Measuring atomic bomb-derived ^{14}C levels in human remains to determine Year of Birth and/or Year of Death

Award Number: 2005-IJ-CX-K013

Author(s): Gregory W. L. Hodgins

Abstract

Between 1955 and 1963 above-ground nuclear testing generated high concentrations of radioactive carbon (^{14}C) in the earth's atmosphere. After a 1964 moratorium on above ground testing, atmospheric levels of ^{14}C began falling as the radioactivity passed from the atmosphere into the oceans and the biosphere. Virtually every organism living since the 1950's is labeled with higher than normal levels of ^{14}C, including humans. The rapid year-to-year changes in atmospheric levels of ^{14}C within this time frame, combined with rapid transfer of atmospheric ^{14}C into the food chain via photosynthesis, mean that the distribution of bomb-derived ^{14}C within the humans depends upon birth year, diet, and the dynamics of tissue replacement within the human body. The radiocarbon content of tissues is fixed at the time of death. Thus, the distribution of ^{14}C in different tissue compartments can potentially be used to determine Year-of-Birth and/or Year-of-Death for people who lived and died in the past half century.

This study measured ^{14}C-levels in post mortem tissues from 36 humans whose birth and death dates were known. Ultimately 276 measurements were completed on nine different tissue fractions: tooth enamel, bone apatite, bone collagen, bone lipid, skin collagen, skin lipid, hair, nails, and blood. Different tissues were measured to determine Year-of-Birth and Year-of-Death. The radiocarbon content of tooth enamel was examined as a potential indicator of Year-of-Birth. Tooth enamel does not turnover during life and, for teeth

formed after 1965, enamel radiocarbon content was found to be close to the atmospheric level at the time of tooth formation. The method proved robust for individuals born approximately after 1960. Radiocarbon levels in teeth formed before 1960 were found to contain less radiocarbon than expected. The method predicted Year-of-Birth within 1.5 years of actual birth for 8 of 15 individuals and but inaccuracies increased up to five years in cases where people were born in the 1950s. The accuracy of Year-of-Birth prediction could be improved quite simply if comparisons were made between unknown-age and known-age teeth, rather than comparisons between unknown-age teeth and atmospheric radiocarbon levels.

The radiocarbon content of soft tissues was also measured to examine whether these might be used to determine Year-of-Death. All soft tissues were obtained from individuals who died in 2006. The measurements quantified the variability in radiocarbon levels within a particular tissue within the population. Bone mineral, bone lipid, bone protein, skin protein, skin lipid, hair, fingernail, and blood were subjected to ^{14}C measurement. Of these, blood, nail, and hair radiocarbon levels were found to be closest to the contemporary atmosphere. Variability in the tissue levels due to genetic or dietary factors was not evident. This implies that soft tissue ^{14}C is a good indicator of Year of Death. However, the 36 individuals whose tissues were measured were all from South Eastern United States, and so geographic variability wasn't tested.

At the time of the study, peer-reviewed atmospheric radiocarbon measurement data was only available up to June 2003, so tissue levels had to be compared to extrapolated atmospheric curves.

Differences between atmospheric radiocarbon levels and soft tissue radiocarbon levels reflect the time required for ^{14}C transfer from one compartment to the other. Thus the relative rates of tissue replacement were determined. Absolute replacement rates for soft tissues were not calculated because of the lack of contemporary atmospheric data. Nevertheless, extrapolations bracketing the true values indicated that blood, hair and nail radiocarbon levels lagged atmospheric levels by 0 to 3 years, consistent with a rapid replacement of these tissues. Bone lipids radiocarbon levels lagged atmospheric levels by 6.8 years. The precision of Year-of-Death determinations was found to be of the order of ± 3 years for nails and ± 4 years for bone lipids for the year 2006. These precision estimates encompass 95% of the tissue population variability.

The precision Year-of-Death estimates depends upon the year of measurement. Precision is highest in the 1960's and 1970's, and is progressively decreasing over the past 50 years. The variability was modeled to illustrate the pattern of precision change. As levels approach natural environmental levels, the precision of tissue age determinations based upon radiocarbon content will decrease. Barring future nuclear catastrophe, it would be reasonable to expect the method could be used for Year-of-Birth determinations for people born within the next one or two decades.

Stable carbon and nitrogen isotope measurements on bone collagen from the donor population provided no evidence of diets that might confound the approach. Stable isotope measurements identified one individual whose diet was isotopically characteristic of a C4 vegetarian. Coincidently this person was the only Hispanic in the study. Although very preliminary, these results suggest that dietary stable isotopes might have forensic applications.

The primary advantage of using tissue radiocarbon content to estimate Year-of-Birth and Year-of-Death is that it focuses on a parameter determined by a global environmental phenomenon: namely a uniform worldwide distribution of atmospheric radiocarbon whose level changes measurably on a yearly basis. The transfer of bomb-derived radiocarbon from the atmosphere into tissues via photosynthesis and the food chain generates a global time-stamp for organisms living over the last 60 years. All of this, combined with the fact that levels of bomb radiocarbon in tissues are not significantly altered after death either by radioactive, biological, or chemical decay means the method is robust, regardless of variations in local conditions.

Decay of soft tissues presents the greatest challenge to Year-of-Death estimations based upon ^{14}C. An innovative direction for future research would be to examine the possibility that Year-of-Death estimations might be generated from radiocarbon measurements on the pupal cases of insects that feed on post mortem soft tissues. Pupal cases are composed of chitin, a protein/carbonhydrate complex that is nearly as resistant to decay as bone.

Table of Contents

Executive Summary

Between 1955 and 1963 above-ground nuclear testing doubled radioactive carbon (^{14}C) in the earth's atmosphere. After a 1964 moratorium on above ground testing, atmospheric levels of ^{14}C began falling as the radioactivity passed from the atmosphere into the oceans and the biosphere. Virtually every organism living since the 1950's is labeled with higher than natural levels of ^{14}C, including humans. The rapid year-to-year changes in atmospheric levels of ^{14}C within this time frame, combined with rapid transfer of atmospheric ^{14}C into the food chain via photosynthesis, mean that the distribution of bomb-derived ^{14}C within humans depends upon birth year, diet, and the dynamics of tissue replacement within the human body. The radiocarbon content of tissues is fixed at the time of death. Thus, the distribution of ^{14}C in different tissue compartments can potentially be used to determine Year-of-Birth and/or Year-of-Death for people who lived and died in the past half century.

Using the technique known as accelerator mass spectrometry, this study measured ^{14}C-levels in post mortem tissues from 36 humans whose birth and death dates were known. Ultimately 276 measurements were completed on nine different tissue fractions: tooth enamel, bone apatite, bone collagen, bone lipid, skin collagen, skin lipid, hair, nails, and blood. This was significantly more than the five tissues originally proposed but scientifically well worth the additional effort. Different tissues were measured to determine Year-of-Birth and Year-of-Death. The radiocarbon content of tooth enamel was examined as a potential indicator of Year-of-Birth. Tooth enamel does not turnover during life and, for teeth formed after 1965, enamel radiocarbon content was found to be close to the atmospheric level at the time of tooth formation. The method proved robust

for individuals born after 1960. Radiocarbon levels in teeth formed before 1960 were found to contain less radiocarbon than expected. The method predicted Year-of-Birth within 1.5 years of actual birth for 8 of 15 individuals and but inaccuracies increased up to five years in cases where people were born in the 1950s. The accuracy of Year-of-Birth prediction could be improved quite simply if comparisons were made between unknown-age and known-age teeth, rather than comparisons between unknown-age teeth and atmospheric radiocarbon levels.

The radiocarbon content of soft tissues was also measured to examine whether these might be used to determine Year-of-Death. All of the individuals in this study died in 2006, so the soft tissue measurements quantified the variability in radiocarbon levels within a particular tissue within the population. Bone mineral, bone lipid, bone protein, skin protein, skin lipid, hair, fingernail, and blood were subjected to ^{14}C measurement. Of these, blood, nail, and hair radiocarbon levels were found to be closest to the contemporary atmosphere. Variability in the tissue levels within the population was smaller than expected, and in fact was similar in size to the variability in atmospheric measurement data. This implies that soft tissue ^{14}C is a good indicator of Year of Death: expected variability due to genetic or dietary factors was not evident. The main caveat to this assertion is that the 36 individuals were all from South Eastern United States, and so variability in the total US population might be higher.

At the time of the study, peer-reviewed atmospheric radiocarbon measurement data was only available up to June 2003, so tissue levels had to be compared to extrapolated atmospheric curves. The lack of most recent data was thought to be trivial as direct

measurements on archived plant materials such as seeds from seed banks, could be carried out to fill in the missing measurements for 2003 to the present.

The difference between atmospheric radiocarbon levels and soft tissue radiocarbon levels reflected the time required for ^{14}C transfer from one location to the other. Levels also varied within each individual, reflecting the fact that different tissue compartments have different replacement rates. Absolute replacement rates could not be determined because atmospheric radiocarbon measurements for 2006 have not yet been published. However, they were estimated using extrapolations of atmospheric data sets from 1970 to 2003, and these estimations indicated that blood, hair and nail radiocarbon levels lagged atmospheric levels by 0 to 3 years, consistent with a rapid replacement of these tissues. Bone lipids radiocarbon levels lagged atmospheric levels by 6.8 years. The precision of Year-of-Death determinations was found to be of the order of ± 3 years for nails and ± 4 years for bone lipids for the year 2006. These precision estimates encompass 95% of the tissue population variability. Similar to conventional radiocarbon dating, the precision Year-of-Death estimates depends upon the year of measurement. Precision is highest in the 1960's and 1970's, and is progressively decreasing over the past 50 years. The variability was modeled to illustrate the pattern of precision change. The method does have a limited time frame because the bomb pulse has nearly been removed from the atmosphere. As levels approach natural environmental levels, the precision of tissue age determinations based upon radiocarbon content will decrease to the point of being useless. Barring future nuclear catastrophe, it would be reasonable to expect the method could be used for Year-of-Birth determinations for people born within the next one or two decades.

Individuals who intensively consume marine-foods have depressed tissue radiocarbon levels compared to those whose diet is derived principally from land-based resources. This could, at least theoretically, confound this method. However, stable carbon and nitrogen isotope measurements on bone collagen from the donor population provided no evidence of marine food intake, so it was concluded that the effect was not significant. Interestingly, the stable isotope measurements identified one individual whose diet was isotopically different from the rest of the sample population: and characteristic of a C4 vegetarian. Coincidently this person was the only individual of Hispanic origin in the study. Although very preliminary, these results suggest that dietary stable isotopes might have forensic applications.

The primary advantage of using tissue radiocarbon content to estimate Year-of-Birth and Year-of-Death is that it focuses on a parameter determined by a global environmental phenomenon: namely a uniform worldwide distribution of atmospheric radiocarbon whose level changes measurably on a yearly basis. The transfer of bomb-derived radiocarbon from the atmosphere into tissues via photosynthesis and the food chain generates a global time-stamp for organisms living over the last 60 years. All of this, combined with the fact that levels of bomb radiocarbon in tissues are not significantly altered after death either by radioactive, biological, or chemical decay means the method is robust, regardless of variations in local conditions.

Local environmental conditions of temperature, moisture, surrounding biota etc. are nevertheless important because these determine tissue survival. Decay of soft tissues presents the greatest challenge to Year-of-Death estimations based upon ^{14}C. An

innovative direction for future research would be to examine the possibility that Year-of-Death estimations might be generated from radiocarbon measurements on the pupal cases of insects that feed on post mortem soft tissues. Pupal cases are composed of chitin, a protein/carbonhydrate complex that is nearly as resistant to decay as bone.

It was originally conceived that the formation year of the tissues would be determined by the process of comparing tissue radiocarbon measurements to atmospheric radiocarbon levels, taking into account pre-determining the lag times for the transfer of atmospheric radiocarbon into tissues. However, it seems that the more direct method of comparing levels in known-age and unknown-age tissues would be preferable. For example, Year-of-Birth would be determined for an unidentified human by comparing radiocarbon levels in their teeth to levels in teeth from individuals with known Year-of-Birth. This seems an achievable goal in the case of teeth, but significantly more challenging for soft tissues. The challenge for soft tissues would be locating materials from the last six decades from which to make the known-age measurements. In this case, comparison between tissue levels and atmospheric levels may be the only way forward.

I. Introduction

Research problem

Four fundamental objectives of forensic anthropology focus on time: the establishment of post mortem interval, determination of age, determination of Year-of-Birth and Year-of-Death. Very sophisticated methods have been developed for determining post mortem interval within the timeframe of hours to weeks. These methods are based upon the chemistry and biology of decay. Their accuracy and precision can be astounding. The principles underlying most methods is that time is measured in terms of some chemical or biological process, for example hemoglobin breakdown, tissue oxidation, entomological succession etc. Such processes are inescapably influenced by extrinsic environmental factors such as temperature, environmental moisture levels, and local biota. These factors vary significantly over time and space. Endeavoring to provide chronological information based upon comparisons between the chemical or biological state of benchmark cases and unknowns becomes increasingly inaccurate as post mortem interval increases. What is required for determinations of long post mortem intervals is a chronometer that is unaffected by local environmental conditions.

This project investigates the possibility that atomic bomb-derived radiocarbon, integrated into the tissues of all humans, indeed all living things in the post nuclear age, provides a chronometer that can be used to determine on the order of years, Year-of-Birth, Year-of-Death, and consequently post mortem interval. The principle of the method is based upon the fact that above ground nuclear testing raised atmospheric ^{14}C levels in the late 1950's and early 1960's well above natural levels. The rapid rise and

subsequent decline of atmospheric ^{14}C concentrations has been referred to as the 'bomb-spike' (figure 1).

Bomb ^{14}C has been passing through all living organisms. It enters plant tissues via photosynthesis and animal tissue (including human tissue) through the food chain. The bomb spike was global phenomenon, and so it can serve as a universal time marker: people born in different years accumulate differing amounts of that radiocarbon in their tissues. Tissues formed around the year of birth potentially mark birth year, tissues formed around the time of death mark the Year of Death. As long as tissues survive in the post mortem period, their radiocarbon levels mark when they were formed. Thus the bomb-spike has significant potential as a forensic tool.

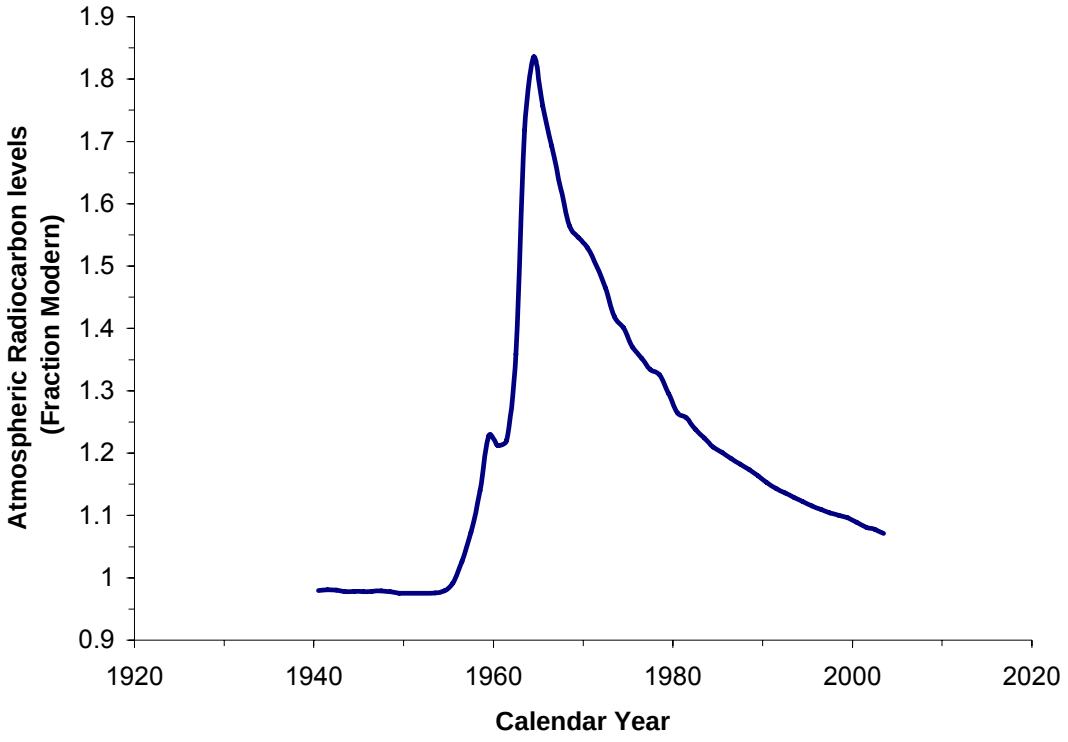

Figure 1: Northern Hemisphere Atmospheric Radiocarbon Levels 1940 to 2003AD. The levels are expressed in terms of Fraction Modern, where 1.0 FM (Fraction Modern) represents the level of atmospheric radiocarbon in 1950 AD without anthropogenic influence. Archaeological material 5730 years or one half-life old, contains 0.5 FM radiocarbon by definition: two halflives, 0.25FM, etc. This scale is used for expressing

recent radiocarbon measurements because 1950 AD was selected as the "zero year" by convention. FM avoids the paradox of expressing radiocarbon dates from the past 60 years in terms of negative years. The curve is a composite of data sets from Stuiver et al. (1998) and Levin and Kromer (2004), and has been annually smoothed.

The principle is straight forward, however predicting precision and accuracy is not. In the context of conventional radiocarbon dating (ie archaeological applications) precision and accuracy vary in a complex manner through time (e.g. Reimer *et al.* 2004). Although the proposed method is fundamentally different from conventional radiocarbon dating, it shares this characteristic.

The basic questions to be addressed are:

What tissues can be used to determine Year-of-Birth?

What tissues are best suited to determination of Year-of-Death?

How much variability is there in radiocarbon levels in a specific tissue within the population?

How does precision change over time?

In order to address these questions, a series of more nuanced questions arise. Because tissues do not form precisely at the time of birth or death, and most tissues are constantly turning over during life, what needs to be determined is how close the formation of a particular tissue is to the event of birth or death. Delving even deeper, bomb radiocarbon, in fact all radiocarbon, enters human tissues from the atmosphere via multi-step food chains: atmosphere to plants then plants to humans, or atmosphere to plants to animals to humans. Not only are there biological time lags in these processes, involving plant and animal growth, there are also socio-economic time lags resulting from patterns of food production, distribution, storage, and consumption. Does all this variability translate into

large differences in the tissue radiocarbon levels in individuals born in the same year, or dying in the same year? These questions address heart of the matter.

The central research goal in this proposal is to determine the atmosphere-to-tissue transport times for a variety of human tissues of known age and origin. How long does transport take? Are transport times similar between individuals or highly variable? The straightforward experiments are documented in what follows, and the results lead to some surprising and encouraging conclusions.

Literature review

Broecker *et al.* (1959), Libby *et al.* (1964), Nydal and Lővseth (1965) and Stenhouse and Baxter (1977) studied the uptake of bomb ^{14}C into the biosphere, including various human tissues. The motivation of early studies was to quantify the human radiation burden. The estimates attempted to assess of the lag time for transfer between atmosphere and human tissues. Investigations determined that dietary input from fruits, vegetables and dairy products were most tightly liked to atmospheric ^{14}C levels, whereas cereal, meat, and fish contained ^{14}C levels lagging well behind the contemporary atmosphere. They all estimated about a one year lag time for ^{14}C levels in soft tissue to reflect atmospheric levels. Jull *et al.* (1995) and Wild *et al.* (2000) were the first to suggest forensic applications of the bomb spike. Jull *et al.* (1995) focused on bone and found uptake was complex. Wild *et al.* (2000) measured several soft tissues and found that the ^{14}C content of hair (a continuously synthesized, non-turning over tissue) lagged approximately one year behind atmospheric ^{14}C levels. Biological studies of tissue replacement also provided data on other tissues. Pulse labeling studies of blood proteins

has shown that replacement rates of this tissue can be on the order of a few months (Buchholz *et al.* 1999).

Slowly replaced tissues: bone collagen

Bone collagen is the primary structural protein in bone. Studies of collagen residence time in human bone have shown that it increases during growth and stabilizes to about 20 years in adults (Jull *et al.* 1995). This value is only an approximation, as bone is a complex and dynamic tissue, continuously recycled and remodeled. For example, Ubelaker, Buchholz, and Stewart (2006) demonstrated that trabecular and cortical bone remodel at differing rates. Collagen residence times greater than 30 years have been demonstrated in elderly individuals (Stenhouse and Baxter 1977, Wild *et al.* 2000). With such long residence times, the rate of bomb-carbon uptake will change depending upon life stage, that is, whether skeletal growth, skeletal maintenance or skeletal atrophy are taking place. Because bone remodeling is slow and variable, the bomb radiocarbon content of bone is not strongly linked to either Year of Birth or Year of Death. However, the fact that it is resilient to decay merits its inclusion in this study.

Non-turning over tissues: tooth enamel, neural cell DNA, and eye lens crystallines

Human adult teeth are formed both *in utero*, and during childhood (Nolla 1960, Costa 1986). The timing is well constrained. Although the initial proposal was to examine the radiocarbon content of dentin tissue, a study by Spalding *et al.* (2005a), published just as this research began, investigated tooth enamel radiocarbon content as a potential indicator of Year-of-Birth, and so I elected to follow the Spalding protocols. Subsequently, a study by Cook *et al.* (2006) compared the enamel with the combined

dentin/cementum collagen fraction extracted from the root. Their results were consistent with root tissues being formed after crowns, and highlighting the possibly that the radiocarbon content is altered somewhat by replacement.

Work by Spalding *et al.* (2005b) and Bhardwaj *et al.* (2006) on neural tissue DNA, and by Lynnerup *et al.* (2008) on eye lens crystalline proteins have confirmed that other tissue components in the body besides tooth enamel, once formed, do not turnover during life. Theoretically the radiocarbon content of these could also be exploited to mark Year-of-Birth. Neural tissue DNA is embedded within metabolically active cells, and these within matricies of other fast replacement tissues so complex purifications would be necessary, and Lynnerup *et al.* found that eye lenses decay within three days post mortem. For the moment, neither seems a viable alternative to tooth enamel and dentin.

What tissues are suited to forensic ^{14}C measurement?

Different tissues are suited for addressing different questions: non-replacement tissues for establishing year of birth, fast-replacement tissues for year of death. However, an additional factor is tissue preservation. Soft tissues such as skin are more susceptible to decay, and may not be available for measurement so alternatives must be considered. In order to apply ^{14}C measurement to forensic analysis, biological replacement is only one half of the equation; *post mortem* tissue preservation is also important.

The research goal in this proposal is to measure the transport time of radiocarbon from the atmosphere to tissues in human biological tissues of known age and origin. Tissue types will be selected based upon replacement rates, resistance to post mortem decay, and forensic relevance. The data will be used to model ^{14}C transport from the atmosphere to the tissues in individuals living from the 1950's to the present. This will be

used to calibrate ^{14}C measurements on tissues of unknown origin, in order to establish year of birth and or year of death.

II. Methods

Sample Population

Tissues were provided by University of Tennessee Forensic Anthropology Center, at Knoxville, Tennessee. These samples were obtained from human remains donated to the Center, as part of their established Body Donation Program. The Program excludes people with known histories of HIV, tuberculosis, MRSA, or hepatitis. The majority of donations come from within a 200 mile radius of Knoxville, TN. Tissue samples were obtained from 36 remains donated to the facility between February and December 2006.

Donor Data

Donors or their family complete a Body Donation Questionnaire that is filed at The Forensic Anthropology Center. This records basic data: sex, race, birth date, height, weight, dental history, medical history, as well as self reported socio-economic status, and habitual activities (jogging, repetitive motions etc.). Birth dates are not verified by birth certificates. Birth and death dates, sex and race were initially made available to the PI for the purposes of this research.

Soft Tissue Sampling Protocols

Soft tissues were obtained at the time of donation.

Blood

Coagulated blood samples were obtained from the aorta using a syringe and large bore needle. Samples of approximately 5 mls were stored in glass vials with foil cap-liners until use. Fingernails were cut from the digits with a scalpel and adhering soft tissue scraped away. These were placed in glass vials. An approximately 1 square inch of

tissue was removed from the scalp of each donor. This sample included hair, epidermal, dermal, and sub-dermal layers and provided both skin and hair tissue. These were shipped in a foil and then transferred to glass vials for storage. All soft tissue samples were stored frozen until use.

Teeth

A minimum of two teeth were obtained from each donor at the time of donation. Although specific teeth were targeted based upon their formation sequence, in practice sampling was determined simply by the availability of intact teeth.

Bone

Approximately 10 grams of bone tissue was obtained from the anterior tibia using a striker saw. The samples included both cortical and trabecular bone. There was some variation in the sampling protocol. Bone samples from donors received early in the sampling phase, that is in the spring and summer, were obtained several months later, after the remains were allowed to naturally skeletonize. Bone samples from donors late in the sampling phase of the project were taken shortly after donation when the other soft tissue samples were obtained. All bone samples were wrapped in aluminum foil, bagged in plastic bags and frozen until use.

Sample Preparation Chemistry

Blood

The blood samples were simply freeze dried in preparation for carbon extraction.

Nail and Hair

Both of these materials are keratinous, and were chemically treated in a similar fashion. A single finger nail was used per individual. It scraped with a scalpel to remove

any dried soft tissue and then a 2-3 mm strip sampled from the 'quick' end. This was cut into sub-millimeter chunks in preparation for treatment. Hair was obtained from the scalp tissue samples. Hair within two centimeters of the scalp surface was sampled for radiocarbon measurement. Estimates by O'Connell *et al.* (1999), based upon stable hair isotope content after dietary change suggested this represents about two months growth.

Both nail and hair samples were subject to solvent extraction using a Soxhlet Apparatus. The solvent series hexane/ethanol/methanol/water was used to extract lipid residues and other hydrophobic contaminants. Samples were treated with each solvent for 24 hours. Then samples were placed in test tubes and soaked in 10 mls of 0.1N HCl over night, rinsed with deionized water, soaked in 10 mls of 0.1N NaOH for 4-5 hours, rinsed with deionized water, and then soaked again in 0.1N HCl for 4 hours to over night, and finally rinsed in deionized water until the pH of the wash solution was above pH 3.5. This acid/base/acid protocol is similar to the modified Longin Method (see Gillespie *et al.* 1984), and is typical in radiocarbon dating sample preparation protocols. Although of limited specificity, the use of hexane, low molecular weight alcohols, and mineral acids and bases minimizes the possibility of contaminating the sample with carbon-containing residues from cleaning solvents.

Skin

Skin samples were used to provide both skin lipid and skin 'collagen' samples. After hair was removed, the scalp samples were divided in two.

Skin Total Lipids

Glassware, scalpels etc used for lipid preparations were thoroughly cleaned and baked at 500°C before use. All reagents were HPLC grade or higher.

Total fatty acids were extracted from skin sub samples using the Folch Method, outlined in Stott *et al.* (1997). Approximately 2 g of the tissue was first lyophilized. The dried tissue was the cut into small chunks with a clean scalpel and the placed in a glass test tube. The skin was then extracted with 6 mls of chloroform:methanol 2:1. The de-fatted tissue was set aside for further treatment. The chloroform phase, containing the total lipid, was filtered using a silica gel column, collected into a screw cap vial and the solvent removed by evaporation under a gentle stream of nitrogen. The residue of total fatty acids was stored in sealed vials under nitrogen at -20°C.

