AMERICAN JOURNAL OF PHYSICAL ANTHROPOLOGY 137:106–112 (2008)

Brief Communication: Paleopathology of the Kiik-Koba 1 Neandertal

Erik Trinkaus,[1]* Blaine Maley,[1] and Alexandra P. Buzhilova[2]

[1]*Department of Anthropology, Washington University, St. Louis, MO 63130*
[2]*Institute of Archaeology, Russian Academy of Sciences, Moscow 117036, Russia*

KEY WORDS postcrania; hypercementosis; DISH; trauma; enthesopathy

ABSTRACT The Kiik-Koba 1 Neandertal partial skeleton (canine, partial hands, partial leg, and feet), of a ≈40-year-old probable male, exhibits a suite of pathological lesions, including hypercementosis, minor fibrous ossifications, pedal phalangeal fracture, and pronounced enthesopathies on the patella and calcanei in the context of no articular degenerations. The first two sets of lesions are related to age in the context of advanced dental attrition and physical strains. The third lesion joins a series of healed minor traumatic lesions among the Neandertals. The last represents either pronounced tendinous inflammation, albeit in the context of no articular degenerations, or a case of diffuse idiopathic skeletal hyperostosis (DISH) in the Late Pleistocene. Kiik-Koba 1 therefore adds to the high incidence of pathological lesions among the Neandertals and, if a diagnosis of DISH is correct, to a high frequency of this disorder among older Neandertals. Am J Phys Anthropol 137:106–112, 2008.

As the Neandertals have been investigated from a paleopathological perspective, it has become increasingly evident that they, or at least their better preserved partial skeletons, exhibit a high frequency of developmental and degenerative abnormalities (Heim, 1982; Trinkaus, 1983, 1985; Ogilvie et al., 1989; Brennan, 1991; Duday and Arensburg, 1991; Berger and Trinkaus, 1995; Smith et al., 2006). Many of these lesions are minor developmental defects reflecting stress periods during development (e.g., dental enamel hypoplasias) or the scars of superficial traumatic lesions that impacted the underlying bone. These lesions may be notable for their commonness (Ogilvie et al., 1989; Berger and Trinkaus, 1995; Cowgill et al., 2007), even though the frequencies of the developmental lesions fall within recent human ranges of variation (Guatelli-Steinberg et al., 2004; Cowgill et al., 2007), but the Neandertals also appear to have sustained a number of more serious injuries and/or systemic abnormalities (Trinkaus, 1983, 1985; Duday and Arensburg, 1991; Crubézy and Trinkaus, 1992; Fennell and Trinkaus, 1997; Schultz, 2006). Given the dearth of individuals in the Neandertal sample that are likely to have lived to at least the fifth decade, their accumulation of such lesions may well provide insight into both their habitual stress levels and sociocultural means of surviving the insults sufficiently to leave diagnosable lesions.

It is in this context that we present a paleopathological description and diagnosis of the Kiik-Koba 1 partial skeleton. These remains were described by their excavator, Bonch-Osmolovskii (1941, 1954), who concluded (1941, p. 18) that the changes evident on the skeletal remains are not pathological but merely reflect normal activities. Subsequently Rokhlin (1965), in a radiographic analysis of the remains, concluded that the ossifications on the patella and calcanei are pathological, as well as the deformity of the right fifth proximal phalanx and the alterations of a few of the pedal distal phalangeal tuberosities. These descriptions, however, are brief, and the exceptional state of preservation of those bones present for Kiik-Koba 1 warrants further discussion of its lesions.

MATERIALS AND METHODS

Kiik-Koba 1: context

The site of Kiik-Koba in the Crimea (45° 03′ N, 34° 18′ E) was excavated by Bonch-Osmolovskii in 1924–1926, during which he uncovered three Middle Paleolithic levels (Levels III, IV, and VI), the deepest (Level VI) resting on bedrock (Bonch-Osmolovskii, 1940). Level III was overlain by a largely sterile deeper portion of Level II, and Levels IV and VI were separated by a largely sterile Level V. The site provided abundant evidence for anthropogenetic alterations, including hearths, pits and burials. The Kiik-Koba 1 adult was buried into a depression, which was excavated through Level VI and apparently into the underlying limestone. A second individual, the Kiik-Koba 2 infant, was buried in close proximity to the adult in a separate grave, similarly excavated through Level VI. Subsequent to its burial, an additional excavation during the Middle Paleolithic disturbed the remains of Kiik-Koba 1 and removed most of the skeleton, leaving a tooth, portions of both hands, the right lower leg, and two virtually complete pedal skeletons (Bonch-Osmolovskii, 1941, 1954). In the process, the tooth and some of the phalanges were mixed into the material in the overlying Level IV.

On the basis of the Middle Paleolithic (Kiik-Koba Mousterian) association of the burial and the presence of both temperate and cold climate fauna (e.g., *Sus*, *Cervus*, *Saiga,* and *Marmota* in Levels IV and VI, plus *Rangifer*

*Correspondence to: Erik Trinkaus, Department of Anthropology, Campus Box 1114, Washington University, St. Louis, MO 63130, USA. E-mail: trinkaus@artsci.wustl.edu

Received 17 December 2007; accepted 11 February 2008

DOI 10.1002/ajpa.20833
Published online 20 March 2008 in Wiley InterScience (www.interscience.wiley.com).

in Level IV), the burial level of Kiik-Koba 1 and 2 has been attributed to the early last glacial or possibly late last interglacial (early OIS 4 or late OIS 5) (Bonch-Osmolovskii, 1940; Klein, 1965).

