated in Kasana, located in the Upper West Region of northern Ghana near the Burkina Faso border. The burial dates to the 17th century and predates slaving activity in the area, which probably began circa the 1870s (Boachie-Ansah, personal communication). Kasana later became a base for Zabarima slave raiders from Mali, which archaeological research suggests resulted in the departure of its organized iron smelting community (Boachie-Ansah, 2005). The sample is a proper control for elemental and isotopic investigations of NYABG individuals’ geographic origins as it represents the interior hinterland of western Africa where many captive Africans would have originated (as opposed to coastal regions to which many were transported from the interior for departure). For this study in particular, it should provide a reliable signal of low environmental lead since the burial predates widespread use of lead technology in the region.

The sample was graciously provided by Mr. James Boachie-Ansah of the University of Ghana, 26 The samples were collected in the summer of 2000 as part of the Central Region Project, an archaeological survey of late 17th- and 18th-century Ghanaian changes in settlement and subsistence patterns, trade networks, and craft production associated with European “culture contact.” This project is directed by Professor Christopher DeCorse of Syracuse University (DeCorse et al. 2000).
Legon who, along with his colleagues, has begun systematic excavation of numerous sites important to the Transatlantic Trade as part of The Slave Route Project initiated by UNESCO in 1994.  

**Sample Preparation**

Preparation of samples for LA-ICP-MS analysis involved an extensive cleaning process for removal of organic and inorganic contaminants followed by embedding and sectioning to produce the microscopically flat surface necessary for laser focusing during ablation (Pollard et al. 2007). The five-day process began with removal of loose debris by manual brushing and cleaning with Millipore 18 MΩ cm distilled deionized (DDI) water (Millipore, Billerica, MA, USA). Each tooth was then soaked for two days in a 1% (v/v) papain solution for removal of protein material. The teeth were rinsed several times with DDI water, cleaned of any lingering organic material via a 30-second bath in 3% (v/v) hydrogen peroxide (diluted from 30% v/v strength; Fisher Scientific, Fairlawn, NJ, USA), rinsed again with DDI water, and allowed to air-dry overnight.

The dry teeth then were fixed in resin using a slightly modified version of the procedure described by Marks and colleagues (1996). Each sample was secured in a 10 mL cuboid plastic mold with glue (instead of copper wire) with the buccal or labial surfacing facing up and embedded in a 5:1 w/w ratio mixture of epoxy resin and hardener (Buehler, Lake Bluff, IL, USA). The embedded samples were placed in a vacuum for 20 minutes to minimize the number of air bubbles that formed throughout the resin and left for two days to harden in the vacuum dessicator. To avoid contamination, all glassware used during the above steps was cleaned with 50% (v/v) nitric acid and rinsed three times with DDI water.

---

Once fixed within the resin, teeth were sectioned bucco-/labio-lingually using a low-speed Isomet cutting unit (Springfield, VA, USA) affixed with a diamond-tipped wafering blade. The blade was cleaned with acetone and cooled and lubricated with a steady stream of DDI water during sectioning. Two thin sections (approximately 150 μm) were taken for histology and a 1- to 2-mm section was taken for LA-ICP-MS and other chemical analyses. Ablation surfaces were polished with a 0.3-μm alumina micropolish-DDI water solution, microscopically inspected for defects, and digitally photographed. As a final precaution against contaminants introduced after processing of the samples, ablation surfaces were etched with 1 mol L⁻¹ hydrochloric acid for 15 seconds, quenched with DDI water, and dried with acetone just prior to laser sampling.

**LA-ICP-MS Measurement**

LA-ICP-MS measurement was conducted using a 266 nm Nd:YAG CETAC-LSX 100 laser-ablation system (CETAC Technologies, Omaha, Nebraska, USA) coupled to a Perkin Elmer Sciex Elan 6000 (Shelton, CT, USA) following instrumental parameters for analysis of teeth established by Kang et al. (2004) (Figure 6.1). Lens voltage and nebulizer gas flow rate were optimized to the ⁸⁸Sr signal from line scans of the National Institute for Standards and Technology glass standard reference material (NIST SRM-612). This process was carried out twice for the lens voltage and once for the argon carrier gas flow. Line scans of the reference materials also served to optimize the energy level and number of shots for the laser system. Optimized operating conditions are summarized in Table 6.2.

Following external calibration (described below, page 135), sectioned teeth were positioned within the ablation chamber platform such that a single line scan would track elemental intensities chronologically, from early- to late-formed lateral enamel. Preliminary research confirmed that a single scan produces replicable data (see Figure 6.2). For the
Figure 6.1: Laser ablation (A) and ICP-MS (B) instrumentation

Table 6.2: Laser ablation and ICP-MS optimized operating conditions

<table>
<thead>
<tr>
<th>Laser ablation operation parameters</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Laser type</td>
<td>Nd:YAG</td>
</tr>
<tr>
<td>Laser mode</td>
<td>Frequency quadrupled 266-nm UV, Q-switched mode</td>
</tr>
<tr>
<td>Repetition rate/Hz</td>
<td>10</td>
</tr>
<tr>
<td>Laser energy/mJ</td>
<td>0.74 – 1.5 at level 13/20</td>
</tr>
<tr>
<td>Sampling scheme</td>
<td>Linear raster scan</td>
</tr>
<tr>
<td>Scanning speed/μm s⁻¹</td>
<td>20</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>ICP-MS operation parameters</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Forward power/kW</td>
<td>1</td>
</tr>
<tr>
<td>Ar gas flow rates/min⁻¹</td>
<td></td>
</tr>
<tr>
<td>Coolant</td>
<td>15</td>
</tr>
<tr>
<td>Auxiliary</td>
<td>1.2</td>
</tr>
<tr>
<td>Nebulizer gas</td>
<td>0.7 – 1.0</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Measurement conditions</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Dwell time (ms)</td>
<td>10-30</td>
</tr>
<tr>
<td>Resolution</td>
<td>High</td>
</tr>
<tr>
<td>Readings/replicates</td>
<td>200</td>
</tr>
<tr>
<td>Isotopes measured</td>
<td>⁴³Ca, ⁶⁴Zn, ⁸⁸Sr, ¹³⁸Ba, ²⁰⁶Pb, ²⁰⁷Pb, ²⁰⁸Pb</td>
</tr>
<tr>
<td>Internal standard</td>
<td>⁴³Ca</td>
</tr>
</tbody>
</table>
Figure 6.2: TriPLICATE scans of B-101 showing reproducible LA-ICP-MS results. This is a graph of the distribution of lead across dental tissues from a separate study. The two peaks represent elevated lead concentrations in secondary dentine on either side of the pulp cavity.

The purpose of standardization, buccal or labial enamel was sampled unless insufficiently intact or otherwise defective, in which case lingual enamel was sampled. Initial ablations included the occlusal surface enamel and traversed the full available crown height, but a more time-efficient strategy was developed for sampling only inner enamel, which best preserves the record of environmental chemical exposures (Humphrey et al. 2008). With the new ablation strategy, scans began in the upper portion of the lateral enamel and proceeded downward and slightly outward toward the cervix (Figure 6.3). In addition to further standardizing the ablation process and scan length (given the varied levels of occlusal wear observed especially for adults), this new strategy minimized the likelihood of detecting maternally-derived lead incorporated into cuspal enamel due to exposure in utero or via breast milk. Data collected for surface and cuspal enamel was excluded from data processing and analysis for all teeth ablated under the original sampling.
Figure 6.3: Epoxy-embedded and polished permanent upper left first molar in bucco-lingual cross-section showing dental tissues and the pulp cavity (Burial 35; 9x). The red arrow indicates the path of laser ablation sampling, from early- to late-formed lateral enamel.

For each sample, LA-ICP-MS measurement generated a large amount of time-resolved data, with a single elemental intensity data point being reported every 1.1 to 1.4 seconds. At a scan speed of $20 \, \mu m \, s^{-1}$, typical data acquisition time was approximately 3 minutes per tooth depending upon the angle of the scan, which varied somewhat across samples in order to avoid ablating fractured or discolored enamel where possible. Care was taken, also, to avoid ablating near the enamel-dentine junction (EDJ) as a number of studies document elevated trace metal levels, including lead, at this feature (e.g., Kang et al. 2004). Ablations were monitored in real-time via remote camera.
Data were collected for the following isotopes: $^{43}$Ca, $^{46}$Ca, $^{64}$Zn, $^{88}$Sr, $^{138}$Ba, $^{206}$Pb, $^{207}$Pb and $^{208}$Pb. Intensity counts per second (cps) were measured for approximately 30 seconds prior to and following each ablation in order to determine argon background intensities. The mass abundance of $^{43}$Ca is measured relatively free from isobaric and polyatomic interferences. Thus, $^{43}$Ca served as the internal standard for normalization, or signal correction against instrumental drift or ablation variations. $^{208}$Pb was the analyte isotope (mass abundance) as it provides a better signal and signal-to-blank ratio compared to those of other lead isotopes. $^{64}$Zn, $^{88}$Sr and $^{138}$Ba data will be incorporated into a future nutritional study. A digital photograph of the ablated tooth section was taken to document the line scan location and fractures or other features within the tooth that might affect the analytical signal, and for creation of chronological age-of-exposure profiles. A sample data and laser conditions record form is included as Appendix A.

**Data Processing and Analysis**

**Quantification**

Data processing began by importing the intensity data into Microsoft Excel for background subtraction and normalization to the $^{43}$Ca signal (see Appendix B). Quantification of the normalized $^{208}$Pb intensity data (i.e., $^{208}$Pb/$^{43}$Ca) was achieved by three-point calibration using a series of calcium phosphate discs spiked with known concentrations of lead (0.12, 2.09 and 18.45 μg g$^{-1}$) and other elements. Calcium phosphate is the primary component of enamel's inorganic hydroxyapatite phase, represented as Ca$_5$(PO$_4$)$_{10}$(OH)$_2$. As part of this study, the standards were being evaluated as external calibrants for tooth analysis. Dr. Peter Outridge of the Geological Survey of Canada kindly provided the standards.

Figure 6.4 shows a plot of mean $^{208}$Pb/$^{43}$Ca ratios from LA-ICP-MS (y-axis) against the
known bulk lead concentrations (x-axis) of the calibration standards. While lead distribution was not totally homogenous for any of the discs, mean $^{208}\text{Pb}/^{43}\text{Ca}$ ratios derived from the individual means of three successive 60-s line scans of each are in very good agreement with the bulk lead concentrations. Typical regression fit of the data yields a linear calibration curve ($y = 0.0162x - 0.0027, R^2 = 0.9999$), indicating the standards' suitability for quantitative LA-ICP-MS lead measurement.

As noted above, LA-ICP-MS lower limits of detection (LODs) can vary substantially depending on sample matrix, operating parameters, and other factors. For a given day, the LOD was calculated as 3 times the standard deviation of pre- and post-ablation argon blanks. The LOD for this study was estimated to be $0.39 \pm 0.29 \mu g \cdot g^{-1} (n = 8)$. Values measured below this concentration are not reliable. For statistical analyses, a value of $0.39 \mu g \cdot g^{-1}$ was used for all teeth measured at or below this concentration and a value of $“<0.39 \mu g \cdot g^{-1“}$ (LOD = 3 x s.d. of...
Distribution and Age Profiling

The 1.1- to 1.4-second data acquisition time increments were converted to micrometer-scale distances based on the laser scan speed of 20 μm s\(^{-1}\). Plotting enamel-lead concentrations against these distances in Excel produced graphs of the microspatial distributions of enamel-lead. These graphs were then superimposed onto the post-ablation photographs such that the distance (x) axes corresponded precisely to the laser tracks. The result was a visual representation of how lead distributes from early- to late-forming lateral enamel in each tooth.

The next step of data processing was to convert these microspatial distribution profiles into chronological age profiles. Here, recent advances in dental anthropology enabled me to convert elemental distributions along the enamel growth axis into age-resolved exposure profiles. These age profiles were produced for individuals whose M1 or I1 and M3 distributions revealed patterns of chronic exposure and/or acute exposure episodes. For these individuals, the post-ablation photographs were modified further by adding estimated age (in years) of enamel formation for each decile of crown completion.

The age estimates were histologically-derived by Reid and Dean (2006) who counted and measured daily (cross-striations) and long-period incremental markings (Retzius lines) in permanent teeth in order to determine fractional growth rates (see Figure 6.5). They report total crown formation time as the sum of cuspal and lateral enamel formation time. Their sample included over 600 teeth from contemporary and historic populations from southern Africa, northern Europe and North America. From these growth rates, Reid and Dean (2006) calculated ages at which each decile of crown height was completed, identifying a previously
unrecognized trend of gradually increasing formation time. Importantly, they observed low geographic variation across populations, suggesting their calculated age ranges are widely applicable for human tooth studies such as this one. Reid and Dean’s (2006) mean estimates of chronological age of enamel formation for permanent anterior teeth (incisors and canines) and molars are presented in Figures 6.6 and 6.7.

Yet, two important sources of variation and potential error should be noted with respect to these chronological age-at-exposure profiles. First, are Reid and Dean’s (2006) assumptions concerning the timing of initiation for crown development in the tooth types included in this study. Those assumptions are: birth for upper and lower M1s, 128 days for upper I1s, 90 days for lower I1s, and 8 years for upper and lower M3s. They cite Antoine (2001) who observed as much as 250 days variation for initial mineralization of some anterior teeth and note, also, that initiation of M3 crown development is particularly variable at the individual and population
Figure 6.6: Estimates for chronological ages of human enamel formation for permanent molars by decile of crown growth. The numbers on the left are mean estimates for the southern African sample and those on the right are mean estimates for the northern European sample. For each decile, an average of these values was used to create the M1 and M3 chronological age profiles lead exposure presented in chapter 8. From Reid and Dean (2006, Figure 4)

levels. Another potential source of error is the amount of occlusal wear observed especially for NYABG adults. For many individuals, age- and diet-related wear resulted in substantial loss of cuspal enamel. Thus, these age profiles are necessarily approximations and are perhaps most accurate for subadults.

Finally, the microspatial distribution-tooth overlay images were critical with respect to two data processing quality control measures. First, they were used to determine whether fluctuations in lead concentration, particularly spikes or other substantial increases, were the result of ablating fractured or defective enamel. Fractures and hypocalcified enamel can yield artificially high lead readings due to a decrease in the $^{43}$Ca internal standard signal used to normalize elemental intensity data prior to quantification. Secondly, from these images, I was able to identify lead-enriched surface enamel zones. These limited zones of elevated lead
Figure 6.7: Estimates for chronological ages of human enamel formation for permanent anterior teeth (incisors and canines) by decile of crown growth. The numbers on the left are mean estimates for the southern African sample and those on the right are mean estimates for the northern European sample. For each decile, an average of these values was used to create the 11 chronological age profiles lead exposure presented in chapter 8. From Reid and Dean (2006, Figure 4)

concentration may reflect remineralization in the oral cavity after the early-life period of crown development that is of interest for this study (Budd et al. 1998; Humphrey et al. 2008). Thus, elevated surface values for lead are not included in calculations of mean enamel-lead. However, these data were retained in the microspatial distribution and age graphs for illustrative purposes and to maintain the integrity of the distance/age axes.

Analysis

The primary analytical goal of this study is to explore variation in the extent, nature and age of lead exposure for NYABG children and adults. The first part of analysis focuses on whole
tooth enamel values overall and by subgroups within the NYABG. The second part involves more
detailed study of the pattern of lead deposition by location/age at development in order to
discern and consider variations within individuals over early childhood as well as among
individuals. Additionally, early and late childhood exposure is compared for a limited number of
adults.

Data analysis proceeded through two main phases: enamel-lead content analysis and
evaluation of lead distribution and age at deposition profiles. The first phase involved
characterizing lead burden for the entire sample set and assessing variation in mean lead
content within and among subadult, modified adult and non-modified adult cohorts. This
process began by determining mean enamel-lead concentrations for each tooth, i.e., the
average of (quantified) data values produced during a single line scan. Individual tooth mean
values were in turn used to calculate subadult, (modified/non-modified) adult, male, female,
etc. group means for statistical analyses.

A combination of non-parametric and parametric statistical tests was necessary to
identify significant differences in mean enamel-lead content. Variables assessed include age,
sex, temporal group, absence/presence of enamel hypoplasia and tooth type. Comparisons of
subadults with adults required non-parametric procedures (the Mann-Whitney U, Kruskall-
Wallis tests and Spearman rank correlation) that do not assume normal distribution or similar
variances of the data; problematic assumptions given the hypothesis that most subadults spent
their early childhoods in relatively high-versus low-lead environments. Parametric tests
(independent samples t-test and paired samples t-test) were used for comparisons involving
only adults (e.g., mean differences for M1s/I1s versus M3s). All statistical procedures were
performed using Statistical Package for the Social Sciences (SPSS) 20.0 (IBM, Armonk, NY, USA).
The second phase of data analysis focused on the evaluation of enamel-lead microspatial distributions and age graphs. The purpose of this phase is to better understand the nature and age of individuals’ experiences of lead exposure by assessing intra-tooth variation. For those individuals with discernible enamel-lead signals, the specific objectives are to determine: (1) whether exposure was chronic or episodic; (2) whether increases in lead concentrations were gradual or acute; and (3) the specific age(s) at which lead burden increased and/or decreased. Building upon interpretations based on mean lead content in this fashion raised interesting and important questions largely unanswerable until now – questions that both expanded and gave focus to the interpretive process. For example, does a relatively high mean lead value reflect consistently high-level exposure, distinct periods of greatly elevated exposure, or perhaps even large “spikes” representing acute exposure events amid generally low-level exposure? Conversely, might a low average value mask single or infrequent acute exposures with potential health significance? Results of analysis appear in the next two chapters, followed in chapter nine by a discussion of their biocultural interpretations and implications.
CHAPTER 7

RESULTS: MEAN ENAMEL-LEAD CONTENT

Results of dental lead analysis are presented in two parts. First, in this chapter, I compare mean enamel-lead concentrations for individuals within and among the main analytical cohorts. In chapter eight, I provide the microspatial distribution and age profiles that illustrate the unique capabilities and value of LA-ICP-MS in dental analysis. Comparing lead content levels within and among groups is the first step towards identifying who was most at-risk for exposure and why. Specifically, analysis of mean lead concentrations suggests the extent of lead exposure for NYABG individuals and groups and enables comparison of the total sample with historical and contemporary populations, including reference populations with documented high- and low-level exposures (Budd et al. 2000, 2004). Here, I report patterns related to age, sex, and temporal grouping. I first present data from permanent first molars (M1) or first incisors (I1) for all individuals. Then, for a subsample of adults, I compare lead content of these early-forming M1 and I1 teeth with those of later-forming third molars (M3).

**Permanent First Molars/Incisors**

Mean enamel-lead concentrations by individual are listed in Table 7.1. Of the 41 M1s or I1s measured, values for teeth from 16 individuals (39%) are below the 0.39 μg g\(^{-1}\) limit of detection (LOD). For statistical analyses, the 0.39 μg g\(^{-1}\) LOD value was assigned to these individuals, all adults: 10 modified and 5 non-modified NYABG individuals and the control sample from Kasana. Analysis of teeth for 25 individuals – 11 subadults, 8 modified adults and 6 non-modified adults – produced readings above the 0.39 μg g\(^{-1}\) LOD.

Figure 7.1 is a box plot graph of M1 or I1 concentrations that illustrates the variation within and among these three groups. Enamel-lead distributions are presented around the
Table 7.1: Enamel-lead concentrations by individual/tooth.

