References and Notes

1. M. Dickerman, *Annu. Rev. Ecol. Syst.* **6**, 107 (1975); B. Montag and T. Montag, *Minn. Med.* **62**, 368 (1979).
2. J. Calhoun, *Sci. Am.* **206**, 139 (February 1962); R. Curtin and P. Dolhinow, *Am. Sci.* **66**, 468 (1978).
3. R. Trivers, in *Sexual Selection and the Descent of Man*, B. Campbell, Ed. (Aldine, Chicago, 1972), p. 136; S. Hrdy, *Ethol. Sociobiol.* **1**, 13 (1979).
4. J. DeFries and G. McLearn, *Evol. Biol.* **5**, 279 (1972).
5. J. Labov, *Behav. Ecol. Sociobiol.* **6**, 297 (1980); F. vom Saal, paper presented at a meeting of the International Society of Developmental Psychobiology, Cincinnati, November 1980.
6. All CF1 male mice used in the studies were housed separately in plastic mouse cages when 35 days old and tested when 70 to 90 days old.
7. Males that had been paired were placed on either side of a wiremesh partition in a box (30 by 30 by 15 cm) for 1 hour. Whatman 2 filter paper was used as the substrate, and when it is placed under a black light fluorescent bulb (GE F15T8-BLB), the individual urine marks can be counted [C. Desjardins, J. A. Maruniak, F. H. Bronson, *Science* **182**, 939 (1973)].
8. Two newborn male or female mice were placed in a cage with a sexually naïve male ($N = 15$ males per group). The results were (i) female pups: infanticide, 44 percent; parental toward, 19 percent; and ignored, 37 percent; (ii) male pups: infanticide, 44 percent; parental toward, 25 percent; and ignored, 31 percent (χ^2 test, $P > .1$). Other sexually naïve males ($N = 30$ per group) were tested for their response to newborn or 7-day-old mice by the same procedure. The results were (i) newborn mice: infanticide, 50 percent; parental toward, 23 percent; and ignored, 27 percent; (ii) 7-day-old mice: infanticide, 43 percent; parental toward, 50 percent; and ignored, 7 percent ($P > .1$).
9. A male mouse virtually never exhibits infanticide toward one newborn mouse and parental behavior toward the other when two young are placed into the male's cage.
10. F. vom Saal, *J. Reprod. Fertil.* **62**, 633 (1981); in *Hormones and Aggressive Behavior*, B. Svare, Ed. (Plenum, New York, in press).
11. ——, S. Pryor, F. Bronson, *J. Reprod. Fertil.* **62**, 33 (1981).
12. M. Smith, *Fed. Proc. Fed. Am. Soc. Exp. Biol.* **39**, 2571 (1980).
13. F. vom Saal, unpublished observations; S. Mantelenakis and M. Ketchell, *J. Reprod. Fertil.* **12**, 391 (1966).
14. Each pregnant female was housed in a cage (30 by 30 by 15 cm) divided in two by a wooden partition. Three hours after a litter was delivered, a male was placed into the empty area for 30 minutes, after which the barrier was removed. Males were thus placed with females before the time of postpartum estrus. The number of days from the introduction of the male to the delivery of the next litter was recorded, and the original young that survived were weaned at this time or when they were 30 days old. The number of young in the new litter that survived to 5 days of age was counted to assess the possibility that some young had been killed.
15. J. Reimer and M. Petras, *J. Mammal.* **48**, 88 (1967); W. Lidicker, *J. Anim. Ecol.* **45**, 677 (1976).
16. J. Maruniak, C. Desjardins, F. Bronson, *Am. J. Physiol.* **233**, E-495 (1977).
17. R. Lande, *Evolution* **34**, 292 (1980); S. Maxon, in *The Biology of Aggression*, P. Brain and D. Benton, Eds. (Sijthoff & Noordhoff, Alphen aan den Rijn, Netherlands, 1981), p. 69.

18. We thank S. Blaffer-Hrdy and J. A. Maruniak for their suggestions during the preparation of this manuscript and J. Broich for her excellent technical assistance. Supported by NIMH grant MH35079 and by University of Missouri Institutional Biomedical Research Support grant RR 07053 from the National Institutes of Health.

