CHAPTER 9
Suspicions
Historical Lead Poisoning

Little boy, box of paints
Licked the brush, joined the saints

We have seen that exposure to lead can cause a wide range of physiological effects, non-beneficial, several acutely hallucinogenic. Were we, as a species, dumb or what? Didn't we know that it was bad? This question particularly comes up with respect to the widespread use of lead in both the Roman and British Empires. Did lead play a role in the course of history in either of these two empires? When did we begin to focus on lead as a public health issue?

These are the questions that we will explore in this chapter.

LEAD AND THE ROMANS

Did lead affect the decline and fall of the Roman empire (Time line J.15)? After about 27 BC Rome, technically a republic, in actuality an oligarchy, began a huge expansion that left a few Romans ruling over large masses of un-Romanized cultures. The officials of the empire owed appointment to personal factors and were responsible directly to the emperor. The western empire had to adsorb non-urban tribes into this system, governed by Roman overlords with excessive scrutiny from the central government. The economy was underdeveloped (mass poverty), with predominance of agricultural labour. Outposts of the empire supplied tax, rent-grain, to Rome. After centuries of expansion the Roman empire imploded. The western legions were unable to stop the tide of the migrating barbarians, even though they had successfully fended off previous Celtic invasions in Europe proper and in Britain itself. In the waning years of the empire soldiers were not only high ranking imports from the barbarian ranks, but poorly trained soldiers from the barbarians. Pay from Rome was often missing. The Roman ruling elite was unable to sustain itself, and the records of family lines of power is sparse. The birthrate of the ruling class plummeted so greatly that the term "aristanasia" (death of the ruling class) has been applied to the Romans.

The reasons for it's fall have intrigued historians for years. There are several historically accepted reasons for the fall of the empire. Among these are the rise and impact of Christianity, economic stagnation, demographic decline with manpower shortages, over taxation, an inability to form alliances within the ruling class, over-exploitation of the masses and natural resources (Brunt 1988; Garnsey and Saller 1987). The one agreed upon fact is that weaknesses within and pressure from without combined to end the Empire.

What role would lead have played, if any, in the internal weakness of the system? The case against lead is as follows. 1) The Romans had a very high production of lead. 2) The Roman use of lead was high. 3) The Roman food intake of lead, particularly among the affluent, was astronomical, and could lead to clinical poisoning. 4) Written accounts of the aristocracy suggests erratic behavior consistent with lead poisoning, contributing to poor decision making. 5) The aristocracy died off unnaturally and had a very low birth rate, consistent with lead poisoning. Adoption was a common method of getting an heir. 6) The sparsely analyzed bone record appears to support the view of elevated lead.

High Production of Lead

World wide production of lead took an abrupt increase during the years of the Roman empire (Figure

1.3) due to it's association with silver. Galena, PbS, forms a solid solution at geologically obtainable high temperatures with silver, in particular: argentite (Ag_2S), miagyrite ($AgSbS_2$), matildite (α-$AgBiS_2$) and aramaysite ($Ag(Bi,Sb)S_2$). This fact was early recognized and lead was thought of as being "married" to silver (PbO = silver stone).

High Use of Lead

The second reason for such a high level of lead production during Roman times is that technologies involving lead were extensively used. Pure lead was used in plumbing, lead cooking vessels, bronzes were made with high lead content, and leaded glaze technology had not yet disappeared from the Mediterranean world. In Chapter 4 we noted that cooking beans in a lead glazed pot could result in 22 Fg Pb/ g beans. So if one were eating two meals of beans a day five days a week the day consumption rate would be 54 g/day (Gersberg and others 1997). Also in that chapter it was noted that there was a uniform recipe of lead glaze throughout the empire which contained 50-80% PbO.

High Intake of Lead

The intake of lead by Romans would have been large from all of these sources. The intake of lead would be particularly high among the aristocracy.
For sweetness the Romans boiled grape juice down to "sapa" in a lead cooking vessel. The probable chemical formula of sapa is $Pb(CH_3CO_2)_2$, lead acetate, a compound which has a sugary taste.

Why did the Romans not use honey or run out to the store and purchase sugar?

Sweetners: Honey

Although honey was well known and cultivated in antiquity it was a luxury of the rich (Toussaint-Samat 1993, p. 17). Egyptians were the first to practice domestication of bees, as noted from Theban wall paintings from 700 B.C., showing pottery, or baked mud, hives (Gould and Gould 1988, p. 5). The cylindrical pots were stacked horizontally. The technology was inherited or transferred to the Greek world, with horizontal stacking of true terracotta pots (Crane 1983, p. 46). The use of the pots allowed some ability to move the hives about.

Romans advanced beekeeping technology by utilizing both pots, and tree or upright hives, but also by using woven baskets, with Columella, suggesting that the terracotta pots were too cold in the winter and too hot in the summer. Horizontal log hives may have been introduced by the Romans (Crane 1983, p. 56). Columella was a Spanish Roman, and favored the horizontal hives over the more northern upright hives. In 180 B.C. a tax on Corsica required 200,000 pounds of beeswax (100,000 hives worth) (Gould and Gould 1988, p. 10).

Roman legions from Tunisia apparently kept bees in Aude as a hobby with only the consuls eating the honey. Although Spanish honey, derived from the thyme plant was well regarded, Greek honey was the preference of the Romans, particularly of Mount Hymettus, and was part of the luxury items imported into Rome. Honey was used in Roman cooking, as it shows up in Apicius' fish sauces.

Beekeeping was of a sufficiently technical knowledge that most of the beekeeping died out with the collapse of the Roman Empire and wild collecting was again practiced. The art requires knowledge of the fields and placement of the hives, proper construction of the hives, and appropriate access to the hive to collect the honey, as well as the ability to collect a hive of bees to inhabit the hive. After the fall of the Roman Empire beekeeping was not well organized until the time period of Charlemagne who placed a honey tax on the population, effectively converting honey into a feudal right of the overlords. St. John's Church in Novgorod required a 400 ton beeswax tribute, which would have required collection from 1,000,000 wild tree hives.

Sweetners: Sugar

Despite its prevalence in the ancient world, honey did not apparently satisfy the full sweet desire of the populous, perhaps because of the lack of control over its production. The sugar itself, once "tamed" could be more reliably produced. Sugar cane plant had been domesticated as early as 9,000 to 6,000 years ago in New Guinea (Diamond 1997). Botanists Artschwager and Brandes suggest that sugar cane left New Guinea in three separate time periods, the first around 8,000 B.C. About 6,000 B.C. sugar cane moved to India (Mintz 1985). References to sugar making appear in the *Mahabhashya of Patanjali*, a commentary on Panini's study of Sanskrit, a grammar book (the first) dating to 400-350 B.C. The invasion of India by the Greeks (327 B.C.) brought the sugar cane to the attention of the Greeks. Nearchus, general of Alexander, asserted that " *a reed in India brings forth honey without the help of bees, from which an intoxicating drink is made though the plant bears no*

fruit"[Deerr, #2161].

Dioscorides writes:

"*There is a kind of concreted honey, called saccharon, found in reeds in Arabia Felix, like in consistence to salt, and brittle to be broken between the teeth, as salt is. It is good for the belly and the stomach being dissolved in water and so drank, helping the pained bladder and the reins."*

The development of sugar technology took some time. The problem was not in the plant domestication, but in the ability to move the plant from subtropical areas, (irrigation) and to bring to it a stable and intensive agricultural labor. The plant is propagated from asexual cuttings of the stem. It becomes ripe from 9 to 18 months. Once chopped, the cane is crushed, chopped, pounded, and soaked in liquid. Heating the liquid brings about supersaturation and crystals of sugar begin to form. The remaining liquid is molasses or treacle.

By 500 A.D. a complete description of the production process appears in Indian literature (*Buddhagosa*, or Discourse on Moral Consciousness). Like the technology of majoilica, the technology of sugar was spurred under the Islamic golden age. Heraclius (Emperor of Constantinople) invaded Baghdad in 627 A.D., where he describes sugar as an Indian luxury. He was defeated by the Arabs in 636 A.D. and sugar was introduced to Spain almost simultaneously with the invasion of Spain in 711 A.D. By 900 A.D. sugar cane was in production in the Mediterranean area, but the climate was not optimal and required major advances in irrigation.

Another major problem was in the labor required. Slavery early on became associated with the production of sugar, particularly during the Crusaders' occupation of Jerusalem (1099-1187) and their control of the sugar industry in the Middle East. It was the Crusaders who brought back sugar to the table of Europe (Mintz 1985, p. 28). Albert van Aachen collected the reminiscences of the veterans of the First Crusade (1099-1187):

In the fields of the plains of Tripoli can be found in abundance a honey reed which they call Zuchra; the people are accustomed to suck enthusiastically on these reeds, delighting themselves with their beneficial juices, and seem unable to sate themselves with this pleasure in spite of their sweetness. The plant is grown, presumably and with great effort, by the inhabitants...It was on this sweet-tasting sugar cane that people sustained themselves during the sieges of Elbarieh, Marrah, and Arkah, when tormented by fearsome hunger.

Initially sugar was considered a medicine and/or spice. Albertus Magnus (1250 A.D.) writes of sugar:

It is by nature moist and warm, as proved by its sweetness and becomes dryer with age. Sugar is soothing and solving, it soothes hoarseness and pains in the breast, causes thirst (but less than honey) and sometimes vomiting, but on the whole it is good for the stomach if it is in good condition and free of bile."

The Knights of Malta were planting cane in Acre when conquered by the Saracens in 1291.

The decline of the Mediterranean sugar industry has been attributed to the Black Death (1350s) which caused a tremendous increase in prices of labor-costly goods like sugar. It was this labor shortage that caused sugar production to be linked to slavery. When sugar was found to be easily produced in the New World, the peculiar link between New World history and slavery was made (Galloway 1977). Sugar refinery in Europe did not take place until the production of sugar beets in 1503.

This brief history of sugar cane production indicates that the production of sapa by the Romans was in response to a lack of other alternatives. The Romans, instead, relied upon honey, and upon various concentrates of wine called sapa, defructum and garum.

Sweetner: Sapa or Lead Acetate

Sapa was produced by boiling down wine. The process of producing various concentrates of wine is described by Pliny, Cato, and Palladius. What follows is Palladius' recipe:

Now about the preparation of defrutum, caroenum, and sapa. Although all three are made from the same substance, namely from must, the method of their preparation modifies both their names and their properties. For defrutum has its name from "boiling down" and it is ready when it is reduced to a thick consistency. Caroenum is ready when it has lost one third of it volume with two-thirds remaining, sapa,

Figure 9.1. Sugar of Lead (Lead acetate) can be obtained by boiling wine in lead vessels. It was used to stabilize wines (prevent oxidation) and to sweeten them.

Table 9.1: Sweetness of Some Compounds Relative to Sucrose

Compound	Relative Sweetness*
Acesulfame K	20,000
Glucose	74
Fructose	173
Lactose	16
Sucrose	100
Maltose	33
P-4000	400,000
Saccharin	50,000
Aspartame	16,000

*Sweetness is relative to sucrose at 100

Table 9.3: Estimated Daily Intake of Lead

source: Nriagu's Table 6.2 p. 401

	µg Pb/day
Pre-technological Societies (NAS, 1980)	2.4
UK (1980)	6-37
Roman Slave	15-77
Tolerable Limit (WHO, 1977)	43
Roman Aristocrat	160-1520
U.S. Adults (NAS, 1980)	30-50

when it has been reduced to one-third. The latter is improved when quinces are cooked with it and fig wood is added to the fire.

All three authors are specific that the best sweetener was made by boiling in a lead container. Pliny writes*: "lead and not copper jars should be used"*. Palladius' also specifies lead containers:

Some people put the must in leaden vessel and by boiling it reduce it by a quarter, and other by a third. There is no doubt that anyone who boiled it down to one-half would be likely to make a better, thick form of must and therefore more profitable for use, so much so that it can actually be used, instead of must boiled down to one-third, to preserve the must produced from old vineyards.

This recipe was reproduced in 1977, and resulted in a paste containing 1000 mg lead/L. A single teaspoon of this material per day could cause chronic lead poisoning, particularly because lead acetate is one of the few soluble lead salts, and therefore, more biologically available (Esisinger 1977).

A number of substances that activate human receptors for sweetness. The original synthesis of aspartamine gave a bitter taste until it was stereochemically resolved. This, in addition, to the variable sweetness of the stereochemically different sugars sucrose and lactose (Table 9.1) suggest that the sweetness receptor site is chiral or stereochemically controlled.

An inspection of all of the sweeteners listed suggests that a common feature is a carbonyl (C=O) functionality alpha (next to) to a CH group, as well as alpha to an NH or OH group. Lead diacetate has two acetate groups and could add water to other binding sites. Because of the lone pair on lead we find stereochemical activity (recall our discussion of lead tetracetate as a catalyst, Chapter 7). Lead acetate has been known for centuries as sugar of lead, and imparts a high degree of sweetness to wines or foods to which it is added (Figure 9.1).

According to Nriagu, the Roman aristocracy must have consumed a large amount of sapa since nearly 80% of the recipes in Apicius' cookbook contain the addition of one of the three types of leaded wine sweetener (Table 9.2). In addition sapa was used as an **adulterant** for wines. When a wine harvest was not very good, lacking in body and sweetness sapa, was added. It had the additional advantage of "preserving" the wine against further oxidation. Columella (12.21) gives us directions in which to add sapa to wine:

Table 9.2.
Pork Stew with Apples
Apicius 168

Minutal Matianum: Adicies in caccabum oleum, liquamen, cocturam, concides porum, coriandrum, esicia minuta. Spatulam porcinamcoctam tessellatim concides cum sua sibi tergilla. Facies ut simul coquantur. Media coctura mala Matiana purgata intrinsecus, concisa tessllatim mittes. Dum coquitur, teres piper, cuminum, coriandrum viridem vel semen, mentam, laseris radicem, suffundes acetum, mel liquamen, defritummodice et ius de suo sibi, aceto modico temperabis. Facies ut ferveat. Cum ferbuerit, tractam confringes et ex ea obligas, piper asparges et inferes.

