THE EVOLUTION OF PREGNANCY SICKNESS AS PROTECTION TO THE EMBRYO AGAINST PLEISTOCENE TERATOGENS

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ABSTRACT: Pregnancy sickness may have evolved during the Pleistocene as a protection to the embryo against the wide array of toxic foods available to the mother. The nausea and food aversions that characterize pregnancy sickness deter pregnant women from consuming many of the pungent toxic defenses--particularly secondary plant compounds --that potential food sources use against predators. Many substances that are mildly toxic to adults are extremely toxic to embryos that are undergoing organogenesis, causing malformation or abortion. The selective advantage of pregnancy sickness in bringing to term healthy offspring may have been significant.

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Introduction

Pregnancy sickness is commonly considered to be a side effect of pregnancy, conferring no benefits *per se*. Although the biochemical mechanisms triggering the first-trimester nausea, vomiting, and food aversions are not understood, pregnancy sickness is often attributed to the pregnancy hormone human chorionic gonadotropin (HCG), because the peak incidence of pregnancy sickness coincides with the maximal levels of HCG (Danforth, 1977); however, experiments to establish a correlation between various pregnancy hormone levels and severity of pregnancy sickness have led to negative results (Soules *et al.*, 1980). The sparse literature on pregnancy sickness has generally overlooked the possibility that even if pregnancy sickness is related to hormone levels, the reaction of nausea to the hormone could have evolved as a selective advantage.

The influence of pregnancy sickness on maternal diet suggests that pregnancy sickness may have evolved due to selection pressures to minimize maternal ingestion of teratogens during pregnancy. Selection pressures for pregnancy sickness can be assumed if pregnancy sickness is shown to deter first-trimester pregnant women from consuming the more toxic, potentially teratogenic substances of their usual diets. Hook (1976) measured first-trimester changes in maternal consumption of the toxic agricultural substances caffeine, alcohol and tobacco, but his focus on toxic substances that are ingested purposely for their toxicity, and on the advantages since the evolutionarily recent advent of agriculture of avoiding cultivated toxins, led to inconclusive results regarding a fetoprotective role for pregnancy sickness. Rather, an investigation of the link between dietary toxins and the evolution of pregnancy sickness should take into account the vast array of toxic compounds in food sources that humans have had to confront throughout their evolutionary history, and the selective advantages to Pleistocene hunter-gatherers of developing means for minimizing toxic intake, such as through their processing

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and cooking of foods. This paper will argue that pregnancy sickness evolved in the Pleistocene, and enhanced the chances of survival for the embryo by discouraging the mother from ingesting the many toxic substances that were prevalent in the Pleistocene environment.

Why Pregnancy Sickness Evolved

Human strategies for finding sources of food are coupled with strategies for detoxifying sources of foods. As experimental omnivores, humans eat a wide and ever-changing array of foods, including mammals, fish, reptiles, birds, insects, fruits, seeds, roots and leaves. Because plants and animals are in a coevolutionary race with their predators for survival (Van Valen, 1973), most species of plants and some species of animals develop toxic defenses against predators (Tu, 1977; Freeland & Janzen, 1974). Plants produce toxic secondary compounds (Keeler, 1983; Duke, 1985), which they commonly advertise to potential predators with pungent or bitter tastes and odors (Westoby,1977). Plant toxins can cause severe and sometimes fatal harm to mammals, including neurological, renal, endocrine, or reproductive disturbances (Conning, 1983; Freeland & Janzen, 1974). However, the biochemical specificity of a toxic defense does not protect against all potential predators, because many foods that are highly toxic for one species may place only slight demands on the detoxification abilities of another species, for whom the toxic food is, on balance, a nutritious food source (Conning, 1983; Freeland & Janzen, 1974). Animal foraging patterns commonly include means of minimizing toxic intake, such as sampling potentially toxic new foods in small doses to determine or build tolerance to them (Freeland & Janzen, 1974). Experimenting with small doses of novel toxic foods can cause digestive enzymes or bacterial gut flora that degrade the toxins to increase to levels that can handle subsequent larger doses of the toxic foods (Freeland & Janzen, 1974). Many species prevent overloading their systems with any particular toxin by diversifying their diets to include a range of toxins (Freeland & Janzen, 1974). Humans have variable additional lines of defense against the toxicity of food sources, such as processing plant and animal foods to circumvent their most toxic parts, and cooking foods to destroy toxins (Tooby & DeVore, 1986; Stahl, 1984). By adapting to the defenses of many plants and animals faster than these plants and animals can evolve new defenses (Tooby & DeVore, 1986), humans are able to exploit a great number of diverse toxic foods for their diets.

