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# Biological variables in forager fertility performance: a critique of Bongaarts' model

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**BIOLOGICAL VARIABLES IN  
FORAGER FERTILITY PERFORMANCE:  
A CRITIQUE OF BONGAARTS' MODEL**

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## BIOLOGICAL VARIABLES IN FORAGER FERTILITY PERFORMANCE:

### A CRITIQUE OF BONGAARTS' MODEL

#### The model

Bongaarts (1978, 1980) has presented a model in which indices for intermediate variables that are important in controlling fertility performance in human populations are specified quantitatively. Four variables are isolated; these are: 1. proportion married among females; 2. contraceptive use and effectiveness; 3. prevalence of induced abortion; 4. duration of postpartum infecundability. The variables are associated in the model with rate modulating indices: 1. index of proportion married:  $C_m = f(a)/(f(a)/m(a))$ , where  $m(a)$  is the proportion of women currently married and  $f(a)$  is their age specific fertility rate schedule (note that Bongaarts specifies that only legitimate births should be counted); 2. index of contraception ( $C_c$ ), based on estimates of contraceptive use and effectiveness along with an estimate of female sterility; 3. index of induced abortion ( $C_a$ ), a function of total fertility rate and total abortion rate; 4. index of postpartum infecundability ( $C_i = 20/18.5 - i$ , where  $i$  is the mean duration of postpartum infecundability in months; see below for discussion).

These indices are translated into population specific fertility rates as follows:

$$\text{TFR} = C_m \times \text{TM}, \quad (1)$$

$$\text{TM} = C_c \times C_a \times \text{TN}, \quad (2)$$

$$\text{TN} = C_i \times \text{TF}, \quad (3)$$

$$\text{TF} = \text{TN}/C_i. \quad (4)$$

TFR is the total fertility rate defined as total age-specific fertility ( $f(a)$ ). TM is the total marital fertility rate defined as the total age-specific marital fertility ( $f(a)/m(a)$ ). TN is the total natural marital fertility rate defined as fertility uninhibited by any cultural factor other than sanctioned

than sanctioned marriage (including consensual unions). TF is the total fecundity rate defined as the rate of ability to reproduce, but again, only within the legitimate realm of marriage. Bongaarts derives a constant value (15.3) for this parameter.

Bongaarts (1980:14, Fig. 2) demonstrates that model estimates of total fertility rates agree closely with observed TFR in thirty selected cases, 97% of observed variation being explained. The model, thus, is potentially a powerful tool for estimating in a quantitative manner the effects of specified intermediate variables in all societies. More importantly, it suggests means for policy intervention measures which can be effective in influencing fertility performance in desired directions. It is, therefore, worthwhile to examine closely the contribution of each intermediate variable to total fertility performance in order to arrive at analytically more satisfying understandings of the mechanisms by which these variables exert their effects. The examination must search for the underlying variables that provide the driving power of those more tangible variables labelled intermediate.

A naturally breeding population (in the definition of Henry 1961:81) offers the best avenue for such an investigation because variables can be controlled independently according to their action upon either social or biological parameters of fertility. The  $\frac{Zu}{\text{Oasi}}$  (!kung San) of southern Africa were, until recently, a population of this kind. A large amount of prospective data relating to the fertility performance of these people over a significant period of time have been obtained; these data will be applied to Bongaart's model in order to clarify its variables. But, first, it will be necessary to consider some of the major characteristics of the model itself.

Three model indices ( $C_m$ ,  $C_c$ ,  $C_a$ ) are solely sociological measures; underlying these are social institutions which have ideological and behavioral

components that are not addressed either by the model or by Bongaarts in his discussions. Nevertheless, in order to remove ambiguity from the use of these indices in this paper, their referents in natural populations will be clarified. The standard definition of natural fertility is that of Henry (1961: 81): fertility in the absence of deliberate parity dependent birth control. Now, restricting fertility to marriage is certainly "deliberate parity dependent birth control" and is imposed by human societies not so much to restrict biological procreation as to give the results of procreation their proper social identity. In societies of all types, marriage is frequently consequent upon demonstrated fertility (whether by accident or design) and much of the low frequency of "illegitimacy" reported for non-contracepting societies (Nag 1968) can be attributed to Howell's (1979:232) observation that "people are highly effective in adjusting marriage to match the fait accompli of pregnancy." Bongaarts (1980, Table 4) documents the fact that those populations which interfere least with conception have the highest index of marriage; also, in these populations TM more closely approximates TN than it does in others.

The model makes these relations clear. By definition, a naturally breeding population is one in which  $C_c = 1$  (no contraception) and  $C_a = 1$  (no induced abortion). In this case,  $TM = TN$  (from 2), and when  $C_m = 1$ ,  $TFR = TN$  (from 1). Consequently, in societies that place no restrictions on mating, other than those defined as incest,  $TFR = TM = TN$ . Accordingly,  $TFR = C_i \times TF$  (substituting in 3) and total fertility rate is depressed from potential fecundity limits only by postpartum infecundability. In any case, it can be shown that  $C_m$  does not add explanatory power to the model but merely shifts its focus from TM to TFR. Table 1 reproduces Bongaarts' 1980, Table 3 plus relevant parts of Table 2; in addition, column 8 of Table 1 lists a model MTM

calculated by  $TM = C_c \times C_a \times TN$  (2). Clearly, this equation -- which excludes  $C_m$  -- is as good an estimator of fertility performance as is  $TFR = C_m \times C_c \times C_a \times C_i \times TF$ , the expanded form of (1) used by Bongaarts (1978, 1980). Excluding  $C_m$  is equivalent to setting it at 1 and the tabulated results of doing so provide theoretical justification for ignoring the distinction between TFR and TM in naturally breeding populations.

Empirical support is found in the Žu/Ōasi case. Marriage occurs early, usually being formalized shortly after menarche. Both women and men, however, enter into sexual unions that are independent of first marriage, and stable marital unions are, as Howell observes, routinely established on the basis of an accomplished pregnancy rather than vice versa. Stochastic perturbations that affect a women's mating behavior do occur (differential availability of sanctioned mates, divorce, widowhood) but these have their greatest effect on the youngest and oldest cohorts in the reproductive pool. Continual cohabitation probably does lead to higher frequency of intercourse and, hence, presumably, to higher probability of conception (see Bongaarts 1976 for a recent, thorough consideration of this point). Again, the youngest women -- because they have not established long-term unions -- and older widows are most effected. Howell (1979:241, Table 12.7) gives probability of remarriage for divorced Žu women as 0.5 at one month and, for those women remaining unmarried after that, as 0.6 at one year; for widows, the probabilities are 0.4 and 0.2 respectively. Thus, divorce results in only short periods of non-cohabitation and has little effect on mating. Howell notes that reproductively able women remarry more quickly than do others and that most widows who remain unmarried are post-menopausal. Clearly, differential access to mates is more important than is marriage in controlling fertility in zu society. An estimation of its effect must be included. The course adopted in

this presentation will be to assume that  $C_m$  is an index of mating rather than of marriage and to set it at 1.0 for all but the youngest and oldest cohorts in an age specific fertility schedule. The terminal cohorts will be calculated in the usual way. For comparison, a  $C_m$  index calculated according to Bongaarts' model will also be presented.

