Chapter 5 – Factors Affecting Bone Density

This chapter intends to highlight the main variables that control and affect bone density levels. This information can be used in chapter 8 (results and discussion) when the reasons for variation in the experimentally derived data are explored. Each factor will be discussed in turn.

5.1: Introduction

The previous chapters have highlighted the importance of bone density as a mediating factor for the mechanical destruction of archaeological bone as a result of taphonomic processes. It is clear from this that an understanding of how bone density varies between and within individuals will contribute significantly to the archaeologist's ability to interpret bone assemblages reliably. Archaeologists already have a keen understanding that bone density can vary according to taxa and element. Occasionally, an appreciation of the effects of other variables (for example, age at death, sex or diet) on bone density has been shown. Unfortunately such recognition is often vague and seldom backed up by experimental data.

This project will address questions of bone density variation in sheep skeletons. Perhaps unsurprisingly, there is little literature that refers explicitly to this topic. Instead, the vast majority of the literature that addresses the effects of various factors on bone density concerns live animals and most of this relates specifically to humans. This section will draw upon such material, where relevant, in order to provide an indication of how different variables might affect the bone density of the sample.

Weber (1999 p194) notes that for humans, "genetic factors, age, race, general health, exercise, cigarette smoking as well as nutritional factors ... determine the risk for osteoporosis". This is in general agreement with Ott (1991 p1646), who claims that "currently identified determinates of peak bone mass include race and heredity, hormonal status, nutrition, activity, weight and strength". Below is a discussion of some of the main determinates listed by Weber (1999) and Ott (1991) (and others) and how they might affect the density of a skeleton.

5.2: The Impact of Each Factor on Bone Density

5.2.1: Skeletal location

Perhaps the most obvious factor that is associated with variation in bone density is skeletal location. Different bones (or parts of bones) at different skeletal locations have for a long time been known to have different bone densities. Many archaeological studies (discussed in the next chapter) have endeavoured to establish how bone density varies across the skeletons of a variety of different species.

Variation in bone density according to skeletal location has been attributed to the function of bones during the life of an animal. According to Wolf's law, bony structures remodel in order to fit them to their function (Currey 1984 p252). In other words, the form and chemical constitution of bones change so that they are best able to withstand the mechanical pressures to which they are subjected. This adaptation can take place over an evolutionary time scale (eg the development of bone shafts as hollow cylinders) or within the lifetime of an individual (eg the increase in density of the arm bones of racket sports players (Kannus *et al* 1995 – see below). Such adaptation frequently takes the form of changes in bone density.

Valuable insights into the variation of bone density across the skeleton (and its causes) have been provided by Kreutzer (1992) and Pavao and Stahl (1999). These authors have linked differences in bone density across skeletons of different species with the functional anatomy of those species. In these studies, weight bearing bones, or those associated with impact involved in locomotion, were shown to be among the densest in the skeleton.

The variation of bone density according to skeletal location is well established. This variation is a reflection of variation in the mechanical stresses experienced by different bones throughout the normal life of an animal. Currey (1984) has described at length the adaptive reaction of the skeleton to these mechanical stresses.

5.2.2: Age

The main focus of this project is to explore how bone density varies with the age of an animal. It seems natural, therefore, that age is the first variable to be addressed here.

Trotter and Hixon (1974) carried out a survey of 426 human skeletons ranging from 16 weeks' gestation to 100 years of age. They recorded an increase in bone density between 30 weeks before birth and approximately 20 years of age. This more or less