<table>
<thead>
<tr>
<th>Burial</th>
<th>Tooth</th>
<th>Cohort</th>
<th>[Pb] (μg g⁻¹)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subadults</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>LRM1</td>
<td>SA</td>
<td>2.86</td>
</tr>
<tr>
<td>22</td>
<td>LRM1</td>
<td>SA</td>
<td>4.66</td>
</tr>
<tr>
<td>35</td>
<td>ULM1</td>
<td>SA</td>
<td>10.5</td>
</tr>
<tr>
<td>39</td>
<td>LRM1</td>
<td>SA</td>
<td>6.09</td>
</tr>
<tr>
<td>43</td>
<td>LRM1</td>
<td>SA</td>
<td>2.41</td>
</tr>
<tr>
<td>126</td>
<td>LLM1</td>
<td>SA</td>
<td>11.6</td>
</tr>
<tr>
<td>138</td>
<td>URM1</td>
<td>SA</td>
<td>2.30</td>
</tr>
<tr>
<td>180</td>
<td>ULM1</td>
<td>SA</td>
<td>1.20</td>
</tr>
<tr>
<td>219</td>
<td>LRM1</td>
<td>SA</td>
<td>14.7</td>
</tr>
<tr>
<td>244</td>
<td>LLM1</td>
<td>SA</td>
<td>4.35</td>
</tr>
<tr>
<td>405</td>
<td>URM1</td>
<td>SA</td>
<td>3.98</td>
</tr>
<tr>
<td>Modified adults</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>LLM1</td>
<td>MA</td>
<td>&lt;0.39</td>
</tr>
<tr>
<td>6</td>
<td>ULM3</td>
<td>MA</td>
<td>&lt;0.39</td>
</tr>
<tr>
<td>9</td>
<td>LLM1</td>
<td>MA</td>
<td>1.34</td>
</tr>
<tr>
<td>9</td>
<td>LRM3</td>
<td>MA</td>
<td>0.69</td>
</tr>
<tr>
<td>23</td>
<td>URM1</td>
<td>MA</td>
<td>&lt;0.39</td>
</tr>
<tr>
<td>47</td>
<td>LLM1</td>
<td>MA</td>
<td>1.31</td>
</tr>
<tr>
<td>68</td>
<td>LRM3</td>
<td>MA</td>
<td>&lt;0.39</td>
</tr>
<tr>
<td>101</td>
<td>LRI1</td>
<td>MA</td>
<td>7.80</td>
</tr>
<tr>
<td>101</td>
<td>LLM3</td>
<td>MA</td>
<td>7.38</td>
</tr>
<tr>
<td>106</td>
<td>LRM1</td>
<td>MA</td>
<td>0.93</td>
</tr>
<tr>
<td>106</td>
<td>LRM3</td>
<td>MA</td>
<td>&lt;0.39</td>
</tr>
<tr>
<td>115</td>
<td>LLM1</td>
<td>MA</td>
<td>&lt;0.39</td>
</tr>
<tr>
<td>115</td>
<td>LRM3</td>
<td>MA</td>
<td>&lt;0.39</td>
</tr>
<tr>
<td>151</td>
<td>LRM1</td>
<td>MA</td>
<td>&lt;0.39</td>
</tr>
<tr>
<td>165</td>
<td>LLM1</td>
<td>MA</td>
<td>&lt;0.39</td>
</tr>
<tr>
<td>165</td>
<td>LRM3</td>
<td>MA</td>
<td>&lt;0.39</td>
</tr>
<tr>
<td>241</td>
<td>URI1</td>
<td>MA</td>
<td>&lt;0.39</td>
</tr>
<tr>
<td>243</td>
<td>ULI1</td>
<td>MA</td>
<td>0.89</td>
</tr>
<tr>
<td>243</td>
<td>URM3</td>
<td>MA</td>
<td>&lt;0.39</td>
</tr>
<tr>
<td>266</td>
<td>URM1</td>
<td>MA</td>
<td>0.56</td>
</tr>
<tr>
<td>266</td>
<td>URM3</td>
<td>MA</td>
<td>&lt;0.39</td>
</tr>
<tr>
<td>270</td>
<td>LLM1</td>
<td>MA</td>
<td>&lt;0.39</td>
</tr>
<tr>
<td>270</td>
<td>LLM3</td>
<td>MA</td>
<td>&lt;0.39</td>
</tr>
<tr>
<td>366</td>
<td>LLM1</td>
<td>MA</td>
<td>&lt;0.39</td>
</tr>
<tr>
<td>367</td>
<td>ULM1</td>
<td>MA</td>
<td>0.76</td>
</tr>
<tr>
<td>377</td>
<td>ULM1</td>
<td>MA</td>
<td>2.82</td>
</tr>
<tr>
<td>384</td>
<td>ULM1</td>
<td>MA</td>
<td>&lt;0.39</td>
</tr>
<tr>
<td>397</td>
<td>ULM1</td>
<td>MA</td>
<td>&lt;0.39</td>
</tr>
</tbody>
</table>
Table 7.1: Enamel-lead concentrations by individual/tooth.

<table>
<thead>
<tr>
<th>Burial</th>
<th>a Tooth</th>
<th>b Cohort</th>
<th>c [Pb] (μg g⁻¹)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-modified adults</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>ULM1</td>
<td>NMA</td>
<td>&lt;0.39</td>
</tr>
<tr>
<td>25</td>
<td>URI1</td>
<td>NMA</td>
<td>0.40</td>
</tr>
<tr>
<td>25</td>
<td>LRM3</td>
<td>NMA</td>
<td>&lt;0.39</td>
</tr>
<tr>
<td>49</td>
<td>ULM1</td>
<td>NMA</td>
<td>1.72</td>
</tr>
<tr>
<td>63</td>
<td>LRM3</td>
<td>NMA</td>
<td>2.08</td>
</tr>
<tr>
<td>135</td>
<td>LRM3</td>
<td>NMA</td>
<td>&lt;0.39</td>
</tr>
<tr>
<td>150</td>
<td>LRM1</td>
<td>NMA</td>
<td>&lt;0.39</td>
</tr>
<tr>
<td>172</td>
<td>LLM1</td>
<td>NMA</td>
<td>&lt;0.39</td>
</tr>
<tr>
<td>172</td>
<td>LLM3</td>
<td>NMA</td>
<td>2.31</td>
</tr>
<tr>
<td>176</td>
<td>LRM1</td>
<td>NMA</td>
<td>&lt;0.39</td>
</tr>
<tr>
<td>176</td>
<td>LLM3</td>
<td>NMA</td>
<td>0.61</td>
</tr>
<tr>
<td>179</td>
<td>LRM1</td>
<td>NMA</td>
<td>1.60</td>
</tr>
<tr>
<td>179</td>
<td>LLM3</td>
<td>NMA</td>
<td>&lt;0.39</td>
</tr>
<tr>
<td>196</td>
<td>LRM1</td>
<td>NMA</td>
<td>&lt;0.39</td>
</tr>
<tr>
<td>262</td>
<td>LRM3</td>
<td>NMA</td>
<td>&lt;0.39</td>
</tr>
<tr>
<td>323</td>
<td>LRM1</td>
<td>NMA</td>
<td>4.35</td>
</tr>
<tr>
<td>323</td>
<td>LRM3</td>
<td>NMA</td>
<td>&lt;0.39</td>
</tr>
<tr>
<td>324</td>
<td>ULM1</td>
<td>NMA</td>
<td>1.39</td>
</tr>
<tr>
<td>324</td>
<td>LRM3</td>
<td>NMA</td>
<td>1.96</td>
</tr>
<tr>
<td>335</td>
<td>LLM1</td>
<td>NMA</td>
<td>0.42</td>
</tr>
<tr>
<td>335</td>
<td>URM3</td>
<td>NMA</td>
<td>&lt;0.39</td>
</tr>
<tr>
<td>Kasana</td>
<td>ULM1</td>
<td>control</td>
<td>&lt;0.39</td>
</tr>
</tbody>
</table>

a Tooth type/position key: L or U = lower or upper; L or R = left or right; M or I = molar or incisor; 1 or 3 = first or third. All teeth are from the permanent dentition (e.g., LLM1 = permanent lower left first molar).

b SA = subadult; MA = modified adult (i.e., w/CMT); NMA = non-modified adult (i.e., without CMT)

LOD of Pb = < 0.39 μg g⁻¹

median for each group (subadults: 4.35 μg g⁻¹; modified adults: 0.39 μg g⁻¹; non-modified adults: 0.40 μg g⁻¹), which is represented by the bold horizontal line within each box. The box represents the interquartile range (IQR), i.e., the 25th (lower edge) to 75th (upper edge) percentiles, and the “whiskers” extend to the lowest and highest non-outlier values. The value ranges for each of the adult subsamples are increased substantially by the presence of extreme
Figure 7.1: Box plot of M1 or I1 enamel-lead concentrations by analytical cohort for NYABG individuals.

outliers Burials 101 and 377 within the modified adults and Burial 323 as an outlier for non-modified adults.\(^{28}\) Values for the bottom half of both adult samples cluster tightly around the medians due to the large number of individuals measured below the 0.39 μg g\(^{-1}\) detection limit.

**Subadults and Adults**

The overall mean enamel-lead value for NYABG M1s or I1s is 2.42 ± 3.38 μg g\(^{-1}\) (range: 0.39 to 14.7 μg g\(^{-1}\); n=40). The mean enamel-lead concentration for subadults is 5.88 ± 4.4 μg g\(^{-1}\) (n=11); more than 5 times that of the overall adult sample (1.11 ± 1.56 μg g\(^{-1}\); n=29). This difference is statistically significant (Mann-Whitney: \(z = -4.295\); \(p < .001\)). The subadult mean

\(^{28}\) An outlier, indicated in Figure 7.1 by a circle, is defined as a sample with a value between 1.5 and 3 times the IQR. An extreme outlier has a value greater than 3 times the IQR and is indicated by an asterisk.
also is significantly higher than those of both modified (1.13 ± 1.77 μg g⁻¹; z = -3.946; p < .001) and non-modified (1.06 ± 1.16 μg g⁻¹; z = -3.435; p < .001) adults. The difference between the mean values for modified and non-modified adults (z = - .388; p = .698) is not significant.

In addition to their higher average lead content, children’s teeth exhibit a much wider distribution of values than both adult groups. Subadult M1 or I1 values range from 1.20 to 14.7 μg g⁻¹. By comparison, the distributions of modified and non-modified adult M1 or I1 values are similar and narrow, ranging from the 0.39 detection limit to 7.80 μg g⁻¹ and 4.10 μg g⁻¹, respectively. Subadults show the greatest variation and non-modified adults, the least.

**Age**

Figure 7.2 further illustrates the enamel-lead variation present both within and among groups as well as a general pattern to the relationship of enamel-lead to age. Of the 40 NYABG M1s or I1s analyzed, this scatterplot graph includes data for 37 individuals with age-at-death estimates. Estimated ages are reported as midpoints of age ranges determined from osteological indicators. Included among them are 11 subadults, 15 modified adults and 11 non-modified adults. While highly variable particularly among subadults, overall lead concentrations tend to decrease with age. This negative correlation is significant according to Spearman’s rho (r = -.497, p < .01). Lead concentrations decrease slightly when adults are considered exclusively but this correlation is not significant (r = .146, p = .476) (see Figure 7.3).

Ninety percent (n = 26) of all adults have lead concentrations below 2 μg g⁻¹ and most (72%; n = 21) are below 1 μg g⁻¹, the suggested lower limit for technological exposure (Budd 2004; see chapter 6). By comparison, all but 1 of the subadult M1 or I1 values are above 2 μg g⁻¹, with a small majority (55%; n = 6) measured between 2 and 6 μg g⁻¹. A few modified adults,
Figure 7.2: Scatterplot of M1 or I1 enamel-lead concentrations by age-at-death for NYABG individuals. Most adult values are below 2 μg g⁻¹ while most subadult values fall between 2 and 6 μg g⁻¹. Burials 323 and 377, fall within this range of relative mid-level exposure. The lowest subadult value is 1.20 μg g⁻¹ (Burial 180). The remaining five individuals with the highest concentrations include 4 subadults and 1 modified adult, Burial 101. Thus, the first part of the analysis shows measurable lead levels in most teeth and a clear trend of great variation and greater concentrations in teeth of children. One implication to be discussed later is that children are most likely to have grown up in the Americas.
Figure 7.3: Scatterplot of M1 or I1 enamel-lead concentrations by age-at-death for NYABG adults. Lead concentration does not correlate significantly with age for adults.

**Sex**

The 40 NYABG individuals in the study sample included 16 females, 11 males, and 13 individuals of undetermined sex. Of the 27 individuals positively assessed as either female or male, all were adults with the exception of Burial 180, an approximately 12-year old girl. Sex could not be determined for 10 subadults and 3 adults. Table 7.2 lists the mean lead concentrations by sex and analytical cohort. Comprised mostly of subadults, the undetermined sex group had the highest mean enamel-lead concentration as well as the widest distribution of values (4.98 ± 4.59 μg g⁻¹; range: 0.39 – 14.7; n=13), followed in both respects by males (1.75 ± 2.32 μg g⁻¹; range: 0.39 – 7.80; n=11) and, lastly, females (0.81 ± 0.68 μg g⁻¹; range: 0.39 – 2.82;
Table 7.2: NYABG M1 or I1 enamel-lead concentrations by sex and analytical cohort.

<table>
<thead>
<tr>
<th>Sex</th>
<th>N</th>
<th>[Pb] (μg g⁻¹)</th>
<th>Min-Max</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Females</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subadults</td>
<td>1</td>
<td>1.20</td>
<td>N/A</td>
</tr>
<tr>
<td>Mod. adults</td>
<td>8</td>
<td>0.83 ± 0.83</td>
<td>0.39 – 2.82</td>
</tr>
<tr>
<td>Non-mod. adults</td>
<td>7</td>
<td>0.73 ± 0.57</td>
<td>0.39 – 1.72</td>
</tr>
<tr>
<td>Total</td>
<td>16</td>
<td>0.81 ± 0.68</td>
<td>0.39 – 2.82</td>
</tr>
<tr>
<td><strong>Males</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subadults</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Mod. adults</td>
<td>8</td>
<td>1.61 ± 2.54</td>
<td>0.39 – 7.80</td>
</tr>
<tr>
<td>Non-mod. adults</td>
<td>3</td>
<td>2.98 ± 1.94</td>
<td>1.60 – 4.35</td>
</tr>
<tr>
<td>Total</td>
<td>11</td>
<td>1.75 ± 2.32</td>
<td>0.39 – 7.80</td>
</tr>
<tr>
<td><strong>Undetermined</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subadults</td>
<td>10</td>
<td>6.35 ± 4.36</td>
<td>2.30 – 14.7</td>
</tr>
<tr>
<td>Mod. adults</td>
<td>2</td>
<td>0.39 ± 0.0</td>
<td>0.39</td>
</tr>
<tr>
<td>Non-mod. adults</td>
<td>1</td>
<td>0.39</td>
<td>0.39</td>
</tr>
<tr>
<td>Total</td>
<td>13</td>
<td>4.98 ± 4.59</td>
<td>0.39 – 14.7</td>
</tr>
</tbody>
</table>

* Individuals of undetermined sex were not included in group comparisons.

n=16). The mean male concentration is thus a little over twice that for females. An independent samples t-test indicates that this difference is not statistically significant (t = -1.308; p = .217). Although the results are not significant, the sample sizes are low and thus a gender difference, perhaps related to dietary or caretaking practices, cannot be ruled out entirely. It is also worth reminding the reader that the concentrations are from early life. Thus, while they are from adults of osteologically determined sex, the enamel-lead is from enamel that is a window onto infancy and early childhood.

As Figure 7.4 shows, the majority of values for both groups fall within the < 0.39 (LOD) to 2 μg g⁻¹ “adult” range identified in the previous section. The median concentration for females is 0.41 μg g⁻¹ compared to 0.89 μg g⁻¹ for males. Burials 101 and 323 are again (male)
Figure 7.4: Box plot of M1 or I1 enamel-lead concentrations by sex for NYABG individuals. The median value is higher for males although the distributions are similar with the exception of the outliers, particularly Burial 101.

Figure 7.5: Box plot of M1 or I1 enamel-lead concentrations by sex and analytical cohort for NYABG adults. The median values fall below 2 µg g⁻¹ for males and females for both groups. Non-outlier tooth values are more widely distributed for non-modified than for modified individuals.
outliers and Burial 377 is the only female outlier. Hers is the only female value above 2 µg g⁻¹. Figure 7.5 compares concentrations for modified and non-modified males and females. With the exception of a few extreme outliers, the distribution of tooth values is wider for non-modified adults. Median concentrations are slightly higher for modified (0.48 µg g⁻¹) than for non-modified females (0.40 µg g⁻¹). For males, the opposite is observed: median value is 0.64 µg g⁻¹ for non-modified males as compared to 1.60 µg g⁻¹ for modified males.

Temporal Groups

Temporal variation in M1 or I1 enamel-lead concentration is illustrated in figure 7.6. This graph is a representation of lead exposure among NYABG individuals from approximately 1735 AD through the American Revolutionary era (see chapter four). It does not include the Early period (prior to ~ 1735 AD), which was represented in the study sample by just a single individual, Burial 23, whose mean concentration was below the 0.39 µg g⁻¹ LOD. Subadults, modified adults and non-modified adults are fairly evenly distributed among the Middle (circa 1735 to 1760), Late-Middle (circa 1760 to 1776) and Late periods (post-1776) although the Late-Middle group includes only 1 non-modified adult. Overall mean concentration increases when comparing teeth from the Middle (n = 17; 2.71 ± 3.58 µg g⁻¹) and Late-Middle (n = 7; 4.56 ± 5.07 µg g⁻¹) Groups (see Table 7.3). The lead distributions for these two groups are comparable and all outliers are subadults: Burials 35 and 126 for the Middle Group and Burial 219 for the Late-Middle. Mean lead concentration decreases significantly from the Late Middle to the Late Group (1.22 ± 1.39 µg g⁻¹) (Mann-Whitney: z = -2.227; p = .026).

Table 7.3 provides mean M1 or I1 concentrations for the three main analytical cohorts and shows distinct trends for each across temporal groups. Subadult mean values decrease
Figure 7.6: Box plot of M1 or I1 enamel-lead concentrations by temporal group for NYABG individuals showing variation from the mid-1730s through the post-American Revolutionary era. The highest median value (2.85 μg g$^{-1}$) was measured for the Late-Middle cohort corresponding approximately to the period of 1760 through 1776. Median values for the Middle and Late groups are 1.31 μg g$^{-1}$ and 0.40 μg g$^{-1}$, respectively.

steadily across all periods; slightly from the Middle to Late-Middle Groups and then considerably for the Late Group. Following a general pattern, subadult values are consistently higher than those of modified and non-modified adults. For modified adults, mean concentrations increase from the Middle to Late-Middle periods and then decrease to their lowest levels for the Late period. Non-modified adult values follow the opposite pattern, decreasing from the Middle to Late-Middle periods before increasing to their highest levels in the Late period. Among adults, mean concentrations are higher for non-modified than for modified adults for the Middle and Late periods. Mean concentration is higher for modified adults in the Late-Middle Group. Note, however, that this group includes a single non-modified individual with concentration measured below LOD. The typical sex pattern of higher enamel-lead concentrations for males persists for
Table 7.3: NYABG M1 or I1 enamel-lead concentrations by temporal group and analytical cohort.

<table>
<thead>
<tr>
<th>Temporal group</th>
<th>n</th>
<th>Mean ± SD</th>
<th>Min-Max</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Middle</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subadults</td>
<td>5</td>
<td>7.37 ± 3.47</td>
<td>3.98 – 11.6</td>
</tr>
<tr>
<td>Mod. adults</td>
<td>8</td>
<td>0.67 ± 0.42</td>
<td>0.39 – 1.34</td>
</tr>
<tr>
<td>Non-mod. adults</td>
<td>4</td>
<td>0.98 ± 0.67</td>
<td>0.40 – 1.72</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>17</td>
<td>2.71 ± 3.58</td>
<td>0.39 – 11.6</td>
</tr>
<tr>
<td><strong>Late-Middle</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subadults</td>
<td>3</td>
<td>6.66 ± 6.97</td>
<td>2.86 – 14.7</td>
</tr>
<tr>
<td>Mod. adults</td>
<td>3</td>
<td>3.86 ± 3.55</td>
<td>0.93 – 7.80</td>
</tr>
<tr>
<td>Non-mod. adults</td>
<td>1</td>
<td>0.39</td>
<td>0.39</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>7</td>
<td>4.56 ± 5.07</td>
<td>0.39 – 14.7</td>
</tr>
<tr>
<td><strong>Late</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subadults</td>
<td>3</td>
<td>2.62 ± 1.60</td>
<td>1.20 – 4.35</td>
</tr>
<tr>
<td>Mod. adults</td>
<td>6</td>
<td>0.50 ± 0.20</td>
<td>0.39 – 0.89</td>
</tr>
<tr>
<td>Non-mod. adults</td>
<td>6</td>
<td>1.25 ± 1.59</td>
<td>0.39 – 4.35</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>15</td>
<td>1.22 ± 1.39</td>
<td>0.39 – 4.35</td>
</tr>
</tbody>
</table>

* The sample included a single individual from the Early Group (Burial 23 with enamel-lead concentration measured below the 0.39 μg g⁻¹ LOD.

all three temporal groups although sample sizes are small and the differences are not statistically significant (see Table 7.4).

**Comparison of Permanent First Molars or Incisors and Third Molars**

A comparison of early- and late-forming tooth enamel may reveal changes in lead exposure, uptake or skeletal deposition during an individual’s life. I analyzed M3s for 20 NYABG adults for this study. The mean concentration for this group is 1.14 ± 1.74 μg g⁻¹, virtually the same as that observed for the NYABG adult M1 or I1 sample (1.11 ± 1.56 μg g⁻¹; n=29). Seventy percent (n=14) of the M3 teeth produced measurements below the LOD (< 0.39 μg g⁻¹) as compared to 52% (n=15) of the M1 or I1 teeth (see Table 7.1).
Table 7.4: NYABG M1 or I1 enamel-lead concentrations by temporal group and sex.

<table>
<thead>
<tr>
<th>a  Temporal group</th>
<th>n</th>
<th>Mean ± SD</th>
<th>Min-Max</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Middle</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>8</td>
<td>0.73 ± 0.53</td>
<td>0.40 – 1.72</td>
</tr>
<tr>
<td>Male</td>
<td>3</td>
<td>1.01 ± 0.54</td>
<td>1.31 – 1.34</td>
</tr>
<tr>
<td>Undetermined</td>
<td>6</td>
<td>6.20 ± 4.21</td>
<td>0.39 – 11.59</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>17</td>
<td>2.71 ± 3.58</td>
<td>0.39 – 11.59</td>
</tr>
<tr>
<td><strong>Late-Middle</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>2</td>
<td>1.89 ± 1.36</td>
<td>0.93 – 2.82</td>
</tr>
<tr>
<td>Male</td>
<td>2</td>
<td>4.10 ± 5.24</td>
<td>0.39 – 7.80</td>
</tr>
<tr>
<td>Undetermined</td>
<td>3</td>
<td>6.66 ± 6.97</td>
<td>2.41 – 14.7</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>7</td>
<td>4.56 ± 5.07</td>
<td>0.39 – 14.7</td>
</tr>
<tr>
<td><strong>Late</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>6</td>
<td>0.55 ± 0.32</td>
<td>0.39 – 1.20</td>
</tr>
<tr>
<td>Male</td>
<td>5</td>
<td>1.52 ± 1.66</td>
<td>0.39 – 4.35</td>
</tr>
<tr>
<td>Undetermined</td>
<td>4</td>
<td>1.86 ± 1.89</td>
<td>0.39 – 4.35</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>15</td>
<td>1.22 ± 1.39</td>
<td>0.39 – 4.35</td>
</tr>
</tbody>
</table>

The sample included a single individual from the Early Group (Burial 23 with enamel-lead concentration measured below the 0.39 µg g⁻¹ LOD.