Skin 'tissue'

The skin preparation protocol targeted the insoluble 'collagen' fraction. However, the resulting preparation was no doubt a complex mixture of biomolecules. After lipid extraction with the Folch Method, the remaining skin tissue was washed with deionized water by centrifugation and extracted with the same acid-base-acid washing protocol as the hair and nail samples. The remaining insoluble fraction was lyophilized in preparation for combustion.

Bone Lipid

The exterior surface of each bone sample was first cleaned by scraping with a scalpel. Approximately five grams of bone was lyophilized, and then crushed using a mortar and pestle. Lipids were extracted from the bone powder using the Folch Method, outlined in Stott *et al.* (1997) with chloroform:methanol 2:1. Samples were ultrasonicated for 30 minutes during extraction, and total lipid fraction decanted to cleaned test tubes. The bone powder was extracted a second time with chloroform:methanol and the lipid fractions pooled. Total Lipid fractions were then

filtered through a 10 ml silica gel column and the solvent evaporated under a stream of nitrogen. The recovered Total Lipid Fractions were transferred to foil-lined screw cap vials and stored at -20°C under nitrogen.

Bone Carbonate Carbon

Bone mineral carbonate carbon was extracted using the method of Haynes (1968). The defatted bone powder recovered from lipid extraction procedures and washed extensively with water and then lyophilized. Approximately one gram of this material was finely crushed with a mortar and pestle and transferred to one arm of a Y-shaped hydrolysis vessel. Five milliliters of anhydrous phosphoric acid were pipetted into the other arm, and the vessel evacuated and sealed. The acid and bone powder were mixed inside the vessel and the hydrolysis vessel incubated over night at 65°C. Carbon dioxide from the reaction was recovered from the head-space, purified and quantified using the cryogenic isolation methods described below.

Bone Collagen

The fraction known in the radiocarbon field as 'collagen' was isolated from 0.5 grams of bone. Although predominantly type I Collagen, this fraction is by no means biochemically pure. The isolation method was based upon the modified Longin Method (Gillespie *et al.* 1984). The bone was first de-fatted by chloroform:methanol extraction, crushed to a powder using a mortar and pestle, and then carried through the acid-base-acid extractions in test tubes. The bone powder was washed with multiple changes of 0.1N HCl until the bone powder stopped bubbling and no longer neutralized the acid. The acid extractions dissolve bone mineral and released carbon dioxide was discarded. The insoluble collagen residue was washed extensively with water, and then extracted

with multiple changes of 0.1 N NaOH. Base treatment extracts base soluble humic acid contaminants that could be derived from the burial environment. A final acid and water washes leave an insoluble collagen residue suspended in a weakly acidic solution. The collagen was then solubilized by heating to 65°C over night, and filtered through a GFC glass fiber filter. Purified collagen was recovered from the solution by lyophilization.

Tooth Enamel

Teeth were identified by visual comparison to reference materials. In some cases extensive wear made precise identification difficult. Carbon was extracted from tooth enamel using the method of Spalding *et al.* (2005a). Briefly, the tooth was cut in two at the cervical line using a diamond band saw. The recovered crown was soaked in 10 ml of 10N NaOH at 70°C over night with periodic sonication to promote dissolution of the non-enamel tissue. The NaOH solution was changed daily and the softened tissue manually scraped away until only enamel remained. The enamel was washed extensively with deionized water, lyophilized and stored in vials until further processed. Carbon was isolated from the enamel by acid hydrolysis and recovered as carbon dioxide using hydrolysis vessels described above.

Combustion and Graphitization

Organic samples (ie. other than bone apatite and enamel) were combusted to extract carbon using standard procedures. Briefly, samples were combined with a stoicheometric excess of CuO in quartz test tubes. The tubes were connected to a vacuum line and atmospheric gases pumped away. The tube was sealed and the contents heated to > 900°C. Above 450°C, CuO decomposes and the oxygen is available for combustion. The carbon dioxide was cryogenically separated from other combustion gases, and

manometrically quantified by expansion into a known volume. Carbon dioxide was converted to graphite using the method of Slota *et al.* (1987).

Elemental Analysis and Stable Isotope Mass Spectrometry

The stable isotope composition of carbon dioxide from each sample was determined on a VG Isotech stable isotope mass spectrometer, using laboratory standards calibrated to vPDB (Coplen 1994). Results are expressed as $\delta^{13}C$ per mil (‰) relative to vPDB, with a precision of ± 0.1‰.

CN ratios and nitrogen stable isotope values ($\delta^{15}N$) were measured on bone collagen samples. This was carried out by combusting collagen in a Carlo Erba NC1500 elemental analyzer connected upstream of the stable isotope mass spectrometer. Nitrogen stable isotope ratios were determined using laboratory standards calibrated to AIR and expressed as $\delta^{15}N$ per mil (‰), with a precision of ± 0.3‰.

Accelerator Mass Spectrometry

Radiocarbon measurements were carried out in a National Electrostatics Corporation 3.0 MeV Tandem Accelerator Mass Spectrometer using Oxalic Acid I (NIST SRM4990B) as the internal standard. All results were background subtracted and isotope fractionation corrected. The results are expressed as Fraction Modern ($F^{14}C$, or FM) according to Reimer, Brown and Reimer (2004). Fraction Modern ($F^{14}C$) according to equation (1)

$$F^{14}C = A_{SN} / A_{ON} \quad (1)$$

According to Stuiver and Polach (1977) and Donahue *et al.* (1990), where A_{SN} is the isotope fractionation corrected Sample 14/13 isotope ratio, and A_{ON} is the isotope

fractionation corrected Oxalic Acid Standard 14/13 isotope ratio. Alternatively, $F^{14}C$ is sometimes its percent equivalent, percent Modern Carbon (pMC) according to equation (2).

$$pMC = F^{14}C \times 100\% \quad (2)$$

Calibration of Tooth Enamel Formation Dates

The calibration curve IntCal04, (Reimer *et al.* 2004) was used for the calibration of Pre-Bomb enamel samples, ie. those containing less than 100 pMC. This calibration curve was created from thousands of high resolution measurements. The measurement uncertainties in this data set are explicit. The calibration program OxCal3.10 was used to calculate probabilities associated with each calendar age distribution and to generate calibration graphics.

The formation time of Post-Bomb tooth enamel samples (containing greater than 100 pMC) were determined by comparison of their ^{14}C content to atmospheric measurements published in Levin and Kromer (2004), and modified by Hua and Barbetti (2005). The Levin and Kromer curve was derived from a concatenation of measurements of atmospheric ^{14}C-carbon dioxide made on a bimonthly basis from 1959 to 2003 at two locations, Vermunt,, Austria and Schauinsland, Germany. Hua and Barbetti (2005) estimated the distribution Post Bomb radiocarbon levels in different Zones of the Northern hemisphere based upon modeled Atmospheric Circulation. Zone 2 covers the continental United States.

Conversion of ^{14}C measurements to calendar dates was accomplished by calibration program Calibomb3.0 (Reimer, Brown and Reimer 2004). The Zone 2 curve was selected and smoothed to 1.0 years. The minimum resolution was set at 0.2 years.

Smoothing dampens the influence of the fluctuations in atmospheric levels seen in the raw data and instructions the recommend a smoothing time equal to the formation time of the sample. A one year crown formation time was selected. This was shorter than the formation time observed by Nolla (1960), but seemed a reasonable compromise as the greater smoothing obliterated too many details of the curve. The Resolution of 0.2 years indicates that events less than 0.2 years apart are consolidated.

Calibration of Tissue ^{14}C measurements from Extrapolated of Atmospheric Radiocarbon Levels

The different human tissues from 2006 showed different mean ^{14}C contents and different amounts of scatter. An attempt was made to compare these to the mean ^{14}C content and scatter in the measurements of the contemporary atmosphere. Unfortunately, the Levin and Kromer (2004) data only extends to 2003, so atmospheric ^{14}C levels and their scatter had to be estimated. Several estimations were done as a sensitivity analysis. Estimations of the atmospheric mean ^{14}C values in 2007 were made by fitting both linear and 2nd order polynomial curves to the subset of Levin and Kromer (2004) measurements spanning 1985 to 2003. These curves were then extrapolated to 2007. The linear and 2nd order polynomial extrapolations are assumed to bracket the true atmospheric values.

The scatter in the Levine and Kromer (2004) raw atmospheric measurements was characterized by first generating smoothed curves using 6 and 12 point running averages of the raw data. This ‘smoothing’ corresponded to approximately to 3 and 6 month time intervals. The scatter for these two curves was calculated as the root mean square of the residuals between individual measurements and their corresponding running average values. These values were determined to be 0.0059 and 0.0093 $F^{14}C$ respectively. These

represent the standard deviations of the 3 and 6 month smoothed curves. These standard deviations were then plotted on the linear and 2nd order polynomial extrapolations extending to 2007.

Estimated Calibrations of Soft Tissue Samples

Manual calibration using graphical methods were carried out on extrapolated curves.

Extrapolations of Atmospheric Radiocarbon Levels 2003 to 2008.

Unlike the IntCal04 data set, the Post-Bomb atmospheric Calibration curves do not quantify measurement uncertainties. This is because only single measurements were made at each time point. However the time course of atmospheric measurements shows considerable scatter. This scatter contributes to the uncertainty of Post-Bomb calibration calculations and so a sensitivity analysis was carried out to examine its contribution.

The Levin and Kromer (2004) raw data was smoothed using either 6, 12, or 18 month time windows. The standard deviations of the scatter from each smoothed curve were calculated. The root mean square of the residuals from the raw data and the smoothed curve was calculated to be 0.0059 pMC for the one year smoothed curve. This value used to estimate the standard deviation of extrapolated curves for the time period 2003 to 2010.

III. Results

The birth and death dates of the 36 donors who provided tissues for this study are shown in Table 1. All of the individuals died in 2006. The age range spanned 31.2 to 93.8 years. There were 17 males and 19 females. The self-identified racial make-up was 30 white (83%), 4 Black, 1 Hispanic, and 1 American Indian. The donors died within a 100 mile radius of Knoxville, Tennessee. The lifetime geography of these people is unknown, but presumed to center on the South-Eastern United States.

Table 1: Donor Population Statistics:
Donor Year-of-Birth, Year-of-Death, Sex, and Race Data, and Mean Year-of-Death for Sample Population

Number	ID #[1]	Sex	Ancestry	Date of Birth	Date of Death	Age (Years)
1	65-06	M	WHITE	06/23/75	09/02/06	31.2
2	33-06	M	HISPANIC	08/10/72	02/20/06	33.6
3	32-06	F	WHITE	06/03/66	04/22/06	39.9
4	74-06	F	WHITE	05/26/64	09/17/06	42.3
5	63-06	F	WHITE	10/31/62	08/26/06	43.8
6	25-06	F	WHITE	01/17/62	04/07/06	44.2
7	69-06	F	WHITE	04/17/61	08/20/06	45.4
8	87-06	M	WHITE	01/07/60	11/02/06	46.9
9	98-06	M	BLACK	06/18/59	10/20/06	47.4
10	75-06	M	BLACK	10/19/58	09/25/06	48.0
11	92-06	M	WHITE	03/02/58	11/16/06	48.7
12	78-06	F	WHITE	04/27/57	10/10/06	49.5
13	89-06	F	WHITE	05/06/56	11/10/06	50.5
14	93-06	M	BLACK	01/18/56	11/16/06	50.9
15	17-06	F	WHITE	09/13/55	02/25/06	50.5
16	40-06	F	WHITE	05/29/55	06/16/06	51.1
17	95-06	M	WHITE	11/07/52	11/29/06	54.1
18	62-06	F	BLACK	11/24/51	08/08/06	54.7
19	68-06	F	WHITE	10/20/51	09/04/06	54.9
20	80-06	M	WHITE	12/01/50	10/03/06	55.9
21	100-06	F	WHITE	11/16/49	11/19/06	57.0
22	19-06	M	WHITE	04/20/46	01/28/06	59.8
23	101-06	F	WHITE	12/18/45	12/02/06	61.0
24	57-06	F	AM.INDIAN	12/09/45	08/12/06	60.7
25	81-06	F	WHITE	07/26/45	10/20/06	61.3
26	31-06	M	WHITE	04/21/42	03/15/06	63.9
27	61-06	M	WHITE	06/17/37	08/20/06	69.2
28	16-06	M	WHITE	06/26/35	02/26/06	70.7

29	76-06	M	WHITE	02/27/35	10/04/06	71.6
30	79-06	M	WHITE	08/18/33	10/16/06	73.2
31	30-06	F	WHITE	07/02/30	04/21/06	75.9
32	83-06	F	WHITE	06/20/30	10/21/06	76.4
33	18-06	M	WHITE	10/01/24	02/24/06	81.5
34	39-06	F	WHITE	01/02/21	06/08/06	85.5
35	60-06	M	WHITE	05/03/17	08/12/06	89.3
36	94-06	F	WHITE	01/31/13	11/10/06	93.8
				Average DoD (years):	2006.6	
				Std Dev (years):	0.266	

1. Forensic Anthropology Center ID Number

Donor Life Spans Relative to Atmospheric ^{14}C Spike

The level of atomic-bomb derived radiocarbon into different tissue compartments is determined by multiple factors. Two factors that likely predominate are the timing of tissue formation relative to the bomb-spike, and the rate of tissue replacement. The life spans of the donor individuals relative to atmospheric radiocarbon levels are shown in Figure 2.

The donor population is diverse in terms of Year-of-Birth relative to the atomic bomb-derived ^{14}C spike, and so presents a useful sample for this initial investigation. Figure 2 also draws attention to the first two decades of life. Within this time frame, all adult teeth are formed, and full adult stature is attained. After this time, growth transitions from net tissue gain to an approximately steady state of tissue replacement.

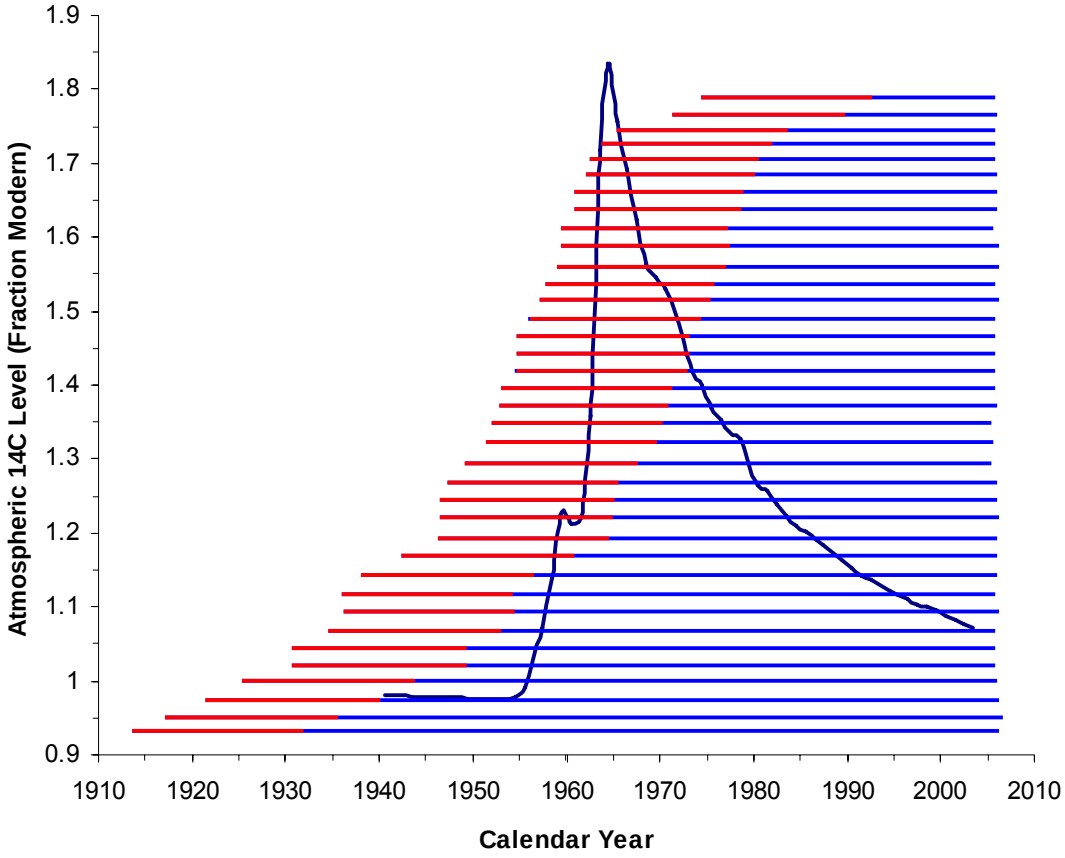

Figure 2: Life Spans of 36 Donors compared to Atmospheric Radiocarbon Levels. The average annual atmospheric ^{14}C levels (modified from Levin and Kromer 2004) are shown in dark blue and plotted as Fraction Modern ^{14}C. The life spans of individual donors are represented as a 36 timelines superimposed over the atmospheric ^{14}C curve. These lines span from Years of Birth (ranging from 1912 to 1975), to Year of Death (all in 2006). The first 20 years of each timeline is highlighted in red. This is the period within which adult dentition and full skeletal mass is achieved.

In Table 2, the donor population is divided into four categories relative to the timing of the atmospheric ^{14}C spike. These categories provide useful framework for interpreting the distribution of atomic bomb-derived ^{14}C into different tissue compartments. This categorization does not take into account the time-lag of the transfer of atmospheric ^{14}C into various human tissues.

Table 2: Examining Donor Population Growth Phase relative to the Timing of the Atmospheric ^{14}C Spike

Birth years	Number of Donors	Characteristic
1913 to 1937	10	Adult stature achieved before bomb-spike
1942 to 1946	5	Adult stature achieved during rising portion of bomb spike
1947 to 1964	18	Growth occurring during both rising and falling portion of bomb spike
1966 to 1975	3	Growth to adult stature achieved during falling portion of the bomb spike

Analyzing Dietary Complexity of the Donor Population based upon Bone Collagen Stable Isotope Values

Radiocarbon enters human tissues by dietary intake and subsequent tissue synthesis. Atmospheric radiocarbon is first incorporated into plants tissues through photosynthesis, and when humans consume plants directly, or consume them indirectly by eating herbivorous or omnivorous animals, it becomes available for incorporation into human tissues. Different time lags are associated with different food chains. The shortest chain length from the atmosphere to human tissues occurs through the direct human consumption of plants and fruits. Temporally longer chains occur when humans consume terrestrial animals, as time is required to grow animal feed and for the animal to reach a consumable size, (for example, for chickens this may only add an additional few months, but for cattle this can be 1-3 years). The longest lag occurs when humans consume marine organisms as there is a considerable lag involving the dissolution of atmospheric gases into ocean water. The human consumption of marine organisms adds considerable complexity to the analysis of tissue ^{14}C and so identifying individuals whose diet has included substantial marine foods in crucial.

An established tool for identifying dietary input is bone collagen carbon and nitrogen stable isotope analysis. Bone collagen turn over is approximately decadal (Jull *et al.* 1995) so bone collagen stable isotopes provide information about the average diet over that time-period. Bone collagen stable isotope values were measured on the donor population to generate a picture of dietary diversity. The underlying question being asked was whether or not there was evidence that some individuals consumed marine foods, a circumstance that might affect tissue ^{14}C levels in a complex manner. Fortunately, marine dietary input is easy to identify on this basis, as it is characterized by the combination of high $\delta^{13}C$ and high $\delta^{15}N$ values.

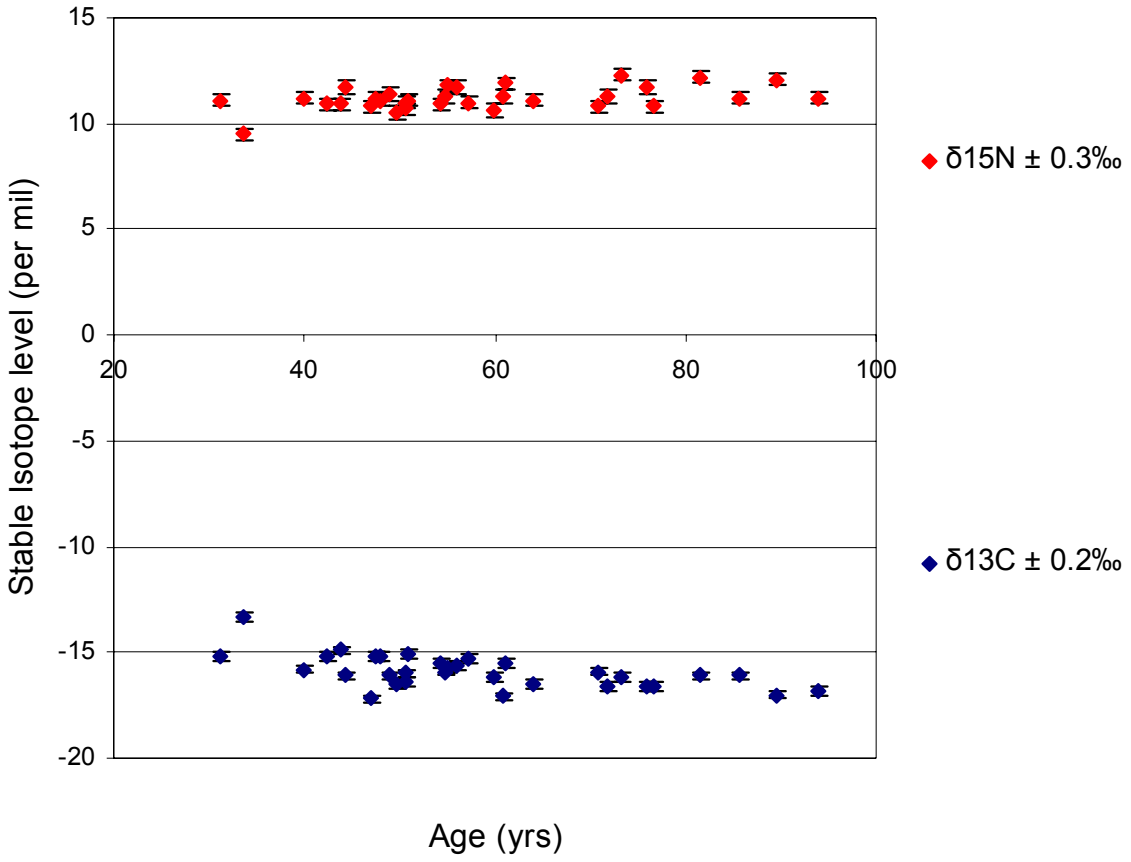

Figure 3: Bone Collagen $\delta^{13}C$ and $\delta^{15}N$ versus Age. Measurements are shown for 32 Donors: four Donors (40-06, 61-06, 69-06, and 81-06) did not provide collagen.

The results shown in figure 3 indicate a slight trend in both isotope values with age: younger individuals have slightly higher $\delta^{13}C$ values ($\delta^{13}C$ = -0.0309Age - 14.139, R^2 = 0.3895), and slightly lower $\delta^{15}N$ values ($\delta^{15}N$ = 0.0161(Age) + 10.253, R^2 = 0.2149).

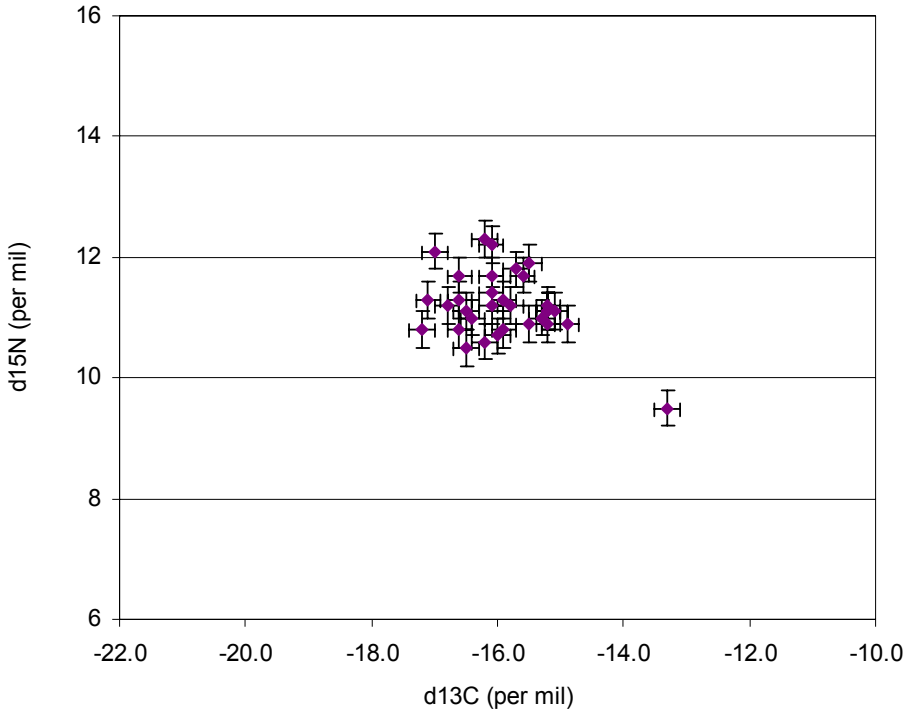

Figure 4 Bone Collagen $\delta^{13}C$ versus $\delta^{15}N$. Excluding the single outlier, the mean values for $\delta^{13}C$ and $\delta^{15}N$ are -16.0‰ ± 0.63‰ and 11.2‰ ± 0.47‰ respectively.

The plot of $\delta^{13}C$ versus $\delta^{15}N$ is shown in figure 4 It reveals the remarkably uniform dietary stable isotopes for the donor population, with the exception of a single individual (33-06). The donor population appears to be predominantly terrestrial omnivores. The donor population shows evidence of corn-based dietary input ($\delta^{13}C$ = -16.0), presumably through both direct and indirect sources such as the use of corn-based sweeteners, and corn-fed animals. This is in contrast with data published by O'Connell *et al.* (2001) on bone stable isotope values found in British populations ($\delta^{13}C$ = -19.71, $\delta^{15}N$ = 10.34, *n* =

8). The difference stable isotope signatures likely reflect differences between the US and European diet. A single individual in this study, donor 33-06, appears to have a high corn/low meat diet ($\delta^{13}C$ and $\delta^{15}N$ are -13.3‰ and 9.5 ‰ respectively). High $\delta^{13}C$ values can be indicative of either a marine-based diet, or a diet based upon plants that utilize the C4 carbon fixation biochemistry, such as corn. A high meat or marine diet would generate high bone collagen $\delta^{15}N$ values. Given the low $\delta^{15}N$ for 33-06, a reasonable hypothesis is that 33-06 was vegetarian.

The dietary uniformity of the donor population was surprising, but very helpful as it appears that marine foods were not significant. It would be very useful to examine the larger US population to see if this is a widespread trend or a situation unique to the Forensic Anthropology Center Donor population.