Some toxic substances that exert minimal effects on the adult are teratogenic to developing embryos (Beck, 1973; Duke, 1985). Certain toxins that circulate in the blood of a pregnant mother diffuse across the placenta and enter the bloodstream and cells of the embryo. Other toxins affect the placenta or affect chemicals in the mother that regulate the nourishment of the embryo (Waddell & Marlowe, 1981). Toxins can directly or indirectly disrupt the chemical signals that direct tissue and organ differentiation (Heinonen, 1977). Differentiation that proceeds incorrectly can lead to the malformation or death of the embryo.

Pregnancy sickness causes nausea, vomiting and food aversions in response to many foods that contain toxins, and induces pregnant women to eat foods that are bland. Because many foods advertise their toxicity by emitting volatile secondary compounds, foods that cause the chemoreceptors of the human nose and tongue to register pungent or bitter odors and tastes are more likely to be toxic to humans than bland foods (Oakley, 1986). Bitter tastes signal the presence of toxins (Rozin & Vollmecke, 1986). Women who experience pregnancy sickness become much less tolerant of foods with pungent or bitter odors and tastes, such as garlic, spicy foods, and all but the most bland vegetables (Willson & Carrington, 1976). They generally become intolerant of smoke and fumes, which are common sources of toxins, to the extent that nausea is often triggered by strong cooking fumes (Hale, 1984). Cooking fumes not only alert the woman to the potential toxicity of the substance being cooked, but can be toxic as well. Although cooking is a highly effective method for killing parasites in meats and for destroying toxins in plants, cooking can also volatilize some toxins, which can then be inhaled. Primitive tools used for cooking, such as sticks or leaves, may contain resins or other toxins that can be converted into toxic fumes and inhaled. The nausea brought on by such potentially toxic fumes deters pregnant women from subjecting herself, and thus her embryo, to noxious inhalants.

Pungent meats, as well as pungent plants, may induce nausea in women experiencing pregnancy sickness. Meats acquire a pungent odor when they start to rot and to attract bacteria. Certain bacteria that parasitize the human digestive tract produce substances that are highly toxic (Alcock, 1983). By avoiding meats suggestive of rot, the pregnant woman safeguards her embryo against these bacterial toxins.

Pregnancy sickness discourages pregnant women from ingesting the more toxic foods of their usual diets. This view implies that pregnancy sickness may also cause pregnant women to become much less experimental about choosing foods to eat, because novel foods are potentially toxic. In short, women who have pregnancy sickness eat bland foods, which in general carry fewer toxins than do the pungent or bitter foods; consequently they inflict less toxic harm on their developing embryos, and so increase their chances of delivering healthy offspring.

Selection Pressures for Pregnancy Sickness in the Pleistocene

If we view current hunter-gathering as fairly (albeit imperfectly) representative of Pleistocene hunter-gathering, then we can infer that selection pressures for the evolution of pregnancy sickness would have been significant during the Pleistocene, because hunter-gatherer feeding ecology is characterized by experimentation with a large diversity of foods of varying degrees of toxicity. Although during wet seasons or times of abundance, hunter-gatherers may choose to eat only a fraction of the huge variety of plants and animals that they recognize as edible, in the marginal environments that can result during dry seasons or droughts, they must broaden their diets

to include a range of less palatable foods (Lee & DeVore, 1976, p.44). Eileen O'Brien and Charles Peters (personal communication) have pointed out that the diversification of diets to include more bitter species of plants would have increased toxic intake, and that for this reason, periods of scarcity would have provided the greatest selection pressures for pregnancy sickness. Under especially scarce conditions during and since the Pleistocene, diversification of diets may have been accompanied by expansion into new regions with unfamiliar flora, the toxicity of which had to be determined by experience. Pregnancy sickness, however, would have discouraged first-trimester pregnant women from experimenting with novel toxic foods, in favor of incurring short-term nutritional costs and sustaining viable embryos.

