Death by smallpox

investigating the relationship between anaemia and viruses

in 18th and 19th century South Africa

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The historical record combined with the presence of large numbers of individuals exhibiting skeletal responses to anaemia (porotic hyperostosis and cribra orbitalia; PH and CO) are the main reasons for investigating the presence of smallpox in three South African communities, Griqua, Khoe, and 'Black' African, during the 18th and 19th centuries. The smallpox virus (variola) raged throughout South Africa every twenty or thirty years during the eighteenth and nineteenth centuries and was responsible for the destruction of entire communities. It has an 80 to 90 per cent fatality rate among non-immune populations (Aufderheide & Rodríguez-Martín 1998; Young 1998) and all ages are susceptible. The variola virus can only survive in densely populated areas and therefore sedentary communities, such as those present in agricultural and pastoral based societies, are more susceptible to acquiring the disease.

Smallpox may remodel bone in the form of osteomyelitis variolosa ('smallpox arthritis') (Aufderheide & Rodríguez-Martín 1998; Jackes 1983; Ortner & Putschar 1985) which causes the reduction of longitudinal bone growth (Jackes 1983). However, since smallpox only remodels bone in very few individuals and solely in children the only method for unconditionally determining the presence of the smallpox virus in a skeletal population is by performing DNA and PCR analyses.

Survival from smallpox affords the individual natural immunity for the remainder of their life. The virus is undetectable in a smallpox survivor as they will possess the antibodies for the disease and therefore will have gained natural immunity for the remainder of his or her life. Therefore, positive identification of the viral DNA will only be present in individuals who died from the virus. Although the ancient DNA results were inconclusive, in this project, for the smallpox virus, its presence in these communities during the nineteenth and twentieth centuries combined with the high rates of PH and CO suggest a possible relationship between the cause of death and smallpox epidemics.

Presently, there is no direct link between smallpox and porotic hyperostosis or cribra orbitalia, however, there is a link between smallpox and iron deficiency anaemia. Since viruses lack the vital features for energy production, they are entirely dependent upon a living cell for reproduction. The host cell provides the necessary energy, raw material, and machinery for reproduction (Aufderheide & Rodriguez-Martin 1998); viruses require iron in order to survive.

The Griqua community is represented by five different settlements: Campbell, Danielskuil, Philippolis, Bethulie and de Tuin in the Northern Cape and Free State provinces (figure 1). All of these sites are dated to the nineteenth century although one of the individuals in the Danielskuil sample may be from the early 20th century. These people were primarily pastoralists, growing gardens of marijuana and hemp and hunting animals; however large scale agriculture was introduced by the missionaries in 1804 (Legassick 1989).

The Khoe skeletal remains are associated with an indigenous population who died during the smallpox epidemic of 1866 in the town of Colesberg (Slome 1929; South African Museum Catalogue) (figure 1). The Khoe labourers lived on the edge of the town in family-based communities, i.e. men, women, and children all residing together in an area of proximity; the Khoe community provided the rural and urban population with a cheap source of labour. The population was almost entirely pastoral. The most important agricultural product was wheat, then barley and rye, oats, maize, and finally peas and beans (Government Documents, G.1-1866; Van de Sandt

1843). With the exception of the inhabitants of the village who were mostly shop keepers or artisans, the entire population was engaged in stock farming of sheep and cattle.

The historical and ecological information suggests that the people buried at the Wolmaransstad cemetery (figure 1) were 'Black' Africans, from Sotho-Tswana communities, who were eventually colonised and reduced to farm labourers for the benefit of Colonial farm owners. The people living in this community were engaged in an agricultural-based subsistence (Maggs 1976).

