RECONSTRUCTING THE CHILDHOOD DIET OF AN 18TH TO 19TH CENTURY LAND-OWNING FAMILY IN BRUNSWICK COUNTY, NORTH CAROLINA

by

Corinne Taylor

August, 2020

Director of Thesis: Dr. Megan Perry

Major Department: Anthropology

Breastfeeding and weaning practices can greatly impact a child's immune system development and nutritional status, later causing long-term health effects. This research explores the relationships between weaning practices, metabolic disease, and childhood frailty in an 18th to 19th century coastal North Carolina land-owning family. Ten individuals were recovered from the 2017 and 2018 field seasons at the Gause cemetery at Seaside (GCAS), and six of the ten individuals were under the age of eight. Most of the GCAS individuals experienced non-specific physiological stress in the form of either dental enamel hypoplasias (DEH) and/or cribra orbitalia. The $\delta^{15}N$ and $\delta^{13}C$ values received from incremental dentin collagen of 13 teeth and 10 bone collagen samples were used to analyze dietary and trophic level shifts that occurred during weaning and post-weaning periods. Additionally, radiographic and histological analysis were performed on the first permanent molars of the adult from Grave 2, and two subadults from Grave 9 and Grave 10 Burial 1, to determine whether they experienced a metabolic disease during childhood. The GCAS sample had a diet consisting of C_3 plants with supplementation of marine sources or C_4 plants. The GCAS sample ceased weaning at age 2.5 years with a weaning diet that largely consisted of C_3 plants with a larger contribution of C_4 plants than adults. Stable isotopes incremental dentin values were compared to DEH formation ages. Much but not all of

the DEH coincided with the weaning period of the GCAS sample. Only the adult individual from Grave 2 experienced interglobular dentin (IGD) between the ages of 2.5 and 3 years suggesting they were vitamin D deficient. Evidence from analysis of stable isotopes and dental histology indicates that weaning age and metabolic disease did not notably increase childhood frailty in this family.

RECONSTRUCTING THE CHILDHOOD DIET OF AN 18TH TO 19TH CENTURY LAND-OWNING FAMILY IN BRUNSWICK COUNTY, NORTH CAROLINA

A Thesis

Presented To the Faculty of the Department of Anthropology

East Carolina University

In Partial Fulfillment of the Requirements for the Degree

Master of Arts, Anthropology

by

Corinne Taylor

August, 2020

Title: RECONSTRUCTING THE CHILDHOOD DIET OF AN 18TH TO 19TH CENTURY LAND-OWNING FAMILY IN BRUNSWICK COUNTY, NORTH CAROLINA

By

Corinne Taylor

APPROVED BY:
DIRECTOR OF THESIS: _____ Megan A. Perry, PhD

COMMITTEE MEMEMBER: _____ Charles Ewen, PhD

COMMITTEE MEMEMBER: _____ James Loudon, PhD

COMMITTEE MEMEMBER: _____ Robert Tykot, PhD

DEPARTMENT CHAIRPERSON: _____ I. Randolph Daniel Jr., PhD

DEAN OF THE GRADUATE SCHOOL: _____ Paul J. Gemperline, PhD

ACKNOWLEDGMENTS

I would first like to thank Mr. J.R. Robinson for providing me the opportunity to study his ancestor's remains and delve deeper into the Gause family history and daily lives of North Carolina land-owning families in 18th and 19th century. I would also like to thank Dr. Megan Perry for supporting and guiding me through each stage of my research. Furthermore, I would like to express my gratitude to Dr. Robert H. Tykot for teaching and instructing me through the process of prepping stable isotope samples, and Dr. Andrea Vianello for his help prepping some of my stable isotope samples. Special thanks also to Dr. James Loudon, Dr. Charles Ewen, and Dr. Robert H. Tykot for serving as my thesis committee members. Additionally, I am deeply grateful to Courtney Grace Olah for providing assistance in prepping teeth to be examined and analyzed for interglobular dentin. Finally, I would like to thank my friends and family for helping and supporting me along this journey and for providing endless love and support every step of the way.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

CHAPTER ONE:

INTRODUCTION

Overview

The Gause family was a prominent land owning family during the late 18th to early 19th century in Brunswick County, North Carolina. They were well-known for their participation in the Revolutionary War and their presence in early Brunswick county politics (David & Bender, 2009). Today, they are still commemorated for their part in Brunswick County's history and are connected to a few modern locales, like Gause Landing. While no known original structures associated with the Gause family exist, there are two familial burial sites in Brunswick County, the Gause Cemetery and the Gause Tomb and Cemetery. The first established burial site is the Gause Cemetery at Seaside in Sunset Beach, which seems to date between the late 18th and mid-19th century. The 1830s Gause Tomb near Ocean Isle Beach may have been a second familial burial site, but the surrounding cemetery could have been in use since the early 1800s (Landmark Preservation Associates, 2010).

In recent years, the Gause Cemetery at Seaside and Gause Tomb and Cemetery have become sites of interest for Gause family descendant J. R. Robinson. In 2015, Mr. Robinson purchased and cleared the land containing the Gause Tomb and Cemetery and refurbished the tomb (Slattery 2015). He also purchased the land containing the Gause Cemetery at Seaside in 2016, and in 2017 he contacted the Department of Anthropology at East Carolina University to conduct excavations of the graves in preparation for reburial near the Gause Tomb. An initial field season was conducted at the Gause Cemetery at Seaside (GCAS) in 2017 which excavated three graves each containing one individual and one possible grave

(Quintana, 2019). In 2018, excavation of the Gause Cemetery continued with the recovery of seven individuals from five excavated burials (Long, 2019). After each field season, researchers determined and analyzed age, sex, and ancestry, and documented pathological lesions of these individuals. From these analyses, researchers found the excavated graves largely contained children and both the adults and children had non-specific indicators of childhood physiological stress like dental enamel hypoplasias or cribra orbitalia (Long, 2019; Quintana, 2019). This current project builds upon the analyses of these ten individuals by delving deeper into the lived experience of the individuals from the GCAS site through the exploration of diet and dietary deficiencies in human bone and teeth. Additionally, research of historical documents and archaeological reports supplemented the analysis and aided in understanding the subsistence strategies of elite families during the 18th to 19th century. The analysis includes a stable isotope analysis of bone and incremental dentin collagen from each individual at the GCAS site and histological and radiographic analysis of the first molars from the individuals from Grave 2, 9, and 10 Burial 1. The purpose of this study is to determine whether breastfeeding and weaning practices, as well as metabolic disease, were factors that led to high child mortality in this population.

Intellectual merit

Currently, only a few stable isotope studies have focused on the diets of elite families in the southeastern United States (e.g. Cullen & Owsley, 2011; France et al., 2014; Owsley et al., 2018; Seeman, 2011; Trinkley & Hacker, 2015). Furthermore, even fewer have examined the breastfeeding and weaning practices and metabolic diseases encountered by elite plantation-owning families (Owsley et al., 2018; Seeman, 2011). Although this study has a relatively small

sample size (N=10), a good deal of information has been garnered on the diet and health of early 18th century to late 19th century rural elite plantation-owning families of the southeastern U.S., and can help comparative research. The information obtained from this study also contextualizes that contained in historical accounts and documents. Lastly, this study provides an example of the benefits of collaborative research between disciplines and between bioarchaeologists and the descendant community. Collaboration between disciplines can help answer broader questions, for example in this study methods and analyses from bioarchaeology, paleopathology, chemistry, and history were used to identify reasons behind the GCAS high childhood frailty. Additionally, collaboration with descendant communities can provide knowledge that cannot be found elsewhere. J.R. Robinson, a direct descendent of William Gause, Jr., was continually contacted during this study to provide and receive information on his ancestors.

Broader impacts

Stable isotope analysis provides information to help interpret the diet and, sometimes, weaning practices used by the deceased individuals. Additionally, the analysis of metabolic disease provides additional information as to whether malnutrition was experienced in their daily lives. The information from these analyses illuminates the daily lifestyle of these individuals and perhaps why children died in this family. Although the GCAS site is also not the only 18th century to 19th century site with a high child mortality in the southeastern United States, this research delves into the possible factors causing this phenomenon like diet and breastfeeding and weaning practices (Cullen & Owsley, 2011; Owsley et al., 2018; Trinkley & Hacker, 2015). Furthermore, as discussed, this research illustrates the benefits of collaboration with the descendant community. J. R. Robinson plans to have the information obtained from his ancestors

be accessible to the public and descendent community. Thus, this research will have the immediate impact of helping descendants like J.R. Robinson and the public learn more about the Gause family and daily life at that time.

CHAPTER TWO:

BACKGROUND

Historical background

Although the Gause family is well known in Brunswick County for namesake locations like the Gause cemetery and Gause Landing, only limited historical accounts of their plantation and livelihood exist. Gause Landing today refers to a quiet neighborhood facing the Intercoastal Waterway about 1000 feet from the Causeway Drive Bridge leading to Ocean Isle Beach (Figure 2.1). The Gause Cemetery at Seaside is approximately 2.5 miles to the west near the intersection of NC State Highway 904 and Seaside Drive, a few feet inland across from Tubb's Inlet (Figure 2.1). Another Gause landmark near Gause Landing is the Gause Tomb, originally built around 1838, situated in a larger cemetery near the intersection of Hale Swamp Road and NC Highway 179 (Figure 2.1). This cemetery appears to be slightly more recent than the Gause Cemetery at Seaside, as discussed further below.

During the first half of the 18th century the Gause family lived in Prince George Parish, now known as Horry County, South Carolina. William Gause, Sr. (1710?-1761) settled near Little River, SC, and was known for being an innkeeper near Gause's Inlet, known today as White Point Swash (Berry, 1982; Johnston, 2020; Quintana, 2019; Todd, n.d.). On February 6, 1751, he obtained a plantation and 400 acres at Starr Bluff on the Waccamaw River in Horry County, SC, and three slaves from Nathan Frink (Berry, 1982, 1988, 1996; Frink, 1751). It is unknown whether he lived there in his final years (Figure 2.1).

Figure 2.1: Map displaying the locations of Little River, GCAS site, Gause Landing, and Gause Tomb and Cemetery (Google Maps, 2020)

William Gause, Sr. had six sons and one daughter with Ann Bryan (1718-1812): Needham Gause (1733-1794), John Gause, Sr. (1735-1783), Susanna Gause (born 1740), Benjamin A. Gause (1741-1783?), Charles Gause (1743-1807), William Gause, Jr. (1745-1801), and Bryan Gause (died ~1804) (B.W. Gause, 1804; J. Gause, 1783; N. Gause, 1794, 1801; W. Gause, 1801; Johnston, 2020; Quintana, 2019; Smith, 2011; Todd, n.d.; U.S. Census Bureau, 1790). According to genealogists and historical documents, all of William Gause, Sr.'s children lived and owned land in Brunswick County, NC, by the 1790s (B.W. Gause, 1804; J. Gause, 1783; W. Gause, 1801; Johnston, 2020; Quintana, 2019; Todd, n.d.; U.S. Census Bureau, 1790). Additionally, based on deeds and wills, a few of William Gause, Sr.'s children, John Gause, William Gause, Jr., and Needham Gause, potentially acquired the land near Tubb's Inlet, on which the Gause Cemetery at Seaside sits. Since one of William Gause, Sr.'s children possibly owned the land containing the Gause Cemetery during the time in which the cemetery was in use, some of the individuals found at GCAS could be from either one of his children's families, and so research was done on his children's families and their deed records.

One possible owner of the land containing the GCAS while it was in use was John Gause, Sr., whose son John Julius Gause, Jr. had the Gause Tomb built. The earliest documentation of John Gause, Sr. owning land in Brunswick County is of 190 acres between Lockwood Folly and Shallotte River deeded in 1777, after he fought in the Revolutionary War (J. Gause, 1777a, 1777b). He married Susannah Frink (1746-1809) in 1760. They had four sons and two daughters: Benjamin (1762-1825), Charles (born 1764), Charlott(e) (m. Galloway) (1767-1838), Needham (1769-1816), Elizabeth (m. Christie) (1771-1850), and John Julius, Jr. (1774-1836) (Figure 2.2) (Christie Family Tree, 2020; J. Gause, 1783; J. J. Gause, 1836; Linda Family Tree, 2020). Daughters of John, Sr., Elizabeth and Charlott(e), did not receive land from their father (J.

Gause, 1783). Charlott(e) married Nathaniel Galloway (1766-1834) and stayed in Brunswick County until at least 1830, while Elizabeth moved to Georgia by 1820 (Christie Family Tree, 2020; U.S. Census Bureau, 1800, 1820, 1830). In his 1783 will, John Gause, Sr. bequeaths his "Seashore plantation", possibly one of the pieces of land acquired in 1777, to his wife Susanna, to be split between Charles and Benjamin after her death with "Randals Branch [being] the dividing line between them" (J. Gause, 1783). Benjamin moved to South Carolina in 1800, before his mother's death (Christie Family Tree, 2020; U.S. Census Bureau, 1800) and Charles' whereabouts by this time is undocumented, unless some of the Brunswick County land transactions before 1805 can be attributed to him, not his uncle Charles who died in 1808. Thus, who ended up with John Gause, Sr.'s "Seashore plantation" after Susanna's death is unclear, although at some point his son, John Julius, Jr., ended up with land near the Gause Tomb and GCAS. John Julius, Jr. originally was bequeathed a tract of land that his father had purchased from Steven Daniell near "Skippers(?) Neck", while his brother Needham received land known "by the name of the Swamp house(?)" (J. Gause, 1783).

After his father's death, John Julius, Jr. acquired more land on the north side of the Shallotte River and in the town of Shallotte (J. J. Gause, 1788, 1836). John Julius had three wives, Elizabeth Bacot Gause (his first cousin) (born 1778), Maria Theresa Bruard (1784-1814), and Emily R. Miller (1792-1881), and with them had approximately 10 children (Figure 2.2) (Berry, 1982; K. Gause, 2000). It is ambiguous as to which wife provided which children. Genealogy sources believe that either Elizabeth Bacot Gause or Maria Theresa Bruard had Ann Marie (m. Wilson) (1799-1837), Mariah J. B. (born 1800), John Peter (1802-1858), George W. (born 1802), Frederick B. "Fitz" (1803-1880), Samuel Sidney (1804-1867), and Julius L. (1809-1887) (Christie Family Tree, 2020; J. J. Gause, 1836; K. Gause, 2020; The Complete Genealogy

Reporter, 2016). Jane M. (born 1820), Margaret L. (m. Campbell) (1823-1894), and Julia E. are suspected to be Emily R. Miller's children (Christie Family Tree, 2020; J. J. Gause, 1836; K. Gause, 2020; The Complete Genealogy Reporter, 2016). Additionally, in 1805, three Gause children from an indeterminate branch of the family, Benjamin William (1789-1826), Bryan (born 1792), and Elizabeth J. "Eliza Jane" (m. Blackwell) (born 1794) were adopted by John Julius, Jr. (Brunswick County Court, 1805; B.W. Gause, 1826; J. J. Gause, 1824). Benjamin William, Elizabeth J., and Ann Marie stayed in Brunswick County, but it is unclear whether Julia E., Jane M., George W., Bryan and Mariah J. B. stayed as well (Christie Family Tree, 2020; B.W. Gause, 1826; J. J. Gause, 1824; The Complete Genealogy Reporter, 2016). The rest of John Julius's children Fredrick B., Margaret L., Samuel S., Julius L., and John Peter moved out of the state. (Christie Family Tree, 2020; The Complete Genealogy Reporter, 2016; U.S. Census Bureau, 1850; U.S. Census Bureau, 1860).

In his will, John Julius, Jr. requests for his body to be placed in a vault with his two deceased wives (presumably Elizabeth Bacot Gause and Maria Theresa Bruard) at the "old burying grounds" until a new tomb can be built at the "old family grounds" on the plantation, "late the residence of Samuel Gause, deceased" (J.J. Gause, 1836). Once the new tomb was constructed, his executors would arrange for his body, those of his wives, Mr. and Mrs. Bruard (his in-laws), and his children to be interred in the structure. Samuel, the previous owner of the land, likely does not refer to John Julius, Jr.'s son, Samuel S., but John Julius, Jr.'s cousin Samuel (discussed below), the son of William Gause, Jr. (S. Gause, 1811). The location of the "old burying grounds" containing the vault where John Julius, Jr. would be temporarily interred until the new tomb was constructed is not clear, but family lore identifies it as GCAS.

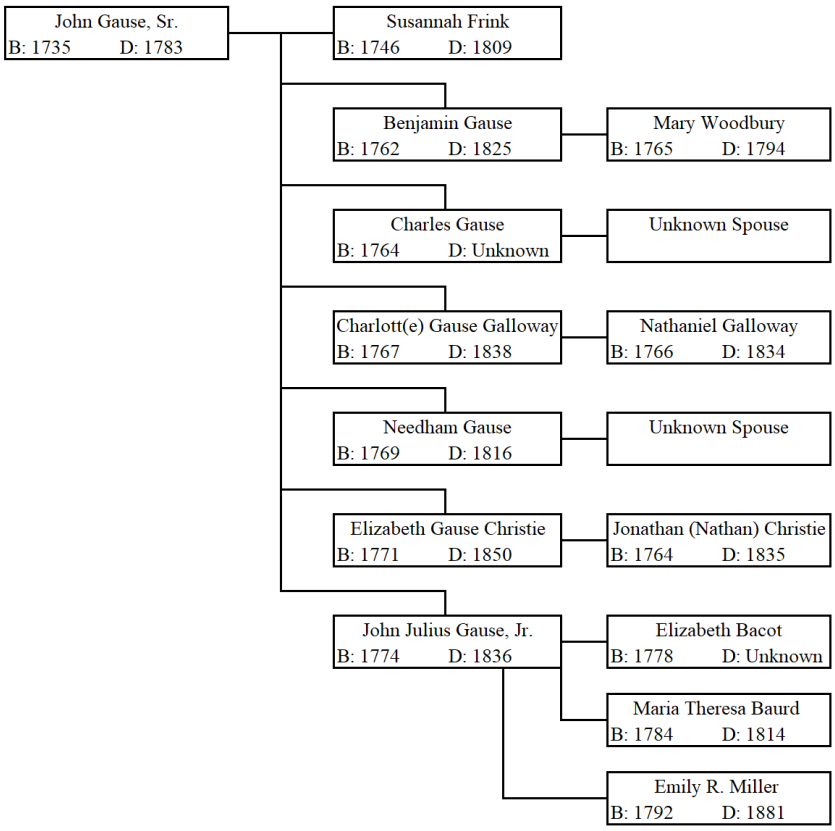

Figure 2.2: John Gause's, Sr. Family (based on information from Christie Family Tree, 2020; J. Gause, 1783; J. J. Gause, 1836; Linda Family Tree, 2020)

William Gause, Jr., the brother of John Gause, Sr., also acquired extensive landholdings along the coast and inland and could have owned the GCAS land. He owned 760 acres of land in Brunswick County, NC, on Ocean Isle Beach near Tubb's Inlet, close to the Gause Cemetery at Seaside, and the east side of the Waccamaw River adjacent to that of his brother Needham's (W. Gause, 1770a, 1770b). After the war, he obtained land in Shallotte Swamp, Lockwoods Folly, and land near Saus(c)epan Creek and Shallotte inlet, which is near Gause Landing, the suspected location of William's, Jr. Naval Store Plantation and the Gause Manor (W. Gause, 1778, 1783, 1788, 1796). He participated in the Revolutionary War alongside his brothers John, Sr., Charles, and Needham (Berry, 1982; The North Carolina Daughters of the American Revolution, 1932).

Additionally, according to family folklore, William Gause, Jr. lost his leg during the war, but no historical documents detail this loss.

William Gause, Jr. not only was a public official but also had relationships with President George Washington and Methodist Bishop Francis Asbury. On April 27, 1791, George Washington recorded in his diary his visit with the Gause family, when he sat with William, Jr. over breakfast (Berry, 1982; David & Bender 2009). Bishop Francis Asbury also frequently visited William, Jr. and discussed in his diary his visits with the Gause family, including his sorrow upon William Gause, Jr.'s death in 1801 (Berry, 1982; W. Gause, 1801). In 1777, William Gause, Jr. was appointed as Justice of the Peace of Brunswick County along with his brother Needham and in 1778 as a member of the House of Commons for Brunswick County (Daughters of the American Revolution, 2020; David & Bender 2009; Lee, 1980; The Weekly Standard, 1844). William Gause, Jr. married Mary Wingate (1750-1776) and then Elizabeth Bacot (1746-1801) and with them had seven children: Needham (1771-1792), Samuel (1773-1811), Sarah (1775-1801), Elizabeth Bacot (born 1778), (Reverend) William Bacot, Jr. (1778-1860), Martha (m. Wilson) (1781-1844), and Peter (1783-1823) (Figure 2.4) (Christie Family Tree, 2020; P. Gause, 1823; S. Gause, 1811; W. Gause, 1801; W. Jr. Gause, 1858; The Complete Genealogy Reporter, 2016; Todd, 2020; U.S. Census Bureau, 1850-1885).

Before his death, William, Jr. sold land to his son Samuel that included 210 acres of the tide marsh and an island near Tubb's Inlet, which is in the area of the GCAS (S. Gause, 1801). In his will, William, Jr. split his remaining landholdings that included Tubb's Beach between his sons, with William Bacot, Jr. receiving the western half excluding half of Tubb's Beach and the adjoining marsh (land presumably already purchased by Samuel) and Peter the eastern half along with the portion of land including half of Tubb's Beach and the adjoining marsh and 50 acres

containing the plantation house, (presumably at Gause Landing) to be first given to Elizabeth, William, Jr.'s wife, for her use until her death. Thus Samuel, William Bacot, Jr., or Peter could have ended up with the land containing the GCAS. Other land west of Lockwoods Folly River was given to his grandchildren, Benjamin William Gause and William Wilson (W. Gause, 1801). It is unclear whether William Gause's children, Martha Gause Wilson and Sarah Gause, left Brunswick County, NC, but William Bacot, Jr., Elizabeth, and Peter stayed and had families (P. Gause, 1823; W. Jr. Gause, 1858). As previously mentioned, Elizabeth married her first cousin, John Julius Gause, Jr., while Peter married Sarah Goodman and William Bacot, Jr. married Martha Frink (1780-1828), then after her death, Piercy Purefoy (1804-1860) (Figure 2.4) (Christie Family Tree, 2020). Shortly before his death in 1823, Peter requested that his wife Sarah be his executrix and oversee the distribution of his land (P. Gause, 1823). Apparently some of Peter's land ended up with his daughter, Sarah, which is mentioned in her husband William James Gause's will (W. J. Gause, 1824). The will also mentions that William James is indebted to Peter Gause's estate at the time of his death, suggesting he might have bought out some of Peter's other children's land inheritance to add to that received via Sarah.

William Bacot Gause in his 1860 will, split his landholdings from the beach (i.e., Tubb's Beach) to the main road (modern North Carolina State Highway 903) in addition to the "back lands" lying near Caw Caw Swamp five ways between his wife Piercy and his sons Olin, Emory, Lucien, and McCarroll B., with Piercy getting the parcel including the buildings at the seashore plantation, presumably at Gause Landing (W. Jr. Gause, 1858). An 1865 map shows a semi-circle of 4 buildings near what today is called Gause Landing, with a path or road leading to a building identified as belonging to "Gass" (likely Gause), which could identify lands owned by William Bacot Gause (Figure 2.3) (Blackford, 1865; Landmark Preservation Associates, 2010).

Figure 2.3: 1865 Survey Map of Brunswick County. The yellow circle contains the name "S. Frink," the green circle shows where Tubb's Inlet is, and the blue circle contains the name "Gass" (Blackford, 1865)

Samuel Gause moved to Bladen County, NC, with his wife, Margaret Councel Gause (1777-1839) and had six children: William James (1794-1824), Samuel Cyprus (born 1805), Mary (1808-1895), Harriet (born 1809), Margaret (1810-1839), and Anna (born 1812), and then, before his death (1811), moved back to Brunswick County, NC (Christie Family Tree, 2020; S. Gause, 1811; W. J. Gause, 1824). His son, William James married Sarah, the daughter of Peter Gause as noted above, and William James' 1824 will mentioned that both the land he received from his father and that his wife received from her father be divided between his wife Sarah and his daughter Mary Jane upon his death (W.J. Gause, 1824). One parcel of this land likely contained the "old family ground on the plantation late the residence of Samuel Gause, deceased" where John Julius, Jr. wished his tomb to be built, as discussed below.

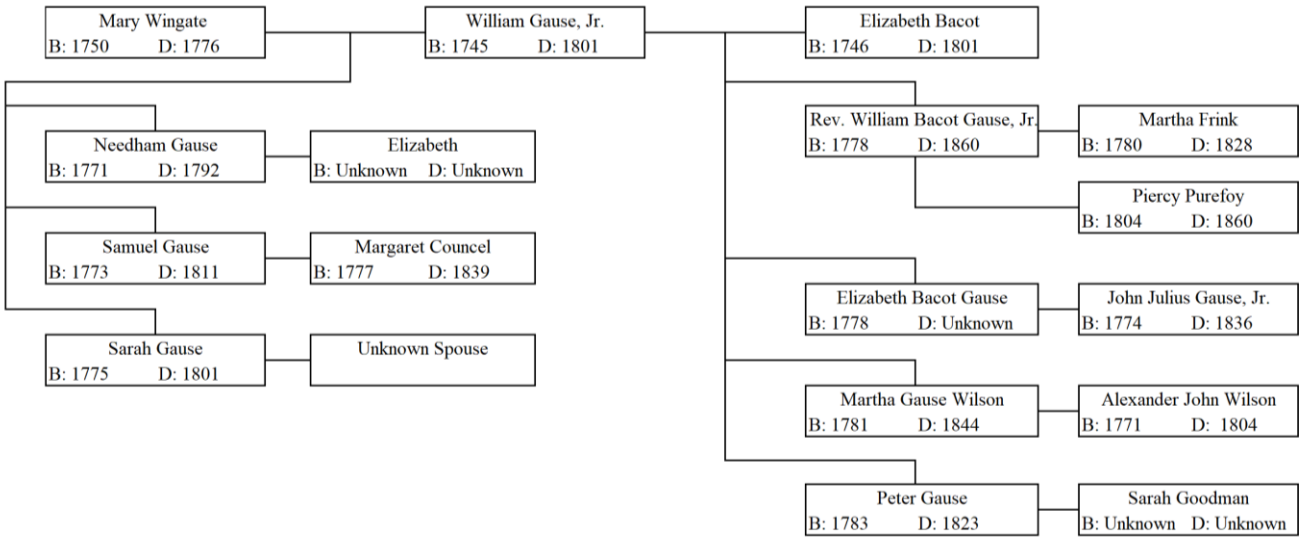

Figure 2.4: William Gause's, Jr. Family (based on information from Christie Family Tree, 2020; Daughters of the American Revolution, 2020; P. Gause, 1823; S. Gause, 1811; W. Gause, 1801; W. Jr. Gause, 1858; The Complete Genealogy Reporter, 2016; Todd, 2020)

Needham Gause, the son of William Gause, Sr., also owned land near the sea according to his will, and land near Indigo Branch and Caw-Caw Swamp near Shallotte (N. Gause, 1794). He married Hannah Frink (1737-1821) and had four sons and one daughter: John (1769-1817),

Needham, Jr. (1775-1818), Bryan (1775-1850), William (1789-1860), and Elizabeth (Figure 2.5) (Christie Family Tree, 2020; N. Gause, 1794). Genealogists suspect based on census and will records that John and William moved to another state, and Bryan and Needham, Jr. stayed in Brunswick County, NC (Christie Family Tree, 2020).

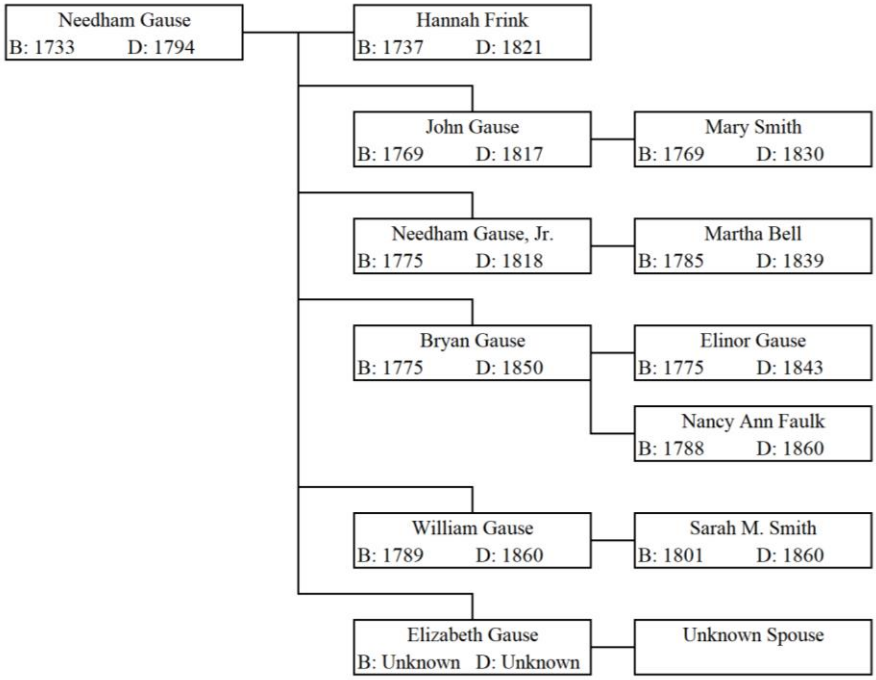

Figure 2.5: Needham Gause's Family (based on information from Christie Family Tree, 2020; N. Gause, 1794)

Charles Gause was a Revolutionary War veteran along with his brothers and also obtained land in Brunswick County near Lockwoods Folly River. However, most of his land and his attention was focused on his holdings near Smithville, now known as Southport, NC (C. Gause, 1779, 1785, 1806). Charles Gause helped get approval for Joshua Potts' petition for the building of this new Brunswick County town located near Fort Johnston (Lee, 1980). After acceptance of the petition by the legislature in 1790-1800, he was appointed as a commissioner of Smithville until 1801 (Lee, 1980). After his death in 1807, Charles Gause, Sr. left his estate

and belongings to his wife, daughters, and grandchildren, Elizabeth Davis and Charles Gibbs, and was buried on his plantation, now known as the Shrub Hill plantation in Winnabow, NC (Figure 2.6) (C. Gause, 1806; Smith, 2011).