9 October 1981; revised 8 December 1981

Malnutrition and Fertility

Direct measurements of plasma prolactin concentrations in lactating women on both supplemented and nonsupplemented diets in Gambia (*1*) do not support Bongaarts' (*2*) view that the length of lactational amenorrhea is mainly dependent on the suckling stimulus. Plasma prolactin concentrations fell more rapidly among mothers on supplemented diets although the frequency of breastfeeding remained the same (*1*). Also, seasonal variations in plasma prolactin and milk yield were associated with significant differences in maternal nutrition and physical labor and were not explained by the frequency of infant feeding (*1, 3, 4*). The prolonged high prolactin concentrations found in undernourished mothers may ensure milk synthesis when food intake is limited. "The lower hormonal levels associated with improved maternal nutrition may shorten the period of post-partum infertility despite prolonged breast feeding" (*1*).

Lactational amenorrhea can range from a median of 5.3 months in Boston to 17.7 months in Zaire (*5*). In a prospective study in Bangladesh, Chen *et al.* (*6*) found that the median duration of lactational amenorrhea with a surviving child was 17 months. This long period was related both to child feeding practices and to the poor nutritional status of the mother and child. These authors noted that "while fertility was high by Western standards, it was well below the biological maximum, particularly in the 20–24 age group," and was not explained by use of contraceptives or induced abortion. A seasonal variation in number of births was observed, as well as a seasonal effect on termination of lactational amenorrhea; the frequency of termination rose at the peak of the major rice harvest. Chen *et al.* concluded that the interaction of maternal nutrition and child feeding practices "suggests that a nutritional programme could possibly shorten the duration of lactational amenorrhea by 50 percent or more. If this were to occur, it would shorten the average birth interval by about 20 percent, producing a corresponding rise in the birth rate, unless some other means of fertility control were introduced and accepted."

The lactation data cited by Bongaarts from Kippley and Kippley (*7*) are atypical and incomplete. The data are reported as "number of experiences," which is not defined, and these differ markedly from the number of mothers. There are no data on age, parity, or nutrition of the mothers.

An important omission in the Bongaarts discussion of the interaction of nutritional status and the frequency of suckling is the fact that after 6 months of age, and sometimes earlier, as was found in the Gambia (*4*), breast milk alone is not sufficient for the proper growth and health of the infant (*8*). Continued nursing, however, is important because breast milk is often the only source of high-quality protein.

Bongaarts' evaluation of the significance of the delaying effect of undernutrition on age of menarche (*9*) is based on the assumption that menarche is an independent event in the reproductive span. Data on both historical and contemporary populations, however, show that the age of menarche is integrated with rates of physical growth and reproductive potential; the age of menarche of a population is thus part of a consistent biological history (*10, 11*). For example, among the seasonally, marginally nourished Bush people of the Kalahari (*12*), the average age of menarche is 15.5 to 16.0 years (*13*). A young woman who marries at age 16 has a low probability of having a child the first year because of the long period of adolescent subfecundity, which lasts until physical growth is completed. In fact, the average maternal age at the birth of a first child among the Bush people is 20 to 21 years (*13*). In the United States, however, where the mean age of menarche is now 12.8 years, girls have completed their growth and the period of adolescent subfecundity by age 16 (*11*); a 16-year-old bride, therefore, would be at high risk of pregnancy the first year of marriage.

Although Bongaarts doubts a secular trend in age of menopause, recent data, which classified women under the most conservative definition of the menopause—12 consecutive months without a cycle—show that the menopause takes place now on average at 51 years of age in a well-nourished society (*14*), whereas historically it was 47 to 48 years (*10*). These data, therefore, indicate a secular trend of age of menopause similar to the secular trend in age of menarche of 3 or 4 months per decade (*9, 15*). It has also been reported that fat women have a later age of menopause than do slender women (*16*), and plump girls have an earlier age of menarche (*10, 16*). Therefore, the reproductive span of well-nourished, noncontraceptive women on average is longer than that of undernourished women (*10, 16, 17*). Of course, in most well-nourished societies reproduction

0036-8075/82/0305-1272$01.00/0

ends long before the natural age of menopause because of the use of contraception.