steady increase was punctuated immediately following birth by a sudden drop and then recovery of bone density (see figure 5.1).
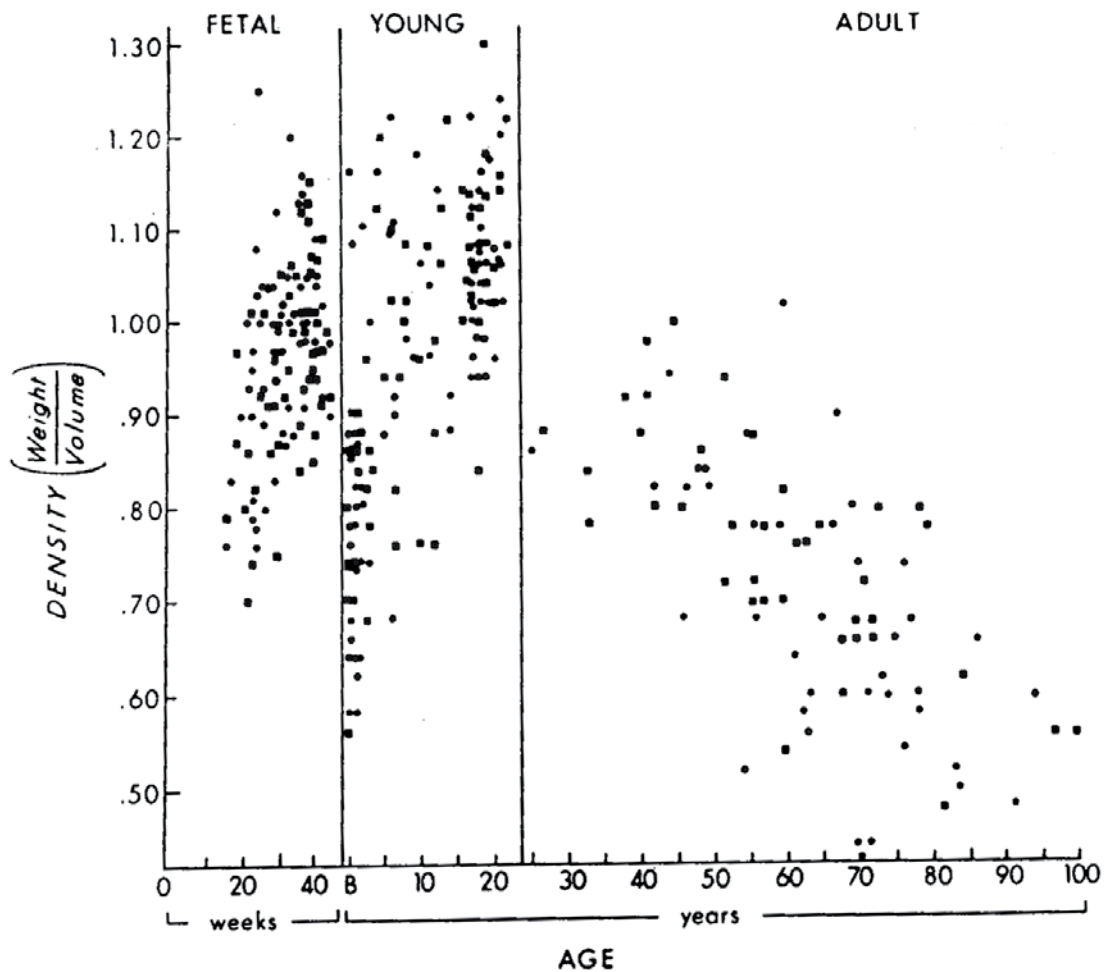

Figure 5.1: Showing the results of research by Trotter and Hixon onmale and female humans. Around the time of birth there is a sudden loss of bone density, followed by an increase in early life. After approximately 20 years of age, bone density can be seen to reduce. After Trotter and Hixon (1974 p12 Fig 2).

Trotter and Hixon (1974 p10) have suggested that this feature of bone development is the result of a rapid growth of bone size, without a concordant increase in bone weight. This feature is probably directly connected to the similar temporary drop of calcium levels and calcium:nitrogen ratio in very young human bone (Dickerson 1962b p60). Similar observations have been made for the bone of dogs (Burns and Henderson 1936 p1208), pigs (Dickerson 1962a p49), rabbits (Weidmann and Rogers 1958 p339) and cats (Burns and Henderson 1936 p1208, Weidmann and Rogers 1958 p339). The exact

timing and extent of these changes is both species and site specific and may be the result of rapid bone growth in the first days of life (Burns and Henderson 1936 p1208)

Other studies that have focussed on bone changes in early life have broadly repeated the observations described above (Binford and Bertram 1977 p109, Currey 1969 p462 and Mueller *et al* 1966 p145 and Thomas *et al* 1991 pp52 - 53). However, none of these works examined individuals that were less than six months old and so the features associated with the first few weeks of life, described above, were not recognised. The potential impact of early changes in bone density on archaeological interpretation has been recognised by Guy *et al* (1997).

