

An Adaptationist Approach to Pregnancy Sickness

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Pregnancy sickness, commonly referred to as morning sickness, is a set of symptoms that occurs in some women during the first trimester of pregnancy. Women with pregnancy sickness may experience one or more of these symptoms: food aversions, nausea, and vomiting (Profet, 1992). Profet argued that food aversions, nausea, and vomiting of pregnancy evolved during the course of human evolution to protect the embryo against maternal ingestion of toxins abundant in natural foods. She suggested that pregnancy sickness represents a lowering of the usual human threshold of tolerance to toxins in order to compensate for the extreme vulnerability of the embryo to toxins during organogenesis, the period of maximum susceptibility to toxins.

Profet suggested that since pregnancy sickness generally has been assumed to be a side effect of pregnancy hormones, its possible benefits have rarely been explored. Although hormones that signal the onset of pregnancy may trigger pregnancy sickness, Profet (1992) argued, the central adaptationist question is whether this triggering would have been selectively advantageous in the human environment of evolutionary adaptation, the environment of a Plio-Pleistocene forager. She suggested that an adaptationist approach can help to understand physiological phenomena, such as pregnancy sickness, that are commonly assumed to be anomalies or pathologies (Profet, 1992). An examination of the plausibility of Profet's adaptation hypothesis requires that several issues be addressed. First, it will be necessary to consider the purported benefits that outweigh the costs of pregnancy sickness. In addition, the occurrence of pregnancy sickness cross-culturally has important implications for Profet's argument. It will also be useful to consider the occurrence of pregnancy sickness among other mammals, particularly primates. The discussion of these issues has as its ultimate consideration, whether pregnancy sickness is an adaptation against toxins or simply a side effect of other physiological processes.

Although Profet has proposed an adaptationist approach to understanding pregnancy sickness, it is important to briefly consider the critiques against such adaptationist interpretations. Gould and Lewontin (1979) asserted that it is important to consider that organisms are constrained as integrated wholes. The selection of a particular trait might involve simultaneous effects in other traits. Therefore, the mere utility of a feature does not necessarily account for the primary evolutionary reason for its existence. Gould and Lewontin (1979) suggested, then, that seemingly adaptive traits may be epiphenomena of non-adaptive structures.

Profet (1992) discussed possible selection pressures that favored pregnancy sickness. Plants have evolved a wide range of chemical weapons, or toxins, which they synthesize in their tissues to poison their numerous predators, including parasites, mammals, insects, and fungi. These toxins can cause neurological, renal, endocrine, metabolic, hepatic, reproductive, or other impairment in mammals. The impairment may be temporary, permanent, or lethal, depending on the doses ingested and on the metabolic defenses of the predator (Freeland and Janzen, 1974). Mammals, in turn, have evolved defenses against toxins in order to exploit plants for their nutrients. In particular, these defenses include detoxification enzymes manufactured by the liver and surface tissues of various other organs, such as the skin and the lungs. The presence of toxins induces cells of these organs to increase the production of detoxification enzymes (Freeland and Janzen, 1974).

The coevolutionary struggle between plant and predator has resulted in plant toxins which impair predator physiology and in predator physiological and behavioral mechanisms for detecting toxins, for determining dangerous thresholds of toxins, and for detoxifying and eliminating ingested toxins (Freeland and Janzen, 1974). Both plant and predator benefit from the predator being able to detect toxins in plants. The predator's detection of toxins may deter their ingestion of plants that emit cues of toxicity. While this prevents the predator's ingestion of harmful substances, it also saves the plant from being ingested. As a result of the coevolution of plant signals of toxicity and predator mechanisms for perceiving this toxicity, plant toxins tend to be correlated with odors and tastes that their predators find aversive (Freeland and Janzen, 1974). Profet suggested that humans and other experimental plant predators, however, often learn which pungent or bitter tastes are associated with the toxins that their physiological systems tolerate well and even acquire "tastes" for some of those that signal important sources of nutrients (Profet, 1992).

