Stable isotope ratios and dental health on the island of Lesvos, Greece: evidence for diet and lifeway. S.J. GARVIE-LOK, Department of Archaeology, University of Calgary, Calgary, AB T2N 1N4, Canada.

Populations on the Greek island of Lesvos have historically had access to both terrestrial and marine resources. However, the relative degree of dependence on these in various periods and the impact of ethnicity on resource use have been unclear. This study examines diet and ethnicity in two archaeological populations, one late Byzantine (Christian) and one late Ottoman (Muslim), using stable isotope analysis of human bone collagen and the frequency of lesions of dental disease.

The stable carbon and nitrogen isotope ratios obtained for both populations suggest a diet primarily based on terrestrial resources. This may reflect customs favouring the consumption of meat and dairy products over that of fish. However, it may also reflect a preference for molluscs and small fish low on the food chain, whose stable isotope values depart less from terrestrial values than do those of marine organisms of higher trophic level.

The stable isotope ratios indicate little difference in dietary focus between Byzantine and Ottoman populations. Some difference is seen in the dental data, with the Ottoman population showing an increased prevalence of dental caries, antemortem tooth loss and periodontal disease. This may reflect an increased availability of refined carbohydrates in the later period.

The most marked difference between the two populations is in the distribution of stable isotope ratios. While values for the Byzantine population cluster tightly, the Ottoman population shows several individuals with extreme outlying values. Rather than reflecting higher dietary variability in the Ottoman period, this appears to indicate higher mobility, with some individuals coming into the community from elsewhere in the Ottoman empire.

These data improve our understanding of resource use in the Aegean in recent centuries. As well, they suggest increased residence mobility in Ottoman times, an idea that could be further examined using stable oxygen or strontium isotope analysis.

Mutations, parental age, and offspring longevity: new ideas and findings. L.A. GAVRILOV, N.S. GAVRILOVA, G.N. EVDOKUSHKINA and V.G. SEMYONOVA, Center on Aging, NORC and University of Chicago, Chicago, IL 60637.

Individuals born to older parents may suffer from the load of deleterious mutations. The human spontaneous mutation rate for DNA base substitutions is reported to be very high, presumably more than one new mutation per zygote (Crow, 1997, PNAS USA, 94: 8380-86). The mutation rate is much higher in male sperm cells than in female ovaries and increases with paternal age due to large number of cell divisions in the male germ line (Crow, 1997). In this study we have checked whether human longevity is affected by increased mutation load expected for the progeny of older fathers. For this purpose the high quality data (more than 15,000 records) on European royal and noble families were collected, computerized and analyzed. The data on offspring lifespan were adjusted for historical trends and fluctuations in life expectancy of human birth cohorts. Also, in order to avoid bias in estimation of the offspring life span, only extinct cohorts were analyzed (born in 1800-1899).

We found (after controlling for maternal age at reproduction, paternal and maternal longevity and sex-specific cohort life expectancy) that adult daughters (30+ years) born to older fathers (45-55 years) live shorter lives and for each additional year of paternal age the daughters loose about 0.5 ± 0.2 year of their life span. In contrast to daughters the sons are not significantly affected by delayed paternal parenting. This result was also confirmed after taking into account additional confounding variables (nationality, birth order, cause of death and loss of parents before age 20) using multiple regression on nominal variables. Since only daughters inherit paternal X-chromosome, this sex-specific life span shortening for daughters born to older fathers might indicate that genes affecting longevity and sensitive to mutation load are probably concentrated in X chromosome.

Another interesting finding is that familial resemblance between offspring and parental lifespan is higher for children born to younger parents as expected for genetic reasons (higher genetic diversity of younger parents). This study was supported by NIA grants P20 AG12857, AG13698-01 and AG16138-01A1.

Mechanisms of familial transmission of human longevity. N.S. GAVRILOVA, L.A. GAVRILOV, V.G. SEMYONOVA and G.N. EVDOKUSHKINA, Center on Aging, NORC and University of Chicago, Chicago, IL 60637.

Recent scientific debates on the future of human longevity and its possible biological limits has revealed a great need for direct biological evidence for such longevity limit if it really exists (Gavrilov, Gavrilova, 1998, *Science*, 281: 1611-1615). For this purpose the familial transmission of human longevity from parents to daughters (more than 4,000 cases for adult daughters born in 1800-1880) was studied, since daughters did not have high violent losses due to military service.

The familial transmission of human longevity from mother to daughter is essentially non-linear with very weak resemblance before maternal life span of 85 years (regression slope of daughters life span on maternal life span, $b = 0.04 \pm 0.02$, $n = 3,756$ cases, $p = 0.05$) and very high additive heritability for longer lived mothers ($b = 0.53 \pm 0.26$, $n = 484$, $p < 0.05$). This indicates that maternal age of 85 years could be considered as a demarcation line for women longevity. Women who live above this age are fundamentally (biologically?) different from other women in the sense that their daughters live significantly longer. Thus life expectancy at 85 years could be a biological limit for validity of extrapolative approach in forecasting of human life expectancy for women.

Similar study of familial transmission of human longevity

from *father* to daughter revealed a demarcation point at 80 years, suggesting that this age might represent a limit for validity of extrapolative approach to male life expectancy. The familial transmission of human longevity from father to daughter is also non-linear with very weak resemblance before paternal life span of 80 years ($b = 0.03 \pm 0.02$, $n = 3,842$, $p = 0.18$) and very high additive heritability for longer lived fathers ($b = 0.36 \pm 0.16$, $n = 763$, $p < 0.05$).