In a pot put oil, garum, broth, chopped leeks, coriander, and small meat patties. Dice cook pork shoulder with its rind. Cook everything together when this is half-cooked, put in Matian apples that have been cored and cut into pieces. During the cooking, grind pepper, cumin, fresh coriander or coriander seeds, mint and silphium root; pour in vinegar, honey, garum, a bit of sapa , and cooking broth. Mix with a bit of vinegar. Bring to a boil. When it has boiled, break in pieces and thicken, sprinkle with pepper and serve.

Serves 4-6
1 lb pork
3/4 lb ground meat
1Tbs olive oil
1Tbs garum
2 leeks, chopped
1 Tbs minced fresh coriander
1/2 cup stock
1 lb cooking apples, cored and cut

For sauce:
1tsp pepper
1 Tbs total, cumin, coriander, and mint
1 garlic clove pressed for its juice
1 Tbs vinegar
1 tsp garum
1/2 cup sapa
sufficient stock
1 Tbs. Flour

Heat the olive oil in a casserole, then add the garum, leeks, and coriander. Add the diced pork and the meatballs, letting it cook a while to flavor the meat, then moisten with a bit of stock and continue to cook. When the meat is half done, add the apple pieces. Shortly before the cooking time is complete, add the sauce composed of the first eight sauce ingredients listed above, and thicken with flour.

The recipe is attributed to Gaius Matius, a friend of Julius Caeasar.

Must of the sweetest possible flavor will be boiled down to a third of its original volume and when boiled down, as a I have said above, is called defructum. When it is cooled down, it is transferred to vessel and put in store that use may be made of it after a year. But it can also be added to wine nine days after it has cooled; but it is better if it has remained undisturbed for a year. ....

Adulteration of wines was recognized explicitly as medically unsound. The descriptions given match those of lead poisoning. Pliny (5.9) writes:

Wine adulterated with gypsum is injurious to the nerves, induces headaches and fever and is bad for the

Figure 9.2: Thomas Couture's *romans of Decadence*, 1847, engage in an orgy of wine drinking and debauchery: Http://www.bestpriceart.com/painting/?image-couture-couture1.jpg&tc-cgfa

bladder... The addition of sapa and defrutum to wines may cause headaches, intoxication and stomach trouble....from excessive use of such wines arise dangling....paralytic hands.

while Dioscorides (5.11) notes the continuous drinking of doctored wines: *is pernicious, ruins the nerves, and causes melancholia*. Adulteration of wines continued even with the fall of the Empire and was common in "modern" accounts.

The intake of Romans of wine is so high that the aristocracy must have consumed large amounts of lead adulterated wine. Pliny lists 185 brands of available wine. The average consumption per year was estimated to be 1-5 L/day/person. Lucullus used 4 million liters for his triumphal banquet. (Figure 9.2 shows an 18th century version of a decadent Roman banquet). The huge intake of wine resulted in gout, a disease of the liver, which is consistent with symptoms of lead poisoning. Musonium (20-90 A.D.) describes the aristocracy and their gout and colic:

The masters are less strong, less healthy, less able to endure labor than servants; countrymen more strong than those who are bred in the city, those that feed meanly than those who feed daintily; and that, generally, the latter live longer than the former. Nor are there any other persons more troubled with gouts, dropsies, colics, and the like, than those who, condemning simple diet, live upon prepared dainties.

The total amount of lead consumed by the Romans is estimated to be 3 to 25 times greater than modern man's intake (Table 9.3).

Lead Poisoning Symptoms Well Described in Roman Literature

Many of the later emperors had symptoms consistent with lead poisoning, in some cases making them possibly paranoid. Claudius (41-54) had disturbed speech, weak limbs, an ungainly gait, tremors, fits of excessive and inappropriate laughter, and unseemly anger, and he often slobbered (Nriagu 1983). He had many attacks of stomachache that were so fierce that he contemplated suicide.

Caligula was sound neither in body nor mind. As a boy

Figure 9.3. Statue of the Emperor Claudius
http://www.personal.kent.edu/~bkharvey/roman/slclaud.htm

Figure 9.4. Caligula
http://www.vroma.org/~bmcmanus/caligula.html

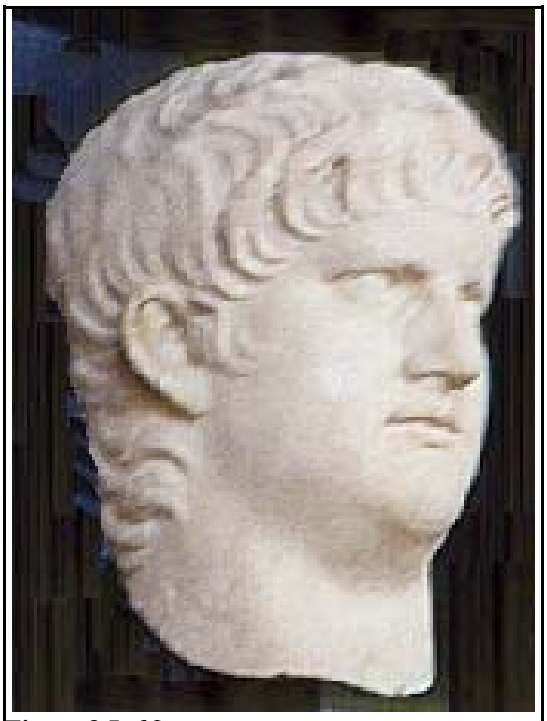


Figure 9.5. Nero
http://www.ga.k12.pa.us/academics/MS/8th/romanhis/Forum/QuinnC/

he was troubled with the falling sickness, and while in his youth he had some endurance, yet at times because of sudden faintness he was hardly able to walk, to stand up, to collect his thoughts, or to hold up his head. He himself realized his mental infirmity, and thought at times of going into retirement and clearing his brain (Nriagu 1983).

Nero, of course, goes down in history as having set Rome on fire for fun and playing a fiddle while watching it burn (Figures 9.5 and 9.6).

Death of the Aristocracy: Infertility

Lead in high amounts can kill fetoes. One account of female workers in a white lead pigment factory from industrial Britain (1914) says that women used the spontaneous abortive effects of lead as a type of "sponge" in which to prevent their own poisoning. They felt that the most harmful effects were passed on

Figure 9.6. John Williams Waterhouse's 1878 *The Remorse of Nero after the Murder of his Mother.*
http://www.artrenewal.org/museum/w/Waterhouse_John_William/page4.html

to the aborted children (Oliver 1914)).

Of 35 married men who lived in the aristocratic quarters in Roman Troy 18 were childless, 10 only had one offspring, and 7 had two or three children (Gilfillan 1965). The written records of wills indicating estates left to freedmen suggests lack of progeny. The rolls of the Senate showed little consistency of family line. Aelius Spartianus bemoans:

Indeed, when I reflect on the matter, it is sufficiently clear that no great man has aleft a son who is excellent and useful, for such men either die without children or for the most part have children of such a kind that it would have been better for the human race if they had died without descendants.... What of Scipio? What of the Catos, who were such great men? What about Caesar? What about Tullius for whom especially it would have been better not to have had children? What about Augustus, who did not even have a good son by adoption, although he had the power of choosing from all men?

An alternative explanation for the custom of adoption is that Augustus tried to reduce social mobility at the top by encouraging senators to marry, bear children, and keep property within the family. Senators were, however, happy with adoption of adult sons. The low life expectancy at birth (~25), the late age of marriage of men (late 20s), and, therefore, the generational age gap (about 40), reduced effects of paternal authority over sons. Few fathers, around 20%, were alive at the time of their son's marriage. This lack of family ties made inheritance of wealth outside the family in patron/client, patron/protege, and friends important.

The Roman Bone Record

A few archeological studies confirm the estimated excessive intake of lead by the aristocracy (Table 9.4 and Figures 9.7 and 9.8). (Recall that lead is easily deposited in bones and leads, being more insoluble than calcium.) The bone lead shows up as two distinct populations in Figure 9.10, the majority of bones with little or no lead and a second population of high bone lead levels. This latter population has been suggested to correspond with the aristocracy.

You may be skeptical at attributing the distribution of bone lead into two separate populations (aristocracy and slave). This same bimodal distribution

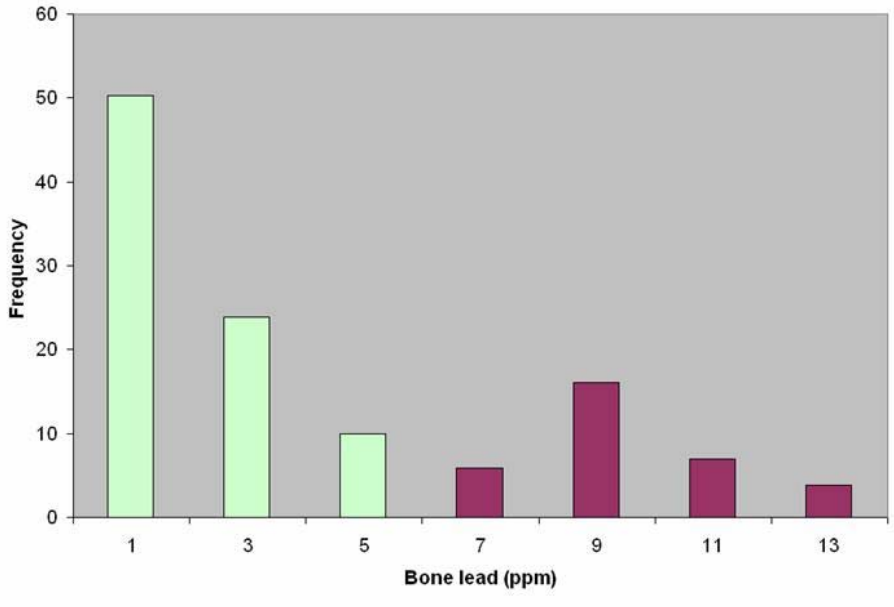


Figure 9.6. Bone lead concentration of a Roman population. Data Source: G. A. Bacchus 1982, Sci. Total Environ. 24:121-131.

North Elham Park	Anglo-Saxon	Rib (5)	55
Barton	9th-12th A.D.	Rib (101)	71
King's Lynn	Medieval	Rib (4)	104

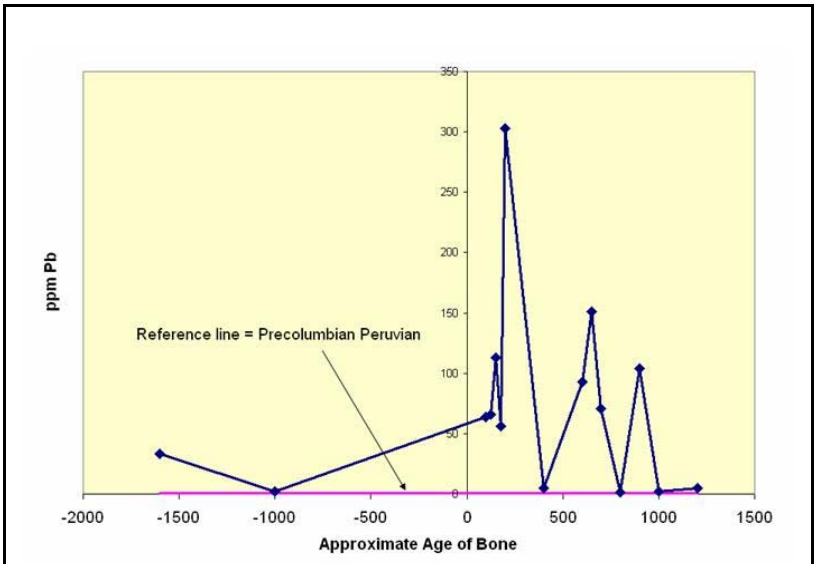


Figure 9.7. Data from Table 9.4 graphed with approximate ages of bones. (Data should be viewed with scepticism unless analytical procedure for each bone is known).

more historically accessible populations: colonial European-American plantation owners (185 ppm bone lead (range 128-258)) and African-American plantation slaves (35 ppm bone lead (range 9-96)). In the colonial American context, lead was a luxury item associated with lead-lined storage containers. Furthermore, the distribution of bones into separate burial populations facilitated easy identification (Aufderheide and others 1985; Aufderheide and others 1981; Corruccini and others 1987; Wittmers and others 2002).

In a more historically accessible time period, bone lead of a sugar (rum) processing plantation in the Caribbean showed a slave population with an average bone lead concentration of 120 ppm. Lead exposure derived from the use of lead stills in the distillation of fermented molasses. The historical literature indicates symptoms of acute lead poisoning: mild abdominal pain, muscle paralysis, convulsions and death (Handler and others 1986).

The total bone lead concentration can be used to estimate the degree of intoxication, making some important assumptions. First we assume that the bone has not been altered by the process of diagenesis which can result in leaching of lead to the soil or by deposition of the lead from the soil (or lead coffin?) to the bone. This may not be such a great assumption. Careful work has been performed with archaeological samples suggesting that not only does lead deposit differentially into the bone in life depending upon bone type, subject age, and has been observed with

subject gender, but post-death changes in the bone may be influenced by whether or not the bone is trabecular (see Chapter 8). For those interested in the chemistry associated with determining the pre-death lead content of archaeological bone please see the end of the chapter.

