

- FLORES, C., ET AL. 2000. "Genetic affinities among human populations inhabiting the sub-Saharan area, Northwest Africa, and the Iberian Peninsula," in *Prehistoric Iberia: Genetics, anthropology, and linguistics*. Edited by A. Arnaiz-Villena, pp. 33–50. New York: Kluwer Academic/Plenum Press.
- . 2001. Y-chromosome differentiation in Northwest Africa. *Human Biology* 73. In press.
- FUSTÉ, M. 1958. *Algunas observaciones acerca de la antropología de las poblaciones prehistórica y actual de Gran Canaria*. Las Palmas: Ediciones de El Museo Canario.
- GONZÁLEZ, R., AND A. TEJERA. 1990. *Los aborígenes canarios: Gran Canaria y Tenerife*. Oviedo: Ediciones Istmo.
- GRAVEN, L., ET AL. 1995. Evolutionary correlation between control-region sequence and restriction polymorphisms in the mitochondrial genome of a large Senegalese Mandenka sample. *Molecular Biology and Evolution* 12:334–45.
- GUASCH, J., ET AL. 1952. Los factores hemáticos en España, excepto en el País Vasco. *Medicina Clínica* 18:268–71.
- HAMMER, M. F., ET AL. 1997. The geographic distribution of human Y chromosome variation. *Genetics* 145:787–805.
- HSIEH, C.-L., AND H. E. SUTTON. 1992. Mitochondrial and nuclear variants in a U.S. Black population: Origins of a hybrid population. *Annals of Human Genetics* 56:105–12.
- HURLES, M. E., ET AL. 1999. Recent male-mediated gene flow over a linguistic barrier in Iberia, suggested by analysis of a Y-chromosomal DNA polymorphism. *American Journal of Human Genetics* 65:1437–48.
- LADERO-QUESADA, M. A. 1979. *Los primeros europeos en Canarias*, s. XV–XVI. Las Palmas: Cabildo Insular de Gran Canaria.
- LARRUGA, J. M., ET AL. 1992. Human enzyme polymorphism on the Canary Islands. 5. Western islands. *Gene Geography* 6:159–66.
- LOBO-CABRERA, M. 1982. *La esclavitud en las Canarias orientales en el siglo XVI*. Las Palmas de Gran Canaria: Excmo. Cabildo Insular de Gran Canaria.
- MACAULAY, V., ET AL. 1999. The emerging tree of West Eurasian mtDNAs: A synthesis of control-region sequences and RFLPs. *American Journal of Human Genetics* 64:232–49.
- MACÍAS-HERNÁNDEZ, A. 1988. Fuentes y principales problemas metodológicos de la demografía histórica de Canarias. *Anuarios de Estudios Atlánticos* 34:51–158.
- MARTELL, M., ET AL. 1986. Human enzyme polymorphism on the Canary Islands. 1. Gran Canaria Island population. *Human Heredity* 36:41–44.
- MORILLA, J. M., ET AL. 1988. Human enzyme polymorphism on the Canary Islands. 2. African influence. *Human Heredity* 38:101–5.
- ONRUBIA-PINTADO, J. 1987. Les cultures préhistoriques des Îles Canaries: État de la question. *L'Anthropologie* 91:653–78.
- . 1992. "Canaries (Iles)," in *Encyclopédie Berbère XI Bracelets-Caprarienses*. Edited by G. Camps, pp. 1731–55. La Calade, France: Édisud.
- PÉREZ, M. J., ET AL. 1991. Human enzyme polymorphism on the Canary Islands. 4. Eastern islands. *Human Heredity* 41:385–90.
- PINTO, F., ET AL. 1994. Human enzyme polymorphism on the Canary Islands. 6. Northwest African influence. *Human Heredity* 44:156–61.
- . 1996a. Blood group polymorphisms in the Canary Islands. *Gene Geography* 10:171–79.
- . 1996b. Sub-Saharan influence on the Canary Islands population deduced from G6PD gene sequence analysis. *Human Biology* 68:517–22.
- . 1996c. Genetic relationship between the Canary Islanders and their African and Spanish ancestors inferred from mitochondrial DNA sequences. *Annals of Human Genetics* 60:321–30.
- RANDO, J. C. 1999. Composición genética y posible origen de las poblaciones humanas canarias deducidos del polimorfismo de su ADN mitocondrial. Ph.D. diss., Universidad de La Laguna, Tenerife.
- RANDO, J. C., ET AL. 1998. Mitochondrial DNA analysis of Northwest African populations reveals genetic exchanges with European, Near-Eastern, and sub-Saharan populations. *Annals of Human Genetics* 62:531–50.
- . 1999. Phylogeographic patterns of mtDNA reflecting the colonization of the Canary Islands. *Annals of Human Genetics* 63:413–28.
- ROBERTS, D. F., ET AL. 1966. Blood groups and the affinities of the Canary Islanders. *Man* 1:512–25.
- SALAS, A., ET AL. 1998. MtDNA analysis of the Galician population: A genetic edge of European variation. *European Journal of Human Genetics* 6:365–75.
- SCHWARZFISCHER, F., AND K. LIEBRICH. 1963. Serologische Untersuchungen an prähistorischen Bevölkerungen insbesondere an altkanarischen Mumien. *Homo* 14:129–33.
- SCHWIDETSKY, I. 1956. Anthropologische Beobachtungen auf Tenerife. *Homo* 7:142–52.
- . 1975. *Investigaciones antropológicas en las Islas Canarias: Estudio comparativo entre la población actual y la prehistórica 10*. Tenerife: Publicaciones del Museo Arqueológico de Tenerife.
- SCOZZARI, R., ET AL. 1999. Combined use of biallelic and microsatellite Y-chromosome polymorphisms to infer affinities among African populations. *American Journal of Human Genetics* 65:829–46.
- TORRONI, A., ET AL. 1998. MtDNA analysis reveals a major late Paleolithic population expansion from southwestern to northeastern Europe. *American Journal of Human Genetics* 62:1137–52.
- UNDERHILL, P. A., ET AL. 1997. Detection of numerous Y chromosome biallelic polymorphisms by denaturing high-performance liquid chromatography. *Genome Research* 7:996–1005.
- VERNEAU, R. 1887. Rapport d'une mission scientifique dans l'Archipel Canarien. *Archives des Missions Scientifiques et Littéraires* 3d series, 18:567–817.
- WÖLFEL, D. J. 1930. Sind die Ureinwohner der Kanaren ausgestorben? *Zeitschrift für Ethnologie* 62:282–302.