The threshold of tolerance for toxicity sufficient to protect adults against dangerous levels of toxins may be insufficient to protect developing embryos. For many toxins, doses that have negligible effects on the adult are teratogenic or even lethal to embryos (Moore and Persaud, 1993). Teratogenesis can occur when toxins enter the maternal bloodstream and diffuse through the placenta. Toxins can initiate teratogenesis by mutating the genes, interfering with cell division, changing the characteristics of the cell membrane, or causing other disturbances that lead to disruption of chemical signal, to cell death, or to lack of functional cell maturation. Toxins also can harm the embryo indirectly by interfering with the placental or maternal chemicals that regulate the nourishment of the embryo (Moore and Persaud, 1993).

Profet's argument can be better understood by first considering the nature of pregnancy sickness. Pregnancy sickness coincides with organogenesis, the embryonic period of maximum vulnerability to teratogens. Human

organogenesis takes place from approximately day 20 to day 56 after conception and entails formation of the limbs and all the major organ systems, including the central nervous system, heart, eyes, ears, and external genitalia. Therefore, major morphological malformations can occur during this period, although for some organ systems the sensitive developmental periods extend through the 14th week (Moore and Persaud, 1993). Tierson et al. (1986) reported that pregnancy sickness among women in their study usually began within 2 to 4 weeks after conception, peaked about 6 to 8 weeks after conception, fell off after 8 weeks, and disappeared around 17 weeks.

Neither organogenesis nor observable pregnancy sickness occurs during the first 2 weeks after conception. During this time embryonic cells proliferate rapidly but do not differentiate much morphologically. Consequently, disruption of embryonic development at this stage is more likely to cause cell death, which leads either to cell replenishment or to embryonic death, rather than malformation (Moore and Persaud, 1993). Also, toxins that are absorbed into the maternal bloodstream are less likely to reach the embryo if the placenta, which connects the embryo to the maternal bloodstream, has not yet formed. While the embryo implants in the uterus 6 to 7 days after conception, it does not form a placenta capable of absorbing maternal blood until 15 days after conception (Moore and Persaud, 1993). During the first week after conception, the embryo is thought to obtain nourishment by absorbing uterine gland secretions while traveling down the uterine tube, and during implantation, by digesting uterine endometrial cells. Toxins that are absorbed into the maternal bloodstream can potentially reach these initial sources of nourishment. However, these sources are much less direct pathways for toxins than is the maternal blood, which nourishes the embryo from the third week until birth (Moore and Persaud, 1993).

Profet (1992) discussed that the fitness costs incurred by women who experience pregnancy sickness can include inadequate nutrition, due to a decrease in consumption of nutritious sources of food, and lower productivity because of feeling ill. She suggested that pregnancy sickness usually ends by the second trimester; otherwise, it would probably inflict nutritional costs on the fetus that outweighed the benefits of maternal aversions to food toxins. Tierson et al. (1986) reported that food cravings and aversion during pregnancy can reduce nutrient intake. They found that those who reported pregnancy sickness experienced weight loss during the first trimester. Profet (1992) suggested that nutritional costs, however, are usually of small consequence to the embryo, who weighs only a few grams by the end of organogenesis and therefore has small nutritional demands. However, Tierson et al. (1986) found that women ultimately experiencing live birth outcomes, consumed more nutrients during the first trimester.

Noting that some first-trimester vitamin deficiencies have been implicated in teratogenesis, Profet (1992) suggested that pregnancy sickness might appear

maladaptive because it induces aversions to important sources of folate and other vitamins and micronutrients, such as some vegetables, that are essential for normal embryonic development. She went on to suggest, however, that the nutritionally adverse effects of pregnancy sickness on embryonic health are probably exacerbated in modern industrial societies. Pleistocene hunter-gatherer women with sufficient fat reserves to conceive are unlikely to have been vitamin deficient at conception because much of their caloric intake prior to conception would have come from vegetables and fruits. Folate reserves from a normal Pleistocene diet would have been sufficient to withstand the temporary deficiencies of pregnancy sickness. Much of the caloric intake of women in modern industrial societies, on the other hand, comes from nonplant foods or processed foods low in vitamins (Profet, 1992).