The larger percentage of M3s below the LOD may indicate a general decrease in lead exposure during adolescence for NYABG adults. However, an analysis of paired early- and late-forming tooth data for 16 NYABG adults indicates that M1 or I1 concentrations for most of these individuals are below the 1 µg g⁻¹ upper limit for non-technological exposure. Thus, these individuals most likely spent their early years (when M1s and I1s form) as well as their adolescent years (when M3s form) in low-lead environments.

Figure 7.7 shows the distributions of M1 or I1 and M3 mean enamel-lead concentrations. The median concentration for both groups is 0.39 µg g⁻¹ and the ranges of their concentration values are similar: <0.39 (below LOD) to 7.80 µg g⁻¹ for M1 or I1 teeth and <0.39
Figure 7.7: Box plot comparison of M1 or I1 and M3 enamel-lead concentrations for NYABG adults. The median concentration for both groups is 0.39 μg g⁻¹, the limit of detection, but the range of values is considerably narrower for M3s.

to 7.38 μg g⁻¹ for M3s. Burial 101 has the highest lead concentrations and is an extreme outlier for both tooth types. Other extreme outliers within the M3 group are Burials 63, 172 and 324. While the mean concentrations and range of values for both groups are very similar, the distribution of non-outlying M3 values is narrower and – contra the M1 or I1 group – none of these values exceed 1 μg g⁻¹, the suggested threshold for technological exposure.

Paired M1 or I1 and M3 enamel-lead concentrations are available for 16 individuals, 5 of which – Burials 9, 101, 172, 179 and 324 – appear in Figure 7.8. I highlight these individuals because they do not conform to the trend of low M1 or I1 concentrations (i.e., < 1 μg g⁻¹) and lower M3 concentrations (below LOD [< 0.39 μg g⁻¹]) observed for a slight majority (n = 9 or 56%) within this group. This general pattern indicates that these individuals spent their earliest as well their adolescent years in environments with low-level non-anthropogenic or background lead
Figure 7.8: Paired NYABG M1 or I1 and M3 enamel-lead concentrations. Relatively stable concentrations for M1s or I1s and M3s suggest a stable (high- or low-) lead environment throughout childhood and adolescence. Substantial variation between tooth types for an individual may indicate migration during childhood years.

exposure. Individuals whose concentrations deviate from this trend likely had different experiences. For example, like most individuals with paired M1 or I1 and M3 data, Burial 101 shows little inter-tooth variation. However, this individual differs from the general pattern of low-level and decreasing lead exposure as his high concentrations for I1 and M3 teeth suggest little change in infant and adolescent lead environments. These data seem to indicate that Burial101 grew up under conditions of relatively high-level lead exposure.

Of particular interest are those individuals whose increases or decreases in early- to late-forming tooth concentrations cross Budd et al.’ (2004) suggested 1 μg g-1 threshold of non-technological exposure. Such findings may indicate migration from low- to high-lead environments, or vice versa. This seems to be the case for Burial 172, an individual whose M1 and M3 lead concentrations suggest movement from a low-lead (presumably African) to a high-lead (presumably American) setting. Meanwhile, Burials 9, 179 and 323 may also have migrated
as children albeit from high- to low-lead environments given their age-related decreases in enamel-lead concentration. This latter finding is fascinating as it points to the potential importance of regional variation of diasporic conditions associated with different enslavement regimes in the Americas and, possibly, within Africa.

**Conclusions**

In this chapter I have shown that enamel-lead concentrations varied greatly within and across groups and sometimes for different teeth of the same individual. Mean M1 or I1 concentrations were highest for children; over five times that of adults, whether considered collectively or as modified and non-modified subgroups. Mean concentration for males was higher than that of females and early-life exposure was greatest for individuals in the Late-Middle group, i.e., those buried between approximately 1760 and 1776, the years leading up to the American Revolutionary War. For most individuals with measured M1 or I1 and M3 concentrations, M1 or I1 values were higher than but comparable to those measured in their later-forming M3 teeth, indicating a degree of stability in environmental lead conditions. For some, however, inter-tooth variation suggests exposure increased or decreased substantially with age.

Comparisons of mean concentrations begin to tell a story about lead exposure in 18th-century America and in the lives of enslaved New Yorkers. Yet, for all these data reveal, they also potentially mask important patterns with respect to the age and nature (i.e., chronic versus acute) of exposures. The unique promise of the method developed for this study is its ability to capture such experiences. Therefore, in the next chapter I describe enamel-lead microspatial and age distributions. I then explore the potential relevance of these and other findings in chapter 9.
CHAPTER 8

RESULTS: ENAMEL-LEAD PROFILES

This second chapter of analytical results focuses on the microspatial distribution and chronological age of enamel-lead deposition. I present the data in two sections. In the first, I describe various enamel-lead distribution patterns observed amongst NYABG individuals. In the second section, I combine these patterns with estimated ages of enamel formation to produce a series of age-at-deposition profiles. These profiles reveal the natures as well as the extent of early life lead exposure. As in the preceding chapter, I compare measured lead across individuals, groups and tooth types (earlier developing M1s or I1s versus later developing M3s) in order to characterize variation in the extent of enamel-lead deposition at each of these levels.

The spatial distribution and age profiles presented below take full advantage of the laser ablation capabilities of the ICP-MS. A unique analytical feature of laser ablation is its ability to detect tissue level distributions of lead. Here, this feature is particularly useful for identifying intra-tooth (and by extension intra-individual) temporal trends that enable a more in-depth analysis of the timing and amount of lead content than can be attained from mean or “total” enamel-lead data alone. Potentially, these profiles can shed light on the varied nature and perhaps, by extension, the actual sources of lead exposure for NYABG individuals and groups. Throughout this chapter, distribution graphs appear in log scale for selected – and in linear scale for all – teeth that yielded clear analytical signals. For comparative purposes, log scale graphs for all of these teeth are included as Appendices B-D, although the condensed scale may serve to obscure some of the patterns described below.²⁹

²⁹ A log scale is useful for presenting data values that range widely. Rather than actual values, logarithms of values are graphed on a scale that increases exponentially according to an
**Microspatial Pb Distribution Profiles**

Thirty-two teeth – 28 M1 or I1s and 4 M3s – from 29 NYABG individuals produced clear lead signals via LA-ICP-MS analysis, from which I graphed microspatial distributions for early-to-late formed core enamel developed during infancy. This sample consists of: M1s from all 11 subadults included in this study; 8 M1 or I1s and 2 M3s from 8 modified adults; and 9 M1 or I1s and 2 M3s from 10 non-modified adults. Comparison of their distributions reveals four general patterns of intra-tooth variation, described below and presented as log scale graphs in Figures 8.1-8.4. For each of these graphs, surface enamel peaks have been removed in order to emphasize the core enamel-lead trends that are the focus of this chapter. These interesting features are retained in all other graphs.

**Pattern A: A steady lead signal** that does not deviate much from the mean enamel-lead concentration reported in chapter seven (see Figure 8.1). The lack of major increases or decreases in lead concentration suggests lead is fairly evenly distributed throughout the enamel. Subadult, modified adult and non-modified adult teeth exhibit this pattern.

**Pattern B: A lead concentration that trends upward** (see Figure 8.2). This pattern of increasing lead deposition is evident amongst subadults and a few non-modified adults. Lead concentrations sometimes increase rather quickly and span a wide range of values, as is the case with Burial 126 (M1). For other individuals such as Burial 324 (M1 and M3), lead levels rise slowly by comparison and stay within a relatively limited range.

**Pattern C: The inverse of pattern B; for teeth with this pattern, lead concentrations decrease** from early- to late-formed enamel, i.e., with an individual’s chronological age. Here, again, the rate of change may vary, from the relatively rapid drop in lead concentrations seen for Burial 9’s M1 to the slight, more gradual decrease observed for that of Burial 335 (see Figure assigned base – here, 10. The result is a compressed scale that reduces analytical noise and enables easier comparison of trends across orders of magnitude.
8.3). Teeth of four modified adults and a single non-modified adult exhibit this pattern.

**Pattern D:** A more complicated, random or otherwise mixed lead signal, i.e., one that lacks an overall or unidirectional trend. Instead, zones of increasing or decreasing lead concentration are often discernible (see Figure 8.4). Teeth from several subadults, modified
adults and non-modified adults have this mixed lead signal. None of these patterns vary by sex or temporal group. Teeth exhibiting these different patterns are listed in Table 8.1.

These four patterns represent the intra-tooth variation observed for all subadult M1 teeth. However, LA-ICP-MS analysis of over half of the modified adult and about a quarter of the
Table 8.1: NYABG M1 or I1 enamel-lead microspatial distribution patterns by analytical cohort.

<table>
<thead>
<tr>
<th>Distribution Pattern</th>
<th>n</th>
<th>Burial(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Steady Pb signal (A)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subadults</td>
<td>5</td>
<td>43, 138, 180, 244, 405</td>
</tr>
<tr>
<td>Mod. Adults</td>
<td>2</td>
<td>47, 367</td>
</tr>
<tr>
<td>Non-mod. Adults</td>
<td>3</td>
<td>12, 150, 179</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>9</td>
<td></td>
</tr>
<tr>
<td><strong>Increasing Pb signal (B)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subadults</td>
<td>4</td>
<td>7, 35, 39, 126</td>
</tr>
<tr>
<td>Mod. Adults</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Non-mod. Adults</td>
<td>1</td>
<td>176, 324</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>5</td>
<td></td>
</tr>
<tr>
<td><strong>Decreasing Pb signal (C)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subadults</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Mod. adults</td>
<td>4</td>
<td>9, 106, 266, 366</td>
</tr>
<tr>
<td>Non-mod. Adults</td>
<td>1</td>
<td>335</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>5</td>
<td></td>
</tr>
<tr>
<td><strong>Mixed Pb signal (D)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subadults</td>
<td>2</td>
<td>22, 219</td>
</tr>
<tr>
<td>Mod. adults</td>
<td>2</td>
<td>101, 377</td>
</tr>
<tr>
<td>Non-mod. adults</td>
<td>3</td>
<td>25, 49, 323</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>7</td>
<td></td>
</tr>
</tbody>
</table>

non-modified adult M1 or I1s failed to produce stable lead signals above the 0.39 µg g\(^{-1}\) LOD.

Distribution graphs for many of these teeth are blank with the exception of surface enamel peaks or occasional signal spikes from ablation of fractured enamel. Thus, they are not useful for determining trends even for limited portions of the tooth crown. To illustrate, I include the graph for the Kasana control sample as Figure 8.5. This lack of a clear distribution pattern is evident for all teeth with mean lead concentrations below the LOD (0.39 µg g\(^{-1}\)) as well as those of Burials 6, 165 and 243, modified adults with quantified M1 or I1 concentrations.

Finally, only four (20%) of the M3s analyzed for this study produced stable lead signals;
two teeth from modified adults (Burials 9 and 101) and two teeth from non-modified adults (Burials 63 and 324). Their spatial distribution graphs appear in the following section and in Appendices C and D. The M3 lead signals for Burials 9, 63 and 101 are mixed, with no clear lead deposition trend while that of Burial 324 indicates a slight but continuous increase for most of the measured period. For Burial 63, there was no M1 or I1 available for comparison of lead levels or distribution. Intra-tooth variation for the other individuals and patterning of distributions (or lack thereof) across subadults, modified and non-modified adults is described below and discussed in more detail in chapter nine.

**Chronological Age Profiles**

In this section, I present enamel-Pb distribution/age profiles for NYABG subadults, modified adults and non-modified adults. These are visual representations of the intensity and timing of lead deposition at specific chronological ages. Each profile consists of a micro-

Figure 8.5: Enamel-lead distribution graph for the Kasana control tooth sample. Throughout much of the crown, lead concentrations are below instrument detection limits (0.39 $\mu$g g$^{-1}$). The signal spikes at the beginning and latter portion of the graph represent fractures within the tooth crown where low calcium readings due to less ablated enamel produce artificially high lead values.
photograph of a longitudinal cross section of an ablated tooth scaled to Reid and Dean’s (2006) enamel formation timing estimates with the corresponding microspatial distribution graph superimposed upon it. The graphs are positioned over the photographs such that the ablation tracks, indicated by red lines, are located in place of the x-axes.

Subadult distribution/age profiles are immediately below, followed by profiles for modified adults and, lastly, non-modified adults. Each profile includes: (1) the age and sex estimates of the individual, (2) the mean enamel-lead concentration for the sampled portion of the tooth, and (3) a detailed description of the enamel-lead distribution, including the lead signal pattern and an explanation of significant fluctuations or sustained increases and decreases with age.

**Subadults**

![Enamel-Pb/age graph for Burial 7 (LRM1), a 3- to 5-year-old subadult of undetermined sex. Mean enamel-lead concentration for this tooth is 2.86 μg g⁻¹. The red line indicates the location of the ablation track. This is an example of an increasing lead signal. Lead concentrations rise gradually until about 2.3 years of age when they decrease prior to a surface enamel peak. Note that the zone of highest lead concentration coincides with sampling near the dentine-enamel junction (DEJ). This zone is indicated by the red arrow.](image)
Figure 8.6b: Enamel-Pb/age graph for Burial 22 (LRM1), a 2.5- to 4.5-year-old subadult of undetermined sex. Mean enamel-lead concentration for this tooth is 4.66 μg g⁻¹. This is an example of a mixed and particularly noisy lead signal. Note that several peaks in the analytical signal are due to ablation of fractured enamel. Disregarding the first of these (indicated by the red arrow), lead concentrations rise from about 1.3 to 1.5y, then decrease until becoming somewhat stable after about 1.8 years.

Figure 8.6c: Enamel-Pb/age graph for Burial 35 (ULM1), an 8- to 10-year-old subadult of undetermined sex. Mean enamel-lead concentration for this tooth is 10.52 μg g⁻¹. This is an example of an increasing lead signal. Lead concentrations begin on the high end of the spectrum for NYABG individuals and rise fairly consistently from about 1.7 to 2.3y. Following a major fluctuation around 2.6y, lead levels again rise – and at a faster pace – throughout the remainder of the sampled enamel (i.e., until circa 2.8y). A similarly rapid increase in lead levels within late-formed enamel is seen for Burial 126.
Figure 8.6d: Enamel-Pb/age graph for Burial 39 (LRM1), a 5- to 7-year-old subadult of undetermined sex. Mean enamel-lead concentration for this tooth is 6.09 μg g\(^{-1}\). This is another example of an increasing lead signal. A slight, gradual increase in lead concentration begins around 1.5y and becomes more pronounced beginning at about 2y. Note, however, that lead levels decrease between 2.6 and 2.9y – and the presence of large occlusal and cervical surface enamel peaks.

Figure 8.6e: Enamel-Pb/age graph for Burial 43 (LRM1), a 2.5- to 4.5-year-old subadult of undetermined sex. Mean enamel-lead concentration for this tooth is 2.41 μg g\(^{-1}\). This is an example of a steady lead signal that indicates no major increases or decreases in lead concentration.
Figure 8.6f: Enamel-Pb/age graph for Burial 126 (LLM1), a 3.5- to 5.5-year-old subadult of undetermined age. Mean enamel-lead concentration for this tooth is 11.59 μg g⁻¹. This is an example of an increasing lead signal. Lead concentrations begin to increase just before 1.8y and, following a period of steady by relatively slow increase, rise dramatically beginning at about 2.3y. A similar zone of rapidly increasing lead concentrations in late-formed enamel is seen for Burial 35. Note that this tooth also has an area of raster ablation near the y axis and between 1.5-1.8 years from an earlier elemental signature analysis (ESA) study.

Figure 8.6g: Enamel-Pb/age graph for Burial 138 (URM1), a 3- to 5-year-old subadult of undetermined age. Mean enamel-lead concentration for this tooth is 2.30 μg g⁻¹. This is an example of a steady lead signal, with no major increases or decreases in lead concentration.
Figure 8.6h: Enamel-Pb/age graph for Burial 180 (ULM1), an 11- to 13-year-old female. Mean enamel-lead concentration for this tooth is 1.20 μg g$^{-1}$. This lead signal for this tooth is steady, with no major increases or decreases in lead concentration. Note the occlusal surface enamel peak.

Figure 8.6i: Enamel-Pb/age graph for Burial 219 (LRM1), a 4- to 5-year-old subadult of undetermined age. Mean enamel-lead concentration for this tooth is 14.70 μg g$^{-1}$, the highest value observed amongst NYABG individuals. This is an example of a mixed lead signal. Lead concentrations increase steadily from 1.2y, peak between 1.4 and 1.5y, and then decrease through 1.7y. From this point, lead levels are relatively stable given the large amount of lead concentration oscillations for this sample. Note that the occlusal surface enamel peak is small compared to those observed for most other subadults.
Figure 8.67j: Enamel-Pb/age graph for Burial 244 (LLM1), a 5- to 9-year-old subadult of undetermined age. Mean enamel-lead concentration for this tooth is 4.35 \( \mu g \) g\(^{-1}\). This is an example of a steady lead signal indicating no major increases or decreases in lead concentration. Note that the occlusal surface enamel peak is small relative to those observed for most other subadults.

Figure 8.6k: Enamel-Pb/age graph for Burial 405 (URM1), a 6- to 10-year-old subadult of undetermined age. Mean enamel-lead concentration for this tooth is 3.98 \( \mu g \) g\(^{-1}\). This is an example of a steady lead signal. A few minor fluctuations occur but there are no major increases or decreases in lead exposure/deposition. Note the presence of occlusal and cervical surface enamel peaks. In addition to the linear ablation, this tooth was also ablated in three other directions around mid-crown as part of an earlier study.
Figure 8.7a: Enamel-Pb/age graph for Burial 9 (LLM1), a 35- to 45-year-old female. Mean enamel-lead concentration for this tooth is 1.34 μg g$^{-1}$. Overall, lead concentrations decrease with distance/age. The slight increase in lead levels in the late-formed enamel likely results from sampling near the dentine-enamel junction (DEJ). Note the presence of occlusal and late-formed enamel surface peaks.

Figure 8.7b: Enamel-Pb/age graph for Burial 9 (LRM3), a 35- to 45-year-old female. Mean enamel-lead concentration for this tooth is 0.69 μg g$^{-1}$. This suggests low-level early-childhood exposure/deposition despite several spikes of considerable intensity, the largest of which exceeds the occlusal surface enamel peak. Her lead signal is mixed, decreasing from 9.3 to 9.7y and then increasing slowly after the large spike until about 10.4y. From this point, the signal decreases briefly (with a few more spikes) before rising again as the line scan approaches the crown cervix and dentine-enamel junction (DEJ).
Figure 8.7c: Enamel-Pb/age graph for Burial 47 (LLM1), a 35- to 45-year-old male. Mean enamel-lead concentration for this tooth is 1.31 μg g⁻¹. Disregarding the high concentrations at the latter portion of the graph, which correspond to fractured enamel, this is an example of distribution of a steady signal with no major increases or decreases in lead concentration. Note the lack of an occlusal surface enamel peak.

Figure 8.7d: Enamel-Pb/age graph for Burial 101 (LRI1), a 26- to 35-year-old male. Mean enamel-lead concentration for this tooth is 7.80 μg g⁻¹, the highest value observed for an NYABG adult. His “mixed” lead signal is consistently in the high range for NYABG individuals and indicates several periods of increased exposure/deposition prior to 1.6y and around 2.2y.
Figure 8.7e: Enamel-Pb/age graph for Burial 101 (LLM3), a 26- to 35-year-old male. Mean enamel-lead concentration for this tooth is 7.38 μg g⁻¹, quite similar to that observed for this individual’s M1. Likewise, the lead signal for this tooth is also mixed. Lead concentrations are consistently high with a period of marked increase; here, between 9.6 and 9.9y. Concentrations for the discolored portion of the tooth, which corresponds to a large carie, are not included in the mean calculation. Note the presence of an occlusal surface enamel peak.

Figure 8.7f: Enamel-Pb/age graph for Burial 106 (LRM1), a 25- to 35-year-old female (probable). Mean enamel-lead concentration for this tooth is 0.93 μg g⁻¹. For this tooth, lead concentrations decrease, albeit slightly, with distance/age.
Figure 8.7g: Enamel-Pb/age graph for Burial 266 (URM1), a 25- to 35-year-old female. Mean enamel-lead concentration for this tooth is 0.56 μg g⁻¹. This is another example of a decreasing lead signal. Note the presence of occlusal and (fractured) late-formed enamel surface peaks.

Figure 8.7h: Enamel-Pb/age graph for Burial 366 (LLM1), a 34- to 62-year-old adult of undetermined sex. Mean enamel-lead concentration for this tooth is > 0.39 μg g⁻¹. Lead concentrations decrease gradually with distance/age. The increased lead signal at 1.5y is due to ablation of fractured enamel. Note the presence of occlusal and late-formed enamel surface peaks.
Figure 8.7i: Enamel-Pb/age graph for Burial 367 (ULM1), a 25- to 35-year-old female (probable) adult of undetermined sex. Mean enamel-lead concentration for this tooth is 0.76 μg g⁻¹. This is a steady signal with no major increases or decreases in concentration indicating minimal lead exposure.

Figure 8.7j: Enamel-Pb/age graph for Burial 377 (ULM1), a 33- to 58-year-old female. Mean enamel-lead concentration for this tooth is 2.82 μg g⁻¹. This is an example of a steady lead signal. Lead levels rise modestly around 1.3 and 1.9y but overall there are no major increases or decreases in lead exposure/deposition.
Non-modified Adults

AGE (y)

1.2 1.3 1.4 1.5 1.7 1.9 2.0 2.3 2.6 2.8 3.0

$^{208}\text{Pb}$ Concentrations

Figure 8.8a: Enamel-$\text{Pb}$/age graph for Burial 12 (ULM1), a 35- to 45-year-old female. Mean enamel-lead concentration for this tooth is $< 0.39 \, \mu\text{g g}^{-1}$. The lead signal is quite steady with no major fluctuations. Note the surface enamel peak at the end of the graph.