Kiik-Koba 1: preservation, age-at-death and sex

Kiik-Koba 1 retains the right C_1 and 73 skeletal elements of the hand, leg, and foot. The hands have the right trapezium and first and third metacarpals, four ulnar proximal phalanges, six middle phalanges, both pollical distal phalanges, and two ulnar distal phalanges. They are complete with damage to three of the proximal phalangeal heads and one distal pollical phalangeal base. The right patella, tibia, and fibula are largely complete, with marginal damage to the proximal tibial epiphysis and the fibular head. The pedal skeletons retain all of the tarsals, metatarsals, proximal phalanges and hallucal sesamoid bones, plus six middle and nine distal phalanges. The only pedal bones with more than minor edge damage are the calcanei; the right one was crushed laterally from the peroneal tubercle to the tuberosity, and the left one lost all of the lateral body and tuberosity. In addition, two of the lateral distal phalanges lack their distal tuberosities.

The Kiik-Koba 1 remains, as well as the Kiik-Koba 2 infant postcrania, have morphological affinities to western Eurasian Neandertals, including known or biomechanically inferred body proportions, scapular morphology, pollical anatomy, tibial morphology, talar proportions, and pedal phalangeal proportions (Vlček, 1972, 1973, 1975, 1977; Trinkaus, 1975a, in press; Trinkaus and Hilton, 1996; Trinkaus and Rhoads, 1999). Of these features, the body proportions, scapular morphology, and pollical anatomy indicate Neandertal affinities; the other features are shared with archaic *Homo* (Trinkaus, 2006).

The age-at-death of Kiik-Koba 1 can only be based on the C_1 occlusal attrition. The crown retains only a thin ring of enamel, 2.3 mm labially, 1.8 mm lingually, and 0.5 mm mesially and distally. Similar occlusal wear among the Neandertals is associated with ages-at-death (based on femoral histology and pelvic fibrocartilagenous surfaces) in the fourth or fifth decade (Trinkaus and Thompson, 1987; Trinkaus, 1995), similar to or moderately older than the 35–40 years suggested by Rokhlin (1965).

Alexeev (1978) inferred that Kiik-Koba 1 was female. However, its tibial maximum length (349 mm) is within the values for the pelvically sexable male Neandertals (363 ± 17 mm, 340–386 mm, $N = 4$) and above those of the two pelvically sexable females (311 and 319 mm). Moreover, an estimated stature of ≈164 cm and an inferred body mass of ≈78 kg (Ruff et al., 1997), both based on the tibial length and assuming Neandertal body proportions, place it among the larger of the Neandertals and close to pelvically sexed males (La Chapelle-aux-Saints 1, Feldhofer 1, Kebara 2, and Shanidar 1 and 3). Kiik-Koba 1 is therefore probably male.

RESULTS

The canine

The Kiik-Koba 1 mandibular canine (Fig. 1) presents moderately advanced hypercementosis, in that it covers all of the root except the cervical 4–5 mm of the labial side. It is relatively thin and does not change the normal contour of the root. This extra laying down of cement on the root has been associated with pronounced occlusal attrition and/or periodontal inflammation (Corruccini et al., 1987; Hillson, 1996), and it is therefore not surprising given the wear on this tooth.

The hand remains

The 17 preserved hand remains, which derived from both hands (10 right, 5 left, and 2 indeterminate) are free of pathological alterations. There are marked insertions for the extensor digitorum tendons on the bases of the middle phalanges. There are strong flexor sheath crests on the proximal phalanges. The flexor digitorum superficialis insertions are moderately raised on the middle phalanges. In addition, there are well-marked edges, especially radially, to the flexor pollicis longus tendon insertions around the proximal pollical phalangeal palmar bases. However, none of these muscle insertions consists of more than a raised area of bone for the fibrous attachment, and they are therefore not enthesopathies *sensu stricto* [the term "enthesopathy," or "enthesophyte" (Jurmain, 1999; Villotte, 2006), is here restricted to protruding spicules of bone following tendinous insertions into the bone and representing calcifications of that fibrous tissue]. All of the articular surfaces are normal, with no evidence of subchondral porosity or ossifications of the capsular attachments.

The patella

As noted by Rokhlin (1965: 220), the Kiik-Koba 1 patella has a massive enthesopathy for the quadriceps femoris tendon (Fig. 2). The enthesopathy covers the entire original tendinous attachment surface of the bone. It extends ≈5 mm superior of the normal proximal margin of the bone. There are smaller enthesopathies along the anterodistal margin, especially laterally, where the patellar ligament continues the quadriceps femoris fibers to the proximal tibia. The principal anterolateroproximal bony growth is ≈24 mm wide, ≈8 mm thick, and ≈23 mm high; these are respectively 47%, 31%, and 48% of the overall dimensions of the patella.

This enthesopathy is partly separated from the original tendinous surface by a space on the lateral two-thirds of the proximal margin and the proximal half of the lateral surface. The proximal gap is up to ≈8 mm wide and extends mediolaterally ≈25 mm. The lateral gap is ≈18 mm high. The space appears to be largely the product of the anterior and anterosuperior projection of the new bone formation, but there has been some anterolateroproximal resorption of the original bone. The enthesopathy is completely separate from the articular margin and the attachment for the joint capsule, especially proximally and laterally (Fig. 2).

The patellofemoral articular facets are normal, with only a slight elevation of the midproximal margin (the distal margin is abraded) and minimal irregularity of the subchondral bone (Fig. 2). There is no subchondral porosity, and the facets are normally slightly concave with a larger lateral than medial facet [unlike many archaic *Homo* patellae (Carretero et al., 1999; Trinkaus, 2000)], indicating normal knee function.

The tibia, fibula, and talocrural articulation

The right lower leg bones and their articulations with the right talus, as well as the talocrural surfaces of the left talus, are completely normal. The proximal articular

Fig. 1. Views of the Kiik-Koba 1 right mandibular canine. Mes: mesial; Dist: distal; Lab: labial; Ling: lingual. Scale in millimeters.