Data show also that weight loss in women does not have to be in the starvation range, as Bongaarts suggests, before menstrual cycles cease. Not only do menstrual cycles in nonanorectic as well as anorectic women cease with weight loss in the range of 10 to 15 percent of normal weight for height (*18–20*), but this weight loss is accompanied by hypothalamic dysfunction (*18*) and reduction of concentrations of the hormones that control ovulation (*20*). Knuth *et al.* (*21*) state that "dietary treatment of patients with amenorrhea and loss of weight may replace gonadotropin therapy for induction of ovulation in a significant proportion of patients with anovulatory infertility." It has also been found that physical exercise and hard work may delay menarche (*22–24*) and cause irregularity and cessation of cycles (*22, 23, 25*), although the mechanisms are not as yet known.

Finally, Bongaarts' interpretation of the causes of differences in age-specific marital fertility rates historically, such as the 18th-century Canadian population, must be viewed with caution since there are no details of the components of diet or physical growth rates for these populations.

ROSE E. FRISCH
Harvard Center for Population Studies, Cambridge, Massachusetts 02138

References and Notes

1. P. G. Lunn, S. Austin, A. M. Prentice, R. G. Whitehead, *Lancet* **1980-I**, 623 (1980).
2. J. Bongaarts, *Science* **208**, 564 (1980).
3. R. G. Whitehead, M. Hutton, E. Müller, M. G. M. Rowland, A. M. Prentice, A. Paul, *Lancet* **1978-II**, 178 (1978).
4. A. A. Paul, E. M. Müller, R. G. Whitehead, *Trans. R. Soc. Trop. Med. Hyg.* **73**, 686 (1979).
5. J. Knodel, *Science* **198**, 1111 (1977).
6. L. C. Chen, S. Admed, M. Gesche, W. H. Mosley, *Popul. Stud. (London)* **28**, 277 (1974).
7. S. K. Kippley and G. F. Kippley, *J. Obstet. Gynecol. Neonatal Nurs.* **1**, 15 (1972).
8. N. Scrimshaw and N. Behar, *N. Engl. J. Med.* **27**, 137 (1965); *ibid.*, p. 193.
9. J. M. Tanner, *Growth at Adolescence* (Blackwell, Oxford, ed. 2, 1962).
10. R. E. Frisch, *Science* **199**, 22 (1978); in *Patterns and Determinants of Natural Fertility*, H. Leridon and J. Menken, Eds. (Ordina, Liege, 1979), pp. 137–147.
11. ——, *Hum. Biol.* **48**, 353 (1976); *Fed. Proc. Fed. Am. Soc. Exp. Biol.* **39**, 2395 (1980).
12. L. Van Walt, E. N. Wilmsen, T. Jenkins, *J. Clin. Endocrinol. Metab.* **46**, 658 (1978).
13. N. Howell, *The Demography of the Dobe !Kung* (Academic Press, New York, 1979).
14. L. J. B. Jaszmann, in *The Management of the Menopause and Post-Menopausal Years*, S. Campbell, Ed. (University Park Press, Baltimore, 1976), pp. 11–23.
15. G. Backman, *Acta Anat.* **4**, 421 (1948).
16. B. Sherman, R. Wallace, J. Bean, L. Schlabaugh, *J. Clin. Endocrinol. Metab.* **52**, 488 (1981).
17. R. E. Frisch, *Nat. Hist.* **89**, 16 (1980).
18. R. A. Vigersky, A. E. Andersen, R. H. Thomson, D. L. Loriaux, *N. Engl. J. Med.* **297**, 1141 (1977).
19. M. P. Warren *et al.*, *J. Clin. Endocrinol. Metab.* **40**, 601 (1975); R. E. Frisch and J. W. McArthur, *Science* **185**, 949 (1974).
20. R. M. Boyar *et al.*, *N. Engl. J. Med.* **291**, 862 (1974).
21. U. A. Knuth, M. G. R. Hill, H. S. Jacobs, *Br. J. Obstet. Gynaecol.* **84**, 801 (1977).
22. R. E. Frisch, G. Wyshak, L. Vincent, *N. Engl. J. Med.* **303**, 17 (1980).
23. R. E. Frisch *et al.*, *J. Am. Med. Assoc.* **246**, 1559 (1981).
24. M. P. Warren, *J. Clin. Endocrinol. Metab.* **51**, 1150 (1980).
25. E. Dale, D. H. Gerlach, A. L. Wilhite, *Obstet. Gynecol.* **54**, 47 (1979).