Radiocarbon Levels in Human Tissues

The Master Database of Tissue Radiocarbon Measurements is shown in attached Table 3. The rational for making these measurements was to investigate whether levels of radiocarbon in different human tissues could predict Year-of-Birth, and/or Year-of Death, based upon the differential uptake of elevated levels of atmospheric radiocarbon. Tooth Enamel Radiocarbon content was measured as a potential tool for identifying Year-of-Birth. Measurements on bone tissues (bone collagen, bone apatite, and bone lipid) were made to characterize variability in the metabolic uptake of atomic bomb-derived radiocarbon. And finally, radiocarbon measurements were made on various soft tissues (blood, hair, skin, nails, and skin lipid) to investigate the potential of these to indentify the Year-of-Death. Data addressing each of these three objectives are presented below.

^{14}C Measurements in Tooth Enamel as Predictors of Year-of-Birth

Spalding *et al.* (2005a) suggested that tooth enamel radiocarbon content can be used to determine the Year-of-Birth for individuals who lived during the atomic age, based on the fact that tooth enamel, once formed, does not turn over during life, and specific teeth form at fixed times after birth.

The pattern of adult tooth formation was studied by Nolla (1960). She described the process using annual dental X-radiographs obtained from Michigan school children (twenty-five girls and twenty-five boys), as they grew from approximately three years to seventeen years of age. She divided tooth formation into ten stages. In her scheme, tooth crown formation initiates at Stage 2, is one-third complete at Stage 3, two-thirds complete at Stage 4, almost complete at Stage 5, and complete at Stage 6. Root growth continues in Stages 7 through 10. Tooth formation was studied in both maxillary and mandibular teeth. An example of Nolla's data is shown in Figure 5 which summarizes mandibular tooth formation in boys. Only subtle differences were obtained for boys maxillary tooth formation and for tooth formation in girls (shown in Table 4). Nolla's data is particularly well suited to this study, as it is based upon American Children from the 1940's to the 1960's. However variation in the timing of tooth formation was not discussed muc by Nolla, and must be examined when applying the method to a broad population.

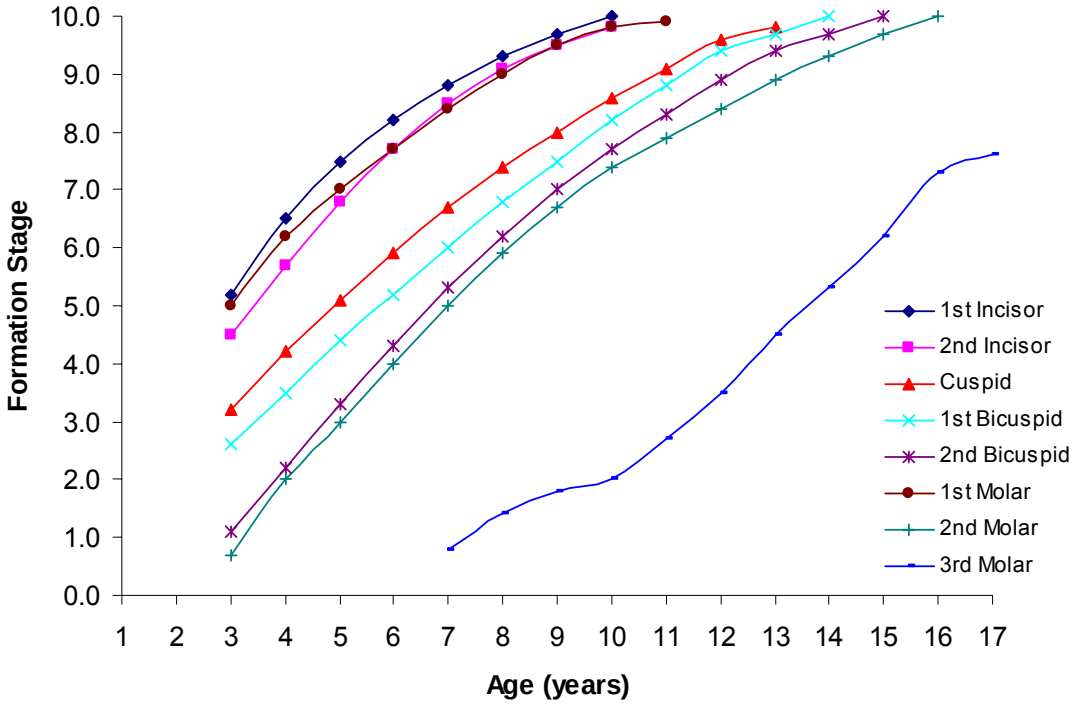

Figure 5. Mandibular Tooth Formation in Boys (from Nolla 1960). The data show the range of ages by which time crown formation (Stage 6) is complete in different teeth. Crown completion ranges from 3.5 years of age in the 1st Mandibular Incisor, to 15 years of age for the crown of the 3rd mandibular molar. Note also the duration of crown formation (Stage 2 to 6). Although not known for the first four teeth, crown formation extends over four years for the 1st and 2nd Bicuspid and the 2nd molar.

Table 4: Years of Age for Complete Tooth Crown Formation (Stage 6). Data from Nolla 1960.

	1st Incisor	2nd Incisor	Cuspid	1st Bicuspid	2nd Bicuspid	1st Molar	2nd Molar	3rd Molar
Boys Mandible	3.2	3.8	5.4	6.3	7.2	3.4	7.6	14.2
Boys Maxilla	4.1	5.0	5.8	6.8	7.6	4.2	7.6	13.6
Girls Mandible	3.1	3.6	5.0	5.6	6.8	3.4	6.6	12.7
Girls Maxilla	4.0	4.7	5.2	6.1	6.6	3.8	6.8	12.1

Because the Spalding *et al.* (2005a) study appeared just as this research was being initiated, I decided to utilize their protocols so that our results could be directly compared. A second study by Cook *et al.* (2006) also appeared, and so the context of

these measurements has been further expanded. Moreover, because these two studies were already available, the decision was made to carry out a minimum of measurements to test the method, and devote resources to other aspects of the research that had not been previously investigated by others.

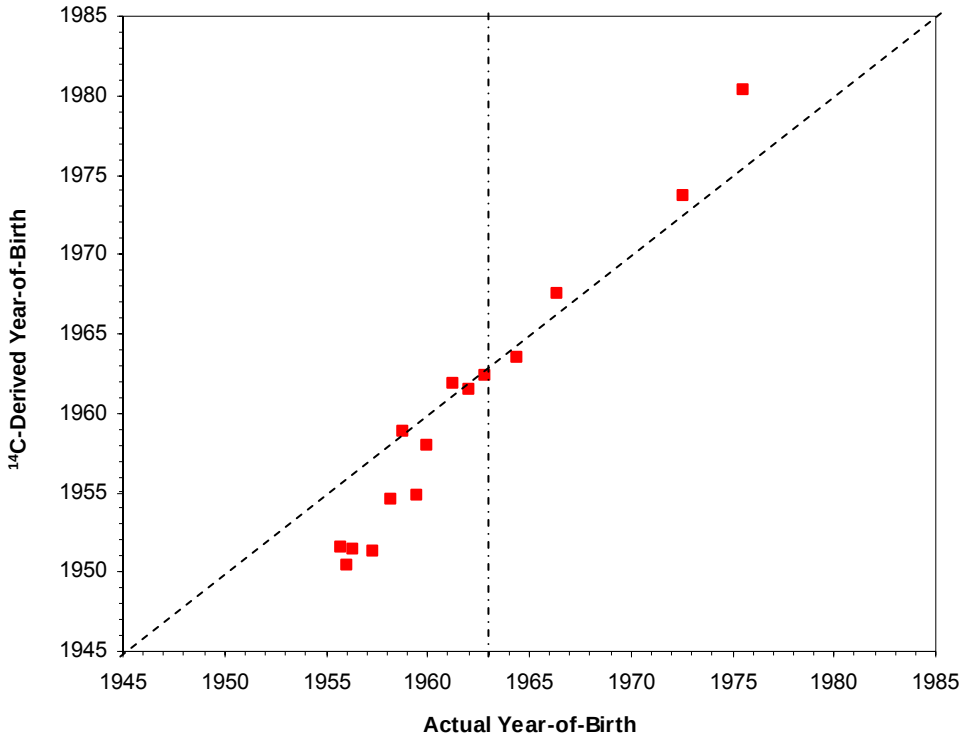

Figure 6: Comparison of Tooth Enamel ^{14}C-Derived Birth Year with Actual Birth Year. The diagonal dashed line represents one-to-one correspondence. The vertical dot-dash line indicates the approximate peak of Northern Hemisphere Atmospheric radiocarbon levels. Teeth from 15 Donors were measured

Teeth were obtained from the donor population. In two instances, tooth identification was ambiguous and so these individuals were excluded from the study. Crown enamel was purified according the method of Spalding *et al.* (2005a), and radiocarbon measurements were obtained. Teeth from fifteen of the donors were found to contain elevated levels of radiocarbon. These elevated measurements were compared to post 1950 atmospheric radiocarbon levels using the Calibomb Calibration Program, specifying

and the Northern Hemisphere mid-latitude (Zone 2) calibration curve, smoothed with a one year filter. Calibration established the putative Year-of- Crown-Formation and then the Nolla Data shown in Table 4 was used to extrapolate back to the Year-of-Birth. Finally, these measurements were compared to actual Year-of-Birth data and this comparison is shown in Figure 6. In half the cases, the ^{14}C content of tooth enamel accurately determined actual Year-of-Birth. However the results tended to diverge from the one-to-one line for individuals living during the early portion of the bomb-spike. The tooth enamel from these individuals has a lower radiocarbon content than expected. Thus the method calculates a Year-of-Birth that is earlier than the actual Year-of-Birth. It is unlikely this is a result of experimental error, as recent contamination would tend to increase sample ^{14}C content over expected values.

The results show great promise, however it must be pointed out that these experiments were not a rigorous test of the method. With only two exceptions, the ^{14}C-derived Years-of-Birth were generated from single tooth measurements per donor individual. As both Spalding *et al.* (2005a) and Cook *et al.* (2006) point out, a minimum of two measurements from teeth forming at different ages is required to establish which side of the bomb-spike the teeth were formed on. The ^{14}C-Derived Year-of-Birth estimates in this study, derived from single tooth measurements were guided by knowledge of the actual year of birth. This must also have been the case in the Spalding *et al.* (2005) study, as several estimates in that study were also derived from single tooth measurements.

The data shown in Figure 6 is directly compared to the results obtained by Spaldling *et al.* (2005a) and Cook *et al.* (2006) in figure 7. Comparison reveals several

differences between the studies. First, the sample population in this study is generally older than the earlier studies, and this influences the expected pattern of tooth enamel labeling within each individual. Individuals born after about 1962 will be forming all of their adult tooth enamel after the 1963-64 peak in atmospheric radiocarbon levels. Sequentially developing teeth in these individuals will have progressively lower radiocarbon levels. Individuals born before 1962 will have patterns of tooth enamel labeling that are more complex because teeth are forming when atmospheric radiocarbon levels are both the rising and falling. Adding to this complexity is the fact that the rate of change in atmospheric radiocarbon levels in the period leading up to the peak is much higher than the period after the peak. Moreover, given the evidence in Nolla (1960) that enamel formation occurs over a period of four years, enamel radiocarbon uptake in teeth forming during the rising portion of the curve will be complex. This aspect of the method has not been adequately discussed in the existing literature.

The second observation about Figure 7 is that the estimated ages in this study are more divergent from the one-to-one line than the previous studies. This is especially true of individuals born before 1960, and presumably forming tooth enamel near the years with peak atmospheric radiocarbon levels. Differences between studies are apparent in spite of the fact that Spalding *et al's* protocols were replicated. Further studies are necessary to more thoroughly exclude the possibility of process contamination, and investigate other possible explanations such as enamel carbon exchanging with dead-carbon from bicarbonate-based tooth paste, during life.

A final observation is that the combined data set shows that the method tends to estimate a Year-of-Birth that is too early more often than too late – in other words, the

rate of uptake of atmospheric radiocarbon into tooth enamel appears variable, but on average involves a time lag of the order of one to two years.

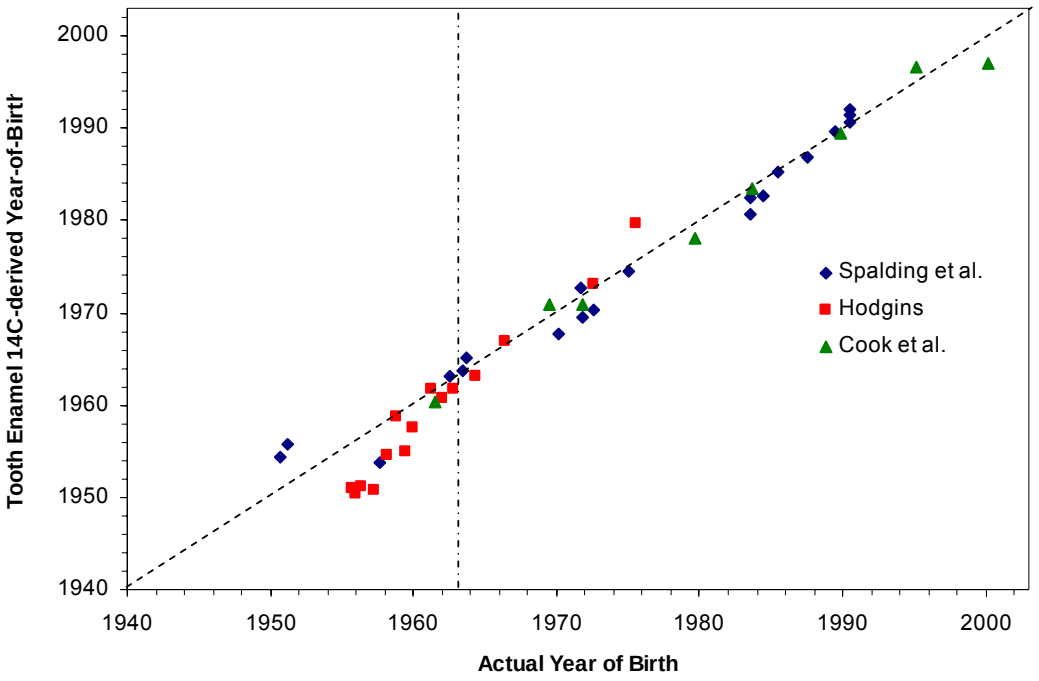

Figure 7: Comparing results from three studies using tooth enamel radiocarbon content to derive Year-of-Birth. The fifteen data points from this study (red squares) are combined with twenty measurements by Spalding *et al.*(2005a) (blue diamonds) and eight measurements by Cook *et al.* (2006) (green triangles).

In reality, the underlabeling of teeth in the 1950's probably does not present problem for forensic determination of Year-of-Birth. In practice a "Tooth ^{14}C content vs Year-of-Birth" calibration curve derived from tooth measurements of known-age Americans should be constructed, and this used to assign Year-of-Birth for unknowns. Moreover, similar curves could be constructed from other geographic regions, in order to

investigate whether variations in diet (as noted in figure 4 of this study) have an effect on tooth ^{14}C uptake during the early bomb-spike.

^{14}C Measurements on Bone fractions: collagen, bone apatite, and bone total lipids

Bone is a complex tissue with many fractions: apatite mineral, collagen, and non-collagen proteins. The replacement rates of these fractions may differ considerably. It has been previously demonstrated that collagen radiocarbon content can not directly provide a reliable estimate of either Year-of-Birth or Year-of-Death (Jull *et al.* 1995, Wild *et al.* 2000) etc. However such measurements serve several purposes. First, bone is decay resistant. The biomineral complex might protect labile fractions that would otherwise be lost through tissue decay. Moreover, bone mineral, or some of the non-collagen proteins might have higher replacement rates and thus their radiocarbon contents that might indicate the Year-of-Death. As mentioned previously, bone collagen stable isotope values indicate the individual's average diet, and avoid the potential confusion of a significant marine diet. Finally, the replacement rate of collagen might provide clues as to the general metabolic rate of the individual.

In these experiments, bone samples from each donor were fractionated into collagen, bone mineral (apatite), and bone total lipid fractions. The radiocarbon content of each of these was determined and is shown in Figure 8. The radiocarbon content of bone mineral has not previously been systematically analyzed. It is generally avoided in archaeological bone because of the potential for contamination by groundwater carbonates. Although this might be a factor in forensic applications, our assumption is

that such contamination is not a factor in these measurements. Most of the bone samples were collected from a controlled environment, only weeks to months post mortem from surface rather than buried contexts.

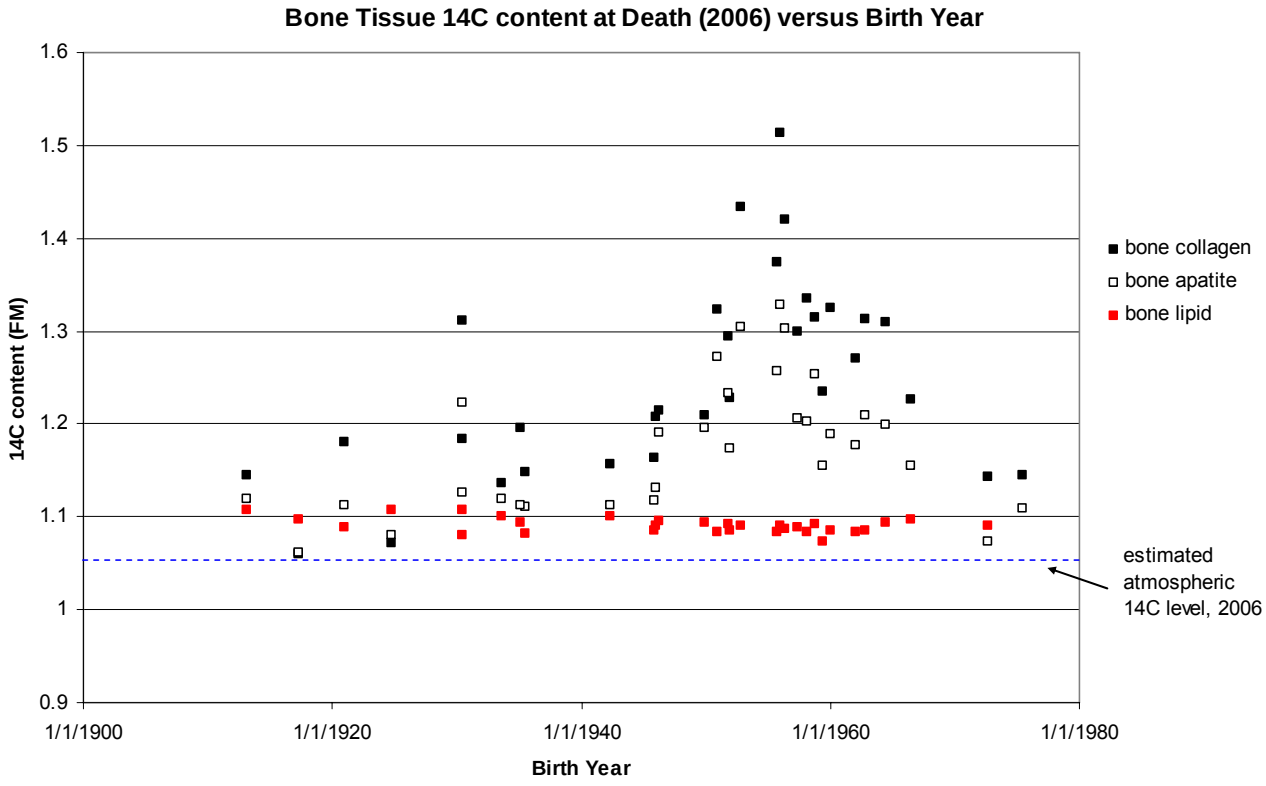

Figure 8: The radiocarbon content of bone collagen, bone apatite, and bone lipid fractions from 36 donor individuals who died in 2006. The data are arranged according to the birth year of each donor. The measurement uncertainties are of the order of ± 0.004 FM and thus the Y-error bars are covered by the markers.

Previous estimates of the rate of collagen replacement rates are based on a limited number of measurements. Estimations in the range of 20-30 years have been published by Stenhouse and Baxter 1977, Jull *et al.* 1995, Wild *et al.* 2000, and Ubelaker *et al.* 2006. The data in figure 8 show that such blanket estimations are misleading. Bone replacement rates are characterized more by variability than uniformity. This is clearly shown in the measurements on individuals born before 1940. These individuals achieved adult stature

and so their skeletons must have contained the ambient pre-bomb 20th Century radiocarbon content of approximately 0.97 Fraction Modern. Slight differences might have existed, attributable to varying diet (intake of marine foods), and perhaps a regional Seuss Effect. Elevated levels of radiocarbon in these individuals can be attributed exclusively to tissue replacement.

Variability in replacement rates is clearly evident in the bone collagen radiocarbon contents of individuals born on or near the same year. For example, note the difference in levels between the two individuals born in 1930, the five individuals born in 1949-52, and the three individuals born in 1955-56. Elevated levels result from high replacement rates when atmospheric radiocarbon content was high. Low levels are most likely the result of limited replacement. The skeletons of the individuals born in 1917 and 1924 are barely above ambient radiocarbon levels. It appears these individuals generated hardly any new skeletal material after 1955.

The replacement rate of apatite appears to be related to collagen replacement, but it appears to be approximately 50% faster. Given the intimate structural association of collagen and apatite mineral, such correlation is not surprising. However, the fact that replacement rates are not the same was unexpected. The measurements suggest the ratio of collagen to apatite replacement rates is not constant between individuals.

The radiocarbon content of bone lipid was surprisingly uniform across the donor population. The fact that its radiocarbon carbon content for all individuals was slightly higher than that of the ambient atmosphere in 2006 indicates the lipid fraction turns over at a much higher rate compared to both apatite and collagen. Lipid can function as an energy storage molecule so its rapid replacement is not surprising. These findings suggest

that bone lipid might be a good candidate for Year-of-Death determination. That is, if lipids survive within bone even after other soft tissues have fully decayed away.

Further research is necessary to investigate the origins of replacement rate variability. Genetics and diet is likely important factors. Physical activity must also play a role. Bone disease, bone injury and bone healing might also influence bone radiocarbon content. The data in Figure 7 might provide clues. For example, it would be interesting to research the life histories of the two individuals born in 1930 to try to identify factors that might have caused one of them to remodel their skeletons more extensively than the other.

^{14}C Measurements on Fast replacement Tissues: toward establishing Year-of-Death

The working hypothesis is that soft tissues that rapidly turn over should have ^{14}C contents close to the ambient atmosphere radiocarbon level of the Year-of-Death. Because atmospheric radiocarbon levels have been descending towards natural levels since 1964, and the donor population in this study all died in 2006, it was expected their soft tissue levels would be equivalent to, or slightly higher than ambient atmosphere radiocarbon levels of 2006. Slightly higher levels would be generated if significant time was required for the photosynthetic uptake of atmospheric carbon at the base of the food chain, dietary uptake by the individual, and tissue synthesis and replacement. Central to this research was characterizing the magnitude of the time-lag, and its variability within the population for different soft-tissue types. Hypothetically, a tissue that would be a good candidate for a Year-of-Death marker would be one that had a short lag (rapid replacement), low variability within the donor population, and be decay resistant.

In total nine tissue types were selected for measurement from the 36 donors, although not every tissue was available from each donor. The radiocarbon contents of these tissues were measured (Table 3) and the mean and standard deviations of each tissue type are presented in Figure 9. The tissues with the lowest radiocarbon values (ie closest to atmospheric levels in 2006) and the lowest standard deviations within the sample set were blood, hair and nails. Skin and bone lipid values have slightly higher radiocarbon values, but relatively low scatter. Surprisingly, skin collagen radiocarbon content was quite variable, suggesting it is not a good candidate for Year-of-Death determination. These results are presented in greater detail below.

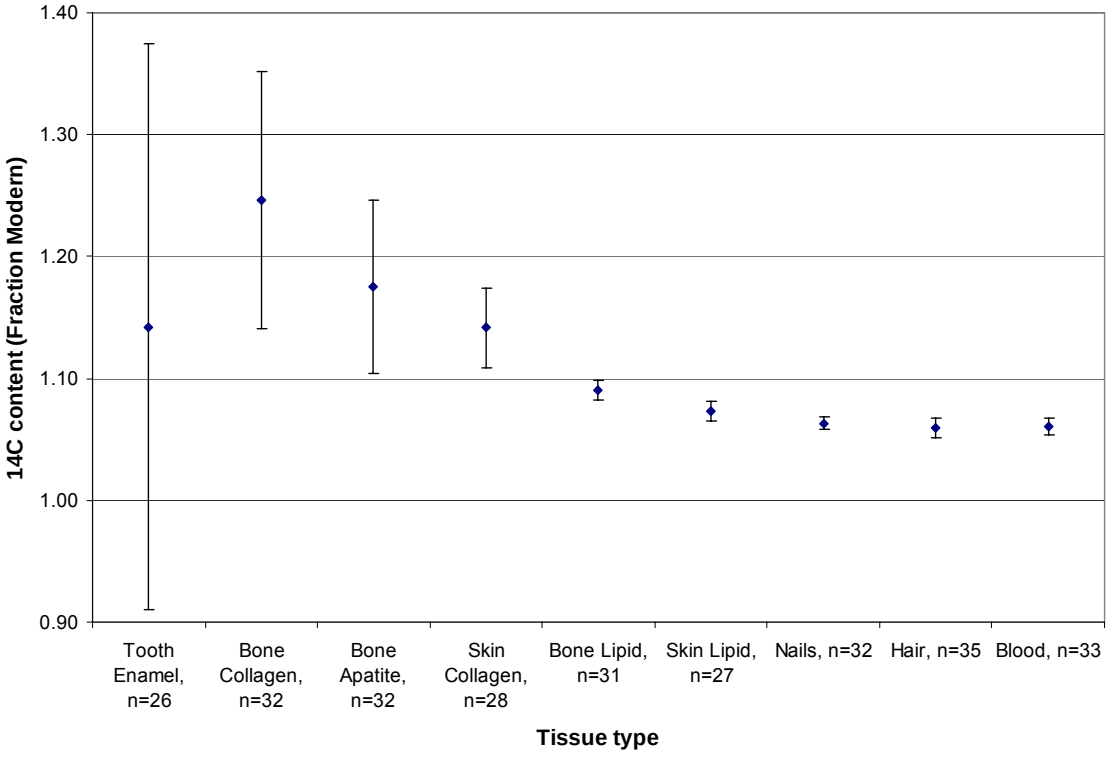

Figure 9: Tissue ^{14}C contents for 36 Individuals who died in 2006. The donor population mean and one sigma standard deviation values are shown.

^{14}C Measurements on Blood, Nails, and Hair and Bone Lipid

Although blood is probably not a good candidate tissue as a Date-of-Death marker due to its susceptibility to decay, my hypothesis was that blood radiocarbon content should be the closest to the dietary radiocarbon content and hence atmospheric radiocarbon levels. It is a tissue that has a very high replacement rate – for example red blood cell half-life has been measured at about 180 days (Buchholz *et al.* 1999). For this reason, the blood measurements were assumed to define the minimum radiocarbon content.