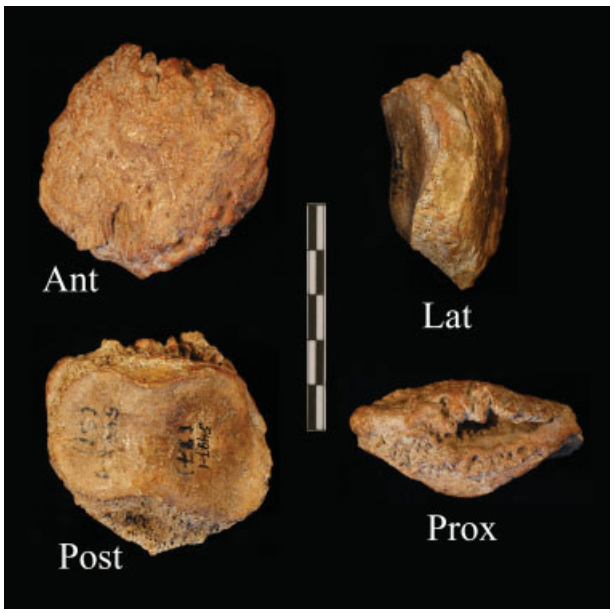

Fig. 2. Anterior (Ant), lateral (Lat), posterior (Post) and proximal (Prox) views of the Kiik-Koba 1 right patella. Scale in centimeters.

margins of the tibia are missing, but the subchondral condylar bone and the intercondylar space are normal. The complete right and one-sided left talocrural articulations show no pathological changes. There is modest rugosity of the tibial tuberosity, the soleal line, and the distal tibiofibular ligament attachments, but all of them are well within normal ranges of variation, especially for robust Pleistocene *Homo*.

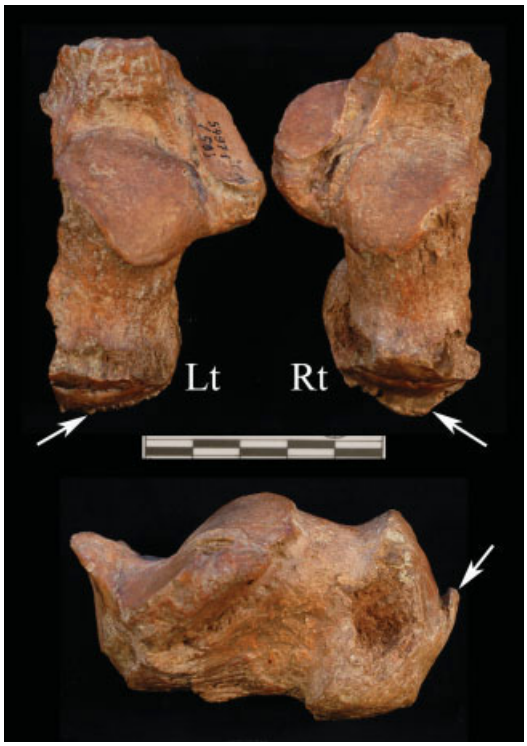

Fig. 3.

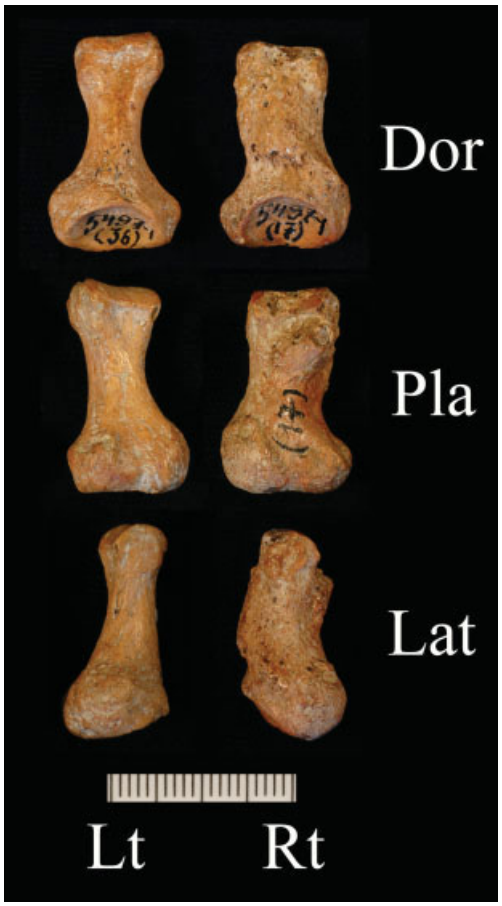

Fig. 4.

Fig. 3. Dorsal view (above) of the Kiik-Koba 1 left (Lt) and right (Rt) calcanei, and lateral view (below) of the Kiik-Koba 1 right calcaneus. The large right and the damaged and less prominent left Achilles tendon enthesopathies are indicated by arrows. Scale in centimeters.

Fig. 4. Views of the Kiik-Koba 1 normal left (Lt) pedal proximal phalanx 5 and the abnormal right (Rt) one. Dor: dorsal; Pla: plantar; Lat: lateral. Scale in millimeters.

The subtalar skeletons

Except for damage to the lateral calcanei, especially on the left, the tarsals and metatarsals, plus the four hallucal sesamoid bones, are essentially complete. All of their articular surfaces are normal. There are minor ossifications of intertarsal connective tissue in three places. There are small bony spurs in the left sulcus tali but none in the right one; the associated anterodorsal surfaces on the calcanei show only moderate rugosity. There is a relatively distal ossification of the long and/or short plantar ligament on the right calcaneus, but no ossifications of the insertions on the plantar cuboid or metatarsal bases; the medial and lateral processes are normal. There is a small spur of bone, 3.5 mm high, 3.0 mm thick, and 7.0 mm proximodistal on the middle of the dorsomedial margin of the left intermediate cuneiform bone; there is no change to the adjacent medial cuneiform bone.

