Differences in Dietary Intake as a Function of Sexual Activity and Hormonal Contraception

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Abstract: As a consequence of the need to downregulate some maternal immune responses so as to tolerate paternal genetic material following conception, the luteal phase of the menstrual cycle is associated with increased susceptibility to infection. Because meat was one of the primary sources of foodborne pathogens throughout our evolutionary history, Fessler (2001) predicted a decrease in meat intake during the luteal phase; the current research provides the first test of this prediction. Based on the assumption that any such behavioral changes would be hormonally mediated, we also investigated the effects of varying levels of exogenous hormones on meat consumption by examining dietary intake in women using hormonal contraceptives. Lastly, because, from a functional perspective, immunomodulation is unnecessary during anovulatory cycles and in women who are not currently sexually active, luteal phase compensatory behavioral prophylaxis was predicted to be absent in these contexts. Although we find that women who are sexually active eat less meat than those who are not, we do not find support for the core prediction regarding effect of cycle phase on meat consumption, nor do we find support for the ancillary prediction that meat consumption would be influenced by the presence or withdrawal of exogenous hormones. We replicate the finding that periovulatory total food intake is decreased compared to the rest of the cycle and find that sexually active women show a greater periovulatory decrease in food intake than sexually inactive women.

Keywords: meat, menstrual cycle, progesterone, sexual activity, hormonal contraception, behavioral prophylaxis, immunity

Introduction

Meat has been an important source of calories and nutrients throughout our evolutionary history (Mann, 2000; Milton, 1999). However, meat is also a principal source
of foodborne illness, as animals carry pathogenic endosymbionts and parasites, and microbes proliferate on meat (reviewed in Fessler, 2002). Triggered by alterations in hormone levels, female immune functioning varies systematically across the menstrual cycle, potentially influencing the costs of meat consumption. In this paper we test four adaptationist predictions regarding meat consumption as a function of hormone levels and behavioral cues of conception risk.

Progesterone is an important hormone, levels of which vary over the course of a woman’s menstrual cycle, and undergo substantial changes over the lifespan. Progesterone causes immunomodulation, the downregulation of certain components of immune response during the first trimester of pregnancy and during the luteal phase of the menstrual cycle. This is mediated by progesterone induced blocking factor (PIBF). PIBF shifts the maternal immune system towards anti-inflammatory signals.

The female menstrual cycle consists of functionally distinct phases marked by characteristic variations in hormonal levels. The highest levels of progesterone outside of pregnancy characterize the luteal phase of the menstrual cycle. The luteal phase is the period after the rupture of the ovarian follicle in which the corpus luteum secretes progesterone (Hatcher and Nannoum, 2004). During the luteal phase, the body prepares for conception and implantation of the blastocyst by down-regulating inflammatory immune responses. This immunomodulation is an adaptation that prevents the maternal immune system from attacking the fertilized egg which is 50% unrelated to the mother. The downregulation of facets of immunity during the luteal phase is apparent in a variety of ways. During the luteal phase levels of inflammation-enhancing cytokines (chemical messengers that mediate immune responses) decline, and the responsiveness of first-line cellular defenses, such as natural killer cells, is downregulated. Correspondingly, susceptibility to a variety of pathogens increases during the luteal phase, and chronic infections that are normally managed through inflammatory responses are exacerbated. Finally, indirect evidence of these changes in immune function can be found in cyclic alterations in the severity of autoimmune diseases, as conditions, such as rheumatoid arthritis, in which excess inflammation causes pathology, are ameliorated, while those, such as lupus erythematosus, in which excess anti-inflammatory signals are implicated, worsen (for reviews of luteal phase changes in immune functioning, infectious illness, and autoimmune pathology, see Doyle, Swain Ewald, and Ewald, 2007; Fessler, 2001).

From a functional perspective, the goal of luteal phase immunomodulation is tolerance of the half-foreign conceptus. Implantation and subsequent fetal/placental development are highly invasive events of a type that, in the absence of immunomodulation, would normally trigger an aggressive inflammatory response. Accordingly, once implantation occurs, the downregulation of inflammatory responses escalates, regulated by rising levels of progesterone and PIBF, with corresponding increases in vulnerability to infection (reviewed in Fessler, 2002). While the escalation of immunomodulation in pregnancy constitutes the ultimate culmination of the process that begins in the luteal phase, in this paper we concern ourselves solely with the latter.