The form of the model used in the remainder of this presentation, therefore, will be:

$$TFR = C_m \times C_i \times TF. \quad (5)$$

This equation also overcomes the redundancy that has crept into the model where TN and TM are derivations of TFR. The components of  $C_i$ , which are both sociological and biological, will be considered in relation to the test case to which I now turn.

#### The zu/oasi case: 1963 - 1973

The  $\check{z}u/\check{o}asi$  are a division of the !kung (San) group of northern Bushmen who live in Botswana, Namibia, and Angola. The group referred to in this paper lives in northwestern Botswana (Fig. 1). Anthropologically, they are among the classic foraging peoples who are characterized as obtaining all of their food from wild plant and animal sources (Lee 1979). While it is now known that the picture is considerably more complex than this and that pastoralist economies were established in the region more than 1000 years ago (Wilmsen 1981), the group studied has been only marginally pastoralist within living memory. Until 1979 these people obtained 75% - 95% of their subsistence from non-domesticated plants and animals (Table 2) and met the criterion of natural breeding.

Howell (1979:135) examined the reproductive histories of all women in this group who had completed their fertility by 1973 and found, on the basis of age

specific fertility of all cohorts, no evidence of deliberate fertility limitation. She also found that the congruence of marriage and paternity was high not because the nonmarital conception rate was low but because marriage was adjusted to paternity (Howell 1979:232; see also Harpending 1971). My data substantiate these observations and extend them to 1979. In late September 1979, introduced food relief programs made available large quantities of refined carbohydrates and other foods, thereby stabilizing the diet for a period of one year (Table 2). About this time, oral contraceptives were also introduced.

Table 3 is an age specific fertility schedule for women in the population during the years 1963 - 1973. From column 1 of this table,  $f(a) \times 5$  yields  $TFR = 4.275$ . It should be borne in mind that this is not a completed fertility schedule because all of the cohorts represented were still in their reproductive years. An estimate of completed fertility may be obtained from Howell (1979:124) who gives  $TFR = 4.691$  for 62 women who had completed their reproductive lives in 1973. Both values are among the lowest ever recorded. In this paper, the lower value (4.275) will be used in subsequent calculations because it is not possible to calculate  $m(a)$  for the older women; hence,  $C_m$  cannot be estimated. Relevant calculations are included in Table 3.  $C_m$  is 0.945, or by Bongaarts' method, 0.882.  $C_i$  may be estimated. Howell (1979: 134) gives 35.4 + 13.9 months as the birth interval for 1963 - 1973 which suggests approximately 24 months postpartum infertility for these women. Following the procedure of Bongaarts (1980:27), who equates postpartum infertility primarily with amenorrhea due to lactation, and extrapolating his table, yields  $C_i = 0.471$ . Model TFR may then be calculated:  $0.882 \times 0.471 \times 15.3 = 6.356$  or  $0.945 \times 0.471 \times 15.3 = 6.811$ . These values are essentially in agreement and are more than 50% higher than observed TFR. Even if  $TFR = 4.691$



is used as representative of *zu* completed fertility, the model overestimates performance by 40%.

Aside from the fact that neither model value approximates the observed TFR, the procedure is in error because  $C_i = 0.471$  if attributed to breastfeeding alone requires a mean lactation duration of 36 months according to Bongaarts' method. This is the entire length of the observed birth interval. TFR and  $C_m$  are observed values in this case and, thus, are fixed; if TF is assumed to be invariate, then  $C_i$  required to achieve the observed fertility performance may be calculated by  $TFR/C_m \times TF = 4.275/0.882 \times 15.3 = 0.295$  or, using 0.882, = 0.317. To achieve this level, according to Bongaarts (1980, appendix table), postpartum amenorrhea would have to extend for 44-48 months corresponding to about 60-64 months of continuous lactation. Obviously, such routines were not maintained by *zu/oasi* women. In fact, the longest reported estimates of mean birth intervals is  $44 \pm 1.7$  months (Howell 1979:135; Lee 1979:321; Konner and Worthman 1980) and the longest mean duration of lactation about 36 months. We must search further.

The *zu/oasi* case: 1976 - 1980

Table 4 is a complete log of the reproductive events at /ai/ai (see Fig. 1 for location) during 1976 - 1980, inclusive. Table 5 is an age specific fertility schedule of these events, and like its counterpart Table 3, is not a completed fertility schedule; all cohort members retain potential fertility. There were 47 births to 32 of 41/42 women at risk during the period. Observed TFR = 6.88. A remark should be made, however; no births occurred in cohorts (40-44) and (45-49). Following on previous performance, 0.65 and 0.22 births respectively would be expected in these cohorts. It is likely that the small sample size accounts for this lack of births, but it is worth noting that these are the cohorts identified by Howell (1979:185) as having a higher than average secondary infertility in 1963-1973.

Model indices may be calculated. From Table 5,  $C_m$  is 0.891 (discounting unattached states -- which, in any case, were fertile) and 0.837 (Bongaarts' method). Mean birth interval during this period is 27.2 months (Table 4); this value is identical to that reported by Konner and Worthman (1980) who also give 24 months as the mean duration of lactation for these  $\check{z}$ u in 1975, a figure in substantial accord with my observations in 1976 - 1980.  $C_f$ , estimated as before, is then 0.563. Model TFR is calculated:  $0.837 \times 0.563 \times 15.3 = 7.210$  and  $0.891 \times 0.563 \times 15.3 = 7.670$ . These results are reasonably close to observed TFR = 6.88. When it is considered that completed TFR would probably be close to 7.50, the model results approximate contemporary  $\check{z}$ u fertility performance even more closely.

A slight adjustment to model TFR must be made to account for the low incidence of oral contraception that began in the community in September 1979. In that month, a 39-year-old, multiparous, married woman began oral contraception; she was lactating at the time and left the area shortly thereafter. In December 1979, two sexually active, nulliparous, unmarried school-girls aged 17 and 18 began regular oral contraception. These two women, but not the 39-year-old who departed, are considered in  $C_c$  values (0.950 or 0.990). Applying these values to model TFR yields results ranging from 6.85 to 7.60, a marginal decrease and still well within the observed and estimated TFR range 6.88 - 7.50. Note that this rate is among the highest reported.

