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Reproductive Immunosuppression and Diet

An Evolutionary Perspective on Pregnancy Sickness and Meat Consumption[1]

by Daniel M. T. Fessler

Pregnancy sickness, a suite of "symptoms" that frequently co-occur during pregnancy, may be an adaptation providing behavioral prophylaxis against infection. Maternal immunosuppression, necessary for tolerance of the fetus, results in gestational vulnerability to pathogens. Throughout the period of maximal vulnerability, dietary behavior is significantly altered via changes in nausea susceptibility and olfaction and the development of marked aversions and cravings. Of food types, meat is both the most likely to carry pathogens and the principal target of gestational aversions and pregnancy taboos. Because meat was prominent in ancestral human diets but hygienic procedures that effectively eliminate the risk of meat-borne infection are recent, such pathogens likely constituted a source of selective pressure on pregnant females throughout human history. Both the relatively low protein and energy demands of the first trimester and the existence of nonmeat alternatives would have allowed for the evolution of time-limited gestational meat-avoidance mechanisms. Complementing these mechanisms, gestational cravings target substances that may influence immune functioning and affect the availability of iron in the gastro-intestinal tract, thereby limiting the proliferation of iron-dependent pathogens. Clinical and ethnographic findings are examined in light of these proposals, and directions for future research are outlined.

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Around the world, people recognize that pregnancy is associated with nausea, vomiting, and marked changes in dietary preferences, a pattern termed pregnancy sickness (Flaxman and Sherman 2000).[2] Nausea and vomiting generally serve to protect the individual from the harmful effects of ingested toxins and toxin-producing pathogens; vomiting expels the offensive substance, while nausea, a highly aversive experience, anchors remarkable one-trial learning, motivating future avoidance of the substance (Bernstein 1999). The complex and coordinated nature of these features, the specificity of their eliciting conditions, and the obvious benefits that they provide all suggest that normal nausea and vomiting are part of an adaptation, a trait that has evolved through natural selection because of the advantages that it furnishes to its possessors. However, in contrast to the usual circumstances associated with nausea and vomiting, many pregnant women experience these symptoms after ingesting foods which others around them eat without ill effect—indeed, it seems that pregnant women often become nauseated *before* ingestion has occurred and, most notably, experience nausea and vomiting at far higher frequencies than do other members of their communities. The striking differences between the circumstances of normal nausea and vomiting and those of pregnancy (or "morning") sickness call for explanation.

A large body of research addresses possible physiological mechanisms underlying pregnancy sickness. Recent

2. The Bengkulu of southwestern Sumatra term pregnancy "the good sickness," a reference to the nausea, vomiting, and strange aversions and cravings which appear early in gestation. For the !Kung San of the Kalahari desert, these are diagnostic criteria for pregnancy (Shostak 1981). Soranus of Ephesus, a 2d-century Greek gynecologist, described the same pattern in association with early pregnancy (Temkin 1991), and the Maasai (Spencer 1988) and Tiv (Bohannan and Bohannan 1969) do likewise. Cravings, nausea, and vomiting are hallmarks of early pregnancy among the Hokkien Taiwanese (Barnett 1971) and Tongans (Ostraff et al. 2000, Morton n.d.); Culpepper's 1651 *Directory for Midwives* notes the same (Fairweather 1968), and these symptoms also occur among the Saraguro (Finerman 1985) and Hawaiians (Green and Beckwith 1924). Unique gestational cravings are recognized by the Jivaro (Harner 1972) and the Tahitians (Levy 1973). Present-day midwives from such diverse countries as Sierra Leone, Chile, the Netherlands, Pakistan, Vanuatu, Iran, Jamaica, and Nicaragua agree that nausea and vomiting occur in early pregnancy (O'Brien, Relyea, and Lidstone 1997), and anthropologists report the same for highland Ecuador (Weigel et al. 2000) and Truk (Fischer 1998 [1963]). Clinical surveys report nausea and vomiting in association with pregnancy in 75 groups in Western Europe, North America, Japan, India, Kenya, Nigeria, Sri Lanka, and Hong Kong (Flaxman and Sherman 2000), and ethnographers record the same among indigenous groups in North and South America, the Caribbean, Oceania, Australia, Asia, the Middle East, and Africa (reviewed in Weigel 1985 and Flaxman and Sherman 2000).

investigations focus on specific isoforms of human chorionic gonadotrophin (hCG) (cf. Jordan et al. 1999; also O'Connor et al. 1998), with attention being paid to the influence of hCG on thyroid functioning (Tareen et al. 1995). While some investigators have employed evidence regarding proximate mechanisms to argue that pregnancy sickness is merely a nonfunctional or even dysfunctional by-product of hormonal changes accompanying pregnancy (see Weigel 1985 for review and Profet 1992 and Flaxman and Sherman 2000 for additional discussion), others have postulated that pregnancy sickness, like normal nausea and vomiting, is a functional adaptation. Specifically, the greater sensitivity of the mother has been explained as a means of compensating for the special vulnerability of the embryo or fetus (hereafter generically termed the "developing organism").

To date, adaptationist explanations have taken a number of forms. Hook (1976, 1978, 1980) and Tierson, Olsen, and Hook (1985) initially proposed that pregnancy sickness protects the developing organism from a heterogeneous collection of toxins such as those thought to occur in coffee. Weigel (1985) generalized this list to include a range of teratogens and pathogens. Noting that pregnancy sickness is concentrated in the first trimester, when the developing organism is most vulnerable to insult, Profet (1988, 1992) expanded the emphasis on teratogens, arguing that pregnancy sickness principally protects the developing organism from secondary compounds produced by plants as defensive agents. Profet also noted that cooked meat, which contains carcinogens, may be teratogenic and therefore could constitute a secondary target of prophylactic avoidance.

If pregnancy sickness is an adaptation, natural selection should have imbued it with discriminatory specificity; in order to preclude the costly avoidance of beneficial substances, only those foods that posed a significant and recurrent threat should have acquired nauseogenic salience during pregnancy. To test adaptationist explanations, I therefore began compiling studies of gestational food aversions. It soon became apparent that, in general, contrary to Profet's central thesis, plant foods were not a central target of such aversions. Rather, in most studies, the principal targets were meat and other animal products. Troubled by an absence of evidence in support of Profet's secondary assertion that cooked meat may be teratogenic and recalling Weigel's observation regarding the hazards of infection during pregnancy (and Profet's own brief reference to the risks posed by spoiled meat [1992:334]), I noted that meat is a common source of infection and pregnancy sickness coincides with a period of marked maternal immunovulnerability, a time of danger for both the developing organism and the mother. While developing the ensuing hypothesis that a principal function of pregnancy sickness is the provision of prophylactic protection from meat-borne pathogens during reproductive immunosuppression, I learned from Paul Sherman that he and Samuel Flaxman had just completed a comprehensive meta-analysis of studies of pregnancy sickness and had reached the same conclusion. Flaxman and Sherman's subsequent publication (2000) also documents the incidence of pregnancy sickness in a large number of societies and summarizes patterns of gestational cravings.

This paper builds on and complements Flaxman and Sherman's important work in a number of ways. First, seeking additional evidence of the unique salience of meat during pregnancy, I survey the targets of pregnancy taboos in 73 cultures. Second, because adaptations are thought to arise in response to highly specific selection pressures, I document that (*a*) the timing of nausea, vomiting, and aversions coincides with the period of maximal immunovulnerability and (*b*) meat-borne pathogens possess attributes that make them particularly dangerous during precisely this period. Third, because adaptations arise only in response to recurrent selection pressures, I present evidence that meat-borne pathogens have probably constituted a source of selective pressure throughout much of human history. Fourth, because intrinsic trade-offs are thought to constrain the efficiency with which any single adaptive goal is pursued, I demonstrate that the benefits of avoiding meat during early pregnancy are likely to have generally exceeded the costs. Fifth, because adaptations are best identified on the basis of the complex nature of their designs, extending suggestions made by Profet and others I introduce evidence for the existence of an integrated suite of sensory and ingestive changes in early pregnancy. I then explore the possible utility of gestational cravings, arguing that they often target substances that affect the availability of iron in the gastrointestinal tract, thereby limiting the proliferation of iron-dependent pathogens. Finally, I address problems with the meat-avoidance hypothesis, compare it with other explanations of pregnancy sickness and related phenomena, and present testable predictions derived from it.

Meat Avoidance during Pregnancy

Building on the work of Hook (1976, 1978, 1980) and Tierson et al. (1985), Profet (1988, 1992) has argued that pregnancy sickness involves the patterned development of a large number of marked aversions to previously favored foods. The acquisition of aversions is closely tied to the experience of nausea, whether as a result of or in anticipation of ingestion (reviewed in Flaxman and Sherman 2000; also Dye, Jones, and Hill 1998). Increased nausea and vomiting are the hallmark of pregnancy sickness, and, correspondingly, Flaxman and Sherman's meta-analysis reveals a substantial increase in the number of aversions among pregnant versus nonpregnant women (2000:129). However, while the association of gestational aversions with nausea fits the general pattern, gestational aversions exhibit unique properties: In Western subjects, whereas food aversions acquired in response to nausea and vomiting generally persist for years or decades (Mattes 1991), nearly all gestational aversions disappear with parturition (Worthington-Roberts et al. 1989, Pope, Skinner, and Carruth 1992). The combination of the greatly increased occurrence of aversions dur-

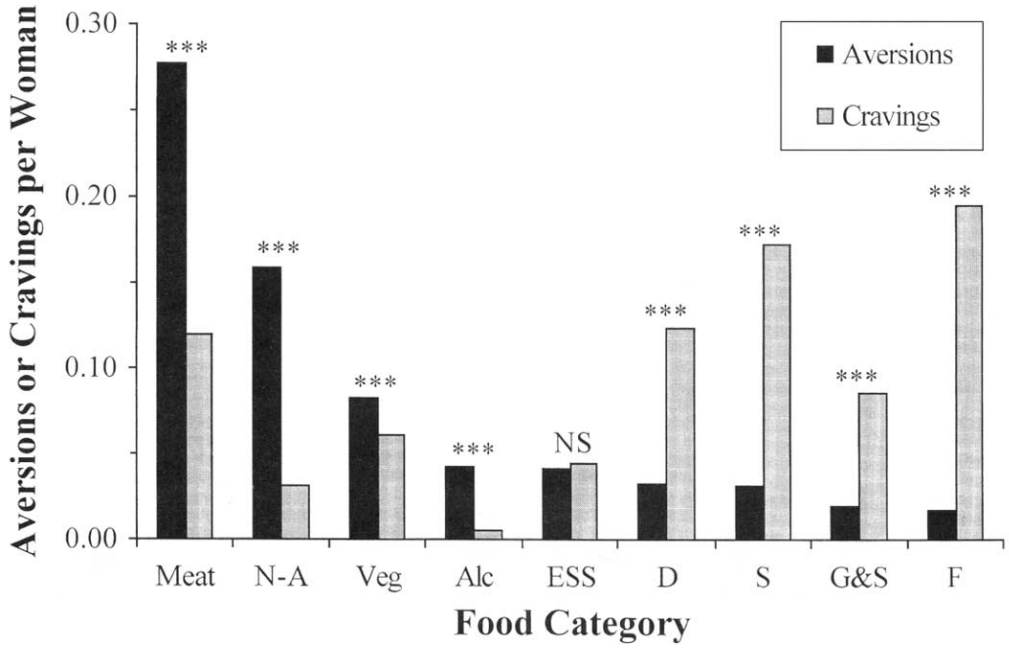

FIG. 1. *Average number of food aversions and cravings of pregnant women, all trimesters (Flaxman and Sherman 2000: fig. 7). Aversions based on 20 studies including 5,432 women and cravings on 21 studies including 6,239 women (see table 1).* ***, *P < 0.001;* NS, *no significant difference.* Meat, *meats, fish, poultry, and eggs;* N-A, *nonalcoholic beverages;* Veg, *vegetables;* Alc, *alcoholic beverages;* ESS, *ethnic, strong and spicy foods;* D, *dairy products and ice cream;* S, *sweets, desserts, and chocolate;* G&S, *grains and starches;* F, *fruits and fruit juices.*

ing pregnancy and their uniquely time-limited nature supports Profet's and others' assertions that pregnancy exercises a distinctive influence on food avoidance. In order to determine whether gestational aversions are the product of an evolved adaptation and, if so, what its function is, we must therefore establish which foods are avoided during pregnancy.

Flaxman and Sherman (2000) conducted a meta-analysis of 20 studies of gestational aversions (covering 5,432 women) and 21 studies of gestational cravings (6,239 women) (see fig. 1) and found that meat is clearly the principal target of aversions (see also Knox, Kremer, and Pearce 1995, Dye, Jones, and Hill 1998). Although the studies of aversions surveyed by Flaxman and Sherman include investigations in Saudi Arabia, Jamaica, Sudan, and South Africa, 16 of the 20 employ Western subjects. In general, claims regarding species-typical adaptations produced by natural selection are best supported by data drawn from a wide range of human societies. Accordingly, it is important to examine meat eating during pregnancy in a broader range of non-Western subjects. In many traditional societies, food taboos and related beliefs are an important determinant of gestational ingestive behavior. While taboos may sometimes constitute wholly exogenous constraints, a number of observations suggest that they may in part reflect women's own experiences. Accordingly, any patterning of the categories of foods targeted by taboos is of potential interest.

A search of the Human Relations Area Files, other ethnographies, and the nutrition literature revealed 73 societies for which investigators provide information on gestational food taboos. In tabulating the findings (tables 1 and 2), I employed the following conventions: In some instances, an entire category of food was proscribed, while in other cases only specific types of food were taboo; in the latter, a given society was counted once regardless of how many items were proscribed (because categorical proscriptions cannot be quantitatively compared with specific proscriptions, the units being compared were whole societies rather than taboos). In some cases there was a lack of concordance either between two ethnographers or within one ethnography; I labeled these "type B" cases, in contrast to the simpler "type A." In type B cases, positive evidence was given priority over negative; since this was done for all food types, it did not inflate the count for any given category. Likewise, when one source indicated categorical proscriptions while another indicated specific ones, only the former was recorded. In the hopes of mitigating Galton's Problem, data are presented by geographical region showing the number of contributing societies.[3]

3. The total number of societies listed in the two tables exceeds the number of actual societies because a society appears in both tables if it has categorical proscriptions for one food type and specific proscriptions for another food type.

TABLE 1
Targets of Taboos on General Categories of Food

Region and Type of Report	Number of Societies	Target						
		Meat	Vegetables	Fruit	Dairy Products	Sweets	Spicy Foods	Starches
North Africa								
Type A[1]	1	–	–	–	–	1	–	–
Type B[2]	1	1	–	–	–	–	–	–
Sub-Saharan Africa								
Type A[3]	6	4	1	1	1	1	–	–
Type B	–	–	–	–	–	–	–	–
Europe and Euro-America								
Type A[4]	1	–	–	1	–	–	–	–
Type B	–	–	–	–	–	–	–	–
Middle East								
Type A[5]	1	–	–	–	–	–	1	–
Type B	–	–	–	–	–	–	–	–
East Asia								
Type A[6]	2	–	1	–	–	–	1	–
Type B	–	–	–	–	–	–	–	–
South Asia								
Type A[7]	1	–	–	–	–	–	1	–
Type B	–	–	–	–	–	–	–	–
Southeast Asia								
Type A[8]	1	–	–	–	–	–	1	–
Type B[9]	1	1	–	–	–	–	–	–
Australia								
Type A	–	–	–	–	–	–	–	–
Type B[10]	1	1	–	–	–	–	–	–
Oceania								
Type A[11]	1	1	–	–	–	–	–	–
Type B	–	–	–	–	–	–	–	–
Central America								
Type A	–	–	–	–	–	–	–	–
Type B[12]	1	1	–	–	–	–	1	–
South America								
Type A[13]	5	3	1	–	1	1	1	–
Type B	–	–	–	–	–	–	–	–
North America								
Type A[14]	2	1	–	–	–	–	2	–
Type B	–	–	–	–	–	–	–	–
Total	25	13	3	2	2	3	8	0

NOTE: Type A, concordance among ethnographies; B, lack of concordance among or within ethnographies.
SOURCES: 1, Calame-Griaule and Dieterlen (2000[1960]), Paulme (1940), van Beek (2000[1992]), Bohannan and Bohannan (1969); 2, Kaufman (2000[1960]), Hanks (2000[1963]); 3, Kagwa et al. (1934), Mair (1934), Gilks and Orr (1931), Merker (1910), Spencer (1998), Aunger et al. (1999), Israel and Nestor (1982*a*), Demissie, Muroki, and Kogi-Makau (1988), Ominde (1952); 4, Laguerre (1984); 5, Abdalla (1982); 6, Pritham and Sammons (1993),Liulan (1999); 7, Stevenson (1920), Briggs (1920); 8, Manderson (1981); 9, Kaufman (2000[1960]), Hanks (2000[1963]); 10, Róheim (1993); 11, Whitehead (2000); 12, Chapin (1983), Nordenskiöld (1998[1938]); 13, Karsten (1932), Peirson (1998[1967]), Elick (1969); 14, Turner (1972), Clark (1959).

Using these data, I conducted exact binomial tests of the proposition that the proportion of meat taboos (described in resampling terminology as "successes") occurring in this sample was equal to the proportion of every other type of food taboo observed. The total number of successes was calibrated to reflect the proportion of samples within each geographic region displaying the taboo (i.e., 9 out of 10 societies within the same region would return a success rate of 0.9 for that region). Type A and type B ethnographies were pooled, and the number of successes was rounded to the nearest integer for the analysis. For the general food taboos, five out of six binomial tests executed returned p values less than 0.05 (see appendix). The only other food taboo that achieved proportion levels statistically indistinguishable from meat was spicy foods (cf. Flaxman and Sherman 2000). When tested against the mean of all nonmeat taboos, the p values were significant. For the specific food taboos all seven tests returned p values less than 0.05. When the data from general and specific food taboos were pooled (taking care to ensure that only one type was counted), all p values were below 0.05. Of 20 binomial tests, 19

TABLE 2
Targets of Taboos on Specific Food Items

Region and Type of Report	Number of Societies	Target						
		Meat	Vegetables	Fruit	Dairy Products	Sweets	Spicy Foods	Starches
North Africa								
Type A[1]	1	1	–	–	–	–	–	–
Type B[2]	1	–	–	–	–	1	–	–
Sub-Saharan Africa								
Type A[3]	17	13	1	5	2	2	2	6
Type B	–	–	–	–	–	–	–	–
Europe and Euro-America								
Type A[4]	3	3	–	–	–	1	–	–
Type B	–	–	–	–	–	–	–	–
Middle East								
Type A[5]	1	–	1	1	–	–	–	–
Type B	–	–	–	–	–	–	–	–
East Asia								
Type A[6]	5	3	–	2	–	–	1	1
Type B	–	–	–	–	–	–	–	–
South Asia								
Type A[7]	5	4	1	3	1	–	–	–
Type B	–	–	–	–	–	–	–	–
Southeast Asia								
Type A[8]	5	5	1	4	–	2	2	2
Type B	–	–	–	–	–	–	–	–
Australia								
Type A	–	–	–	–	–	–	–	–
Type B[9]	1	1	–	–	–	–	–	–
Oceania								
Type A[10]	6	6	1	3	–	–	–	–
Type B[11]	1	1	–	1	–	–	–	–
Central America								
Type A[12]	1	1	1	1	–	–	–	–
Type B[13]	1	1	–	–	–	–	–	–
South America								
Type A[14]	10	9	–	3	–	1	1	–
Type B	–	–	–	–	–	–	–	–
North America								
Type A[15]	3	3	–	1	–	–	–	–
Type B[16]	1	1	–	1	–	–	–	–
Total	62	52	6	25	3	7	6	9

NOTE: Type A, concordance among ethnographies; B, lack of concordance among or within ethnographies.
SOURCES: 1, Calame-Griaule and Dieterlen (2000[1960]); 2, Kaufman (2000[1960]); 3, Brock (1953), Calonne-Beaufaict (1998[1921]), Evans-Pritchard (1998[1932, 1937]), Richards (1939), Helitzer-Allen, Kendal, and Wirima (1997), Israel and Nestor (1981*a*, *b*, 1982*b*, *c*), Costello (1986), Ominde (1952); 4, Pavlovic (1973), Sanders (1949), Fife and Fife (1966); 5, Abdalla (1982); 6, Pritham and Sammons (1993), Wheeler and Tan (1983), Burkhardt (1954), Gould-Martin (1976), Liulan (1999); 7, De Silva (1995), Kar (1993), Ferro-Luzzi (1973), Brown, Shah, and Worth (1968), Rizvi (1979), Panikkar (1918); 8, Adriani and Kruyt (1996[1951]), Man (1975), Bolton (1972), Covarrubias (1938); 9, Róheim (1993); 10, Thompson (1940), Beaglehole and Beaglehole (1938, 1941), Jelliffe and Jelliffe (1964), Blackwood (1935), Schieffelin (1990); 11, Malinowski (1929), Seligmann (1910); 12, Cosminsky (1994); 13, Chapin (1983); 14, Da Silva (1962), Chapman (1982), Jackson (1983), Reichel-Dolmatoff (1971), Becher (1960), Taylor (1950), Parsons (1945), Bianco et al. (1990), Pereira et al. (1989), Kracke (1981), Johnson (n.d.), Elick (1969); 15, Hilger (1951), Clark (1959); 16, Jones (1914), Emmons (1991).

returned p values less than 0.05, thus rejecting the null hypothesis. This analysis affirms the profound overrepresentation of meat taboos in the sampled societies.

Food taboos in pregnancy may reflect (*a*) the moralization of a prevailing pattern (i.e., pregnant woman avoid meat, and people notice this and create a rule), (*b*) an institutionalized understanding of a causal relationship (i.e., meat eating is observed to have negative consequences and so is proscribed), or (*c*) a self-serving institution created for the benefit of those in power (i.e., meat is valuable and men seek to monopolize it; pregnancy epitomizes womanhood and therefore foregrounds rules which benefit men at women's expense). In addition, meat may have special salience as a target of taboos, and therefore pregnancy taboos may reflect a larger pattern of proscriptions.

In some cases, the ethnographic record suggests processes of introspection and observation. For example, Aranda women describe numerous dreams linking pregnancy, bitter tastes, meat eating, and nausea. Some

informants report that "the woman may eat meat if she wishes to do so, but the unborn child does not like it, and may show its resentment by causing sickness"; others state outright that meat should be avoided (Róheim 1933:251). In Tamilnad, India, meat and eggs are sometimes proscribed because they induce vomiting and also because they are "hot" foods (Ferro-Luzzi 1973); "hot" foods are often taboo during the first trimester because they are thought to be abortifacient (Pool 1986). Papaya, another "hot" food, is an even more prevalent target: mango, elsewhere a common focus of cravings, is also avoided because it is "hot" (Ferro-Luzzi 1973). While the latex present in papaya skin does indeed induce uterine contractions (Cherian 2000), there are no reports of such an effect for mango. Therefore Tamilnad pregnancy beliefs suggest taboo formation based on observations of causality combined with symbolic elaboration and extension.[4] Although the level of detail presented in most ethnographies is insufficient to allow for similar analyses of other cases, these examples suggest that gestational proscriptions may be in part a function of women's experiences with meat.

In at least 10 of the 65 meat-proscribing cultures (Matsigenka, Kaluki, Orang Asli, Island Carib, Eastern Toraja, Andaman Islanders, Lau Fijians, Chippewa, Buka, and Kagwahiv), patriarchy cannot explain the taboos, since some or all meat is proscribed for both the pregnant woman and her husband. Further evidence against patriarchy as a universal explanation comes from the observation that these taboos often do not apply to pre- and postreproductive-age women (Simoons 1994).

Additional ethnological analysis (reported elsewhere [Fessler and Navarrete n.d.]) reveals that the patterned focus on meat evident in pregnancy taboos also appears in other food taboos, suggesting that similar factors underlie the formation of both pregnancy and nonpregnancy taboos. One possibility is therefore that pregnancy taboos are not informative with regard to postulated endogenous changes in women's attitudes toward meat during pregnancy. However, a second possibility is that both pregnancy and nonpregnancy taboos focus on meat because, given its unique combination of high nutritional value and high risk of pathogen ingestion, humans possess an evolved ambivalence toward it. Animal products have unparalleled salience as elicitors of disgust, and in both humans and nonhumans proteinaceous foods are the principal target of conditioned aversions. Food taboos in general may reflect such reactions. Pregnancy thus appears to entail an amplification of reactions that are present outside of pregnancy, and hence pregnancy taboos may spring in part from endogenous responses to meat.[5] In sum, while it is impossible to discern the exact causes of all gestational taboos, it is at least plausible that the prominence of meat as a target of proscriptions in part reflects women's spontaneous subjective experiences. Accordingly, a parsimonious explanation of the concordance between Flaxman and Sherman's and my results is that the spontaneous avoidance of meat during pregnancy is widespread across populations.

Timing of Pregnancy Sickness

As noted earlier, acquired food aversions are intimately linked to nausea and vomiting. Numerous investigators report that, in Western subjects, nausea and vomiting are concentrated during the first trimester (Jarnfelt-Samsioe, Samsioe, and Velinder 1983, MacIntyre 1983, Klebanoff et al. 1985, Weigel 1985, Tierson, Olsen, and Hook 1986, Vellacott, Cooke, and James 1988, Gulick, Shaw, and Allison 1989, Rodin and Radke-Sharpe 1991, Gadsby, Barnie-Adshead, and Jagger 1997; but see also Lacroix, Eason, and Melzack 2000). The same pattern is reported in widely disparate preindustrial societies. Consistent with the link between nausea, vomiting, and acquired aversions, Flaxman and Sherman (2000) demonstrate that, in a subsample of the above studies of Western subjects, pregnancy-related food aversions are significantly concentrated in the first trimester and decline thereafter (see also Knox, Kremer, and Pearce 1995).

The timing of pregnancy sickness has been singled out by some adaptationists as evidence of evolved design. To date, these arguments have focused on the delicate nature of organogenesis, a process that occurs primarily during the first trimester (Profet 1988, 1992; Flaxman and Sherman 2000). Organ construction is equivalent to shooting an arrow at a target—the consequences of a slight deflection of the trajectory will be much greater if they occur early in the arrow's flight than if they occur later. Similarly, early deviations in the organization of organogenic cells have greater consequences than later deviations. The period of organogenesis thus constitutes a window during which insult to the developing organism, be it from teratogens or from infection, is likely to have the most wide-ranging consequences (reviewed in Flaxman and Sherman 2000). Moreover, the dangers inherent in this somewhat precarious phase are greatly exacerbated by the fact that, *during precisely this period*, the mother's ability to resist infection is severely compromised.

4. A common rationale is the (probably accurate) belief that meat consumption increases birth weight, an effect which women seek to avoid (despite the costs to infant survivorship) where labor is seen as difficult and dangerous.

5. Meat is so prominent in nonpregnancy taboos that even dramatic increases in endogenous responses during pregnancy would not increase the centrality of meat in pregnancy taboos relative to other taboos. No claim is being made as to any functional benefit provided by either pregnancy or nonpregnancy taboos. On the contrary, both are best viewed as accidental consequences of the effects of individual propensities on the generation and diffusion of cultural knowledge (see Fessler and Navarrete n.d.).

Reproductive Immunosuppression

Far from being a reflection of godly perfection, human beings, like other living things, are largely a collection of compromises. Given the precarious nature of embryonic development, in an ideal world women would be most immunologically robust during early pregnancy. However, this cannot occur because pregnancy brings two dichotomous objectives into conflict. On the one hand, pregnancy necessitates harboring a foreign body, since half of the genetic material of the developing organism is paternally derived. On the other hand, the immune system identifies and attacks foreign bodies. Accordingly, pregnancy can occur only if maternal immunological defenses are initially restrained (see Loke and King 1997).

Lymphocytes, crucial components of the immune system, compose both specific and nonspecific defenses. T and B cell lymphocytes are specific; each clonal cell line binds only to a single antigen. Other lymphocytes, including natural killer (NK) cells, are nonspecific; they will attack any entity that shows indications of foreignness or dysfunction. Lymphocytes both produce and are in part regulated by a class of soluble polypeptides called cytokines. Although the dynamics of cytokine-mediated interactions among lymphocytes are complex, in general there is an opposition between two types of cytokines, referred to as Th1 and Th2. Th1 cytokines are generally proinflammatory, that is, they promote aggressive cellular response, notably including the excitation of NK cells. Th2 cytokines are generally anti-inflammatory, blocking the effect of Th1 cytokines and reducing the responsiveness of NK cells (Szekeres-Bartho and Wegmann 1996).

In normal menstrual cycling, progesterone levels remain low through the follicular phase, begin to rise just after ovulation, peak in the midluteal phase, and then decline again by the end of the cycle. However, in the event that conception occurs, instead of dropping, progesterone levels continue to climb, peaking in the first trimester (Costea et al. 2000). High progesterone levels stimulate the spleen to produce progesterone-induced blocking factor (PIBF). PIBF stimulates production of Th2 cytokines (Szereday, Varga, and Szekeres-Bartho 1997), with the result that there is a profound decrease in NK cell activity (Szekeres-Bartho and Wegmann 1996). NK cells, with their ability to attack any and all invaders, constitute a fundamental threat to the developing organism, and hence progesterone (indirectly) reduces their activity.[6] Progesterone also exercises direct effects, inhibiting lymphocyte proliferation in response to invasion (Borel et al. 1999). Trophoblasts also produce progesterone, increasing local concentrations of the hormone (see Stites and Siiteri 1983 for review). As pregnancy progresses, progesterone production shifts from the corpus luteum to the placenta (Hansen 1998), and there is a corresponding change in the distribution of the hormone over time, resulting in higher concentrations in the vicinity of the developing organism. There is also a progressive rise in the production of anti-inflammatory cytokines by cytotrophoblasts themselves, directly inhibiting local NK activity (see Roth et al. 1996; Szereday, Varga, and Szekeres-Bartho 1997). Together, this multiplex restraint of lymphocyte activity allows for the implantation of the (half-) foreign blastocyst in the endometrium and the resulting development of the embryo and placenta.

