THESIS

CRAZY BONES: EVIDENCE OF INSANITY IN THE SKELETAL COLLECTION OF THE COLORADO STATE INSANE ASYLUM

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ABSTRACT

CRAZY BONES: EVIDENCE OF INSANITY IN THE SKELETAL COLLECTION OF THE COLORADO STATE INSANE ASYLUM

Excavations of a forgotten cemetery of the Colorado Insane Asylum yielded the skeletons of approximately 155 individuals buried between 1879 and 1899. The collection exhibited a number of abnormalities that could be related to pathological conditions that may have been the cause of insanity for those present. The skeletons were examined for abnormalities, especially to the crania, and these abnormalities were compared to conditions that may have afflicted Victorian populations and resulted in insanity. In particular, several crania, while normal in appearance, were extremely heavy. Radiographs were taken of these crania to see if any pathological conditions known to be associated with both heavy crania and insanity could be found.

Forty of the skeletons were tested for the presence of heavy metals, specifically focusing on the individuals who had signs of syphilis. It was hoped that, as mercury was the most common treatment for syphilis at the time, the presence of mercury might be useful in distinguishing syphilitic infection from other pathologies of the bone. Arsenic, copper, lead, manganese and zinc levels were also examined, as all were metals common in the period of the asylum, and all are associated with mental disruptions in high enough doses. Although it is not possible to definitely link skeletal remains to specific mental conditions, some abnormalities that seem to be linked to insanity were found.

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Chapter I: Introduction

The study of human skeletal remains can be used to determine a great deal about the life of an individual, but it is rarely used to elucidate more esoteric possibilities, such as the mental state of the individual when alive. Most mental disorders, both those recognized now such as schizophrenia and bipolar disorder, as well as those of the Victorian period such as intemperance and chronic masturbation, do not leave traces on the skeleton. However, some catalysts for institutionalization from the period do manifest osteologically. One way to examine these "deviant" mental states is by looking at collections where the individuals present were known to have been insane by the standards of the time when they were alive, such as an asylum skeletal collection. There are very few skeletal collections of asylums from any period and even fewer that have been studied, making any widespread comparison impossible and their study all the more critical.

The Pueblo Insane Asylum cemetery, dating from 1879-1899, is one such collection. From the opening of the Colorado Mental hospital in Pueblo Colorado in 1879 until the resignation of the first superintendent, Dr. Thombs, in 1899, a cemetery was maintained on the grounds. Although it does not account for all the recorded deaths at the asylum during the period, the graveyard did contain up to 200 individuals, of which roughly 155 have been excavated (Mitchell et al. 2002; Magennis 2002). As the graveyard was used only by the asylum as a final resting place for patients, these skeletons represent only individuals deemed insane by their contemporaries, allowing for the study of abnormalities that may reflect the inmates' mental condition in life.

The results of illness can be seen both as physical abnormalities and in chemical traces that reveal treatment and possible reasons for incarceration in the institution; in particular,

congenital abnormalities, unusual skulls and signs of tertiary syphilis as well as traces of elements known to be associated with mental disturbances. Of 155 skeletons, there are a disproportionate number that have unusual features, particularly on the crania. In most cases, this would likely be hereditary, but there is no indication of familial relations in the admittance records and not having anyone to take care of them was a leading decider of whether people ended up in the state asylum (Mitchell et al. 2002).

Without the soft tissue, only a few avenues of study are available for the understanding of insanity in this collection. The first is any deformities or abnormalities in the crania. Although the lumps and bumps of the skull do not reflect personality and mental state as the phrenologists of an early period believed, many changes in the bone do indicate more widespread changes in the body that include insults to the brain. These can include injuries, some diseases, congenital disorders and perhaps even age-related degradation, such as dementia.

A second avenue of exploration is the chemical composition of the bone. It is well established that in high enough quantities, many elements, particularly heavy metals, can cause mental problems and insanity. When present in high enough doses to affect mental functions, these elements are frequently incorporated into bone. As the elements are inorganic, they do not break down with the passage of time and so leave a record in the bones of the contamination present in life. Testing the bones for these elements can therefore reveal their presence and possibly the cause of insanity for some individuals.

The first step in trying to find osteological traces of insanity in a Victorian collection is to understand the history of insanity, the theories of its treatment and causes as well as what conditions were considered insane during the period the asylum was in operation, which is in the second chapter. This information not only reveals what osteological changes should be looked

for, but also their meaning, both as a manifestation of symptoms and how these manifestations might have been understood and treated by Victorian doctors.

The osteological implications of those diagnoses are considered in the third chapter. This includes diseases that would affect both the skeleton and brain function. This is also where the effects, doses and possible exposure routes for trace element analysis need to be considered. It is important to know not only how the elements affect the body, but also how they might have entered it to begin with and what implications those exposure routes have.

Once the background has been established, it is possible to look at the Pueblo Insane Asylum collection in the proper context. This context informs the methodology and what information is examined. In particular, the search for cranial abnormalities and the processes used to gather the trace element analysis of the bones. This is the focus of the fourth chapter.

Finally, the results of the analysis of the asylum collection are reported in the fifth chapter. Although it cannot properly be compared to any other collection at this time, the results can inform the study of other asylum collections, creating a baseline for what expected results might be. In time it may indeed be possible to determine insanity from skeletal remains.

Chapter II: History and Theories of Mental Illness

Insanity and the people who fall under its purview do not exist in a vacuum. The societal expectations, responses, treatments and even biological realities behind it change with different times and conditions. The Colorado Insane Asylum is no different. Colorado built the asylum in response to larger concerns about the rising numbers and changing character of insanity during the end of the 1800s, and these changes reflect in its creation, administration and who was admitted to the institution. In particular, there were concerns sweeping the industrialized world in the later 1800s that insanity was rising at an alarming pace and that something had to be done to stem the tide of mental illness and track down the causes of the disease itself. This lead to many different schools of thought, from the psychologists, neurologists and psychoanalysts, but none came to a solid agreement as to the root of the problem and the percentage of the population deemed insane continued to rise into the 20th century (Torrey and Miller 2001).

There has been a great deal of discussion among modern historians about whether this increase represents an actual increase in biological mental disturbance or a society that was becoming increasingly rigid and thus more likely to label any deviation "insane", even when it did not represent a mental disorder as the term is now understood (McGovern 1986). During the 1800s, the United States was as concerned as Europe about this rise, but was not on the cutting edge of treatment or philosophical understanding of the causes of insanity. As such, the main concern was to protect the public from the insane and the insane from mistreatment by the public rather than the creation of cures, although some treatments for symptoms did exist. It was in this climate that the Pueblo Insane Asylum was conceived and built.

History of the Colorado Insane Asylum

The Colorado Insane Asylum was established in October of 1879 in Pueblo, Colorado as the official place for those who were deemed mentally unfit and had nowhere else to go (Mitchell et al. 2002). Before the establishment of the asylum, the Colorado state government sent the insane to other states' asylums at what was reported to be great expense, or held in prisons where they were at risk of abuse from other inmates and could not receive care for their specific conditions (Colorado Weekly Chieftain 1879). Thus the asylum was developed as both a humanitarian enterprise and cost saving venture. It opened under superintendent Dr. Thombs, a former Civil War doctor who had a separate practice in the city of Pueblo. The institution started with 13 patients in the first year, 1879, and by 1899, the last year the cemetery was in use, there were over 400 patients, of which only about 40 were deemed curable (Mitchell et al. 2002). During the 20 year period from 1879 until Dr Thombs left in 1899, 1,938 people had been admitted to the asylum at some point. Of those, 506 died in the asylum in the first 20 years, 314 died later and 1,035 were released.

This number of patients created severe overcrowding as well as funding issues as it was estimated that each patient cost $300 a year to maintain (Mitchell et al. 2002). As with many state asylums in the United States during the period, there was not enough funding for new buildings or to purchase sufficient food. During the use of the hospital, labor provided by the inmates themselves was utilized to farm, raise a dairy herd and to plant and maintain orchards. To help offset the cost of care, the inmates who were well enough to do manual labor handled much of what could be done on the grounds, including construction of some of the buildings, creating and maintenance of the gardens and orchards, laundry and sewing of much of the clothing, even the strait jackets for other inmates. Underfunding was so severe that by the end of

Dr Thombs' tenure in 1899, the asylum was nearly $19,000 in debt. The commissioners investigating the asylum noted that "beyond a certain point, economy in the management of this institution is criminal. This class of people is the last class under the state's care for pinching and stringent economy to be practiced upon" (Mitchell et al. 2002).

Given the chronic funding issues, it is no surprise that a large number of burials occurred on the grounds. As most patients were neither local nor did they have the financial resources to pay for their own burials, the institution had to rely on what the state could provide. It is clear from the orderly nature of the burials in the cemetery and the fact that coffins were used, no matter how rudimentary, that the hospital was not trying to hide the deaths or the cemetery. Likely the issue was entirely economic. To bury on the grounds was of little expense since the labor to build the coffin and dig the grave could be handled by the inmates. To bury in the town cemetery, the asylum would have needed to hire someone to take the body into town, as is indicated in the budget did sometimes happen. In addition, the coffin had to be sturdy enough to make the trip and it was necessary to purchase plots in Roselawn Cemetery, the pauper cemetery for the city of Pueblo, at a cost of $5 per burial (Mitchell et al. 2002). The records for the asylum do not mention where the 506 people who died there during the period were interred. As death certificates for the state were not required until after 1900, there is no way to track who is among those in the asylum cemetery collection and who was interred elsewhere. Thus, it is not possible to tie any listed diagnosis from the intake records to any of the skeletons buried on the grounds, greatly limiting the amount of information that can be used from the contemporary records.

Theories of Insanity in the 1800s

The asylum was created in the framework of the society in which it existed and this is reflected in the patients they received and how they were treated. Although cemeteries on the grounds of asylums do not seem to be the norm for the period, in many other ways the asylum fits well within the medical establishment of the late 1800s. Any history of medicine must include the theories that drive its practice and ideas of mental illness and its causes are no different. People deemed insane in industrialized societies in the 18th, 19th and 20th centuries have received considerable attention from social historians in regards to the homogenization of society as a whole, how complex societies treat their most vulnerable members and how and why someone might be labeled deviant or insane. In particular, the evident astronomical rise in insanity in industrialized Canada, Europe and the United States has been widely studied from a number of perspectives (McGovern 1986, 1987; Sutton 1991; Torrey and Miller 2001). Just as with commentators concerned with the seeming alarming increase in the numbers of insane at the time, there is currently no agreement as to why insanity increased and how much is attributable to an increasingly homogenizing society, and how much of the increase represents a real rise in the number of people suffering mental ailments (McGovern 1987; Sutton 1991; Torrey and Miller 2001). The best way to examine the issue is to analyze both the changing philosophical understanding of insanity in the period as well as the medical realities they reflect.

Until the Enlightenment in the 17th and 18th centuries, beliefs about the root of insanity fell into two categories, the spiritual and the physical. The more generally popular and widely believed theory was that spirits, demons or a lack of connection with God caused objectionable conduct which included sexual deviancy, insanity, addiction and other such behaviors (Torrey and Miller 2001). A second school of medical thought, never as popular, but dating back to the

ancient Greeks, found that as with all physical ailments, mental issues arose from an imbalance of humors or fluids in the body. During the Enlightenment, both of these theories were largely supplanted in the medical profession with the idea that it was a flaw in a person's ability to reason that caused insanity rather than a spiritual or biological failing (Dwyer 1987; Rush 1835).

Insanity in the United States is largely absent until the 1750s. Before this period, the records indicate that insanity was exceptionally rare, especially compared to countries where most of the colonists originated (Torrey and Miller 2001). This is not entirely surprising since until the colonies became established, no infrastructure existed for the care of those unable to care for themselves and so afflicted individuals would not have been able to leave their home countries and make the arduous journey across the Atlantic. As the colonies became more established in the size of the population as well as their infrastructures, insanity became an increasing issue (Torrey and Miller 2001). Things started to change between the 1750s and 1800. Between the founding of the colonies and 1750, there is no record that the colonists felt the need for asylums or any need to care for the mentally ill. From 1750 to 1800 however, this became an issue of some concern with six of the 13 colonies considering the creation of mental institutions due to the rising number of insane individuals in their societies. Not surprisingly, during this period the first asylum in the country was built, constructed in Williamsburg in 1773. At the time, the insane seemed to be drawn largely from the upper classes and clergy, so insanity as a sign of moral failing was not a widespread belief as it would later become (Torrey and Miller 2001). In part for this reason, those deemed insane were kept at home with their families who had the means to take care of them and so there was less need for asylums than in later periods.

The actual cause of insanity was poorly understood at this time, so it was widely held that faulty reasoning was the primary cause of insanity, a philosophy that held through much of the 1800s (Dwyer 1987; Rush 1835; Torrey and Miller 2001). Among the many important members of society who felt reason to be the underlying issue in insanity was Benjamin Rush, who was both a popular author and a prominent psychiatrist at the beginning of the 1800s. His book, *Medical inquiries and observations upon the diseases of the mind*, was the first American book to cover the pathology of insanity. He describes insanity as:

> "By deraignment in understanding, I mean every departure of the mind in its perceptions, judgments, and reasonings, from its natural and habitual order; accompanied with corresponding actions. It differs from delirium, whether acute, or chronic, in being accompanied with departure from habitual order, in incoherent conduct, as well as conversation. The latter, however...is evinced by the correctness with which they sometimes perform certain mechanical and menial pieces of business. Madness is to delirium what walking in sleep is to dreaming. It is delirium, heightened and protracted by a more active and permanent stimulus upon the brain" (Rush 1830; 10).

The cause of insanity was thus neither supernatural nor the physical body, but rather the mind's ability to appropriately comprehend its surroundings. As reason was an inherent part of humanity, many of the sufferers of these delusions were considered treatable if they were given the right care to restore their ability to properly reason. Indeed, many of the asylums in the first part of the 1800s got their funding through grandiose claims of the money the government would save by removing the insane from alsmhouses where many were kept, treating their mental disturbances, and then returning them back into society as productive members (Dwyer 1987). Unfortunately, the conditions at the asylum were little better in many cases than the almshouses from which a large number of the inmates were drawn. Taxes were very low during the period and insanity seemed to rise faster than new facilities could be built, so asylums remained overcrowded and underfunded (Goodheart 2003; Sutton 1991; Torrey and Miller 2001).

This created a crisis that culminated with the crusade of Dorathea Dix in the 1840s to improve the conditions of the asylums. She traveled around the country touting both the need to restore human dignity to the insane and also the ability to cure them. The latter was based on amazing rates of curability claimed by many asylum superintendents such as Dr. Awl whose assertions were so grand he was facetiously knows as Dr. cure-Awl (Torrey and Miller 2001). These doctors achieved their exceptional results using the moral therapy invented in Europe by Philipe Pinel and William Tuke. Moral therapy worked under the premise that insanity was simply lost reason or misperception of reality which could be corrected. The unfortunate sufferer was to do normal human activities such as farming and taking care of the grounds to return to a normal mental state. The theory was that by acting like a human, they would regain that most basic human element, reason (Dwyer 1987; Torrey and Miller 2001). The insane were also prevented from reading or over stimulating their minds as this was considered the cause of a number of problems, especially depression (Torrey and Miller 2001). This perceived curability, economy and humanitarianism in handling of the insane created a wave of asylum building across the Eastern and to a lesser extent, the Southern United States that lasted until the Civil War.

After the war ended, it became increasingly clear that the previous optimism about the curability of insanity was misplaced and the numbers of insane were ever increasing while the number of insane who were deemed cured decreased. This, combined with scandals over living conditions, superintendents marrying former patients, financial mismanagement and in some cases the doctors being alcoholics or insane themselves, soured people on the idea of asylums and the moral cure more generally (McGovern 1987; Torrey and Miller 2001). The biggest blow

came not from the sensationalism of the misbehavior of a few, however, but in 1876 from a very public report by Pliney Earle about the dismal cure rates (Torrey and Miller 2001).

This loss of public trust opened the door to an entirely new group of doctors and theories about the causes and cures of insanity. The heads of the asylums were psychiatrists, largely trained in the US. The new group consisted of the neurologists who were trained in the latest science in Europe and brought it back to the United States. Both the psychiatrists and the neurologists vied for dominance in prestige, money and public acceptance throughout the 1870s and 1880s, publishing articles discouraging each other, taking different sides in major trials where insanity was in question and suing each other for libel. The neurologists were unable to unseat most of the psychiatrists already in power, but with help from the general public and the dramatic report from Nellie Bly in 1887 about the conditions of the asylums, the neurologists were able to supplant the psychiatrists in theory and methodology (Dwyer 1987; Torrey and Miller 2001). Much of this came from Charles Spitztka, a young member of the neurologist movement and one of their most prominent and vocal supporters. Spitzka complained that reliance on the moral treatment created a group of asylum superintendents who knew everything about the gardens, buildings and other activities improved by the moral treatment and nothing about the patients or insanity itself (Torrey and Miller 2001). His textbook on insanity was hugely influential. In it, Spitzka defined insanity as:

> "Insanity is a term applied to certain results of brain disease and defect which invalidate mental integrity...The disease manifesting mental deraignment may be an inflammation of the brain and its membranes, or a brain injury--a brain wasting, or an original brain defect--a mal-nutrition, or a reflex disturbance of that organ; and in such conditions must we seek for the groundwork of a scientific nomenclature and classification of the morbid state underlying mental deraignment" (Spitzka 1889; 18).

Insanity thus became reduced to the gross anatomy of the brain and nervous system and was attributable to lesions and physical degradation of these systems. Although other methods of understanding mental issues, such as psychotherapy, did develop at this time, the idea of physical abnormalities being the cause of insanity remained the dominant scientific explanation of insanity well into the 20th century. This switch away from a loss of reason and therefore humanity might seem to allow for more sympathy and respect for the mentally ill, as they were now perceived as being simply physically disabled in the same way as people with other ailments, but this was not the case. Instead, these physical abnormalities acquired a reputation as either the natural results of moral failings, such as venereal disease or alcoholism, or due to the degradation of race and fears that immigrants with a hereditary predisposition to mental disabilities were overrunning the country (Brandt 1985; Sutton 1991).

Asylum Demographics

Although the theories surrounding insanity and its causes changed dramatically during the 19th century, in many ways the realities for the insane did not. In particular, the conditions and demography of insanity changed very little over the course of the century. The most dramatic change happened during the early 1800s when insanity went from a problematic condition of the rich and respectable to the scourge of the poor. There is no clear reason why the rates of insanity rose so precipitously during the 19th century nor why the demographics shifted to over-represent the poor, but there are a few likely possibilities. The first is that there were medical improvements that extended life and allowed people who might have died from other physical conditions to live long enough to manifest their compromised mental condition (Torrey and Miller 2001). As medical improvements tend to start with the wealthy and move

down through society, this could explain the change in the demographics of insanity, which followed a similar path (Dwyer 1987; Torrey and Miller 2001).

Another possibility is immigration. Immigration during the majority of the 1800s in the United States consisted mainly of those where were not the middle class white Protestants who had previously made up most of the immigrant population joining the United States. Starting with the Irish in the 1840s and continuing through the 20th century, people from marginalized groups made their way to the United States in large numbers. Although many groups immigrated to the United States, it was the Catholics, poor, unskilled laborers and young adults with limited family connections who most raised the ire of the already established groups within the United States (Torrey and Miller 2001). Furthermore, once these questionable immigrants were in the United States, they tended to have far more children than the longer established, higher class individuals. This, combined with the emerging fields of social Darwinism and eugenics, lead to fears that the immigrants were likely to entirely overtake the proper people with the new, inferior stock prone to mental illness. As Irish immigrants gave way to those from Southern and Eastern Europe the specific people who were a danger to respectable society changed, but the niche they held and what they represented to the established members of society did not (Brandt 1985).

The overrepresentation of insanity among the poor immigrant groups is unlikely to have been entirely based on racism however; the demographics of who emigrated may also be a cause. Before the 1840s, mainly middle class families emigrated, a group who were not considered a threat to the established values and norms of American society. This changed when immigration shifted to poor, young individuals (Brandt 1985; Torrey and Miller 2001). Those without family connections generally had nowhere to turn for support, and if they were unable to

take care of themselves, relied on the welfare of the country rather than their kinsman, costing taxpayer money. The lack of family oversight that would normally restrain destructive behaviors might also have allowed for overindulgence in activities than can cause mental issues, such as alcoholism and fighting (McGovern 1987). Finally, there is the age of those who immigrated. Among the poor groups, it was predominantly young adults. A very large number of currently recognized mental conditions, such as schizophrenia, first manifest among young adults, so those coming over were in the stage of life most likely to first reveal compromised sanity (APA 2000; Torrey and Miller 2001).

Whatever the cause of the shifts in the class and age of the insane in America, it had profound effects on the perception of insanity. When confined to the higher classes, insanity was simply the lack of reason. When mental illness became a disease of the poor and disenfranchised it stopped being a novelty and started being a moral failing. After all, if the brain could misperceive the world badly enough to need institutionalization, what prevented it from misunderstanding morality as a whole or even being the root cause of poverty? This understanding was both because of the perceived link between moral failings and mental failings but also because in many cases the insane had nowhere else to go as they lacked the resources to get help from anywhere but the state.

Marginalized groups continued to make up a disproportionate contribution to the asylum population throughout the 1800s, particularly the Irish (McGovern 1987; Torrey and Miller 2001). Although there were a number of theories as to the Irish's supposed propensity to insanity, including their over-love of their homeland, fiery tempers and propensity to drink, the poverty and age of the Irish immigrants was likely the biggest contributor to their overrepresentation in asylums. Irish were not the only minority group considered particularly

prone to insanity. African Americans were thought to be sane while they were slaves and the group with the highest levels of insanity when free, but this was based mainly on the doctored census of 1840 by pro slavery factions. Due to segregation and racism, African Americans were rarely admitted to the asylums, and so were not an important demographic component of institution populations during the 1800s (Torrey and Miller 2001). Native Americans were also seen as more prone to insanity than white, civilized people, but for racial reasons and their very different beliefs about mental illness, they were rarely incarcerated in asylums (Torrey and Miller 2001). Bucking this trend were the Chinese, who had the lowest insanity rate of any group in the United States in the late 1800s and those that were deemed insane were rarely committed to asylums (Goodheart 2003; Torrey and Miller 2001). Whereas in the early part of the century, immigrants and marginalized groups were admitted to institutions in higher numbers because they were thought to lack the same abilities to reason, they remained a large part of asylum populations because their race was perceived as genetically inferior (Brandt 1985; McGovern 1986).

