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Introduction and Diagnosis

The study of disease in earlier societies stands firmly in the purview of the discipline of the history of medicine. There are a number of ways in which the subject can be approached, such as from the study of extant medical texts or of pictorial or plastic art and artefacts. However, the most direct approach is from the examination of the physical remains of our ancestors. Each approach has its limitations and calls for different skills. The preservation of documentary evidence is subject to random vagaries, at least as great as the preservation of human remains. The medical historians must be able to set their interpretation of the written word in the context of the various theories of disease extant at the time the original was written. Medical historians will also almost certainly need to be proficient in some languages other than their own. Those who attempt to diagnose disease from paintings, pots or sculpture will need to be familiar with the artistic conventions of the artists whose artefacts they study.

Palaeopathologists have the advantage of being able to study directly the remains of the diseased, although usually only in part, and this ability may be the only factor in their favour. The principal disadvantage that constrains palaeopathologists is that their study is restricted largely to those diseases that affect the skeleton – preserved soft tissues being decidedly unusual in most parts of the world. Skeletal diseases are uncommon, as most diseases affect the soft tissues; this is certainly the case for the killing diseases. Thus, it is generally impossible for palaeopathologists to determine the cause of death of those they examine, which is a great pity because the knowledge of how the causes of death might have changed over the centuries would add greatly to our understanding of the history of disease.[1]

[1] This problem does not, of course, solely affect palaeopathologists. Even though they have written accounts of disease and, in more recent times, mortality statistics, historians also find it difficult to determine the causes of death of our ancestors. One cause of the difficulty is due to the inability to recognise precisely what diseases earlier authors were actually describing. There is considerable scepticism among many historians

The other major problem is that there is no agreed system for diagnosing disease in the skeleton to which all (or even the majority of) palaeopathologists subscribe, thus rendering comparisons between different studies somewhat arbitrary at best, and invalid at worst. The problem is not alleviated at all by the fact that most palaeopathologists have no medical training and so have not been subject to the discipline of the diagnostic treadmill and are not always inclined to base their diagnosis on clinical evidence, or rein in their imaginations. On this account it seems appropriate to meet the difficulty with diagnosis head on and dispatch it with all haste.

DIAGNOSIS IN PALAEOPATHOLOGY

In the clinical setting, medical students are taught the traditional, formalised approach to diagnosis, which begins by taking a detailed history from the patient, eliciting all the present complaints, followed by the past medical history and family, social and occupational histories. Any drugs that are being taken are noted and then the patient is examined to discover any abnormal signs in the various organ systems. The next step is to compile a list of all the conditions that might conceivably cause the signs and symptoms, beginning with the most likely and proceeding downwards to the esoteric and frankly improbable. In practice, such a list – the so-called differential diagnosis – is seldom created except for special purposes, such as writing up, or presenting a case report, when the aim is then to astonish the audience with the brilliance of the presenter. It is much more likely that following the first steps in the procedure, the clinician will have a good idea of the problem and will then arrange for a series of investigations by which the provisional diagnosis can be confirmed. Ancillary investigations may include *inter alia* blood tests, biochemistry, radiology (plain X-rays and scans), virology, bacteriology, ultrasound, endoscopy, biopsy and, as a last resort, invasive surgery. As the results roll in, the provisional diagnosis may be – and frequently is – revised.[2] To aid diagnosis, a number of algorithms, flow

about the reliability of death registration (G Alter and A Carmichael, Studying causes of death in the past. Problems and models, *Historical Methods*, 1996, 29, 44–48).

[2] This procedure may sound familiar to those with a knowledge of Bayes' theorem and, indeed, the claim has been made that clinicians are natural bayesians (CJ Gill, L Sabin and CH Schmidt, Why clinicians are natural bayesians, *British Medical Journal*, 2005, 330, 1080–1083). Bayes' theorem states that the pre-test probability of an hypothesis being true multiplied by the likelihood ratio (the weight of new evidence) produces the post-test probability. Clinicians certainly do change their minds about the probability of a diagnosis being true as new evidence emerges to improve the odds of being correct, but the similarity to the formal Bayesian

charts and various other aids have been designed to make the procedure both more reliable and more consistent.[3]