Figure 8.8b: Enamel-$\text{Pb}$/age graph for Burial 25 (URI1), a 20- to 24-year-old female. Mean enamel-lead concentration for this tooth is $0.40 \, \mu\text{g g}^{-1}$. Its lead signal is mixed, rising and falling (occasionally below detection limit) throughout the crown. Note the presence of a surface enamel peak. Note, also, that the graph for this tooth represents a series of four line scans. The separate scans were necessary in order to sample the lingual enamel, which is less affected by the hypocalcification indicated by the cream and brown discoloration throughout the labial enamel.
Figure 8.8c: Enamel-Pb/age graph for Burial 49 ULM1, a 40- to 50-year-old female. Mean enamel-lead concentration for this tooth is $1.72 \, \mu g \, g^{-1}$. The lead signal is mixed. Lead concentrations decrease for a brief period (between 1.3 and 1.4y) and then rise slowly, peaking just after 2.0y and decreasing again thereafter. Note, however, the overall relatively poor condition of this tooth (enamel and dentine) and that the signal increase coincides with sampling of discolored, hypocalcified enamel. Occlusal and late-formed enamel surface peaks are present.

Figure 8.8d: Enamel-Pb/age graph for Burial 63 (LRM3), a 35- to 45-year old male. Mean enamel-lead concentration for this tooth is $2.08 \, \mu g \, g^{-1}$. This is an example of a mixed lead signal. Lead concentrations are below detection limits until 9.9y and then fluctuate substantially, reaching fairly high levels for this population, before peaking at the crown surface.
Figure 8.8e: Enamel-Pb/age graph for Burial 150 (LRM1), a 20- to 28-year-old female. Mean enamel-lead concentration for this tooth is < 0.39 μg g⁻¹. The lead signal is steady and concentrations are consistently low (under 1 μg g⁻¹) until the late-formed enamel surface peak.

Figure 8.8f: Enamel-Pb/age graph for Burial 176 (LRM1), a 20- to 24-year-old male. Mean enamel-lead concentration for this tooth is < 0.39 μg g⁻¹. This is an example of an increasing lead signal, with concentrations rising slowly from about 1.8 to 2.6y. Note that the peaks on either side of this graph result from ablating fractured, not surface, enamel. Note also two areas of raster ablation.
Figure 8.8g: Enamel-Pb/age graph for Burial 179 (LRM1), a 25- to 30-year-old male. Mean enamel-lead concentration for this tooth is 1.60 μg g\(^{-1}\). This is an example of a steady lead signal, with no major fluctuations besides an increase due to sampling fractured enamel just before the surface enamel peak.

Figure 8.8h: Enamel-Pb/age graph for Burial 323 (LRM1), a 19- to 30-year-old male. Mean enamel-lead concentration for this tooth is 4.35 μg g\(^{-1}\); relatively high for NYABG adults. The lead signal is mixed. Lead concentrations rise roughly threefold, from about 2 to 6 μg g\(^{-1}\), between 1.3 and 1.4y. The signal then decreases until approximately 1.7y and, from that point, concentrations fluctuate between 4 and 8 μg g\(^{-1}\) throughout the remainder of the core enamel. Note the presence of two surface enamel peaks.
Figure 8.8i: Enamel-Pb/age graph for Burial 324 ULM1, a 25- to 35-year-old female. Mean enamel-lead concentration for this tooth is 1.39 μg g⁻¹. The lead signal increases continuously with distance/age. Note the occlusal surface enamel peak and the large spike in the late-formed enamel.

Figure 8.8j: Enamel-Pb/age graph for Burial 324 (LRM3), a 25- to 35-year-old female. Mean enamel-lead concentration for this tooth is 1.96 μg g⁻¹, slightly higher than that of her M1. Beginning at 9.4y, the lead signal increases until almost 10.4y. Thus, the main trend observed for this tooth, like that of her M1, is a gradual increase in lead concentration. Here, however, the signal decreases slightly as it nears the cervical region of the crown.
Figure 8.8k: Enamel-Pb/age graph for Burial 335 (LLLM1), a 25- to 35-year-old female. Mean enamel-lead concentration for this tooth is 0.42 μg g$^{-1}$. Lead concentrations are consistently low (i.e., under 1 μg g$^{-1}$) and decrease gradually with distance/age with the exception of the surface enamel peak.

The distribution patterns of enamel-lead described and illustrated above are not group-specific although some clustering is evident for subadults and modified adults. Nine of the 11 subadults exhibit either a steady (pattern A) signal or increasing (pattern B) lead concentrations with age. Two subadults had random or mixed signals identified as pattern D while none showed evidence of decreasing lead deposition with age (pattern C). Similarly, three patterns – A, C and D – are found amongst modified adults, with a majority represented by patterns C (decreasing lead deposition with age) or D (random). None of the modified adults had increasing lead concentrations. All four patterns could be found amongst non-modified adults. It may be worth noting that the subadult (Burial 219), modified adult (Burial 101) and non-modified individuals (Burial 323) with the highest enamel-lead concentrations for their respective groups all had random/mixed early life signals, possibly indicating high-level but also episodic exposure.
Early Versus Late Enamel-lead

I lastly report on a comparison of mean lead concentrations of core enamel formed before and after age two, which marks a critical period when children are most vulnerable to environmental lead (see chapter five). I compared mean concentrations for “early” (pre-age two) and “late” (post-age two) stages of M1 or I1 enamel development for 27 of the 28 individuals with age profiles. For most M1s, line scans captured enamel that formed over the course of approximately 12 or 13 months, from about 1.4 or 1.5 until 2.6 years of age. Scans of I1 teeth for Burials 25 and 101 sampled a greater age range, particularly for late enamel. The results for subadults, modified adults and non-modified adults are listed by individual/tooth in Table 8.2 and by sex and temporal group in Tables 8.3 and 8.4, respectively.

For the entire sample, mean lead concentration increases from 2.85 ± 3.27 μg g⁻¹ (range: 0.21 to 15.3) for early enamel to 3.67 ± 4.92 μg g⁻¹ (range: 0.15 to 21.04) for late enamel. Sixteen (59%) of the NYABG individuals – including 6 subadults, 6 modified adults and 4 non-modified adults – follow this pattern of higher mean concentrations for late enamel. The difference between early and late enamel lead levels was minimal or even negligible for some individuals such as Burial 244 whose mean concentrations decreased from 4.35 to 4.34 μg g⁻¹. Others experienced far greater change in lead deposition during the first years of life. The greatest difference in early and late lead levels occurs for Burial 126, for example, a subadult whose mean concentrations increased nearly fivefold after age two, from 4.26 to 21.0 μg g⁻¹. Overall, subadults follow the general pattern of more lead deposition after age two and have the highest mean concentrations of all subsamples: 4.86 ± 4.01 μg g⁻¹ and 6.95 ± 6.08 μg g⁻¹ for early and late enamel, respectively. For adults, modified and non-modified, early enamel values are greater. The decrease in early to late mean concentration is slight for both adult

---

30 Sampling captured only early enamel formed prior to age two for Burial 323, a non-modified adult male (see Table 8.2; Figure 8.5h).
Table 8.2. NYABG early and late M1 or I1 enamel-lead concentrations.

<table>
<thead>
<tr>
<th>Burial</th>
<th>Sample</th>
<th>Early (Pre-2y)</th>
<th>Late (Post-2y)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subadults (n = 11)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>LRM1</td>
<td>1.75</td>
<td>5.16</td>
</tr>
<tr>
<td>22</td>
<td>LRM1</td>
<td>5.50</td>
<td>3.09</td>
</tr>
<tr>
<td>35</td>
<td>ULM1</td>
<td>8.54</td>
<td>14.2</td>
</tr>
<tr>
<td>39</td>
<td>LRM1</td>
<td>4.28</td>
<td>8.50</td>
</tr>
<tr>
<td>43</td>
<td>LRM1</td>
<td>2.13</td>
<td>2.85</td>
</tr>
<tr>
<td>126</td>
<td>LLM1</td>
<td>4.26</td>
<td>21.0</td>
</tr>
<tr>
<td>138</td>
<td>URM1</td>
<td>2.45</td>
<td>2.09</td>
</tr>
<tr>
<td>180</td>
<td>ULM1</td>
<td>1.26</td>
<td>1.08</td>
</tr>
<tr>
<td>219</td>
<td>LRM1</td>
<td>15.3</td>
<td>9.96</td>
</tr>
<tr>
<td>244</td>
<td>LLM1</td>
<td>4.35</td>
<td>4.34</td>
</tr>
<tr>
<td>405</td>
<td>URM1</td>
<td>3.69</td>
<td>4.19</td>
</tr>
<tr>
<td>Mean ± SD</td>
<td></td>
<td>4.86 ± 4.01</td>
<td>6.95 ± 6.08</td>
</tr>
<tr>
<td>Modified adults (n = 8)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>LLM1</td>
<td>1.51</td>
<td>1.08</td>
</tr>
<tr>
<td>47</td>
<td>LLM1</td>
<td>1.24</td>
<td>1.49</td>
</tr>
<tr>
<td>101</td>
<td>LRI1</td>
<td>7.34</td>
<td>8.31</td>
</tr>
<tr>
<td>106</td>
<td>LRM1</td>
<td>0.94</td>
<td>0.84</td>
</tr>
<tr>
<td>266</td>
<td>URM1</td>
<td>0.65</td>
<td>0.42</td>
</tr>
<tr>
<td>366</td>
<td>LLM1</td>
<td>0.41</td>
<td>0.29</td>
</tr>
<tr>
<td>367</td>
<td>ULM1</td>
<td>0.78</td>
<td>0.73</td>
</tr>
<tr>
<td>377</td>
<td>ULM1</td>
<td>2.93</td>
<td>2.62</td>
</tr>
<tr>
<td>Mean ± SD</td>
<td></td>
<td>1.98 ± 2.30</td>
<td>1.97 ± 2.66</td>
</tr>
<tr>
<td>Non-mod. adults (n = 9)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>ULM1</td>
<td>0.27</td>
<td>0.17</td>
</tr>
<tr>
<td>25</td>
<td>URI1</td>
<td>0.29</td>
<td>0.43</td>
</tr>
<tr>
<td>49</td>
<td>ULM1</td>
<td>1.76</td>
<td>1.45</td>
</tr>
<tr>
<td>150</td>
<td>LRM1</td>
<td>0.36</td>
<td>0.15</td>
</tr>
<tr>
<td>176</td>
<td>LRM1</td>
<td>0.21</td>
<td>0.42</td>
</tr>
<tr>
<td>179</td>
<td>LRM1</td>
<td>1.53</td>
<td>1.74</td>
</tr>
<tr>
<td>323</td>
<td>LRM1</td>
<td>4.35</td>
<td></td>
</tr>
<tr>
<td>324</td>
<td>ULM1</td>
<td>1.12</td>
<td>2.14</td>
</tr>
<tr>
<td>335</td>
<td>LLM1</td>
<td>0.48</td>
<td>0.36</td>
</tr>
<tr>
<td>Mean ± SD</td>
<td></td>
<td>1.15 ± 1.33</td>
<td>0.86 ± 0.79</td>
</tr>
</tbody>
</table>

Sample mean ± SD | 2.85 ± 3.27 | 3.67 ± 4.92 |

*a* Tooth type/position key: L or U = lower or upper; L or R = left or right; M or I = molar or incisor; 1 or 3 = first or third. All teeth are from the permanent dentition (e.g., LLM1 = permanent lower left first molar).

*b* Only early enamel was sampled for this individual.
Table 8.3. NYABG M1 or I1 early and late enamel-lead concentrations by sex and analytical cohort.

<table>
<thead>
<tr>
<th>Sex</th>
<th>n</th>
<th>Early (Pre-2y) [Pb] (μg g⁻¹) (mean ± SD)</th>
<th>Min-Max</th>
<th>Late (Post-2y) [Pb] (μg g⁻¹) (mean ± SD)</th>
<th>Min-Max</th>
</tr>
</thead>
<tbody>
<tr>
<td>Females</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subadults</td>
<td>1</td>
<td>1.26</td>
<td>N/A</td>
<td>1.08</td>
<td>N/A</td>
</tr>
<tr>
<td>Mod. Adults</td>
<td>4</td>
<td>1.33 ± 1.08</td>
<td>0.65 – 2.93</td>
<td>1.15 – 0.99</td>
<td>0.42 – 2.62</td>
</tr>
<tr>
<td>Non-mod. adults</td>
<td>6</td>
<td>0.71 ± 0.60</td>
<td>0.27 – 1.76</td>
<td>0.78 ± 0.82</td>
<td>0.15 – 2.14</td>
</tr>
<tr>
<td>Total</td>
<td>11</td>
<td>0.99 ± 0.79</td>
<td>0.27 – 2.93</td>
<td>0.94 ± 0.82</td>
<td>0.15 – 2.62</td>
</tr>
<tr>
<td>Males</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subadults</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Mod. Adults</td>
<td>3</td>
<td>3.36 ± 3.45</td>
<td>1.24 – 7.34</td>
<td>3.63 ± 4.06</td>
<td>1.08 – 8.31</td>
</tr>
<tr>
<td>Non-mod. adults</td>
<td>3</td>
<td>2.03 ± 2.11</td>
<td>0.21 – 4.35</td>
<td>1.08 ± 0.93</td>
<td>0.42 – 1.74</td>
</tr>
<tr>
<td>Total</td>
<td>6</td>
<td>2.70 ± 2.66</td>
<td>0.21 – 7.34</td>
<td>2.61 ± 3.23</td>
<td>0.42 – 8.31</td>
</tr>
<tr>
<td>Undetermined</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subadults</td>
<td>10</td>
<td>5.23 ± 4.04</td>
<td>1.75 – 15.3</td>
<td>7.54 ± 6.07</td>
<td>2.09 – 21.0</td>
</tr>
<tr>
<td>Mod. Adults</td>
<td>1</td>
<td>0.41</td>
<td>N/A</td>
<td>0.29</td>
<td>N/A</td>
</tr>
<tr>
<td>Non-mod. adults</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Total</td>
<td>11</td>
<td>4.79 ± 4.10</td>
<td>0.41 – 15.3</td>
<td>6.88 ± 6.16</td>
<td>0.29 – 21.0</td>
</tr>
</tbody>
</table>

Subsamples, especially modified adults for whom these values are nearly identical (see Table 8.2). For early enamel, the subadult value is over twice that of modified adults and over four times that of non-modified adults. Late enamel mean concentration for subadults is over four times that of modified adults and over eight times the non-modified adult value.

As indicated in Table 8.3, overall mean concentration decreases slightly after age two for males and females. Early and late enamel mean values are nearly threefold higher for males than for females. Male individual values also range much more widely for both age ranges. Within male and female cohorts, most of the differences observed between early and late mean concentrations for modified and non-modified adults are also minimal. For individuals with sex estimates, only non-modified adult males show a substantial difference in early and late enamel
Table 8.4. NYABG M1 or I1 early and late enamel-lead concentrations by temporal group and analytical cohort.

<table>
<thead>
<tr>
<th>a Temporal group</th>
<th>n</th>
<th>Early (Pre-2y)</th>
<th>Min-Max</th>
<th>Late (Post-2y)</th>
<th>Min-Max</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Middle</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subadults</td>
<td>5</td>
<td>5.25 ± 1.95</td>
<td>3.69 – 8.54</td>
<td>10.2 ± 7.47</td>
<td>3.09 – 21.0</td>
</tr>
<tr>
<td>Mod. adults</td>
<td>4</td>
<td>0.99 ± 0.49</td>
<td>0.41 – 1.51</td>
<td>0.90 ± 0.51</td>
<td>0.29 – 1.49</td>
</tr>
<tr>
<td>Non-mod. adults</td>
<td>4</td>
<td>0.91 ± 0.67</td>
<td>0.29 – 1.76</td>
<td>1.10 ± 0.86</td>
<td>0.36 – 2.14</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>13</td>
<td>2.60 ± 2.49</td>
<td>0.29 – 8.54</td>
<td>4.54 ± 6.37</td>
<td>0.29 – 21.0</td>
</tr>
<tr>
<td><strong>Late-Middle</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subadults</td>
<td>3</td>
<td>6.39 ± 7.72</td>
<td>1.75 – 15.3</td>
<td>5.99 ± 3.63</td>
<td>2.85 – 9.96</td>
</tr>
<tr>
<td>Mod. adults</td>
<td>3</td>
<td>3.74 ± 3.28</td>
<td>0.94 – 7.34</td>
<td>3.92 ± 3.90</td>
<td>0.84 – 8.31</td>
</tr>
<tr>
<td>Non-mod. adults</td>
<td>1</td>
<td>0.21</td>
<td>N/A</td>
<td>0.42</td>
<td>N/A</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>7</td>
<td>4.37 ± 5.34</td>
<td>0.21 – 15.3</td>
<td>4.31 ± 3.67</td>
<td>0.42 – 9.96</td>
</tr>
<tr>
<td><strong>Late</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subadults</td>
<td>3</td>
<td>2.69 ± 1.56</td>
<td>1.26 – 4.35</td>
<td>2.50 ± 1.67</td>
<td>1.08 – 4.34</td>
</tr>
<tr>
<td>Mod. adults</td>
<td>1</td>
<td>0.65</td>
<td>N/A</td>
<td>0.42</td>
<td>N/A</td>
</tr>
<tr>
<td>Non-mod. adults</td>
<td>4</td>
<td>1.63 ± 1.90</td>
<td>0.27 – 4.35</td>
<td>0.69 ± 0.91</td>
<td>0.15 – 1.74</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>8</td>
<td>1.90 ± 1.67</td>
<td>0.27 – 4.35</td>
<td>1.43 ± 1.49</td>
<td>0.15 – 4.34</td>
</tr>
</tbody>
</table>

a Data for the Early Group are not presented because the enamel-lead concentration for the sole representative was below the LOD (< 0.39 μg/g).

b Only early enamel was sampled for non-modified adult Burial 323 LRM1.

values, i.e., a decrease of 0.95 μg g⁻¹. In contrast to the overall adult pattern, late enamel values increase slightly for non-modified adult females and for modified adult males.

I provide data on temporal group differences in early and late enamel-lead concentrations in Table 8.4. Within the Middle group (circa 1735 to 1760), mean enamel lead generally increases after age two. Only modified adults exhibit a very slight increase in mean late enamel concentration. Subadults and non-modified adults follow the major trend. The only significant difference in early and late enamel-lead values for this time period is observed for subadults, for whom the late enamel value is nearly double that of early enamel. Subadult
values are higher than modified and non-modified adults within all temporal groups, but the differences are greatest in this period.

For the Late-Middle period (circa 1760 to 1776), overall mean concentration is highest for early enamel although the difference between early and late enamel values is not very large; a decrease of 0.06 μg g⁻¹. Lead concentration is also higher in early enamel for the Late (post-1776) period. Across temporal groups, early enamel mean concentration increases from the Middle to the Late-Middle period and then decreases to its lowest level for the Late period. Late enamel values decrease slightly from the Middle to Late-Middle period and then more substantially – again, to the lowest level – for the Late group.

**Conclusions**

As captured in microspatial and age distributions, enamel-lead was prevalent and concentrations were especially high and variable for subadults. At the individual level, distribution patterns indicate that levels of early-life deposition remained stable, increased, decreased or reflected some combination of these trends. Increasing or fluctuating (“random”) lead concentrations may be accounted for under conditions of early American industrialization where lead use and exposure opportunities abounded. Decreasing concentrations are less easily explained but perhaps reflect age-related metabolic changes, declining maternal contributions, or other factors. This question will be explored further.

While none of these patterns are exclusive to specific groups, teeth of subadults were most likely to reveal chronic (steady) or increased deposition. Recall that all subadults or children appear to have been born in New York or somewhere else in the Americas. Age profiles suggest that increased deposition, probably due to greater childhood exposure, often occurred around and after age two. Random or combined patterns were also useful for determining
specific ages when lead deposition/exposure changed substantially (see, for example, Burial 219
[Figure 8.6i]). In the next chapter, I attempt to interpret these data, drawing upon historical,
archaeological and other skeletal biological research for necessary context.
CHAPTER 9
DISCUSSION

This study builds upon a number of recent bioarchaeological investigations that use dental chemistry to probe the lives of historic individuals. Like genetic research, chemical studies of the body serve as the basis for reconstructing collective and individual histories – intimate life stories – once unimaginable or beyond analytical reach. Arguably, in some respects, chemical life profiles are even more informative than those produced from alleles. Chemical elements and isotopes can quite possibly reveal a wider range of actual experiences as opposed to ancestral backgrounds or health susceptibilities that may not have directly influenced an individual’s well-being. The chemistry of the body, and teeth especially, reflect what actually happened. The hard part, now that we can measure tooth chemistry, is interpreting the possible significance for individuals and groups. That question – (What does it mean?) – is the focus of this chapter.

Researchers are now capable of exploring simultaneously numerous trace element interactions and patterns of isotopic variation from skeletal remains and are using these data to identify and compare experiences of residential mobility, nutrition and pollution within and across populations or within a single lifetime (Dolphin and Goodman 2009; Prowse et al. 2010; Webb et al. 2005). Combined with historical data, multi-elemental/isotopic analysis of dental enamel in particular is a powerful means of reconstructing, refining and even reshaping human pasts by offering new perspectives on old questions (Goodman et al. 2009; Turner and Armelagos 2012; Price et al. 2012; Schroeder et al. 2009). This is not the extent of their value, however. In addition to confirming or challenging existing interpretations of past peoples, combinations of elemental and isotopic analyses can open new biological windows onto often complex social, cultural and individual histories whose interpretations may require entirely new
theories of bodies as “sites of memory” (Morrison 1995) and material culture (Sofaer 2006), and
new understandings of human pasts in relation to the present (Armelagos 2003; Singleton
2010).