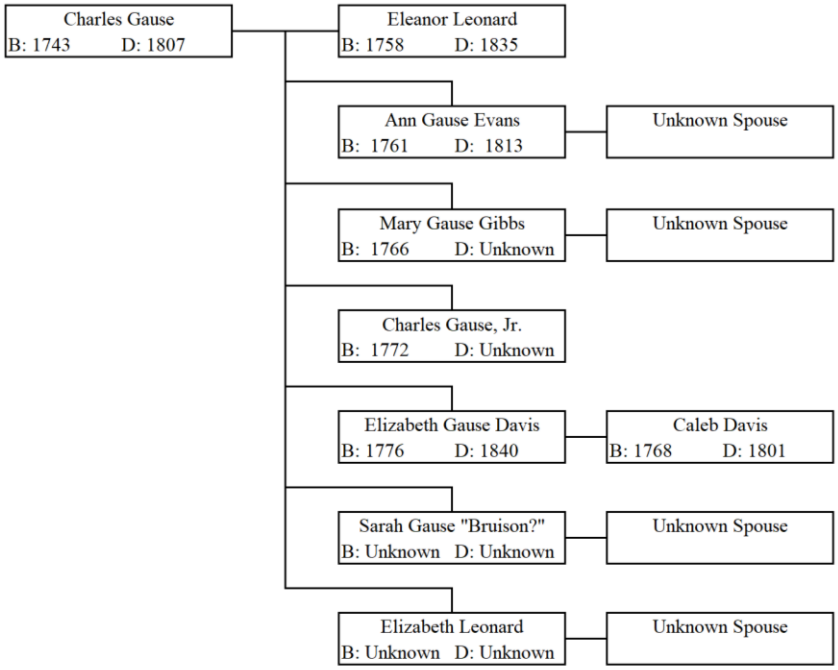

Figure 2.6: Charles Gause's Family (based on information from Christie Family Tree, 2020; C. Gause, 1806; Smith, 2011)

There is little historical documentation of Susannah, Bryan, and Benjamin, the other children of William Gause, Sr. In the U.S. 1790 Census of Brunswick County, a "Susanna Gause" is listed, but because John Gause, Sr. died in 1783 it could be his wife, Susanna Frink Gause (Berry, 1982; J. Gause, 1783; U.S. Census Bureau, 1790). Additionally, a "Bryan Gause" and a "Benjamin Gause" are listed in the 1790 Census (U.S. Census Bureau, 1790). There are also bills of sale in Brunswick County, NC, associated to a Bryan Gause, with the earliest dating from 1790 (B. Gause, 1790a, 1790b) when he sold some slaves, household items such as a hand mill and feather beds, and livestock to Benjamin Gause (B. Gause, 1790b). This bill of sale

might suggest that Bryan Gause moved out of Brunswick County. Furthermore, there is a will of a Bryan W. Gause in Horry County, SC, which dates to 1804, but contains little definitive evidence of his residence (B.W. Gause, 1804). It is also suspected by genealogists and historians that Benjamin Gause moved back to Horry County, SC, and was married to Ann Frink (1755-1814) (Berry, 1982; Christie Family Tree, 2020; K. Gause, 2000).

Finally, one other individual who had land possibly including the Gause Cemetery at Seaside was Samuel R. Frink (1786-1862), who began acquiring Brunswick County land in between that owned by William Gause, Jr. and his brother Needham Gause in 1811. Indeed, the 1865 map (Figure 2.3) identifies that S. Frink owned land near Seaside. The Frinks and the Gauses led intertwined lives, often intermarrying, such as Samuel's aunt Suzanna Frink to John Gause, Sr. or his great aunt Hannah Frink to Needham Gause (Christie Family Tree, 2020), or serving as witness to important events, such as John Julius Gause, Jr. at Samuel's wedding to Elizabeth Bellune in 1807 (State of North Carolina, 1807), or as executor of wills, such Samuel being executor of John Julius Gause, Jr.'s will (J. Gause, 1836). Samuel's land acquisitions included near Shingletree Swamp (S. Frink, 1811; 1840), Little Caw Caw swamp (S. Frink, 1847), east of the Waccamaw River (S. Frink, 1819), and the beach land adjacent to Needham's sons, Needham, Jr. and Bryan (S. Frink, 1818; 1820). It is possible that one of these land acquisitions included the parcel containing the Gause Cemetery at Seaside. Samuel and his family, however, are buried in marked graves in the Frink family cemetery probably located on his original land near Calabash, NC (Find-a-Grave, 2020), and thus likely did not inter any deceased in the Seaside Cemetery. If Samuel Frink did purchase land containing the GCAS, the date of one of these land acquisitions may mark the *terminus ante queum* of use of the cemetery.

Based on the above information, the most likely families who interred their dead at the Gause Cemetery at Seaside are those of John Gause, Sr., William Gause, Jr., and Needham Gause. Additionally, from the description of the land William Gause, Jr. owned, it is likely the Gause Cemetery at Seaside was on William, Jr.'s property, some of which was inherited by his sons, William Bacot and Peter, and some sold to his son, Samuel, although it is not clear which son received the land containing the cemetery. John Gause, Sr. and Needham Gause also owned land to the east and west of this property and could have also used the cemetery as their familial burying ground.

As noted above, there are two known burial sites associated with the Gause family in Brunswick County, NC: the Gause Tomb and Cemetery and the Gause Cemetery at Seaside. It is not clear how the Gause Cemetery at Seaside may be related temporally or lineally to the cemetery surrounding the Gause Tomb, nor if and when this land was acquired by Samuel Frink prior to his death in 1862. The oldest surviving grave marker recovered from the Gause Tomb and Cemetery belongs to Samuel Russ who died in 1829, although in 1961 genealogist Ida Brooks Kellum reported she had found wooden markers for Thomas Frink (Gause) dating to 1802 and Duncan M. Gause from 1808 (David and Bender, 2009). Both the Russ and the Frink families owned land in the area and in the case of the Frinks, often intermarried with the Gauses, so all three families may have used the cemetery.

The large Gause Tomb is thought to be the new vault John Julius Gause requested to be built after his death in May, 1836 (J. J. Gause, 1836). As previously discussed, John Julius dictates in his will that he would like to be interred in the old family vault on the "old burying ground", believed to be Gause Cemetery at Seaside, until a new vault can be built on the "old family ground" on land recently owned by the late Samuel Gause (J. J. Gause, 1836). The land

Samuel Gause purchased from his father, William Gause, Jr. on November 1801, was 620 acres of land near Tubb's Inlet and Little River as well as the parts of the mainland across the sound from Tubb's Inlet, which could have contained the Gause Tomb and Cemetery (S. Gause, 1801). This land fell to his son, William James's (1826), who upon his death willed said land to his wife, Sarah Ann Gause, and child, Mary Jane Gause, after the debt he owed was paid (W. J. Gause, 1824). There is no documentation of who owned the land when the tomb was built, whether it was acquired by John Julius Gause, Jr. before his death, it was sold to someone else, or it was still owned by Sarah Ann and Mary Jane.

The Gause Cemetery at Seaside (GCAS) could be the "old burying ground" mentioned in John Julius, Jr.'s will as well as be the location of John Julius Gause, Jr.'s deceased wives, Mrs. and Mr. Bruard (his in-laws), and children. Additionally the cemetery possibly sat on William, Jr.'s land, but it is unclear as to whether it was sold to Samuel or willed to William Bacot or Peter. Family lore indicates the cemetery was in use during the late 18th century to early 19th century and an architectural survey from the 1960s reports it contained scaled brick tombs, typical of those constructed by plantation elite, and one included a mortar inscription block with an illegible 1830s date (Landmark Preservation Associates, 2010).

Excavation of the Gause Cemetery at Seaside

In 2017, J. R. Robinson, one of the direct descendants of the Gause family and landowner of the GCAS, sought to have his purported ancestors' remains at the GCAS exhumed and studied in preparation for cemetery rehabilitation. He contacted the Department of Anthropology at East Carolina University to oversee the investigation and excavations were conducted in 2017 and 2018 under the direction of Dr. Charles Ewen and Dr. Megan Perry. The 2017 and 2018 field

excavations and initial skeletal analysis formed the basis for ECU students Jorge Quintana's (2019) and Madison Long's (2019) MA theses. A total of 10 individuals were recovered from eight graves generally oriented east to west and organized into at least three rows (Figure 2.7). The 2017 field season excavated four burials (Graves 1, 2, 6, 8), only three of which contained human remains (Graves 2, 6, 8) (Quintana, 2019). Grave 1 was determined to have been misidentified as a burial, which was confirmed during the 2018 field season. The 2018 field season excavated five burials (Graves 3, 9, 10, 11, 12) and recovered seven individuals (Long, 2019). One grave, Grave 10, contained multiple individuals: the lower burial, Grave 10 burial 1, included a 6 to 8-year-old child and the remains of a wooden coffin, and the upper burial, Grave 10 burial 2, contained the remains of two neonates (birth ± 1.5 months) in the remnants of a single wooden coffin. Three other graves, Graves 4, 7 and 13, were identified but not excavated, and more are thought to exist within the fence enclosure and underneath the surrounding modern streets.

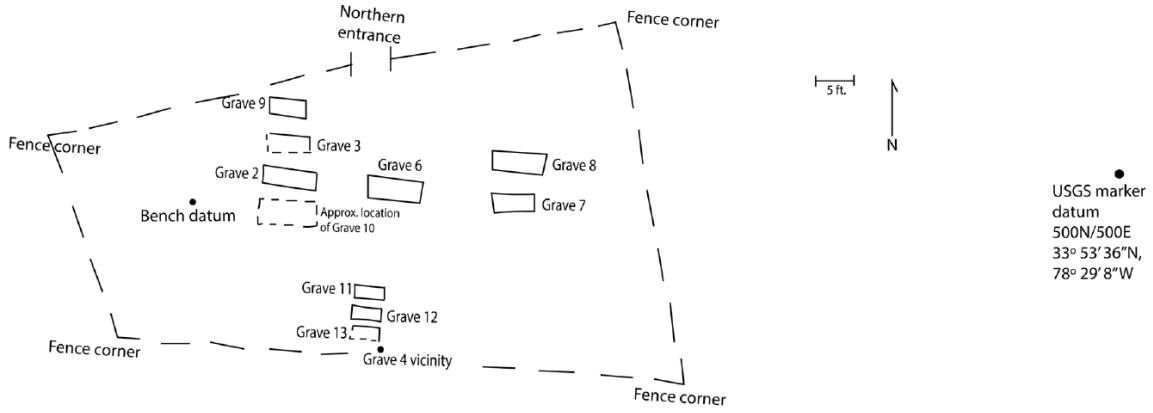

Figure 2.7: Site map of GCAS showing the documented graves and modern fence enclosure (Long, 2019)

The skeletal remains recovered each season underwent osteological analysis focusing on the age, sex, stature, ancestry, and pathologies of each individual (Table 2.1). The eight excavated graves were found to contain six children and four adults, with three of the children interred together in Grave 10. Most of the individuals displayed evidence of childhood physiological stress such as dental enamel hypoplasias (Graves 2, 3, 6, 8, 9, 10 burial 1) and cribra orbitalia (Graves 9, 10 burial 1 and 2), and potential evidence of metabolic conditions evident in flaring of the sternal rib ends (Grave 11) (Long, 2019; Quintana, 2019). Dental enamel hypoplasias (DEH) are an episodic disturbance that presents as a defect along the enamel of the tooth (Goodman & Rose, 1990; Hillson, 2005; Lewis, 2007). The nature of the defect can vary and present itself as a single line, a broad furrow, or isolated pits (Hillson 2005). DEH are known to occur during nutritional stress and physical stress and are often associated with breastfeeding and weaning practices because of the nutritional stress experienced during this time (Goodman & Rose, 1990; Hillson, 2005; Lewis, 2007). Cribra orbitalia is described as porosity of the upper eye orbits which tends to manifest in childhood and is associated with anemia due to multiple causes such as megaloblastic anemia and blood loss from parasitism (Holland & O'Brien, 1997; Rivera & Lahr, 2017; Walker et al., 2009). In addition, the individual excavated from Grave 11 had discernible beading and flaring of the sternal ends of the ribs, which is often associated with rickets or scurvy (Brickley & Ives, 2008; Long, 2019).

The adult individuals showed evidence of joint degeneration in the form of arthritis (Grave 3), osteophytic lipping (Grave 2), and Schmorl's nodes (Grave 2). Osteophytic lipping, bone spurs that surround the edge of vertebral bodies, and Schmorl's nodes, herniation of the nucleus pulposus into adjacent vertebral bodies, are pathologies often associated with trauma

from labor intensive activities, but often can indicate normal age-related degeneration (Lee, 2011; Ortner, 2003).

Other pathologies observed with the GCAS individuals were antemortem tooth loss, caries, mastoiditis, and congenital conditions such as occipitalization of 1st cervical vertebra and os calcaneus secundarius. Antemortem tooth loss and caries were present in all of the GCAS adults. These pathologies are often associated with diets high in carbohydrates because plaque bacteria ferment carbohydrates which produces organic acids that demineralize the enamel, dentin, and cementum (Hillson, 2005). Additionally, the build-up of acid from morning sickness, the loosening teeth from increased hormone levels, gingivitis, and limited attention to oral health can affect the oral health of pregnant mothers (Silk et al., 2008). Furthermore, if a mother has high caries levels, their children also have a high susceptibility of getting caries (Silk et al., 2008).

The occipitalization of the atlas and os calcaneus secundarius are both congenital conditions. Occipitalization of the atlas (seen in Grave 2) is the fusion of the atlas to the occipital condyles and can cause neurological symptoms from headaches to numbness in the limbs (Sharma et al., 2008). Os calcaneus secundarius, another congenital disease in the GCAS sample, is an accessory ossicle between the calcaneus, cuboid, talus, and navicular bone, which can cause ankle pain (Krapf et al., 2015). Lastly, mastoiditis (seen in Grave 2) is caused by untreated middle ear infections and presents as a perforation of the outer surface of the mastoid because of the osteoclastic resorption of the pneumatized mastoid cells (Flohr &Shultz, 2009). This infection is primarily seen in children and adults in pre-antibiotic times (Flohr & Shultz, 2009).

Table 2.1: Biological information of the individuals analyzed from the 2017 and 2018 GCAS project (Long, 2019; Quintana, 2019)

Grave #	Skeletal Age	Estimated Sex	Estimated Ancestry	Estimated Stature	Observed Pathologies
2	30-39 years	Male	European	66.9”-71.7”	Antemortem tooth loss (AMTL); dental abscesses, dental enamel hypoplasias (DEH); osteophytic lipping of vertebrae; Schmorl’s nodes; mastoiditis
3	20-29 years	Female	European	55.7”-61.2”	DEH; caries; AMTL; calculus on all teeth present in mandible; osteoporotic bone loss with cortical thinning across entire skeleton; arthritis observed on C1, C2, & acetabulum
6	25-34 years	Female	European	61.6”-66,7”	AMTL; dental abscesses; DEH; occipitalization of 1st cervical vertebra (congenital condition)
8	20-25 years	Male	European	70.3”-75.2”	AMTL; dental caries; DEH; os calcaneus secundarius (congenital condition)
9	7-8 years	N/A	N/A	N/A	DEH; cribra orbitalia
10 Burial 1	6-8 years	N/A	N/A	N/A	DEH; cribra orbitalia with evidence of healing and active lesions
10 Burial 2 Individual 1	Birth ± 1.5 mos.	N/A	N/A	N/A	Cribra orbitalia with active lesions; bone loss on occipital, unsided parietal, and frontal; bone formation on frontal and parietals
10 Burial 2 Individual 2	Birth ± 1.5 mos.	N/A	N/A	N/A	Cribra orbitalia with active lesions; bone loss on occipital, unsided parietal, and frontal; bone formation on frontal and parietals

Table 2.1 (continued)

11	18 mos. ± 6 mos.	N/A	N/A	N/A	Flaring sternal end, beading, and lateral straightening on both first ribs and several ribs 3-10
12	Birth ± 2 mos.	N/A	N/A	N/A	Cortical erosion across entire skeleton present

The most recent excavations of GCAS also found evidence possibly linking the Gause family to the cemetery and supporting its date. A decorated coffin lid was found in Grave 11 with brass tacks spelling out "J.A.G. Æ 1Y 5M" (Figure 2.8) (Long, 2019). Assuming the last initial stands for Gause, this coffin lid supports the association of GCAS to the Gause family (Long, 2019). Additionally, the decoration of the coffin lid, tack design with fabric lining, and the organization style, the initials and date, all suggest a mid-18th century date (Long, 2019). Other dateable artifacts include metal straight pins and plain bone buttons found with the individuals from Grave 3, 6, 8, 9, 10, 11, and 12. The straight pins were commonly used during the 18th century to fasten burial shrouds, and the plain bone buttons were often used during the 17th and 18th centuries (Long, 2019). Furthermore, the Gause Cemetery's gable-lidded coffin design, the grave shaft pattern, and the barrel-roofed surface marker date to the 19th century

(Quintana, 2019). Thus, the material cultural and architectural evidence supports the predicted date of the late 18th and early 19th centuries.

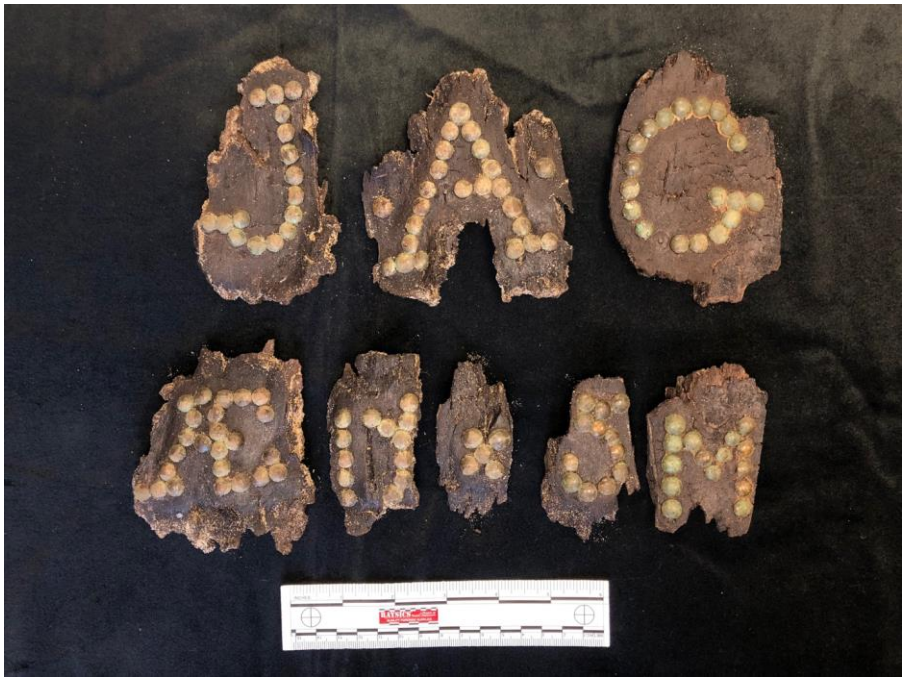

Figure 2.8: Coffin wood with brass tacks from Grave 11 (Long, 2019)

As discussed, many of the GCAS individuals experienced some form of pathology related to physiological stress, particularly those experienced during childhood like dental enamel hypoplasias, cribra orbitalia and possible metabolic deficiencies. Weaning is a process that can cause physiological stress in infants. Human breastmilk provides essential antibodies that provide the infant immunity from diseases or illnesses (Katzenberg et al., 1996; Lewis, 2007; Mays, 2010). Replacing human milk with a milk substitute or ceasing breastfeeding too early could increase susceptibility to infection in infants with immune systems incapable of mounting an effective response, and could also significantly impact their cognitive and emotional development (Brickley & Ives, 2008; Brickley et al., 2014; Chinique de Armas & Pestle, 2018; Katzenberg et al., 1996; WHO, 2003; Wolf, 2018). Additionally, reducing the duration of breastfeeding can potentially condense the interbirth interval (Chinique de Armas &

Pestle, 2018). Traditionally, the reduction of the interbirth and weaning interval have been associated with the adoption of agriculture and different subsistence strategies (Chinique de Armas & Pestle, 2018). Recent studies have shown there to be no correlation between cease weaning age or length of the weaning process and subsistence (Chinique de Armas & Pestle, 2018; Schurr, 2018).

During weaning, an infant's health can also be impacted when supplementing nutritionally-rich breastmilk with a diet lacking in the necessary nutrients for growth and development (Brickley & Ives, 2008; King et al., 2018; Tsutaya & Yoneda, 2015; WHO, 2003). The change in diet can sometimes cause the juvenile to obtain insufficient quantities of necessary nutrients, such as calcium, iron, and vitamin D (Brickley et al., 2014; King et al., 2018). Weaning diets largely based on, the C_4 plant, maize have been known to cause iron deficiency because it is protein deficient and low in iron (King et al., 2018). Thus, this study seeks further bioarchaeological analysis, stable isotope, and histological analysis of the Gause family to characterize their diet and identify whether or not weaning practices or vitamin D deficiency can be associated with the GCAS's high child mortality.

Diet and subsistence in 18th and early 19th century coastal North Carolina

Despite the Gause family's prominence in early Brunswick County history, there is little information regarding the personal lives of the Gause family and the day-to-day operations of their plantation. General historical information on Brunswick County in the late 1700s and early 1800s indicate that most plantation families primarily grew rice in addition to other crops such as corn, wheat, cotton, legumes, American beans, and sweet potatoes (W. Jr. Gause, 1858; Gorrell, 1857; Lee, 1980; Omstead, 1863; U.S. Census Bureau, 1860a; State of Agricultural Society of

North Carolina, 1855). Although they had rice crops, descendants and historians state the Gause family plantation was known for having their main production be in naval stores (Bender, 2017; W. Jr. Gause, 1858). Additionally, according to Needham's, John Julius', Jr., Charles', and John, Sr.'s wills, the Gause family also raised cattle, goats, hogs, and sheep (C. Gause, 1806; J. Gause, 1783; J. J. Gause, 1836; N. Gause, 1794). Furthermore, household gardens provided plantation families with green peas, cucumbers, corn, cabbage, wheat, watermelons, and strawberries (Gorrell, 1857; Omstead, 1863; Sprunt, 1916). They also hunted deer and wild turkeys and fished for oysters (Swift, 1904). Items such as lettuce, Irish potatoes, pork, sugar, coffee, limes, apples, red and white onions, beets, and corn brought in through trading routes and sold through stores also contributed to plantation families' diets (DeNeale, 1855; Sprunt, 1916). The trading routes were either with New York or states bordering North Carolina (DeNeale, 1855; Sprunt, 1916; The Wilmington Daily Herald, 1855).

Additionally, in the southern United States, plantation families mostly ate rich and heavy foods like wine and fatty meat because they believed diet influenced behavior, and that what one ate would affect how one acted in life (Klepp, 1994; Rathburn, 1987; Ross, 1993). As a result, their diet of meat, fruit, milk, butter, and wheat were thought to incite passions, while a diet of enslaved people consisting of corn, rye, potatoes, and cheap cuts of meat was intended to procure obedience (Klepp, 1994; Rathburn, 1987; Ross, 1993).

While historical documents frequently outline political and economic aspects of families, they rarely focus on quotidian details such as family planning, pregnancy, and infant care. These aspects are important because they give insight into the health of the community and aspects in one's daily life that might influence mortality rates. In the early 19th century, most United States doctors lacked medical knowledge of sanitation and disease (McMillen, 1990; Reilly, 2016).

Additionally, doctors had little knowledge of prenatal care and identifying pregnancy in its early stages (McMillen, 1990; Southern Historical Collection, 1853-1873:6). For example, doctors frequently prescribed bleeding or simple tonics to sick pregnant women (McMillen, 1980; McMillen, 1990; Southern Historical Collection, 1853-1873:6). Because of most doctors' lack of knowledge on the subject of pregnancy and disease, most women experienced some illness related to pregnancy as well as unrelated illnesses like malaria (McMillen, 1990). In her letters to Maria Edgeworth, Rachel Mordecai Lazarus discussed the constant sickness she felt after giving birth to her second and third child, sometimes believing she was going to die (Lazarus, 1825, 1828). Most women feared pregnancy and childbirth because they feared illness and, as a result, death (McMillen, 1980).

Unfortunately, the consequence of a weakened state from pregnancy and childbirth was sometimes death, for both the mother and the infant. In the United States during the 19th century, the infant mortality rate was at least 21.6% (Censer, 1984; McMillen, 1980; McMillen, 1990; Roser et al., 2019). From historical studies, it is predicted that in North Carolina at least 230 children died per 1,000, and that 1 out of 4 children did not survive until their 5th birthday (Censer, 1984). Furthermore, society pushed women to have multiple children and provide a large family (Censer, 1984; McMillen, 1980; McMillen, 1990). In 1800, the average family size in the Antebellum South was 7.04 children per woman of childbearing age (Leavitt, 1986; McMillen, 1990:32). When considering stillbirths and miscarriages at that time, the average family size was quite large, but it came at a detriment to women (Leavitt, 1986; McMillen, 1980). According to the 1850 U.S. Census Mortality Schedule, 104 of the 2,706 women in North Carolina who died that year perished during childbirth (McMillen, 1980:71), placing North Carolina at the fourth highest childbirth death rate in the United States (McMillen, 1980:71).

Furthermore, according to the Brunswick County records, 11 women between the ages of 20 and 35 died from childbirth (U.S. Census Bureau, 1850-1885). This statistic does not include the women who died from puerperal fever, a post-delivery infection that ran rampant in the South during the 1850s and 1860s and killed many women in the lower southern states (McMillen, 1980:71). In 1850 in the United States, over 520 women died from puerperal fever, and by 1860, an additional 1,202 women died (McMillen, 1980:72). Although believed to be influenced by climate, puerperal fever was primarily caused by the patient's exposure to unsanitary conditions and contamination from doctors attending to the delivery (Leavitt, 1986; McMillen, 1980; McMillen, 1990).

Besides illnesses and the dangers of childbirth, women also likely experienced nutritional and physiological strains during pregnancy, which not only affected the mother's health, but also the fetus'. When pregnant, a mother can experience undernutrition which can be regulated by stored nutrients (Beaumont et al., 2015). If these stores are depleted, the mother will deprive not only herself of nutrients, but also the fetus. This can result in a low birth weight of the infant and affect their response to nutrition (Beaumont et al., 2015; Norton, 1994; Viteri, 1994). Additionally, long periods of undernutrition can permanently change the mother's and child's metabolic efficiency (Beaumont et al., 2015). With anemic mothers, there is also a shorter pregnancy and higher rates of fetal and infant deaths (Viteri, 1994).

In addition to the frequent ailments experienced by mothers, children in the 18th and 19th century U.S. also frequently experienced illness, and in certain cases, death. After childbirth, the domestic duties of the mother solely focused on the development and health of their child (McMillen, 1980). This usually involved the maternal duty to breastfeed. Most mothers in post-colonial and antebellum North Carolina breast-fed their infants (McMillen, 1990), and only in

times of dire circumstances did higher class mothers use other sources (Child, 1837; McMillen, 1990). To most women, the dire circumstances entailed either their own death or the possibility of passing a disease to their child (Censer, 1984; Child, 1837; McMillen, 1990). Rachel Mordecai Lazarus embodied these rules as she continued to breastfeed her second child even when she felt ill, but stopped breastfeeding her third child when she felt like she was very near death (Lazarus, 1825, 1828).

Breastmilk is very important to an infant's growth and development. Before birth, a fetus will store nutrients like vitamin D and iron to sustain themselves for the first couple of months after birth (Lewis, 2007). If the mother has a nutritional deficiency during this time, the infant will receive limited nutrients for its nutritional stores and predispose them to a nutritional deficiency (Lewis, 2007). After birth, the best source of nutrition for infants is their mother's breastmilk (Lewis, 2007). Breastmilk contains over 100 different nutritional and immunological components vital to human health, one of those constituents being immunoglobulins (Brickley & Ives 2008; Lewis, 2007; McMillen, 1990; Ross, 1981; Tsutaya & Yoneda, 2015). The antibodies found within breastmilk such as IgA molecules provide the needed support against pathogens in the surrounding environment (Lewis, 2007; Rose, 1981). Additionally, the mother's health can have a large impact on not only the nutritional quality of the milk but also whether milk is produced (Allen, 1994; Lewis, 2007). For example, if a mother is deficient in a micronutrient, such as vitamin B-12, the concentration of the vitamin is lowered in the breastmilk, which causes the infant to be deficient as well and possibly develop mental and physiological problems (Allen, 1994).

Wet-nursing and bottle-feeding were other options available to mothers for infant feeding in 17th and 18th century North Carolina. While wet-nursing and bottle-feeding were not ideal,

they did provide nutrition to infants (Child, 1837; McMillen, 1990; Trinkley & Hacker, 2015). However, there was no guarantee a wet-nurse was healthy and disease free. Thus, wet-nurses were often a family friend or other known individuals (Child, 1837; McMillen, 1990; Trinkley & Hacker, 2015).

Bottle-feeding was a more precarious prospect for the infant, for not only did it fail to provide crucial antibodies but the formula itself, usually consisting of two parts cow or preferably donkey milk, one part water, and a little bit of sugar. Rice water and gum-arabic were added once teething began (usually around 6-9 months). Overall, bottle-feeding was not very nutritious (Child, 1837:36; McMillen, 1990; Southern Historical Collection, 1853-1873; Trinkley & Hacker, 2015). Many children became very ill when subsisting on a diet of animal milk and sugar. If the milk spoiled, it allowed for bacteria to grow, sometimes causing cholera infantum to develop, an illness that affects the bowels (McMillen, 1990; Trinkley & Hacker, 2015). According to the federal census in 1860, cholera infantum was five times as likely to occur in the South compared to the North (McMillen, 1990:115; U.S. Census Bureau, 1860b). In Brunswick County, at least six infants died from cholera infantum between 1860 and 1880 (U.S. Census Bureau, 1850-1885).

As discussed, understanding aspects like family planning, pregnancy, and infancy are important to the understanding of daily life during that time but also to understanding levels of stress within a community. From historical documentation, parts of North Carolinians' lives during the 18th and 19th century can be illustrated like the hardships met during pregnancy and after, the illnesses experienced and the high mortality rate. The Gause family also lived during this time and possibly had a similar lifestyle, including the individuals interred at the Gause cemetery at Seaside.

Investigations of diet and weaning practices using stable isotopes

Both diet and weaning practices are integral to culture and understanding the daily lives of the people studied. Characterizing the diet can help determine not only the food available to the community but also, with additional evidence, their trade and agricultural practices. As discussed, weaning practices can have a large impact on the immune system and nutritional sufficiency of children and, when studied, can determine whether weaning practices were a factor of high child frailty and child mortality experienced by the population. When experiencing adverse nutrition early in life, there is a strong chance the child will experience the metabolic syndrome (Almond & Currie, 2011). In addition to the biological profiles and historical documents, stable isotope analysis will provide more information on the GCAS individuals' diets and weaning practices.

Stable carbon and nitrogen isotope analysis was first used by archaeologists to understand ancient diets in the late 1970s (DeNiro & Epstein, 1978; Katzenberg, 2008; Schoeninger & DeNiro, 1984; Schoeninger et al., 1983; Tykot, 2018; van der Merwe &Vogel, 1978). They found carbon and nitrogen isotope ratios from bone collagen could tell a lot about a person's diet from the type of food source (terrestrial or marine) to the type of plants (C_3, CAM, or C_4) they were consuming (DeNiro & Epstein, 1978; Schoeninger & DeNiro, 1984; Schoeninger et al., 1983; van der Merwe &Vogel, 1978). Since these studies, applications of isotope analysis in archaeology have led to more in-depth analyses on past diet and migration with the addition of oxygen, strontium, sulfur, and lead isotopes.