These results are also consistent with the predictions of evolutionary theory of aging and mutation accumulation theory in particular that the additive genetic variance for human life span should increase with parental longevity (Gavrilova et al., 1998, *Human Biology*, 70: 799-804). This study was supported by NIA grants P20 AG12857, AG13698-01 and AG16138-01A1.

Complex allometry of brain size scaling among mammals. B. R. GELVIN, California State University, Northridge, CA 91330, G. H. ALBRECHT, University of Southern California, Los Angeles, CA 90033, & J. M. A. Miller, University of California, Los Angeles, CA 90095.

The relationship between brain size **Y** and body size **X** has long been described by the simple allometry equation: $\log(\mathbf{Y}) = \log(\boldsymbol{b}) + \boldsymbol{k} \cdot \log(\mathbf{X})$. Early studies of mammals reported $\boldsymbol{k} \approx 0.67$, presumably reflecting volume to surface area relationships (Bonin, '37; Jerison, '73). Later studies reported $\boldsymbol{k} \approx 0.75$, presumably reflecting metabolic scaling (Martin, '81; Eisenberg, '81; Armstrong, '83; Hofman, '83; Worthy & Hickie, '86). Using Worthy & Hickie's data, Gelvin & Albrecht ('90) confirmed Count's ('47) long ignored observation of curvilinearity in logarithmic plots of brain weight versus body weight that can be fit by a 2° polynomial: $\log(\mathbf{Y}) = \log(\boldsymbol{b}) + \boldsymbol{k}_1 \cdot \log(\mathbf{X}) + \boldsymbol{k}_2 \cdot [\log(\mathbf{X})]^2$.

We have assembled a new database of over 700 species of adult mammals. Brain weights (or volumes) and body weights were abstracted from primary literature sources. We used *Mammal Species of the World* (Wilson & Reeder, '93) to control and standardize taxonomy. For each species, we used the best available data taking into account sample sizes, sexual dimorphism, subspecific variation, and the origin (wild vs. captive) and condition (health) of specimens.

Our results from least-squares polynomial regression affirm that brain size scaling is best described by a curvilinear model rather than simple allometry. "Instantaneous allometry coefficients" — i.e., the slopes of tangents to the curved polynomial regression line analogous to the $\boldsymbol{k}$'s of simple allometry — progressively decrease as body size increases. Simple allometry coefficients like $\boldsymbol{k} = 0.67$ or $\boldsymbol{k} = 0.75$, as artifacts of an inadequate linear model, cannot be used to support hypotheses based on geometric or metabolic principles even though such factors must play some role in affecting the pattern of brain size differences among mammals. Furthermore, the common practice of using simple allometric regression to predict a species' brain size or calculate its encephalization quotient yields biased estimates, especially for small and large mammals.

Brain size scaling has neither a simple form nor a common explanation across the size range of mammals. Rather, the relationship of brain size to body size displays a complex allometric pattern that must have multifaceted explanations that vary both by body size and taxonomic grouping.

Cheaters never prosper: Social mechanisms maintaining honest signals of status in vervet monkey scrotal color. M.S. GERALD, Department of Anthropology, University of California, Los Angeles, 90095

Previous experiments introducing pairs of unfamiliar adult male vervet monkeys (Cercopithecus aethiops sabaeus) matched for size, but differing in scrotal color, revealed that color conveys social status and predicts the potential for contact aggression to arise (Gerald, 1999). The present investigation examined interactions involving "mutant" "Cheaters", signaling high status. The purpose of this study was to estimate the costs and benefits of cheating, as a means to identify potential mechanisms maintaining color as an honest signal of status. Cheaters were Pale males, painted to resemble naturally Dark (dominant) males. Clear painted males served as Control subjects. Average rates of aggression (contact and non-contact), displays and affiliative behavior were calculated to assess potential costs and benefits of cheating.

Relative to Control males, Cheaters suffered fewer threats ($U=26.5$, $N_1=10$, $N_2=13$, $p=0.021$) and attacks ($p=0.01$) from Pale males. Furthermore, Cheaters received more affiliative overtures from Pale males ($U=26.5$, $p=0.008$) than Controls. By contrast, Dark males threatened ($U=22.5$, $N_1=10$, $N_2=10$, $p=0.012$) and subjected Cheaters to more non-contact ($U=22.5$, $p=0.012$) and contact aggression ($p=0.01$) than Control males. While Pale males interacted affiliatively, even more often with Cheaters ($U=36.5$, $p=0.000$) than with naturally Dark males, Dark males did not exhibit any evidence of distinguishing Cheaters from other naturally Dark males.

Evolutionarily stable strategy models of cooperative signaling demonstrate that when signaling benefits both the signaler and receiver, signals need not be costly for the maintenance of honest signals (i.e. Johnstone 1998). The results of these experiments suggest support for this model. While Cheaters may reap benefits from engaging in friendly interactions with Pale males, they increase their vulnerability to threats and aggression when interacting with Dark males. These costs of cheating may function to maintain honest signals of status. Other hypothetical costs and benefits will also be discussed.

Ontogenetic allometry of the skull. R.Z.GERMAN, Biological Sciences, University of Cincinnati, Cincinnati, OH 45221-0006

Charles Oxnard's use of complex statistical methods to examine the functional basis of both craniofacial and postcranial morphology transformed studies of allometry and scaling. His results, that sexual dimorphic allometry and scaling vary among structures within a set of taxa, suggests a particular course of investigation for ontogenetic studies of growth in mammalian skulls.

Variation in craniofacial skeletal morphology characterizes mammals and has a clear functional basis in demands of feeding. Yet all infant mammals