Maternal basal metabolic rate often decreases in the first trimester and increases in the third trimester, perhaps to compensate for decreased nutritive intake in the first trimester (Moore and Persaud, 1993). Profet (1992) suggested that severe nutritional deprivation in the first trimester often can be compensated for by adequate nutrition in the second and third trimesters. However, as already mentioned, Tierson et al. (1986) reported higher rates of live birth among women who had adequate nutrition during the first semester. Since nutritional deficiencies experienced by the mother during the latter trimesters can lead to growth retardation of the fetus (Moore and Persaud, 1993), Profet (1992) believed that the mother's food behavior during this period should reflect the emphasis in fetal development on growth rather than the emphasis in embryonic development on organ differentiation. Therefore, she reported that the maternal threshold for tolerating toxicity, after decreasing during the first trimester, increases during the second and third trimesters.

Similarly, Profet (1992) speculated that maternal aversions to pungent aromas and tastes deter ingestion of these foods in favor of bland foods containing lower, less detectable concentrations of toxins and so prevent embryonic exposure to high levels of toxin. In making this argument, Profet drew on accounts that discussed perceptual changes in the palatability of foods that can occur during pregnancy due to changes in olfactory chemoreceptive sensitivity to substances suggestive of toxins. It is suggested that in the first trimester, during embryonic organogenesis, women become hyperosmotic, that is, develop increased olfactory acuity, and thus are likely to develop aversions to previously tolerated foods. By the third trimester, however, women become hypoosmotic and thus are unlikely to maintain aversions to these foods. This hypoosmoticism may be necessary to counter the food aversions that developed during the first trimester hyperosmoticism, so that the mother is not deterred from consuming important sources of nutrients during a period of rapid fetal growth. However, in addition to Tierson et al.'s (1986) finding that inadequate nutrition during the first trimester was associated with poor pregnancy outcome, Brown et al. (1997) found that nausea and vomiting did not prevent women from consuming foods that Profet considered harmful. Furthermore,

Brown et al. reported that the consumption of these foods did not produce adverse pregnancy outcomes.

Profet (1992) discussed that it has been found that in early pregnancy, gastric motility, or the movement of food from the stomach to the intestines, decreases. This slows the rate of absorption of foods, including their toxic constituents, into the bloodstream and confines them to the stomach for a longer period of time, thus increasing the opportunity to expel foods through vomiting. Profet (1992) suggested that inhibition of gastric motility may be an adaptation to slow absorption of toxins because this inhibition also occurs in nonpregnant mammals after ingestion of substances that induce nausea. The small intestine also displays reduced motility during pregnancy, prolonging transit times for foods. The gastrointestinal absorption of toxins is spread out over a longer period of time during pregnancy so that the peak toxic load on the liver is decreased. Therefore, at any given time, the liver is more likely to have sufficient metabolic resources to thoroughly handle incoming toxins. However, Profet (1992) noted, gastrointestinal motility is decreased throughout pregnancy, not just during the first trimester. She suggested that this could mean that some reduction in toxicity is important for the fetus as well as the embryo or that the decrease in gastrointestinal motility has a function in addition to reducing toxicity, such as preventing vitamin depletion.