The Colorado asylum had a similar population demographic as United States asylums more generally. According to the 1880 census, the most detailed ever taken in the country in regards to mental illness, Colorado had a very low overall rate of insanity compared to the rest of the country. The rate for Colorado was 0.5 insane for every 100,000 people, on par with other frontier states with the exception of California, which had "the craziest people in the world" (Torrey and Miller 2001). The rate for California and New England was up to three insane for every 100,000 people (Torrey and Miller 2001). The Colorado asylum intake records help elaborate who entered the asylum and why, further breaking down the demographic and admission trends. Not including those whose admission cause was simply listed as unknown, the

most common reasons for commitment were hereditary with 273 cases reported, 266 with intemperance, 133 with syphilis and 98 with ill health, not including the more specific cases that included "ill health". In cases of ill health, abhorrent behavior might have been corrected when the physical cause was treated. Treatment of typhus for example, which was listed as an intake reason, removes the fever, hallucinations and ranting that likely caused the need for institutionalization (Mitchell et al. 2002).

The theories on the causes of insanity were not just split along temporal lines however, as there was also a component of sex, especially by the end of the 19th century. Although nothing was written specifically about the possible differences in the cause of insanity in men and women for Colorado specifically, the admission records do elucidate a number of admission reasons that were sex specific. For men, this was an overtaxing of the brain or using up their physical resources so that the brain did not have enough energy to function, or that it exacerbated existing physical abnormalities in the brain. For women, the majority of mental instability was believed to result from problems with their reproductive organ (Goodheart 2003; McGovern 1987; Sicherman 1985). The term "hysteria" which doctors of the period commonly used to refer female mental illness comes from the Greek work for uterus.

Although nothing was written specifically about the possible differences in causes of mental illness between men and women in the asylum papers, the admission records do elucidate a number of such cases (Mitchell et al. 2002). “Unknown” and “heredity” were listed as the main admission causes for women, but there were quite a few ailments that were female specific, all relating to the reproductive system. The largest group of these included those afflictions related to women who did not have a regular menstrual cycle. There are many causes of menstrual irregularity in women; some are simply due to factors such as weight, but menstrual

problems can also result from conditions that also effect mental state, such as hormonal problems and syphilis. As hormones were not understood, the suppression of the menses, menstrual irregularity, suppression and second climacteric were all believed to stem from insufficient energy as was a common problem for men (McGovern 1987). In this case, women were perceived to have fewer mental abilitis than men, so they over used them trying to keep up with modern society. In doing so, females overextended their bodies so much they did not have the strength or resources to menstruate (Goodheart 2003). William Hammond, one of the founders of the neurologists in America, wrote, "in some women it [menstruation] is very irregularly performed from the first, and this deraignment, when it exists, is a fruitful source of a great variety of nervous and debilitated conditions from which so many women of modern society suffer" (Hammond 1883, 105).

The next largest group of female specific ailments at the Colorado Insane Asylum were those with an admitting cause listed as puerperal conditions (Mitchell et al. 2002). The term puerperal referred to any ill health, both mental and physical, that occurred immediately after childbirth. Puerperal fever was a severe bacterial infection caused by insufficient sanitation among doctors or birth attendants delivering babies and lead to an alarmingly high mortality rate for both mothers and infants delivered in hospitals (Theriot 1989). As the cases in question were in a mental health facility and not a medical hospital, the puerperal state/mania conditions listed on the intake record are likely postpartum psychosis rather than bacterial infection. The 14 cases where childbirth was listed as the cause of insanity are likely also postpartum psychosis (McGovern 1987; Theriot 1989). It was seen as almost natural and expected for a woman to get some form of pregnancy-related mental condition. **"**The profound changes induced in the female organism by the condition of *pregnancy* could scarcely leave the mind untouched" (Hammond

1883; 114). The remainder of female specific cases at the asylum include seduction, abortion, ovarian disease and uterine disease (Painter et al. 2002). Abortion and seduction can reflect either a considered moral failing which resulted from insanity or could simply be physical problems that, due to their reproductive nature, were sent to the asylum.

Although men did find themselves admitted to the asylum for problems related to their sexuality, the Colorado Asylum records show that this was not considered the major catalyst for most mental breakdowns (Mitchell et al. 2002). Victorian society believed men had a much more robust constitution and so it took severe mental stress or physical exertion to bring on their mental disorders or create physical brain abnormalities. As men were considered more able to handle the rigors of modern life than their female counterparts, inability to accept or deal with financial adversity meant that the individual must be mentally unsound (Torrey and Miller 2001). This does bear out in the admission record where all those admitted for failed business and financial issues are male. Being physically overwhelmed to the point of mental instability was even more common in admission records than financial stress. Additionally, all the cases of ill health and exposure, exposure, overwork, sunstroke and all but one case of overwork and anxiety admitted were men. It should be noted that it may also be that these patients had physical issues and nowhere else to go so they were sent to the asylum as it took indigent cases that other hospitals and institutions of the time would not (Dwyer 1987; McGovern 1986; Torrey and Miller 2001).

One final point on sex differences is clear. Of the 266 cases of intemperance, 254 are men. Although roughly 75% of the asylum was male, in line with the population for Colorado more generally, 95% of the alcoholics were men (Mitchell et al. 2002). The difference is largely attributable to the different ways men and women self medicated during the period. For men,

drinking and visiting saloons and taverns was a major part of the social fabric, especially for the poor. Saloons were often called Poor Man's Clubs as a counter to the gentlemen's clubs wealthy men used to socialize. Not only did this make it more socially acceptable for men to drink, but when they overindulged, it was much more public and disruptive (Powers 1998). For women, drinking was largely taboo. Instead, respectable females took patent medicine that did not list ingredients but which usually contained high levels of alcohol and opiates such as morphine or laudanum (Shikes 1982; Torrey and Miller 2001). This meant that "drunk" or alcoholic behavior was more likely to be attributed to the woman's ailments that necessitated the medicine in the first place rather than the dubious content of the medication. As self-medicating was confined to the home, far fewer people were likely to see or report a woman and so she would not be institutionalized for it. Even in the cases where a woman was deemed insane due to her alcoholism, many doctors would be hesitant to publicly proclaim socially unacceptable behaviors on the part of their clients and so may have listed a different root cause for insanity (Brandt 1985; Shikes 1982; Smith and Brown 2001).

The Differential Diagnosis of Insanity

Although cultural constructions play a large part in who is deemed insane and how they are handled, there are certain commonalities in diagnosis and described symptoms that suggest at least some conditions transcend the place and time in which they were described. In the period of the asylum, the major categories of insanity were labeled somewhat differently from doctor to doctor as there was not an official diagnostic manual as is currently the case. However, by the end of the 1800s, the major groupings generally include monomania, mania, melancholia, dementia and paresis (Spitzka 1889). A comparison of the listed groups of mental disorders to

diagnostic criteria of the present would suggest a somatic basis for some mental conditions "if there is a strong neurobiological basis for 'delusion', then the name under which it has traveled throughout history or the concepts used to explain it or whether or not it has been considered a 'symptom' will matter little, for the behavior will persistently re-appear in a recognizable form in successive historical periods" (Berrios 1996; 11). Thus a comparison of the descriptions of symptoms from the Victorian period to current diagnostic standards can clarify which disorders more likely reflect social biases of the period and which represent real and enduring somatic issues.

Monomania first became widely known at the beginning of the 19th century. It was widely popular in literary and artistic circles for how unusual it was compared to previous known mental disturbances. By the middle of the 1800s it had lost much of its luster due to the perceived commonality of it (Torrey and Miller 2001). Due to the rather lofty literary heights it attained, monomania obtained many meanings in the popular imagination of the period. A stricter, medical definition of it is a

> "mental condition in which a single faculty or class of faculties or associations become diseased the mind generally remaining healthy…where there appears to be an incontrollable tendency to steal, to squander, to drink, to destroy, are of common occurrence and are supposed to be compatible with the exercise of intelligence and with the discharge of many of the ordinary duties of life…they [those with monomania] fail to detect the incongruities and absurdities with which they are associated or having detected the real character of these errors are unable or unwilling to cast them out or to disregard them" (Chambers and Chambers 1887; 535)

Although the term had largely fallen out of use by the 20th century, the issues described remained very much present. The *Diagnostic and Statistical Manual of Mental Disorders*, created by the American Psychiatric Association in 1958, is the most widely regarded and used book on mental disorders and their diagnosis in the United States (APA 2000). Although lacking

a category of monomania, they do have a listing for impulse-control disorders. In the latest edition, the DSM IV, describes these as "the failure to resist an impulse, drive, or temptation to perform an act that is harmful to the person or to others. For most of the disorders in this section, the individual feels an increasing sense of tension or arousal before committing the act and then experiences pleasure, gratification, or relief at the time of committing the act" (APA 2000). Although the language used is different, the description of the disorders is remarkably consistent. They both include the idea of someone who in most situations appears to fall within the normal bounds of mental understanding but in regards to a specific topic or idea, is unable to control their thoughts, impulses and actions relating to it, even when they are detrimental to the life of the afflicted. Although some of what might fall under the purview of monomania is broken into other diagnostoc classes in the modern period, kleptomania, pathological gambling, pyromania and a number of self-destructive behaviors all continue to be classed in this group, just as the compulsion to steal, to squander, and to destroy were listed as common expressions of monomania in 1889 (APA 2000; Berrios 1996; Chambers and Chambers 1889).

Although monomania waned in popularity as a diagnosis for mental problems, many others remained consistently diagnosed throughout the 19th century, including mania. In an era without any understanding of the causes of insanity and much debate and change in the way it was understood and classified, the easiest and most consistent way to comprehend mental troubles was by looking at the symptoms and how they manifested. Generally, this was done by which symptoms seemed the most powerful in the afflicted. In monomania, this was an uncontrolled obsession, in mania, an overexcitement or agitation (Berrios 1996). Spitzka (1889; 131) described it well when he said "mania is a form of insanity characterized by an exalted emotional state which is associated with a corresponding exaltation of other mental and nervous

functions. The typical condition of the maniac may be summarized in one phrase: loosening of the inhibitions or checks, both those of organic and those of mental life." The idea of an overreaction to stimulus to the point that it was detrimental to normal functioning is not confined to the 19th century. Any number of conditions, including the taking of some stimulants and some vitamin deficiencies can cause it, but the most common form now recognized is bipolar disorder, formally characterized as manic depressive for its alternating manic episodes (Berrios 1996). Indeed, according to the DSM, this manic state is the critical symptom for differentiating it from other mood affective disorders. In severe cases, the maniac can go days without sleeping, has an inability to remain focused and lacks all self-control, frequently leading to lavish spending or substance abuse (APA 2000). As such it correlates very well with the condition so succinctly described by Spitzka. Clearly, although mania fits well within certain cultural constructs of the time, such as women having manic or hysterical episodes due to overstimulation of their feeble minds, it also has a more enduring quality that strongly suggests a basis that transcends the culture in which it is diagnosed.

The opposite of mania was melancholia. In this case, it is best defined as:

> "a form of insanity whose essential and characteristic feature is a depressed i.e. subjectively arising painful emotional state which may be associated with a depression of other nervous functions. At its height the melancholic disorder is the antithesis of the maniacal. Just as every gesture and every thought of the maniac betrays his exalted emotional state so the attitude and expression of the melancholiac his thoughts and his delusions illusions and hallucinations if he have these announce the dominant emotion of sadness or psychical pain (Spitzka 1989; 140).

It is important to note that melancholia was not simply a case of someone being depressed, but a collection of symptoms that hampered their functioning in any meaningful way in society that are best characterized by their extremely depressive nature. That fact makes melancholia less clear as a direct modern diagnosis. Most likely, it combined people who

suffered from depression so severe that they were unable to properly care for themselves, but also the majority of cases of schizophrenia (Berrios 1996). Although people suffering under severe depression may have some level of delusion, it tends to be fairly minor and not severe enough to require institutionalization. In schizophrenia on the other hand, the delusions and hallucinations are the clearest diagnostic criteria (APA 2000). Not all cases of schizophrenia are so defined by depression, but the largest group, paranoid schizophrenics, does experience mental disturbances of a very negative nature. A much smaller subclass, the catatonic schizophrenic who is almost entirely unresponsive to outside stimulus, likely also would have fallen into the melancholic category (APA 2000).

The remaining forms of schizophrenia probably fell within the group of dementia. Although it is now defined almost exclusively in terms of aging, in the 1800s it simply meant any kind of mental deterioration (Berrios 1996). As schizophrenia is almost always asymptomatic until the late teens or early twenties, a sufferer would appear to go into a sudden cognitive decline that would best be described as young dementia at the time (APA 2004; Berrios 1996). Without any other overriding characteristic such as depression, this deterioration was in and of itself considered the most diagnostic symptom (Spitzka 1889). Such declines were generally seen as inevitable in the afflicted and were usually attributed to their heredity (Berrios 1996). Senile dementia, although remarkably unchanged in its descriptions between the 1800s and the present day, offered a very difficult philosophical conundrum for early 19th century doctors. With people living long enough to acquire dementia in any real number for the first time in the 19th century, the question of its inevitability became paramount (Berrios 1996). If a person was a rational being with a soul, as asserted in the early part of the century, then their basic nature should remain unchanged with time. If, however, the brain was subject to the same

ravages of time as the rest of the body, as the neurologists claimed, then such declines were not abnormal (Berrios 1996; Spitzka 1889). Although no consensus was reached about the meaning of its existence, the diagnostic criteria of senile dementia was agreed upon. It consisted mainly of a loss of short term memory and especially the ability to create new memories. If retroactive memory happened, it tended to lose the most recent events and slowly go backwards, nearly identical to modern descriptions of Alzheimer's disease today (APA 2000; Berrios 1996).

Unlike senile dementia which is remarkably similar in description across two centuries, paresis is the least likely class of diagnosis from the Victorian period to still be a cause of mental illness in the modern world. This reflects the remarkable advances in treatment and the understanding of disease far more than any change in beliefs about what constitutes insanity (Berrios 1996). Paresis is the loss of voluntary muscle control which can include tremors, paralysis and seizures (Spitzka 1889). There are a substantial number of conditions that can cause these symptoms, the most common of which in the 1800s were syphilis, some chemical exposures and epilepsy (Berrios 1996; Brandt 1985). Although syphilis can manifest in a variety of ways, one of the most common in its tertiary stage is neurosysphilis. So often was this the cause of paresis during the period that in many cases, including in the Colorado Insane Asylum, the two terms could be used interchangeably (Berrios 1996; Mitchell et al. 2002). As it is now treatable with antibiotics, neurosyphilitics were largely removed from the realm of the insane not due to changing societal norms, but to the wonders of modern medicine (Brandt 1996; Quetel 1990). A similar case can be said for epilepsy. With the discovery of Phenobarbital in 1919, there was, for the first time, an effective medication for treating seizures, removing the need for hospitalization of epileptics and allowing them to live normal lives outside asylums (Steele 2005).

Syphilis in the Victorian Era

Although sometimes diagnosed as a form of paresis, syphilis was such a dominating presence in 19th century psychiatry that it often remained separate from other classes of mental disorder. Syphilis, a venereal disease, is caused by spirochete bacteria in the *Treponoma* genus. Although there is still a great deal of debate about the origins of syphilis, the first reliable account of its existence comes from Naples in 1595. From there, it spread quickly through Europe and throughout the world shortly thereafter (Parascandola 2008). Despite many centuries of research since its appearance, little changed in the knowledge and understanding of syphilis until the mid-1800s at which point a number of developments in Europe helped to classify and better understand, although not treat, the disease.

The first was the separation of syphilis from gonorrhea which was the other major sexually transmitted disease of the period. This allowed the symptoms of syphilis to be more clearly noted and from that, the discovery of its different stages (Parascandola 2008; Quetel 1990). Before an understanding of germ theory and microbes, the latency period common in syphilis made its tertiary stage difficult to associate with the initial infection. After this was discovered, syphilis stopped being simply an unpleasant venereal disease and became a terrifying time bomb that could manifest years later in a panoply of ways, including deterioration of the brain or nervous system, neurosyphilis (Parascandola 2008; Quetel 1990). In the United States, this discovery in combination with the Civil War made syphilis both far more prevalent, and far more frightening than it had been previously and accounted for much of the concern over the rise in insanity during the period if not the rise itself (Parascandola 2008). Wars have a tendency to relax certain social restraints and move groups of people across large geographic distances. This,

combined with the female camp followers who often acted as prostitutes, was a perfect breeding ground for spreading a venereal disease (Parascandola 2008).

The situation in the later part of the century, when people moved West to look for gold, was little different than war time and this included the Colorado gold rush. The prospectors who came West were mostly unattached men, going to new locations with limited moral controls where the main contingent of women was prostitutes (Parascandola 2008; Steele 2005). It is not surprising then that there are well over 100 cases of syphilis listed on the intake records of the Pueblo Insane Asylum for the 1800s (Painter et al. 2002). As the tertiary stage of syphilis had a multitude of manifestations, and given the disrespectability associated with it, it is possible the number of inmates that could attribute their disease to syphilis may be a great deal higher than recorded.

Another of the ways syphilis may show up in the asylum is in the category of heredity, which was the single largest reason given for admission to the Colorado Insane Asylum in the 1800s. In this period before genetics was understood, heredity referred to any condition or trait that passed from parent to child. As such, although not genetic, congenital syphilis may often have been the root cause of hereditary admissions to the asylum as well (Parascandola 2008). When a pregnant mother is infected with syphilis, she can pass it to the child through the placenta, the birth canal or breast milk. There are a number of abnormalities that can result from congenital syphilis, not all of which were clearly attributable to syphilis in the days before the bacteria was discovered. In modern cases, congenital syphilis results in neurosyphilis in nearly one quarter of those infected (Woods 2009). As it can manifest anywhere from birth to roughly 20 years of age, it would be easy for doctors with no test for the syphilis bacteria to call

congenital syphilis a hereditary issue rather than recognize it as a result of the same processes that control venereal syphilis (Musher 1999).

Treatments of Mental Disorders in the 19th Century

Diagnosis of specific metal disorders, whatever their cause, became increasingly common as the century wore on. In the beginning of the 19th century, doctors felt insanity arose from a person's inability to reasonably and logically organize the pieces of information they took in. As this was the case regardless of the specific symptoms expressed, the treatment for mental disorders was equally homogenous. As insanity was divided into diagnostic categories towards the end of the 1800s, so too were the treatments for the conditions divided (Barrios 1996; Spitzka 1889; Torrey and Miller 2001).

In the period of initial asylum building in the early 1800s, moral therapy dominated the treatment options. Inmates were kept on a regular schedule of productive work. By doing the tasks of normal individuals, the hope was that the thought processes of the deranged would also be normalized. This also kept them from over stimulating their brains, which was widely held to overwhelm the brain and thus cause the breakdown in logical thought (Dwyer 1987; McGovern 1986). Although not very effective as a treatment in its own right, it did remove the inmates from either sitting idly all day or confinement as they would have experienced in the almshouses. In the Colorado asylum, treatment consisted of a mix of the older labor or moral therapy and the newer medical-based options. Dr. Thombs touted several benefits of the moral therapy for his patients, including exercise, training in self-sufficiency, time spent outdoors and a chance to restore normalcy of mind through normalcy of actions. On a more practical level it filled in

budget shortfalls by covering building, food production and household necessities (Mitchell et al. 2002).

The new treatments added to this labor were improvement of physical symptoms with healthy living and medicines. As with moral therapy, the medical treatments of the neurologists at the end of the 1800s existed mainly as a way to control symptoms and normalize behavior (Torrey and Miller 2001). It was hoped that by stimulating the depressed and calming the manic, chemicals could provide the relief nature had not (Hammond 1883; Spitzka 1889). Toward the end of the 1800s, there was remarkably little requirement for certification as a doctor, not only allowing the proliferation of patent medicine, but also a number of competing schools of thought as to the best way to treat ailments. Among the neurologists and most in the psychiatric profession, giving the afflicted something that would counter their symptoms was the dominant school of thought. However, most doctors borrowed from many paradigms, including the osteopaths who believed manipulation of the skeleton would promote self-healing, and homeopathy which saw treatments as being effective if they induced symptoms similar to the disease in healthy people (Steele 2005; Shikes 1986).

As cures for mental issues were in such short supply, most treatment for mental illness focused on controlling symptoms. The Colorado Insane Asylum did keep a list of these treatments in order to receive compensation for them, but did not leave any notes as to their amounts or use (Mitchell et al. 2002). As such, it is impossible to tell from the list of medicines alone what treatments went with any given condition nor what the philosophical basis was for using the treatments. Most likely, the superintendent of the asylum followed the neurologists' treatment theories if not the specific medications. This almost certainly involved giving calming drugs to the manic, stimulants to the melancholic and purges to the sufferers of paresis. Not only

would these be the most popular treatment methods during the period, but they would also have made the patients more manageable, which was always important in the overburdened asylum (Mitchell et al. 2002). Many of the medicines listed for the asylum were also likely to treat physical ailments that would not have been related to insanity, but would be unpleasant and in many cases dangerous to the sufferer. Treatment for insanity itself would generally have been broken down in much the same way as the diagnosis was. Since the causes of most complaints were not known, those that presented with similar symptoms were generally treated the same way.

