tary carpels per flower occur in other Winteraceae (*3, 7*) including several species of *Bubbia* (*5*).

Leroy illustrated what is labeled as a dorsal view of the gynoecium [figure 1B of (*1*)]. In the absence of developmental studies it would seem impossible to determine the morphologically dorsal side of such a structure in the center of the flower. If the gynoecium is in fact unicarpellate, then by analogy with other Winteraceae, the figure would not illustrate a dorsal view but rather one in which the dorsal and ventral surfaces are at the left and right sides of the illustration (*7*). Additional evidence is clearly needed before the gynoecium of *B. perrieri* can convincingly be termed compound. Thus, in the absence of illustrations showing cellular details in transversely sectioned material it would seem premature to consider two vertical grooves on diametrically opposed sides of the gynoecium as representing sutures. Evidence on gynoecial vascularization also is essential; cleared gynoecia with stained vascular bundles should unequivocally show the number of carpels, particularly as the results could be compared with similar preparations of other Winteraceae illustrated by Bailey and Nast (*5*). In view, then, of insufficient evidence at present for a compound ovary in *B. perrieri*, we suggest that this taxon should not currently be proposed as the basis for a new subfamily in the Winteraceae. Nor do we consider that the apparent differences Leroy cited in inflorescence structure between *B. perrieri* and other Winteraceae support such a concept. For example, although Leroy (*1*) noted that a special characteristic of *B. perrieri* was that, in the reproductive shoot, the bract function is filled by specialized scales rather than by ordinary leaves, bract scales have been recorded in other Winteraceae (*2, 4, 8*).

Of necessity, Leroy (*1*) was only able to examine floral material from herbarium specimens of two parts of a flowering plant. We suggest that if, in fact, the material he examined is bicarpellary, had further material been available for examination, unicarpellate gynoecia might have been discovered. This suggestion is based on the observation (*7*) that, although most flowers of the reduced montane winteraceous species *Pseudowintera traversii* are unicarpellate, 15 percent have two carpels that, in material examined, are congenitally fused at their bases. In this plant, some immature fused carpels were found in which, at maturity, a unilocular syncarpous gynoecium would develop (*7*). Bicarpellate gynoecia in *P. traversii* have their carpels fused in a manner quite different from that proposed by Leroy for *B. perrieri*. In view of the significance of this plant, clarification of its gynoecial structure is essential.

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It is interesting that Tucker and Sampson did not reject the possibility of a unilocular bicarpellate gynoecium in the Winteraceae and point to their own observation of an incipient stage of it in *Pseudowintera*. What they found incipient and sporadic may well be the general condition elsewhere.

Bubbia species with short stigmatic regions have a continuous O-shaped placenta below their stigmas, whereas "*B*" *perrieri* has two separate placentas. Its stigmatic lobes are far from reminiscent of anything known to me in the Winteraceae. A detailed description of them has appeared elsewhere (*1*). What is surprising in this plant is not that it would have only one carpel, according to the Capuron's description, but that this carpel is erected and capped with two large stigmatic lobes opposite one another. In the opinion of Tucker and Sampson, it would seem premature to consider the two vertical grooves as representing sutures. Why then, do they give no explanation of these remarkable grooves, both of which occupy the place of ordinary sutures and are completely unknown in other Winteraceae. Tucker and Sampson argue that my figure 1B (*2*) perhaps does not show the dorsal side since no developmental study was made to ascertain the gynoecium interpretation. But if the two carpels are supposed to be centrally united, which is my hypothesis, where are the dorsal sides, if not in the plane perpendicular to the junction plan?

Even if one disregards this hypothesis, the enigmatic genus is worth considering as a subfamily of its own. Under my hypothesis, the Madagascan plant is no longer an enigmatic one. Moreover, contrary to Tucker and Sampson, the particular character of the inflorescence is not so much its specialized scales as its large development and its gradual formation from the vegetative zone. For all of these reasons, I feel I must hold to my first view. It is of course entirely possible that unicarpellate gynoecia are also found in "*B*" *perrieri*, as hypothesized by Tucker and Sampson. The question is then, How many stigmatic lobes would they have? The scantiness of the available material is of course deplored, and attempts are being made to have fresh material gathered in Madagascar for in-depth studies.

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Difference Between Postpartum and Nutritional Amenorrhea

By the fact that it follows a pregnancy, postpartum amenorrhea reported by S. L. Huffman *et al.* (*1*) differs fundamentally from the secondary amenorrhea due to undernutrition that we described (*2*) in which weight loss below a threshold weight for height results in amenorrhea and failure to ovulate. Our weight and height data were obtained from anorectic and nonanorectic young women at the time of loss of normal cycling due to undernutrition and at the time of restoration of cycles after increased food intake. None of our subjects had ever conceived.