Profet (1992) presented findings from studies which suggest that in early pregnancy the maternal rate of blood flow to the kidneys increases significantly, almost doubling by the end of the second trimester and declining thereafter. The glomerular filtration rate, or the rate at which molecules are absorbed from the bloodstream to the kidneys, increases by up to 70%, thereby increasing the rate of elimination of toxins. During late pregnancy the glomerular filtration rate generally declines, which may prevent excess excretion of water-soluble vitamins and nutrients during the time of rapid fetal growth. Profet (1992) pointed out that if selection pressures to reduce embryonic exposure to toxins were indeed significant during the course of human evolution, one might expect that mechanisms would have evolved to simply accelerate the rate at which the mother detoxifies toxins during the first trimester of pregnancy by, for example, increasing the number of detoxification enzymes. Mechanisms that increased the efficiency with which the liver detoxifies plant compounds might even have eliminated the need for pregnancy sickness. However, she noted that it has been found that accelerating the detoxification process is problematical. A necessary step in enzymatic degradation of certain types of inert compounds to water-soluble excretable compounds entails activating them to toxic intermediate metabolites. Therefore, accelerating the maternal rate of enzymatic degradation of compounds would also accelerate the rate at which these toxic metabolites are formed. This could pose various dangers for the embryo.

Profet (1992) presented discussions that speculate that the same physiological and psychological mechanisms may underlie both pregnancy sickness and the food aversions, nausea, and vomiting that occur in response to high concentrations of food toxins in nonpregnant humans. The vomiting response in humans and other animals often involves a part of the brain stem called the chemoreceptor trigger zone, which lies in the region of the brain known as the area postrema. This region is involved in inducing conditioned taste aversions to a wide variety of ingested and injected substances. The CTZ is extensively bathed with blood and cerebrospinal fluid, which it samples for toxic constituents. Chemoreceptors of the CTZ are specific for many different types of toxic molecules and induce nausea and vomiting when levels of toxins exceed certain thresholds.

Profet (1992) discussed that it has been reported that one possible mechanism for pregnancy sickness is that pregnancy hormones cause an increase in blood flow to the CTZ and, consequently, an increase in toxins that reach the CTZ from the bloodstream. This might recalibrate the CTZ response to toxins in the bloodstream, resulting in heightened sensitivity, and the triggering of nausea and vomiting to low concentrations of food toxins. It has been hypothesized that control of blood flow to the CTZ is regulated by receptors on the surfaces of blood vessels to the CTZ that expand or contract these vessels in response to particular substances circulating in the bloodstream. Thus, Profet (1992) suggested, a role for the CTZ in inducing pregnancy sickness might be demonstrated by finding receptors on blood vessels to the CTZ that bind pregnancy hormones.

At implantation, the embryonic placenta begins to synthesize the hormone human chorionic gonadotropin (HCG). After release into the maternal bloodstream, HCG induces the maternal ovary to produce the steroid hormones estradiol and progesterone, which are necessary for stimulating uterine growth and suppressing ovulation. The placenta also synthesizes estradiol and progesterone directly and, by the 6th week after conception, synthesizes them at sufficient levels to maintain the pregnancy without hormonal support from the maternal ovary (Moore and Persaud, 1993). HCG levels in the maternal bloodstream peak by about the 8th week after conception, fall precipitously after the 10th week, and remain low from the 14th week on. Estradiol levels rise steadily throughout pregnancy until birth. Progesterone levels rise immediately after conception, plateau from about weeks 2 through 8, then rise steadily until birth (Moore and Persaud, 1993).

The hormones traditionally viewed as most likely triggering pregnancy sickness are HCG and estradiol. HCG has been considered because its peak levels coincide with pregnancy sickness. Estradiol has been considered because high levels of this hormone cause nausea in nonpregnant women, such as in some women taking birth control pills (O'Brien and Zhou 1995). Profet (1992) discussed that it has been reported that the hormonal factors that are most

likely to influence pregnancy sickness are increases in estradiol, in the estradiol/progesterone ratio, and perhaps in HCG. Estradiol, an estrogen, appears to profoundly affect the CTZ by increasing its sensitivity to circulating toxins, perhaps by binding to receptors that line the blood vessels of the CTZ and dilating these vessels to produce greater blood flow. Profet (1992) acknowledged, however, that estradiol levels continue to rise after pregnancy sickness ends, so if estradiol causes CTZ sensitivity during early pregnancy, the question is what shuts off pregnancy sickness after the first trimester.