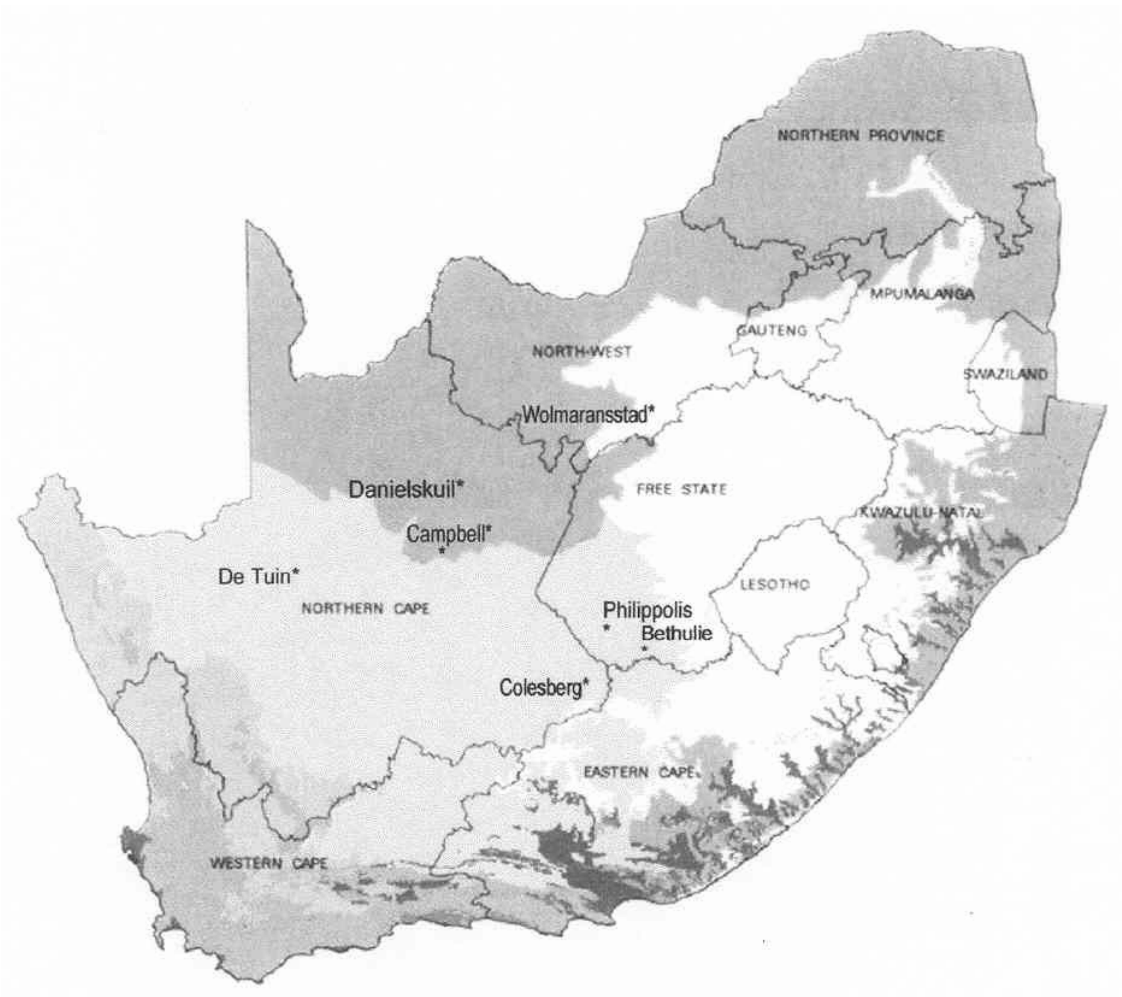

Figure 1: Map of South Africa with archaeological sites employed for this project (Developed from Low and Rebelo 1996).

The relationship between anaemia and smallpox

Porotic hyperostosis and cribra orbitalia (PH and CO) are similarly appearing skeletal responses to anaemia, PH occurring on the skull vault (figure 2) and CO on the orbital roof (figure 3). They are manifested as pitting on the bone; it looks as though someone took a sharp pin and made tiny holes in a piece of clay.