Although progesterone concentrations are highest at the feto-maternal interface, systematically elevated progesterone levels are sufficient to suppress the immunoreactivity of NK cells throughout the body (see Szekeres-Bartho 1990). As a consequence, the pregnant woman is vulnerable to infection by pathogens. Moreover, should infection occur, the feto-maternal interface is especially susceptible to attack.[7] However, this weakness is partially compensated for through an increase in phagocytosis, the engulfing of diseased cells.[8] Compensatory increases in phagocytosis take place progressively, with the greatest increases in some forms of phagocytic activity not occurring until the second trimester (Barriga, Rodriguez, and Ortega 1994), while peak activity is reached during the third trimester (Shibuya et al. 1987). Likewise, although amniotic fluid stimulates the migration of leukocytes, presumably enhancing phagocytic defense of the developing organism, it does not have this effect until the second trimester (Gleicher et al. 1980). Finally, it appears that some reduction in maternal immunosuppression occurs once gestation is well-established, as progesterone levels peak in the first trimester and there are progressive increases in maternal systematic levels of a number of Th1 cytokines following the end of this period (Vassiliadis et al. 1998).[9] Together, these patterns indicate that the initial phase of pregnancy constitutes a period of maximal maternal susceptibility to pathogens.

Pathogens pose a threat not only to the mother but to the developing organism as well. Eventually, the latter will be protected by a second line of defense, as the placenta is capable of generating nonspecific immune reactions (Paradowska et al. 1996). However, the true pla-

6. Maternal immunotolerance is multifaceted. In the latter part of the luteal phase CD56bright, a distinct type of NK cell, partially displaces other NK cells. CD56bright is inhibited by trophoblasts at the feto-maternal interface (Biassoni et al. 1999). The developing organism is protected against T cells through induction of apoptosis in cells which are sensitized to it, leading to the deletion of those clonal lines (see Zhang et al. 1999).

7. Systemic Th2 cytokine production increases progressively across the three trimesters. Some investigators have found this to be matched by increases in Th1 cytokine production, resulting in little net change over pregnancy (Matthiesen et al. 1998), but others have found the opposite pattern (Marzi et al. 1996).

8. Invasive tissues appear to be partially protected against maternal macrophages (cf. Lu et al. 1989), possibly through the expression of a special identifier protein (integrin-associated protein/CD47) (cf. Oldenborg et al. 2000). Increases in systemic levels of phagocytosis may be shackled to the maturation of the placenta because of the importance of expression of CD47 by placental cells, thus sheltering the developing organism from maternal macrophages.

9. An exception is a *drop* in IL-2 in the third trimester (Vassiliadis et al. 1998).

centa does not emerge until the 12th week, becoming fully formed at 18 to 20 weeks (Beers and Berkow 1999). Correspondingly, transfer of maternal gamma globulin (IgG) greatly increases after the end of the first trimester, providing the fetus with additional, specific, defenses (see Webster 1998). The fetal immune system can ultimately provide some defense against infection, but it is likely that significant quantities of many of the most important immune cells are not present until after the first trimester (Webster 1998, Reháková et al. 1998, Senogles et al. 1979). Hence, for a number of reasons, the vulnerability of the developing organism to pathogens is likely to be highest at precisely the same time that maternal vulnerability is highest, in the first trimester.

The immunological vulnerability of the mother and the developing organism during the first trimester is likely to have constituted a source of selective pressure during human evolution. Given that selection shapes not only physiology but also behavior, one can ask whether behavioral patterns can compensate for reduced pathogen resistance by decreasing the likelihood of transmission. In the case of some dangerous diseases, the answer is clearly no. Cytomegalovirus, for example, although not a threat to immunocompetent individuals, poses a grave risk to pregnant women and their offspring (see Forbes 1989), particularly early in pregnancy (Kumar and Prokay 1983), but natural selection has not shaped female behavior so as to mitigate this danger. Transmission occurs through contact with infected individuals or their urine. Children under five excrete more pathogens than others and hence pose a threat to unborn siblings (Bello 1992). To reduce the risk of infection, a pregnant woman would therefore need to avoid her young children during the first trimester, and in ancestral environments this would have endangered valuable existing offspring.

In contrast to the adaptive dilemma posed by socially transmitted pathogens such as cytomegalovirus, a significant class of diseases that threaten pregnant women can be avoided through affordable behavioral changes. Ingestion is a common pathway for infection, and hence increased dietary selectivity can have prophylactic effects.

Meat, Pathogens, and Pregnancy

Of all food types, animal protein (including meat, poultry, eggs, and seafood, hereafter generically termed "meat") is the most dangerous. Meat is the source of a wide range of pathogens that pose a grave threat to pregnant women and developing organisms (see Smith 1999) (table 3). Defense against these pathogens hinges upon proinflammatory cytokine activity and, conversely, is often hindered by anti-inflammatory cytokines. Consistent with the portrait of feto-maternal immunology presented earlier and in keeping with the susceptibility of organogenesis to insult, these pathogens generally pose the greatest risk early in pregnancy.

Many of the organisms listed in table 3 are present in a wide variety of game animals, either pathogenically or endosymbiotically. In some cases, pathogens are distributed throughout an infected animal; in other cases, they are confined to an animal's gastrointestinal tract but are easily spread during butchering. Still others are simply ubiquitous in the environment, with a propensity to proliferate on meat. As a result of these factors, culturally prescribed avoidance of particular species or selective consumption of body parts provides little protection from infection—all parts of any animal are potentially dangerous. Although infected or contaminated meat can be safely eaten if it is cooked thoroughly, this is not an easy thing to accomplish, or gauge, when cooking over a fire. Moreover, thorough cooking provides imperfect protection if processing practices leave hands or implements contaminated. Although simple hygienic measures can prevent infection, such practices are likely to be (and to have been) rare in most small-scale societies. Accordingly, while the advent of cooking in hominid evolution likely greatly reduced pathogen exposure associated with meat eating, for immunosuppressed females the residual risks will have remained significant.

Faunal remains and morphological features indicate that meat has constituted a significant part of the diet of our species since its inception, and it is likely that this association dates to the very beginnings of our genus (see Aiello and Wheeler 1995, Milton 1999, Sponheimer and Lee-Thorp 1999). Although portions of some societies today are vegetarian, all available evidence indicates that meat is likely to have played a critical role in the diet of every member of our species prior to the advent of agriculture (cf. Hill 1982, Kaplan et al. 2000, Mann 2000), and, while agriculture may have diminished the importance of meat, it has not eliminated it. Together, these factors make it likely that meat-borne diseases have constituted a significant source of selective pressure on pregnant women for much of human history.

If meat has been an important component in the human diet for most of our history, is it feasible (or, more specifically, was it feasible in ancestral environments) for pregnant women to avoid meat? As the case of cytomegalovirus illustrates, if meat avoidance was prohibitively expensive, no amount of benefit gained would have selected for the behavior. It is therefore important that, from an energetic perspective, human pregnancy is an extremely gradual affair: When scaled for maternal body size, the daily increase in the energy demands of pregnancy is lower in humans that in any other mammal (Prentice and Whitehead 1987). The energetic requirements of the first trimester are low relative to those of the second and third as a consequence of both the small mass of the developing organism and associated tissues and metabolic changes that may occur early in pregnancy, allowing more efficient use of available energy (see Catalano 1999). Provided that sufficient energy is consumed, dietary restriction during this period is therefore far less problematic than in later phases of preg-

TABLE 3
Attributes of Some Meat-Borne Pathogens That Pose a Threat to Pregnant Women

Pathogen	Environmental Distribution	Sequelae	Limiting Cytokine Response	Exacerbating Cytokine Response	Period of Most Dangerous Infection
Toxoplasma gondii	All warm-blooded animals can host; common in many mammals and birds[1]	Abortion, stillbirth, congenital deformities, fetal and maternal neurological and retinal damage[2]	Th1:IL-12,IL-6,TNF-alpha, GM-CSF, IFN-gamma, maternal IgG[3]	TH2:IL-4[3]	Early pregnancy[4]
Listeria monocytogenes	Ubiquitous; infects or is endosymbiotic with many mammals and marine organisms; proliferates on meat[5]	Maternal death, abortion, neonatal death, and meningitis; > 25% fatality rate with antibiotic treatment[6]	Th1: IL-12, IFN-gamma, TNF-alpha, IL-8, GM-CSF, IL-6[7]	Th2: IL-10,IL-4[7]	Early pregnancy[8]
Escherichia coli	Ubiquitous; infects many animals; proliferates on meat[9]	Maternal death, intrauterine death[10]	Th1: IL-1, IL-2, IFN-gamma[11]	TH2: IL-10[12]	Early pregnancy[13]
Brucella abortus, B. melitensis	Carried by many mammals and marine organisms[14]	Abortion, fetal death, preterm delivery[15]	Th1:IL-12, IFN-gamma, TNF-alpha[16]	Th2: IL-10, IL-4[17]	?
Shigella dysenteriae	Ubiquitous; often water-borne, but proliferates on meat[18]	Maternal dehydration, electrolyte imbalance, death, abortion, preterm delivery[13]	Th1: IL-8, IL-2[19]		Early pregnancy[13]
Campylobacter jejuni	Ubiquitous; infects or is endosymbiotic with many mammals and birds; proliferates on meat[20]	Maternal dehydration, electrolyte imbalance, death, abortion, preterm delivery, stillbirth[21]	Th1: TNF-alpha, IL-8[22]		Early pregnancy[23]
Clostridium perfringens	Carried by many mammals and birds; proliferates on meat[9]	Abortion[24]	Th1: IL-1, IL-6, TNF-alpha, IFN-gamma[25]		?
Leptospira	Infects many mammals and birds[26]	Abortion, stillbirth > 50% of cases[27]	Th1: TNF-alpha, IL-2[28]		Early pregnancy[29]

SOURCES: 1, Hejlícek, Literák, and Nezval (1997; 2, Remington, McLeod, and Desmonts (1995), Galván Ramírez et al. (1995); 3, Thouvenin et al. (1997), Suzuki (1999); 4, Beazley and Egerman (1998; 5, Cooper and Walker (1998); 6, Farber and Peterkin (1991); 7, Nakane et al. (1999); 8, Abram and Doric (1997); 9, Pepin, Russo, and Pardon (1997); 10, Moyo et al. (1995); 11, Chopra et al. (1997); 12, Zimmer et al. (1996); 13, Sechser et al. (1976); 14, Corbel (1997); 15, Makhseed et al. (1998); 16, Oliveira et al. (1998); 17, Fernandes et al. (1996); 18, Mwenye et al. (1997); 19, Sansonetti et al. (1999); 20, Cabrita et al. (1992); 21, Goh and Flynn (1992); 22, Pancorbo et al. (1999); 23, Simor and Ferro (1990); 24, Lantelme et al. (1995); 25, Wallace et al. (1999); 26, Treml and Nesnalova (1993); 27, Carles et al. (1995); 28, Petros, Leonhardt, and Engelmann (2000); 29, Shaked et al. (1993).

nancy.[10] Moreover, meat is often of dietary importance not as a source of calories but as a source of protein, and, given the relative leanness of many game animals, this is particularly likely to have been true in ancestral foraging populations (Milton 1999; also Mann 2000).

In animal models, protein restriction during early pregnancy stimulates placental growth (Perry et al. 1999). In a study of a Dutch famine, Lumey (1998) shows that human maternal undernutrition during the first trimester increases placental weight. Meat supplies are quickly exhausted during famines, and hence Lumey's results suggest that humans share the pattern of enhanced placental growth in response to protein restriction. The probable goal is a compensatory increase in the surface area across which nutrients from the mother are supplied to the developing organism (see Dwyer et al. 1995). There is a ceiling to such benefits; a large ratio between the weight of the placenta and that of the infant is predictive

10. Zhou, O'Brien, and Relyea (1999) found a negative association between intensity of vomiting during pregnancy and birth weight and consider it to be principally due to the link between the intensity of vomiting and its duration across gestation. Women who experience severe vomiting late in pregnancy are thought to suffer inadequate nutritional intake during the period of maximal fetal demands. Singh and Rosen (n.d.) found an association between adult fluctuating asymmetry and maternal third- (but not first- or second-) trimester nausea, a finding which can be interpreted as indicating that the negative impact of pregnancy sickness–induced dietary restriction is greatest late in pregnancy. These findings can be viewed as suggesting that the benefit/cost ratio of pregnancy sickness decreases across gestation, and this is consistent with its predominance early in pregnancy.

of future health problems (Langley-Evans et al. 1996). However, such disproportionate ratios most likely occur when the maternal diet is restricted throughout pregnancy, a condition that impacts fetal health in a variety of ways. Although prolonged protein deprivation during pregnancy is detrimental, decreased protein intake that is restricted to the first trimester may be minimally harmful (cf. Langley-Evans et al. 1996), harmless (cf. Dwyer et al. 1995, Muaku et al. 1995), or even beneficial (cf. Perry et al. 1999). Lastly, while meat is an important source of protein, it is not the only source, and hence it is possible that compensatory increases in the consumption of other foods might mitigate the reduction in protein intake entailed by meat avoidance.

Hypoferraemia (iron-deficiency anaemia) during the first trimester is associated with poor pregnancy outcome (see Schwartz and Thurnau 1995). Meat is a principal source of iron, a critical determinant of cellular growth, and hence avoidance of meat early in pregnancy could conceivably have detrimental consequences. However, as I will show, there is evidence that mild hypoferraemia may actually be beneficial and that iron availability is influenced by multiple dietary factors.

In sum, well-nourished women can apparently afford to avoid meat during the first trimester, making it possible for selection to have shaped the psychophysiological determinants of behavior in such a way as to shield immunosuppressed pregnant women against the threat of meat-borne pathogens.

Additional Design Features of Pregnancy Sickness

Having established that, as least with regard to the hazards posed by meat-borne pathogens, pregnancy sickness evinces the outward signs of an adaptation produced by natural selection, keeping in mind Williams's (1966) admonition that adaptations are best identified on the basis of the complex nature of their designs I turn now to a more detailed examination of the subjective changes that affect ingestion during pregnancy.

Profet (1992) and Erick (1995) suggest that the increase in nausea susceptibility and changes in food preferences co-occur with an increase in olfactory acuity (cf. Dickens and Trethowan 1971). This is a plausible suggestion given that olfaction is a principal avenue for the remote detection of food properties and plays an important role in the elicitation of nausea (cf. Fernández-Marcos et al. 1996).

Yeremina (1976) examined 439 pregnant Siberian women and found enhanced epithelial motility and olfactory sensitivity during the first and second trimesters, but methods are not described. A longitudinal study of 20 German women failed to find an influence of either pregnant state or stage of pregnancy on olfactory acuity. However, compared with controls, pregnant women rated four of five food-related odors as less palatable, a trend which was most significant for peanut odor (Laska et al. 1996). Gilbert and Wysocki (1991) compared 13,610 pregnant women with 277,228 nonpregnant women between the ages of 20 and 40 in the United States and found that pregnant women failed to display enhanced acuity to five of six odorants but (*a*) were less willing to eat three of the six odorants, (*b*) rated three odorants as less pleasant than they were rated by nonpregnant women, and (*c*) were less willing to wear perfume.

Hansen and Glass (1936) report "olfactory perversions" among pregnant German women: Individuals who have spent their whole lives in animal husbandry describe being unable to bear the smell of animals, particularly their excreta, when pregnant, and the same is true with regard to fish odor.[11] Fairburn, Stein, and Jones (1992:667) recorded gestational aversions among 80 out of 100 subjects and noted that "an altered sense of smell appeared to underlie the majority of the aversions." O'Brien, Relyea, and Lidstone (1997) asked 124 women to record elicitors of nausea during pregnancy; odors were highly nauseogenic, an effect which subjects blamed on profound alterations in the sense of smell. Knox, Kremer, and Pearce (1995) asked 100 pregnant Irish women about alterations in smell and found that 39% reported heightened olfactory awareness; this change was strongly correlated with experiencing cravings and aversions (fried food and cooking meat being particularly aversive) but not with the incidence of nausea or vomiting. Three days or less postpartum Cooksey (1995) asked 280 women, "Did you experience craving to smell anything special?" Fourteen percent reported strange olfactory cravings never before experienced, and 1.4% spontaneously reported alterations in olfaction. Cantoni et al. (1998) collected retrospective data from 500 German women, finding that 68% reported changes in olfactory perception (principally increased olfactory responsiveness) during pregnancy, most markedly during the first trimester. Seventy-four percent remembered perceiving specific odors as markedly less pleasant than usual, while 21% recalled increases in olfactory pleasure; meat and smoke were prominent stimuli for the former class, while fruit and flowers were prominent for the latter. Fifty-eight percent reported nausea in response to odors, and 75% reported changes in diet, with both effects concentrated in the first trimester. Changes in olfactory perception were positively correlated with both nausea and changes in consumption.

Looking beyond Western cultures, Al-Kanhal and Bani (1995) list "unpleasant smell" as an important reason given by pregnant Saudi women for avoiding particular foods. Morton (n.d.) describes a powerful association between odor and nausea among pregnant Tongan women, and Beaglehole and Beaglehole (1941) similarly report both reduction in appetite and abnormal sensitivity to bad smells as indices of the onset of pregnancy in Tonga. Obeyesekere (1985) reports "olfactory distortions" in-

11. The researchers also tested olfactory acuity using the odors of rubber, rose, and almonds. They describe a deficit in the third trimester relative to the postnatal period but do not report the results from their examinations earlier in pregnancy.

volving revulsion among pregnant women in Sri Lanka. Beaglehole and Beaglehole (2000 [1935]) note that early in pregnancy the Hopi woman is "without appetite, since food, especially meat, smells bad." In conclusion, although acuity per se may be unchanged in pregnancy, marked alterations do indeed occur in olfactory experience.

As Profet (1992:334) notes, olfactory cues are an extremely accurate index of degree of spoilage caused by bacteria in meat. Although systematically collected data on olfactory preferences are generally absent from the ethnographic corpus, it is my impression that, while cultural variation exists, such putrid odors are nauseogenic in most populations (cf. Tuzin 1986)—selection seems to have crafted an aversion to these odors, presumably because of the risk of pathogen ingestion. Correspondingly, mechanisms that increase the aversiveness of putrid odors during pregnancy are likely to decrease the probability that spoiled meat will be eaten. However, an exclusive focus on putrid odors would provide only partial protection, since many pathogens, particularly those that are endosymbiotic with prey species, are unlikely to produce a detectable odor. Accordingly, the goal of prophylaxis would be best served by hedonic changes such that the odor of meat in general is aversive. A plausible phylogenetic scenario is therefore one in which putrid meat was the initial target of olfactory safeguards and these measures were subsequently expanded to include animal flesh in general (J. Moore, personal communication).

Profet (1992:334) suggests that the odor of cooking meat is particularly aversive for pregnant women, and Knox, Kremer, and Pearce (1995) robustly confirm this. Cooking is likely to release many of the volatile constituents of meat odor and can thus be seen as an amplification of the aversive signal. Flaxman and Sherman (2000:127) report that, second to meat, a principal target of gestational aversions is "nonalcoholic beverages," of which coffee is the primary contributor. As Profet (1992: 352) points out, odor is an important element in gestational aversions to coffee (cf. Knox, Kremer, and Pearce 1995). On the plant, however, coffee cherries have little odor, and this suggests that, contrary to Profet's phytotoxin hypothesis, the aversion to coffee may be a consequence of the misfiring of a mechanism designed to detect volatile meat odors.

Whereas odor seems more likely to signal danger in meats than in vegetable foods, the converse would seem to be true of taste. Arguing that bitter tastes index phytotoxicity, Profet (1992:333) postulated an increased aversion to bitter tastes in pregnancy. This prediction has recently been confirmed by Duffy et al. (1998), making it more difficult to dismiss the phytotoxin hypothesis despite the low frequency of reported aversions to vegetables.[12]

12. Sipiora et al. (2000) found that hyperemetic pregnant women exhibited different patterns of bitter-taste perception from those with lower levels of vomiting.

Gestational Cravings

Both the ethnographic and the clinical literature report marked cravings in conjunction with pregnancy. A number of investigations have considered the possibility that such cravings may have adaptive value; typically, explanations focus on the ingestion of sugars or micronutrients (cf. Jeans, Smith, and Stearns 1952, Hook 1980, Tierson, Olsen, and Hook 1985, Demissie, Muroki, and Kogi-Makau 1998). Consistent with the changes in olfactory experience discussed above, there appears to be an association between cravings and olfaction (Pelchat and Schaefer 2000, Cooksey 1995), and cravings appear primarily in the first trimester (Wijewardene, Fonseka, and Goonaratne 1994, Flaxman and Sherman 2000). As is true of aversions, cravings are clearly motivationally relevant, as both experiences importantly influence women's diets (Harries and Hughes 1957; Hook 1978, 1980; Darwish, Amine, and Abdalla 1982; Schwab and Axelson 1984; Tierson, Olsen, and Hook 1985; Knox, Kremer, and Pearce 1995; Pope, Skinner, and Carruth 1997).

DAIRY PRODUCTS AND PROTEIN

Flaxman and Sherman report a high frequency of cravings for milk and other dairy products and a low frequency of aversions. While the attractiveness of meat declines during pregnancy, the attractiveness of other sources of protein may rise in a complementary fashion—the propensity to experience cravings is significantly associated with the propensity to experience aversions (Knox, Kremer, and Pearce 1995), suggesting a compensatory mechanism (Demissie, Muroki, and Kogi-Makau 1998). Such compensatory adjustments can explain the lack of negative impact of nausea and vomiting (severity of which is associated with number of aversions [Rodin 1989, Crystal, Bowen, and Bernstein 1999]) on postpartum measures of maternal nutritional status (Weigel et al. 2000).

Humans may be able to detect protein using olfactory, gustatory, and possibly postingestive cues (cf. Westerterp-Plantenga and Wijckmans-Duijsens 1996, Crovetti et al. 1998). By increasing the appeal of cues of protein (while still making meat aversive), some of the costs of meat avoidance could be reduced. In ancestral environments, plant foods, such as nuts and seeds, and insects would have been the primary sources of nonmeat protein (Southgate 1991).[13] Cravings for dairy products may thus

13. In addition to protein, important nutrients include the essential fatty acids necessary for proper neurological development. As is true of protein, many of these nutrients can be obtained from nuts and seeds (Ollis, Meyer, and Howe 1999). In contemporary circumstances, nuts and seeds may sometimes be hazardous to pregnant women because they contain mycotoxins produced by molds (Peraica et al. 1999). However, the risk of mold proliferation is greatest when nuts or seeds are stored in large quantities (Peraica et al. 1999), and hence it is not clear that mycotoxins would have constituted a significant source of selective pressure prior to extensive reliance on agriculture. Nevertheless, given that nuts and seeds may contain

constitute an evolutionarily recent manifestation of a mechanism designed to motivate the search for alternative protein sources.[14]

CLAYS AND CALCIUM

Geophagy is found in many societies (Reid 1992, Wiley and Katz 1998, O'Brien, Relyea, and Lidstone 1997). Despite persistent efforts to classify it as a pathology or culture-bound syndrome (cf. Grigsby et al. 1999), it is likely that geophagy is often adaptive (see Reid 1992, Wiley and Katz 1998). Animal models indicate that geophagy is often elicited by nausea (Mitchell, Winter, and Morisaki 1977, McCaffrey 1985). Many primates and other animals engage in geophagy in conjunction with the consumption of substances that contain high concentrations of toxins (Krishnamani and Mahaney 2000, Gilardi et al. 1999). Provenancial evidence suggests that hominid geophagy may date back to *Homo erectus*, if not beyond (Johns and Duquette 1991). Both nonhuman animals and humans are generally quite selective about the type of earth consumed, and olfactory cues may be significant in the attraction to particular earths (Forsyth and Benoit 1989, Krishnamani and Mahaney 2000; cf. Harner 1972).[15] Clays, the most frequent targets of geophagy, possess a structure that makes them extremely absorbent, resulting in remarkable detoxifying attributes (see Johns and Duquette 1991, Reid 1992, Wiley and Katz 1998, Krishnamani and Mahaney 2000). Given its distribution across taxa, it is likely that there is an evolved attraction to clays during nausea; societies may then recognize this attraction, as well as its beneficial consequences, and institutionalize the behavior (Wiley and Katz 1998).

Geophagy is associated with pregnancy, sometimes exclusively so, in many societies (Wiley and Katz 1998; cf. Harner 1972). Both the greater incidence of nausea and the greater importance of detoxification are likely to result in increases in the behavior during this period (Wiley and Katz 1998; Profet 1992:345). However, although geophagy is present across Africa, it is rare or absent in societies in which pregnant women consume significant quantities of milk and other dairy products (Wiley and Katz 1998). Milk differs from clays in that it lacks the surface-area properties that make the latter highly absorbent. Correspondingly, milk is a relatively poor detoxifier and has no antinauseogenic properties (cf. Hu, Lagomarsino, and Luo 1998, Lindseth and Lindseth 1995). However, both many of the targeted clays (Wiley and Katz 1998) and milk possess a high calcium content.

Calcium demands rise during pregnancy as a result of both the increase in maternal plasma and the developing organism's progressively escalating requirements. These increased demands are anticipated by enhanced maternal intestinal absorption of calcium beginning in the first trimester (Ritchie et al. 1998). Both fetal demand and maternal absorption increase markedly during the second trimester, the period of fetal skeletal formation. Grave consequences accompany a failure of the latter to keep pace with the former.

Preeclampsia is a potentially dangerous condition characterized by maternal hypertension after the 20th week and a shift to proinflammatory cytokines and cellular activity (Saito et al. 1999). Inadequate calcium consumption increases the risk of preeclampsia, and supplementation reduces it (Crowther et al. 1999, Lopez-Jaramillo 2000), apparently because of the role which calcium plays in the system whereby diseased cells incite immunological response (cf. von Dadelszen, Wilkins, and Redman 1999, Hojo et al. 1999). Demands for calcium thus derive from both the structural importance of calcium for the developing organism and the necessity of maintaining sufficient calcium levels to preclude maternal immunological rejection of it. These considerations are so important that maternal bone matter is routinely sacrificed to ensure adequate calcium levels (cf. Black et al. 2000). Increases in the subjective appeal of calcium-rich clays and dairy products during the first and second trimesters may thus be understood as an adaptation designed to enhance the stockpiling and, later, the supply of calcium in order to meet increasing fetal demands for calcium (cf. Hook 1978, 1980; Wiley and Katz 1998).

Iron is required for all cellular growth. However, unlike calcium, iron is toxic at high concentrations, and the body is unable to store substantial quantities for future needs. Moreover, to make matters worse, the presence of calcium inhibits the absorption of dietary iron and meat is a principal source of iron. Hence, when viewed with regard to the developing organism's need for iron, the existence of both cravings for calcium and aversions to meat appear bizarre and maladaptive. However, cravings for fruit shed light on the resolution to the apparent paradox posed by the need simultaneously to avoid pathogens, ensure an adequate calcium supply, and maintain iron levels.

secondary compounds (cf. Gilardi et al. 1999) (and given Laska et al.'s olfactory data), broad generalizations about the costs and benefits of nut and seed consumption in ancestral environments are premature. Eusocial insects, the preferred targets of human consumption (Defoliart 1995), themselves face strong pathogen risk and may have evolved hygienic practices and other specialized adaptations in response (cf. Rosengaus et al. 1998, 1999). As a consequence, at least with regard to those pathogens that afflict both humans and their insect prey, eusocial insects may be a less risky source of protein than meat.

14. An obstacle to adaptive explanations of dairy-product cravings is the fact that dairy products are a potential source of many of the pathogens described above (Vasavada 1988). Conventional evolutionary psychology (cf. Tooby and Cosmides 1992) holds that the approximately 6,000 years (Bökönyi 1969) since humans are thought to have begun consuming the milk of domesticated animals is too short a period for the evolution of an aversion to milk. However, the presence of adult lactase production in populations with a history of animal milk consumption (see Durham 1991) suggests that adaptations may evolve or be modified within such a period.

15. Geophagy carries the risk of infection from soil-borne parasites (Geissler et al. 1997). However, children seem particularly likely to suffer this cost, perhaps as a result of inadequate selectivity.

FRUIT AND VITAMIN C

Flaxman and Sherman (2000:127) find that fruit is the single most common target of cravings and is rarely aversive. For Bengkulu women, fruit is *the* prototypical food craved during pregnancy, and, similar patterns appear elsewhere.[16] In ancestral environments, fruit is likely to have been an important source of carbohydrates (Southgate 1991). The human preference for sweet tastes, a prominent factor in the desire for fruit, is most likely an adaptation that facilitates caloric maximization. Sweet foods in general are a prominent target of gestational cravings (Flaxman and Sherman 2000, Taggart 1961, Obeyesekere 1985, Wijewardene, Fonseka, and Goonaratne 1994, Knox, Kremer, and Pearce 1995). Hook (1978, 1980) argues that these cravings facilitate the expansion of energetic reserves in anticipation of the demanding later phases of pregnancy.

A number of sources report cravings for *unripe* fruit (Adriani and Kruyt 1996 [1951], my field notes, Wijewardene, Fonseka, and Goonaratne 1994). Likewise, in disparate populations many subjects experience cravings for sour or savory foods.[17] In comparison with a simple desire for sweet things, such preferences seem less likely to maximize the caloric content of foods. Rather, the craving for sweet and the craving for sour are related, as fruits often combine these two tastes—in the ancestral past, fruit was the principal source of these gustatory experiences.