Archeaological Bone Record for the Romans

We will now try to relate the bone record of the Romans (Table 9.4) to blood lead levels they would have been experiencing. Several recent studies have shown that the bone lead content in live subjects correlates reasonably well with blood lead (linearly) using either the skull bone or the finger bone. These studies mostly relate to the question of post-occupational storage of bone lead and the effect on post-occupational toxicosis. The fundamental assumption is that in a model of skeletal sub-compartments, the lead in bones should be considered readably exchangeable and constitute a source of lead input to the blood (Marcus 1983; Rabinowitz and others 1976; Rosen 1988; Schutz and others 1987b). Figure 9.9 shows the correlation between Tibial lead concentration (μg Pb/g bone mineral) and between the cranial bone lead and blood lead. Both of these show a reasonable linear fit (Erkkila and others 1992) with slopes of 0.535 and 0.667 for blood lead to calcaneus and tibia, respectively with a probability of error of <0.1%. Blood lead concentrations can be related to bone lead concentrations by Equations 8.1 and 8.2.

Reference man (Snyder and others 1984) has a skeletal mass of 10 kg with 4 kg as cortical wet bone mass and 1 kg as trabecular wet bone mass. Ashed weights corresponding to the bone mineral are given as 2200 g and 500 g, respectively. From this we create a conversion of 4 kg wet cortical bone per 2.2 kg dry cortical bone (factor of 1.81) and of 1 kg wet trabecular bone to 0.5 kg dry trabecular bone (factor of 2). Assuming that the Roman rib bones are best described as trabecular, we compute the wet value ppm (303/2 = ~151.5 ppm).

Comparing this value to the correlation slope or directly to the Figure 9.9 or equations 8.1 and 8.2 we estimate that the average Roman aristocrat would have had a blood lead concentration of 1.2 μmole Pb/L or (Fmole x {207 Fg/Fmole} x {0.1 L/dL}) 25 μg Pb/dL. This value on the physiological charts (Figure 8.2) comes out to imply a range of symptoms of fatigue and low erythrocyte production, and the beginning of low of hearing acuity. By comparison, the Peruvian bones would result, using the same assumptions, in 4 μg Pb/dL, which is below the physiologic response levels observed 1997.

To review the bone lead argument we have had to assume that the bones were not contaminated or altered on burial. We further had to assume what type of bone was analyzed in the reported study (cortical vs trabecular). Based on these assumptions we calculated

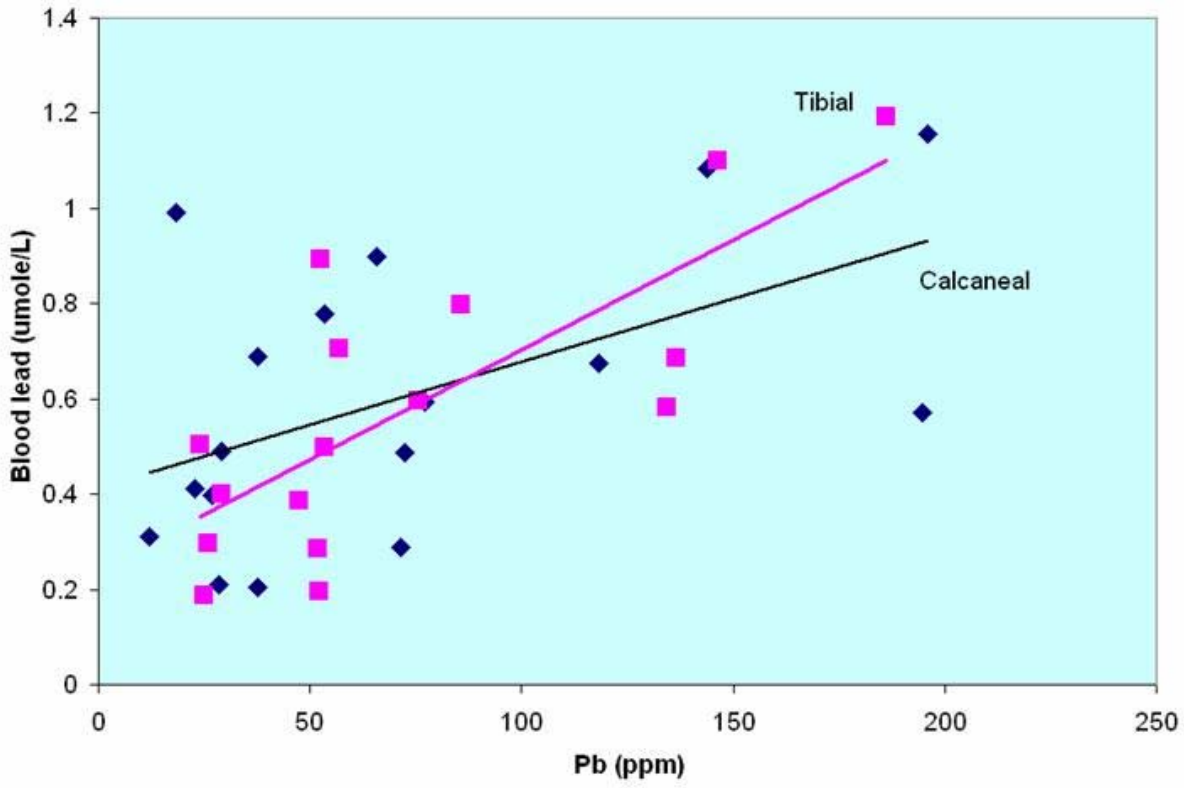


Figure 9.9. Blood lead values change with respect to bone lead. See also equations 8.1 and 8.2. Data source: Erkkila, J., R. Armstrong, V. Riihimkai, D. R. Chettle, A. Paakkari, M. Scott, L. Somervale, *Br. J. Ind. Med.* 1992, 49, 631-644.

a lead/wet of bone, which we related to blood lead, which in turn is related to the toxicity the person would have experienced due **solely** to the bone material as a source of toxicity. If the individuals whose bones were examined were actively depositing lead into the bone material we might expect higher levels of blood lead, leading to even more severe symptoms.

This high level of intake has been suggested to result in a country in which the rulers were all poisoned leading, ultimately, to the downfall of the empire. This claim was first made by Gilfillan (Gilfillan 1965).

In the interests of fairness we should note that modern Italian scientists dispute Nriagu's hypothesis. As they point out, while some of the emperors exhibited clear symptoms of acute clinical lead poisoning, the fall of the Roman empire did not come until several centuries later.

As we noted in the argument about diffusion vs anti-diffusion of metallurgy to China, one can not "prove" to any degree of certainty the relative weight of the causative factors in this type of post-fact "experiment".

EARLY LEAD WARNINGS

The history of lead use immediately raises the question of whether or not people were just plain lazy or stupid to be exposing themselves to so much toxic material. Many people did observe the ill effects of lead, but the combination of poor communications (no books, no paper, illiterate populations, and the lack of understanding of the mechanism of lead toxicity) made most of the public health predictions unattended.

The early history of lead poisoning has been described in Nriagu's text *Lead and Lead Poisoning in Ancient History*. Much of what follows is derived from Nriagu's research.

Nikander, Greek physician from 2nd B.C. (Major 1945) bemoans the use of lead:

The harmful cerussa, that most noxious thing
Which foams like the milk in the earliest spring
With rough force it falls and the pail beneath fills
This fluid astringes and causes grave ills.
The mouth it inflames and makes cold from within
The gums dry and wrinkled, are parch'd like the skin
The rough tongue feels harsher, the neck muscles grip
He soon cannot swallow, foam runs from his lip
A feeble cough tries, it in vain to expel
He belches so much, and his belly does swell
His sluggish eyes sway, then he totters to bed
Complains that so dizzy and heavy his head
Phantastic forms flit now in front of his eyes
While deep from his breast there soon issue sad cries
Meanwhile there comes a stuporous chill
His feeble limbs droop and all motion is still
His strength is now spent and unless one soon aids
The sick man descends to the Stygian shades.

An anonymous Greek verse curses intemperance listing effects that would be related to lead adulterated wines:

Hence gout and stone afflict the human race;
Hence lazy jaundice with her saffron face;
Palsy, with shaking head and tott'ring knees
And bloated dropsy, the staunch sot's disease;
consumption, pale with keen but hollow eye
And sharpened feature, shew'd that death was nigh
The feeble offspring curve their crazy sires,
And, tainted from his birth, the youth expires.

Pliny (born AD 22 and died in the eruption of Vesuvius, 79 AD) (Beagon 1992; French and Greenawy 1986) follows this up with the admonition:

For medicinal purposes lead is melted in earthen vessels, whilst it is being melted the breathing passages should be protected....otherwise the noxious and deadly vapour of the lead furnace is inhaled; it is harmful to dogs with special rapidity (Waldron 1973).

On adulterated wines he notes that '*from the excessive use of such wines arise dangling.. ..paralytic hands'*

Dioscorides (41-68 A.D.) notes that such sapa doctored wines " most hurtful to the nerves"

Vitruvius (100s, B.C.) in his architectural text gives a very clear warning about lead:

Water is much more wholesome from earthernware than from lead pipes. For it seems to be made injurious by lead because cerusse is produced by it; and this is said to be harmful to the human body. Thus if what is produced by anything is injurious, it is not doubtful but that the thing is unwholesome in itself. We may take example by the workers in lead who have complexions affected by pallor. For when, in casting, the lead receives the current of air, the fumes from it occupy the members of the body and rob the limbs of the virtues of the blood. Therefore it seems that water should not be brought in lead pipes if we desire to have it wholesome.

Many other writers of the Roman period note the ill effects of white lead, including Thomas (*Alexipharmaca* lines 75-85), and *Scribonius Largus (Compositiones*, 183-184) who gives as symptoms of white-lead poisoning the whitening of both the tongue and the commissures between the teeth, nausea, vertigo, dimming of sight, and dyspnea. Aetoios (4.1) writes on the effect of swallowing ceruse or litharge:

We can observe heaviness of the stomach and excruciating, twisting pain, similar to volvulus, especially around the navel, there is anxiety and general agitation. The body becomes livid and takes on a leaden hue. Finally, burning pains are felt in the articulations. If the disease takes a turn for the worse, paralysis of the extremities appears as well as delirium and convulsive trembling.

Jumping ahead we find Paul of Aegina in 700 A.D. giving the first description of a lead poisoning **epidemic** which was attributed to Italy (continuing the

custom of adulterating wines?):

I consider moreover a colicky affection, which still becomes violent from a kind of collection of humors, which took its origin from regions in Italy, moreover in many other places in Roman territory whence the like contagion of a pestilential plague. Wherefore in many cases it passed into epilepsy, to some there came loss of motion with sensation unhurt, to many both, and of those who fell victims to the epilepsy, very many died. Of those indeed who were paralyzed, not a few recovered, for the cause which attacked them ended by crisis.

800 years later both Agricola (1526) and Paracelsus (1533) chroniclers of mining and technical arts were describing health of miners. Agricola specifically notes the effect of arsenic and mine gnomes. Paracelsus published a book *On Miners sickness and other miners' diseases.*

In 1616 Francois Citois (the physician to Cardinal Richelieu (the shadow behind the French throne at this time)) published an account of the symptoms of pallor, disturbances of the mind and of vision, insomnia, fainting, stomach pains, loss of appetite, nausea, vomiting, inflames abdomen, convulsions and paralysis. This he called colica Pictonum since it was a disease found in Poitou (a region of tart wines) which he blamed on the supernova of 1572.

Bernardino Ramazzini (1633-1714) wrote *De Morbis artificum diatriba* (Diseases of workers) which quite clearly indicated the effects of lead and the occupations of workers likely to be exposed to lead and therefore to be lead poisoned. He listed workers likely to experience lead poisoning as metalworkers, gilders, potters, and painters. Lead miners suffered from

"*dysnopea, phthisis (asthma), apoplexy (sudden loss of muscular control); paralysis, cachexia (a general wasting of body due to chronic disease), swollen feet, loss of teeth, ulcerated gums, pains in the joints, and palsy." (Ramazzini 1964) "these then are the diseases that afflict men who handle lead: first their hands become palsied, then they become paralytic, splenetic, lethargic, cachetic,.... toothless... cadaverous and the color of lead*.

In 1650 Samuel Stockhausen, a physician in Goslar, in Germany's Hartz Mountains, described an illness "Huttenkatze" thought to be caused by gnomes within the mountains. He noted that only miners and smelter workers exposed to lead dust got the disease.

Vernatti's report to the British Royal Society on workers involved in white lead manufacture was given in 1678.

In 1690 Eberhard Glockel, doctor to the Ulm (Germany) monasteries, tended monks ill with excruciating abdominal pains. He too developed symptoms on drinking the monastery's wines. He deduced from a ducal wine master that sweetened (lead acetate adulterated) wines were the culprit. He published an account of this episode in 1697 entitled:

A Remarkable Account of the Previously Unknown WINE DISEASE
which in 1694, 95, and 96 was caused by sour wine sweetened with litharge, resulting in many cruel symptoms in towns, monasteries, and castles, occasionally also in the countryside, and had the consequence that many persons of high and low rank were severely afflicted, or even lost their lives.

The Duke Eberhard Ludwig of Würtlemberg proclaimed the, apparently, first law banning lead adulterants in 1697. Henderson in his 1824 account of the history of wine (Henderson 1824) discusses the adulteration of wines with sapa and relates:

In the following year, some offenders against this law were punished by banishment or hard labour; and a wine cooper at Eslingen, who afterwards ventured to revive this nefarious trade, and had induced several persons, in various places, to follow it, was condemned to lose his head, while the possessors of the adulterated wines were severely fined, and the wines themselves thrown out.

The practice of adulterating wines continued. Inferior wines, those with high acidity, were the most susceptible to adulteration. Henderson warns the reader to beware of Rhine, Moselle, Cape or Teneriffe.