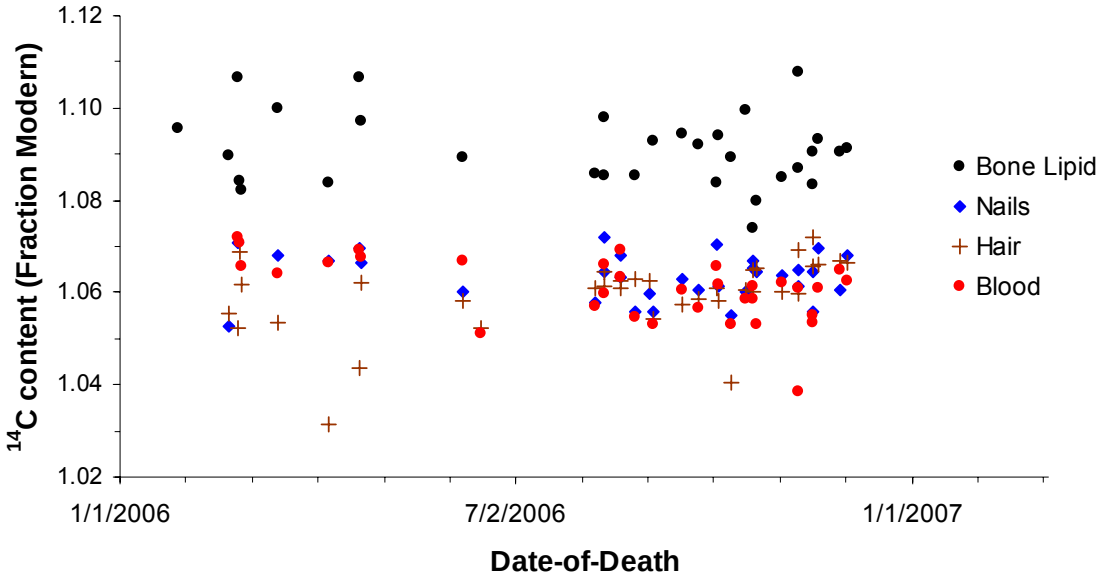

Figure 10: Blood, Hair, Nails and Bond Lipid radiocarbon content versus Date of Death. The Y-error bars have been omitted for clarity. The measurement uncertainties are of the order of ± 0.0040 FM.

Figure 10 shows that the radiocarbon content of hair and nails is similar to that of blood. Both are extracellular structures comprised of keratin, and both are continuously synthesized during life. Hair was sampled within 1 cm of the scalp, and nail samples

were obtained closest to the quick. The results shown in figure 10 suggest that their hair nail and blood radiocarbon contents are closely linked to dietary radiocarbon content. Keratin is somewhat resistant to decay, and so these hair and nails might be candidate marker tissues to indicate Year-of-Death. Figure 10 also clearly shows bone lipid radiocarbon content is higher than the other three materials, consistent with it having a slower replacement rate. A close examination of the data suggests that there is variability between tissues. In other words, if an individual had a high bone lipid radiocarbon values compared to the rest of the donors, it did not necessarily mean that other tissues in that individual were also elevated.

Finally, the radiocarbon content of some of the tissues appeared to be lower than the ambient environment. One of the low measurements was a blood sample. This was obtained from the oldest donor in the population. Three of the lower measurements were on hair samples. Two of these were checked by repeated measurements. The low values did not correlate with age. The six lowest measurements, and in fact ten of the fifteen lowest values were from women's hair. This could be genetic or metabolic; however, the influence of hair dyes or other hair products might be a factor and should be investigated. If hair products such as hair dyes are produced from petroleum products, all of which are radiocarbon free, then the presence of hair dyes might depress the overall radiocarbon content. Solvent residues from Soxhlet extractions must also be considered.

III. Discussion of Findings

Comparing Tissue Radiocarbon Content to Atmospheric Levels

Determining Year-of-Death from tissue radiocarbon content presented three challenges: how to deal with noise in the atmospheric data set; how to account for

unknown rates of tissue formation and/or replacement; and the lack of published atmospheric data from 2003 up to the present date. The first challenge was be addressed by data smoothing, the second by direct measurement, and the third by extrapolating the trend of atmospheric radiocarbon level reduction before 2003 out beyond 2006.

Curve Smoothing

Atmospheric radiocarbon levels fluctuate both randomly and periodically. The data sets such as Levin and Kromer (2004) were assembled from bimonthly atmospheric samples obtained from mountain-top test stations. The high altitude sampling was selected to avoid the more complex fluctuations in radiocarbon levels that occur closer to sea level, as a consequence of both natural and anthropogenic processes. For example, ground level atmospheric ^{14}C is depressed in winter months in urban areas compared to levels at altitude due to increased fossil fuel burning for heating. Nevertheless, even the high altitude data sets are noisy.

Random fluctuations in atmospheric measurements could spuriously affect comparisons to tissue ^{14}C measurements, consequently time-averaging, or "smoothing" of the atmospheric data sets was required. Curve smoothing involved specifying a time window (eg 3 months, 6 months, one year), and averaging the measurements within that window. The window was advanced one data point, and then the average recalculated. Repetition of this algorithm generates a series of "average" points, and this defines the smoothed curve. The Calibomb software allows the user to specify the degree to which raw atmospheric data is smoothed. It is recommended that the degree of smoothing should be matched to the formation time of the object being measured. Consequently for this project, determining tissue replacement or formation times was a prerequisite for

generation of the calibration curves. Moreover, it is clear that several curves using different degrees of smoothing should be applied to different tissues from the same individual.

Tissue Formation and/or Replacement

In the case of tooth enamel, which does not turnover, formation times for different teeth were based upon the empirical observations of Nolla 1960, (shown in Figure 5). For most adult teeth, crown formation, defined as progressing from stage 3 to stage 6, occurred over a period of 36 months. Therefore this study suggests that atmospheric calibration curves used for Year-of-Birth estimations based upon tooth enamel ^{14}C should be smoothed at least by this amount.

Hair and nails are similar to teeth in that they too do not turnover. They are inert organic polymers that are continuously extruded from epidermal tissues. Thus the formation rates of these materials needed to be determined. Tissues such as blood, skin and bone are continuously breaking down and being re-synthesized. They exist in a steady state. Thus, replacement time rather than formation time determines the rate of radiocarbon uptake from dietary sources.

In initial experiments, a six month formation/replacement time was selected as a first approximation of the formation/replacement rates. This was based upon empirical measurements of red blood cell life-times of 180 days (Buchholz *et al.* 1999). The experimental strategy was to initially use a six month smoothing function for the atmospheric data set, and then determine replacement times empirically by plotting the ^{14}C contents of known-age tissues against atmospheric ^{14}C measurements. In other words,

empirically test the hypothesized formation/replacement rates, and then modify the curve smoothing function as needed for each tissue type.

This approach would have worked fine had the experiments been conducted before 2003, however the final challenge was that existing atmospheric calibration data sets do not extend beyond that year so extrapolation of existing measurements was required.

Extrapolating Atmospheric Data Sets

The Northern Hemisphere atmospheric radiocarbon curve of Levin and Kromer (2004) ends in 2003. The donor tissues for this study were from individuals who died in 2006 so a short-term solution was to extrapolate the atmospheric data. Obviously, the long term solution is to extend the existing data set by direct measurement of known-age plant tissues such as archived seeds, and this has been proposed in the next phase of the research.

Curve fitting for the extrapolation was not straight forward. The kinetics of carbon dioxide exchange between the atmosphere, the oceans, the terrestrial landscape and the biosphere are complex and so the reduction in atmospheric levels over time do not follow a simple decay kinetics (reference). A pragmatic strategy was adopted: the data were extrapolated beyond 2006 using both linear and second order polynomial extrapolations assuming that these two curves would bracket the true values. It is likely that a second order polynomial extrapolation overestimates contemporary atmospheric radiocarbon levels and a linear extrapolation underestimates them. The extrapolations, along with the soft tissue measurements shown Figure 10, are show in Figure 11A and B.

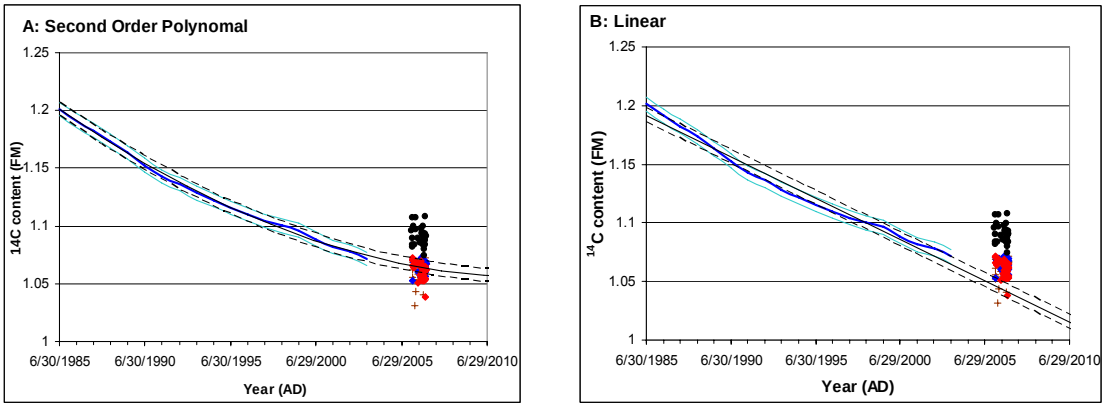

Figure 11A-B: Extrapolation of Atmospheric Radiocarbon Levels Beyond 2003: A, 2nd Order Polynomial extrapolation; B, Linear Extrapolation. The smoothed semi-annual Northern Hemisphere Atmospheric Radiocarbon Curve is plotted in dark blue extending from 1985 to 2003. This line is bracketed by light blue curves at ± 0.007 Fraction Modern that define the one sigma scatter in the atmospheric data. This uncertainty was calculated as the root mean square in a 12 point running average (approximately 6 months) of the Levin data set extending from 1970 to 2003. Superimposed over these curves are: (A), second order polynomial, and; (B) linear curves (solid black) that extrapolate atmospheric values to 2010. These extrapolations are bracketed at ±0.007 FM (black dashed). Tissue radiocarbon measurements shown in Figure 10 are plotted relative to these curves.

Part of the extrapolation involved quantifying the noise in the existing atmospheric measurements, and then assuming this same noise level for the period of extrapolation. Noise was quantified using the Levin and Kromer (2004) data set. A 12-point running average (approximately 0.5 year) was calculated for the raw atmospheric measurements from 1970 to 2003. Nineteen seventy was selected as the starting year for this analysis in order to exclude the earlier, noisier measurements that do not characterize later atmospheric conditions. The root mean square of the differences between the raw and smoothed curves was calculated to be 0.007 FM. The linear and 2nd order polynomial extrapolations were then bracketed by curves offset by plus and minus this amount, as shown in Figures 11 to 13.

The tissue measurements in Figures 11A and B fall either on or slightly above the atmospheric curves depending upon the method of extrapolation. Plots of individual tissue types on the 2nd order polynomial extrapolated atmospheric curves are shown in Figures 12A-D. These plots suggested that most blood, hair and nail samples had radiocarbon contents equivalent to, and in a few cases even slightly lower than, the contemporary atmosphere. This reflects a very high replacement rate, and underscores the effectiveness of these tissues as Year-of-Death markers. The closeness of the match to atmospheric levels might be exaggerated because the 2nd order polynomial extrapolation overestimates atmospheric levels in 2006. In similar plots using linear extrapolations (not shown), all tissues plot above atmospheric values. What is important is that the standard deviations of the measurements of each tissue type are nearly equivalent to the standard deviations within the atmospheric measurements themselves.

The mean and standard deviation of the measurements on the four tissues are shown in Table 5. These measurements fulfill a fundamental objective of this study. Although the measurement precision for single samples is of the order of ± 0.004 FM (see Table 2), calculating a Year-of-Death based upon that value would be overly optimistic because it does not take into account the variation within the population. The standard deviations shown in Table 5 for each tissue type would be more appropriate values.

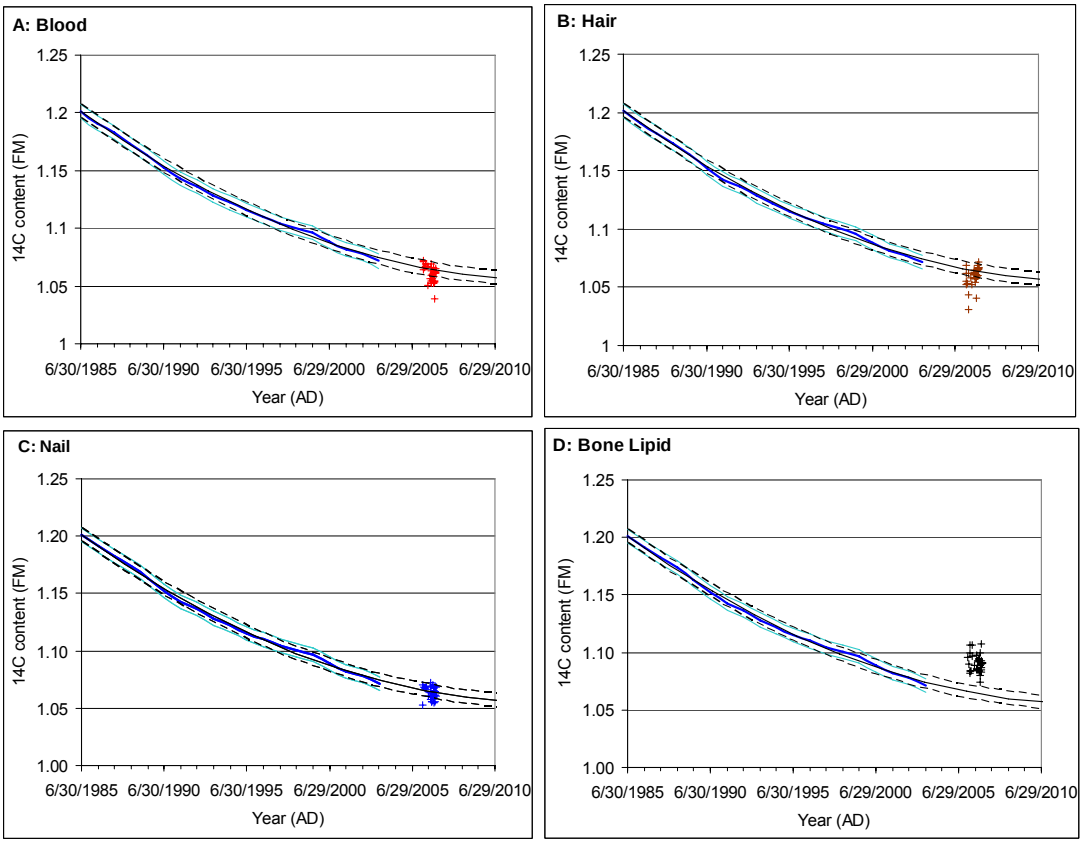

Figure 12 A-D: Individual tissue type radiocarbon measurements plotted against the polynomial extrapolated atmospheric radiocarbon curve: A, Blood; B, Hair; C, Nail; and D, Bone Lipid.

Once again, it is noteworthy that the standard deviation in each set of tissue measurements is nearly identical to the one sigma standard deviation of the atmospheric radiocarbon measurements.

Table 5: Mean and Standard Deviation in Tissue Radiocarbon Measurements. Compare these values to the ±0.007 FM variation in the published atmospheric measurements (Levin and Kromer 2004).

	Blood, n=33	Hair, n=32	Nails, n=35	Bone Lipid, n=31
Mean (FM):	1.0607	1.0595	1.0634	1.0909
Sd (± FM):	0.0069	0.0081	0.0051	0.0080

Quantifying the time-lag between atmosphere and tissue

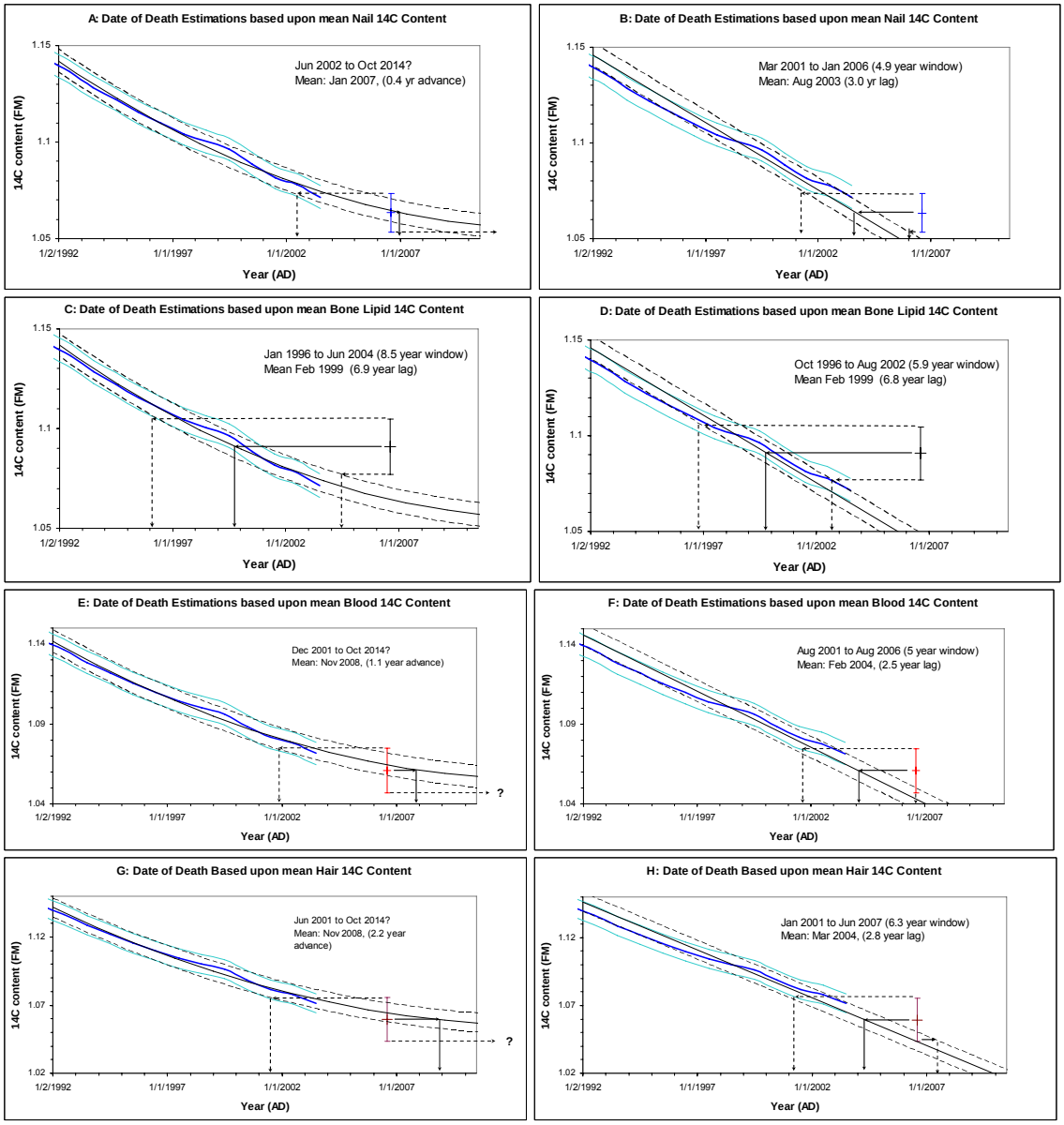

Figure 13 A-H: Determining the lag time of atmosphere-to-tissue radiocarbon transfer and the uncertainty of Year-of-Death estimations based upon tissue radiocarbon content. Estimates based on the radiocarbon content of fingernail (A and B), bone lipid (C and D), blood, (E and F), and hair (G and H) are shown plotted against either 2nd order polynomial (left column) or linear (right column) extrapolations of known atmospheric radiocarbon levels. A mean death year of 2006.6 was used throughout this study (see Table 1). The mean and 2 σ uncertainty of each tissue measurement was plotted against the mean and extrapolated ± 1 σ atmospheric radiocarbon values, and the X intercepts determined for each. The X-intercept of the mean values were taken as the atmosphere-to-tissue time lag, and range between the X-intercepts of the minus 2 σ and the plus 2 σ tissue measurements with the plus 1 σ and the minus 1 σ atmospheric radiocarbon levels defined the precision of that estimate.

When tissue values plot above the atmospheric curve, it indicates the time lag for the transfer of atmospheric radiocarbon into that tissue. This lag is partially related to the rate of tissue replacement. Somewhat analogous to quantifying the formation year of a particular tooth enamel for determining the Year-of-Birth based upon enamel radiocarbon content, determining Year-of-Death based upon soft tissue radiocarbon content requires quantifying the duration of the time lag for that tissue.

The values shown in Table 5 were used to determine the mean time lag for the transfer of atmospheric radiocarbon into each tissue type as well as the precision of Year-of-Death estimates based upon the different tissue types. This was accomplished by plotting the numbers in Table 5 against extrapolated atmospheric radiocarbon curves (Figure 13 A-H) and determining the intercepts.

Figure 13C and D show mean tissue radiocarbon values that were lower than the mean extrapolated atmospheric values. As previously mentioned this highlights the shortcomings of the 2nd order polynomial extrapolation. Where possible tissue lag and Year-of-Death estimation precisions were estimated from plots 12A-H. For example, Figure 13B shows that finger nail radiocarbon content lags three years behind atmospheric values assuming a linear extrapolation, and that the precision of a Date-of-Death estimation based upon finger nail radiocarbon content is of the order of ± 2.5 years. Figures 13C and 13D indicate that bone lipid radiocarbon content lags 6.8 or 6.9 years respectively, behind atmospheric values, and that Year-of-Death estimations based upon bone lipid has an uncertainty of ± 4.2 years if the 2nd Order polynomial validly estimates atmospheric radiocarbon levels, and an uncertainty of ± 3.0 years if atmospheric levels are estimated using a linear extrapolation.

Plots 13 A, E and G show extrapolated Year-of-Death estimations extrapolated into the future. Obviously one would ignore future dates in real applications. The extrapolations were plotted to show the magnitude of the uncertainties that would be expected from tissues obtained from individuals whose Year-of-Death occurred in 2006, but the remains were discovered in future decades. The size of the uncertainty would be reduced in past decades according to the slope of the bomb spike.

Quantifying Uncertainty of Year-of-Death Estimates based upon hypothetical tissue measurements spanning 1955 to 2003

The precision of the Year-of-Death estimations such as those shown in Figure 13 A-depends upon both the population variability of ^{14}C levels in particular tissues, and when death occurred in relation to the bomb spike. In order to illustrate how these variables affect the uncertainty of Year-of-Death estimations, a series of calibrations were performed on hypothetical tissues assigned radiocarbon levels equivalent to the years between 1955 and 2003. These mock tissue measurements were also assigned measurement uncertainties that ranged from ±0.4‰ FM to ± 1.0‰ FM, reflecting the possibility of differing levels of variability within the population. These mock measurements were then calibrated using the Calibomb software program specifying the Levin and Kromer 2004 data set smoothed to semi-annual resolution. The magnitudes of the resulting uncertainties were then plotted as a function of the Year-of-Death. This is shown in Figure 14.

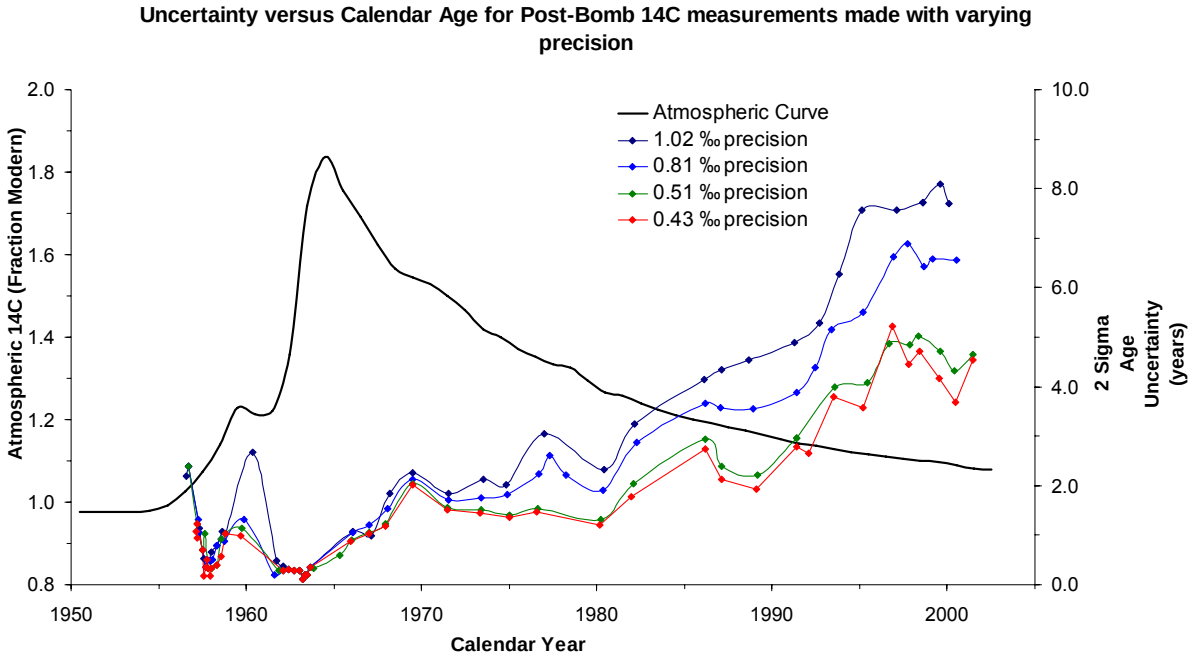

Figure 14: Precision of Year-of-Death Estimation as a function of Year-of-Death Measurement. Calibration was carried out on mock series of tissue measurements made with 0.4 to 1.0‰ precision. The calibration data set was the Levin and Kromer (2004) Northern Hemisphere Atmospheric Radiocarbon curve smoothed semi-annually.

Figure 14 shows that as the precision of the tissue measurement decreases, the precision of the Year-of-Death estimate also decreases. The variability in tissue radiocarbon levels within the population will limit the true measurement precision, and this effect is most pronounced in the later decades (1990 to 2003). For example in 1970, Year-of-Death 2 σ uncertainty is ± 2 years regardless of whether the tissue measurements were assigned 0.4‰ or 1.0‰ uncertainty. However, by 1996, those same differences in measurement precision double the uncertainty in the Year-of-Death estimation from ± 4 to ± 8 years. Recall from Table 5 that the 1 sigma uncertainties for Blood, Hair, and Nails, were 0.7, 0.8 and 0.5‰ FM.

Figure 14 illustrates that the precision of Year-of-Death estimates are quite sensitive to small fluctuations in the slope of the atmospheric radiocarbon curve. In reality, these atmospheric fluctuations are dampened by carbon flow from the atmosphere

into plant, animal and human tissues as carbon flows up the food chain. This dampening can be simulated by time-averaging algorithms that are not just helpful, but necessary for proper estimation of precision. The accuracy of Year-of-Birth and/or Year-of-Death based upon elevated tissue radiocarbon levels requires appropriate curve smoothing, and it has become clear in this research that simply time-averaging the raw atmospheric data by an amount equivalent to the estimated formation age of the tissue in question is not sufficient. The real duration of time averaging that must be considered is the integrated time for carbon flow up the entire food chain from atmosphere through plant and animal, into human tissue.