Although Hook (1976) has suggested that pregnancy sickness may have evolved in response to agriculture, diets comprised of agricultural foods depend on the cultivation of a few staple foods that have been selected in part for their minimal deleterious effects, and lack the diversity of hunter-gatherer diets (Woodburn, 1968; Dunn, 1968). In general, humans, like other mammals, try to minimize the toxicity of their diets by selecting foods whose toxins can be degraded easily by digestive enzymes, by processing, or, for humans, by cooking (Tooby & DeVore, 1986; Stahl, 1984); exceptions to this behavior--the ingestion of wild or cultivated substances specifically for their toxicity, such as recreational or medicinal drugs--occur in both hunter-gatherer and agricultural societies (Schleiffer, 1973; Weiner, 1972; Watt & Breyer-Brandwijk, 1962). Although various agricultural societies, unlike hunter-gatherer societies, have incorporated complex spices into their culinary activities, John Tooby (personal communication) has pointed out that eating spices may have the effect of fooling the palate into believing the diet is more diverse than it is, thus compensating for the limited diversity and toxicity of agricultural diets relative to hunter-gatherer diets. Spices may induce the "sensory-specific satiety effect"--in which the perceived diversity of foods consumed influences the amount of food consumed--that has been shown to occur in humans, who consume more food in total when offered a variety of foods than when offered only their favorite food, and in rats, who consume more of a particular food when different odorants have been added to the food (Rozin & Vollmecke, 1986). By mimicking the nutritional diversity of hunter-gatherer diets through varied chemosensory stimulation, spices may induce satiety in otherwise bland agricultural diets.

During years of drought or other periods of scarcity, however, some agriculturalists must diversify their diets, like hunter-gatherers, by gathering wild plants for food sources (Scudder, 1962). Eileen O'Brien and Charles Peters (personal communication) have pointed out that toxic load increases significantly when agricultural diets are supplemented by more toxic, less palatable, wild plants. For example, the Gwembe Tonga agriculturalists of Zambia resort to wild famine foods during periods of hunger, and drink a beverage made from *Tamarindus indica*, "though not without stomach aches, which are only partially avoided by adding ashes to the brew to reduce its bitterness" (Scudder, 1971, p. 29). They also eat the pods of leguminous plants

such as the *Xeroderris stuhlmanni*, "although not without headache and other symptoms of stress" (Scudder, 1971, p. 30). Although generally the agriculturalists of an area readily distinguish the edible from the highly toxic plants, sometimes conditions force them to move to a new area whose flora exhibits a chemically distinct arsenal of secondary compounds. The consequences of experimenting with novel wild plants are examined in Scudder's (1971) study of the Zambian government's 1958 resettlement of the Gwembe Tonga. Almost 10% of the 600 relocated settlers within the Lusitu basin died of the "Lusitu condition", a sudden affliction whose symptoms and circumstances strongly indicate poisoning by wild plants gathered for food (Scudder, 1971). The serious risk of wild plant toxins to adults underscores the extreme risk of plant toxins to the much more vulnerable embryo. Agriculturalists throughout much of agricultural history, then, may have especially benefitted by pregnancy sickness when they resorted to pre-agricultural, or hunter-gatherer, means for survival.