When these wines have an unusual degree of sweetness, a darker colour than their age and body seem to warrant, and particularly when the use of them is followed by pains of the stomach, we may presume, that they have been adulterated with lead.

He had good reason to warn of adulterated wines as Graham's Treatise on Wine-Making (1770) has several recipe's requiring adulteration of wine with lead:

To hinder wine from turning
Put a pound of melted lead, in fair water, into your cask, pretty warm, and stop it close.

To soften Grey Wine
Put in a little vinegar wherein litharge has been well steeped, and boil some honey, to draw out the wax. Strain it through a cloth, and put a quart of it into a tierce of wine,and this will mend it.

In 1738 John Huxham describes symptoms "*said to infest the county of Devon, amongst the populace especially, and those who were not very elegant and careful in their diet*". This came to be known as Devonshire colic, which occurred every autumn, exacerbated by drinking beer or cider (cider presses.)

In 1753 Lind, while experimenting with citric acid for scurvey treatment noted that glazes treated with citric juice produced lead acetate and warned about the possible effects. In 1754 Dehaen describes the same symptoms as shown in the Pictonum colic which he terms Poitiers colic. In 1757 Tronchin published an essay suggesting that colica Pictonum, also known as **saturnine** colic, was caused by rain water passing over leaden roofs.

This was followed shortly by the work in 1767 of Sir George Baker who reported to the Royal College of Physicians that Devonshire Colic came from cider presses in Devon were lead lined (Meiklejohn 1954). He wrote a pamphlet entitled "*An examination of several means by which the poison of lead may be supposed frequently to gain admittance into the human body, unobserved, and unsuspected*:(McCord 1953):

It seems not improbable, that, if we had an opportunity of making an accurate inquiry, we might see reason to conclude, that the disease, called popularly the dry-belly-ach, which is common as well in the northern colonies of America, as in the islands of the West-Indies, ought to be referred wholly to lead, as its cause.

My suspicions, concerning this subject, have been greatly confirmed by the authority of Dr. Franklyn of Philadelphia. That gentleman informs me, that, at Boston, about forty years ago leaden worms were used for the distillation of rum. In consequence thereof, such violent disorders were complained of by the drinkers of new rum, that the government found it expedient to enact a law, forbidding the use of any worms, except such only as were made of pure block-tin. This law having been enacted, the dry colic was much less frequently heard of than before. But the law was complied with only inpart; for from that time to the present, instead of block-tin, they have used a pewter, containing a large portion of lead. Dr. Franklyn likewise informed me that the colic of Poitou is not so frequent a disease in any of the colonies, as it was formerly; and that the reason, commonly assigned, is that the people now drink their punch very weak in comparison with that what they were formerly accustomed too; which used to be rum and water in equal quantities. He added, that they now also drink their punch, with more juice of fresh limes in it; and, as that juice, joined to certain laxative medicines, is part present their common remedy, when any are seized with the disease, so it is generally considered as the best preservative against it.

I am likewise informed by a gentleman, who resided many years in the Bahama-Islands, that the dry-belly-ach has hardly been known in those islands, since the inhabitants have left off the distillation of rum. The same gentleman informs me, that the people of the Bahama-islands drink very large quantities of small punch, made extremely acid by the juice of limes, many of the labouring people to the amount of at least two gallons every day.

It is hoped, that what has here been thrown out concerning the most probable cause of the colic, which is endemical both in the West-Indies and on the continent of America, may appear to those, who have opportunities of making a more accurate inquiry to be not unworthy of their attention.

In 1775 a medical dissertation from Scotland of Roberston (*The Medical Virtues of Lead, see Chapter 6*) discusses the work of various physicians experimenting on the poisonous effects of intravenous injection of lead into dogs and extensively quotes the work of Dr. Baker.

As Baker mention, Benjamin Franklin (Figure 9.10) was an "expert" on lead poisoning (McCord 1953; McCord 1954). Franklin writes:

To Benjamin Vaughan

Philadelphia July 31, 1786

Dear Friend,

I recollect that when I had the great Pleasure of seeing you at Southampton, now a 12 month since, we had some Conversation on the bad Effects or Lead taken inwardly; and that at your Request I promis'd to send you in writing a particular account of several Facts I there mentioned to you, of which you thought some good use might be made. I now sit down to fulfill that Promise.

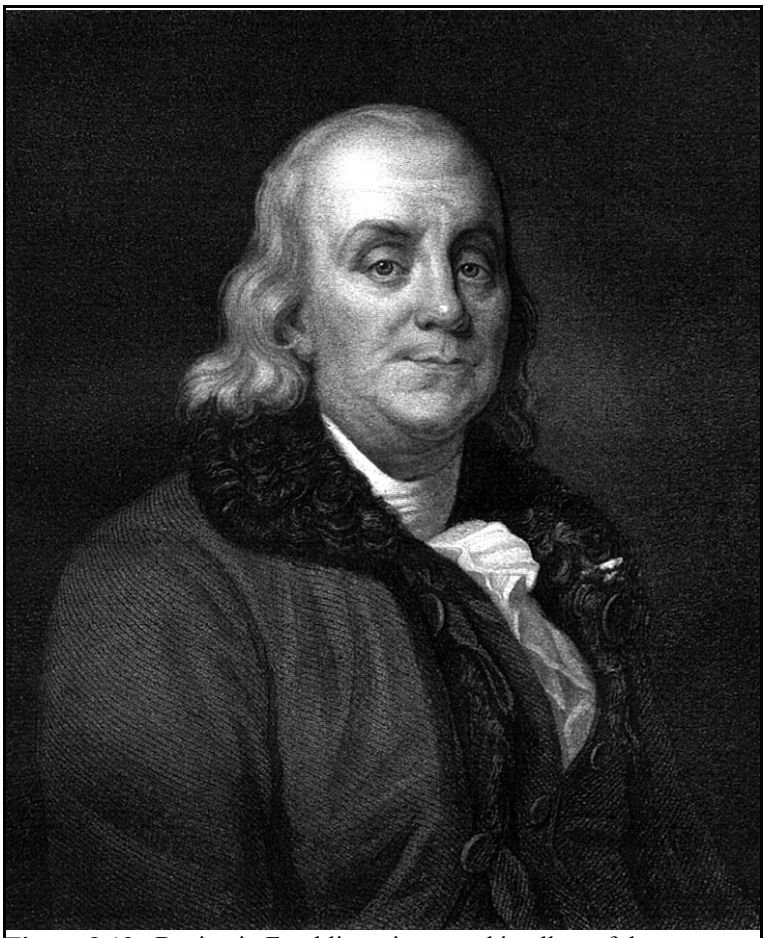


Figure 9.10. Benjamin Franklin, printer, and intellect of the American Revolutionary period noted occupational and public health issues related to lead poisoning. Photo source: http://teachpol.tcnj.edu/amer_pol_hist/thumbnail15.html

The First Thing I remember of this kind was a general Discourse in Boston, when I was a Boy, of a Complaint from north Carolina against new England Rum, that it poisn'd their People, giving them the Dry Bellyach, with Loss of the use of the Limbs. The Distilleries being examin'd on the occasion, it was found that several of them used Leaden Still-heads and Worms, and the Physicians were of the Opinion, that the Mischief was occasioned by the Use of Lead. The Legislature of the massachusetts thereupon pass'd an Act, prohibiting under severe Penalties the Use of such Still-heads and Worms thereafter. Inclos'd I send you a Copy of the Acct. Taken from my printed Law-Book.

In 1724, being in London, I went to work in the printing-House of Mr. Palmer, Bartholomew Close, as a Compositor. I there found a practice, I had never seen before, of drying a Case of Types (which are wet in Distribution) by placing it sloping before the fire. I found this had the additional Advantage, when the Types were not only dry'd but heated, of being comfortable to the Hands working over them in cold weather. I therefore sometimes heated my case when the Types did not want drying. But an old Workman, observing it, advis'd me not to do so, telling me I might lose the use of my Hands by it, as two of our Companions had nearly done, one of whom that us'd to earn his Guinea a Week, could not then make more than ten shillings, and the other, who had the Dangles, but seven and six-pence. This, with a kind of obscure Pain, that I had sometimes felt, as it were in the Bones of my Hand when working over the Types made very hot, induced me to omit the Practice. But talking afterwards with Mr James, a letter-Founder in the same Close, and asking if his People, who work'd over the little furnaces of melted Metal, were not subject to that Disorder, he made light of any danger from the Effluvia, but ascribed it to the particles of the metal swallow'd with their Food by slovenly Workmen, who went to their Meals after handling the Metal, without well washing their fingers, so that some of the metalline Particles were taken off by the Bread and eaten with it. This appeared to have reason in it. But the Pain I had experienc'd made me still afraid of those Effluvia.

....

In America I have often observ'd that on the Roofs of our shingled Houses, where Moss is apt to grow in northern Exposures, if there be anything on the Roof painted with white Lead, such as Balusters, or Frames of dormant Windows, &c., there is constantly a streak on the shingles from such Paint down the Eaves, on which no Moss will grow (Figure 3.32), but the wood remains, constantly clean and free from it. We seldom drink RainWater that falls on our Houses, and if we did perhaps the small quantity of Lead descending from such Paint might, not be sufficient to produce any sensible ill Effects on our Bodies. But I have, been told of a case in Europe, I forgot the Place, where a whole Family was afflicted with what we call Dry Bellyach, or colica Pictonum, by drinking RainWater. It was at a Country-Seat, which being situated too high to have the Advantage of a Well, was supply'd with Water from a Tank, which received the Water from the leaded Roofs. This had been drunk several years without Mischief;

but some young Trees planted near the House growing up above the Roof, and shedding their Leaves upon it, it was suppos'd that an Acid in those leaves had corroded the Lead they cover'd and furnished with Water of that with its baneful Particles and Qualities.

When I was in Paris with Sir John Pringle in 1767, he visited la Charité, a Hospital particularly famous for the cure of that Malady, and brought from thence a Pamphlet containing a List of the Names of Persons, specifying their Professions or Trades, who had been cured there. I had the curiosity to examine that List, and found that all the Patients were of Trades, that, some way or other, use or work in Lead, such as Plumbers, Glaziers, painters, &c., excepting only two kinds, Stonecutters and Soldiers. These I could not reconcile with my Notion, that lead was the cause of that Disorder. But on my mentioning this Difficulty to a Physician of that Hospital, he inform'd me that the Stonecutters are continually using melted Lead to fix the Ends of iron Balustrades in Stones; and that the Soldiers had been employ'd by Painters, as Labourers, in Grinding of Colours.

This, my dear Friend, is all I can at present recollect on the Subject. You will see by it, that the Opinion of this mischievous Effect from lead is at least above Sixty Years old; and you will observe with Concern how long a useful Truth may be known and exist, before it is generally receiv'd and practis'd on.

I am, ever, yours most affectionaly,
B. Franklin

Franklin is the publisher of an essay by Evans Cadwalader called entitled "Essay on the West-India Dry Gripes" (1780) in which the dry gripes (cramping) is traced to rum drinking, where lead is picked up in the distillation process. Cadwalader's description of the Dry Gripes is quite, shall we say, "gripping":

The European Phyficians give an Account of a Difeafe fimilar to the Dry-Gripes, calling it cholica Pictonum, becaufe moft frequent at Poictiers. They are both attended with exceffive griping Pains in the Pit of the Stomach and Bowels, which are much diftened with Wind; violent and frequent Reachings to vomit, fometimes bringing up fmall Quantities of bilious Matter; at other times there is a Senfation, as if the Bowels were drawn together by Ropes; great Coftivenefs, and frequently a continual Inclination to go to Stool without voiding any thing.

The Ducts which open into the Inteftines, and excern a Mucus to moiften and lubricate them, are obftructed and glewed up with a Vifcofity not eafily refolvable; and through the whole courfe of the diftemper, the Faeces are extremely dry and hard, in fmall Lumps like Bullets. The inteftines are drawn up towards the Back with almoft continual convulfive Twitches. The Pains are frequently fo sharp, that the Patient will fall on the Floor, and cry out violently in the greatft Agony.

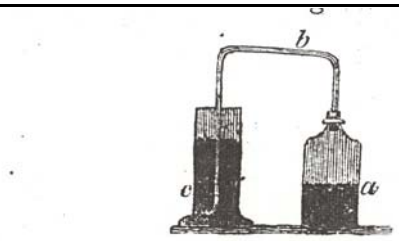
In 1783 the Royal Society offered a prize to those who could devise a means of reducing lead dust in white lead manufacture.

In 1820 F. C. Accum published his "Treatise on the adulteration of food and culinary poisons exhibiting fraudulent sophistications of bread, beer, and wine". He quotes Sir George Baker's account of poisoning by lead pipes:

A gentleman was the father of a numerous offspring, having had one-and-twenty children, of whom eight died young, and thirteen survived their parents. During their infancy, and indeed ***until they had quitted the place of their usual residence, they were all remarkably unhealthy;*** *being particularly subject to disorders of the stomach and bowels. The father during many years, was paralytic; the mother, for a long time, was subject to colics and bilious obstructions. After the death of the parents, the family sold the house which they had so long inhabited. The purchaser found it necessary to repair the pump. This was made of lead; which upon examination was found to be so corroded, that several perforations were observed in the cylinder, in which the bucket plays; and the cistern in the upper part was reduced to the thinness of common brown paper, and was full of holes, like a sieve (Accum 1820).*

Accum indicates that one of the most delicate tests (at that time) was passing sulphuretted hydrogen gas through the water, which gives a brown colloidal suspension (PbS) (Figure 9.11).