IV. Conclusions

This study is by no means the first investigation of bomb radiocarbon as a forensic tool. Indeed Jull *et al.* (1995), Wild *et al.* (2000), Cook *et al*. (2006), and Ubelacker *et al.* (2006) had forensics in mind. However, none of these studies examined variability in the population, and this is clearly critical for a meaningful evaluation of the approach.

A total of measurements 276 measurements were made on 9 tissue types from 36 individuals. The objective was to identify tissue types best suited to determination of Year-of-Birth, and Year-of-Death based upon tissue Radiocarbon Content, and to calculate the precision of those determinations.

Tooth enamel radiocarbon content was examined for the determination of Year-of-Birth. Using published sample preparation protocols, tooth enamel radiocarbon was found to be capable of estimating Year of Birth with a mean accuracy of 0.4 years for the period 1961 to 1980, and 4.4 years for the period 1955 to 1960. The lowered accuracy

for the early period reflects an observed under labeling of teeth in early years of bomb-spike. This suggests that it would be best to abandon the strategy that compares tooth and atmospheric radiocarbon levels in favor of a direct comparison of unknown tooth radiocarbon levels with a database of radiocarbon measurements from known-age teeth.

The radiocarbon content of skeletal tissues was found to be highly variable. This was expected given the relatively slow replacement of bone. However, it was particularly important to demonstrate the variability in bone collagen and apatite radiocarbon content between individuals of a similar age. The slow and variable rate of bone replacement means bone collagen and apatite are poor indicators of either Year-of-Birth, or Year-of-Death. In contrast to bone collagen and bone mineral, bone lipid radiocarbon levels were found to be much closer to contemporary atmospheric levels, and remarkably similar within the sample population. Although bone lipid turned over more slowly than other soft tissues, the low population variability suggested its potential as an indicator of Year-of-Death. The potential advantage of targeting lipids is that they are more likely to survive in the burial environment compared to soft tissue components composed of water soluble components such as proteins and/or carbohydrates.

The radiocarbon contents of five soft tissue types, blood, nails, hair, skin lipid, skin collagen, were investigated. The radiocarbon contents of nails, hair and blood were found to be remarkably similar to each other, and all were close to assumed contemporary atmospheric radiocarbon values. This suggests these would be good markers for Year-of-Death, based upon ^{14}C content. The precision of the Year-of-Death estimations were of the order of ± 3 to 4 years for this data set. However, precision would vary according to when death occurred in relation to the peak of the bomb spike.

In contrast to the suggestion that Year-of-Birth estimations should be based upon direct comparisons between radiocarbon measurements from unknown and known-age teeth, Year-of-Death estimations from soft tissue radiocarbon measurements will probably always dependent upon comparisons between tissues and atmospheric radiocarbon levels. The reason is simply that it is unlikely a comprehensive reference sample set of known-age soft tissue samples from the past 60 years could be assembled for measurement. It is unlikely that formaldehyde or gluteraldehyde preserved tissue specimens could be used, as both of those chemicals contain carbon themselves and their preservative properties depend upon covalent bonding to the tissues they preserve. A comparison of soft tissue radiocarbon levels with atmospheric radiocarbon levels thus involves greater uncertainties than direct tissue-to-tissue comparisons. However, the existing evidence is that the transfer is rapid enough as to make blood, hair and nail tissue levels appear identical to the contemporary atmosphere.

Differences in the eating habits of individuals potentially pose a problem for the method. The reason is that consumption of high levels of sea food has the effect of depressing tissue radiocarbon levels. This affect has been noted in archaeological investigations involving radiocarbon dating of coastal communities whose dietary intake was dominated by the consumption of sea foods. However, this effect did not appear to be a problem in this study of a modern population. Stable isotope measurements on the bone collagen from the sample population indicated, with one exception, a remarkably homogeneous diet, with no evidence significant marine food intake. The one exception was a single individual who appeared to have a very high corn/legume-based diet. Such a diet would not perturb tissue radiocarbon levels, but the significance of the observation is

that this individual was the sole Hispanic in the sample set. The result suggests that carbon and nitrogen stable isotopes might prove valuable for the forensic identification of culturally determined dietary habits. This suggests a new avenue of research.

Implications for policy and practice

These results have immediate implications for public policy and practice. At the very least detection of elevated levels of radiocarbon in human remains definitively places the life and death of that individual within the past 60 years. Moreover, these experiments demonstrated that measurement of radiocarbon levels in teeth can determine Year-of-Birth within 4 years, but usually with much higher accuracy. This study was not designed to provide statistical measurements of precision because the sample population, though all dying in 2006, were born in different years. This reduced the number of data points available in a particular year for precision calculation.

The routine application for forensic determination of Year-of-Birth would require more measurements on teeth from geographically and temporally dispersed individuals to assemble a reference data set. Such an undertaking is a practical and reasonable project. The unanswered questions, such as why radiocarbon uptake in individuals in the late 1950's is lower than expected, or the degree to which dietary differences might alter tooth ^{14}C content, means it is likely that the variability in the US population is greater than the variability seen in the small population in this study. However, a possible reduction in precision by no means invalidates the method. Moreover, assembling a reference set of known-age teeth with which to address those questions seems a surmountable challenge.

Fortuitously, the measurement of enamel radiocarbon levels is entirely compatible with protocols for the extraction of DNA from teeth, in which the crown is discarded as a waste product (reference).

Radiocarbon measurements in blood, nail, hair, and bone lipids show potential for the determination of Year-of-Death. The crucial observation from this study is that blood nails and hair have remarkably similar radiocarbon contents, and these levels are very close to ambient atmospheric levels. In other words, the replacement of these tissues is very rapid. Moreover, hair and nails are relatively resistant to decay and so might serve as valuable indicators of Year-of-Death. However, more measurements are needed to investigate whether the sampled population truly represents the larger US population.

Determining Year-of-Death from soft tissues has the obvious drawback that they are highly susceptible to post mortem decay. Soft tissue preservation does occur in some environments, moreover, a strategy to overcome this is suggested below.

The results of the study demonstrated that the precision Year-of-Death determination is variable, and is diminishing as time goes on. However, within the context of this study, uncertainties of the order of ± 3 to 4 years were determined for individuals dying in 2006.

The strength of this approach compared to other methods, is that radiocarbon measurements have the potential to provide significant forensic metrics, at extreme post mortem intervals. The radiocarbon levels in tissues are fixed during life, and thus are unaffected post-mortem by environmental conditions such as temperature, humidity, etc, that can confound forensic methods that determine post-mortem interval based upon

diagenetic or taphonomic processes. Consequently this approach would be complementary to such methods.

Implications for further research

Tooth Enamel Radiocarbon Research

We did not devote resources to measurements of multiple teeth. Our results would benefit from systematic measurements on several teeth from each individual. Measurements on multiple teeth are necessary to establish which side of the bomb spike the tooth enamel was formed on. Cook *et al.* (2006) suggest using different fractions within the same tooth, but given the deviations we have seen, measurements on multiple teeth are advisable.

Unlike the measurements on fast replacement tissues, the teeth measurements did not provide indications of variability with the population. To carry out such a study, one would have to obtain teeth from thirty individuals born in the same year, and measure the ^{14}C content of the same tooth from each individual. Such a study is now merited, given the nature of the disagreements between our results and those of Spalding *et al.* (2005a) and Cook *et al.*(2006). Finally, our data show tooth enamel was underlabeled compared to the other published studies. It will be important to investigate whether this reflects experimental, geographic, or other differences.

Tissue Preparation Protocol Research

Organic solvents were used in several tissue preparation protocols. Although these solvents are routinely used in conventional radiocarbon dating, a reviewer pointed out

that the possible effect of solvents on radiocarbon content of the processed tissues such as bone lipid and hair have not been properly investigated. It is essential that future investigations address this question through fully controlled experiments.

US-wide investigation of Dietary Variability and its affect on tissue radiocarbon levels

Our results on the bone collagen carbon and nitrogen stable isotope values indicated a remarkable homogeneity in the diet of the sample population, with one exception. There was no indication that anyone's diet consisted of large quantities of marine foods, and this bodes well for the potential accuracy of the method, if the current results are representative of the larger US population. A single outlier, showed stable isotope values that were consistent with a high corn/legume-based diet, raising the possibility that carbon and nitrogen stable isotope values could be used to determine eating habits from skeletonized human remains. The outlying values were obtained from the single Hispanic individual and so it is possible that collagen stable isotope values could be used as weak evidence of racial origin and/or socioeconomic status.

Looking for Proxies of Soft Tissue Radiocarbon Content

The results of this study indicate that soft tissues such as blood, nails, hair, and to a lesser extent skin and bone lipids, have radiocarbon contents close the contemporary atmosphere, and thus can serve as a marker for Year-of-Death. As previously mentioned, the difficulty with such an approach is that soft tissues are most susceptible to decay. What is required are decay-resistant proxies of soft tissue radiocarbon content. I propose that such a proxy might be found in the pupal cases of insects that feed on decaying soft tissues. Various fly species of the family *Calliphoridae* lay eggs on carrion, and their

larvae feed on decomposing soft tissues. Consequently, the radiocarbon content of the larvae assumes that of the host organism. The emergent adult fly also has the same radiocarbon carbon content as the host organism's soft tissues. Although the adults leave, the discarded pupal cases stay in the soil beneath the decaying carrion. The pupal cases, were synthesized by the larvae and thus should contain the same radiocarbon content as the host tissues. The pupal cases are composed of chitin which is quite resistant to decay. My hypothesis is that the recovery of insect pupal cases from beneath skeletonized human remains, might prove to be robust indicators of Year-of-Death.

Reconstruction of the Atmospheric Radiocarbon Levels after 2003AD

As evident in the results dealing with soft tissue radiocarbon measurement, the study suffered from a lack of available data on atmospheric radiocarbon levels. The key data set compiled in Levin and Kromer (2004) only presents atmospheric measurements up to the summer of 2003. If this method were to be adopted for forensic determinations of Year-of-Death, it would be important to update this data to the present.

The Levin and Kromer (2004) data sets are based upon measurements obtained from directly sampled atmospheric gases. Consequently, would not be possible to fill in the missing data points for the time period 2003.5 to the present in precisely the same manner. However, it would be possible to infer atmospheric levels in the past from measurements of the radiocarbon content of archived leaves and/or seeds from annual plants. In fact, such measurements might be more relevant to this application, as plant tissues and seeds are consumed as food sources, and also have time-integrated radiocarbon contents.

Thus, a necessary future project should be the assembly of archived seeds and or plant tissues from various regions of the US, from years past. A portion of these materials should be from years overlapping the known atmospheric measurements (ie, before 2003 AD) and the rest from 2003 to the present. Radiocarbon measurements on these samples would be used to extend the Levin and Kromer (2004) data set up to the present. This curve could then be used for contemporary forensic dating studies.

V. References

Bhardwaj R D, Curtis M A, Spalding K L, *et al.* (2006) Neocortical neurogenesis in humans is restricted to development. *Proceedings of the National Academy of Science* 103 (33):12564-12568

Broecker W S, Schulert A, Olson E A (1959) Bomb Carbon-14 in Human Beings. *Science* 130:331-332.

Buchholz, B A, Arjomand A, Dueker S R, Schneider P D, Clifford A J, and Vogel J S (1999) *Anal. Biochem.* 269:348.

Cook G T, Dunbar E, Black S M, Xu S (2006) A preliminary assessment of Age at Death Determination using the nuclear weapons testing ^{14}C of Dentine and Enamel. *Radiocarbon* 48(3):305-313.

Coplen, T.B. (1994) Reporting of Stable Hydrogen,Carbon, and Oxygen Isotopic Abundances *Pure and Applied Chemistry* 66(2):273-276.

Costa, R. L. (1986) Determination of age at death: dental analysis. In Zimmerman, M. R. and Angel, J. L. (eds) *Dating and Age Determination of Biological Materials*, 248-269. Croom Helm, London.

Donahue, D. Linick, T.W. and Jull A.J.T. (1990) Isotope Ratio and Background Corrections for Accelerator Mass Spectrometry Radiocarbon Measurements *Radiocarbon* 32(2):135-142.

Gillespie R, Hedges R E M, Wand J O (1984) Radiocarbon Dating of Bone by Accelerator Mass Spectrometry *Journal of Archaeological Science* 11:165-170.

Haynes V (1968) Radiocarbon: Analysis of Inorganic Carbon of Fossil Bone and Enamel *Science* 161 (3842):687-688.

Hua Q, Barbetti M (2005) Influence of Atmospheric Circulation on Regional CO_2-^{14}C Differences *Journal of Geophysical Research: Atmospheres* 112: D9

Jull A J T, Kalin R M, Burns K (1995) Bomb-derived ^{14}C in Human Bones: AMS application to Forensic Science. American Chemical Society, 205th Annual Meeting, Anaheim, CA, April 1995.

Levin I, Kromer B (2004) The tropospheric $^{14}CO_2$ level in mid-latitudes of the Northern Hemisphere (1959-2003). *Radiocarbon* 46:1261-1272.

Libby W F, Berger R, Mead J F, Alexander G V, Ross J F (1964) Replacement Rates for Human Tissue from Atmospheric Radiocarbon *Science* 146:1170-1172.

Lynnerup N, Kjeldsen H, Heegaard S, Jacobsen C Heinemeier J (2008) Radiocarbon Dating of the Human Eye Lens Crystallines Reveal Proteins without Carbon Turnover throughout Life. *PLoS One (www.plosone.org)* 1: 1-3.

Nolla C.M. (1960) The Development of Permanent Teeth *Journal of Dentistry of Children* 27:254-266.

Nydal R, Lővseth K (1965) Distribution of Radiocarbon from Nuclear Tests *Nature* 208:1029-1030.

O'Connell, T. C. and Hedges, R.E.M. (1999) Investigations into the Effect of Diet on Modern Human Hair Isotopic Values *American Journal of Physical Anthropology* 108:409-425.

Reimer P.J., Brown T. A. and Reimer R. W. (2004) Discussion: Reporting and Calibration of Post-Bomb ^{14}C Data. *Radiocarbon* 46(3):1299-1304.

Reimer, P J, Baillie M G L, Bard E, Bayliss A, Beck J W, Bertrand C JH, Blackwell P G, Buck C E, Burr G S, Cutler K B, Damon P E, Edwards R L, Fairbanks R G, Friedrich M, Guilderson T P, Hogg A G, Hughen K A, Kromer B, McCormac G, Manning S, Ramsey C Bronk, Reimer R W, Remmele S, Southon J R, Stuiver M, Talamo S, Taylor F W, van der Plicht J, Weyhenmeyer C E (2004) IntCal04 terrestrial radiocarbon age calibration, 0-26 cal kyr BP. *Radiocarbon* 46(3):1029-1058

Slota, P.J., Jull, A.J.T., Linick, T.W. & Toolin, L.J. (1987). Preparation of small samples for ^{14}C accelerator targets by catalyst reduction of CO. Radiocarbon 29, 303-306.

Spalding K.L., Buchholz B. A, Bergman L.E., Druid H., Frisen J. (2005a) Age written in teeth by Nuclear Tests *Nature* 437(7057):333-334.

Spalding K L, Bhardwaj R D, Buchholz B A, Druid H and Frisén J (2005b) Retrospective Birth Dating of Cells in Humans *Cell* 122(1):133-143.

Stenhouse M. J. and Baxter M. S. (1977) Bomb ^{14}C as a biological tracer. *Nature* 267: 828-832.

Stott A W, Evershed R P, Tuross N. (1997) Compound-specific approach to the δ^{13}C analysis of cholesterol in fossil bone. *Organic Geochemistry* 26 (1/2): 99-103.

Stuiver M, Polach H A (1977) Reporting of ^{14}C data.*Radiocarbon* 19(3):355-363.

Stuiver M, Reimer PJ, Bard E, Beck JW, Burr GS, et al. (1998) INTCAL98 radiocarbon age calibration, 24000—cal BP. *Radiocarbon* 40:1041-1083.

Ubelaker D H, Buchholz B A, Stewart J E B (2006) Analysis of Artificial Radiocarbon in Different Skeletal and Dental Tissue Types to Evaluate Date of Death. *Journal of Forensic Sciences* 51(3):484-488.

Wild E M, Arlamovsky K A, Golser R, Kutschera W, Priller A, Puchegger S, Rom W, Steier P, Vycudilik W (2000) ^{14}C dating with the bomb peak: An application to forensic medicine. *Nuclear Instruments and Methods in Physics Research* B172: 944-950.

VI. Dissemination of Research Findings

Scientific Conferences

American Academy of Forensic Sciences 58th Annual Meeting: National Institute of Justice, Office of Science and Technology, Investigative and Forensic Sciences Division, General Forensics R&D Grantees Meeting, Seattle Washington, February 21, 2006

Poster presentation:
Measuring Atomic Bomb-Derived ^{14}C-Levels in Human Remains to Determine Year-of-Birth and/or Year-of-Death
Gregory W. L. Hodgins

American Academy of Forensic Sciences 59th Annual Meeting: National Institute of Justice, Office of Science and Technology, Investigative and Forensic Sciences Division, General Forensics R&D Grantees Meeting, San Antonio, Texas, February 20, 2007

Oral Presentation:
Measuring Atomic Bomb-Derived ^{14}C-Levels in Human Remains to Determine Year-of-Birth and/or Year-of-Death
Gregory W. L. Hodgins

National Institutes of Justice Annual Conference, Arlington Virginia, July 23-25 2007. Panelist on Forensic Anthropology Session "When Bones Talk: The Role of Forensic Anthropology in Cases Involving Unidentified Remains"

Oral Presentation:
Measuring Atomic Bomb-Derived ^{14}C-Levels in Human Remains to Determine Year-of-Birth and/or Year-of-Death
Gregory W. L. Hodgins

11th International Conference on Accelerator Mass Spectrometry, Rome Italy, September 14-19, 2008

Oral Presentation:
"Radiocarbon Measurements on Modern Human Tooth Enamel"

Gregory W.L Hodgins, Autumn Carey, Marcus B. Lee, Lindsay H. Trammel, and Richard L. Jantz

(Upcoming)
American Academy of Forensic Sciences 61st Annual Meeting, Denver, Colorado
February 16-21, 2009

Oral Presentation:
"Year-of-Death Determination based upon the measurement of Atomic Bomb-Derived Radiocarbon in Human Soft Tissues"
Gregory W.L Hodgins, Autumn Carey, Lindsay H. Trammel, Lee Meadows Jantz, and Richard L. Jantz

Oral Presentation:
"*Insect Pupal Casings as Decay-Resistant Reservoirs of Human Soft Tissue Radiocarbon Content"*
Gregory W. L. Hodgins

Invited Talks

Forensic Anthropology Center, University of Tennessee, Knoxville, TN, January 26, 2006.
"Life and Death in the Nuclear Age: Measuring Bomb-Derived ^{14}C in Human Remains"
Gregory W. L. Hodgins

Barrow Arctic Science Consortium, Barrow, Alaska, May 24, 2007.
"Helping the Dead Speak: Measuring Atomic Bomb-Derived Radiocarbon in Human Remains for the Forensic Determination of Year-of-Birth and/or Year-of-Death"
Gregory Hodgins

Library of Congress,Washington DC, December 12 2007:
Topics in Preservation Science Lecture Series: *"ÁMS Radiocarbon Dating of Museum Objects"* Gregory W. L. Hodgins

Publications

Gregory W.L Hodgins, Autumn Carey, Marcus B. Lee, Lindsay H. Trammel, and Richard L. Jantz (Submitted) Radiocarbon Measurements on Modern Human Tooth Enamel, *Nuclear Instruments and Methods in Physics Research(B)*

Gregory W.L Hodgins, Autumn Carey, Lindsay H. Trammel, Lee Meadows Jantz, and Richard L. Jantz (Planned) Year-of-Death Determination based upon the measurement of Atomic Bomb-Derived Radiocarbon in Human Soft Tissues. *Journal of the American Academy of Forensic Sciences*

Gregory W. L. Hodgins (Planned) Insect Pupal Casings as Decay-Resistant Reservoirs of Human Soft Tissue Radiocarbon Content. *Journal of the American Academy of Forensic Sciences*

Gregory W. L. Hodgins (Planned) Bone collagen stable isotope values in human remains as indicators of diet, socio-economic class and/or race.

VII. Acknowledgements

Many people contributed their time and expertise to this work. I would like to acknowledge the help of Professor Richard Jantz, Director, and Dr. Lee Meadows Jantz, Coordinator of the Forensic Anthropology Center, University of Tennessee, Knoxville, for providing the tissue samples used in this study. Lindsay Trammel, a graduate student in the Anthropology Department, University of Tennessee, carried out the actual sampling tasks with skill and perseverance. Professor Richard Evershed, and Dr. Lorna Corr, University of Bristol, UK advised me on lipid extraction methods. At the University of Arizona, students Bongi Bishop and Jessica Cerezo made important contributions during the early stages of the research. The bulk of the sample processing chemistry was undertaken with skill and determination by Autumn Carey and Marcus Lee, and AMS measurements were carried out by the staff of the NSF-Arizona AMS Facility, University of Arizona. Professor Tim Jull, and colleagues Drs. Warren Beck and George Burr of the AMS Lab also supported the work with helpful comments and suggestions. Finally, I would like to thank the National Institutes of Justice for the funding and administrative support required to complete this research.

VIII. Figures

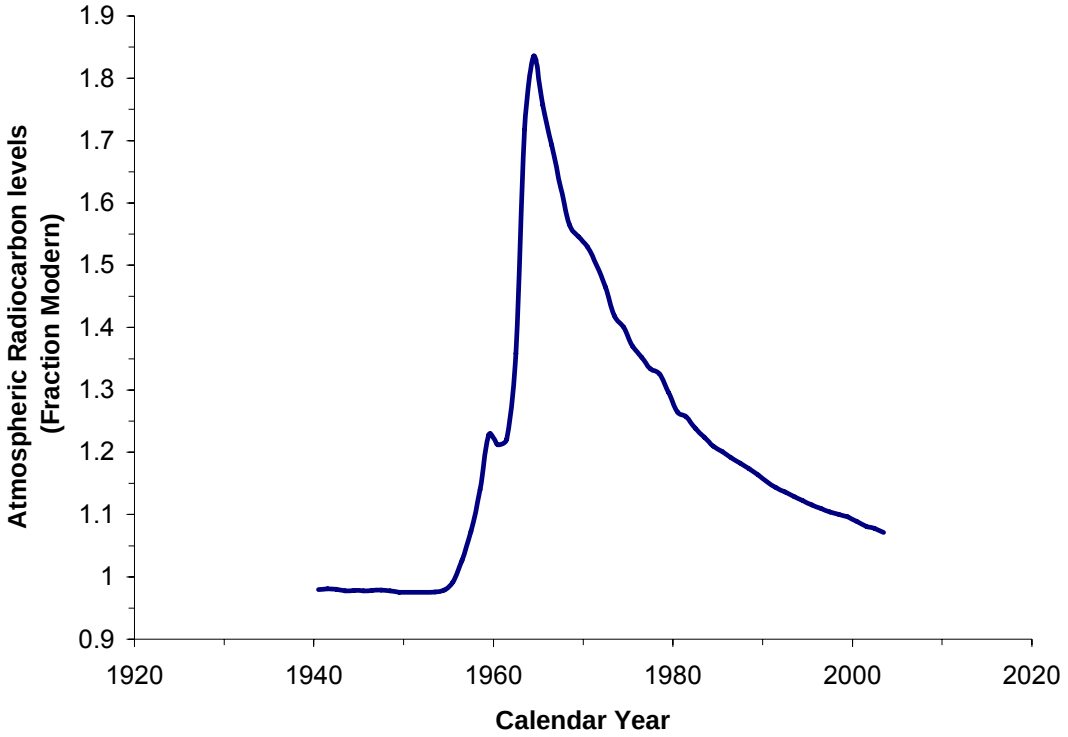

Figure 1: Northern Hemisphere Atmospheric Radiocarbon Levels 1940 to 2003AD. The levels are expressed in terms of Fraction Modern, where 1.0 FM (Fraction Modern) represents the level of atmospheric radiocarbon in 1950 AD without anthropogenic influence. Archaeological material 5730 years or one half-life old, contains 0.5 FM radiocarbon by definition: two halflives, 0.25FM, etc. This scale is used for expressing recent radiocarbon measurements because 1950 AD was selected as the "zero year" by convention. FM avoids the paradox of expressing radiocarbon dates from the past 60 years in terms of negative years. The curve is a composite of data sets from Stuiver et al. (1998) and Levin and Kromer (2004), and has been annually smoothed.

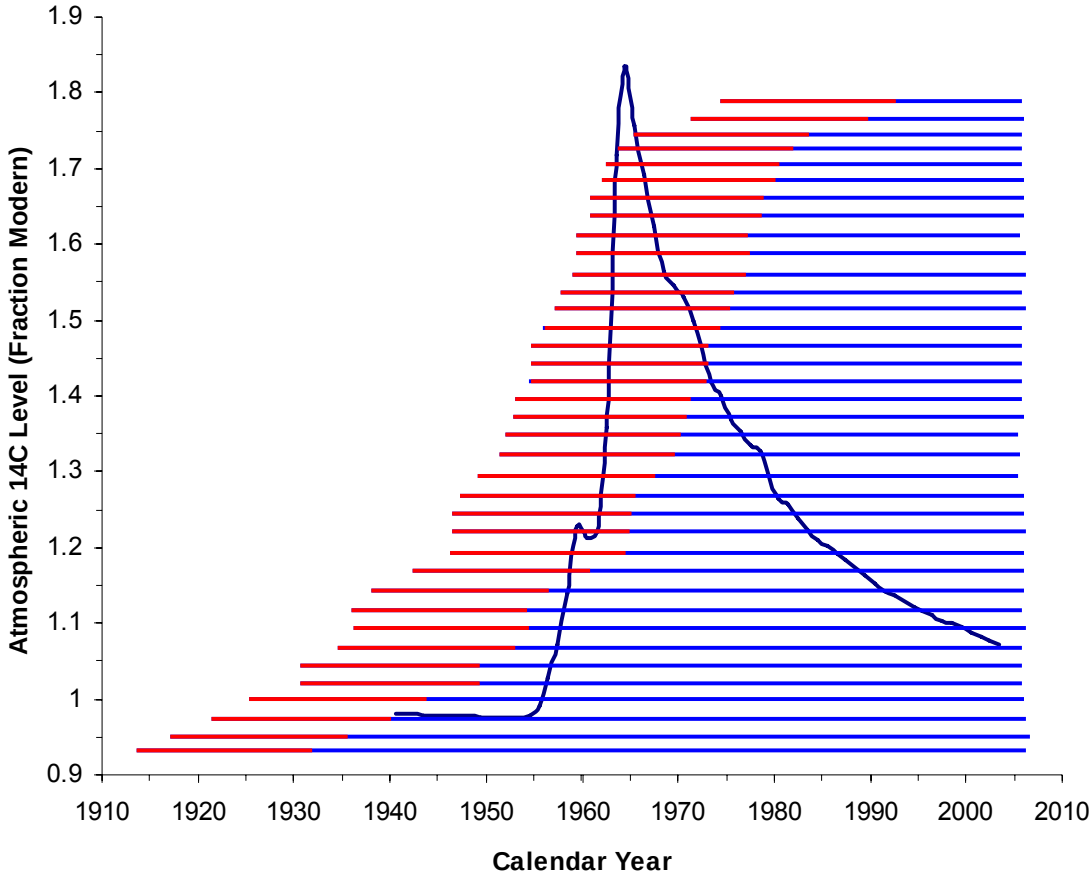

Figure 2: Life Spans of 36 Donors compared to Atmospheric Radiocarbon Levels. The average annual atmospheric ^{14}C levels (modified from Levin and Kromer 2004), is shown in dark blue, and plotted as Fraction Modern ^{14}C. The lifespans of individual donors are represented as a 36 timelines superimposed over the atmospheric ^{14}C curve. These lines span from Years of Birth (ranging from 1912 to 1975), to Year of Death (all in 2006). The first 20 years of each timeline is highlighted in red. This is the period within which adult dentition and full skeletal mass is achieved.