This confusion between nutritional amenorrhea and postpartum amenorrhea and the physical changes associated with each type would be clarified by longitudinal data on weight changes for individual women from parturition throughout the course of lactation (if present) to resumption of regular ovulatory cycles. These data are needed for each racial or ethnic group.

A woman who has become pregnant

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must have ovulated: by definition, therefore, her weight must have been *above* the minimum weight for height necessary for ovulation. British data show that during pregnancy, women store additional fat (*3*). Thus, at the time of parturition these women would have a relatively high fat/lean ratio; if a woman did not nurse, or if her infant died soon after birth, she might be expected to conceive again in a short time. In Bangladesh, Mosley (*4*) has observed that the interval to conception is very short if an infant dies soon after birth. In the absence of hard data, the most reasonable assumption is that the majority of lactating women have weights for a particular height above the minimal weight necessary for ovulation at least during the early part of the lactational period. Therefore, lactating women would not necessarily have to *gain* weight before resuming regular cycles. Only those women who had been marginal in their nutrition before and during pregnancy, and then also inadequately nourished while lactating, would be expected to fall below the minimal weight for cycling and have to gain weight before resumption of menses.

Huffman *et al*. unfortunately have no weight for height data, or even weight data for postpartum Bangladesh women at the time of resumption of menses. Futhermore, there are no data telling *how long* women who are menstruating 13 to 21 months postpartum *have been* menstruating within each classification: 1 month? 6 months? 12 months? The weight data of these women are uninterpretable without this information and without associated height for each woman. Age of the mother, parity, feeding of supplementary foods, or not, to the infant, all are important factors which also must be considered, in addition to the nutrition and work output of the mother, to clarify the complex hormone interactions affecting the duration of postpartum amenorrhea.

Other workers (*5*) differ from Huffman *et al*. in finding an effect of the nutrition of the mother on the length of time of postpartum amenorrhea. What weights are involved for a particular height at the resumption of menses for lactating women of differing ages and parities are not known for any ethnic group, so far as we know.

Women in Bangladesh at the completion of their growth are about 10 to 12 kg lighter in weight, and shorter (no exact data available), than the North American women on whom the Frisch-McArthur minimal weight standards for nutritional amenorrhea are based (*2*). It is not known whether an equation derived from actual measurements of body water of North American girls and young women (*6*) can be used to estimate body water of Bangladesh women. Therefore, it is especially important to have direct measurements of height, weight, and body composition of Bangladesh women at the time of completion of growth. Meaningful comparisons of Bangladesh women then could be made with other groups, both postpartum and during the periods when food supplies are so low that nonpregnant women become amenorrheic (*7*).

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We are pleased that Frisch and McArthur clearly acknowledge the difference between postpartum amenorrhea and amenorrhea induced by acute malnutrition. It is well established that starvation leads to an amenorrheic state, and it was not the purpose of our study (*1*) to determine the minimal nutritional status necessary for menstrual cycles. Rather, we were testing a theory that prolonged postpartum amenorrhea is a function of chronic malnutrition. As stated by Frisch and McArthur (*2*), "If a minimum of stored fat is necessary for normal menstrual function, one would expect that . . . poorly nourished lactating women would not resume menstrual cycles as early after parturition as well-nourished women." In a more recent article (*3*), Frisch states again that "lactational amenorrhea is longer after parturition" in an undernourished than in a well-nourished woman. We believe our data indicate that these conclusions are misleading and that malnutrition has only a trivial effect on the duration of postpartum amenorrhea.

In their comments above, Frisch and McArthur restate shortcomings of our data set that we acknowledged (*1*), caused by our lack of longitudinal data. We did not know the weights of our subjects at resumption of menses. But one would expect that, if nutritional status influenced the duration of postpartum amenorrhea, menstruating women would have significantly different weight-for-height distributions from those of amenorrheic women at the same interval postpartum, and we showed that such was not the case. In response to the comment on the lack of data on weight for particular heights, Table 1 is here provided. Again, it is evident that there was only a very small difference between the mean weights of menstruating and amenorrheic women of similar height.

The suggestion that at parturition women have relatively high fat-to-lean ratios is based on British data. More relevant to our study is a study of 82 women in India (*4*) that showed the difference between weight before pregnancy and weight immediately after delivery to be less than 1 kg. Their mean weight before pregnancy was 41.91 kg and immediately after delivery was 42.59 kg. Ebrahim (*4*) states that in countries existing on marginal diets, body stores of nutrients are low and the average gain in pregnancy is 5 to 6 kg, with no stores laid down during pregnancy. It is therefore doubtful that the fat stores at the termination of pregnancy are significantly higher than at conception. Women whose infants die soon after birth have relatively short intervals to conception because there is no suckling stimulus to maintain the hormonal profile preventing ovulation.