Fessler (2001) hypothesized that, given the potential hazards of meat consumption, a reduced attraction to meat during the luteal phase would be adaptive as changes in dietary behavior could prophylactically compensate for increased luteal phase susceptibility to foodborne illness. This hypothesis builds on the notion that gestational aversions to meat serve a prophylactic function (Fessler, 2002; Flaxman and Sherman, 2000), the idea being
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that the increased vulnerability to infection that is a consequence of the need to tolerate foreign genetic material during pregnancy is not limited to gestation, but rather also occurs, in an anticipatory (and somewhat muted) form, during the luteal phase, with concomitant changes in susceptibility to foodborne illness.

Fluctuations in dietary behavior across the menstrual cycle have been the focus of numerous investigations. However, whereas it has been repeatedly shown that females’ total energy intake increases during the luteal phase, findings vary with regard to patterns of macronutrient consumption during this period. Some researchers find a decrease in protein intake during the luteal phase (Alberti-Fidanza, Fruttini, and Servili, 1998), while others report a significant luteal increase in protein intake (Chapell and Hackney, 1997; Gallant, Bowering, Short, Turki, and Badawy, 1987; Lyons, Truswell, Mira, Vizzard, and Abraham, 1989); most simply find no significant effect of phase on protein intake (Johnson, Corrigan, Lemmon, Bergeron, and Crusco, 1994; Tarasuk and Beaton, 1991; Wurtman, Brzezinski, Wurtman, and LaFerrere, 1989). For comprehensive reviews of the diet and menstrual cycle literature (see Dye and Blundell, 1997; Fessler, 2001; and Vlitos and Davies, 1996).

The studies cited above vary substantially in their methodology and participant characteristics. Some of these studies deal specifically with women with premenstrual syndrome or late luteal phase dysphoric disorder (LLPDD) (Gallant et al., 1987; Wurtman et al., 1989). More importantly, because none of these studies were designed with the compensatory prophylaxis hypothesis in mind, meat intake was not examined directly; rather, food types were collapsed into macronutrient categories that encompass foods of varying pathogenic potential.

Consistent with the fact that menstrual cycle variation in immune functioning is hormonally mediated, hormonal contraceptives (HCPs) have been shown to have immunosuppressive effects. The principal exogenous progestins found in commonly prescribed oral contraceptives ameliorate inflammatory autoimmune diseases, reduce the number of natural killer (NK) cells, and lower natural cytotoxic activity. Correspondingly, women on both conventional and triphasic oral contraceptives report more respiratory illness (a clear index of enhanced vulnerability to pathogens) and more gastrointestinal distress (a possible indication of enhanced vulnerability to pathogens) than nonusers (Auerbach, Hafner, Huber, and Panzer, 2002; Scanlan, Werner, Legg, and Laudenslager, 1995). The major exogenous progestin that has not been found to have immunosuppressive effects, Desogestrel (Auerbach et al., 2002), was not used by any participants in our hormonal contraception using sample. For an extensive review of the effects of HCPs on immune functioning, see Doyle, Swain Ewald, and Ewald (2007).

Combined HCPs (so called because they contain both the synthetic hormones ethinyl estradiol and progestinone) essentially “flatline” the levels of naturally occurring estrogen and progesterone by using synthetic hormones to inhibit endogenous production of their endogenous equivalents. HCPs have two main phases: the “active” phase in which women are taking both synthetic hormones, and the “hormone free interval” or placebo period in which women receive no synthetic hormones (by either skipping HCPs entirely or taking placeholder pills that contain no hormones) (Sulak, Carl, Gopalakrishnan, Coffee, and Kuehl, 2004; Sulak, Scow, Preece, Riggs, and Kuehl, 2000). During the active phase HCPs keep a steady amount of ethinyl estradiol in the woman’s bloodstream, thus supplying the constant hormone levels that prevent ovulation. Depending on the
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formulation, combined HCPs supply either a steady level (e.g., Nuva ring) or a variable level (e.g., Ortho Tri-Cyclen) of progestins during the active phase. The drop in progestins that occurs during the hormone-free interval triggers the onset of menstruation. Given that progestins have immunosuppressive effects similar to endogenous progesterone, we hypothesize that exogenous progestins should reduce meat intake. Conversely, we expect to see an increase in meat intake during the hormone-free interval, when there is a lapse in progestin and concomitant immunosuppressive effects.