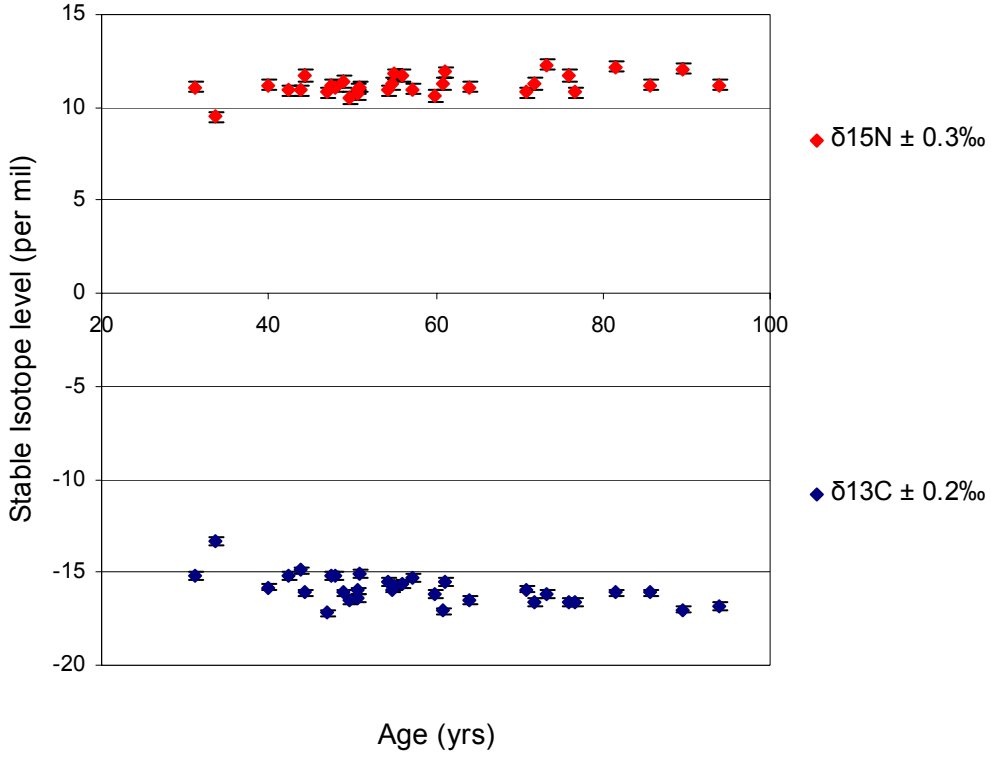

Figure 3: Bone Collagen $\delta^{13}C$ and $\delta^{15}N$ versus Age. Measurements are shown for 32 Donors: four Donors (40-06, 61-06, 69-06, and 81-06) did not provide collagen.

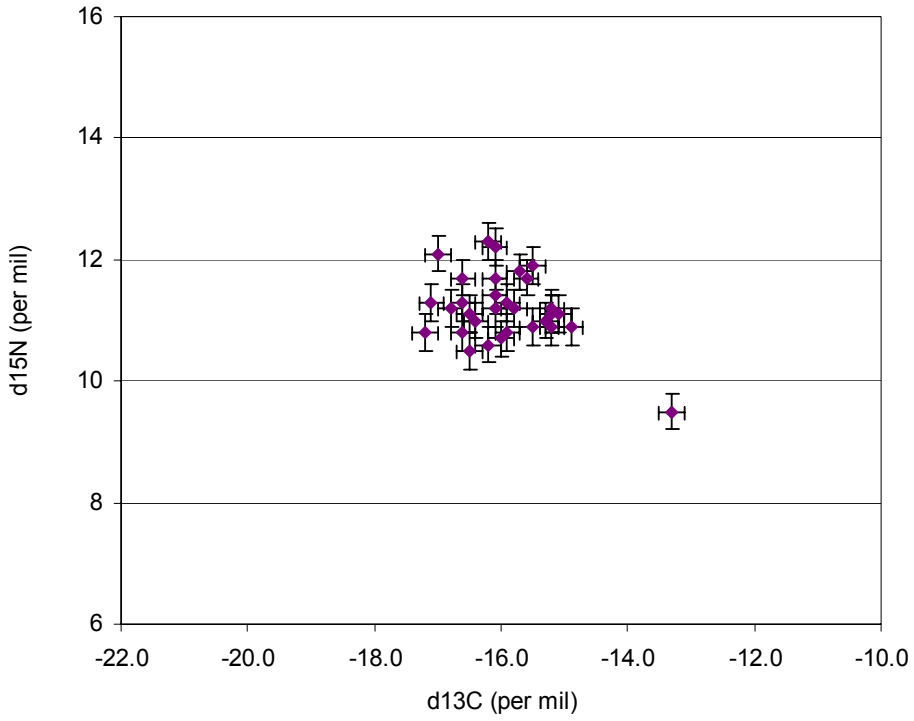

Figure 4: Bone Collagen δ13C versus δ15N. Measurements are shown for 32 Donors: four Donors (40-06, 61-06, 69-06, and 81-06) did not provide collagen.

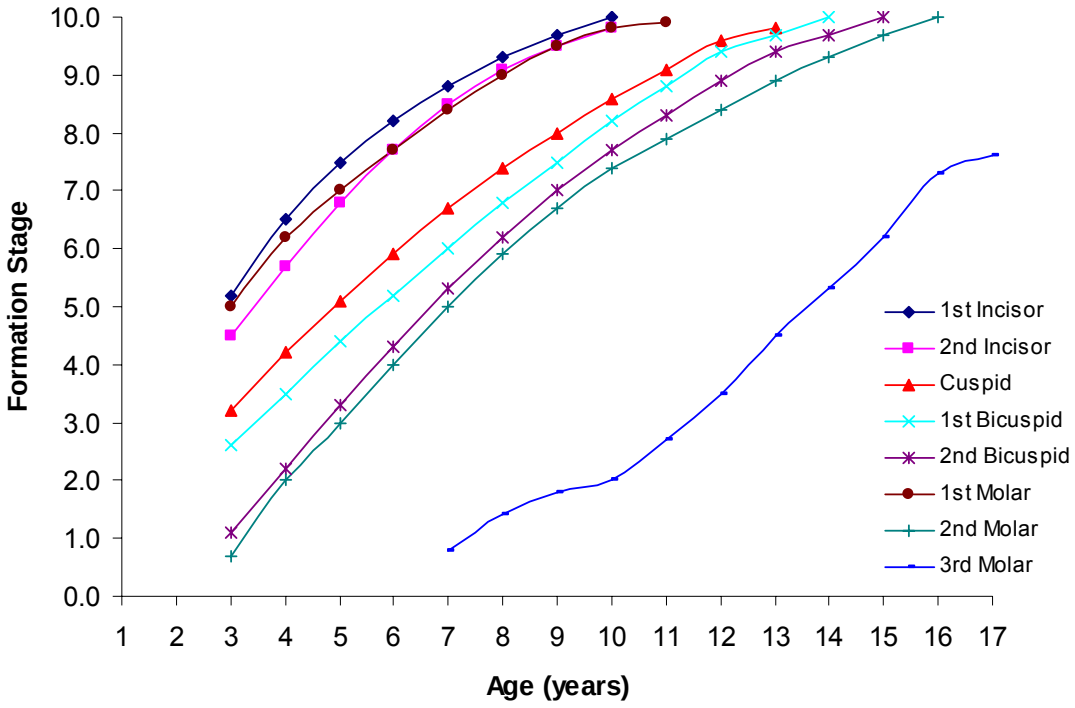

Figure 5: Mandibular tooth Formation in Boys (from Nolla 1960).

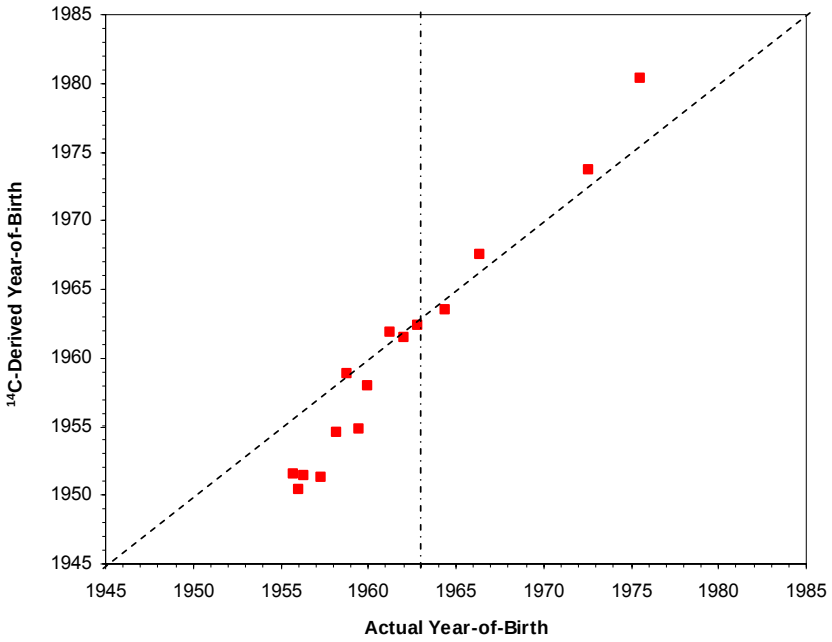

Figure 6: Comparison of Tooth Enamel ^{14}C-Derived Birth Year with Actual Birth Year. The dashed line represents one-to-one correspondence. The vertical dot-dash line indicates the approximate peak of Northern Hemisphere Atmospheric radiocarbon levels. Teeth from 15 Donors were measured.

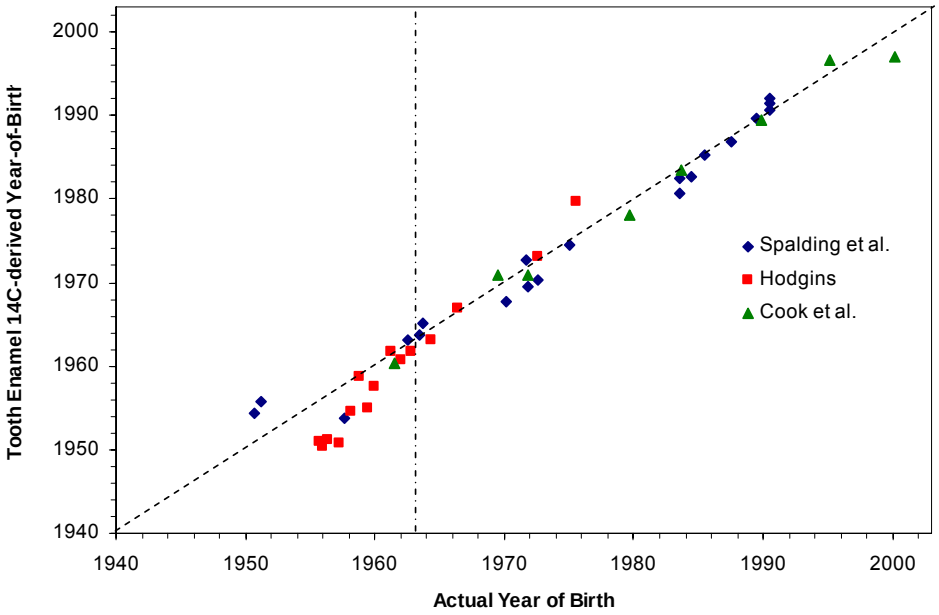

Figure 7: Comparing results from three studies using tooth enamel radiocarbon content to derive Year-of-Birth. The fifteen data points from this study (red squares) are combined with twenty measurements by Spalding *et al.*(2005a) and eight measurements by Cook *et al.* (2006).

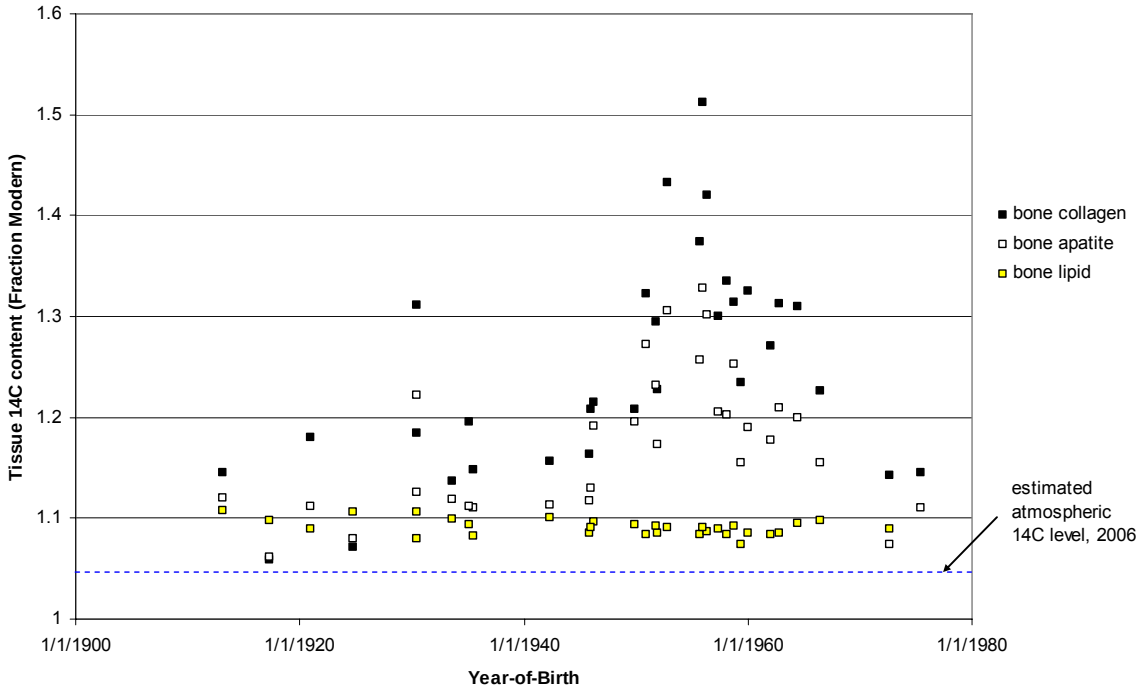

Figure 8: The radiocarbon content of bone collagen, bone apatite, and bone lipid fractions from 36 donor individuals who died in 2006. The data are arranged according to the birth year of each donor. Individuals born before 1940 would have achieved adult stature before atmospheric radiocarbon levels rose due to above-ground nuclear testing. Consequently elevated bone radiocarbon levels in these individuals are a consequence of tissue replacement alone. Ambient atmospheric radiocarbon levels before the atomic age were 0.97 Fraction Modern.

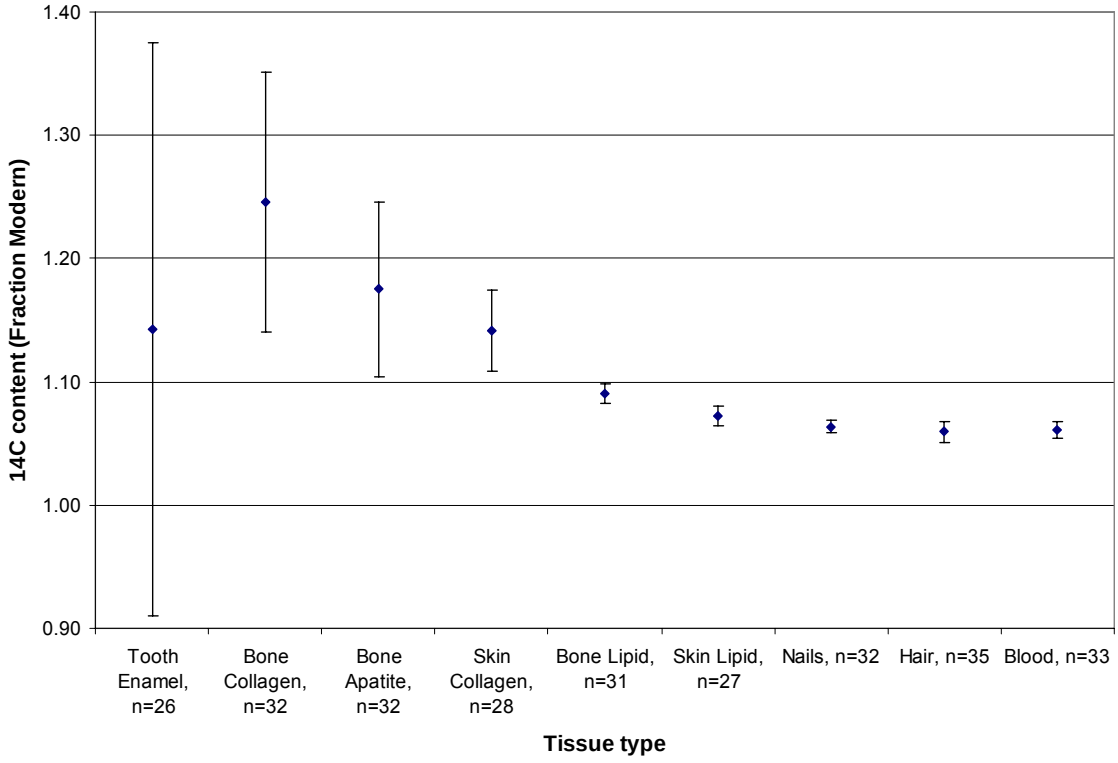

Figure 9: Tissue ^{14}C contents for 36 Individuals who died in 2006. The bars represent one standard deviation.

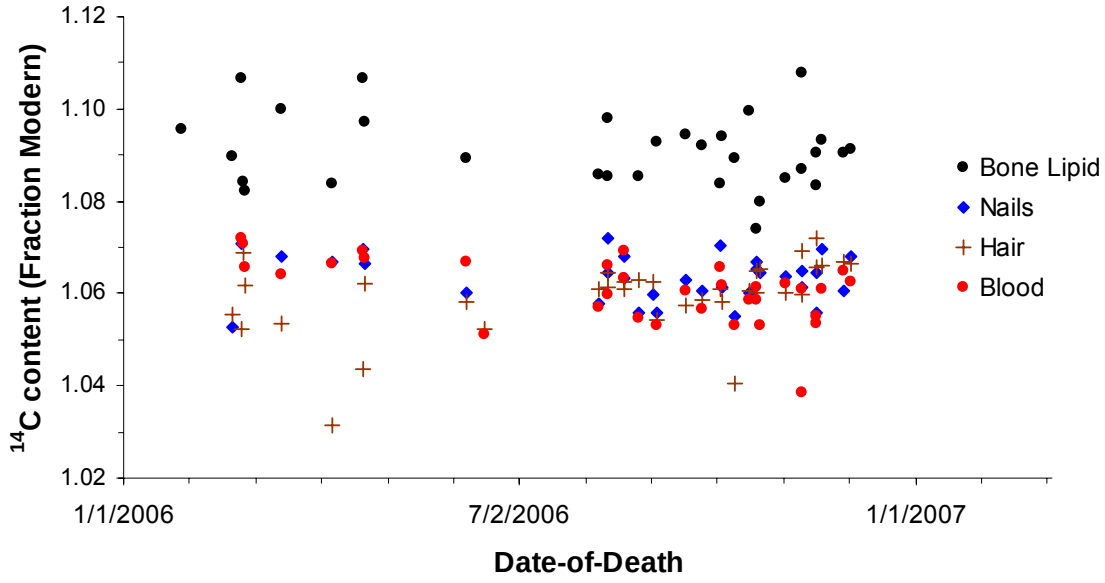

Figure 10: Blood, Hair, Nails and Bond Lipid radiocarbon content versus Date of Death.

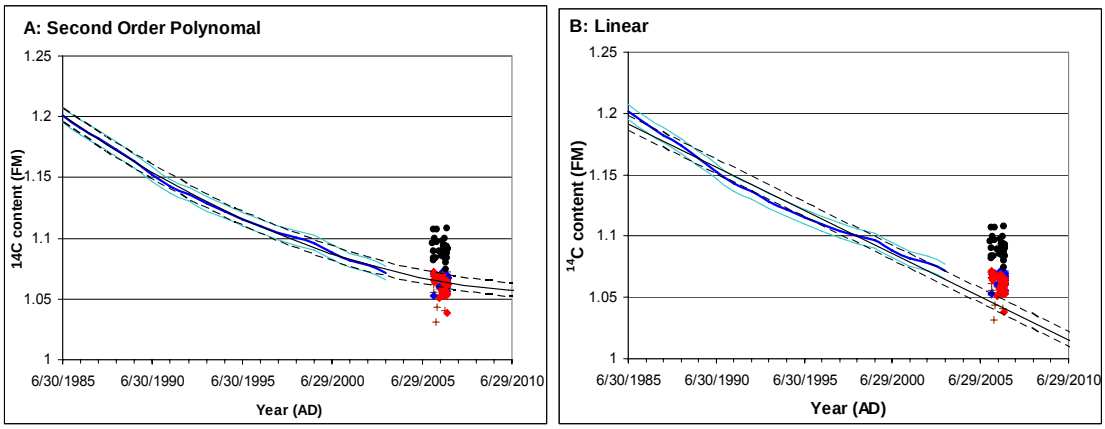

Figure 11A-B: Extrapolation of Atmospheric Radiocarbon Levels Beyond 2003: A, 2nd Order Polynomial extrapolation; B, Linear Extrapolation. The smoothed semi-annual Northern Hemisphere Atmospheric Radiocarbon Curve is plotted in dark blue extending from 1985 to 2003. This line is bracketed by curves at ± 0.007 Fraction Modern. This uncertainty was calculated as the root mean square in a 12 point running average of the Levin data set extending from 1970 to 2003. Superimposed over these curves are: (A), second order polynomial, and; (B) linear curves that extrapolate atmospheric values to 2010. These extrapolations are bracketed at ±0.007 FM. Tissue radiocarbon measurements shown in Figure 10 are plotted relative to these curves.

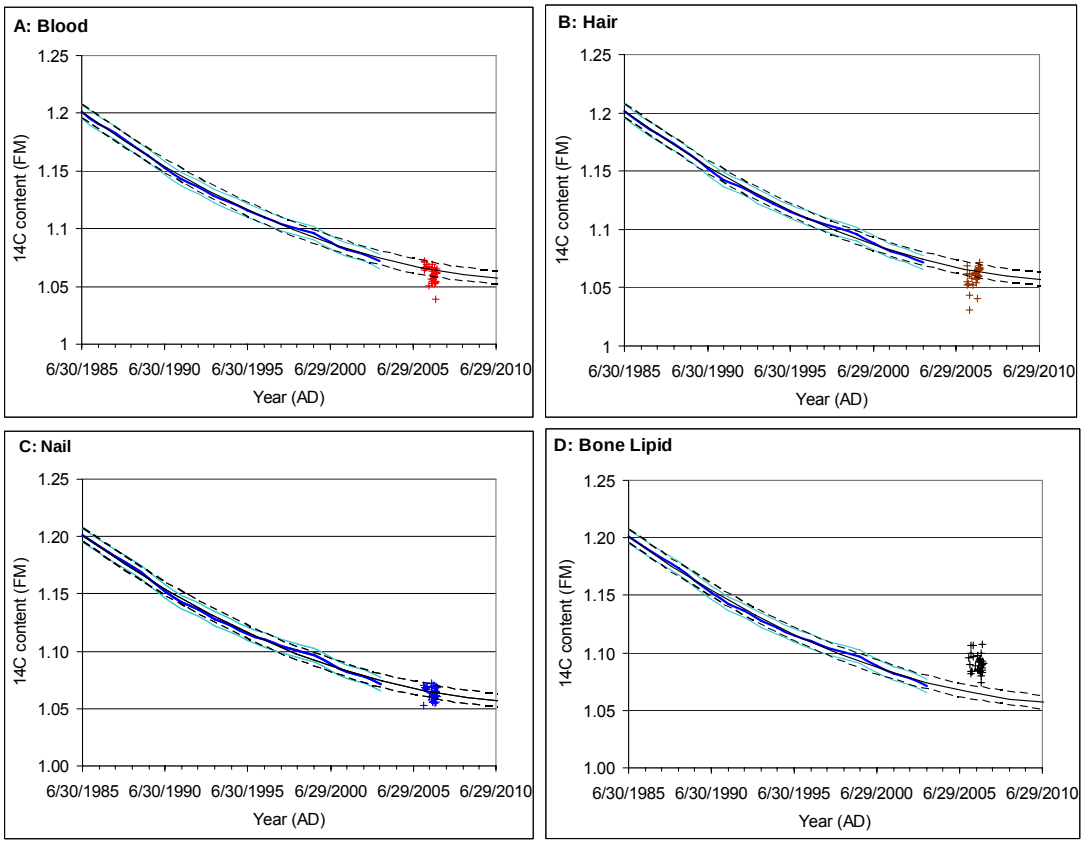

Figure 12 A-D: Individual Tissue Type radiocarbon measurements plotted against the polynomial extrapolated atmospheric radiocarbon curve: A, Blood; B, Hair; C, Nail; and D, Bone Lipid.