The principal changes to the subtalar skeleton are large enthesopathies for the triceps surae (Achilles) tendon insertions into the calcaneal tuberosities (Fig. 3). The better preserved right one is 15.8 mm wide at its base, at the midtuberosity dorsoplantar angling of the tuberosity. It then narrows to 8.5 mm wide at its dorsal end, extending 6.0 mm dorsally and posteriorly from the original tuberosity surface. The left calcaneus has evidence of a similar enthesopathy, but damage precludes determining its original extent.

The pedal phalanges

With two exceptions, the right fifth proximal phalanx and the right first distal phalanx, the pedal phalanges are normal. Rokhlin (1965) noted the absence of the tuberosities of two lateral distal phalanges and suggested that this might be osteomyelitis due to trauma or frostbite. It is unclear whether these bones sustained antemortem lesions or postmortem damage, and preservative on the bones precludes proper assessment of the exposed trabeculae. Even if Rokhlin's interpretation is correct, the effect was minimal and would only have affected the distal portions of these two distal phalanges.

Along 12.7 mm of the middorsal proximal margin of the right distal hallucal phalanx, there is an irregular but only slightly projecting and rounded bony growth. This represents a minor ossification of the insertion for the extensor hallucis longus tendon. A similar but less pronounced rugosity is present on the proximodorsal right proximal hallucal phalanx for the insertion of the extensor hallucis brevis muscle. Both are relatively common, and they are better seen as rugosities from muscle hypertrophy rather than from a pathological process.

The fifth right proximal phalanx, however, is clearly abnormal (Fig. 4). There is a dorsal convexity to the shaft, dorsoplantar, and mediolateral midshaft expansions (midshaft diameters of 9.1 and 9.3 mm respectively versus 6.1 and 6.0 mm on the left), a slight shortening of the bone (articular length of 19.5 mm versus 20.8 mm on the left), an S-shaped parallel-sided contour to the bone in dorsal and plantar views as opposed to the normal straight hour-glass shape of the left bone, and a general irregularity of the subperiosteal surface. The proximal articulation and capsular attachment are normal, as is the trochlear surface of the head; there is no change on the associated fifth metatarsal head. These alterations of the right fifth proximal phalanx are best seen as the result of a fracture of the diaphysis. The radiograph of the bone (Rokhlin, 1965; p. 223) reveals no sign of the original fracture line, indicating complete healing of the trauma. The bone's normal articulations indicate no loss of function once the lesion had healed.

DISCUSSION

The pathological lesions on Kiik-Koba 1 consist of three forms: dental hypercementosis, pedal phalangeal fracture, and tendinous/ligamentous ossifications. The first alteration is age and probably dental attrition related. The second lesion joins a long list of direct and indirect lesions from traumatic injuries known for the Neandertals, most of them nondebilitating and none of them impairing locomotion (Lumley, 1973; Trinkaus, 1983, 1985; Berger and Trinkaus, 1995; Dawson and Trinkaus, 1997; Schultz, 2006; Mann and Monge, 2006). Given the minimal role of the fifth digit in weight support and traction (Stott et al., 1973; Warren et al., 2004), it should have had little effect on mobility. The third form of lesion is more complicated.

The simplest interpretation of the large patellar and calcaneal enthesopathies, plus the small one on distal hallucal phalanx and the osteophytes on the talus and intermediate cuneiform bone, is that they represent independent results of localized tendinous or ligamentous collagen and/or fibrocartilagenous calcification as reactions to strain (Benjamin et al., 2000; Villotte, 2006). In this case, the small bony spurs on the tarsals could be the products of strains on the associated ligaments, although each adjacent bone into which the connective tissue would insert shows no new bone growth. The bony growths on the patella, calcanei, and distal hallucal phalanx could therefore be seen as the products of either age (although the age-at-death of Kiik-Koba 1 may not be sufficiently advanced for that to be a factor), overuse and/or localized trauma of the associated muscles, even though associations between enthesopathies and activities are hard to document (Jurmain, 1999).

If these lesions were the products of independent strains, then it is curious that the patella shows massive enthesopathy formation, whereas the tibial tuberosity insertion of the patellar ligament is normal. Similarly, there is bilateral ossification of the triceps surae insertions, but the soleal lines on the tibia and fibulae exhibit their normal rugosity. Moreover, none of the other major plantarflexors, intrinsic or extrinsic, exhibits the consequences of similar tendinous strains, and none of the associated articulations shows any subchondral bone or capsular attachment degeneration.

A different interpretation, at least for the patella, as suggested by Rokhlin (1965), views the enthesopathies as the result of strain and/or impact from kneeling or similar biomechanical overloading of the knee. There is a large squatting facet on the distal tibia, ≈18 mm wide and 6.8 mm high, that matches a large facet, 9.8 mm proximodistal and from 13.8 to 17.4 mm wide, on the dorsal neck of the right talus. Kiik-Koba 1, as with many Neandertals (Trinkaus, 1975b; Heim, 1982), frequently engaged in squatting and may have habitually knelt. However, clinical analyses of knee degenerations associated with habitual squatting and kneeling primarily document osteoarthritic changes of the patellofemoral and tibiofemoral articular surfaces, including articular cartilage and menisceal degeneration plus articular margin osteophyte formation (Kivimäki et al., 1992; Jensen

and Eenberg, 1996; Zhang et al., 2004; Tangtrakulwanich et al., 2006, 2007). Ossification of the quadriceps femoris tendon and the presence of patellar "spurs" are principally associated with quadriceps femoris tendon rupture (Ferretti et al., 1983; Hardy et al., 2005). Given the absence of knee osteoarthritis, it appears unlikely that the patellar enthesopathies were the result of habitual kneeling or squatting, even though they could have been associated with a quadriceps femoris injury.