Male Contribution to Diet and Female Reproductive Success among Foragers¹

FRANK MARLOWE
 Department of Anthropology, Peabody Museum,
 Harvard University, Cambridge, Mass. 02138, U.S.A.
 (fmarlowe@fas.harvard.edu). 7 VI 01

Male-female bonds in humans have long been attributed to the need for male assistance with provisioning of offspring (Westermarck 1929, Lovejoy 1981, Lancaster and Lancaster 1983). Recently, however, attention has been drawn to several features of modern foragers (and presumably our ancestors) that challenge the paternal-investment theory of human pair bonding: (1) Men in some

© 2001 by The Wenner-Gren Foundation for Anthropological Research. All rights reserved 0011-3204/2001/4205-0009\$1.00

1. I thank Nick Blurton Jones, Barry Hewlett, and three anonymous referees for their helpful comments.

foraging societies contribute less to the diet than women do (Hiatt 1974, Kelly 1995) yet marriage still exists. (2) Forager women are less dependent on husbands when they can gather food and when they live with their own kin, and postmarital residence is much more flexible among foragers than in traditional agricultural societies (van den Berghe 1979, Marlowe 2000a). (3) Hunting provides such unpredictable returns that it should be a poor strategy for provisioning children (Hawkes, O'Connell, and Blurton Jones 1991). (4) The foods men acquire (e.g., meat, fish, and honey) tend to be shared widely outside the household (Kaplan and Hill 1985, Bliege Bird and Bird 1997, Hawkes, O'Connell, and Blurton Jones 2001), with the result that the wives of good foragers may receive no more food than others in camp. (5) The effect of fathers' absence on offspring survival does not predict marital stability as well as do men's mating opportunities (Blurton Jones et al. 2000). These challenges to the paternal-investment theory have left some asking just how important provisioning by men is to forager women.

Females should allocate available energy to reducing offspring mortality and/or increasing fertility in ways that maximize their own reproductive success (Lack 1968, Smith and Fretwell 1974). Therefore, in this paper I use demographic data on foragers to test whether male provisioning—for which male contribution to diet serves as a proxy—enhances female reproductive success. While others have analyzed the demography of some foraging populations (Hewlett 1991; Bentley, Jasienska, and Goldberg 1993; Kelly 1995; Pennington 1996; Sellen and Mace 1997, 1999) and some have analyzed male contribution to diet among foragers (Lee 1968, Hiatt 1974, Barry and Schlegel 1982, Ember 1978, Kelly 1995), here I consider the two together. In addition, I control for habitat quality. The comparative approach I adopt ignores variation within societies, which would be more informative about cause and effect, but allows me to generalize across all foraging societies. I will show that male contribution to diet does enhance female reproductive success and go on to discuss the implications of this for our mating system.