Trotter and Hixon (1974 p12) also noted a decrease in the bone density of humans that begins at some stage between the mid-twenties and mid-forties and results in approximately 15.6g of bone being lost from the adult skeleton every year (Trotter and Hixon 1974 p11). A more recent study has refined the timing of the earliest stages of this decrease to the early twenties (Teegarden *et al* 1999 p1014). If this decrease is particularly rapid or if the bone density is low at the time of its onset, osteoporosis may result.

The majority of studies that have focussed on bone density changes in later life were concerned with osteoporosis in humans (Brickley 1997, Currey 1979b p461, Kneissel *et al* 1994 p543, Lees *et al* 1993, Ortner and Putschar 1981 p289, Trotter and Hixon 1974 p5). Other factors that control the impact of osteoporosis (sex, nutrition, breed etc.) will be discussed in the relevant sections below. Age related osteoporosis is comparatively rare in sheep, since they tend to be killed by their herders once they become barren (before osteoporosis can develop) (pers. com. John Martin 15.11.00). There is a lack of data on the effects of old age on the bone density of sheep. This project will aim to redress this.

There can be little doubt that the density of an animal's bones changes as it matures and grows old. Previous studies have identified a rapid increase of density in fetal bone, followed by a sudden decrease immediately after birth. Density levels quickly recover from this dip and continue to increase until the age of about 20 (in humans) when it starts to fall gradually. The precise nature of these bone changes is dependent on the species and bone site involved, as well as other factors (which will be discussed below). Little is known about the nature of these changes in sheep and it is an intention of this project to redress this.

5.2.3: Disease

There is a wide range of diseases that can have either a positive or a negative impact on the density of a subject's bones. It would be inappropriate to explore here exactly how each of these numerous disorders affects bone density or which factors control them, as this has been done elsewhere (Baker and Brothwell 1980). Instead, this section aims to explore the potential impact of disease on bone density in more general terms.

Osteoporosis is probably the bone disorder most commonly detected in humans. Osteoporosis is a condition that is characterised by a reduction in the amount of bone tissue in the skeleton, leaving the bones weak and the sufferer prone to bone fracture. It occurs when the natural process of bone remodelling is disrupted to such an extent that the patient is at serious risk of bone fracture (Brickley 1997 p35). The factors that contribute to this disruption are numerous and often little understood. As mentioned in the previous section, age is an important factor in controlling the progress of bone loss, with older individuals being more prone to (or more affected by) osteoporosis. Other factors that have been suggested to contribute to osteoporosis will be mentioned in the relevant sections below. First, some of the *pathological* conditions that have been shown to lead to osteoporosis will be described.

Parasitic infestation has the potential to affect the skeleton. A series of experiments on sheep by Wilson and Field (1983) demonstrated that infestation by the larvae of the parasite *Trichostrongylus colubriformis* resulted in 30% less phosphorus being absorbed through the animals' digestive tracts. The infected animals also excreted phosphorus and calcium into their small intestines. These two processes eventually led to a measurable loss of mineral from the skeleton. Animals that are densely stocked will be more prone to parasitic infestation and in cases of infestation of an entire herd, abnormalities of the skeleton may be more difficult to detect since they will not stand out as being unusual (Baker 1978 p111).

A pathological mechanism by which mineral can be removed from the skeleton is chronic renal failure (Baker 1978 p111). Such cases can lead to over activity of the parathyroid gland, resulting in excessive resorption of mineral from the skeleton – especially from the skull and vertebrae. In extreme cases this disorder can leave the live skeleton with a rubbery form (hence the common name for the condition: "rubber jaw"). The affected bones will also be light and fragile, typically with a perforated skull (Baker and Brothwell 1980 pp 54 - 55). This condition is relatively rare and unlikely to have a considerable impact in the archaeological collection. Also, the physical symptoms

described by Baker and Brothwell (1980 pp 54 - 55) have, where they occur, been relatively easily identified in archaeological material.