She went on to discuss the findings that estradiol/progesterone ratio may significantly affect pregnancy sickness, because progesterone exerts some effects that are opposite of estradiol. Progesterone may dampen the effects that estradiol has on blood vessels that lead to the CTZ. During the peak period of pregnancy sickness, from weeks 2 to 8 after conception, the estradiol/progesterone ratio increases between 5- and 10-fold. When the rate of change of this ratio slows considerably, 8 weeks after conception, pregnancy sickness decreases. In addition, the rise and fall of HCG levels are neatly timed with pregnancy sickness. If HCG helps to regulate pregnancy sickness, then it might do so by altering the effectiveness of estradiol and progesterone. For example, it may stimulate or inhibit the presence of receptors for these steroids on target tissues or alter the rate of steroid metabolism in the liver. Thus, Profet (1992) speculated that the net influence of a particular estradiol/progesterone ratio on the CTZ might be different in the presence of HCG.

Profet (1992) suggested that although pregnancy sickness can occur at any time during the day, many women consistently experience pregnancy sickness upon rising in the morning, hence the term "morning sickness". She drew on other studies to discuss that gastric and intestinal motility decrease during pregnancy, significantly delaying digestion and absorption of food constituents. The bacteria that extensively colonize the intestines of all mammals manufacture enzymes that interact with the contents of the intestines to produce a vast array of metabolites, many of which are toxic. During the first trimester of pregnancy the stomach becomes less acidic due to decreased secretions of hydrochloric acid, which may encourage bacterial colonization of the stomach. Chemoreceptors that register toxicity in the gastric region would be likely to induce nausea and vomiting in response to toxic metabolites produced from the gastrointestinal bacterial metabolism of meals eaten the night before. The CTZ would also react to the bacterially activated food toxins that are seeping into the bloodstream from the digestive tract.

She went on to report that since the intestines contain toxins produced by bacterial interaction with food eaten the previous evening, then filling the stomach with foods that are bland, low in toxicity, dilutes the concentration of toxins in the intestines and blocks the bacterially activated toxins from stimulating the chemoreceptors in the gastric region and from being absorbed

into the bloodstream in high concentrations to reach the CTZ. Profet suggested that the nearly constant state of nausea experienced by some women during the first trimester of pregnancy may be caused by CTZ and gastric hypersensitivity to the toxic metabolites that are manufactured constantly during digestion by intestinal bacteria. Although meals tend to be eaten at discrete intervals, they may be digested continuously throughout the day and night. Also the low levels of toxins contained in virtually all plant foods may be slowly absorbed into the circulation throughout the day, because of slower gastrointestinal absorption of food during early pregnancy. This could cause the hypersensitive CTZ to produce continuous nausea. Gastrointestinal bacteria, like metabolic enzymes of the liver, inactivate some types of toxin but activate other types. Therefore, she believed that the decrease in stomach acidity that enables bacteria to colonize the stomach may serve the dual function of enabling the detoxification of many compounds before they are absorbed into the circulation and of ensuring that some compounds that would have been activated to toxic metabolites in the liver are instead activated in the intestines, where they can trigger chemoreceptors in the gastric region and be more easily expelled through vomiting.

Profet (1992) acknowledged that the cross-cultural occurrence of pregnancy sickness has important implications for her argument. In particular, she suggested, pregnancy sickness should be ubiquitous among pregnant women of hunter-gatherer societies because she considered them to represent the closest approximation to Pleistocene hunter-gatherer feeding ecology. She suggested that selection pressures for pregnancy sickness would have been most intense in the environment of a Plio-Pleistocene forager, particularly during periods of scarcity. Similarly, Profet (1992) described current hunter-gatherer feeding ecology as being characterized by experimentation with wide variety of food sources, particularly during dry seasons or drought years when marginal environments necessitate broadening the diet to include more bitter, and therefore more toxic, plant foods (Profet, 1992).