MATERIALS AND METHODS

The sample included every society that derived all or almost all of its subsistence from foraging and had data on at least one of the relevant variables, a total of 161. Many of these data come from previous compilations (Hewlett 1991, Kelly 1995, Hill and Hurtado 1996). The variables examined were the following:

1. Primary biomass (kg/m^2), a measure of the resource abundance or quality of a habitat. These data were taken from Kelly (1995) or calculated using his formulas for deriving primary biomass from primary production—the plant biomass expected given the effective temperature and precipitation. Even though there is relatively less edible food for humans as primary biomass increases (per kilogram), the absolute amount of edible food increases because there are more kilograms per square meter (Kelly 1995). Primary biomass (henceforth simply biomass) may

be a crude measure of habitat quality, but it is an important control variable in this analysis. Because there was no formula for arctic biomass, arctic societies were excluded from all tests that control for biomass.

2. Male contribution to diet, the percentage of the diet produced by men as opposed to women. In some cases, the data available are only estimates based on the sex that tends to acquire certain foods and the percentage of those foods in the diet (Barry and Schlegel 1982, Kelly 1995), but I used actual measurements wherever they were available. For example, in the case of the Hadza, I weighed all food entering camp, converted this to kilocalories by type of food, and then calculated the percentage of the total kilocalories contributed by adult males. (Ideally, one would measure actual consumption by all members of a household while in camp and out foraging and the exact amount of kilocalories acquired by individuals on their own versus the amount given to them by others over a long period of time and for all households.)

3. Infant mortality, the percentage of children who die within the first year of life (0–1 yr).

4. Juvenile mortality, the percentage of children who die before age 15 (0–15 yr).

5. Postinfant juvenile mortality, mortality between ages 1 and 15 years.

6. Total fertility rate (TFR), the mean number of children women have borne by the time they reach menopause.

7. Age at weaning, the mean age at which children are weaned.

8. Interbirth interval, the mean number of years between births, whether the previous child survived or died before the birth of the subsequent child.

9. Female reproductive success, the number of children surviving to age 15, calculated by multiplying TFR by juvenile survivorship ($100\% - \text{juvenile mortality}$) and dividing by 100. This takes into account both fertility and mortality and is the best measure of the target of natural selection.

All tests are either Pearson correlations or multiple linear regressions, with $p < 0.05$ recognized as significant.

RESULTS

Variation in male contribution to diet. Male contribution to diet varies from 25 to 100%, with a mean of 64% (S.D. = 18.3, $n = 95$). Because there is less edible plant food for women to gather in colder climates, male contribution is higher at higher latitudes, where effective temperature is lower ($r = -.512$, $p < 0.0005$, $n = 82$). Male contribution is also greater where biomass is greater ($r = .300$, $p < 0.031$, $n = 52$), but because there is no formula for calculating the biomass of arctic environment this correlation is inflated by the exclusion of low-biomass arctic habitats, where the importance of male provisioning is indisputable. In environments where effective temperature is greater than 13°C (between about latitudes 45°N and 45°S , mean male contribution to diet is 55% (S.D. = 15.9, $n = 36$). Not sur-

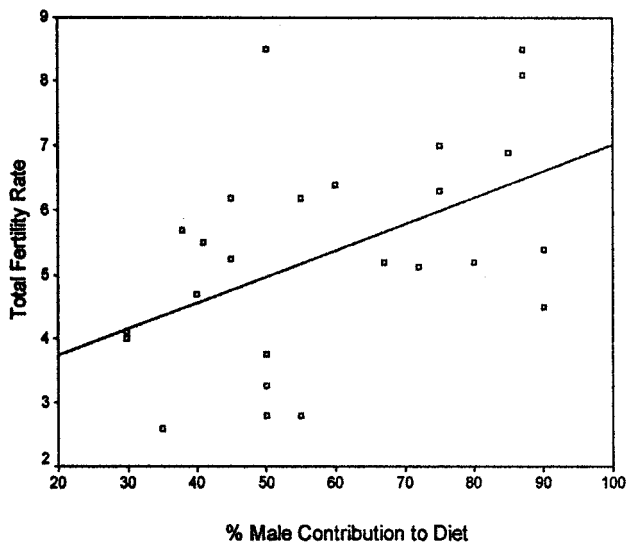


FIG. 1. Total fertility rate as a function of male contribution to diet ($\beta = .497$, $p = 0.010$, d.f. = 24).