For much of human history, fruit was a key source of vitamin C (Southgate 1991). There is a clear correlation between fruit consumption during pregnancy and serum vitamin C levels (Ortega et al. 1998). Vitamin C provides protection against a number of teratogenic substances and unfavorable uterine conditions (cf. Usami, Tabata, and Ohno 1999, Maritz and van Wyk 1997). Animal models and human studies suggest that vitamin C enhances proinflammatory cytokine production (Schwager and Schulze 1998, Jeng et al. 1996), thus partially counteracting the shift to anti-inflammatory cytokines engendered by high progesterone levels. Vitamin C may also greatly enhance the productivity of activated macrophages (Mizutani et al. 1998). While such actions might seem to expose the developing organism to maternal immunological attack, this danger is perhaps mitigated by vitamin C's simultaneous reduction of NK cell activity (Huwyler, Hirt, and Morell 1985). Mathews, Yudkin, and Neil (1999) found that (*a*) vitamin C was the only micronutrient predictive of birth weight and placental weight (recall that a larger placenta is a good thing, provided that birth weight rises as well) and (*b*) these effects occurred only with regard to vitamin C consumption early in pregnancy.[18] A craving for foods high in vitamin C during the first trimester thus ensures a supply of the micronutrient which suffices to meet the needs of both the mother and the developing organism.

Vitamin C enormously enhances the absorption of iron, in part through preventing the formation of insoluble iron compounds. It thus counteracts the detrimental influence of calcium on iron absorption (see Lynch and Cook 1980). One simple possibility, therefore, is that, while meat avoidance reduces dietary iron intake and calcium consumption reduces absorption, the resulting decrement in iron availability is compensated for through increased vitamin C consumption. Although the basic outlines of this explanation may be correct, actual events are likely more complex.

Gestational Hypoferraemia

I noted earlier that extreme hypoferraemia during the first trimester can have detrimental consequences. However, pregnancy is frequency characterized by a progressive increase in hypoferraemia to the point that, by the third trimester, the vast majority of pregnant women have an iron profile equivalent to that of an extremely iron-deprived nonpregnant woman (Hollán and Johansen 1993). Fetal demands for iron increase progressively and do so independent of the level of maternal reserves (Bentley 1985). Because iron levels are relative to total blood volume, the significant increase in plasma also necessarily results in lower iron levels (Hollán and Johansen 1993). Finally, iron regulation may play a critical role in fighting disease.

Because microbial and protozoan pathogens are dependent on iron, the availability of iron can be a limiting factor in pathogen proliferation. Accordingly, the body's response to pathogen invasion can be viewed as a delicate balancing act between meeting the need for iron and withholding iron from the invader (see Sunder-Plassmann, Patruta, and Horl 1999 for review). Hypoferraemia in general may thus constitute an adaptive response to pathogen threat (Hollán and Johansen 1993, Nesse and Williams 1995). Likewise, gestational hypoferraemia is probably both normal and highly functional (Goodlin 1982, Montgomery 1990, Hollán and Johansen 1993). There is a strong association between maintenance of high iron levels and the risk of low birth weight and preterm delivery (Goldenberg et al. 1996), outcomes associated with maternal infection. All of the meat-borne

16. See Taggart (1961 [Scottish]), Obeyesekere (1985) and Wijewardene, Fonseka, and Goonaratne (1994 [Sri Lankan]), Dye, Jones, and Hill (1998 [English]), Thompson (1940 [Fijian]), Textor (1973 [Thai]), Knox, Kremer, and Pearce (1995 [poor Irish]), cf. Norgan, Ferro-Luzzi, and Durnin (1974 [New Guinean]), Dufour, Reina, and Spurr (1999 [Colombian]).

17. See Harries and Hughes (1957), Fairburn, Stein, and Jones (1992 [English]), Taggart (1961 [Scottish]), Knox, Kremer, and Pearce (1994 [Irish]), Posner, McCottry, and Posner (1957), Schwab and Axelson (1984), Finley et al. (1985), Tierson, Olsen, and Hook (1985), Pope, Skinner, and Carruth (1992 [U.S.]), Kehrer (reported in Hansen and Langer 1935 [German]), Al-Kanhal and Bani (1995 [Saudi]), Walker et al. (1985 [South African]), Rizvi (1980 [Bangladesh]), Obeyesekere (1985), Wijewardeme, Fonseka, and Goonaratne (1994 [Sri Lankan]), Foll (1959 [Burmese]), Laderman (1981 [Malay]), Textor (1973 [Thai]), Kaufman (2000 [1960] [Thai]), Levy (1973:143 [Tahitian]).

18. Mathews et al. play down the clinical significance of these findings for well-nourished British women; however, these effects are likely to be amplified under conditions more typical of ancestral circumstances.

pathogens listed in table 3 are highly dependent on iron (Dimier and Bout 1998, Fisher and Martin 1999, Lopez-Goni and Moriyon 1995, Chart, Scotland, and Rowe 1989, Coker and Obi 1991, but see also Schwartz et al. 1996, Miles and Maskell 1985, Staneck, Henneberry, and Cox 1973).

The threat of disease is greatest during the first trimester, yet hypoferraemia increases over the course of pregnancy. If hypoferraemia provides disease protection, why is the pattern not reversed? First, in keeping with the importance of iron for cell growth and replication, iron transfer occurs as soon as sufficient placentation has taken place (Gulbis et al. 1994), and first-trimester hypoferraemia is associated with negative birth outcomes (Schwartz and Thurnau 1995). Second, maternal iron levels must not only accommodate immediate fetal demands but also be calibrated in light of the upcoming progressively increasing fetal needs. Maternal iron absorption rates increase ninefold from the first trimester to the third (Barrett et al. 1994). Nevertheless, despite increased absorption, maternal iron levels continue to drop with each week of gestation (Milman, Graudal, and Agger 1995)—a trend which is evidently due to increasing fetal demands, since the effect is more substantial in multiple gestations (Ben Miled et al. 1989). Therefore, although hypoferraemia during the first trimester could conceivably provide enhanced protection from pathogens, both the initial demands of the developing organism for iron and the rapidity of the increase in those demands preclude beginning the developmental process with low maternal iron levels. The converse considerations explain why rates of maternal absorption do not increase sooner. Although greatly enhancing iron absorption during the first trimester would decrease the strain which the developing organism places on maternal iron stores, an abundance of iron during the period of maximum immunovulnerability would facilitate pathogen proliferation. The lag between increases in absorption rates and increases in demands on maternal stores can thus be seen as an attempt to minimize disease vulnerability while still meeting the needs of the developing organism.

Many ingested pathogens begin the process of proliferation prior to extensive tissue penetration. Accordingly, the presence of iron in the gastrointestinal environment may constitute a critical determinant of the hazards posed by ingested pathogens (Stockman 1981). A number of primates consume calcium-rich clays when suffering heavy parasite loads (Krishnamani and Mahaney 2000), one benefit of which may be a reduction in the availability of iron in the gastrointestinal tract. Many studies find an association between geophagy and anemia in humans. Although some investigators, noting the presence of iron in soils, have argued that anemia precedes and motivates geophagy, the preferential focus on calcium-rich clays with a marked ability to inhibit the absorption of iron suggests that causality occurs in the opposite direction (see Reid 1992, Patterson and Staszak 1977, Hochstein 1968).[19]

Geophagy is a form of pica, the ingestion of inedible substances. Gestational pica occurs at differing frequencies in diverse populations but is likely grossly underreported in some groups given the atmosphere of shame and secrecy which often surrounds it (Cooksey 1995). Pica provides definitive evidence of purely endogenous modification of gestational ingestion, since overwhelming compulsions are experienced even by women who are unaware that others have similar experiences, are baffled by their desires, and worry that their behavior is "crazy." These cravings lead to ingestion in spite of fears that it will harm the developing organism or the mother herself. Olfaction appears to play a central role in pica cravings (Cooksey 1995, Simpson et al. 2000).

Inedible substances ingested as a consequence of gestational cravings include cornstarch, laundry starch, plaster, ashes, coal, chalk, burnt matches, baking soda, baking powder, flour, cigarette ashes, egg shells, magnesium carbonate, powdered detergent, toothpaste, whitewash, corn meal, raw oatmeal, raw rice, raw potatoes, dry powdered milk, and baby powder (Jeans, Smith, and Stearns 1952, Harries and Hughes 1957, Posner, McCottry, and Posner 1957, Walker et al. 1985, Cooksey 1995, Al-Kanhal and Bani 1995, Knox, Kremer, and Pearce 1995, Simpson et al. 2000, Smulian, Motiwala, and Sigman 1995, Rainville 1998, Rizvi 1980; also Levy 1973; O'Brien, Relyea, and Lidstone 1997). Many of these materials are highly absorbent, and, as in the case of geophagy, this may allow them to mitigate the danger posed by ingested toxins. Likewise, just as geophagy occurs among many animals, some nonhuman primates consume charcoal in response to high levels of phytotoxins (Krishnamani and Mahaney 2000). Both the significant toxin-neutralizing properties of many clay and nonclay targets of pica and the presence of comparable pica in nonmeat-eating species lend support to Profet's claim that phytotoxin avoidance is an important adaptive function of pregnancy sickness.

Other forms of pica may also have multiple benefits. In addition to being highly absorbent, many of the substances listed above have a marked ability to inhibit the absorption of iron.[20] Some, such as chalk, plaster, white-

19. Clinical trials showing that iron supplementation reduces geophagy and related practices have been adduced as evidence that anemia causes geophagy. However, such studies overlook the implicit influence which the treatment process exercises over the behavior under scrutiny (Reid 1992),. Nevertheless, the fact that some clays targeted in geophagy actually release iron (Johns and Duquette 1991) makes it impossible to rule out this possibility.

20. An additional substance which is widely craved, ice (Knox, Kremer, and Pearce 1995, Cooksey 1995, Smulian et al. 1995, Rainville 1998), has neither of these properties. Hadjadj et al. (1990) suggest that ice craving may be due to ice's palliative effects on the gastrointestinal consequences of hypoferraemia. However, subjective accounts emphasize a powerful craving for the substance itself rather than relief experienced after ingestion (Cooksey 1995). It is unlikely that an evolved system would motivate ingestion of materials which have been unavailable for many populations over most of their history. Crushed ice resembles a mineral powder, and women report craving ice which is crushed in a particular fashion

wash, egg shells, and baby powder, have this effect because of the presence of calcium; others act through different pathways. For example, amylophagia, the consumption of indigestible starch, is epidemiologically associated with hypoferraemia. Although the direction of causality has been debated (Ephros and Lee 1988), animal models show that starch binds dietary iron, inhibiting its uptake (Thomas, Falko, and Zuckerman 1976, but see also Morais et al. 1996).

Extending an observation of Minturn and Weiher (1984), Flaxman and Sherman (2000) report that, of the 27 societies listed in the Human Relations Area Files for which there is information pertaining to gestational nausea and vomiting, the 7 societies in which ethnographers noted the absence of these symptoms are significantly more likely to rely on maize as a dietary staple. Corn is rich in phytic acid, an iron-binding substance that greatly reduces the bioavailability of dietary iron (cf. Wrobel et al. 1999). In contrast, polished white rice contains less than half as much phytic acid as corn (Liu et al. 1993) and exercises minimal influence on the availability of iron (Tuntawiroon et al. 1990). Minturn and Weiher found a positive association between rice consumption and pregnancy sickness. In Flaxman and Sherman's assessment, none of the 7 societies reportedly lacking pregnancy sickness rely on rice as a primary staple. Among the Toraja, for whom rice is the primary staple, pregnant women are reported to experience a powerful aversion to rice, preferring to eat only corn (Adriani and Kruyt 1996 [1951]). Although it is not clear what the proximate cues might be, it is possible that pregnant women experience a strong attraction to corn (but see Pike 1997) or an aversion to rice (cf. Obeyesekere 1985, Wijewardene, Fonseka, and Goonaratne 1994, O'Brien, Relyea, and Lidstone 1997:55) or both because the relative ability of these staples to bind iron somehow influences the frequency of nausea and vomiting.[21]

Meat is extremely rich in iron, and hence meat eating poses a double threat to an immunosuppressed woman, as she risks both ingesting pathogens and supplying them with the fuel which allows them to multiply within her gastrointestinal tract. Nevertheless, not all pregnant women are averse to all meat all of the time. Some of the variability in the severity and duration of pregnancy sickness may be due to the existence of heritable differences with regard to the balance point in the trade-off between optimal prophylaxis and optimal nutrition. Likewise, there may sometimes be utility in gambling by consuming meat. This possibility is enhanced in light of the centrality of meat in the diets of some hunter-gatherer groups, making total meat avoidance possibly very costly in some ancestral populations. How, then, can the risks of meat eating be reduced? One possibility is that, although complete decontamination of meat is unlikely to have been feasible for most of human history, the same is not true of manipulation of the availability of iron in the gastrointestinal environment.

Consumption of substances that inhibit iron absorption significantly reduces the availability of iron from meat (Thomas, Falko, and Zuckerman 1976). Women may thus be able to decrease the risks of meat eating by consuming such substances in conjunction with or soon after meat. Yet, given that vitamin C has the potential to enhance iron absorption (and does so independent of the presence of calcium [Hallberg et al. 1992]), it might seem that regulation of iron availability in the gastrointestinal environment would necessitate a complex timing mechanism for separating the ingestion of substances that inhibit the absorption of iron from the ingestion of substances that are rich in vitamin C. However, the need for such a mechanism may be obviated by the fact that vitamin C has minimal influence on the absorption of the iron available in meat (heme iron), exercising its effects primarily on the absorption of the iron available in plant foods (nonheme iron) (Lynch et al. 1985). Accordingly, a diet rich in both substances that inhibit iron absorption, such as calcium and indigestible starch, and foods high in vitamin C may provide additional protection from meat-borne pathogens while still meeting the iron requirements of both the mother and the developing organism.

Problematic Aspects

ABSENCE OF UNIVERSALITY

Flaxman and Sherman analyzed 56 studies involving 79,146 pregnancies and 64,876 women and found a mean proportion of women experiencing nausea and vomiting of 62%, with population frequencies ranging from 35 to 84% (2000:119) (see also Emelianova et al. 1999, Weigel et al. 2000). Ethnographic reviews by Weigel (1985) and Flaxman and Sherman (2000) have revealed that, though widely distributed, pregnancy sickness is reportedly absent in some groups. If it is an evolved adaptation, why don't all women experience it?

Pregnancy sickness spans a range of subjective and behavorial phenomena, from dramatic bouts of vomiting to subtle changes in tastes. Studies which discriminate between nausea and vomiting find that the former is more pervasive than the latter (Weigel 1985, Tierson, Olsen, and Hook 1986, Vellacott et al. 1988, Dye, Jones, and Hill 1998, Zhou, O'Brien, and Relyea 1999, Weigel et al. 2000). Subtle changes in food preferences are probably even more prevalent, but because most surveys focus on nausea and, to an even greater degree, vomiting they are almost certainly underrepresented in the data (Profet 1992). Underreporting is probably exacerbated by the fact that the processes of learning and memory for-

(Cooksey 1995). Cravings for ice may thus be a misfiring of a mechanism aimed at other substances. The consumption of harmful substances such as paint (Smulian, Motiwala, and Sigman 1995) is likely the consequence of similar misfirings, and the same probably applies to many strange olfactory cravings (cf. Cooksey 1995).

21. A reviewer has pointed out that the major iron-binding substances in the human diet are plant tannins; however, foods such as tea that are rich in tannins may be avoided because of gustatory changes (cf. Flaxman and Sherman 2000, but see also Knox, Kremer, and Pearce 1995).

mation involved in the development of food aversions are such that many aversions probably shape behavior in an unconscious fashion (see Bernstein 1999). Lastly, expectations are an important determinant of the salience and subjective impact of a variety of aspects of pregnancy, and such expectations vary both within populations and between them (compare Waldenstrom 1999 with Levy 1973; also Mead 1949:219–20; O'Brien, Relyea, and Lidstone 1997). All of these factors can contribute to variation in the frequency of pregnancy sickness without calling into question the proposition that pregnancy sickness is an adaptation.

Humans are remarkably flexible, able to exist in a wide range of environments and subsist on a wide range of foods. It is likely that this flexibility is ancient, for our lineage has occupied diverse ecosystems for hundreds of thousands of years. Even within local settings, seasonal and year-to-year fluctuations in food availability will continue to favor dietary flexibility. Nausea, vomiting, and gestational aversions constrict dietary options and inflict energetic costs on pregnant women. An optimizing adaptation can be expected to balance the benefits of behavioral prophylaxis against a woman's ability to bear the attendant costs where that ability is a function of both the availability of less hazardous alternative foods and the woman's overall nutritional status. Consistent with the latter assumption, between-population comparisons of progesterone levels indicate a positive relationship with nutritional status (Bentley 1999), suggesting a possible reduction in immunosuppression and pregnancy sickness under conditions of dietary stress. More directly, mid-upper-arm circumference and triceps skinfold thickness—measures of energetic reserves—are positively associated with the experience of aversions (Demissie, Muroki, and Kogi-Makau 1998), and there appears to be a correlation between the severity of nausea and vomiting and body mass index (reviewed in Huxley 2000, but see also Weigel 1985). Variation in nutritional status both within and between populations is therefore likely to contribute to variation in pregnancy sickness, and it is plausible that variation in the availability of alternatives to meat will have a similar effect. To date, investigators have not controlled for such variation, and hence reported differences in the frequency of pregnancy sickness shed no light on its utility or lack thereof.

Multiple gestations are associated with increased pregnancy sickness (Brandes 1967, Fairweather 1968, Walker et al. 1985). Multiple gestations can thus contribute to within-population variation in pregnancy sickness and, because populations differ substantially in their rates of twinning (Bortolus et al. 1999), to between-population variation as well.

Although pregnancy sickness exhibits heritable variation (reviewed in Flaxman and Sherman 2000), this does not invalidate the claim that it is an adaptation. Animal models reveal heritable variation in the propensity to develop food aversions in response to nausea (reviewed in Mattes 1991), but there is overwhelming evidence that this is an adaptation (reviewed in Bernstein 1999). With regard to both learned aversions and pregnancy sickness, the goal of avoiding ingestible hazards often conflicts with the goals of achieving optimal nutrition and optimal foraging efficiency. Because different environments favor different balance points in this trade-off, gene flow and/or environmental change may allow for a diversity of alleles, producing substantial variation in the tendency to prioritize one goal over the other. In sum, observable variation, a highly overdetermined phenomenon, does not weaken the argument that pregnancy sickness is an adaptation.

POSITIVE EFFECTS OF MEAT CONSUMPTION ON GESTATIONAL NAUSEA

Recently, Jednak et al. (1999) found that high-protein meals, including those containing meat, have a palliative effect on first-trimester nausea. This finding clearly runs counter to predictions derived from the meat-avoidance hypothesis, particularly since the subjects were well-nourished Western women. However, given the small sample size ($n = 14$), this study must be replicated before definitive conclusions can be reached.

OUTCOME MEASURES

Adaptationist explanations of pregnancy sickness are often both attacked (i.e. Brown, Kahn, and Hartman 1997) and defended (i.e., Weigel 1985, Profet 1992, Flaxman and Sherman 2000, Huxley 2000) on the basis of correlations between presence or severity of pregnancy sickness and pregnancy outcome in Western clinical populations. However, the industrialized nation-state is an evolutionarily novel environment, and it is not clear that the selective pressures which may have shaped pregnancy sickness remain unchanged.[22] In such societies, meat production, shipment, and storage are closely regulated. Public education efforts reduce infection resulting from inadequate hygiene during preparation or insufficient cooking, and obstetricians routinely warn pregnant women to handle meat carefully and cook it thoroughly. Rates of infection proportionate to meat consumption are therefore doubtless substantially lower than would have been true in most ancestral populations. It is therefore difficult to measure whether pregnancy-induced dietary changes offer prophylactic protection against infection by pathogens, and in the absence of extensive non-Western clinical data inference must be relied upon. Consistent with the importance of protein intake during the second and third trimesters, in North America maternal vegetarianism is associated with low birth weight and premature delivery (see Sanders 1994). However, in Karachi, where public health measures are limited in

22. Additionally, there are questions of causality. Although an association between lack of pregnancy sickness and increased risk of first-trimester abortion is marshaled as evidence of pregnancy sickness's prophylactic effects (Weigel 1985, Flaxman and Sherman 2000), it is unclear whether the problem lies in a deficiency of pregnancy sickness or in an inadequate shift to anti-inflammatory activity, causing both termination and an absence of pregnancy sickness.

comparison, the converse is true, as meat eating during pregnancy is correlated with lower birth weight and intrauterine growth retardation (Fikree et al. 1994), outcomes associated with many meat-borne pathogens.

CRAVINGS FOR MEAT

Flaxman and Sherman (2000:127) report that, while meat is the principal target of aversions, it is not infrequently a target of cravings as well. If meat is so hazardous that a special avoidance-motivating mechanism has evolved, why would it ever be craved? Two mutually compatible explanations exist. First, as Flaxman and Sherman (2000) have argued, given the importance of adequate protein intake, meat may be craved in populations where it is the principal or even the sole source of protein. The goals of immunodefense and protein acquisition may be reflected in potentially antagonistic aversions and cravings—when alternative protein sources are available, aversions and cravings do not conflict, but when this is not the case, one may win out over the other depending on endogenous cues of need. However, this explanation alone is insufficient to account for all of the data. The need for protein is minimal during the first trimester and maximal during the third trimester, yet Flaxman and Sherman (2000:127) report that cravings for meat decline from the first to the third.

Food is a highly marked cultural domain, one in which symbolic factors independent of utility are likely to play a role (Rozin 1990). For example, Wijewardene, Fonseka, and Goonaratne (1994), investigating subjective experience in pregnant Sri Lankan women, found that 47% reported craving meat and that cravings across food categories were more than ten times as common among the poor than among the rich. Cravings were positively correlated with belief in spirit possession and marriage after a love affair. Sri Lankan spirit possession has been explained as a means of wish fulfillment, and Sri Lankan women who marry following a love affair are exhibiting considerable independence in a world of arranged marriages (Obeyesekere 1981). Accordingly, these two factors suggest that, at either a conscious or an unconscious level, these women recognize that they are in a position to manipulate their social environment. De Silva (1995) notes that pregnancy is the only time when a Sri Lankan woman can make demands about her food, and Obeyesekere (1985) concludes that Sri Lankan women actively exploit beliefs about pregnancy cravings so as to control those who are normally superior to them. He points out that women crave an array of foods, including those which are either expensive and rare or taboo to pregnant women, and he notes that meats fall into both categories (cf. Wijewardene, Fonseka, and Goonaratne 1994:97)—indeed, women may even "crave" nonfood items such as clothes and jewelry (1985:660).

The pattern described above is not unique to Sri Lanka. In diverse societies ethnographers record the belief that the mother and/or child will suffer adverse consequences if maternal cravings are not indulged.[23] In both Bengkulu (my data) and Maryland (Schwab and Axelson 1984), women explicitly state that pregnancy cravings constitute an opportunity for them to both exercise power over their husbands and obtain things which are normally unavailable or forbidden; the same appears to be true among the Bemba (Richards 1939), Fijians (Thompson 1940), Trukese (Fischer 1998 [1963]); Hokkien Taiwanese (Barnett 1971), Maasai (Spencer 1988), Javanese (Zeitlin et al. 1995), Morelos Mexicans (Ingham 1986), Bangladeshi (Rizvi 1980), and Saramaka (Price and Price 1991). Of all food categories, meat is the most universally valued (Stanford 1999). Hence, if any single category is likely to be the target of manipulative "cravings," it is meat (cf. Hanks 2000 [1963], Spencer 1988), and this is especially likely to be true when meat is scarce and difficult to obtain (cf. Demissie, Muroki, and Kogi-Makau 1998). Accordingly, the high frequency of cravings for meat may reveal more about the pervasiveness of gender inequality (cf. Stanford 1999) than it does about endogenous motivations produced by evolved mechanisms.

CORRELATIONS WITH PROGESTERONE LEVELS

If pregnancy sickness functions to compensate for the greater vulnerability entailed by reproductive immunosuppression, then, given that progesterone plays a key role in the process leading to reduced maternal immune activity, we should expect progesterone levels to be associated with the severity of gestational nausea and vomiting. However, empirical results are mixed on this count. Consistent with the prediction, in addition to being associated with more severe nausea and vomiting in comparison with singleton pregnancies, multiple gestations also involve higher levels of progesterone (Johnson, Abbas, and Nicolaides 1994) (and, correspondingly, are associated with greater risk of infection [Mascola, Ewert, and Eller 1994]). Recent work has focused on the finding that women experiencing pregnancy sickness exhibit gastric slow-wave dysrhythmias, and, consistent with the prophylaxis hypothesis, the same dysrhythmias can be artificially produced in nonpregnant women through administration of progesterone (alone or in combination with estradiol) in doses equal to those occurring during pregnancy (Walsh et al. 1996). However, in contrast to these confirmatory results, studies of the relationship between progesterone levels and severity of gestational nausea and vomiting report either no correlation (Masson, Anthony, and Chau 1985, O'Connor et al. 1998) or a *negative* correlation (Jarnfelt-Samsioe, Bremme, and Eneroth 1986). These findings constitute a significant challenge to the prophylaxis hypothesis. Either the hypothesis is wrong or the core hypothesis is correct but

23. For example, Bengkulu (my data), Javanese (Zeitlin et al. 1995), Maasai (Spencer 1988), Tlingit (Kan 1989), Thai (Hanks 2000 [1963]), East Toradja (Adriani and Kruyt 1996 [1951]), Ifugao (Barton 1998 [1955]); Morelos, Mexico (Ingham 1986), Hadiya, Ethiopia (Demissie, Muroki, and Kogi-Makau 1998), Bangladesh (Rizvi 1980), Saraguro (Finerman 1985), Tonga (Ostraff et al. 2000), the United States (Edwards, McSwain, and Haire 1954), Schwab and Axelson (1984).

the relationship between progesterone and reproductive immunosuppression is more complex than described herein. Given the significance of these findings for these questions, and given that only two of the three studies confirm one another, further investigation in this area is vital.

SEX DURING PREGNANCY

Reproductive immunosuppression presumably increases vulnerability not only to ingested pathogens but also to sexually transmitted diseases. Cross-species comparison among extant primates (Nunn, Gittleman, and Antonovics 2000) suggests that, given probable patterns of human mating, sexually transmitted diseases have likely been a source of selective pressure throughout our history. We might therefore expect that, if selection to avoid pathogens was strong enough to shape ingestion during pregnancy dramatically, it would also have been strong enough to favor a reduction in behaviors leading to sexual contact. Consistent with this prediction, pregnancy is associated with a decline in libido, coital frequency, and orgasm. However, in stark contrast to the chronology predicted by the prophylaxis hypothesis, this decline increases successively throughout pregnancy and is most marked during the third trimester (Kumar, Brant, and Robson 1981, Hart et al. 1991).[24]

Competing Explanations of Meat Avoidance

As noted earlier, Profet (1992:334) argues that selection favored gestational meat avoidance because the rapidly proliferating tissues of the embryo are highly vulnerable to mutagens and cooked meat contains carcinogenic substances. North American diet and lifestyle increase the risk of many cancers. In contrast, investigation of hundreds of deaths among the !Kung, Ache, and Hadza has not uncovered a single case of cancer despite extensive meat consumption in each of these societies (N. Blurton Jones, personal communication). Hunter-gatherers thus appear not to suffer the consequences that attend meat eating in North America, presumably in part because other dietary and lifestyle factors mitigate the carcinogenic effects of meat. This calls into question the antiquity of selection pressures that Profet associates with meat eating. Moreover, even in North America, clinical results suggest that meat consumption during pregnancy does not increase childhood cancer rates (see Sarasua and Savitz 1994).

Speth (1991) asserts that selective pressure for a meat-avoidance mechanism in pregnant women stems from the fact that, under conditions of restricted caloric intake, high levels of protein consumption are dangerous. Speth notes that (*a*) most of the calories derived from game are in the form of protein and (*b*) periodic plant scarcity drives foragers toward greater meat consumption, increasing the risk of excess protein intake by pregnant women. However, the constriction of available resources is significant in proportion to the caloric needs of the individual—the higher those needs, the more a scarcity of plant foods is likely to result in dangerous increases in the caloric contributions of protein. Aversions are concentrated during the first trimester, when energetic demands are low, and taper off over the second and third trimesters, as energetic demands climb—the opposite pattern from that which follows from Speth's hypothesis.

Other Perspectives on Pregnancy Sickness

Huxley (2000) proposes that first-trimester nausea and vomiting are an adaptation that reduces caloric intake in order to stimulate early placental growth. This hypothesis is inconsistent with the concentration of cravings for sweets, fruits, and other high-calorie foods during the same period. It is likely that enhanced placental growth is a compensatory response to environmentally determined scarcity rather than a goal in itself (cf. Kadyrov et al. 1998).

Deutsch (1994) hypothesizes that the evolution of concealed ovulation created selective pressure for a mechanism that reduces the frequency of sexual intercourse during pregnancy in order to protect the developing organism. This hypothesis is consistent with the risk of sexually transmitted diseases discussed above. However, despite the uniquely human nature of gestational vomiting, absence of estrus advertisement is found in many primates (Sillen-Tullberg and Møller 1993). Moreover, empirical studies indicate that pregnancy sickness does little to inhibit sexual behavior (Robson, Brant, and Kumar 1981). Deutsch also argues that pregnancy sickness signals others that the woman requires social support. However, a woman's fitness is ill served by a signal that unnecessarily inflicts costs on herself, her kin, and her mate, particularly given that pregnancy is accompanied by cost-free signals such as darkening of the areola and amenorrhea that are emically recognized under conditions approximating those of ancestral societies (Finerman 1985, Shostak 1981, Flaxman and Sherman 2000).

Knox, Kremer, and Pearce (1995), noting that women who suffer from allergies experience more gestational aversions than those who do not, argue that such aversions may serve to protect the developing organism from allergens. This proposal is supported by the observations that in utero exposure to food allergens does indeed increase the risk of developing an allergy (Frank et al. 1999) and the propensity to develop allergies has a heritable component (Rancé et al. 1999), suggesting that there would be benefits to scaling dietary allergen avoidance in proportion to the mother's own propensity to develop allergies. However, if the objective is to shelter the developing organism from dietary substances that may provoke an immune response, the timing of gestational

24. It is possible that the benefits of reassuring (or confusing) males as to paternity militate against a reduction in sexual behavior early in pregnancy, but we must be careful to avoid ad hoc rationalizations.

aversions should parallel the ontogeny of the fetal immune system, yet the exact opposite is true. Furthermore, the patterning of gestational aversions should closely match the allergenic potential of foods, and this does not occur with any regularity. While eggs, a common allergen, are indeed a sometime target of gestational aversions, peanuts, another common allergen (Rancé et al. 1999), are not notably avoided. Conversely, beef, which elicits allergic reactions at only one-fifth the frequency of peanuts (Fiocchi, Restani, and Riva 2000), is a principal target of aversions.

