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Ancient avian osteopetrosis: the current state of knowledge

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Abstract. The avian leucosis group are caused by viruses which have been viewed seriously by the poultry industry, due to the losses caused. In ancient remains, the virus may also produce pathognomonic changes to the long bones. The dense bone growth, called osteopetrosis, results eventually in massive thickening of long bone surfaces and infilling ("occlusion") of the marrow cavity. In birds, bone expansion of the diaphysis is the result of excessive osteoblast proliferation and bone growth. No other bird pathology results in such bone expansion, and it should not be difficult to discriminate between avian osteopetrosis and other osteitis or trauma. This paper therefore calls attention to the exciting prospects for building up an epidemiological picture of an ancient bird disease. As a contribution to the palaeo-epidemiology of this disease, particular attention will be given to chicken populations from British archaeological sites.

Key words: *Gallus*, chicken, osteopetrosis, Roman, leucosis virus.

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Although a wide range of chicken diseases were known in the nineteenth century, and were referred to in poultry keeping publications, the microbiology of most conditions was only known to a very limited degree, and it did not include osteopetrosis (BROWN 1892). Leucotic diseases were being described by 1868, but were known only to specialist researchers (PAYNE and FADLEY 1997).

PEARL et al. (1919) provided a more extensive account of *Gallus* diseases, and although osteopetrosis was still not recognised until 1938, it was seen that so-called "filterable agents" caused a range of pathology and tumour development. Surprisingly, even as late as 1969, Hungerford's 'Diseases of Poultry' did not mention osteopetrosis, although by then it had been well described. The bone pathology has now been well researched and published (HOLMES 1964; BAKER 1965; BILTZ and PELLEGRINO 1965; SIMPSON and SANGER 1968). Limited comment has also been made in archaeological publications (LUFF and BROTHWELL 1993; PRUMMEL 1987), although generally neglected in the field of zooarchaeology. For this reason, the disease is reviewed in some detail here, and an attempt is made to evaluate its geographic distribution through time in the British Isles. This has been made possible through the help and advice of Terry O'CONNOR, Polydora BAKER, Umberto ALBARELLA, Jim PAYNE, Deborah JAQUES, John BAKER, Dale SERJEANTSON and Rosemary LUFF.

Osteopetrosis – aetiology

Changes to the bones are caused by the chronic action of the avian leucosis virus group (ALV), which belongs to the family Retroviridae. The diseases produced by these viruses today commonly result in a 1-2% mortality of birds, but occasionally up to 20% or more. There can also be a reduc-

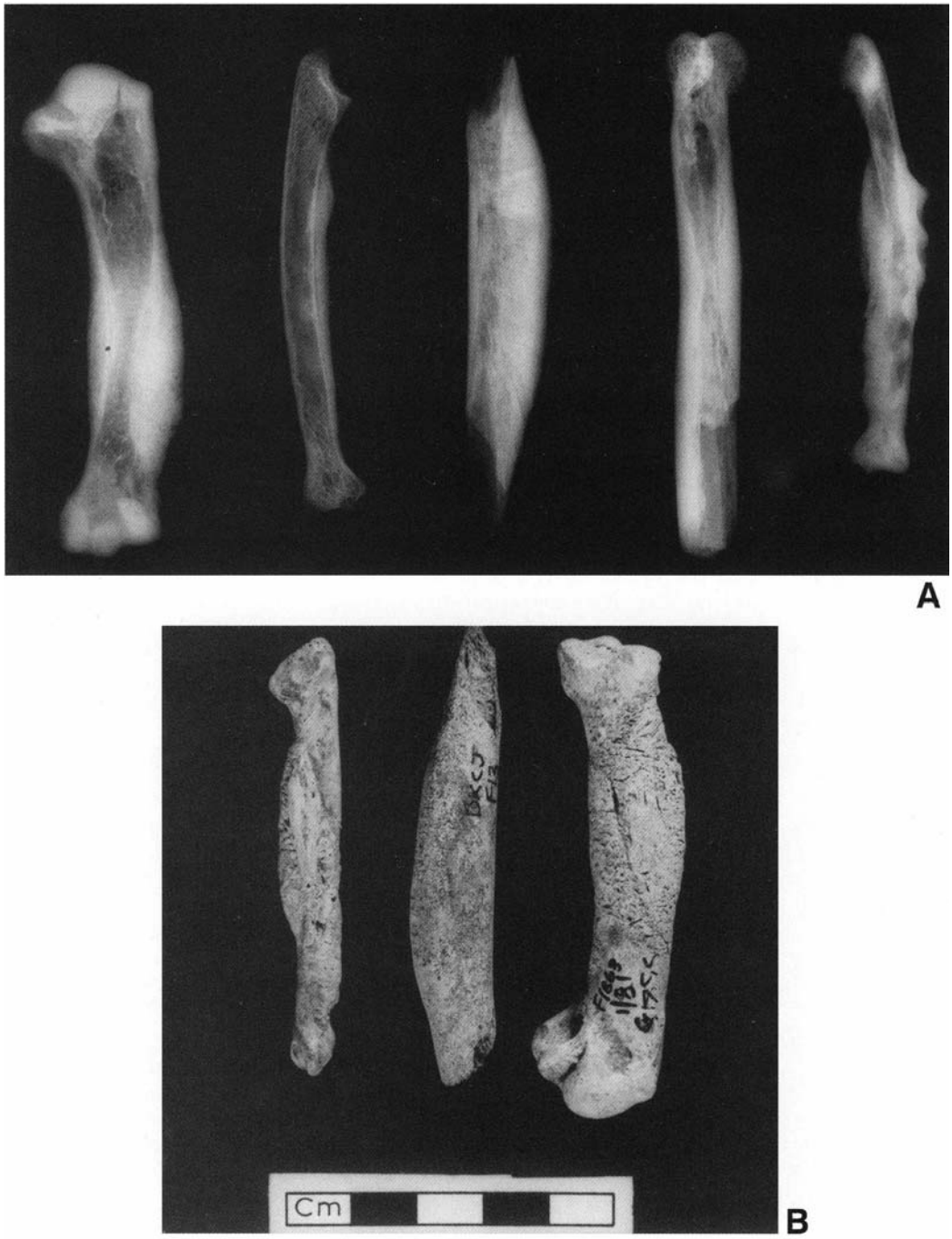

Fig. 1. Pathological changes in avian osteopetrosis from Roman Colchester. A – X-rays of long bones showing dense extra bone both externally and internally; B – surface detail of the swollen bones.