prisingly, male contribution to diet is lower where more of the diet comes from gathering ($r = -.742$, $p < 0.0005$, $n = 62$) and higher where more of the diet comes from fishing ($r = .513$, $p < 0.0005$, $n = 62$). It is not significantly higher where more of the diet comes from hunting ($r = .238$, $p < 0.063$, $n = 62$). It is higher in the New World (70%, S.D. = 14.9, $n = 67$) than in the Old World (50%, S.D. = 17.9, $n = 27$), even when effective temperature is greater than 13°C to exclude arctic societies (62%, $n = 20$ versus 47%, $n = 16$). Both hunting and fishing contribute more to the diet in the New World.

Variation in subadult mortality. Mean infant mortality is 22.32% (7.8–46%, S.D. = 9.4, $n = 18$). Infant mortality may be largely due to diseases that paternal investment can do little to ameliorate. Since diseases should be more prevalent in warmer, wetter habitats, we might expect infant mortality to increase with biomass, and to a degree it appears to. Mean juvenile mortality is 45.08% (22–61%, S.D. = 9.4, $n = 20$). Perhaps because the immune system improves with age and exposure, juvenile and postinfant juvenile mortality appear to decrease slightly with increasing biomass, suggesting that infant and juvenile mortality have different causes (and see Sellen and Mace 1999).

Variation in female fertility and reproductive success. Mean TFR is 5.28 (.81–8.5, S.D. = 1.68, $n = 46$). We might expect fertility to be higher in high-biomass habitats, and to a degree it appears to be ($r = .130$, n.s.). However, it should also be affected by mortality, since when a nursling dies and nursing ceases there should be a shorter interval to the next birth. Alternatively, more children may put more strain on parental resources, resulting in higher offspring mortality. TFR is significantly correlated with juvenile mortality ($r = .530$, $p = 0.016$, $n = 20$) but not with infant or postinfant juvenile mortality.

Therefore it is probably the mortality of nurslings up to three or four years old rather than infants below one year that is most closely correlated with TFR, but there are too few populations for which nursling mortality rates are available to test this.

Mean age at weaning is 2.62 years (1–4.5, S.D. = .84, $n = 41$) and is slightly less where TFR is higher. Mean interbirth interval is 3.22 (1.75–4.83, S.D. = .76, $n = 18$). The later women wean, the longer lactational subfertility should delay the next birth, and, as expected, interbirth interval is longer when age at weaning is greater ($r = .803$, $p = 0.016$, $n = 8$). Neither age at weaning nor interbirth interval is correlated with biomass. Mean female reproductive success is 3.11 (1.93–5.27, S.D. = .79, $n = 20$) and appears to increase with biomass ($r = .359$, n.s.).

The impact of male contribution to diet on subadult mortality. There is no relationship between male contribution to diet and infant mortality, even when both biomass and TFR are controlled. The same is true of juvenile mortality and postinfant juvenile mortality, although when biomass and TFR are controlled postinfant juvenile mortality is slightly but not significantly lower where male contribution to diet is greater ($\beta = -.632$, $p = 0.213$, d.f. = 6). The potential benefit of greater male provisioning to older, weaned children in contrast to infants would be straightforward, since their nutritional status could be directly enhanced. Given the small number of cases, all we can say is that male contribution to diet may lower postinfant juvenile mortality but certainly does not appear to lower infant mortality. This again suggests that infant and postinfant juvenile mortality have different causes and that if we could analyze

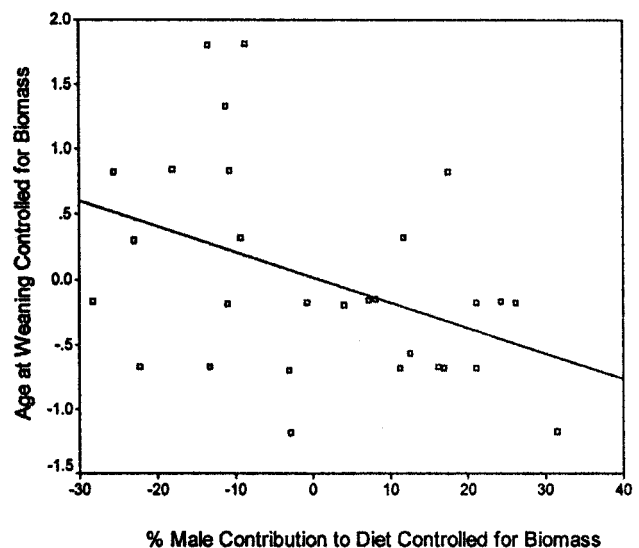


FIG. 2. Age at weaning as a function of male contribution to diet, controlling for primary biomass of habitat ($\beta = .437$, $p = 0.028$, d.f. = 26).

postweaning juvenile mortality (children 4–15 years old), we might see an effect of male contribution to diet.