In the case of mental illness, it was understood that the brain was the cause of the issues, but beyond that there was little to be done and most medical theorists had their own pet theories as to the specific way the brain was being affected. Hammond, leader of the neurologist movement in the United States, believed that blood flow to the brain was largely at issue. In particular, mania and other "excited" conditions were caused by congestion of blood in the brain while depression and catatonic conditions resulted from insufficient cranial blood flow (Hammond 1883). It is worth noting that there were four patients admitted to the asylum for brain congestion, so Dr. Thombs likely believed at least some of Hammond's work (Mitchell et al. 2002). Spitzka, the other leader of the neurologists, felt that lesions, inflammation and other physical abnormalities of the brain and nerves were to blame and advocated autopsies so at least the cause of insanity could be known, although this did little for treatment at the time (Spitzka 1889).

The other major theory of the causes of physical abnormalities in the brain evolved from the much earlier theory of humors and the balance of different fluids in the body (Quetel 1990). In an era before a clear understanding of contagion and microbes, many diseases were attributed

to toxins that built up in the system. Some of these may have been obvious, such as lead poisoning, but the etiology of others was less clear (Quetel 1990). The treatments advocated in these cases usually centered on draining the poisonous agent from the body, using purges and less often by the end of the 1800s, bleeding (Shikes 1986; Steele 2005). The medicine list from the asylum shows a mix of medications indicated by all of these schools of thought and mostly they were used interchangeably to best fit the specific case and symptoms. Alcohol appears in at least 21 medications on the list, either on its own or in the form of tinctures, and many of the patent medicines were high in alcohol as well. This does indicate that the asylum did not employ a large number of "teetotalers", who were common at the time, although they did have a high number of alcoholics on the intake records (McGovern 1988; Mitchell et al. 2002; Smith and Brown 2001). Alcohol was used externally as an antiseptic and internally as a tonic and stimulant. Tinctures were various substances, usually botanical, preserved in alcohol, and they would have been given based on the properties of said substance and not for the alcoholic content (Hammond 1883; Spitzka 1889).

Alcohol was certainly not the only depressant used at the institution and there were a number of popular sedatives that were available. Among the most common sedatives of the period were opiates, both on their own and in patent medicine. Laudanum, opium and various forms of morphine were effective both at calming inmates as well as inducing sleep, which maniacs frequently would not do otherwise (APA 2000; Mitchell et al. 2002). Another popular sleep drug was chloroform which also acted as a powerful anesthetic (Shikes 1986). Chloral hydrate was listed several times on the medication list, so it was likely a favorite of Dr. Thombs. It is an effective sedative, but only in small doses; in higher doses, it is a hypnotic and can induce hallucinations, so care had to be taken in its use so as not to make the patient more agitated.

However, it was also widely regarded as safer than chloroform and is not addicting like the opiates (Hammond 1883; Shikes 1986).

Various forms of bromide were also on the medication list and were considered safer than the opiates or the chloral hydrate, but also less effective in many cases, although Hammond felt bromide drew blood away from the brain, making it an ideal treatment for those in manic states where there was too much blood in head (Hammond 1883; Spitzka 1889). Although effective as a sedative, bromide was also the only effective treatment commonly used for epilepsy before phenobarbital became available in the early 1900s (Fox and Fox 1871; Steele 2005). As such, it is difficult to determine whether the various listings for bromide acted primarily as calming agents or to prevent seizures. Sedatives and sleep aids were almost certainly not entirely restricted to maniacs and may have been used in any cases where a patient needed to be controlled and mechanical restraints were not desirable. Strait jackets, special beds and other devices were fairly common in asylums of the period and straitjackets were used to some degree at Pueblo, but were also considered inhumane by many members of the general public, so a medicine-based solution may have been preferable in a number of cases (Dwyer 1986; Mitchell et al. 2002).

Just as the sedatives helped to reduce the responsiveness of the manic patients, stimulants could help draw out and animate the melancholic ones. For the depressive and catatonic patients, there were a large number of stimulants also on the list of medications from the asylum (Painter et al. 2002). Many of the stimulants used were botanicals that are still common in modern diets such as guarana and kola, which are both high in caffeine (Smith and Brown 2001). Although not as powerful as other stimulants, they are fairly harmless plant extracts that could safely be given with few concerns about poisoning or addiction. Another popular treatment for catatonic

and depressive conditions was digitalis. In high doses it is fatal, but in low ones it can improve blood flow and is still used as a heart medication today (Shikes 1986). Hammond felt it improved blood flow to the brain while Spitzka felt digitalis reduced inflammation, so it had wide support, even if the reasons for it supposed effectiveness were unclear (Hammond 1883; Spitzka 1889).

In cases where a stronger treatment was called for, there were a number of options now considered highly unsafe. Cocaine was present on the medicine list as well and was very popular in Europe at the time, both as a treatment for depression and as an aid to psychoanalysis. The highly addictive nature of the drug was only starting to become widely known during the period and so there were few restrictions on its use (Quetel 1990). Unlike cocaine, the highly poisonous nature of strychnine was well known in the 1800s, but it remained a very popular treatment option where other medications were deemed insufficient. Strychnine shows up several times and in a number of forms on the list of medications. It is very toxic and causes severe seizures and muscle contractions, but as catatonic patients often did not move for days at a time, strychnine's ability to force muscle contractions made it the most desirable treatment for their condition (Hammond 1883; Spitzka 1889). Unlike in mania, poor blood flow was thought to cause inflammation or congestion and was often blamed for melancholic conditions, so purges of any kind were rarely recommended for melancholia (Hammond 1883; Spitzka 1889; Steele 2005).

Unlike disorders with a seemingly clear etiology and presentation of symptoms, monomania was not considered treatable by medications. There does not seem to have been clear agreement on the type or nature of treatment and most likely, treatment varied widely based on the specific obsession of the monomaniac (Berrios 1996; Hammond 1883; Spitzka 1889). In

cases where they were destructive, sedatives, physical restraints and other methods to protect the patients from themselves were likely the most common course of action (Dwyer 1987). There was a great issue with patients who did not eat and were somewhat obsessed with not doing so and this can be a manifestation of impulse control disorders (APA 2000). In these cases, meat broths and feeding tubes were the most popular remedies and purges were never indicated (Hammond 1883; Spitzka 1889). As the majority of the times sufferers of monomania were fairly functional, they were likely only medicated when there was concern about their specific obsession, and not at all times.

Monomania was not the only diagnostic group that was difficult to treat as paresis could arise from a number of disparate causes. The treatment options for paresis where based mainly on whether the doctor thought the condition was caused by syphilis or something else (Hammond 1883; Quetel 1990; Spitzka 1889). Until the end of the 19th century, there was a great deal of debate about whether syphilis should be treated. Many, even in the medical profession, felt that it was punishment for the sexual sins of the afflicted and that treating it would thwart god's will (Brandt 1985). Towards the later part of the 1800s however, it became increasingly clear that while many suffers had received it from illicit sexual contact, many more were the moral wives and children of problematic men and were as such innocent victims of the disease (Brandt 1985). Not only did it legitimize the condition to an extent, it also offered more avenues for treatment and more legitimacy to those who dealt with the disease.

Since its appearance centuries earlier, the main treatment for syphilis was mercury. It could be applied to skin in hopes of removing the lesions and therefore the disease, it could be vaporized and inhaled or it could be given as a tonic (Quetel 1990; Rasmussen et al. 2008; Tucker 2007). The popularity of mercury waned substantially during the first part of the 1800s

with a school of thought that blamed inflammation for the symptoms (Quetel 1990). However, with the fall in popularity of this paradigm and the discovery of new forms of mercury in the 1860s, it regained much of its dominance as a treatment (Quetel 1990). By this point the dangers of inhaling mercury were better known, so most treatments were cutanous and a few were tonics, but vaporized forms were no longer acceptable (Quetel 1990, Shikes 1986). Another popular option was to purge the body in hopes of draining out the toxin responsible (Quetel 1990, Steele 2005). One final remedy that was often popular was potassium. It was first recommended in France in the early 1800s and caught on in the United States after it was highly recommended by Hammond, who felt it was superior to either mercury or arsenic as a treatment (Hammond 1883; Quetel 1990). Arsenic was used occasionally in the period, but did not gain supremacy as a syphilis treatment until the discovery of Salvarsan in 1910 (Brandt 1985).

In cases where paresis was not believed to have been caused by syphilis, either because the doctor, such as Spitzka, did not believe there was a link between the two conditions or because another cause was known, the treatments varied. In these cases, the disease was treated as epilepsy, toxin-based or by the other symptoms. There were few effective treatments for epilepsy in the 19th century, although bromide was somewhat helpful (Hammond 1883; Shikes 1986). When the suspected cause was an outside toxin or inflammation was the suspected agent, purging the body to remove the poison was considered the best option (Spitzka 1889; Steele 2005). If none of these cases fit, then the sufferer was treated for their other symptoms such as catatonia, mania, etc. according to the doctrines of that diagnosis (Spitzka 1889).

The history of insanity does provide valuable context for understanding the Colorado Insane asylum collection, but the biology of mental illness is also critical to properly interpreting any results obtained from specific collections. Understanding what conditions were present, how

they were understood and what treatments were used defines what irregularities should be searched for in the skeletal collection and to inform what those irregularities might mean.

Chapter III: The Biology and Contamination of Human Bone

The treatments, theories and history of insanity are critical for understanding its nature in a skeletal collection such as that from the Colorado Insane Asylum, but the history itself cannot fully account for all the biological realities of the patients' lives or conditions. Although the majority of mental disturbances still recognized today do not leave any trace in skeletal remains, this is not universally true. Cranial abnormalities, syphilitic lesions and the chemical composition of the bones themselves are physical manifestations of mental conditions that can leave traces osteologically.

Bone Structure

Bone as a biological structure is largely unique in its ability to resist decomposition when the rest of the human body usually does not. Although not a full accounting of the life or biology of the individual to which they once belonged, bones can reflect some of the biological realities of the individual due to their unique design. Bone serves several functions, including acting as the structure for the body, as a source of protection for other vital organs, to create blood cells and some fat cells as well as to maintain chemical homeostasis, such as for calcium. To do this, bone has a unique combination of strength and flexibility stemming from its structure and combination of organic and mineral components.

Bone contains trace elements and minerals, but is composed primarily of a combination of collagen and hydroxyapatite, which is crystallized calcium phosphate (Ortner 2003; White and Folkens 2005). The bone matrix, which is the osteological structure, is built out of collagen with hydroxyapatite embedded within it. To build the matrix, osteoblast cells create osteoids which are collegen rich, calcium free cells. The osteoids attract hydroxyapatite which calcifies the cell.

Once an osteoid is fully calcified, it is an osteocycte, which maintains the bone cells. To maintain itself, allow for sufficient blood flow, maintain calcium homeostasis of the body and to respond to stimulus such as disease and injury, the bone is constantly changing and rebuilding (Ortner 2003; White and Folkens 2005). Although the same materials make up the basis of all the bone, it is not all arranged in the same manner. The cortical bone is the dense outer layer which has tightly packed osteocytes and protects the inner portion, which is made up of cancelous bone. Cancelous or trabecular bone is spongy which allows for improved blood flow compared to the cortical bone and more easily enables the creation of cells such as white blood cells (Ortner 2003; White and Folkens 2005). Because of its vital importance, bone has the ability to heal itself in response to injury. In cases of damage to the bone, such as a traumatic injury or infection, osteoblasts are signaled to rebuild bone on the site of damage. In order to repair the damage quickly, the new bone laid down is much more disorganized in structure than the normal bone, giving it a woven appearance. With time, the woven bone will be converted to more structurally sound lamellar bone (Ortner 2003; White and Folkens 2005).

As with any biological function, there are variations and imperfections in the system which can leave unique traces in the bone. These traces are chemical elements and metals which are not considered normal parts of the bone matrix. The presence of any elements aside from calcium and phosphate in the bone is considered abnormal, but not necessarily pathological. The incorporation of other elements into the bone reflects the presence of that element in the body at the time a section of bone was rebuilding itself. Since this process is constantly occurring to maintain bone integrity, a partial record of the contaminants within the body at any given time can be preserved. The record is only partial as not all elements can be incorporated into the bone and the relative levels of any element in bone will not reflect exactly how much was in the blood

stream due to variables such as the particular element, the form it was in, the way it entered the body or the age of the individual when they were exposed. Despite these shortcomings, the amount of any given element in the skeleton can be at least somewhat indicative of the elements present in life and the levels of the elements an individual experienced. Although a large number of elements can make their way into the bone structure those that mimic calcium or phosphate chemically or those that have an affinity for one of those elements are the most likely to be absorbed (Lambert et al. 1985).

Bone chemistry is not the only thing that is reflected osteologically, as changes that affect the body more generally may also appear in the skeleton. Many diseases, both bacterial and viral, have the ability to cause changes to bone tissue and leave a lasting mark. These infections rarely affect the bone exclusively and in most cases diseases leave no trace on bone at all. This can happen because the infected person died or fought off the disease before it was able to have an impact on the bone. It can also be that the condition an individual suffered from did not enter the bone as most diseases do not (Ortner 2003). In cases where there is osteological involvement, osteitis, the inflammation of the bone due to a pathogen, results. In many cases, these inflammations are non-specific and cannot be attributed to a specific pathogen, although some, such as syphilis, result in distinct lesion patterns making it possible to diagnose the disease. Other bone modifications, such as osteoporosis or its opposite, hyperostosis, can be caused by a number of agents, such as genetic predisposition, which may not have other effects on the body, or hormonal imbalances, which usually manifest with a number of other symptoms. Finally, there are skeletal changes that do not have a clear cause and may not be malignant at all, including many tumors of the bone (Larsen 1999).

Cranial Pathologies

Among the modifications to the skeleton than can be benign or pathological are cranial changes such as increased bone density and thickened areas. There are a large number of conditions that can cause this to happen to the skeleton as a whole, but very few that would modify the skull while leaving the rest of the skeleton untouched (Ortner 2003). Of these cranial conditions, the majority are genetic and quite rare. In a collection as small as the Colorado Insane Asylum, having several individuals with the same pathology likely reflects environmental conditions as rare genetic conditions would not appear multiple times among genetically unrelated individuals unless the condition had behavioral components causing the sufferers to be institutionalized. There are a number of conditions that are either environmental or show a mix of environmental and genetic components and are therefore more likely to be found in the asylum collection.

The most common bone disease to combine a genetic and environmental trigger is Paget's disease or osteitis deformans (Ortner 2003; Ralston 2008). Paget's disease is a fairly common disorder affecting between 1-3% of people over 40 years of age of European descent (Ortner 2003; Ralston 2008). It is more common in men and increases in prevalence with age (Ortner 2003). There is a great deal of debate about the exact cause of the condition, but development of Paget's disease seems to require both a genetic predisposition and an environmental trigger (Ortner 2003; Ralston 2008). There are a number of gene alterations that could be responsible for the disease, which seem particularly tied to British ancestry (Ralston 2008). The environmental trigger is less clear, with the most common suggestions being a virus or nutritional deficits (Ortner 2003; Ralston 2008). Paget's disease generally only afflicts a

single bone or adjacent bones with no clear reason as to why the particular bone is the focus of the disease, but which may be tied to injury or stress (Ortner 2003; Ralston 2008).

Whatever the etiology, Paget's disease develops in three stages which leave distinct marks on the bone. In the first stage, the osteoclasts that break down bone become overactive, thinning the bone and weakening it. In the second stage, the osteoblasts respond to the injury by creating new woven bone in the location where it had been weakened. This new bone tends to be lower in hydroxyapatite than normal bone. Finally, the osteoblasts continue to build and rebuild the bone, making it much thicker than before the initial damage but also structurally less sound due to the demineralization and the structural weakness of woven bone (Ortner 2003; Poncelet 1999). The bone is thus very distinct in Paget's disease, being both unusually thick and covered in a mosaic pattern on the surface from the different additions of woven bone. Another thing that separates Paget's from most other osteological disorders is that it never afflicts the whole skeleton and in most cases affects only one bone (Ortner 2003). It is mainly confined to the axial skeleton and affects the skull in 42% of cases (Ortner 2003; Poncelet 1999). When in the cranium, it can cross suture lines easily, is usually confined to the interior surface of the cranial vault and not the exterior of the skull and rarely affects the facial bones, leaving a skull that appears "normal" without detailed examination (Ortner 2003).

Although Paget's disease is osteological, in as many as 10% of the cases, there is also a neurological component as well (Poncelet 1999). One of the less common genes responsible for Paget's disease is also commonly associated with myelopathy and frontal lobe dementia (Poncelet 1999; Ralston 2008). Even patients without the genetic causes of Paget's disease that is specifically tied to frontal lobe dementia, there are a number of ways in which the mental faculties can be affected (Poncelet 1999). The most common seem to come from micro-fractures

and collapsing of the skull base. This can restrict blood flow, put pressure on the nerves and spinal cord and block drainage of spinal fluid, causing hydrocephalus (Poncelet 1999). The bone overgrowth in Paget's disease can also reduce the cranial capacity, putting pressure on the brain itself. In these cases and in hydrocephalus, the effects most resemble dementia with a loss of memory, inability to create new memories and antipathy (Poncelet 1999). In the cases where the collapse of the base of the skull puts pressure on the spine, idiopathic Parkinson's disease, uncontrollable muscle spasms or paraplegia may result (Poncelet 1999; Ralston 2008). In rare cases where the increased growth of Paget's disease puts pressure on the cerebral hemispheres, epilepsy has been noted (Poncelet 1999). Also uncommon is ossification of the dura mater, which decreases cranial capacity and potentially damages the brain structure. Finally, the amount of blood needed to maintain the excessive amounts of bone may draw blood away from adjacent organs that also need it, which, in combination with the reduced blood flow that can be a result of micro-fractures, can also cause a host of mental problems (Poncelet 1999). Thus, although Paget's disease is clearly osteological, its presence in a skeleton can also be a good indicator of potential mental problem on the part of the afflicted.

Although fairly common, Paget's disease is by no means the only condition that regularly causes cranial hyperostosis. Another condition to cause increased cranial bone thickness is *hyperostosis frontal interna* in which the internal section of the frontal bone is thickened and in some cases denser than unafflicted bone. It can be confined to a small area of the frontal bone or extend all the way around the parietals and into the occipital bone, although the latter is very rare (Barber et al. 1997; Hershkovitz et al. 1999). Generally, it is fairly uniform with a billowing appearance, but it can also have larger growths on the frontal bone. In cases where it does extend beyond the lambdoid suture, the thickening is most pronounced around the frontal bone

and decreases smoothly towards the posterior of the cranium (Hershkovitz et al. 1999; May et al. 2010).

There is some debate about the exact stages for *hyperostosis frontal interna*, but generally four levels are agreed upon. The lowest level involves a small growth on the interior frontal bone, usually bilateral but not always so. It is generally smooth in shape and size without nodules. It may be difficult or impossible to see in x-rays and only close analysis of the bone or CT scans will reveal its presence (Barber et al. 1997; Hershkovitz et al. 1999). The second stage involves a growth with a much larger surface area but which does not extend past the frontal bone. It may have a few ridges or nodules but they are uncommon at this stage. The third stage tends to show a billowing appearance on the internal bone surface and can extend past the frontal bone to the parietals. The final and most severe stage involves a large, domed area, often extending past the frontal bone that is bilateral and can double the thickness of the frontal bone (Barber et al. 1997; Hershkovitz et al. 1999). *Hyperostosis frontal interna* is not the only condition that can cause thickening of the frontal bone, but is different from other hyperostoses of the skull in a few critical ways, no matter the stage. The appearance is smooth, never porous, and the bone itself does not usually appear pathological in texture as is the case with cancerous lesions. It must involve the frontal bone which is also the location of the greatest severity. The edges are smooth and usually dome like, not the irregular edges found in many other ostomas. Finally, there is no growth or change on the external cranial bone (Barber et al. 1997; Hershkovitz et al. 1999; May et al. 2010). Although the bone increases in thickness, the density of it remains the same.

In addition to the appearance of *hyperostosis frontal interna*, the age and sex of those who have the condition are very specific, making it easier to diagnose, if the age and sex of the

skeleton are known. It becomes more common with increasing age, peaking around age 60, and is far more common in women than men, at a ratio of anywhere from 9:1 to as high as 100:1 (Barber et al. 1997; Ortner 2003). As the cranium generally thins with age, any thickening of it is an indication of something outside of the expected norm (May et al. 2010). Estimates run as high as 40% of women over 40 years old in the modern world having *hyperostosis frontal interna* (Hershkovitz et al. 1999; May et al. 2010).

There is a great deal of debate whether this rate of occurrence is part of normal aging and has remained consistent through time or if it is a reflection of something in modern society. Most studies of *hyperostosis frontal interna* in skeletal collections from earlier periods do show much lower occurrence which is attributable to three possible scenarios. The first is that as it is age dependent, *hyperostosis frontal interna* will only show up in collections with a reasonable number of elderly individuals, something which is less likely in past periods where there were generally shorter life spans (Larsen 1999). Another possibility is that the levels of *hyperostosis frontal interna* are the same, but the methods of examining the condition in archaeological collections makes it much harder to find minor cases than can be diagnosed with CAT scans as in most modern cases (Barber et al. 1997). Finally, *hyperostosis frontal interna* may be caused by some contaminant or condition that has become more common in the modern era that remains as yet unidentified.

Although there are a number of possible causes for *hyperostosis fronal interna*, the most likely cause is hormonal. If this is true, the hormone responsible has not been isolated nor is there agreement whether it is even pathological (Nikolic et al. 2010; Phillips 2001). The high frequency of the condition in modern society would seem to indicate *hyperostosis fronal interna* is not pathological, but there is evidence to the contrary and at the time of the Colorado Insane

Asylum, doctors believed *hyperostosis frontal interna* was not only pathological, but tied specifically to insanity. Although the exact causal mechanism was unknown, it was theorized that the brains of the insane atrophied and shrank; the body compensated by filling in the area with bone and the condition was most common among the senile insane and epileptics (Lewis 1889; Stewart 1928). Although this explanation is no longer favored, many of the observations about the links between *hyperostosis frontal interna* and insanity remain valid. Hawkins and Martin (1965) found that x-rays of mental patients were twice as likely to have *hyperostosis frontal interna* as compared to non-mental hospital patients when adjusted for age and sex.