A bone disorder more commonly noted in the archaeological record is rickets. Rickets can affect herbivores as well as carnivores (but more commonly the former) and results in a variety of morphological changes (Baker and Brothwell 1980 p49) as well as a reduction in the density of the skeleton (Trotter and Hixon 1974 p2). The condition affects the rapidly growing bones of young animals and can be caused by an imbalance of the calcium:phosphorus ratio in the diet of cats, dogs and chickens, or a deficiency of vitamin D in the diet of herbivores.

The three pathological conditions, briefly described above, are known to result in a reduction in the density of the bones of the sufferer. Different animals have different susceptibilities to these disorders and they often affect the skeleton in a non-uniform manner. Some conditions do not affect the skeleton at all and some (eg osteodystrophia fibrosa (Baker and Brothwell 1980 p61) and osteopetrosis (Trotter and Hixon 1974 p2)) result in an increase in skeletal mass. A full description of a much wider range of pathological conditions, including how they can affect bone mass and the relative mineral content of the skeleton, is available in Baker and Brothwell (1980 especially page 48).

A lack of predictability as well as a lack of research means that it is practically impossible to use density data relating to pathological bone to predict the taphonomic destruction of such material for any particular assemblage. However, pathological conditions are generally rare within an assemblage and they can often be identified by a simple macroscopic examination. Consequently, the preferential removal or preservation of diseased bone is unlikely to have a serious impact on an assemblage as a whole and any potential for misinterpretation can be identified.

This section began with the statement that osteoporotic bone could be more susceptible to taphonomic destruction. The following section examines some of the factors that can control the onset and progression of osteoporosis.

Pathological conditions can either increase or decrease the density of the bones that they affect. However, these conditions are usually rare and, when they do occur, can be easily identified. An exception to this rule is in parasitic infestation, when the disorder may be ubiquitous and so may not be recognised as being unusual. Perhaps the

most common disorder that leads to bone loss is osteoporosis. The factors that can contribute to this condition will be discussed below.

5.2.4: Physical exercise

It has been recognised for some time that physical exercise can have an impact (positive or negative) on the bone density of an individual. Research into this matter has focussed on the reduction or prevention of osteoporosis in humans. This section will summarise the findings of some of this research as well as exploring studies into the effects of exercise on animals.

Numerous studies have demonstrated that individuals engaged in a regular regime of exercise will have a higher bone mass (a higher bone density) (Ekenman *et al* 1995 p358, Johnston and Slemenda 1993 pS54, Kannus *et al* 1995 p29, Lees *et al* 1993 p675, Ott 1991 p1646). However, Bourrin *et al* (1994 p2001), in their experiments on rats, noted that when the level of exercise was very high, bone mass was reduced (by 6%). The level of exercise at which its effects change from promoting bone density to reducing it is not known.

The effects of exercise on bone density will not necessarily be apparent in the entire skeleton. Bartosiewicz *et al* (1993) has identified an increase of both size and density of the metapodia of cattle that were used as draught animals, as opposed to those of cattle not engaged in such strenuous activities. Similarly, the deleterious effects of over-exercise in rats were limited to the weight bearing bones (Bourrin *et al* 1994 p2001). This non-uniformity of bone change is graphically illustrated by the findings of Kannus *et al* (1995 p29), who reported that the bone density of a sample of racket sports players was elevated only in the dominant arm (by a factor of 2 - 4).