The impact of male contribution to diet on female fertility and reproductive success. Although greater male contribution to diet may or may not reduce subadult mortality, its impact on fertility is clear. Where male contribution to diet is greater, women's fertility (TFR) is higher ($\beta = .497$, $p = 0.010$, d.f. = 24) (fig. 1). Controlling for biomass, the positive effect of male contribution to diet on TFR is even greater ($\beta = .740$, $p = 0.003$, d.f. = 20). Greater male contribution to diet must allow females to maintain ovarian function better by foraging less and spending less energy or by weaning earlier or both. Age at weaning is lower where male contribution to diet is greater ($r = .279$, $p = 0.081$, $n = 40$) and significantly lower controlling for biomass ($\beta = -.437$, $p = 0.028$, d.f. = 26) (fig. 2).

Because greater male contribution to diet appears to help women wean earlier and have more children, we might expect it to shorten the interbirth interval. However, higher mortality will also have this effect, since a woman can more quickly resume cycling after the death of a child and the end of nursing. Interbirth interval is therefore not a straightforward measure of reproductive success, since it can be short because of high fecundity or because of high mortality. When juvenile mortality is controlled, interbirth interval is shorter where male contribution to diet is greater, though not significantly. Here the lack of significance can be attributed to the inclusion of the Batak, for whom births are concentrated early in life, when interbirth interval tends to be shorter. Mean age at last birth is 26.3 for the Batak (Eder 1987), compared with 39 for other foragers (Kaplan et al. 2000),

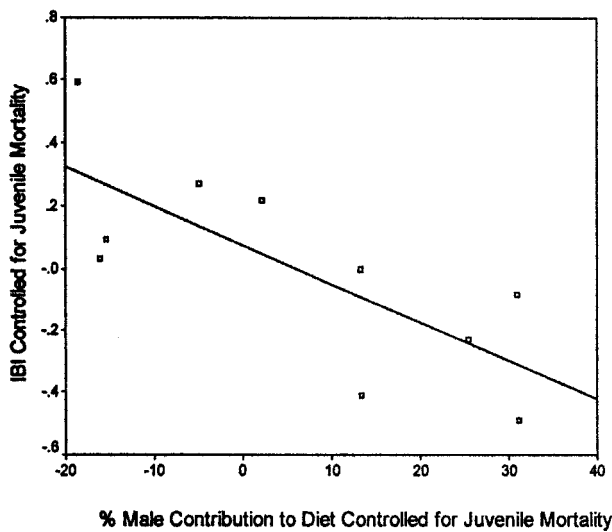


FIG. 3. Interbirth interval as a function of male contribution to diet, controlling for juvenile mortality, with the Batak excluded ($\beta = -.765$, $p = 0.007$, d.f. = 7).

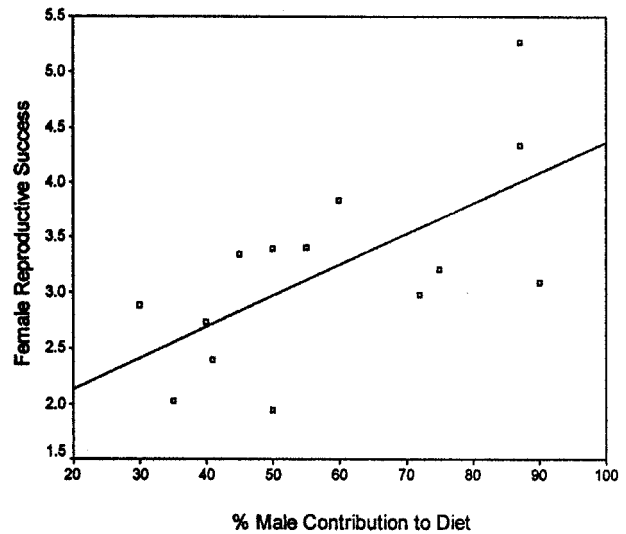


FIG. 4. Female reproduction success as a function of male contribution to diet ($\beta = .648$, $p = 0.0012$, d.f. = 12).