An alternative approach is to view the small tarsal and phalangeal growths as the products of localized strains, but to consider the large patellar and calcaneal enthesopathies as a reflection of a systemic disorder. The primary systemic disorder that involves these patellar and calcaneal changes, associated with the absence of osteoarthritis, is diffuse idiopathic skeletal hyperostosis [DISH, also known as Forestier's Disease or hyperostotic disease (Forestier and Rotes-Querol, 1950; Resnick et al., 1975; Rogers et al., 1985; Utsinger, 1985; Crubézy, 1990)]. DISH is normally defined by the presence of flowing ossifications of the anterior longitudinal ligaments across several (usually $\geq$4) vertebral bodies without involvement of the disk spaces and by the absence of vertebral articular facet osteoarthritis. While the primary diagnosis of DISH is vertebral, it usually involves extra-spinal bilateral enthesopathies, particularly at the olecranon processes (triceps brachii tendons), the patellae (quadriceps femoris tendons), and the calcaneal tuberosities (triceps surae tendons). There may be additional enthesopathies, particularly on the foot, the pelvis, and the femoral trochanters. In particular, Crubézy (1990) has emphasized that paleopathologically, in the absence of preserved vertebrae, there must be no inflammatory disease within peripheral joints, bony spurs must be greater than 3 mm in length, they need to be bilateral and symmetrical, and they need to minimally involve the olecranons, patellae, and calcanei.

Clinical studies of DISH have shown a male bias, with male-to-female ratios averaging $\approx$2:1 (Julkunen et al., 1975; Utsinger, 1985; Jankauskas, 2003; Kacki and Villotte, 2006). Population frequencies are variable and highly age dependent after the fifth decade, but the condition is almost never seen in individuals <40 years of age and frequencies among adults <60 years of age tend to be <20% and frequently <5% (Henrard and Bennett, 1973; Julkunen et al., 1975; Arlet and Mazières, 1985; Cassim et al., 1990; Weinfeld et al., 1997). The etiology of DISH is not known, but it has been associated with a variety of other systemic disorders, including diabetes mellitus (see below).

Kiik-Koba 1 does not preserve any of the vertebral column or the ulnae, and it retains only one patella. It is therefore not possible to confirm a diagnosis of DISH (cf. Crubézy, 1990). However, the bilateral presence of the calcaneal tuberosity ossifications, the massive enthesopathy on the patella, the absence of osteoarthritis, and the minor bony spurs on the tarsals, considered together, point to a feasible diagnosis of DISH. And the probable male sex and age-at-death of $\approx$40 years fit with the diagnosis.

At the same time, and perhaps not independently, one might be able to view the Kiik-Koba 1 enthesopathies as an indirect result of hyperinsulinemia from adult onset (Type II) diabetes mellitus (DM). It has been suggested (Littlejohn, 1985; Rosenbloom and Silverstein, 1996; Arkkila and Gautier, 2003) that the systemic changes of DM affect tendons and promote the formation of enthesopathies, especially in the insertions of the major weight-supporting muscles. However, more detailed assessments of quadriceps femoris and triceps surae tendinous alterations with DM have found either no association between them (Tambolo et al., 1995; Altinel et al., 2007) or a possible or limited association between them (Holmes and Lin, 2006; Akturk et al., 2007). Clinical studies are therefore not conclusive as to whether the enthesopathies on Kiik-Koba 1 could be the result of Type II DM.

There is also a more general clinical history of associating DISH with DM (Julkunen et al., 1971; Pastan and Cohen, 1978; Denko et al., 1994; Kiss et al., 2002; Arkkila and Gautier, 2003; Crispin and Alcocer-Varela, 2003; Mader et al., 2005; Sencan et al., 2005), although many of the analyses were unable to document a statistically significant association between the two conditions, especially when appropriate multiple comparison corrections (Proschan and Waclawiw, 2000) are applied. Most often the two conditions are equally associated with comorbidities, especially obesity, such that it is difficult to determine if DISH is a consequence or a comorbidity of DM. Indeed, paleopathological studies (e.g., Janssen and Maat, 1999; Rogers and Waldron, 2001; Jankauskas, 2003; Kacki and Villotte, 2006; Blondiaux et al., 2007) generally support a correlation between higher nutritional status and the incidence of DISH.

The absence of other skeletal changes in Kiik-Koba 1 commonly associated with DM, such as general bone mineral loss (Arkkila and Gautier, 2003; Strotmeyer and Cauley, 2007) and osteoarticular deterioration of the hand and foot (Ardic et al., 2003), further make a diagnosis of DM unlikely. Moreover, the low probability of both chronic obesity and long-term survival with DM in the Middle Paleolithic make it unlikely that DM was the cause of the DISH-like skeletal changes on this Neandertal.

DISH has been diagnosed in the Shanidar 1 Neandertal (Crubézy and Trinkaus, 1992), and it is possible that it was present in the less complete Shanidar 4 skeleton (Trinkaus, 1983). Of the other sufficiently complete older ($\geq$40 years of age) Neandertal partial skeletons (N = 4), there is no evidence of DISH in any of the three males (La Chapelle-aux-Saints 1, La Ferrassie 1, and Feldhofer 1) or the one female (La Ferrassie 2). Yet, if both Shanidar 1 and 4 plus Kiik-Koba 1 endured this condition, that would indicate that 50% of the older male Neandertal partial skeletons exhibit it (95% CI: 12–88%), 43% of the older Neandertals including La Ferrassie 2 (95% CI: 10–82%), or 33% of the total sample if Shanidar 4 is considered too incomplete (95% CI: 4–78%). Despite the small sample sizes (and consequent large 95% CIs), which reflect the dearth of older Neandertals in the fossil sample (Trinkaus, 1995), these mean frequencies are relatively high compared to most recent human observations (see above).