#### Comparison of the cases

It is clear that the intermediate variables specified in the Bongaarts model are capable of accounting for contemporary  $\check{z}$ u fertility performance but not for that of 1963 - 1973. Howell (1979:186) plots expected numbers of births for each year 1910 - 1970 against reported births of 165  $\check{z}$ u women who were reproducing during that period; except for the two cohorts noted above,

performance conforms reasonably well to expectations. The 1963 - 1973 case may, therefore, be taken to be typical of previous decades in what may be termed traditional time. Consequently, the model fails for the greater part of zu history.

Both the contemporary and the traditional cases are "natural" with respect to reproduction; neither places extrinsic controls upon mating nor attempts to constrain conception. (The fact that effective contraception was introduced at the end of the 1976 - 1980 case, distorts that situation slightly, but only two women had adopted its use by August 1980 and only 0.338 ( $2 \times 0.169$ ) additional births might have been expected had they not done so.) The contemporary case conforms to the usually accepted expectation of natural fertility, that of parity-independence and high TFR (Henry, 1961). The traditional case, however, does not: its characteristic is one of low TFR coupled with parity-independence.

On this evidence, a definition of "natural" fertility drawn from historical societies, all of which are administered, must be more closely examined. Such societies interfere with the realization of fertility in some way, either by limiting marriage (low  $C_m$ ) in conjunction with inhibiting nonmarital fertility ( $T_M$  higher than TFR), or by effective contraception (low  $C_c$ ), or both. All of the societies cited by Bongaarts are such administered societies, with the possible exception of Bangladesh. The value of an unmanaged case such as is offered by studies of Zu/Oasi is that it allows us to specify in unambiguous terms the constituents of naturalness and, thus, to examine more closely those parameters that are free to vary independently of social constraint. We may, thereby, reach a fuller understanding of the reproductive process and be better able to construct effective policy for influencing fertility performance.

It was argued above that it is preferable to speak of an index of mating rather than an index of marriage in naturally breeding societies. In the Žu 76-80 case, five of the 47 observed births (three to unmarried women, and two fathered by men not the husbands of the mothers), would be eliminated if legitimacy were enforced as an acceptance criterion. These five births are 11% of the total; eliminating them would distort the description of Žu fertility.  $C_m$ , as index of mating, in both Žu cases fluctuates around 0.9 with most of the departure from 1.0 accounted for in the relatively unproductive youngest and oldest cohorts. This Žu ( $C_m$ ) value is probably representative of natural societies in general; we may therefore conclude that in such societies  $C_m$  accounts for little of the variance in fertility.

The Žu cases demonstrate clearly that parity independence coupled with varying fertility -- rather than fixed high fertility -- may be characteristic of naturally reproducing human populations. This variation cannot be accounted for by prohibitions on mating. The question then centers upon fecundability and its constraints.

#### Postpartum infecundability

Arguments over postpartum infecundability tend to be univariate in construction. There are, however, both social and biological components of that reproductive state. Social constraints are expressed as postpartum abstinence and prescriptions concerning breastfeeding and supplementation of infant diets. For the Žu case, during the period under direct consideration in this analysis (1963 - 1980), postpartum abstinence did not exceed 2-3 months, a period far too short to have any effect on  $C_i$ . Both the social and the practical ideal dictate long demand breastfeeding; the infant is carried on the mother's back and is allowed to suckle at will. In the past, breastfeeding continued for 36 months or longer, and in 1980 it was still practiced for about 24 months.

Bronte-Steward et al. (1960:194) report, however, that in 1952 <sup>v</sup>Zu women supplemented infant diet as soon as the child could sit unaided. Howell (1979:121) observes that all mothers supplement infant diets after a few months by pre-masticating or pounding foods with mortar and pestle. My observations confirm this practice through 1980. These observations are in accord with Wray's (1978:222) statement that breast milk alone cannot sustain normal infant growth beyond six months. Typically, as in the <sup>v</sup>Zu case, lactation is prolonged much more than is abstinence and, hence, has a greater potential for influencing fertility. Accordingly, Bongaarts has defined  $C_i$  exclusively as a function of lactation, but it must be recognized that what is actually measured is the social practice of breastfeeding. A biological effect -- suppression of ovulation -- is attached to this measurement and is assumed to be the principal biological effect (aside from abnormalities) upon natural fertility performance (Bongaarts 1980:568). However, biological parameters, perturbations in which may influence fertility performance, underlie fecundity as well as fecundability; that is, not only  $C_i$  but also TF may vary.

The principal competing models that purport to describe and explain these parameters attempt either to relate body weight to fecundity or to see natural fertility controlled primarily by lactation. Frisch (1972, 1978; Frisch and McArthur 1974; Frisch and Revelle 1970) is the leading exponent of a fatness hypothesis. Short (1976) and McNeilly (1979) may be considered the leading spokesmen for the lactation hypothesis although Tietze (1961) and Gonzalez (1964) anticipated current developments in this field. In reality, both hypotheses, because their mechanisms are set ultimately in body composition, are sub-species of an encompassing body composition hypothesis.

The hypotheses are usually considered separately -- even antagonistically -- much to the detriment of knowledge. A growing body of literature makes it

clear, however, that such a separation is not tenable (Wray 1978; Whitehead et al. 1978; Paul et al. 1979; Lunn et al. 1981; see Harrell 1981 for a current summary available to anthropologists). Let us consider some current studies on the relation between nutrition, lactation, and fertility, after which we proceed to an overview of endocrinology relating to the issue, and then examine relevant data from the  $\bar{z}$ u cases.

Almost everyone has questioned the Frisch hypothesis (Johnston et al. 1971, Johnston et al. 1975; Huffman 1978; Delgado et al. 1979) and, indeed, it is vague and unspecified as presented. But as Huss-Ashmore (1980) observes, it has not yet been tested and seldom is nutritional status considered along with parity. Bongaarts (1980; Delgado et al. 1979) attempts such a test. He (Bongaarts 1980: 567) uses data obtained by the INCAP study in Guatemala (Delgado et al. 1978) to argue that there is no nutritional effect on length of postpartum amenorrhea by showing that there is no significant difference in duration of amenorrhea between women of low and of high nutrition (measured by weight); he also claims to find no difference associated with caloric intake. Close reading of these data reveal, however, that there is no difference in caloric intake among these women when differences in their weights are considered (mean weight of low group is 43.7 kg, of high group is 55.6 kg; mean caloric intake/day is 1309 kcal and 1960 kcal respectively). Mean intake is about 29 kcal/kg of body weight for both groups. As no other measure of nutritional level is given, no conclusion about nutritional effect can be drawn. Similarly, when citing Chowdhury (1978), Bongaarts fails to heed that author's warning that his Bangladesh study may be biased because 17.6% of subjects of lowest weight ( $\bar{x}$ =38.5 kg, .26 kg/cm) had not conceived in five years compared with 6.7% of these with highest weights ( $\bar{x}$  = 42.4 kg, .33 kg/cm).