partly because women may ingest a sterility-inducing plant after a difficult birth or on reaching a desired number of children (Eder 1987). When the Batak are excluded and juvenile mortality is controlled, greater male contribution to diet does predict a shorter interbirth interval ($\beta = -.765$, $p = 0.007$, d.f. = 7) (fig. 3). When biomass is also controlled, the effect of male contribution to diet is not significant ($\beta = -.585$, $p = 0.076$, d.f. = 5), but if nursing mortality could be controlled there would likely be a stronger effect.

Since greater male contribution to diet increases TFR, which in turn is positively correlated with juvenile mortality, it is not clear whether we should expect greater male contribution to diet to reduce subadult mortality. What matters is whether it leads to greater female reproductive success, the result of trade-offs between fertility and mortality, and it does. The greater male contribution to diet, the higher female reproductive success ($\beta = .648$, $p = 0.012$, d.f. = 12, $n = 14$) (fig. 4). Controlling for biomass makes the effect of male contribution to diet even stronger ($\beta = .861$, $p = 0.007$, d.f. = 10).

Male contribution to diet predicts female reproductive success mainly because it raises TFR. Female reproductive success is not significantly correlated with infant mortality, juvenile mortality, postinfant juvenile mortality, interbirth interval, or age at weaning. The only demographic variable correlated with it is TFR ($r = .808$, $p < 0.0005$, $n = 20$). Controlling for biomass, and regardless of which other demographic variables are entered in a regression analysis, TFR still has the strongest significant relationship to female reproductive success.

Male contribution to diet and TFR are much greater in the New World, especially in South America, than in

the Old World. Although some areas significantly predict female reproductive success, male contribution to diet remains the best predictor of female reproductive success throughout the world, even after controlling for area. The surest way to increase female reproductive success is to increase TFR, and that is what greater male contribution to diet appears to do.

DISCUSSION

Human mortality in the first year is the same as that of chimpanzees. However, by age 5 chimpanzee mortality is 40% (Hill et al. 2001), considerably higher than among human foragers. This suggests that there is little extra that forager women can do to reduce mortality in the first year but that provisioning by men may help around the time of weaning. When there is little else females can do to reduce offspring mortality, they should use energy coming from males to increase fertility. We can even expect females to accept some increase in mortality if that is the price of sufficiently increased fertility to yield the highest reproductive success in the end. They might, for example, prefer that males forage rather than provide direct care even if direct care could help reduce infant mortality.

Without help from males, females might get enough children weaned to reproduce themselves but take longer and lose out to females getting help. The regression line in figure 4 shows that reproductive success is expected to be about 2.2 when male contribution to diet is 20%, whereas the mean reproductive success for all foragers is 3.11. Even with low rates of mortality, 2.2 children reaching age 15 could simply be too few. But a male contribution to diet of only 20% would still be a gain for females unless males ate more of the food in camp than the 20% they brought in. There is no reason male contribution to diet must be over 50% to represent a real benefit to females; what matters is the amount of food over and above personal consumption that males contribute.

What explains the variation in male contribution to diet? As noted, it is greatest in cold climates, where there is little that females can gather. The higher level of male contribution in the New World might be due to cultural inertia, if migrants through the Arctic maintained high levels of male contribution even after moving into other habitats, or to high mortality due to diseases in the New World to which humans had less resistance, with high mortality requiring high TFR and high male contribution. Differences in types of foods available may be important. Females may contribute a greater percentage of the diet in low-biomass habitats because in such places tubers, which are usually acquired by women, are a more important source of food. In high-biomass habitats, females may need to provide more direct care to prevent higher nursing mortality, and if this caregiving interferes with their foraging (see Hurtado et al. 1992, Marlowe n.d.) males may have to provide more food. Alternatively, female preference for providers may everywhere

push male contribution up to the highest level males can afford, given the opportunity costs.