I have taken a different approach from combining multiple chemical methods and instead have focused on a single element: lead. LA-ICP-MS analysis involved the measurement of concentrations for various elements at tissue level, which should shed light on nutritional and other aspects of health status for NYABG individuals. Yet, this dissertation study focuses solely on lead as a central “element of diaspora” with unique biomonitoring value, the exploitation of which can illuminate otherwise hidden aspects of African-American ethnogenesis. As explained in chapter five, this singular focus is warranted for the NYABG population on at least three fronts.

First, lead use was prevalent in the colonial Americas but limited in those parts of West Africa where most enslaved individuals were captured. Since the 1980s, bioarchaeologists have recognized lead’s usefulness for distinguishing African- from American-born individuals based upon this distribution of lead technology throughout the 18th-century Atlantic world (Handler et al. 1986; Corruccini et al. 1987; Aufderheide et al. 1981, 1988). Analysis of tooth enamel at micrometer resolution by LA-ICP-MS is a methodological update of this approach. In addition to mean concentrations, laser sampling measures lead’s distribution within a tooth along a time/age-axis. As a result, it is now possible to assess the nature and intensity of exposure during developmentally important stages.

Secondly, by focusing on colonial-era exposure during childhood, this study has begun to fill several important gaps in the scientific literature. Much of what is known about lead poisoning amongst enslaved Africans has been inferred from historical accounts of lead sources or derived from bone-lead analysis (e.g., Handler et al. 1986; Rathbun 1987). Although the basis
for confirming the “hidden epidemic” of lead poisoning in colonial Barbados, bone chemistry is fraught with interpretive limitations and pitfalls with respect to the reconstruction of biogenic, age-based exposures (Barbosa et al. 2005; Wittmers 2002). By comparison, early-life lead exposure and possible health consequences may be more accurately reconstructed and inferred via tooth enamel. However, such studies are limited in number within anthropology (see Goodman et al. 2009 for an exception). As the first study to apply an explicitly political ecological framework to the quantitative measurement of enamel-lead via LA-ICP-MS, these findings should prove of interest to anyone concerned with the history of lead poisoning and environmental health as well as social bioarchaeology and the bioarchaeology of childhood, important emerging areas of study.

Third, lines of inquiry and evidence from lead analysis potentially converge in interesting ways to reveal both biological and cultural consequences of social vulnerability. Lead poisoning is yet another dimension of the “biology of poverty” (Thomas 1998; Leatherman and Thomas 2001). What new insights into patterns of lead exposure in early New York does a critical biocultural framework offer? How might study findings influence future studies of African diasporic biohistory and culture? As we shall see, a political ecological approach can expand the scope of inquiry and, thereby, of knowledge concerning the cultural geography of slavery.

Enamel-lead data presented in the preceding two chapters reveal considerable variation with respect to the extent, timing and nature of early-life lead burden. Throughout the remainder of this chapter, I explore the significance of this variation for reconstructing this diasporic population’s biocultural beginnings. The discussion unfolds in three parts as I consider how the results of analysis may provide or supplement information useful for determining: (1) natality or geographic origins, (2) sources and pathways of lead exposure, and (3) possible
health consequences for NYABG individuals. Discussion revolves around the following major study findings, which suggest who was most vulnerable to lead.

- A slight majority (53%) of the NYABG sample – all adults – appears to have been born in Africa. American-born individuals include subadults, modified adults and non-modified adults. The relatively high enamel-lead concentrations suggestive of American natality for four modified adults are quite intriguing in light of the longstanding hypothesis that dental modification indicates African birth (Handler 1995; Handler et al. 1986; Schroeder et al. 2012).

- Age-based variation was measured in several ways. M1 or I1 mean concentration is significantly higher and distribution of values is wider for subadults than for both modified and non-modified adults. Individual M1 or I1 tooth means are quite variable but tend to cluster into three ranges. Most adult teeth have values below 2 μg g⁻¹. Most subadult teeth fall between 2 and 6 μg g⁻¹, and a small number of individuals have values above 6 μg g⁻¹. A review of the enamel-lead literature, including a study of early-life lead exposure amongst enslaved Barbadians (Schroeder et al. 2013) discussed below, suggests that most individuals in this study experienced relatively low-level exposures. However, some individuals – mostly subadults – quite possibly experienced severe symptoms of lead poisoning.

- Comparison of lead concentrations in enamel formed before and after age two also suggests subadults, on average, experienced lead-enriched early childhood environments. Of four individuals for whom M1/I1-M3 comparisons were possible,
three exhibit a slight increase in mean concentration from M1s to late-forming M3s while a slight decrease is observed for the fourth.

- Lead burden differed by sex as well, with mean M1/I1 concentration for males over twice that of females. This pattern is not statistically significantly. However, it is consistent amongst modified and non-modified adults and across temporal groups, a reflection of variation in the sources or intensity of lead exposure, perhaps, or possibly of sex-based differences in early life lead metabolism and biokinetics.

- Overall, lead levels decrease within the sample cohort from approximately 1735 through the end of the 18th century. However, the decline in lead concentrations is not steady but occurs despite a slight increase circa 1735 until the onset of the Revolutionary War. This finding is somewhat surprising given a general increase in lead usage and, thus, environmental lead during the 18th century.

- Four enamel-lead distribution patterns are evident for M1/I1 and M3 teeth. With age (i.e., over microspatial distance), the lead signal either (1) is relatively steady or flat, (2) increases, (3) decreases, or (4) fluctuates randomly. Interestingly, none of these patterns are exclusive to any demographic group (age, sex or temporal) although not all groups are represented by each pattern.
Natality

Identifying enslaved individuals’ geographic origins is a fundamental concern of African diasporic bioarchaeology and was a primary goal of the African Burial Ground Project. The natal estimations presented above represent refinements of those developed by Goodman et al. (2009) through elemental signature analysis, which involved semiquantitative LA-ICP-MS measurement of lead in relation to numerous other trace metals. Enamel-lead concentrations serve as an independent chemical source of information regarding individuals’ birthplaces and early residences. The current estimates will be further refined through the addition of strontium, oxygen and carbon isotope data. Here, I integrate these chemical data with skeletal, historical and cultural data as an initial step toward clarifying and contextualizing observed patterns. African Burial Ground researchers have identified the primary sources and routes from which enslaved Africans arrived in early New York (see chapter four). Historical and genetic research indicates that most enslaved New Yorkers originated in West and West Central Africa. Through pirating, some Malagasy were imported into New York from Southeast Africa (Madagascar). While enamel-lead data does not pinpoint specific regions within Africa or the Americas where a person was born, determining continental natality for NYABG individuals is a major step toward understanding how slavery and the processes of enslavement influenced individual health trajectories and patterns of cultural production.

Before addressing the question of individuals’ geographic origins directly, it is worth pointing out that the mean sample concentration of 2.90 μg g\(^{-1}\) for M1/I1s does not rank highly among reported enamel-lead values for human populations of known technological exposure, which range from 0.04 to 82 μg g\(^{-1}\) (Fergusson and Purchase 1987; Budd et al. 2004). It should be noted, however, that the wide variety of methodologies employed in studies of enamel-lead complicates comparison of values across studies, which entail a range of tissue preparation,
sampling and analytical techniques. Here, direct comparison of NYABG core enamel-lead concentrations with those reported for other populations may prove somewhat dubious in the absence of other quantitative LA-ICP-MS data. Nonetheless, this relatively low sample mean concentration is plausible given the mixed residential origins of early African New Yorkers that would have included regions with varying levels of environmental lead.

The first step in assessing African and American natality from enamel-lead concentrations was to determine an upper limit value associated with low-level or non-technological exposure. I used the threshold of 1 μg g\(^{-1}\) suggested by Budd et al. (2004; see chapter 6). I then compared mean concentrations for the entire sampled portion of M1 or I1 teeth and the portion developed after age two for each individual. While the former provides a fuller depiction of early-childhood exposure, the latter likely best reflects environmental lead conditions without direct maternal input via breastfeeding. No substantial differences were found with respect to the 1 μg g\(^{-1}\) threshold. For all individuals, total mean concentrations above or below the threshold correspond to similarly high- or low-level exposure exclusively after age two. Thus, mean (total) M1 or I1 concentrations were deemed appropriate for natal estimation, with values above 1 μg g\(^{-1}\) attributed to American natality and those below considered indicative of natal Africans. Based upon this criteria, enamel-lead data suggest that a slight majority (53%; \(n = 21\)) of the 40 NYABG individuals with sampled M1 or I1s were born in Africa. This subsample includes the following 14 (78%) adults with CMT and 7 (64%) non-modified adults.


**Non-modified adults**: Burials 12, 25, 150, 172, 176, 196 and 335

The American-born cohort includes subadults and individuals from both categories of adults.

**Subadults**: Burials 7, 22, 35, 39, 43, 126, 138, 180, 219, 244 and 405

**Modified adults**: Burials 9, 47, 101 and 377
**Non-modified adults**: Burials 49, 179, 323 and 324

These findings compare quite well with the hypothesized residential origins. American natality was deemed most likely for subadults given slavers’ general preference for importing captives – most often young adult males – capable of carrying out the difficult labor of building early New York and the unlikelihood that children, particularly young children, withstood the rigors of the Middle Passage in appreciable numbers. Historian Paul Lovejoy (2006) observes that there were very few children age 2 – 5 in the Transatlantic Slave Trade. However, age and sex structure of imported captives varied over time and regionally with labor demands. For example, Medford and co-workers (2009) indicate that slightly more females than males were imported to British colonies from the Bight of Biafra between 1658 and 1713.

Some very young children did make the journey as well. Although relatively few in number, Medford et al. (2009) note that children as young as four years old were imported to New York to learn trades and provide domestic labor. West Central Africa was a primary source of enslaved children but throughout coastal Africa, slave traders balanced the demand for healthy and strong young adults and the need to quickly fill cargoes, even if this entailed the purchase of children (Lovejoy 2006). Thus, although mean subadult values were four to five times higher than modified and non-modified adult values, respectively, it is interesting that all of the children appear to have been born in the Americas. It is perhaps noteworthy, also, that the lowest subadult values belong to Burial 180, the oldest subadult at age 12. Burial 180’s mean M1 and post-2 year-old concentrations – 1.20 and 1.08 μg g⁻¹, respectively – are not much higher than the suggested 1.0 μg g⁻¹ background limit.

There were no expectations with respect to the specific geographic origins of most non-modified adults. As hypothesized, these individuals originated throughout Africa and the Americas. The sole exception is Burial 323, a 19-to-30-year-old male for whom prior strontium
isotope analysis indicated probable American natality (Goodman et al. 2009). Interestingly, Burial 323’s M1 and M3 $^{87}\text{Sr} / ^{86}\text{Sr}$ ratios were lower than most subadults’ and below the 0.711 to 0.712 hypothesized local Manhattan range, possibly indicating Caribbean origins (see chapter 4, Figure 4.6). Interestingly, this value is just above Price et al.’s (2012) predicted range of 0.7077 to 0.7092 for Campeche, Mexico.

The chemical indication that a small number of modified adults may have been born in the Americas is intriguing. Several researchers left unresolved the question of modified individuals’ geographic origins (e.g., Ortner 1966; Stewart 1942), but in most instances where natality is estimated, CMT are attributed to residential origins in Africa. Evidence for this perspective comes in part from ethnohistorical accounts of slave traders in coastal Africa who selected against individuals with dental modification. For example, Newson and Minchin (2004) report 82 cases of missing teeth from a list of daños (defects) recorded for 291 captives shipped from Angola and Upper Guinea to Cartagena, Colombia between December 1623 and September 1633. The authors attribute most cases to poor nutritional and dental health, but also note several entries referring to “...‘missing teeth, two below and two above’ that might suggest deliberate removal...” (Newson and Minchin 2004: 25). If these were in fact cases of intentional tooth ablation – whether or not slave traders recognized them as such – these cultural “defects” would have resulted in discounted prices for the affected individuals upon arrival in the Americas; a strong disincentive for including individuals thus modified in slave cargoes. Selection against CMT by slave traders may at first seem an indication that dental modification necessarily happened in the Americas. However, it is important to realize that slave traders’ biases in Africa reflected slaveholders’ aesthetic tastes and concern over the health and fitness of enslaved Africans upon arrival in the Americas. These were primary economic considerations.
Ethnohistoric and skeletal chemistry of diasporic communities provide direct evidence of dental modification as a marker of African natality. As detailed in chapter five, Jerome Handler and co-workers (Handler et al. 1982, 1986; Corruccini et al. 1987) articulated this view compellingly as part of their bioarchaeological investigation of the Newton Plantation in Barbados, finding low bone-lead concentrations for modified individuals. In a follow-up study involving 18th-century runaway ads, Handler (1994: 118) observes that indelibly altered teeth would have presented a major problem during escape attempts and, thus, concludes that “Investigators who discover remains of persons of identifiable African ancestry showing signs of tooth mutilation can conclude with a certain degree of confidence that such persons were born in Africa and not in the New World.” While the skeletal studies of Newton Plantation remains involved bone-lead analysis, recent tooth chemistry from sites in Mexico and the Caribbean also support the association of CMT with African natality (Price et al. 2006, Cucina et al. 2011; Schroeder et al. 2012).

Perhaps understandably, then, most interpretations of CMT emphasize reasons that enslaved Africans would have discontinued dental modification under slavery in the Americas – despite the practice’s ethnographically documented presence in later diasporic Africans (e.g., Ortiz 1929; Higman 1998). Slave trader bias against modified individuals in Africa and the difficulties that CMT would have posed for runaways in the Americas certainly help to explain the limited number of individuals with CMT reported thus far from the diasporic skeletal record. But do these factors also explain the exclusive association of CMT with African geographic origins, i.e., the implied sudden “disappearance” of a cultural practice then widespread in western Africa?

The possible American origins of Burials 9, 47, 101 and 377 – adults with CMT – suggest otherwise. This finding is fascinating given the universal association of CMT with African natality
noted above and raises interesting and important questions about biology as a medium of contested cultural and social power. Specifically, it underscores the importance of theorizing diasporic maintenance as well as discontinuance of dental modification. Most current studies account only for the latter possibility. As a result, and probably unintentionally, they tend to align theoretically with the position that the Middle Passage and enslavement “denuded black people of any ancestral heritage from Africa” (Brown 2009: 1241). From this perspective, slavery was a form of “social death” through alienation that rendered the enslaved culturally empty vessels (Patterson, 1982). Once dominant within the historiography of slavery, the “culture loss” position has always been contentious; one side in the major debate over the presence and nature of “Africanisms” or cultural “retentions” and “transformations” in the Americas (Brown 2009, 2010; Perry and Paynter 1995; Mintz and Price 1992; Price 2010; Walker 2001).

In the extreme, this position juxtaposes elite power on one hand and agency under oppression on the other, as mutually exclusive rather than shared, coexisting realities. In other words, the prevailing focus on CMT as defective or maladaptive in the context of slavery presents only one side of an equation that fails to account, conversely, for what Scott (1985) has termed “weapons of the weak,” or the will and power of people to maintain and assert both their individuality and collective humanity even under conditions of extreme oppression (see also Levine 1977). There is little question that diasporic Africans abandoned cultural dental modification more quickly than other expressive biocultural ties to Africa such as “country marks” (cicatrization), quite possibly due to external social pressures suggested by Handler (1994) and others. However, association of dental modification solely with natal Africans would seem to indicate their complete disavowal of its practice on emic terms as well. Although possible, this level of complicity with the sudden demise of a longstanding cultural practice invested with social and ethnic meaning is unlikely and at odds with current theories of diaspora
as both a condition and a process (Olaniyan and Sweet 2010) as well as emerging understandings of how cultural and historical memories are forged and reproduced (Eyerman 2004; Patterson and Kelley 2000; Scott 1991). This argument is also inconsistent with archaeological and historical models that are revealing in great detail how various aspects of African lifeways persisted in the Americas in spite of slavery and in response to regionally-specific labor needs to produce “Afro-Atlantic” or “Atlantic African” cultural zones (Ogundiran and Falola 2007; Yelvington 2006; Lovejoy 1997).

Critical perspectives on human biology and culture can serve to “rehistoricize” early diasporic peoples by providing interpretive alternatives that incorporate a wider range of complex social and individual experiences (Harrison 2006). With respect to the perception of CMT as defects, one might envision different scenarios in which this logic encouraged some individuals to modify teeth. One such case might involve the chipping, filing or removal of teeth in order to reduce their or their loved ones’ market value in an effort to prevent being traded. Depending on various factors such as how strongly a particular slaveowner felt about CMT or the nature of the affected individual’s typical work regime (e.g., domestic versus industrial, which might influence the degree of freedom individuals enjoyed to engage in body modification), the risk of punishment may have been present, but possibly outweighed by the importance of preserving tradition and family. At the other extreme, it is at least as easy to envision the perpetuation of dental modification as “underground culture,” in which patterns were more subtle, perhaps recognizable only to the initiated, and social significance of CMT now reflected new, diasporic realities of American ethnogenesis and racial formation. Unfortunately, few, if any, interpretations of dental modification offer or entertain rationales for its continuance in diasporic settings. Such considerations are important for understanding dental modification as a biocultural “transformation” (Blakey 2009).
From a broader, critical vantage point, the observation of dental modification in 18th-century New York, while unique, is less surprising than one might think. Indeed, it is perhaps more remarkable to note that all other bioarchaeological instances of CMT are attributed to natal Africans. This new perspective is a major shift in African diaspora “biohistoriography”; a shift best facilitated through political ecological analysis of social and economic forces that constrain but do not completely determine cultural options and choices (Blakey 2001; Goodman and Leatherman 1998b; Leatherman and Thomas 2001). Specifically, a political ecological framework might prove useful for assessing why the first and only skeletal case of diasporic dental modification to date occurred in New York. Was New York (or the colonial North in general) an environment that uniquely allowed for the perpetuation of cultural dental modification or should one expect to find similar cases elsewhere in the Americas?

Just as economic considerations of slave traders in coastal Africa limited the number of modified individuals who entered the Middle Passage, attention to the constrained choices of slaveholders and the enslaved may help to explain the specific geographic and temporal distribution of dental modification in early America. With respect to geography, it is instructive to consider the practice of “dumping” whereby a disproportionate number of enslaved Africans who were “[b]roken, enfeebled, and generally unfit for plantation labor... found their way to northern ports when no one else would purchase them” (Berlin 1998: 47). As one of the last destinations for many slave voyages, it was not uncommon for New York slaveholders to complain of the “recalcitrant nature” or low quality of the enslaved Africans who arrived in their markets, sometimes as rejects from the West Indies or the South (Berlin 1998; Medford et al. 2009). Reports of dumping do not constitute direct evidence that American-born individuals modified their teeth. Yet, the higher concentration of modified individuals in the North that would have resulted from dumping may well have produced an environment of relative
familiarity of dental modification that made it more attractive as a cultural option for the enslaved and possibly more tolerable for slaveholders.

One important point when considering whether dental modification persisted in New York is the proportion of individuals who arrived directly from Africa versus those who first landed in the Caribbean or the South and then re-embarked to the city. For example, the restricted importation of “unseasoned,” “salt water Africans” following the rebellion of 1712 or the “great conspiracy” to burn down the city in 1741 would have limited, to an extent, their cultural influence in diasporic communities, including, presumably, any influence they wielded in perpetuating a practice like dental modification. Similarly, increased surveillance following rebellions – e.g., bans on the number of individuals who could gather for funerals or other social events – would have limited opportunities to modify teeth. As indicated above, however, it is unlikely that such restrictions would have resulted in the disappearance of even so brazen a form of cultural expression as dental modification. It is quite possible that, under such circumstances, CMT came to represent a form of resistance more attractive to enslaved individuals than before.

Finally, this runaway advertisement from a Tennessee newspaper underscores the need to excavate the potentially multivalent meanings of CMT carefully (Perry and Paynter 1999):

“One Kentucky master described a runaway in 1815 as having ‘a black streak on his nose, which is very plain, it extends on his left cheek near the size of one little finger.’ ‘I filed several notches between several of his upper fore teeth, which I expect is also very plain,’...’ (Nashville Whig, 8, 15; cited in Franklin and Schweninger 1999: 217)

In most cases, dental modification indicates African natality. Clearly, however, dental modification continued in American settings, and in ways more diverse than often realized. This slaveowners’ use of incisal notching is a strong reminder of just how complex the biocultural record – the experiences and histories embedded in bones and teeth – can be. So, too, is the observation that just over 20% of modified NYABG adults apparently were American-born. This
unique finding warrants further investigation. Isotopic elemental ratios (Sr, C and O) should help to affirm or clarify their geographic origins as well as those of a small number of individuals whose “borderline” natal estimations are based on enamel-lead concentrations just above or below the 1 μg g⁻¹ g environmental exposure threshold. For example, Burial 101’s ⁸⁷Sr/⁸⁶Sr isotopic ratio falls within the “Manhattan” range of values while that of B-47 suggests possible Caribbean origins. Isotopic analysis might also help to clarify whether Burial 9 migrated at some point from a lead-enriched to a low-lead environment as might be indicated by comparison of his M1 and M3 concentrations. Eventually, such information will also prove useful in narrowing down possible sources of lead exposure and their health implications for NYABG individuals, topics that I explore via enamel-lead concentrations and profiles in the following sections.

**Sources and pathways of exposure**

Pinpointing sources of skeletal lead is a difficult task. When assessing technologically exposed individuals, it is particularly challenging to distinguish actual sources from amongst numerous possible exposures associated with widespread industrial and household use. There is the possibility, also, that skeletal lead is intergenerational in origin, involving maternal transfer *in utero* or through breastfeeding. Body burden, therefore, may reflect exposures over several lifetimes – and beyond. As well, some methods need to contend with diagenesis, further confounding simple source interpretations.

Still, some researchers have attempted to identify individuals’ specific sources of lead exposure with interesting results. Following their recent study of the “Mozart cranium,” for example, Stadlbauer et al. (2007) suggest that lead-containing medicines may have contributed to the famous composer’s elevated enamel and hair concentrations. Initial analysis of hair and bone samples of Ludwig van Beethoven led researchers to conclude that lead-contaminated
wine and medicinal salves may have contributed to his death as well. These findings are plausible although some have challenged this interpretation (Eisinger 2008).