Wray (1978) is called upon by Bongaarts (1980:567) to question results obtained by Chavez and Martinez (1973) with regard to a nutrition-lactation

link in Mexico; and, indeed, those results are questionable. Notice, however, that Wray (1978:201-203) also examines the INCAP data. He notes that in this study women who gained relatively little weight during pregnancy, lost more weight postpartum, and remained below their pre-pregnant weight while lactating were able (emphasis mine) to lactate for significantly shorter periods (less than half as long) than were women who gained and maintained highest weights (Fig. 2 reproduces Wray 1978, Fig. 1). He finds "that duration of lactation decreases by as much as 60 percent" when maternal caloric intake is extremely low (1978:203). The findings of Aono et al. (1976) are relevant here; their experiments indicate that if nutritional environment is held constant, individual mammary glands increase milk production in response to increased prolactin levels; as nutritional status declines, more prolactin is required to evoke equivalent milk synthesis. Paul et al. (1979) consider this relationship at length and posit a competition for nutrients between maternal tissue requirements and milk production in marginally nourished women. Whitehead et al. (1978) examine the relation of nutrition to milk production and discuss the feedback mechanisms involved.

Lunn et al. (1980) show a direct relation between maternal nutrition and prolactin levels in a study that controlled for suckling frequency and activity of mothers. Fig. 3 reproduces their results. Gambian women on native dietary regimes of 1490 kcal/day and 1650 kcal/day had prolactin levels above those at which ovulation might be suppressed (860  $\mu$ U/ml: Delvoye et al. 1978) for periods longer than a year (up to 22 months) while 45% of women who were supplemented by the investigators to 2300 kcal/day were below that level at 6 - 7.5 mo. At about one year, all of the supplemented group were below 860  $\mu$ U/ml. All of these women in the three strata suckled their infants on demand. Lunn et al. (1980:625) conclude that higher prolactin levels will be

needed to maintain adequate milk production in undernourished women if that hormone preferentially channels energy to the breast for milk synthesis. Delvoe et al. (1977) calculate that suckling frequencies of 6 times/day are sufficient to maintain prolactin levels high enough to suppress ovulation for 12 months in adequately nourished women. It is worth noting that the longest observed durations of postpartum amenorrhea associated with demand lactation are on the order of fourteen months in well-nourished industrial societies (Kippley and Kippley, 1974) and tend to be shorter than this length of time (Salber et al. 1965). Where nutritional status is reduced, amenorrhea associated with lactation reaches, but seldom exceeds, twenty-four months (Huffman 1978).

These investigations, taken together, underscore the need to examine body habitus ecology systematically rather than merely to correlate variables in piecemeal fashion. To follow such a course diverts attention from variables, as such, to the interactive restructuring of variables in the body environment. Steroid metabolism is a logical place to begin.

#### Steroid metabolism and transport

Current clinical investigations into the relations between body habitus and steroidal function will be summarized quickly; a more detailed synthesis with respect to the problem at hand is forthcoming (Wilmsen n.d.)

The original hypothesis that estrogen production arises not exclusively by glandular secretion but additionally by extraglandular formation via utilization of circulating C<sub>19</sub> steroidal precursors at sites other than ovaries or adrenal glands was formulated in a series of papers by MacDonald, Siiteri, Grodin, and Rombaut (MacDonald et al. 1967; MacDonald et al. 1969; Grodin et al. 1973). These investigators found that, of all potential androgen precursors, circulating androstenedione is most efficiently aromatized to



estrogen in both normal males and non-pregnant females (Grodin et al. 1973:207), that in normal, young adult females 44 mg/day and in similar males 18 mg/day of estrone may arise from plasma borne androstenedione (MacDonald et al. 1967:1103), and that utilization of circulating androstenedione accounts for 10% - 50% of estrone production in normal, non-pregnant, ovulating females -- the percentage varying with the menstrual cycle (MacDonald et al. 1969:770). The same extent of conversion was found in adrenalectomized and oophorectomized females; therefore, "an extraglandular source of estrogen of potentially sizable magnitude may arise from plasma borne C<sub>19</sub> precursors" (MacDonald et al. 1967:1104). Grodin et al. 1973:207 report similar function in castrate females; these findings "implicate adipose tissue as a significant site of production and/or metabolism of estrone derived from circulating androstenedione . . . [via] . . . slow release of estrone derived from androstenedione from body fat deposits" (Grodin et al. 1973:213).

Since then, many studies have expanded these findings. Nimrod and Ryan (1975) report a testosterone to estradiol conversion in abdominal and breast fat tissue in addition to an androstenedione to estrone pathway. Longcope et al. (1978) substantiate this finding and suggest that, assuming a constant rate throughout the body, 20% - 25% of the extragonadal conversion of androgens to estrogens is accounted for by muscle and 10% - 15% by adipose tissues. These latter workers also find aromatization in brain, kidney, and bone. Finally, Perel and Killinger (1979) find human adiposite precursors to estrogen conversion and Forney et al. (1981:192) find rate dependent conversion correlated significantly with "calculated body component mass of adipose tissue" as well as adipose cell number. Their studies demonstrate that the capacity of adipose tissue to aromatize estrogens from androgens is a function of body component mass of adipose tissue (Forney et al. 1981:198).

The studies just cited were mainly concerned with reproductive hormones in the human female. Similar functioning has been demonstrated in males. Lee et al. (1977) found testosterone, follicle stimulating hormone, and cholesterol (but not luteinizing hormone) to be significantly depressed in nutritionally deprived males. Stanik et al. (1981) find changes in rate of peripheral conversion of androstenedione and testosterone to estrone and estradiol accompanying changes in weight of obese men. Klibanski et al. (1981) report that depressed testosterone in fasting men is not accompanied by decreased luteinizing hormone levels; therefore, fasting does not primarily effect testicular function. It is clear that body habitus effects male steroid function as it does female.

Recent indications that body composition affects steroidal transport are also relevant. Nisker et al. (1980) and Dunn et al. (1981) report that variations in body weight are associated with the capacity of sex-hormone-binding globulin (SHBG) and albumin to transport testosterone and estradiol. Two conclusions of significance to arguments of diet related functions in fertility are reached by these authors: "Changes in body-fat composition may thus influence reproductive performance though the intermediate effects of serum SHBG, which regulates the availability of estradiol to the brain and other target tissues" (Nisker et al. 1980). 2. "These results emphasize the importance of considering albumin binding in in vivo studies of sex hormone physiology" (Dunn et al. 1981:65). When considered in conjunction, peripheral aromatization of C<sub>19</sub> steroids and their transport by liver synthesized and tissue stored proteins indicate a potentially significant role for dietary elements in fertility performance. Adipose tissue may act as a steroid sink from which androgens and estrogens are released to blunt short-term alterations in circulating levels.