Stable isotopes are nonradioactive forms of an element that differ in atomic mass (Katzenberg, 2008; Price, 2015; Schoeninger & Moore, 1992). The two most commonly occurring stable carbon isotopes in nature are ^{13}C and ^{12}C (DeNiro & Epstein, 1978; Schoeninger

& Moore, 1992; van der Merwe &Vogel, 1978). The ratio of these two isotopes, denoted $\delta^{13}C$ and expressed in parts per mil (‰) when compared to a standard (generally Pee Dee Belemnite) reflects the plants and animals the individual was eating. In bone collagen, carbon isotopes provide information about dietary protein rather than the whole diet because of the structural makeup of collagen (Katzenberg, 2008). There are three types of plants with different photosynthetic pathways, C_4 plants, C_3 plants, and CAM plants, which produce varied $\delta^{13}C$ ranges. C_4 plants such as maize, millet, sorghum, and tropical grasses, have a more positive $\delta^{13}C$ value than C_3 plants (DeNiro & Epstein, 1978; Price, 2015; Schoeninger & Moore, 1992; Tsutaya & Yoneda, 2015; van der Merwe &Vogel, 1978). C_3 plants are found in temperate areas and are usually grasses, most fruits and vegetables, potatoes, rice, wheat, and most cultivated grains (Beaumont & Montgomery, 2016; Emery et al., 2015; Price, 2015). CAM plants are usually succulents and have $\delta^{13}C$ values that are very similar to C_4 values (Schoeninger & Moore, 1992). The $\delta^{13}C$ values for C_4 plants usually range between -9 to -16‰ and for C_3 plants, -20 to -35‰ (Katzenberg, 2008; Schoeninger & Moore, 1992). In addition to plant life, $\delta^{13}C$ values can fluctuate depending on whether marine or terrestrial organisms are being consumed. Marine organisms encounter several different carbon sources compared to terrestrial life (Schoeninger & Moore, 1992). These carbon sources cause marine organisms to reflect $\delta^{13}C$ values between typical C_3 and C_4 values (Schoeninger & Moore, 1992).

Nitrogen can also provide some information about the individual's diet. Nitrogen has two stable isotopes ^{15}N and ^{14}N, which, when compared in a ratio, $\delta^{15}N$ (also expressed in ‰), can tell the trophic level of an individual (Price, 2015; Schoeninger & DeNiro, 1984; Schoeninger et al., 1983). Organisms have an enrichment value of 2-3‰ over the values of the food they consume (Price, 2015; Schoeninger & Moore, 1992). Marine organisms and terrestrial organisms

can also be distinguished when looking at $\delta^{15}N$ values because marine organisms tend to have higher $\delta^{15}N$ values than terrestrial organisms because they are exposed to a higher concentrations of ^{15}N (Schoeninger & DeNiro, 1984; Schoeninger & Moore, 1992). In addition to diet, $\delta^{15}N$ values can be affected by physiological stress (Fuller et al., 2005; Katzenberg & Lovell, 1999; Schoeninger & DeNiro, 1984). When the body is under stress, it goes into a catabolic state. This state causes $\delta^{15}N$ values to increase because the body is taking the stored protein and metabolizing the ^{14}N isotope (Fuller et al., 2005; Katzenberg & Lovell, 1999).

Besides diet, stable isotope analyses can identify breastfeeding and weaning practices used by past populations. Children relying on breastmilk have enriched $\delta^{15}N$ compared to the source of the milk, and those relying on human milk will have a relatively higher $\delta^{15}N$ value than the adults at a site (Fogel et al., 1989; Katzenberg, 1993; Katzenberg et al., 1996; Schoeninger & Moore, 1992; Schurr, 1997). Once the child starts weaning off of breastmilk and having solid foods incorporated into their diet, their $\delta^{15}N$ level drops to adult levels (Fogel et al., 1989; Katzenberg, 1993; Schurr, 1997). The pattern of decrease in $\delta^{15}N$ across skeletal tissues forming at different ages may indicate the ages weaning began and ended (Fogel et al., 1989; Katzenberg, 1993). This pattern varies if the child's diet has been supplemented with animal milk, formula, or weaning foods (Fogel et al., 1989; Fuller et al., 2006; Katzenberg, 1993), which will cause the child's $\delta^{15}N$ values to be lower than a solely breastfeeding infant (Fogel et al., 1989; Katzenberg, 1993).

Stable carbon isotopes also provide insight into weaning practices by identifying foods used to supplement breastmilk, particularly if infants are fed a specialized diet different from adults (Fuller et al., 2006; Katzenberg, 1993; Tsutaya & Yoneda, 2015). They also provide

insight to when weaning begins and ends. Breastfeeding infants have approximately 1‰ increase in $\delta^{13}C$ values compared to their mothers (Fuller et al., 2006; Tsutaya & Yoneda, 2015).

In most recent studies of weaning practices in stable isotope analysis, researchers have looked into the effect nutritional and immunological stress has on stable isotopes. Beaumont and colleagues (2015) found bone turnover rate is not similar between infants and adults, so when a mother is acutely ill, her slow bone turnover rate means that bone collagen nitrogen values reflecting this stress may not strongly impact her overall nitrogen value. However, due to the rapid bone growth and turnover in fetuses and infants this impact would not be "swamped" by older bone values as they are in the mother. They also found bone collagen does not represent the diet of the individual at the time of death because of bone turnover and because of differential growth, resulting in different values and patterns of change in bone collagen versus dentin collagen nitrogen and carbon values with age (Beaumont et al., 2015; Beaumont et al., 2018). This divergence can partially result from patterns of bone and dentin growth and formation during stress. During illness or a period of nutritional deficiency, dentin in teeth continues to form and thus captures the associated increase in $\delta^{15}N$. Bones, on the other hand, halt growth, and thus they do not capture dietary isotopes during this period (Beaumont et al., 2015; Beaumont et al., 2018).

The U.S. 18th century to 19th century diet for higher-status adults in both rural and urban areas relied heavily on meat and cereals (Ross, 1993). The Gause family is expected to have had a diet of various types of marine and terrestrial meat as well as maize and rice, based on historical sources (U.S. Census Bureau, 1860a; C. Gause, 1806; J. Gause, 1783; J. J. Gause, 1836; N. Gause, 1794; W. Jr. Gause, 1858; Gorrell, 1857; Lee, 1980; Omstead, 1863; State of Agricultural Society of North Carolina, 1855; Swift, 1904). Stable isotope analysis of Mid-

Atlantic and southern 18th and 19th century U.S. populations have found a heavy reliance on the C_4 crop, maize, as opposed C_3 plants like wheat which was most commonly consumed further north (France et al., 2014; Owsley et al., 2018; Seeman, 2011; Schurr, 2018; Trinkley & Hacker, 2015; Ubelaker & Owsley, 2003; Vigeant et al., 2017). It has been suggested by some studies with populations that have both C_3 and C_4 signatures that this variation in diet could be due to immigration (Schurr, 2018). As noted earlier, diets also varied because of socioeconomic status, with "lower class" individuals having lower $\delta^{15}N$ values than "upper class" individuals from 18th and 19th century sites across North America (France et al., 2014). At Orton Plantation, also in coastal Brunswick County, this typical high-class diet was supplemented by fish (Trinkley & Hacker, 2015), a finding also observed among U.S. soldiers who fought and died in the war of 1812 in Ontario, Canada (Emery et al., 2015; Raynor & Kennett, 2008). In general, Mid-Atlantic and southeastern U.S. communities in the 18th and 19th centuries relied on a diet consisting of both C_3 and C_4 terrestrial plants.

The diets of children, as opposed to adults, in these communities may have included the same foods, but seem to have been more restricted. In 18th and 19th century America, weaning diets were made up of broth, bread, pap (a mixture of corn or other grain flour and milk or water), and panada (a varied combination of flour, cereals, butter, milk, and broth) (Child, 1837; Fildes, 1995; Mays, 2010; McMillen, 1990; Schmidt, 1976). However, unlike breastmilk, animal milk and pap or panada did not provide sufficient nutrition for a growing infant, and often lacked or even prevented absorption of trace elements such as vitamin D, calcium, or iron (Brickley & Ives, 2008; Lewis, 2007; Schmidt, 1976). The dependence on maize and the cultivation of rice by adults in the southern U.S. (France et al., 2014; Owsley et al., 2018; Seeman, 2011; Trinkley & Hacker, 2015; Ubelaker & Owsley, 2003; Vigeant et al., 2017) likely meant that infants

consumed pap or panada made from maize or rice and may show a higher $\delta^{13}C$ value (maize) or a lower $\delta^{13}C$ value (rice) depending upon which cereal grain was consumed more. Owsley and colleagues (2018) found in their study of individuals buried at the 18th century Darnall's Chance site in Prince George's County, Maryland, that the weaning diet consisted of mostly C_4 plants while the adult diet contained more C_3 plants, which went against 19th century recommendations for weaning. When examining the individuals found at the Old Frankfort Cemetery (1810-1850) in Frankfort, Kentucky, Schurr (2018) found there to be more variability in weaning diet, which follows Mrs. Child's (1837) recommendations. Mrs. Child suggests in *The Family Nurse; or Companion of the Frugal Housewife* that rice, bread, milk, water, gum-arabic, arrow-root, and/or sugar be used as ingredients in weaning foods (Child, 1837). This would lead to signatures that read both C_3 and C_4 because sugarcane is a C_4 plant while rice, gum-arabic, and arrow-root are C_3 plants.

Similar to dietary composition, weaning ages in 18th and 19th century England and the U.S. depended on the individual's socioeconomic status. In England, higher status mothers tended not to breastfeed their children but instead relied on either a wet nurse or milk from an alternative source, such as cows or goats (Mays, 2010; Newman & Gowland, 2017). Weaning ended around 1 to 2 years of age depending on one's socioeconomic status in England during the 17th to 20th century (Fildes, 1995; Mays, 2010; Newman & Gowland, 2017; Schmidt, 1976). Infants born into a higher economic status were given a wet nurse or dry nursed (given pap or panada), which did not provide the nutrients needed, while infants born into a lower economic status were breastfed for a shorter period of time and weaned at an earlier age because the mother needed to work (Britton et al., 2018; Fildes, 1995; Newman & Gowland, 2017). In North America during the 18th and 19th century, weaning ages varied widely, with infants introduced to

weaning foods, such as broth, bread, pap, and panada between 3 to 12 months of age and fully weaned by 6 months to 2 years of age (Censer, 1984; Child, 1837; Fildes, 1995; McMillen, 1990; Schmidt, 1976). Modern clinical studies have shown that weaning should start between 4 and 6 months and last 2 years or beyond (Grueger et al., 2013; Mayo Clinic Staff, 2019; Victora et al., 2016; WHO, 2020).

Isotopic evidence for weaning and childhood diet has been explored in many areas of the globe to help understand infant morbidity and mortality patterns in the past (e.g., Beaumont et al., 2015; Beaumont et al., 2018; Dupras & Tocheri, 2007; Garland & Reitsema, 2018; Katzenberg & Pfeiffer, 1995; Mays, 2010; Schurr, 2010). Stable isotope analyses at historic North American sites suggest the cease weaning age varied between sites (Katzenberg & Pfeiffer, 1995). Collagen samples from Prospect Hill cemetery in Toronto (19th century) suggest weaning ended around 2 years of age, while the collagen samples from St. Catherine's Island, Georgia (16th century) deduced that weaning was completed between 1.5 and 3.5 years of age (Garland & Reitsema, 2018; Katzenberg & Pfeiffer, 1995). Additionally, no direct correlation was established when applying results from pre-historic and historic North American sites to a demographic measure of fertility (Schurr, 2010). Other studies in the Eastern hemisphere have looked at the mother's bone mineral density compared to weaning age and the differences in diet between *utero* (pre-weaning, deciduous dentin) and post-birth (post-weaning, permanent dentin) (Dupras & Tocheri, 2007; Mays, 2010). These studies have found mothers experience low bone mineral density possibly due to prolonged breastfeeding (Mays, 2010).

Besides looking at age-related trends in an entire sample as discussed in the studies above, weaning practices can also be determined individually by sampling multiple sections of the dental dentin. Incremental sampling can be useful for getting a more precise timeline of

weaning practices and to look into an individual's childhood diet and weaning practices (Beaumont et al., 2015; Beaumont et al., 2018; Czermak et al., 2018; Garland & Reitsema, 2018; Mays, 2010). When observing incremental dentin and weaning practices, Garland and Reitsema (2018) found a significant variation in weaning practices between individuals from St. Catherine's Island, Georgia. Other studies have used incremental sampling of the dentin and found differences between bone and dentin stable isotope values and their relationship to physiological stress (Beaumont et al., 2015; Beaumont et al., 2018). Furthermore, in order to get a more precise chronological age of the incremental sample, Czermak and colleagues (2018) have tried to perfect the method by using microscopy images of the teeth as guides for incremental sampling.

Bioarchaeological investigation of metabolic disease

As noted above, a nutritionally inadequate diet during and after weaning could impact infant health. All of the individuals, except for the individuals from Grave 11 and 12, from the Gause cemetery displayed physiological stress indicators like dental enamel hypoplasias and cribra orbitalia. These indicators, including the possible identification of rickets or scurvy in the sample in the form of sternal rib end flaring, may signal poor maternal nutrition or a nutritionally inadequate supplemental diet in terms of vitamins C or D during weaning (Brickley & Ives, 2008; Brickley et al., 2014). Vitamin D plays a crucial role in maintaining calcium homeostasis and bone mineralization and it is usually absorbed and synthesized following exposure to UVB radiation or ingestion (Brickley et al., 2014; Holick, 1996). Vitamin D is then metabolized in the liver and hydroxylated in the kidneys to produce a more active form of vitamin D, $1,25(OH)_2D$ (Brickley et al., 2014; Holick, 1996). $1,25(OH)_2D$ manages calcium homeostasis in the blood by

improving the efficiency of intestinal calcium absorption and directly activating osteoclast production and the mineralization of calcium in bones (Bitzan & Goodyer, 2019; Brickley et al., 2014; Holick, 1996). Any failure point in the synthesis of vitamin D or its pathway may cause the individual to develop a deficiency in vitamin D.

The main factors that can cause this failure and resulting deficiency are lack of UVB radiation from sunlight, vitamin D-poor diet, genetic mutations, and comorbidity with certain diseases. Humans can only synthesize vitamin D via exposure to UVB radiation, thus, vitamin D deficiency may result from any factors that inhibit this process, such as seasonal fluctuations in sunlight, skin pigmentation, or cultural practices that inhibit sun exposure (Brickley & Ives, 2008; Holick, 1996; Holick, 2006). Foods naturally high in vitamin D such as eggs and fish, or vitamin-D fortified meat, cereals, or milk can prevent vitamin D deficiency (Brickley & Ives, 2008; Holick, 1996; Holick, 2006). Since fortification of vitamin D in food did not start until the early 20th century (Brickley & Ives, 2008; Holick, 1996; Holick, 2006), this was not a resource for pre-20th century populations. The only source adequate to provide sufficient levels of vitamin D during the 18th and 19th century was UVB exposure (Brickley & Ives, 2008; Holick, 1996; Holick, 2006). Additionally, some genetic disorders like vitamin D-dependent rickets type 1 and autosomal dominant hypophosphatemic rickets can cause or lead to vitamin D deficiency and could have been experienced in 18th and 19th century U.S. populations. Vitamin D-dependent rickets type 1 is a rare autosomal recessive disorder caused by the loss of function in the 25-hydroxyvitamin-D-1 α-hydroxylase gene and results in the inability for 25(OH)D to convert to vitamin D in the kidneys (Brickley & Ives, 2008; Yan et al., 2011). This hereditary disorder usually manifests itself before the individual is 2 years old (Brickley & Ives, 2008; Yan et al., 2011). Autosomal dominant hypophosphatemic rickets is a hereditary disorder caused by

mutation of the FGF23 gene which causes renal phosphate wasting and a depletion in the total body phosphorous stores (Brickley & Ives, 2008; Econs & McEnery, 1997).

Vitamin D in fetuses and breastfeeding infants is supplied via the mother's placenta or breastmilk, and thus, any deficiency in the mother will be passed on to the infant (Brickley & Ives, 2008; Littleton, 1998; Newman & Gowland, 2017). This deficiency can be reduced if the infant is exposed to UVB radiation after birth (Brickley & Ives, 2008; Holick, 1996; Holick, 2006; Newman & Gowland, 2017). As weaning progresses, breastmilk, which typically has adequate levels of vitamin D in healthy mothers, is slowly replaced by supplemental foods, which may or may not have sufficient levels of calcium and vitamin D (Brickley et al., 2014; Littleton, 1998). In America during the 18th and 19th centuries, cereals were a staple for weaning. Cereals are high in phytates, which cause less calcium to be absorbed and can contribute to the development of vitamin D deficiency (Brickley & Ives, 2008; Lewis, 2007; Littleton, 1998; Schmidt, 1976). Overall, the combination of malnutrition and limited exposure to sunlight among American infants and children could have resulted in vitamin D deficiency within the population during the 18th and 19th centuries (Lewis, 2007; Schmidt, 1976).

Vitamin D deficiency can have numerous detrimental effects to the body, but its involvement in malabsorption of minerals means it most dramatically affects the skeletal system. Individuals deficient in vitamin D will not have sufficient skeletal absorption of calcium and phosphorus, important elements in bone mineralization. The lack of either of these minerals hinders mineralization of bone protein (osteoid), particularly in the long bones, pelvis, ribs, and cranium (Brickley et al., 2014; Holick, 1996; Holick, 2006; Ortner & Mays 1998). Prolonged vitamin D deficiency can eventually result in bone diseases such as rickets in children and osteoporosis or osteomalacia in adults. The low bone mineral density in osteomalacia results in

an increased risk for buckling or compression bone fractures, often called “pseudofractures” (Brickley & Ives, 2008; Brickley et al., 2014; Waldron, 2009). Rickets only emerges in vitamin D-deficient subadults undergoing bone growth and development and is indicated by poor mineralization at the epiphyseal plates and low resistance to biomechanical stress in the long bones. The cartilaginous cells at the ends of the metaphyses change arrangement as the bone undergoes stress due to crawling and walking, resulting in a “frayed” and flaring metaphyseal area often accompanied by abnormal curvature, or bowing, of the long bone diaphysis (Brickley & Ives, 2008; Brickley et al., 2014; Holick, 2006; Ortner & Mays, 1998). If the vitamin D deficiency is resolved during childhood, the bone can remodel, correcting more mild deformities and making any childhood deficiency difficult to identify in adult skeletons (D’Ortenzio et al., 2016; Holick, 2006; Waldron, 2009).

Additionally, vitamin C deficiency can also be present when an adult or child is experiencing malnutrition. Unlike most mammals, humans cannot synthesize vitamin C or ascorbic acid and so must retrieve it through dietary sources (Brickley & Ives, 2008; Lewis, 2007). Vitamin C is essential to the production and formation of type 1 collagen, which forms the connective tissues of bone, cartilage, blood vessels, and skin (Brickley & Ives, 2008; Hasegawa et al 2011; Lewis, 2007). Vitamin C or ascorbic acid helps form type 1 collagen by being a cofactor in the hydroxylation of lysine and proline (Brickley & Ives, 2008; Lewis, 2007; Pinnell, 1985). Hydroxylysine maintains collagen’s structural integrity while hydroxylproline helps collagen be secreted from fibroblasts and osteoblasts (Pinnell, 1985; Hasegawa et al., 2011). Additionally, vitamin C influences the adherence of osteoblasts to the bone matrix by indirectly affecting the structural makeup of the collagen fibrils (Angelo et al., 2019; Hasegawa

et al., 2011). Thus, any failure in obtaining or synthesizing vitamin C can lead to the individual having a vitamin C deficiency.

The daily requirements of vitamin C vary and depend upon sex and age. Infants need 40 to 50 mg/day while children need less than half of the daily requirements of infants (15mg/day) (Edwards, 2019; Popovich et al., 2009). Additionally, pregnant women's vitamin C daily requirements are 85mg/day and only increase when breastfeeding (120mg/day) (Edwards, 2019; Popovich et al., 2009). The most common foods that contain vitamin C are fruits and vegetables (Angelo et al., 2019; Brickley & Ives, 2008). Trace amounts of vitamin C are also contained in milk, meat, and fish (Angelo et al., 2019; Brickley & Ives, 2008). When digested, vitamin C cannot be stored in the body because it is water soluble, and so limited supplementation and access to any of these foods and a daily intake of less than 10 mg/day in an individual's diet can cause vitamin C deficiency (Brickley & Ives, 2008; Fain, 2005). Additionally, vitamin C is heat, oxygen, and UV sensitive and will decrease in content when exposed to these elements (Brickley & Ives, 2008). Therefore, pasteurized products or un-fresh fruit and vegetables are not suitable sources for the supplementation of vitamin C (Brickley & Ives, 2008; Fain, 2005). Some genetic factors can also affect vitamin C levels in the body, for example, human plasma protein haptoglobin (Hp). Haptoglobin (Hp) has three different phenotypes, Hp 1-1, Hp 2-1, and Hp 2-2 (Delanghe et al., 2011). Hp prevents oxidative damage caused by heme iron in free hemoglobin (Hb) (Delanghe et al., 2011). Unlike Hp 1, Hp 2 is inefficient at binding to Hb and so vitamin C is used (Delanghe et al., 2011). Thus, any individual with the Hp 2 allele will be more susceptible to vitamin C deficiency (Delanghe et al., 2011).

Like vitamin D, vitamin C is supplied to fetuses and breastfeeding infants via the mother, and so any deficiency the mother has is passed on to their child (Brickley & Ives, 2008;

Delanghe et al., 2011). Generally, vitamin C is abundant in breastmilk and so vitamin C deficiency is not often seen in societies that breastfeed, but rather in societies that use artificial feeding (Brickley & Ives, 2008). In the United States during the 18th and 19th century, an artificial source most commonly used with the addition of other ingredients was cow milk (Child, 1837:36; McMillen, 1990; Rajakumar, 2001; Southern Historical Collection, 1853-1873; Trinkley & Hacker, 2015). Cow milk contains less vitamin C than breastmilk, and therefore, is less likely to contain adequate levels of vitamin C (Brickley & Ives, 2008). Additionally, weaning foods during the 18th and 19th century were mostly made up of dried cereal grains and corn, which have little to no vitamin C and could have contributed to the development of a vitamin C deficiency (Brickley & Ives, 2008; Child, 1837; Griffiths, 2020). Overall, the substitution to breastmilk and malnutrition of infants and children in the United States in the 18th and 19th century could have resulted in scurvy (Brickley & Ives, 2008; Griffiths, 2020; Rajakumar, 2001).

Vitamin C deficiency, or scurvy, has a large effect on both the soft and hard tissues found within the body (Brickley & Ives, 2008). Scurvy weakens the collagen structure of blood vessels which results in hemorrhaging throughout the body like hemarthrosis in the joints due to the separation of the periosteum from the bones (Brickley & Ives, 2008). Scurvy can also cause the gums to swell and recede and the periodontal ligament to deteriorate, which could cause the teeth to become loose and fall out (Brickley & Ives, 2008). Additionally, prolonged vitamin C deficiency can lead to structurally weakened and remodeled bone. With infants and children, scurvy can start to manifest itself in children 4 to 10 months of age (Lewis, 2007; Ortner, 2003). It appears as porosity and periosteal formation on the cranialfacial bones, such as the sphenoid, mandible, maxilla, frontal, and zygomatics, the pelvis, the scapulae, the long bones, and the ribs

(Brickley & Ives, 2008; Ortner, 2003). Furthermore, the absence of functional collagen causes cortical bone fractures to occur in the ribs near the costochondral joint and the metaphysis of long bones (Brickley & Ives, 2008; Lewis, 2007; Ortner, 2003). Adults undergo similar manifestations as children; however, vitamin C deficient adults do not experience cortical bone fractures in the long bones (Brickley & Ives, 2008; Ortner, 2003). Additionally, adults with scurvy can develop osteopenia in the vertebrae (Brickley & Ives, 2008).

Therefore, both vitamin C and D deficiencies can cause flaring of the sternal rib ends, but the lack of other diagnostic features makes the exact cause unclear. In order to identify a possible cause of the sternal rib end changes, a feature unique to rickets in dental dentin was studied to identify vitamin D deficiency as a cause. As previously mentioned, rickets can cause changes to molar pulp chamber shape, particularly the height of the pulp horns (D'Ortenzio et al., 2018b), and the development of interglobular dentin (IGD) (D'Ortenzio et al., 2016). Pulp chamber anomalies in molars can be observed through buccal-lingual radiographs from which the pulp horns and chamber of the molars are measured. Past vitamin D deficiency is reflected by a 2:1 ratio of pulp horn heights and pulp horn widths of <1 mm (D'Ortenzio et al., 2018b). Because it is non-destructive in nature, this method is a good diagnostic first step in identifying whether an individual had vitamin D deficiency in childhood.

The mineralization of the dentin is also slowed in individuals with vitamin D deficiency, causing spaces to form that contain incompletely coalesced calcium salts known as interglobular dentin (IGD) (D'Ortenzio et al., 2016). The presence of IGD is identified through histological analysis of tooth thin sections. The number of events and severity of periods of deficiency can be identified by the density and location of IGD (D'Ortenzio et al., 2016). In addition, the age of the episodes can be estimated by linking calcification and eruption times of the dentin with the

location of the IGD because, unlike bone, dentin ossification (dentineogenesis) occurs in layers and is not replaced or turned over (Brickley et al., 2019; D'Ortenzio et al., 2016, 2018a). The dentin cells form the layers by moving inward, then laying down the dentin incrementally starting at the tooth cusps. Each incremental layer represents a year or more (Brickley et al., 2019). Assessment of IGD must rule out other factors such as diagenesis, contour lines of Owen, marbling, and developmental interglobular dentin (DIGD), which is imperfectly calcified dentine that is usually located near the dentinal periphery and does not align with incremental lines (D'Ortenzio et al., 2018a). Diagenesis rarely occurs in teeth, but when it does occur, it can obscure evidence of IGD (D'Ortenzio et al., 2018a). Contour lines of Owen appear as lines that run parallel to the enamel, but unlike IGD the lines do not have scalloped characteristics (D'Ortenzio et al., 2018a). Marbling is a normal variation that occurs in the dentin which appears mottled but does not have the empty spaces seen with IGD. Lastly, DIGD is thought to form when the thick sections of dentine do not mineralize completely which leave spaces in the matrix and can look like low grade IGD, but its size, amount, and location appear natural (D'Ortenzio et al., 2018a).

Skeletal evidence of vitamin D in the past has been linked to cultural practices in addition to a vitamin D deficient diet. Prior to the Industrial Revolution, high status individuals experienced vitamin D deficiency because of cultural practices that limited their exposure to sunlight, such as swaddling during infancy and makeup during adulthood (Giuffra et al., 2015). In a study on the Medici family who lived during the 16th and 17th centuries, Giuffra and colleagues (2015) found skeletal indicators of active rickets in individuals 4 years and younger and recovering rickets in individuals aged 5 years and older. Stable isotope analysis revealed that the children began weaning at the age of 2. Breastmilk is not an adequate source of nutrition after

6 months of age and thus rickets probably developed as a result of this cultural practice of weaning children later. By the Industrial Revolution (18th and 19th centuries), both upper- and lower-class individuals were experiencing vitamin D deficiency in the urban settlements (Holick, 2006; Newman & Gowland, 2017; Schmidt, 1976; Saunders et al., 2002). In England, Newman and Gowland (2017) found that urban upper-class infants experienced vitamin D deficiency which they attributed to community fashion trends, such as early weaning and having a wet nurse. Conversely, the lower class likely experienced vitamin D deficiency because of malnutrition, excessive time spent indoors, and pollution (Newman & Gowland, 2017; Mays et al., 2006).

Although most studies (e.g. Newman & Gowland, 2017; Schmidt, 1976; Saunders et al., 2002) have discussed vitamin D deficiency as it affected urban dwelling individuals, it could also occur in individuals living in rural environments. In a rural Dutch town, skeletal indicators of vitamin D deficiency were found in some subadult remains, and based on historical documentation, attributed to cultural practices, like swaddling, and weaning diet (Veselka et al., 2015). The weaning diet consisted of cow or goat milk mixed with cereals which was similar to historic accounts of North American weaning diet (Lewis, 2007; Schmidt, 1976; Veselka et al., 2015) Furthermore, because these children from a rural area developed vitamin D deficiency, this population could serve as a good comparison for vitamin D deficiency in children from the Gause family (Lewis, 2007; Schmidt, 1976).

While many examples of vitamin D deficiency have been noted in 17th and 19th century Britain and Western Europe (Giuffra et al., 2015; Newman & Gowland, 2017; Mays et al., 2006; Veselka et al., 2015), rickets has been less documented in the U.S. during the same period. Most examples of rickets have been found in the skeletal remains of adult enslaved and free

populations of African descent. In the 18th and 19th century, most African Americans from Virginia, Maryland, the Carolinas, and Northern states experienced rickets either seasonally or long-term (Kelley & Angel, 1987; Klepp, 1994). Kelley and Angel (1987) found tibial bowing, residual evidence of childhood rickets, in more than 50% of African American females and males in a 19th century population buried near Catoctin Furnace, Maryland. At this time, African American diet was typically low in calcium, which is needed for vitamin D synthesis (Brickley & Ives, 2008; Kelley & Angel, 1987). Together, the low calcium diet, skin pigmentation, and the limited sunlight during the winter seasons in the Mid-Atlantic States, could have contributed to rickets being present in the population (Kelley & Angel, 1987; Klepp, 1994).

As discussed, one individual from the GCAS site, an 18-month-old (± 6 months) in Grave 11, showed skeletal signs of rickets or scurvy in the ribs in the form of flaring of the sternal end, lateral straightening and beading of the left and right first ribs and six rib 3-10 fragments (Figure 2.9) (Long, 2019). Weaning practices can be a contributing factor to vitamin D and vitamin C deficiency because of the change in diet and nutritional sufficiency (Brickley & Ives, 2008; Brickley et al., 2014; Lewis, 2007). Weaning diets during the 18th and 19th century were supplemented with foods like cereals and animal milk which lacked or prevented sufficient levels of calcium, vitamin C, and vitamin D (Brickley & Ives, 2008; Lewis, 2007; Schmidt, 1976). Although macroscopically vitamin D and vitamin C deficiency were not expressed in the other individuals from the Gause cemetery, they still could have experienced nutritional deficiencies in early childhood. Histological analysis can help identify childhood vitamin D deficiency by identifying IGD (D'Ortenzio et al., 2016).

Figure 2.9: Signs of metabolic disease observed in the left first rib of the individual from Grave 11 (Long, 2019)

Summary

Breastfeeding and weaning practices play a significant role in the maintenance and survival of a population (Katzenberg et al., 1996). If changes in practices move a body away from homeostasis, infant mortality could rise (Katzenberg et al., 1996). Breastfeeding and weaning are both important stages in infant and childhood development because they provide the infant with necessary antibodies to ensure survival (Katzenberg et al., 1996; Lewis, 2007; Mays, 2010). Weaning foods also factor heavily into infants' nutrition and can result in illness, including vitamin D deficiency, when nutritionally inadequate (Brickley & Ives, 2008; Brickley et al., 2014). The notable number of infants and children in the sample excavated from Gause cemetery at Seaside may indicate that childhood environment, including breastfeeding and

weaning practices, age and diet, could have impacted childhood health and mortality. Thus, I expect the GCAS stable isotope values from incremental dentin and bone collagen will depict a weaning age earlier than 2 years, different from current medical guidelines, and a weaning diet consisting of maize, a C_4 plant, which is an important component of the historic diet in Maryland and North Carolina (Grueger et al., 2013; Mayo Clinic Staff, 2019; Owsley et al., 2018; Seeman, 2011; Trinkley & Hacker, 2015; WHO, 2020; Victora et al., 2016). Additionally, the GCAS adults' bone collagen values should reflect a diet consisting of C_4 plants with the supplementation of marine meat, similar to the diets expressed by the Orton and Foscue sites in North Carolina (Seeman, 2011; Trinkley & Hacker, 2015).

Furthermore, if individuals at the Gause cemetery experienced a nutritionally deficient weaning diet and vitamin D deficiency during childhood, their weaning ages will correspond with the ages at which they experienced insufficient levels of vitamin D. Techniques such as stable isotopes analysis and histological analysis can help determine weaning ages and whether vitamin D deficiency was experienced by the individual even if not expressed macroscopically. Overall, these techniques and results will further delve into the daily lives of coastal North Carolinians and the possible factors that led to the high child mortality seen at the GCAS site.