Hyperostosis frontal interna has also been found in much higher levels in one other asylum from roughly the same period as the Colorado hospital. Phillips (2001) examined roughly 100 burials from a cemetery from the asylum used between the 1880s and 1896 in Oneida County, New York and found that the average thickness of the frontal bones was somewhat higher than would be expected for a normal collection. Thus, even in a collection from the Victorian period examined visually, there appears to be a higher number of those with the condition than among the crania of people not suspected of having mental issues.

A number of conditions likely account for all the manifestations of hyperostosis frontal interna. One of the most cited, including by Phillips (2001) in the discussion of the Oneida collection, is Morgagni-Morel-Stewart syndrome, or metabolic craniopathy, a hormonal disorder that is at least partially genetic and afflicts mainly older women. In addition to the *hyperostosis frontal interna*, it is also tied to hirsutism, obesity and mental disruptions including dementia and seizures (Hershkovitz et al. 1999; Phillips 2001). This mental component does

make it a highly credible explanation for unusually high levels of *hyperostosis frontal interna* in a mental hospital collection.

Not all cranial thickening has the characteristic appearance of *hyperostosis frontal interna* and as genetic disorders are unlikely to account for multiple cases with similar appearances in the Colorado Insane Asylum, environmental factors must also be examined. In particular, there are very few conditions or substances that can cause both thickening and an increased density of the bone. Among these is a class of anticonvulsants in the hydantoin family (Kattan 1970; Lefebvre et al. 1972). The most popular of these medications, Dilantin, or diphenylhydantoin, is well recorded as causing both an increase in skull density, but also in some cases, an extreme thickening of the cranium as well (Kattan 1970; Lefebvre et al. 1972). As an anti-epileptic drug, it would seem very possible that Dilantin might have been administered to the patients at the asylum, but the formula was patented by Heinrich Biltz in1908 and not used as a medication until much later (Lefebvre et al. 1972). However, hydantoin had been known to science since at least 1867 (Miller 1867) so it is possible it was used as a medication. It does not appear specifically on the list of medications for the asylum, but there were a few medications that did not appear on the list and were only recorded in the journal Dr. Thombs kept (Mitchell et al. 2002). It is also possible hydantoin was on the list and under another name that is now considered archaic and no longer applied to medicines in the hydantoin group.

Syphilitic Pathology of the Bone

Syphilis is best known for its affliction of the skin and brain, but it can also attack bones. While there are a number of possible causes of cranial hyperostosis, other distinct skeletal alterations, such as caries sicca, are confined to a known cause (Larsen 1999). Syphilis has a

very wide set of possible manifestations that made it somewhat difficult to diagnose, particularly in the tertiary stage, before the bacterium responsible was isolated in 1905 (Parascandola 2008). Before this discovery, the multitude of symptoms of tertiary syphilis meant that many cases were almost certainly mistaken for other diseases, including diagnoses of patients in the asylum. This would have given rise to different treatments which might also have affected the seeming side effects of syphilis (Ortner 2003). For this reason, it is nearly impossible to accurately trace the prevalence of syphilis in the 1800s nor all its likely presentations in the era before effective treatments.

Although a number of specific factors and possibly even symptoms may have changed over the years, much of the basic presentation of syphilis did not. The descriptions that linked the first two stages of the disease to the tertiary stage are remarkably similar to modern understanding of the symptoms (Quetel 1990). The first stage of syphilitic infection occurs in the days following exposure and manifest as skin lesions that last for several weeks before disappearing. It is painless and is usually on the genitals. At this stage the infection has not spread systemically through the body (Ortner 2003). For this reason, one common treatment during the Victorian period was the cauterizing of the chancre in hopes of removing the disease before it spread, but this was more often simply disfiguring rather than effective (Brandt 1985). The second stage marks the transition from a localized infection to a systemic one and is characterized by rashes on large sections of the body (Ortner 2003).

After this point, the infection goes into a latency period which can last several years. In many individuals this is the last stage and tertiary symptoms never develop. This tertiary period made it very difficult to link the initial stages of syphilis to the varied symptoms of the final stage and many doctors remained unconvinced of the link between the tertiary stages and

venereal syphilis until scientists definitively isolated the pathogen from the brain of paresis sufferers (Parascandola 2008; Quetel 1990). The final, tertiary stage of syphilis usually manifests with the onset of symptoms 2-10 years after the initial infection although this can vary from one year to over 50 after the initial infection (Ortner 2003). Although not entirely discrete categories, the tertiary stage of syphilis is generally classed as asymptomatic, cardiovascular, neurosyphilis or gummatous, or late benign syphilis (Gjestland 1955). Cardiovascular syphilis generally manifests as damage to the heart and commonly coexists with neurosyphilis.

To further complicate the matter, even within neurosyphilis there are a number of presentations of the disease depending on the structures infected by the syphilis bacteria and the severity of the infection. Many cases are asymptomatic, but of those that do present with symptoms, meningeal, paretic and dorsic are the possible forms (Swartz et al. 1999). All neurosyphilis starts as meningeal where the spirochetes get into the meninges of the brain. Once there, the bacteria cause inflammation of the tissue surrounding the blood vessels and the lack of blood and nutrients to adjacent structures results in cell necrosis (Swartz et al. 1999). Stiff neck, nausea and severe headaches are the most common symptoms, but blindness and hearing loss are also reported (Swartz et al. 1999).

In some cases, the bacteria spread further into the brain. If the syphilis spirochetes spread into the cortex and destroy the neurons, then paresis is the common result. In the early stages of paresis, there is memory loss, personality changes and headaches. If left untreated, these degenerate into dementia, confusion, occasionally mania or depression and paralysis (Swartz et al. 1999). The final presentation of neurosyphilis is Tabes Doralis in which the spinal cord itself is affected. In these cases extreme pain, trouble with locomotion, especially walking, and incontinence result (Swartz et al. 1999). It is common to have more than one form of

neurosyphilis and so in many cases the symptoms overlap and do not divide into discrete categories.

The final form of syphilis, gummatous or late benign, rarely overlaps with neurosyphilis. However it is possible, generally only when gummatous lesions form inside the brain itself with the symptoms being based on the size and location of the growth (Gjestland 1955). For the most part, gummatous syphilis affects the bones and skin of the sufferer, although the lesions can manifest in any part of the body. A gumma is an area of necrotic tissue that causes inflammation in the surrounding flesh; it is unique to syphilis and so makes it a valuable diagnostic tool in osteological studies (Ortner 2003). When on the bones, gummas form most commonly on the tibia and cranial vault, especially the frontal bone and the nasal bones. The unique mark they leave is called caries sicca which are small grooves formed by the vascular involvement of the gumma with a smooth, often raised center from the reactivity of the bone to the gummatous lesion (Ortner 2003). These occur in 1.5-10% of cases of untreated tertiary syphilis.

Other forms of syphilis also leave bone lesions, but they are not distinct to syphilis in the way caries sicca are. The most common of these non-distinct lesions is bilateral osteomylities of the posterior of the tibiae creating "saber shins" (Ortner 2003). Other lesions of syphilis are similar to those found in tuberculosis and leprosy. However, these syphilitic lesions affect different bones and over stimulate the osteoblasts, creating bone re-growth not seen in most other bacterial infections (Larsen 1999; Ortner 2003). The intake records from the Colorado Insane Asylum also indicate that syphilis was likely the most common form of bacterial infection among the inmates based on admission and death records.

To see if syphilis was indeed present among the asylum population, Leslie Johnson (2009) evaluated the skeletons using the diagnostic criteria put forth by Hackett (1965). She found no

caries sicca and only one case with definitive signs of syphilis, in skeleton A4. Four skeletons all showed lesion patterns that strongly suggested the individual had syphilis, but which did not meet the criteria for certainty. Ten individuals also had clear signs of an infection that was consistent with syphilis, but could also be due to a number of other conditions (Johnson 2009).

Chemical Composition of Bone

Disease is not the only factor that can influence bone and reflect the stresses and dangers of everyday life. The bone can also be a repository of elements that although not normally part of bone composition were drawn out of the blood stream and incorporated into the bone matrix. This process can be of great importance in understanding the life of the person to whom a skeleton belonged as it gives some measure of the contaminants to which they were exposed. Of particular interest are those elements that in large doses can cause symptoms in the body that mimic those that Victorian society deemed indicative of insanity such as seizures, memory loss and extreme emotional disturbances. Although in high enough doses almost any element can cause some of these symptoms, the elements of particular significance are those which regularly produce these symptoms and were common enough in Victorian Colorado for the asylum population to have had a high likelihood of exposure. The elements most likely to be absorbed by bone, produce insanity and be found in Colorado at the time are arsenic, copper, lead, manganese, mercury and zinc.

Elemental Exposure Routes in Victorian Colorado

During the late 19th century, there were a number of pathways through which contaminants and heavy metals could enter the body and therefore the bone. In 1859, small

amounts of gold were found in Cherry Creek, near what is now Denver. As news trickled east, it started a gold rush to the state. The little gold in Cherry Creek was quickly played out, but veteran miners soon realized that the real wealth was up in the mountains and it is here that the bonanzas were found. In the frantic pace of gold digging, silver was also discovered in large quantities (Smith 2009). Most mining in Colorado was "hard rock" during this period, rather than soft rock, which was usually coal based. Hard rock mining involved placing charges in the rock and once blown, the miners would dig through the rubble to remove the profitable materials (Smith 2009). The mines during this period had almost no ventilation and so whatever was in the rocks also tended to be in the lungs of the miners. Silicosis was the major concern, but breathing the dust is also a good vector for the introduction of heavy metals into the body (Derikson 1988; Shikes 1986).

Even once the minerals were extracted from the mine, there were various routes of exposure to heavy minerals. In gold mining, hydraulic systems were the most common way to extract the precious mineral from its less valuable surroundings. The rock coming out of the mine was broken down into small chunks and mercury was added, which would amalgamate with the gold, but not the other, less valuable materials (Smith 2009). This mixture was flushed through sluices into troughs that captured only the amalgamate. As much as 10-30% of the mercury added to the water flowed downstream (Bowie 1905). There were no filters so any material not considered precious enough to save entered the water system, exposing anyone downstream. This was particularly problematic in Colorado since the mines tended to be at higher altitudes than the settlements, which put everyone downstream and thus in the path of the contaminated water (Derikson 1988; Neubert et al. 2011; Smith 2009). Therefore, elements that can easily be absorbed through ingestion would be high not only among the miners but also

anyone who was in close proximity to the mines. Alternatively, those elements that are not well absorbed through the intestinal tract but through touch or inhalation would be notably higher in the miners.

The one exception to the problem of mine tailings in the water was the city of Denver. As the capital and largest city in Colorado, it was also the county from which the majority of the patients at the state hospital in Pueblo were admitted. The water in Denver was mainly drawn from the Deep Rock well, an underground aquifer with limited exposure to the surface run-off from the mines and other sources (Shikes 1986). Until 1883, water in Denver was delivered by the Deep Rock Company, but this started to change with the laying of water pipes in 1883. Pipes of the period had high quantities of lead, so although mining was not a contamination problem in Denver per se, the water likely still had some dangerous elements (Emsley 2001; Shikes 1986).

Mining was not the last stage in the metal extraction process nor was it the only stage if metal processing where individuals not directly involved could be exposed to the metals. The final stage of metal processing was smelting to remove imperfections in the ore. This meant burning off non-precious elements that might still be combined with those of commercial value. This made a particularly toxic smoke that got into the lungs of the smelter workers just as it did during the mining process (Derikson 1988; Shikes 1986; Smith 2009). There was also no oversight of the pollution this process created, potentially allowing the toxic smoke into the lungs of anyone adjacent to the areas where smelters operated.

Mining itself was not the only source of elemental contamination for residents of the state. Pueblo, as with most of Colorado in the mid to late 1800s, owed its existence to metals. Although Pueblo did not have the rich mineral deposits of Cripple Creek or Leadville, the city was centrally located and ideal for processing the materials from richer areas into more

useable forms. There were a number of smelters in the city but the main focus was on the steel mill (Smith 2009). Instead of removing potentially hazardous materials, steel-making added them. Steel is a mix of iron and carbon that is exceptionally strong, but also highly prone to rust. To protect it from deterioration, other materials are frequently added. In the Victorian period, these included lead, manganese and zinc (Derickson 1988). The heat needed to create steel evaporates some of the materials used, allowing them into the air and presumably into the lungs and bodies of those unfortunate enough to be in the area.

Although metal working was a major source of elemental contamination both to those who worked with it as a profession and anyone in the area, there were a number of other occupations that had exceptionally high elemental exposure risks. Anyone who worked in construction risked exposure to various elements through wood, glass and paint; wood was treated to protect it against insects and fungi, glass was treated with many elements for clarity and color and paint contained a wide variety of inorganic coloring agents (Emsley 2001). For women, who generally did not work in these fields, many of the exposure routes were domestic. Many cosmetics, especially skin whiteners, contained hazardous materials. Candles, cooking pots and even foods could all contain dangerous elements when exposed to them in high enough quantities (Farrer 1993; Emsley 2001).

The final source of contamination for many people was medicine. Before the 20th century, the American government had no labeling requirements, so medicines might contain any number of hazardous ingredients. Doctors also wrote glowing praise about a large number of substances now known to be poisonous and there was no standardization of amounts given, elemental purity or how they were administered, leaving an endless variety of ways a person might be exposed to problematic substances.

Arsenic

During the Victorian period, arsenic had already acquired its reputation as a poison, but that did not prevent its use in a wide variety of situations, not the least of which was medicine. Arsenic changed in popularity as a medication throughout the 1800s and early 1900s. It was considered a cure-all in the antebellum period but declined in use after the Civil War ended until its revival in 1910 as a fairly effective syphilis treatment (Brandt 1985). The first wave of popularity stemmed from the use of white arsenic in Fowler's Solution, a health tonic created in 1786 that remained popular into the 20th century (Emsley 2001). There were as many conditions arsenic was purported to treat as there were doctors using it, but there were some conditions for which arsenic was widely regarded as an effective treatment (Liu et al. 2008). The most universal uses were to treat recurrent fevers, such as with malaria, pain and swelling as well as some breathing problems. It was also popular for skin conditions that resulted in lesions or eruptions including syphilis, leprosy and psoriasis (Liu et al. 2008; Wood 1860).

Arsenic and its use was not homogenous, however. There are different forms of arsenic, which vary in appearance and use. Red, white and yellow arsenic were used interchangeably by some doctors and very specifically prescribed based on color by other doctors (Emsley 2001; Wood 1860). The asylum medical list has a single entry for "pills, arsenic 100" so it seems unlikely it was highly thought of by Dr Thombs (Painter et al. 2002). Medicine was certainly not the only way arsenic might enter the body during the period. It was commonly included in paint as a dye, in the creation of glass, as a pesticide, to whiten skin and to protect wood (Emsley 2001; Fowler et al. 2007).

Although exposure to arsenic from intentional use is possible, for those in Colorado at the end of the 1800s accidental exposure from mining is a much more likely source of arsenic

poisoning. In the southern part of the state, arsenic is commonly found in ore beds containing both silver and gold while in the rest of the state arsenic sulphate ore is commonly found only with gold (Neubert et al. 2011; Smith 2008). This means arsenic could enter the water supply anywhere and potentially be ingested by all residents. Arsenic was also an important part of copper smelting, as it was used in a process which aerosolizes it (Smith 2008).

Just as there were different routes of exposure from arsenic, so too are there different effects from the various forms. In cases of red, white and yellow arsenic, its low bioavailability prevents as much as 90% of arsenic in circulation from being absorbed and it is urinated out. The remaining 10% gets picked up by organs and bone (Feussner et al. 1979). The amount absorbed varies greatly as to the type of arsenic, however. For gray arsenic, the amount that makes into the blood stream when ingested is up to 90% (Fowler et al. 2007). Unlike ingestion where the form of arsenic dictates the percentage absorbed, 30-34% of inhaled arsenic will enter the blood stream. It is nearly impossible for it to enter the body through the skin eliminating that as a pathway for arsenic to enter the bloodstream and from there, to the bone (Fowler et al. 2007; Liu et al. 2008).

Once in the body, arsenic is not distributed equally, being found mainly in the kidneys and liver, and lungs if it is inhaled (Fowler et al. 2007). Arsenic is chemically similar to phosphate so some does make its way into the bones, although not in high levels. The exact mechanism by which arsenic causes severe problems in the body is not well understood, but it seems to damage cell mitochondria (Fowler et al. 2007). As such, the effects of high doses seem to mimic other poisons that are known mitochondrial disruptors. In low but chronic exposure, arsenic causes heart problems, cancer, pulmonary issues if inhaled, or digestive and stomach issues if ingested (Fowler et al. 2007). In acute doses, headaches, lethargy, seizures and

confusion also occur (Fowler et al. 2007; Oakberg et al. 2000). Arsenic is not particularly neurotoxic and thus it is only in very high doses that mental symptoms are liable to manifest (Fowler et al. 2007; Liu et al. 2008). As it was considered safe enough for human consumption during the 1800s, these effects from arsenic were likely attributed to other causes. So although arsenic poisoning is not listed as an intake reason on the asylum records, it is certainly possible that it created the symptoms of insanity for some of the inmates.

Copper

Not all elements were considered to have the medicinal qualities of arsenic. One of these elements is copper, which was not actively ingested during the late 19th century. However, it was very common and used in a wide variety of objects. It is a critical component of both bronze and brass so any production of those materials could be a source of exposure. It was also very popular as a pesticide and to make roofs for high end buildings (Emsley 2001). A much more likely source of copper for most people was cooking pots. Due to copper's exceptional ability to conduct heat, it makes an ideal material for pots and pans, which can leave small traces in the food, especially when the food is acidic (Farrer 1993; Emsley 2001). There are not large copper deposits in Colorado and so it was not actively mined in the state during the 1800s, but it is fairly common in ore deposits for other minerals that were mined and so it also may have gotten into the water supply, especially in mining towns (Neubert et al. 2011; Smith 2009).

Unlike many other metals, copper is a vital part of many cells in the human body. For this reason, the amount absorbed through the stomach and intestines varies widely by how much the body already has circulating and how much has been ingested in one sitting. The absorption rate can vary anywhere from 12.4-79%, although it usually remains on the higher end of that

amount (Mercer et al. 2002; Ellingsen et al. 2007). There is far less information on the amount absorbed through inhalation or cutanously, although both routes are known pathways for copper to enter the blood stream (Ellingsen et al. 2007). Once absorbed, copper enters the liver where it is stored to maintain copper homeostasis until it is needed by other organs (Mercer et al. 2002). Copper homeostasis is critical because if there is more copper than chaperone cells for it, the remainder will oxidize and damage any cells it contacts. If there is too little copper, then the enzymes that rely on it will not function (Ellingsen et al. 2007; Mercer et al. 2002). The enzymes that utilize copper have a number of functions, including electron transport, as a connective element in collagen, for neurotransmitter synthesis and mainly to protect against the oxidative effects of other elements. The brain in particular needs copper, consuming 35% of the amount in a healthy individual (Mercer et al. 2002).

Although vital, copper is also damaging if not bound to the proper molecules for transport through the body. It is very difficult for most people to overdose on copper and when it does happen, hepatoxicity is the main concern. However, in the cases where copper builds up past the level the liver can hold and the chaperone cells can safely transport, copper can be extremely neurotoxic (Lutsenko et al. 2002;, Mercer et al. 2002). This is very rare in normal individuals, but common in Wilson's disease, an autosomal recessive gene disorder. As many as 1 in 100 people may be carriers for the gene, making it common enough to have been present in a sample as small as the Colorado Asylum (Ala et al. 2007).

Although the most common feature of Wilson's disease is hepatoxicity, in nearly half of cases there is a psychological component as well. The body is unable to excrete copper efficiently, allowing it to build up in the tissues. Once the liver reaches carrying capacity for copper, it sends the remainder to the cells that would normally require it, such as the brain, but in

much higher levels (Lutsenko et al. 2002). It can also build up in collagen, which will over-represent copper in the bone, although not to the degree as other tissues (Ellingsen et al. 2007; Lutsenko et al. 2002). The most common manifestation of this build-up in the brain is idiopathic Parkinson's disease, characterized by movement problems such as slowness, rigidity, tremors and loss of balance and muscle control. Slightly less common symptoms are depression, anxiety, memory problems and a loss of inhibitions (Ala et al. 2007; Lutsenko et al. 2002; Multhaup et al. 2002). In severe cases, patients with Wilson's disease have been misdiagnosed as having schizophrenia (Lutsenko et al. 2002). Thus, although copper is not normally associated with mental disorders as are some other metals, it is still capable of producing a large spectrum of mental disturbances recognized during the Victorian Period. It is likely that anyone in the asylum who had high copper levels represent cases of Wilson's disease if indeed copper toxicity resulted in their symptoms and not occupational exposure or adulterated food and water.

Lead

Unlike many other elements, by the end of the 1800s it was clear that in many cases lead was dangerous to human life. In previous eras, lead had been ingested on a fairly regular basis, even being used as a sweetener in some cases (Emsley 2001; Farrer 1993; Shikes 1986). By the time of the asylum, however, the dangers of ingesting lead were fairly well known and using it as a sweetener or for any internal medicine was illegal (Shikes 1986). In fact, eight people were admitted to the asylum for lead poisoning or plumbism, and as early as 1860 it was raised as a possible cause of the dreaded mountain fever that plagued some high altitude mining communities (Mitchel et al. 2002; Shikes 1986). It does appear on the medicine list for the asylum in one form, sugar lead, which is more commonly known as lead acetate (Harlan 1838).