This very brief summary is sufficient to indicate a substantial relation between body composition and fertility performance. All of the intrinsic variability, as well as the observed variation, in fertility performance noted in this paper may be accounted for by the processes just adumbrated. Body habitus, lactation, menstrual function, and fertility are interlinked through endocrinological networks. Diet is implicated, especially in marginal cases. Seldom, however, are all pertinent variables available for simultaneous testing. They are available for the  $\bar{z}$ u cases to which we now return.

#### Underlying variables

Summaries of these data pertinent to the present discussion are given here. Complete data sets and analyses may be found in Wilmsen (n.d.) Components of  $\bar{z}$ u dietaries were presented in Table 2; these data were collected in 1975-1980 and have been reported in greater detail (Wilmsen 1982). Lee's (1968) data suggest that the values given for 1975-76 bush diets are good indicators of the 1963-73 condition. The important things to notice are the differences in absolute amounts of foods and in dietary components for the given periods. Traditional bush diets provide about 2100 kcal/person/day, 85% of which is derived from vegetable foods. Cattlepost diets provide about 2300 kcal/person/day, of which only 65% is supplied by vegetables. The difference in the latter diet is accounted for by domestic milk and maize meal. In 1979-80, these domestic products accounted for 81% of all diets. Seasonal variation in consumption characterized the earlier periods but was truncated in 1979 by government intervention. Table 6 displays the differences in weights associated with these diets; seasonal variation of about 6% is apparent. What is not apparent is that bush derived diets lead to about 10% lower body weight than do cattlepost diets ( $\bar{X}$ =38 kg; 0.25 kg/cm as opposed to  $\bar{X}$ =42 kg; 0.29 kg/cm). These data are in good agreement with those of Truswell and Hansen (1976:171-174), of Lee (1979:304), and of Howell (1979:194-199).

Associated cholesterol levels stratified according to diet are displayed in Table 7. Differences between cattlepost (75-76) and supplemented žu (79-80) are not significant but those between these strata and bush žu are ( $p < 0.05$ , all periods). A 40% rise in cholesterol is apparent with change in diet. Hormonal levels follow a similar pattern (Table 8). Unsupplemented žu women display depressed testosterone and estradiol levels which rise by 182% and 24%, respectively, with improved diet and are now (1979-80) at levels found among normal women in many parts of the world. Konner and Worthman (1980) report similar estradiol levels in their study. None of the women represented in Table 8 were pregnant or lactating; their prolactin levels fall within normal laboratory range. It is worth noting that Dale et al. (1979) report comparable data in their study of women distance runners whose reproductive diaries are similar to those of the unsupplemented žu women considered in this paper.

As dietaries and weights of žu women displayed marked seasonal variation in the past, the birth histories of these women were examined for associated seasonality. As is apparent in Figure 4, women on traditional bush diets experienced marked seasonality of births with 52% of all births occurring nine months after their diet induced peak weights were attained in June - August. Supplemented women, both those who consume cattlepost diets and those (all women in 79-80) who received government food supplements, experience no such marked seasonality.

### Discussion

By any measure, traditional bush diet consuming žu women display reduced fertility performance: total fertility, age specific fertility, and seasonal fertility are all suppressed. Supplemented women display no such pattern; on the contrary, their fertility follows a pattern familiar in other parts of the

world where national-state interventive measures are applied. Each of the models examined may account for part of this transformation but each is limited in its power. Lactation, alone, cannot account for the very long birth intervals recorded among  $\check{Z}$ u/ $\check{O}$ asi before 1963. Body weight does not explain the moderate intervals observed between 1963 and 1973. The intermediate variable model cannot account for the transition because it assumes constant biological substrates. The first two models suffer from a univariate focus, and, although they are often employed to assess fertility, the lactation model actually addresses fecundability and the weight model addresses fecundity.

Bongaarts' model has the merit of incorporating a number of variables and of specifying fecundability and fecundity functions of fertility. But it is a static model which sees fertility as an end product of highly constrained and redundant -- rather than interactive -- variables. When both the biology and the sociology of reproduction fall within a certain (modern) range, a static model may have predictive as well as heuristic value. The model works, that is, when the biological components of fecundability and fecundity are approximately constant and the parameters of reproductive physiology have relatively high values. In such cases, the purely sociological variables (marital restriction of mating, contraception, prescriptive lactation) may be manipulated to achieve some specific fertility rate. But in those cases where the biological factors are themselves altered by exogenous agencies, the interactive nature of the underlying variables must become the focus of investigation.

Such an interactive model must be grounded in reproductive ecology. The estradiol suppressing activity of prolactin on the ovaries has been strongly supported by both laboratory and field studies; this is a lactation induced phenomenon that inhibits ovulation. This activity, however, may be itself

inhibited by peripheral aromatization of estradiol in the presence of sufficiently high concentrations of circulating  $C_{19}$  precursors, thus permitting earlier resumption of full hypothalamic function and return to ovulatory status. On the other hand, prolactin blocking of estradiol synthesis may be enhanced in women of poor body habitus. These are phenomena of body composition. Adiposity, per se, need not be the controlling variable. The cholesterol fraction of peripheral body fat may be more important; this  $C_{27}$  steroid is the substrate from which the  $C_{19}$  steroids androstenedione and testosterone are metabolized, and these in turn are the immediate precursors of estrogens. Lipolysis is in part diet and nutrition dependent. These interactive reactions are not likely to have marked consequences in most situations that may be observed today, but in marginally nourished populations the effects may fluctuate about permissive levels at one or more of the interactive sites. In populations like presupplemented  $\bar{Y}$ u and in groups that purposefully alter body habitus (runners, ballet dancers) this could alter reproductive performance by affecting fecundity.

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Table 1. Model estimates of TFR compared with model estimates of TM.