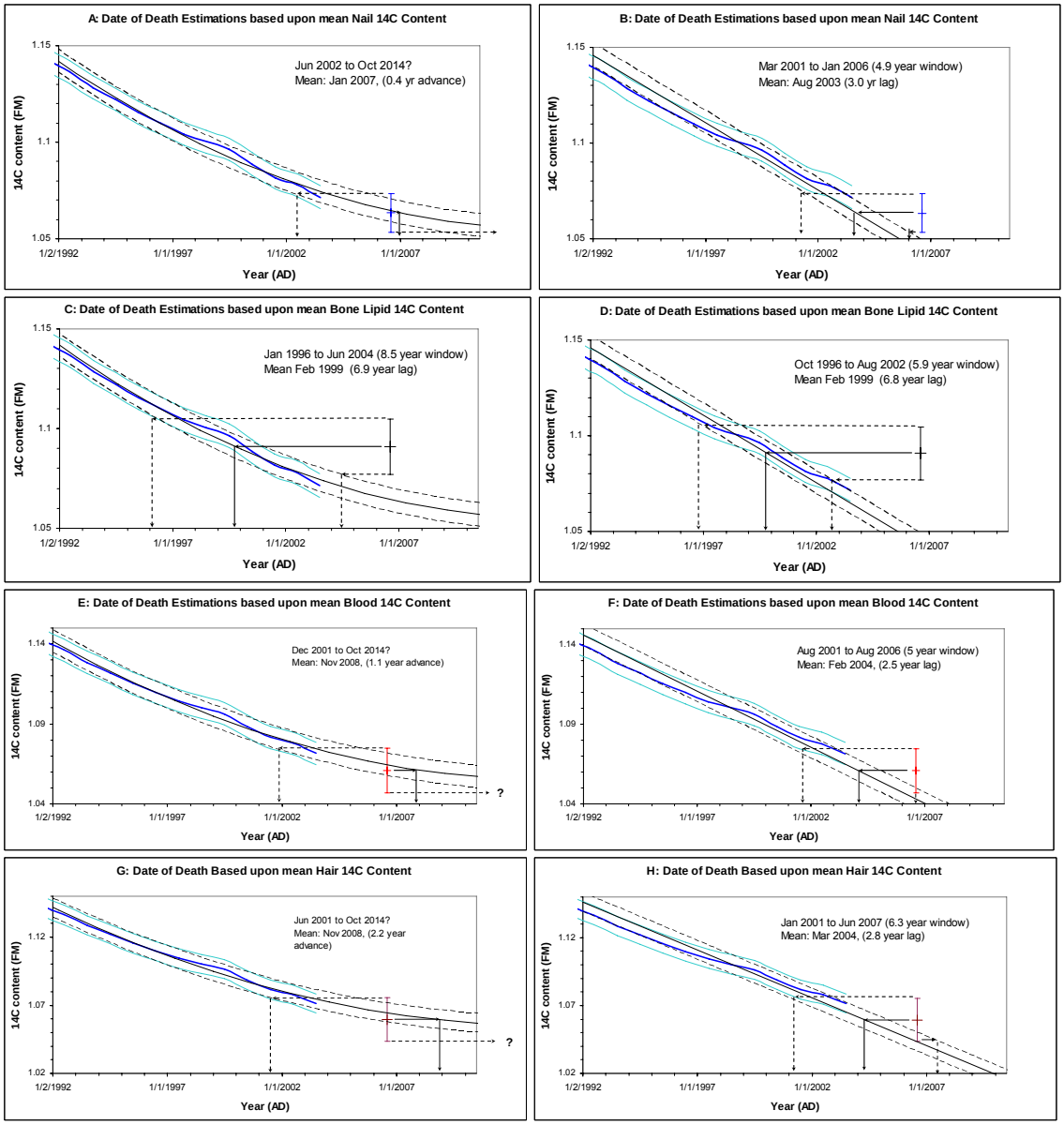

Figure 13 A-H: **Determining the lag time of atmosphere-to-tissue radiocarbon transfer** and the uncertainty of Year-of-Death estimations based upon tissue radiocarbon content. Estimates based on the radiocarbon content of fingernail (A and B), bone lipid (C and D), blood, (E and F), and hair (G and H) are shown plotted against either 2nd order polynomial (left column) or linear (right column) extrapolations of known atmospheric radiocarbon levels. A mean death year of 2006.6 was used throughout this study (see Table 1). The mean and 2 σ uncertainty of each tissue measurement was plotted against the mean and extrapolated ± 1 σ atmospheric radiocarbon values, and the X intercepts determined for each. The X-intercept of the mean values were taken as the atmosphere-to-tissue time lag, and range between the X-intercepts of the minus 2 σ and the plus 2 σ tissue measurements with the plus 1 σ and the minus 1 σ atmospheric radiocarbon levels defined the precision of that estimate.

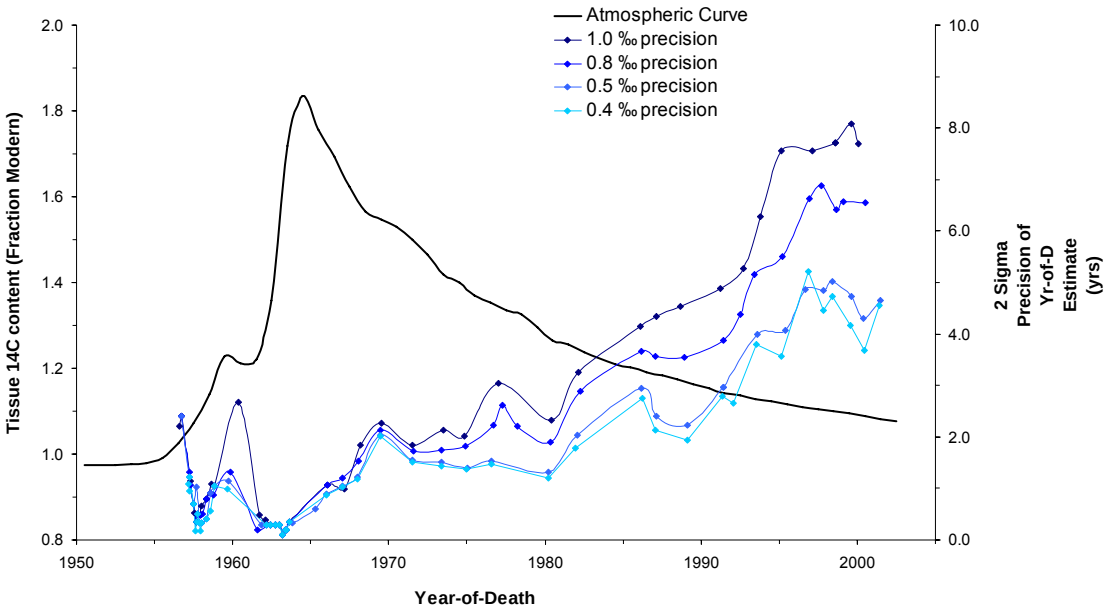

Figure 14: **Precision of Year-of-Death Estimation** as a function of Year-of-Death Measurement. Calibration was carried out on mock series of tissue measurements made with 0.4 to 1.0‰ precision. The calibration data set was the Levin and Kromer (2004) Northern Hemisphere Atmospheric Radiocarbon curve smoothed semi-annually.

IX. Tables

Table 1: Donor Population Statistics; Donor Year-of-Birth, Year-of-Death, Sex, and Race Data, and Mean Date-of-Death for Sample Population

Number	ID #[1]	Sex	Ancestry	Date of Birth	Date of Death	Age (Years)
1	65-06	M	WHITE	06/23/75	09/02/06	31.2
2	33-06	M	HISPANIC	08/10/72	02/20/06	33.6
3	32-06	F	WHITE	06/03/66	04/22/06	39.9
4	74-06	F	WHITE	05/26/64	09/17/06	42.3
5	63-06	F	WHITE	10/31/62	08/26/06	43.8
6	25-06	F	WHITE	01/17/62	04/07/06	44.2
7	69-06	F	WHITE	04/17/61	08/20/06	45.4
8	87-06	M	WHITE	01/07/60	11/02/06	46.9
9	98-06	M	BLACK	06/18/59	10/20/06	47.4
10	75-06	M	BLACK	10/19/58	09/25/06	48.0
11	92-06	M	WHITE	03/02/58	11/16/06	48.7
12	78-06	F	WHITE	04/27/57	10/10/06	49.5
13	89-06	F	WHITE	05/06/56	11/10/06	50.5
14	93-06	M	BLACK	01/18/56	11/16/06	50.9
15	17-06	F	WHITE	09/13/55	02/25/06	50.5
16	40-06	F	WHITE	05/29/55	06/16/06	51.1
17	95-06	M	WHITE	11/07/52	11/29/06	54.1
18	62-06	F	BLACK	11/24/51	08/08/06	54.7
19	68-06	F	WHITE	10/20/51	09/04/06	54.9
20	80-06	M	WHITE	12/01/50	10/03/06	55.9
21	100-06	F	WHITE	11/16/49	11/19/06	57.0
22	19-06	M	WHITE	04/20/46	01/28/06	59.8
23	101-06	F	WHITE	12/18/45	12/02/06	61.0
24	57-06	F	AM.INDIAN	12/09/45	08/12/06	60.7
25	81-06	F	WHITE	07/26/45	10/20/06	61.3
26	31-06	M	WHITE	04/21/42	03/15/06	63.9
27	61-06	M	WHITE	06/17/37	08/20/06	69.2
28	16-06	M	WHITE	06/26/35	02/26/06	70.7
29	76-06	M	WHITE	02/27/35	10/04/06	71.6
30	79-06	M	WHITE	08/18/33	10/16/06	73.2
31	30-06	F	WHITE	07/02/30	04/21/06	75.9
32	83-06	F	WHITE	06/20/30	10/21/06	76.4
33	18-06	M	WHITE	10/01/24	02/24/06	81.5
34	39-06	F	WHITE	01/02/21	06/08/06	85.5
35	60-06	M	WHITE	05/03/17	08/12/06	89.3
36	94-06	F	WHITE	01/31/13	11/10/06	93.8
				Average DoD (years):	2006.6	
				Std Dev (years):	0.266	

1. Forensic Anthropology Center ID Number

Table 2: Examining Donor Population Growth Phase relative to the Timing of the Atmospheric ^{14}C Spike

Birth years	Number of Donors	Characteristic
1913 to 1937	10	Adult stature achieved before bomb-spike
1942 to 1946	5	Adult stature achieved during rising portion of bomb spike
1947 to 1964	18	Growth occurring during both rising and falling portion of bomb spike
1966 to 1975	3	Growth to adult stature achieved during falling portion of the bomb spike

Table 3: Database of Tissue Radiocarbon Measurements (See below) The Table Columns are as follows: AA, the Arizona Accelerator Mass Spectrometry Laboratory Measurement Number; Lab Numb, the Sample Identification Number and Laboratory Book Reference Number; Person ID, the Forensic Anthropology Center Donor ID Number; Birth Date (mm/dd/yyyy); Age (years); Death Date (mm/dd/yyyy); Sex; Tissue Type; d13C, the tissue $\delta^{13}C$ measurement express in per mil (‰) notation, with a one sigma uncertainty of ± 0.1‰; F(^{14}C), the tissue radiocarbon measurement expressed as fraction modern; ± is the one sigma uncertainty expressed in Fraction Modern; Notes.

Table 4: Years of Age for Complete Tooth Crown Formation (Stage 6). Data from Nolla 1960.

	1st Incisor	2nd Incisor	Cuspid	1st Bicuspid	2nd Bicuspid	1st Molar	2nd Molar	3rd Molar
Boys Mandible	3.2	3.8	5.4	6.3	7.2	3.4	7.6	14.2
Boys Maxilla	4.1	5.0	5.8	6.8	7.6	4.2	7.6	13.6
Girls Mandible	3.1	3.6	5.0	5.6	6.8	3.4	6.6	12.7
Girls Maxilla	4.0	4.7	5.2	6.1	6.6	3.8	6.8	12.1

Table 5: Variation in Tissue Radiocarbon Measurements from Donor Population. Compare these values to the variation in the published atmospheric measurements (Levin and Kromer 2004), calculated to be ±0.007 FM.

	Blood, n=33	Hair, n=32	Nails, n=35	Bone Lipid, n=31
Mean (FM):	1.0607	1.0595	1.0634	1.0909
Sd (± FM):	0.0069	0.0081	0.0051	0.0080

Tissue: *Blood*

AA	Lab Numb	Person ID	Birth Date	Age	Death Date	Sex	Tissue	d13C	F (^{14}C)	±	Note
AA70803	X4837A	16-06B	6/26/1935	70	2/26/2006	M	blood	-19.3	1.0658	0.0045	
AA70805	X4839A	17-06B	9/13/1955	50	2/25/2006	F	blood	-18.9	1.0707	0.0040	
AA70804	X4838A	18-06B	10/1/1924	81	2/24/2006	M	blood	-19.5	1.0719	0.0045	
		19-06B	4/20/1946	59	1/28/2006	M	blood				no sample
AA70806	X4840A	25-06B	1/17/1962	44	2/26/2006	F	blood	-20.3	1.0664	0.0040	
AA70810	X4844A	30-06B	7/2/1930	75	4/21/2006	F	blood	-19.7	1.0691	0.0040	
AA70802	X4836A	31-06B	4/21/1942	63	3/15/2006	M	blood	-19.1	1.0639	0.0095	
AA70808	X4842A	32-06B	6/3/1966	39	4/22/2006	F	blood	-16.9	1.0675	0.0040	
AA73585	X6386	33-06B	8/10/1972	33	2/20/2006	M					no sample
AA70809	X4843A	39-06B	1/2/1921	85	6/8/2006	F	blood	-19.6	1.0670	0.0046	
AA70807	X4841A	40-06B	5/29/1955	51	6/16/2006	F	blood	-18.9	1.051	0.0044	
AA73601	X6402A	57-06B	12/9/1945	60	8/12/2006	F	Blood	-18.5	1.0662	0.0042	
AA73610	X6411A	60-06B	5/3/1917	89	8/12/2006	M	Blood	-18.2	1.0596	0.0042	
AA73619	X6420A	61-06B	6/17/1937	69	8/20/2006	M	Blood	-21.2	1.0691	0.0042	
AA73624	X6425A	62-06B	11/24/1951	54	8/8/2006	F	Blood	-19.2	1.0569	0.0038	
AA73633	X6434B	63-06B	10/31/1962	43	8/26/2006	F	Blood	-17.4	1.0547	0.0038	
		65-06B	6/23/1975	31	9/2/2006	M					no sample
AA73650	X6451A	68-06B	10/20/1951	54	9/4/2006	F	Blood	-21.5	1.0529	0.0037	
AA73659	X6460A	69-06B	4/17/1961	45	8/20/2006	F	Blood	-21.5	1.0635	0.0041	
AA73665	X6466A	74-06B	5/26/1964	42	9/17/2006	F	Blood	-17.7	1.0605	0.0041	
AA73676	X6477A	75-06B	10/19/1958	47	9/25/2006	M	Blood	-18.2	1.0565	0.0037	
AA73685	X6486A	76-06B	2/27/1935	71	10/4/2006	M	Blood	-19.1	1.0617	0.0041	
AA73694	X6495A	78-06B	4/27/1957	49	10/10/2006	F	Blood	-19.1	1.053	0.0046	
AA73703	X6504A	79-06B	8/18/1933	73	10/16/2006	M	Blood	-19.1	1.0584	0.0041	
AA73712	X6513A	80-06B	12/1/1950	55	10/3/2006	M	Blood	-18	1.0657	0.0041	
AA73721	X6522	81-06B	7/26/1945	61	10/20/2006	F	Blood	-19.8	1.0614	0.0042	
AA73726	X6527A	83-06B	6/20/1930	76	10/21/2006	F	Blood	-19	1.053	0.0041	
AA73735	X6536	87-06B	1/7/1960	55	11/2/2006	M	Blood	-19.6	1.0622	0.0042	
AA73744	X6545A	89-06B	5/6/1956	50	11/10/2006	F	Blood	-19	1.0608	0.0041	
AA73753	X6554	92-06B	3/2/1958	48	11/16/2006	M	Blood	-19.1	1.0534	0.0041	
AA73762	X6563	93-06B	1/18/1956	50	11/16/2006	M	Blood	-18.1	1.0549	0.0042	
AA73771	X6572A	94-06B	1/31/1913	93	11/10/2006	F	Blood	-21.8	1.0385	0.0043	
AA73780	X6581	95-06B	11/7/1952	54	11/29/2006	M	Blood	-18	1.0648	0.0042	
AA73789	X6590A	98-06B	6/18/1959	47	10/20/2006	M	Blood	-18.5	1.0585	0.0042	
AA73798	X6599A	100-06B	11/16/1949	57	11/19/2006	F	Blood	-18.7	1.0611	0.0042	
AA73807	X6608A	101-06B	12/18/1945	60	12/2/2006	F	Blood	-18.7	1.0624	0.0037	

Tissue: *Nails*

AA	Lab Numb	Person ID	Birth Date	Age	Death Date	Sex	Tissue	d13C	F (^{14}C)	±	Note
AA70828	X4862A	16-06N	6/26/1935	70	2/26/2006	M	Nails	-18.1	1.0631	0.0045	
		17-06N	9/13/1955	50	2/25/2006	F	Nails				no sample
AA70826	X4860A	18-06N	10/1/1924	81	2/24/2006	M	Nails	-18.4	1.0706	0.0045	
		19-06N	4/20/1946	59	1/28/2006	M	Nails				no sample
AA70825	X4859A	25-06N	1/17/1962	44	2/26/2006	F	Nails	-18.5	1.067	0.0045	
AA73566	X6367A	30-06N	7/2/1930	75	4/21/2006	F	Nails	-20	1.0695	0.0038	
AA73573	X6374	31-06N	4/21/1942	63	3/15/2006	M	Nails	-18.3	1.0682	0.0043	
AA73580	X6381	32-06N	6/3/1966	39	4/22/2006	F	Nails	-16.7	1.0665	0.0049	
AA73589	X6390	33-06N	8/10/1972	33	2/20/2006	M	Nails	-20.2	1.0528	0.0043	
AA73596	X6397	39-06N	1/2/1921	85	6/8/2006	F	Nails	-19.6	1.0603	0.004	
		40-06N	5/29/1955	51	6/16/2006	F					no sample
AA73605	X6406A	57-06N	12/9/1945	60	8/12/2006	F	Nails	-18.6	1.0643	0.0041	
AA73614	X6415A	60-06N	5/3/1917	89	8/12/2006	M	Nails	-18.8	1.072	0.0042	
AA73623	X6424A	61-06N	6/17/1937	69	8/20/2006	M	Nails	-19.4	1.0634	0.0041	
AA73628	X6429A	62-06N	11/24/1951	54	8/8/2006	F	Nails	-18.6	1.0577	0.0042	
AA73637	X6438A	63-06N	10/31/1962	43	8/26/2006	F	Nails	-17.5	1.0558	0.0042	
AA73645	X6446A	65-06N	6/23/1975	31	9/2/2006	M	Nails	-20.7	1.0599	0.0042	
AA73654	X6455A	68-06N	10/20/1951	54	9/4/2006	F	Nails	-20.3	1.0559	0.0042	
AA73663	X6464	69-06N	4/17/1961	45	8/20/2006	F	Nails	-19.6	1.0681	0.0041	
AA73669	X6470	74-06N	5/26/1964	42	9/17/2006	F	Nails	-16.9	1.0628	0.0041	
AA73680	X6481	75-06N	10/19/1958	47	9/25/2006	M	Nails	-19.7	1.0605	0.0041	
AA73689	X6490	76-06N	2/27/1935	71	10/4/2006	M	Nails	-17.8	1.0613	0.0041	
AA73698	X6499A	78-06N	4/27/1957	49	10/10/2006	F	Nails	-18.9	1.0551	0.0042	
AA73707	X6508A	79-06N	8/18/1933	73	10/16/2006	M	Nails	-19.2	1.06	0.0041	
AA73716	X6517A	80-06N	12/1/1950	55	10/3/2006	M	Nails	-17.6	1.0704	0.0041	
AA73725	X6526	81-06N	7/26/1945	61	10/20/2006	F	Nails	-20	1.0669	0.0047	
AA73730	X6531A	83-06N	6/20/1930	76	10/21/2006	F	Nails	-18.4	1.0643	0.0041	
AA73739	X6540	87-06N	1/7/1960	55	11/2/2006	M	Nails	-18.3	1.0638	0.0041	
AA73748	X6549	89-06N	5/6/1956	50	11/10/2006	F	Nails	-17.8	1.0614	0.0047	
AA73757	X6558	92-06N	3/2/1958	48	11/16/2006	M	Nails	-17.5	1.0643	0.0041	
AA73766	X6567A	93-06N	1/18/1956	50	11/16/2006	M	Nails	-17.6	1.056	0.0045	
AA73775	X6576	94-06N	1/31/1913	93	11/10/2006	F	Nails	-20.2	1.0648	0.0045	
AA73784	X6585	95-06N	11/7/1952	54	11/29/2006	M	Nails	-16.7	1.0606	0.0042	
AA73793	X6594A	98-06N	6/18/1959	47	10/20/2006	M	Nails	-17.5	1.0653	0.0045	
AA73802	X6603A	100-06N	11/16/1949	57	11/19/2006	F	Nails	-18.5	1.0696	0.0041	
AA73811	X6612	101-06N	12/18/1945	60	12/2/2006	F	Nails	-20.2	1.0681	0.0044	

Tissue: *Hair*

AA	Lab Numb	Person ID	Birth Date	Age	Death Date	Sex	Tissue	d13C	F (^{14}C)	±	Note
AA70819	X4853A	16-06H	6/26/1935	70	2/26/2006	M	hair	-17.7	1.0616	0.0051	
AA70813	X4847A	17-06H	9/13/1955	50	2/25/2006	F	hair	-17.4	1.0689	0.0044	
AA70815	X4849A	18-06H	10/1/1924	81	2/24/2006	M	hair	-17.2	1.0524	0.0088	
		19-06H	4/20/1946	59	1/28/2006	M					no sample
AA70811	X4845A	25-06H	1/17/1962	44	4/7/2006	F	hair	-17.7	1.0230	0.0130	
AA70820*	X4854A	25-06H*	1/17/1962	44	2/26/2006	F	hair	-16.2	1.0398	0.0044	Soxhlet
AA70816	X4850B	30-06H	7/2/1930	75	4/21/2006	F	hair	-17.3	1.0435	0.0087	
AA70812	X4846A	31-06H	4/21/1942	63	3/15/2006	M	hair	-17.0	1.0535	0.0057	
AA70818	X4852	32-06H	6/3/1966	39	4/22/2006	F	hair	-14.5	1.0620	0.0044	
AA73586	X6387	33-06H	8/10/1972	33	2/20/2006	M	Hair	-18.9	1.0554	0.0042	
AA70817	X4851A	39-06H	1/2/1921	85	6/8/2006	F	hair	-18.4	1.0583	0.0044	
AA70814	X4848A	40-06H	5/29/1955	51	6/16/2006	F	hair	-14.4	1.0522	0.0044	
AA73602	X6403	57-06H	12/9/1945	60	8/12/2006	F	Hair	-17.1	1.0614	0.0042	
AA73611	X6412	60-06H	5/3/1917	89	8/12/2006	M	Hair	-17.9	1.0659	0.0042	
AA73611	X6412RA	60-06H	5/3/1917	89	8/12/2006	M	Hair	-17.6	1.0631	0.0042	Soxhlet
AA73620	X6421	61-06H	6/17/1937	69	8/20/2006	M	Hair	-17.6	1.0624	0.0042	
AA73625	X6426A	62-06H	11/24/1951	54	8/8/2006	F	Hair	-19	1.0608	0.0042	
AA73634	X6435	63-06H	10/31/1962	43	8/26/2006	F	Hair	-16.1	1.0628	0.0042	
AA73642	X6443	65-06H	6/23/1975	31	9/2/2006	M	Hair	-20.6	1.0604	0.0042	
AA73642	X6443RA	65-06H	6/23/1975	31	9/2/2006	M	Hair	-19.5	1.0646	0.0042	Soxhlet
AA73651	X6452	68-06H	10/20/1951	54	9/4/2006	F	Hair	-19.7	1.0524	0.0043	
AA73651	X6452R	68-06H	10/20/1951	54	9/4/2006	F	Hair	-19.3	1.0557	0.0042	Soxhlet
AA73660	X6461	69-06H	4/17/1961	45	8/20/2006	F	Hair	-15.7	1.061	0.0042	
AA73666	X6467	74-06H	5/26/1964	42	9/17/2006	F	Hair	-16.9	1.0574	0.0041	
AA73677	X6478	75-06H	10/19/1958	47	9/25/2006	M	Hair	-18.9	1.0585	0.0084	
AA73686	X6487	76-06H	2/27/1935	71	10/4/2006	M	Hair	-17.6	1.0583	0.0043	
AA73695	X6496	78-06H	4/27/1957	49	10/10/2006	F	Hair	-17.7	1.0405	0.0041	
AA73704	X6505	79-06H	8/18/1933	73	10/16/2006	M	Hair	-16.9	1.0605	0.0042	
AA73713	X6514	80-06H	12/1/1950	55	10/3/2006	M	Hair	-16.4	1.0609	0.0041	
AA73722	X6523	81-06H	7/26/1945	61	10/20/2006	F	Hair	-16.7	1.0603	0.0042	
AA73727	X6528	83-06H	6/20/1930	76	10/21/2006	F	Hair	-17.6	1.0651	0.0041	
AA73736	X6537A	87-06H	1/7/1960	55	11/2/2006	M	Hair	-16.5	1.0603	0.0043	
AA73745	X6546	89-06H	5/6/1956	50	11/10/2006	F	Hair	-15.4	1.0691	0.0041	
AA73754	X6555	92-06H	3/2/1958	48	11/16/2006	M	Hair	-16.5	1.0656	0.0042	
AA73763	X6564A	93-06H	1/18/1956	50	11/16/2006	M	Hair	-16.6	1.0719	0.0048	
AA73772	X6573A	94-06H	1/31/1913	93	11/10/2006	F	Hair	-19	1.0599	0.0043	
AA73781	X6582	95-06H	11/7/1952	54	11/29/2006	M	Hair	-15.3	1.0667	0.0042	
AA73790	X6591A	98-06H	6/18/1959	47	10/20/2006	M	Hair	-16.5	1.0648	0.0042	
AA73799	X6600A	100-06H	11/16/1949	57	11/19/2006	F	Hair	-16.8	1.066	0.0043	
AA73808	X6609	101-06H	12/18/1945	60	12/2/2006	F	Hair	-16.8	1.0666	0.0042	