Most likely, it was used externally as an astringent. Lead acetate was the main ingredient in a large number of popular astringents at the time, including salt of Saturn and Goulard's powder (Harlan 1838).

Although medicine is a possible source of lead, the most likely source is mining. In Colorado the majority of the silver ore is encased in lead carbonate (Smith 2008). This means that the miners inhaled it after blasting apart the rocks, drank it when the tailings got into the water and inhaled it again when the last of it was burned off during the smelting process. This would have exposed a great deal of the population of Colorado to very high levels of lead and make it difficult to differentiate miners from those who simply drank run off.

Even without mining, lead was ubiquitous in the Victorian period and exposure to it nearly inevitable. Common routes of ingestion included the pipes that carried water which were usually lead-based, the solder on cans for food and some colors of ceramic glazes (Emsley 2001; Farrer 1993; Karri et al. 2008). For construction, it was a common component in glass, it occurs as a roofing material and was nearly universal in paint. It has a high resistance to rust and so it was sometimes used as a coating for steel. In the home, it was a common element in face whiteners for women and it was applied to candle wicks (Emsley 2001; Karri et al. 2008). Thus, it is nearly impossible to isolate a likely source of lead in an individual and indeed there were probably multiple routes of exposure for most people.

Just as with other metals, the amount of lead that enters the blood stream and from there the bone varies according to the method of exposure. In cases of inhalation, 10-60% enters the body. The ability to absorb it through ingestion varies widely based on the amount of calcium the body has circulating. Because of this, the amount absorbed through the stomach and intestines varies widely from 4-70% (Skerfving and Bergdahl 2007). It is also possible to absorb

through the skin, such as when used in cosmetics but the exact amounts that make it to the bloodstream through this route are unknown. Once the lead has entered the body, it is mainly picked up by the bone matrix; as much of 90% of the circulating blood levels of lead become incorporated into the bone, making it invaluable for osteological testing (Karri et al. 2008; Needleman 2004). Lead $^{2+}$ is so chemically similar to calcium^{2+} that the body is unable to recognize the difference between them (Cory-Slechta and Pounds 1995; Skerfving and Bergdahl 2007).

Because lead deposits so readily in the bone, the amounts there have been well-studied and a conversion exists for the expected bone volume of lead based on the circulating blood levels. It varies by age, sex and health, but overall in adults, levels above 50 μg/dl in the blood translate to 160ppm in the bone (Bower et al. 2007). This makes it possible to correlate the amount of lead found in the bones with the expected symptoms in the body from lead poisoning. At bone levels of 160ppm, lead can cause depression, agitation, seizures, dementia, personality changes, irritability, headaches and muscle control issues. In higher doses, encephalitis, delirium and seizures are common (Cory-Slechta and Pounds 1995; Needleman 2004; Skerfving and Bergdahl 2007). Although the way in which lead causes this toxicity is not well understood, it seems to be related to the chemical similarity to calcium. Calcium homeostasis is not only critical for the bones, but also the brain and especially the nerves. When lead replaces calcium in these pathways, it can damage the structure of the nerves, cause an over-release of neurotransmitters controlled by calcium and dull the ability of neurons to receive information and stimulus (Cory-Slechta and Pounds 1995).

Because it is so well studied in bone and because of the clear psychological effects that can result from lead intoxication, it has been studied in relation to the Colorado Asylum

previously. Bower et al. (2007) tested 33 skeletons from the 1992 excavation of the cemetery, and found that 29 individuals exhibited lead levels higher than what is now considered the safe level for adults and three were above the level considered dangerous. Thus it is clear both from the records of the asylum and from previous work on the skeletons that lead poisoning did afflict patients at the asylum and was possibly a widespread cause of mental disruptions.

Manganese

Manganese, like lead, is an extremely common mineral in Colorado geology, but was far less ubiquitous in industry and domestic use. Although the 12th most common mineral in Colorado, manganese has an uneven geographic distribution and the largest deposits of it are in the same areas that were intensively gold-mined in the Victorian period (Neubert et al. 2011). It was not actively mined during the period, but the manganese portion of the debris removed from gold was likely utilized. Because of its distribution, manganese was almost certainly part of the contamination from the goldmines. In a modern test of water flowing near the Sweet Home Mine west of Denver, the manganese levels were 94 times higher than maximum levels allowed by the state for drinking water (Neubert et al. 2011). Presumably, a similar level of contamination was present in many areas during the 1800s.

Although exposure from the mines is almost certainly the source of any intake of manganese by the patients at the asylum, the element was also used in paint, early batteries and glassmaking (Emsley 2001). In those materials, the exposure most likely happened only during production, not to consumers using the items, so they can be eliminated as sources of manganese toxicity. However, there was one industry, steel making, where manganese was used that could have affected people who ended up in the asylum (Elsner and Spangler 2005; Emsley 2001). A

number of metals can be added to steel to increase its resistance to rust, strength or durability and one of the most popular both in the late 1800s and the present is manganese. It is unclear if the steel mill in Pueblo used manganese during the Victorian period, but if so, it could have evaporated in the steel making process and entered the air or the water from the cooling of the metal, making the industry another possible source of manganese contamination in the skeletons from the asylum (Elsner and Spangler 2005; Emsley 2001).

Unlike other metals, the side effects of manganese seem to vary greatly depending on the exposure method. If ingestion is the method, a great deal is needed to produce any ill effects since manganese is not absorbed well into the body, with only 3-5% absorbed through food or water intake (Chu et al. 1995; Lima et al. 2011). Inhalation on the other hand not only allows 30-60% of the particles into the blood, but seems to make it easier for them to cross the blood-brain barrier than when ingested (Chu et al. 1995). There is no record that it can be absorbed through the skin and so that is unlikely to be a route for manganese poisoning (Saric and Lucchini 2007). Once in the body, manganese is mainly picked up by the bones and brain. It is not only preferentially deposited in the brain, but once there, remains there far longer than in any other tissue of the body, allowing toxic levels to develop (Chu et al. 1995; Lima et al. 2011). It is not entirely clear why it is picked up by the bone or how it affects the brain, but it may destroy cell mitochondria, overload cells with calcium, oxidize dopamine or deplete the strength of the blood-brain barrier, allowing aluminum and un-chaperoned iron into the brain (Chu et al. 1995).

Whatever the etiology, the effects of manganese poisoning are devastating. In cases of ingestion, the symptoms vary widely, but generally lack psychological involvement, whereas cases of inhalation, especially by miners, are characterized by such symptoms (Chu et al. 1995; Saric and Lucchini 2007). When inhaled, the symptoms usually do not appear for a few months.

When symptoms do occur, manic episodes and impulse control issues are the most common manifestations, although hallucinations have also been reported (Chu et al. 1995). If the exposure continues, the disease moves to a second phase characterized by severe depression and issues with movement and coordination. At this stage the condition is still treatable, but if it progresses to the final level, the deterioration can be halted, but not repaired. It is this stage that is classically known as manganism and is frequently misdiagnosed as Parkinson's disease (Chu et al. 1995; Emsley 2001; Saric and Lucchini 2007). The common symptoms in the final level include motor system disturbances, inability to move muscles with any speed, muscle rigidity, dystonia, speech problems, dementia and mood disorders (Chu et al. 1995). As with lead, these symptoms were known to be associated with manganese intoxication at the time of the asylum, as manganism was first discovered among miners in 1837, making it plausible that the doctors at the asylum were aware of the dangers of manganese (Emsley 2001). However, with no tests for manganese the doctors of the period could administer, and a large number of possible causes for idiopathic Parkinson's disease, it is not surprising there is no record of manganism at the asylum whether it was actually present or not.

Mercury

Another element that was not listed on the intake records but which was very common in the 19th century is mercury. Mercury has a long history of medical use and showed up no less than four times on the list of medicines for the asylum, in the forms of blue ointment, corrosive sublimate, mercury and citron ointment (Mitchell et al. 2002). As with arsenic, different forms of mercury were used for different conditions. Although it was used in a variety of forms to handle complaints as varied as cleaning wounds and relieving constipation, it was particularly

used as the dominant treatment for syphilis during the period (Shikes 1986). As a treatment for syphilis, it has been administered orally, topically and through inhalation (Rasmussen et al. 2008). By the late 19th century, the dangers of inhaling mercury were well established, so this method of treatment was no longer used (Shikes 1986). Although there is no documentation of how mercury specifically was used at the asylum, corrosive sublimate and ointments were applied topically and so it is clear that in the majority of cases mercury was used externally (Tucker 2007).

Medicine was a very common route of exposure to mercury, but there were a panoply of ways an individual in the 1800s could be exposed to mercury. It was a critical component in extracting gold from non-precious rocks using running water. Any mercury lost in the process, and there was a great deal, entered the drinking water supply (Buckie 1905). It became a very popular dental filling after 1895 and was a critical component in many medical devices (Emsley 2001). As with many other elements, it was also commonly used for preserving wood, as a paint pigment, a pesticide and in the production of glass. It was an effective skin whitener and so was part of many cosmetics as well. It also removes hair, making it a vital part of the felt hat production of the period. In fact, the mercury used is widely credited for the term "mad hatter" (Brandt 1985; Emsley 2001).

Mercury's reputation as a cause of madness is well earned. Even in small doses, it can cause severe neurotoxicity. Although neurological disorders vary based on the type of mercury, when ingested the stomach and intestines absorb up to 30% of the element (Berlin et al. 2007). Inhalation is by far the most dangerous method of exposure as mercury can pass through the alovear membrane into the blood stream. Up to 90% of mercury in vaporous form can enter the blood (Chang and Verity 1995). In animal studies, 10 times as much inhaled mercury makes its

way into the brain as injections, suggesting that this method of exposure is also likely the most neurotoxic (Chang and Verity 1995).

As mercury is one of the few elements commonly applied to the skin during the period, both as a medicine and cosmetic, cutaneous exposure must be considered as well. Mercury's ability to get through the skin into the blood stream depends mainly on the form, with elemental mercury being poorly absorbed while alkylmercury is much more readily absorbed. Overall, 1-8% of mercury applied to the skin gets into the body, making it a possible route of exposure, but far less likely to be enough to cause poisoning (Berlin et al. 2007). Once in the body, mercury rapidly binds to red blood cells and oxidizes which allows it to cause damage throughout the system. As with manganese, mercury seems to remain in brain tissue much longer than other parts of the body, increasing its toxicity and generally building up to levels 3-6 times that found in the blood (Chang and Verity 1995). The bones have little affinity for mercury, but it does seem to change calcium homeostasis, keeping the bones from releasing calcium as much as they normally would. This change in bone turnover is the most likely method for mercury to enter the bones (Berlin et al. 2007).

The horrifying side effects of mercury poisoning in the body account for its use in a number of medicines, especially skin treatments. When mercury is applied to the skin, itching and burning is common and this irritation can remove some lesions caused by other conditions (Berlin et al. 2007). In cases where mercury was inhaled as a treatment, extreme salivation is common (Chang and Verity 1995; Rammusen et al. 2008). During periods where diseases were believed to be caused by an imbalance of fluids or "humors" in the body, this salivation was believed to draw out the causal agent of the disorder (Brandt 1985). The other common side effects of mercury poisoning, uncontrolled emotional displays, memory loss, insomnia and brain

damage, could all be attributed to the diseases mercury was used to treat (Chang and Verity 1995; Parascandola 2008). Thus it is very difficult without chemical testing to tell whether many cases of madness were attributable to the disease or the treatment for it when mercury was involved.

Zinc

Unlike many other metals, zinc is not only vital to proper functioning of the body, it is also not neurotoxic. However, it can still lead to other conditions that mimic mental problems. It is not present in high levels naturally in Colorado soil, so any exposure likely came through human activities (Neubert et al. 2011). Zinc was popular in the period as a roofing material which might make it a source of exposure to construction workers, but few others. More likely, any exposure to high levels of zinc came from combining it with other metals. It is a critical component of brass, so anyone involved in the creation of brass or fabricating anything from brass would have at least some zinc exposure (Emsley 2001). The most likely source of toxic zinc levels for anyone in Colorado at the end of the 1800s is through the steel mill. There are a number of ways to counter this rust on steel, among the most popular of which is to coat steel in zinc, which is called galvanized steel. The term galvanizing was not used in reference to steel production during the period, but the process of protecting steel with zinc dates to the early 1800s, so it is very possible that high levels of zinc were used in Pueblo at the steel mill (Emsley 2001).

One other human source of zinc exposure in Pueblo is through medicine. "Sulph zinc" is listed on the pharmacopeia for the asylum and almost certainly refers to zinc sulphate, more popularly known as "white vitriol" during the period. It was mainly used externally as an effective antiseptic but also had a secondary use as emetic and was considered to be the fastest

one available at the time (Hamilton and Godkin 1894). Either of these uses is unlikely to have created dangerous levels of zinc in the blood stream unless combined with other sources, such as an overabundance in food or through inhalation while working.

Zinc is a critical element for human survival and so like copper, the rate at which it is absorbed through ingestion varies based on the amount the body needs. The amount of zinc that enters the blood stream through food and water sources varies between 20-60% (Sandstead and Au 2007). Unlike other vital elements, the human body has no specific storage system for zinc in case of deficiency, but the majority does end up in the bones, making it ideal for chemical tests. Both zinc and calcium bind to phytate and it is through this medium that zinc enters the bones (Sandstead and Au 2007).

Unlike most other metals, zinc is not neurotoxic and even in very high doses it does not seem to disrupt brain functioning. However, although vital, zinc is also damaging if not bound to the proper molecules for transport through the body. As copper also needs these chaperone cells, high levels of either zinc or copper in the blood tend to cause deficiencies in the other (Mercer et al. 2002; Sandstead and Au 2007). Zinc overabundance is one of the most common causes of copper deficiency and in these cases, there are severe neurological symptoms. Copper is critical to the protection of nerves, and deficiencies frequently result in the demyelization of nerves, leading to weakness, poor muscle control, balance issues and in severe cases, paralysis (Mercer et al. 2002).

Although nerves are well established as the main organ to suffer damage from zinc toxicity if copper deficiency is present, there may also be problems with the brain. The brain is low in anti-oxidizing cells and the ones that do exist are mainly copper- based, so any disruption in copper can result in severe physical damage to the brain structures. Problems with copper

deficiency or metabolism in the brain have also been linked a large number of neurodegenerative disorders, including genetic amyotrophic lateral sclerosis (ALS), Alzheimer's disease, non-idiopathic Parkinson's disease and brain prion diseases including Creutzfeldt–Jakob disease (Multhaup et al. 2002). Thus, although zinc cannot directly be linked to mental problems it can produce symptoms that would have been labeled paresis during the period. Further study may also reveal that various forms of dementia arise from the complex interactions of an overabundance of zinc in the body, further adding to its importance as marker of mental issues.

An understanding of the diseases and possible forms of contamination are valuable for understanding bone more generally, but critical for looking at specific cases, such as the Colorado Insane Asylum so that any findings in the specific collection can be contextualized and understood. Without this, no meaning can be derived from any results obtained.

Chapter IV: Research Methods

With the context of the Colorado Insane Asylum burials thus established, it is possible to examine the bones for evidence of conditions or contamination that might suggest the possible biological basis for the mental disorders suffered by some in the asylum. Among the ways this can be understood are through the excavations of the burials themselves, examination of the skeletons, especially the crania, and by doing chemical testing to look for possible neurotoxic agents. There are a large number of possible pathologies that can present in the cranium as an indication of insanity and in the case of the Colorado Insane Asylum collection. Of these, the clearest abnormality in the crania were some that were extremely heavy compared to other individuals and to the post cranial remains of the skeletons with heavy skulls. For the trace element analysis, samples were taken and processed in a way that would best preserve the neurotoxic elements most likely to be present as well as to minimize contamination.

Excavations of the Cemetery

The excavations that yielded this unique collection were carried out in several stages some years apart. When new buildings needed to be added to the asylum property, earth movers revealed the location of an unmarked and largely unknown cemetery. Water and sewer lines ran through the cemetery, particularly row A, so it was not entirely undisturbed before the excavation. The 1992 excavation revealed six rows of burials containing 18-25 interments per row. Although some skeletons were incomplete, approximately 131 individuals were found. When further expansion of the building was called for in 1998, another series of internments were encountered and excavated in 2000.

An earth mover cleared the parameter of the new section of the cemetery and the rest of the work proceeded with hand tools. Some of the burials discovered in the second excavation seemed to be a continuation of row A, while other skeletons discovered in 2000 seem to have been disturbed and reburied previous to the 1992 excavation. Twenty four skeletons were found, but this number may inflate the overall size of the collection as some of the partial ones are likely portions of the incomplete skeletons from the 1992 excavation. All the skeletons that were in the area have been disinterred; however a paved street runs over part of what was once the cemetery and it is possible more burials remain under the paved street.

The records for the asylum do not list who was buried on the property. Five hundred and three people died at the asylum during the time burials on the grounds took place, so it is impossible to say with any accuracy who was buried and therefore what their specific mental disorder might be (Painter and Kennedy 2002). Once the skeletons were sorted into separate individuals as much as possible, each was examined to determine age and sex, and pathological condition or signs of trauma were identified. Many individuals were incomplete, highly fragmented or part of group internments that made the determination of age and sex impossible. For the 113 individuals with well preserved pelves, age was determined by examining both the auricular surface and pubic symphysis. In the cases where the ages from these two methods did not agree, both features were compared to find the best fit for age. For sex, 131 individuals had pelves or crania complete enough to use for analysis. A litany of features were scored as definitely male, probably male, undetermined, probably female or definitely female to find the sex for each individual (Johnson 2009).

Cranial Measurements

In the examination of the skeletons a few stood out for having no visible signs of pathology, but whose crania were extremely heavy. The rest of their skeletons appeared to be normal, leaving the cranium as the only apparent abnormality. There is a wide range of variability in bone density and bone weight among any group; however the range of variability in weights suggests that at least some of the outliers were pathological. Each skeleton was examined and those with crania that were most intact were weighed and examined. Those skeletons where there was no head or where the remains of the skull were largely fragmented were not considered. Those skulls from individuals that are in more or less complete form were weighed both to find any that were unusually heavy or light and also to establish the mean weight for the collection to better clarify the meaning of any exceptionally heavy specimens. There were 59 skulls complete enough to be weighed which was done using a manual scale in grams. The heaviest two individuals, 00-13 and A6, were substantially heavier than would be expected and there was a gap between them and the next heaviest in weight as shown in table 1. These two, as well as the next heaviest, and a skull that had no apparent pathologies and was close to the mean weight for the collection, were chosen to be x-rayed. Differences in bone thickness, density, or any patterns in the distribution of these peculiarities were the primary focus.

The heaviest two were X-rayed using a digital X-ray machine at the Colorado State University veterinary center and the controls were radiographed at the Hartshorn Health Center on the Colorado State University campus. This was also a digital x-ray machine. Both sets of radiographs were taken by different technicians and machines, but the results are comparable. Two x-rays were taken of each skull; a profile and anterior view so that the full

expanse of each skull could be examined. They x-rays went onto disk with different x-ray display programs. Although the programs were different, they had all the same basic features, including the ability to take measurements of the skull at actual size. No comparable measurements have been published of skulls from inmates of Victorian asylum, so the measurements on the x-rays were done in accordance with modern studies to give a control for the thickness of the heaviest skulls. Ross et al. (1998) took 1 cm squares of both the frontal and the parietal bones on the right and left and measured the bone thickness to create a listing of the expected thickness. To make the comparisons as applicable as possible, the measurements on the x-rays from the Colorado Insane Asylum were taken in the same locations. It should be noted that the X-ray captures the entire skull and its curvature so there is a possibility that the x-rays would portray the skulls as thicker than they actually are. The level of detail in the pictures allows for a fairly reliable look at where the bone curves however. Due to the angle the radiographs were taken from, it was not possible to reliably measure the parietal in the same location Ross et al. (1998) used.

The weights, frontal thickness measurements and the x-ray images themselves were then compared to radiographs where a pathology had been reliably diagnosed. Ideally, any measurements and x-ray images from the Colorado Insane Asylum would be compared to skeletal collections that are contemporaneous. These data, if they exist, have not been published thus far and so modern collections are the basis for comparison.

Trace Element Testing in Bone

Visual examination of the bones can reveal a great deal, but certainly not everything that can be learned from the skeleton is visible. In order to obtain a fuller understanding of the

skeletons, especially as regards syphilis, chemical testing was done on bone samples from some of the individuals likely to have syphilis since it was commonly treated with mercury. Although a very large number of elements can be tested for, in this study the focus were those that were both widely present in the late 19th century and which can cause mental disruptions in high doses. This left six metals as possible contaminants; arsenic, copper, lead, manganese, mercury and zinc. There are a number of methods that can test for these elements, but due to the volatility of mercury, it cannot be tested by most methods that can be used for the other five likely elements. The relatively low melting point for mercury means that most methods evaporate the mercury before it can detected (Rasmussen et al. 2008; Tucker 2007). As such, inductively coupled plasma mass spectrometry, ICP-MS, was used to investigate these contaminants in the bone since this chemical method looks at the ions and evaporates all the elements. As evaporation is a part of the process for all elements, mercury's tendency to vaporize at low temperatures is not a problem. The skeletons were selected based on their perceived pathology or as controls for the pathological specimens.

Bone samples from 40 skeletons and the soil from five of the graves was used for testing. The soil samples were taken at the time of the initial 1992 excavation and came from the burials labeled A4, C1, C10, C25 and F13. The first goal of the testing was to see if there was a difference in the element levels, particularly mercury, of the skeletons diagnosed as potentially having syphilis versus controls that did not having the appearance of pathology. Samples were taken from the 15 skeletons that Johnson (2009) identified as having lesions consistent with or potentially consistent with syphilis. Another skeleton, 00-3, was selected due to extremely carious teeth, which can be a sign of mercury poisoning. Finally, 00-13 was sampled as it was

the heaviest skull in the collection and elemental analysis might help account for the weight difference from the other crania.