	TFR	MTR	TM	MTM	C <sub>m</sub>	C <sub>c</sub>	C <sub>a</sub>	C <sub>i</sub>
Bangladesh	6.34	6.73	7.43	7.89	.853	.928	1.0	.556
Colombia	4.24	4.47	7.54	7.94	.562	.613	1.0	.847
Guatemala	7.01	6.54	9.74	9.09	.720	.971	1.0	.612
Indonesia	4.69	4.95	6.64	7.01	.706	.751	1.0	.610
Jordan	6.41	6.48	8.60	8.70	.745	.779	1.0	.730
Korea	4.16	3.90	7.10	6.65	.586	.780	.848	.658
Panama	4.29	4.02	6.80	6.37	.631	.483	1.0	.862
Peru	5.11	4.75	8.91	8.29	.573	.734	1.0	.738
South Lebanon	4.68	4.65	8.18	8.13	.572	.680	1.0	.781
Sri Lanka	3.53	3.35	6.88	6.53	.513	.694	1.0	.615
Syria	7.00	6.44	9.59	8.81	.730	.789	1.0	.730
Turkey	5.60	6.01	7.18	7.71	.780	.690	1.0	.730
Denmark	1.78	2.09	3.21	3.77	.555	.282	.940	.930*
Finland	1.61	1.17	3.13	2.28	.514	.180	.889	.930*
France	2.21	2.40	4.26	4.62	.519	.334	.973	.930*
Hungary	1.80	1.70	2.92	2.75	.617	.340	.568	.930*
Poland	2.09	2.34	4.78	5.35	.438	.424	.886	.930*
UK	2.38	2.35	3.91	3.86	.609	.274	.989	.930*
US	2.34	2.74	3.71	4.34	.631	.305	.999	.930*
Yugoslavia	2.11	2.32	3.69	4.06	.571	.378	.754	.930*
Bavaria	4.45*	4.89	11.89	13.10	.374	1.0	1.0	.856
Crulai	5.60	5.83	9.89	10.30	.566	1.0	1.0	.673
Grafenhausen	4.74*	4.54	10.73	10.27	.442	1.0	1.0	.671
Hutterites	9.50	9.15	12.96	12.48	.773	1.0	1.0	.816
Ile de France	6.10	5.50	12.08	10.89	.505	1.0	1.0	.712
Oschelbron	5.06*	5.31	10.60	11.12	.477	1.0	1.0	.727
Quebec	8.00	7.80	12.72	12.39	.629	1.0	1.0	.810
T. au Perche	6.00	6.77	10.15	11.46	.591	1.0	1.0	.749
Waldeck	4.41*	4.57	9.97	20.34	.442	1.0	1.0	.676
Werdum	3.78*	3.95	9.37	9.79	.403	1.0	1.0	.640

TFR = total fertility rate

MTR = model estimate of TFR

TM = total marital fertility rate

MTM = model estimate of TM

C<sub>m</sub>, C<sub>c</sub>, C<sub>a</sub>, C<sub>i</sub> = indices as in text

\*indicates that rate/index is itself an estimate made by Bongaarts

All entries from Bongaarts 1980b Tables 2 and 3 except TM calculated by Wilmsen

MTR estimates TFR with overall deviations as follows: developing countries, 1%; developed countries, 5%; historic groups, 1%.

MTM estimates TM with overall deviations as follows: developing countries, 1%; developed countries, 5%; historic groups, 2%.

Table 2. Daily per capita caloric intake.

	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec	Jan	Feb	Mar	Apr
T diet 75-76												
vege	2572	2370	2183	1845	1738	1564	1653	1146	994	918	1838	2718
meat	4	207	111	253	79	286	108	126	502	462	336	396
milk	40								165	165		
meal					29	29	29	136			136	
	2616	2577	2294	2098	1846	1879	1790	1408	1661	1545	2310	3114
D diet 75-76												
vege	1799	2183	2134	1814	1701	1601	1341	971	559	675	1450	1888
meat	1291	49	20	38	40	21	46	52	398	280	259	448
milk	495	330	330	330	165	165	165	330	660	660	660	495
meal			175	175	175	175	175	350	350	350		
	3573	2560	2659	2357	2081	1971	1727	1703	1967	1965	2369	2831
S diet 75-76												
vege		42			?			224				537
meat		232			250			275				50
milk		858			297			429				858
meal*		1251			715			1519				894
		2384			1262+			2447				2339

T diet = traditional diet

D diet = domestic foods in diet

S diet = supplemental diet

\*includes sugar

Sep 79 vege mainly Acacia and Terminalia gums for which caloric value not available.

Table 3. Age specific fertility rates of  $\bar{z}$  women in 1963-1973.

Age group	f(a)	m(a)	f(a)/m(a)	M(a)	f(a)/M(a)
15-19	.063	.56	.113	.56	.113
20-24	.208	.90	.231	1.00	.208
25-29	.238	1.00	.238	1.00	.238
30-34	.183	.94	.195	1.00	.183
35-39	.107	.79	.135	1.00	.107
40-44	.043	1.00	.043	1.00	.043
45-49	.013	.93	.014	.93	.013
	.855		.969		.905

f(a) = age specific fertility rate

m(a) = proportion of women currently married

M(a) = as m(a) with all cohorts except youngest and oldest set at 1.00

Entries for f(a) from Howell 1979:139 (Table 7.1),  
and for m(a) from Howell 1979:234 (Table 12.1).

Table 4. Log of reproductive events at /ai/ai, 1976-1980.

ID	age	76	status	1976	1977	1978	1979	1980	parity	notes	BI
283	14							1	1	1	
292	15						1		1	1	
724	15		U					1	1	1	
523	16					1	1		2	2	21
124	17				1		1		2	2	20
414	17		S			1		1	2	2	19
039	18						1		1	1	A in 76
114	20		D		1			1	2	2	77 died in 78
442	20					A			0	1	
252	21					1	1		2	4	18
992	21				1			P	1	1	
322	22				1	L-----			1	3	
652	22				1		1		2	3	30
243	23					1		1	2	4	19
642	23			1				P	1	3	
132	24		U					1	1	1	
635	24							1	1	1	A in 75
532	25			1				1	2	2	46
582	26					1		1	2	4	27
723	26				1		1		2	2	21
462	27				1			P	1	4	
832	27			1		1		1	3	3	24-18
432	30			1			1		2	5	40
472	30				1				1	5	
672	30			1		1			2	5	76 died in 76
222	32						1		1	7	
953	33			1				P	1	6	
922	34			1			1		2	6	79 died in 79
542	35					1	L-----		1	5	
822	35				1			1	2	6	31
552	36				1			A	1	5	
902	39			1			CL-----		1	6	
(N = 32)				8	10	8	10	11	47		X = 27.2

A = aborted  
S = stillbirth  
P = pregnant  
C = contracepting  
L = left area  
BI = birth interval  
U = unmarried  
D = divorced

Table 5. Number of births per year and age specific fertility rates of  $\bar{z}_u$  women in 1976-1980.