The purpose of a diagnosis is – as it has always been – to offer the patient a prognosis and, where indicated, some treatment. For the latter purposes, it is immaterial what name is attached to the disease that affects the patient. Diagnostic nomenclature is a rag-bag of terms, some descriptive, some anatomical, some denoting a specific infection, some with virtually no meaning at all. Old and new terms are frequently mixed in a miscellany that has been likened to a room full of furniture from different periods, from Georgian sideboards to glass coffee tables. However, because the aim of the clinician is simply to say, you have a disease that I will treat with such and such a drug, from which you will recover completely, the illogicality of diagnosis causes scarcely a ripple on the medical mill pond.[4]

There are few clinicians who will readily admit to the fact that diagnosis is a rather hit-and-miss affair that tends to be conducted at a subliminal level. The clinician is apt to recognise a diagnosis by its 'jizz'; this is a term that bird watchers use to refer to what one might call the 'totality' of a bird. Bird watchers recognise a marsh harrier, for example, by the sum of its appearance and behaviour. Most clinicians do the same with disease. They recognise its salient features and then expend their energy substantiating their hunch. Most lay people are somewhat disillusioned when (or if) they learn that diagnosis remains more art than science; they are even more alarmed when (or, again, if) they find out how prone the procedure is to error.

There have been many studies of the accuracy of diagnosis, most frequently by comparing a clinical diagnosis with that determined at autopsy. The results have never been very reassuring. In one recent study of fifty-three previously published reports, it was found that up to half of all diagnoses were seriously in error, and

procedure is more apparent than real and it is not very likely, in fact, that most clinicians would consider themselves bayesians (MW Cooper, Should physicians be bayesian agents? *Theoretical Medicine*, 1992, 13, 349–361).

[3] See, for example, those published by the American Academy of Family Physicians (aafp.org). There is now a web site which will offer diagnoses in response to a list of symptoms (www.isabelhealth.com) and some doctors are now using Google as an aid to diagnosis.

[4] In truth, doctors are not much interested in discussing disease and diagnosis in any philosophical sense and, indeed, have some difficulty in saying what precisely they mean by 'disease' or its antithesis 'health'. These matters are discussed in detail in: *What is disease?* (edited by JM Humber and RF Almeder), Humana Press, Totowa, 1997 and *Health, disease and illness* (edited by AL Caplan, JJ McCartney and DA Sisti), Georgetown University Press, Washington, 2004. The allusion to diagnostic furniture is from RE Kendall, *The role of diagnosis in psychiatry*, Blackwell, Oxford, 1975, p 20. Despite its age, this is still probably the best account of the state of diagnosis in medicine. For some more recent thinking on models of disease see C Del Mar, J Doust and P Glasziou, *Clinical thinking. Evidence, communication and decision-making*, Oxford, Blackwell, 2006, pp 27–37.

Table 1.1. *Error rates in clinical diagnosis detected at autopsy*

Type of patient	Number of studies	Overall error rate (%)	Class I error rate (%)*
General inpatients	13	12.6–38.0	1.5–12.0
Adult medical	5	14.0–41.0	7.0–16.0
Adult intensive care	7	19.8–27.9	7.0–12.0
Surgical	5	15.7–49.8	2.6–20.7
Paediatric	3	6.4–13.1	4.3–6.5
Neonatal or paediatric intensive care	5	0.3–28.0	2.1–11.8
Others	5	4.1–33.3	0–9.5

* Errors that might have affected outcome
Data from Shojania et al. (2003)[5]

that in up to one-fifth, the error was sufficiently serious that the patient received the wrong treatment (see Table 1.1). The authors of the review did not believe that the errors had resulted in many avoidable deaths, but overall, their conclusions were not a boost for diagnostic acumen and do little to instil confidence in the procedure.

Now, if clinicians, with a host of information at their disposal get their diagnoses wrong so frequently, how much more likely is it that palaeopathologists will fare any better when they have so *little* information on which to base their conclusions? The answer should be, not very likely at all, although one is not infrequently astonished by the certainty that some authors attach to their diagnoses, sometimes seeming to possess gifts denied to most of us.

If one looks at the clinical criteria for diagnosing a common condition, osteoarthritis (OA) of the knee, that have been developed by the American College of Radiologists[6] (Table 1.2), it is immediately obvious that this is only a minimal help to the palaeopathologist. There is no way in which pain or stiffness can be determined in the skeleton; crepitus cannot be elicited; osteophytes as a lone phenomenon are not necessarily indicative of OA; and even deciding that a skeleton may be that of

[5] KG Shojania, EC Burton, KM McDonald and L Goldman, Changes in rates of autopsy-detected diagnostic errors over time. A systematic review, *Journal of the American Medical Association*, 2003, 289, 2849–2856.