Wine was frequently still adulterated with lead. Addition of lead clarifies white wine.



take one part of sulphuret of antimony of commerce, break it into pieces of half the size of split pease, put it into the flask, and pour upon it four parts of common concentrated muriatic acid (spirit of salt of commerce). Sulphuretted hydrogen gas will become disengaged from the materials in abundance, and pass through the water in the vial (*c*). Let the extrication of the gas

Figure 9.11. Image from Acum's text of 1820 for measurement of lead.

Wine merchants persuade themselves that the minute quantity of lead employed for that purpose is perfectly harmless, and that no atom of lead remains in the wine. Chemical analysis proves the contrary.

Acum recites the case of The Gentleman;

A gentleman who had never in his life experienced a day's illness, and who was constantly in the habit of drinking half a bottle of Madeira wine after his dinner, was taken ill, three hours after dinner, with a severe pain in the stomach and violent bowel colic, which gradually yielded within twelve hours to the remedies prescribed by his medical adviser. The day following he drank the remainder of the same bottle which was left the preceding day, and within two hours afterwards he was again seized with the most violent colliquative pains, headach, shiverings, and great pain over the whole body. His apothecary becoming suspicious that the wine he had drunk might be the cause of the disease, ordered the bottle from which the wine had been decanted, to be brought to him, with a view that he might examine the dregs, if any were left. The bottle happening to slip out of the hand of the servant, disclosed a row of shot wedged forcibly into the angular bent-up circumference of it. On examining the beads of shot, they crumbled into dust.

A later text also dealt with the falsifications of food (Mitchell, 1848) (Mitchell 1848).

In 1830-38 Tanquerel des Planches re-analyzed the hospital admission data for La Charité Hospital in Paris. He found that of the 1213 patients admitted from lead colic, 490 were employed in the manufacture of lead compounds, 390 were painters, 61 were potters, 55 were copper and bronze founders, 35 were lapidaries, 25 were refiners, 14 were plumbers, 11 worked in lead shot factories, and 8 were glaziers or worked in glass factories.

In 1840 Burton, physician at St. Thomas's Hospital in London, published a paper entitled on the remarkable effect on human gums produced by the absorption of lead: blue lines, also known as Burton's Lines. The blue lines are caused by a deposit of lead sulphide in the cells of the lining of the mouth, with the sulphur coming from the decay of food along the teeth.

The various world navies were also cognizant of lead poisoning. An epidemic of lead poisoning was found in the French fleet of 1876-7 where vinegar and apple juice (anti-scurvy) were placed in the water supply where the acid accelerated corrosion of the lead pipes (Schadewaldt 2967). We noted in Chapter 7, under the invention of tin cans, that Capt. De Long was sufficiently aware of lead poisoning to suspect it as an agent in his sick bay on the Arctic expedition of the Jeannette in 1891.

Lead and the English: THE FRANKLIN EXPEDITION

We saw that the English, perhaps as a legacy of the Romans, or perhaps due to their own abundance of lead sources, made use of lead widely and pervasively. It was in the plumbing, the machinery, as food adulterants, in the wines, in the glazes, and in the cosmetics. One other peculiar use of the English was the invention of the tin can soldered with lead.

This invention was propelled by a need to combat scurvy to extend the time allowed for exploration by the Navy. The English, following their successful naval battles with Napoleon, found an outlet for the ambitions of the officers, and for national pride, in attempting to find a Northwest Passage, rather like the Russian Sputnik and American space programs of the 1960s.

The Historical Setting

The search for the Northwest Passage begins immediately after Colombus' voyage proving that the earth was round. It did not take long for the English to realize that a trip over the North Pole, if it could be realized, would avoid the Portugeuse dominated route around the tip of Africa, and would shave months and 2,000 miles off the trip to the far east.

The goal was instantaneous riches. "The said islands (Cathay) abound in Gold, Rubies, Diamonds, Bolasses, Granates, jacints, and other stones and pearls" wrote Master Rober Thorne to Sir Edward Lees, ambassador in Seville for King Henry VIII.

The first expedition was under the command of Martin Frobisher (Figure 9.12). Frobisher was an unsavory character who first went to sea at the age of 15. At one point he was held hostage for 9 months in the Castle at El Mina (chief depot of the West African slave trade). After this time he became a pirate preying equally on Protestant and Catholic ships. By 1569 he was arrested for piracy and resurfaces as a patroller for Queen Elizabeth in the Irish channel. He very nearly became involved in a Catholic uprising in Ireland but became enamored of the search for the Northwest Passage. This passage was proven to exist by Sir Humphrey Gilbert (half brother to Sir Walter Raleigh):

All which learned men and painful travellers have affirmed with one consent and voice is that America is an Island; and there that lyeth a great Sea between it, Cathay and Greenland, by which any one of our country that will give the attempt may with small danger pass to Cathaya, the Molucca, India, and all other places in the East in much shorter time than either the Spaniards or Portugals doeth or may do (Thomson 1975; Thorne 1986).

Frobisher set out with the backing of the

Figure 9.12. Martin Frobisher, first of the English Arctic explorers, previously a pirate.The bodlein Library, Oxford. In James P. Delgado, Across the Top of the World.

Muscovy Co in the Gabriel (15-30 tons, Lbs 152, crew 18), the Michael (20-25 tons, lbs 120, crew 17) and a pinnace (7-10 tons, crew 17). The total cost of the expedition was close to $1,500 lbs. They carried 20 compasses, 18 sand glasses, armillary sphere, adjustable sundial, treatise on navigation, a large English bible, a blank chart, and the instruction of Master John Dee, famed astrologer and alchemist, who instructed the officers in the occult mysteries of navigation. We encountered Dee in our discussion of alchemy in the court of the Holy Roman Emperor, Rudolf II, Chapter 6.

Frobisher "discovered' Baffin Island where he met with the Inuit. He captured one Inuit by tempting him with a bell into approaching to arms length. The Inuit was taken back to England for "scientific study" and died shortly on arrival. On his second trip Frobisher returned with a piece of rock from Baffin Island, which after shopping it around to a third "authority" was found to contain a little gold. The third authority, when asked why he was the only one to find gold, replied "Ah Signor, nature needs to be flattered."

The discovery of gold changed the nature of the expedition from one of exploration to a gold rush. A 3rd expedition was outfitted at a cost of 20,000 lbs. Baffin Island was trenched and about 1,000 tons of worthless ore was brought back. A record of the arrival of the gold miners, the trenching and the kidnaping of the Inuit was recorded faithfully in the oral history of the Inuit. Some 300 years later they gave testimony of their encounters with Frobisher similar to the written records of the English.

Further attempts to find the Northwest Passage were carried forward, leading to the trips of Samuel Champlain (St. Lawrence and Great Lakes explorer) and Henry Hudson (1570) who discovered the Hudson Bay, convinced that it was the Northwest Passage. He was unable to prove this because his crew turned mutinous.

At the end of the Napoleonic wars and the decisive victory of the English military, England was poised to again mount searches for the Northwest Passage. The defeat of the French and Spaniards at Trafalgar in 1815 was the most glorious moment for the English navy (Houston). This time the goal was not so much the desire for a faster trip around the world, but for the glory of the taming of the north. Naval officers vied to become leaders of the exploration. Three go down in history: Lt. John Franklin, Ross, and William Edmund Parry. Franklin (Figure 9.16) by the late 1830s was the veteran of 4 Arctic explorations, including an overland trip by canoe(Davis 1995). On May 18, 1845 the 370 ton Erebus (Captain Fitzjames) and the slightly smaller Terror (Cap. Crozier), 139 officers and men, provisions for 3 years (lead soldered

Figure 9.13. Sir John Franklin, leader of the Franklin expedition of 1845 to find the Northwest Passage. Source of image: Beattie, O. B., *Buried In Ice.* Source: Bridgeman/Art Resources.

Figure 9.14. Lady Jane Franklin. Source: Across the Top of the World; National Portrait Gallery.

tin food) set off under the command of Franklin. The men never returned, many relief expeditions set out after them, and due to these relief expeditions the Northwest Passage was indeed charted (although not sailed for another 50 years).

In 1850 the Admiralty gave up hope of finding survivors, but not Lady Franklin who put up her own personal money to finance other search parties (Figures 9.14 & 9.15). She badgered the Admiralty into searching for the records if not for the men.

What secrets may be hidden within those wrecked or stranded ships we know not, what may be buried in the graves of our unhappy countrymen or in caches not yet discovered we have yet to learn. The bodies and graves which we were told of have not yet been found; the books (journals) have not been recovered, and thus left in ignorance and darkness with so little obtained and so much yet to learn can it be said and is it fitting to pronounce that the fate of the expedition is ascertained? (Woodman 1991).

The Franklin party was the only Northwest

Lady Franklin's Lament
(Traditional)
Also known as a Sailor's Dream, in a compilation by Paul Clayton, from a ship's log dated 1850's

'Twas homeward bound one night on the deep
Slung in my hammock fast asleep
I had a dream which I thought was true
Concerning Franklin and his bold crew

"Twas as we neared the English shore
I heard a lady sadly deplore
she cried aloud and seemed to say
Alas my husband is so long away

'Twas seven years since that ship of fame
First bore my husband across the main
With hearts undaunted and courage stout
To seek a Northwest Passage out

To seek a passage around the pole
one hundred seaman brave and bold
With hearts undaunted and courage true
"Tis what no man on Earth can do

There was Captain Osbourne of Scarborough town
Brave Petty and Winslow of high renown
there was Captain Ross and many more
in vain they cruised 'round the Arctic shore

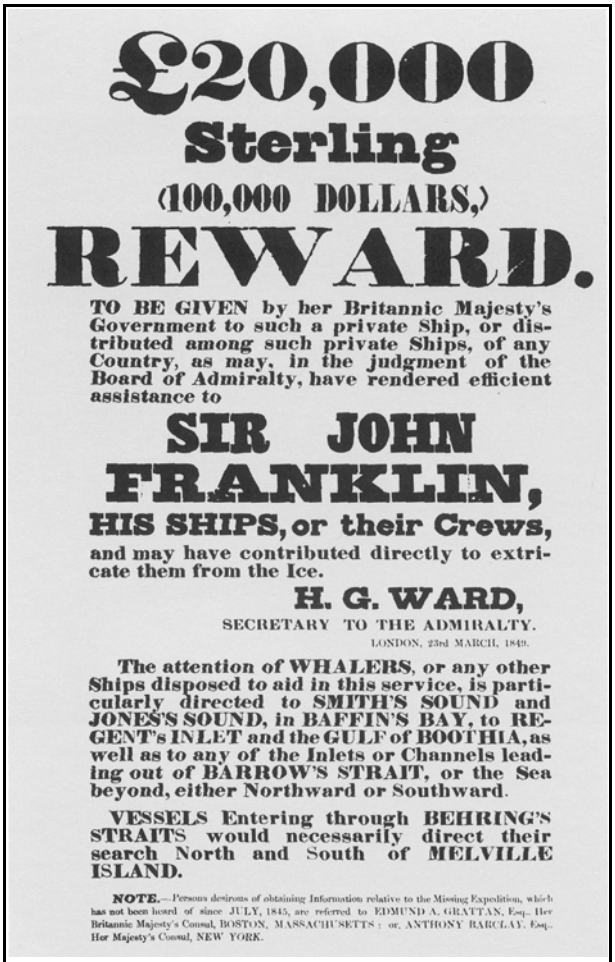
Oh they sailed east and they sailed west
on Greenland's coast where they thought the best
Amid hardships and dangers, they vainly strove
On mountains of ice their ships were hove

On Baffin's Bay where the whalefish blows
is the fate of Franklin that no one knows
Ten thousand pounds I would freely give
To find my husband still did live

And to bring him back to this land of life
Where once again I could be his wife
I'd give all the wealth that I ere shall have
But I fear alas he has found his grave

Now a voice deep within that I can't control
Is assurance to me of his peace of soul
Oh arctic seas what you now have sealed
At judgement day shall be revealed

'Twas homeward bound one night on the deep
Slung in my hammock fast asleep



£20,000
Sterling
(100,000 DOLLARS,)
REWARD.

TO BE GIVEN by her Britannic Majesty's Government to such a private Ship, or distributed among such private Ships, of any Country, as may, in the judgment of the Board of Admiralty, have rendered efficient assistance to

SIR JOHN FRANKLIN,

HIS SHIPS, or their Crews,

and may have contributed directly to extricate them from the Ice.

H. G. WARD,
SECRETARY TO THE ADMIRALTY.
LONDON, 23rd MARCH, 1849.

The attention of WHALERS, or any other Ships disposed to aid in this service, is particularly directed to SMITH'S SOUND and JONES'S SOUND, in BAFFIN'S BAY, to REGENT's INLET and the GULF of BOOTHIA, as well as to any of the Inlets or Channels leading out of BARROW'S STRAIT, or the Sea beyond, either Northward or Southward.

VESSELS Entering through BEHRING'S STRAITS would necessarily direct their search North and South of MELVILLE ISLAND.

NOTE.—Persons desirous of obtaining Information relative to the Missing Expedition, which has not been heard of since JULY, 1845, are referred to EDMUND A. GRATTAN, Esq., Her Britannic Majesty's Consul, BOSTON, MASSACHUSETTS; or, ANTHONY BARCLAY, Esq., Her Majesty's Consul, NEW YORK.

Figure 9.15 Reward poster for news of the Franklin Espedition. Source: James P. Delgado: Across the Top of the World (National Maritime Museum).

Figure 9.16: Supposed fate of the Franklin crew. Sir Edwin Landseer's *Man Proposes, God disposes*. *Source, Beattie's Frozen in Time.* http://www.rhul.ac.uk/Visitors-Guide/images/man-proposes.html

Figure 9.17. Area explored by Franklin's crew. Data source: O. Beattie and J. Geiger, 1987 *Frozen In Time*. Saskatoon, Canada: Western Producer Prairie Books

Expedition to never return a single crew member. The fact that it had been equipped with the most modern of technology (boilers, tinned food, metallized hulls to break ice), the most able of arctic explorers, and the success of Lady Franklin in keeping it before the public eye created a public sensation. Figure 9.16 shows a popular conception of the fate of Sir Franklin's crew as shipwrecked and meat for polar bears (Sir Edwin Landseer: Man Proposes, God disposes). The "truth" was fully as grizzly as feared.