CONCLUSION

The paleopathological lesions on the Kiik-Koba 1 partial skeleton therefore make it similar to other known older Neandertal partial skeletons, all of which exhibit some form of degenerative lesion(s) (Heim, 1982; Trinkaus, 1983, 1985; Crubézy and Trinkaus, 1992; Fennell and Trinkaus, 1997; Schultz, 2006). The dental and many of the skeletal changes of Kiik-Koba 1 can be seen as the accumulated effects of a Middle Paleolithic lifestyle, plus survival to $\approx$40 years of age, combining high dental attrition, mobility, and risk of minor trauma. The patellar

and calcaneal enthesopathies could be similar to the other lesions, or they could be subsumed into one condition, diffuse idiopathic skeletal hyperostosis (DISH). Since the primary manifestation of DISH involves intervertebral ossifications, the appendicular diagnosis of this condition in Kiik-Koba 1 would imply that he suffered from a stiffening of the vertebral column in addition to any discomfort from the tendinous ossifications.

These lesions reflect both moderately high levels of risk, compared with many recent human skeletal series, as well as survival among these late archaic humans. If these appendicular lesions accurately reflect DISH, then they reflect a moderately high incidence of this systemic degenerative condition among these late archaic humans.

ACKNOWLEDGMENTS

We are grateful to Y.K. Chistov, V.I. Khartanovich, A. Kozintsev, and A.A. Sinitsyn for their assistance and kind permission, which has made the study of the Kiik-Koba Neandertal remains in the Kunstkamera, St. Petersburg, possible.

LITERATURE CITED

Akturk M, Ozdemir A, Maral I, Yetkin I, Arslan M. 2007. Evaluation of achilles tendon thickening in type 2 diabetes mellitus. Exp Clin Endocrinol Diabetes 115:92–96.

Alexeev V. 1978. Palaeoanthropology of the globe and formation of human races. The Paleolithic (in Russian). Moscow: Nauka.

Altinel L, Kose KC, Degirmenci B, Petik B, Acarturk G, Colbay M. 2007. The midterm effects of diabetes mellitus on quadriceps and patellar tendons in patients with knee arthrosis: a comparative radiological study. J Diabetes Complications 21: 392–396.

Ardic F, Soyupek F, Kahraman Y, Yorgancioglu R. 2003. The musculoskeletal complications seen in type II diabetics: predominance of hand involvement. Clin Rheumatol 22:229–233.

Arkkila PE, Gautier JF. 2003. Musculoskeletal disorders in diabetes mellitus: an update. Best Pract Res Clin Rheumatol 17:925–970.

Arlet J, Mazières B. 1985. La maladie hyperostosique. Rev Med Interne 6:553–564.

Benjamin M, Rufai A, Ralphs JR. 2000. The mechanism of formation of bony spurs (enthesophytes) in the achilles tendon. Arthritis Rheum 43:576–583.

Berger TD, Trinkaus E. 1995. Patterns of trauma among the Neandertals. J Archaeol Sci 22:841–852.

Blondiaux J, Alduc-Le Bagousse A, Demondion X, Delahaye F, Niel C. 2007. Maladie hyperostosique et maladie goutteuse, une diathèse familiale en Normandie: Thaon, Calvados. Bull Mém Soc Anthropol Paris ns 19:7–20.

Bonch-Osmolovskii GA. 1940. Paleolit Kryma 1: The Cave of Kiik-Koba (in Russian with French summary). Moscow-Leningrad: Izdatelstvo Akademii nauk SSSR.

Bonch-Osmolovskii GA. 1941. Paleolit Kryma 2: the hand of the fossil man from Kiik-Koba (in Russian with French summary). Moscow-Leningrad: Izdatelstvo Akademii nauk SSSR.

Bonch-Osmolovskii GA. 1954. Paleolit Kryma 3: the skeleton of the foot and leg of the fossil man from the Cave of Kiik-Koba (in Russian). Moscow-Leningrad: Izdatelstvo Akademii nauk SSSR.

Brennan MU. 1991. Health and disease in the Middle and Upper Paleolithic of southwestern France: a bioarcheological study. Ph.D. thesis, New York University.

Carretero JM, Lorenzo C, Arsuaga JL. 1999. Axial and appendicular skeleton of homo antecessor. J Hum Evol 37:459–499.

Cassim B, Mody GM, Rubin DL. 1990. The prevalence of diffuse idiopathic skeletal hyperostosis in African blacks. Br J Rheumatol 29:131–132.

Corruccini RS, Jacobi KP, Handler JS, Aufderheide AC. 1987. Implications of tooth root hypercementosis in a Barbados slave skeletal collection. Am J Phys Anthropol 74:179–184.

Cowgill LW, Trinkaus E, Zeder MA. 2007. Shanidar 10: a Middle Paleolithic immature distal lower limb from Shanidar Cave, Iraqi Kurdistan. J Hum Evol 53:213–223.

Crispin JC, Alcocer-Varela J. 2003. Rheumatologic manifestations of diabetes mellitus. Am J Med 15:753–757.

Crubézy E. 1990. Diffuse ideopathic skeletal hyperostosis: diagnosis and importance in paleopathology. J Paleopathol 3:107–118.