Profet's phytotoxin-avoidance explanation of pregnancy sickness is neither conclusively supported nor conclusively disproven by the available evidence. Flaxman and Sherman (2000) find only intermediate levels of aversions to vegetables, while my investigation reveals that vegetables have minimal salience as a target of pregnancy taboos. Given both the high concentration of secondary compounds in spices and the overt nature of the olfactory and gustatory cues thereof, Profet's hypothesis predicts strong aversion to spicy foods. Flaxman and Sherman (2000:127) find only low levels of aversions, however, and these are matched by equivalent levels of cravings. Similarly, I found that, although spicy foods were the only nonmeat category to be tabooed with any frequency, such taboos were far less common than meat taboos. Higher rates of aversions and lower rates of cravings are reported for the category "nonalcoholic beverages," including coffee and tea, but it is difficult to know what to make of these aversions given that the olfactory cues that likely play a role in them are not prominent in nature. In contrast to those mixed results, studies of gustation strongly support Profet's hypothesis, as sensitivity to bitterness, the hallmark of phytotoxicity, increases dramatically during pregnancy. Likewise, both the association between phytotoxin ingestion and pica across species and the demonstrable toxin-neutralizing effects of many clays and other inedible substances ingested during pregnancy support the assertion that phytotoxins may have contributed to the selective pressures shaping pregnancy sickness.

Following a line of argument that complements my position and that of Flaxman and Sherman, Haig (1993) suggests that pregnancy sickness may be a result of maternal-fetal conflict over the level of maternal investment. Because immunosuppression puts the mother at risk while immunoreactivity inflicts costs on the developing organism, it is conceivable that the mother and the developing organism differ with regard to the optimal level of immunosuppression. Maternal-fetal conflict is likely to be greatest early in a woman's reproductive career, as the value of the given developing organism is lower relative to future offspring than later in life. However, although some studies report the predicted inverse relationship between pregnancy sickness and maternal age (Weigel 1985, Klebanoff et al. 1985), many others find no association (see Wiegel et al. 2000). Given that the conditional fitness value of male and female offspring differs as a function of maternal rank (Trivers and Willard 1973), we might expect an association between pregnancy sickness and female fetal sex among high-status women and the opposite association among low-status women. A number of large studies of Western subjects have found that, among women suffering severe gestational vomiting (hyperemesis gravidarum) during the first (but not second or third) trimester, there is an increased frequency of female births (reviewed in James 2001; also see del Mar Melero-Montes and Jick 2001; for nonsignificant trends in subclinical cases, see Perry et al. 1999, Zhou, O'Brien, and Relyea 1999). However, these analyses do not include socioeconomic data. In a small study ($n = 120$), Vilming and Nesheim (2000) found the same association in Norwegian women, yet also found that immigrants from developing countries lacked any association between hyperemesis and fetal sex. This is intriguing given the lower status of immigrant women relative to Norwegians; however, the lack of a positive correlation with male fetal sex among immigrants is not consistent with the hypothesis.

As noted earlier, many observers have suggested that pregnancy sickness is an epiphenomenal consequence of pregnancy. It is difficult to reconcile this null hypothesis with the integrated nature of its various features. Both the timing and the content of aversions and cravings appear to complement patterns of immunological vulnerability in a fashion which, if not an adaptation, is a remarkable coincidence.

There are strong parallels between the nausea, vomiting, and cravings of pregnancy sickness and cyclical experiences of nonpregnant women, suggesting that pregnancy may amplify regular subjective changes. However, rather than indicating that pregnancy sickness is not an adaptation, these parallels are best interpreted as evidence that an equivalent relationship between immune status and diet pertains during the luteal phase of the menstrual cycle (see Fessler n.d.).

Animal Models of Pregnancy Sickness

Pregnant females of many species will be confronted by trade-offs between the nourishing qualities of particular food items and the hazards which consumption entails for the mother and the developing organism. It is likely that species having highly specialized diets will possess adaptations, such as elaborate physiological mechanisms or the ability to fast, which mitigate the dangers entailed by those diets. In contrast, omnivores, with their wider range of dietary choices, will be more likely to exhibit greater food selectivity during pregnancy (cf. Profet 1992: 355). Such a behavioral pattern is proximately produced through changes in the attractiveness of various food items, and changes in nausea susceptibility will likely be one avenue whereby this is achieved.

Rats, being highly omnivorous, present a model for testing the meat-avoidance hypothesis. Unfortunately, published results are insufficient in this regard. Dietary self-selection experiments using pregnant rats reveal that protein consumption is initially equivalent to the lowest preconception levels, followed by a progressive increase

over the course of gestation (Leshner, Siegel, and Collier 1972). This change results from changes in the subjective appeal of different macronutrients, since, if progressive increases in protein consumption are artificially foiled, an apparent aversion to available foods develops (Wilson 1987, 1997). While such investigations indicate the presence of a progressive increase in the need for protein over the course of pregnancy equivalent to that found in humans, because these experiments were conducted using nonmeat protein sources they tell us little about the adaptive changes in dietary selectivity which are at issue here. Similarly, although Czaja (1975) found distinct food rejection during the first half of pregnancy in rhesus macaques, despite his attempts to draw parallels with human pregnancy sickness it is difficult to interpret these results, since the animals were offered only uniform processed monkey chow. Similar problems plague studies of domestic dogs (reviewed in Flaxman and Sherman 2000).

Directions for Future Research

The following are empirically testable predictions that stem from the hypothesis that pregnancy sickness is an adaptation which partially compensates for reproductive immunosuppression:

1. Omnivorous meat-eating animals will avoid meat, particularly that which shows signs of spoilage, during early gestation.

2. Such animals will increase their consumption of substances which influence iron availability.

3. In human and nonhuman alike, the consumption of substances that inhibit iron absorption will co-occur with signs of infection risk (including both nausea and exposure to or consumption of meat).

4. In vivo studies of food-borne pathogens will reveal reduced rates of pathogen proliferation and tissue penetration when many of the targets of pica are introduced into the digestive tract.

5. The odor of meat in general and odors of spoilage in particular will be among the most aversive odors for women early in pregnancy.

6. Meat and spoilage odors will be more aversive to women in the first trimester than to women in later stages of pregnancy, nonpregnant women (particularly those not in the luteal phase of the menstrual cycle), and men.

7. The strength of both pregnancy sickness in general and aversions to meat in particular will be a function of both a woman's nutritional state and the availability of nonmeat sources of protein, essential fatty acids, iron, and calories.

8. Using olfactory, gustatory, or postingestive cues or some combination thereof, humans will be able to gauge the calcium content of foods; pregnant women will be particularly good at this task or particularly attracted to high-calcium substances or both.

9. Cravings for fruit in general and unripe fruit in particular will focus primarily on fruits high in vitamin C and low in harmful secondary compounds; conversely, fruits targeted by pregnancy taboos (e.g., papaya) will often be high in secondary compounds, particularly when unripe.

10. Cravings for meat during the first trimester will show a positive correlation with a woman's degree of subordination and powerlessness and/or with the cultural significance of meat.

11. Among highly empowered women, cravings for meat will be more common in late pregnancy than in early pregnancy.

In conclusion, while the meat-avoidance hypothesis parsimoniously accounts for a wide range of findings, many questions remain. Although the phytotoxin-avoidance hypothesis fares less well, it too accounts for some important features of pregnancy sickness. While it is clear that pregnant women are well advised to avoid pathogens, given that adaptationist explanations of pregnancy sickness are still in a preliminary phase of evaluation it would be premature to offer more specific clinical recommendations.

Appendix: Statistical Tests

TABLE A
Food Taboos

	Meat	Vegetables	Fruit	Dairy	Sweets	Spicy	Starches	Non-meat
General								
Observed k	5.767	0.867	1.167	0.367	0.867	5.200	–	1.693
Proportion of total	0.481	0.072	0.097	0.031	0.072	0.433	–	0.141
Probability ($k >=$ *Meat* k)	N.A.	0.000	0.000	0.000	0.000	0.426	–	0.003
$p <= .05$	N.A.	Yes	Yes	Yes	Yes	No	–	Yes
Specific								
Observed k	9.565	2.102	4.966	0.318	1.451	1.451	0.953	1.873
Proportion of total	0.797	0.175	0.414	0.026	0.121	0.121	0.079	0.156
Probability ($k >=$ *Meat* k)	N.A.	0.000	0.004	0.000	0.000	0.000	0.000	0.000
$p <= .05$	N.A.	Yes	Yes	Yes	Yes	Yes	Yes	Yes
All								
Observed k	10.362	2.361	4.775	0.372	1.884	3.291	0.899	2.264
Proportion of total	0.947	0.197	0.398	0.031	0.157	0.274	0.075	0.189
Probability ($k >=$ *Meat* k)	N.A.	0.000	0.000	0.000	0.000	0.000	0.000	0.000
$p <= .05$	N.A.	Yes	Yes	Yes	Yes	Yes	Yes	Yes

NOTE: "Observed k" reflects the number of successes of each specified food taboo for each category, with each success (k) calibrated to reflect the proportion of successes within each geographic region (i.e., if four out of five sampled taboos in a region are meat taboos, then 0.80 success is recorded for that region); the successes for all 12 regions are then summed and their values presented as "Observed k." "Proportion of total" is the proportion of successes contributed by each of the taboo types across all 12 geographic regions. The proportion of total for meat taboos is compared with the proportion of total for each of the other types of taboo, and, for each comparison, separate binomial tests are conducted regarding the probability that k for the given type of food taboo will be greater than or equal to k for meat. Each p value is the product of an exact binomial test against the null hypothesis that the proportion of successes for meat taboos will be equal to the proportion observed for the given nonmeat category.

Comments

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Fessler argues that pregnancy sickness and gestational food aversions protect the mother and her developing organism from infection in the period of immunosuppression necessary during early pregnancy. It is clear that nausea and/or vomiting are a fairly universal experience of pregnancy, indicating that these symptoms have a biological origin and may serve an adaptive function. It is argued that vomiting causes the expulsion of dangerous foods, while nausea encourages women to eliminate the offending food from their diet via learned food aversions.

The mechanism that links nausea to aversions is that of taste-aversion learning. This is a form of classical conditioning in which an organism comes to avoid a food (the conditioned stimulus) that has previously been paired with an unpleasant experience such as nausea (the unconditioned stimulus). Strong aversions can be acquired in a single trial, with long delays between exposure to the food and the unpleasant experience (Bernstein 1991). It is therefore possible that nausea experienced during pregnancy acts as an unconditioned stimulus, and through conditioning foods associated with nausea are experienced as noxious, leading to the strong aversions to specific foods (conditioned response) that are reported in pregnancy.

If a taste-aversion learning mechanism is responsible for the development of food aversions during pregnancy, then there should be a clear temporal association between the onset of nausea and the onset of food aversions. Fessler correctly mentions that pregnancy sickness and gestational food aversions are significantly concentrated in the first trimester. Our own data showed that 87% of women who reported gestational food aversions also experienced some nausea during their pregnancy. Furthermore, there was a close temporal association between the week of first onset of nausea and the week of first onset of food aversions. In 64% of the women who experienced nausea and food aversions during pregnancy, the first occurrence of nausea was reported in either the week preceding the first food aversion or the same week as the first food aversion (Bayley et al. n.d.). These findings are consistent with the taste-aversion learning mechanism that Fessler suggests.

The paper asserts that meat is the principal target of gestational food aversions. However, in some studies coffee and tea have been identified as the most frequent target of aversions, followed by meat/high-protein dishes

(Dickens and Trethowan 1971, Dye, Jones, and Hill 1998). According to Fessler, changes in olfactory sensitivity during pregnancy result in an increased ability to detect the odour of meat, which results in gestational food aversions that ensure avoidance and therefore limit the chance of infection. We acknowledge that aversions to meat may have served an adaptive function prior to hygienic procedures. However, it is also plausible that gestational food aversions may simply be an accidental by-product of pregnancy sickness. A strong odour may make a food a more salient target of a learned food aversion. Thus, any food with a strong odour may be a particularly potent target. Fessler argues that aversions to coffee are a result of "the misfiring of a mechanism" designed to detect meat odour. Although this is a possibility, it seems unlikely that an adaptive mechanism would produce "errors" in such a large proportion of the pregnant population. Protein sources are also significantly overrepresented as targets of aversions in cancer patients receiving chemotherapy treatment (Bernstein, Webster, and Webster 1982). Presumably a plausible anthropological explanation is that aversions to meat are more likely to occur in an immune-suppressed individual vulnerable to infection. However, there appears to be no obvious adaptive explanation for the large number of aversions to meat reported by members of the general population (Midkiff and Bernstein 1985). This is indicative that many food aversions may be accidental rather than purposeful by-products of sickness.

Research indicates that gestational food aversions disappear with parturition (Worthington-Roberts et al. 1989). This contrasts with the finding that in the normal population food aversions acquired in response to sickness last for years (Garb and Stunkard 1974). Fessler interprets this as evidence that pregnancy exerts a distinctive influence on food avoidance and that gestational aversions are an evolved adaptation. However, the time-limited nature of gestational aversions can be explained in terms of attribution. The discrepancy between the persistence of food aversions in a normal and a pregnant population may reflect the variation in attributions for the cause of illness made by the two groups. Normal individuals typically attribute their illness to the consumption of the food that became aversive (Logue, Ophir, and Strauss 1981). In contrast, pregnant women should relate their sickness to their pregnancy. Therefore, pregnant women may be more willing to consume a previously aversive item outside pregnancy.

In summary, pregnancy sickness and food aversions are closely related, and classical conditioning is a likely candidate for the mechanism that links these events. Food-aversion learning may have initially developed as an adaptive mechanism. However, during pregnancy the development of food aversions may be nothing more than the accidental by-product of pregnancy sickness, which in certain cases may even lead to the avoidance of foods which do not represent a threat to either the mother or her developing embryo (e.g., fruit, chocolate).

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Fessler is to be commended for presenting an innovative view of a subject relatively neglected by both anthropologists conducting cross-cultural research and those conducting research on women's issues. Nevertheless, the cross-cultural study of practices related to pregnancy dates back almost 50 years, to the groundbreaking thesis of Barbara Ayres (1954) and her later publication based on this research (1967). Although Ayres applies the cross-cultural method to a smaller sample of societies than Fessler and tests entirely different hypotheses, her important early study should have been cited. A product of its own time, Ayres's study focuses on psychological issues. Also a product of its time, Fessler's article mentions no psychological explanations of "pregnancy sickness" (not even the age-old interpretation that such symptoms may be related to a mother's ambivalence concerning her pregnancy) but focuses on adaptation and biological issues.

There are a number of conventions regarding cross-cultural research that Fessler has failed to observe. The reader should be provided with a list of the societies in the sample; tables 1 and 2 should have indicated not just the presence of the variables but reports of their absence, and it would have been helpful to have the ethnographic bibliography of the sample of societies separated from the general bibliography. "Galton's Problem" has haunted cross-cultural research since its inception, and it is not solved by simply choosing societies from an assortment of geographic areas. Fessler might have consulted the various published samples carefully drawn to a variety of specifications by, for example, Murdock and his coworkers. (For a published cross-cultural sample of societies with sources particularly rich in information concerning women, see Brown, Sarah, and Pilato 1994.) Finally, although the Human Relations Area Files are an invaluable tool for cross-cultural research, they should never be the only source of information (for a variety of reasons) but should be used in combination with the resources of an ethnographic library.

Fessler notes that although he has identified it as an adaptive practice with implications for survival, pregnancy sickness is totally absent in some societies. The explanations provided are not particularly convincing, and this weakens his presentation considerably. He ascribes "centrality" to meat in the diet of hunter-gatherers in general, apparently unaware of the overwhelming quantity of gathered vegetable foods consumed by hunter-gatherers living in low latitudes (Lee 1968). However, the question does remain how northern hunter-gatherers like the traditional Inuit coped with a diet that allowed for almost no substitution for meat. Fessler's suggestion that dairy products could be substituted for meat fails to take account of the fact that in a vast area of the world people do not consume dairy products. It is unfortunate that he does not explain his labeling of a variety of foods such as fish, seafood, eggs, and poultry

as "meat." Viewing these foods as interchangeable in relation to his hypotheses should have been given justification. Fessler's mentioning of cultural factors such as gender inequality suggests interesting areas for further inquiry. Indeed, if the definition of an engaging paper is that it raises new and exciting questions, this one certainly qualifies.

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On June 27, 2001, I participated in a workshop entitled "Foodborne Infections in Pregnancy" at the annual meeting of the Teratology Society. Workshop speakers made two messages clear: (1) certain food-borne pathogens, while generally not dangerous to immunocompetent, nonpregnant adults, can be dangerous to pregnant women and their embryos/fetuses, and (2) meats are the foods that are most likely to be sources of these pathogens. These facts raise the question whether there are mechanisms associated with pregnancy that might help mitigate the increased danger of food-borne infection. In his comprehensive analysis, Fessler concludes that available evidence is consistent with the hypothesis that "pregnancy sickness"—the nausea, vomiting, and food aversions and cravings that occur in the early part of about two-thirds of all pregnancies—provides protection from infections, especially infections caused by meat-borne pathogens.

Fessler's synthesis is an important contribution for three reasons. First, he considers several alternative hypotheses about the possible function of pregnancy sickness and, in doing so, independently replicates research that Paul Sherman and I conducted. We found strong evidence to support the hypothesis that pregnancy sickness protects pregnant women and their embryos/fetuses from food-borne pathogens and toxins (Flaxman and Sherman 2000). Fessler cites nearly 350 works, 87% of which were not cited in our analysis, and there is a striking correspondence between his conclusions and our own. I hereafter refer to his conclusions and ours as the "prophylaxis hypothesis." Given (*a*) the desire of most women for their pregnancies to have positive outcomes, (*b*) the worldwide prevalence of pregnancy sickness, and (*c*) the fact that researchers have typically found pregnancy sickness difficult to explain, replication of findings about the prophylaxis hypothesis is very important.

Second, Fessler's extensive cross-cultural analysis of pregnancy sickness and taboos adds substantially to the information available on patterns of diet in pregnancy in non-Western cultures. This is crucial for testing any functional hypothesis about the relationship between pregnancy and food, and it should be a useful resource.

Third, Fessler provides data that address several questions about the prophylaxis hypothesis that arose following the publication of our results (Flaxman and Sherman 2000):

1. Does pregnancy sickness occur at the time when pregnant women and embryos are most sensitive to pathogens? Fessler found evidence that *both* maternal and embryonic/fetal vulnerability to pathogens are greatest when pregnancy sickness is most common (in the first trimester). In addition, gestational infection by several common food-borne pathogens is most dangerous in early pregnancy (table 3).

2. Have members of our species been exposed to contaminated meats frequently enough over our evolutionary history to have evolved aversions to them? As Fessler shows, all the available evidence suggests that meat and associated pathogens have likely been significant parts of humans' diets since our species's inception.

3. Nausea and vomiting are associated with reduced chances of miscarriage, but are the symptoms *effects* rather than causes? Investigators (e.g., Stein and Susser 1991, Haig 1993) have hypothesized that—contrary to the prophylaxis hypothesis—pregnancy sickness might be a nonfunctional by-product of the physiological changes (e.g., hormones) that accompany pregnancies that do not end in miscarriage. There are four problems with this "by-product hypothesis." First, it makes the critical prediction that pregnancy sickness should occur in nearly every pregnancy that does not end in miscarriage. However, many women who do not experience nausea and vomiting carry their pregnancies to term (e.g., data in Weigel and Weigel 1989). While this finding has been carefully quantified in Western populations only, the by-product hypothesis predicts that pregnancy sickness should occur whenever a woman's physiological state is adequate for the maintenance of a pregnancy, irrespective of her social, economic, or nutritional status. Second, some investigators have suggested that correlations between pregnancy sickness and hormone levels support the by-product hypothesis. However, pregnancy sickness, like any biological phenomenon, must have an underlying physiological mechanism and, as Fessler's analysis suggests, *should* be related to hormonal profiles under the prophylaxis hypothesis because hormones modulate maternal immunosuppression Third, Fessler describes enormous variability in the incidence of pregnancy sickness across populations. This is inexplicable under the by-product hypothesis unless the same magnitude of between-population variation in the ability of women to carry pregnancies is demonstrated. Fourth, the by-product hypothesis does not explain the clear and consistent patterns of gestational food aversions, cravings, and taboos that Fessler describes.

I agree with Fessler that it would be premature to offer specific clinical recommendations based on the prophylaxis hypothesis. However, it is important to note that extreme levels of nausea and vomiting (e.g., hyperemesis gravidarum) are *not* adaptive and obviously require treatment. Also, the prophylaxis hypothesis does *not* imply that pregnant women should avoid meats altogether. Smith (1999) provides an extensive list of the ways in which pregnant women can avoid the dangers of food-borne infections.

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The clinical observation that pregnant women who experience morning sickness have a greater chance of a positive outcome of the pregnancy, especially when sickness is combined with vomiting, gives emphasis to Fessler's hypothesis that pregnancy sickness may be a protective mechanism based on selection during evolution. He presents a very detailed overview of the factors in favour of and against his hypothesis. As a gynaecologist-obstetrician working in a Western country, I am not acquainted with the wide range of human societies that Fessler includes in his study, but I believe that the following points should be added to his results: In my opinion, the genesis of pregnancy sickness is multifactorial and the relevance of these symptoms in the psychosocial context of the pregnant woman should receive a little more attention.

For vomiting and nausea, a variety of predisposing factors such as stress, inadequate information about pregnancy or delivery, and problems in the marital relationship are well documented (Katon et al. 1980–81), Klebanoff et al. 1985, Leeners, Sauer, and Rath 2000). In addition, feelings of ambivalence about the pregnancy and unconscious rejection of the child are known to influence the intensity of pregnancy sickness (Ringler and Krizmanits 1983; Tylden 1991).

The incidence of nausea and vomiting in pregnancy varies with the psychosocial environment. The incidence was reduced, for example, during World Wars I and II and in the first postwar years (Jarnfelt-Samisoe, Samisoe, and Velindes 1983). Given that storage of meat was probably inadequate during those years, Fessler's hypothesis would predict an increase in incidence. Because incidences of psychosomatic diseases are often reduced in times characterized by life-threatening events, these changes suggest an important role for psychosomatic factors in the causation of pregnancy sickness. Interestingly, pregnancy sickness often occurs when pregnancy is confirmed by tests in the sixth to eighth weeks of gestation—that is, when the woman has proof that she is pregnant (Roemer 1967). (Obviously, this fact is relevant only for countries where pregnancies are confirmed by such a test.) In addition, the intensity of symptoms often decreases when the pregnant woman is separated from her family, for example, for a hospitalization, and increases when she returns to it (Fuchs et al. 1980).

Nausea, vomiting, and food aversions occur in a period when there is no other clear sign of pregnancy. Fessler mentions that women put themselves in a position to manipulate their social environment by developing pregnancy sickness or food aversions/cravings. The symptom "morning sickness" may lead to an increase of social support, permitting women to concentrate on their pregnancy and reduce their activity not only at home but also in the workplace. El-Mallakh, Liebowitz, and Hale (1990) found that nausea and vomiting may be particularly intense when the husband is present—that is, it may play an important role in getting support from the partner.

Only humans and a few primates, in contrast to most mammals, engage in sexual intercourse during pregnancy (Solberg, Butler, and Wagner 1973). Therefore, the significance of sexual activity for stabilizing a partnership seems to play a more important role than the protection of pregnant women against sexually transmitted diseases mentioned by Fessler as a hypothetical selection mechanism throughout our history. In addition, Robertson (1946) found that the likelihood of nausea and vomiting diminishes with the ability of a couple to maintain its accustomed frequency of sexual activity during pregnancy.

In conclusion, Fessler has written a fascinating article on pregnancy sickness and food aversions from an evolutionary perspective, but I believe that psychosomatic aspects should be taken into account. While psychosomatic factors are certainly not the only reasons for the development of pregnancy sickness, food aversions, or cravings, they may play an additional important role in the intensity of symptoms.

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Fessler's paper elegantly reviews the fascinating proposal that pregnancy sickness is a mechanism through which nature protects both mothers and embryos. The available data strongly support the hypothesis that pregnancy sickness serves an adaptive, prophylactic function. The author's perspective is well discussed and referenced. I would like to comment on the concept of reproductive immunosuppression from a different angle. The participation of reproductive cyclicity in the immune-neuroendocrine network is a way in which nature protects the time of ovulation and overcomes the stress of reproduction. The gonadal status of female mammals plays a crucial role in preventing an exaggerated immune response during particular phases of the ovulatory cycle and in early pregnancy (Nappi, Bonneau, and Rivest 1997). Although the exact modes and sites of action employed by gonadal steroids to exert immunoregulatory effects have not been clearly established, many experimental studies have demonstrated that the integrity of the immune response depends on the fine-tuning performed by these steroids on the multiple components of the immune system (Grossman 1984). In this context, the neuroendocrine system plays a pivotal role at both the central and the peripheral level. Indeed, immune challenges convey information to the brain to allow coordination of the metabolic, behavioral, and endocrine changes necessary for adaptation. Apart from the action of circulating cytokines, brain-derived cytokines may regulate specific challenges to the central nervous system directly or in response to input from immune signals

in systemic circulation. Increased production of cytokines is an essential feature of the early stages of immune activation and has the ability to activate the hypothalamus-pituitary-adrenal (HPA) axis by stimulating neuroendocrine corticotropin-releasing hormone (CRH) neurons (Rivest 1995). HPA function is dynamic over the gonadal cycle, and plasma fluctuations of gonadal steroids inflect the HPA-axis's sensitivity to stress by inducing neuronal changes capable of interfering with the activity of neuroendocrine CRF (Nappi and Rivest 1995). In addition, hormonal fluctuations are responsible for cyclical rhythms in neuropeptides, neurotransmitters, and neuromodulators orchestrating adaptive behaviors such as food intake, emotional reactions, and cognitive adjustments. In particular, appetite control mechanisms and food cravings throughout the menstrual cycle, with a luteal-phase peak in energy intake, offer survival advantages related to the genetic predisposition for storage of calories as fat in order to sustain gestation (Wade, Schneider, and Li 1996). Working together, these hormone-dependent events allow the female organism to perform reproductive tasks. At the same time, neuropeptides, growth factors, and cytokines are expressed in peripheral organs and tissues, where they interact with afferent endocrine messages to modulate a great variety of biological functions that need to be finely controlled to ensure adaptation to pregnancy (Reis et al. 2000). Avoiding specific exogenous insults during the period of maximal vulnerability to infections can then be crucial to implantation, placental development, regulation of uterine blood flow, and fetal growth and maturation.

In conclusion, the capacity of female animals to modulate the intensity with which adaptive circuitries are activated during immunogenic processes is likely to be an elegant sexual dimorphism participating in the adjustment of responses to the physiological demands of the menstrual cycle and reproductive performance. Unexplained infertility, recurrent abortion, or uteroplacental insufficiency may be the result of triggering factors that overcome the ability of the female immune-neuroendocrine system to adapt during emergencies. Be that as it may, Fessler's evolutionary perspective on pregnancy sickness is highly plausible.

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The ethnographic data examined by Fessler (and Flaxman and Sherman [2000]) validate the salience of early-pregnancy meat aversions and sweet cravings across cultures. This is a productive direction to pursue for understanding the etiology of pregnancy sickness, as I suggested in a review of Profet's book (O'Connor 1999). Fessler examines how specific cravings and aversions might interact with immunological, hormonal, nutritional, and metabolic pathways and together produce the suite of purportedly adaptive symptoms of nausea, vomiting, cravings, and aversions.

Fessler's hypothesis is complex, because specific cravings and aversions do not fit into a neat explanatory package. But, as he acknowledges, neither do they fit easily into a complex package. There are several aversions and cravings that conflict with each other or with some aspect of the theory; this highlights that we know rather little about the biology we are trying to explain. In recognition of this, Fessler lists specific and well-targeted predictions.

There are now several well-developed theories of the evolution of pregnancy sickness: Profet's phytotoxin theory (1992), Haig (1993) and Forbes's (n.d.) maternal-fetal conflict theory, and Flaxman and Sherman's (2000) and Fessler's prophylaxis theory. To evaluate these theories, outcome measures of health and survival coupled with systematically collected biological and ethnographic data are necessary. Additionally, adaptationist theories should accommodate the well-established biological features of pregnancy sickness. Two of the strongest and most consistent findings are that the risk of early spontaneous abortion is lower in women with pregnancy sickness (e.g., Weigel and Weigel 1989) and that higher levels of human chorionic gonadotropin (hCG) are associated with nausea and vomiting (e.g., O'Connor et al. 1998). Fessler does not incorporate either of these.

A significant proportion of early pregnancy loss, ranging from 23% to 79% during weeks 6–12 (the peak timing of pregnancy sickness), depending on the stage of gestation and the age of the mother (Boué, Boué, and Gropp 1985), is a result of genetic abnormalities. Is this genetic damage a result of phytotoxins and pathogens that women with pregnancy sickness avoid? Or are phytotoxins and pathogens causing nongenetic losses, with this type of loss making up a significant (but as yet unobserved) proportion of losses? Or are losses a result of genetic or other events occurring in gametes prior to fertilization? Given the strength and pervasiveness of the link between pregnancy sickness and risk of fetal loss, this is an area to examine in more detail.

Seven of ten studies support a positive association between levels of hCG and pregnancy sickness (e.g., in addition to the studies listed by Fessler, positive associations are reported in Mori et al. 1988, Goodwin et al. 1992, Schoeneck 1942, and Kauppila et al. 1984). Of five studies examining progesterone, four find no association with pregnancy sickness (references cited by Fessler plus Soules et al. 1980, Kauppila et al. 1984). Fessler's theory is thus undermined more than he recognizes by the hormonal data. In contrast, the maternal-fetal conflict theory includes specific evolutionary mechanisms incorporating hCG into an adaptive complex.