My goal is to identify the most likely sources of lead for NYABG infants and young children. Some major exposures may be inferred from contemporary environmental health research. For example, common sources of contemporary pediatric lead poisoning such as leaded paint and contaminated soil and dust certainly posed a threat for children in early New York as well. To identify other likely sources of lead exposure for NYABG individuals, I turn to the historical record. Three studies are particularly useful for this purpose. The first study is *Historical Perspectives of the African Burial Ground: New York Blacks and the Diaspora*, the history report of the African Burial Ground Project in which Medford et al. (2009) identify several possible exposures including alcohol and pewter used for tools and food storage containers. Medford and co-workers (2009) also list forms of labor through which enslaved Africans would have been lead-exposed. The second study is *Lead and Lead Poisoning in Early America* by Carey McCord (1954), a prominent industrial hygienist and amateur historian of the twentieth century. Originally published as a series of essays in the journal *Industrial Medicine and Surgery* (McCord was an editor of this journal), his studies provide one of the most comprehensive accounts of lead use in the American colonial and early national periods (circa. 1600 to 1850). It is a critical resource for gauging the full scope of the threat posed by environmental lead for in the 18th century.

Lastly, in *Brush with Death: A Social History of Lead Poisoning*, Christian Warren (2000) identifies three primary modes or social pathways of human lead exposure: universal, occupational and pediatric. As Warren (personal communication) notes, these categories are not mutually exclusive, but overlap and indeed tend to “leak at every seam.” While meant to

---

31 See chapter five for a full description of Warren’s (2000) categories of human lead exposure.
inform, they – like all attempts to capture or conceptualize fluid lived experiences – have the capacity to miss and/or mask important connections. Like all biosocial boundaries, they can be more real and meaningful at certain times than others.

Warren (2000: 5) acknowledges as much when he observes that “Occupational and pediatric lead poisoning can be studied in isolation, but referring each to the other and both to larger political, scientific, and social issues in which they were embroiled is far more informative. ...Workers, children, and workers’ children – these subjects must be integrated.”

This call for integrative and ecological analysis is duly noted and, for the purposes of this study, I emphasize that pediatric toxicity often results from both universal and occupational exposures. By definition, universal lead sources are distributed broadly (though not necessarily evenly) and will reach all segments of a population, including children. Yet, certain behaviors can place children at greater risk of exposure to universal sources such as lead-contaminated soil, as will be clear from the discussion of pica (geophagy) below. As well, epidemiological research indicates that children of industrial lead workers are at heightened risk of lead poisoning. Thus, some occupational exposures become pediatric exposures. Nonetheless, these broad and sometimes overlapping categories provide a valuable framework for tracking the distribution of anthropogenic environmental lead across specific segments of a population. As we shall see, the framework’s utility extends well beyond twentieth-century United States. Here, it proves indispensable for assessing the web of early-life exposures – and within that web, the most likely sources – for NYABG children.

**Alcohol**

Table 9.1 summarizes potential sources and modes of environmental lead exposure for NYABG individuals. Biological anthropologists will recognize alcohol and pewter as important
Table 9.1: Potential sources of lead exposure for the NYABG population.

<table>
<thead>
<tr>
<th>Universal</th>
<th>Occupational</th>
<th>Pediatric</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alcohol</td>
<td>Mining</td>
<td>Foods and beverages</td>
</tr>
<tr>
<td>Foods and beverages</td>
<td>Painting</td>
<td>Drinking water</td>
</tr>
<tr>
<td>Drinking water</td>
<td>Plumbing</td>
<td>Soil (via hand to mouth activity)</td>
</tr>
<tr>
<td>Medicines</td>
<td>Ammunition casting</td>
<td>Dust</td>
</tr>
<tr>
<td>Soil</td>
<td>Pewter or brass working</td>
<td>Lead-glazed pottery</td>
</tr>
<tr>
<td>Dust</td>
<td>Fishing (e.g., casting weights)</td>
<td>Pica?</td>
</tr>
<tr>
<td>Lead pipes</td>
<td>Glassmaking</td>
<td></td>
</tr>
<tr>
<td>Lead paint</td>
<td>Glazing</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Alcohol production</td>
<td></td>
</tr>
</tbody>
</table>

Sources: McCord (1954); Medford et al. (2009)

sources of lead for enslaved Africans owing largely to the work of Jerome Handler and colleagues (1986, 1987) who linked adulterated rum in particular to epidemic lead poisoning in Barbados. As discussed in chapter five, contamination of rum from the West Indies occurred when producers used still heads and worms made from the metal during distillation (Wittmers et al. 2002). Adulterated wine resulted from use of lead-fitted presses and storage vats lined (or tinned) with lead alloy (Mushak 2011). Medford et al. (2009) observe that pewter tools and storage vats most likely were used in New York distilleries as well. While pewter, strictly speaking, refers to an alloy in which the dominant metal, tin, is hardened through inclusion of small amounts of antimony, copper or bismuth, McCord (1954) notes that colonists often used the term in reference to “any metal shaped into useful objects.” Lead content of colonial pewter ranged widely, with some objects consisting wholly of lead while others contained none. Mushak (2011: 33) reports that “Pewter ware containing high fractions of lead – up to 50% - was

---

32 Available now are a number of social histories of rum that touch upon its role in the lead poisoning of early America including archaeologist Fred Smith’s (2005) Caribbean Rum: A Social and Economic History.
popular for about 600 years, until the early nineteenth century, because of durability and attractive appearance, and the wide variety of dining and food-related objects made of this alloy.”

Exposure via alcohol did not occur solely from unintentional contamination. Following a practice dating at least to the Roman Empire, alcohol producers in Europe and early America used lead to sweeten and retard unwanted fermentation in various alcoholic beverages (Lessler 1988). In this way, lead – referred to as *sapa* by the Romans – found its way into beer, cider, wine and various foods. In such instances, alcohol production presented an occupational hazard as those who oversaw the fermentation process were at great risk of lead exposure via inhalation of vapors.

Despite the likelihood of numerous occupational exposures, McCord (1954) concludes that lead poisoning, primarily, was a “consumer affliction.” Rum consumption was ubiquitous throughout the Americas during the 18th-century; such a part of the cultural fabric and of daily life that some laborers received liquor as payment for their services.

...[T]here were instances of black and white laborers being routinely supplied with alcohol. For instance, an invoice submitted by William Dudgale details the expenses incurred in preparation of an execution. The invoice preparer claims to have ‘Paid negro hire, cartage, hire of ladders, ropes... with liquor to carpenter and negros’.... Similarly, when the City Ferry House was under repair, John Deane submitted a bill for expenses that included the labor of at least four black men, two of whom were known as ‘Negroe Ben and Negroe Roben.’ Aside from the wages the men received, there was a charge for ‘Liquor at Sundry times for all the workmen’ (New York Municipal Archives, Unfiled Papers of the Common Council) (Medford et al. 2009: 83-84).

All rum was not created equal. Those who consumed underaged “new rum” produced from the first distillation were especially susceptible to acute lead poisoning. It is safe to assume that payments to enslaved and other poor laborers in the form of alcohol consisted primarily, if not exclusively, of such cheap, low-quality liquor. Whether inadvertent or intentional, the
introduction of lead into alcohol produced an Atlantic world plague; a universal exposure that likely posed the greatest threat for enslaved and other poor laborers.

Eventually, the various symptoms of lead poisoning associated with rum consumption drew the attention of colonial doctors. Concerned, too, were rum manufacturers, as “rumors” of their product’s detrimental health effects threatened this very lucrative market. From North Carolina, for example, came complaints that New England rum “causes people who drink it to have great pain in the belly with constipation [i.e., the “dry gripes”] and later weakness of the wrists and ankles [peripheral neuropathy]” (Fitz 1938). In response, on September 3, 1723, Massachusetts Bay Colony legislators passed *An Act for Preventing Abuses in Distilling of Rum and Other Strong Liquors, with Leaden Heads or Pipes*. This ordinance was one of the first pieces of American public health legislation.

Here, the central question is whether or not contaminated alcohol contributed to elevated enamel concentrations observed for young children? Rum consumption on the part of enslaved women is well-documented (Medford et al. 2009), as is the transfer of endogenous lead from mother to child during pregnancy and via breastfeeding (Téllez-Rojo et al. 2002). Women need not have consumed leaded alcohol while pregnant for this transfer to occur. Pregnancy and lactation are periods of accelerated remineralization involving release of skeletal lead back into the bloodstream. Once liberated, lead from alcohol and other sources deposited in bone years earlier could be incorporated into the developing fetus or breast milk. Indirectly, then, NYABG infants and children may have been subject to alcohol-derived lead.

Unfortunately, my focus on first molar lead deposition between one and three years of age does not lend itself to a more definitive answer regarding lead’s role as a “legacy pollutant.” Future studies of co-buried women and children dyads possibly related biologically may offer more insight concerning this topic. Analysis of co-buried NYABG children’s deciduous teeth
should prove especially useful and interesting. Deciduous teeth begin developing in utero and, thus, reflect more directly mother-child-environment interactions, the complexity and “mixed” chemical signals of which will present novel interpretive challenges (e.g., Dolphin and Goodman 2009). What is clear from my brief historical survey, however, is that early New Yorkers would have been lead-exposed in numerous ways other, and perhaps more impactful, than alcohol. Here, again, it is useful to recall McCord’s (1954) depiction of colonial-era lead poisoning as first and foremost a consumer affair – and then to ask, “What did children consume?”

**Water**

Today’s parents are not encouraged to supplement infant diets with water until at least six months of age. This was not the case during the 18th century when drinking water transported and collected via lead pipes and vessels was another important source of cumulative lead exposure from the earliest days of life (Handler et al. 1986; Mushak 2011). According to McCord (1954), the first substantial case histories of lead poisoning in early America aside from those attributed to alcohol are related to lead plumbing. Bored-out logs served as the first water mains in colonial America. Apparently, colonists did not bring sheet lead from Europe for plumbing use during the 17th century. By the late 1700s, however, references to lead pipes were common especially in the Middle Atlantic and Northeast. In most cases, these pipes did not reach the interior of the home, but were used for gutters and downspouts. The combination of lead plumbing and lead or lead-painted roofing put all who relied on runoff drinking water at risk for exposure. Often, illness was attributed to well water drawn in lead buckets or rain water “conveyed by leaden pipes, or which had fallen upon roofs covered with this metal, and afterwards been retained in vessels” (Orfila 1818, cited in McCord 1954), as in this case of acute lead poisoning from 18th-century West Indies.
... [A] Gentleman who possessed many slaves, built a spacious house which was covered with shingles... [and] painted with red lead. The rain that fell upon this roof, was conveyed by pipes into an open cistern of Lead for the use of the family; the individuals of which had been peculiarly incident to violent, and sometimes fatal colics (Percival 1774, cited in McCord 1954: 31). In 1786, Benjamin Franklin penned a letter now famous among lead epidemiologists.

Writing to British diplomat and friend, Benjamin Vaughan, Franklin expressed his concerns about lead’s “baneful” qualities and the problem of contaminated drinking water.

In America I have often observ'd that on the Roofs of our shingled Houses, where Moss is apt to grow in northern Exposures, if there be anything on the Roof painted with white Lead, such as Balusters, or Frames of dormant Windows, etc., there is constantly a streak on the Shingles from such Paint down to the Eaves, on which no Moss will grow, but the wood remains, constantly clean and free from it. We seldom drink Rain Water that falls on our Houses; and if we did perhaps the small quantity of Lead descending from such Paint might, not be sufficient to produce any sensible ill Effects on our Bodies. But I have, been told of a case in Europe, I forgot the Place, where a whole Family was affli...cted with what we call Dry Bellyach, or Colica Pictonum, by drinking Rain Water. It was at a Country-Seat, which being situated too high to have the Advantage of a Well, was supply'd with Water from a Tank, which received the Water from the leaded Roofs. This had been drunk several Years without Mischief; but some young Trees planted near the House growing up above the Roof, and shedding their Leaves upon it, it was suppos'd that an Acid in those Leaves had corroded the Lead they cover'd and furnished the Water of that with its baneful Particles and Qualities (Franklin 1981[1786]: 274).

Thus, awareness of the health problems posed by corroded lead water delivery systems dates at least to the late 18th century.

In 1994, the U.S. Environmental Protection Agency (EPA) estimated that up to 20% of total childhood lead exposure in the United States may result from drinking water (Miranda et al. 2007). Still, researchers typically investigate water-Pb levels as a secondary exposure, i.e., when other major exposures such as paint, soil or dust cannot be confirmed (Brown and Margolis 2012). Recent controversies over substitution of the disinfectant chloramine for chlorine in Washington, DC and Greenville, NC have brought a new focus to this issue, however (Brown and Margolis 2012; CDC 2005; Maas et al. 2005). In both cities, the altered water chemistry resulting from the shift to chloramine is associated with elevated residential tap
water levels, particularly in areas with the highest concentrations of lead service pipes. Increased attention to water-Pb as a major source of historical and contemporary lead toxicity appears warranted.

Foods and other beverages

Water and alcohol were not the only dietary sources of lead. Colonial methods and instruments of food preparation, storage and consumption provided many other opportunities for lead contamination; once again, a mix of intentional and inadvertent exposures. Lead was an important ingredient used to enhance the presentation and taste of certain foods. For example, bakers used white lead to sweeten and whiten bread while both white and red lead salts were red pepper additives (Lessler 1988). Grape juice cooked down in lead and lead-lined pots was a commonly used sweetening agent (Mushak 2011). In an unfortunate tradeoff, lead helped to prevent the unsavory taste that resulted when other metals such as copper leached into foods, unknowingly at the potential cost of lead poisoning. Acidic liquids and foods such as vinegar, applesauce and tomatoes were especially prone to lead contamination via leaching from cooking and storage vessels (McCord 1954). Molasses produced by boiling sugar cane in lead-containing kettles was another significant source of lead exposure for individuals of all ages (Handler and Lange 1978; Medford et al. 2009), and one to which young children conceivably were quite susceptible.

Dietary lead was a universal problem with class-stratified dimensions. Both wealth and poverty buffered individuals from certain exposures while increasing the likelihood of others. For example, use of metal kitchenware and utensils was uncommon. Access even to low-grade, high-lead content items was limited primarily to people of high social class position. Impoverished, most early Americans used wooden utensils if any at all. According to McCord
(1954: 17), “Utensils of no type were plentiful.” As a result, risk of culinary exposure from lead or lead-containing tankards, pitchers, utensils, etc. generally increased with social status (Aufderheide et al. 1988). While the wealthiest families could afford sterling silver or the highest quality (lead-free) pewter utensils, even they could scarcely avoid consuming food somehow tainted during its preparation and/or storage.

Not all impoverished people were safe from lead-based utensils. Aufderheide et al. (1981, 1988) observe that enslaved and other domestic laborers would have had regular access to foodstuffs in the homes where they labored. From the perspective of early-life lead exposure, it also is important to note that so, too, would infants and young children who accompanied domestic laborers during their daily routines. Still, for most, access to pewter reflected high social standing and wealth and resulted in a social gradient of dietary lead exposure, with increasing consumption of toxic foods and beverages with social class.

Poverty also meant unique exposure opportunities for enslaved individuals. Throughout the Americas enslaved people prepared, stored and served foodstuffs using lead-glazed ceramics variously referred to as “Colono-Ware,” “Yabbas” and other regional names (Hauser and DeCorse 2003). These poorly fired coarse earthenware vessels were important sources of lead, the solubility of which increased when combined with certain colorants and fluxing agents such as copper oxide (Handler et al. 1986; McCord 1954). As with other lead-based food preparation and storage vessels, acidic foods, sauces and beverages can leach substantial amounts of lead from glazed surfaces. Anthropological, epidemiological and chemical studies of contemporary populations confirm the transfer of bioavailable lead from glazed ceramics into foods at levels capable of inducing clinical lead poisoning (Hailey 1994; Tunstall and Amarasiriwardena 2002; Perez et al. 2010). Fumes and soil contaminated through localized production of lead-glazed ceramics would have contributed to elevated lead levels as well. Thus,
social class position influenced the source and extent of culinary lead intake in important, if not always predictable, ways.

**Dust and soil**

The last major source of lead considered in this chapter is that of dust and soil. NYABG children would have been exposed to leaded dust and soil through some of the same pathways available to children today including housing contaminated by leaded paint. Additionally, some children quite likely were exposed occupationally through time spent in high-lead work environments. “Take-home” lead brought home on the bodies and clothing of lead workers may have been a significant source of lead for others. Infants and children who ingested leaded dust and soil were at high risk of lead poisoning.

Most contemporary cases of pediatric lead poisoning in the United States result from contaminated dust and soil (American Academy of Pediatrics 2005). The primary source of the contamination is flaking or deteriorating lead-based paint in or near homes, often found in paint chips and in dust on window sills, walls and floors as well as painted toys and other objects readily available to children. Although banned from use on interior surfaces since the 1970s, leaded paint still stalks many children, mainly those who live in old structures built prior to 1940. Dermal transfer and inhalation of lead particles can occur, but ingestion is the primary concern. Thus exposed, children may absorb nearly 50% of ingested lead from the intestinal tract (Berney 1996).

For children, paints containing the sweet-tasting pigment white lead, or lead carbonate, constitute a special hazard – and an old one. It was not until the early 19th century that white lead production became profitable in the United States, the result of exclusionary trading practices associated with the War of 1812. However, earlier generations of Americans found the
environmental toxin irresistible and widespread use of this British import preceded the war. Warren (2000: 45-46) notes that, “[f]rom colonial days, Americans demanded white lead for paints, enamel, cosmetics, and medicines, and its manufacture, a process of ‘corroding’ metallic lead in acidic vapors, was one of the first chemical industries in the United States.” Then, as now, the intense and indiscriminate mouthing behavior characteristic of infants and young children during the first two to three years of life rendered them uniquely vulnerable to this sweet, versatile poison. Possibly, enslaved children in the North may have been exposed to leaded paint in domestic settings in greater amounts than their southern counterparts. On southern plantations, enslaved individuals typically lived some distance from the main house, often in unpainted cabins. In northern urban settings, however, it was more common for enslaved women and children to reside in separate quarters within a slaveowner’s home.

Quite possibly, NYABG individuals were exposed to lead through pica, “the craving and purposive consumption of items that the consumer does not consider to be food for more than a month” (Young 2011: 3-4). Frequently, studies of historical and contemporary populations identify pregnant women and children as most likely to engage in pica behavior. For children, it is the “purposive” nature of pica that distinguishes it from infant mouthing behavior. The intensity of infant mouthing decreases drastically beginning at about age one such that intentional discovery and consumption of pica substances may be attributed to a normally developing child after age two (Young 2011). Although traditionally pathologized in the medical literature, pica’s broad geographic range and some recent research suggests its current classification as an eating disorder may be problematic.

---

33 Fessler and Abrams (2004) hypothesize that avid infant mouthing evolved as a mechanism for calibrating the immune system to the local disease ecology.
34 Interestingly, a recent study by Golden et al. (2012) found pica to be more common amongst Malagsay men but the authors note that taboos against speaking about pregnancy may have led to underreporting on the part of pregnant women.
The term *pica* covers the consumption of numerous substances and other terms are necessary to indicate the specific item being consumed. The primary focus here is on geophagy, the ingestion of earth (soil, dirt and clay).\(^{35}\) Geophagy appears in primary accounts of slavery in the Caribbean and the American South but most likely was not limited to these regions (Handler 2009, Higman 1995; Kiple 2002 [1984]). Medford et al. (2009) speculate that enslaved New Yorkers with hookworm infection, which often leads to iron-deficiency anemia, may have resorted to consuming earth.

Actual physiological mechanisms and functional consequences of pica remain something of a puzzle. Since at least the 18\(^{th}\) century, medical explanations for pica have focused on its possible relationship to poor nutrition, with colonial doctors hypothesizing a causal role for hunger. Current research explores the more specific question of whether pica behavior is a response to micronutrient (e.g., iron or zinc) deficiencies. From her thorough review of the scientific literature, Young (2011) concludes that hunger does not cause pica and that tests of the micronutrient deficiency hypothesis thus far have yielded mixed results, with dietary supplementation only inconsistently leads to cessation of the practice. Pica may actually lead to mineral deficiencies as certain substances have been shown to inhibit absorption of nutrients from the gut into the bloodstream. Sometimes prescribed for religious or medicinal purposes, other geophagic substances exhibit detoxifying properties.

Whatever their sources, as Young (2011) notes, geophagic and other pica cravings are not indiscriminate. Rather, the types of substances deemed suitable for consumption are culturally defined and vary according to age and status. For example, according to Kim and Nelson (2012), children who engage in geophagy are more likely than pregnant women to eat

\(^{35}\) Other common pica substances include ice, starch, chalk, glue, flour, ash, paper and hair. Easily produced, a longer list would reveal even greater diversity with respect to smells, textures and tastes of items selected for consumption.
topsoil, the easily accessible surface layer of earth containing the highest concentrations of lead, arsenic and other metals. Meanwhile, some studies of pregnant women with elevated blood-lead levels have revealed lead-glazed pottery consumed during pregnancy to be the probable source of exposure for the women as well as their newborns (e.g., Hamilton et al. 2001; Klitzman et al. 2002).

Although geophagy is a documented mechanism of toxic chemical exposure in children, bioarchaeologists have yet to explore the likely role it played in early-life lead intake and lead poisoning for enslaved individuals. Given the contaminated soil certainly produced from increasing levels of industrial activity during 18th-century (discussed below), geophagy may help to explain why mean subadult enamel-lead concentrations exceed significantly those of adults. Most intriguingly, this form of pica may also account, in part, for the higher mean concentrations observed after age two in subadults. By this time, infant mouthing and breastfeeding (as a means for transferring maternal lead) played minor roles, if any, in exposing children to lead. The post-two-year-old increase is rather dramatic for several individuals such as Burial 126 (see chapter 8, Figure 8.6f).