Crubézy E, Trinkaus E. 1992. Shanidar 1: a case of hyperostotic disease (DISH) in the Middle Paleolithic. Am J Phys Anthropol 89:411–420.

Dawson JE, Trinkaus E. 1997. Vertebral osteoarthritis of the La Chapelle-aux-Saints 1 Neandertal. J Archaeol Sci 24:1015–1021.

Denko CW, Boja B, Moskowitz RW. 1994. Growth promoting peptides in osteoarthritis and diffuse idiopathic skeletal hyperostosis—insulin, insulin-like growth factor I, growth hormone. J Rheumatol 21:1725–1730.

Duday H, Arensburg B. 1991. La pathologie. In: Bar-Yosef O, Vandermeersch B, editors. Le Squelette Moustérien de Kébara 2. Paris: C.N.R.S. p 179–193.

Fennell KJ, Trinkaus E. 1997. Bilateral femoral and tibial periostitis in the La Ferrassie 1 Neandertal. J Archaeol Sci 24: 985–995.

Ferretti A, Ippolito E, Mariani P, Puddu G. 1983. Jumper's knee. Am J Sports Med 11:58–62.

Forestier J, Rotes-Querol J. 1950. Senile ankylosing hyperostosis of the spine. Ann Rheum Dis 9:321–330.

Guatelli-Steinberg D, Larsen CS, Hutchinson DL. 2004. Prevalence and the duration of linear enamel hypoplasia: a comparative study of Neandertals and Inuit foragers. J Hum Evol 47:65–84.

Hardy JR, Chimutengwende-Gordon M, Bakar I. 2005. Rupture of the quadriceps tendon: an association with a patellar spur. J Bone Joint Surg 87B:1361–1363.

Heim JL. 1982. Les hommes fossiles de La Ferrassie II. Arch Inst Paléontol Hum 38:1–272.

Henrard JC, Bennett PH. 1973. Etude epidémiologique de l'hyperostose vertébrale. Enquête dans une population adulte d'Indiens d'Amérique. Rev Rhum Mal Ostéoartic 40:581–591.

Hillson S. 1996. Dental anthropology. Cambridge UK: Cambridge University Press.

Holmes GB, Lin J. 2006. Etiologic factors associated with symptomatic achilles tendinopathy. Foot Ankle Int 27:952–959.

Jankauskas R. 2003. The incidence of diffuse idiopathic skeletal hyperostosis and social status correlations in Lithuanian skeletal materials. Int J Osteoarchaeol 13:289–293.

Janssen HAM, Maat GJR. 1999. Canons buried in the "Skiftskapel" of the Saint Servaas Basilica at Maastricht A.D. 1070–1521. A paleopathological study. Barge's Anthropol (Leiden) 5:1–31.

Jensen L, Eenberg W. 1996. Occupation as a risk factor for knee disorders. Scand J Work Environ Health 22:165–175.

Julkunen H, Heinonen OP, Knekt P, Maatela J. 1975. The epidemiology of hyperostosis of the spine together with its symptoms and related mortality in a general population. Scand J Rheumatol 4:23–27.

Julkunen H, Heinonen OP, Pyorala K. 1971. Hyperostosis of the spine in an adult population. Its relation to hyperglycaemia and obesity. Ann Rheum Dis 30:605–612.

Jurmain R. 1999. Stories from the skeleton. Amsterdam: Gordon and Breach.

Kacki S, Villotte S. 2006. Maladie hyperostotique et mode de vie: Intérêt d'une démarche bio-archéologique. Exemple du cimetière du Couvent des Soeurs Grises de Beauvais (Oise), XV^{e}-XVIII^{e} siècles. Bull Mém Soc Anthropol Paris ns 18:55–64.

Kiss C, Szilágyi M, Paksy A, Poór G. 2002. Risk factors for diffuse idiopathic skeletal hyperostosis: a case-control study. Rheumatology (Oxford) 41:27–30.

Kivimäki J, Riihimäki H, Hänninen K. 1992. Knee disorders in carpet and floor layers and painters. Scand J Work Environ Health 18:310–316.