There are other hormonal details that are problematic for Fessler. He says that progesterone peaks in the first trimester, and it does exhibit a minor peak then, but it continues to increase across pregnancy and reaches its highest levels just before parturition (Johnson and Everitt 1995). Another confounding issue is population variation in progesterone levels. Progesterone is surprisingly low

in non-Western populations (e.g., Ellison et al. 1993, O'Connor et al. n.d.), and it is these populations that have high exposure to pathogens (as well as potential dietary stress). Thus, progesterone is lowest in populations where lower levels might be of some benefit if in fact it served an immunosuppressive function. But the prevalence of nausea and vomiting (and perhaps cravings and aversions) is also significantly lower in these populations (cf. Walker et al. 1985, Gadsby, Barnie-Adshead, and Jagger 1993). These observations might be explained by the need to balance energy or nutrient reserves against the need for pathogen protection. However, as Fessler notes, this would seem to weaken the case for his complex system of pathogen protection, and, moreover, energy and nutrient needs are minimal in the first trimester of human pregnancy. I think that most of Fessler's theory on cravings and aversions remains tenable without the progesterone/immunosuppression link, but it would certainly benefit from including a link with hCG and more specific links with early pregnancy loss.

The maternal-fetal conflict theory, though fitting nicely with the hormonal and fetal-loss data, fails to provide an explanation for meat aversions and sweet cravings. Research on pregnancy cravings and aversions is still in an early stage. Most of the research cited by Fessler (and Flaxman and Sherman) is based on anecdotal accounts in the ethnographic literature. Prospective collection and quantitative analysis of data are rare. This sort of work might help to resolve some of the conflicting information Fessler presents on cravings and aversions. He suggests that those that do not fit neatly into his scenario are "misfirings" of the adaptive apparatus. An alternative interpretation is that the mechanism is more general (e.g., hCG contributes to nausea and vomiting), and sensitive to "learned" (in the biological sense) aversions and cravings.

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The argument that meat-borne pathogens are a more important selective force than plant-borne teratogens exposes certain oversimplifications in Profet's models and seems to fit the data better, but things are likely to be more complex. First, we should not interpret immune suppression among newly pregnant women only as evidence of a mechanism to protect the implanted blastocyst and embryo from immunological attack. Immune suppression might also function as a nutrient-sparing adaptation because an up-regulated immune response draws on energy stores and competes for available protein and micronutrients. Second, marginally nourished people are immune-suppressed, more susceptible to infection by pathogens and parasites and once infected subject to illnesses of longer duration and greater severity. Under conditions where conception increases nutritional stress of the mother, indicators of immune suppression might result from pathology rather than functional adaptation. Third, improved nutrition generally allows a better response to disease and among the undernourished always results in an increase in symptomatology. For example, supplementation of children with both zinc and iron is consistently associated with increases in upper respiratory infection (responses to Oppenheimer 2001). Thus, a very careful approach is needed to identify the direction of causation for associations between meat-eating, pregnancy, and immune function. Fourth, micronutrient needs may have shaped the evolution of pregnancy sickness in ways Fessler has yet to consider. Pregnant women may become more averse to bitter tastes to reduce intake of the tannins that inhibit assimilation of iron, zinc, and calcium. Similarly, pica and geophagy can be interpreted as mechanisms for increasing intakes of key nutrients rather than for absorbing pathogens in the gut (Wiley and Katz 1998).

We should also remain skeptical of the notion that the nutritional and developmental costs of meat avoidance during pregnancy are phenotypically tolerable. Trace elements like iron, zinc, and iodine, which occur in low concentrations or with poor bioavailability in most nonanimal foods but are highly bioavailable from meat, are critical determinants of uterine and postneonatal growth and cognitive development (Commission on the Nutrition Challenges of the 21st Century 2000). For example, iron deficiency is associated with preterm birth, low birth weight, perinatal mortality, infant and young child mortality, developmental delays, lowered IQ, poor school achievement, decreased play activity in children, lethargy and fatigue, decreased work productivity in repetitive tasks, reduced physical work capacity, and maternal mortality. There is a tight correlation between measures of maternal, fetal, neonatal, and child iron status (Allen 2001). On this evidence, the benefits of reduced iron status during pregnancy in protecting against infectious disease would have to rise steeply to counterbalance such profound fitness costs.

If indirect evidence that pregnancy sickness protects against meat-borne pathogens (or food-borne teratogens) is strong, how can we test the prediction that the associated fitness benefits outweigh the costs under most conditions? A rare cohort study in a Turkana sample (Pike 1999), which ostensibly demonstrates significant costs of nausea and vomiting during pregnancy (NVP), in fact highlights the difficulties of doing this. First, although presence of NVP is reported to increase fetal, perinatal, and neonatal mortality more than twofold, it is not possible to assess the biological significance of this apparent cost or estimate the magnitude of the putative fitness differential because the actual mortality rates are not reported. Second, although anthropometric indications of reduced nutritional status among women with NVP suggest that there may be nutritional costs, one cannot rule out alternative interpretations that NVP is itself associated with poorer diets prior to pregnancy, reduced quality and quantity of food in the household during the pregnancy observed, maternal depletion (as a function of age and parity), or increased exposure to dis-

ease or susceptibility to infection because actual food consumption by NVP and non-NVP subjects is not assessed. Third, the results would be consistent with the antiteratogenic hypothesis if by some mechanism mothers entering pregnancy in poor condition were more likely to experience NVP (leading to better outcomes than if they had not). Retrospective case-control designs lack power, and a better test of pregnancy sickness or NVP as an adaptation to avoid pathogens or teratogens is to compare the outcomes and NVP symptoms of women exposed to dietary pathogens or teratogens, all other things being equal.

Fessler's efforts are ambitious and result in a workable conceptual framework, but are there any practical implications? Most nutritionists would be alarmed by the suggestion that food restriction during pregnancy might be a good thing. In many developing countries the impact of supplementation interventions aimed at reducing poor pregnancy outcomes has been less than predicted, in part because of a lack of compliance motivated by fear of the labor complications thought to be associated with delivering a large baby (e.g, Gittelsohn, Thapa, and Landman 1997). Such fears are usually shared by traditional birth attendants and instituted in pregnancy food taboos, and a behavioral response dubbed "eating down during pregnancy" (Nichter and Nichter 1983) often frustrates interventions to improve maternal nutrition during pregnancy. If it is true that meat-based dishes elicit pregnancy sickness and are associated with increased illness, then these fears will be reinforced by such interventions.

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"Morning sickness," the common term for gestational nausea and vomiting, is a complete misnomer. Symptoms occur throughout the waking hours, not just in the morning, and whereas "sickness" implies pathology the majority of women experience the symptoms and bear healthy babies. Indeed, women who experience symptoms within the normal range (i.e., excluding hyperemesis gravidarum) are significantly less likely to miscarry than women who do not (Flaxman and Sherman 2000). The medical acronym NVP ("nausea and vomiting of pregnancy") is more objective, descriptive, and appropriate.

Why does NVP occur? Like other questions in biology, this one can be analyzed from proximate and ultimate perspectives. Answers to questions about *how* NVP symptoms are brought about (their underlying mechanisms) and *why* they occur (their reproductive consequences) are complementary, not mutually exclusive. Complete understanding requires explanations at both levels of analysis (Sherman 1988).

Indeed, Fessler addresses both issues. His descriptions of the intricate hormonal mechanisms underlying NVP, especially involving human chorionic gonadotropin, the fact that these mechanisms trigger immediate expulsion and subsequent avoidance of food rather than other conceivable symptoms (e.g., headache, back pain, etc.), and their coincidence with the most sensitive period of embryogenesis (weeks 5–18) provide compelling evidence of functional design. Gestational nausea and vomiting are uniquely associated with specific food aversions that appear spontaneously in the first trimester and usually disappear by the third; in contrast, nausea triggered by food poisoning outside pregnancy can create aversions that last a lifetime.

The evidence that Fessler marshals supports the meat-avoidance hypothesis—that NVP protects mothers and embryos from parasites and pathogens in meat products. Pregnant women are especially susceptible to diseases because from conception on their immune response is depressed to avoid rejecting an embryo which is like a foreign tissue because half its genes are paternally derived (Haig 1993). Meat products are particularly dangerous because of the pathogens they may contain (see Fessler's table 3). During gestation (1) meats are the most common targets of women's aversions (fig. 1) and cultural food taboos (tables 1 and 2), (2) smells and tastes of meats are particularly aversive, and (3) cravings are primarily for fruits, sweets, and dairy products.

Support for the meat-avoidance hypothesis does not mean that the Hook-Profet phytotoxin-avoidance hypothesis should be discarded, as I think Fessler would agree. Every day we ingest plants' defensive chemicals, and we enjoy spices, chocolate, and coffee because they contain these chemicals. However, plant secondary compounds can sometimes be teratogens and abortifacients (Sherman and Billing 1999). NVP may protect embryos by reducing maternal ingestion of these toxins.

Gestational food aversions support both hypotheses. Among various food categories that Flaxman and Sherman (2000) analyzed (Fessler's fig. 1, "nonalcoholic (caffeinated) beverages," "vegetables," and "ethnic, strong, and spicy foods" all include items that contain phytotoxins. Combined into a single supercategory, they account for as many per capita aversions as meat products. Also consistent with phytotoxin avoidance, vegetables and nonalcoholic beverages are the second- and third-most-aversive food categories. and these aversions decline as pregnancy progresses.

When we and Fessler began our inquiries, a key question was whether NVP was a cause or a consequence of a viable pregnancy—in other words, whether it was functional or an uncomfortable, superfluous side effect of a hormonally mediated tug-of-war between vigorous embryos and mothers over maternal resources. Fessler's comprehensive, balanced analysis lends further support to the functional interpretation, which I also favor (see Flaxman and Sherman 2000; Sherman and Flaxman 2001, n.d.).

Fessler touches more lightly on another important question: Is NVP merely an evolutionary anachronism today? I suspect that it is not. After all, food-borne pathogens and phytotoxins are still major health and economic concerns, and the relationship between NVP and

reduced miscarriages has been documented many times in recent years.

This does not imply that pregnant women should always avoid meats and vegetables. Nowadays food-borne pathogens are reduced by packaging and preservatives, cooking and irradiation, and refrigeration and freezing; most commercial vegetables have minimal protective chemicals because of selection for bland taste. Conversely, however, there is no evidence that women will improve their pregnancy outcomes by forcing themselves to eat aversive foods. Doing so might even have the opposite effect if it exposed them to harmful pathogens or phytotoxins.

For pregnant women the take-home message is clear and positive: normal levels of gestational nausea and vomiting will not hurt the embryo. Indeed, there are good evolutionary reasons for emetic responses to smells and tastes of meat products and bitter-tasting plants during gestation. NVP is neither a disease ("a departure from health") nor an unselected side effect of mother–offspring conflict. Rather, it serves a useful, prophylactic function. Henceforth NVP should be recognized and treated for what it is: an adaptation.

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Fessler has written an excellent comprehensive review of current theories and understanding surrounding early pregnancy sickness and its association with food aversions and cravings. Building on Flaxman and Sherman's (2000) earlier work, he provides clear evidence that meat is a principal target of food aversion during early pregnancy in both Westernized and non-Westernized women and that meat taboos are ubiquitous in traditional societies across the globe. He argues that pregnancy sickness evolved as an adaptive mechanism that serves to protect women from meat-borne pathogens during reproductive immunosuppression. This is an intriguing hypothesis but one that is difficult to prove for several reasons. According to Smith (1999), pregnant women are no more vulnerable to infection by most food-borne pathogens than the general population, although specific pathogens (e.g., Hepatitis E virus, *Coxiella burnetii*, *Listeria monocytogenes*, and *Toxoplasma gondii*) can produce serious illness in the mother and/or fetus. These organisms are not unique to meat but can be present in a wide range of foods including vegetables and dairy products, the latter being a common target of cravings in pregnancy. The parasite *T. gondii* can be transmitted to humans in food, water, or soil, suggesting multiple sources of exposure (Smith 1999). Thus, pregnant women are exposed to pathogens from a variety of sources, not limited to meat.

It is striking that no comprehensive theory on the cause or purpose of pregnancy sickness has emerged in spite of the many studies that have been devoted to this topic. This may be because of the lack of uniform measures of pregnancy sickness. Some researchers use the presence or absence of nausea and/or nausea and vomiting as the sole determinant of pregnancy sickness (e.g., Tierson, Olsen, and Hook 1986) while others consider nausea and vomiting as separate symptoms, taking into account symptom severity (Whitehead, Holden, and Andrews 1992). Simply tabulating the presence or absence of symptoms leads to the assumption that the origins of nausea and vomiting in pregnancy are the same and that vomiting is a result of nausea. However, prospective studies indicate that women can experience varying degrees of nausea and/or vomiting and that severity on one axis does not necessarily indicate severity on the other (Whitehead, Holden, and Andrews 1992). It may be that nausea and vomiting serve different roles in early pregnancy. Establishing reliable measures of the incidence and severity of pregnancy sickness is necessary for determining pregnancy sickness cause and effect. Likewise, establishing a basic understanding of cravings and aversions in pregnancy is hampered by the lack of uniform definitions and quantitative measures. Even if standardized definitions are developed, the self-report nature of cravings and aversions limits the ability to determine the uniformity of reporting across individuals.

Anthropological studies have placed a great deal of emphasis on pregnancy cravings and aversions as a window into the dietary needs of the pregnant woman. Despite the logic of this argument, research has not convincingly demonstrated that cravings serve to identify and redress bodily needs. For example, pregnant women with gestational diabetes who metabolize simple sugars poorly show increased preferences for sweet carbohydrates and crave fruit and fruit juices as avidly as pregnant women without this disease (Tepper and Seldner 1999). As Fessler points out, pregnancy places multiple nutritional demands on women that are often conflicting. Nevertheless, the hypothesis that aversions and cravings discretely guide food choices during pregnancy assumes an extraordinary level of precision in the nutrient regulatory system, precision that has yet to be demonstrated in humans and seems unlikely to develop as a result of physiologic changes associated with pregnancy. At least in Westernized societies, food cravings appear to be more closely associated with mood than with food deprivation (Hill, Weaver, and Blundell 1991). Thus, it is possible that mood changes during pregnancy may be more predictive of food cravings and aversions than pregnancy sickness.

To understand appetitive changes in pregnancy, perhaps we need to abandon the traditional view that the collection of symptoms commonly called "pregnancy sickness" is unique to pregnancy and begin to explore commonalities between pregnancy sickness and other conditions or diseases which share either symptoms or physiological changes. For example, it would be important to determine whether other immunosuppressed individuals such as organ-transplant recipients and those

who are HIV-positive experience symptoms similar to pregnancy sickness as well as food cravings and aversions. Similarly, the experience of nausea, vomiting, cravings, and aversions should be examined in women receiving fertility drugs. For the present, Fessler's provocative article challenges us to consider a broad range of alternatives with regard to the origins of this condition.

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Fessler has weighed in on the debate over the functional significance of pregnancy sickness, food aversions, taboos, and cravings during pregnancy. In challenging Profet's popularized hypothesis that pregnancy sickness functions to reduce exposure of the embryo to toxins, Fessler argues that it is not so much plant-based toxins as meat-borne pathogens that threaten its survival. He advances the hypothesis that not only pregnancy sickness but these other common dietary modifications of pregnancy collectively serve to reduce meat-borne infection during early pregnancy. Infection can have severe consequences for the embryo, but at the same time the mother's immune system must be restrained so as to avoid the destruction of embryonic cells, which display foreign antigens. This poses a dilemma for the mother and embryo which may be resolved by a host of physiological and behavioral adaptations.

Fessler's model is predicated on complex, finely tuned relationships between maternal behavior and the physiology of pregnancy, particularly as they affect iron status during the first trimester. However, it is the model's very complexity that worries me, because (1) behavior is highly susceptible to the exigencies of the cultural and natural environment in which a pregnant woman finds herself and (2) it is not clear what the *net* effects of the various behavioral and physiological changes are (especially given the problems associated with 1). For example, the embryo has limited immunological defenses of its own and needs protection from the mother's and from infections derived from maternal meat consumption. It needs iron and calcium for its development, but pathogens also require iron, and calcium inhibits iron absorption. So, women are nauseated by meat and hence avoid it so as to reduce infection, but then they crave fruit, which contains Vitamin C, which enhances non-heme (plant) iron absorption and offsets some of the dampening of maternal immune function. The result is that women should desire other sources of nonmeat iron and the means to enhance its absorption (Vitamin C from fruit) and at the same time crave calcium-rich foods and engage in pica (items that provide no nutrients), which, however, undermine iron absorption. A delicate calibration of iron uptake is supposedly achieved through these dietary manipulations such that the embryo's needs are met while those of meat-borne pathogens are thwarted.

Fessler's argument is that the net effect of these simultaneous forces enhances birth outcome. There are so many countervailing forces at work here that I am unconvinced that their net effect would necessarily be as predicted. My skepticism is based in part on the fact that the balancing act is entirely contingent on the availability of nonmeat sources of iron, calcium, and Vitamin C, and such plants are notably constrained by seasonality, especially in temperate environments. What happens if a woman finds herself newly pregnant during the winter or in the Arctic (where the only source of Vitamin C may be raw meat)? While the model cannot be expected to account for every variation of human population and lifestyle, it should be able to explain broad environmental trends. Clearly, cross-population tests that link differences in maternal diet, food availability, immune and pathogen status, and birth outcome are desperately needed.

Other empirical issues also call for resolution. For example, given that cooking is considered a major advance in food detoxification and decontamination, what is the evidence for morbidity or mortality derived from meat-borne infection, either from the fossil/archeological record or among contemporary hunter-gatherers? Second, what do we know about dietary changes during pregnancy among wild carnivores or other omnivores? What special mechanisms do they have to protect embryogenesis while consuming parasite-rich raw meat? Fessler notes that gestational vomiting is unique to humans. Why don't we see it among other omnivorous organisms? Rats are unable to vomit and successfully employ geophagy to detoxify their omnivorous diet, as many humans do, especially during pregnancy. Lastly, milk cravings are reportedly common during pregnancy, but before pasteurization milk would also have been a major source of pathogens and might be expected to elicit aversive responses similar to those for meat.

Fessler is to be credited with bringing together multiple lines of evidence consistent with his hypothesis. Obviously his argument would have been strengthened by data that could speak to its complexity, since those provided only serve to confirm Flaxman and Sherman's (2000) and others' findings that meats are the most common targets of food taboos during pregnancy and at other times. The paper is best seen as a springboard for the generation of new specific hypotheses for interactions between cultural themes and mandates, maternal behavior, and the physiology of pregnancy. It forces us to (re)consider the adaptive significance of some striking behavioral changes that many women experience during pregnancy.

Reply

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I thank the commentators for their extensive remarks. The participation of individuals with such diverse backgrounds is most heartening, for it exemplifies the kind of interdisciplinary exchange that is needed if we are to arrive at a fuller understanding of pregnancy sickness.

Brown is unhappy with my presentation of the taboo material. Unfortunately, space constraints necessitated the most streamlined format possible. She also wonders why I did not employ existing holocultural samples. Quite simply, I wanted a more extensive data set than any published to date. In addition, by organizing my sample using categories larger than commonly recognized culture areas, I reduced the likelihood that any two societies from different categories would share significant historical links. I agree with Brown that the Human Relations Area Files, while useful, should not be the sole source of information—that is why, as stated in the text, I employed a variety of materials, drawn from multiple literatures. Lastly, Brown questions my decision to classify fish, seafood, eggs, and poultry as "meat." While these items are clearly amenable to widely varying symbolic loadings, as I note in the paper, from the perspective of risk of pathogen transmission animal protein as a whole is distinct from plant foods.

Citing Lee's dated paper, Brown suggests that I overstated the importance of meat in the diet of hunter-gatherers. However, as referenced in my paper, contemporary reviews of the subject employing much more extensive data sets support the centrality of meat in probable ancestral diets (Hill 1982, Kaplan et al. 2000, Mann 2000). Brown does raise the interesting question of how societies such as the traditional Inuit, for whom meat was an irreplaceable source of calories, coped in the face of the hypothesized gestational aversion to meat. As is suggested by my prediction of an inverse relationship between the intensity of aversions to meat and the availability of nonmeat sources of protein, essential fatty acids, iron, and calories, it may be that, as in many other appetitive contexts, narrowing the range of possibilities causes a reduction in the aversiveness of the available choices. I would be most interested in hearing from investigators who have worked with women in such societies.

Flaxman, one of the originators of the prophylaxis hypothesis, evaluates competing explanations. He dismisses the by-product hypothesis in light of variation in pregnancy sickness, since, according to him, the by-product hypothesis predicts that "pregnancy sickness should occur whenever a woman's physiological state is adequate for the maintenance of a pregnancy." Although I do not find the by-product hypothesis persuasive, I nonetheless disagree with this statement. The hormonal profile of pregnancy exhibits both within- and between-individual variation. It is therefore conceivable that, if pregnancy sickness were a by-product of either a specific hormonal profile or, more reasonably, a specific range of ratios among hormones, it would vary substantially from one pregnancy to another.

Taking a more favorable view of the by-product hypothesis, O'Connor implies that it can be reconstrued as a learning hypothesis, with high levels of human chorionic gonadotropin somehow changing the milieu in a fashion that facilitates conditioning. As Bayley and Dye make clear, this possibility cannot be dismissed, given that meat is also a principal target of conditioned aversions possessed by nonpregnant individuals (see Fessler and Navarrete n.d.). Nevertheless, the conditioning-as-by-product explanation is plagued by a number of problems. First, in contrast to Bayley and Dye's interpretation, the centrality of meat in conditioned aversions in the general population does not challenge the foundations of the prophylaxis hypothesis. In both humans and other animals the dedicated mechanisms responsible for one-trial conditioning in response to toxicosis preferentially target high-protein foods (Bernstein 1999). This focus is consistent with the argument that, because of the greater risk of pathogen transmission, meat is always likely to be hazardous. Hence, consistent with the increased vulnerability to pathogens attending reproductive immunosuppression, it seems that pregnancy increases the sensitivity of a functional targeted system that is present in nonpregnant individuals as well.

The second problem with the conditioning-as-by-product explanation is that gestational aversions are distinctly time-limited, in contrast to typical conditioned aversions that persist for years. Bayley and Dye attempt to account for this with a cognitive explanation wherein individuals attribute nausea and vomiting to a cause; nonpregnant individuals attribute illness to food poisoning, hence the aversion persists, but pregnant individuals attribute illness to pregnancy, hence the aversion ends with parturition. This explanation is at odds with a large literature on conditioned food aversions suggesting that they are the product of implicit rather than explicit memory formation (Bernstein 1999). Indeed, the striking thing about conditioned food aversions is that they are acquired *despite* conscious awareness that one's current nausea is a consequence of pregnancy, chemotherapy, or motion sickness (Arwas, Rollick, and Lubow 1989). Consistent with the hypothesis that conditioned food aversions are the product of an autonomous psychological mechanism dedicated to protecting the organism from ingestible hazards, conscious contextual attribution plays little role in these experiences. Attribution thus cannot explain the uniquely time-limited nature of gestational aversions, hence this feature continues to set gestational aversions apart from other food aversions. This suggests that, if conditioning plays a role in the genesis of gestational aversions, it is a special kind of conditioning, subject to highly specific parameters that do not operate outside of pregnancy.

Both Brown and Leeners point out that numerous psychosocial factors can influence the severity of pregnancy

sickness. Although the picture is not quite as clear-cut as Leeners implies (cf. Simpson et al. 2001), granting that psychosocial factors affect pregnancy sickness in no way detracts from the larger question why pregnancy sickness exists in the first place—of all the things that an ambivalent mother could do, why vomit? By way of analogy, the fact that psychosocial factors influence the experience of pain (Proctor, Gatchel, and Robinson 2000) does not call into question the more elementary explanation that pain is the product of a dedicated system (Treede 1995), one that presumably exists in order to motivate withdrawal from injurious contexts, facilitate learning and memory, and motivate future avoidance. Still, to gain the fullest understanding possible, we clearly do need to take account of psychosocial influences. For example, Leeners suggests that a positive correlation between the severity of pregnancy sickness and the presence of the husband indicates that nausea and vomiting may serve a socially manipulative function. This explanation is wholly congruent with my account of gestational cravings for valued goods. More generally, having carefully observed a young Sumatran woman who evinced peculiar cravings, became hyperemetic, and manifested locally recognized signs of spirit possession whenever she was forced to reside in her husband's village, I am convinced that, while based on evolved psychological mechanisms, these symptoms are overdetermined, occurring with widely varying degrees of conscious awareness and control.

Nappi points out that hormonal regulation of the immune response is not limited to early pregnancy but occurs during the menstrual cycle as well. She also notes that food intake varies across the menstrual cycle, with a caloric peak during the luteal phase (i.e., just prior to implantation in the event of conception). These observations are highly relevant to the topic at hand, as the luteal phase constitutes the period of preparation for implantation and gestation. In a forthcoming paper (Fessler n.d.) I review evidence of reproductive immunosuppression during the luteal phase, compare behavioral changes during the luteal phase and early pregnancy, and argue in favor of a pregnancy-sickness-like mechanism that operates during the luteal phase. I am thus once again seeking to explain the evolved foundations for a set of experiences that is, at the same time, clearly also subject to psychosocial influences (cf. Fontana and Palfai 1994).

Criticizing the anecdotal nature of much of the ethnographic literature that I cite, O'Connor repeatedly calls for both more systematic data collection and greater use of outcome measures, sentiments that are echoed by Tepper and Crystal-Mansour. I enthusiastically agree, with the caveat that, as I argue in the paper, these avenues will shed light on pregnancy sickness only to the extent that they are pursued across widely varying cultural and socioeconomic contexts. Like the proverbial drunk looking for his keys under the street lamp "because that's where the light is," at present investigators are prone to collect extensive outcome data and examine gestational experiences using highly reliable methods primarily where subjects are most accessible, namely, in Western maternity clinics and the like. Of related interest, Sellen provides useful guidelines for the sort of studies that are needed, pointing out that outcome measures must be calibrated against both baseline mortality rates and potential exposure to pathogens and teratogens. In the same vein, Wiley appropriately calls for more data on meat-borne infections among hunter-gatherers.

Noting the considerable empirical support for an association between pregnancy sickness and high levels of human chorionic gonadotropin, O'Connor promotes the maternal-fetal conflict theory. However, while she acknowledges that maternal-fetal conflict cannot account for the targeted nature of gestational aversions and cravings, O'Connor overlooks the failed predictions derived from this theory that I describe in the paper. Moreover, in championing hCG at the expense of progesterone as a hormonal trigger, O'Connor does not do justice to the evidence pertaining to the latter. Reviewing the endocrinological literature, O'Connor states that "of five studies examining progesterone, four find no association with pregnancy sickness (references cited by Fessler plus Soules et al. 1980, Kauppila et al. 1984)." As I note in the paper, the absence of such a correlation may well prove to be an obstacle to the prophylaxis hypothesis. Nevertheless, the situation is not as well defined as O'Connor suggests. First, although Soules et al. indeed found no association with progesterone, they also found no association with hCG, an unusual result and one that I would have expected to have led O'Connor to question their methods. Second, O'Connor misconstrues the Kauppila et al. study. Kauppila et al. made no measurements of progesterone whatsoever; rather, they measured SP1 (pregnancy-specific beta 1-glycoprotein), a trophoblast specific marker that is not correlated with progesterone (Baumann, Bersinger, and Birkhäuser 1998).

Continuing her attack on the prophylaxis hypothesis, O'Connor notes that progesterone levels increase across pregnancy, peaking just before parturition, yet pregnancy sickness is concentrated in the first trimester. However, this objection is premised on the assumption that there is a continuous association between progesterone levels and vulnerability to pathogens. As I detailed in the paper, the complex interplay between mother and fetus is such that, for a number of reasons, immunological vulnerability is greatest in the first trimester; after this period, rising progesterone levels are likely to be of secondary importance given, for example, progressive increases in phagocytosis and rising levels of some Th1 cytokines. Accordingly, the strongest physiological test of the prophylaxis hypothesis (one which, had I thought of it at the time, I would have included in the paper) is not a predicted association between the severity of pregnancy sickness and levels of progesterone but a predicted association between the severity of pregnancy sickness and the degree of Th2 predominance.

O'Connor points out that between-population variation in progesterone levels corresponds with variation in nutritional state, arguing that such evident plasticity contradicts the prophylaxis explanation of pregnancy sickness. I see no such contradiction. On the contrary, I

explicitly predicted that the strength of pregnancy sickness should be a function of a woman's nutritional state. Moreover, to extend the refined prediction outlined above, all else being equal, the degree of Th2 predominance should be contingent on the mother's preconception robustness (cf. Nappi's comments above). As Sellen aptly points out, however, the relationship between nutritional state, immune functioning, and infection is a complex one. Is immunosuppression simply an energy-saving response to dietary constriction? Or, alternatively, do across-the-board reductions in physiological robustness result in increased rates of infection, with pathogens then manipulating immune responses for their own benefit? These are important questions reaching far beyond the domain of pregnancy sickness, questions that must be answered before discrete predictions regarding pregnancy sickness can be tested.

Sellen argues for greater attention to micronutrient needs in understanding gestational cravings and aversions. I agree that this is a potentially productive line of investigation. However, it is important to keep in mind that behavior such as geophagy can be multifunctional, and we should not rule out one explanation in order to investigate another. Sellen also questions the nutritional feasibility of gestational meat avoidance given the importance of meat as a source of trace elements (and, I might add, essential fatty acids). The model that I sketched of complementary cravings and aversions was intended to address precisely this issue. However, it is admittedly only a model and requires more rigorous testing before any firm conclusions can be reached.