Labor was another way that people were exposed, and exposed others, to lead and lead-contaminated dust and soil. Table 9.2 lists occupations of household heads for New York City households with black residents in 1703 and indicates specific routes of lead exposure. Some of the occupations listed have long been linked to industrial lead poisoning, e.g., painters, printers and glaziers (Franklin 1981[1786]). Others garnering less attention from epidemiologists

---

36 The impact of labor is an important dimension of a developing social bioarchaeological focus on childhood. Some bioarchaeologists emphasize the need to differentiate childhood as a phase of the human life span marked by biological immaturity and accelerated growth and developed from the social designation “child,” with its culturally defined parameters and meanings (e.g., Halcrow and Tayles 2011). This focus on unique challenges faced by children in the past mirrors the emergence of “children’s health” as an area of study in public health. Here, the distinction between chronological and social age categories draws attention to the prospect of lead exposure as a consequence of child labor.
Table 9.2: Distribution of blacks in New York City households in 1703, by occupation of household head. Reproduced from Medford (2009: 56)

<table>
<thead>
<tr>
<th>Occupation</th>
<th>Number of Households</th>
<th>Black Males (over 16)</th>
<th>Black Females (over 16)</th>
<th>Black Male Children</th>
<th>Black Female Children</th>
<th>Total Blacks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Merchant</td>
<td>50</td>
<td>49</td>
<td>57</td>
<td>19</td>
<td>20</td>
<td>145</td>
</tr>
<tr>
<td><strong>Ship’s master</strong></td>
<td>17</td>
<td>11</td>
<td>17</td>
<td>7</td>
<td>5</td>
<td>40</td>
</tr>
<tr>
<td>Bolter</td>
<td>7</td>
<td>14</td>
<td>5</td>
<td>3</td>
<td>0</td>
<td>22</td>
</tr>
<tr>
<td>Brewer</td>
<td>3</td>
<td>8</td>
<td>4</td>
<td>5</td>
<td>1</td>
<td>18</td>
</tr>
<tr>
<td>Gentleman</td>
<td>8</td>
<td>6</td>
<td>10</td>
<td>1</td>
<td>0</td>
<td>17</td>
</tr>
<tr>
<td>Cordwainer</td>
<td>10</td>
<td>7</td>
<td>5</td>
<td>1</td>
<td>2</td>
<td>15</td>
</tr>
<tr>
<td>Victualler</td>
<td>6</td>
<td>4</td>
<td>4</td>
<td>3</td>
<td>4</td>
<td>15</td>
</tr>
<tr>
<td>Baker</td>
<td>10</td>
<td>6</td>
<td>4</td>
<td>3</td>
<td>1</td>
<td>14</td>
</tr>
<tr>
<td>Cooper</td>
<td>7</td>
<td>7</td>
<td>4</td>
<td>1</td>
<td>0</td>
<td>12</td>
</tr>
<tr>
<td><strong>Mariner</strong></td>
<td>7</td>
<td>4</td>
<td>5</td>
<td>1</td>
<td>1</td>
<td>11</td>
</tr>
<tr>
<td>Carpenter</td>
<td>3</td>
<td>4</td>
<td>3</td>
<td>2</td>
<td>0</td>
<td>9</td>
</tr>
<tr>
<td>Bricklayer</td>
<td>3</td>
<td>3</td>
<td>3</td>
<td>3</td>
<td>0</td>
<td>9</td>
</tr>
<tr>
<td>Attorney</td>
<td>3</td>
<td>1</td>
<td>4</td>
<td>1</td>
<td>2</td>
<td>8</td>
</tr>
<tr>
<td><strong>Blacksmith</strong></td>
<td>3</td>
<td>2</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>6</td>
</tr>
<tr>
<td>Goldsmith</td>
<td>2</td>
<td>0</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td>Sailmaker</td>
<td>2</td>
<td>1</td>
<td>3</td>
<td>1</td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td>Painter</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td><strong>Shipwright</strong></td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>Chirurgeon</td>
<td>3</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Blockmaker</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Printer</td>
<td>1</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Shopkeeper</td>
<td>1</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Butcher</td>
<td>2</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Yeoman</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td><strong>Boatman</strong></td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Weaver</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Tailor</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Barber</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td><strong>Glazier</strong></td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Silversmith</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>159</td>
<td>134</td>
<td>143</td>
<td>58</td>
<td>43</td>
<td>378</td>
</tr>
</tbody>
</table>

and historians of medicine still were capable of generating significant exposure. For example, Warren (personal communication) emphasizes the risky nature of trades associated with boating such as “Ship’s master” and “Mariner” given the many uses of lead at sea and the practice of casting molten lead below-decks. The range of potentially lead-based work that
enslaved New Yorkers performed was much broader than can be inferred from the occupations of heads of households since “... transience was the norm; even workers with particular skills changed employment seasonally [and] summer’s painter might be winter’s longshoreman. ...

Even if the census listed an individual as a ‘fisherman,’ he may have spent his winters working in a print shop” (Warren, personal communication). Or, he might have labored in a lead mine in Dutchess County, New York where the nation’s lead mining industry began circa 1740.37

Work as a possible pathway of early-life lead exposure and intake requires some explanation. First, without question, children engaged in work that resulted in lead contact. As Rosner et al. (2005: 297) observe, “For much of American history, children worked alongside their parents – planting, harvesting, and tending crops, and eventually toiling in factories and cities.” 18th-century New York, like other industrializing urban centers, afforded workers ample opportunity for lead exposure; a risk, therefore, shared by child and adult laborers alike. Children who resided in households headed by occupationally exposed adults – such as those indicated in Table 9.2 – were in jeopardy of following those adults into trades requiring significant lead contact.

Here, the crucial question, then, is not whether but when occupational lead became a salient aspect of the childhood “leadscape.” Specifically, did labor begin early enough to influence patterns of enamel-lead variation found in M1, I1 and/or M3 teeth? Medford et al. (2009) report advertisements for the labor of enslaved children as young as age six. By this time,

37 McCord (1954) provides a brief history of the origins of the lead mining industry in the United States. The first colonial European lead operations may have occurred in 1621 near Jamestown, Virginia. These would have been minor operations. As noted, Dutchess County, New York became the site of the first commercially viable lead mine around 1740. About a quarter-century later, in 1767, Frederick Philipse leased a silver-lead mine from the British Government for development. The Philipse were a major mercantile and slaveowning family based in New York. The Revolutionary War also created demand for lead mining in New York. As ammunition stores waned, the Colonial Congress in 1777 urged more extensive development of the state’s lead deposits using forced labor of prisoners of war. By the early 19th century, the Mississippi River Valley had become the seat of American lead mining activities.
M1 and I1 crown development is complete so enamel-lead concentrations for these teeth would not reflect time spent laboring at industrial or other high-risk sites. More useful for assessing direct occupational exposure are M3 teeth, the sampled portion of enamel for which formed between 9.3 and 11.3 years of age. Of the individuals with measurable M3 enamel-lead concentrations, the outlier Burial 101 appears most likely to have spent a portion of his later childhood in a high-lead (possibly work) environment. His M3 concentration of 7.38 μg g⁻¹ is the highest measured and far exceeds the 1 μg g⁻¹ upper limit for natural or background exposure and is high relative even to most M1s and I1s. Also, his M3 lead signal is consistently high, indicating the constant or chronic nature of exposure consistent with labor in a high-lead environment (see Figure 8.7e). Occupational exposure is a highly plausible explanation for these observations.

M1 and I1 teeth may not have recorded personal experiences of occupational lead exposure, but their enamel-lead concentrations may reflect potentially significant secondhand exposure. Consider, for example, a recent crisis of acute lead poisoning in northwestern Nigeria and its impact on young children. In early 2010 meningitis surveillance activities conducted by Médecins Sans Frontières (MSF, or Doctors Without Borders) and local Nigerian public health officials uncovered widespread pediatric lead poisoning in the country’s rural Zamfara region. This situation necessitated intervention from a multiagency, international rapid response team who identified artisanal gold mining and ore-processing within villages as the source of lead exposure (Lo et al. 2012). Typically, gold mining does not yield lead but Zamfara is something of a “geological anomaly” (Living on Earth 2012) where lead ore is found in conjunction with

38 Hansen (2012) describes the situation in Nigeria as resulting from a perfect storm of geochemical uniqueness, inadequate occupational and environmental health awareness and protocols, and political negligence. Unfortunately, the fundamental issue of poverty and the increased vulnerability to chemical “social toxins” that it creates is not unique. Throughout the global South, millions of impoverished people resort to small-scale or artisanal mining, with all of its associated health hazards, as a source of primary or supplemental income.
gold ore close to the earth’s surface. Furthermore, millennia of weathering have resulted in partial oxidization of these lead ore deposits and conversion of lead sulfide into more bioavailable lead carbonate (Hansen 2012).

Ore-processing activity within villages exposed children to extremely high levels of lead in dust and soil. Samples from some villages measured at > 1,200 μg g⁻¹, far above the current EPA hazard standard of 400 μg g⁻¹ for areas of bare soil where children play (EPA 2001). The resulting morbidity and death was widespread and unprecedented. For example, Dooyema et al. (2012) found that, of 204 children less than five years of age, all were lead poisoned and 97% had blood-lead levels ≥ 45 μg dl⁻¹, requiring chelation therapy. Zamfara State officials banned ore-processing operations within villages in 2012 and, after months of delay, released funding for soil remediation in 2013, but not before approximately 400 children died and thousands more exhibited convulsions, brain damage and other symptoms of severe lead poisoning. Beyond rare, the crisis in Zamfara may be the worst-ever documented outbreak of clinical and fatal lead poisoning; a vivid illustration of the danger that artisanal mining presents for lead workers and their children.

With this region’s historical ties to American slavery, the current situation also raises the interesting question of whether some enslaved individuals were significantly lead-exposed prior to the Middle Passage. The topic of anthropogenic lead exposure in West Africa during this period is rarely, if ever, discussed in the bioarchaeological literature. Yet, the recent discovery of near-surface gold-lead ore deposits in Zamfara may suggest historical as well as contemporary risk of lead exposure. Falola and Heaton (2008) explain that Zamfara and other Hausa states relied upon free and enslaved labor to produce grain, livestock, leather goods and other commodities including gold. Also economically important was the trade in enslaved people themselves. In the early 18th century, these Hausa states shifted the focus of their commercial
relations from the trans-Saharan routes leading to North Africa toward increasingly more lucrative coastal West African and, ultimately, American markets. Additional historical research is necessary to gauge the nature and extent of gold mining and ore-processing in this region; activities that could have resulted in contaminated soil and households, thereby affecting children. Other possible sources of lead in western Africa during this time include European trade goods like rum and guns requiring lead bullets or shot. However, circulating primarily amongst elites, these high-status items would not have had a major impact on West African disease ecology as seen, for example, with rum throughout the Americas, and related pediatric exposure most likely was limited.

My goal in this section has been to convey the breadth of the sources of lead available to NYABG individuals while highlighting those sources from which early-life exposure was most likely. Early studies of colonial-era lead poisoning involved measurement of bone-lead. While identifying other possibilities, these studies tended to focus on alcohol as a primary source of lead; understandably, given that bone-lead concentration reflects lifetime exposure and rum clearly played an important role in producing an early American “dry gripes” epidemic. However, there were other, more likely sources of early-life exposure during the colonial and early national periods including foods, water and other beverages contaminated by lead and pewter cooking and storage vessels. Importantly, historical and epidemiological studies suggest that contemporary sources of pediatric lead exposure such as leaded paint and soil also would have impacted children in early New York in a major way. So, too, would have lesser known pathways like pica and “take-home” occupational lead. In the last section of this chapter, I explore the question of what varied levels of lead exposure via these different sources meant for the health of NYABG infants and young children.
Health consequences

As explained in chapter five, lead adversely affects numerous systems in the human body. The effects of lead are dose-dependent and determined by its source and chemical form as well as the age, diet and physiological and nutritional status of the exposed individual. Symptoms of lead poisoning in humans range from impaired nerve conduction to fatal encephalopathy and, in young children, lead targets the still-developing central nervous system (Needleman 2004). Here, I compare results of this study to another recent analysis of enamel-lead amongst enslaved Africans: Schroeder et al.’s (2013) study of the Newton Plantation in Barbados in which the authors attempt to correlate early life lead exposure with clinical symptoms of lead poisoning. I consider what specific health effects can be inferred from the range of enamel-lead variation observed in early-forming M1/I1 teeth.

Using digestion ICP-MS, Schroeder et al. (2013) measured lead in dental enamel from 26 enslaved Africans buried at the Newton Plantation. Their analysis included an early forming tooth from each individual; M1s and one deciduous molar for a single individual for whom an M1 was not present. The authors report a mean enamel-lead concentration of 11 µg g⁻¹ and a range of 0.2 to 47.3 µg g⁻¹. The mean enamel-lead concentration for Newton individuals is considerably higher than that observed for African New Yorkers (2.9 µg g⁻¹; range: below LOD [<0.39] to 14.7 µg g⁻¹). Such a large difference may simply reflect higher levels of pediatric exposure in Barbados, or methodological differences in the two studies may be a factor, as discussed below.

Based on the estimated enamel-to blood-lead ratio of 10:1 proposed by Grobler et al. (2000) and the scale of severity of lead poisoning symptoms employed by Handler et al. (1986), Schroeder et al. (2013) conclude that most children in their study either were asymptomatic or would have experienced mild symptoms of lead poisoning during their earliest years. These
symptoms would have included occasional abdominal pains and headaches. Over a third of the
children, however, had concentrations above 80 µg dl\(^{-1}\) and “probably suffered more severe
symptoms such as frequent colic, seizures, paralysis, and even life-threatening coma”
(Schroeder et al. 2013: 208). While lead concentrations do not correlate with age-at-death, the
authors note with interest that two individuals with very high derived blood-lead values – 209
and 473 µg dl\(^{-1}\) – died before the age of ten.

Table 9.3 lists NYABG individuals’ M1/I1 enamel- and derived blood-lead concentrations.
According to Grobler et al.’s (2000) model, blood-lead concentrations for these individuals
ranged from 1.3 to 230 µg dl\(^{-1}\). Here, too, the majority of individuals (27, or 84 %) would have
exhibited mild symptoms of lead poisoning according to Handler et al.’s scale. Four individuals
would have shown moderate to severe symptoms such as vomiting, weakness and convulsions,
and one (Burial 138) might have experienced very severe symptoms such as paralysis or spasms
or been subject to coma.\(^{39}\) All of the individuals for whom symptoms would have been
moderate, severe or very severe were subadults. Of 32 individuals, nine would have had blood-
lead levels below the current 5 µg dl\(^{-1}\) reference level at which the CDC recognizes a child
between the ages of one and five as lead-exposed and recommends case management to
prevent future exposure (CDC 2012).

If the difference in blood-lead concentrations for enslaved Barbadians and New Yorkers
reflects genuine diversity in environmental lead levels, exposure was more variable and greater
for young children in Barbados than in colonial/early national New York. However, this is
difficult to discern without knowing whether Schroeder et al.’s (2013) sampling strategy entailed
analysis of core enamel exclusively, as did this study (see chapter six), or allowed for analysis of

\(^{39}\) Handler (1996) suggests that lead-induced convulsions or spasms might have led to
differential treatment in life, including accusations of witchcraft, and in death through mortuary
treatment.
Table 9.3: Enamel-lead and blood-lead derived concentrations by individual/tooth (following Grobler et al. [2000]).

<table>
<thead>
<tr>
<th>Burial</th>
<th>a Sample</th>
<th>b Cohort</th>
<th>Enamel (μg g⁻¹)</th>
<th>c Estimated Blood (μg g⁻¹)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subadults</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>LRM1</td>
<td>SA</td>
<td>2.86</td>
<td>28.6</td>
</tr>
<tr>
<td>22</td>
<td>LRM1</td>
<td>SA</td>
<td>4.66</td>
<td>46.6</td>
</tr>
<tr>
<td>35</td>
<td>ULM1</td>
<td>SA</td>
<td>10.5</td>
<td>105</td>
</tr>
<tr>
<td>39</td>
<td>LRM1</td>
<td>SA</td>
<td>6.09</td>
<td>60.9</td>
</tr>
<tr>
<td>43</td>
<td>LRM1</td>
<td>SA</td>
<td>2.41</td>
<td>24.1</td>
</tr>
<tr>
<td>126</td>
<td>LLM1</td>
<td>SA</td>
<td>11.6</td>
<td>116</td>
</tr>
<tr>
<td>138</td>
<td>URM1</td>
<td>SA</td>
<td>2.30</td>
<td>230</td>
</tr>
<tr>
<td>180</td>
<td>ULM1</td>
<td>SA</td>
<td>1.20</td>
<td>120</td>
</tr>
<tr>
<td>219</td>
<td>LRM1</td>
<td>SA</td>
<td>14.7</td>
<td>147</td>
</tr>
<tr>
<td>244</td>
<td>LLM1</td>
<td>SA</td>
<td>4.35</td>
<td>43.5</td>
</tr>
<tr>
<td>405</td>
<td>URM1</td>
<td>SA</td>
<td>3.98</td>
<td>39.8</td>
</tr>
<tr>
<td>Modified adults</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>LLM1</td>
<td>MA</td>
<td>&lt;0.39</td>
<td>(3.9)</td>
</tr>
<tr>
<td>6</td>
<td>ULM1</td>
<td>MA</td>
<td>&lt;0.39</td>
<td>(3.9)</td>
</tr>
<tr>
<td>9</td>
<td>LLM1</td>
<td>MA</td>
<td>1.34</td>
<td>13.4</td>
</tr>
<tr>
<td>9</td>
<td>LRM3</td>
<td>MA</td>
<td>0.69</td>
<td>6.9</td>
</tr>
<tr>
<td>23</td>
<td>URM1</td>
<td>MA</td>
<td>&lt;0.39</td>
<td>(3.9)</td>
</tr>
<tr>
<td>47</td>
<td>LLM1</td>
<td>MA</td>
<td>1.31</td>
<td>13.1</td>
</tr>
<tr>
<td>68</td>
<td>LRM3</td>
<td>MA</td>
<td>&lt;0.39</td>
<td>(3.9)</td>
</tr>
<tr>
<td>101</td>
<td>LRI1</td>
<td>MA</td>
<td>7.80</td>
<td>78</td>
</tr>
<tr>
<td>101</td>
<td>LLM3</td>
<td>MA</td>
<td>7.38</td>
<td>73.8</td>
</tr>
<tr>
<td>106</td>
<td>LRM1</td>
<td>MA</td>
<td>0.93</td>
<td>09.3</td>
</tr>
<tr>
<td>106</td>
<td>LRM3</td>
<td>MA</td>
<td>&lt;0.39</td>
<td>(3.9)</td>
</tr>
<tr>
<td>115</td>
<td>LLM1</td>
<td>MA</td>
<td>&lt;0.39</td>
<td>(3.9)</td>
</tr>
<tr>
<td>115</td>
<td>LRM3</td>
<td>MA</td>
<td>&lt;0.39</td>
<td>(3.9)</td>
</tr>
<tr>
<td>151</td>
<td>LRM1</td>
<td>MA</td>
<td>&lt;0.39</td>
<td>(3.9)</td>
</tr>
<tr>
<td>165</td>
<td>LLM1</td>
<td>MA</td>
<td>&lt;0.39</td>
<td>(3.9)</td>
</tr>
<tr>
<td>165</td>
<td>LRM3</td>
<td>MA</td>
<td>&lt;0.39</td>
<td>(3.9)</td>
</tr>
<tr>
<td>241</td>
<td>URM1</td>
<td>MA</td>
<td>&lt;0.39</td>
<td>(3.9)</td>
</tr>
<tr>
<td>243</td>
<td>ULI1</td>
<td>MA</td>
<td>0.89</td>
<td>8.9</td>
</tr>
<tr>
<td>243</td>
<td>URM3</td>
<td>MA</td>
<td>&lt;0.39</td>
<td>(3.9)</td>
</tr>
<tr>
<td>266</td>
<td>URM1</td>
<td>MA</td>
<td>0.56</td>
<td>5.6</td>
</tr>
<tr>
<td>266</td>
<td>URM3</td>
<td>MA</td>
<td>&lt;0.39</td>
<td>(3.9)</td>
</tr>
<tr>
<td>270</td>
<td>LLM1</td>
<td>MA</td>
<td>&lt;0.39</td>
<td>(3.9)</td>
</tr>
<tr>
<td>270</td>
<td>LLM3</td>
<td>MA</td>
<td>&lt;0.39</td>
<td>(3.9)</td>
</tr>
<tr>
<td>366</td>
<td>LLM1</td>
<td>MA</td>
<td>&lt;0.39</td>
<td>(3.9)</td>
</tr>
<tr>
<td>367</td>
<td>ULM1</td>
<td>MA</td>
<td>0.76</td>
<td>7.6</td>
</tr>
<tr>
<td>377</td>
<td>ULM1</td>
<td>MA</td>
<td>2.82</td>
<td>28.2</td>
</tr>
<tr>
<td>384</td>
<td>ULM1</td>
<td>MA</td>
<td>&lt;0.39</td>
<td>(3.9)</td>
</tr>
<tr>
<td>397</td>
<td>ULM1</td>
<td>MA</td>
<td>&lt;0.39</td>
<td>(3.9)</td>
</tr>
<tr>
<td>Non-modified adults</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>ULM1</td>
<td>NMA</td>
<td>&lt;0.39</td>
<td>(3.9)</td>
</tr>
<tr>
<td>25</td>
<td>URI1</td>
<td>NMA</td>
<td>0.40</td>
<td>4.0</td>
</tr>
</tbody>
</table>
Table 9.3: Enamel-lead and blood-lead derived concentrations by individual/tooth (following Grobler et al. [2000]).