CHAPTER THREE:

METHODS

Stable isotope methods

The study's samples came from the ten individuals exhumed from the Gause Cemetery. The determination of the beginning and end of the weaning process and the characterization of the childhood and adult diets was accomplished through analyzing bone and dentin collagen for $\delta^{13}C$ and $\delta^{15}N$. Out of concern for the friability of the sample and there being limited collagen, larger increments were taken from the dentin samples from three location: superior 3/4ths of the crown, from 3/4ths of the crown to the upper $1/4^{th}$ of the root, and from the upper $1/4^{th}$ of the root to the apex (see the example in Figure 3.1). The age associated with each section sampled was determined using AlQahtani and colleagues' (2010) dental age estimations. The teeth sampled include the deciduous first molars and second molars, and the permanent premolars, first molars, and second molars (Table 3.1). Multiple teeth were utilized from some individuals to reflect a longer childhood time span. The bones sampled from the individuals were either a rib, clavicle, femur, or cranium (Table 3.1). Care was taken to avoid sampling elements showing notable taphonomic or pathological alterations as these processes could change the elemental composition of the bone (Katzenburg & Lovell, 1999; Tsutaya & Yoneda, 2015).

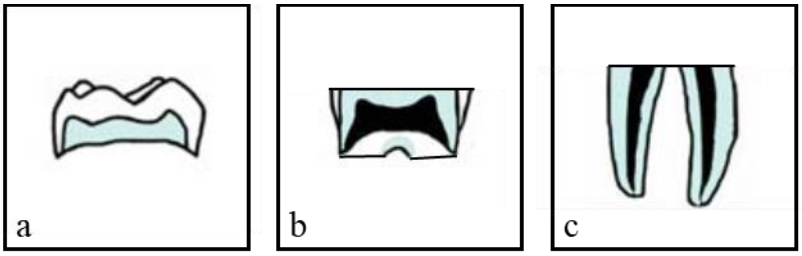

Figure 3.1: Locations of incremental sampling of a first molar. a) 3/4ths of the crown; b) 3/4ths of the crown to 1/4th of the root; c) 1/4th of the root to the apex (AlQahtani et al., 2010)

Table 3.1: Samples taken from the GCAS individuals

Context	Age	Teeth	Age(s) reflected by tooth dentin samples	Bone	Latest age range reflected by bone sample
Grave 2	30-39 years	LLM1 and URM2	2.5-9.5 and 5.5-14.5 years	Left rib	30-39 years
Grave 3	20-29 years	ULPM1 and URPM2	4.5-13.5 and 4.5-13.5 years	Left rib	20-29 years
Grave 6	25-34 years	URM1	2.5-8.5 years	Right clavicle	25-34 years
Grave 8	20-25 years	ULM1 and ULM2	2.5-8.5 and 5.5-14.5 years	Left rib	20-25 years
Grave 9	7-8 years	URM1 and LLM1	2.5-8.5 and 2.5-9.5 years	Left rib	7-8 years
Grave 10 Burial 1	6-8 years	ULm2 and LLM1	4.5 mos.-2.5years and 2.5-9.5 years	Left rib	6-8 years
Grave 10 Burial 1 Individual 1	Birth ± 1.5 mos	N/A	N/A	Right temporal	Birth ± 1.5 mos
Grave 10 Burial 1 Individual 2	Birth ± 1.5 mos	N/A	N/A	Right temporal	Birth ± 1.5 mos

Table 3.1 (continued)

Grave 11	18 mos ± 6 mos	LLm1 crown	10.5 mos.	Left femur	18 mos ± 6 mos
Grave 12	Birth ± 2 mos	N/A	N/A	Left femur	Birth ± 2 mos

The incremental dentine and whole bone samples were processed in the Laboratory for Archaeological Science and Technology (LAST) at the University of South Florida in Tampa, Florida, under the direction of Dr. Robert H. Tykot. The procedures followed for extracting collagen from bone and dentin were based on LAST's procedures, summarized as follows:

First, the enamel was removed from the sampled teeth to expose the dentin. The teeth were then cut vertically and then horizontally into respective sections (see Figure 3.1) using a 1/8th inch blade with a QEP 2500 tabletop wet saw. The sections were then weighed and it was determined that all of the tooth would have to be used to have a weight of 200 mg. Before extracting the collagen, the sectioned solid dentin and solid bone samples were ultrasonicated with water in a Fisher FS60 ultrasonicator to remove dirt and other adherent materials. The samples were then dried and approximately 200 mg from the individual sectioned dentin and at least 1 gram from the bone were extracted, weighed, and placed in 0.1 M sodium hydroxide (NaOH) for 24 hours to remove humic acids (Ambrose, 1990). The sodium hydroxide was poured off and the samples were rinsed with water. The samples were then demineralized in 2% hydrochloric acid (HCl) for six days. During the first, second, and fifth day, while replacing the hydrochloric acid, both the bone and dentin samples were cut with a scalpel into smaller pieces. After six days of acid treatment, the hydrochloric acid was poured off and the samples were rinsed three times with water and one time with distilled water, followed by a second 24-hour treatment of 0.1 M sodium hydroxide. Finally, after implementing the same pouring off processes as the HCl for the NaOH, the samples were placed in 2:1:0.8 defatting mixture of

methanol, chloroform, and distilled/deionized water for 24 hours then replaced with deionized water at least five times. They were then dried for at least 24 hours in vials and weighed. In addition, the bone and dentin samples were broken into smaller pieces and duplicate ~1 mg samples were placed in tin cups. Two samples were taken from each bone and sectioned dentin and averaged in order to control for variation within the sample. Any sample leftover was stored at LAST in case they needed to be run again or for future isotopic analysis.

After the samples were prepped, they were run through the Carlo-Erba NA2500 EA coupled with the Delta+XL isotope ratio mass spectrometer in the Paleolab at the University of South Florida. The Paleolab reported the carbon and nitrogen isotope values of collagen ($^{13}C/^{12}C$ and $^{15}N/^{14}N$) in percent per mil (‰) and compared them against Vienna Pee Dee Belemnite (VPDB) and Ambient Inhalable Reservoir (AIR) standards. The precision of the stable isotope ratio mass spectrometry analysis was ± 0.15‰ for $\delta^{13}C$ and ± 0.18‰ $\delta^{15}N$ (1 σ). Collagen yields and C:N ratios were used to test the reliability of the sample (Ambrose, 1990; DeNiro, 1985; Tykot, 2018). All the samples yielded more than 1% collagen and all C:N ratios fell between 2.9 and 3.6 which is the expected C:N ratio range for well-preserved collagen (Ambrose, 1990; DeNiro, 1985; Tykot, 2018). Additionally, the adult dentin values were compared to the subadult dentin values using a Wilcoxon rank-sum test, and the variation in $\delta^{15}N$ values at different ages of tissue formation, before 2 years, between 4 and 6 years and between 6 and 15 years of age in both individuals dying as subadults and adults were studied using a Levene's test.

Although great care was taken to not sample teeth with pathologies, two of the sampled teeth, the right maxillary second molar from the individual in Grave 2 and the left maxillary first molar from the individual in Grave 8, had caries. Pathologies, like caries, can change the chemical composition of the bone or dentin, changing the stable isotope values. Therefore, these

teeth were removed from any further analysis (Katzenberg & Lovell, 1999). In some cases, multiple teeth of the same tooth type (for example, left and right first molars) or multiple bone samples representing the same ages of tissue formation were sampled from individuals and in these cases the $\delta^{13}C$ and $\delta^{15}N$ values were averaged.

Radiographic and histological methods

Childhood periods of vitamin D deficiency were identified through radiography and histology of the teeth. Initial radiographs were taken of the permanent first molars of individuals in Graves 2 and 9 and Grave 10 burial 1. Buccal-lingual radiographs were taken using a standard dental x-ray in the Radiology Department at the School of Dental Medicine at East Carolina University at 40 to 42 pluses. Dimensions of the radiographed pulp chambers (pulp horn heights, widths, and the pulp chamber) and the pulp horn heights ratios were measured and calculated in ImageJ using the Straight-Line tool (see Figure 3.2) (D'Ortenzio et al., 2018b).

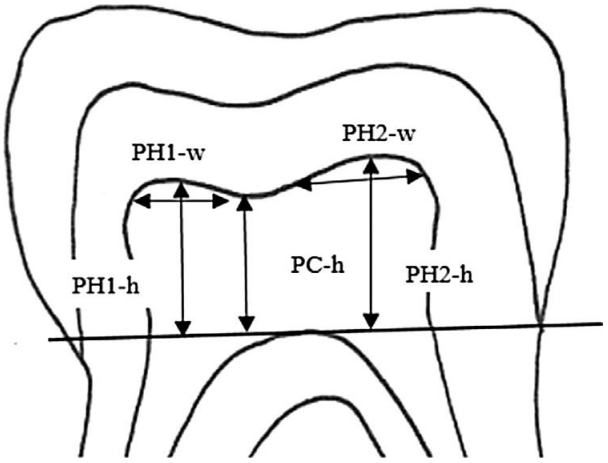

Figure 3.2: Diagram of a generic permanent molar and measurements taken in ImageJ. PH1-h and PH2-h are the height of left and right pulp horns. PH1-w and PH2-w are the width of the left and right pulp horns. PC-h is the pulp chamber height (D'Ortenzio et al., 2018b)

After radiographic analysis, the first right molars from Grave 2, Grave 9, and Grave 10 burial 1 were selected for thin sectioning and microscopic identification of interglobular dentin

(IGD). Following similar procedures found in Saunders and colleagues' (2007) and D'Ortenzio and colleagues' (2016) work, the teeth were embedded in a 2.22:1 mixture by weight of Buehler EpoThin™ 2 Epoxy resin and Buehler EpoThin™ 2 Epoxy hardener in silicone ice trays. After 24 hours, the samples were adhered using yellow dental sticky wax on their buccal sides to a chuck and sectioned along the mesial-distal plane from the buccal cusps to the root apices using a Buehler Isomet slow-speed saw with a 0.3mm thick diamond wafering blade. The exposed section was polished using a sequence of 400, 600, 800, and 1200 μm grit paper then glued onto a slide using UV resin. The second cut was made 0.8 mm from the first to create a 200 to 300 μm section. The section was lapped using the same grits as above down to a 100 μm thickness and then polished using Buehler 1.0 micron polisher and a microcloth pad.

The dentine of the thin sections was observed for IGD using a Laxco LMC4-PL27-035184 polarizing microscope. Pictures were taken with a Moticam X^2 at 4X magnification using the application Motic Images Plus. Any IGD present was scored by severity following D'Ortenzio and colleagues' (2016) scale: Grade 0, no interglobular dentin is seen and only normal dentin is present, Grade 1, the amount of interglobular dentin is less than 25% in relation to the surrounding normal dentin, Grade 2 is 50%, and Grade 3 is 75% or more interglobular dentin (see Figure 3.3). The age at which the individuals experienced vitamin D deficiency was estimated based on knowledge of dentin formation timing compiled and augmented by Brickley and colleagues (2019; Figure 3.4).

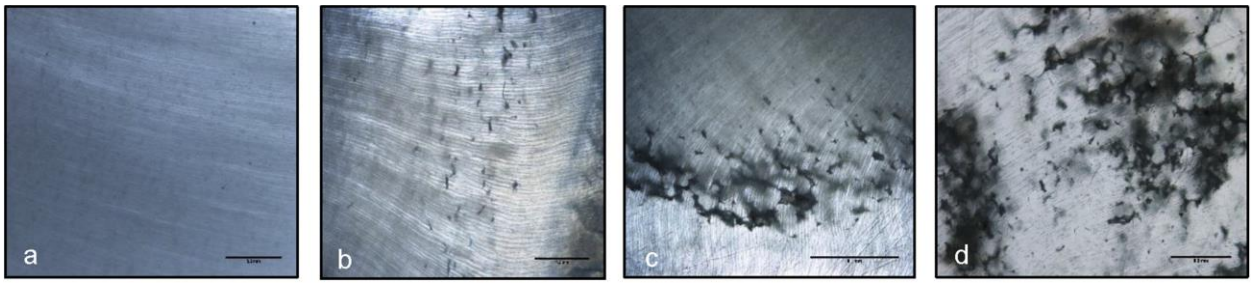

Figure 3.3: Pictures and examples of IGD and Grade. a) Grade 0; b) Grade 1; c) Grade 2; d) Grade 3 (D'Ortentzio et al., 2016)

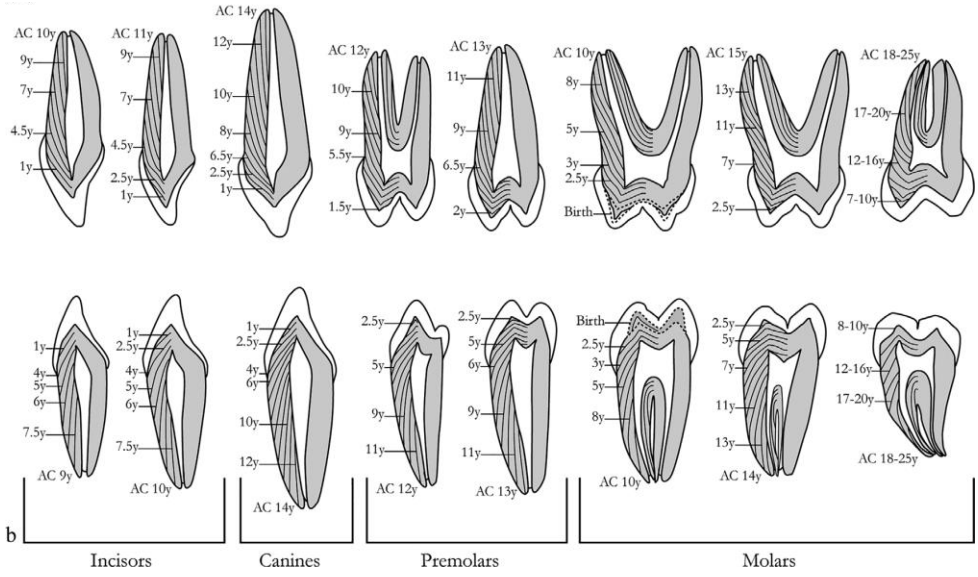

Figure 3.4: Diagram of the permanent teeth and the approximate ages in which the dentin mineralizes (Brickley et al., 2019)

CHAPTER FOUR:

RESULTS

Stable isotope analysis

The overall bone and dentin $\delta^{13}C$ ranged from -15.6‰ to -9.5‰ with an average of -13.5‰ (±1.4). In addition, the bone and dentin $\delta^{15}N$ ranged from 10.5‰ to 14.2‰ with a mean of 12.1‰ (± 0.9) (Table 4.1). The majority of these samples (73%) were from teeth, so it is not surprising that the mean and standard deviation of the dentin values were close to the same values of the overall sample ($\delta^{13}C$ mean=-13.3±1.5‰, $\delta^{15}N$ mean=12.1±0.9‰). The bone carbon values ($\delta^{13}C$ mean=-14.0±0.9‰) were slightly higher than the mean dentin value, but the bone nitrogen isotope values only varied from the dentin value by 0.1% ($\delta^{15}N$ mean=12.2±0.8‰).

The bone $\delta^{13}C$ values (mean=-13.9±1.1‰) of the adults (>20 years of age) in the sample do not differ significantly from that of children dying between 6 and 8 years of age (mean=-13.8±0.8‰) or infants between 0 and 1.5 years of age (mean=-14.1±0.8‰). However, the $\delta^{15}N$ values of adult bone (mean=11.9±0.2‰) and children between 6 and 8 years of age (mean=11.4±0.2‰) are notably lower than the bone values of infants dying between 0 and 1.5 years old (mean=12.9±0.8‰). The mean infant bone $\delta^{13}C$ values differ only slightly from the overall adult bone mean, while the $\delta^{15}N$ values are 1‰ higher.

Table 4.1: GCAS skeletal samples' $\delta^{13}C$ and $\delta^{15}N$ values

USF sample #	**Context**	**Sample**	**Sample type**	**Age at death of individual**	**Age of Tissue Formation**	**$\delta^{13}C$**	**$\delta^{15}N$**
38159a	Grave 2	LLM1 crown	dentin	30-39 years	2.5	-13.7	12.2
38159a	Grave 2	LLM1 crown	dentin	30-39 years	2.5	-13.4	14.2
38159b	Grave 2	LLM1 middle	dentin	30-39 years	4.5	-13.6	12.0
38159b	Grave 2	LLM1 middle	dentin	30-39 years	4.5	-13.0	11.5
38159c	Grave 2	LLM1 root	dentin	30-39 years	9.5	-14.0	12.1
38159c	Grave 2	LLM1 root	dentin	30-39 years	9.5	-13.1	11.8
3161	Grave 2	L ribs	bone	30-39 years	30-39	-13.3	11.7
38161	Grave 2	L ribs	bone	30-39 years	30-39	-14.3	11.8
38164a	Grave 3	ULPM1 crown	dentin	20-29 years	4.5	-15.4	12.5
38164a	Grave 3	ULPM1 crown	dentin	20-29 years	4.5	-15.4	13.1
38164b	Grave 3	ULPM1 middle	dentin	20-29 years	7.5	-14.1	12.4
38164b	Grave 3	ULPM1 middle	dentin	20-29 years	7.5	-15.4	12.4
38164b	Grave 3	ULPM1 root	dentin	20-29 years	13.5	-15.5	12.3
38164c	Grave 3	ULPM1 root	dentin	20-29 years	13.5	-15.6	12.5
38164c	Grave 3	URPM2 crown	dentin	20-29 years	4.5	-15.3	13.2
38165a	Grave 3	URPM2 crown	dentin	20-29 years	4.5	-15.4	12.6
38165a	Grave 3	URPM2 middle	dentin	20-29 years	7.5	-14.6	13.0
38165b	Grave 3	URPM2 middle	dentin	20-29 years	7.5	-14.7	12.2
38165c	Grave 3	URPM2 root	dentin	20-29 years	13.5	-14.4	12.2

Table 4.1 (continued)

38165c	Grave 3	URPM2 root	dentin	20-29 years	13.5	-13.4	12.1
38166	Grave 3	L ribs	bone	20-29 years	20-29	-13.5	11.7
38166	Grave 3	L ribs	bone	20-29 years	20-29	-14.0	11.7
38162a	Grave 6	URM1 crown	dentin	25-34 years	2.5	-12.4	11.3
38162a	Grave 6	URM1 crown	dentin	25-34 years	2.5	-12.2	11.3
38162b	Grave 6	URM1 middle	dentin	25-34 years	3.5-4.5	-12.8	12.4
38162b	Grave 6	URM1 middle	dentin	25-34 years	3.5-4.5	-12.9	12.3
38162c	Grave 6	URM1 root	dentin	25-34 years	8.5-9.5	-13.2	12.8
38162c	Grave 6	URM1 root	dentin	25-34 years	8.5-9.5	-13.0	12.7
38163	Grave 6	R clavicle	bone	25-34 years	25-34	-12.4	11.7
38163	Grave 6	R clavicle	bone	25-34 years	25-34	-12.8	12.0
38168a	Grave 8	ULM2 crown	dentin	20-25 years	5.5	-15.1	11.4
38168a	Grave 8	ULM2 crown	dentin	20-25 years	5.5	-15.5	11.7
38168b	Grave 8	ULM2 middle	dentin	20-25 years	7.5	-14.9	12.1
38168b	Grave 8	ULM2 middle	dentin	20-25 years	7.5	-14.8	12.2
38168c	Grave 8	ULM2 root	dentin	20-25 years	14.5	-15.5	12.8
38168c	Grave 8	ULM2 root	dentin	20-25 years	14.5	-14.3	12.8
38169	Grave 8	L ribs	bone	20-25 years	20-25	-15.6	12.0
38169	Grave 8	L ribs	bone	20-25 years	20-25	-15.3	12.2
38155a	Grave 9	URM1 crown	dentin	7-8 years	2.5	-11.7	10.8
38155a	Grave 9	URM1 crown	dentin	7-8 years	2.5	-11.3	14.1
38155b	Grave 9	URM1 middle	dentin	7-8 years	3.5-4.5	-13.1	11.3
38155b	Grave 9	URM1 middle	dentin	7-8 years	3.5-4.5	-12.4	11.4
38155c	Grave 9	URM1 root	dentin	7-8 years	8.5-9.5	-14.4	11.7
38155c	Grave 9	URM1 root	dentin	7-8 years	8.5-9.5	-14.9	12.3
38156a	Grave 9	LLM1 crown	dentin	7-8 years	2.5	-12.1	11.1
38156a	Grave 9	LLM1 crown	dentin	7-8 years	2.5	-11.5	13.6
38156b	Grave 9	LLM1 middle	dentin	7-8 years	4.5	-12.4	11.1
38156b	Grave 9	LLM1 middle	dentin	7-8 years	4.5	-12.2	11.2

Table 4.1 (continued)

38156c	Grave 9	LLM1 root	dentin	7-8 years	9.5	-14.0	11.7
38156c	Grave 9	LLM1 root	dentin	7-8 years	9.5	-13.8	11.5
38157	Grave 9	L ribs	bone	7-8 years	7-8	-14.8	11.6
38157	Grave 9	L ribs	bone	7-8 years	7-8	-14.2	11.3
38153a	Grave 10 B1	LLM1 crown	dentin	6-8 years	2.5	-12.3	10.5
38153a	Grave 10 B1	LLM1 crown	dentin	6-8 years	2.5	-12.2	10.7
38153b	Grave 10 B1	LLM1 middle	dentin	6-8 years	4.5	-11.2	11.6
38153b	Grave 10 B1	LLM1 middle	dentin	6-8 years	4.5	-11.6	11.5
38153c	Grave 10 B1	LLM1 root	dentin	6-8 years	9.5	-12.7	12.0
38153c	Grave 10 B1	LLM1 root	dentin	6-8 years	9.5	-12.1	11.2
38158a	Grave 10 B1	ULm2 crown	dentin	6-8 years	4.5 mos.	-11.2	12.2
38158a	Grave 10 B1	ULm2 crown	dentin	6-8 years	4.5 mos.	-9.5	13.1
38158b	Grave 10 B1	ULm2 middle	dentin	6-8 years	1.5	-10.3	13.0
38158b	Grave 10 B1	ULm2 middle	dentin	6-8 years	1.5	-12.6	10.8
38158c	Grave 10 B1	ULm2 root	dentin	6-8 years	2.5	-12.2	10.5
38158c	Grave 10 B1	ULm2 root	dentin	6-8 years	2.5	-12.2	10.7
38154	Grave 10 B1	L ribs	bone	6-8 years	6-8	-13.4	11.4
38154	Grave 10 B1	L ribs	bone	6-8 years	6-8	-12.9	11.2
38170	Grave 10 B2 Ind1	R temporal	bone	newborn	0	-12.9	12.1
38170	Grave 10 B2 Ind1	R temporal	bone	newborn	0	-13.1	12.3
38171	Grave 10 B2 Ind2	R temporal	bone	newborn	0	-14.0	12.9
38171	Grave 10 B2 Ind2	R temporal	bone	newborn	0	-14.3	12.9
38172	Grave 11	LLm1 crown	dentin	18 mos +/- 6 mos	10.5 mos.	-12.0	13.8
38172	Grave 11	LLm1 crown	dentin	18 mos +/- 6 mos	10.5 mos.	-11.8	13.9
38173	Grave 11	L Femur	bone	18 mos +/- 6 mos	18 mos +/- 6 mos	-14.3	14.1

Table 4.1 (continued)

38173	Grave 11	L Femur	bone	18 mos +/- 6 mos	18 mos +/- 6 mos	-14.1	14.1
38174	Grave 12	L Femur	bone	birth +/- 2 mos	birth +/- 2 mos	-15.1	12.3
38174	Grave 12	L Femur	bone	birth +/- 2 mos	birth +/- 2 mos	-15.2	12.3

` Plotting isotope values by age of tissue formation (Figure 4.1) shows the $\delta^{15}N$ dentin values decline between 10.5 months and 1.5 years of age before flattening out, and a similar decline is seen in the $\delta^{13}C$ dentine values, but at 4.5 months. This graph also shows that the pattern of dietary changes in childhood varies by tissue type (Figure 4.1). The $\delta^{15}N$ bone values do not decline until 1.5 years of age, unlike the $\delta^{15}N$ dentin values. Similarly, the $\delta^{13}C$ bone values are lower than the $\delta^{13}C$ dentine values and rise between 1.5 and 6.5 years of age while the $\delta^{13}C$ dentine values decline.

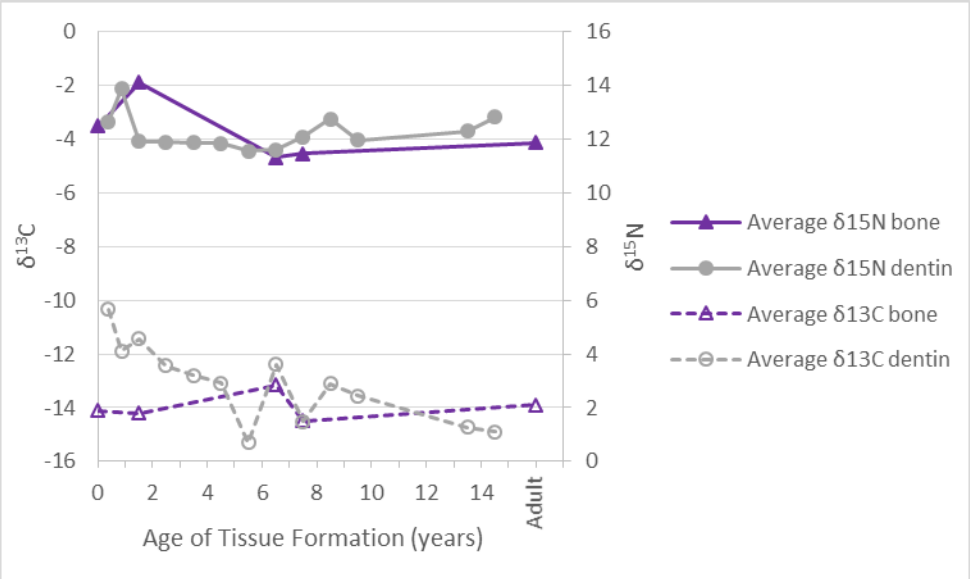

Figure 4.1: Mean isotope values by tissue type and age of formation

Recent studies comparing $\delta^{13}C$ and $\delta^{15}N$ in different tissues co-forming in the same individual have found these values may not be directly comparable (Beaumont et al., 2018). While the studies comparing $\delta^{13}C$ in co-forming tissues showed less of a difference, the divergence in $\delta^{15}N$ seems to stem from the differential impact of physiological stress on the growth of these tissues (Beaumont et al., 2018). Only two cases of co-forming tissues from a

single individual occur in this sample: bone and the first molar root dentin samples from Grave 9, a child dying between 6 and 7 years, and from Grave 10 Burial 1 Individual 1, a child dying between 6 and 8 years of age. In both cases, the bone samples have isotope values slightly lower than the co-forming dentine, although the extent of this difference varies by individual, as discussed further in the individual-level analyses below.

Infants (Grave 10 Burial 2 Individuals 1 and 2, Grave 11, and Grave 12)

Four individuals in the sample died during infancy, three of who, Grave 10 Burial 2 Individuals 1 and 2 and Grave 12, were neonates. These neonates were poorly preserved and very little to no dental remains were discovered. Therefore, they are represented only by bone samples forming during the period immediately before death. On the other hand, the 18-month-old (±6 months) individual in Grave 11 was represented by dentine from a lower left deciduous first molar forming during the period around birth and a bone sample representing the period before death at 18 months (± 6 months).

The newborns show slight variation in $\delta^{13}C$ bone values (±0.8‰) (see Figure 4.2), paralleling the slight variation in the amount of C_3 versus C_4 plants consumed seen in the general adult sample (±1.1‰). The 18-month-old's (± 6 months) diet in Grave 11 is similar to the newborns' based on their $\delta^{13}C$ values. However, this infant has slightly higher $\delta^{15}N$ dentin and bone values than the $\delta^{15}N$ bone values of the newborns and the adult $\delta^{15}N$ bone average, which reflects the expected 2-3‰ offset from their mother's values (DeNiro & Epstein, 1981; Fogel et al., 1989), assuming the mother of this infant had a diet similar to the adults in this sample. Although slightly higher than the adult average, the other individuals, Grave 12 and Grave 10 Burial 2 Individuals 1 and 2, did not show the expected offset.

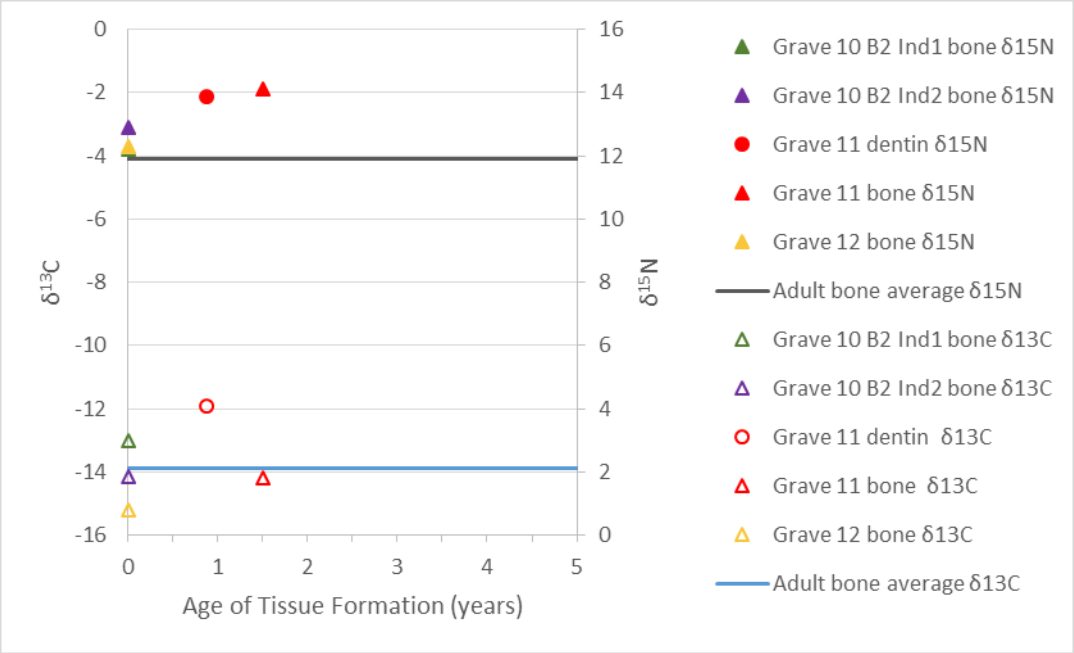

Figure 4.2: Results of $\delta^{15}N$ and $\delta^{13}C$ stable isotopes in relation to age of tissue formation from the individuals in Grave 10 Burial 2 Individuals 1 and 2, Grave 11, and Grave 12

Grave 2

The $\delta^{13}C$ and $\delta^{15}N$ values from the mandibular first molar of the 30 to 39-year-old male individual in Grave 2 are plotted in Figure 4.3. The $\delta^{13}C$ values of dentin do not shift dramatically between the represented ages of 2.5, 4.5, or 9.5 years of age nor differ much from their bone values or the adult $\delta^{13}C$ bone average. From 2.5 to 4.5 years, $\delta^{15}N$ dentin values decrease by 1.5‰ to level off near the adult bone average. This shift in $\delta^{15}N$ values between 2.5 and 4.5 years could represent the end of weaning and the stabilization to adult nitrogen values. When considering the differences between the dentin values formed at 9.5 years and the adult bone values, there is only a 0.3‰ difference for $\delta^{13}C$ and a 0.2‰ difference for $\delta^{15}N$. Although these tissues do not co-form, it is expected that the post-weaning dentin values should be similar

to the average adult diet. These slight differences seen may reflect a difference in tissue type, which will be expanded upon in the discussion.