Although he is one of the originators of the prophylactic meat-avoidance hypothesis, Sherman also argues in favor of Profet's phytotoxin-avoidance hypothesis. Referring to the results of Flaxman and Sherman's (2000) metaanalysis (reprinted as my figure 1), Sherman notes that combining three categories of plant products that are targets of aversions ("nonalcoholic beverages," "vegetables," and "ethnic, strong, and spicy foods") into a single metacategory accounts for as many per capita aversions as those contributed by the category "meat." While I both acknowledge the key contributions that Sherman has made to the study of pregnancy sickness and recognize that some support does exist for the phytotoxin hypothesis, for several reasons I do not find this line of argument persuasive. First, among the three summed categories, the primary contribution (> 50%) derives not from the prototypical category "vegetables" but rather from "nonalcoholic beverages," a rather ambiguous class. Indeed, the influence of "nonalcoholic beverages" is so great that combining it with other categories, such as "alcohol" and "dairy," produces a sizable metacategory of aversions regardless of the prominence or lack thereof of phytotoxins in these categories. Second, the tactic of combining categories of aversions obscures the fact that "vegetables," which we might expect to be the core of a phytotoxin metacategory, are craved nearly as often as they are avoided. Similarly, there is no difference between the rate of cravings and the rate of aversions for the ostensibly highly teratogenic category "ethnic, strong, and spicy foods." Hence, although neither hypothesis is conclusively proven at present, I continue to believe that the evidence in favor of the phytotoxin hypothesis is considerably weaker than that in favor of the meat-avoidance hypothesis.

Tepper and Crystal-Mansour offer a number of interesting ideas, but their remarks indicate an incomplete grasp of the logic of evolutionary explanations. They question both the vulnerability of pregnant women to pathogens and the importance of meat as a source of infection. On the first question, from the perspective of evolutionary fitness the key issue is not simply whether rates of infection differ between pregnant women and others but rather whether the consequences of infection differ between these groups. As discussed in the paper, many infections that produce only minor symptoms in healthy nonpregnant individuals can have a catastrophic impact on a pregnant woman and the developing organism she carries. On the second question, Tepper and Crystal-Mansour must keep in mind that evolutionary arguments are premised upon reconstructions of probable recurrent circumstances faced by ancestral populations. For example, among contemporary women in industrialized societies an important avenue for *T gondii* infection (besides eating raw, undercooked, or improperly prepared meat) is contact with cat feces, because cats pass the parasite's oocysts in their stools (Jones et al. 2001). However, because our ancestral mothers unquestionably ate infected animals more often than they changed cat litter, meat will have consistently constituted the more important source of *T. gondii* infection over evolutionary history.[1]

Discussing gestational aversions to coffee, Bailey and Dye make a similar mistake when they ask why an adaptive mechanism would accidentally misfire in a large proportion of the pregnant population. The answer is simply that coffee is an evolutionarily novel stimulus—on an evolutionary time scale, the processing and consumption of this now-pervasive drug began but a moment ago, hence we would not expect natural selection to have eliminated the propensity for erroneous responses to its odor.

Tepper and Crystal-Mansour doubt that cravings "serve to identify and redress bodily needs." However, they misunderstand my position. Rather than postulating some complex and precise calibration of desires in response to a careful monitoring of the internal milieu, I simply suggest a set of default concomitants of pregnancy, motivations that, most of the time, for most women, would have been adaptive over evolutionary history. Accordingly, the example cited by Tepper and Crystal-Mansour, that pregnant women with gestational diabetes crave sweets and fruit as much as those without, does not address the issue—if pregnancy simply flips a

1. Water-borne toxoplasmosis is similarly likely due to the evolutionarily novel circumstances of reliance on reservoirs in conjunction with high cat population densities (Aramini et al. 1999). More complex is the issue of evolutionary disequilibrium in the case of diary products, a problem raised by Wiley's observation that milk is a source of infection (see n. 14 above)

"cravings-for-fruits-and-carbohydrates switch" somewhere in the mind, then blood sugar levels (and associated feedback mechanisms) may be irrelevant to the experience of those cravings. By the same token, contrary to Tepper and Crystal-Mansour's interpretation, the prophylaxis hypothesis makes no predictions regarding the behavior or experiences of immunosuppressed organ transplant recipients or HIV sufferers, since the postulated evolved mechanism would likely respond to indices of the early stages of pregnancy rather than to the generic state of immunosuppression. Lastly, noting that Western studies support a link between cravings and mood, Tepper and Crystal-Mansour suggest that "mood changes during pregnancy may be more predictive of food cravings and aversions than pregnancy sickness." I have used the term "pregnancy sickness" to refer to a suite of changes, including appetitive ones. Mood changes may well be components of the mechanisms that shape ingestive behavior during pregnancy, a proximate explanation that in no way competes with my ultimate explanation of the utility of such changes (cf. Daly and Wilson 1983:14–19).

Wiley expresses skepticism about the complexity of my model of cravings, noting that the proposed system's functionality is contingent on the availability of various nutrients and food types and such availability varies across seasons and settings. However, to the extent that the given substances are potentially available, even if difficult to obtain, strong cravings are precisely the sort of subjective experiences needed to motivate women to seek out these substances or to urge others to procure them on their behalf. Of course, over human history there are likely to have been times and circumstances in which the given materials were simply unavailable. However, contrary to Wiley's supposition, it is not clear that such episodes would have sufficed to preclude either the evolution or the maintenance of the proposed mechanisms. By way of analogy, humans live in places that are too hot, too cold, too wet, or too dry, but this variation has not precluded the existence of mechanisms that generate a desire for and an attempt to obtain more hospitable conditions (on the contrary, it is likely that it is precisely such mechanisms that have motivated the technological innovations that have made it possible for humans to survive in such environments). Wiley also raises the important question of the nature of equivalent mechanisms operating in nonhuman carnivores and omnivores. Although a considerable amount is known about cross-species variation in placentation (Enders and Blankenship 1999), I have been unable to find equivalent materials on reproductive immunosuppression. As I noted in the text, published investigations of nonhuman gestational dietary changes suffer extensive (but not unavoidable) methodological shortcomings.

Sherman concludes his comments by advocating the depathologizing of pregnancy "sickness." I concur, and, moreover, I suggest that this should be part of a larger agenda, that of depathologizing pregnancy and female reproductive functioning as a whole (Boston Women's Health Book Collective 1996). That said, I share Sellen's concern that academic discussions of pregnancy sickness may lead to misguided and premature decisions by individuals or institutions. I am therefore grateful to Flaxman for pointing out that the prophylaxis hypothesis does not imply that pregnant women should not eat meat—provided that adequate precautions are taken, meat constitutes a valuable part of the diet. Similarly, in no way do I wish to suggest that pregnancy taboos enhance the welfare of women subjected to them. While some taboos, such as those on papaya, may have a functional component, the vast majority, including nearly all of those on meat, are probably best viewed as non- or dysfunctional outgrowths of the interaction of social processes and evolved psychological propensities (see Fessler and Navarrete n.d. for a complete discussion).

I am pleased that Wiley concludes by describing the paper as a springboard. I will consider my efforts successful if, regardless of the ultimate fate of the prophylaxis hypothesis, the paper serves to motivate additional research into the causes, consequences, and cultural concomitants of pregnancy sickness. Despite the human propensity to extend territoriality and social competition into the realm of ideas, as scientists we are fortunate to engage in a pursuit in which we can take just as much joy in being proven wrong as in being proven right.

References Cited

ABDALLA, SABIHA M. 1982. Food habits during pregnancy and lactation in Iraq. *Food and Nutrition Bulletin* 4(3):14–16.

ABRAM, M., AND M. DORIC. 1997. Primary *Listeria monocytogenes* infection in gestating mice. *Folia Microbiologica* 42: 65–71.

ADRIANI, N., AND A. C. KRUYT. 1996 (1951). *The Bare'e-speaking Toadja of Central Celebes (the East Toradja)*. New Haven: Human Relations Area Files.

AIELLO, L. C., AND P. WHEELER. 1995. The expensive-tissue hypothesis: The brain and the digestive system in human and primate evolution. CURRENT ANTHROPOLOGY 36:199–221.

AL-KANHAL, M. A., AND I. A. BANI. 1995. Food habits during pregnancy among Saudi women. *International Journal for Vitamin and Nutrition Research* 65:206–10.

ALLEN, L. H. 2001. Biological mechanisms that might underlie iron's effect on fetal growth and preterm birth. *Journal of Nutrition* 131:581S–89S. [DWS]

ARAMINI, J. J., C. STEPHEN, J. P. DUBEY, C. ENGELSTOFT, H. SCHWANTJE, AND C. S. RIBBLE. 1999. Potential contamination of drinking water with *Toxoplasma gondii* oocysts. *Epidemiology and Infection* 122:305–15.

ARWAS, S., A. ROLNICK, AND R. E. LUBOW. 1989. Conditioned taste aversion in humans using motion-induced sickness as the US. *Behaviour Research and Therapy* 27:295–301.

AUNGER, R., R. C. BAILEY, G. R. BENTLEY, P. T. ELLISON, A. M. HARRIGAN, AND M. JENIKE. 1999. Women's strategies to alleviate nutritional stress in a rural African society. *Social Science and Medicine* 48:149–62.

AYRES, BARBARA. 1954. Personality determinants of food and sex taboos during pregnancy. Ph.D. diss., Radcliffe College, Cambridge, Mass. [JKB]

———. 1967. "Pregnancy magic: A study of food taboos and sex avoidances," in *Cross-cultural approaches: Readings in comparative research.* Edited by Clellan Ford, pp. 111–25. New Haven: HRAF Press. [JKB]

BARNETT, W. K. 1971. *An ethnographic description of Sanlei*

Ts'un, Taiwan, with emphasis on women's rules: Overcoming research problems caused by the presence of a great tradition. Ann Arbor: University Microfilms.

BARRETT, J. F., P. G. WHITTAKER, J. G. WILLIAMS, AND T. LIND. 1994. Absorption of non-haem iron from food during normal pregnancy. *British Medical Journal* 309:79–82.

BARRIGA, C., A. B. RODRIGUEZ, AND E. ORTEGA. 1994. Increased phagocytic activity of polymorphonuclear leukocytes during pregnancy. *European Journal of Obstetrics, Gynecology, and Reproductive Biology* 57:43–46.

BARTON, R. F. 1998 (1955). *The mythology of the Ifugaos*. New Haven: Human Relations Area Files.

BAUMANN, C. K., N. A. BERSINGER, AND M. H. BIRKHÄUSER. 1998. Placental protein 14 and pregnancy-specific beta 1-glycoprotein in women with suppressed, normal, and stimulated follicular maturation. *Gynecological Endocrinology* 12:231–41.

BAYLEY, T. M., L. DYE, S. JONES, M. DE BONO, AND A. J. HILL. n.d. Food cravings and aversions during pregnancy: Relationships with nausea and vomiting. MS. [TMB, LD]

BEAGLEHOLE, E., AND P. BEAGLEHOLE. 1938. *Ethnology of Pukapuka*. Honolulu: Bernice R. Bishop Museum Bulletin.

———. 1941. *Pangai: Village in Tonga.* Memoirs of the Polynesian Society 18.

———. 2000 (1935). *Hopi of the Second Mesa*. Menasha, Wis. American Anthropological Association/New Haven: Human Relations Area Files.

BEAZLEY, D. M., AND R. S. EGERMAN. 1998. Toxoplasmosis. *Seminars in Perinatology* 22:332–38.

BECHER, HANS. 1960. *The Surara and Pakidai, two Yanoama tribes in northwest Brazil*. Hamburg: Kommissionsverlag Cram, de Gruyter

BEERS, M. H., AND R. BERKOW. 1999. 17th edition. *The Merck manual of diagnosis and therapy*. Whitehouse Station, N.J.: Merck Research Laboratories.

BELLO, C. S. 1992. Transmission of cytomegalovirus in the Gambia. *West African Journal of Medicine* 11:140–45.

BEN MILED, S., D. BIBI, N. KHALFI, R. BLIBECH, Y. GHARBI, R. CASTALLI, AND N. KHROUF. 1989. Iron stocks and risk of anemia in twins. *Archives de l'Institut Pasteur, Tunis* 66:221–41.

BENTLEY, D. P. 1985. Iron metabolism and anaemia in pregnancy. *Clinical Haematology* 14:613–28.

BENTLEY, G. R. 1999. Aping our ancestors: Comparative aspects of reproductive ecology. *Evolutionary Anthropology* 7: 175–85.

BERNSTEIN, I. L. 1999. Taste aversion learning: A contemporary perspective. *Nutrition* 15:229–34.

BERNSTEIN, I. L., M. M. WEBSTER, AND I. D. WEBSTER. 1982. Food aversions in children receiving chemotherapy for cancer. *Cancer* 50:2960–63. [TMB, LD]

BIANCO, A. M., E. DESAI, I. D. DESAI, A. M. DOELL, M. I. JANSEN, S. A. OFFICIATI, J. E. DE OLIVEIRA, AND Y. VAN SEVEREN. 1990. Nutritional needs assessment of rural agricultural migrants of southern Brazil: Designing, implementing, and evaluating a nutrition education program. *World Review of Nutrition and Dietetics* 61:64–131.

BIASSONI, R., C. BOTTINO, R. MILLO, L. MORETTA, AND A. MORETTA. 1999. Natural killer cell-mediated recognition of human trophoblast. *Seminars in Cancer Biology* 9: 13–18.

BLACK, A. J., J. TOPPING, B. DURHAM, R. G FARQUHARSON, AND W. D. FRASER. 2000. A detailed assessment of alterations in bone turnover, calcium homeostasis, and bone density in normal pregnancy. *Journal of Bone and Mineral Research* 15:557–63.

BLACKWOOD, BEATRICE. 1935. *Both sides of Buka Passage: An ethnographic study of social, sexual, and economic questions in the north-western Solomon Islands*. Oxford: Clarendon Press.

BOHANNAN, PAUL, AND LAURA BOHANNAN. 1969. *A source book on Tiv religion in* 5 *volumes.* Vol.1. *Cosmos, soma, psyche, and disease*. New Haven: Human Relations Area Files.

BÖKÖNYI, S. 1969. "Archaeological problems and methods of recognizing animal domestication," in *The domestication and exploitation of plants and animals*. Edited by P. J. Ucko and G. W. Dimbleby, pp. 219–29. London: Duckworth.

BOLTON, J. M. 1972. Food taboos among the Orang Asli in West Malaysia: A potential nutritional hazard. *American Journal of Clinical Nutrition* 25:789–99.

BOREL, I. M., S. M. FREIRE, E. RIVERA, A. CANELLADA, R. A. BINAGHI, AND R. A. MARGNI. 1999. Modulation of the immune response by progesterone-induced lymphocyte factors. *Scandinavian Journal of Immunology* 49:244–50.

BORTOLUS, R., F. PARAZZINI, L. CHATENOUD, G. BENZI, M. M. BIANCHI, AND A. MARINI. 1999. The epidemiology of multiple births. *Human Reproduction Update* 5: 179–87.

BOSTON WOMEN'S HEALTH BOOK COLLECTIVE. 1996. 25th anniversary edition. *The new Our bodies, our selves: A book by and for women.* New York: Simon and Schuster.

BOUÉ, A., J. BOUÉ, AND A. GROPP. 1985. Cytogenetics of pregnancy wastage. *Advances in Human Genetics* 14:1–57. [KO]

BRANDES, J. M. 1967. First-trimester nausea and vomiting as related to outcome of pregnancy. *Obstetrics and Gynecology* 30:427–31.

BRIGGS, GEORGE W. 1920. *The Chamars*. London and New York: Oxford University Press.

BROCK, R. G. C. 1953. "Some notes on the Zande tribe as found in the Meridi District (Bahr El Ghazal Province)," in *Ethnographic survey of Africa*, part 9, *East Central Africa*. Edited by Daryll Forde. London: International African Institute.

BROWN, J. E., E. S. KAHN, AND T. J. HARTMAN. 1997. Profet, profits, and proof: Do nausea and vomiting of early pregnancy protect women from "harmful" vegetables? *American Journal of Obstetrics and Gynecology* 176:179–81.

BROWN, JUDITH K., THERESE SARAH, AND DENISE PILATO. 1994. "A bibliography for the cross-cultural study of women's lives," in *Women among women: Anthropological perspectives on female age hierarchies.* Edited by Jeanette Dickerson-Putman and Judith K. Brown, pp. 1214–40. Urbana: University of Illinois Press. [JKB]

BROWN, M. J., N. K. SHAH, AND R. M. WORTH. 1968. Food habits and food intake in Nepal. *Tropical Geographic Medicine* 20:217–24.

BURKHARDT, VALENTINE R. 1954. *Chinese creeds and customs*. Hong Kong: South China Morning Post.

CABRITA, J., J. RODRIGUES, F. BRAGANÇA, C. MORGADO, I. PIRES, AND A. P. GONÇALVES. 1992. Prevalence, biotypes, plasmid profile, and antimicrobial resistance of *Campylobacter* isolated from wild and domestic animals from northeast Portugal. *Journal of Applied Bacteriology* 73:279–85.

CALAME-GRIAULE, G., AND G. DIETERLEN. 2000 (1960). *Dogon alimentation*. New Haven: Human Relations Area Files.

CALONNE-BEAUFAICT, ADOLPHE DE. 1998 (1921). *Azande: Introduction to a general ethnography of Ubangi-Uele and Aruwimi basins*. New Haven: Human Relations Area Files.

CANTONI, P., R. HUDSON, H. DISTEL, AND M. LASKA. 1998. Changes in olfactory perception and dietary habits in the course of pregnancy: A questionnaire study. *Chemical Senses* 245:58.

CARLES, G., E. MONTOYA, F. JOLY, AND C. PENEAU. 1995. Leptospiroses et grossesse: Etude de 11 cas en Guyane Française. *Journal de Gynécologie, Obstetrique et Biologie de la Reproduction* 24:418–21.

CATALANO, P. M. 1999. Pregnancy and lactation in relation to range of acceptable carbohydrate and fat intake. *European Journal of Clinical Nutrition* 53:S124–35.

CHAPIN, NORMAN M. 1983. Curing among the San Blas Kuna of Panama. Ph. D. diss., University of Arizona, Tucson, Ariz.

CHAPMAN, ANNE. 1982. *Drama and power in a hunting society: The Selk'nam of Tierra del Fuego*. Cambridge and New York: Cambridge University Press.

CHART, H., S. M. SCOTLAND, AND B. ROWE. 1989. Production of vero cytotoxin by *Escherichia coli* and *Shiga* toxin by *Shigella dysenteriae* 1 as related to the growth medium and availability of iron. *Zentralblatt für Bakteriologie* 272:1–10.

CHERIAN, T. 2000. Effect of papaya latex extract on gravid and non-gravid rat uterine preparations in vitro. *Journal of Ethnopharmacology* 70:205–12.

CHOPRA, U., H. VOHRA, S. CHHIBBER, N. K. GANGULY, AND S. SHARMA. 1997. TH1 pattern of cytokine secretion by splenic cells from pyelonephritic mice after in-vitro stimulation with hsp-65 of *Escherichia coli*. *Journal of Medical Microbiology* 46:139–44.

CLARK, M. 1959. *Health in the Mexican-American culture*. Berkeley: University of California Press.

COKER, A. O., AND C. L. OBI. 1991. Effect of iron-dextran on lethality of Nigerian isolated of *Campylobacter jejuni* in mice. *Central African Journal of Medicine* 37:202–3.

COMMISSION ON THE NUTRITION CHALLENGES OF THE 21ST CENTURY. 2000. Global nutrition challenges: A life cycle approach. *Food and Nutrition Bulletin* 21:18–34. [DWS]

COOKSEY, N. R. 1995. Pica and olfactory craving of pregnancy: How deep are the secrets? *Birth* 22:129–37.

COOPER, J., AND R. D. WALKER. 1998. Listeriosis. *Veterinary Clinics of North America, Food Animal Practice* 14: 113–25.

CORBEL, M. J. 1997. Brucellosis: An overview. *Emerging Infectious Diseases* 3:213–21

COSMINSKY, S. 1994. "Childbirth and change: A Guatemalan study," in *Ethnography of fertility and birth*. Edited by Carol P. MacCormack. Prospect Heights, Ill: Waveland Press.

COSTEA, D. M., L. K. GUNN, C. HARGREAVES, R. J. HOWELL, AND T. CHARD. 2000. Delayed luteo-placental shift of progesterone production in IVF pregnancy. *International Journal of Gynaecology and Obstetrics* 68:123–29.

COSTELLO, CHRISTINE A. 1986. Maternal and child health in rural Uganda: The role of nutrition. Ph.D. diss., University of Pennsylvania, Philadelphia, Pa.

COVARRUBIAS, MIGUEL. 1938. *Island of Bali*. New York: Knopf.

CROVETTI, R., M. PORRINI, A. SANTANGELO, AND G. TESTOLIN. 1998. The influence of thermic effect of food on satiety. *European Journal of Clinical Nutrition* 52:482–88.

CROWTHER, C. A., J. E. HILLER, B. PRIDMORE, R. BRYCE, P. DUGGAN, W. M. HAGUE, AND J. S. ROBINSON. 1999. Calcium supplementation in nulliparous women for the prevention of pregnancy-induced hypertension, preeclampsia, and preterm birth: An Australian randomized trial, FRACOG and the ACT Study Group. *Australia and New Zealand Journal of Obstetrics and Gynaecology* 39:12–18.

CRYSTAL, S. R., D. J. BOWEN, AND I. L. BERNSTEIN. 1999. Morning sickness and salt intake, food cravings, and food aversions. *Physiology and Behavior* 67:181–87.

CZAJA, J. A. 1975. Food rejection by female rhesus monkeys during the menstrual cycle and early pregnancy. *Physiology and Behavior* 14:579–87.

DALY, M., AND M. WILSON. 1983. 2d edition. *Sex, evolution, and behavior.* Boston: Willard Grant Press.

DARWISH, O. A., E. K. AMINE, AND S. M. ABDALLA. 1982. Food habits during pregnancy and lactation in Iraq. *Food and Nutrition Bulletin* 4:4–16.

DA SILVA, P. ALCIONILIO BRÜZZI ALVES. 1962. *The indigenous civilization of the Uaupés*. São Paulo: Centro de Pesquisas de Iauareté.

DEFOLIART, G. R. 1995. Edible insects as minilivestock. *Biodiversity and Conservation* 4:306–21.

DEL MAR MELERO-MONTES, M., AND H. JICK. 2001. Hyperemesis gravidarum and the sex of the offspring. *Epidemiology* 12:123–24.

DEMISSIE, T., N. M. MUROKI, AND W. KOGI-MAKAU. 1998. Food aversions and cravings during pregnancy: Pregnancy and significance for maternal nutrition in Ethiopia. *Food and Nutrition Bulletin* 19:20–26.

DE SILVA, W. I. 1995. Towards safe motherhood in Sri Lanka: Knowledge, attitudes, and practices during the period of maternity. *Journal of Family Welfare* 41:18–26.

DEUTSCH, J. A. 1994. Pregnancy sickness as an adaptation to concealed ovulation. *Rivista di Biologia/Biology Forum* 87: 277–95.

DICKENS, G., AND W. H. TRETHOWAN. 1971. Cravings and aversions during pregnancy. *Journal of Psychosomatic Research* 15:259–68.

DIMIER, I. H., AND D. T. BOUT. 1998. Interferon-gamma-activated primary enterocytes inhibit *Toxoplasma gondii* replication: A role for intracellular iron. *Immunology* 94:488–95.

DUFFY, V. B., L. M. BARTOSHUK, R. STRIEGEL-MOORE, AND J. RODIN. 1998. Taste changes across pregnancy. *Annals of the New York Academy of Sciences* 855:805–9.

DUFOUR, D. L., J. C. REINA, AND G. B. SPURR. 1999. Food and macronutrient intake of economically disadvantaged pregnant women in Colombia. *American Journal of Human Biology* 11:753–62.

DURHAM, W. H. 1991. *Coevolution: Genes, culture, and human diversity*. Stanford: Stanford University Press.

DWYER, C. M., A. J. A. MADGWICK, S. S. WARD, AND N. C. STICKLAND. 1995. Effect of maternal undernutrition in early gestation on the development of fetal myofibres in the guinea-pig. *Reproduction Fertility and Development* 7: 1285–92.

DYE, L., S. JONES, AND A. J. HILL. 1998. Food aversions and cravings during pregnancy: A temporal association with nausea and vomiting. *International Journal of Obesity* 22:S130.

EDWARDS, C. H., H. MC SWAIN, AND S. HAIRE. 1954. Odd dietary practices of women. *Journal of the American Dietetic Association* 30:976–81.

ELICK, JOHN WILLIAM. 1969. An ethnography of the Pichis Valley Campa of eastern Peru. Ph.D. diss., University of California, Los Angeles, Calif.

ELLISON, P. T., S. E. LIPSON, M. T. O'ROURKE, G. R. BENTLEY, A. M. HARRIGAN, C. PANTER-BRICK, AND V. J. VITZTHUM. 1993. Population variation in ovarian function. *Lancet* 342:433–34 [KO]

EL-MALLAKH, R. S., N. R. LIEBOWITZ, AND M. S. HALE. 1990. Hyperemesis gravidarum as a conversion disorder. *Journal of Nervous and Mental Disease* 178:655–59. [BL]

EMELIANOVA, S., P. MAZZOTTA, A. EINARSON, AND G. KOREN. 1999. Prevalence and severity of nausea and vomiting of pregnancy and effect of vitamin supplementation. *Clinical and Investigative Medicine* 22:106–10.

EMMONS, GEORGE T. 1991. *The Tlingit Indians*. Seattle: University of Washington Press/New York: American Museum of Natural History.

ENDERS, A. C., AND T. N. BLANKENSHIP. 1999. Comparative placental structure. *Advanced Drug Delivery Reviews* 38: 3–15.

EPHROS, H. D., AND H. Y. LEE. 1988. Amylophagia and iron deficiency anemia. *Oral Surgery, Oral Medicine, Oral Pathology* 66:448–49.

ERICK, M. 1995. Hyperolfaction and hyperemesis gravidarum: What is the relationship? *Nutrition Reviews* 53:289–95.

EVANS-PRITCHARD, E. E. 1998 (1932). *Heredity and gestation, as the Azande see them*. New Haven: Human Relations Area Files.

———. 1998 (1937). *Witchcraft, oracles, and magic among the Azande*. New Haven: Human Relations Area Files.

FAIRBURN, C. G., A. STEIN, AND R. JONES. 1992. Eating habits and eating disorders during pregnancy. *Psychosomatic Medicine* 54:665–72.

FAIRWEATHER, D. V. 1968. Nausea and vomiting in pregnancy. *American Journal of Obstetrics and Gynecology* 102: 135–75.

FARBER, J. M., AND P. I. PETERKIN. 1991. *Listeria monocytogenes*, a food-borne pathogen. *Microbiological Reviews* 55: 476–511.

FERNANDES, D. M., X. JIANG, J. H. JUNG, AND C. L. BALDWIN. 1996. Comparison of T cell cytokines in resistant and susceptible mice infected with virulent *Brucella abortus*

strain 2308. *FEMS Immunology and Medical Microbiology* 16: 193–203.

FERNÁNDEZ-MARCOS, A., M. MARTÍN, J. J. SANCHEZ, A. RODRIGUEZ-LESCURE, A. CASADO, J. A. LÓPEZ MARTIN, AND E. DIAZ-RUBIO. 1996. Acute and anticipatory emesis in breast cancer patients. *Supportive Care in Cancer* 4:370–77.

FERRO-LUZZI, G. E. 1973. Food avoidances of pregnant women in Tamilnad. *Ecology of Food and Nutrition* 2:259–66.

FESSLER, D. M. T. n.d. An evolutionary perspective on luteal phase immunosuppression and meat eating. *Rivista di Biologia/Biology Forum*. In press.

FESSLER, D. M. T., AND C. D. NAVARRETE. n.d. Meat is good to taboo: Dietary proscriptions as a product of the interaction of psychological mechanisms and social processes. MS.

FIFE, A. E., AND A. FIFE. 1966. *Saints of sage and saddle: Folklore among the Mormons*. Gloucester, Mass.: Peter Smith.

FIKREE, F. F., H. W. BERENDES, F. MIDHET, R. M. D'SOUZA, AND R. HUSSAIN. 1994. Risk factors for intrauterine growth retardation: Results of a community-based study from Karachi. *Journal of the Pakistan Medical Association* 44:30–34.

FINERMAN, R. D. 1985. Health care decisions in an Andean Indian community: Getting the best of both worlds. Ph.D. diss., University of California, Los Angeles, Calif.

FINLEY, D. A., K. G. DEWEY, B. LONNERDAL, AND L. E. GRIVETTI. 1985. Food choices of vegetarians and nonvegetarians during pregnancy and lactation. *Journal of the American Dietetic Association* 85:678–85.

FIOCCHI, A., P. RESTANI, AND E. RIVA. 2000. Beef allergy in children. *Nutrition* 16:454–57.

FISCHER, A. 1998 (1963). *Reproduction in Truk*. New Haven: Human Relations Area Files.

FISHER, C. W., AND S. E. MARTIN. 1999. Effects of iron and selenium on the production of catalase, superoxide dismutase, and listeriolysin O in *Listeria monocytogenes*. *Journal of Food Protection* 62:1206–9.

FLAXMAN, S. M., AND P. W. SHERMAN. 2000. Morning sickness: A mechanism for protecting mother and embryo. *Quarterly Review of Biology* 75:113–48.

FOLL, C. V. 1959. An account of some of the beliefs and superstitions about pregnancy, parturition, and infant health in Burma. *Journal of Tropical Pediatrics* 5:51–59.

FONTANA, A. M., AND T. G. PALFAI. 1994. Psychosocial factors in premenstrual dysphoria: Stressors, appraisal, and coping processes. *Journal of Psychosomatic Research* 38:557–67.

FORBES, B. A. 1989. Acquisition of cytomegalovirus infection: An update. *Clinical Microbiological Review* 2:204–16.

FORBES, S. n.d. Does pregnancy sickness protect or signal embryo quality? *Trends in Ecology and Evolution.* In press. [KO]

FORSYTH, C. J., AND G. M. BENOIT. 1989. "Rare, ole, dirty snacks": Some research notes on dirt eating. *Deviant Behavior* 10:61–68.