Profet (1992) cited examples of nausea, vomiting, and food aversions among the !Kung of the Kalahari Desert, the Efe Pygmies of Zaire, and the Aborigines of Australia as being understood as indications of pregnancy. She also discussed the practice of eating clay to prevent or counteract vomiting among pregnant women throughout Indonesia, Oceania, and Africa, as cross-cultural evidence of pregnancy sickness (Profet, 1992).

However, the evidence she presented suggested that food cravings and food aversions are highly influenced by cultural factors. There has been much debate about the physiological versus cultural influences on food preferences during pregnancy (Wiley and Katz, 1998). For instance, in addition to the practice of geophagia, the consumption of particular foods, has come to be associated with, and expected during pregnancy in respective cultures (Reid, 1992; Wiley and Katz, 1998).

In their study of the influence of cravings and aversions on diet among 400 pregnant women in Albany, New York, Tierson et al. (1985) found that 76% of the women reported craving at least one item, while 85% reported at least one aversion. They reported that the respondents attributed the majority of cravings and aversions to endogenous factors. These were reasons coming from within the woman, such as nausea or vomiting, or a response like "the food item just tasted better." These reasons contrast with exogenous factors, or reasons from without, such as maternal concern for fetal health, a physician's orders to avoid certain items or the seasonality of food items (Tierson et al., 1985). Tierson (1997a) suggested that it is possible that women might express cravings for items they would prefer to eat anyway, such as chocolate, but which they would feel constrained against eating when not pregnant.

Minturn and Weiher (1984), in their cross-cultural study of the influence of diet on morning sickness, concluded that morning sickness is not a universal symptom associated with pregnancy but a condition which is influenced by diet. They reported that, of the thirty societies available in the Human Relation Area Files (HRAF) that had some information on morning sickness, eight societies reported no morning sickness. They found that maize was the staple in seven of these eight societies and in none of the societies with morning sickness. Profet (1992) suggested that niacin deficiency is common among people whose staple food is untreated maize. She discussed, therefore, how the nutritional deficiencies commonly found among people with maize-based diets may disrupt the physiological processes that induce pregnancy sickness. For example, since niacin deficiency causes disturbances of the central nervous system, it may disrupt pregnancy sickness by affecting the CTZ. In addition, it may be difficult to discern pregnancy sickness from the gastrointestinal disorders, such as nausea and vomiting, that niacin deficiency induces. Finally, she argued that niacin may be essential for inducing pregnancy sickness since it causes vasodilation, which increases blood flow throughout the body, including throughout the CTZ. However, Minturn and Weiher (1984) reported that vitamin B6 is involved in suppressing pregnancy sickness and that maize may be involved in facilitating the body's production of this vitamin.

Tierson et al. (1986) found that 89.4% of the 414 pregnant women in their study reported some symptoms of nausea and/or vomiting. They reported that the incidence of vomiting was about 55%. Similarly, Klebanoff et al. (1985) found that 56% of the 9098 pregnant women in their study reported vomiting during pregnancy. Profet (1992) believed that the reported percentage of women who experience nausea and vomiting is probably lower than actual rates because many studies asked participants to recall symptoms, after the peak period of pregnancy sickness had already passed. Furthermore, she suggested that the main problem with prevalence studies is that they define pregnancy sickness as the presence of nausea or vomiting rather than as the presence of food aversions, nausea, or vomiting (Profet, 1992). She speculated that because the

nausea of pregnancy sickness is often caused by the odor of certain foods, a pregnant woman may avoid them in order not to experience nausea and vomiting (Profet, 1992). Profet argued that, if pregnancy sickness were redefined to include food aversions, which she believed are the main purposes and symptoms of pregnancy sickness, prevalence would likely approach 100% (Profet, 1992). This speculation, however, weakens her argument. A prevalence rate of 100% would demonstrate that pregnancy sickness offers no protective effect.