A consideration of the importance of meat in the human diet suggests that cyclic factors may not be the sole determinants of reproductive immunomodulation and, concomitantly, meat consumption. Meat plays a prominent role in the lives of many contemporary hunter-gatherers, constituting between 23% and 80% of the diet (Waguespack, 2005). Although there is debate regarding the exact manner in which meat-eating influenced hominid evolution, as underscored by recent work focusing on the importance of dietary long-chain fatty acids (see Finch and Stanford, 2004), it is reasonable to suppose that, for ancestral women, frequent abstention from meat eating would have been costly. In its original formulation, the compensatory prophylaxis hypothesis predicts luteal phase reductions in meat consumption in all nonpregnant women (Fessler, 2001). However, if the function of luteal phase immunomodulation is to facilitate tolerance of the conceptus, in light of the costs of this immunomodulation, we might expect such changes to occur only in cycles in which conception is likely. Correspondingly, the hypothesized luteal phase increase in costly behavioral prophylaxis should be absent during anovulatory cycles, and should similarly not occur in women who are not sexually active. Women who are using HCPs, though not ovulating, have a steady level of progestins that may mimic the luteal phase, and therefore also decrease meat consumption. In addition to failing to segregate meat from other sources of protein, all studies to date have failed to consider the variables of sexual activity and exogenous hormone administration.

Predictions

The above reasoning generates four predictions regarding changes in meat intake:
1) Rates of meat intake will decrease in the luteal phase relative to other phases of the menstrual cycle.
2) Rates of meat intake will be lower during the active phase of the HCP cycle than during the hormone-free phase.
3) Naturally cycling sexually active women will eat less meat than naturally cycling women who are not sexually active.
4) Women using HCPs should show the same overall meat intake as sexually active naturally cycling women.

Methods

Female participants were recruited from the Introduction to Psychology subject pool at the University of Texas at Austin. Participants received course credit for their participation. All participants were treated in accordance with the ethical guidelines of the Human Subjects Review Board at the University of Texas at Austin, including safeguards for their privacy and confidentiality. Participants completed a background questionnaire regarding their height, weight, health history, sexual activity, dietary habits, allergies,
prescription medication, and exercise habits. Participants were also directed to a web-based survey, which they were to fill out on a daily basis, addressing dietary and other behaviors (the survey can be viewed at http://www.epjournal.net/filestore/dietsurvey.html). Students in the Introduction to Psychology subject pool are required to earn five experimental credits (or write a paper) as a class requirement; participants earned one experimental credit for each week that they filled out the dietary survey. If participants did not fill out the survey for 48 hours, they were sent a reminder email with their subject number and a link to the survey. Participants were asked to complete the daily online survey at approximately the same time each day. The survey addressed food intake in the last 24 hours. Survey items included questions about the number and size of servings of specific types of meat eaten (red meat, chicken, fish and shellfish, pork), as well as all of the other major food groups, including milk and milk products, eggs, vegetables, fruits, lentils, and starches. Participants were told how much of a given food category and subcategory counted as one serving. Additionally, for most food types we provided a familiar reference object or amount to help participants estimate serving sizes (e.g., one serving is 1 oz processed cheese = size of a pair of dice or your thumb, etc.).