If pregnancy sickness did indeed evolve among Pleistocene hunter-gatherers, rather than among agriculturalists, then pregnancy sickness should be found to be a symptom of pregnancy among current hunter-gatherers. Although data are very scant concerning the early pregnancies of hunter-gatherer women, anthropologists have noted pregnancy sickness among the !Kung of the Kalahari Desert, the Efe Pygmies of Zaire, and the Aborigines of Australia. In Shostak's (1981) monograph on the !Kung woman she calls Nisa, Nisa recalls that in early pregnancy, "Whenever I ate meat, I threw up, and whenever I ate sweet berries I threw up. But when I ate water root or pounded gwia leaves or do roots, [foods which are extremely bland (Shostak, personal communication)] I didn't throw up" (p. 186). Indeed, nausea, vomiting and unexplained dislikes for certain foods are among the first signs that the !Kung recognize that a woman is pregnant (p. 178). Nisa's mother-in-law even says to her, "If you are throwing up like this, it means you have a little thing inside your stomach" (p. 187). Nadine Peacock (personal communication) reports that in the context of questioning six Efe Pygmy women on miscarriage, one woman mentioned that the realization of pregnancy comes about when "food tastes bad". Kaberry (1939) refers to vomiting of pregnant Australian Aboriginal women (p. 42) and to food taboos that are placed on the pregnant women as "a means of protecting the child developing within her womb, though she herself may sicken if she disregards them" (p. 241).

The connection between plant toxins and abortion is recognized by many hunter-gatherer women, who ingest or inhale the smoke from plant toxins in order to terminate unwanted pregnancies. !Kung women attempt to induce abortions by ingesting chemical agents made from plants, although the success of this method has not been documented (Shostak, 1981). Mbuti Pygmy women try to induce abortions through ingesting vegetable decoctions made of gorogoro bark or tebvo liana (Turnbull, 1965a, p. 222) or by burning and inhaling the smoke from the highly toxic ikanya bark (p. 232). Efe Pygmy women claim to know of a plant from the forest that induces abortion (Nadine Peacock, personal communication). Thus, hunter-gatherer women not only

recognize nausea, vomiting and food aversions as symptomatic of pregnancy, but recognize the utility of plant toxins for inducing abortion.

The Adaptive Value of Pregnancy Sickness

Pregnancy sickness usually begins within 2-4 weeks after conception, peaks near 8 weeks after conception and disappears by 14 weeks, the beginning of the 2nd trimester (Willson & Carrington, 1976). If pregnancy sickness has evolved to protect the embryo from teratogenic substances, then the actual periods of sickness experienced by the mother should coincide with the periods that the embryo is most vulnerable to teratogenic damage by toxins in the mother. The embryo is vulnerable to harm by toxins that enter the mother's bloodstream because the embryo depends on maternal blood for nutrients. However, the embryo takes 15 days from conception to form a placenta that is able to absorb maternal blood (Tuchmann-Duplessis, David & Haegel, 1971). During the 1st week after conception, while travelling down the uterine tube and through the uterus, the embryo is nourished by uterine secretions and probably by uterine gland secretions (Hamilton & Mossman, 1972); during the 2nd week, when implanting itself in the uterus, the embryo may derive nourishment by digesting the cells of the mother's endometrium (Hamilton & Mossman, 1972). Although toxins in the mother's bloodstream could potentially affect these initial sources of nourishment and thereby hurt the embryo, these sources are much less direct sites for toxins than is the blood, which directly bathes the placenta from the 3rd week until birth. Before the 3rd week, therefore, the embryo should not be affected greatly by teratogenic substances of the mother. Experimental evidence has shown, indeed, that before implantation, teratogenic substances ingested by the mother generally do not cause adverse effects on the embryo (Persaud, 1985). During the 3rd week, the embryo begins to absorb nutrients from maternal blood and begins to differentiate rapidly (Hamilton & Mossman, 1972). The embryo becomes very vulnerable to teratogenic harm at this stage, by gaining greater exposure to toxins that can interfere with cell differentiation. The onset of pregnancy sickness coincides with this stage.