Additionally, Brown et al. (1997) found that women with nausea and/or vomiting were no more likely to avoid pungent and bitter foods identified by Profet as harmful than women without these disorders. Furthermore, they found that women who consumed the proscribed foods were no more likely to have an adverse pregnancy outcome (miscarriage, fetal death, or an infant born with one or more congenital anomalies) than were women who did not consume the foods. Despite recent discussion to the contrary, previous studies generally reported an increased risk of spontaneous abortion among women who did not experience pregnancy sickness (Klebanoff et al., 1985; Petitti, 1986; Tierson et al., 1986). Even if we were to entertain the possibility of an association between pregnancy sickness and favorable pregnancy outcome, the association is generally attributed to estrogen and/or human chorionic gonadotropin (HCG) levels that are too low to induce pregnancy sickness, and that are at the same time, too low to maintain pregnancy. It is believed that, consequently, these low hormonal levels result in spontaneous abortion (Klebanoff et al., 1985; Petitti, 1986; Tierson, 1997b). Along these lines, Weigel and Weigel (1989a) present evidence to further counter Profet's suggestion that food aversions should be included in defining pregnancy sickness. They classified nausea and vomiting of pregnancy into three levels: no symptoms, nausea only, and nausea with vomiting. They found that only vomiting was associated with a decreased miscarriage risk, and that women who had nausea without vomiting had a miscarriage risk equal to that in the overall sample.

Tierson (1997b) discussed the difficulties in establishing causal explanations for pregnancy sickness. He suggested that even though they explain the same event, an explanation of the origin of pregnancy sickness at the proximate level, hormonal changes, may be viewed as being in opposition to an ultimate level of explanation, that is, the level of evolutionary significance or purpose. He suggested that the proclivity for explaining behaviors in an adaptive context often neglects to consider that many biological features may be side effects of other processes. Tierson (1997b) explained that while selection may be operating, it may not be operating in the manner we suspect. For example, although pregnancy sickness may reduce fetal exposure to toxins, selection may be selecting for earlier fetal maturation, and pregnancy sickness may be a side effect of this selection (Tierson, 1997b). Similarly, since Weigel and Weigel (1989b) reported favorable pregnancy outcomes for those who

experienced pregnancy sickness, Profet (1992) suggested that a correlation between pregnancy sickness and pregnancy sickness among women with hormone levels sufficient for maintaining pregnancy might support the hypothesis that pregnancy sickness protects the embryo against toxins. This will be necessary in order to rule out, as already mentioned, the generally accepted explanation that hormones insufficient to induce pregnancy sickness are likewise insufficient to maintain pregnancy.

Profet (1992) also suggested that comparative studies among pregnant nonhuman mammals on the detection and avoidance of toxins could provide an additional means for testing the hypothesis that human pregnancy sickness is an adaptation to prevent maternal ingestion of teratogens. She suggested that while vomiting as a normal aspect of early pregnancy has been documented only in humans, and nausea in nonhuman mammals may be too subjective a symptom for an observer to detect, food aversions in pregnant mammals could be detected by determining changes in dietary preferences. Profet (1992) suggested, however, that selection pressures are more intense for humans than any other mammal because they exploit a vast array of different plants. However, Freeland and Janzen (1974) emphasized the importance for all mammals to continually consume novel foods as protection in the event that their primary food sources become extinct.

Profet (1992) also argued that because not all modern toxins emit the cues that are necessary for triggering the aversions of pregnancy sickness and because the plant foods have become less toxic through selective breeding, the selection pressures maintaining the mechanisms for detecting and avoiding Pleistocene toxins during pregnancy may have been decreasing since the advent of agriculture. In fact, Profet speculated that the variability in pregnancy sickness among women in industrial societies may be due to variations in dietary toxicity or to decreasing selection pressures for detecting and avoiding substances that emit Pleistocene cues of toxicity (Profet, 1992). She proposed a comparison of variability among women in industrial societies and among women in hunter-gatherer societies in order to determine whether pregnancy sickness is more variable among the former.