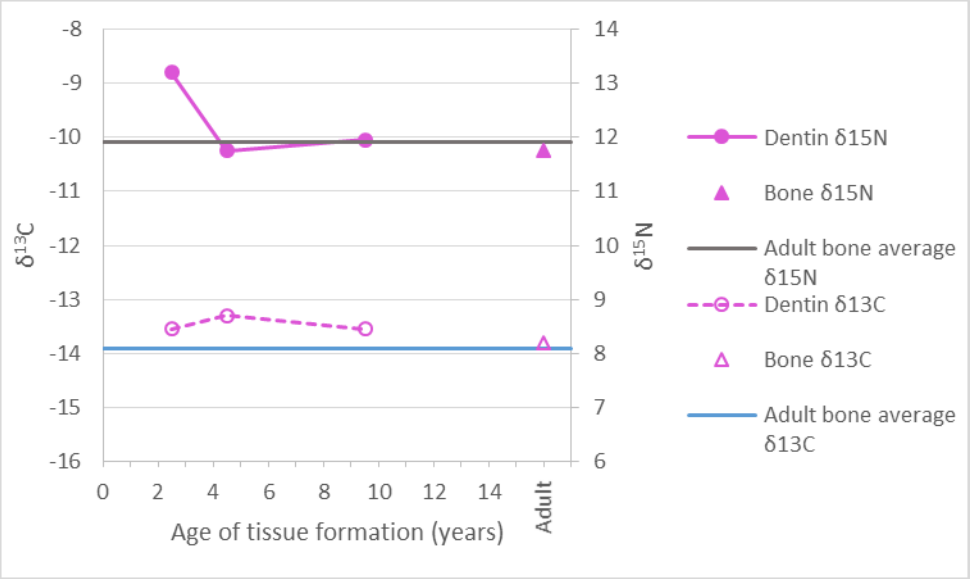

Figure 4.3: Results of $\delta^{15}N$ and $\delta^{13}C$ stable isotopes in relation to age of tissue formation from the 30 to 39-year-old adult male in Grave 2

Grave 3

The averaged $\delta^{15}N$ and $\delta^{13}C$ values from the maxillary first and second premolars representing 4.5, 7.5, and 13.5 years of age in the 20 to 29-year-old female from Grave 3 demonstrates a pattern similar to the male in Grave 2, but at a different magnitude of change (Figure 4.4). The $\delta^{13}C$ values in dentin see a 1.0‰ increase between 4.5 and 7.5 years but then stabilize through 13.5 years, and the $\delta^{15}N$ declines very slightly during these ages. There is almost a 1‰ increase of $\delta^{13}C$ between the last available dentin sample forming at 13.5 years of age and the bone sample representing the years before death about 10 years later. Similarly, there is a decline in $\delta^{15}N$ between the last dentine and the adult bone sample.

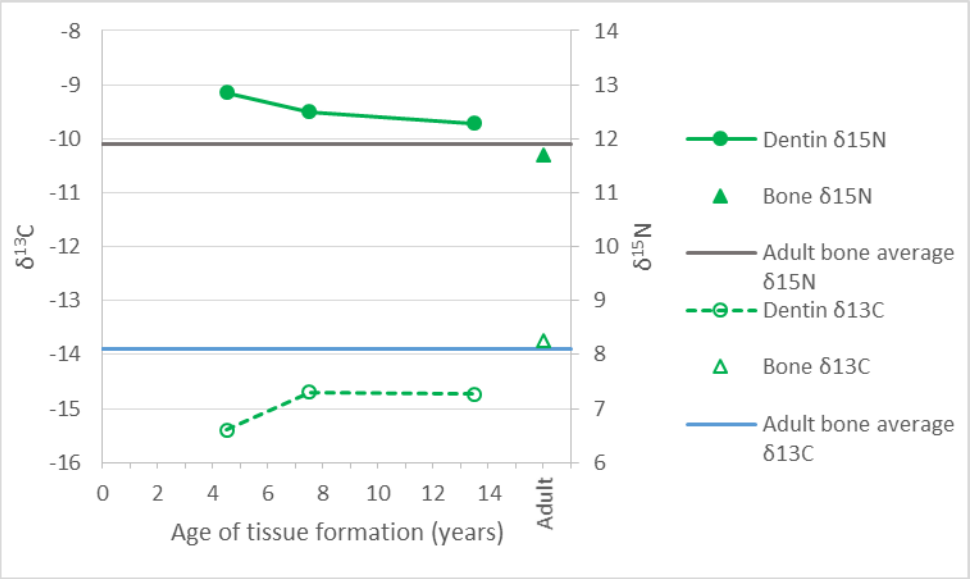

Figure 4.4: Results of $\delta^{15}N$ and $\delta^{13}C$ stable isotopes in relation to age of tissue formation from the 20 to 29-year-old female in Grave 3

Grave 6

Figure 4.5 displays the $\delta^{15}N$ and $\delta^{13}C$ values from the maxillary first molar representing 2.5, 3.5, and 8.5 years of age and the bone of the 25 to 34-year-old female in Grave 6. In this case, the $\delta^{15}N$ values increase by 1‰ between 2.5 and 3.5 years of age and then increase another 0.5‰ between 3.5 and 8.5, while the $\delta^{13}C$ values decrease about 1‰ between 2.5 and 8.5 years of age. Similar to the female in Grave 3, the adult bone values representing the years before death are slightly less enriched in $\delta^{15}N$ and more enriched in $\delta^{13}C$ than the latest childhood dentine values, in this case from 8.5 years of age.

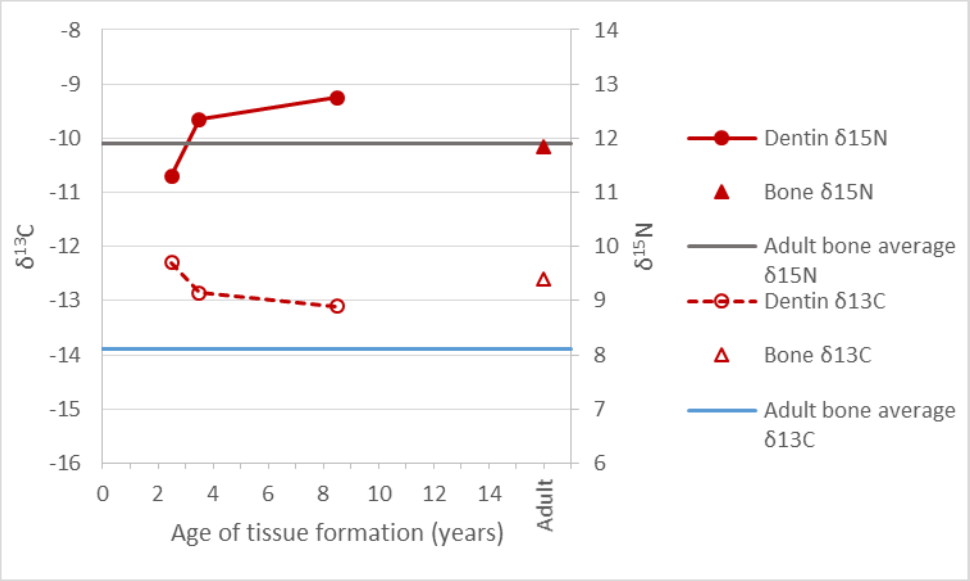

Figure 4.5: Results of $\delta^{15}N$ and $\delta^{13}C$ stable isotopes in relation to age of tissue formation from the 25 to 34-year-old female in Grave 6

Grave 8

The $\delta^{13}C$ and $\delta^{15}N$ values from the 20 to 25-year-old male in Grave 8 are plotted in Figure 4.6. The $\delta^{13}C$ dentin values from the maxillary second molar increase slightly (0.5‰) between 5.5 and 7.5 years, and then level off between 7.5 and 14.5 years. However, the adult bone value from the years before death is about 0.5‰ more negative than the latest childhood dentin values, forming only 5 to 10 years earlier. The $\delta^{15}N$ dentin values increase by 1.3‰ between 5.5 to 14.5 years, similar to the pattern seen in Grave 6. The oldest dentin value is almost 1.0‰ higher than the bone value representing the years before death.

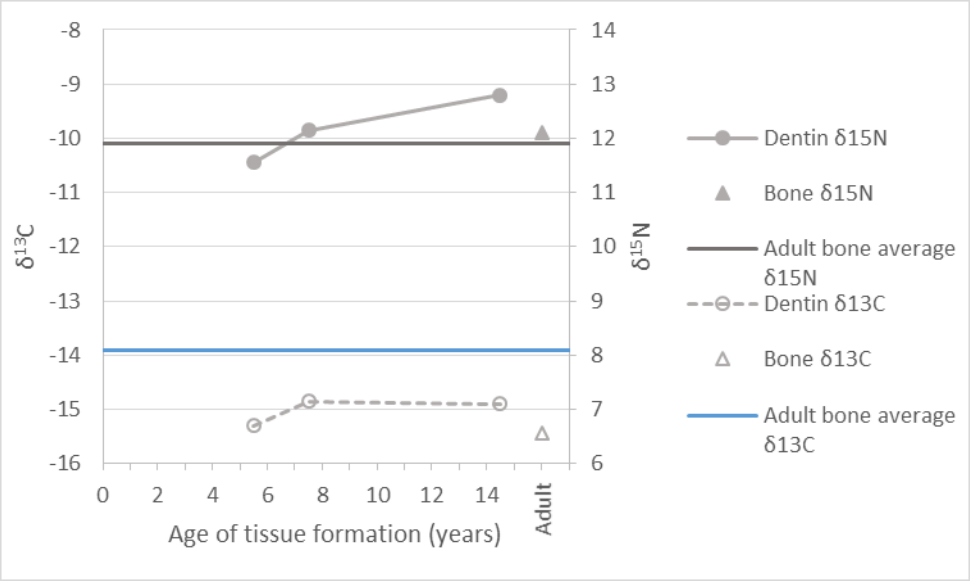

Figure 4.6: Results of $\delta^{15}N$ and $\delta^{13}C$ stable isotopes in relation to age of tissue formation from the 20 to 25-year-old male in Grave 8

Grave 9

The averages of the $\delta^{15}N$ and $\delta^{13}C$ values from the maxillary and mandibular first molars' dentin of the 7 to 8-year-old child from Grave 9 are graphed in Figure 4.7. The $\delta^{15}N$ values decrease almost 1‰ between the ages of 2.5 and 4.5 years before increasing very slightly between 4.5 and 7.5 years of age. The decrease observed between 2.5 and 4.5 could represent the end of weaning and the stabilization of the $\delta^{15}N$ values. As noted above, the co-forming dentin and bone $\delta^{15}N$ values only slightly differ. The $\delta^{13}C$ values decrease between 2.5 and 3.5 years and then increase between 3.5 and 4.5 years before a decrease of 2‰. The co-forming bone and dentin from the subadult in the period before death show very similar $\delta^{13}C$ values.

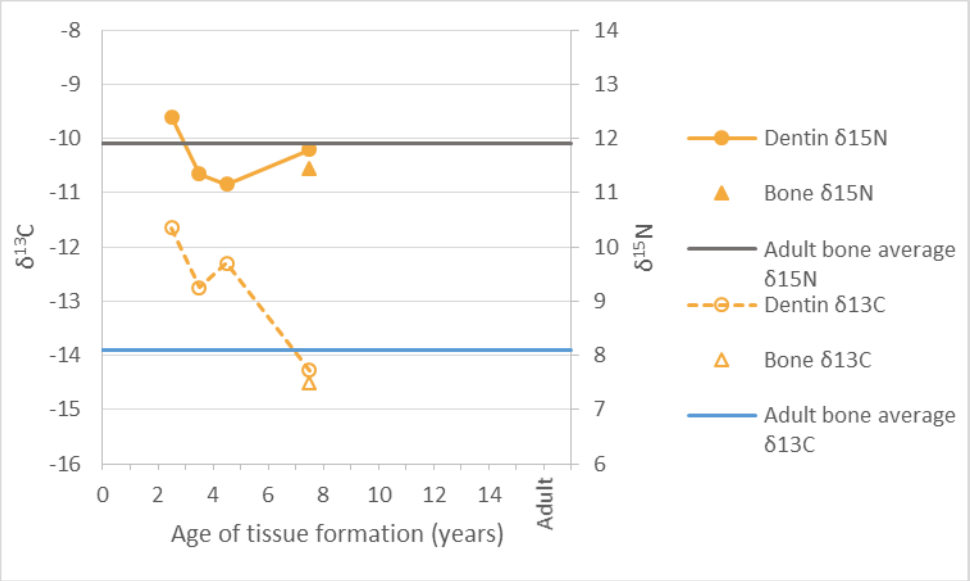

Figure 4.7: Results of $\delta^{15}N$ and $\delta^{13}C$ stable isotopes in relation to age of tissue formation from the 7 to 8-year-old in Grave 9

Grave 10 Burial 1

The $\delta^{15}N$ and $\delta^{13}C$ values from the 6 to 8-year-old in Grave 10 Burial 1 are graphed in Figure 4.8. The dentin $\delta^{15}N$ values from the deciduous maxillary second molar and permanent mandibular first molar decline noticeably by over 2.1‰ from 4.5 months to 2.5 years of age, which was matched by a decline of over 1.9‰ in $\delta^{13}C$. From 2.5 to 6.5 years, ^{15}N dentine became increasingly enriched, resulting in an over 1‰ increase in $\delta^{15}N$. Unlike $\delta^{15}N$, $\delta^{13}C$ values follow a similar pattern to Grave 9 in which dentin values decrease from 4.5 months to 2.5 years, then increase between 2.5 and 4.5 years, then decrease again until the period before death at around 6.5 years. The co-forming bone and dentin in Grave 10 Burial 1, unlike Grave 9, show slightly less similar nitrogen and carbon isotope values. Additionally, the co-forming bone and dentin carbon isotope values are slightly higher than the average adult bone carbon value, while the individual's nitrogen values are slightly lower.

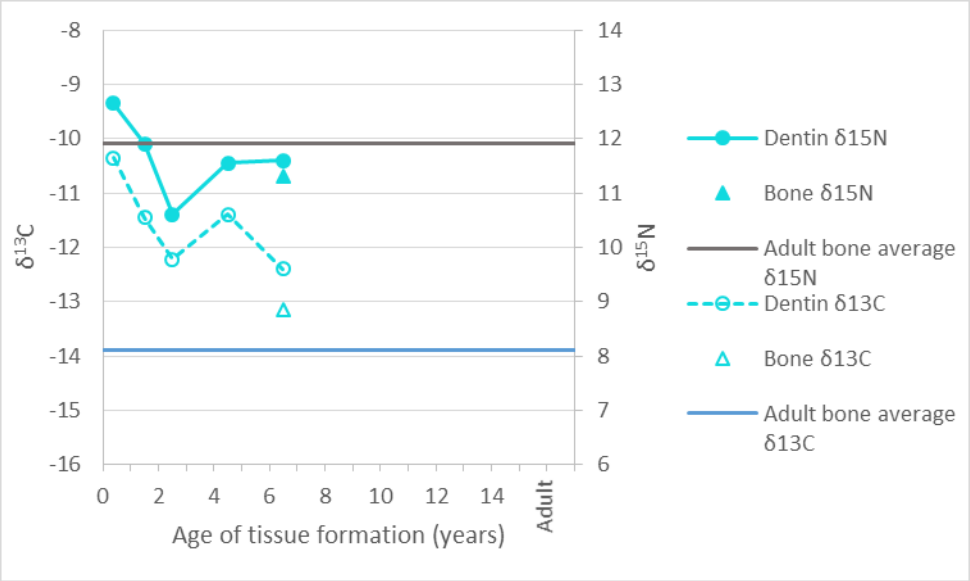

Figure 4.8: Results of $\delta^{15}N$ and $\delta^{13}C$ stable isotopes in relation to age of tissue formation from the 6 to 8-year-old in Grave 10 Burial 1

Adults and Subadults

Some differences in $\delta^{13}C$ and $\delta^{15}N$ between individuals dying as adults versus those dying in infancy or childhood may identify not only differences between adult and childhood diets, but also between diets of those more or less susceptible to childhood mortality. Although there is some overlap of the standard deviations, those dying as infants and children have higher $\delta^{13}C$ dentine values than those dying as adults (Figure 4.9). This can also be seen when assessing different ages of dentin formation, which is presented in Table 4.2. A Wilcoxon rank-sum test confirmed the significance of this difference not only in tissues forming during the perinatal and weaning period before 2 years of age ($Z=-2.279$, $p=0.023$) but also between 4 and 6 years of age ($Z=2.279$, $p=0.023$). The $\delta^{15}N$ ratios in those dying as children versus those living into adulthood does not differ significantly in the younger age categories but those dying as children have lower $\delta^{15}N$ values in tissues forming between 6 and 15 years of age than those dying later in life ($Z=-2.354$, $p=0.019$). However, only three samples from two children represent tissues forming

during this age range, and the dentin was forming just before death. Indeed, the relatively small samples sizes here indicate these results should be taken with caution.

The individual-level analysis of isotope ratios by age also indicates a generally consistent diversity and pattern of change in diet within this group throughout childhood. Although the age in which weaning finishes is rather homogeneous, the $\delta^{15}N$ values vary more or less at different ages (Table 4.2). Both individuals dying as adults and subadults display higher diversity in their $\delta^{15}N$ ratios in tissues forming before 2 years of age than the other age categories (Levene's F 7.572, p=0.002). This level of variation decreases in tissues forming between 4 and 6 years of age and is least in tissues forming between 6 and 15 years of age.

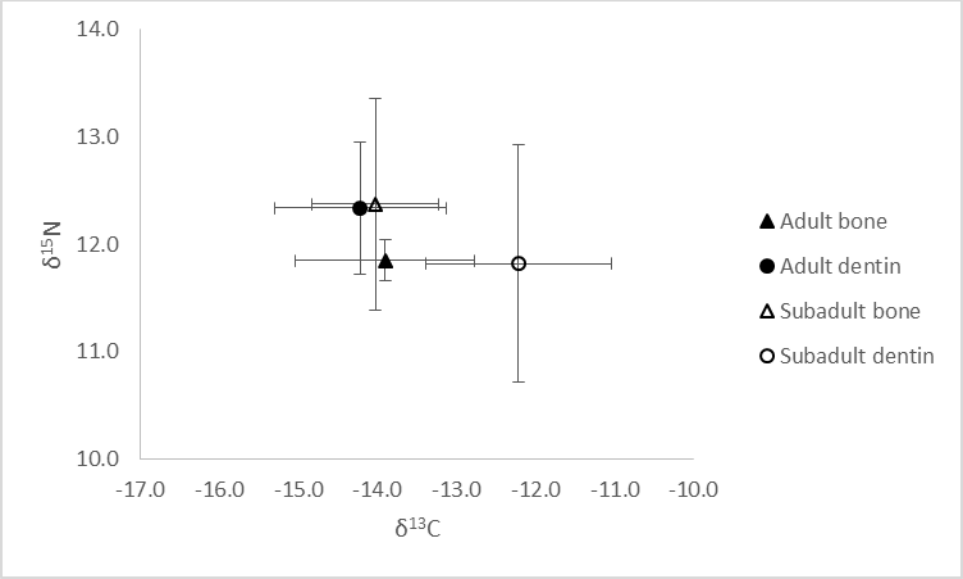

Figure 4.9: Comparison of $\delta^{15}N$ to $\delta^{13}C$ in bone and dentin between individuals dying as children versus those dying in adulthood in the GCAS skeletal sample

Table 4.2: Carbon and nitrogen isotope values from different ages of dentin formation in those dying as children and those dying as adults at GCAS

	Children		Adults	
Age of dentin formation	$\delta^{13}C$	$\delta^{15}N$	$\delta^{13}C$	$\delta^{15}N$
<2 years	-11.7 ±0.6‰	12.1 ±1.2‰	-13.5 ±1.1‰	12.6 ±1.1‰
4-6 years	-12.2 ±0.7‰	11.4 ±0.2‰	-14.4 ±1.1‰	12.1 ±0.7‰
6-15 years	-13.7 ±1.1‰	11.7 ±0.2‰	-14.5 ±1.1‰	12.3 ±0.3‰

Non-Specific physiological stress and stable isotopes

The high percentage of dental enamel hypoplasias (DEH) and other indicators of childhood stress also indicated that childhood was a period of high morbidity in the Gause family. Weaning practices and childhood diet may have been one factor that led to a stressful childhood. While previous studies have found difficulties with linking the presence and timing of non-specific indicators of stress to weaning (Blakey et al., 1994; Larson, 2015; Lewis, 2007; Wood, 1996), these patterns may illuminate ages at which children suffered from and survived stress. Within the Gause sample, 39 out of 71 teeth (excepting all 3rd molars) showed at least one DEH, and these were seen in every individual except those dying before 2 years of age (Long, 2019; Quintana, 2019). The overall observed DEH occurred between approximately 1 and 6 years of age (Long, 2019; Quintana, 2019). Figures 4.10, 4.11, 4.12, 4.13, 4.14, and 4.15 depict time spans of often repetitive DEH formation, shown by the blue boxes, within the context of each individual's stable isotope results to determine whether the DEH coincide with systematic

stress during the weaning process, post-weaning stress, or neither (Beaumont et al., 2018; Katzenberg & Lovell, 1999; Sandberg et al., 2014).

Grave 2

The 30 to 39-year-old male from Grave 2 had multiple dental enamel hypoplasias on 15 out of the 30 teeth present (Quintana, 2019). The ages in which these DEH occurred are between the ages 2 and 6 years (Quintana, 2019). The isotopes values show this is a period possibly reflecting the later stages of weaning and/or a shift from childhood diet to an adult diet (Figure 4.10).

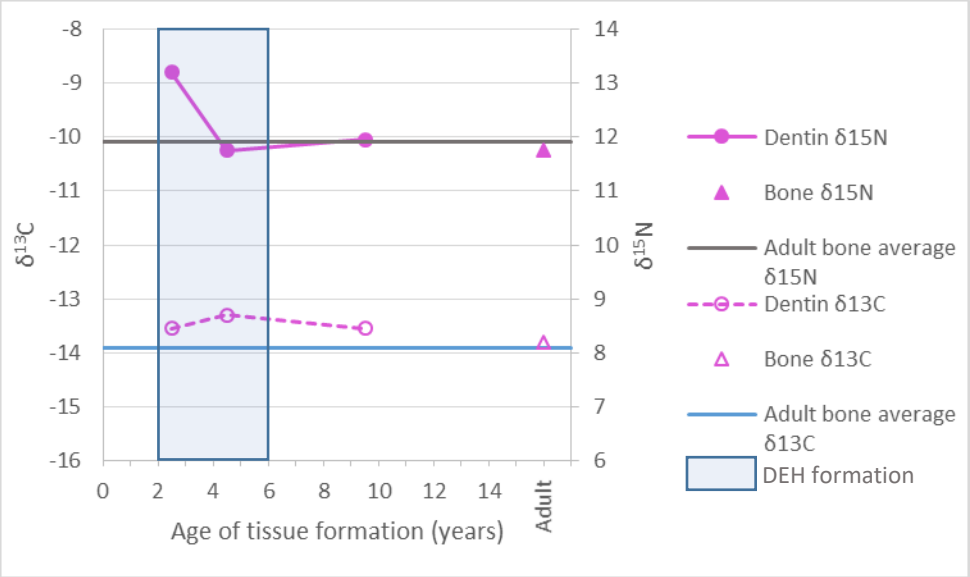

Figure 4.10: Results of $\delta^{15}N$ and $\delta^{13}C$ stable isotopes in relation to age of tissue formation compared to DEH formation from the 30 to 39-year-old adult male in Grave 2

Grave 3

The 20 to 29-year-old female from Grave 3 has a total of 13 dental enamel hypoplasias, which were observed on 6 of the 17 teeth present (Long, 2019). The earliest observed DEH occurs at 3.6 years and the latest observed DEH occurs at 5.7 years on the mandibular first

premolars (Long, 2019). The incisors, the crowns of which form starting at approximately 6 months of age until 4.5 years of age (AlQahtani et al., 2010), do not show any sign of DEH and thus the lack of DEH formation before 3.6 years is not a factor of sampling bias. Assessing this in light of the isotope data, DEH in this individual is not linked to weaning-related stress, but to issues faced in later childhood (Figure 4.11).

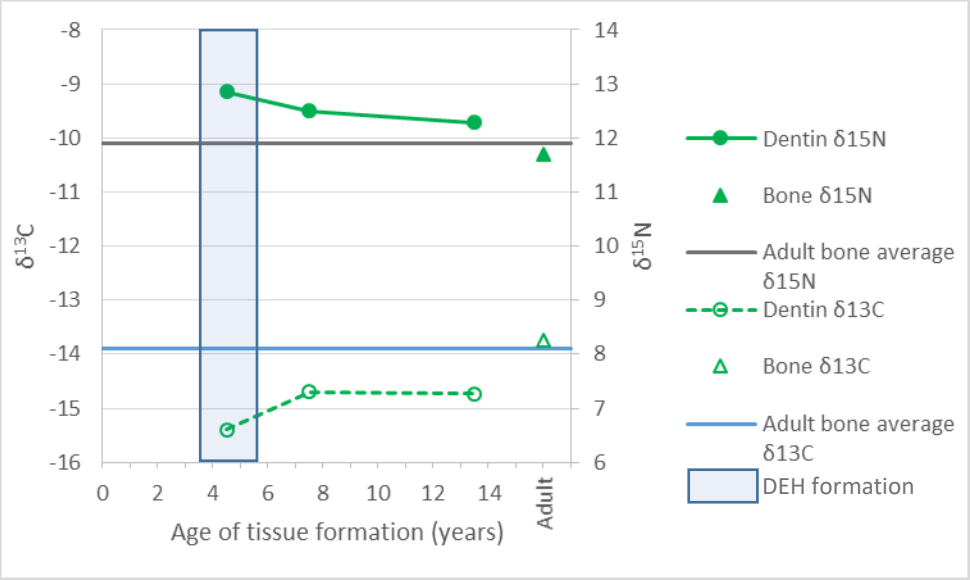

Figure 4.11: Results of $\delta^{15}N$ and $\delta^{13}C$ stable isotopes in relation to age of tissue formation compared to DEH formation from the 20 to 29-year-old female in Grave 3

Grave 6

Multiple dental enamel hypoplasias were observed on the left mandibular first incisor, right mandibular canine, left maxillary canine, and right maxillary first premolar of the 25 to 34-year-old individual from Grave 6 that have ages of formation between 1 and 6 years of age (Quintana, 2019). Teeth forming later, like the second premolars and first molar, were observable (Quintana, 2019) but showed no DEH which means the lack of DEH after 6 years of age is not due to sampling bias. Although there is a subtle increase in $\delta^{15}N$ values and decrease in $\delta^{13}C$ between the ages of DEH formation, this increase is most likely due to changes in diet from

the addition of freshwater animals or forestry animals rather than stress (Figure 4.12) (Beaumont & Montgomery, 2016). Thus, the DEH does not coincide with any nutritional or physiological stress that would have been expressed in the $\delta^{15}N$ and $\delta^{13}C$ values.

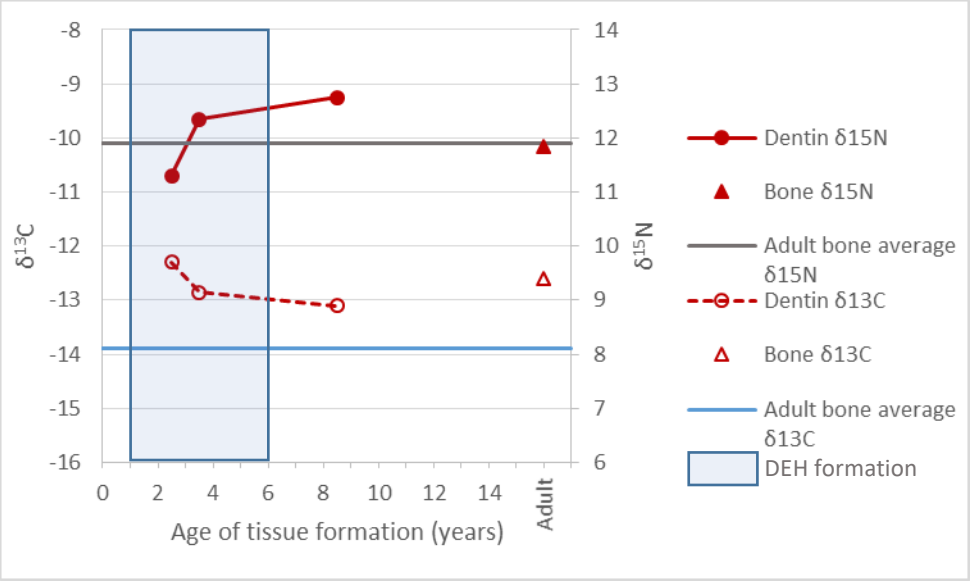

Figure 4.12: Results of $\delta^{15}N$ and $\delta^{13}C$ stable isotopes in relation to age of tissue formation compared to DEH formation from the 25 to 34-year-old female in Grave 6

Grave 8

The 20 to 25-year-old individual from Grave 8 had multiple DEH on 4 out of the 21 present teeth (Quintana, 2019) that represent successive, repetitive periods of stress between 1 to 5 years of age (Quintana, 2019). The premolars representing later periods of childhood, including the ages from which we have isotope data, were present but show no observable DEH (Figure 4.13). The incremental dentin samples for this individual do not coincide with the likely age that weaning occurred, but the ages of DEH formation do overlap with the likely weaning age, which suggest this was a period of repeated stress.

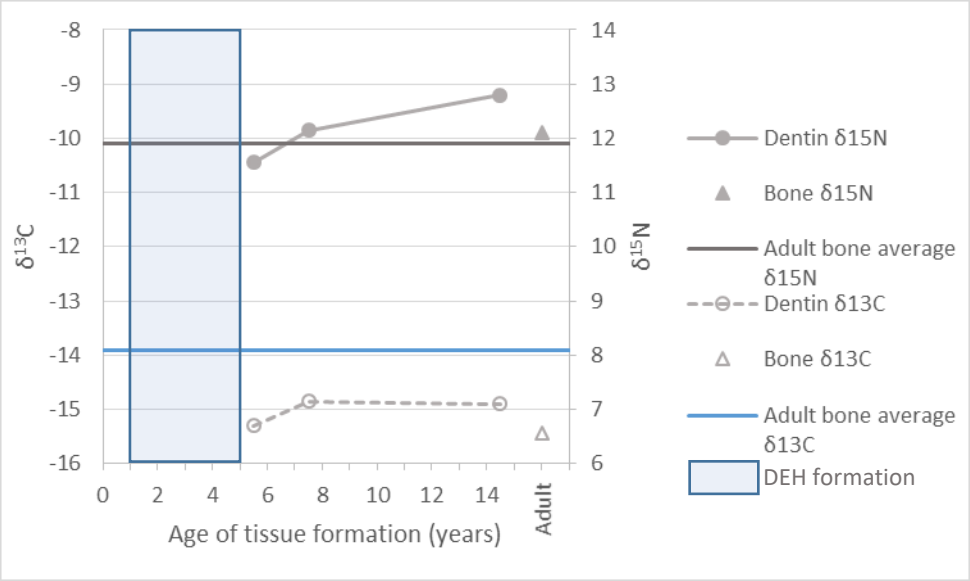

Figure 4.13: Results of $\delta^{15}N$ and $\delta^{13}C$ stable isotopes in relation to age of tissue formation compared to DEH formation from the 20 to 25-year-old male in Grave 8

Grave 9

Out of the 12 teeth present, the 7 to 8-year-old in Grave 9 only had one tooth, the right permanent maxillary first incisor (RI^1), with dental enamel hypoplasias (Long, 2019). Only three dental enamel hypoplasias were observed on RI^1 and the ages in which they occurred range from 1.9 to 4.0 years old (Long, 2019). The lack of teeth sensitive to DEH formation that develop after the second incisor crown, such as the canines and premolars, hinders assessment of DEH formation later in childhood. In this case, DEH are clearly associated with the weaning period (Figure 4.14). However, stress between the cessation of weaning and this individual's death between 6 and 8 years cannot be observed through DEH due to the lack of dental crowns that formed during this period.