Tissue: *Skin Collagen*

AA	Lab Numb	Person ID	Birth Date	Age	Death Date	Sex	Tissue	d13C	F (^{14}C)	±	Note
		16-06SC	6/26/1935	70	2/26/2006	M					no sample
		17-06SC	9/13/1955	50	2/25/2006	F					no sample
		18-06SC	10/1/1924	81	2/24/2006	M					no sample
		19-06SC	4/20/1946	59	1/28/2006	M					no sample
AA73560	X6361	25-06SC	1/17/1962	44	2/26/2006	F	Skin Collagen				no sample
AA73565	X6366A	30-06SC	7/2/1930	75	4/21/2006	F	Skin Collagen	-18.9	1.1687	0.0053	
AA73572	X6373	31-06SC	4/21/1942	63	3/15/2006	M	Skin Collagen	-18.1	1.1227	0.0052	
AA73579	X6380A	32-06SC	6/3/1966	39	4/22/2006	F	Skin Collagen	-15.7	1.1192	0.0052	
AA73588	X6389	33-06SC	8/10/1972	33	2/20/2006	M	Skin Collagen				no sample
AA73595	X6396A	39-06SC	1/2/1921	85	6/8/2006	F	Skin Collagen	-17.3	1.1377	0.0052	
		40-06SC	5/29/1955	51	6/16/2006	F	Skin Collagen				no sample
AA73604	X6405	57-06SC	12/9/1945	60	8/12/2006	F	Skin Collagen				no sample
AA73613	X6414A	60-06SC	5/3/1917	89	8/12/2006	M	Skin Collagen	-17	1.1752	0.0054	
AA73622	X6423A	61-06SC	6/17/1937	69	8/20/2006	M	Skin Collagen	-16.7	1.1213	0.0052	
AA73627	X6428	62-06SC	11/24/1951	54	8/8/2006	F	Skin Collagen	-16.7	1.1954	0.0055	
AA73636	X6437A	63-06SC	10/31/1962	43	8/26/2006	F	Skin Collagen	-16.2	1.1228	0.0053	
AA73644	X6445A	65-06SC	6/23/1975	31	9/2/2006	M	Skin Collagen	-17.4	1.1062	0.0051	
AA73653	X6454A	68-06SC	10/20/1951	54	9/4/2006	F	Skin Collagen	-17.3	1.2312	0.0055	
AA73662	X6463A	69-06SC	4/17/1961	45	8/20/2006	F	Skin Collagen	-16.1	1.1374	0.0052	
AA73668	X6469A	74-06SC	5/26/1964	42	9/17/2006	F	Skin Collagen	-16	1.1273	0.0052	
AA73679	X6480A	75-06SC	10/19/1958	47	9/25/2006	M	Skin Collagen	-15.2	1.1158	0.0053	
AA73688	X6489A	76-06SC	2/27/1935	71	10/4/2006	M	Skin Collagen	-17.4	1.1411	0.0054	
AA73697	X6498A	78-06SC	4/27/1957	49	10/10/2006	F	Skin Collagen	-17.5	1.107	0.0053	
AA73706	X6507A	79-06SC	8/18/1933	73	10/16/2006	M	Skin Collagen	-16.9	1.1736	0.0054	
AA73715	X6516A	80-06SC	12/1/1950	55	10/3/2006	M	Skin Collagen	-16.1	1.1465	0.0045	
AA73724	X6525R	81-06SC	7/26/1945	61	10/20/2006	F	Skin Collagen	-16.6	1.1477	0.0045	
AA73729	X6530	83-06SC	6/20/1930	76	10/21/2006	F	Skin Collagen	-17.5	1.0773	0.0052	
AA73738	X6539A	87-06SC	1/7/1960	55	11/2/2006	M	Skin Collagen	-16.8	1.1106	0.0053	
AA73747	X6548A	89-06SC	5/6/1956	50	11/10/2006	F	Skin Collagen	-17.1	1.1561	0.0056	
AA73756	X6557A	92-06SC	3/2/1958	48	11/16/2006	M	Skin Collagen	-16.6	1.1502	0.0053	
AA73765	X6566	93-06SC	1/18/1956	50	11/16/2006	M	Skin Collagen	-17.4	1.194	0.0056	
AA73774	X6575A	94-06SC	1/31/1913	93	11/10/2006	F	Skin Collagen	-17.4	1.1353	0.0054	
AA73783	X6584A	95-06SC	11/7/1952	54	11/29/2006	M	Skin Collagen	-16.5	1.1117	0.0053	
AA73792	X6593	98-06SC	6/18/1959	47	10/20/2006	M	Skin Collagen	-16.3	1.1579	0.0053	
AA73801	X6602A	100-06SC	11/16/1949	57	11/19/2006	F	Skin Collagen	-15.7	1.1479	0.0053	
AA73810	X6611A	101-06SC	12/18/1945	60	12/2/2006	F	Skin Collagen	-17.1	1.1258	0.0054	

Tissue: *Skin Lipid*

AA	Lab Numb	Person ID	Birth Date	Age	Death Date	Sex	Tissue	d13C	F (^{14}C)	±	Note
		16-06SL	6/26/1935	70	2/26/2006	M					no sample
		17-06SL	9/13/1955	50	2/25/2006	F					no sample
		18-06SL	10/1/1924	81	2/24/2006	M					no sample
		19-06SL	4/20/1946	59	1/28/2006	M					no sample
AA73559	X6360	25-06SL	1/17/1962	44	2/26/2006	F	Skin Lipid				no sample
AA73564	X6365A	30-06SL	7/2/1930	75	4/21/2006	F	Skin Lipid	-24.0			no sample
AA73571	X6372A	31-06SL	4/21/1942	63	3/15/2006	M	Skin Lipid	-23.7	1.0806	0.0063	
AA73578	X6379A	32-06SL	6/3/1966	39	4/22/2006	F	Skin Lipid	-22.9	1.0826	0.0054	
AA73587	X6388	33-06SL	8/10/1972	33	2/20/2006	M	Skin Lipid				no sample
AA73594	X6395	39-06SL	1/2/1921	85	6/8/2006	F	Skin Lipid	-23.8	1.0511	0.0043	
		40-06SL	5/29/1955	51	6/16/2006	F	Skin Lipid				no sample
AA73603	X6404	57-06SL	12/9/1945	60	8/12/2006	F	Skin Lipid				no sample
AA73612	X6413A	60-06SL	5/3/1917	89	8/12/2006	M	Skin Lipid	-24.2	1.0772	0.0042	
AA73621	X6422A	61-06SL	6/17/1937	69	8/20/2006	M	Skin Lipid	-20.7	1.0754	0.0042	
AA73626	X6427A	62-06SL	11/24/1951	54	8/8/2006	F	Skin Lipid	-23.6	1.0718	0.0042	
AA73635	X6436A	63-06SL	10/31/1962	43	8/26/2006	F	Skin Lipid	-23.5	1.065	0.0042	
AA73643	X6444A	65-06SL	6/23/1975	31	9/2/2006	M	Skin Lipid	-25.3	1.0727	0.0042	
AA73652	X6453A	68-06SL	10/20/1951	54	9/4/2006	F	Skin Lipid	-25.7	1.0678	0.0042	
AA73661	X6462A	69-06SL	4/17/1961	45	8/20/2006	F	Skin Lipid	-23.1	1.0761	0.0042	
AA73667	X6468A	74-06SL	5/26/1964	42	9/17/2006	F	Skin Lipid	-23.3	1.0798	0.0043	
AA73678	X6479A	75-06SL	10/19/1958	47	9/25/2006	M	Skin Lipid	-21.5	1.0787	0.0042	
AA73687	X6488A	76-06SL	2/27/1935	71	10/4/2006	M	Skin Lipid	-23.9	1.0648	0.0042	
AA73696	X6497A	78-06SL	4/27/1957	49	10/10/2006	F	Skin Lipid	-24.7	1.0715	0.0042	
AA73705	X6506A	79-06SL	8/18/1933	73	10/16/2006	M	Skin Lipid	-24.3	1.0789	0.0043	
AA73714	X6515A	80-06SL	12/1/1950	55	10/3/2006	M	Skin Lipid	-23.5	1.0804	0.0043	
AA73723	X6524A	81-06SL	7/26/1945	61	10/20/2006	F	Skin Lipid	-23.8	1.0744	0.0043	
AA73728	X6529A	83-06SL	6/20/1930	76	10/21/2006	F	Skin Lipid	-24.7	1.0617	0.0042	
AA73737	X6538A	87-06SL	1/7/1960	55	11/2/2006	M	Skin Lipid	-24.2	1.0727	0.0042	
AA73746	X6547A	89-06SL	5/6/1956	50	11/10/2006	F	Skin Lipid	-22.0	1.0788	0.0042	
AA73755	X6556A	92-06SL	3/2/1958	48	11/16/2006	M	Skin Lipid	-23.8	1.0646	0.0042	
AA73764	X6565A	93-06SL	1/18/1956	50	11/16/2006	M	Skin Lipid	-23.6	1.0785	0.0042	
AA73773	X6574A	94-06SL	1/31/1913	93	11/10/2006	F	Skin Lipid	-24.8	1.055	0.0041	
AA73782	X6583A	95-06SL	11/7/1952	54	11/29/2006	M	Skin Lipid	-23.0	1.0813	0.0042	
AA73791	X6592A	98-06SL	6/18/1959	47	10/20/2006	M	Skin Lipid	-21.0	1.0698	0.0042	
AA73800	X6601A	100-06SL	11/16/1949	57	11/19/2006	F	Skin Lipid	-23.6	1.0765	0.0043	
AA73809	X6610A	101-06SL	12/18/1945	60	12/2/2006	F	Skin Lipid	-25.0	1.0745	0.0042	

Tissue: *Bone Lipid*

AA	Lab Numb	Person ID	Birth Date	Age	Death Date	Sex	Tissue	d13C	F (^{14}C)	±	Note
AA70829	X4863	16-06BL	6/26/1935	70	2/26/2006	M	Bone Lipid	-24	1.0821	0.0044	
AA70833	X4867	17-06BL	9/13/1955	50	2/25/2006	F	Bone Lipid	-24.6	1.084	0.0043	
AA70836	X4870A	18-06BL	10/1/1924	81	2/24/2006	M	Bone Lipid	-24.4	1.1067	0.0045	
AA73555	X6356A	19-06BL	4/20/1946	59	1/28/2006	M	Bone Lipid	-23.5	1.0956	0.0056	
AA73561	X6362A	25-06BL	1/17/1962	44	2/26/2006	F	Bone Lipid	-24.7	1.0839	0.0054	
AA73568	X6369A	30-06BL	7/2/1930	75	4/21/2006	F	Bone Lipid	-24.9	1.1067	0.0054	
AA73575	X6376A	31-06BL	4/21/1942	63	3/15/2006	M	Bone Lipid	-24.2	1.0998	0.0051	
AA73582	X6383A	32-06BL	6/3/1966	39	4/22/2006	F	Bone Lipid	-23.7	1.0971	0.0056	
AA73591	X6392A	33-06BL	8/10/1972	33	2/20/2006	M	Bone Lipid	-23.8	1.0897	0.0055	
AA73598	X6399A	39-06BL	1/2/1921	85	6/8/2006	F	Bone Lipid	-25.0	1.0891	0.0066	
		40-06BL	5/29/1955	51	6/16/2006	F	Bone Lipid				no sample
AA73607	X6408A	57-06BL	12/9/1945	60	8/12/2006	F	Bone Lipid	-24.4	1.0853	0.0052	
AA73616	X6417A	60-06BL	5/3/1917	89	8/12/2006	M	Bone Lipid	-24.4	1.0979	0.0043	
		61-06BL	6/17/1937	69	8/20/2006	M	Bone Lipid				no sample
AA73630	X6431A	62-06BL	11/24/1951	54	8/8/2006	F	Bone Lipid	-23.7	1.0858	0.0043	
AA73639	X6440A	63-06BL	10/31/1962	43	8/26/2006	F	Bone Lipid	-24.0	1.0854	0.0043	
AA73647	X6448	65-06BL	6/23/1975	31	9/2/2006	M	Bone Lipid	M			no sample
AA73656	X6457A	68-06BL	10/20/1951	54	9/4/2006	F	Bone Lipid	-25.1	1.0927	0.0043	
		69-06BL	4/17/1961	45	8/20/2006	F	Bone Lipid				no sample
AA73671	X6472A	74-06BL	5/26/1964	42	9/17/2006	F	Bone Lipid	-24.6	1.0943	0.0043	
AA73682	X6483A	75-06BL	10/19/1958	47	9/25/2006	M	Bone Lipid	-20.7	1.0919	0.0043	
AA73691	X6492A	76-06BL	2/27/1935	71	10/4/2006	M	Bone Lipid	-24.3	1.0939	0.0043	
AA73700	X6501A	78-06BL	4/27/1957	49	10/10/2006	F	Bone Lipid	-24.6	1.0892	0.0043	
AA73709	X6510A	79-06BL	8/18/1933	73	10/16/2006	M	Bone Lipid	-24.4	1.0997	0.0043	
AA73718	X6519A	80-06BL	12/1/1950	55	10/3/2006	M	Bone Lipid	-24.1	1.0836	0.0043	
		81-06BL	7/26/1945	61	10/20/2006	F	Bone Lipid				no sample
AA73732	X6533A	83-06BL	6/20/1930	76	10/21/2006	F	Bone Lipid	-24.3	1.0798	0.0042	
AA73741	X6542A	87-06BL	1/7/1960	55	11/2/2006	M	Bone Lipid	-24.5	1.0848	0.0043	
AA73750	X6551A	89-06BL	5/6/1956	50	11/10/2006	F	Bone Lipid	-22.6	1.0871	0.0043	
AA73759	X6560A	92-06BL	3/2/1958	48	11/16/2006	M	Bone Lipid	-24.0	1.0834	0.0042	
AA73768	X6569A	93-06BL	1/18/1956	50	11/16/2006	M	Bone Lipid	-23.2	1.0903	0.0043	
AA73777	X6578A	94-06BL	1/31/1913	93	11/10/2006	F	Bone Lipid	-24.9	1.1078	0.0043	
AA73786	X6587A	95-06BL	11/7/1952	54	11/29/2006	M	Bone Lipid	-21.9	1.0904	0.0043	
AA73795	X6596A	98-06BL	6/18/1959	47	10/20/2006	M	Bone Lipid	-20.9	1.0741	0.0042	
AA73804	X6605A	100-06BL	11/16/1949	57	11/19/2006	F	Bone Lipid	-23.4	1.0932	0.0043	
AA73813	X6614	101-06BL	12/18/1945	60	12/2/2006	F	Bone Lipid	-24.8	1.0911	0.0043	

Tissue: *Bone Apatite*

AA	Lab Numb	Person ID	Birth Date	Age	Death Date	Sex	Tissue	d13C	F (^{14}C)	±	Note
AA70831	X4865	16-06BA	6/26/1935	70	2/26/2006	M	Bone Apatite	-10	1.1103	0.0072	
AA70835	X4869	17-06BA	9/13/1955	50	2/25/2006	F	Bone Apatite	-10.6	1.2566	0.005	
AA73554	X6355	18-06BA	10/1/1924	81	2/24/2006	M	Bone Apatite	-10.5	1.0794	0.0043	
AA73557	X6358	19-06BA	4/20/1946	59	1/28/2006	M	Bone Apatite	-9.9	1.1905	0.0046	
AA73563	X6364	25-06BA	1/17/1962	44	2/26/2006	F	Bone Apatite	-9.8	1.1766	0.0046	
AA73570	X6371	30-06BA	7/2/1930	75	4/21/2006	F	Bone Apatite	-11.1	1.222	0.0047	
AA73577	X6378	31-06BA	4/21/1942	63	3/15/2006	M	Bone Apatite	-9.8	1.1126	0.0051	
AA73584	X6385	32-06BA	6/3/1966	39	4/22/2006	F	Bone Apatite	-8.9	1.1544	0.0045	
AA73593	X6394	33-06BA	8/10/1972	33	2/20/2006	M	Bone Apatite	-8	1.0733	0.0052	
AA73600	X6401	39-06BA	1/2/1921	85	6/8/2006	F	Bone Apatite	-10.8	1.1118	0.0053	
		40-06BA	5/29/1955	51	6/16/2006	F	Bone Apatite				no sample
AA73609	X6410	57-06BA	12/9/1945	60	8/12/2006	F	Bone Apatite	-10.2	1.1175	0.0053	
AA73618	X6419	60-06BA	5/3/1917	89	8/12/2006	M	Bone Apatite	-11.1	1.0618	0.0051	
		61-06BC	6/17/1937	69	8/20/2006	M	Bone Apatite				no sample
AA73632	X6433	62-06BA	11/24/1951	54	8/8/2006	F	Bone Apatite	-9.7	1.1727	0.0054	
AA73641	X6442	63-06BA	10/31/1962	43	8/26/2006	F	Bone Apatite	-9.1	1.2099	0.0055	
AA73649	X6450	65-06BA	6/23/1975	31	9/2/2006	M	Bone Apatite	-9.4	1.1097	0.0053	
AA73658	X6459	68-06BA	10/20/1951	54	9/4/2006	F	Bone Apatite	-10.5	1.2323	0.0056	
		69-06BA	4/17/1961	45	8/20/2006	F	Bone Apatite				no sample
AA73673	X6474	74-06BA	5/26/1964	42	9/17/2006	F	Bone Apatite	-8.5	1.1991	0.0055	
AA73684	X6485	75-06BA	10/19/1958	47	9/25/2006	M	Bone Apatite	-9	1.2532	0.0057	
AA73693	X6494	76-06BA	2/27/1935	71	10/4/2006	M	Bone Apatite	-10.8	1.1119	0.0053	
AA73702	X6503	78-06BA	4/27/1957	49	10/10/2006	F	Bone Apatite	-10.1	1.2055	0.0055	
AA73711	X6512	79-06BA	8/18/1933	73	10/16/2006	M	Bone Apatite	-10.7	1.1193	0.0054	
AA73720	X6521	80-06BA	12/1/1950	55	10/3/2006	M	Bone Apatite	-10.1	1.2714	0.0057	
		81-06BC	7/26/1945	61	10/20/2006	F	Bone Apatite				no sample
AA73734	X6535	83-06BA	6/20/1930	76	10/21/2006	F	Bone Apatite	-11.5	1.1258	0.0053	
AA73743	X6544	87-06BA	1/7/1960	55	11/2/2006	M	Bone Apatite	-11.1	1.1896	0.0055	
AA73752	X6553	89-06BA	5/6/1956	50	11/10/2006	F	Bone Apatite	-9.9	1.3019	0.0058	
AA73761	X6562	92-06BA	3/2/1958	48	11/16/2006	M	Bone Apatite	-9.9	1.2029	0.0055	
AA73770	X6571	93-06BA	1/18/1956	50	11/16/2006	M	Bone Apatite	-10.3	1.3283	0.0058	
AA73779	X6580	94-06BA	1/31/1913	93	11/10/2006	F	Bone Apatite	-11.1	1.1196	0.0053	
AA73788	X6589	95-06BA	11/7/1952	54	11/29/2006	M	Bone Apatite	-9.3	1.3051	0.0058	
AA73797	X6598	98-06BA	6/18/1959	47	10/20/2006	M	Bone Apatite	-8.9	1.1544	0.0052	
AA73806	X6607	100-06BA	11/16/1949	57	11/19/2006	F	Bone Apatite	-9.8	1.1957	0.0072	
AA73815	X6627	101-06BA	12/18/1945	60	12/2/2006	F	Bone Apatite	-10.9	1.1304	0.0054	

Tissue: *Bone Collagen*

AA	Lab Numb	Person ID	Birth Date	Age	Death Date	Sex	Tissue	d13C	F (^{14}C)	±	d15N	Note
AA70830	X4864A	16-06BC	6/26/1935	70	2/26/2006	M	Bone Collagen	-15.9	1.148	0.0046	10.8	
AA70834	X4868A	17-06BC	9/13/1955	50	2/25/2006	F	Bone Collagen	-16.4	1.3743	0.006	11	
AA70837	X4871A	18-06BC	10/1/1924	81	2/24/2006	M	Bone Collagen	-16.1	1.0712	0.0044	12.2	
AA73556	X6357A	19-06BC	4/20/1946	59	1/28/2006	M	Bone Collagen	-16.2	1.215	0.0054	10.6	
AA73562	X6363A	25-06BC	1/17/1962	44	2/26/2006	F	Bone Collagen	-16.1	1.2711	0.0057	11.7	
AA73569	X6370A	30-06BC	7/2/1930	75	4/21/2006	F	Bone Collagen	-16.6	1.3114	0.0058	11.7	
AA73576	X6377A	31-06BC	4/21/1942	63	3/15/2006	M	Bone Collagen	-16.5	1.1564	0.0054	11.1	
AA73583	X6384A	32-06BC	6/3/1966	39	4/22/2006	F	Bone Collagen	-15.8	1.2259	0.0055	11.2	
AA73592	X6393A	33-06BC	8/10/1972	33	2/20/2006	M	Bone Collagen	-13.3	1.1422	0.0053	9.5	
AA73599	X6400A	39-06BC	1/2/1921	85	6/8/2006	F	Bone Collagen	-16.1	1.1799	0.0054	11.2	
		40-06BC	5/29/1955	51	6/16/2006	F	Bone Collagen					no sample
AA73608	X6409A	57-06BC	12/9/1945	60	8/12/2006	F	Bone Collagen	-17.1	1.1629	0.0053	11.3	
AA73617	X6418A	60-06BC	5/3/1917	89	8/12/2006	M	Bone Collagen	-17	1.0589	0.0054	12.1	
		61-06BC	6/17/1937	69	8/20/2006	M						no sample
AA73631	X6432	62-06BC	11/24/1951	54	8/8/2006	F	Bone Collagen	-15.9	1.2279	0.0056	11.3	
AA73640	X6441	63-06BC	10/31/1962	43	8/26/2006	F	Bone Collagen	-14.9	1.3131	0.0057	10.9	
AA73648	X6449A	65-06BC	6/23/1975	31	9/2/2006	M	Bone Collagen	-15.2	1.1453	0.0053	11.1	
AA73657	X6458	68-06BC	10/20/1951	54	9/4/2006	F	Bone Collagen	-15.7	1.2941	0.0057	11.8	
		69-06BC	4/17/1961	45	8/20/2006	F						no sample
AA73672	X6473A	74-06BC	5/26/1964	42	9/17/2006	F	Bone Collagen	-15.2	1.3092	0.005	10.9	
AA73683	X6484A	75-06BC	10/19/1958	47	9/25/2006	M	Bone Collagen	-15.2	1.3141	0.0057	11.1	
AA73692	X6493A	76-06BC	2/27/1935	71	10/4/2006	M	Bone Collagen	-16.6	1.1951	0.0046	11.3	
AA73701	X6502A	78-06BC	4/27/1957	49	10/10/2006	F	Bone Collagen	-16.5	1.2998	0.0058	10.5	
AA73710	X6511	79-06BC	8/18/1933	73	10/16/2006	M	Bone Collagen	-16.2	1.1366	0.0053	12.3	
AA73719	X6520A	80-06BC	12/1/1950	55	10/3/2006	M	Bone Collagen	-15.6	1.3225	0.0056	11.7	
		81-06BC	7/26/1945	61	10/20/2006	F	Bone Collagen					no sample
AA73733	X6534A	83-06BC	6/20/1930	76	10/21/2006	F	Bone Collagen	-16.6	1.1835	0.0055	10.8	
AA73742	X6543A	87-06BC	1/7/1960	55	11/2/2006	M	Bone Collagen	-17.2	1.3251	0.0059	10.8	
AA73751	X6552	89-06BC	5/6/1956	50	11/10/2006	F	Bone Collagen	-16	1.4199	0.0061	10.7	
AA73760	X6561	92-06BC	3/2/1958	48	11/16/2006	M	Bone Collagen	-16.1	1.3346	0.0059	11.4	
AA73769	X6570A	93-06BC	1/18/1956	50	11/16/2006	M	Bone Collagen	-15.1	1.5126	0.0056	11.1	
AA73778	X6579	94-06BC	1/31/1913	93	11/10/2006	F	Bone Collagen	-16.8	1.1445	0.0053	11.2	
AA73787	X6588A	95-06BC	11/7/1952	54	11/29/2006	M	Bone Collagen	-15.5	1.4331	0.0061	10.9	
AA73796	X6597A	98-06BC	6/18/1959	47	10/20/2006	M	Bone Collagen	-15.2	1.2351	0.0056	11.2	
AA73805	X6606A	100-06BC	11/16/1949	57	11/19/2006	F	Bone Collagen	-15.3	1.2086	0.0055	11.0	
AA73814	X6615	101-06BC	12/18/1945	60	12/2/2006	F	Bone Collagen	-15.5	1.2073	0.0048	11.9	

Tissue: *Teeth*

AA	Lab Numb	Person ID	Birth Date	Age	Death Date	Sex	Tooth ID	d13C	F (^{14}C)	±	Note
		16-06E	6/26/1935	70	2/26/2006	M					no sample
AA70832	X4866	17-06E	9/13/1955	50	2/25/2006	F	7R Max lat incisor	-9.7	1.05	0.0041	
AA70822	X4856	18-06E	10/1/1924	81	2/24/2006	M	2nd R mand molar	-9.3	0.881	0.011	
AA70821	X4855	19-06E	4/20/1946	59	1/28/2006	M	3rd R max molar	-7.3	0.9344	0.0049	
AA70823	X4857	25-06E	1/17/1962	44	2/26/2006	F	2nd L Max molar	-8.3	1.5932	0.0067	
AA73567	X6368	30-06E	7/2/1930	75	4/21/2006	F	24L	-9.4	0.9466	0.005	
AA73574	X6375	31-06E	4/21/1942	63	3/15/2006	M	24L	-7.7	0.9613	0.0089	
AA73581	X6382	32-06E	6/3/1966	39	4/22/2006	F	6 R Max Canine	-6.9	1.4648	0.0054	
AA73590	X6391	33-06E	8/10/1972	33	2/20/2006	M	12L 1st Max Premolar	-2.7	1.2737	0.0048	
AA73597	X6398	39-06E	1/2/1921	85	6/8/2006	F	5R 1st Max Premolar	-8.5	0.9627	0.0038	
		40-06E	5/29/1955	51	6/16/2006	F					no sample
AA73606	X6407	57-06E	12/9/1945	60	8/12/2006	F	2R 2nd Max Premolar	-9.5	0.9454	0.0037	
AA73615	X6416	60-06E	5/3/1917	89	8/12/2006	M	21 L mand 1st premolar	-8.4	0.9714	0.0039	
		61-06E	6/17/1937	69	8/20/2006	M					no sample
AA73629	X6430	62-06E	11/24/1951	54	8/8/2006	F	26R mand lat incisor	-8.7	0.9468	0.0039	
AA73638	X6439	63-06E	10/31/1962	43	8/26/2006	F	2nd R mand molar	-7.3	1.5663	0.0064	
AA73646	X6447	65-06E	6/23/1975	31	9/2/2006	M	5R 1st Max Premolar	-7.9	1.1899	0.0046	
AA73655	X6456	68-06E	10/20/1951	54	9/4/2006	F	2R 2nd Max molar	-8.1	0.9886	0.004	
AA73664	X6465	69-06E	4/17/1961	45	8/20/2006	F	21L 1st mand premolar	-7	1.4844	0.0055	
AA73670	X6471	74-06E	5/26/1964	42	9/17/2006	F	5R 1st Max Premolar	-7.6	1.5436	0.0064	
AA73681	X6482	75-06E	10/19/1958	47	9/25/2006	M	28R mand 1st premolar	-7.2	1.6628	0.0061	
AA73690	X6491	76-06E	2/27/1935	71	10/4/2006	M	10L max lat incisor	-10.2	0.96	0.0048	
AA73699	X6500	78-06E	4/27/1957	49	10/10/2006	F	22L mand canine	-8.8	1.0639	0.0042	
AA73708	X6509	79-06E	8/18/1933	73	10/16/2006	M	4R max 2nd premolar	-8.2	0.965	0.0047	
AA73717	X6518	80-06E	12/1/1950	55	10/3/2006	M	L mand incisor				
		81-06E	7/26/1945	61	10/20/2006	F					no sample
AA73731	X6532	83-06E	6/20/1930	76	10/21/2006	F					
AA73740	X6541	87-06E	1/7/1960	55	11/2/2006	M	26R mand lat incisor	-9.9	1.3138	0.0061	
AA73749	X6550	89-06E	5/6/1956	50	11/10/2006	F	11L max canine or 12L	-8.7	1.1027	0.0043	
AA73758	X6559	92-06E	3/2/1958	48	11/16/2006	M	24L mand central incisor	-9.5	1.1249	0.0044	
AA73767	X6568	93-06E	1/18/1956	50	11/16/2006	M	5R 1st Max Premolar	-7.7	1.0825	0.0053	
AA73776	X6577	94-06E	1/31/1913	93	11/10/2006	F	25R or 5R	-9.4	0.9883	0.0095	
AA73785	X6586	95-06E	11/7/1952	54	11/29/2006	M	27R mand canine	-8.7	0.9712	0.0054	
AA73794	X6595	98-06E	6/18/1959	47	10/20/2006	M	13L max 2nd molar	-7.1	1.4167	0.0083	
AA73803	X6604	100-06E	11/16/1949	57	11/19/2006	F	27R mand canine	-8.5	0.9485	0.0047	
AA73812	X6613	101-06E	12/18/1945	60	12/2/2006	F	7R Max lat incisor	-8.2	0.9496	0.0048	