Klein RG. 1965. The Middle Paleolithic of the Crimea. Arctic Anthropol 3:34–68.
Littlejohn GO. 1985. Insulin and new bone formation in diffuse idiopathic skeletal hyperostosis. Clin Rheumatol 4:294–300.
de Lumley MA. 1973. Anténéandertaliens et Néandertaliens du bassin Méditerranéen occidental Européen. Etud Quatern 2:1–626.
Mader R, Dubenski N, Lavi I. 2005. Morbidity and mortality of hospitalized patients with diffuse idiopathic skeletal hyperostosis. Rheumatol Int 26:132–136.
Mann A, Monge J. 2006. A Neandertal parietal fragment from Krapina (Croatia) with a serious cranial trauma. Period Biol 108:495–502.
Ogilvie MD, Curran BK, Trinkaus E. 1989. The incidence and patterning of dental enamel hypoplasias among the Neandertals. Am J Phys Anthropol 79:25–41.
Pastan RS, Cohen AS. 1978. The rheumatologic manifestations of diabetes mellitus. Med Clin North Am 62:829–839.
Proschan MA, Waclawiw MA. 2000. Practical guidelines for multiplicity adjustment in clinical trials. Control Clin Trials 21:527–539.
Resnick D, Shaul SR, Robins JM. 1975. Diffuse idiopathic skeletal hyperostosis (DISH): Forestier's disease with extraspinal manifestations. Radiology 115:513–524.
Rogers J, Waldron T. 2001. DISH and the monastic way of life. Int J Osteoarchaeol 11:357–365.
Rogers J, Watt I, Dieppe P. 1985. Palaeopathology of spinal osteophytosis, vertebral ankylosis, ankylosing spondylitis, and vertebral hyperostosis. Ann Rheum Dis 44:113–120.
Rokhlin DG 1965. Diseases of ancient men (bones of the men of various epochs—normal and pathological changed) (in Russian). Moscow-Leningrad: Publishing House "Nauka".
Rosenbloom AL, Silverstein JH. 1996. Connective tissue and joint disease in diabetes mellitus. Endocrinol Metab Clin North Am 25:473–483.
Ruff CB, Trinkaus E, Holliday TW. 1997. Body mass and encephalization in Pleistocene Homo. Nature 387:173–176.
Schultz M. 2006. Results of the anatomical-palaeopathological investigations on the Neanderthal skeleton from Kleine Feldhofer Grotte (1856) including the new discoveries from 1997/2000. In: Schmitz RW, editor. Neanderthal 1856–2006. Mainz am Rhein: Verlag Philipp von Zabern. p 277–318.
Sencan D, Elden H, Nacitarhan V, Sencan M, Kaptanoglu E. 2005. The prevalence of diffuse idiopathic skeletal hyperostosis in patients with diabetes mellitus. Rheumatol Int 25:518–521.
Smith FH, Smith MO, Schmitz, RW. 2006. Human skeletal remains from the 1997 and 2000 excavations of cave deposits derived from Kleine Feldhofer Grotte in the Neandertal Valley, Germany. In: Schmitz RW, editor. Neanderthal 1856–2006. Mainz am Rhein: Verlag Philipp von Zabern. p 187–246.
Stott JRR, Hutton WC, Stokes IAF. 1973. Forces under the foot. J Bone Joint Surg 55B:335–344.
Strotmeyer ES, Cauley JA. 2007. Diabetes mellitus, bone mineral density, and fracture risk. Curr Opin Endocrinol Diabetes Obes 14:429–435.
Tambolo C, Poli M, Mantovani G, Bressan F, Bambara LM. 1995. Enthesopathies and diabetes mellitus. Clin Exp Rheumatol 13:161–166.
Tangtrakulwanich B, Chongsuvivatwong V, Geater AF. 2006. Associations between floor activities and knee osteoarthritis in Thai Buddhist monks: the Songkhla Study. J Med Assoc Thai 89:1902–1908.
Tangtrakulwanich B, Chongsuvivatwong V, Geater AF. 2007. Habitual floor activities increases risk of knee osteoarthritis. Clin Orthop Relat Res 454:147–154.
Trinkaus E. 1975a. A functional analysis of the Neandertal Foot. Ph.D. thesis, University of Pennsylvania.
Trinkaus E. 1975b. Squatting among the Neandertals: a problem in the behavioral interpretation of skeletal morphology. J Archaeol Sci 2:327–351.
Trinkaus E. 1983. The Shanidar Neandertals. New York: Academic Press.
Trinkaus E. 1985. Pathology and the posture of the La Chapelle-aux-Saints Neandertal. Am J Phys Anthropol 67:19–41.
Trinkaus E. 1995. Neanderthal mortality patterns. J Archaeol Sci 22:121–142.
Trinkaus E. 2000. Human patellar articular proportions: recent and Pleistocene patterns. J Anat (Lond) 196:473–483.
Trinkaus E. 2006. Modern human versus Neandertal evolutionary distinctiveness. Curr Anthropol 47:597–620.
Trinkaus E. Kiik-Koba 2 and Neandertal axillary border ontogeny. Anthropol Sci (in press).
Trinkaus E, Thompson DD. 1987. Femoral diaphyseal histomorphometric age determinations for the Shanidar 3, 4, 5 and 6 Neandertals and Neandertal longevity. Am J Phys Anthropol 72:123–129.
Trinkaus E, Hilton CE. 1996. Neandertal pedal proximal phalanges: diaphyseal loading patterns. J Hum Evol 30:399–425.
Trinkaus E, Rhoads ML. 1999. Neandertal knees: power lifters in the Pleistocene? J Hum Evol 37:833–859.
Utsinger PD. 1985. Diffuse idiopathic skeletal hyperostosis. Clin Rheum Dis 11:325–351.
Villotte S. 2006. Connaissances médicales actuelles, cotation des enthésopathies: Nouvelle méthode. Bull Mém Soc Anthropol Paris ns 18:65–85.
Vlček E. 1972. Antropologie neandertálského dítěte z Kiik-Koby v Sovětském svazu. Zprávy 25:36–40.
Vlček E. 1973. Postcranial skeleton of a Neandertal child from Kiik-Koba, U.S.S.R. J Hum Evol 2:537–544.
Vlček E. 1975. Morphology of the first metacarpal of Neanderthal individuals from the Crimea. Bull Mém Soc Anthropol Paris Série 13 2:257–276.
Vlček E. 1977. Rekonstruktion des Postkranial-Skeletts eines Säuglings des Neandertalers aus Kiik-Koba in der UdSSR. Ärztliche Jugendkunde 68:173–179.
Warren GL, Maher RM, Higbie EJ. 2004. Temporal patterns of plantar pressures and lower-leg muscle activity during walking: effect of speed. Gait Posture 19:91–100.
Weinfeld RM, Olson PN, Maki DD, Griffiths HJ. 1997. The prevalence of diffuse idiopathic skeletal hyperostosis (DISH) in two large American Midwest metropolitan hospital populations. Skeletal Radiol 26:222–225.
Zhang Y, Hunter DJ, Nevitt MC, Xu L, Niu J, Liu LY, Yu W, Aliabadi P, Felson DT. 2004. Association of squatting with increased prevalence of radiographic tibiofemoral knee osteoarthritis: the Beijing Osteoarthritis study. Arthritis Rheumatol 50:1187–1192.