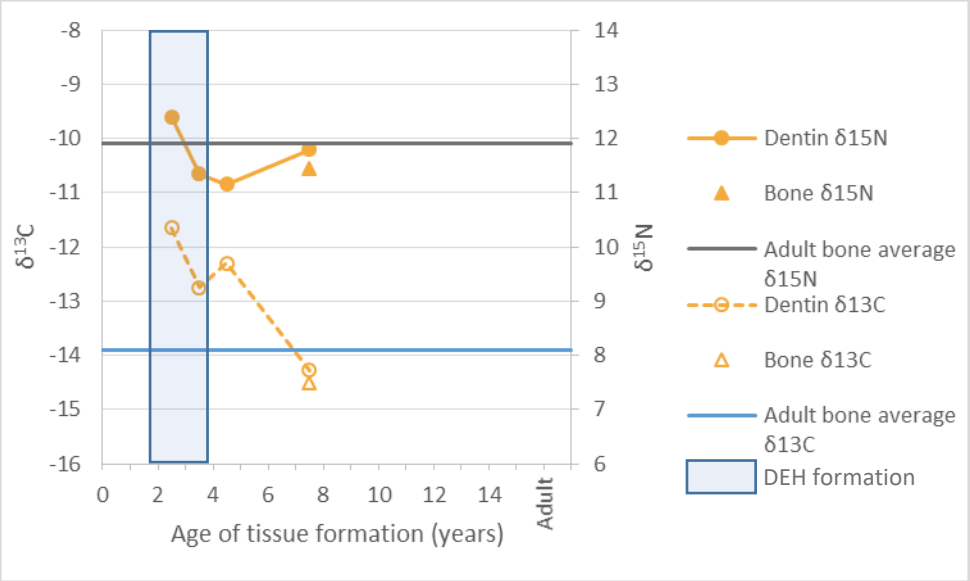

Figure 4.14: Results of $\delta^{15}N$ and $\delta^{13}C$ stable isotopes in relation to age of tissue formation compared to DEH formation from the 7 to 8-year-old in Grave 9

Grave 10 Burial 1

The 6 to 8-year-old in Grave 10 Burial 1 has a total of 27 DEH and 9 out of the 18 permanent teeth exhibit at least one DEH (Long, 2019). The DEHs form between 1.9 to 4.8 years (Long, 2019), which cease during formation of the premolar crowns, suggesting the lack of DEH in later childhood is not a factor of sampling bias. In this individual, stress leading to DEH formation seems to begin during the late weaning period and continue during a period of post-weaning dietary shift (Figure 4.15).

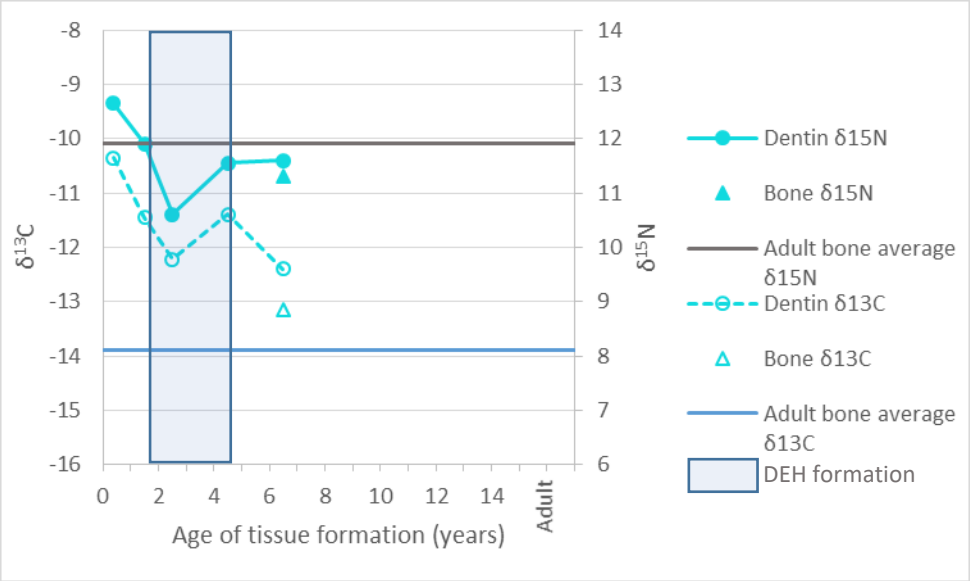

Figure 4.15: Results of δ15N and δ13C stable isotopes in relation to age of tissue formation compared to DEH formation from the 6 to 8-years-old in Grave 10 Burial 1

Childhood vitamin D deficiency

Radiograph and Microscopy Results

The radiographs of the right mandibular first molars from Grave 2 and Grave 9 and the left mandibular first molar from Grave 10 Burial 1 were analyzed for evidence of childhood rickets. The radiographs of these teeth show the first permanent molar from the adult individual in Grave 2 has a constricted and chair-shaped pulp chamber possibly indicative of rickets (Figure 4.16). The pulp chamber measurements of this molar found one pulp horn with a width < 1 mm and a pulp horn height ratio greater than 2:1, further indicating vitamin D deficiency may have occurred during childhood (Table 4.3). The subadult individuals from Grave 9 and Grave 10 Burial 1 also have chair-shaped pulp chambers (Figures 4.17 and 4.18), but only Grave 10 Burial 1 has a pulp horn width < 1mm, and the other pulp horn measurements and pulp horn height ratios are within normal range. These results suggest the individual from Grave 2 experienced vitamin D deficiency during formation of the first molar's pulp chamber formation.

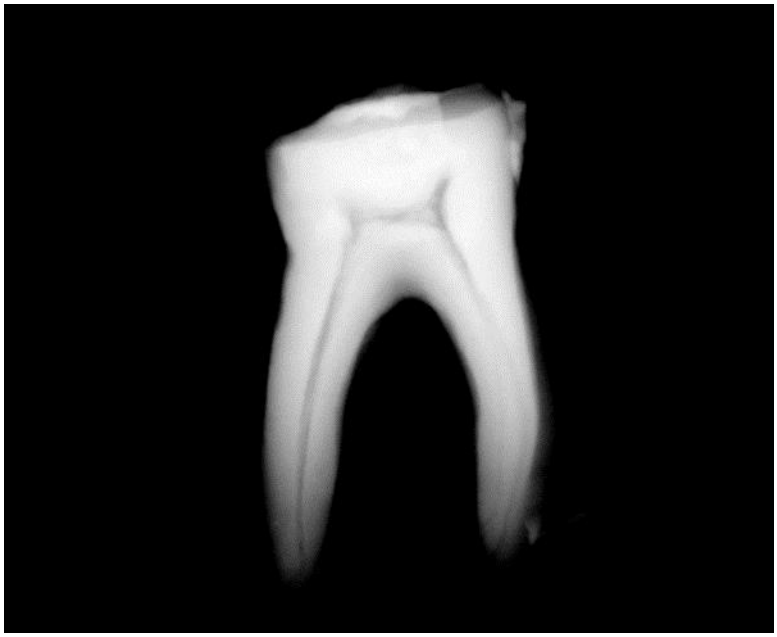

Figure 4.16: Radiograph of the buccal side of the right mandibular first permanent molar of the individual in Grave 2

Table 4.3: Measurements (mm) and Ratios of the first permanent molars from some of the skeletal individuals analyzed by GCAS

Grave No.	Type of Tooth	Pulp Chamber Height	Pulp Horn Height-1	Pulp Horn Width-1	Pulp Horn Height-2	Pulp Horn Width-2	Ratio of Pulp Horn Height
2	LRM1	0.227	0.616	0.713	1.803	1.037	2.93:1
9	LRM1	2.535	3.802	1.486	4.807	1.617	1.26:1
10 Burial 1	LLM1	1.932	2.801	0.998	3.477	1.312	1.24:1

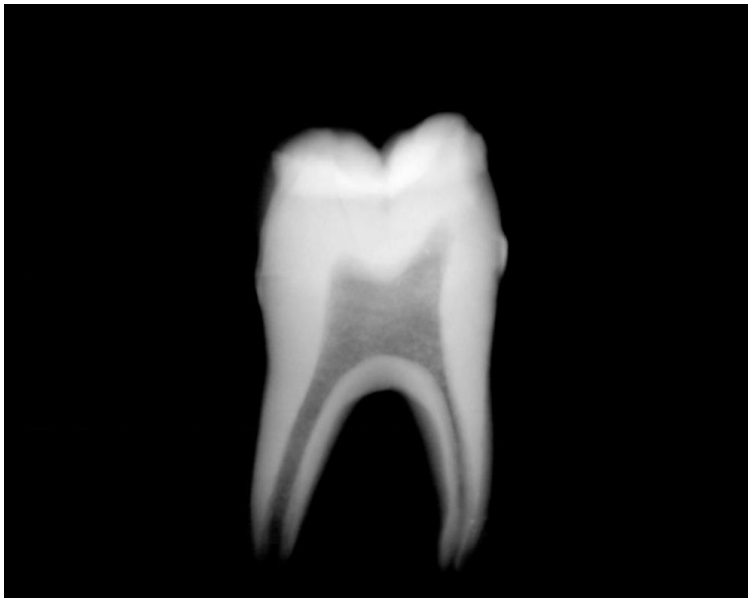

Figure 4.17: Radiograph of the buccal side of the right mandibular first permanent molar of the individual in Grave 9

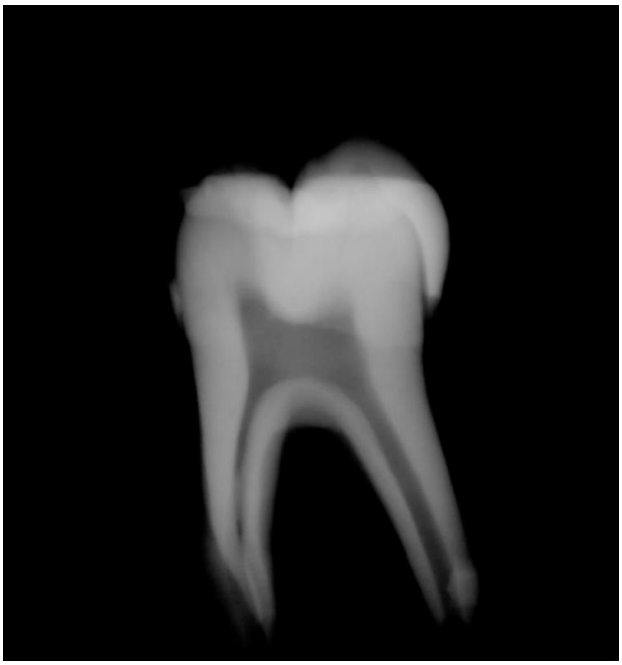

Figure 4.18: Radiograph of the buccal side of the mandibular left first permanent molar of the individual in Grave 10 Burial 1

The histological analysis further supports the radiographic analysis. The adult in Grave 2 displays interglobular dentin (IGD) (Figure 4.19a), while the subadults from Grave 9 and Grave

10 Burial 1 do not (Figure 4.19b, c). The individual from Grave 2 has Grade 1 IGD. It was hard to determine the age of dentin formation because some of the enamel and the early forming dentin of the molar were worn down. Based on the remainder of the dentino-enamel junction (DEJ), the IGD formed between 2.5 to 3 years of age (Table 4.4). This corresponds to a period of decreased $\delta^{15}N$ values in the individual as indicated above.

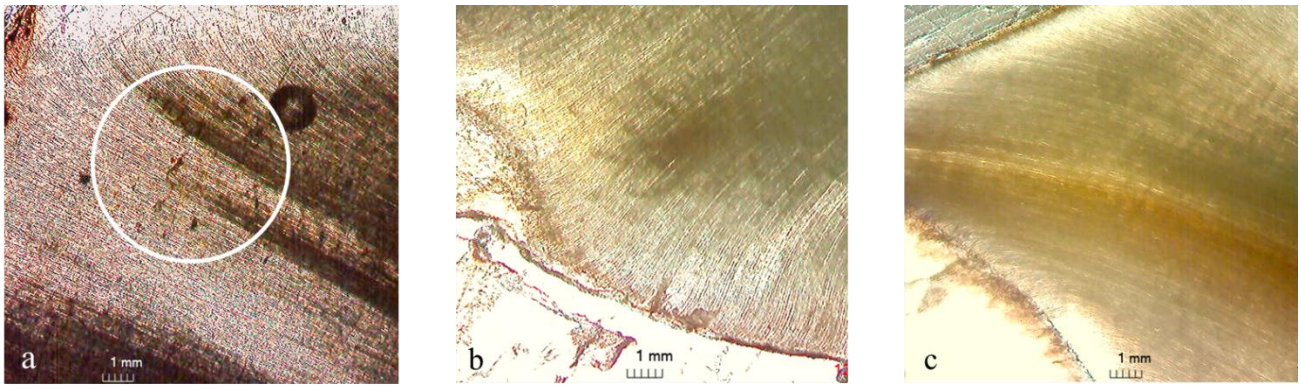

Figure 4.19: a) Histological image of dentin from the individual in Grave 2 (IGD severity Grade 1). The concentration of IGD is circled in white. b) Individual from Grave 9 (Grade 0); c) Individual from Grave X Burial 1 (Grade 0).

Table 4.4: Grade of severity and estimated age of the first permanent molars from some of the skeletal individuals in GCAS

Grave No.	Type of Tooth	Location	Grade	Estimated Age
Grave 2	LRM1	Mesial Crown and Distal Crown	1	2.5 - 3 years
Grave 9	LRM1	Absent	0	N/A
Grave 10 Burial 1	LRM1	Absent	0	N/A

CHAPTER FIVE:

DISCUSSION

As previously discussed, the GCAS sample is small (N=10). More than half of the sample contains children (60%), which suggests this population was experiencing high childhood mortality (Long, 2019; Quintana, 2019). Other mid-Atlantic 17th to 19th century sites (e.g. Cullen & Owsley, 2011; Owsley et al., 2018; Trinkley & Hacker, 2015) have similar proportions of children in their sample, ranging from 60% to 70%. The bioarchaeological research done on these sites (e.g. Cullen & Owsley, 2011; France et al., 2014; Owsley et al., 2018; Trinkley & Hacker, 2015; Ubelaker & Owsley, 2003) though has not fully delved into the possible factors, like diet and breastfeeding and weaning practices, that could have contributed to the early demise of these children. Breastfeeding and weaning practices can heavily impact a child's immunological development and nutritional status and sometimes cause nutritional and physiological stress (Brickley & Ives, 2008; Brickley et al., 2014; Katzenberg et al., 1996; Lewis, 2007; Mays, 2010). The nutritional and physiological stress associated with breastfeeding and weaning practices are sometimes linked with non-specific physiological stress, like dental enamel hypoplasias. However, dental enamel hypoplasias have been linked to other nutritional, physiological and environmental stressors (Blakey et al., 1994; Katzenberg et al., 1996; Larson, 2015; Lewis, 2007; Reitsema & Mcllvaine, 2014; Wood, 1996). The two infants and two children in the GCAS sample experienced some form of non-specific physiological stress before their death, dental enamel hypoplasias and/or an ongoing condition leading to cribra orbitalia (Long, 2019). Additionally, all the adults in the GCAS sample had dental enamel hypoplasias. Here, breastfeeding and weaning practices and childhood and adult diets were analyzed through stable isotope analysis of incremental dentin in order to assess possible factors of the GCAS

samples' childhood frailty (Beaumont et al., 2015; Beaumont et al., 2018; Garland & Reitsema, 2018; Mays, 2010). Additionally, the observation of micronutrient deficiencies like vitamin D can provide information on whether the weaning diet of the GCAS sample is nutritionally poor (Brickley & Ives, 2008; Brickley et al., 2014). Histological analysis is effective in determining vitamin D deficiency in childhood (D'Ortenzio et al., 2016, 2018b). The results of both methods were used to help determine the weaning age and diet of the GCAS sample and delve deeper into the daily lives of early European North Carolinians.

Overall diet

The isotopic study of the Gause family's childhood diet using $\delta^{13}C$ and $\delta^{15}N$ bone and dentin collagen helped illuminate the overall diet and weaning practices used by the Gause individuals buried in the cemetery. As previously discussed, $\delta^{13}C$ reflects the dietary composition of an individual in terms of C_3 versus C_4 plants and marine versus terrestrial resources. The $\delta^{13}C$ collagen values of the overall sample (bone and dentin) range between -15.6‰ and -9.5‰ ($\underline{x}$= -13.5 ± 1.4‰) suggesting they had both C_3 and C_4 plants and/or marine organisms in their diet. The $\delta^{15}N$ collagen ratios ranging between 10.5‰ and 14.2‰ and ($\underline{x}$ = 12.1 ± 0.9‰) seem to confirm this contribution of marine organisms to the diet. It is important to note the depicted overall diet used data from infants, children and adults and tissues that formed in multiple stages of life.

Comparison of bone collagen $\delta^{13}C$ and $\delta^{15}N$ values from the GCAS skeletal sample to comparable 18th and 19th century sites in the mid-Atlantic and the southeastern regions of the United States provided context for these results. This comparative analysis included adults and children older than 4 years of age buried in the Woodville Cemetery (Doms et al., 1995; France

et al., 2014), Richards Family Vault (Cullen & Owsley, 2011), Orton Plantation (Trinkley & Hacker, 2015), Foscue Plantation (Seeman, 2011), and Darnall's Chance House (Owsley et al., 2018). The Orton Plantation and Foscue Plantation sites are located in southeastern North Carolina (Seeman, 2011; Trinkley & Hacker, 2015). The individuals excavated from the Orton Plantation cemetery in Brunswick County, NC, are associated with Roger Moore and his family, who established the plantation in the mid-18th century and was involved in settling adjacent Brunswick Town (Trinkley & Hacker, 2015). The early 19th century Foscue burial crypt in Jones County, NC, was used by the Foscue family who owned Foscue Plantation (Seeman, 2011). The other sites used here come from the larger Mid-Atlantic region. The 18th to 19th century cemetery associated with Darnall's Chance House in Prince George's County, MD, includes one series of landowners, the Lee Family (Owsley et al., 2018). The elite yet urban family of Mr. Alfred Richards, the first man to manufacture brick in Washington D.C., was interred in the late 19th and early 20th century Richards Family Vault, located in the Historic Congressional Cemetery in Washington D.C. (Cullen & Owsley, 2011). On the other hand, the late 18th to early 19th century Woodville cemetery in Delaware, near the intersection of Routes 13 and 406, is associated with a tenant farmstead, the Woodville Farm Site (Doms et al., 1995).

Table 5.1: Means and standard deviations of $\delta^{13}C$ and $\delta^{15}N$ from 18th to early 20th century Mid-Atlantic and North Carolina sites

Site	Location	Sample size (4 + years of age)	$\delta^{13}C$ Bone Collagen Mean (1σ)	$\delta^{15}N$ Bone Collagen Mean (1σ)	Reference
Woodville Cemetery	Delaware	6	-12.0 ± 1.6	11.2 ± 0.6	Doms et al., 1995; France et al., 2014
Richards Family Vault	Washington D.C.	5	-14.1 ±1.5	11.9 ± 0.5	Cullen & Owsley, 2011
Orton Plantation	North Carolina	4	-15.0 ± 0.7	11.1 ± 0.3	Trinkley & Hacker, 2015
Foscue Plantation	North Carolina	3	-13.4 ± 0.8	10.9 ± 0.4	Seeman, 2011
Darnall's Chance House	Maryland	5	-14.4 ± 0.6	11.3 ± 0.6	Owsley et al., 2018
Gause Cemetery at Seaside	North Carolina	6	-13.9 ± 1.0	11.7 ± 0.3	This study

These sites have $\delta^{13}C$ means and standard deviations ranging from -16‰ and -10‰, indicating varied C_3 and C_4 contributions to their diets (Table 5.1; Figure 5.1). However, the level of animal protein is relatively similar across the sites except the Richards Family vault and the GCAS. These two sites have moderate $\delta^{13}C$ values and higher $\delta^{15}N$ values, likely indicating they had a substantial marine as well as terrestrial component to their diet. Exploitation of marine resources by the Gause family is not unexpected considering their plantation's coastal location. The Richards Family, an economically prominent family buried in the Historic Congressional Cemetery in Washington D.C. also relied substantially on freshwater and/or marine resources and/or a notable contribution of animals consuming maize (Cullen & Owsley, 2011).

Additionally, when looking at possible socioeconomic patterns, landowners and industry leaders versus tenant farmers, the sole tenant farmer site, Woodville Cemetery, tends to have higher $\delta^{13}C$ values yet similar $\delta^{15}N$ than sites represented by landowners or industrialists. These values suggest the tenant farmers had a higher contribution of C_4 plants and animals that consumed C_4 plants in their diet. Living in a rural versus urban environment also does not seem to result in notable differences in either isotope ratio.

Inner and outer coastal North Carolinian diets also varied in terms of plant types, meat versus plant resources, and marine contributions to diet. The Roger Moore family buried near Orton Plantation on the Cape Fear River does not show similarly elevated $\delta^{15}N$ values as the Gauses. Additionally, the Roger Moore family's $\delta^{13}C$ values suggest a stronger reliance on wheat, rice, or other C_3 sources than the Gause family. The Foscue family living more inland near the Trent River, which feeds into the Neuse River inlet, has average $\delta^{15}N$ ratios similar to

the Moore Family but had a higher contribution of C_4 plants in their diet, likely maize, than either the Moore or Gause families.

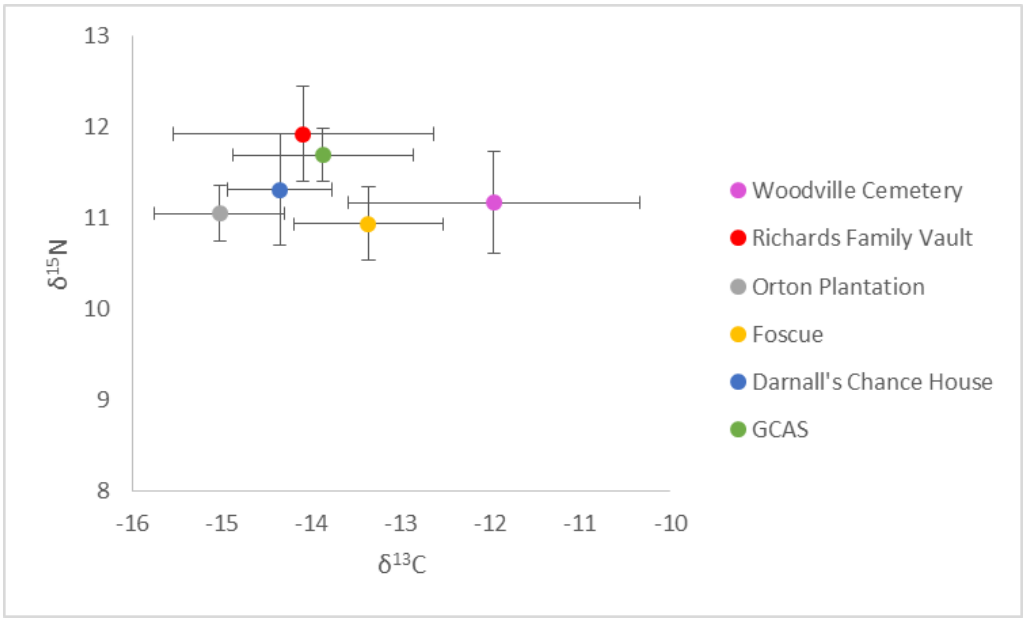

Figure 5.1: Mean and standard deviation of bone collagen $\delta^{13}C$ and $\delta^{15}N$ values from GCAS and comparative regional sites listed in Table 5.1

Thus, from the geographic comparison, it is clear the Gause family relied on a mixture of C_3 and C_4 sources and both terrestrial and marine sources. From historical documents, it is likely the individuals from the Gause Cemetery consumed rice, potatoes, peas, onions, and wheat (C_3 plants) in addition to maize and sugar (C_4 plants) from their garden, local farms, or long-distance trade (DeNeale, 1855; Gorrell, 1857; Omstead, 1863; Sprunt, 1916; State of Agricultural Society of North Carolina, 1855; The Wilmington Daily Herald, 1855; U.S. Census Bureau, 1860a). Furthermore, the Gause family owned cattle, sheep, goats, and hogs, which were likely fed on corn, sweet potatoes, and wild grasses (C. Gause, 1806; J. Gause, 1783; J. J. Gause, 1836; N. Gause, 1794; Lee, 1980). The Gause family and other families in Brunswick County also had easy access to coastal marine resources, including oysters, sturgeon, herring, shad, and other freshwater fish (Swift, 1904; Lee, 1980). However, it is important to note the marine effect on

$\delta^{13}C$ values likely modified the GCAS samples $\delta^{13}C$ signatures and so it is unclear as to whether the GCAS sample had a stronger C_3 or C_4 plant signature.

Weaning practices and childhood diet

The age-related changes in $\delta^{15}N$ in dentin within the Gause family indicates the weaning process began by 10.5 months of age and ceased by 1.5 years (see Figure 5.2). The slight increase 1.2‰ in $\delta^{15}N$ from 4.5 to 10.5 months of age may reflect the normal pattern of residual effects of receiving nitrogen from the intrauterine environment in addition to solely breastfeeding, but also could indicate the level of maternal physiological stress during this period (Beaumont et al., 2015; Millard, 2000). The 2.0‰ decline in $\delta^{15}N$ from 10.5 months to 1.5 years of age reflects the period of a gradual decline in breastfeeding and increased supplementation of solid foods. This age and duration of the weaning period parallels most available colonial U.S. historical accounts suggesting an infant should start the weaning process between 3 and 12 months of age and continue until 6 months to 2 years of age (Censer, 1984; Child, 1837; Fildes, 1995; McMillen, 1990; Schmidt, 1976). Weaning in historic North America was recommended to start as late as 1 year of age, not the modern recommendation of 6 months of age, for fear of infantile diarrheal diseases (Child, 1837; Lewis, 2007; McMillen, 1990). The GCAS overall weaning period deviates from modern recommended practices based on clinical research that suggest the weaning period should start at 4 to 6 months and extend until 1 to 2 years of age or longer. (Grueger et al., 2013; Mayo Clinic Staff, 2019; Victora et al., 2016; WHO, 2020). The prolonged breastfeeding, without additional food sources, seen in the GCAS sample, and suggested in historical documents, may have been harmful to the infants' growth and development because breastmilk is inadequate at supplying enough protein and nutrients, like

iron, to infants in the later stages of developmental growth, after 6 months (Grueger et al., 2013; Ross, 1981).

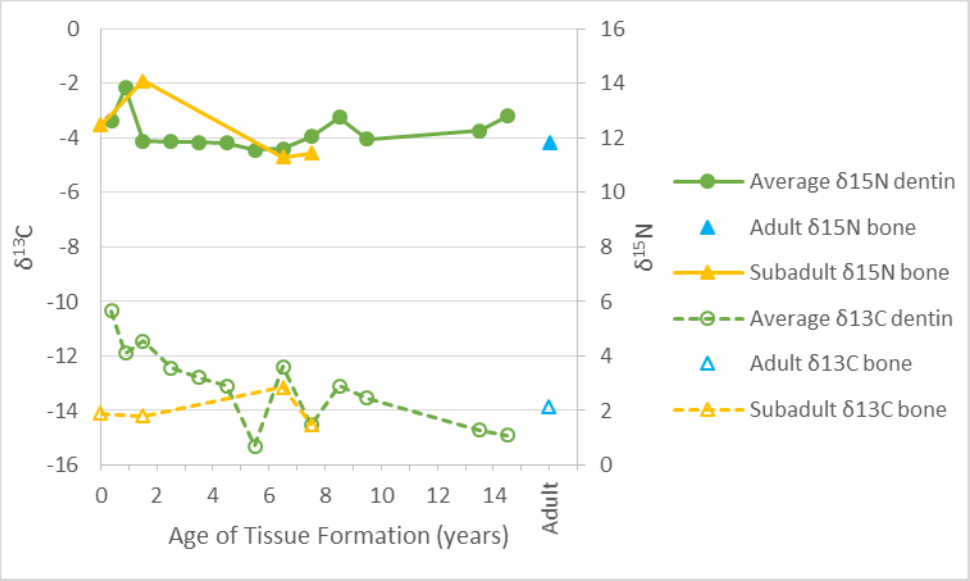

Figure 5.2: Averaged Mean $\delta^{15}N$ and $\delta^{13}C$ dentin collagen values and subadults and adults bone collagen values from the GCAS skeletal sample

The $\delta^{13}C$ dentin values from the Gause family reveal a slightly different pattern than the $\delta^{15}N$ ratios (Figure 5.2). Instead of values increasing between 4.5 months and 10.5 months, they decrease by 1.6‰ and show the GCAS sample started with a larger component of C_4 plants at 4.5 months then added some C_3 sources to the diet by 10.5 months. From 10.5 months to 5 years, $\delta^{13}C$ values decrease another 3.4‰ during the weaning and some of the post-weaning periods. The $\delta^{13}C$ dentin values decline and rise between 5.5 and 7.5 years of age and are close to those seen in adult bone collagen. Overall, $\delta^{13}C$ in dentin declined 4.9‰ from 4.5 months until 7.5 years. If weaning indeed began at around 10.5 months of age as shown by the $\delta^{15}N$ values, the maternal diet during breastfeeding contained more C_4 sources than the first solid foods introduced to the infant and the general adult population based on the bone $\delta^{13}C$. As noted above, the Gause family diet reflected a mix of C_3 and C_4 sources along with a notable contribution of

marine and terrestrial foods. Although women can have a 1‰ offset from their exclusively breastfed children (Fuller et al., 2006; Herrscher et al., 2017; Tsutaya & Yoneda, 2015), pregnant and lactating women in the Gause family could have had a diet with more maize or marine sources than the typical adult diet, resulting in higher $\delta^{13}C$ value than average adults. The children likely were fed weaning foods that had a larger portion of wheat, rice, and other C_3 sources than that consumed by the mother, which according to historical sources would have been combined with milk, broth and/or water in the form of pap, panada, or bread (Censer, 1984; Child, 1837; Fildes, 1995; McMillen, 1990; Schmidt, 1976). Other weaning food recommendations were arrow-root (C_3 plant) or gum-arabic (C_3 plant) dissolved in water with sugar (C_4 plant) and milk (Child, 1837).