Furthermore, the effect of physical activity on the skeleton tends to vary according to the age of the individual in question. Bourrin *et al* (1994 p1999) reported that bone density only fell in over-exercised *young* animals. Kannus *et al* (1995 p29) reported that human females who started playing racket sports before the menarche would be more likely to have a higher bone density than those who took up their sport later in life, regardless of the number of years they had been playing. There is little doubt that high levels of physical activity in early life can increase bone density. Similar levels of exercise in later life will not increase an individual's bone density, but will serve to reduce the rate at which bone is lost with the onset of old age (Ekeman *et al* 1995 p358). The point at which this transition occurs (or if it occurs at all in some animals) is unclear, but in human females it seems likely to be at the menopause.

With the exception of extreme cases (Bourrin *et al* 1994), exercise will increase bone density. However, the nature and extent of this effect are dependent on the age of the animal, and the type and intensity of the exercise involved. These findings are potentially important since there might be expected to be a difference in bone density (and therefore potential to survive taphonomic processes) between draught animals and those that are penned or tethered and so have little opportunity to exercise. Any attempt to use modern reference material to record the bone density of any given taxa or taxon (in this case, sheep) should take into account potential biases caused by differences in the levels of exercise of individuals from within the sample.

5.2.5: Sex

Among the main hormones associated with bone growth are oestrogens. These hormones, associated with ovarian activity, have been implicated as being of central importance in osteoporotic bone loss among older women. Disruption of ovarian function can be caused by the menopause or by surgical ovarectomy and will result in the disruption of bone turnover, and subsequent osteoporotic bone loss (Brickley 1997 p51). It is well established that women are more prone to osteoporosis than men (Ekenman *et al* 1995 p356, Kneissel 1994 pp543 - 544, Lees *et al* 1993 p674, Silberg and Silberg 1971 pp406 - 411). Men are affected by osteoporosis (albeit to a lesser extent), although the causal factors are less fully understood (Gooren 1998 p155). No matter what the cause, osteoporotic bone is less dense and more prone to fracture than normal bone. There is little evidence that taxa other than humans are susceptible to osteoporotic bone loss in old age (see section 5.2.2).

There are factors other than age that can contribute to a decrease in the bone density of females rather than males. The stresses of pregnancy and lactation can result in mineral deficiencies in the body of the mother (and seldom of the offspring (Benzie *et al* 1955 p426)), leading to bone loss (Lees *et al* 1993 p675, Ott 1991 p1646). It will become apparent that bone loss due to parity and lactation will only occur when the diet of the mother is in some way deficient (see section 5.2.6). Consequently, this matter will be discussed in more detail in the next section, which focuses on the effects of diet on bone density.

A factor that is likely to cause a reduction in bone density in early life is castration. Davis (2000 p383) notes that the epiphyses of females will naturally fuse at an earlier age than those of males. However, if the males are castrated at an early age, a

third pattern of bone development emerges. In such cases, the bones of castrates will fuse much later than those of both males and females (Davis 2000 p383). This delay results in longer limb bones in castrates and has been observed in a variety of taxa (Silberg and Silberg 1971 pp444 - 447). The age at which an animal is castrated is of some importance here, since the development of only the bones that are unfused when castration occurs will be retarded, (Davis 2000 p382). It is not clear that a delay in skeletal fusion will subsequently lead to either higher or lower bone density. By using an experimental sample that includes castrates as well as males and females, this project will be able to begin to address this question. Although no direct link between epiphysial fusion and bone density has been demonstrated, research has demonstrated that the bones of castrates can be porous or more fragile. Silberg and Silberg (1971 pp444 - 447) have provided an extensive review of the literature that examines the skeletal effects of castration in a wide variety of animals. This review includes references to studies that have noted that castration produces a less robust skeleton in sheep (Hammond, J. (1932) *The Growth and Development of Mutton Qualities in Sheep*. Oliver and Boyd, Edinburgh, cited in Silberg and Silberg (1971 p445)) and higher porosity in humans (Labhart and Corvoisier (1950) *Helvetica Medica Acta* Vol. 17 p457, cited in Silberg and Silberg 1971 p444. Ravault, Vignon and Fraisse *Revue du Rhumatisme* Vol 17 p247, cited in Silberg and Silberg 1971 p444).