Historical Reconstruction of Events

The various search parties did find physical evidence of the crew, as well as rock cairns containing a few cryptic notes. Based on this evidence and on "selected" testimony from the Inuit the fate of Franklin's expedition was determined. The prevailing reconstruction was that the ships wintered by Beechey Island (Figure 9.17) the first winter and by King Williams Island the second winter. An outbreak of scurvy had them abandoning ship and hauling large tins of food and stoves and other unsuitable material south on man pulled sleds. Evidence of cannibalism was found (carefully piled long bones with saw marks for the extraction of marrow) and it remained a mystery as

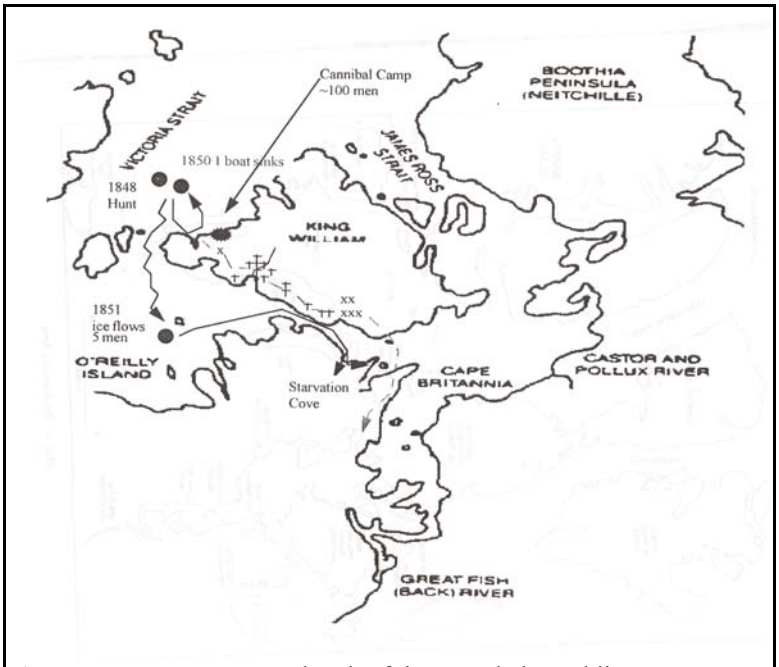


Figure 9.18. Reconstructed path of the stranded Franklin crew.

to why the men resorted to this in the presence of sufficient food stores.

An alternative reconstruction of the events based on accepting the Inuit testimony in its entirety (partly based on their accurate recollections of Frobisher's party) is shown in Figure 9.18. The ships were still locked in ice by the end of the second winter. A small hunting crew was sent out in 1848 to combat scurvy. This crew returned to the two ships. The Inuit participated in a large scale hunt with the crew in 1849, a hunt that may have triggered subsequent years of famine. One of the ships was suddenly crushed by ice (as seen by the Inuit) before it's stores could be off loaded. The crew and the remaining officers removed to the shoreline, where they proceeded south to walk to Hudson Bay outposts. Many of the crew, refusing to move on, returned to camp near the boat. 45 others proceeded south along the King William shore, traveling fewer and fewer miles between camps and building careful graves for each of the fallen. This . crew encountered a band of Inuit and pleaded for food They had rotting gums and blackened teeth and were clearly of poor health. The Inuit gave them some food but slipped away at night, perhaps in fear of dangerously reducing their own food supply. The number dead increased with each camp until only some three men were left to die without graves at the Todd Islands, where they had hoped to sail across to the river. The 60-70 crew members camped off shore from the remaining boat resorted to systematic cannibalism. A small party of men refusing to participate in cannibalism returned to the boat. The boat was carried south in an ice flow in 1851 where it again froze, but a small boat was launched with four men attempting to go by boat to the river. These men perished in the vicinity of Starvation Cove (Woodman 1991). Conventional wisdom has it that these men perished of scurvy. The tinned food, heated in the tinning process would have destroyed any vitamin C necessary to prevent scurvy. Scurvy is a complex disease resulting from vitamin C, niacin, riboflavin, and thiamine deficiency. It is aggravated by isolationsensory deprivation and environmental stress and could lead to hallucinations, withdrawal, and schizophrenia. Physically, men become anemic, weak, have rotting gums and teeth.

Figure 9.20. Rusted tin can from the Franklin expedition. Figure from Beattie's *Buried In Ice.*

Table 9.5: Lead Content of Franklin Crew, ppm

Data dource: Beattie and Saville, 1983

Material	Franklin Crew	General Public	Unexposed Public	Exposed Public
Aorta	6.96	1.94	2.96	6.84
Liver	34.27	1.60	1.03	1.93
Kidney	20.42	10.90	0.78	0.66
Lung	4.09	0.55	0.22	0.31
Spleen	9.88	0.53	0.23	0.32
Stomach	10.4	0.88	0.09	0.1
Bladder	1.42	0.39		
Muscle	3.40	0.34		1.0
Intestine	1.72	0.31		
Skin	6.53	0.10	0.19	0.08
Bone	128	22.36		74.00
Hair (10 cm from root)	225-565			
Inuit bone	5.1			
Caribou bone	2.0			
Modern bone	29.8			
Modern hair	1-8			
Ice from grave	<0.1			

Lead Stands Accused

The fact that 9 officers (27% of the officers) and 15 men (15%) had died by the end of the first year, the fact that many stores of canned food were found (suggesting adequate food, but perhaps suggesting food placed earlier in hopes of land exploration), the fact that had the Englishmen attempted to adapt to the Inuit style of life (furs and seal hunting) they could have survived (many died in their thin woolen, English outfits), and the fact that the recovered tin cans, and bones, all contained similar isotope ratios of lead, as well as elevated lead concentrations, led Beattie to suggest that the judgement of the officers failed under the influence of lead poisoning.

We can list Beattie's arguments as follows (Kowal and others 1991), (Kowal and others 1989). A) Lead was readily available in the tins. B) There was an elevated level of lead in the sailors. C) The lead in the sailors was not endogenous (preceding the trip). D) The lead level was high enough to affect the events.

Argument A: Lead was available from the tin cans

The expedition was equipped with a large amount of tinned cans: 4220 kg canned lemon juice, 16,750 canned litres of alcohol, 6,028 canned liters of vinegar; 910 canned liters of wine; 2,640 canned litres of pickles; 773 liters canned cranberries; 8,000 tins of meat, 11,628 litres of tinned soup, 546 kg tinned pemmican, and 4,037 kg tinned vegetables.

These cans were manufactured barely 5 years after invention and contained large amounts of lead in the solder joints. The cans could contain up to 30% lead in the inside seam. Figure 9.20 shows a rusted tin can from the expedition.

The lead from the tin cans could get transferred to the sailors. One case of this was mentioned in Chapter 7, with respect to another arctic expedition, that of DeLong and the Jeannette. Beattie argues that tin cans could transfer the lead to the food. Old tin cans opened show an increase in lead content, particularly if the interior is not enameled. The amount of lead in an opened and stored open (leftover) tin can of fruit showed lead at 0.01 to 0.20 ppm on opening

Figure 9.21 Franklin crew member John Hartnell, exhumed for analysis. Image source: Beattie *Buried in Ice.*

and 0.19 to 0.48 at day 5 (Capar 1978). If eating from an open container one could obtain 1.32 µg lead/day.

If one assumes that the store of food was eaten at a "reasonable" pace, it could be argued that large daily amounts of lead from the tinned food would be ingested. Beattie (Beattie) assumes that if each sailor ate1 can of food/day **with all the lead transferred from can to food to body,** the amount ingested with be 700 ng/day, which would easily reach clinical (classically high) symptoms of lead poisoning over a four year period.

These arguments related to availability and transfer of lead during the course of the expedition have been disputed by Farrer. Farrer(Farrer 1989; Farrer 1993) does not dispute that there was lead available from the tin cans. He does argue that lead is **not** transferred from the tin to the food. In a case of dueling experts, Farrer points out that chemically lead is less susceptible to oxidation than tin (Page and others 1974). While this is true, the amount of oxidation will depend a lot on the particular can, the lacquer used, and the isolation of the lead solder from tin can by the lacquer (to create an isolated lead electrolytic cell). Natural food chelating reagents would affect solubility of the lead also, as we showed in Chapter 7. Tinned foods with lead solder are generally quite safe according to Farrer's experts (Boudene 1979). He finds literature about contemporary tin cans (1820-1850) which indicate little lead in the food. For example, one set of tin cans from 1856 were opened and analyzed to find less than 2 ppm lead (Warnock and Wills 1991), which Farrer contends is a **low** amount of lead. Other analyses (dating to the 1930s) indicate that lead is not measurable in tin cans recovered from Capt. Parry's 1824 expedition, nor that of Capt. Belcher's 1852 expedition(Drummond and Macara 1938; Macara 1928). This latter piece of evidence we can toss out since the limits of detection were quite crude in that time period. < 2 ppm does not mean 0 ppm!

Even if lead was present in large amounts in the tins this does not mean that the general population of sailors would be lead poisoned. Farrar computes that the tinned food was not present in sufficient quantities that it could contribute a large intake of lead. The amount of food intake from tins was relative low for the total number of sailors present and other sources of food on the boats. We can make a small calculation based on the expected duration of the voyage (3 years) and the total number of sailors (139 of whom 15 were officers). Beattie uses the amount of one can of food (soup or meat)/day for each sailor for 3 years, an amount which would supply only 17 sailors. This would suggest that Beattie's worst case scenario could not be achieved and that lead intake would have to be considerably less than 700 ng/day.

Argument B. Higher (Than Normal) Lead Levels were found in the Sailors

The remains of some of the 129 member crew

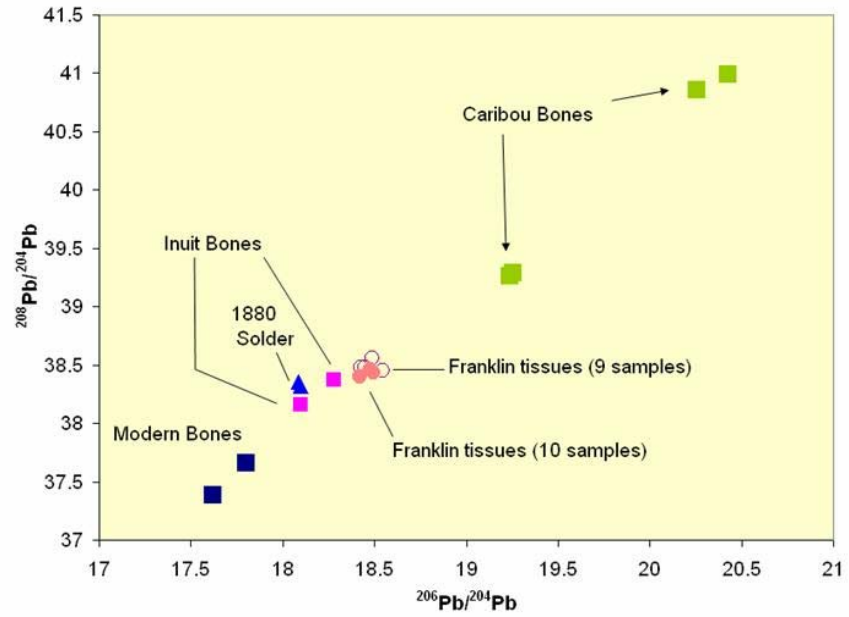


Figure 9.22. Isotope ratio plot of data from the Franklin expedition . Data replotted from Koval, Beattie, Baadsgaard, Krahn, *J. Arch. Sci.,* 1990, 18, 193-203.

were uncovered in the early 1980s by Beattie and co-workers and found highly lead contaminated (Figure 9.21). The bones and tissue recovered from autopsy of the thawed corpses (Amy and others 1986) and of newly recovered skeletons (Beattie and Saville 1983) show high lead (Table 9.5). A control for this argument is that the amount of lead from caribou and Inuit bones of same time period was low and of a different isotope pattern (indicating the recovered bones are not contaminated).

The results are not due to diagenesis of the bone material due to arctic climate and the fact that few of the recovered remains were actually in contact with thawed soil.

Based on the results of the individuals recovered Beattie extrapolates to the general condition of the 139 member crew. The lead observed in the recovered remains would indicate generalized lead poisoning of the crew.

Farrar points out that John Hartnell (Figure 9.21), upon autopsy, showed no signs of the Burton's lead line or other non-bone lead manifestations of lead poisoning. This, he argues, implies that there was no lead poisoning among the sailors. This is a poor argument because Burton's lead line would only be observed in individuals in the later phase of acute lead toxicosis. Farrar further states that Beattie's own autopsy showed that John Torrington had clearly been previously examined by the ships doctor and the probable cause of death, judging from the organs removed in that autopsy, and judging by the bacterial investigation of the more modern autopsy would have suggested tuberculous and generally wasting (Notman and others 1987).

Even if one concedes that there is a high amount of lead in John Torrington's thawed body, this has not implications for the rest of the crew, Farrer argues. John Torrington died early in the expedition (within 6 months) and would have had a higher exposure to lead because of his sick bay status permitting extra tinned rations. (This seems a contradiction of Farrar's assertion that there was, in fact, no lead in the tins.)