The overall patterns seen in the dentin may mask differences between individuals dying during childhood versus those living into adulthood, and individual weaning patterns. The level of diversity particularly in nitrogen samples steadily decreases with increasing age of dentin formation, but this variation is seen equally in individuals dying in childhood versus those surviving into adulthood, and no difference is seen based on childhood vs. adult mortality in the mean values of $\delta^{15}N$ except in dentin forming after 6 years of age. The early-age diversity in $\delta^{15}N$ has been hypothesized to reflect the mutable physiological and metabolic effects of pregnancy, and presumably, lactation on both the mother and the child, rather than diversity in diet (Beaumont et al. 2015; Burt & Garvie-Lok, 2013). On the other hand, the variation seen in $\delta^{13}C$ in the younger age categories is reflecting distinctly different diets seen in those dying in childhood versus in adulthood, with those linked to childhood mortality having a larger C_4 component in their (or their mother's) diet before 2 years and between 4 and 6 years of age. Infant nails' and bone stable isotope values do not reflect the dietary intake of breastmilk until 21

weeks after birth, so any values before will reflect birth and *in utero* values (Herrscher et al., 2017). Thus the slight offset or variation seen in the GCAS infants stable isotope values were likely due to individual variation, like differences in isotopic fractionation, short term changes in the mother's diet before birth, how long they survived postpartum, or physiological stress (Beaumont et al., 2015; Herrscher et al., 2017; Reitsema, 2013). These factors seem to have impacted the risk of childhood mortality in this sample.

The relationship between $\delta^{13}C$ and $\delta^{15}N$ in the individual-level analysis may illuminate what is causing this pattern--that is, whether it is due to dietary change, physiological stress, or other factors. In most cases, such as the adults in Graves 2, 3, and 6, and the tissues forming after 3 years of age for the child buried in Grave 9 have shifts in $\delta^{15}N$ and $\delta^{13}C$ with similar timing but opposite directions, indicating an increase in C_3 sources and thus a shift to a more negative $\delta^{13}C$ ratio is linked to an increase in $\delta^{15}N$ and vice versa. This pattern is expected, as a shift in diet as indicated by $\delta^{13}C$ should also be reflected by a change in $\delta^{15}N$. However while Grave 8 shows a parallel change in between 5.5 and 7.5 years in both isotope ratios, in this case in the same direction, between 7.5 and 14.5 years $\delta^{13}C$ remains stable while there is a 0.5‰ (standard error 0.1‰) increase in $\delta^{15}N$. While this is a rather small shift, this increase in $\delta^{15}N$ without a parallel change in $\delta^{13}C$ might reflect a period of stress between these ages (Beaumont et al. 2015, 2018). Unfortunately, there are no samples of dentin for this individual that formed between these ages, 7.5 and 14.5, that could indicate this increase in $\delta^{15}N$ and stabilization of $\delta^{13}C$ was a continuous trend during this period. The child buried in Grave 10 Burial 1 also shows parallel shifts in both isotope ratios between birth and 6 years of age, but from 6 until their death at between 6-8 years of age, $\delta^{13}C$ decreases approximately 1‰ while $\delta^{15}N$ stays the same. If $\delta^{15}N$ decreased along with $\delta^{13}C$, following the pattern earlier in this individual's life, then a stationary nitrogen isotope

ratio and a decline in $\delta^{13}C$ would indicate that $\delta^{15}N$ may be higher than it should be, and thus is reflecting a period of physiological stress in the years before death.

The two newborns buried together in Grave 10 Burial 2 were hypothesized to have been twins who died during the perinatal period. Since they would have been exposed to the same intrauterine environment, their $\delta^{15}N$ and $\delta^{13}C$ ratios should be similar within the range of analytical error. However, this is not the case. Individual 1 from Grave 10 Burial 2's $\delta^{13}C$ and $\delta^{15}N$ values are higher than individual 2 from Grave 10 Burial 2's by 1.15‰ and 0.7‰. It is also possible they were two newborns from different mothers who just happened to be buried together for convenience's sake, but little is known whether maternal tradeoffs during pregnancy could differently impact twins in utero.

Non-Specific Physiological Stress and Stable Isotopes

Overall, although most of the GCAS individuals had DEH, not all of the DEH formations can be linked to weaning processes or physiological or nutritional stress. Certain teeth, particularly the posterior dentition, are less likely to develop DEH, not only in terms of their sensitivity, but also due to their crown morphology (Guatelli-Steinberg et al., 2012; Hillson & Bond, 1997). However, in most individuals all periods of childhood are represented.

Variation between Types of Tissues

As with other studies (e.g., Beaumont et al., 2015, 2018) dentin and bone collagen $\delta^{15}N$ and $\delta^{13}C$ values differed in co-forming tissues and the sample-level patterns also varied between bone and dentin. In Figure 5.2, the dentin and bone $\delta^{15}N$ values do not share a similar pattern until 7.5 years of age. Instead of the $\delta^{15}N$ values increasing between 4.5 and 10.5 months and decreasing between 10.5 months and 1.5 years as seen in the dentin, the bone values instead

increase between 0 and 1.5 years, then decrease between 1.5 years and 6.5 years. The same can be said when looking at the dentin and bone $\delta^{13}C$ values. Instead of decreasing in $\delta^{13}C$ values between 4.5 months and 6.5 years as seen in the dentin values, the bone values increase by 1‰. The last dentin and bone adult stable isotope values also are not similar. Even though the bone and dentin samples do not occur at the same age, their values were predicted to be alike because the individual would likely be consuming an "adult" diet at the time of dentin formation. Additionally, the children from Grave 9 and Grave 10 burial 1 had co-forming dentin and bone, and in the case of Grave 9 the bone value was 0.4‰ lower in $\delta^{15}N$ values and 0.2‰ lower in $\delta^{13}C$ values than dentin. For Grave 10 burial 1, the bone value was 0.3‰ lower in $\delta^{15}N$ and 0.8‰ lower in $\delta^{13}C$ than dentin.

This variation between bone and dentin stable isotope values can result from differences in turnover rates by tissue type in addition to the differential impact of physiological stress on absorption of isotopes in bone and dentin. Bone cells turnover faster in children than adults, and this turnover rate becomes progressively slower throughout childhood (Beaumont et al., 2018). Thus, the bones sampled include new and old bone cells and their absorbed stable isotopes, constituting different periods of time before death, with the amount of antemortem time increasing with age. However, dentin remains stable, and so the dentin stable isotope values closely represent the diet and physiology of the individual immediately surrounding the period of its formation, although some previously-absorbed isotopes still circulating in the body could be incorporated as well (Beaumont et al., 2015, 2018). Thus, other studies have seen an offset between bone and dentin isotope values with bone $\delta^{15}N$ and $\delta^{13}C$ values being lower than the dentin values (Beaumont et al., 2015, 2018). The differences in $\delta^{15}N$ and $\delta^{13}C$ bone and dentin

values observed in the individuals from Grave 9 and 10 burial 1 and the overall sample could have resulted from differences in bone and dentin turnover rates.

However, an added factor stems from the impact of physiological or nutritional stress on bone and dentin cells. Osteoblasts cease to produce new bone collagen when a child is undergoing stress, while dentinoblasts continue to produce and absorb carbon and nitrogen (Beaumont et al., 2018). Physiological stress generally results in higher $\delta^{15}N$ values than normal because the stress causes the individual's body to go into a catabolic state and synthesize proteins from their own body tissues, which increases the trophic level shift (Beaumont et al., 2018; Katzenberg & Lovell, 1999). Additionally, nutritional stress causes $\delta^{13}C$ values to lower because of the recycling of body fat stores (Beaumont & Montgomery, 2016; Beaumont et al., 2018). The individuals from Grave 9 and 10 Burial 1 and the overall sample had similar $\delta^{15}N$ values for both bone and dentin. Furthermore, Grave 10 Burial 1 individual's $\delta^{15}N$ dentin values stay stagnant when the $\delta^{13}C$ dentin and bone values decrease; however, Grave 9 individual's $\delta^{15}N$ dentin values rise 0.7‰ between 4.5 and 7.5 years while the $\delta^{13}C$ dentin values decreased 2.0‰. The change in $\delta^{15}N$ and $\delta^{13}C$ values observed in the 7 to 8-year-old from Grave 9 is likely from dietary changes due to eating game and freshwater animals rather than signs of physiological or nutritional stress because of the small increase in $\delta^{15}N$ and close to similar values in $\delta^{15}N$ between bone and dentin. However, the child in Grave 10 Burial 1 may have been undergoing physiological stress during the period both tissues were forming shortly before death.

Childhood vitamin D deficiency

Out of the three individuals analyzed (Grave 2, Grave 9, Grave 10 Burial 1), only the individual from Grave 2 presented IGD indicative of vitamin D deficiency between 2.5 and 3

years of age. This age coincides with a decline in $\delta^{15}N$ and a slight increase in $\delta^{13}C$ that may reflect the last stages of weaning or a post-weaning dietary shift to a more plant-based and slightly more C_4 diet. Most cereal C_3 plants are high in phytates, which can contribute to the development of vitamin D deficiency because phytates stop calcium absorption (Brickley & Ives, 2008; Lewis, 2007; Littleton, 1998; Schmidt, 1976). The influence of metabolic deficiencies such as rickets or scurvy on $\delta^{15}N$ and $\delta^{13}C$ has strong research potential.

The one infant in Grave 11 with skeletal signs of rickets or scurvy did have other present and observable dentition; however, none of the dentition was thin sectioned and analyzed for IGD in this study. This individual though did have dentin, the crown of a deciduous first molar representing 10.5 months of age, and bone, the femur representing the period just before death at 18 months ± 6 months, that was sampled for stable isotope analysis. Bone and dentin collagen $\delta^{15}N$ ratios differ due to multiple factors, as noted above, and in this case the bone ratio is slightly higher than the dentin. This difference may stem from these samples reflecting different points in the weaning process, mirroring, but slightly older than the increase between 4.5 and 10.5 months seen in the combined dentin sample described above. This difference could also indicate this individual was under acute physiological stress before death, which can produce higher $\delta^{15}N$ ratios. However, the dentin values are not higher than the bone values, which would be predicted because of the decline in osteoblast activity in bone during stress. In general, however, it does not appear that metabolic deficiencies were large factors in the Gause family's high childhood frailty.

Implications

Overall, the individuals sampled from the Gause Cemetery at Seaside reflect a varied diet that consisted largely of C_3 sources and some contribution of marine or C_4 sources. Additionally, the GCAS sample fully ceased weaning by the age of 2.5 years with early diets consisting of more C_4 sources than later childhood or adulthood. Additionally, most of the GCAS samples, including Grave 2, 6, 8, 9, and 10 Burial 1, had DEH that coincided with weaning. Furthermore, the differences seen between stable isotope values in bone and dentin are most likely from differences in turnover rate between the tissues, although in some cases it could be due to physiological stress. Lastly, vitamin D deficiency was experienced by the 30-39 year-old male from Grave 2, who survived the condition according to the histological assessment of IGD. This period of deficiency from 2.5 to 3 years is associated with DEH development as well as a decline in $\delta^{15}N$ and a rise in $\delta^{13}C$, which may reflect dietary change in the late weaning or post-weaning stage. Neither of the children from Grave 9 and Grave 10 Burial 1 showed any IGD in their lower first molars. This indicates that metabolic disease was a not large factor in the GCAS sample's high childhood frailty.

CHAPTER SIX:

CONCLUSION

The overall diet of the individuals from the GCAS site consisted largely of C_3 with some C_4 plants or more likely, marine resources. The overall diet depicted compliments the historical documentation of an 18th to 19th century diet, but variation within the three southeastern North Carolina cemeteries suggests dietary diversity between plantations in the region (Cullen & Owsley, 2011; DeNeale, 1855; Doms et al., 1995; France et al., 2014; C. Gause, 1808; J. Gause, 1783; J. J. Gause, 1836; N. Gause, 1794; Gorrell, 1857; Lee, 1980; Omstead, 1863; Owsley et al., 2018; Seeman, 2011; Sprunt, 1916; State of Agricultural Society of North Carolina, 1855; Swift, 1904; The Wilmington Daily Herald, 1855; Trinkley & Hacker, 2015; U.S. Census Bureau, 1860a). Additionally, the weaning diet consisted of C_4 plants with a gradual supplementation of C_3 plants, which reflected the historical diet suggested by Child (1837). The overall sample mean of $\delta^{15}N$ in dentin may indicate that weaning ceased at 1.5 years. However, some variability exists when the isotope ratios are observed on the individual level, which indicates that weaning may have ended between 2.5 and 4.5 years. As previously discussed, according to clinical studies as well as anthropological studies, weaning should begin around 4 to 6 months and cease at 1 to 2 years of age or beyond (Dettwyler, 1995; Grueger et al., 2013; Lewis, 2007; Mayo Clinic Staff, 2019; Victora et al. 2016; WHO, 2020). Thus, the ceased weaning age was not a factor to the high child mortality rate seen at the GCAS site.

The patterning of $\delta^{15}N$ and $\delta^{13}C$ ratios demonstrates that even though $\delta^{15}N$ in dentin can reflect the impact of physiological stress on the isotope ratio, most shifts in $\delta^{15}N$ seen here match changes in $\delta^{13}C$, and thus are largely diet related. However, some individuals such as Grave 10 Burial 1 may have been experiencing stress before death, indicated not only by divergence in

$\delta^{15}N$ and $\delta^{13}C$ between approximately 5.5 to 6.5 years, but also by the repeated DEH formation before this divergence and the slight offset between dentin and bone $\delta^{15}N$ just around the time of death. Based on the lack of IGD in this person's first molar, it seems that they did not suffer from vitamin-D deficiency before their death. Additionally, the weaning period in the GCAS sample seems to coincide with a period of repeated DEH formation, either based on direct comparison between DEH timing and isotope values from around those specific ages, or knowledge on when weaning generally occurred in this group and DEH formation.

From the histological analysis, it was determined out of the three individuals sampled, Grave 2, 9, and 10 Burial 1, only one individual, Grave 2, showed signs of vitamin D deficiency. The IGD found in the dentin from the individual in Grave 2 formed between 2.5 and 3 years of age. When compared with the stable isotope results, this may be related to dietary change in the late weaning or post-weaning period. The impact of metabolic disease on isotope ratios is not clear but one study has suggested that both $\delta^{15}N$ and $\delta^{13}C$ values increase when an individual has a metabolic disease such as scurvy (Nicholls et al., 2020). The 18-month-old (± 6 month) in Grave 11 with rib beading, which can result from either rickets or scurvy, did not display any signs of physiological stress in their stable isotopes that would indicate experience of a metabolic deficiency.

Overall, this study helps bring to light the foods that these individuals consumed and how they were used to raise their families as well as provides a unique perspective of the daily lives of coastal North Carolinians. Additionally, this study provides an example of the benefits of interdisciplinary research by demonstrating the knowledge that can be gained about diet and metabolic diseases on a historic population through paleopathology, history, bioarchaeology, and chemistry. Furthermore, the study demonstrates that working with the descendant community

can be beneficial to the archaeologist. Throughout this study, J.R. Robinson provided valuable information about his ancestors as well as intends to help promote archaeology by releasing information obtained from his ancestors accessible to the public and other descendants.

Although this study provides some valuable information on the diet of the GCAS sample, the GCAS's small sample size hinders the ability to make broader interpretations about childhood health and metabolic diseases experienced in the population. Thus, the rest of the GCAS site must be excavated and the remains analyzed in order to further interpret the lived experiences of the Gause family.

Future research

Isotopic and histological analysis of dentition

In addition to the continued excavation of the GCAS site, isotopic and histological analysis should continue to be performed on the remains. If more individuals were excavated from GCAS, isotopic analysis would help strengthen patterns in the sample and would help identify differences between adult and subadult weaning patterns and diet. Additionally, if apatite were sampled from the GCAS individuals and compared to collagen samples a greater association could be made on whether C_3 or C_4 plants were being consumed and possibly when breastfeeding stopped because breastfed infants have higher $\delta^{18}O$ than post-weaning infants (Dupras & Tocheri, 2007; Kellner & Schoeninger, 2007). Furthermore depending on the facilities available, more teeth could be sampled from not only excavated individuals from future field seasons, but also the permanent first molar from the individual in Grave 8, in order to get a more comprehensive view of their weaning patterns.

Vitamin D was present during childhood in the 30 to 39-year-old male from Grave 2 and possibly the 18-month-old (± 6 months) from Grave 11. With the continued excavation and analysis of the GCAS site, additional histological analysis including the 18-month-old (± 6 months) from Grave 11 should be performed on any of the first permanent molars of the individuals to determine whether a large part of the population experienced a vitamin D deficiency. Additionally, the findings from the histological analysis should be compared to the findings from the stable isotope analysis. This comparison will help determine whether diet or illness were factors that caused metabolic disease, or possibly whether metabolic disease has an impact on stable isotopes values. As previously mentioned, foods were not fortified with vitamin D and so the only sources for vitamin D had to come from natural sources. Furthermore, the weaning diet at the time was high in phytates which can contribute to the development of vitamin D deficiency (Brickley & Ives, 2008; Lewis, 2007; Littleton, 1998; Schmidt, 1976). Thus, the diet or lying in bed from illness likely caused vitamin D deficiency to form (Lewis, 2007; Schmidt, 1976).

Chemical analysis of the hair

Besides dentin and bone, other forms of tissues provide information about diet, like hair. During the recovery and analysis of the adult female from Grave 3, hair was found covering two-thirds of her skull. Although short and not useful for physical observation and analysis, the recovered hair can be used in analyses like stable isotopes and trace elements. When analyzing hair collagen, the $\delta^{15}N$ and $\delta^{13}C$ values represent the diet of the individual during their last years of life (Price, 2015; Webb et al., 2015). Trace elements linked to nutritional deficiency present in archaeological hair samples are calcium, copper, and zinc (Bergfield, 2007). In addition to stable

isotopes and trace elements, cortisol, a stress hormone, can be measured in archaeological hair samples (Webb et al., 2015). Since cortisol is only produced during times of stress, the levels of cortisol can help determine whether the female from Grave 3 experienced stress in adulthood (Webb et al., 2015). Overall, these studies can help provide a glimpse into adult life in the 18th and 19th century, which cannot be provided by dental enamel hypoplasia or fully detailed by bone stable isotopes.

BIBLIOGRAPHY

Allen, L. H. (1994). Maternal Micronutrient Malnutrition: Effects on Breast Milk and Infant Nutrition, and Priorities for Intervention. *SCN News*, (11), 76.

Almond, D., & Currie, J. (2011). Killing Me Softly: The Fetal Origins Hypothesis. *Journal of Economic Perspectives*, 25(3), 153-172.

AlQahtani, S. J., Hector, M. P., & Liversidge, H. M. (2010). Brief Communication: The London Atlas of Human Tooth Development and Eruption. *American Journal of Physical Anthropology*, 142, 481-490.

Ambrose, S. H. (1990). Preparation and Characterization of Bone and Tooth Collagen for Isotopic Analysis. *Journal of Archaeological Science*, 17, 431-451.

Angelo, G., Delage, B., & Weaver, C. (2019). *Bone Health in Depth*. Oregon State University. Retrieved from https://lpi.oregonstate.edu/mic/health-disease/bone-health#vitamin-C

Beaumont, J., Atkins, E., Buckberry, J., Haydock, H., Horne, P., Howcroft, R., Mackenzie, K., & Montgomery, J. (2018). Comparing Apples and Oranges: Why Infant Bone Collagen May Not Reflect Dietary Intake in the Same Way as Dentin Collagen. *American Journal of Physical Anthropology*, 167, 524-540.

Beaumont, J., & Montgomery, J. (2016). The Great Irish Famine: Identifying Starvation in the Tissues of Victims using Stable Isotope Analysis of Bone and Incremental Dentin Collagen. *PLoS ONE*, 11(8), 1-21.

Beaumont, J., Montgomery, J., Buckberry, J., & Jay, M. (2015). Infant Mortality and Isotopic Complexity: New Approaches to Stress, Maternal Health, and Weaning. *American Journal of Physical Anthropology,* 157, 441-457.

Bender, V. (2017). *The Plantation Years*. Vern Bender. http://vernbender.com/the-plantation-years-1721-to-1774-at-ocean-isle-beach/

Bergfield, R. A. (2007). *Dietary Analysis of Archaeological Hair Samples from Peru*. (Master's Thesis, University of Missouri-Columbia).

Berry, C. B. (1982). William Gause. *The Independent Republic Quarterly*, 16(4), 4-17.

Berry, C. B. (1988). A History of Star Bluff, Horry County, S.C.. *The Independent Republic Quarterly*, 22(3), 30-34.

Berry, C. B. (1996). What Has Happened to Gause's Swash?. *The Independent Republic Quarterly*, 30(4), 3-11.

Bitzan, M., & Goodyer, P. R. (2019). Hypophosphatemic Rickets. *Pediatric Clinic North America Journal*, 66(1), 179-207.

Blackford, B. L. (1865). *Map of part of Brunswick County showing the approaches to Wilmington, N.C.* [survey map]. Retrieved from https://dc.lib.unc.edu/cdm/singleitem/collection/ncmaps/id/214

Blakey, M. L., Leslie, T.E., & Reidy, J. P. (1994). Frequency and Chronological Distribution of Dental Enamel Hypoplasia in Enslaved African Americans: A Test of the Weaning Hypothesis. *American Journal of Physical Anthropology*, 95, 371-383.

Brickley, M., & Ives, R. (2008). *The Bioarchaeology of Metabolic Bone Disease*. Elsevier.

Brickley, M. B., Kahlon, B., & D'Ortenzio, L. (2019). Using Teeth as Tools: Investigating the Mother-Infant Dyad and Developmental Origins of Health and Disease Hypothesis using Vitamin D Deficiency. *American Journal of Physical Anthropology,* 171(2), 1-12.

Brickley, Megan B., Moffat, T., & Watamaniuk, L. (2014). Biocultural Perspectives of Vitamin D Deficiency in the past. *Journal of Anthropological Archaeology,* 36, 48-59.

Britton, K., McManus-Fry, E., Cameron, A., Duffy, P., Masson-MacLean, E., Czére, O., Smith, N., Stones, J., Winfield, A., & Müldner, G. (2018). Isotopes and New Norms: Investigating the Emergence of Early Modern U.K. Breastfeeding Practices at St. Nicholas Kirk, Aberdeen. *International Journal of Osteoarchaeology,* 28, 510-522.

Brunswick County Court, North Carolina. (1805, January). ORPHANS Benjamin, Bryan, Elizabeth Gause in Jan 1805 Brunswick Co. Minutes. *Minutes of Court of Pleas and Quarter Sessions.* Retrieved from https://www.ancestry.com/mediaui-viewer/tree/28038949/person/12036432093/media/920c89dc-28d0-49ca-9f47-57242ae7c58a

Burt, N. M. & Garvie-Lok, S. (2013). A New Method of Dentin Micro-sampling of Deciduous Teeth for Stable Isotope Ratio Analysis. *Journal of Archaeological Science,* 40, 3854-3864.

Censer, J. T. (1984). *North Carolina Planters and their Children: 1800-1860.* Louisiana State University Press.

Child. (1837). *The Family Nurse; or Companion of the Frugal Housewife*. Charles J. Hendee Co.

Chinique de Armas, Y. & Pestle, W. (2018). Assessing the Association between Subsistence Strategies and the timing of weaning among indigenous archaeological populations of the Caribbean. *International Journal of Osteoarchaeology*, 28, 492-509.

Christie Family Tree. (2020). Ancestry. Retrieved from https://www.ancestry.com/family-tree/person/tree/28038949/person/12097896786/facts

Cullen, E. N., & Owsley, D. (2011). Osteological Analysis of Remains from the Richards' Family Vault. *Stevenson University Forensics Journal*, 2, 33-39.

Czermak, A., Schermelleh, L., & Lee-Thorp, J. (2018). Imaging-Assisted Time-Resolved Dentine Sampling to Track Weaning Histories. *International Journal of Osteoarchaeology*, 28, 535-541.

Daughters of the American Revolution. (2020). *North America, Family Histories, 1500-2000 for Martha Gause*. Retrieved from https://bit.ly/32cnTfG

David, F. R., & Bender, V. J. (2009). *The Gause Plantation at Ocean Isle Beach Its Tomb & The Tomb Raiders*. Donning Company Publishers.

Dettwyler, K. A. (1995). A Time to Wean: The Hominid Blueprint for the Natural Age of Weaning in Modern Human Populations. In Patricia Stuart-Macadam & Katherine A. Dettwyler (Eds.), *Breastfeeding: Biocultural Perspectives* (pp. 39-74). Routledge.

Delanghe, J. R., Langlois, M. R., Buyzere, M. L., Na, N., Ouyang, J., Speeckaert, M. M., & Torck, M. A. (2011). Vitamin C deficiency: more than just a nutritional disorder. *Genes and Nutrition*, 6(4), 341-346.

DeNeale, W.M. H. (1855, November 27). Receiving Constantly. *The Tri-Weekly Commercial*. p. 3.

DeNiro, M. J. (1985). Postmortem Preservation and Alteration of in *Vivo* Bone Collagen Isotope Ratios in Relation to Paleodietary Reconstruction. *Nature*, 317, 806-809.

DeNiro, M. J., & Epstein, S. (1978). Influence of Diet on the Distribution of Carbon Isotopes in Animals. *Geochimica et Cosmochimica Acta*, 45, 341-351.

DeNiro, M. J. & Epstein, S. (1981). Influence of Diet on the Distribution of Nitrogen Isotopes in Animals. *Geochimica et Cosmochimica Acta*, 42, 495-506.

Doms, K., Hoseth A., Silber, B. H., Grettler, D. J., Gentile, S. M., & Faulls, F. D. (1995). *Archaeological investigations of the Scott's run project area, the Route 72/13 intersection improvements project area, and the Woodville Grave Site (7NC-E-98A), State Route 1 Corridor, New Castle County, Delaware* (Report). University of Delaware, Department of Anthropology, Center for Archaeological Research. Delaware Department of Transportation Archaeology Series No. 147.

D'Ortenzio, L., Kahlon, B., Peacock, T., Salahuddin, H., & Brickley, M. (2018). The rachitic tooth: Refining the use of interglobular dentine in diagnosing vitamin D deficiency. *International Journal of Paleopathology*, 22, 101-108.

D'Ortenzio, L., Ribot, I., Kahlon, B., Bertrand, B. Bocaege, E., Raguin, E., Schattmann, A., & Brickley, M. (2018). The rachitic tooth: The use of radiographs as a screening technique. *International Journal of Paleopathology*, 23, 32-42.

D'Ortenzio, L., Ribot, I., Raguin, E., Schattmann, A., Bertrand, B.,Kahlon, B., & Brickley, M. (2016). The Rachitic Tooth: A Histological Examination. *Journal of Archaeological Science,* 74, 152-163.

Dupras, T. L. & Tocheri, M. W. (2007). Reconstructing Infant Weaning Histories at Roman Period Kellis, Egypt Using Stable Isotope Analysis of Dentition. *American Journal of Physical Anthropology,* 134, 63-74.

Econs, M. J., & McEnery, P. T. (1997). Autosomal Dominant Hypophosphatemic Rickets/Osteomalacia: Clinical Characterization of a Novel Renal Phosphate-Wasting Disorder. *Journal of Clinical Endocrinology and Metabolism*, 82(2), 674-681.

Edward, E. (2019). *Metabolic Disease in Subadult Skeletal Remains from Late Ottoman-Era Tell Hisban, Jordan* (Master's Thesis, East Carolina University). Retrieved from the Scholarship.

Emery, M. V., Prowse, T. L., Schwarcz, H. P., & Brickley, M. (2015). Exploring Dietary Variability in a War of 1812 Skeletal Collection from Stoney Creek, Ontario, Using Stable Carbon and Nitrogen Isotopes. *Historical Archaeology*, 49(4), 54-70.

Fain, O. (2005). Review Musculoskeletal Manifestations of Scurvy. *Joint Bone Spine*, 72, 124-128.

Fildes, V. (1995). The Culture and Biology of Breastfeeding: An Historical Review of Western Europe. In P. Stuart-Macadam & K. A. Dettwyler (Eds.), *Breastfeeding: Biocultural Perspectives* (pp. 101-126). Aldine De Gruyter.

Find-a-Grave. (2009). *Samuel Frink*. https://www.findagrave.com/memorial/38773179

Flohr, S., & Shultz, M. (2009). Mastoiditis-Paleopathological Evidence of a Rarely Reported Disease. *American Journal of Physical Anthropology*, 138, 266-273.

Fogel, M. L., Tuross, N., & Owsley, D. W. (1989). Nitrogen Isotope Tracers of Human Lactation in Modern and Archaeological Populations. *Annual Report of the Director of the Geophysical Laboratory*, 88, 111-117.

France, C. A. M., Owsley, D. W. and Hayek, L. C. (2014). Stable Isotope Indicators of Provenance and Demographics in 18th and 19th century North Americans. *Journal of Archaeological Science*, 42, 356-366.

Frink, N. (1751, February 6). *South Carolina, Wills and Probate Records, 1670-1980* [deed]. Retrieved from https://bit.ly/3iY8JAj

Frink, S. (1811, May 11) *North Carolina, Land Grant Files, 1693-1960* [deed]. Retrieved from https://bit.ly/3027dEX

Frink, S. (1818, December 5) *North Carolina, Land Grant Files, 1693-1960* [deed]. Retrieved from https://bit.ly/3fo5i3X

Frink, S. (1819, November 25) *North Carolina, Land Grant Files, 1693-1960* [deed]. Retrieved from https://bit.ly/3gSLVjP

Frink, S. (1823, November 28) *North Carolina, Land Grant Files, 1693-1960* [deed]. Retrieved from https://bit.ly/3euDpGh

Frink, S. (1840, December 3) *North Carolina, Land Grant Files, 1693-1960* [deed]. Retrieved from https://bit.ly/32aQfXx

Frink, S. (1847, November 9) *North Carolina, Land Grant Files, 1693-1960* [deed]. Retrieved from https://bit.ly/3fAw9tP

Fuller, B. T., Fuller, J. L., Harris, D. A., & Hedges, R. E. M. (2006). Detection of Breastfeeding and Weaning in Modern Human Infants with Carbon and Nitrogen Stable Isotope Ratios. *American Journal of Physical Anthropology,* 129, 279-293.

Fuller, B. T., Richards, M. P., & Mays, S. A. (2003). Stable Carbon and Nitrogen Isotope Variations in Tooth Dentine Serial Sections from Wharram Percy. *Journal of Archaeological Science,* 30, 1673-1684.

Fuller, B. T., Fuller, J. L., Sage, N. E., Harris, D. A., O'Connell, T. C., & and Hedges, R. E. M. (2005). Nitrogen Balance and $\delta^{15}N$: Why You're Not What You Eat during Nutritional Stress. *Rapid Communications in Mass Spectrometry,* 19, 2497-2506.

Garland, C. J., & Reitsema, L. J. (2018). Variation in Early Life Diet and Stress during Early Spanish Colonization a Life History Perspective from Individuals Interred at the Fallen Tree Mortuary Site (9LI8) on St. Catherines Island, Georgia. *Early Georgia: The Society for Georgia Archaeology*, 46(1-2), 51-80.