[6] R Altman, E. Asch, D. Bloch, D. Bole, K. Borenstein, K. Brandt, W Christy, TD Cooke, R Greenwald, M Hochberg, D Howell, D Kaplan, W Koopman, S Longley, H Mankin, DJ McShane, R Medsger, R Meenan, W Mikkelsen, R Moskowitz, W Murphy, B Rothschild, M Segal, L Sokoloff and F Wolfe, Determination of criteria for the classification and reporting of osteoarthritis: classification of osteoarthritis of the knee, *Arthritis and Rheumatism*, 1986, 29, 1039–1049.

Table 1.2. *Clinical criteria for the classification of osteoarthritis of the knee*

Clinical and radiological	Clinical
Knee pain	Knee pain
+	+
at least one of the following:	at least three of the following
Age >50 years	Age >50 years
Stiffness for less than 30 minutes	Stiffness for less than 30 minutes
Crepitus	Crepitus
+	Bony tenderness
osteophytes	Bony enlargement
	No palpable warmth

Data from Altman et al. (1986)[7]

a man or woman aged over fifty at death is not always a straightforward matter to determine. What is true for OA of the knee is true for many other diseases that affect the skeleton. Thus, another strategy must be adopted for diagnosing lesions in the skeleton, albeit firmly based on clinical evidence.[8]

The palaeopathologist can obtain a limited amount of information about his 'patients'. The skeleton can be examined directly, or at least as much of it as is present,[9] and the visual inspection can be supplemented by radiography, although this is often not as informative as one might hope because it is a relatively insensitive technique.[10] It may also be possible to carry out a small number of ancillary tests, such

[7] R Altman, E. Asch, D. Bloch, D. Bole, K. Borenstein, K. Brandt, W Christy, TD Cooke, R Greenwald, M Hochberg, D Howell, D Kaplan, W Koopman, S Longley, H Mankin, DJ McShane, R Medsger, R Meenan, W Mikkelsen, R Moskowitz, W Murphy, B Rothschild, M Segal, L Sokoloff and F Wolfe, Determination of criteria for the classification and reporting of osteoarthritis: classification of osteoarthritis of the knee, *Arthritis and Rheumatism*, 1986, 29, 1039–1049.

[8] Those who espouse the quaint notion that clinical evidence may not be suitable for use in palaeopathology will probably wish to read no further, but continue along the primrose path with their like-minded colleagues (M Brickley and M Ives, Skeletal manifestations of infantile scurvy, *American Journal of Physical Anthropology*, 2006, 129, 163–172).

[9] Unfortunately that is often by no means the whole skeleton. It is a regrettable fact of the palaeopathologist's life that the most interesting skeletons (pathologically) are often the least complete, sometimes because the disease affecting the bones makes them more liable to post-mortem damage. (The factors affecting bone preservation are considered by CM Stojanowski, RM Seidermann and GH Doran, Differential skeletal preservation at Windover Pond: causes and consequences, *American Journal of Physical Anthropology*, 2002, 119, 15–26.)

[10] Juliet Rogers and her colleagues, for example, found that radiography was much less able to detect osteoarthritic changes than direct examination. For example, changes were noted in sixteen knees by direct examination but radiographically in only two (J Rogers, I Watt and P Dieppe, Comparison of visual and radiographic detection of bony changes at the knee joint, *British Medical Journal*, 1990, 300, 367–368).

as histology (usually not very helpful)[11] and ancient DNA (aDNA) analysis (which may be helpful in confirming the diagnosis of some infectious diseases)[12] and that is about it. The diagnosis, therefore, is almost always based solely on the morphology and distribution of the changes found in the skeleton on direct examination.[13]

To make a diagnosis based on this meagre information and to ensure conformity, I suggest that the palaeopathologist should use what epidemiologists refer to as an operational definition (see Chapter 13). An operational definition would take the form of a set of criteria that must be fulfilled in order for the disease to be recognised, similar to the criteria set out for the clinical diagnosis of OA of the knee shown in Table 1.2. However, there would be no criteria relating to symptomatology. In a few cases the appearances of the lesion, or the radiological signs, are so characteristic that they are said to be pathognomonic of the condition, that is, they fit this, and only this disease. Not many bone diseases have pathognomonic signs. Therefore, one might say, for example, that disease D would be said to be present if two major criteria were fulfilled, or three of five minor criteria.