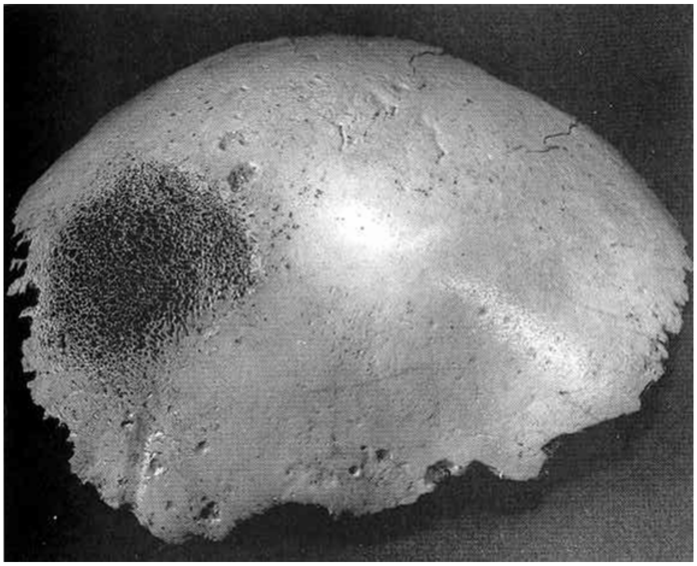

Figure 2: Porotic Hyperostosis (PH) – skeletal response to anaemia (Aufderheide & Rodríguez-Martín 1998:349).

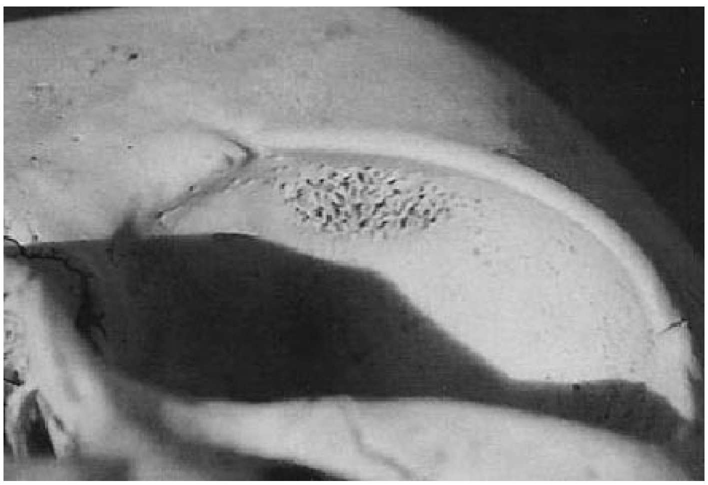

Figure 2: Cribra Orbitalia (CO) – skeletal response to anaemia (Fairgrieve & Molto 2000:324).

Although the primary consensus for the cause of PH and CO is iron deficiency anaemia, many factors can explain its occurrence. PH and CO are linked to diets that are either low in iron or that contain substances that interfere with iron absorption, otherwise known as the 'maize-dependency' model (El-Najjar 1976; El-Najjar & Robertson 1976; El-Najjar *et al.* 1975, 1976, 1982; Henschen 1961; Lallo *et al.* 1977; Mensforth *et al.* 1978). Tannins which are found in vegetables inhibit iron absorption. Cereal grains, especially maize, have a very low iron content and contain phytic acid which binds to any iron present, rendering it unusable (Garn 1992). Adding to the problem is that many cereal grains and beans lack the enzyme phytase which counteracts the phytic acid. Also, haeme iron (found in animal organs and skeletal muscle) has about a 20 to 40 per cent absorption rate while non-haeme iron (found in grains and vegetables) has an approximately 5 per cent absorption rate when consumed alone, i.e. without meat (Arthur & Isbister 1987; Dallman 1986; Holland & O'Brien 1997). Therefore populations dependent on a vegetable staple will suffer higher rates of iron deficiency anaemia which will create the characteristics of PH and CO.

The high rates of PH and CO in the Griqua, Colesberg and Wolmaransstad communities could be linked to their agricultural economies; maize has a low iron content and the phytic acid chelates any iron that may be present. However, there could also be other factors involved.

Agricultural subsistence strategies produced a deterioration of hygienic conditions that accompanied population growth and an increase in sedentism, which created prime situations for pathogenic infestation. Populations with high rates of iron deficiency anaemia generally also have high rates of infectious disease. During an infectious process the availability of iron for invading microorganisms is limited by two factors: firstly, the rapid sequestration of iron in tissue storage forms, and second, the presence of iron transport proteins i.e. unsaturated transferrin and lactoferrin (transferrin present in milk). These iron transport proteins have strong binding affinities for iron that allow them to withhold iron from invading micro-organisms; bacteria and viruses require iron to reproduce. Lactoferrin is released by phagocytising white blood cells in areas of inflammation to help restrain bacterial/viral growth in the immediate area. However, in iron deficient patients lymphocyte numbers are reduced (Fletcher *et al.* 1975). There is also a redistribution of iron into the liver, spleen and marrow which has two effects: decreasing the availability of iron throughout body fluids and increasing the percentage of unsaturated transferrin circulating in the body.