Within the first 8 weeks after conception, the cells of the embryo differentiate to form the rudiments of the various organs (Johnson, 1983). After this period of fudamental differentiation the embryo becomes a fetus, which grows much larger, but undergoes much less differentiation (Persaud, 1985). In general, the embryo is far more susceptible to damage by external chemical agents than is the fetus. Although some chemical agents can exert greater harm at later stages of development, due to their affect on the nervous system, or on the particular genes that are expressed in later stages, or on the particular nutritional requirements of later stages, the embryo is in danger of suffering many more severe disturbances than is the fetus (Persaud, 1985). During this stage of embryonic differentiation, the mother experiences the worst pregnancy sickness. Thus, the most severe period of the mother's pregnancy sickness coincides with the most sensitive period of the embryo's

development.

Pregnancy sickness causes the mother to consume less variety of foods, and so less variety of nutrients, and often causes a decrease in the mother's appetite, and consequently a decrease in the amount of nutrients she ingests. Before the 2nd trimester, the fetus weighs less than an ounce, has scant nutritional needs, and so is usually unharmed by a low maternal food intake. But during the 2nd trimester and especially the 3rd trimester, the fetus gains significant weight and increases its nutritional requirements. The mother needs to increase her intake of food in order to properly nourish herself and the fetus. Nutritional deficiencies incurred by the mother can cause teratogenic effects for the fetus (Shepard, 1980). The mother's reaction to foods during this period should reflect the priority in fetal development to growth rather than the priority in embryonic development to organ differentiation. Indeed, pregnancy sickness usually ends by the beginning of the 2nd trimester, the stage at which the nutritional demands of the fetus require a substantial increase in food consumed by the mother.

The actual period of pregnancy sickness, then, corresponds to the period that pregnancy sickness should most benefit the embryo. Pregnancy sickness covers that period of embryonic development when the embryo is most harmed by toxins in the mother's blood and least harmed by loss of appetite by the mother.

Several studies conducted in the last 30 years in modern societies have shown that women who experience severe pregnancy sickness have significantly higher pregnancy success rates than women who experience mild or no pregnancy sickness. Klebanoff, Koslowe, Kaslow and Rhoads (1985), in their study of 9098 pregnant women, found that the abortion rate after the 14th week of pregnancy for women who experience pregnancy sickness severely enough to cause vomiting is 3.4%, whereas the rate for women with mild to no pregnancy sickness is 5.3%. Brandes (1967), in a study of 7027 pregnant women, found that the abortion rate before the 20th week of pregnancy was 2.8% for women with pregnancy sickness, compared to 6.6% for women without pregnancy sickness. Yerushalmy and Milkovich (1965), in a study of 3853 pregnant women, found that the abortion rate before the 20th week of pregnancy was 3.8% for women with pregnancy sickness, compared to 10.4% for women without pregnancy sickness. They also found that fetuses of women with pregnancy sickness suffered lower rates of severe and nontrivial birth defects than did the fetuses of women without pregnancy sickness. Medalie's (1957) study of 100 pregnant women records 0 abortions out of 52 women with severe pregnancy sickness, in contrast to 11 abortions out of 48 women (22.9%) with mild to no pregnancy sickness. Samuel Wasser (personal communication) has pointed out, however, that the connection between pregnancy sickness and spontaneous abortion may be spurious, because if pregnancy sickness occurs in response to the hormone HCG, then insufficient levels of HCG might fail to cause pregnancy sickness as well as cause abortion. On the other hand, because experiments have failed to correlate levels of HCG or other pregnancy hormones with severity of pregnancy sickness (Soules *et al.*,

1980), the studies relating degree of pregnancy sickness to abortion rates may indeed support the thesis that pregnancy sickness protects the embryo from teratogenic harm by the mother.