FRANK, L., A. MARIAN, M. VISSER, E. WEINBERG, AND P. C. POTTER. 1999. Exposure to peanuts in utero and in infancy and the development of sensitization to peanut allergens in young children. *Pediatric Allergy and Immunology* 10: 27–32.

FUCHS, K., E. PALDI, H. ABRAMOVICI, AND B. A. PARETZ. 1980. Treatment of hyperemesis gravidarum by hypnosis. *International Journal of Clinical and Experimental Hypnosis* 28:313–22. [BL]

GADSBY, R., A. BARNIE-ADSHEAD, AND C. JAGGER. 1993. A prospective study of nausea and vomiting during pregnancy. *British Journal of General Practice* 43:245–48. [KO]

———. 1997. Pregnancy nausea related to women's obstetric and personal histories. *Gynecologic and Obstetric Investigation* 43: 108–11.

GALVÁN RAMÍREZ, M. D. L. L., J. L. SOTO MANCILLA, O. VELASCO CASTREJÓN, AND R. PÉREZ MEDINA. 1995. Incidence of anti-*Toxoplasma* antibodies in women with high-risk pregnancy and habitual abortions. *Revista da Sociedade Brasileira de Medicina Tropical* 28:333–37.

GARB, J. L., AND A. J. STUNKARD. 1974. Taste aversions in man. *American Journal of Psychiatry* 131:1204–07. [TMB, LD]

GEISSLER, P. W., D. L. MWANIKI, F. THIONG'O, AND H. FRIIS. 1997. Geophagy among school children in western Kenya. *Tropical Medicine and International Health* 2:624–30.

GILARDI, J. D., S. S. DUFFEY, C. A. MUNN, AND L. A. TELL. 1999. Biochemical functions of geophagy in parrots: Detoxification of dietary toxins and cytoprotective effects. *Journal of Chemical Ecology* 25:897–922.

GILBERT, A. N., AND C. J. WYSOCKI. 1991. Quantitative assessment of olfactory experience during pregnancy. *Psychosomatic Medicine* 53:693–700.

GILKS, J. L., AND J. B. ORR. 1931. *Studies in nutrition: The physique and health of two African tribes*. London: His Majesty's Stationery Office.

GITTELSOHN, J., M. THAPA, AND L. T. LANDMAN. 1997. Cultural factors, calorie intake, and micronutrient sufficiency in rural Nepali households. *Social Science and Medicine* 44: 1739–49. [DWS]

GLEICHER, N., P. BEERS, T. D. KERENYI, C. J. COHEN, AND S. B. GUSBERG. 1980. Leukocyte migration enhancement as an indicator of immunologic enhancement. I. Pregnancy. *American Journal of Obstetrics and Gynecology* 136: 1–4.

GOH, J., AND M. FLYNN. 1992. *Campylobacter jejuni* in pregnancy. *Australia and New Zealand of Obstetrics and Gynaecology* 32:246–48.

GOLDENBERG, R. L., T. TAMURA, M. DUBARD, K. E. JOHNSTON, R. L. COPPER, AND Y. NEGGERS. 1996. Plasma ferritin and pregnancy outcome. *American Journal of Obstetrics and Gynecology* 175:1356–59.

GOODLIN, R. C. 1982. Why treat "physiologic" anemias of pregnancy? *Journal of Reproductive Medicine* 27:639–46.

GOODWIN, T. M., M. MONTOR, J. H. MESTMAN, A. E. PEKARY, AND J. M. HERSHMAN. 1992. The role of chorionic gonadotropin in transient hyperthyroidism of hyperemesis gravidarum. *Journal of Clinical Endocrinology and Metabolism* 75:1333–37. [KO]

GOULD-MARTIN, KATHERINE. 1976. *Women asking women: An ethnography of health care in rural Taiwan*. Ann Arbor: University Microfilms.

GREEN, L. C., AND M. W. BECKWITH. 1924. Hawaiian customs and beliefs relating to birth and infancy. *American Anthropologist* 26:130–46.

GRIGSBY, R. K., B. A. THYER, R. J. WALLER, AND G. A. JOHNSTON JR. 1999. Chalk eating in middle Georgia: A culture-bound syndrome of pica? *Southern Medical Journal* 92: 190–92.

GROSSMAN, C. J. 1984. Regulation of the immune system by sex steroids. *Endocrine Reviews* 5:435–55. [REN]

GULBIS, B., E. JAUNIAUX, J. DECUYPER, P. THIRY, D. JURKOVIC, AND S. CAMPBELL. 1994. Distribution of iron and iron-binding proteins in first-trimester human pregnancies. *Obstetrics and Gynecology* 84:289–93.

GULICK, E. E., V. SHAW, AND M. ALLISON. 1989. Dietary practices and pregnancy discomforts among urban blacks. *Journal of Perinatology* 9:271–80.

HADJADJ, M. L., F. MARTIN, AND D. FICHET. 1990. Anémie par carence en fer et pagophagie: A propos d'un cas. *Revue de Medecine Interne* 11:236–38.

HAIG, D. 1993. Genetic conflicts in human pregnancy. *Quarterly Review of Biology* 68:495–532.

HALLBERG, L., L. ROSSANDER-HULTEN, M. BRUNE, AND A. GLEERUP. 1992. Calcium and iron absorption: Mechanism of action and nutritional importance. *European Journal of Clinical Nutrition* 46:317–27.

HANKS, J. R. 2000 (1963). *Maternity and its ritual in Bang Chan*. New Haven: Human Relations Area Files.

HANSEN, P. J. 1998. Regulation of uterine immune function by progesterone—lessons from the sheep. *Journal of Reproductive Immunology* 40:63–79.

HANSEN, R., AND L. GLASS. 1936. Über den Geruchssinn in

der Schwangerschaft (On changes in smell during pregnancy). *Klinische Wochenschrift* 15:891–94.

HANSEN, R., AND W. LANGER. 1935. Über Geschmacksveränderungen in der Schwangerschaft (On changes in taste during pregnancy). *Klinische Wochenschrift* 14:1173–76.

HARNER, M. J. 1972. *The Jívaro, people of the sacred waterfalls*. Garden City, N.Y.: American Museum of Natural History by Doubleday/Natural History Press.

HARRIES, J. M., AND T. F. HUGHES. 1957. An enumeration of the "cravings" of some pregnant women. *Proceedings of the Nutrition Society* 16:xx–xxi.

HART, J., E. COHEN, A. GINGOLD, AND R. HOMBURG. 1991. Sexual behavior in pregnancy: A study of 219 women. *Journal of Sex Education and Therapy* 17:86–90.

HEJLÍCEK, K., I. LITERÁK, AND J. NEZVAL. 1997. Toxoplasmosis in wild mammals from the Czech Republic. *Journal of Wildlife Diseases* 33:480–85.

HELITZER-ALLEN, DEBORAH L., CARL KENDAL, AND JACK J. WIRIMA. 1997. "Beliefs and practices during pregnancy in a Yao community in Malawi," in *Issues and perspectives in health care in contemporary Sub-Saharan Africa*. Edited by Ezekiel Kalipeni and Philip Thiuri, pp. 145–64. Lewiston, Queenston, and Lampeter: Edwin Mellen Press.

HILGER, M. INEZ. 1951. *Chippewa child life and its cultural background*. Washington, D.C.: U.S. Government Printing Office.

HILL, A. J., C. F. WEAVER, AND J. E. BLUNDELL. 1991. Food cravings, dietary restraint, and mood. *Appetite* 17:187–97. [BJT, SC]

HILL, K. 1982. Hunting and human evolution. *Journal of Human Evolution* 11:521–44.

HOCHSTEIN, G. 1968. "Pica: A study in medical and anthropological explanation," in *Essays in medical anthropology*. Edited by T. Weaver, pp. 88–96. Athens: University of Georgia Press.

HOJO, M., M. SUTHANTHIRAN, G. HELSETH, AND P. AUGUST. 1999. Lymphocyte intracellular free calcium concentration is increased in preeclampsia. *American Journal of Obstetrics and Gynecology* 180:1209–14.

HOLLÁN, S., AND K. S. JOHANSEN. 1993. Adequate iron stores and the "Nil nocere" principle. *Haematologia* 25:69–84.

HOOK, E. B. 1976. "Changes in tobacco smoking and ingestion of alcohol and caffeinated beverages during early pregnancy: Are these consequences, in part, of feto-protective mechanisms diminishing maternal exposure to embryotoxins?" in *Birth defects: Risks and consequences*. Edited by S. Kelly et al., pp. 173–83. New York: Academic Press.

———. 1978. Dietary cravings and aversions during pregnancy. *American Journal of Clinical Nutrition* 31:1355–62.

———. 1980. Influence of pregnancy on dietary selection. *International Journal of Obesity* 4:338–40.

HU, S., J. J. LAGOMARSINO, AND Y.-J. LUO. 1998. Drinking milk or water has no effect on the severity of optokinetic rotation-induced symptoms of motion sickness. *Aviation, Space, and Environmental Medicine* 69:1158–61.

HUWYLER, T., A. HIRT, AND A. MORELL. 1985. Effect of ascorbic acid on human natural killer cells. *Immunology Letters* 10:173–76.

HUXLEY, R. R. 2000. Nausea and vomiting in early pregnancy: Its role in placental development. *Obstetrics and Gynecology* 95:779–82.

INGHAM, J. M. 1986. *Mary, Michael, and Lucifer: Folk Catholicism in central Mexico*. Austin: University of Texas Press.

ISRAEL, RON, AND JOANNE NESTOR. Editors. 1981*a*. *Maternal and infant nutrition reviews: Zaire, a guide to the literature*. Newton, Mass.: International Nutrition Communication Service.

———. 1981*b*. *Maternal and infant nutrition reviews: Liberia, a guide to the literature*. Newton, Mass.: International Nutrition Communication Service.

———. 1982*a*. *Maternal and infant nutrition reviews: Tanzania, a guide to the literature*. Newton, Mass.: International Nutrition Communication Service.

———. 1982*b*. *Maternal and infant nutrition reviews: Cameroon, a guide to the literature*. Newton, Mass.: International Nutrition Communication Service.

———. 1982*c*. *Maternal and infant nutrition reviews: Ghana, a guide to the literature*. Newton, Mass.: International Nutrition Communication Service.

JACKSON, JEAN E. 1983. *The fish people: Linguistic exogamy and Tukanoan identity in northwest Amazonia*. Cambridge and New York: Cambridge University Press.

JAMES, W. H. 2001. The associated offspring sex ratios and cause(s) of hyperemesis gravidarum. *Acta Obstetricia et Gynecologica Scandinavica* 80:378–79.

JARNFELT-SAMSIOE, A., K. BREMME, AND P. ENEROTH. 1986. Steroid hormones in emetic and non-emetic pregnancy. *European Journal of Obstetrics and Gynecology and Reproductive Biology* 21:87–100.

JARNFELT-SAMSIOE, A., G. SAMSIOE, AND G. M. VELINDER. 1983. Nausea and vomiting in pregnancy: A contribution to its epidemiology. *Gynecologic and Obstetric Investigation* 16:221–29.

JEANS, P. C., M. B. SMITH, AND G. STEARNS. 1952. Dietary habits of pregnant women of low income in a rural state. *Journal of the American Dietetic Association* 28:27–34.

JEDNAK, M. A., E. M. SHADIGIAN, M. S. KIM, M. L. WOODS, F. G. HOOPER, C. OWYANG, AND W. L. HASLER. 1999. Protein meals reduce nausea and gastric slow wave dysrhythmic activity in first trimester pregnancy. *American Journal of Physiology* 277:G855–61.

JELLIFFE, E. F. P., AND D. B. JELLIFFE. 1964. Children in ancient Polynesian Hawaii. *Clinical Pediatrics* 3:604–13.

JENG, K. C., C. S. YANG, W. Y. SIU, Y. S. TSAI, W. J. LIAO, AND J. S. KUO. 1996. Supplementation with vitamins C and E enhances cytokine production by peripheral blood mononuclear cells in healthy adults. *American Journal of Clinical Nutrition* 64:960–65.

JOHNS, T., AND M. DUQUETTE. 1991. Detoxification and mineral supplementation as functions of geophagy. *American Journal of Clinical Nutrition* 53:448–56.

JOHNSON, ALLEN. n.d. *Families of the forest: The Matsigenka Indians of the Peruvian Amazon*. Los Angeles: University of California Press.

JOHNSON, M. H., AND B. I. EVERITT. 1995. 4th edition. *Essential reproduction.* Oxford: Blackwell Science. [KO]

JOHNSON, M. R., A. ABBAS, AND K. H. NICOLAIDES. 1994. Maternal plasma levels of human chorionic gonadotrophin, oestradiol, and progesterone in multifetal pregnancies before and after fetal reduction. *Journal of Endocrinology* 143: 309–12.

JONES, LIVINGSTON F. 1914. *A study of the Thlingets of Alaska*. New York: Fleming H. Revell.

JONES, J. L., A. LOPEZ, M. WILSON, J. SCHULKIN, AND R. GIBBS. 2001. Congenital toxoplasmosis: A review. *Obstetrical and Gynecological Survey* 56:296–305.

JORDAN, V., S. K. GREBE, R. R. COOKE, H. C. FORD, P. D. LARSEN, P. R. STONE, AND C. E. SALMOND. 1999. Acidic isoforms of chorionic gonadotrophin in European and Samoan women are associated with hyperemesis gravidarum and may be thyrotrophic. *Clinical Endocrinology* 50:619–27.

KADYROV, M., G. KOSANKE, J. KINGDOM, AND P. KAUFMANN. 1998. Increased fetoplacental angiogenesis during first trimester in anaemic women. *Lancet* (North American Edition) 352:1747–49.

KAGWA, APOLO, ERNEST B. KALIBALA, MAY EDEL, AND JOHN ROSCOE. 1934. *The customs of the Baganda*. New York: Columbia University Press.

KAN, SERGEI. 1989. *Symbolic immortality: The Tlingit potlatch of the nineteenth century*. Washington, D.C.: Smithsonian Institution Press.

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.

KAR, R. K. 1993. Reproductive health behavior of Nocte women in Arunachal Pradesh. *Social Change* 23(4): 40–52.

KARSTEN, RAFAEL. 1932. *Indian tribes of the Argentine and Bolivian Chaco: Ethnological studies*. Helsingfors: Akademische Buchhandlung.

KATON, W. J., R. K. RIES, J. A. BOKAN, AND A. KLEINMAN. 1980–81. Hyperemesis gravidarum: A biopsychological perspective. *International Journal of Psychiatry in Medicine* 10:151–62 [BL]

KAUFMAN, H. K. 2000 (1960). *Bangkhuad: A community study in Thailand*. Locust Valley, N.Y.: Association for Asian Studies/New Haven: Human Relations Area Files.

KAUPPILA, A., M. HEIKINHEIMO, H. LOHELA, AND O. YLIKORKALA. 1984. Human chorionic gonadotropin and pregnancy-specific beta-1-glycoprotein in predicting pregnancy outcome and in association with early pregnancy vomiting. *Gynecologic and Obstetric Investigation* 18:49–53. [KO]

KLEBANOFF, M. A., P. A. KOSLOWE, R. KASLOW, AND G. G. RHOADS. 1985. Epidemiology of vomiting in early pregnancy. *Obstetrics and Gynecology* 66:612–16.

KNOX, B., J. KREMER, AND J. PEARCE. 1994. Food fads during pregnancy: A question of taste? *Appetite* 23:288.

———. 1995. A survey of dietary urges and consumption during pregnancy in Belfast working-class women. *Social Sciences in Health* 1:125–44.

KRACKE, WAUD H. 1981. "Don't let the piranha bite your liver: A psychoanalytic approach to Kagwahiv (Tupi) food taboos," in *Working papers on South American Indians*. Edited by K. Kensiger and W. Kracke, pp. 91–142. Bennington, Vt.: Bennington College.

KRISHNAMANI, R., AND W. C. MAHANEY. 2000. Geophagy among primates: Adaptive significance and ecological consequences. *Animal Behaviour* 59:899–915.

KUMAR, R., H. A. BRANT, AND K. M. ROBSON. 1981. Childbearing and maternal sexuality: A prospective survey of 119 primiparae. *Journal of Psychosomatic Research* 25:373–83.

KUMAR, M. L., AND S. L. PROKAY. 1983. Experimental primary cytomegalovirus infection in pregnancy: Timing and fetal outcome. *American Journal of Obstetrics and Gynecology* 145: 56–60.

LACROIX, R., E. EASON, AND R. MELZACK. 2000. Nausea and vomiting during pregnancy: A prospective study of its frequency, intensity, and patterns of change. *American Journal of Obstetrics and Gynecology* 182:931–37.

LADERMAN, C. 1981. Symbolic and empirical reality: A new approach to the analysis of food avoidances. *American Ethnologist* 8:463–93.

LAGUERRE, MICHEL S. 1984. *American odyssey: Haitians in New York City*. Ithaca: Cornell University Press.

LANGLEY-EVANS, S. C., S. J. WELHAM, R. C. SHERMAN, AND A. A. JACKSON. 1996. Weanling rats exposed to material low-protein diets during discrete periods of gestation exhibit differing severity of hypertension. *Clinical Science* 91: 607–15.

LANTELME, P., I. MOHAMMEDI, S. DUPERRET, J. M. VEDRINNE, B. ALLAOUCHICHE, AND J. MOTIN. 1995. Septicémie à *Clostridium perfringens* associée à une toxi-infection alimentaire et un avortement. *Annales Françaises d'Anesthésie et de Réanimation* 14:359–61.

LASKA, M., B. KOCH, B. HEID, AND R. HUDSON. 1996. Failure to demonstrate systematic changes in olfactory perception in the course of pregnancy: A longitudinal study. *Chemical Senses* 21:567–71.

LEE, RICHARD. 1968. "What hunters do for a living, or How to make out on scarce resources," in *Man the hunter*. Edited by Richard Lee and Irven DeVore, pp. 30–48. Chicago: Aldine. [JKB]

LEENERS, B., I. SAUER, AND W. RATH. 2000. Early pregnancy sickness and vomiting/Hyperemesis gravidarum: Actual knowledge regarding psychosomatic aspects. *Zeitschrift für Geburtshilfe und Neonatologie* 204:128–34. [BL]

LESHNER, A. I., H. I. SIEGEL, AND G. COLLIER. 1972. Dietary self-selection by pregnant and lactating rats. *Physiology and Behavior* 8:151–54.

LEVY, R. I. 1973. *Tahitians: Mind and experience in the Society Islands*. Chicago: University of Chicago Press.

LINDSETH, G., AND P. D. LINDSETH. 1995. The relationship of diet to airsickness. *Aviation, Space, and Environmental Medicine* 66:537–41.

LIU, X., HAN, XIAOJIC, LI, AND YUJIU. 1993. Determination of phytic acid in common plant foods. *Journal of China Medical University* 22:110–12.

LIULAN, WANG. 1999. "Food and herbal medicine for childbirth care among the Chinese minority in northern Thailand," in *The Tokyo International Forum on Conservation and Sustainable Use of Tropical Bioresources*. Tokyo: New Energy and Industrial Technology Development Organization.

LOGUE, A. W., I. OPHIR, AND K. E. STRAUSS. 1981. The acquisition of taste aversions in humans. *Behavior Research and Therapy* 19:319–33. [TMB, LD]

LOKE, Y. W., AND A. KING. 1997. Immunology of human placental implantation: Clinical implications of our current understanding. *Molecular Medicine Today* 3:153–59.

LOPEZ-GONI, I., AND I. MORIYON. 1995. Production of 2,3-dihydroxybenzoic acid by *Brucella* species. *Current Microbiology* 31:291–93.

LOPEZ-JARAMILLO, P. 2000. Calcium, nitric oxide, and preeclampsia. *Seminars in Perinatology* 24:33–36.

LU, C. Y., R. W. REDLINE, C. M. SHEA, L. B. DUSTIN, AND D. B. MC KAY. 1989. Pregnancy as a natural model of allograft tolerance: Interactions between adherent macrophages and trophoblast populations. *Transplantation* 48:848–55.

LUMEY, L. H. 1998. Compensatory placental growth after restricted material nutrition in early pregnancy. *Placenta* 19: 105–11.

LYNCH, S. R., AND J. D. COOK. 1980. Interaction of vitamin C and iron. *Annals of the New York Academy of Sciences* 355: 32–44.

LYNCH, S. R., S. A. DASSENKO, T. A. MORCK, J. L. BEARD, AND J. D. COOK. 1985. Soy protein products and heme iron absorption in humans. *American Journal of Clinical Nutrition* 41:13–20.

MC CAFFERY, R. J. 1985. Appropriateness of kaolin consumption as an index of motion sickness in the rat. *Physiology and Behavior* 35:151–56.

MACINTYRE, S. 1983. "The management of food in pregnancy," in *The sociology of food and eating*. Edited by A. Murcott. Aldershot: Gower Publishing.

MAIR, LUCY, P. 1934. *An African people in the twentieth century*. London: Routledge

MAKHSEED, M., A. HAROUNY, G. ARAJ, M. A. MOUSSA, AND P. SHARMA. 1998. Obstetric and gynecologic implication of brucellosis in Kuwait. *Journal of Perinatology* 18: 196–99.

MALINOWSKI, BRONISLAW. 1929. *The sexual life of savages in northwestern Melanesia*. 2 vols. New York: Horace Liveright.

MAN, EDWARD H. 1975. *Aboriginal inhabitants of Andaman Islands*. Delhi: Sanskaran Prakashak.

MANDERSON, LENORE. 1981. Vietnamese behavioral and dietary precautions during pregnancy. *Ecology of Food and Nutrition* 11(1):145–53.

MANN, N. 2000. Dietary lean red meat and human evolution. *European Journal of Nutrition* 39:71–79.

MARITZ, G. S., AND G. VAN WYK. 1997. Influence of maternal nicotine exposure on neonatal rat lung structure: Protective effect of ascorbic acid. *Comparative Biochemistry and Physiology C: Pharmacology, Toxicology, and Endocrinology* 117:159–65.

MARZI, M., A. VIGANO, D. TRABATTONI, M. L. VILLA, A. SALVAGGIO, E. CLERICI, AND M. CLERICI. 1996. Characterization of type 1 and type 2 cytokine production profile in physiologic and pathologic human pregnancy. *Clinical and Experimental Immunology* 106:127–33.

MASCOLA, L., D. P. EWERT, AND A. ELLER. 1994. Listeriosis: A previously unreported medical complication in women with multiple gestations. *American Journal of Obstetrics and Gynecology* 170:1328–32.

MASSON, G. M., G. ANTHONY, AND E. CHAU. 1985. Serum

chorionic gonadotropin, schwangerschaftsprotein 1, progesterone, and estradiol levels in patients with nausea and vomiting in early pregnancy. *British Journal of Obstetrics and Gynaecology* 92:211–15.

MATHEWS, F., P. YUDKIN, AND A. NEIL. 1999. Influence of maternal nutrition on outcome of pregnancy: Prospective cohort study *British Medical Journal* 319:339–43.

MATTES, R. D. 1991. Learned food aversions: A family study. *Physiology and Behavior* 50:499–504.

MATTHIESEN, L., C. EKERFELT, G. BERG, AND J. ERNERUDH. 1998. Increased numbers of circulating interferon-gamma- and interlukin-4-secreting cells during normal pregnancy. *American Journal of Reproductive Immunology* 39: 362–67.

MEAD, M. 1949. *Male and female: A study of the sexes in a changing world*. New York: William Morrow.

MERKER, MERITZ. 1910. *The Masai: Ethnographic monograph of an East African Semite people*. Berlin: Dietrich Reimer (Ernst Vohsen).

MIDKIFF, E. E., AND I. L. BERNSTEIN. 1985. Targets of learned food aversions in humans. *Physiology and Behavior* 34: 839–41. [TMB, LD]

MILES, A. A., AND J. P. MASKELL. 1985. The antagonism of tetracycline and ferric iron in vivo. *Journal of Medical Microbiology* 20:17–26.

MILMAN, N., N. GRAUDAL, AND A. O. AGGER. 1995. Iron status markers during pregnancy: No relationship between levels at the beginning of the second trimester, prior to delivery, and post partum. *Journal of Internal Medicine* 237:261–67.

MILTON, K. 1999. A hypothesis to explain the role of meat-eating in human evolution. *Evolutionary Anthropology* 8:11–21.

MINTURN, L., AND A. W. WEIHER. 1984. The influence of diet on morning sickness: A cross-cultural study. *Medical Anthropology* 8:71–75.

MITCHELL, D., W. WINTER, AND C. M. MORISAKI. 1977. Conditioned taste aversions accompanied by geophagia: Evidence for the occurrence of psychological factors in the etiology of pica. *Psychosomatic Medicine* 39:402–12.

MIZUTANI, A., H. MAKI, Y. TORII, K. HITOMI, AND N. TSUKAGOSHI. 1998. Ascorbate-dependent enhancement of nitric oxide formation in activated macrophages. *Nitric Oxide* 2:235–41.

MONTGOMERY, E. 1990. Iron levels in pregnancy, physiology or pathology? Assessing the needs for supplements. *Midwifery* 6:205–14.

MORAIS, M. B., A. FESTE, R. G. MILLER, AND C. H. LIFSCHITZ. 1996. Effect of resistant and digestible starch on intestinal absorption of calcium, iron, and zinc in infant pigs. *Pediatric Research* 39:872–76.

MORI, M., A. NOBUYUKI, H. TAMAKI, K. MIYAI, AND O. TANIZAWA. 1988. Morning sickness and thyroid function in normal pregnancy. *Obstetrics and Gynecology* 72:355–59. [KO]

MORTON, H. n.d. Pregnancy and childbirth in Tonga. MS.

MOYO, S. R., S. A. TSWANA, L. NYSTROM, S. BERGSTROM, J. BLOMBERG, AND A. LJUNGH. 1995. Intrauterine death and infections during pregnancy. *International Journal of Gynaecology and Obstetrics* 51:211–18.

MUAKU, S. M., L. E. UNDERWOOD, P. L. SELVAIS, J. M. KETELSLEGERS, AND D. MAITER. 1995. Maternal protein restriction early or late in rat pregnancy has differential effects on fetal growth, plasma insulin-like growth factor-I (IGF-I), and liver IGF-I gene expression. *Growth Regulation* 5:125–32.

MWENYE, S., N. GUMBO, M. MAVELA, D. E. PETERSON, S. SIZIYA, AND G. WOELK. 1997. Risk factors associated with contracting dysentery during *Shigella dysenteriae* type 1 outbreak in Harare, 1993. *Central African Journal of Medicine* 43:111–12.

NAKANE, A., K. YAMADA, S. HASEGAWA, D. MIZUKI, M. MIZUKI, S. SASAKI, AND T. MIURA. 1999. Endogenous cytokines during a lethal infection with *Listeria monocytogenes* in mice. *FEMS Microbiology Letters* 175:133–42.

NAPPI, R E., M. J. BONNEAU, AND S. RIVEST. 1997. Influence of the estruous cycle on c-*fos* and CRF gene transcription in the brain of endotoxin-challenged female rats. *Neuroendocrinology* 65:29–46. [REN]

NAPPI, R. E., AND S. RIVES. 1995. CRF and stress-related reproductive failure: The brain as a state of the art of the ovary as a novel clue? *Journal of Endocrinological Investigation* 18: 872–80. [REN]

NESSE, R. M., AND G. C. WILLIAMS. 1995. *Why we get sick: The new science of Darwinian medicine*. New York: Times Books.

NICHTER, M., AND M. NICHTER. 1983. The ethnophysiology and folk dietetics of pregnancy: A case study from South India. *Human Organization* 42:235–45. [DWS]

NORDENSKIÖLD, ERLAND. 1998 (1938). *An historical and ethnological survey of the Cuna Indians*. New Haven: Human Relations Area Files.

NORGAN, N. G., A. FERRO-LUZZI, AND J. V. DURNIN. 1974. The energy and nutrient intake and the energy expenditure of 204 New Guinean adults. *Philosophical Transactions of the Royal Society of London B Biological Sciences* 268:309–48.

NUNN, C. L., J. L. GITTLEMAN, AND J. ANTONOVICS. 2000. Promiscuity and the primate immune system. *Science* 290:1168–70.

OBEYESEKERE, G. 1981. *Medusa's hair: An essay on personal symbols and religious experience*. Chicago: University of Chicago Press.

———. 1985. Symbolic foods: Pregnancy cravings and the envious female. *International Journal of Psychology* 20:637–62.

O'BRIEN, B., J. RELYEA, AND T. LIDSTONE. 1997. Diary reports of nausea and vomiting during pregnancy. *Clinical Nursing Research* 6:239–52.

O'CONNOR, K. A. 1999. Review of: "Pregnancy sickness: Using your body's natural defenses to protect your baby-to-be," by Margie Profet. *American Journal of Physical Anthropology* 108: 377–79. [KO]

O'CONNOR, K. A., E. BRINDLE, D. J. HOLMAN, N. A. KLEIN, M. R. SOULES, K. L. CAMPBELL, F. KOHEN, C. J. MUNRO, W. L. LASLEY, AND J. W. WOOD. n.d. Urinary enzyme-immunoassays for population-based research on reproduction: Estrone-3-glucuronide and pregnanediol-3-glucuronide. MS.

O'CONNOR, K. A., D. J. HOLMAN, E. BRINDLE, S. H. BARSOM, AND J. W. WOOD. 1998. Reproductive hormones and pregnancy-related sickness in a prospective study of Bangladeshi women. *American Journal of Physical Anthropology* suppl. 26:172.

OLDENBORG, P. A., A. ZHELEZNYAK, Y. F. FANG, C. F. LAGENAUR, H. D. GRESHAM, AND F. P. LINDBERG. 2000. Role of CD47 as a marker of self on red blood cells. *Science* 288:2051–54.

OLIVEIRA, S. C., J. S. HARMS, E. L. RECH, R. S. RODARTE, A. L. BOCCA, A. M. GOES, AND G. A. SPLITTER. 1998. The role of T cell subsets and cytokines in the regulation of intracellular bacterial infection. *Brazilian Journal of Medical and Biological Research* 31:77-84.