Argument C: The Lead Level Did not precede the Trip and must come from the trip itself

Beattie argues that the amount of lead in the recovered tissues and bones would not be present in the general English population of the time. Farrar suggests that the bone lead concentration of the sailors may be indicative of that population of sailors in 1800s England. This is akin to suggesting pervasive lead poisoning of the entire working male population of England. There is some justification for this assumption. As Nriagu points out lead induced gout was symptomatic of both the Romans and of the British.

In order to prove that the lead could not be due to an endogenous source of lead (pre-voyage), Beattie excavated the frozen remains of the sailors and sampled for hair and tissue. In Chapter 8 it was noted that the kinetic half life of lead in the body differs based on what type of bone it is deposited in and in what type of tissue. In order to rule out lead exposure preceding the expedition, tissue samples, with a half life for lead of 35 to 40 days, were collected. The half life of skeletal lead has been estimated to be between 5.6 and 16 years (Nilson and others 1991). The

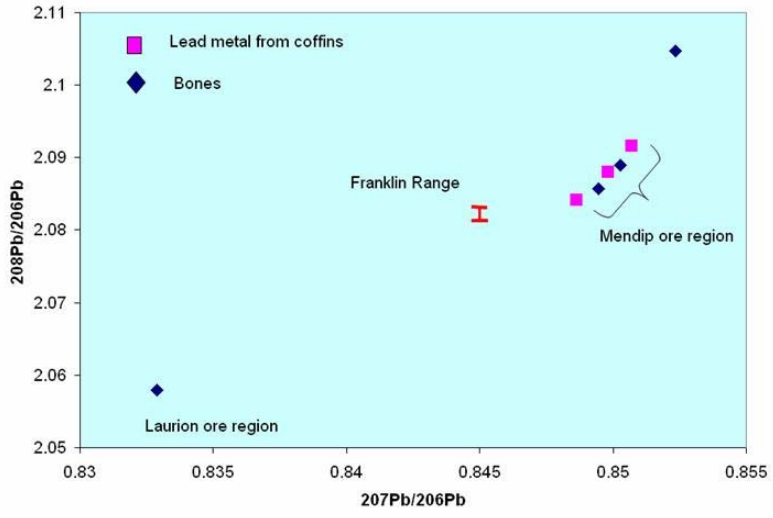


Figure 9.24. Isotope variation for Roman remains found in Poundbury. Data replotted from T. I. Molleson, D. Eldridge, N. Gale, *Ox. J. Arch.*, 1986, 5, 2, 249.

analysis indicated that the tissue content of lead was extremely high, indicating recent ingestion of lead. The level of lead in the hair was high enough to suggest clinically acute case of lead poisoning which would lead to hallucinogenic behavior. This data is corroborated by the isotope match between the lead in the tins and the crew. (See Figure 9.22).

The recovered bones all have same isotope pattern. If lead derived from pre-sail exposure, then they should have a wider spread of isotope ratios. Farrar argues that the isotope match is a **false** positive. Isotope matches can only tell you when two things do not match, but not that two things derive from the same source. Further, the isotope ratio of Roman bones found in England (Molleson and others 1986) match those of the sailor, suggesting that the method is insufficiently precise to show geographical variation **within** England (Figure 9.23). This is wrong. There is a wide spread of lead isotopes through out the ore bodies in England. When Beattie's data is converted to 208/206 ratios the data varies from 2.0816 to 2.0831 which does not begin to match the spread of the English ore bodies (2.075 to 2.1).

In effect, Farrer is arguing that there would be no variation in Englishmen so that the coincidence of all Franklin crew individuals (5) with the tinned lead isotope does not demonstrate that their bone lead is affected by the tin, but that was original, or prior to sailing. This seems a little bit of a stretch, assuming that all Englishmen would have no discernable isotope change. Farrer uses the fact investigators of the Roman bones, upon finding one individual with a Greek lead mine isotope signature, cautioned against uncritically assuming the individual was a Grecian immigrant.

Farrar goes on to argue that the very fact that there is consistency in the bone lead isotope pattern implies that the source of lead must have preceded voyage. He bases this argument on the longer half life of lead in bones.

Based on the type of bone lead values found for non-occupational exposed persons (Drasch and others 1987) he suggests that a typical value of lead in the bone is variable and unreliable for comparison to archaeological bones.

Even so, he goes on to calculate the expected blood lead value from the bone concentration. He assumes that if one has a blood volume of 5 L an increase in the lead from 20 µg/100 mL to 40 µg/100 mL requires the extraction of 1 mg of lead from the bone. Since the Pb/Ca ratio in the bone of a normal person is 1/5000 this would require the release of 5 g Ca to result in a blood Ca level of 100 mg Ca/100 mL which is 10 times the normal value. This, Farrer, suggests, implies that it is *impossible*, for a high bone lead value to produce a high blood lead level.

This shows some chemical naivete for Farrer is assuming that the **rate** of metabolism out **and** in to bone is the **same** as the corresponding rates of Ca out and into the bone. He further assumes that the lead blood equilibrium would have to be co-incident with the calcium blood equilibrium. Neither of these assumptions are necessarily true. More recent models of bone metabolism indicate that lead can be rapidly deposited to a short term bone material (trabecular) and then stored to the cortical bone, where it has a longer half life, the two compartment model (Wittmers and others 1988). The lead stored in both bones can affect the blood lead. A case in point is of a case recently reported (1994) in which a lady with childhood pica (eating of paint chips and soil) and adult exposure to home renovations was experience lead poisoning symptoms (persistent fatigue, insomnia, difficulty concentrating, abdominal cramps, weight loss, muscle and joint aching, and tremor for several months) some 7 years after the fact (Goldman and others 1994). X-ray fluorescence of the tibia showed 154 µg Pb/g bone

Table 9.6 Ascorbic Acid (VitaminC) content

Source	Ascorbic Acid (mg/100 g) raw	Cooked or preserved
Seal flesh	0.5-3	0.5-2.5
Seal liver	18-35	14-30
Whale skin (narwhal)	18	
Whale skin (Beluga)	35	
Cod roe	44	
Broccoli boiled	140 mg/155 g	
Cabbage (boiled) 1 cup	40-50 mg/170 g	
Baked potato (with skin)	30/200 g	
Boiled potato	5-15mg/200 g	
Orange	66 mg/180 g	
Strawberries, 1 cup	88 mg/150 g	
Sailors Diets		
Gooseberries	60-65 mg/100 mL	0
Spruce pine needles	65-200 mg/100 g	
Oranges by Lind's recipe, fresh	240	
Oranges by Lind's recipe, 28 days	60	
Canned soups	0	

Data Source: Carpenter, 1986

mineral (unexposed individual should have 5-10µg Pb/g bone mineral). The blood level of this patient was 53 µg/dL. Since no other current source of lead presented itself, this blood lead level had to come from the bone material. This implies that bone lead does turn over, and contradicts the theory that once it is deposited it would not affect the behavior of the men within the two year period. Chelatable lead in urine of workers was linearly related to vertebral bone lead (trabecular) but poorly related to the finger bone lead (cortical, compact, or dense) bone, consistent with the two part model of lead mobilization from bone (Schutz and others 1987a).

Argument D: The amount of lead was high enough to affect the events

Beattie, having shown that there was a source of lead, that the sailors had a high amount of lead, that the amount of lead was in short term storage and of an isotope match with the source, but not with English spread, argues that the amount of lead present was sufficiently high to cloud judgement. This would explain the reason why the survivors tried to walk south carry a cast iron stove on a sled, as well as why they were so poorly clothed and died of malnutrition even with ample stores of tinned food. He argues that the disproportionate deaths of the commanding officers would suggest a disproportionate exposure to lead, consistent with higher intake of lead from a higher percent intake of "luxury" items like tinned food. A greater level of lead poisoning among the commanding officers would certainly lead to problems in decision making.

His data shows that the main body of sailors would experience enough lead poisoning to be under physiologic stress. The level of lead in the body would be consistent with anemia, fatigue, lack of musculature control, and hallucinogenic behavior consistent with the poor judgement at hauling the sleds full of tin cans, lockets, trinkets, stoves, etc. The high bone lead content (128 ppm) for John Torrington, coal stoker on a dry weight basis (this was an ashed measurement as opposed to X-ray fluorescence) can be converted to a wet bone lead content in the same fashion that the bone lead content for the Romans was calculated. We find that John Torrington would have a wet bone lead concentration of about 60, which would correlate to blood lead levels of about 0.7 Fgram Pb/L (14.5 Fg Pb/dL) consistent with weakness and anemia. While this level would not be enough to cause hallucinogenic behavior it would certainly contribute to diminished stamina and recovery of weak sailors.

Based on these arguments Beattie concludes that the contribution of lead to the health of the sailors would be a "significant" factor (along with scurvy) in the health of the crew and officers, and therefore in the decisions made. Farrar argues that Beattie has not proven his main points and that scurvy would be a predominate factor in the events.

Other Objections to Lead as a Causative Factor

Other historians also have a lukewarm response to Beattie's hypothesis. An entirely different argument against lead poisoning contributing significantly to the fate of the Franklin crew is made by Woodman (Woodman 1991). Woodman contends that any crew of the size of Franklin's would have met the same fate. Once they were trapped in the ice and food stores dwindled they would not have been able to survive because of sheer numbers and the inadequate

Figure 9.25 Woodcut of early Dutch explorers trapping and killing polar bears to obtain fresh meat and so ward off scurvy during a winter in the Arctic . Image resides in the Doe Library. Source Carpenter: The History of Scurvy and Vitamin C.

natural resources of the arctic. Only dividing into smaller hunting crews (5-6) would any have had a chance. The crew would have had to adopt the Inuit eating style also, since the only apparent source of vitamin C was to eat raw flesh, since cooking destroys the vitamin C efficacy. (The non-Inuit called the Inuit Eskimos, deriving from a Cree Indian description: Raw Flesh Eaters, referring to how the Inuit ate animal meat. A particular delicacy is the raw intestines of Polar bears.) The vitamin C content of raw liver is quite high (Table 9.6) (Carpenter 1986).

Not all Europeans scorned the Inuit diet in their survival attempts. An English whaling expedition sent a small party ashore who became stranded in May until the fleet returned the following summer. Rowing to another bay they found a cabin used for extracting oil from the whales. They ate sparingly and with great hunger of bear, deer, walruses they killed. Their food was gone in Jan, but in Feb they killed a bear.

Upon this bear we fed some twenty days....This only mischance we had with her, that upon eating her liver, our very skins peeled off: for my own part, I being sick before, tho I lost my skin, yet recovered I my health upon it.

A 1596 party of Dutch had a similar experience (Figure 9.26) as well as that of 4 Russians sailors stranded in 1743 for six years with only an axe, knife, kettle, and tinder. They found an iron nail and board with a long hook which they used to kill reindeer, but were unable to cook the meat. One of them had once wintered in the arctic and

he instructed them to eat raw and frozen meat cut into small pieces and drink the warm blood of the reindeer, and lastly, to eat as much as possible cochlearia (scurvy grass), the only grass that grew on the island, and that but sparingly....Three of the sailors who made use of this regime kept entirely free of this complaint....the fourth had an unconquerable aversion to the blood...and the malady made such progress that he was subject to cruel suffering....without the power of moving his hand to his mouth.

9.2.D. Summary

Like the case of the Romans, valid arguments can be made to sustain both points of view. What is clear is that the bone record for the Franklin expedition was high enough to suggest severe health effects from the lead. It is not entirely clear if the lead was endogenous to the sailor (all of England poisoned) or whether it derived from shipborne source, among which are the tin cans. Whether lead poisoning contributed **significantly** to the events that unfolded is an entirely separate question.

PART II: Chemistry of Archaeological Bone

In order to perform an analysis of the historical skeletal population under investigation, it is necessary to ascertain the postmortem stability of the bone mass. Bone consists of a tailored or specifically woven inorganic/organic material to increase the tensile strength of the material. Alive it has the strength of iron but with a very low density. Woven and lamellar bone (formed one month after birth) has collagen (protein fibers) oriented length wise to give additional strength to the bone. The mineral or inorganic phase of bone is generally 60-70%, water is 5-8% and organic material is the remained of which 80% is collagenous proteins.

Most of the organic phase is type I collagen, which is a low solubility, 3 polypeptide chains with 1,000 amino acids each. It consists of a triple helix of two identical α I chains and one α II chain cross-linked by hydrogen bonding between hydroxyproline to produce a linear molecule 300 nm long, which is aligned parallel in an array to produce a fibril, which is in turn bundled to form the fiber, leaving gaps between bundles, and pores between parallel molecules. Mineralization begins in the hole zones. Collagen itself is conservative protein in evolutionary terms. It does not vary in structure and amino acid composition among vertebrate classes. Collagen can survive for several thousand years in cool and stable environments, and retains it stable isotope ratio values against diagenetic onslaught.

The inorganic phase consists of hydroxyapatite $Ca_{10}(PO_4)_6(OH)_6$ where K, Mg, Sr, and Na can substitute for Ca, carbonate for the phosphate, and fluoride for the hydroxide (Table D.4). The geologically crystalline hydroxyapatite is plate-like crystal 20-80 mm long and 2-5 nm thick. Bone apatite are much smaller (20-80 nm in the largest dimension) with many impurities adsorbed onto the surface or incorporated into the crystal. Deposition occurs by crystal growth or by initiation of new crystals, a procedure that is controlled by regulators such as osteocalcin, a caroxyglutamic acid containing protein bone Gla (5.8 kd) with three carboxylated residues (driven by Vitamin K modification), which makes this into a calcium binding protein. Osteocalcin accounts for 10-20% of non-collagenous protein. It's synthesis is enhanced by 1,25-dihydroxyvitamin D. Ions which can be incorporated into the bone matrix are those which replace Ca (Na, K, Sr, Ba, Pb, Zn, Mn, Fe), those which replace phosphate (carbonate), and those which replace OH (F). The elements Fe, Zn, Rb, Sr, and Pb are those that have been measured as replacement within bone during life (O'Connor and others 1980). Fe was found to range from 893 ppm, Zn, at 213 ppm, Rb at 25 ppm, Sr, at 108 ppm, Pb at 25 to 40 ppm, and was related to the type of bone.