Participants
Sixty-seven participants filled out surveys. For all analyses, we excluded participants who did not fill out the survey for at least five days, participants on any type of hormonal medication other than combined HCPs, participants who had been taking HCPs for less than three cycles, participants taking any drug that might alter immune functioning (e.g., anti-allergy drugs), participants taking antidepressants or anti-psychotics, and vegetarians. Participants were also asked about other dietary restrictions (e.g., Kosher, pescetarian, no sugar diet, low carbohydrate diet) but, in this sample, the previous criteria resulted in the exclusion of all subjects who answered that they had such dietary restrictions. For the analyses of predictions 1 and 2, which involved examining diet over time, we excluded participants who did not fill out the survey for at least 12 days. This left a subset of 31 total participants, of which 10 were on HCPs and 21 were naturally cycling. These participants ranged in age from 18 to 22; the mean age was 19.00.

For predictions 3 and 4, we used participants for whom we had at least five days of dietary information. This left a subset of 38 participants, 14 of whom were on HCPs and 24 of whom were naturally cycling. These participants also ranged in age from 18 to 22; the mean age was 18.95.

Estimation of phase day
Using a combination of counting forward and counting back methodology, we estimated the day of ovulation and assigned each daily dietary survey to a phase. Because the luteal phase is less variable in length than the follicular phase (Hatcher and Namnoum, 2004), counting backward from the date of next menstruation is the best way, aside from physiological measures, to estimate date of ovulation and thus menstrual cycle phase. Out of 21 participants, we had obtained both a forward and backward day of menstruation for 17. For those survey days for which we had a date of next menstruation, we used a reverse counting method to estimate phase day (Haselton and Gangestad, 2006). For those days for which we had only a date of onset of previous menstruation, we used the participants’
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previous cycle length to estimate cycle phase, standardizing all cycles to a 29 day cycle (Gangestad and Thornhill, 1998).

The follicular phase was defined as the period from first day of menstruation to the estimated day of ovulation (days 1-14 of the cycle). The luteal phase was defined as the period from the day after estimated ovulation to the onset of subsequent menstruation (days 15-29). Assigning each dietary survey to either the follicular or luteal phase, we then averaged meat intake within phases, resulting in a design in which each of the 21 naturally cycling participants had two scores, representing their average meat intake for each of the two phases.

Results

Categories of participants did not differ significantly in age or body mass index. For prediction 1, that rates of meat intake would decrease luteally, we employed a paired samples t-test. For most results t-tests as well as effect sizes are reported. Cohen’s $d$ is a measure of effect size that is the difference between means divided by the standard deviation. According to Cohen, $d$ of 0.20 indicates a small relationship, 0.40 indicates a moderate relationship, and 0.80 indicates a strong relationship (for more information, see Cohen, 1988). The results of a paired $t$-test of luteal total meat intake versus nonluteal total meat intake revealed no significant main effect of phase [$t(20) = -1.21$, $p_{\text{one-tailed}} = 0.17$, Cohen’s $d = 0.13$]. Separating the data into three phases (menstrual, follicular, and luteal) or correlating meat consumption with estimated progesterone levels based on cycle day (Finn et al., 1988) also yielded nonsignificant results. One reviewer of this article suggested that a comparison between the luteal and follicular phase in compiled animal-derived and non-animal-derived food groups should also be made considering that foods derived from animals would all ostensibly have increased pathogen prevalence. We employed a paired samples $t$-test to investigate the prediction that animal-derived foods (dairy and all meats and fish) would be consumed less in the luteal phase [$t(20) = -1.49$, $p_{\text{two-tailed}} = 0.15$, Cohen’s $d = 0.18$]. Non-animal foods were also consumed slightly, but also not significantly, less in the luteal phase [$t(20) = -1.90$, $p_{\text{two-tailed}} = 0.15$, Cohen’s $d = 0.18$]. We did not find that sexually active naturally cycling women and sexually inactive naturally cycling women showed any difference in luteal phase meat consumption.

For prediction 2, that rates of meat intake will decrease when women are in the active phase of their HCP regiment, we categorized days as being active phase or hormone-free phase, then averaged meat intake within each phase and performed a paired sample $t$-test between phases. The prediction was not supported [$t(9) = 0.20$, $p_{\text{one-tailed}} = 0.42$, Cohen’s $d = 0.04$].