<table>
<thead>
<tr>
<th>Burial</th>
<th>Sample</th>
<th>Cohort</th>
<th>Enamel (μg g⁻¹)</th>
<th>Estimated Blood (μg g⁻¹)</th>
</tr>
</thead>
<tbody>
<tr>
<td>25</td>
<td>LRM3</td>
<td>NMA</td>
<td>&lt;0.39</td>
<td>(3.9)</td>
</tr>
<tr>
<td>49</td>
<td>ULM1</td>
<td>NMA</td>
<td>1.72</td>
<td>17.2</td>
</tr>
<tr>
<td>63</td>
<td>LRM3</td>
<td>NMA</td>
<td>2.08</td>
<td>20.8</td>
</tr>
<tr>
<td>135</td>
<td>LRM3</td>
<td>NMA</td>
<td>&lt;0.39</td>
<td>(3.9)</td>
</tr>
<tr>
<td>150</td>
<td>LRM1</td>
<td>NMA</td>
<td>&lt;0.39</td>
<td>(3.9)</td>
</tr>
<tr>
<td>172</td>
<td>LLM1</td>
<td>NMA</td>
<td>&lt;0.39</td>
<td>(3.9)</td>
</tr>
<tr>
<td>172</td>
<td>LLM3</td>
<td>NMA</td>
<td>2.31</td>
<td>23.1</td>
</tr>
<tr>
<td>176</td>
<td>LRM1</td>
<td>NMA</td>
<td>&lt;0.39</td>
<td>3.9</td>
</tr>
<tr>
<td>176</td>
<td>LLM3</td>
<td>NMA</td>
<td>0.61</td>
<td>6.1</td>
</tr>
<tr>
<td>179</td>
<td>LRM1</td>
<td>NMA</td>
<td>1.60</td>
<td>16</td>
</tr>
<tr>
<td>179</td>
<td>LLM3</td>
<td>NMA</td>
<td>&lt;0.39</td>
<td>(3.9)</td>
</tr>
<tr>
<td>196</td>
<td>LRM1</td>
<td>NMA</td>
<td>&lt;0.39</td>
<td>(3.9)</td>
</tr>
<tr>
<td>262</td>
<td>LRM3</td>
<td>NMA</td>
<td>&lt;0.39</td>
<td>(3.9)</td>
</tr>
<tr>
<td>323</td>
<td>LRM1</td>
<td>NMA</td>
<td>4.35</td>
<td>43.5</td>
</tr>
<tr>
<td>323</td>
<td>LRM3</td>
<td>NMA</td>
<td>&lt;0.39</td>
<td>(3.9)</td>
</tr>
<tr>
<td>324</td>
<td>ULM1</td>
<td>NMA</td>
<td>1.39</td>
<td>13.9</td>
</tr>
<tr>
<td>324</td>
<td>LRM3</td>
<td>NMA</td>
<td>1.96</td>
<td>19.6</td>
</tr>
<tr>
<td>335</td>
<td>LLM1</td>
<td>NMA</td>
<td>0.42</td>
<td>4.2</td>
</tr>
<tr>
<td>335</td>
<td>URM3</td>
<td>NMA</td>
<td>&lt;0.39</td>
<td>(3.9)</td>
</tr>
<tr>
<td>Kasana</td>
<td>ULM1</td>
<td>control</td>
<td>&lt;0.39</td>
<td>(3.9)</td>
</tr>
</tbody>
</table>

*a* Tooth type/position key: L or U = lower or upper; L or R = left or right; M or I = molar or incisor; 1 or 3 = first or third. All teeth are from the permanent dentition (e.g., LLM1 = permanent lower left first molar).

*b* SA = subadult; MA = modified adult (i.e., w/CMT); NMA = non-modified adult (i.e., without CMT)

*c* LOD of Pb = < 0.39 μg g⁻¹

lead-enriched surface enamel. The latter scenario certainly could also explain the much higher values for some individuals buried at the Newton Plantation. Likewise, it is unclear how Grobler et al. (2000) accounted for the fluctuating nature of blood-lead. By most accounts, the relationship of enamel-lead to blood-lead is not yet sufficiently understood or quantified (Barbosa et al. 2005).⁴⁰

⁴⁰ Studies exploring the relationship between enamel- and blood-lead have yielded mixed results. Cleymaet et al. (1991) reported a significant positive correlation between lead concentrations in blood and enamel for Belgian children between the ages of 7.5 and 9.5 years.
Conclusions

While enamel-lead as a biomarker of lead exposure has increased in recent years, inferences of lead poisoning or its specific symptoms for individuals should still be viewed critically. However, the underlying patterns of enamel-lead variation, understood through emerging frameworks of vulnerability and risk (Leatherman 2005; Panter-Brick and Fuentes 2009), are extremely valuable for identifying those most likely to have encountered and suffered from lead. Analysis of NYABG teeth suggests that children and possibly males were at greatest risk of lead exposure – patterns that mirror findings from contemporary lead epidemiology (e.g., de Almeida et al. 2011). Future research combining dental chemistry with skeletal paleopathological indicators of nutritional stress and infectious disease will help to further elucidate the impact that lead had on enslaved New Yorkers within these high-risk groups and as individuals.

On the other hand, de Almeida et al. (2011), in a recent study involving Brazilian schoolchildren, observed no such correlation; a finding they attributed to the different time periods captured by enamel- and blood-lead concentrations. In general terms, enamel records “cumulative” past exposure while blood-lead primarily reflects recent exposure although the latter may also include lead released back into the bloodstream during bone remodeling (see chapter 5).
CHAPTER 10

SUMMARY AND CONCLUDING REMARKS

The African Burial Ground’s reappearance in the early 1990s sparked new interest in the early northern African presence. Historical monographs published in the wake of the cemetery’s excavation clarify and reinterpret aspects of African-American slavery, resistance and racial formations (e.g., Harris 2003; Foote 2004; Lepore 2006). In 2002 Northeast Magazine of The Hartford Courant produced a special report titled “Complicity: How Connecticut Chained Itself to Slavery.” From 2005 to 2007 the New-York Historical Society hosted two major exhibitions on slavery in New York. Slavery in New York explored the city’s role as a “capital of American slavery.” This exhibition was followed by New York Divided: Slavery and the Civil War, which highlighted northern economic ties to southern cotton, sugar and “slave power”; relationships that expanded alongside and in direct contradiction to the growing anti-slavery movement as war approached.

The burial ground’s excavation also brought political struggle over this once again sacred site. The stakes were high. What was to be the burial ground’s final disposition and meaning? What would become of the remains of over 400 children, women and men unearthed there? How would they be memorialized? Could the descendants of enslaved people demand and receive respect, in death for those who had been so profoundly disrespected in life? From this perspective, the struggle over the African Burial Ground was twofold: a protest for the dignity of enslaved Africans whose labor had built the city and for their own dignity and cultural heritage.

41 The exhibition companion volume includes a series of informative essays detailing various aspects of African-American political, cultural and economic life in New Amsterdam and early New York (Berlin and Harris 2005a). As Singleton (2010) observes, the exhibit and some of these essays would have benefited from incorporating more archaeological and skeletal interpretation from the NYABG Project.
rights as descendants. This struggle gave rise to a new model of public anthropology and a new paradigm for descendant community cooperation in the form of the NYABG Project (Purcell 2000; Blakey 2010).

My intent for this dissertation study has been to further the work of the NYABG Project by generating new biohistories of geographic ancestry and health. My specific goal was to use the biomarker enamel-lead to explore: (1) geographic origins, (2) sources and pathways of lead exposure, and (3) health and developmental consequences of exposure for NYABG individuals. Toward this end, I applied quantitative LA-ICP-MS analysis to NYABG teeth (M1s and I1s) that formed during early life. I analyzed a separate set of teeth for which enamel develops during adolescence in order to identify migration-related changes in lead concentration. Culturally-modified teeth offered another window onto the migrations and other lived experiences of NYABG individuals. In this concluding chapter I summarize the study’s important findings in relation to these goals and the broader goals of the NYABG Project and offer suggestions for future research directions.

This study’s primary focus on determining African versus American natality derives from the NYABG Project’s first research goal of identifying geographic and ethnic origins. LA-ICP-MS analysis clearly distinguished individuals and groups with varying degrees of lead burden. Lead was ubiquitous in 18th-century New York and other American regions where enslaved Africans originated or traveled before reaching the city – and certainly much more prevalent than in most parts of western Africa during this period. Thus, relatively high enamel-lead concentrations probably indicate American early-life settings. Working from this assumption, it appears that children were most vulnerable to lead and, thus, most likely to have been born in New York or elsewhere in the Americas. Mean enamel-concentrations for all of the children (n = 11) were
above the threshold for technological exposure (1 µg g⁻¹). In comparison, 28% of the adults – some bearing culturally-modified teeth (CMT) – were identified as possibly American-born.

As a measure of environmental conditions and health, enamel-lead data speak to the question of material quality of life for enslaved Africans. This was the second research area of the NYABG Project. From infancy or earlier, toxic lead entered the bodies of enslaved New Yorkers. Researchers typically emphasize the role that tainted rum played in the “dry gripes” epidemic that afflicted many throughout the colonial Americas. Yet, exposure resulted from multiple sources and pathways. Another well-documented source was food and beverages contaminated during production or through the use of lead-based pewter. Most pediatric exposures were probably similar to those observed today, primarily in economically impoverished communities. These sources would have included leaded paint, dust and soil, and water. Geophagy has not received much attention but may well have been an important source of ingested lead particularly for children and pregnant women. Occupational exposure was a foregone conclusion in print shops, dockyards, and other work settings in rapidly growing and industrializing 18th-century New York City. The modern distinction between childhood and occupational exposure pathways would have mattered less for enslaved laborers for whom apprenticeships and work often began prior to adolescence.

Also related to the issue of material quality of life is the question of whether or not NYABG individuals experienced clinical lead poisoning; a question that, unfortunately, remains unresolved. My hypothetical exploration in chapter 9 aside, it is not yet possible to infer lead poisoning or its symptoms from enamel-lead concentrations. The relationship of enamel-lead to blood-lead – the biomarker for which concentrations are associated with clinical symptoms – is extremely complicated, dynamic, and simply not well understood as yet. What can be said with some degree of confidence is that NYABG children represent a relatively high-lead cohort.
Within this study sample, children and adults with the highest enamel-lead concentrations – i.e., those born into high-lead environments – were most likely to have exhibited clinical symptoms of lead poisoning during early life. Symptoms could have ranged from anemia and lethargy to death.

One finding from this study has particular relevance for the last two NYABG Project research areas, i.e., biocultural transformations associated with the construction of African-American identities and efforts at “humanity maintenance,” or resistance to enslavement. Amongst the individuals identified as natal Americans are 4 adults with CMT. If Burials 9, 47, 101 and 377 were born in the Americas, this finding is a bioarchaeological first. As noted throughout this study, CMT are exclusively linked to African natality in skeletal research. Yet, as an independent test of natality, the enamel-lead data here seem to support the notion that some individuals, at least, attempted to keep this cultural practice alive in the Americas.

Within the limits of sample representativeness, time constraints, and various challenges associated with their engaged anthropological approach (Blakey 2009), project researchers have learned a great deal about African slavery and life in early New York. This study extends that process. Yet, much work remains to be done, and here are some possible methodological and theoretical directions for further bioarchaeometric analysis of the NYABG remains.

1. Quantitative lead analysis of an expanded M1 and I1 sample would enable us to characterize the skeletal sample and test the patterns observed in this study. Some of these patterns, like the mean difference observed between children and adults, seem quite stable. Others, such as the higher concentrations for males and the differences across temporal groups, may simply result from inadequate sample sizes.
2. Likewise, **more comparisons of M1 or I1 versus M3 and other teeth** would help to identify migrants and the age at which migration occurred. Analysis of canines and premolars that form in the developmental interim between M1s or I1s and M3s will enable a more fine-grained construction of geographic ancestries.

3. The assumption of high-level lead exposure in the Americas is reasonable. However, the higher mean enamel-lead concentration reported for enslaved Barbadians by Schroeder et al. (2013) relative to NYABG individuals serves as a reminder that **lead exposure was not uniform throughout the Americas**. Regional diversity of exposure conditions is an important consideration when reconstructing diasporic movements.

4. The opposing assumption of a total lack of **lead exposure in Africa** is also worth revisiting. For example, captive or enslaved individuals who spent their early years in coastal regions of western Africa where Europeans traded and established outposts most likely would register higher enamel-lead concentrations (and/or more acute exposures) than those from the hinterlands.

5. **Beyond lead**, other elemental experiences reside in the enamel archives of teeth. The methodology developed for this study may be used to mine them. For example, future research might also target mercury given the possibility of mercury poisoning due to artisanal silver-mercury amalgamation practices (Dula Amarasiriwardena, personal communication).

6. **Integration of independent lines or “cables” (Wylie 1993) of data** (e.g., elemental, isotopic [e.g., Pb, Sr and O], paleopathological, etc.) will enable us to check for interpretive errors or excesses in studies such as this, and develop
fuller understandings of personal and collective experiences that define and link individuals.

This study has moved the investigation of diasporic origins, migration and health to the microspatial level. With a novel approach to the old question of colonial-era lead exposure – one focused on revealing the nature, extent and timing of lead exposure in early New York – this study has also opened up new avenues of inquiry in the study of slavery. Did resistance efforts in the Americas include dental modification? How will an expanded dental sample and isotopic data further hone and enrich our understanding of enslaved African lifeways? How are migrations and other experiences reflected at the individual level? One of the study’s most exciting implications is the ability to now probe the processes and consequences of enslavement and identity formation with specific knowledge of those individuals that endured the Middle Passage. How do we use these individual biohistories to reconstruct collective experiences?

Importantly, the methodology developed for this study is not limited to the African Burial Ground or African diasporic populations, but may be applied more broadly to questions of migration and health in bioarchaeology and even to studies of contemporary health. Future research will further evolve this new analytical tool by incorporating recent and ongoing technical developments such as certified solid lead standards for calibration for probing variation at the lowest levels of human exposure. The biohistory thus recovered will intersect with and help make whole other histories, e.g., of childhood and environmental health in the early Americas. This new development in historical biomonitoring represents a small but important step towards a much richer and more refined understanding of a once forgotten people and the world they helped build.
APPENDICES
Figure A.1: Sample data and laser conditions record form.

### Sample Record Form

<table>
<thead>
<tr>
<th>Sample</th>
<th>Energy</th>
<th>Rep. Rate</th>
<th># of Shots</th>
<th>Scan Speed/Sec. Pause</th>
<th>Defocus</th>
<th>-X mJ</th>
<th>Reading # (ICP)</th>
<th>Comments/Image #</th>
</tr>
</thead>
<tbody>
<tr>
<td>C(PN)</td>
<td>10</td>
<td>10</td>
<td>20</td>
<td>0</td>
<td></td>
<td>.790</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C(PN)</td>
<td>15</td>
<td>10</td>
<td>20</td>
<td>0</td>
<td></td>
<td>.755</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C(PN)</td>
<td>20</td>
<td>10</td>
<td>20</td>
<td>0</td>
<td></td>
<td>.790</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C(PN)</td>
<td>25</td>
<td>10</td>
<td>20</td>
<td>0</td>
<td></td>
<td>.955</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C(PN)</td>
<td>30</td>
<td>10</td>
<td>20</td>
<td>0</td>
<td></td>
<td>.864</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C(PN)</td>
<td>35</td>
<td>10</td>
<td>20</td>
<td>0</td>
<td></td>
<td>.799</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C(PN)</td>
<td>40</td>
<td>10</td>
<td>20</td>
<td>0</td>
<td></td>
<td>.880</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Sample Record Form

<table>
<thead>
<tr>
<th>Sample</th>
<th>Energy</th>
<th>Rep. Rate</th>
<th># of Shots</th>
<th>Scan Speed/Sec. Pause</th>
<th>Defocus</th>
<th>-X mJ</th>
<th>Reading # (ICP)</th>
<th>Comments/Image #</th>
</tr>
</thead>
<tbody>
<tr>
<td>A(PN)</td>
<td>10</td>
<td>10</td>
<td>20</td>
<td>0</td>
<td></td>
<td>.796</td>
<td></td>
<td></td>
</tr>
<tr>
<td>A(PN)</td>
<td>15</td>
<td>10</td>
<td>20</td>
<td>0</td>
<td></td>
<td>.796</td>
<td></td>
<td></td>
</tr>
<tr>
<td>A(PN)</td>
<td>20</td>
<td>10</td>
<td>20</td>
<td>0</td>
<td></td>
<td>.796</td>
<td></td>
<td></td>
</tr>
<tr>
<td>A(PN)</td>
<td>25</td>
<td>10</td>
<td>20</td>
<td>0</td>
<td></td>
<td>.796</td>
<td></td>
<td></td>
</tr>
<tr>
<td>A(PN)</td>
<td>30</td>
<td>10</td>
<td>20</td>
<td>0</td>
<td></td>
<td>.796</td>
<td></td>
<td></td>
</tr>
<tr>
<td>A(PN)</td>
<td>35</td>
<td>10</td>
<td>20</td>
<td>0</td>
<td></td>
<td>.796</td>
<td></td>
<td></td>
</tr>
<tr>
<td>A(PN)</td>
<td>40</td>
<td>10</td>
<td>20</td>
<td>0</td>
<td></td>
<td>.796</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Sample Record Form

<table>
<thead>
<tr>
<th>Sample</th>
<th>Energy</th>
<th>Rep. Rate</th>
<th># of Shots</th>
<th>Scan Speed/Sec. Pause</th>
<th>Defocus</th>
<th>-X mJ</th>
<th>Reading # (ICP)</th>
<th>Comments/Image #</th>
</tr>
</thead>
<tbody>
<tr>
<td>A(PN)</td>
<td>10</td>
<td>10</td>
<td>20</td>
<td>0</td>
<td></td>
<td>.796</td>
<td></td>
<td></td>
</tr>
<tr>
<td>A(PN)</td>
<td>15</td>
<td>10</td>
<td>20</td>
<td>0</td>
<td></td>
<td>.796</td>
<td></td>
<td></td>
</tr>
<tr>
<td>A(PN)</td>
<td>20</td>
<td>10</td>
<td>20</td>
<td>0</td>
<td></td>
<td>.796</td>
<td></td>
<td></td>
</tr>
<tr>
<td>A(PN)</td>
<td>25</td>
<td>10</td>
<td>20</td>
<td>0</td>
<td></td>
<td>.796</td>
<td></td>
<td></td>
</tr>
<tr>
<td>A(PN)</td>
<td>30</td>
<td>10</td>
<td>20</td>
<td>0</td>
<td></td>
<td>.796</td>
<td></td>
<td></td>
</tr>
<tr>
<td>A(PN)</td>
<td>35</td>
<td>10</td>
<td>20</td>
<td>0</td>
<td></td>
<td>.796</td>
<td></td>
<td></td>
</tr>
<tr>
<td>A(PN)</td>
<td>40</td>
<td>10</td>
<td>20</td>
<td>0</td>
<td></td>
<td>.796</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Sample Record Form

<table>
<thead>
<tr>
<th>Sample</th>
<th>Energy</th>
<th>Rep. Rate</th>
<th># of Shots</th>
<th>Scan Speed/Sec. Pause</th>
<th>Defocus</th>
<th>-X mJ</th>
<th>Reading # (ICP)</th>
<th>Comments/Image #</th>
</tr>
</thead>
<tbody>
<tr>
<td>A(PN)</td>
<td>10</td>
<td>10</td>
<td>20</td>
<td>0</td>
<td></td>
<td>.796</td>
<td></td>
<td></td>
</tr>
<tr>
<td>A(PN)</td>
<td>15</td>
<td>10</td>
<td>20</td>
<td>0</td>
<td></td>
<td>.796</td>
<td></td>
<td></td>
</tr>
<tr>
<td>A(PN)</td>
<td>20</td>
<td>10</td>
<td>20</td>
<td>0</td>
<td></td>
<td>.796</td>
<td></td>
<td></td>
</tr>
<tr>
<td>A(PN)</td>
<td>25</td>
<td>10</td>
<td>20</td>
<td>0</td>
<td></td>
<td>.796</td>
<td></td>
<td></td>
</tr>
<tr>
<td>A(PN)</td>
<td>30</td>
<td>10</td>
<td>20</td>
<td>0</td>
<td></td>
<td>.796</td>
<td></td>
<td></td>
</tr>
<tr>
<td>A(PN)</td>
<td>35</td>
<td>10</td>
<td>20</td>
<td>0</td>
<td></td>
<td>.796</td>
<td></td>
<td></td>
</tr>
<tr>
<td>A(PN)</td>
<td>40</td>
<td>10</td>
<td>20</td>
<td>0</td>
<td></td>
<td>.796</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
APPENDIX B

DATA PROCESSING IN EXCEL

Figure B.1: Data processing in Excel (background subtraction and normalization steps).
Figure C.1: M1 or I1 enamel-lead microspatial distribution graphs (log scale) for NYABG subadults. Surface enamel peaks are indicated by a red arrow.
APPENDIX D

ENAMEL-LEAD MICROSpatial DISTRIBUTION GRAPHs: NYABG MODIFIED ADULTS

Figure D.1: M1 or I1 enamel-lead microspatial distribution graphs (log scale) for NYABG modified adults. Surface enamel peaks are indicated by a red arrow.
Figure E.1: M1 or I1 enamel-lead microspatial distribution graphs (log scale) for NYABG non-modified adults. Surface enamel peaks are indicated by a red arrow.
BIBLIOGRAPHY


238


Handler JS. 1996. A prone burial from a plantation slave cemetery in Barbados, West Indies: possible evidence for an African-type witch or other negatively viewed person. Historical Archaeology 76-86.


255


Purchase NG, Fergusson JE. 1986. Lead in teeth: the influence of the tooth type and the sample within a tooth on lead levels. Sci Total Environ 52:239-250.