Implications of Pregnancy Sickness in Modern Environments

Investigation of the physiological and psychological mechanisms of food aversion in pregnant women might help to predict which types of modern teratogens will elicit pregnancy sickness. Pregnancy sickness should be triggered by environmental cues that are suggestive of toxins that were prevalent in the Pleistocene environment, such as secondary plant compounds. As pointed out by Leda Cosmides (personal communication), evolutionarily novel teratogens, such as industrial toxins, may lack the cues that are necessary to induce pregnancy sickness. Modern women, therefore, may not be protected by pregnancy sickness against ingesting or inhaling certain modern teratogens. Because the mechanisms that trigger pregnancy sickness evolved to protect against an ever-changing variety of toxins, these mechanisms are probably not fine-tuned enough to be able to distinguish Pleistocene toxins from their Pleistocene mimics or from modern variations of Pleistocene toxins. Various substances that are common correlates of Pleistocene toxicity should elicit pregnancy sickness whether or not the specific substances happen to be teratogenic, and whether or not the exact types of substances existed during the Pleistocene. Novel toxic substances that resemble Pleistocene toxic substances by, for example, emitting pungent odors that resemble secondary plant compounds, are more likely to trigger pregnancy sickness than toxic substances that lack Pleistocene forms of advertising their toxicity, such as foods poisoned by odorless chemicals.

Studies relating pregnancy sickness and maternal consumption of the modern toxins coffee, alcohol and cigarettes (Hook, 1976; Little & Hook, 1979), support the hypothesis that pregnancy sickness is elicited by cues suggestive of Pleistocene teratogens. Results of these studies should be interpreted cautiously, however, for although coffee, alcohol and cigarettes, in regular substantial doses, have been implicated as teratogenic (Shepard, 1980), maternal responses to addictive toxic substances do not necessarily reflect maternal responses to other dietary toxins, because the physiological cost of withdrawal from an addictive substance may be greater than the physiological cost of nausea induced by that substance during pregnancy. Notwithstanding, Hook (1976) found that of 295 pregnant women, 18.5% of the women who drank coffee decreased consumption of coffee due to nausea stimulated by the beverage, but many fewer of the women who drank alcohol decreased their consumption of alcohol due to nausea (5.4% wine drinkers decreased wine consumption, 5.7% beer drinkers decreases beer consumption, 7.4% spirits drinkers decreased spirits consumption). Whether aversions to these alcoholic beverages stemmed from aversions to the alcohol or to the plant constituents of the beverages is unclear. With Little (1979), in a study of 210 pregnant women, Hook found that change in alcohol consumption was unrelated to

pregnancy sickness. Caffeine may be more likely than alcohol to induce nausea in pregnant women because caffeine, a bitter-tasting secondary plant compound that is produced by the plant to ward off predators, resembles other toxic plant defenses that would have been commonly encountered by Pleistocene gatherers; whereas alcohol, a by-product of microorganisms and ripened or rotting plants, that serves no protective function for the developing plants, was probably a less significant threat than secondary plant compounds to Pleistocene hunter-gatherers, in the doses that hunter-gatherers obtained from eating plants. Although fermented plants could have been gathered by Pleistocene hunter-gatherers--for example, a type of honey gathered by the Mbuti Pygmies has been found to be fermented (Turnbull, 1965b, p. 169)--alcohol is primarily a product of agriculture. No evidence that Pleistocene hunter-gatherers produced alcohol has been found, and contemporary hunter-gatherers are not known to consume alcohol other than by procuring alcohol from neighboring agricultural tribes (Irv DeVore, personal communication), or, in one documented case, by producing alcohol by means learned from agricultural tribes (Tanaka, 1980, p. 39). Selection pressures for aversions during pregnancy to a toxin like alcohol, which may not be teratogenic in doses obtained from the incidental ingestion of fermented plants, were probably much less critical than selection pressures for aversions to toxins suggestive of secondary plant compounds, minute concentrations of which may cause embryonic malformation.