3 November 1980; revised 10 July 1981

In discussing the relation between nutrition and lactational amenorrhea it is important to distinguish between the effects of maternal and infant nutrition. Supplementing the nursing infant's diet shortens lactational amenorrhea (*1–4*). As the periods of breast-feeding shorten and the intensity of suckling declines, the concentration of the ovulation-inhibiting hormone prolactin also declines, eventually resulting in the return of normal ovulatory function (*1, 5–8*). The question at issue is whether the mother's nutritional status can influence amenorrhea if breast-feeding and suckling patterns are unchanged.

According to Frisch, a study of Gambian women (*9*) conflicts with my view that the length of lactational amenorrhea is mainly dependent on the suckling stimulus. The Gambian study, however, did not measure the suckling stimulus. Lunn *et al.* (*9*) only note that the frequency of breast-feeding of mothers on both supplemented and unsupplemented diets was within the range of 10 to 16 episodes per day. They do not report on other variables that affect amenorrhea, such as the duration of breast-feeding periods, the intensity of suckling, or the amount of dietary supplementation given to infants. One explanation for shorter amenorrhea among mothers with better diets is that they have more food available to supplement their infants, who are then satisfied with shorter and less intense periods of breast-feeding. This is apparently the reason for the shorter amenorrhea intervals among supplemented mothers in a Mexican study (*10–12*). An alternative explanation, suggested by Gray (*13*) is that more vigorous suckling is required by infants of poorly nourished mothers, whose ability to produce milk is reduced by their low nutritional status. Both explanations are consistent with the present understanding of the determinants of lactational amenorrhea (*1, 5, 8*) and are more plausible than Frisch's suggestion about the minimum weight required for the onset of postpartum ovulation (*14*). In fact, we have shown that maternal weights decline throughout postpartum amenorrhea (*15*).

Chen *et al.* (*16*) mention the possibility of an effect of maternal nutrition on postpartum ovulation, but they lacked the data to determine whether this effect existed. Several investigations specifically designed to test this relation in the same area of Bangladesh failed to find a significant effect of maternal nutritional status (*3, 17, 18*). This finding is consistent with the results of the Kippley study (*19*). Since the purpose of this study (*19*) was to collect data from U.S. women with longer than average breast-feeding intervals, the sample is not nationally representative, but this in no way affects the conclusion that women in well-nourished poulations such as the United States can experience lactational amenorrhea lasting well over a year if they practice unrestricted natural breast-feeding. (The number of breast-feeding experiences in this study exceeded the number of women because some women reported data for more than one child.)

Nutritional variations in age at menarche have a small demographic impact because actual childbearing in virtually all societies starts at a later age, at the time of marriage or a consensual union (*12, 20*). Contrary to Frisch's assertion, my evaluation of this relation does not assume menarche to be an independent event. A close correlation between ages at menarche and marriage as well as some adolescent subfecundity were specifically included in my calculations (*12*). The existence of a prolonged period of adolescent subfertility, as suggested by Frisch, is not supported by the data from Howell's study of the Bush people (*21*). The mean age at menarche is 16.6 years (*21*, p. 178), and the mean age at first birth is around 19—18.8 for the older women and 19.9 for the most recent period (*21*, p. 163). This delay is not unusually long, especially if one takes into account the unexposed time between menarche and marriage for a large proportion of women and the substantial incidence of sterility-inducing venereal disease (*21*).