tion in egg production, and the development of various tumour types (PAYNE and FADLEY 1997). If the virus is present, most birds will become exposed to it, but the incidence of clinical pathology will normally be low. Osteopetrosis occurs commonly in affected flocks, being associated with a number of viral strains. Bone changes are most likely to occur if infection begins in the very young chickens. Transmission can in fact be vertically, from hen to progeny through the egg, or horizontally from bird to bird.

Bone pathology

By ten weeks of age, osteopetrosis can be considerable. Long bones are most commonly affected, the diaphysis displaying thick dense bone which expands both internally and externally (Fig. 1). The abnormal bone is thought to be the result of high levels of virus greatly disturbing the growth and differentiation of osteoblasts. It has been suggested by SHANK et al. (1985) that the ability of this virus group to produce such bone changes, depends on specific sequences in the viral genome, and if this is the case, then archaeology provides evidence of the survival of these sequences over at least two thousand years.

From a microevolutionary point of view then, although leucosis viruses are capable of considerable microevolutionary or adaptive change, there is really no evidence that the bone pathology has significantly changed. Moreover, this presumably indicates that the molecular factors which control the bone changes have a relatively stable genetic background. So the bone changes are giving us fairly restricted but precise information on a specific part of the leucosis virus variation.

A British historical geography

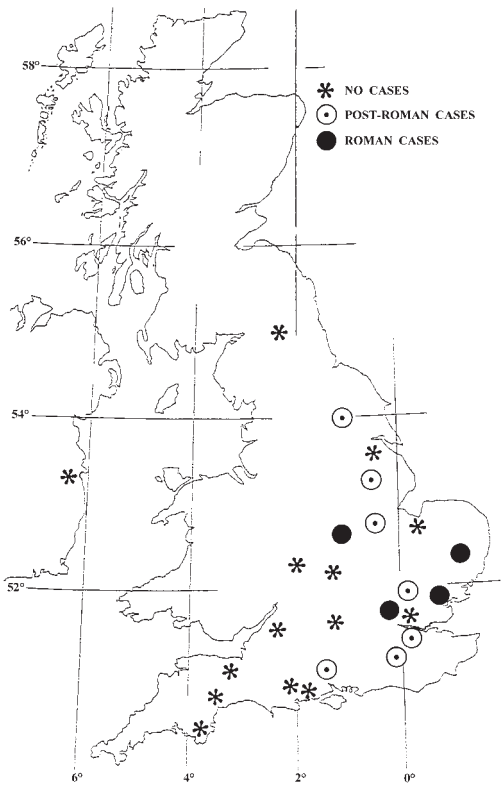

Fig. 2. Early British evidence of osteopetrosis.

So far at most sites in Britain, no cases have been identified, and this may be due to small sample sizes, or perhaps even failure to identify early stages of bone change. But at some sites, one or two cases have been recorded, and at Roman Colchester, 26 cases were identified, some being extremely well developed bone pathology (LUFF and BROTHWELL 1993). Because the number of chicken bones, whole or incomplete, in the archaeological samples is so variable, it was impossible to determine with any reliability, the incidence of osteopetrosis in the *Gallus* populations. But in any case, as the virus causes bone changes in only a small proportion of infected birds, the important fact is that even one case of bone pathology indicates notable infection in the fowl community, at least for a time. Can we attempt then, however crudely, to construct an historical geography of the disease for a particular region? Figure 2 shows the current skeletal evidence for the condition in Britain from Roman to medieval times. Early cases are separated from the post-Roman ones in order to detect any evidence of spatial expansion. Even allowing for some missed identifications, we can at least tentatively suggest that the leucosis virus was established in some chicken flocks in South East England by Roman times, and that there is growing evidence for osteopetrosis by post-Roman times and possibly over a wider area. Even on this limited evidence then, we can begin to suggest an epidemiological history for a virus group producing specific bone pathology.

Regarding a potential source of the infection in another part of Northern Europe during Roman times, there was certainly a viral reservoir in the Netherlands, ready to cross the North Sea. In fact the area of the Roman Castellum at Velsen in the Netherlands, faces across to Colchester and East Anglia. PRUMMEL (1987) found good evidence of the bone pathology at Velsen, and estimated that about 1% of the domestic fowl at this site had osteopetrosis. So all forms of leucosis virus infection in the flock must have been high indeed.

Conclusions

Thus, the value of recording osteopetrosis in earlier fowl populations is that it is an uncommon opportunity to consider the history and epidemology of a specific disease. Even on the current very limited information on early Britain, we can see that it was established, if geographically restricted, by Roman times. It is not yet known if it has a greater antiquity. We now need to see a greater awareness of the bone pathology and then in time we may be able to assemble a bigger picture of its antiquity and spread in Europe and beyond. This must be supported by radiocarbon dates on the pathological bones, in order to get a more sensitive determination of disease movements.

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