The sex of an individual can clearly affect the density of its bones. Hormonal deficiencies in females following interruption of ovarian activity are well known to contribute to osteoporosis in humans. This project will endeavour to establish whether or not the same is true in other taxa. Females are also more prone to osteoporotic bone loss if their diet is deficient during periods of pregnancy or lactation. This will be discussed below. Finally, castration can affect both the density of bones and their fusion times. Whether fusion times are connected with bone density will also be explored in this project.

5.2.6: Nutrition
The fact that the diet of an animal can have an impact on its bone density has already been alluded to (see section 5.2.3 and 5.2.5) and will be discussed here in more detail. A considerable body of literature exists that examines the effects of nutrition on the bones of animals (including sheep). This means that, in this section, it is possible to concentrate specifically on the nutrition of animals, rather than humans.

Reproductively active female animals undergo an annual cycle of bone loss and recovery. This cycle involves a dramatic loss of calcium and phosphorus from the skeleton of the mother during seasons of nutritional stress (winter and early spring (Hindlang and Maclean 1997 p199)) and during lactation. Under normal circumstances, mineral lost in this way will be replenished during the ensuing summer and autumn, leaving the skeleton in a healthy condition the following winter when the cycle can begin again. However, if the diet of the mother is deficient, her skeleton will not be able to recover fully by the following spring. If this cycle of incomplete recovery continues, her skeleton will decrease in density – the trabecular bone being affected before the cortical bone (Benzie *et al* 1955 p426, Horwitz and Smith 1990 p655). The weight bearing bones are affected the least, while the non-weight bearing bones (in sheep, the vertebrae) will lose density first (Benzie *et al* 1955 p437). If these circumstances are allowed to persist, nutritionally stressed animals may have as much as 25% less bone density than those with an adequate diet (Benzie *et al* 1955 p426). The effects of this type of bone density on skeletal strength are complex, but will almost certainly eventually lead to the bones becoming more prone to fracture (Currey and Hughes 1973 pp119 - 122). Observations such as these have enabled the interpretation of cortical bone thinning as being due to the exploitation of sheep for milk (Horwitz and Smith 1991 p33) (such an interpretation is reliant on the assumption that other factors likely to affect bone density remain constant). This cycle of density loss and recovery is seasonal in nature. It is for this reason that the relative densities of sheep that died at different times of the year will be addressed by this project.

Regardless of whether the animal is reproductively active or not, temporary nutritional deficiencies can lead to temporary loss of bone mineral. Furthermore, long-lasting deficiencies in the diet (especially of young animals) can lead to a more permanent reduction in bone density in later life. Dietary deficiencies that are known to affect the skeleton adversely can take the form of either a lack of calcium (Baker and Brothwell 1980 p52, Ott 1991 p1646, Teegarten *et al* 1999) or a lack of vitamin D, C, B_6 or K (Weber 1999).

Without a doubt, the diet of an animal can have a considerable impact on its skeletal health. Animals can usually recover from episodes of seasonal nutritional stress, or pregnancy and lactation. However, if the deficiency is a prolonged one, the impact on the skeleton may be more dramatic, especially if it is combined with periods of pregnancy or lactation. Also, dietary deficiencies in the growing skeleton may be

apparent in later life in the form of a lower peak bone mass. This project will examine the density of bones from both female (with a known history of lambing) and male animals, and will attempt to shed light on the effect of pregnancy and lactation on bone density.

5.2.7: Heredity and breed

Finally, genetic factors are known to have some impact on the density of an animal's bones. Genetic inheritance of bone density levels are especially relevant to archaeological research into animals because the archaeological material will potentially represent a relatively small population of individuals whose interbreeding is managed by their human owners. The reproduction of domesticated animals is necessarily controlled, often in order to produce distinct breeds. A particular breed might be selected (probably unintentionally) for unusually high or low bone density. Similarly, it is quite possible that, because animals from a particular flock might be interrelated, the bone density of these animals might be genetically determined, regardless of the breed in question. Consequently, knowledge of the variation of bone density due to breed and heredity would be useful when interpreting archaeological assemblages.