While Profet discussed various features of pregnancy sickness as evidence of an adaptive design to deter maternal ingestion of teratogens, she also suggested avenues for further testing of her hypothesis. Further consideration of the plausibility of this adaptation argument requires more evidence concerning the critical issues mentioned here: the cross-cultural occurrence of pregnancy sickness; the benefits that supposedly outweigh the costs of pregnancy sickness; and the occurrence of pregnancy sickness among other mammals. This last point necessitates particular emphasis among nonhuman primates. It has been noted that evidence of pregnancy sickness among nonhuman primates is often complicated by the difficulty of recognizing pregnancy (Whitten, personal communication, 1998). As with any adaptationist approach, it is essential to

keep in mind that the apparent adaptive feature may be merely a by-product of other biological processes.

References Cited

Brown JE, Kahn ES, and Hartman TJ (1997) Profet, profits, and proof: do nausea and vomiting of early pregnancy protect women from "harmful" vegetables? *Obstetrics and Gynecology*, 176(1):179-181.

Freeland WJ and Janzen DH (1974) Strategies in herbivory by mammals: the role of plant secondary compounds. *The American Naturalist*, 108(961):269-285.

Gould SJ and Lewontin RC (1979) The spandrels of San Marco and the Panglossian paradigm: a critique of the adaptationist programme. *Proceeding of the Royal Society of London*, 205:581-598.

Klebanoff MA, Koslowe PA, Kaslow R, and Rhoads GC (1985) Epidemiology of vomiting in early pregnancy. *Obstetrics and Gynecology*, 66:612-616.

Minturn L and Weiher AW (1984) The influence of diet on morning sickness: a cross-cultural study. *Medical Anthropology*, Winter, 71-75.

Moore KL and Persaud TVN (1993) *The developing human. Clinically oriented embryology*, 5th ed. W.B. Saunders Company, Philadelphia.

O'Brien B and Zhou Q (1995) Variables related to nausea and vomiting during pregnancy. *Birth*, 22(2):93-100.

Petitti DB (1986) Nausea and pregnancy outcome. *Birth*, 13:4.

Profet M (1992) Pregnancy sickness as adaptation: a deterrent to maternal ingestion of teratogens. In Barkow JH, Cosmides L, and Tooby J (eds): *The Adapted Mind. Evolutionary Psychology and the Generation of Culture*. New York, Oxford University Press, pp. 327-365.

Reid RM (1992) Cultural and medical perspectives on geophagia. *Medical Anthropology*, 13:337-351.

Tierson FD (1997a) Pregnancy, dietary cravings and aversions. In *Encyclopedia of Human Biology*. 2nd ed. New York, Academic Press, 7:57-59.

Tierson FD (1997b) Pregnancy, nausea and vomiting. In *Encyclopedia of Human Biology*. 2nd. ed. New York, Academic Press, 7:75-78.

Tierson FD, Olsen CL, and Hook EB (1985) Influence of cravings and aversions on diet in pregnancy. *Ecology of Food and Nutrition*, 17:117-129.

Tierson FD, Olsen CL, and Hook EB (1986) Nausea and vomiting of pregnancy and association with pregnancy outcome. *American Journal of Obstetrics and Gynecology*, 155:1017-1022.

Weigel MM, and Weigel RM (1989a) Nausea and vomiting of early pregnancy and pregnancy outcome. An epidemiological study. *British Journal of Obstetrics and Gynaecology*, 96:1304-1311.

Weigel MM, and Weigel RM (1989b) Nausea and vomiting of early pregnancy and pregnancy outcome. A meta-analytical review. *British Journal of Obstetrics and Gynaecology*, 96:1312-1318.

Whitten P (1998) Personal communication.

Wiley AS and Katz SH (1998) Geophagy in pregnancy: a test of a hypothesis. *Current Anthropology*, 39(4):532-545.

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