[11] Not usually very helpful because of alterations in the bone substance after death, so-called diagenetic change. (See, for example, J Zapata, C Pérez-Sirvent, MJ Martínez-Sánchez and P Tovar, Diagenesis not biogenesis: Two late Roman skeletal examples, *Science of the Total Environment*, 2006, 369, 357–368). The processes that underlie diagenesis are by no means completely understood (REM Hedges, Bone diagenesis: an overview of processes, *Archaeometry*, 2002, 44, 319–328).

[12] DNA generally survives well only in those bones that have preserved their normal microscopic structure (AN Marinho, NC Miranda, V Braz, AK Ribeiro-Dos-Santos and SM de Souza, Paleogenetic and taphonomic analysis of human bones from Moa, Beirada, and Zé Espinho Sambaquis, Rio de Janeiro, Brazil, *Memórias do Instituto Oswaldo Cruz*, 2006, 101 Suppl 2, 15–23).

[13] It is possible that other techniques will find an application in palaeopathology. For example, micro-CT scanning can provide information on trabecular structure (F Peyrin, M Salome, P Cloetens, AM Laval-Jeanet, R Ritman and P Rüegsegger, Micro-CT examinations of trabecular bone samples at different resolutions: 14, 7 and 2 micron level, *Technology and Health Care*, 1998, 6, 391–401) and can provide information about diseased bone (FJ Rühli, G Kuhn, R Evison, R Müller and M Schultz, Diagnostic value of micro-CT in comparison with histology in the qualitative assessment of historical human skull bone pathologies, *American Journal of Physical Anthropology*, 2007, 133, 1099–1111) although it cannot distinguish woven from lamellar bone, for which polarised light microscopy is needed (G Kuhn, M Schultz, R Müller and FJ Rühli, Diagnostic value of micro-CT in comparison with histology in the qualitative assessment of historical human postcranial pathologies, *Homo*, 2007, 58, 97–115). Backscatter electron microscopy can provide qualitative information on the distribution of bone mineral within a section (AL Boskey, Assessment of bone mineral and matrix using backscatter electron imaging and FTIR imaging, *Current Osteoporosis Reports*, 2006, 4, 71–75) while Raman spectroscopy can supply information on the structure of bone crystals (JS Yerramshetty and O Akkus, The association between mineral crystallinity and the mechanical properties of human cortical bone, *Bone*, 2008, 42, 476–482) and nonlinear resonant ultrasound spectroscopy (NRUS) can be used to assess micro-damage in bone (M Muller, A Sutin, R Guyer, M Talmant, P Laugier and PA Johnson, Nonlinear resonant ultrasound spectroscopy (NRUS) applied to damage assessment in bone, *Journal of the Acoustical Society of America*, 2005, 118, 3946–3952). These techniques may soon find application for research purposes but being mostly expensive and confined to specialist laboratories, none is likely to become widely available to the jobbing palaeopathologist.

To date, few operational definitions have been proposed for use in palaeopathology, and none has been universally agreed upon. The procedure by which diseases are diagnosed in the skeleton often remains something of a mystery, which does little to advance the discipline and nothing to help in making between-study comparison. One of the most interesting aspects of palaeopathology is the potential for comparing the frequency of disease at different times and in different places. With a knowledge of environmental or social factors it might even be possible to suggest how the natural history of some diseases has been influenced by those, or indeed, other factors. It might also conceivably shed some light on the aetiology of diseases of the skeleton. Unless the same criteria are used for diagnosis, however, comparisons are invalid and a great deal of potentially useful information is wasted.

There is no doubt that an operational definition will tend to underestimate the true prevalence of disease in a skeletal assemblage because signs in the skeleton often develop late in the history of a disease, and the early stages are very likely to be overlooked. This deficiency, however, would be more than compensated for by observing strict rules for diagnosis, thereby ensuring the validity of any comparisons that *are* made.