	1976	1977	1978	1979	1980		m(a)	f(a)/m(a)	f(a)/M(a)
15-19 n	0	1	2	2	2	7			
W	6	8	8	9	8	39			
f(a)	.000	.125	.250	.222	.250	.169	.500	.338	.338
20-24 n	0	4	1	3	2	10			
W	11	10	6	5	6	38			
f(a)	.000	.400	.167	.600	.333	.300	.818	.367	.300
25-29 n	3	2	3	2	4	14			
W	7	8	11	9	8	43			
f(a)	.429	.250	.273	.222	.500	.336	.910	.369	.336
30-34 n	4	1	1	1	2	9			
W	6	4	7	8	6	31			
f(a)	.667	.250	.143	.125	.233	.290	1.00	.290	.290
35-39 n	1	2	1	2	1	7			
W	5	6	5	4	5	25			
f(a)	.200	.333	.200	.500	.200	.280	1.00	.280	.280
40-44 n	0	0	0	0	0	0			
W	3	4	1	2	5	15			
f(a)	.000	.000	.000	.000	.000	.000	1.00	.000	.000
45-49 n	0	0	0	0	0	0			
W	4	1	4	4	4	17			
f(a)	.000	.000	.000	.000	.000	.000	.941	.000	.000
n	8	10	8	10	11	47			
Women	42	41	42	41	42	208			
f(a)x5	6.49	6.79	5.42	7.03	8.08	6.88		1.644	1.544
n/W	.190	.244	.190	.244	.262	.226			

f(a) = age specific fertility rate

m(a) = proportion of women currently married

M(a) = as in Table 3

n = number of births

W = number of women



Table 6. Mean weights of adult women (kg).

	Jun	Sep	Dec	Apr
1975-76	40.0	38.6	38.1	39.1
1979-80	41.0	40.3	41.8	42.4

Table 7. Mean serum cholesterol values (mg/100 ml).

	Jun	Sep	Dec	
T diet	127	138	133	N = 22
D diet	200	225	181	N = 14
S diet	183	-	165	N = 38

T diet = traditional diet in 1975-76

D diet = domesticated foods in diet in 1975-76

S diet = supplemental diet in 1979-80

Table 8. Mean endocrine values of adult women (mg/100 ml).

	T	E	
1976	11.3	111.4	N = 17
1979	31.9	137.7	N = 60
% change	182	24	

T = testosterone

E = estradiol

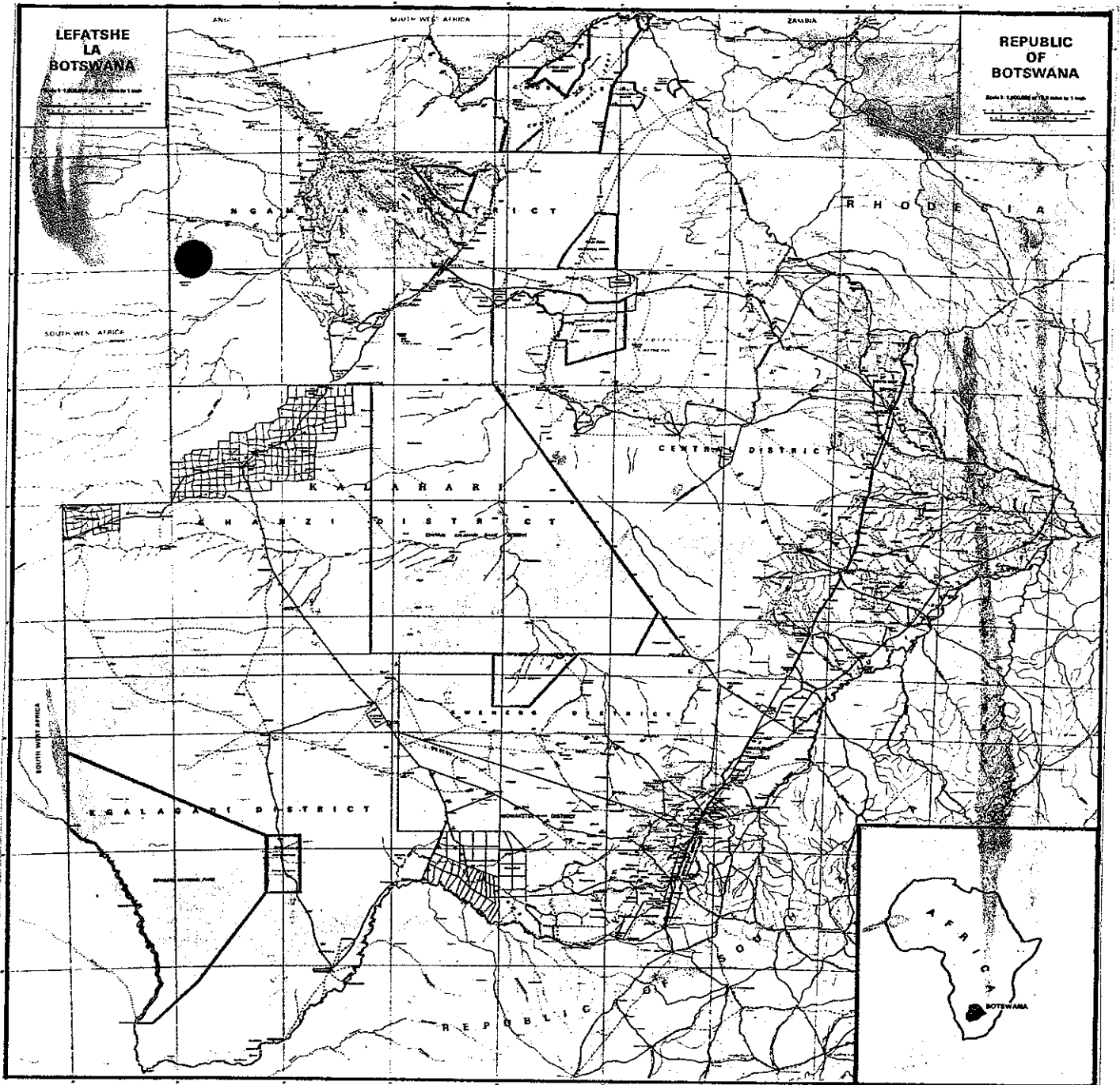


Figure 1. Map of Botswana, showing location of Žu/ǀasi study.