Another possible contributing factor associated with PH and CO is geography. Campbell, Danielskuil, de Tuin, and Wolmaransstad are all geographically located within the 'hookworm belt'(Holland & O'Brien 1997). A single hookworm can remove between 0.2 ml and 0.05 ml of blood per day which can lead to a negative iron balance when large numbers of worms are involved (Cook 1980). Although the prevalence of hookworm in South Africa ranges from 100 per cent to about 40 per cent within individuals the intensities of infection (eggs/g faeces) are generally low by world standards (personal communication Professor Appleton, 2002).

Ground housing as opposed to pile housing provides a greater chance of exposure to pathogens which in turn is related to high levels of iron deficiency anaemia (Dunn 1972). Many of the indigenous people at the Griqua, Colesberg and Wolmaransstad sites may have been living in traditional housing.

The Griqua population shows a high frequency of PH and CO in juveniles. Since increased iron deficiency is related to infectious disease and children are more susceptible to infection than other age groups, due to their low immunity, it would not seem unusual to have large percentages of PH and CO in the childhood years. Iron deficiency in children is also associated with poor maternal diets. As well, if a child is breast fed for longer than four months or weaned onto an iron poor cereal gruel, e.g. maize, iron deficiency anaemia will develop. Goat's milk is also a poor alternative to human milk as it is lacking in folic acid, which in turn can lead to iron deficiency in infants and therefore the skeletal manifestations of PH and CO (Fairgrieve & Molto 2000).

Chronic iron deficiency may also be related to the bovine milk diet. Bovine milk contains only one-tenth as much lactoferrin as human milk. Lactoferrin makes iron unavailable to invading organisms and therefore human milk is a better defence mechanism than cow's milk (Weinberg 1984). Since the community at de Tuin raised goats and cows it is possible that either a prolonged breast feeding or weaning onto goat or cow's milk or a combination were responsible for the PH and CO lesions in the children.

Active versus *healed* PH and CO lesions are important indicators when correlating anaemia with bioarchaeology. Skeletal remains displaying *active* lesions suggest that the 'disease' was still present when the individual died (Grauer 1993). *Healed* lesions suggest that the body was in the process of recovering, or had recovered from, the condition (Grauer 1993).

Stuart-Macadam (1985) states that PH and CO present in adults is most probably representative of an anaemia acquired during early childhood. In this scenario, *active* lesions in adults would represent *unhealed* lesions existing from childhood therefore implying a population chronically iron deficient; the body is not 'fit' enough to repair the skeletal 'damage' of PH and CO. The problem arises when there is both *active* and *healed* lesions present in adult individuals. How can the population be chronically iron deficient and yet still show signs of healed PH and CO?

Although the bony changes associated with PH and CO could be related to maize consumption in all of the samples, the question still remains, why were these people dying? In all three communities, the pitting associated with PH and CO was more frequently categorised as *active* (suggesting that the 'disease process' was in its initial stages when the person died) than as *healed* (suggesting that the body was in the process of recovering, or had recovered from, the condition); smallpox has an 80 to 90 per cent fatality rate among non-immune populations. Since people do not normally die from anaemia, and the smallpox virus raged throughout South Africa during the eighteenth and nineteenth centuries, it is more likely that the high rates of PH and CO are possibly related to smallpox epidemics and not dietary causes.

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References

Appleton, Christopher. 2002. Professor, Centre for Integrated Health Research, Department of Zoology & Entomology, University of Natal, South Africa.

Arthur CK, & Isbister JP. 1987. Iron deficiency, misunderstood, misdiagnosed and mistreated. *Drugs* 33:171-182.

Aufderheide AC, & Rodríguez-Martín C. 1998. *The Cambridge encyclopedia of human paleopathology*. Cambridge: Cambridge University Press.

Cook GC. 1980. *Tropical gastroenterology*. New York: Oxford University Press.