For prediction 3, that naturally cycling women who are sexually active will report less meat intake than those who are not sexually active, we averaged meat intake for the entire period in which the participants filled out surveys and compared daily food intake means using an independent samples $t$-test. Naturally cycling women who were sexually active reported significantly less meat intake than naturally cycling women who were not sexually active (see Table 1, “all meat types”). As evident in Table 1, results were not due to significant differences in total servings of all food types consumed, indicating that the pattern was specific to meat consumption.
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Table 1: t values of the differences between average daily servings of food types between sexually active and sexually inactive naturally cycling women

| Food type   | t    | p    |
|-------------|------|------|
| N           |      |      |
| beef        | .96  | 0.17§|
| pork        | 1.31 | .10§ |
| poultry     | 2.11 | .02§ |
| fish        | .90  | .18§ |
| all meat types | 2.90 | .004*§|
| milk        | .65  | .53  |
| eggs        | -.66 | .51  |
| fruit       | -.16 | .88  |
| vegetables  | .01  | .99  |
| starches    | -.72 | .48  |
| sweets      | .70  | .49  |
| Total servings | .97  | .34  |

*significant at p<.005
§ significant at p<.005
p one-tailed

Table 2: Average daily intake of food types for four groups of women divided by sexual activity and hormone presence

|                  | Naturally cycling and not sexually active | Naturally cycling and sexually active | On hormonal contraception and not sexually active | On hormonal contraception and sexually active |
|------------------|-------------------------------------------|--------------------------------------|--------------------------------------------------|-----------------------------------------------|
|                  | mean SD min max | mean SD min max | mean SD min max | mean SD min max |
| beef             | 2.06 0.58 0.00 2.06 | 0.63 0.33 0.23 1.31 | 1.00 0.58 0.37 1.51 | 0.72 0.60 0.08 1.77 |
| pork             | 0.45 0.49 0.70 1.44 | 0.25 0.24 0.00 0.66 | 0.27 0.28 0.00 0.56 | 0.26 0.20 0.00 0.66 |
| poultry          | 1.40 0.67 0.00 3.00 | 0.93 0.39 0.41 1.60 | 0.81 0.34 0.56 1.20 | 0.66 0.33 0.00 1.07 |
| fish             | 0.34 0.38 0.00 1.21 | 0.22 0.26 0.00 0.67 | 0.46 0.08 0.38 0.52 | 0.36 0.35 0.00 0.88 |
| all meat types   | *3.01 1.06 1.69 5.31 | *2.02 0.51 1.27 2.69 | 1.25 0.51 1.00 1.44 | 1.20 1.10 0.58 4.02 |
| milk             | 1.32 0.81 0.27 3.00 | 1.14 0.55 0.43 2.30 | 0.99 0.84 0.15 1.83 | 1.57 0.74 0.58 3.03 |
| eggs             | 0.28 0.22 0.00 0.64 | 0.38 0.44 0.00 1.64 | 0.56 0.24 0.30 0.75 | 0.41 0.29 0.00 0.77 |
| fruit            | 0.98 0.56 0.06 1.80 | 1.01 0.45 0.34 1.75 | 1.76 0.67 1.05 2.37 | 0.57 0.48 0.00 1.70 |
| veg              | 1.30 0.83 0.44 2.97 | 1.29 0.71 0.19 2.33 | 1.80 0.83 1.27 2.76 | 1.00 0.43 0.41 1.88 |
| starch           | 2.53 1.03 1.46 4.50 | 2.81 0.88 1.00 4.05 | 3.06 0.53 2.50 3.56 | 3.15 1.02 1.25 4.50 |
| sweets           | 1.21 0.94 0.28 3.85 | 0.99 0.59 0.24 2.02 | 1.46 0.59 0.78 1.85 | 1.00 0.50 0.25 1.85 |
| Total servings   | 10.99 3.32 6.34 18.85 | 9.86 2.32 6.68 13.58 | 12.91 0.56 12.44 13.52 | 10.12 2.81 5.63 14.07 |

*significant at p < .005
† Women on HCP have significantly lower total meat intake, p < .05

Prediction 4 held that women using HCPs would show the same overall meat intake as sexually active naturally cycling women. We found that women on HCPs did not have a significantly different level of meat intake compared to naturally cycling sexually active women. In contrast, women on HCPs showed significantly lower meat intake than naturally cycling women who were not sexually active [t(24) = 2.28, p one-tailed < .02, Cohen’s d = 0.90]. We tested to see whether sexual activity or the external administration of progestins
Differences in dietary intake among women were driving the reduction in meat intake among women using HCPs by separating sexually active HCP-using participants from those who were not sexually active. We did not find a significant difference between sexually active and inactive women using HCP \[ t(12) = 0.87, \ p_{\text{one-tailed}} = .20, \ \text{Cohen’s } d = 0.57 \]. However, because there were only three sexually inactive HCP-using participants, we do not place much stock in this result.