As to the comment that "other workers differ [from us] in finding an effect of the nutrition of the mother on the length of time of postpartum amenorrhea," our reading of the cited studies is somewhat different. Knodel's article (*5*) is a review of research on breast-feeding and fertility relationships. He concludes that "the differences [in duration of postpartum amenorrhea] between nutritional groups within the same population do not appear to be large. Variations in breast-feeding practices seem to be a more important determinant." This conclusion hardly supports Frisch's position. Potter (*6*) presents calculations based on a mathematical model. He uses previously published data in a Perrin-Sheps model to quantify the increases in natural fertility expected in changes from prolonged breast-feeding to bottle-feeding. In his calculations the mean duration of postpartum amenorrhea is 6, 10, or 17 months, figures based on studies in Chile, India, and Bangladesh. He speculates that such large differences may be related to nutrition and health, but clearly states that he is not testing a theory but offering a hypothesis.

Delgado *et al.* and Carael do present data that can be used to test the proposed hypotheses, but a critical analysis of these studies raises certain methodological questions. Delgado and co-workers in Guatemala (*7*), examined the effect of dietary supplementation during pregnancy on the length of postpartum amenorrhea. Although dietary differences during pregnancy may result in variations in nutritional status postpartum, such an association is only hypothesized, not demonstrated. More to the point is that the mean duration of postpartum amenorrhea among those with "high" total caloric intakes was only 1 month shorter than those with "low" intakes, a difference of small consequence for fertility. In a separate analysis of this study, women whose average weight varied by more than 10 kg were found to vary in duration of amenorrhea by only 1.6 months (*8*). Carael (*9*) describes differences in mean weights of menstruating and amenorrheic women 0 to 24 months postpartum in a rural area of Zaire. This appears to be the one study offering support to Frisch and McArthur's argument. Although Carael observed rather striking differences in women of 0 to 11 months postpartum, the difference in mean weights beyond this point is very small. Since the average period of postpartum amenorrhea in this population was 18.7 months, and the proportion who resumed menstruation during the early months was small, it is probable that the menstruating women observed in early months were a highly selected group. Other factors that could affect the probability of being amenorrheic such as age, socioeconomic status, and differences in infant feeding practices in particular, were also not taken into account.

Among Hutterites, a North American religious group which does not practice contraception, average duration of postpartum amenorrhea is 6 months (*10*). Since this population is well nourished, it is often referred to as evidence for a nutritional effect on postpartum amenorrhea. However, Hutterite patterns of breast-feeding, as described by Huntington and Hostetler (*11*), differ from those observed in developing countries; semisolid food is given at an early age (by 6 weeks), pacifiers are commonly used, and mothers do not sleep with their infants. The Hutterite mothers observed generally breast-fed their infants for 4 to 5 minutes, and no mother was observed nursing for more than 10 minutes. There was thus far less suckling than among many women in developing countries, whose infants (i) generally do not receive other foods until at least 6 months of age, (ii) are much less likely to be given pacifiers and therefore suckle at the breast for reasons other than obtaining milk, and (iii) generally suckle longer at each feeding. The shorter periods of amenorrhea of the Hutterites in comparison with malnourished women in developing countries is as likely therefore to be related to differences in suckling patterns as to nutrition.

Table 1. Mean weight for height, by menstrual status, of breast-feeding Bangladeshi women 13 to 21 months postpartum.

Height (cm)	Amenorrheic: Mean weight (kg)	Amenorrheic: Standard deviation	Amenorrheic: *N*	Menstruating: Mean weight (kg)	Menstruating: Standard deviation	Menstruating: *N*	Difference in mean weights (%)
<140.0	35.73	3.40	71	35.65	3.03	32	.0
140.0–142.9	37.76	3.71	137	38.15	3.21	80	+1.0
143.0–144.9	38.72	3.67	154	38.99	2.91	81	+0.7
145.0–146.9	39.22	3.71	199	40.54	3.75	109	+3.3
147.0–149.9	40.84	3.64	279	41.38	3.59	171	+1.3
150.0–152.9	42.33	3.57	221	43.11	3.63	138	+1.8
153.0–154.9	43.91	4.02	94	44.45	3.99	67	+1.2
155.0–156.9	44.87	3.37	51	46.58	4.50	36	+3.7
>156.9	45.58	5.13	37	48.21	3.73	25	+5.5

Thus available evidence contradicts or provides only weak support for the theory that malnutrition prolongs postpartum amenorrhea. If such a relation exists, any longitudinal effect would be small. In a subsequent longitudinal phase of our own research we found no increases in maternal weight associated with the return of postpartum menses. Several longitudinal studies, including work in Indonesia and Bangladesh, await more extensive analysis.

The debate on the effect of chronic malnutrition on postpartum amenorrhea and fertility appears at times to be little more than an academic exercise. However, the issue has remained a focus of concern because of the implications for policy. The theorized role of nutrition in increasing fertility has led to proposals to reduce food assistance abroad on the grounds that it will worsen the problem of excessive population growth. Most of the evidence contradicts this theory, and its further propagation in scientific literature could be detrimental to the health of many of the poor in the developing world.

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