Gause, B. (1790, January 27). *Deed for Sale of Enslaved Persons* [deed]. Retrieved from https://www.familysearch.org/ark:/61903/3:1:3QSQ-G98M-H9JX?i=371&cat=299183

Gause, B. (1790, May 3). *Deed of Sale for Enslaved Persons* [deed]. Retrieved from https://www.familysearch.org/ark:/61903/3:1:3QS7-998M-H9NZ?cat=299183

Gause, B. W. (1826, January 10). *North Carolina, Wills and Probate Records, 1665-1997 for Benjamin William Gause* [will]. Retrieved from https://bit.ly/2OkVRqa

Gause, B. W. (1804, March 24). *South Carolina, Wills and Probate Records, 1670-1980 for B. W. Gause* [will]. Retrieved from https://bit.ly/3iWeLSk

Gause, C. (1779, June 20). *North Carolina, Land Grant Files, 1693-1960* [deed]. Retrieved from https://bit.ly/2ZmqDVN

Gause, C. (1785, March 22). *North Carolina Land Grant Files, 1693-1960* [deed]. Retrieved from https://bit.ly/3fn3BDH

Gause, C. (1794, February 25). Record of Deeds from Brunswick County, North Carolina. Book C: 290.

Gause, C. (1808, August 27). *North Carolina, Wills and Probate Records, 1665-1998* [will]. Retrieved from https://bit.ly/2Ctb1Xw

Gause, J. (1777, May 29). Record of Deeds from Brunswick County, North Carolina. Book B: 86.

Gause, J. (1777, June). Record of Deeds from Brunswick County, North Carolina. Book B: 85.

Gause, J. (1783, June 12). *North Carolina, Wills and Probate Records, 1665-1998 for John Gause* [will]. Retrieved from https://bit.ly/3j3d2L5

Gause, J. J. (1788, September 29). Record of Deeds from Brunswick County, North Carolina. Book C: 318.

Gause, J. J. (1824, May 1). Record of Deeds from Brunswick County, North Carolina. Book J: 213.

Gause, J. J. (1836, September). *North Carolina, Wills and Probate Records, 1665-1998 for John Julius Gause* [will]. Retrieved from https://bit.ly/304lv7L

Gause, K. (2000). *Our Gause Roots*. Genealogy.com. https://www.genealogy.com/ftm/g/a/u/Kathy-Gause-TX/BOOK-0001/0002-0001.html

Gause, N. (1794, September 23). *North Carolina, Wills and Probate Records, 1665-1998* [will]. Retrieved from https://bit.ly/3iRWCoJ

Gause, P. (1823, December 23). North Carolina, Wills and Probate Records, 1665-1998 [will]. Retrieved from https://bit.ly/32cI90G

Gause, S. (1801, November 16). Record of Deeds from Brunswick County, North Carolina. Book D: 343.

Gause, S. (1811, July 4). *North Carolina, Wills and Probate Records, 1665-1998* [will]. Retrieved from https://www.ancestry.com/interactive/9061/007600424_00435?pid=2378118&treeid=&personid=&rc=&usePUB=true&_phsrc=Pku472&_phstart=successSource#?imageId=007600424_00501

Gause, W. (1770, April 9). *North Carolina Land Grant Files, 1693-1960* [deed]. Retrieved from https://bit.ly/2ZmtCgY

Gause, W. (1770, April 9). *North Carolina Land Grant Files, 1693-1960* [deed]. Retrieved from https://bit.ly/3etKWoM

Gause, W. (1778, June 19). *North Carolina Land Grant Files, 1693-1960* [deed]. Retrieved from https://bit.ly/2BUGNNp

Gause, W. (1783, October 15). *North Carolina Land Grant Files, 1693-1960* [deed]. Retrieved from https://bit.ly/2C3vthS

Gause, W. (1788, July 11). *North Carolina Land Grant Files, 1693-1960* [deed]. Retrieved from https://bit.ly/2W8KUfu

Gause, W. (1796 December 22). *North Carolina Land Grant Files, 1693-1960* [deed]. Retrieved from https://bit.ly/302fV62

Gause, W. (1801, May 9). *North Carolina, Wills and Probate Records, 1665-1998* [will]. Retrieved from https://bit.ly/2BU5VDX

Gause, W. J. (1824, December 24). *North Carolina, Wills and Probate Records, 1665-1998* [will]. Retrieved from https://www.ancestry.com/interactive/9061/007600424_00518?pid=2378359&treeid=&personid=&rc=&usePUB=true&_phsrc=Pku504&_phstart=successSource

Gause, W. Jr. (1858, March 17). *North Carolina, Wills and Probate Records, 1665-1998* [will]. Retrieved from https://www.ancestry.com/interactive/9061/007600424_00435?pid=2378118&treeid=&personid=&rc=&usePUB=true&_phsrc=Pku472&_phstart=successSource#?imageId=007600424_00467

Giuffra, V., Vitiello, A., Caramella, D., Fornaciari, A., Giustini, D., & Fornaciari, G. (2015). Rickets in a High Social Class of Renaissance Italy: The Medici Children. *International Journal of Osteoarchaeology*, 25, 608-624.

Goodman, A. H., & Rose, J. C. (1990). Assessment of Systemic Physiological Perturbations from Dental Enamel Hypoplasias and Associated Histological Structures. *Yearbook of Physical Anthropology*, 33, 55-110.

Google Maps. (2020). *Gause Cemetery*. Retrieved from https://bit.ly/2OgD6DZ

Gorrell. (1857, February 20). Speech of Mr. Gorrell. *The Greensboro Patriot*. p. 1.

Griffiths, J. (2020). Vitamin Deficiency. In E. Ryan, D. Hill, T. Solomon, T. Endy, & N. Aronson (Eds), *Hunter's Tropical Medicine and Emerging Infectious Diseases* (10th Ed.)(pp.1042-1047).

Grueger, Barbara, Canadian Paediatric Society, & Community Paediatrics Comittee. (2013). Weaning from the breast. *Paediatric & Child Health*, 18(4), 210.

Guatelli-Steinberg, D., Floyd, B. A., Dean, M. C., & Reid, D. J. (2012). Enamel Extension Rate Patterns in Modern Human Teeth: Two Approaches Designed to Establish an Integrated Comparative Context for Fossil Primates. *Journal of Human Evolution*, 63(3), 475-486.

Hasegawa, T., Li, M., Hara, K., Sasaki, M., Tabata, C., Luiz de Freitas, P., Hongo, H. Suzuki, R., Kobayashi, M., Inoue, K., Yamamoto, T., Oohata, N., Oda, K., Akiyama, Y., & Amizuka, N. (2011). Morphological assessment of bone mineralization in tibial metaphyses of ascorbic acid-deficient ODS rats. *Biomedical Research*, 32(4), 259-269.

Herrscher, E., Goude, G., & Metz, L. (2017). Longitudinal study of stable isotope compositions of maternal milk and implications for the Palaeo-diet of infants. *BMSAP Bulletins et mémoires de la Société d'anthropologie de Paris*., 29, 131–139.

Hillson, S. (2005). *Teeth* (2nd Ed.). Cambridge University Press.

Hillson, S. W. & Bond, S. (1997). Relationship of Enamel Hypoplasia to the Pattern of Tooth Crown Growth: a Discussion. *American Journal of Physical Anthropology*, 140, 89-104.

Holick, M. F. (1996). Vitamin D and Bone Health. *Journal of Nutrition,* 126 suppl. 4, 1159-1164.

Holick, M. F. (2006). Resurrection of Vitamin D Deficiency and Rickets. *The Journal of Clinical Investigation,* 116(8), 2062-2072.

Holland, T. D., & O'Brien, M. J. (1997). Parasites, Porotic Hyperostosis, and the Implications of Changing Perspectives. *American Antiquity*, 62(2), 183-193.

Johnson, J. (2020). *Jennifer Johnson Family Tree*. Ancestry. https://www.ancestry.com/family-tree/tree/157761356/family/familyview?cfpid=402073856717.

Katzenberg, M. A. (1993). Age Differences and Population Variation in Stable Isotope Values from Ontario, Canada. In J. B. Lambert & G. Grupe (Eds.), *Prehistoric Human Bone Archaeology at the Molecular Level* (pp. 39-62). Springer-Verlag.

Katzenberg, M. A. (2008). Stable Isotope Analysis: a tool for studying past diet, demography, and life history. In M. A. Katzenberg & S. R. Saunders (Eds.) *Biological Anthropology of the Human Skeleton* (pp. 413-411). John Wiley and Sons.

Katzenberg, M. A., Herring, D., & Saunders, S. R. (1996). Weaning and Infant Mortality: Evaluating the Skeletal Evidence. *Yearbook of Physical Anthropology*, 39, 177-199.

Katzenberg, M. A., & Lovell, N. C. (1999). Stable Isotope Variation in Pathological Bone. *International Journal of Osteoarchaeology*, 9, 316-324.

Katzenberg, M. A., & Pfeiffer, S. (1995). Nitrogen Isotope Evidence for Weaning Age in Nineteenth Century Canadian Skeletal Sample. In A. L. Grauer (Ed.), *Bodies of Evidence: Reconstructing History through Skeletal Analysis* (pp. 221-235). John Wiley and Sons.

Kelley, J. O., & Angel, J. L. (1987). Life Stresses of Slavery. *American Journal of Physical Anthropology,* 74, 199-211.

Kellner, C. M., & Schoeninger, M. J. (2007). A Simple Carbon Isotope Model for Reconstructing Prehistoric Human Diet. *Journal of Physical Anthropology*, 133, 1112-1127.

King, C. L., Snoddy, A. M., Millard, A. R., Grőcke, D. R., Standen, V. G., Arriaza, B. T., & Halcrow, S. E. (2018). A Multifaceted Approach Towards Interpreting Early Life Experience and Infant Feeding Practices in the Ancient Atacama Desert, Northern Chile. *International Journal of Osteoarchaeology*, 28, 599-612.

Klepp, S. E. (1994). Seasoning and Society: Racial Differences in Mortality in Eighteenth-Century Philadelphia. *The William and Mary Quarterly,* 51(3), 473-506.

Krapf, D., Krapf, S., & Wyss, C. (2015). Calcaneus secundarius – a relevant differential diagnosis in ankle pain: a case report and review of the literature. *Journal of Medical Case Reports*, 9(127), 1-7.

Landmark Preservation Associates. (2010). *Comprehensive Historical/Architectural Site Survey of Brunswick County, North Carolina*.

https://www.brunswickcountync.gov/files/planning/2015/04/plan_Final_Report_-_Brunswick_County_Comprehensive_Historical_Architectural_Site_Survey.pdf.

Larson, C. S. (2015). *Bioarchaeology; Interpreting behavior from the human skeleton* (2nd ed.). Cambridge University Press.

Lazarus, R. (1825, October 4). *Letter from Rachel Mordecai Lazarus to Maria Edgeworth*. North American Women's Letters and Diaries. https://nwld.alexanderstreet.com/philologic/NWLD/navigate/14788/1/?byte=13153.

Lazarus, R. (1828, November 3). *Letter from Rachel Mordecai Lazarus to Maria Edgeworth*. North American Women's Letters and Diaries. https://nwld.alexanderstreet.com/philologic/NWLD/navigate/14790/1/?byte=1926

Lee, I. (2011). Imaging Diagnosis of the Degenerative Spine. In D. H. Kim, Y. Kim, & K. Kim. (Eds.), *Minimally Invasive Percutaneous Spinal Techniques* (pp. 58-110). Sanders.

Lee, L. (1980). *The History of Brunswick County North Carolina*. Heritage Press.

Leavitt, J. W. (1986). *Brought to Bed: Childbearing in America: 1750 to 1950*. Oxford University Press.

Lewis, M. E. (2007). *The Bioarchaeology of Children: Perspectives from Biological and Forensic Anthopology*. Cambridge University Press.

Linda Family Tree. (2020). Ancestry. Retrieved from https://www.ancestry.com/family-tree/person/tree/12909/person/330014398122/facts?_phsrc=Pku567&_phstart=successSource

Littleton, J. (1998). A Middle Eastern Paradox: Rickets in Skeletons from Bahrain. *Journal of Paleopathology,* 10(1), 13-30.

Long, M. (2019). *Bioarchaeological Analysis of a Historic North Carolina Family Cemetery* (Master's Thesis, East Carolina University). Retrieved from the Scholarship.

Mayo Clinic Staff. (2019). *Weaning: Tips for Breast-feeding Mothers*. Mayo Clinic. Retrieved from https://www.mayoclinic.org/healthy-lifestyle/infant-and-toddler-health/in-depth/weaning/art-20048440

Mays, S., Brickley, M., & Ives, R. (2006). Skeletal Manifestations of Rickets in Infants and Young Children in Historic Population from England. *American Journal of Physical Anthropology,* 129, 362-374.

Mays, S. (2010). The Effects of Infant Feeding Practices on Infant and Maternal Health in a Medieval Community. *Childhood in the Past,* 3, 63-78.

McMillen, S. G. (1980). Education and Maternal Domesticity: Women in the Southern Social Order, 1830-1860. Master's Thesis, University of North Carolina Charlotte.

McMillen, S. G. (1990). *Motherhood in the Old South: Pregnancy, Childbirth, and Infant Rearing*. Louisiana State University Press.

Millard, A. R. (2000). A model for the effect of weaning on nitrogen isotope ratios in humans. In G. A. Goodfriend, M. J. Collins, M. Fogel, S. A. Macko, & J. F. Wehmiller (Eds.), *Perspectives in amino acid and protein geochemistry*. Oxford, UK: Oxford University Press.

Newman, S. L., & Gowland, R. L. (2017). Dedicated Followers of Fashion? Bioarchaeological Perspectives on Socio-Economic Status, Inequality, and Health in Urban Children from the Industrial Revolution (18th- 19th C), England. *International Journal of Osteoarchaeology*, 27, 217-229.

Nicholls, R., Buckberry, J., Beaumont, J., Črešnar, M., Mason, P., Armit, I., & Koon, H. (2020). A Carbon and Nitrogen Isotopic Investigation of a Case of Probable Infantile Scurvy (6th-4th centuries BC, Slovenia). *Journal of Archaeological Science Reports*, 30, 1-11.

Norton, R. (1994). Maternal Nutrition during Pregnancy as it Affects Infant Growth, Development and Health. *SCN News*, (11), 76.

Omstead, F. L. (1863). *A Journey in the Seaboard Slave States, with Remarks on the Economy*. Mason Brothers. https://www.loc.gov/resource/lhbtn.35036/?st=gallery

Ortner, D. J. (2003). *Identification of Pathological Conditions in Human Skeletal Remains* (2nd ed.). Academic Press.

Ortner, D. J., & Mays, S. (1998). Dry-bone Manifestations of Rickets in Infancy and Early Childhood. *International Journal of Osteoarchaeology*, 8, 45-55.

Owsley, D. W., Bruwelheide, K. S., Barca, K. G., Reidy, S. K., & Fleskes, R. E. (2018). Lives Lost: What Burial Vault Studies Reveal about Eighteenth-Century Identities. In P. K. Stone (Ed.) *Bioarchaeological Analyses and Bodies: New Ways of Knowing Anatomical and Archaeological Skeletal Collections* (pp. 111-145). Springer.

Pinnell, S. R. (1985). Regulation of Collagen Biosynthesis by Ascorbic Acid: A Review. *The Yale Journal of Biology and Medicine,* 58, 553-559.

Popovich, D., McAlhany, A., Adewumi, A., & Barnes, M. (2009). Scurvy: Forgotten but Definitely Not Gone. *Journal of Pediatric Health Care*, 23(6), 405-415.

Price, T. D. (2015). An Introduction to the Isotopic Studies of Ancient Human Remains. *Journal of the North Atlantic,* 7, 71-87.

Quintana, J. R. (2019). *As the Sun Sets, We Remain: A Bioarchaeological Analysis of the Gause Cemetery at Seaside* (Master's Thesis, East Carolina University). Retrieved from the Scholarship.

Rajakumar, K. (2001). Infantile Scurvy: A Historical Perspective. *Journal of the American Academy of Pediatrics*, 108(4), 1-3.

Rathburn, T. A. (1987). Health and Disease at a South Carolina Plantation 1840-1870. *American Journal of Physical Anthropology,* 74, 239-253.

Raynor, L. A., & Kennett, D. J. (2008). Dietary Variability among a Sample of United States Soldiers during the War of 1812. *Historical Archaeology*, 42(4), 76-87.

Reilly, R. F. (2016). Medical and Surgical Care during the American Civil War 1861-1865. *Proceedings (Baylor University. Medical Center)*, 29(2), 138–142.

Reitsema, L. (2013). Beyond Diet Reconstruction: Stable Isotope Applications to Human Physiology, Health, and Nutrition. *American Journal of Human Biology,* 25, 445-456.

Reitsema, L. J., & Mcllvaine, B. K. (2014). Reconciling "Stress" and "Health" in Physical Anthropology: What Can Bioarchaeologists Learn From the Other Subdisciplines?. *American Journal of Physical Anthropology*, 155, 181-185.

Rivera, F., & Lahr, M. M. (2017). New evidence suggesting a dissociated etiology for cribra orbitalia and porotic hyperostosis. American Journal of Physical Anthropology, 76-96.

Roser, M., Ritchie, H., & Dadonaite, B. (2019). *Child and Infant Mortality*. Our World in Data. Retrieved from https://ourworldindata.org/child-mortality#mortality-in-the-past-around-half-died-as-children

Ross, A. (1993). Health and Diet in 19th-Century America: A Food Historian's Point of View. *Historical Archaeology,* 27(2), 42-56.

Ross, L. (1981). Weaning Practices. *Journal of Nurse-Midwifery*, 26(1), 9-14.

Sandberg, P. A., Sponheimer, M., Lee-Thorp, J., & Van Gerven, D. (2014). Intra-tooth stable isotope analysis of dentine: A step toward addressing selective mortality in the reconstruction of life history in the archaeological record. *American Journal of Physical Anthropology*, 155, 281–293.

Saunders, S. R., Herring, A., Sawchuk, L., Boyce, G., Hoppa, R., & Klepp, S. (2002). The Health of the Middle Class: The St. Thomas Anglican Church Cemetery Project. In R. H. Steckel & J. C. Rose (Eds.), *The Backbone of History: Health and Nutrition in the Western Hemisphere* (pp. 130-161). Cambridge University Press.

Saunders, S. R., Chan, A. H. W., Kahlon, B., & Kluge, H. F. (2007). Sexual Dimorphism of the Dental Tissues in Human Permanent Mandibular Canines and 3rd Molars. *American Journal of Physical Anthropology,* 133(1), 735-740.

Schmidt, W. M. (1976). Health and Welfare of Colonial American Children. *American Journal of Diseases of Children,* 130, 694-701.

Schurr, M. R. (1997). Stable Nitrogen Isotopes as Evidence for the Age of Weaning at the Angel Site: A Comparison of Isotopic and Demographic Measures of Weaning Age. *Journal of Archaeological Science,* 24, 919-927.

Schurr, M. R. (2010). Using Stable Nitrogen-Isotopes to Study Weaning Behavior in Past Populations. *World Archaeology,* 30(2), 327-342.

Schurr, M. R. (2018). Exploring Ideas about Isotopic Variation in Breastfeeding and Weaning Within and Between Populations: Case Studies from the American Midcontinent. *International Journal of Osteoarchaeology*, 28, 479-491.

Schoeninger, M. J. & DeNiro, M. J. (1984). Nitrogen and Carbon Isotopic Composition of Bone Collagen from Marine and Terrestrial Animals. *Geochimica et Cosmochimica Acta,* 48, 625-639.

Schoeninger, M. J., DeNiro, M. J., & Tauber, H. (1983). Stable Nitrogen Isotope Ratios of Bone Collagen Reflect Marine and Terrestrial Components of Prehistoric Human Diet. *American Association for the Advancement of Science,* 220(4604), 1381-1383.

Schoeninger, M. J. & Moore, K. (1992). Bone Stable Isotope Studies in Archaeology. *Journal of World Prehistory,* 6(2), 247-296.

Seeman, M. (2011). *An Osteobiographical Analysis of the Foscue Plantation Burial Vault Pollocksville, Jones County, North Carolina* (Master's Thesis, East Carolina University). Retrieved from the Scholarship.

Sharma, M., Singh, B., Abhaya, A., & Kumar, H.. (2008). Occipitalization of Atlas with Other Associated Anomalies of Skull. *European Journal of Anatomy*, 12(3), 159-167.

Silk, H., Douglass, A. B., Douglass, J. M., & Silk, L. (2008). Oral Health during Pregnancy. *American Family Physician*, 77(8), 1139-1144.

Slattery, B. (2015, December 1). Gause's Tomb re-emerges after restoration by descendants. *The Brunswick* Beacon. https://www.brunswickbeacon.com/content/gause%E2%80%99s-tomb-re-emerges-after-restoration-descendants

Smith, B. (2011, March 7). *Capt Charles Gause*. Find A Grave. https://www.findagrave.com/memorial/66592161

Southern Historical Collection. (1853-1873). *Mary Jeffreys Bethell Dairy* (#01737-z). University of North Carolina Chapel Hill, Chapel Hill.

Sprunt, J. (1916). *Chronicles of the Cape Fear River, 1660-1916* (2nd ed.). Edwards and Broughton.

State of Agricultural Society of North Carolina. (1855, November 9). Address of Hon. Thomas Ruffin Alamance. *Wilmington Journal*. p. 2.

State of North Carolina. (1807). *North Carolina, Index to Marriage Bonds, 1741-1868*. 16 July, 1807 Bond # 4513.

Swift, J. (1904). Letters and Documents Relating to the Early History of the Lower Cape Fear, with Introduction and Notes. In K. P. Battle (Ed.), *James Sprunt Historical Monograph* (4). University of North Carolina Press.

The Complete Genealogy Reporter. (2016). *A Genealogy Report for Dawson Blake Gause*. Retrieved from MyHeritage.com

The North Carolina Daughters of the American Revolution. (1932). *Roster of Soldiers from North Carolina in the American Revolution*. Seeman Press.

The Weekly Standard. (1844, May 29). *The Weekly Standard, Raleigh, N.C.* p. 3.

The Wilmington Daily Herald. (1855, October 8). *The Wilmington Daily Herald (Wilmington, North Carolina)*. p. 4.

Todd, S. M. (n.d.). *William Gause Genealogy*. Webtrees. https://webtrees.mstevetodd.com/mediafirewall.php?mid=M53&ged=M%20Steve%20Todd&cb=591203a2

Todd, T. (2020). *Todd Family Tree*. Ancestry. https://www.ancestry.com/family-tree/person/tree/16106562/person/29120720284/facts

Trinkley, M. & Hacker, D. (2015). *Bioanthropological Investigation of the Vaults at Orton Plantation, Brunswick County, North Carolina*. Belvedere Property Management.

Tsutaya, T., & Yoneda M. (2015). Reconstruction of Breastfeeding and Weaning Practices Using Stable Isotope and Trace Element Analyses: a review. *Yearbook of Physical Anthropology,* 156, 2-21.

Tykot, R. H. (2018). Bone chemistry and ancient diet. In C. Smith (Ed.), *Encyclopedia of Global Archaeology* (2nd ed.) (pp. 1-11). Springer.

Ubelaker, D. H., & Owsley, D. W. (2003). Isotopic Evidence for Diet in the Seventeenth-Century Colonial Chesapeake. *American Antiquity*, 68(1), 129-139.

Ubelaker, D. H., Jones, E. B., & Turowski, A. W. (1996). Skeletal Biology of the Patuxent Point Human Remains. In J. King & D. H. Ubelaker (Eds.) (pp. 47-104). The Maryland Historical Trust Press.

U.S. Census Bureau. (1790). *Brunswick County, N.C. 1790 U.S. Census*. Retrieved from http://us-census.org/pub/usgenweb/census/nc/brunswick/1790/pg0022.txt

U.S. Census Bureau. (1800). *Georgetown District, S.C. 1800 United States Federal Census*. Retrieved from https://bit.ly/32cuCpB

U.S. Census Bureau. (1820). *Jefferson, Georgia 1820 United States Federal Census*. Retrieved from https://bit.ly/306tDVx

U.S. Census Bureau. (1830). *Brunswick County, N. C. 1830 U.S. Census*. Retrieved from https://bit.ly/2WcvXca

U.S. Census Bureau. (1850). *Lauderdale County, TN 1850 U.S. Census*. Retrieved from https://bit.ly/32hsNbj

U.S. Census Bureau. (1850-1885). *U.S. Federal Census Mortality Schedules for Brunswick County, NC*. Retrieved from https://www.ancestry.com/search/collections/8756/

U.S. Census Bureau. (1860). *Agriculture, The United States 1860; Compiled from the Original Returns of the Eighth Census*. Retrieved from https://www.census.gov/library/publications/1864/dec/1860b.html

U.S. Census Bureau. (1860). *Barbour County, AL 1860 U.S. Census*. Retrieved from https://bit.ly/2Wcndmp

U.S. Census Bureau. (1860). *Mortality of the United States, 1860*. Retrieved from https://www2.census.gov/library/publications/decennial/1860/statistics/1860d-10.pdf

van der Merwe, N. & Vogel, J. C. (1978). ^{13}C Content of Human Collagen as a Measure of Prehistoric Diet in Woodland North America. *Nature,* 276, 815-816.

Veselka, B., Hoogland, M. L. P., & Waters-Rist, A. L. (2015). Rural Rickets: Vitamin D Deficiency in a Post-Medieval Farming Community from the Netherlands. *International Journal of Osteoarchaeology,* 25, 665-675.

Victora, C. G., Bahl, R., Barros, A. J. D., França, G. V. A., Horton, S., Krasevec, J., Murch, S., Sankar, M. J., Walker, N., & Rollins, N. C. (2016). Breastfeeding 1: Breastfeeding in the 21st century: epidemiology mechanisms, and lifelong effect. *The Lancet,* 387, 475-490.

Vigeant, J., Ribot, I., & Hélie, J. F. (2017). Dietary Habits in New France during the 17th and 18th Centuries: an Isotopic Perspective. *American Journal of Physical Anthropology,* 162, 462-475.

Viteri, F. E. (1994). The Consequences of Iron Deficiency and Anaemia in Pregnancy on Maternal Health, the Foetus and the Infant. *SCN News,* (11), 76.

Waldron, T. (2009). *Paleopathology*. Cambridge University Press.

Walker, P. L., Bathurst, R. R., Richman, R., Gjerdrum, T., & Andrushko, V. A. (2009). The Causes of Porotic Hyperostosis and Cribra Orbitalia: A Reappraisal of the Iron-Deficiency-Anemia. *American Journal of Physical Anthropology,* 139, 109-125.

Webb, E. C., White, C. D., Uum, S. V., & Longstaffe, F. J. (2015). Integrating Cortisol and Isotopic Analyses of Archaeological Hair: Reconstructing Individual Experiences of Health and Stress. *American Journal of Physical Anthropology*, 156, 577-594.

Wolf, J. H. (2018). They Lacked the Right Food: A Brief History of Breastfeeding and the Quest for Social Justice. *Journal of Human Lactation,* 34(2), 226-231.

Wood, L. (1996). Frequency and Chronological Distribution of Linear Enamel Hypoplasia in a North American Colonial Skeletal Sample. *American Journal of Physical Anthropology*, 100, 247-259.

World Health Organization (WHO). (2003). *Global Strategy for Infant and Young Child Feeding, World Health Organization, Geneva*. Retrieved from https://www.who.int/nutrition/publications/infantfeeding/9241562218/en/

World Health Organization (WHO). (2020). *Infant and Young Child Feeding*. Retrieved from https://www.who.int/news-room/fact-sheets/detail/infant-and-young-child-feeding

Yan, Y., Calikoglu, A. S., & Jain, N. (2011). Vitamin D-Dependent Rickets Type 1: A Rare, but Treatable, Cause of Severe Hypotonia in Infancy. *Journal of Child Neurology*, 26(12), 1571-1575.

APPENDIX

$\delta^{13}C$ and $\delta^{15}N$ Values from Comparison Sites

Site	Individual	Age	Time period	Location	Type of tissue	$\delta^{15}N$	$\delta^{13}C$	Reference
Darnall's Chance House	Burial 1	27-32	18th to 19th century	Maryland	bone	11.9	-14.7	Owsley et al., 2018
Darnall's Chance House	Burial 1	27-32	18th to 19th century	Maryland	bone	11.0	-14.7	Owsley et al., 2018
Darnall's Chance House	Burial 2	45-54	18th to 19th century	Maryland	bone	12.3	-14.7	Owsley et al., 2018
Darnall's Chance House	Burial 2	45-54	18th to 19th century	Maryland	bone	12.0	-15.3	Owsley et al., 2018
Darnall's Chance House	Burial 3	20-22	18th to 19th century	Maryland	bone	11.2	-14.3	Owsley et al., 2018
Darnall's Chance House	Burial 3	20-23	18th to 19th century	Maryland	bone	10.9	-13.7	Owsley et al., 2018
Darnall's Chance House	Burial 4	12-14 years	18th to 19th century	Maryland	bone	11.1	-14.0	Owsley et al., 2018

Darnall's Chance House	Burial 5	7-8 years	18th to 19th century	Maryland	bone	10.1	-13.9	Owsley et al., 2018
Foscue	31FOSCUE-ECU-03	34-38	1800-1849	North Carolina	bone	10.5	-13.1	Seeman, 2011
Foscue	31FOSCUE-ECU-04	60+	1800-1850	North Carolina	bone	11.3	-14.3	Seeman, 2011
Foscue	31FOSCUE-ECU-05	60+	1800-1851	North Carolina	bone	11.0	-12.7	Seeman, 2011
Moore family cemetery - Orton Plantation	Vault 1 individual A	40-60	18th century	North Carolina	bone	11.3	-14.3	Trinkley & Hacker, 2015
Moore family cemetery - Orton Plantation	Vault 2 individual A	15-20	18th century	North Carolina	bone	10.7	-15.6	Trinkley & Hacker, 2015
Moore family cemetery - Orton Plantation	Vault 3 Individual A	40-45	18th century	North Carolina	bone	11.3	-15.7	Trinkley & Hacker, 2015
Moore family cemetery - Orton Plantation	Vault 4 Individual A	50+	18th century	North Carolina	bone	10.9	-14.5	Trinkley & Hacker, 2015
Richards Family Vault	Burial 2	25 (based on records)	1851-1920 (died 1851)	Washington D.C	bone	12.0	-13.6	Cullen & Owsley, 2011

Richards Family Vault	Burial 5	39 (based on records)	1851-1920 (died 1865)	Washington D.C	bone	12.7	-13.1	Cullen & Owsley, 2011
Richards Family Vault	Burial 6	72 (based on records)	1851-1920 (died 1894)	Washington D.C	bone	11.6	-14.6	Cullen & Owsley, 2011
Richards Family Vault	Burial 7	48 (based on records)	1851-1920 (died 1892)	Washington D.C	bone	12.0	-12.8	Cullen & Owsley, 2011
Richards Family Vault	Burial 15	13 (based on records)	1851-1920 (died 1920)	Washington D.C	bone	11.3	-16.4	Cullen & Owsley, 2011
Woodville Cemetery	7NCE98A-WOODVILLE-08	late middle age	1790-1850	Delaware	bone	10.9	-10.9	Doms et al., 1995; France et al., 2014
Woodville Cemetery	7NCE98A-WOODVILLE-12	advanced age 60+	1790-1850	Delaware	bone	12.0	-14.3	Doms et al., 1995; France et al., 2014
Woodville Cemetery	7NCE98A-WOODVILLE-01	50+	1790-1850	Delaware	bone	11.2	-11.3	Doms et al., 1995; France et al., 2014
Woodville Cemetery	7NCE98A-WOODVILLE-03	40+	1790-1850	Delaware	bone	10.3	-13.6	Doms et al., 1995; France et al., 2014
Woodville Cemetery	7NCE98A-WOODVILLE-04	middle age	1790-1850	Delaware	bone	11.4	-11.6	Doms et al., 1995; France et al., 2014

Woodville Cemetery	7NCE98A-WOODVILLE-10	50s+	1790-1850	Delaware	bone	11.2	-10.1	Doms et al., 1995; France et al., 2014