What is required for palaeopathology is a set of operational definitions on the lines of the manual produced, for example, by the American College of Psychiatry which is used for both clinical and epidemiological purposes. The present manual makes no claim to be a comprehensive account of skeletal disease. I have, however, suggested operational definitions for some of those diseases in the hope that this will at least promote discussion or perhaps, even acceptance; however, in this last respect, like Corydon, I am unlikely to get what I hope for.[14]

OTHER COMPARISONS

Apart from making comparisons with other contemporary studies on skeletons, it is useful to be able to make comparisons with modern-day data and with data published in bone reports from earlier periods. There is, alas, little prospect of being able to do so. Modern studies almost always use different criteria for making a diagnosis and are carried out on very different populations. For example, some recent studies of the epidemiology of fractures have used referrals to a tertiary trauma

[14] G Mackie, *The eclogues of Virgil, translated into English verse, line for line*, Quebec, Gilbert Stanley, 1847, pp 8–10.

centre,[15] hospital discharge registers,[16] the general population of a city[17] and a general practice research database[18] as their study base. It can scarcely be expected that the incidence or prevalence data that are thereby calculated will have any direct relation to those that might be obtained from the study of a skeletal assemblage or, indeed, with each other. Data from modern epidemiological studies will, therefore, rarely be directly comparable with palaeopathological data in a quantitative sense, although it may be possible to draw some qualitative inferences.[19] For example, suppose one were able to study the distribution of secondary bone tumours, let's say by examining many assemblages using radiography as an aid in collecting cases. If this sort of study were possible, one could compare the distribution of the tumours with that found in a clinical study. Suppose further that the clinical study showed that the major proportion of secondaries from lung and breast tumours concentrated in the thoracic region, followed in order of frequency by the vertebrae, pelvis, upper and lower limbs and the skull.[20] It would then be perfectly reasonable to compare the order in which these regions were represented in the skeletal assemblages, even though the actual percentages may vary due to different methods of investigation. If inconsistencies were found, this might reflect some difference in the behaviour of tumours over time; alternatively the distribution might be found to be so similar that it was reasonable to conclude that the mode of spread showed no substantial differences between the two groups, ancient and modern. One might wish to carry

[15] T Throckmorton and JE Kuhn, Fractures of the medial end of the clavicle, *Journal of Shoulder and Elbow Surgery*, 2007, 16, 49–54.

[16] E Lonnroos, H Kautiainen, P Karppi, T Huusko, S Hartikainen, I Kiviranta and R Sulkava, Increased incidence of hip fractures. A population based-study in Finland, *Bone*, 2006, 39, 623–627.

[17] A Lešić, M Jarebinski, T Pekmezović, M Bumbasirević, D Spasovski and HD Atkinson, Epidemiology of hip fractures in Belgrade, Serbia Montenegro, 1990–2000, *Archives of Orthopaedic and Trauma Surgery*, 2007, 127, 179–183.

[18] F de Vries, C de Vries, C Cooper, B Leufkens and TP van Staa, Re-analysis of two studies with contrasting results on the association between statin use and fracture risk: the General Practice Research Database, *International Journal of Epidemiology*, 2006, 35, 1301–1308.

[19] An additional factor that needs to be stressed here is that modern epidemiological data – certainly those that are published in major journals – are obtained largely from patients or others living and working in the developed countries of Europe, North America and Japan. They will have been following an urban life style which would be about as unlike anything experienced by the majority of past societies studied as can be imagined. Data from rural populations in developing countries who might provide a more suitable comparison group are very hard to come by. Autopsy studies, which probably provide the most reliable data on cause of death – although they cannot provide population incidence or prevalence data – are likely to become increasingly rare in the United Kingdom because of the provision of the *Human Tissue Act 2004* (A Mavroforou, A Giannoukas and E Michalodimitrakis, Consent for organ and tissue retention in British law in the light of the Human Tissue Act 2004, *Medical Law*, 2006, 25, 427–434).

[20] MA Wilson and FW Calhoun, The distribution of skeletal metastases in breast and pulmonary cancer: concise communication, *The Journal of Nuclear Medicine*, 1981, 22, 594–597.

out studies on other diseases to see how similarly they behave qualitatively in a contemporary population.