Frisch compares one of the highest observed means for age at menopause in a modern population with typical values in historical populations. An unbiased comparison would be either between normal values in both cases [47 to 50 years in western countries (*12*) versus 47 to 48 years historically (*22*)], or between highest observed means [51.4 years in the Netherlands (*23*) versus 50.7 for the Bantu in South Africa (*24*)]. These data show no important effect of nutrition on the end of the childbearing years. The same conclusion is reached if one compares mean ages at last birth of poorly nourished and well-nourished populations in which no deliberate birth control is practiced (*12*).

As I have noted elsewhere (*12*), large sudden reductions in weight are certainly associated with disturbances of menstruation and ovulation patterns. This is observed during famines but also among otherwise well-nourished women who go on a starvation diet. However, the question of interest to demographers and nutritionists is whether ovulation and conception rates are affected by the chronic moderate malnutrition that prevails in many of the poorest developing countries. Studies of women in Bangladesh (*17*) and Guatemala (*15*) found no relation between conception rates and levels of nutrition. Apparently, disturbances of ovulation do not occur in women with relatively stable weights even if these weights are low.

Finally, birth intervals and marital fertility rates in 18th-century Canada and in the laboring classes of 19th-century England are virtually the same as in the Hutterite population, despite wide differences in nutritional status (*12*). Hutterites are healthy and well nourished (*25*), while diets were poor in 19th-century England (*22*). Nutritional data for 18th-century Canada are not available, but mortality was high (*26*), and it is unlikely that nutrition levels would approach that of the Hutterites.

On balance, I see no reason to change my conclusion (*12*) that chronic moderate malnutrition of mothers has only a minor effect on their fertility.

JOHN BONGAARTS
Center for Policy Studies, Population Council, One Dag Hammarskjold Plaza, New York 10017

References and Notes

1. A. S. McNeilly, P. W. Howie, M. J. Houston, in *Research Frontiers in Fertility Regulations*, G. I. Zatuchni *et al.*, Eds. (Northwestern Univ. Press, Chicago, 1980), pp. 102–116.
2. A. Perez, P. Vela, R. G. Potter, G. Masnick, *Popul. Stud. (London)* **25**, 491 (1971).
3. S. L. Huffman and A. M. K. Chowdhury, *ibid.* **32**, 251 (1978).
4. P. Buchanan, *Popul. Rep. Ser. J.* **4**, 49 (1975).
5. J. E. Tyson and A. Perez, in *Nutrition and Human Reproduction*, W. H. Mosley, Ed. (Plenum, New York, 1978), pp. 11–27.
6. P. Delvoye, M. Demaegd, J. D. Delogne, C. Robyn, *J. Biosocial Sci.* **9**, 447 (1977).
7. P. W. Howie, A. S. McNeilly, M. J. Houston, A. Cook, H. Boyle, *Br. Med. J.* **283**, 757 (1981).
8. A. S. McNeilly, *Br. Med. Bull.* **35**, 151 (1979).
9. P. G. Lunn, S. Austin, A. M. Prentice, R. G. Whitehead, *Lancet* **1980-I**, 623 (1980).
10. A. Chavez and C. Martinez, *Nutr. Rep. Int.* **7**, 1 (1973).
11. J. D. Wray, in *Nutrition and Human Reproduction*, W. H. Mosley, Ed. (Plenum, New York, 1978), pp. 197–223.
12. J. Bongaarts, *Science* **208**, 564 (1980).
13. R. Gray, in *Determinants of Fertility in Developing Countries: A Summary of Knowledge* (National Academy of Sciences, Washington, D.C., in press).
14. R. Frisch, *Soc. Biol.* **22**, 17 (1975).
15. J. Bongaarts and H. Delgado, in *Patterns and Determinants of Natural Fertility*, H. Leridon and J. A. Menken, Eds. (Ordina, Liege, 1979), pp. 107–133.
16. L. C. Chen, S. Admed, M. Gesche, W. H. Mosley, *Popul. Stud. (London)* **28**, 277 (1974).
17. A. M. K. Chowdhury, in *Nutrition and Human Reproduction*, W. H. Mosley, Ed. (Plenum, New York, 1978), pp. 401–410.
18. W. H. Mosley, in *Patterns and Determinants of Natural Fertility*, H. Leridon and J. A. Menken, Eds. (Ordina, Liege, 1979), pp. 83–104.
19. S. K. Kippley and G. F. Kippley, *J. Obstet. Gynecol. Neonatal Nurs.* **1**, 15 (1972).
20. J. Menken, J. Trussell, S. Watkins, *J. Interdiscip. Hist.* **11**, 425 (1981).
21. N. Howell, *The Demography of the Dobe !Kung* (Academic Press, New York, 1979).
22. R. E. Frisch, *Science* **199**, 22 (1978).
23. L. Jaszmann *et al.*, *Int. J. Fertil.* **14**, 2 (1969).
24. G. Frere, *S. Afr. J. Med. Sci.* **36**, 21 (1971).
25. J. W. Eaton and A. J. Mayer, *Hum. Biol.* **25**, 206 (1953).
26. J. Henripin, *La Population Canadienne au Début du XVIII Siecle* (Institut National d'Etudes Démographiques, Paris, 1954), vol. 22.