Dallman PR. 1986. Biochemical basis for manifestations of iron deficiency. *Annual Review of Nutrition* 6:13-40.

Dunn FL. 1972. Intestinal parasitism in Malayan aborigines (Orang Asli). *Bulletin of the World Health Organization* 46:99-113.

El-Najjar MY. 1976. Maize, malaria and the anemias in the Pre-Columbian New World. *Yearbook of Physical Anthropology* 20:329-337.

El-Najjar MY, Lozoff B, Ryan DJ. 1975. The paleoepidemiology of porotic hyperostosis in the American Southwest: radiological and ecological considerations. *American Journal of Roentgenology and Radiation Therapy* 25:918-924.

El-Najjar MY, Andrews J, Moore G, Bragg DG. 1982. Iron deficiency anemia in two prehistoric American Indian skeletons: a dietary hypothesis. *Plains Anthropologist* 44:447-448.

El-Najjar MY, & Robertson JR. 1976. Spongy bones in prehistoric America. *Science* 193:141-143.

Fairgrieve SI, & Molto JE. 2000. Cribra orbitalia in two temporally disjunct population samples from the Dakhleh Oasis, Egypt. *American Journal of Physical Anthropology* 111:319-31.

Fletcher J, Mather J, Lewis MJ, Whiting G. 1975. Mouth lesions in iron-deficient anemia: relationship to *Candida albicans* in saliva and to impairment of lymphocyte transformation. *Journal of Infectious Disease* 131:44-50.

Garn SM. 1992. The iron-deficiency anemias and their skeletal manifestations. In: Stuart-Macadam P, Kent S, editors. Diet, demography, and disease: changing perspectives on anemia. New York: Aldine De Gruyter. p. 33-62.

Grauer AL. 1993. Patterns of anemia and infection from Medieval York, England. *American Journal of Physical Anthropology* 91:203-13.

Henschen P. 1961. Cribra cranii – a skull condition said to be of racial or geographical nature. *Patholgie et Microbiologie* 24:724-729.

Holland TD, & O'Brien MJ. 1997. Parasites, porotic hyperostosis, and the implications of changing perspectives. *American Antiquity* 62(2):183-93.

Jackes, MK. 1983. Osteological evidence for smallpox: a possible case from seventeenth century Ontario. *American Journal of Physical Anthropology* 60:75-81.

Lallo J, Armelagos GJ, Mensforth RP. 1977. The role of diet, disease and physiology in the origin of porotic hyperostosis. *Human Biology* 49:471-483.

Legassick Martin 1989. The Northern Frontier to c. 1840: The rise and decline of the Griqua people. In: Elphick R, Giliomee H, editors. *The Shaping of South Africa Society, 1652-1840*. Connecticut: Wesleyan University Press. p 358-411.

Low AB, & Rebelo AG. 1996. *Vegetation of South Africa, Lesotho and Swaziland*. Pretoria: Department of Environmental Affairs and Tourism.

Maggs TM. 1976. *Iron age communities of the southern Highveld*. Occasional publications of the Natal Museum, No.2.

Mensforth RP, Lovejoy CO, Lallo JW, and Armelagos GJ. 1978. The role of constitutional factors, diet, and infectious disease in the etiology of porotic hyperostosis and periosteal reactions in prehistoric infants and children. *Medical Anthropology* 2:1-59.

Ortner DJ, & Putschar WGJ. 1985. *Identification of pathological conditions in human skeletal remains*. Washington, DC: Smithsonian Institution Press.

Slome D. 1929. The osteology of a Bushman tribe. *Annals of South Africa Museum* 24:33-60.

South African Museum Catalogue of human skeletal remains. South African Museum, Cape Town.

Stuart-Macadam P. 1985. Porotic hyperostosis: representative of a childhood condition. *American Journal of Physical Anthropology* 66:391-398.

Van de Sandt R. 1843. *Cape of Good Hope Almanac and Annual Register for 1843*. Cape Town: Saul Solomon Press. p 449.

Weinberg ED. 1984. Iron withholding: a defense against infection and neoplasia. *Physiological Reviews* 64(1):65-102.

Government Documents Library, University of Cape Town

G.1-1866 Census of the Colony of the Cape of Good Hope 1865. In: Cape of Good Hope Annexures to Votes and Proceedings 1866.