An additional 23 individuals were selected as not having evidence of skeletal syphilis and were used as controls. To help isolate syphilis as a cause of differences in mercury levels rather than bias, the samples were chosen both based on their age and sex relative to the skeletal collection as a whole. They were also selected based on their proximity to the skeletons with suspected syphilis to help rule out ground contamination. Thus, the controls were selected both to combat bias by age and sex, as well as by conditions in the soil.

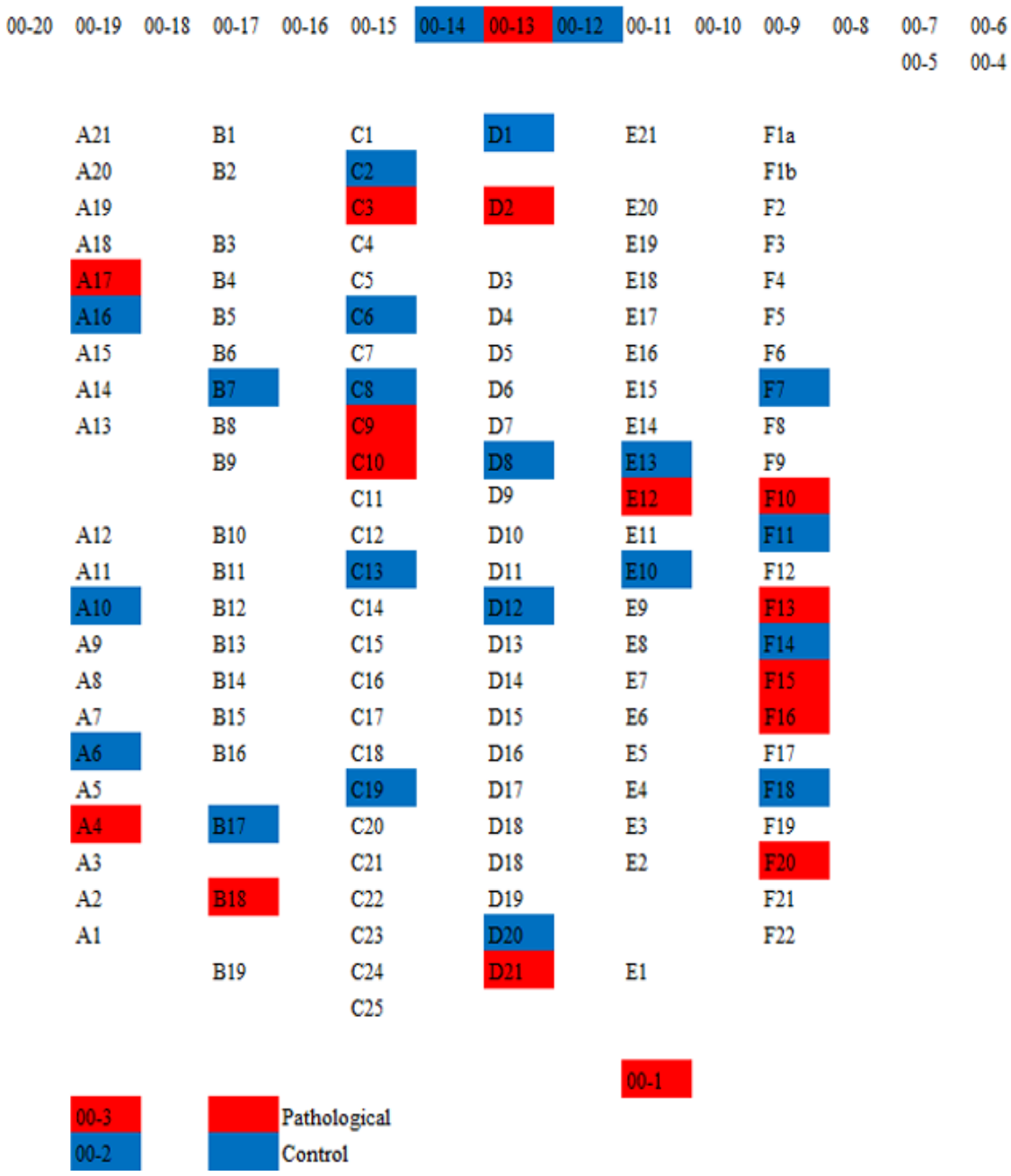

Figure 1: Relative location of the burials, not to scale (Painter and Kennedy 2002)

The age groups were broken down by decade of life and sex and chosen as representative of the skeletal collection more generally. This meant that in the 20-29 year age group, one sample was taken from a male and one from a female, for the 30-39 group there was one female and seven males, for the 40-49 group there was one female and three males, and for the 50 and

older group there was one female and eight males. Furthermore, the controls were taken from graves in close proximity to the pathological skeletons to help establish whether contamination accounted for any of the elemental results.

The bone samples were taken in two different sessions, with the pathological collection in the first and the controls in the second. For the first sampling, a small, toothed, steel blade for a Dremel hand tool was used to cut out a small piece of bone. The advantage was that the blade had a small radius and so did not cut more than the roughly 1cm square of bone needed; however, the blade was very thick so a large amount of bone was pulverized in the process. In the second sampling session, a diamond tip cutting blade was used. Although it had a much larger radius than the blade used in the first sampling, the diamond tip blade was also very thin so far less of the sample was lost; the blade also became dull much more slowly. The samples were taken from either the femur or the tibia, depending on availability. The tibia was preferred in cases such as burial 00-1, where the sign of pathology was on the tibia itself. The femur presented a better choice where either the tibia was unavailable, or where samples had previously been removed from the femur for past testing. The femur and tibia are also ideal because they were usually the best preserved and because long bones, particularly those of the legs, have the lowest level of diagenesis and therefore contamination (Pate and Hutton 1988). Samples were generally taken mid-shaft where the cortical bone was thickest. Any trabecular bone attached to the cortical sample was removed with the Dremel tool, as were any edges that appeared burned from the removal of the sample from the bone.

These samples were turned over to Dr. Nathan Bower of Colorado College to prepare for chemical testing. The periosteal surface of the first six bones, 00-3, 00-13, A4, C10, F15 and F20 were tested in an initial run. These six were not sanded beforehand and so may have had

more contamination from soil residue than the remainder of the sample. In order to further prevent contamination, each sample in the second run was sanded with aluminum grit right before being dissolved as this removed any particles that may have been picked up from the soil or cutting. Everything else in the preparation of the two different testing sessions was the same however. After being sanded, each sample was put into a 15 minute sonic bath with deionized water. The sonic cleaning occurred multiple times until the water ran clear, which usually took three soaks to do. The samples were not washed with anything stronger to prevent leaching of trace elements out of the bones. Each sample was weighed, and these weights were all approximately 100 milligrams. They were then dissolved in approximately 2 milliliters of high purity nitric acid; the exact amount of acid added depended on the size of the initial bone sample. This solution usually took about 8-12 hours to fully dissolve the bone sample. After the sample was completely dissolved, it was triple filtered and then diluted with 5-6 milliliters of deionized water. No microwave digestion was used to speed up the process as this might reduce the amount of elements that could be detected, especially mercury.

The samples were tested in an inductively coupled plasma mass spectrometer, specifically the Agilent 7500 Series ICP-MS machine. It tests by breaking samples down into their component ions and scanning their mass to charge ratio to distinguish the materials present. The machine is very sensitive and can test a wide variety of metals and other non-organic substances. This method is ideal as it is faster and more precise than atomic absorption, which was previously the best way to test for mercury. ICP-MS works by using a plasma torch to break down the sample, which results in a gaseous state where heat dissolves molecular bonds, creating free atoms. The sample is heated to the extent that only the positive ions and negative electrons remain. It is set to scan by positive ions and can distinguish 28 types of elements in the

sample; arsenic, copper, manganese, mercury, lead, zinc, calcium, magnesium, sodium, phosphate, aluminum, iron, vanadium, chromium, cobalt, nickel, selenium, molybdenum, silver, cadmium, antimony, barium, titanium, thorium and uranium. To run the test, a sample is drawn and cooled to nearly freezing to remove any water. It is then sprayed as a vapor by the nebulizer into the plasma chamber where the sample is broken down into its component ions that can be counted, thus given the amount of a specific element in the sample.

The methods for understanding the possible cranial pathologies as well as the metal levels in the bone were select to yield the most reliable results and specifically to address possibly causes of insanity in the skeletons tested. Despite the exacting methods used, the results were not as clear as could be hoped.

Chapter V: Pathological Conditions and Trace Element Results

The results of the examinations of the crania and the ICP-MS testing did add some information to the overall picture of the Colorado Insane Asylum collection. Although the results were not entirely as expected, they did reveal distinctions in the collection that were not previously apparent. The skull examination did reveal some unusual if as yet unidentified conditions and the heavy metal trace elements tied to insanity did reveal some dangerous doses, although not for all the elements examined.

Cranial Weight and Pathology

The weights, measurements and x-rays of the crania did yield results that were outside the expected range for a representative skeletal collection. Given the size of the collection and variability of those interred, it is not surprising to observe a wide variety of skull weights. There is little information available about the average weight of a dry skull so it is impossible to know how heavy these skulls really are compared to a more general population. There has been one small study of modern French crania that found the average weight for a female skull to be 548.07 grams ±94.57 and for men it is 623.44 ±106.83 (Quatrehomme et al. 2011). The average weight for the asylum collection males is 652.34 grams which is very close to the male average from the Quatrehomme et al. (2011) study, and for females it was 595.99 grams so the asylum collection as a whole was somewhat heavier than the modern collection.

Table 1: The age, sex and weight in grams of the complete skulls

ID Info	Sex	Age	Skull weight	ID Info	Sex	Age	Skull weight
A1	male	30-40	630.2	D1	male	20-30	542.6
A3	male	30-40	714.0	D18	female	20-30	583.5
A4	male	30-39	658.4	D19	male	30-39	747.5
A5	male	25-35	622.8	D20	female	40-50	654.7
A6	male	50-60	975.5	E3	male	50+	643.0
A9	male	50-60	691.4	E6	male	15-20	617.2
A11	male	40-50	520.4	E8	male	60+	586.3
A12	male	25-35	635.0	E10	male	30-39	683.1
A14	male	50-60	693.9	E11	male	60+	553.8
A15	male	50-60	597.1	E14	male	30-40	764.8
A16	female	20-29	349.4	E15	male	20-29	612.3
A17	male	20-30	572.8	E17	male	60+	637.0
A18	male	50-60	582.9	E18	male	60+	603.9
B13	male	40-50	571.6	E19	male	40-50	592.8
B14	male	25-40	534.0	E20	female	20-29	659.2
B15	male	30-40	578.3	F5	female	40-50	754.7
B16	male	60+	556.0	F7	male	60+	618.6
B18	female	20-29	610.1	F10	male	30-40	769.7
C5	female	20-29	473.3	F18	male	40-50	803.5
C9	male	60+	648.9	00-1A	male	60+	582.3
C14	male	20-30	658.9	00-1B	male	60+	734.5
C15	male	40-50	622.8	00-1D	female	40-50	567.2
C17	male	40-50	743.4	00-8	female	40-45	474.3
C19	male	25-35	727.2	00-10	male	30-35	653.4
C20	male	60+	617.7	00-13	male	35-40	990.9
C21	female	20-29	797.4	00-18	male	25-30	661.9
C22	female	30-35	792.8	00-19	male	30-35	498.3
C23	female	20-29	435.3	Mean			632.9

The notable difference between the asylum collection and the Quatrehomme et al. (2011) study is how much variability the Colorado State Asylum collection exhibits. The standard deviation for the asylum skulls is 119.8, thus the two heaviest skulls fall outside the second standard deviation as do the three heaviest female skulls. The heaviest two male skulls are 00-13

and A6 although there was no external visual pathology exhibited on either one. C21 was the heaviest female, although C22 and F5 were also quite heavy for the females. It was unclear if the females simply had a greater range of variation than the males. Among the females, there was no clear jump in weight between the heaviest skulls and remainder as there was for the males as shown in figure 2, so it was more difficult to say if the weight in the female skulls were pathological. Thus only the heaviest two male skulls could be reliably considered to be pathological based on the skull weight alone.

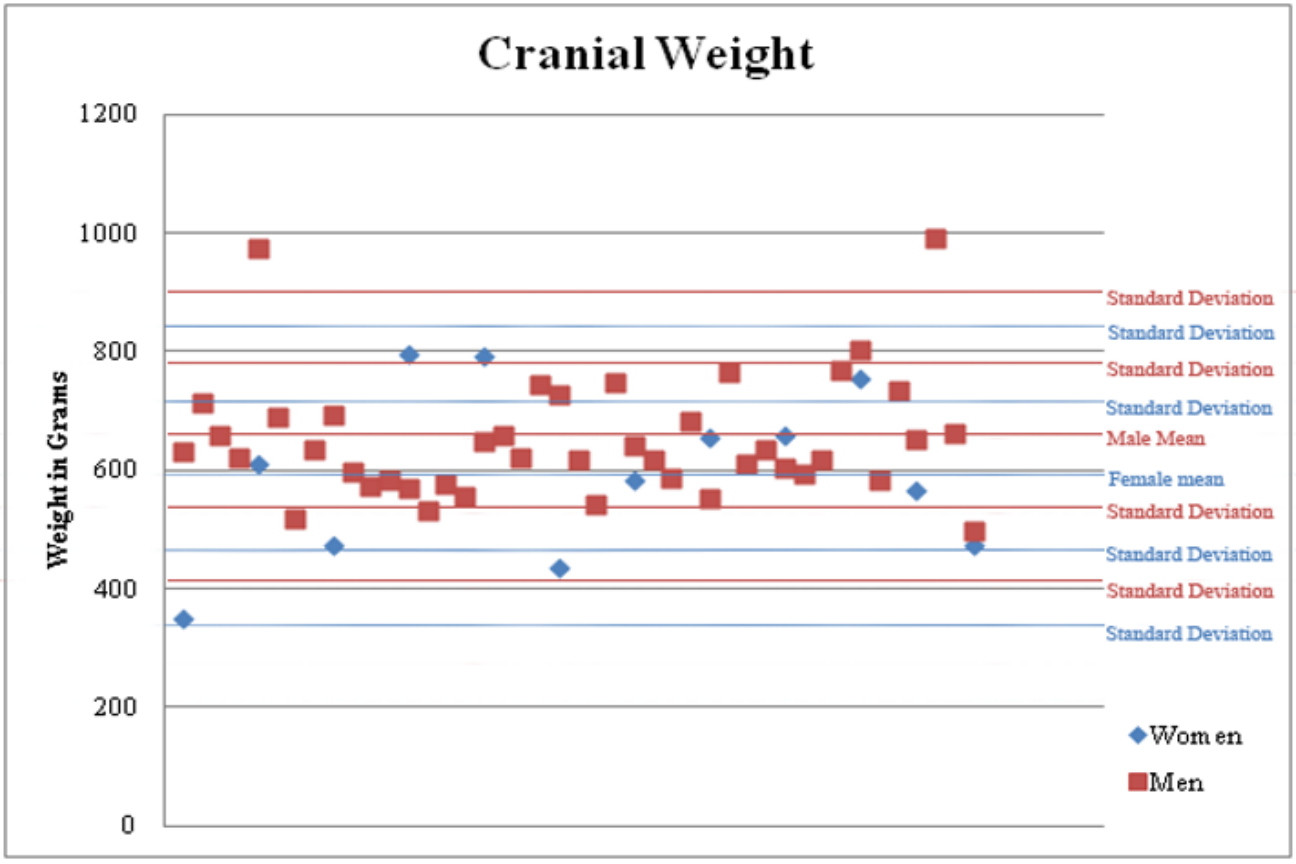

Figure 2: Skull weights with the mean and standard deviations

Cranial Radiograph Images

Since the weight of the two heaviest skulls was abnormal, but their appearances were not, x-rays were used to better understand the nature of their abnormalities and see if there was any indication as to the cause of the additional weight that was not visible to the naked eye.

00-13 is the skull of a male aged 35-40 years. The skull weighed 990.9 grams, making it the heaviest in the collection and nearly a third heavier than the mean weight for the collection. There are no obvious signs of pathology on the exterior of the skull. The teeth were more intact than many other individuals in the collection and the caries were mostly filled with gold indicating some level of quality treatment. The post cranial skeleton showed the individual to be a bit smaller in overall size compared to other males in the Asylum sample. In all other regards, the post cranial skeleton was normal. The frontal area is 13mm thick, making it nearly double the average of 7.68mm observed in a contemporary population from Ross et al. (1998). The frontal density appears uniform across the surface of the brain case and there are no growths or lesions to be noted as seen in figures 3 and 4. Aside from the thickness and apparent density, the skull appears normal and healthy.

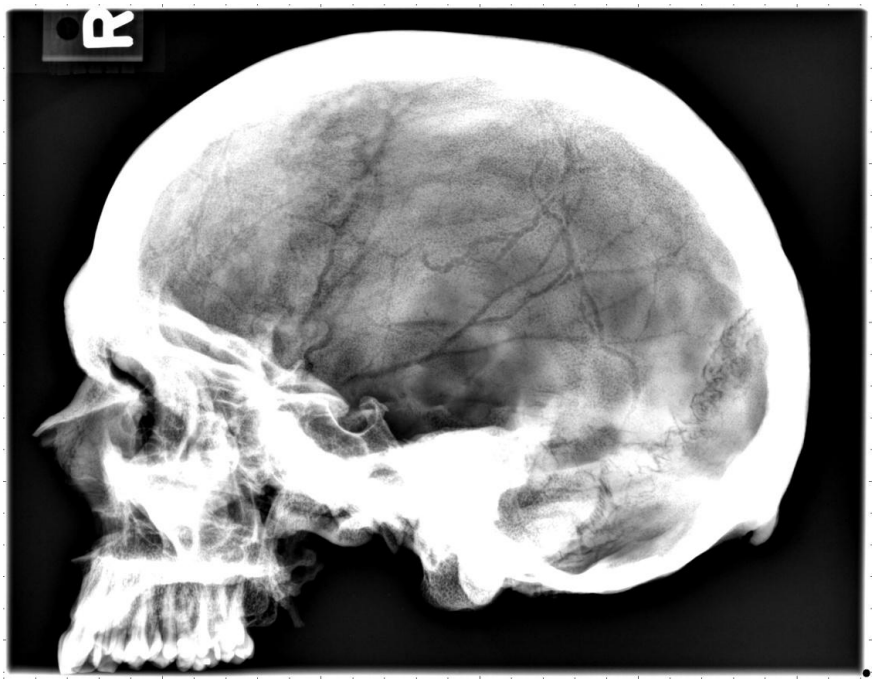

Figure 3: Profile x-ray image of 00-13.

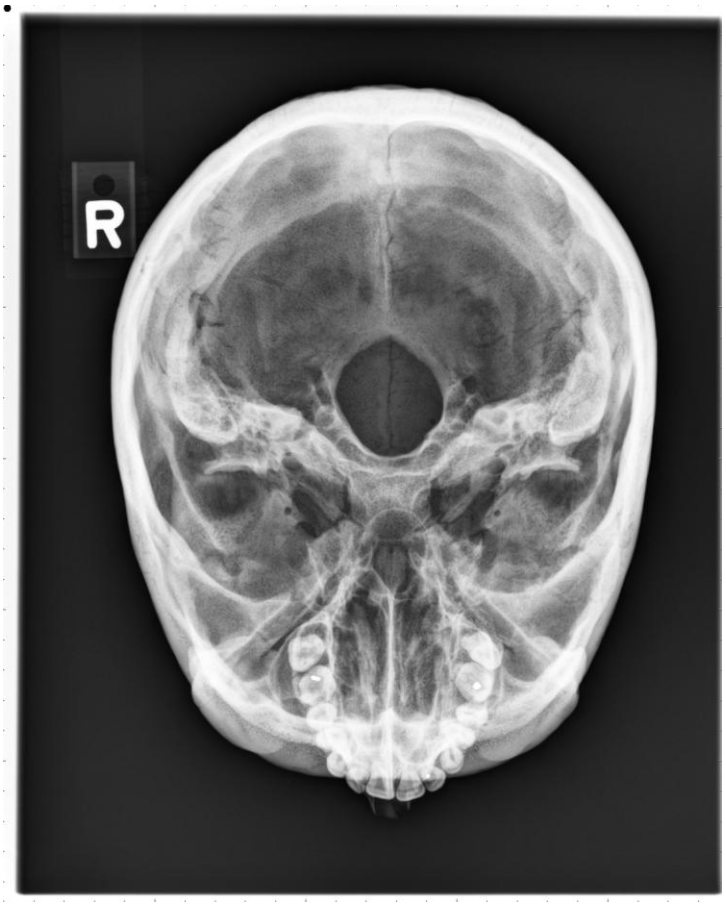

Figure 4: Inferior x-ray image of 00-13.

The next heaviest skull is A6, a male who was 50-60 years old when he died. His skull weighed 975.5 grams. There are no visible abnormalities in the skeleton. He had lost the majority of his teeth and most of the alovear areas showed absorption, indicating that the tooth loss happened some time pre-mortem. The frontal bone is 11mm thick, so thicker than the average for a male his age, but within the expected range of variance Ross et al. 1998). There are no lesions or other signs of pathology, but the skull gets progressively thicker from the frontal bone to the occipital as shown in figure 6. Overall it appears less dense than 00-13 which is not unexpected as this individual was much older and bones generally decrease in mass with age.