One opportunity cost of paternal provisioning is the pursuit of mates. Since male contribution to diet appears to increase fertility rather than reduce mortality, one could argue that it reflects mating rather than parenting effort. If so, is it mating effort directed toward one's own wife or toward all females? Men should pursue a foraging strategy that maximizes their own reproductive success, which means that they may trade off provisioning of wives and children for maximizing mating opportunities through affairs, simultaneous polygyny, or serial monogamy and remarriage of younger wives later in life, a strategy which could be far from optimal for their wives (Marlowe 2000b). Prior to effective tools, it would have been difficult for males to acquire much surplus food for provisioning, and what they could acquire would have been, for the most part, the same food that females acquired. With increased foraging efficiency, males may have gained more by targeting foods different from those females acquired since their trade value would have been greater, resulting in a sexual division of labor. However, once males began to bring in high-nutrient foods such as meat and honey, they would have become more vulnerable to scrounging from everyone in camp, diluting the potential benefit going to wife and children.

Since male contribution to diet is only a proxy for actual provisioning, these results do not confirm the paternal-investment theory of pair bonding. It is conceivable that females could obtain the same benefit from male contribution to diet by trading sex for food at every opportunity as they could from bonding and trading paternity confidence for long-term provisioning. These results do, however, show that male contribution to diet has important consequences for forager women—a necessary condition for the paternal-investment theory. Women begin reproducing later than chimpanzee females and have only a slightly longer reproductive span but manage to exceed them in fertility through earlier weaning (Hawkes et al. 1998) and shorter interbirth intervals (Kaplan et al. 2000). This analysis suggests that one way they achieve this is through male provisioning. While the paternal-investment theory has usually emphasized the benefit of reduced offspring mortality, the real benefit of male provisioning among foragers is increased fertility.

References Cited

- BARRY, H., AND A. SCHLEGEL. 1982. Cross-cultural codes on contribution by women to subsistence. *Ethnology* 21: 165–88.
- BENTLEY, G., G. JASIENSKA, AND T. GOLDBERG. 1993. Is the fertility of agriculturalists higher than that of nonagriculturalists? *CURRENT ANTHROPOLOGY* 34:778–85.
- BLIEGE BIRD, R. L., AND D. W. BIRD. 1997. Delayed reciprocity and tolerated theft: The behavioral ecology of food-sharing strategies. *CURRENT ANTHROPOLOGY* 38:49–78.
- BLURTON JONES, N. G., F. MARLOWE, K. HAWKES, AND J. F. O'CONNELL. 2000. "Paternal investment and hunter-gatherer divorce rates," in *Human behavior and adap-*

- tation: *An anthropology perspective*. Edited by L. Cronk, N. Chagnon, and W. Irons, pp. 65–86. New York: Elsevier.
- EDER, J. F. 1987. *On the road to tribal extinction*. Berkeley: University of California Press.
- EMBER, C. 1978. Myths about hunter-gatherers. *Ethnology* 17: 439–48.
- HAWKES, K., J. F. O'CONNELL, AND N. G. BLURTON JONES. 1991. Hunting income patterns among the Hadza: Big game, common goods, foraging goals, and the evolution of the human diet. *Philosophical Transactions of the Royal Society of London* 334:243–51.
- . 2001. Hadza meat sharing. *Evolution and Human Behavior* 22:113–42.
- HAWKES, K., N. G. BLURTON JONES, H. ALVAREZ, AND E. L. CHARNOV. 1998. Grandmothering, menopause, and the evolution of human life histories. *Proceedings of the National Academy of Sciences, U.S.A.* 95:1336–39.
- HEWLETT, B. S. 1991. Demography and childcare in preindustrial societies. *Journal of Anthropological Research* 47:1–37.
- HIATT, B. 1974. "Woman the gatherer," in *Woman's role in aboriginal society*. Edited by F. Gale, pp. 4–15. Canberra: Australian Institute of Aboriginal Studies.
- HILL, K., C. BOESCH, J. GOODALL, A. PUSEY, J. WILLIAMS, AND R. WRANGHAM. 2001. Mortality rates among wild chimpanzees. *Journal of Human Evolution* 40:437–50.
- HILL, K., AND A. M. HURTADO. 1996. *Ache life history: The ecology and demography of a foraging people*. New York: Aldine.
- HURTADO, A. M., K. HILL, H. KAPLAN, AND I. HURTADO. 1992. Trade-offs between female food acquisition and child care among Hiwi and Ache foragers. *Human Nature* 3: 185–216.
- KAPLAN, H., AND K. HILL. 1985. Food sharing among Ache foragers: Tests of explanatory hypotheses. *CURRENT ANTHROPOLOGY* 26:223–46.
- KAPLAN, H., K. HILL, J. LANCASTER, AND A. M. HURTADO. 2000. A theory of human life history evolution: Diet, intelligence, and longevity. *Evolutionary Anthropology* 9: 156–85.
- KELLY, R. L. 1995. *The foraging spectrum: Diversity in hunter-gatherer lifeways*. Washington, D.C.: Smithsonian Institution Press.
- LACK, D. 1968. *Ecological adaptations for breeding birds*. London: Methuen.
- LANCASTER, J. B., AND C. S. LANCASTER. 1983. "Parental investment: The hominid adaptation," in *How humans adapt*. Edited by D. Ortner, pp. 33–58. Washington, D.C.: Smithsonian Institution Press.
- LEE, R. B. 1968. "What hunters do for a living, or How to make out on scarce resources," in *Man the hunter*. Edited by R. B. Lee and I. DeVore, pp. 30–48. Chicago: Aldine.
- LOVEJOY, O. 1981. The origin of man. *Science* 211:341–50.
- MARLOWE, F. 2000a. Paternal investment and the human mating system. *Behavioural Processes* 51:45–61.
- . 2000b. The patriarch hypothesis: An alternative explanation of menopause. *Human Nature* 11:27–42.
- . n.d. Why get married? Foraging, mating, and parenting among Hadza hunter-gatherers. MS.
- PENNINGTON, R. 1996. Causes of early human population growth. *American Journal of Physical Anthropology* 99:259–74.
- SELLEN, D. AND R. MACE. 1997. Fertility and mode of subsistence: A phylogenetic analysis. *CURRENT ANTHROPOLOGY* 38: 878–89.
- . 1999. A phylogenetic analysis of the relationship between sub-adult mortality and mode of subsistence. *Journal of Biosocial Science* 31:1–16.
- SMITH, C. S., AND S. D. FRETWELL. 1974. The optimal balance between size and number of offspring. *American Naturalist* 108:499–506.
- VAN DEN BERGHE, P. 1979. *Human family systems*. Prospect Heights: Waveland.
- WESTERMARCK, E. 1929. *Marriage*. New York: Jonathan Cape and Harrison Smith.