On death the chemistry that drives bone changes is related to the chemistry of the soluble fraction of the soil, which will depend upon the pH, the organic matter, mineralogy, and most importantly upon the fluoride, carbonate, and phosphate soil composition. The simplest model for predicting postmortem changes is based on diffusion as the rate limiting step, where diffusion is controlled by the extractable or total elemental composition of the soil, instead of the soluble or exchangeable fraction of the material in the soil.

Several situations may be envisioned (Figure 9.26). Elements (arbitrarily labeled Group I) will leach rapidly from the bone to the soil, where soil kinetic processes will be slow. Consequently, these elements will have an anisotropic, local, soil increase near the bone (Ca). Elements (Group II) will leach from the bone to the soil, where soil kinetic processes will be rapid. These will show isotropic distribution in the soil, and depletion in the bone (Na). Elements in group III show a anisotropic decrease in the soil and increase

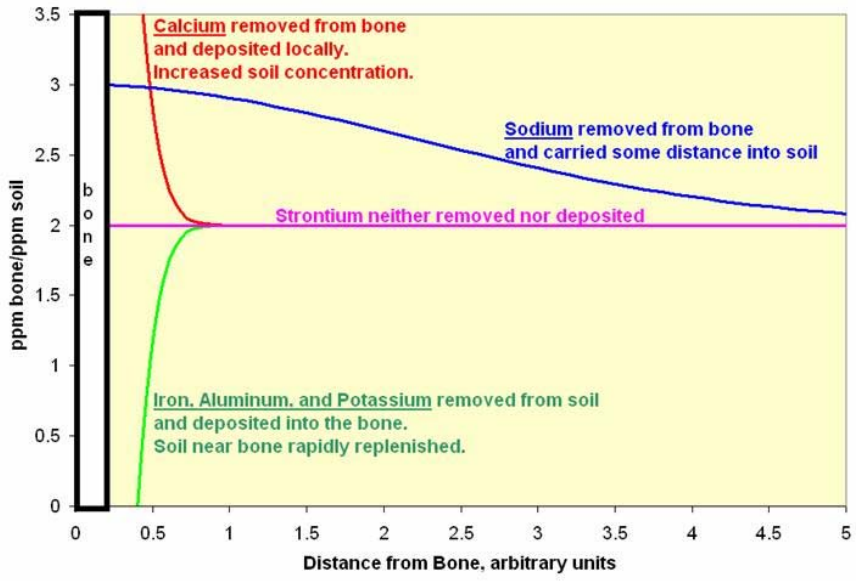


Figure 9.26. Concentration of elements in soil change with respect to buried bone structural changes. Accurate measurement of archaeological bone material must account for bone diagenesis.

in the bone (deposition). Elements in group IV show fast soil kinetic processes which replenish the soil while allowing for deposition within the bone (Fe, Al, K). Finally in group V, elements (Sr) are stable within the bone. This discussion shows that in order to determine postmortem stability soil tests as to total elemental abundance will be misleading (Lambert and others 1984). It is unclear from soil tests what the pattern of behavior is for lead. Early studies suggested that a lower rib than femur concentration of lead was indicative of lead leaching, however data for modern bones in Chapter 8, suggests that the lower rib concentration is inherent in the bone prior to burial.

Another way to test for postmortem changes of the bone is to more carefully examine the structure and physical chemistry of the bone. The attack of the soil component upon the bone will depend upon the bone material which is partitioned into several phases, with the dicalcium phosphate dihydrate being the most immature portion of the bone found at cortical surfaces (Table B.18). Hydroxyapatite is the most dominate form. The dominate form of the bone hydroxapatite, increases in solubility as the pH drops below 6, where the soil can be acidified by soft tissue decomposition, or by microbial decomposition of the collagen. As the hydroxapatite dissolves other materials may be deposited. If the soil is high in carbonates, then the bone should be destabilized as carbonate substitute for phosphate. If the soil is high in F, the bone should be stabilized as F substitutes for OH.

The factors affecting postmortem changes are dissolution, precipitation, re-crystallization, crystal growth and ionic substitution. A very similar argument was made previous with respect to the conversion of calcite into lead zoned crystals (Chapter 1). Precipitation will consist of soil derived water soluble crystals of calcite in voids and fractures of the bone, followed by ion exchange into the interior of the bone, followed by re-crystallization which converts microcrystalline biologically deposited hydroxyapatite to larger well crystallized apatite. Changes should occur outside in, beginning with an ion exchange reaction to the bone/collagen surface, followed by diffusion into the bone interior. Pb^{2+}, Sr^{2+}, CO_3^{2-}, and F^- enter the hydration layer on the surface of the bone. Rapid ionic exchange occurs between surface Ca sites in bone and solution cations, followed by diffusion into crystal interior (Johnson and others 1970). Exchange should occur readily when radii are within 15% (see also discussion of alloys in Chapter 3) (Saleeb and DeBruyn 1972). Sr/Ca are more highly different (r 1.18 to r 1.00 Ca). Synthetic hydroxyapatite in equilibrium with Ca/Sr gives a Sr/Ca ratio of 0.6, suggesting discrimination against Sr(Koutsoukos and Nancollas 1981; Likins and others 1960).

In order to identify ion exchange, Pate, compared the total bone concentration in archaeological bone with the soil solution composition of the soil from which the bone was derived. He also treated the bone with acetic acid at low pH (whole bone dissolution) and at pH 5.8 (dissolve soil calcites). He found that in these Australian soils HCO_3-$^-$ was the predominate soluble anion, followed by chloride and sulfate. Phosphate was minimal. All concentrations increased with depth (consistent with translocation to lower levels). Data indicated that the weak acetic acid solution had higher Ca/P than the whole bone (no rinse

sample) suggesting that calcites ($CaCO_3$) had been deposited. High loss of Ca on ignition also suggests calcite as opposed to calcium phosphates (see Chapter 3 on cuppellation, Chapter 1 on refractories). The transition to gas phase for the apatite occurs at a very high temperature, so should not be lost on ashing, unlike the calcites. Ignition losses were found to be greater for older bones, as well as the Ca/P ratio was large in the pH 5.8 acetic acid was. Pate suggested the following preparation procedure: distilled water wash to remove soluble salts at every 1 min until no further removal is measured. Rinse next for 18-24 hour acetic aid at pH 6 to remove secondary carbonates (calcites). Follow this rinse with 1 minutes washes with pH 5-5.5 acetic acid to remove soil derived apatites. End with 1 minutes washes with pH 4.5 acetic acid to remove low carbonate apatite (the most soluble of the bone apatites) to compare with soil solution composition.

A similar study was carried out by Lambert and co-workers(Lambert and others 1985). Lambert found that intact whole archaeological bone exposed to solutions of soluble salts showed less ion exchange than crushed bone did for all elements investigated (Sr, Zn, Mg, Ca, Na, Pb, Mn, K). The bone material took up a significant amount of lead, and strontium, but little K, Na, or Ca. The data was consistent with exchange reactions observed in earlier literature. These early reports suggest that exchange involves substitution within the bone matrix, deposition into void or defect areas and surface adsorption(Falkenheim and others 1947; Neuman and Mulryan 1950; Neuman and Neuman 1958; Neuman and others). It is suggested that Sr moves into the bone material (similar radius to Ca), as does Zn. Mn and Fe fill voids and defects only, while Na, K, and Ca are on the surface of the bone (Armstrong and Singer 1965; Parker and Toots 1970; Pate and Brown 1985; Samachson 1967; Stoll and Neuman 1958). By analyzing the Ca content in the bathing solution Lambert and co-workers showed an increase in Ca when bone was exposed to Zn, Mg, and Na suggesting that these ions move into Ca containing sites in the bone, instead of simply depositing in the void structure. It has been found that Fe, Mn, and Al appear to be deposited from the soil in the bone (Buikstra and others 1989; Francalacci and Tarli 1988).

Sillen and co-workers suggest that the geochemical deposition of apatite will occur on the surface of the original bone and will have a different trace metal signature, which will affect it's solubility. Apatites with carbonate will be more soluble, those with fluoride less so. They suggest crushing to a powder the bone and sequentially extracting with weak acid (the first 0-5 washes soluble, 5-10 less soluble.) After 25 washes the least soluble material should remain. They further suggest that isotope ratio matching of Sr should be tracked in these (Sillen and others 1989). Sillen earlier suggested that the Ca/P ratio remained the same during despite obvious morphological and density changes in the bone, suggesting that dissolution and re-crystallization occurred on a microscopic scale (Sillen 1981).Changes in t he bone composition were not linear with deposition time, even from the same site, suggesting variation in the chemical process (Klepinger and others 1986).

All of the above studies were performed with intact bone, which was subsequently ashed and dissolved. Other researchers have focused on the collagenous material of bone. The procedure for removing collagen from bone follows several strategies. In the most common method the mineral phase of the bone is removed by dissolution with HCL or EDTA (to chelate away the Ca, and break apart the mineral)(Ambrose 1993). Contaminates of the collagen are lipids, bone apatite, diagenetic carbonates, humic and fulvic acids. Which can be eliminated with simple chemical pre-treatment. The EDTA (C/N) needs to be extensively removed if C/.N ratios and isotopes are to be determined. Demineralization with weak HCl (1-3%) gives collagen with high C/N vs strong HCl (10%). The demineralized material is subsequently treated with NaOH to remove lipid and humic acid contamination. The collagen is gelatinized by heating to 95C in aqueous pH 3 water, followed by filtration/centrifugation.

Alternative methods leave the bone mineral intact: enzyme purification with collagenase, or redox treatment with bleach (sodium hypochlorite). This latter method appears to leave some collagen sealed in the apatite residue. After loss of collagen the bone undergoes more rapid diagenetic process. Tooth enamel on the other hand is stable even with collagen loss.

These results compare well with studies which compare the diagenetic changes of bone associated with the trabecular and compact bone material. It is expected that the trabecular bone material will weather faster in soils than the compact bone material. Since compact bone material is generally higher in lead than trabecular an inversion of this order in historical bones, or a change in the normalized lead content (femur/rib) would suggest postmortem diagenetic changes. This strategy has been pursued with moderate success by a variety of researchers [Edward, 1984 #2142; Lambert,

1984 #2141]. These studies suggest that lead, in the absence of burial in lead coffins or with lead ornaments, is stable diagenetically. A corollary to these studies is that female and youth bones will be more susceptible to diagenetic changes, since these individuals have a higher bone turnover in life, the buried material will be expected to have a higher rate of diagenesis. Studies of buried populations do indicate that young and very old bones are more highly susceptible to contamination (Lambert and others 1979).

Chapter 9: Problems

1. Which part of Rome was most unstable near the end of the Empire?
2. List the six reasons used to support the hypothesis of lead poisoning among the Romans.
3. When did Nero set fire to Rome?
4. When did the Roman republic end?
5. When did the Vandals plunder Rome? From which direction did they advance (see Chapter 2)?
6. What was the estimated daily intake of a Roman aristocrat?
7. Why did sugar take so long to move from India to western European dinner tables?
8. When was sugar introduced to Spain and why?
9. Who was Apiceus?
10. What is the probable chemical formula of sapa?
11. What is the probable chemical reason sapa tastes sweet?
12. Besides cooking what was the main use of sapa?
13. What were the symptoms of a person who imbided too much sweetened wine?
14. What was the average consumption of wine/day/person of the Roman aristocracy? Could you drink that much?
15. What are the symptoms of Caligula that are consistent with lead poisoning?
16. What are some of the reasons given for the lack of family inheritance among the Romans?
17. Define cortical vs trabecular bone.
18. What is an average pre-lead bone concentration of lead?
19. What is an average Roman bone lead concentration?
20. What elements in the bone are most reliable for internal control in checking for contamination or loss of the bone material?
21. What might be an estimate of blood lead for the Roman bones recovered from York?
22. What would the symptoms the individual in question 21?

23 Do you think a convincing argument has been made for lead poisoning among the aristrocracy of Rome?

24. Who was Pliny and what does he have to say about lead?
25. List four episodes of lead induced diseases associated with wines.
26. When were the first laws against sweetened wines introduced?
27. Give several "old" names for diseases that were really episodes of lead poisoning.
28. How does B. Franklin get into our lead story?
29. What is the most significant phrase in B. Franklin's letter, in your opinion?
30. Who was John Dee?
31. Who was Frobisher?
32. When was the search for the Northwest passage initiated and why?
33. Whom did the English wish to "beat"?
34. Who was Rudolf II?
35. What scientific supplies did Frobisher carry with him?
36. Why is the expedition of Frobisher importnat in unraveling the fate of the Franklin expedition some 300 years later?
37. Why did the English take up the quest for the Northwest passage again in 1817-1819?
38. What were the ships that formed the Franklin expedition. How many sailors and crew members were there?
39. Outline the "conventional" historical reconstruction of events of the Franklin Expedition.
40. Outline the alternative reconstruction of events for the Franklin Expedition.
41. What are the most compelling arguments in favor of lead as a cause of the disaster?
42. What are the weakest links in the arguments?
43. What are the isotope ratios of the crewmen and how do they relate to the ore bodies in Europe (see Chapter 2)?
44. Why does it matter what the skeletal half life is in the arguments?
45. Will lead equilibrium with bone material follow the same course as calcium? Why or why not?
46. What does the word Eskimo mean and what is the "real" name of these people?
47. How do these people avoid scurvy?
48. Does the fact that John Hartnell had no Burton line convince you one or the other about the role of lead in the expedition?

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