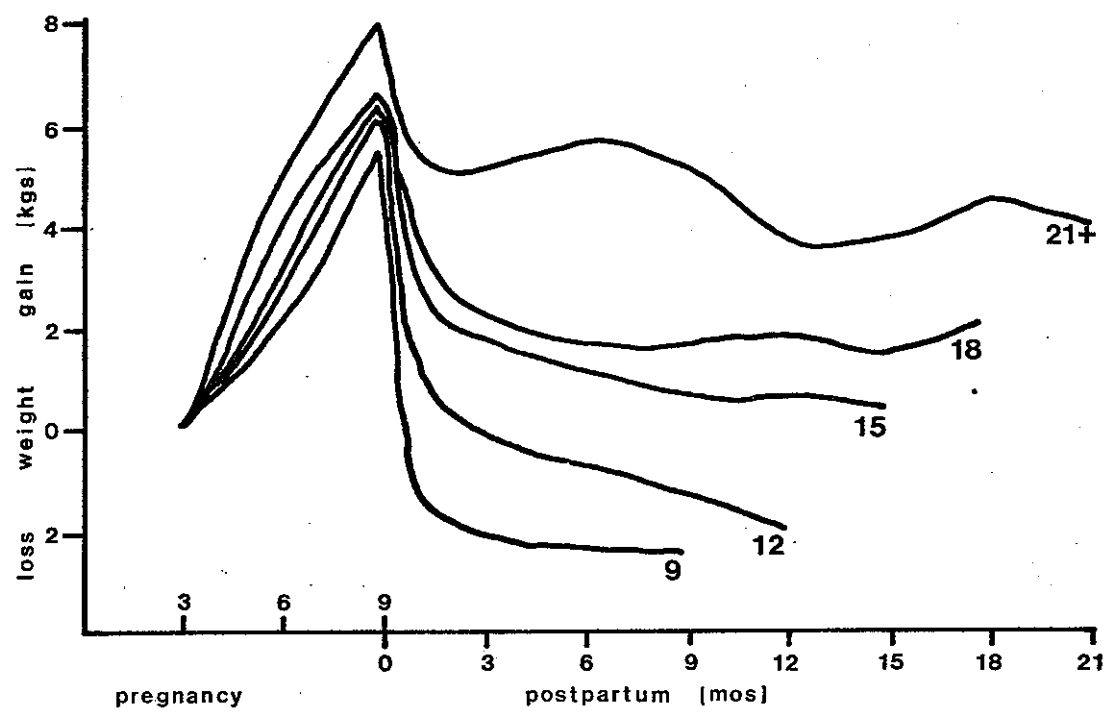


Figure 2. Duration of lactation plotted on weight gain or loss of mothers during pregnancy and lactation.

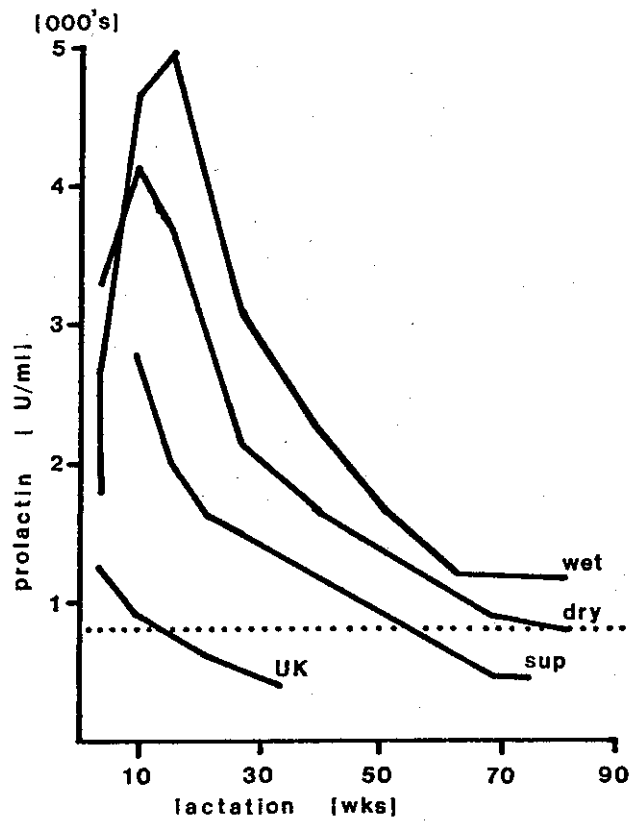


Figure 3. Mean plasma prolactin concentrations in lactating mothers in the United Kingdom and the Gambia. Wet = wet season births (N=128) to Gambian women; dry = dry season births (N=109) to Gambian women; UK = births to Cambridge, England women (N=37).

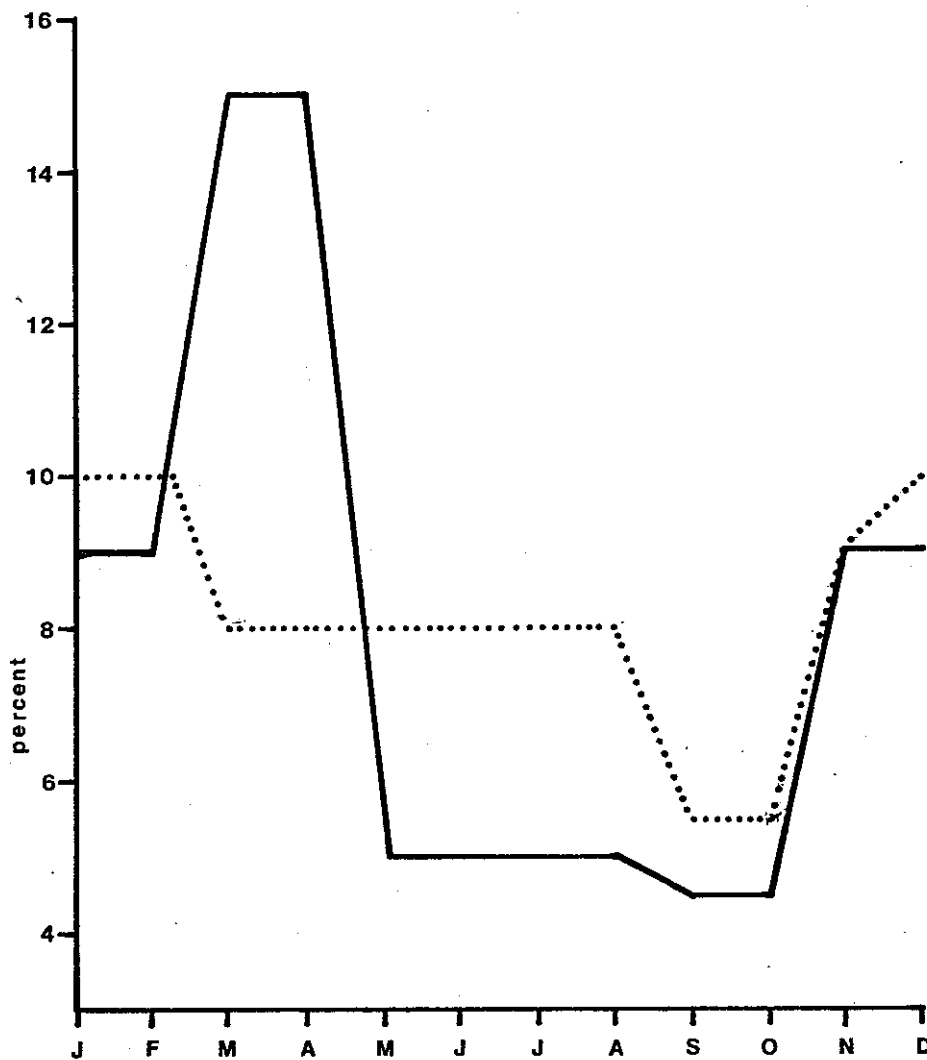


Figure 4. Births to zu/asi women in each month of the year. Solid line = traditional diet (N=127); dotted line = domestic and supplemented diets (N=140).