Research into this area has, again, been primarily concerned with the understanding and prevention or treatment of osteoporosis in humans. However, there is little reason why the conclusions drawn by the medical and clinical literature cannot validly be applied to research into non-humans.

There is a good deal of anecdotal evidence that the bone mineral density of black women is generally higher than that of white or Asian women (Cohn *et al* 1977, Nelson *et al* 1991 p507, Ott 1991 p1646, Trotter *et al* 1960 p53). This difference has been attributed to genetic factors and it is possible that genetically determined higher muscle mass or body size in black women is the primary cause (Cohn *et al* 1977 p177, Nelson *et al* 1991 p511).

Other studies have adopted a less racially based approach to the question. By studying twins, Johnston and Slemenda (1993 pS54) calculated that genetic factors accounted for 60% - 80% of the variation in bone mass within their sample, while environmental factors (nutrition, exercise etc.) accounted for the remaining 20% - 40%. This finding is in broad agreement with Peel and Eastell (1995 p990), Seeman (1989) and Smith *et al* (1973 p2802). Nui (2000 pS102) has suggested that the genetic contribution to bone density is apparent in both juvenile and adult individuals. However, it is likely to be most apparent in early life, before environmental factors have

time to affect the skeleton (Pockock 1987 p709). Furthermore, the density of non-weight-bearing bones will be more susceptible to genetic variation, because any genetically determined variation in weight-bearing bones will quickly be "overwritten" by the effects of environmental factors (eg exercise) (Pockock 1987 p709).

The contribution of genetics to the bone density of sheep has been much less thoroughly researched. Benzie and Gill (1974 p336) and Davis (1987 p40) have noted that some breeds of sheep are less skeletally mature than others. For example, the proximal humerus of Goth sheep is said to fuse at 24-30 months, while the same element in Aragon sheep fuses at 30-42 months (Davis 1987 p40 and references therein). Benzie and Gill (1974 p336) report that the Soay sheep from St Kilda do not reach skeletal maturity until they are 5 years of age – "fully one year later than mainland animals". That a difference in maturation between genetically distinct populations exists is likely. However, whether this difference is the result of genetic or environmental variation has not been conclusively demonstrated. How this difference translates into bone density variation is yet to be established. It is the intention of this project to address this question.

Genetic factors unquestionably have some impact on the bone density of humans. Whether this impact is visible in other animals (in this case, sheep) is less clear. If the findings of studies on human populations are applicable to sheep, it is likely that the impact of genetic factors on the density of their bones will vary according to age and skeletal location. The exact impact of genetic factors on skeletal density is also dependent on the environmental variables with which they interact.

This project will examine animals of a variety of breeds from a number of herds. In doing so, it may be possible to isolate differences in bone density that are due to heredity.

5.3: Summary

It is clear from this review that bone density varies not only according to taxa and skeletal location, but also according to the age, health, level of exercise, sex, nutritional status and genetic inheritance of any individual. These factors are often interrelated (as in the case of age and sex). These factors often differentially affect the skeleton according to element or part of element. The interactions between these factors are often complex and poorly understood, although medical research has provided a

number of guidelines by which their impact on skeletal density can be approximately predicted. The vast majority of research into this topic has focussed on humans, but this project will attempt to provide a better understanding of how the variables discussed in this chapter influence the bones of sheep.

It is worth noting that some variables (eg nutritional status, exercise levels and genetic inheritance (breed)) can rarely be identified from fragmentary archaeological material. Consequently, data on the variability of bone density according to these criteria will be of limited use. Instead, the maximum and minimum possible densities of each relevant skeletal location will produce a range in which the archaeologist knows all material will fall – regardless of factors such as health, level of exercise or nutritional status.

This thesis has so far demonstrated that differential destruction of bone is a potential hindrance to reliable archaeological interpretation. It is mediated largely by bone density and so knowledge of the density of modern bone material can be applied to archaeological material in order to overcome taphonomic biases. It has now been demonstrated that bone density can vary according to factors such as breed, age and sex as well as by taxa and skeletal location and so these factors must be taken into account when interpreting the bone densities from a modern reference sample.