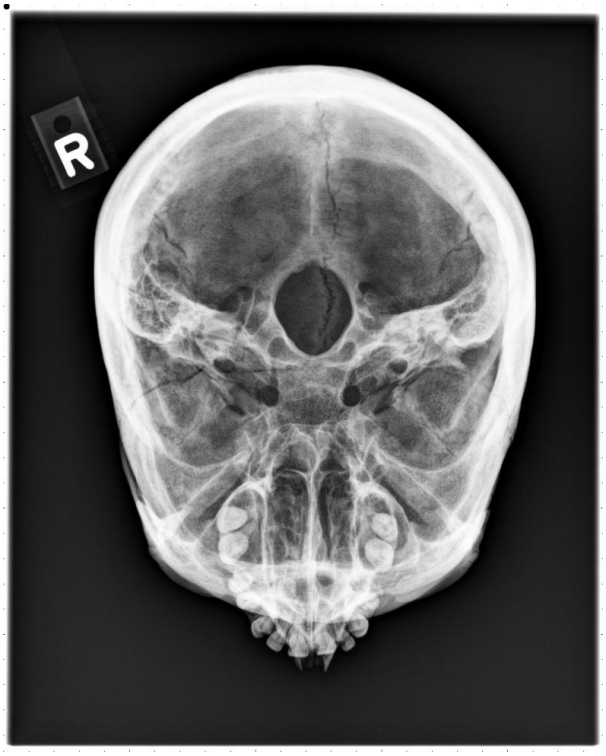

Figure 5: Inferior x-ray image of A6.

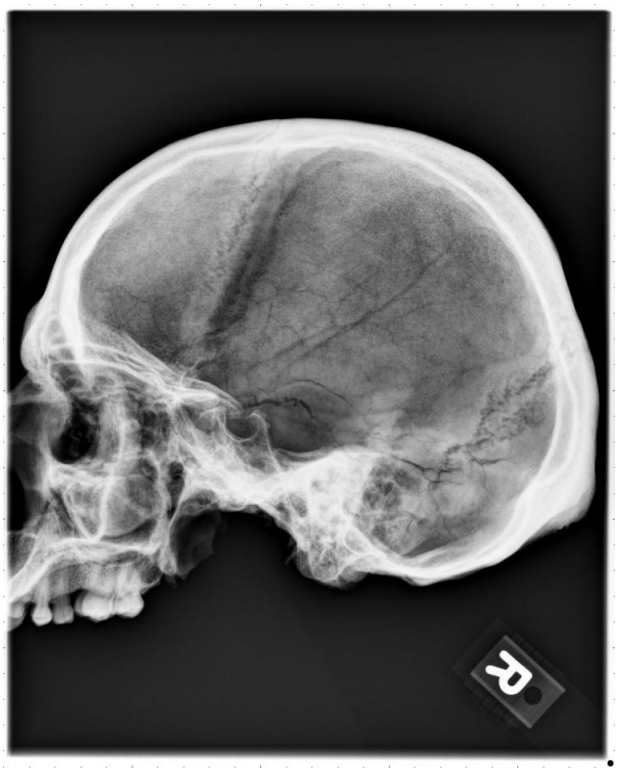

Figure 6: Profile x-ray image of A6.

The first of the controls is the next heaviest skull, F18, a male who was 30-39 years old. His skull weighed 803.5 grams, putting it at the very high end of the range for the collection and between the first and second standard deviation for weight. The frontal bone has one external lesion on the right side just superior to the orbit as seen in figure 7, but the cause of the growth is unclear. He also had a small area of periostisis on the right tibia and fibula, but as they were confined to one side it appeared to be related to an injury rather than systemic infection, which generally manifests bilaterally and thus is unlikely to affect the skull in any way (Ortner 2003). The frontal bone of F-18 is 9mm thick, making it close to the expected thickness for a modern male cranium. Aside from the lesion on the right frontal bone where the skull appears unusually dense, the skull seems otherwise normal.

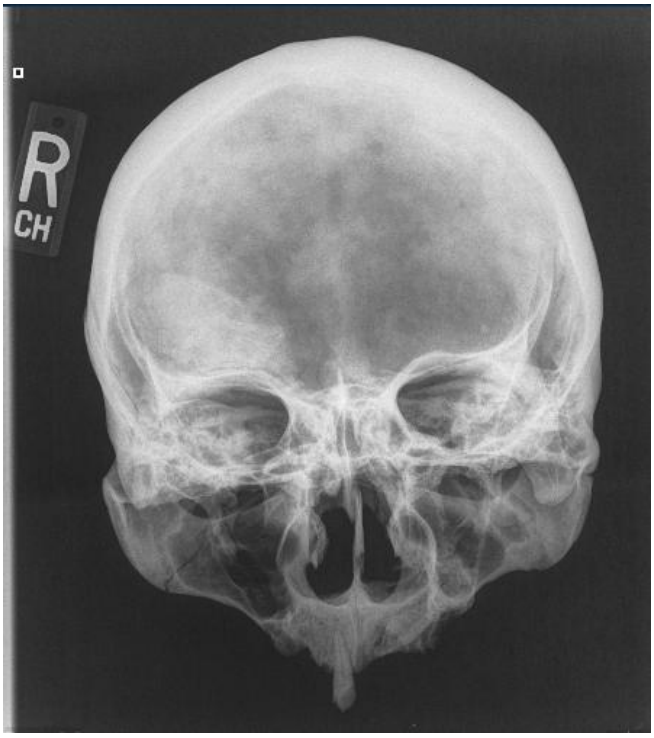

Figure 7: Anterior x-ray image of F18.

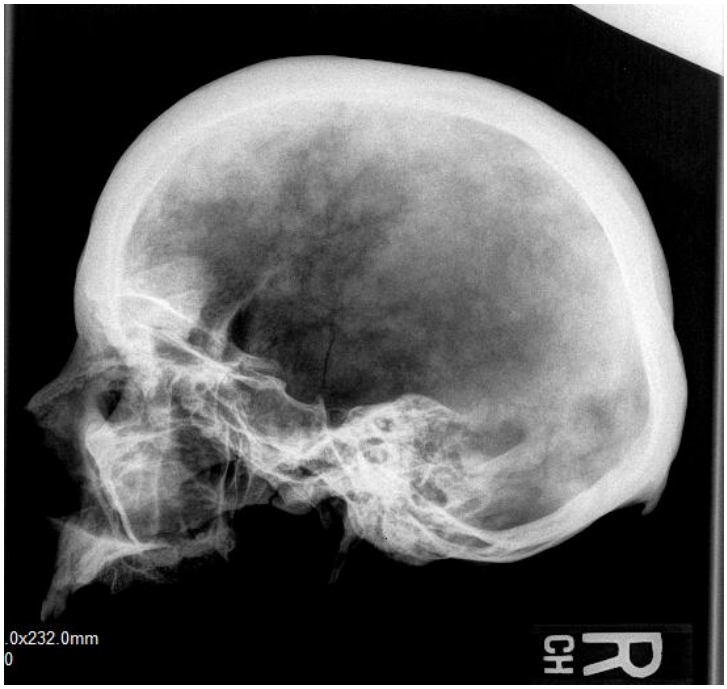

Figure 8: Profile x-ray image of F18.

The final radiograph is of the control C15, a 40-50 year old male whose skull weighed 622.8 grams, very close to the mean for the collection. This individual was selected because it fit the age and sex profile of the heaviest skulls and the skull weight was near the mean. The individual had no visible pathology that might affect the skull weight or thickness, making him an ideal selection as a control. The only notable sign of disease was very poor teeth, with nearly all of them missing. The frontal bone is 7mm thick, making it just below the expected average for contemporary adult males (Ross et al. 1998). The x-rays, figures 9 and 10, revealed no previously unknown pathologies in the skull.

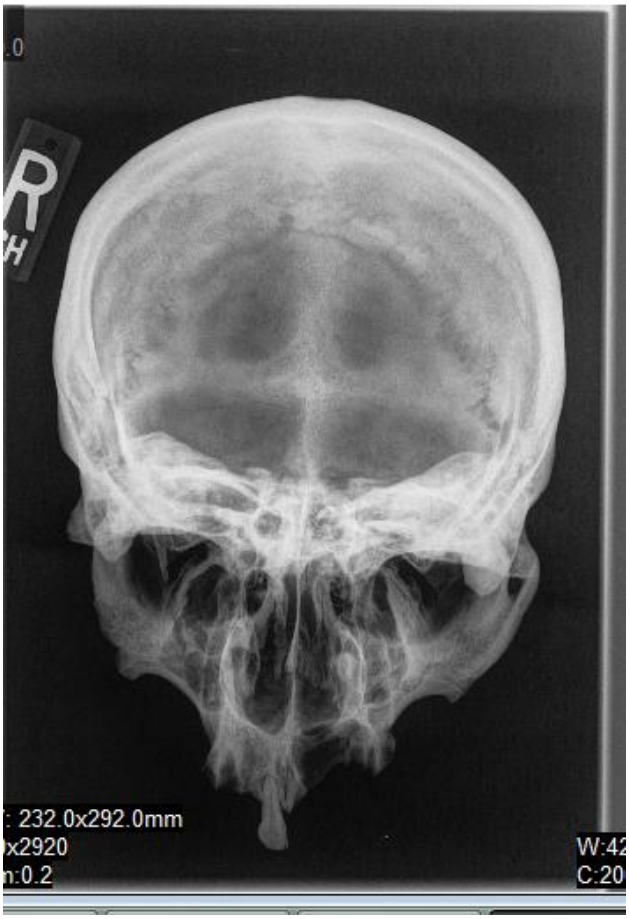

Figure 9: Superior x-ray image of C15.

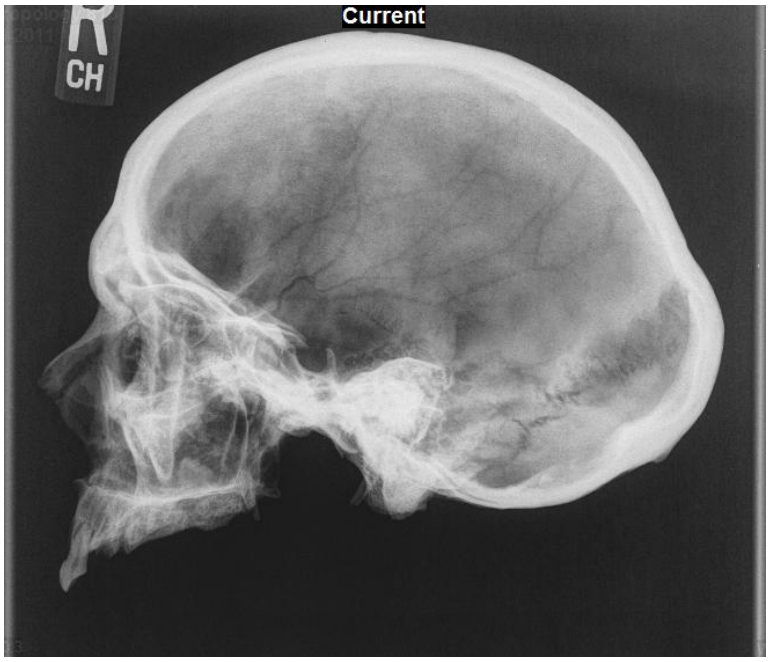

Figure 10: Profile x-ray image of C15.

Taken together, the x-rays and the cranium weight suggest that at least the very heaviest skull is abnormal. However, male skulls get thinner with age, so A6 is especially unusual when considering his advanced age (Ross et al. 1998). F18 fell within the expected range for both weight and thickness, so the weight itself does not suggest any pathology, although the lesion on the frontal bone is likely a sign of disease.

Cranial Pathologies

Taken as a whole, the cranial weights and x-rays do not reveal a clear cause for the unusually heavy skulls. More likely, a number of conditions account for their density. Morgani Stewart Morel syndrome and the accompanying *hyperostosis frontal interna* is nearly exclusive to older women. The three heaviest skulls in this collection are male and aside from A6, under age 50, making this condition an unlikely explanation for the weight (Herzakovitz et al. 1997; May et al. 2010; Ortner 2003). Furthermore, the two heaviest skulls exhibit an overall smooth

appearance to the bone with none of the billowing of *hyperostosis frontal interna*. The skull of 00-13 appears to be uniformly dense, while A6 is thickest at the occipital and thinnest at the frontal. Thus neither of these individuals matches the Morgani Stewert Morel syndrome's profile that mainly involves the frontal bone and only occasionally the parietals to a lesser extent (Barber et al. 2007; May et al. 2010). F18 does have a frontal lesion that matches the appearance and location of *hyperostosis frontal interna*, but the lesion is on the exterior of the bone and therefore more likely a benign growth.

It is much harder to determine if the two heaviest crania had Paget's disease, Dilantin medication intoxication, or another as yet unidentified condition. 00-13 does show a universal thickening of the skull and an increase in bone density over a normal skull which matches the expectations for Dilantin therapy, but whether that medication was available to him is unknown. The final likely explanation is Paget's disease. However, these two crania do not show any sign of the mosaic pattern of bone from the constant remodeling of the tissue which is the defining characteristic of Paget's disease. Usually when it afflicts the skull, Paget's disease is present on the interior which shows the remodeling, not the exterior (Ortner 2003). As the two heaviest skulls are entirely intact, it is extremely dificult to see the internal cranial bone clearly enough to ascertain whether the bone appears extensively remodeled. The radiographs do, however, closely match x-ray images of known cases of Paget's disease, making it the most likely explanation for the unusual weight (White and Folkiens 2005).

Trace Element Results

As there is no way to test the bone for the presence of diseases that causes cranial hyperostosis, the trace elements results could not offer further insight into the pathological

conditions that may be present. However, it can still provide a detailed look at what the individuals had been exposed to in life and whether any of the elements entered the body and therefore the bone in sufficient quantities to account for the presence of mental instability. Arsenic, copper, lead and manganese in high doses would be directly attributable as a cause of insanity for any individuals displaying toxic levels of those elements. Potassium and mercury were both common syphilis treatment of the period, so although indirect, their occurrence in high levels in any of the skeletons could indicate not only the cause of insanity, but also its treatment. Zinc, in high doses, is also an indirect sign of insanity as the high levels are not toxic, but rather an indication of possible low copper levels, which are toxic.

These metals and the other 21 elements the ICP-MS tested gave results for the chemical concentrations that Dr. Nathan Bower converted into parts per million and in the case of mercury, parts per billion so that they could be compared to other studies for the same elements as shown in table 2.

Table 2: Selected results from the ICP-MS chemical testing

Burial Number	K-39 %	Mn-55 ppm	Cu-63 ppm	Zn-66 ppm	As-75 ppm	Hg-201 ppb	Hg-202 ppb	Pb-208 ppm
00-01	0.0049	0.62	0.77	91.72	0.35	6.05	6.53	71.58
00-02	0.0037	1.07	5.50	202.39	0.45	9.52	11.93	44.88
00-03	0.0033	0.68	1.18	65.05	1.38	9.71	9.71	11.17
00-12	0.0030	0.57	3.55	98.18	0.70	3.37	1.96	65.91
00-13	0.0052	3.33	3.21	69.80	1.42	9.98	29.95	45.87
00-14	0.0031	3.30	0.56	104.67	0.53	7.57	5.35	75.95
A4	0.0099	11.77	3.78	93.41	2.61	67.92	77.62	93.94
A6	0.0031	0.94	1.35	114.60	0.58	6.40	6.40	19.71
A10	0.0024	1.47	1.94	93.73	0.43	20.97	22.03	19.78
A16	0.0047	2.97	3.64	168.86	0.67	17.34	18.50	83.25
A17	0.0036	1.00	1.02	123.54	0.26	2.74	3.36	27.90
B7	0.0037	1.01	1.45	140.16	0.54	7.20	6.28	28.71
B17	0.0046	5.32	1.28	80.36	0.22	7.09	5.05	19.50
B18	0.0070	1.77	0.76	94.22	0.08	3.68	2.30	49.26

C2	0.0028	3.16	3.44	112.56	0.31	9.36	8.84	50.85
C3	0.0043	0.71	4.36	90.66	0.33	13.17	16.84	77.54
C6	0.0024	7.61	1.75	88.98	0.47	10.47	11.45	170.97
C8	0.0047	3.26	1.55	118.59	0.40	11.59	14.45	44.07
C9	0.0031	6.31	0.56	99.26	0.47	14.46	17.15	23.71
C10	0.0046	4.97	0.78	57.99	1.28	10.82	10.82	17.76
C13	0.0028	0.71	0.63	82.63	0.06	12.82	15.28	62.51
C19	0.0025	0.61	1.01	84.97	0.12	4.86	6.80	27.68
D1	0.0042	0.33	1.09	172.94	0.21	31.69	39.20	30.37
D2	0.0041	1.46	0.79	98.94	0.24	3.91	6.27	51.80
D8	0.0043	0.69	0.85	139.77	0.21	4.19	7.14	81.75
D12	0.0039	0.61	0.65	98.23	0.48	16.28	16.28	42.61
D20	0.0022	0.55	3.70	103.45	0.39	2.40	3.70	30.63
D21	0.0033	2.67	0.70	95.77	0.13	7.86	8.39	79.09
E10	0.0048	1.45	0.54	108.46	0.21	2.43	2.53	25.79
E12	0.0047	10.87	0.87	112.57	0.32	-0.14	0.28	89.70
E13	0.0030	1.58	1.47	150.09	0.83	2.29	5.04	23.72
F7	0.0050	22.31	1.20	194.40	0.30	49.52	51.72	54.30
F10	0.0036	0.33	32.28	87.06	0.13	6.08	7.18	36.51
F11	0.0025	4.41	2.18	205.25	1.12	0.54	0.98	25.35
F13	0.0050	62.41	2.44	438.91	0.70	0.44	1.32	37.73
F14	0.0059	112.13	3.46	135.22	0.65	6.11	9.09	22.36
F15	0.0028	9.52	0.65	78.69	0.56	0.00	8.96	69.87
F16	0.0024	6.46	3.15	107.27	0.62	7.12	9.80	10.91
F18	0.0038	4.53	1.33	147.31	32.08	16.65	18.40	84.68
F20	0.0038	4.19	12.69	80.65	1.82	10.04	10.04	27.00

The Elements of Insanity

Since the results for iron and aluminum in the skeletons that were exceptionally high may represent digenesis rather than anything they were exposed to in life, those results cannot be taken as meaningful for further analysis. However, this is not true in the cases of other elements that were much higher in some skeletons than could be reasonably accounted for statistically. Each element from the collection was entered separately into a histogram to ascertain whether there were any individuals within the sample who fell outside of the expected range and might therefore represent a particular abnormality. Most of the data had a high degree of skew which

means the data had more variance than could be statistically accounted for and likely contained extraneous information. In order to remove the effects of skew, the logarithm for each element was taken. As some of the results contained negative values, only those elements that had all positive results were used. A histogram was then created from the logs of each element. These histograms revealed that there were a few skeletons with abnormally high levels of arsenic, copper, lead, manganese and zinc.

Most information on the effects of element exposure in humans consider the circulating blood levels of that specific element and do not account for the quantity expected to be incorporated into the bone so a given blood level of an element cannot be correlated to the bone level. However, average amount expected in bone is known for many elements; of particular interest fort this study is arsenic which is found at 0.1-1.6ppm for most people (Emsley 2001). All but four of the skeletons tested from the asylum exhibited arsenic levels within the normal range. Three of those outside the range reported by Emsley (2001) were just under 3ppm and the last one, F18, had an arsenic level of 32.0 ppm. Upon exposure to arsenic, roughly 10% is taken up by all the organs and bone combined, meaning the level in the blood was many times higher than what remained in the skeleton (Feussner et al. 1979; Fowler et al. 2007). Thus this amount of arsenic is very notable and so high that it may have caused the symptoms that institutionalized the individual. It is also very possible that the arsenic indicates a cause of death. Since arsenic was only listed once on the medicine list for the asylum, this amount may represent a successful suicide attempt rather than over medication from the doctor.

Copper is another element that can cause mental symptoms, although generally only in those genetically predisposed to problems with copper metabolism such as in Wilson's syndrome (Ala et al. 2007; Lutsanko et al. 2002). As with arsenic, there has been little study on the amount

of circulating copper in the body and how much of that is taken up by bone. To maintain copper homeostasis, a circulating blood level is normally 1ppm, while the average amount found in the bone is anywhere from 1-25ppm. A single individual, F10, exhibited a copper level of 32.28ppm, a value slightly higher than the highest value in a normal range. This level is not far outside the expected norm and so may not represent anything toxic. However, in Wilson's disease, copper builds up differentially in various organs, so although the amount of copper found is not much higher than in a normal individual, F10 may still represent a case of someone admitted to the asylum for psychiatric symptoms stemming from elemental overload.

At least one individual did exhibit a clear case of elemental overload that was likely the cause of institutionalization and very possibly death in at least one of the individuals tested. Unlike most other elements, it is known roughly how much lead in the blood translates into lead taken up by the bone. The current safe level as defined by the World Health Organization is 32 ppm (Karri et al. 2008). Of the 40 skeletons tested, 23 showed lead levels above this limit and those below were generally very close to the upper limit set by the World Health Organization. This is probably due to the ubiquity of lead during the period. Lead in the late 19th century entered the air and water through mining, chipped off paint, a solder on pipes, cans and cooking pots, was a critical component of ceramic and glass glazes, as the material of choice for many medical devices and was present in tooth fillings among other things (Emsley 2001; Farrer 1993; Shikes 1986). Even with more than half the collection above the safe limit of lead, there was still one individual who stood out because of his exceptionally high lead level; C6 had a lead level of 170.97 ppm. 160ppm is generally the amount in adults where mental and nerve problems manifest (Bower et al. 1997).

Although 170.97 ppm is not high enough to cause encephalitis or certain death, this level of lead could still have been fatal. C6 exhibits a lead level that is much higher than the rest of the sample, suggesting that the exposure happened before he entered the asylum, and was almost certainly the cause of his admittance. It is very possible that he was one of the eight individuals for whom the superintendent listed lead poisoning as the cause of their insanity. Lead in the form of sugar lead was a treatment listed for the asylum and so it is possible his exposure happened once there, but the dangers of lead were fairly well known and it was probably only administered externally (Shikes 1986).