Ethnocentrism and Xenophobia: A Cross-Cultural Study¹

ELIZABETH CASHDAN

King's College Research Centre Human Diversity Project, King's College, Cambridge CB2 1ST, U.K. and Department of Anthropology, University of Utah, 270 S 1400 E Rm 102, Salt Lake City, Utah 84112-0060, U.S.A. (elizabeth.cashdan@anthro.utah.edu). 25 IV 01

People readily though not inevitably develop strong loyalties to their own ethnic group and discriminate against outsiders. In this report I use cross-cultural data to (1) determine the factors that strengthen and weaken these tendencies and (1) ascertain whether they have the same determinants. It is often supposed that ethnocentrism and xenophobia are opposite sides of the same coin, but a few voices have cautioned that this need not be the case.

Van den Berghe (1999) points out that it would be maladaptive for xenophobia to be an inevitable result of ethnocentrism. Ethnic affiliation, he reminds us, usually involves some claim of common ancestry (real or fictive), and a propensity to favor fellow ethnics is no doubt enhanced by this feeling of kinship. But reciprocal relationships with members of other groups can frequently be adaptive also, and it would be foolish to assume an attitude of hostility. The threshold for cooperation may be higher and the insistence on reciprocity may be greater, but a smart opportunist keeps his options open.

Recent experimental work in psychology also suggests that in-group favoritism is not a necessary concomitant of out-group hostility (Rabbie 1982, 1992; Ray and Lovejoy 1986; Struch and Schwartz 1989). While both can be enhanced by competition and external threats (see Sherif 1961 for the classic field experiment), in-group favoritism should be expected only if affiliation with the in-group can successfully counter the competitive threat (Rabbie et al 1974). If a group is unable to be successful, hostility to outsiders may be mirrored by ethnic breakdown and further hostility and competition within the group. Finally, threats can arise from environmental catastrophes as well as from outsiders, and we might expect that such disasters would foster enhanced group loyalty without any concomitant hostility to outsiders.

The cross-cultural data analyzed here provide no support for the proposition that out-group hostility is a nec-

© 2001 by The Wenner-Gren Foundation for Anthropological Research. All rights reserved 0011-3204/2001/4205-0005 \$1.00

1. This research was undertaken as part of the King's College Human Diversity Project. I am particularly indebted to Robert Foley and the King's College Research Centre of Cambridge University for financial support and a stimulating intellectual environment. I also thank Napoleon Chagnon, Carol Ember, Patrick Gray, Hartmut Lang, Alan Rogers, Pierre van den Berghe, and Polly Wiessner for helpful advice.