When trying to make comparisons with old material (that is, those printed more than fifty years ago), there is an immediate problem with nomenclature. Thus, in 1920, R Stockman used four synonyms when discussing osteoarthritis – arthritis deformans, senile arthritis, morbus coxae senilis and spondylitis deformans.[21] When he refers to the history of osteoarthritis, he points out that many other terms were used by even earlier authors, including rheumatic gout, rheumatic arthritis and hypertrophic arthritis.[22] It will not be immediately obvious when reading old reports to which modern condition the author is actually referring, especially if there are no illustrations.

Finally, remember that some diseases that are recognised today will not appear at all in old (or even relatively modern) texts. This is particularly well illustrated by the development of the joint diseases for which fresh entities have been recognised, especially with the development of better means of laboratory diagnosis. If, for example, one reads successive editions of a standard textbook, such as that written by WSC Copeman, it is possible to see how the understanding of the erosive joint diseases was continuously in flux, and to witness the gradual emergence and acceptance of terms such as rheumatoid factor and sero-negative disease.[23] To expect to find an account of the sero-negative joint diseases from prior to the 1970s, therefore, will be met only with grave disappointment.

A NOTE ON HEALTH . . .

Among those who examine human remains, there is frequent reference to the 'health' of the assemblage. This seems to be a reflection of our present obsession with using health as a synonym for disease. The United Kingdom has a Department of Health whose prime responsibility is to offer services to deal with illness, and there are now health, rather than medical, records, as though this will somehow shield the population from the unpleasant business of having to contemplate disease even

[21] R. Stockman, *Rheumatism and arthritis*, W Green & Son, Edinburgh, 1920, chapter IX.
[22] *Ibid*, pp 118–120.
[23] WSC Copeman, *Textbook of the rheumatic diseases*, Livingstone, Edinburgh, 1948. Reference to rheumatoid factor first appears in the third (1964) edition. Not until the fourth (1969) edition, however, is its nature 'so well established that it is no longer necessary to discuss the several arguments against it' (p 187). It is in this edition that the first reference to the term sero-negative is given.

though it is manifestly disease, rather than health, that is of major concern. However one defines health – the World Health Organisation's notion of it being 'a state of complete physical, mental and social well being and not merely the absence of disease or infirmity' does not seem in the least realistic[24] – it is certain that an examination of the skeleton is not going to enable anything to be said about health, even in its most basic form as being the absence of disease. It may very well be the case that there is little evidence of bone or joint disease in an assemblage. However, to then infer that the individuals were in good health seems perverse, given that they are all dead and that it is probable that at least a third to a half of them will have died prematurely; this is a very strange notion of health. Perhaps other indicators of health could be adduced? The expectation of life might show that one assemblage enjoyed a greater expectation of life than another and might therefore be considered healthier, but anyone who relied on such data, given the virtual impossibility of assigning an accurate age at death to a skeleton, would be a brave soul indeed.

There is, in fact, no means of knowing the state of health during life of the individuals who come to comprise an assemblage of skeletons, especially because what determines their actual state of health depends not only upon the diseases that affected their internal organs, but their mental state, their diet[25] and many other environmental and social factors, about which there is little to be known from the state of their bones. It is best to recognise that all palaeopathologists can comment upon is disease and settle for that; it may be less than desirable but it is what we have.

. . . AND MUMMIES

In this book, most of the emphasis is on human skeletal remains because these are much more common in most contexts than mummified material. This is especially true of those countries in which the majority of palaeopathologists work. Even when mummified material is available, it is often not as informative as one might think. For example, the internal organs may have been removed during mummification or

[24] R Saracci, The World Health Organisation needs to reconsider its definition of health, *British Medical Journal*, 1997, 314, 1409–1410.

[25] It is possible to deduce whether the diet of an individual contained a lot of meat or fish, or neither. Originally this was based on trace-element analysis of bone (see, for example, KB Byrne and DC Parris, Reconstruction of the diet of the Middle Woodland Amerindian population at Abbott Farm by bone trace-element analysis, *American Journal of Physical Anthropology*, 1987, 74, 373–384) but this method was susceptible to contamination from the soil, and more reliable results are now obtained from analysis of the stable isotopes of carbon and nitrogen found in collagen (S Lösch, G Grupe and J Peters, Stable isotopes and dietary adaptations in humans and animals at pre-pottery Neolithic Nevalli Cori, southeast Anatolia, *American Journal of Physical Anthropology*, 2006, 131, 181–193).