Findings by Little and Hook (1979) on the relation of cigarette consumption to pregnancy sickness illustrate the importance of registering cues that induce pregnancy sickness. Of 210 pregnant women, those who smoked cigarettes regularly during or prior to early pregnancy suffered much lower rates of pregnancy sickness (52%) than did non-smokers (79%). Although at first glance these findings seem to confound the relationship between pregnancy sickness and teratogens--Little and Hook even suggest that the reason women with pregnancy sickness have higher pregnancy success rates may simply be that such women are less likely to be smokers--on further inspection, the results serve to emphasize the importance of environmental cues in eliciting pregnancy sickness. Smoking has been shown in some studies to interfere with olfactory and taste chemoreception (Arfman & Chapanis, 1962; Hubert *et al.*, 1980; Cain, 1980), thereby decreasing the smoker's sensitivity to cues that warn against toxic foods. Taste thresholds for bitterness increase for heavy smokers (Kaplan & Glanville, 1964), which means that pregnant women who smoke may be less sensitive than others to the the bitter warnings of toxic plant foods. Although studies with a larger number of women should be performed to determine the response of women with pregnancy sickness to modern teratogens, the studies by Hook strengthen the view that pregnancy sickness is induced by cues suggestive of Pleistocene toxins.

Selection pressures favoring pregnancy sickness may be lower today than in the Pleistocene, because modern exposure to a wide range of artificially-created toxins may have diminished the importance of means for avoiding

Pleistocene toxins. Even though the benefit of having pregnancy sickness--protecting the embryo from Pleistocene teratogens--may have been higher in the Pleistocene relative to the cost of having pregnancy sickness--the risk of malnourishment from ingesting fewer nutrients, the decrease in productivity due to feeling sick, and the slight risk of death due to excessive vomiting (hyperemesis gravidarum)--the prevalence of pregnancy sickness in modern societies is significant: 75% of pregnant women suffer from pregnancy sickness (Little & Hook, 1979), and 50% become nauseated enough to vomit (Klebanoff *et al.*, 1985). The variableness of pregnancy sickness among women in modern societies may perhaps indicate a decrease in selection pressures for pregnancy sickness, although data for comparison on the variableness of pregnancy sickness among hunter-gatherer women is lacking. If new teratogens are artificially created at a rate faster than teratogens evolved in the Pleistocene, and in forms radically at variance with the forms of Pleistocene toxins, then the mechanisms triggering pregnancy sickness will not be able to evolve quickly enough to respond to the teratogens and to protect the embryo. In this case, pregnancy sickness eventually may become much less predominant among humans.

Conclusion

Humans are the only mammals known to experience pregnancy sickness, where vomiting due to pregnancy is the indication of pregnancy sickness (Fairweather, 1968). However, data on pregnancy sickness in mammals are scant, and other mammals display means for avoiding toxins, such as by developing aversions to foods which make them ill (Garcia & Koelling, 1969; Rozin & Kalat, 1971). Experimental omnivores and herbivores, in particular, confront significant pressures to avoid the elaborate toxic defenses of plants. Chacma baboons, for example, adapt to toxic food sources by processing most plant and animal foods before eating them, and by ingesting the more toxic plants later in the day after ingesting enough other foods to dilute the concentration of toxins (Hamilton, 1978). Studies could be performed to discover whether other mammals change their taste preferences during pregnancy, which might indicate a mild form of pregnancy sickness, or might indicate another mechanism of food aversion that has evolved to deter females from inflicting toxins on their embryos.

Effects of pregnancy sickness on the human embryo could be measured through experiments that show the extent to which pregnant women avoid ingesting or inhaling common toxins during the first trimester of pregnancy. Such tests might compare the toxicity of the diets of pregnant women who have pregnancy sickness, pregnant women who do not have pregnancy sickness, and women who are not pregnant. Tests of women in both agricultural and hunter-gatherer cultures could be compared to find out whether pregnancy sickness is as prevalent in both cultures and whether pregnancy sickness is induced by similar substances in both cultures. An experiment suggested by Eileen O'Brien (personal communication) to test the degree of a pregnant woman's sensitivity

to cues for toxic substances, is to measure responsiveness by pregnant women to seasonality cues in plants that have the dual characteristic of being edible during one season but highly toxic in another season. Conclusions of such experiments concerning the effectiveness of pregnancy sickness should help pregnant women determine whether or not to welcome the experience of pregnancy sickness.

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