The manganese levels in the bone were not as high as those for lead, but they are no less significant. Manganism usually becomes symptomatic some months after exposure and manganese has a very short half life in the blood, so it is nearly impossible to tell what levels of exposure are needed to induce profound psychological changes. However, as it lasts in the bone much longer than in the blood, it is possible to tell what an expected and safe amount of manganese is, and from that infer what higher levels may indicate.

Pejovic-Milic et al. (2009) found the bone levels of manganese for a population of welders with higher than expected blood levels of manganese versus controls. In that study, the average manganese level in the bone was 0.1ppm for the control population and for the welders it was 2.9ppm. At those levels, the welders did not show any signs of manganese-related toxicity, so the level at which it is likely dangerous is higher. In the asylum collection, 22 individuals fell below the average for welders. Most of those that were above the level were only above it by a few ppm, and all but five of the skeletal results were below 10ppm; of those above, three fell below 50ppm. The two remaining were F13 at 62.41ppm and F14, at 112.13ppm. Although there is no established baseline for how much manganese in bone can be interpreted as

representing manganism, the remarkably high amounts found in F14 would almost certainly explain his admission to the asylum and any symptoms he may have displayed. To protect itself from manganese overdose, the human body will stop absorbing it through ingestion once a critical level is reached. However, the body has no way to do this with inhaled manganese, making the very high levels shown in F14 a likely indicator of inhalation exposure. Given that the industries that used manganese would be unlikely to aerosolize this amount of the element, gold mining in the areas where manganese is a major component of the earth is the most likely explanation.

Unexpectedly, mercury did not match the other neurotoxic elements and showed no sign of either outliers or any individuals that had levels high enough to suggest mercury treatments. The syphilis versus control data which was the main concern of the elemental testing failed to yield results that would indicate syphilis through high mercury levels. The results also do not match the expected numbers based on other syphilitic studies. Rasmussen et al. (2008) used ICP-MS to test a large number of Danish skeletons for the presence of mercury where syphilitic lesions were visible. In the uninfected population they found that the average mercury level varied between 0-100ppb while in those with syphilitic lesions it ranged from 100-600ppb. The highest amount of mercury found in the asylum collection is 77.62ppb, putting it well below the expected amount for mercury treatment and solidly within the range for normal exposure.

The mercury levels make the asylum collection an anomaly with regards to syphilitic lesions, but there are a few possible explanations. The first is that although mercury was the most common treatment for syphilis during the period, it was by no means the only one. Potassium, arsenic and a host of herbal treatments were also used and as Dr. Thombs left no records as to his beliefs on the best cures for syphilis, anything on the extensive medical list for

the asylum could have been used (Brandt 1985; Mitchell et al. 2002). However, if this were the case, it seems unlikely that the one skeleton that could most definitively by listed as syphilitic, A4, would also be the one with the highest levels of mercury, and by a fair degree.

The lack of detailed records also leaves enough ambiguity that sampling error might be the responsible agent. The criteria and evidence for diagnosing syphilis that a Victorian doctor used and those used by someone looking at osteological remains are widely different. Syphilis was infamous for the multitude of forms it could take and before modern testing for it became available in 1905, those people labeled syphilitic and treated accordingly may not have actually had the bacteria and therefore would not show any of the characteristic markings of it on the bone. Conversely, many people who had syphilis might have been misdiagnosed as having another condition and so were not treated with the standard syphilis pharmacopeia. Thus they might display the bone lesions clearly demarcating syphilis, without any of the expected chemical results. Finally, the attempt to diagnose syphilis in the bones might account for the sampling error. As bone involvement in tertiary syphilis only happens in 10-20% of cases, it is possible that the Victorian diagnosis was correct, but that among those buried in the cemetery, there were no traces left osteologically (Ortner 2003). There is also the possibility of modern misdiagnosis of the lesions on the bone. Not one skeleton in the collection had the caries sicca that defines syphilis osteologically and only one skeleton could be assumed to have syphilis with any surety (Johnson 2009). Other than that one case, the others tentatively diagnosed as having syphilis in the collection might have suffered from other infections that were treated with other medications.

There is one final explanation for the low levels of mercury present in the sampled bones. Rasmussen et al. (2008) were looking at syphilitic skeletons where the preferred treatment was

mercury inhalation therapy. Before the 1800s, vapor therapy was the predominant method of administering mercury to syphilitic patients (Rasmussen et al. 2008; Tucker 2007). As almost all the elemental mercury inhaled enters the blood stream, a great deal of it would also have the opportunity to be incorporated into the bone matrix. On the other hand, by the period of the asylum, inhalation therapy was illegal in the United States and the predominant form of mercury used was external (Shikes 1986). The use of mercury only externally seems to be the likely case in the asylum, where one of the treatments was listed simply as mercury, which can be taken in any form, but the other three listings, blue ointment, corrosive sublimate and citron ointment are all for external use only (Emsley 2001; Mitchell et al. 2002). This is a very important distinction as up to 90% of the mercury inhaled enters the blood stream, but only 1-8% on the skin can be absorbed, making cutaneous exposure much less likely to show up archaeologically (Berlin et al. 2007; Chang and Verity 1995).

Zinc also manifests somewhat unusually for the archaeological record as it is not a neurotoxin, but can result in very low copper levels, which does cause neurological symptoms. There is no record of how much zinc in the bone would represent toxcicity and the accompanying dangerous decline in copper, but there are some rough guidelines. The average expected amount of zinc in the blood is approximately 7ppm with levels in bone ranging from 75-170ppm as it stores zinc for the body (Emsley 2001). The dangerous doses of zinc can vary widely, but doses as low as double the normal amount in blood tests have been associated with dangerously low levels of copper (Driscoll et al. 2010). Of the 40 skeletons sampled, 35 exhibit the expected normal amount of zinc for healthy individuals and four of those outside that level fall under 200ppm. The notable exception is F13, who showed a zinc level of 438.91ppm. This level is more than double the expected amount and therefore within the possible range of copper

deficiency. As zinc is being used indirectly, it is difficult to tell if this amount truly represents enough to account for the mental symptoms of the patient in zinc associated mental illness. As females were not involved in most mining operations nor working at the steel mill, there is a real possibility that the exposure may have come from medication. Many treatments from the period contained zinc and at least one of these was present at the asylum, sulf zinc (Mitchell et al. 2002).

Diagenesis and Contamination

Once the testing was complete, there was the issue of contamination to consider. The periosteal surface of the first six bone samples was not removed before being processed. The rest of the samples were sanded. Therefore, there may have been some soil still attached to the surface of the bone of those six. Some of the burials may also have faced high levels of contamination due to run-off, particularly from the paved street. Row A, which was adjacent to Hood Avenue, a paved road immediately adjacent to, and over the cemetery, was seemingly subject to the material from repaving and car emissions, including lead before it was banned as a fuel additive (Painter and Kennedy 2002). However, by taking samples from only the cortical portions of long bones after removing the periosteal layer, the amount of contamination was minimized as those sections are the least subject to digenesis (Roels et al. 1994). The graves were only in the ground for approximately 100 years, which also reduces the likelihood of contamination by the soil as generally, the bone preservation was good to excellent.

The ideal way to test for digenesis is to take a sample of soil from each grave and use principal component analysis see if the elements higher in the soil for each grave were also higher in the bones from the respective graves (Buikstra et al. 1989). If there is no correlation between the skeleton and the respective soil sample, then diagenesis was unlikely to have

occurred. As there were only five samples of soil, it was not possible to compare the bones and the soil from the asylum. Having the soil samples did give some insight into what might have contaminated the samples if indeed there was any diagenesis, so that it can be taken into account in any analysis.

The soil was high in aluminum and iron but also contained measurable amounts of potassium, manganese, cobalt, nickel, cadmium, barium, mercury and thorium. These elements were notably higher in the soil than in the skeletons. As the soil had such high levels of iron and aluminum and these levels were higher than in the bone, these two elements were not used in further analysis as any results may represent soil levels rather than living bone levels. This was done out of concern that these two elements were the most likely to reflect digenesis if any has indeed occurred, and using them in further analysis could severely bias the results. The skeletons with the highest levels of these elements, C10 and A4, were used in further analysis, but the high levels of aluminum and iron cannot at this time be attributed to anything they were exposed to in life.

Analysis of the Bone Chemistry

Looking at the outliers for various elements provides extremely valuable information about what the individuals were exposed to in life and may also highlight the possible root cause of insanity for those with high levels of some elements. However, those outliers do not explain the chemistry of the group as a whole or elucidate any broader implications from the chemical testing results. In order to better understand the results as a whole, a multivariate analysis method is needed.

Although there are a number of mathematical methods that can reveal patterns in multivariate data when the goal is to reduce the number of variables, principal component analysis is the most valuable tool. By looking at the amount of variance that is shared by the different variables, called communality, it is possible to see where the different results overlap and where they differ. Because the method is linear, positive correlations mean that as one variable increases, so too will the others. The line of best fit for the data that are created with principal component analysis is the first principal component, meaning it is the factor that most powerfully influences the connections between the different variables. The second principal component is always orthogonal to the first. The second accounts for the amount of variance that occurs from the line of best fit. The number of components is only bound by the number of variables for the data set, but as each successive component reflects a smaller amount of the variance than the last, it is possible to eliminate some of the smaller components that do not contain much information, simplifying the data and condensing the results. The amount of variance that any component accounts for can be represented mathematically by its eiganvalue. Although it is still debated, it is common to disregard eiganvalues below one as they are deemed too small to add anything substantive to level of variance within the data (Jolliffe 2002; Shlens 2005).

Principal component analysis requires linear expression, which most of the elements from the asylum did not have. As such, the logarithm of the data was taken and the analysis was done based on the logarithm. Iron and aluminum were not useable as any results obtained from them might represent digenesis rather than somethingabout the life of the individual based on the soil samples. That left the six elements linked to insanity, arsenic, copper, lead, manganese, mercury and zinc. Because zinc and copper are already known to be inversely represented in the human

body, this correlation could confuse any results from the other elements, so they were left out as well. This meant that the elements used for analysis were arsenic, lead, manganese and mercury. The logarithms were taken of each of the elements and then these data were combined to find the principal component analysis. As there were four variables tested, there were four Eiganvalues, 1.38, 1.05, .99, and .59.

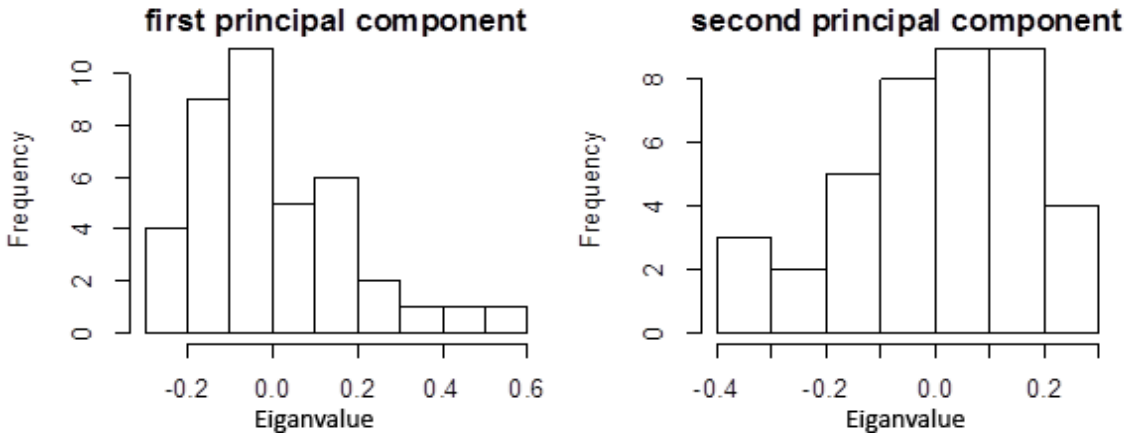

Figure 11: Representations of the logarithms of the first and second principal components

The first principal component accounted for roughly 35% of the variation found within the sample. The results were: 0.34*log(Hg) + 0.12*log(Pb) + 0.61*log(Mn) + 0.70*log(As). This means that the majority of the variation comes from the covariance of manganese and arsenic. The second component accounted for the presence of mercury and lead as being inversely related to manganese and arsenic as shown in the results: 0.70*log(Hg) + 0.58*log(Pb) - 0.39*log(Mn) - 0.10*log(As). The second component accounts for approximately 26% of the variance in the sample. The third and fourth components added very little which means that nearly 40% of the variance within the samples is not accounted for by the identified variables.

Once the variance was factored into components, it was possible to further break down the results by age and sex. As with many forms of science, the more data points available, the

more accurate the results. Given the small sample size in this study, breaking down the results by age left so few results in each category that nothing substantive could be determined as shown in figure 12.

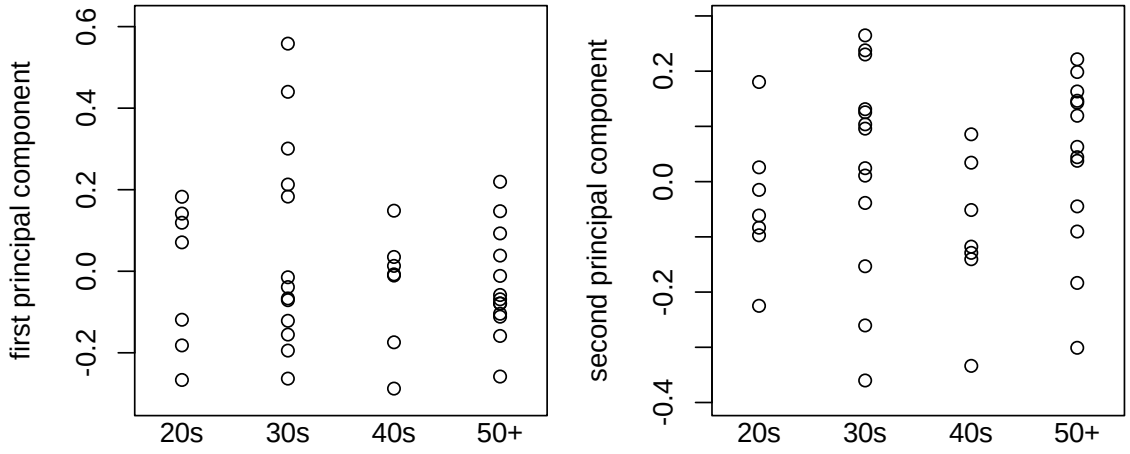

Figure 12: Principal component analysis plotted by age

Although with both the first and the second component there was more variance in the data points for those in their 30s and the 50+ age groups, no other differences appeared. This is unusual as the skeletons of older individuals frequently test higher for contaminants as there had been more time in which to accumulate them. Assuming the data are not severely biased by the small sample size, it appears that age has no effect on the accumulation of these elements in the skeleton.

The sex distribution of the principal component analysis is shown in figure 13. Note that in general, males showed higher values on both the first and second principal components compared to the females. This is especially true of the second component.

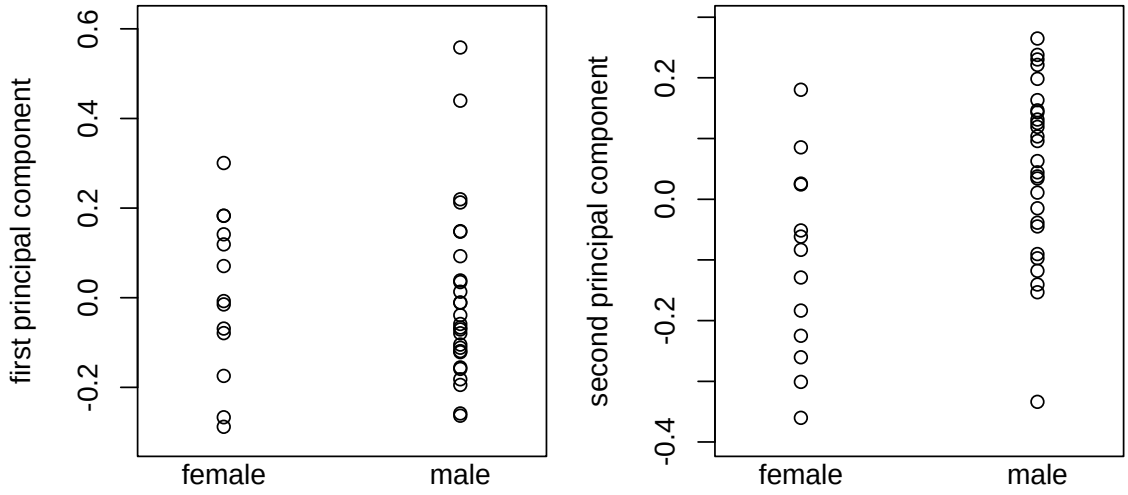

Figure 13. Principal component analysis plotted by sex.

In the case of the first principal component, which represents arsenic and manganese, there is a slight tendency for the males to have higher overall results, but the vast majority of the cases overlap. This suggests that the exposure to these two elements is mainly accidental exposure that hypothetically would have affected both sexes equally. This rules out most occupation exposures and strongly suggests that a communal source, such as contaminated water, is the more likely cause of the observed results. As both manganese and arsenic are common minerals in parts of Colorado, especially where gold and silver mining were occurring during the period, it is easy to see how tailings from the mines would get into the water and flow downstream where the community washed, drank and cooked.

The lead and mercury provide a more differentiated picture. Looking at the second principal component, with the exception of one low result for males, the levels of these elements are overall higher in males than in females, suggesting different levels of exposure to these two elements. Given the high levels of lead observed in some individuals, it is possible that this was partially through the ground water, but as the lead levels in men are consistently higher, it also

suggests they had additional routes of exposure. There are few cases where mercury and lead exposure would have occurred for men but not for women, with mining being an obvious exception. Both elements were also common in a number of construction materials, such as glass and wood, so that is another possible source of toxic exposure for men. Occasionally lead and mercury also had uses in farming, mainly as pesticides, so that is also a way men might have been over-exposed compared to women. The three most common occupations listed for men at the asylum were miners, laborers and farmers, which together constitute 60% of the men in the institution, so the higher levels of lead and mercury are entirely in line with what might be expected given their trades.

After gold was discovered, the population of Colorado exploded with the vast majority of migrants coming from either the Eastern United States or Europe. Therefore, another possibility for exposure to lead and mercury is the location the individuals came from originally. The settlements in Colorado were very recent and so the vast majority of people were not born in the state; of the nearly 2,000 patients that passed through the asylum when the graveyard was in use, only 64 were born in Colorado (Mitchell et al. 2002). The rest of the population came from the Eastern United States, Western Europe, a few from Central Europe, and even one individual from China. This means that the bone chemistry could more accurately reflect the varied locations from which the patients originated or lived in rather than their occupation. There is no way to be sure of the amounts of elements that represent recent exposure versus older contamination without testing the teeth, which do not change in composition over the course of a lifetime, and thus only reflect the elemental conditions of adolescence or younger. This explanation for the levels found also seems likely as those from further away would be less likely

to have family nearby to accept the body for burial, providing a greater chance of being buried on the grounds.

Whatever the underlying cause of the variation that could not be accounted for in the principal component analyses, it is unlikely that being a patient in the asylum or one deemed insane more generally accounts for the results seen. Without a representative collection for the same period and ideally the same area as the asylum it is not possible to account for the causes of elemental variation in the sample. However, if the overall element levels represent asylum life, there should not be sex differentiation nor individuals who fall well outside the expected distribution of elements. This makes the outlier data all the more valuable as it seems to clearly indicate that those who had toxic levels of elements in their bones were not simply the victims of high exposure through the asylum but were actually committed for symptoms commonly associated with high levels of neurotoxins.

Chapter VI: Summary and Conclusions

Whether or not insanity can truly be ascertained from only the skeletal remains of an individual is a question that is not easy to answer. In the case of the Colorado State Asylum collection, it assumes that in the majority of cases, institutionalization reflected real mental disorders and not simply a more stringent standard of normalcy that criminalized unusual behavior. Even in the cases where there is a biological basis for insanity, most currently recognized disorders of the mind do not leave any osteological trace, further reducing the possible skeletal manifestations.

With these caveats in mind, it is possible to make some statements about the collection based on the study of cranial abnormalities and trace element analysis. Although the specific cause could not be ascertained through radiographs, it is clear that there is something abnormal about the heaviest skulls in the collection. The presence of more than one in a small sample does suggest that the nature of the collection itself, an asylum, might have selected for the condition responsible for the heavy crania. Thus, the most likely explanations for the heavy skulls are Paget 's disease, *hyperostosis frontal interna* or Dilantin intoxication.

The results of the bone chemistry analysis are even more telling. Although the mercury did not yield the anticipated results and the mercury levels were in fact quite low overall, many of the other elements did reflect high levels of exposure. In particular, the manganese, lead and arsenic levels exhibited in a few individuals might have been not only a neurotoxin, but also fatal. As these elements are also tied to mental disruptions, it is possible to see how these results can indicate the special nature of an asylum rather than a cemetery of society as a whole.

The first step in clarifying these issues would be to widen the scope of research on the collection itself. Only heavy crania were examined, but there are a number of other notable

abnormalities present, including a high number of individuals showing early cranial suture closure and at least two individuals who appear to have congenital conditions. Testing more of the collection would also be vital. The skeletons that had the most dramatic results for elements such as arsenic and lead were not specifically selected because of their likelihood of having notable element levels but rather because they fit into certain age, sex and cemetery location specifications. Testing of more skeletons might reveal other unexpected results for element exposure.

Once the Colorado Insane Asylum collection has been fully examined, it could be compared to other skeletal collections. Ideally, results from an asylum at the same period but different location, such as the Oneida County Asylum in New York examined by Phillips (2001) and a general community cemetery from late 19th century Colorado could be obtained. By determining how the skeletons from Pueblo most resemble another asylum and where they most resemble an adjacent community, may make it possible to isolate which conditions and elements represent insanity; in the cases where the asylums are similar insanity could be assumed as the cause of the parallels and it would be possible to eliminate results where the Colorado asylum and the control sample are more alike. With this information, one might indeed be able to find insanity in the skeleton.

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