1 December 1981

Eye Movement Patterns in Infants

Birnholz (*1*) has reversed historical fact and thereby misrepresented my concepts of sleep which underlie my current research efforts. He asserted that "Rapid eye movement activity during sleep in infants was . . . rediscovered by Aserinsky and Kleitman." The publication by Aserinsky and Kleitman (*2*) cited by Birnholz makes absolutely no reference to rapid eye movements or REM activity in infants, and neither does any other publication bearing my name. Indeed, an excerpt from my thesis (*3*), which underlies all my earliest publications on sleep is as follows:

> A new type of eye movement was discovered to occur in the sleep of adults and a child but not in infants. Motion pictures confirmed the presence of these eye movements, which were binocularly synchronous, rapid and jerky. It was suggested that they be termed "rapid" eye movements in contrast to the slow eye movements previously reported.

By definition, the term "rapid eye movement" (REM) signifies two conditions. (i) The ocular activity must occur during sleep, and (ii) it must be of a saccadic type. Inasmuch as a saccadic movement can occur in either waking or sleeping, the critical factor in determining whether an eye movement is a REM or not is predicated on recognizing the concurrent state of consciousness. How does one distinguish between sleep and waking? In general, skeletal muscle tone is considerably lower during sleep than in waking, but as any sleep investigator quickly learns, this is not a foolproof guide. A more practical guide is through examination of the pattern of ocular activity. This is predicated on previous investigators having successfully correlated the pattern of ocular movement with other physiological or psychological measures to denote the concurrent state of consciousness. For adult subjects, the normative pattern is distinct and demarcation of defined REM periods is well established. In infants, however, especially those younger than 4 weeks, reliance on ocular pattern to discriminate REM from waking is diffuse, and their shifts from one state of consciousness to another seem erratic. Moreover, while in older subjects the REM state has the invariant characteristic of being preceded by sleep without eye movements, what might tentatively be termed the "REM state" in young infants is preceded by the waking state. The commonly used criteria for identifying REM in an adult are thus no longer tenable in the young infant.

Birnholz's statement that the ocular activity in a 23- or 24-week fetus is REM activity must be a speculation, since it is not known whether the fetus is asleep, or for that matter awake. One might surmise that the fetus is in a primitive, nether state of consciousness and that the primordial ocular activity is not REM but rather the progenitor for both waking eye movements and REM.

EUGENE ASERINSKY
Department of Physiology, Marshall University School of Medicine, Huntington, West Virginia 25701

References and Notes

1. J. C. Birnholz, *Science* **213**, 679 (1981).
2. E. Aserinsky and N. Kleitman, *J. Appl. Physiol.* **8**, 11 (1955).
3. E. Aserinsky, thesis, University of Chicago (1953), p. 76.

10 August 1981; revised 16 December 1981