OLLIS, T. E., B. J. MEYER, AND P. R. HOWE. 1999. Australian food sources and intakes of omega-6 and omega-3 polyunsaturated fatty acids. *Annals of Nutrition and Metabolism* 43: 346–55.

OMINDE, SIMEON H. 1952. *The Luo girl: From infancy to marriage*. London: Macmillan.

OPPENHEIMER, S. J. 2001. Iron and its relation to immunity and infectious disease. *Journal of Nutrition* 131:616S–35S. [DWS]

ORTEGA, R. M., M. E. QUINTAS, P. ANDRES, R. M. MARTINEZ, AND A. M. LOPEZ-SOBALER. 1998. Ascorbic acid levels in maternal milk: Differences with respect to ascorbic acid status during the third trimester of pregnancy. *British Journal of Nutrition* 79:431–37.

OSTRAFF, M., K. ANITONI, A. NICHOLSON, AND G. BOOTH. 2000. Traditional Tongan cures for morning sickness and their mutagenic/toxicological evaluations. *Journal of Ethnopharmacology* 71:201–9.

PANCORBO, P. L., M. A. DE PABLO, E. ORTEGA, A. M. GALLEGO, C. ALVAREZ. AND G. A. DE CIENFUEGOS.

1999. Evaluation of cytokine production and phagocytic activity in mice infected with *Campylobacter jejuni*. *Current Microbiology* 39:129–33.

PANIKKAR, K. M. 1918. Some aspects of Nayar life. *Royal Anthropological Institute of Great Britain and Ireland* 48:254–93.

PARADOWSKA, E., Z. BACH-OLSZEWSKA, J. SENDER, AND W. JAROSZ. 1996. Antiviral nonspecific immunity of human placenta at term: Possible role of endogenous tumor necrosis factors and interferons. *Journal of Interferon and Cytokine Research* 16:941–48.

PARSONS, E. C. 1945. *Peguche, Canton of Otavale, Province of Imbabura, Ecuador: A study of Andean Indians*. Chicago: University of Chicago Press.

PATTERSON, E. C., AND D. J. STASZAK. 1977. Effects of geophagia (kaolin ingestion) on the maternal blood and embryonic development in the pregnant rat. *Journal of Nutrition* 107: 2020–25.

PAULME, D. 2000 (1940). *Social organization of the Dogon*. New Haven: Human Relations Area Files.

PAVLOVIC, J. M. 1973. *Folk life and customs in the Kragujevac region of the Jasenica in Sumdaija*. New Haven: Human Relations Area Files.

PEIRSON, DONALD. 1998 (1967). *Negroes in Brazil.* New Haven: Human Relations Area Files.

PELCHAT, M. L., AND S. SCHAEFER. 2000. Dietary monotony and food cravings in young and elderly adults. *Physiology and Behavior* 68:353–59.

PEPIN, M., P. RUSSO, AND P. PARDON. 1997. Public health hazards from small ruminant meat products in Europe. *Review of Science and Technology* 16:415–25.

PERAICA, M., B. RADIĆ, A. LUCIĆ, AND M. PAVLOVIĆ. 1999. Toxic effects of mycotoxins in humans. *Bulletin of the World Health Organization* 77:754–66.

PEREIRA, I. M., M. J. RONCADA, G. T. STEWIEN, AND M. TRIGO. 1989. Tabus alimentares em região do Norte do Brasil. *Revista de Saude Publica* 23:455–64.

PERRY, D. F., J. DIPIETRO, AND K. COSTIGAN. 1999. Are women carrying "basketballs" really having boys? Testing pregnancy folklore. *Birth* 26:172–77.

PERRY, V. E., S. T. NORMAN, J. A. OWEN, R. C. DANIEL, AND N. PHILLIPS. 1999. Low dietary protein during early pregnancy alters bovine placental development. *Animal Reproduction Science* 55:13–21.

PETROS, S., U. LEONHARDT, AND L. ENGELMANN. 2000. Serum procalcitonin and proinflammatory cytokines in a patient with acute severe leptospirosis. *Scandinavian Journal of Infectious Diseases* 32:104–5.

PIKE, I. L. 1997. Pregnancy sickness and food aversions during pregnancy for nomadic Turkana women of Kenya. *American Journal of Physical Anthropology*, suppl. 24, p. 186.

———. 1999. The nutritional consequences of pregnancy sickness: A critique of a hypothesis. *Human Nature* 11:207–32. [DWS]

POOL, R. 1986. Beliefs concerning the avoidance of food during pregnancy and the immediate post partum period in a tribal area of rural Gujarat, India. *Eastern Anthropologist* 39:251–59.

POPE, J. F., J. D. SKINNER, AND B. R. CARRUTH. 1992. Cravings and aversions of pregnant adolescents. *Journal of the American Dietetic Association* 92:1479–82.

———. 1997. Adolescents' self-reported motivations for dietary changes during pregnancy. *Journal of Nutrition Education* 29: 137–44.

POSNER, L. B., C. M. MC COTTRY, AND A. C. POSNER. 1957. Pregnancy craving and pica. *Obstetrics and Gynecology* 9:270–72.

PRENTICE, A. M., AND R. G. WHITEHEAD. 1987. "The energetics of human reproduction," in *Reproductive energetics in mammals*. Edited by A. S. I. Loudon and P. A. Racey, pp. 275–304. New York: Oxford University Press.

PRICE, R., AND S. PRICE. 1991. *Two evenings in Saramaka*. Chicago: University of Chicago Press.

PRITHAM, URSULA A., AND LUCY N. SAMMONS. 1993. Korean women's attitudes toward pregnancy and prenatal care. *Health Care for Women International* 14:145–53.

PROCTOR, T., R. J. GATCHEL, AND R. C. ROBINSON. 2000. Psychosocial factors and risk of pain and disability. *Occupational Medicine* 15:803–12.

PROFET, M. 1988. The evolution of pregnancy sickness as protection to the embryo against Pleistocene teratogens. *Evolutionary Theory* 8:177–90.

———. 1992. "Pregnancy sickness as adaptation: A deterrent to maternal ingestion of teratogens," in *The adapted mind: Evolutionary psychology and the generation of culture*. Edited by L. C. J. Barkow and J. Tooby, pp. 327–65. New York: Oxford University Press.

RAINVILLE, A. J. 1998. Pica practices of pregnant women are associated with lower maternal hemoglobin level at delivery. *Journal of the American Dietetic Association* 98:293–96.

RANCÉ, F., G. KANNY, G. DUTAU, AND D. A. MONERET-VAUTRIN. 1999. Food hypersensitivity in children: Clinical aspects and distribution of allergens. *Pediatric Allergy and Immunology* 10:33–38.

REHÁKOVÁ, Z., I. TREBICHAVSKY, J. SINKORA, I. SPLÍCHAL, AND M. SINKORA. 1998. Early ontogeny of monocytes and macrophages in the pig. *Physiological Research* 47: 357–63.

REICHEL-DOLMATOFF, GERARDO. 1971. *Amazonian cosmos: The sexual and religious symbolism of the Tukano Indians*. Chicago: University of Chicago Press.

REID, R. M. 1992. Cultural and medical perspectives on geophagia. *Medical Anthropology* 13:337–51.

REIS, F. M., L. COBELLIS, S. LUISI, L. DRIUL, P. FLORIO, A. FALETTI, AND F. PETRAGLIA. 2000. Paracrine/autocrine control of female reproduction. *Gynecology and Endocrinology* 14:464–75. [REN]

REMINGTON, J. S., R. MC LEOD, AND G. DESMONTS. 1995. "Toxoplasmosis," in *Infectious diseases of the fetus and newborn infant*, 4th edition. Edited by J. S. Remington and J. O. Klein, pp. 140–267. Philadelphia: W. B. Saunders.

RICHARDS, A. I. 1939. *Land, labour, and diet in Northern Rhodesia: An economic study of the Bemba tribe*. London: Oxford University Press.

RINGLER, M., AND A. KRIZMANITS. 1983. Psychosomatic aspects of emesis gravidarum: Perceptual and attitudinal patterns of women during early pregnancy. *Zeitschrift für Geburtshilfe und Perinatologie* 187:246–49. [BL]

RITCHIE, L. D., E. B. FUNG, B. P. HALLORAN, J. R. TURNLUND, M. D. VAN LOAN, C. E. CANN, AND J. C. KING. 1998. A longitudinal study of calcium homeostasis during human pregnancy and lactation and after resumption of menses. *American Journal of Clinical Nutrition* 67:693–701.

RIVEST, S. 1995. Molecular mechanisms and neuronal pathways mediating the influence of interleukin-1 on the activity of neuroendocrine CRH motoneurons in the rat. *International Journal of Developmental Neuroscience* 13:135–46. [REN]

RIZVI, N. 1980. Rural and urban food behavior in Bangladesh: An anthropological perspective to the problem of malnutrition. Ph.D. diss., University of California, Los Angeles, Calif.

ROBERTSON, G. G. 1946. Nausea and vomiting of pregnancy. *Lancet* 251:336. [BL]

ROBSON, K. M., H. A. BRANT, AND R. KUMAR. 1981. Maternal sexuality during first pregnancy and after childbirth. *British Journal of Obstetrics and Gynaecology* 88:882–89.

RODIN, J. 1989. Cravings and aversions during pregnancy. *Appetite* 12:76–77.

RODIN, J., AND N. RADKE-SHARPE. 1991. "Changes in appetite variables as a function of pregnancy," in *Chemical senses: Appetite and nutrition*. Edited by M. Friedman, M. G. Tordoff, and M. Kare, pp. 325–40. New York: Marcel Dekker.

ROEMER, H. 1967. "Psychisch bedingte Störungen in der Schwangerschaft: Hyperemesis gravidarum," in *Gynäkologie und Geburtshilfe*, vol. 2. Edited by O. Käser, V. Friedberg, K. G. Ober, K. Thomsen, and J. Zander. Stuttgart: Thieme-Verlag. [BL]

RÓHEIM, G. 1933. Women and their life in central Australia.

Journal of the Royal Anthropological Institute of Great Britain and Ireland 63:207–65.

ROSENGAUS, R. B., A. B. MAXMEN, L. E. COATES, AND J. F. A. TRANIELLO. 1998. Disease resistance: A benefit of sociality in the dampwood termite *Zootermopsis angusticollis* (Isoptera: Termopsidae). *Behavioral Ecology and Sociobiology* 44:125–34.

ROSENGAUS, R. B., J. F. A. TRANIELLO, T. CHEN, AND J. J. BROWN. 1999. Immunity in a social insect. *Naturwissenschaften* 86:588–91.

ROTH, I., D. B. CORRY, R. M. LOCKSLEY, J. S. ABRAMS, M. J. LITTON, AND S. J. FISHER. 1996. Human placental cytotrophoblasts produce the immunosuppressive cytokine interleukin 10. *Journal of Experimental Medicine* 184:539–48.

ROZIN, P. 1990. "The importance of social factors in understanding the acquisition of food habits," in *Taste, experience, and feeding*. Edited by E. D. Capaldi, T. L. Powley, and et al., pp. 255–69. Washington, D.C.: American Psychological Association.

SAITO, S., H. UMEKAGE, Y. SAKAMOTO, M. SAKAI, K. TANEBE, Y. SASAKI, AND H. MORIKAWA. 1999. Increased T-helper-1-type immunity and decreased T-helper-2-type immunity in patients with preeclampsia. *American Journal of Reproductive Immunology* 41:297–306.

SANDERS, IRWIN T. 1949. *Balkan village*. Lexington: University of Kentucky Press.

SANDERS, T. 1994. Good nutrition for the vegetarian mother. *Modern Midwife* 4:23–26.

SANSONETTI, P. J., J. ARONDEL, M. HUERRE, A. HARADA, AND K. MATSUSHIMA. 1999. Interleukin-8 controls bacterial transepithelial translocation at the cost of epithelial destruction in experimental shigellosis. *Infection and Immunity* 67:1471–80.

SARASUA, S., AND D. A. SAVITZ. 1994. Cured and broiled meat consumption in relation to childhood cancer: Denver, Colorado (United States). *Cancer Causes Control* 5:141–48.

SCHIEFFELIN, BAMBI B. 1990. *The give and take of everyday life: Language socialization of Kaluli children*. New York: Cambridge University Press.

SCHOENECK, F. J. 1942. Gonadotropic hormone concentration in emesis gravidarum. *American Journal of Obstetrics and Gynecology* 43:308–12. [KO]

SCHWAB, E. B., AND M. L. AXELSON. 1984. Dietary changes of pregnant women: Compulsions and modifications. *Ecology of Food and Nutrition* 14:143–53.

SCHWAGER, J., AND J. SCHULZE. 1998. Modulation of interleukin production by ascorbic acid. *Veterinary Immunology and Immunopathology* 64:45–57.

SCHWARTZ, D., R. PERRY, D. M. DOMBROSKI, J. M. MERRICK, AND J. GOLDHAR. 1996. Invasive ability of *C. jejuni/coli* isolates from children with diarrhea and the effect of iron-regulated proteins. *Zentralblatt für Bakteriologie* 283: 485–91

SCHWARTZ, W. J., III, AND G. R. THURNAU. 1995. Iron deficiency anemia in pregnancy. *Clinical Obstetrics and Gynecology* 38:443–54.

SECHSER, T., J. VANECEK, H. RASKOVA, Z. JIRICKA, D. MATEJOVSKA, AND V. MATEJOVSKA. 1976. Pregnancy interrupting effects of some bacterial toxins. *Physiologia Bohemoslovaca* 25:155–58.

SELIGMANN, C. G. 1910. *The Melanesians of British New Guinea*. Cambridge: Cambridge University Press.

SENOGLES, D. R., P. S. PAUL, D. W. JOHNSON, AND C. C. MUSCOPLAT. 1979. Ontogeny of T Cells, B cells, and monocytes in the bovine foetus. *Clinical and Experimental Immunology* 36:299–303.

SHAKED, Y., O. SHPILBERG, D. SAMRA, AND Y. SAMRA. 1993. Leptospirosis in pregnancy and its effect on the fetus: Case report and review. *Clinical Infectious Diseases* 17: 241–43.

SHERMAN, P. W. 1988. The levels of analysis. *Animal Behaviour* 36:616–19. [PWS]

SHERMAN, P. W., AND J. BILLING. 1999. Darwinian gastronomy: Why we use spices. *Bioscience* 49:453–64. [PWS]

SHERMAN, P. W., AND S. M. FLAXMAN. 2001. Protecting ourselves from food. *American Scientist* 89:142–51. [PWS]

———. n.d. NVP in an evolutionary perspective. *Obstetrics and Gynecology*. In press. [PWS]

SHIBUYA, T., K. IZUCHI, A. KUROIWA, N. OKABE, AND K. SHIRAKAWA. 1987. Study on nonspecific immunity in pregnant women: Increased chemiluminescence response of peripheral blood phagocytes. *American Journal of Reproductive Immunology and Microbiology* 15:19–23.

SHOSTAK, M. 1981. *Nisa, the life and words of a !Kung woman*. Cambridge: Harvard University Press.

SILLEN-TULLBERG, B., AND A. P. MØLLER. 1993. The relationship between concealed ovulation and mating systems in anthropoid primates: A phylogenetic analysis. *American Naturalist* 141:1–25.

SIMOONS, F. J. 1994. 2d edition, revised and enlarged. *Eat not this flesh: Food avoidances from prehistory to the present*. Madison: University of Wisconsin Press.

SIMOR, A. E., AND S. FERRO. 1990. *Campylobacter jejuni* infection occurring during pregnancy. *European Journal of Clinical Microbiology and Infectious Diseases* 9:142–44.

SIMPSON, E., J. D. MULL, E. LONGLEY, AND J. EAST. 2000. Pica during pregnancy in low-income women born in Mexico. *Western Journal of Medicine* 173:20–25.

SIMPSON, S. W., T. M. GOODWIN, S. B. ROBINS, A. A. RIZZO, R. A. HOWES, D. K. BUCKWALTER, AND J. G. BUCKWALTER. 2001. Psychological factors and hyperemesis gravidarum. *Journal of Women's Health and Gender-Based Medicine* 10:471–77.

SINGH, D., AND V. C. ROSEN. n.d. Effects of maternal body morphology, pregnancy complication, and pregnancy sickness on fluctuating asymmetry in young women. *Evolution and Human Behavior*. In press.

SIPIORA, M. L., M. A. MURTAUGH, M. B. GREGOIRE, AND V. B. DUFFY. 2000. Bitter taste perception and severe vomiting in pregnancy. *Physiology and Behavior* 69:259–67.

SMITH, J. L. 1999. Foodborne infections during pregnancy. *Journal of Food Protection* 62:818–29.

SMULIAN, J. C., S. MOTIWALA, AND R. K. SIGMAN. 1995. Pica in a rural obstetric population. *Southern Medical Journal* 88:1236–40.

SOLBERG, D. A., J. BUTLER, AND N. N. WAGNER. 1973. Sexual behavior in pregnancy. *New England Journal of Medicine* 288:1098–1103. [BL]

SOULES, M. R., C. L. HUGHES, J. A. GARCIA, C. H. LIVENGOOD, M. R. PRYSTOWSKY, AND E. ALEXANDER. 1980. Nausea and vomiting of pregnancy: Role of human chorionic gonadotropin and 17-hydroxyprogesterone. *Obstetrics and Gynecology* 55:696–7000. [KO]

SOUTHGATE, D. A. T. 1991. Nature and variability of human food consumption. *Philosophical Transactions of the Royal Society of London B, Biological Sciences* 334:281–88.

SPENCER, P. 1988. *The Maasai of Matapato: A study of rituals of rebellion*. Bloomington: Indiana University Press in association with the International African Institute.

SPETH, J. D. 1991. Protein selection and avoidance strategies of contemporary and ancestral foragers: Unresolved issues. *Philosophical Transactions of the Royal Society of London B, Biological Sciences* 334:265–70.

SPONHEIMER, M., AND J. A. LEE-THORP. 1999. Isotopic evidence for the diet of an early hominid, *Australopithecus africanus*. *Science* 283:368–70.

STANECK, J. L., R. C. HENNEBERRY, AND C. D. COX. 1973. Growth requirements of pathogenic *Leptospira*. *Infection and Immunity* 7:886–97.

STANFORD, C. B. 1999. *The hunting apes: Meat eating and the origins of human behavior*. Princeton: Princeton University Press.

STEIN, Z., AND M. SUSSER. 1991. Miscarriage, caffeine, and the epiphenomena of pregnancy: The causal model. *Epidemiology* 2:163–67. [SMF]

STEVENSON, MARGARET. 1920. *The rites of the twice-born*. London: Oxford University Press.
STITES, D. P., AND P. K. SIITERI. 1983. Steroids as immunosuppressants in pregnancy. *Immunological Review* 75: 117–38.
STOCKMAN, J. A. 1981. Infections and iron. Too much of a good thing? *American Journal of Diseases of Children* 135: 18–20.
SUNDER-PLASSMANN, G., S. I. PATRUTA, AND W. H. HORL. 1999. Pathobiology of the role of iron in infection. *American Journal of Kidney Disease* 34:S25–29.
SUZUKI, Y. 1999. Genes, cells, and cytokines in resistance against development of toxoplasmic encephalitis. *Immunobiology* 201:255–71.
SZEKERES-BARTHO, J. 1990. Endocrine regulation of the immune system during pregnancy. *Archivum Immunologiae et Therapiae Experimentalis* 38:125–40.
SZEKERES-BARTHO, J., AND T. G. WEGMANN. 1996. A progesterone-dependent immunomodulatory protein alters the Th1/Th2 balance. *Journal of Reproductive Immunology* 31: 81–95.
SZEREDAY, L., P. VARGA, AND J. SZEKERES-BARTHO. 1997. Cytokine production by lymphocytes in pregnancy. *American Journal of Reproductive Immunology* 38:418–22.
TAGGART, N. 1961. Food habits in pregnancy. *Proceedings of the Nutrition Society* 20:35–41.
TAREEN, A. K., A. BASEER, H. F. JAFFRY, AND M. SHAF-IQ. 1995. Thyroid hormone in hyperemesis gravidarum. *Journal of Obstetrics and Gynaecology* 21:497–501.
TAYLOR, D. 1950. The meaning of dietary and occupational restriction among the island Carib. *American Anthropologist* 52: 343–49.
TEMKIN, O. Translator. 1991. Softshell Books edition. *Soranus' gynecology*. Baltimore: Johns Hopkins University Press.
TEPPER, B. J., AND A. C. SELDNER. 1999. Sweet taste and dietary intake in normal pregnancy and pregnancy complicated by gestational diabetes mellitus (GDM). *American Journal of Clinical Nutrition* 70:277–84. [BJT, SC]
TEXTOR, R. B. 1973. *Roster of the gods: An ethnography of the supernatural in a Thai village*. New Haven: Human Relations Area Files.
THOMAS, F. B., J. M. FALKO, AND K. ZUCKERMAN. 1976. Inhibition of intestinal iron absorption by laundry starch. *Gastroenterology* 71:1028–32.
THOMPSON, L. 1940. *Fijian frontier*. San Francisco: American Council, Institute of Pacific Relations.
THOUVENIN, M., E. CANDOLFI, O. VILLARD, J. P. KLEIN, AND T. KIEN. 1997. Immune response in a murine model of congenital toxoplasmosis: Increased susceptibility of pregnant mice and transplacental passage of *Toxoplasma gondii* are type 2-dependent. *Parassitologia* (Rome) 39:279–83.
TIERSON, F. D., C. L. OLSEN, AND E. B. HOOK. 1985. Nausea and vomiting of pregnancy and pregnancy outcome. *American Journal of Physical Anthropology* 66:238.
———. 1986. Nausea and vomiting of pregnancy and association with pregnancy outcome. *American Journal of Obstetrics and Gynecology* 155:1017–22.
TOOBY, J., AND L. COSMIDES. 1992. "The psychological foundations of culture," in *The adapted mind: Evolutionary psychology and the generation of culture*. Edited by Jerome H. Barkow, Leda Cosmides, and John Tooby, pp. 19–136. New York: Oxford University Press.
TREEDE, R. D. 1995. Peripheral acute pain mechanisms. *Annals of Medicine* 27:213–16.
TREML, F., AND E. NESNALOVÁ. 1993. Vy'skyt protilázek proti *Leptospirám* v krevních sérech lovné zvere. *Veterinarni Medicina* 38:123–27.
TRIVERS, R. L., AND D. E. WILLARD. 1973. Natural selection of parental ability to vary the sex ratio of offspring. *Science* 179:90–92.
TUNTAWIROON, M., N. SRITONGKUL, L. ROSSANDER-HULTEN, R. PLEEHACHINDA, R. SUWANIK, M. BRUNE, AND L. HALLBERG. 1990. Rice and iron absorption in man. *European Journal of Clinical Nutrition* 44:489–97.
TURNER, PAUL R. 1972. *The highland Chontal*. New York: Holt, Rinehart and Winston.
TUZIN, D. 1986. Sensory contact and moral contagion in Ilahita. Paper delivered at the 85th annual meetings of the American Anthropological Association, Philadelphia, December 3–7.
TYLDEN, E. 1991. Hyperemesis and psychological vomiting. *Journal of Psychosomatic Research* 12:85–93. [BL]
USAMI, M., H. TABATA, AND Y. OHNO. 1999. Effects of ascorbic acid on selenium teratogenicity in cultured rat embryos. *Toxicology Letters* 105:123–28.
VAN BEEK, MALI W. 2000 (1992). *Becoming human in Dogon*. New Haven: Human Relations Area Files.
VASAVADA, P. C. 1988. Pathogenic bacteria in milk: A review. *Journal of Dairy Science* 71:2809–16.
VASSILIADIS, S., A. RANELLA, L. PAPADIMITRIOU, A. MAKRYGIANNAKIS, AND I. ATHANASSAKIS. 1998. Serum levels of pro- and anti-inflammatory cytokines in non-pregnant women, during pregnancy, labour, and abortion. *Mediators of Inflammation* 7:69–72.
VELLACOTT, I. D., E. J. COOKE, AND C. E. JAMES. 1988. Nausea and vomiting in early pregnancy. *International Journal of Gynaecology and Obstetrics* 27:57–62.
VILMING, B., AND B. I. NESHEIM. 2000. Hyperemesis gravidarum in a contemporary population in Oslo. *Acta Obstetrica et Gynecologica Scandinavica* 79:640–43.
VON DADELSZEN, P., T. WILKINS, AND C. W. G. REDMAN. 1999. Maternal peripheral blood leukocytes in normal and pre-eclamptic pregnancies. *British Journal of Obstetrics and Gynaecology* 106:576–81.
WADE, G., J. SCHNEIDER, AND H. LI. 1996. Control of fertility by metabolic cues. *American Journal of Physiology* 270: E1–E19. [REN]
WALDENSTROM, U. 1999. Experience of labor and birth in 1,111 women. *Journal of Psychosomatic Research* 47:471–82.
WALKER, A. R., B. F. WALKER, J. JONES, M. VERARDI, AND C. WALKER. 1985. Nausea and vomiting and dietary cravings and aversions during pregnancy in South African women. *British Journal of Obstetrics and Gynaecology* 92: 484–89.
WALLACE, F. M., A. S. MACH, A. M. KELLER, AND J. A. LINDSAY. 1999. Evidence for *Clostridium perfringens* enterotoxin (CPE) inducing a mitogenic and cytokine response in vitro and a cytokine response in vivo. *Current Microbiology* 38: 96–100.
WALSH, J. W., W. L. HASLER, C. E. NUGENT, AND C. OWYANG. 1996. Progesterone and estrogen and potential mediators of gastric slow-wave dysrhythmias in nausea of pregnancy. *American Journal of Physiology* 270:G506–14.
WEBSTER, W. S. 1998. Teratogen update: Congenital rubella. *Teratology* 58:13–23.
WEIGEL, M. M. 1985. Early pregnancy associated nausea and vomiting: Maternal risk factors, fetal outcome, and reproductive success. Ph.D. diss., University of California, Los Angeles, Calif.
WEIGEL, M. M., M. E. CAIZA, Y. LASCANO, G. BARRENO, AND L. MOSQUERA. 2000. Early pregnancy nausea and vomiting in a high-altitude Andean population. *International Journal of Gynaecology and Obstetrics* 69:9–21.
WEIGEL, R. M., AND M. M. WEIGEL. 1989. Nausea and vomiting of early pregnancy and pregnancy outcome: A meta-analytical review. *British Journal of Obstetrics and Gynaecology* 96:1312–18. [SMF, KO]
WESTERTERP-PLANTENGA, M. S., AND N. E. WIJCKMANS-DUIJSENS. 1996. The role of macronutrient selection in determining patterns of food intake in obese and non-obese women. *European Journal of Clinical Nutrition* 50:580–91.
WHEELER, ERICA, AND SWEE POH TAN. 1983. From concept to practice: Food behaviour of Chinese immigrants in London. *Ecology of Food and Nutrition* 13:51–57.
WHITEHEAD, H. 2000. *Food rules: Hunting, sharing, and ta-*

booing game in Papua New Guinea. Ann Arbor: University of Michigan Press.

WHITEHEAD, S. A., W. A. HOLDEN, AND P. L. R. ANDREWS. 1992. "Pregnancy sickness," in *Mechanisms and control of emesis.* Edited by A. L. Bianchi, L. Grelot, A. D. Miller, and G. L. King, pp. 297–306. Montrouge: John Libbey Eurotext/ Paris: INSERM. [BJT, SC]

WIJEWARDENE, K., P. FONSEKA, AND C. GOONARATNE. 1994. Dietary cravings and aversions during pregnancy. *Indian Journal of Public Health* 38:95–98.

WILEY, A. S., AND S. H. KATZ. 1998. Geophagy in pregnancy: A test of a hypothesis. CURRENT ANTHROPOLOGY 39: 532–45.

WILLIAMS, G. C. 1966. *Adaptation and natural selection: A critique of some current evolutionary thought*. Princeton: Princeton University Press.

WILSON, J. F. 1987. Severe reduction in food intake by pregnant rats resembles a learned food aversion. *Physiology and Behavior* 41:291–95.

———. 1997. Effects of pregnancy, sucrose, and various low-protein diets on the eating behavior of rats. *Physiology and Behavior* 62:779–82.

WORTHINGTON-ROBERTS, B., R. E. LITTLE, M. D. LAMBERT, AND R. WU. 1989. Dietary cravings and aversions in the postpartum period. *Journal of the American Dietetics Association* 89:647–51.

WROBEL, K., K. WROBEL, G. R. VALTIERRA MARQUEZ, AND M. D. L. RODRIGUEZ ALMANZA. 1999. Studies on bioavailability of some bulk and trace elements in Mexican tortilla using an in vitro model. *Biological Trace Element Research* 68:97–106.

YEREMINA, I. A. 1976. O vliianii beremennosti na funktsional'noe sostoianiae verkhnikh dykhatel'nykh putei. *Zhurnal Ushnykh, Nosovykh i Gorlovykh Boleznei*, no. 4, pp. 28–31.

ZEITLIN, M. F., R. MEGAWANGI, E. M. KRAMER, N. D. COLLETTA, E. D. BABATUNDE, AND D. GARMAN. 1995. *Strengthening the family: Implications for international development*. New York: United Nations University Press.

ZHANG, H. G., X. SU, D. LIU, W. LIU, P. YANG, Z. WANG, C. K. EDWARDS, H. BLUETHMANN, J. D. MOUNTZ, AND T. ZHOU. 1999. Induction of specific T cell tolerance by Fas ligand-expressing antigen-presenting cells. *Journal of Immunology* 162:1423–30.

ZHOU, Q., B. O'BRIEN, AND J. RELYEA. 1999. Severity of nausea and vomiting during pregnancy: What does it predict? *Birth* 26:108–14.

ZIMMER, S., V. POLLARD, G. D. MARSHALL, R. P. GAROFALO, D. TRABER, D. PROUGH, AND D. N. HERNDON. 1996. The 1996 Moyer Award: Effects of endotoxin on the Th1/Th2 response in humans. *Journal of Burn Care and Rehabilitation* 17:491–96.