Previous studies have shown that total caloric intake decreases in the periovulatory period (Fessler, 2003). As an indirect measure of the soundness of our survey method, we tested for replication of this finding using servings as a proxy for calories. Examining days 12, 13, and 14 of the standardized menstrual cycle of the first subset of naturally cycling women (those 21 women for whom we had at least 12 days of dietary information), we averaged the total servings eaten across these three days and compared this with the average daily food intake during the rest of the menstrual cycle using a paired samples \( t \)-test. Results reveal a periovulatory nadir in food intake \( t(20) = -2.24, \ p_{\text{one-tailed}} = .02, \ \text{Cohen’s } d = 0.27 \), akin to that previously reported in the literature, suggesting that our survey method suffices to capture the phenomena of interest. Furthermore, we found that sexually active women were driving this effect. Among women that were not sexually active there was not a significant difference between total serving intake during the fertile window and outside the fertile window though the difference was in the expected direction \( t(10) = -1.33, \ p_{\text{one-tailed}} = .11, \ \text{Cohen’s } d = .22 \). Among sexually active women a paired \( t \)-test revealed a much greater difference in periovulatory total serving intake compared to that outside the fertile window \( t(10) = -2.48, \ p_{\text{one-tailed}} = .02, \ \text{Cohen’s } d = .38 \).

**Discussion**

Neither the core prediction of the compensatory prophylaxis hypothesis regarding the relationship between menstrual cycle phase and meat consumption nor the ancillary prediction regarding the simple effect of exogenous progestins on meat consumption was supported. Support was found for the ancillary prediction that meat consumption would be lower in sexually active than in non-sexually active women; however, given the lack of support for the core prediction, we must entertain the possibility that some third factor is responsible for this finding. That third factor could be where women lived; for example, perhaps women who live with their parents are more likely to eat “home cooked” meals containing more meat, and also less likely to be involved in a sexual relationship.

We found that sexual activity did not just moderate a decrease in meat intake but also those women that were sexually active were driving the periovulatory nadir in total serving intake. As the periovulatory nadir is thought to occur in order to free up time for mating (Fessler, 2003) perhaps being sexually active is an environmental cue that changes energy allocation periovulatorily as well as motivating women to decrease exposure to possible meat-borne contaminants.

Might limitations in our methods have influenced our findings? First, the women in our sample were all relatively young. This is potentially relevant given that young women are more likely to have anovulatory menstrual cycles. We did not employ physiological measures that directly index ovulation; our null results might reflect our inability to differentiate between ovulatory and anovulatory cycles in a dataset potentially containing many of the latter. However, our detection of a periovulatory nadir in food intake casts doubt on this explanation and analogous functionalist reasoning dictates that the midcycle
reduction in food intake ought not to occur in anovulatory cycles—if our dataset captured a sufficient number of ovulatory cycles to reveal the periovulatory nadir, it ought to have also shown a luteal reduction in meat consumption.

We coded as “sexually active” women who reported having had sex in one of the daily surveys completed. However, if immune responsiveness is moderated by sexual activity, then we might reasonably expect a time lag between the cessation of sexual activity and reversion to baseline levels of immune functioning and concomitant compensatory prophylactic behavior. This suggests that the predicted correlations could be disrupted if some of the women whom we coded as not sexually active had been active until recently.

Equivalently problematic, given that University of Texas at Austin demographics suggest that many of our participants were both unmarried and culturally conservative, it is possible that some women who reported not being sexually active in fact were. This may have been particularly problematic in the case of the three women who stated they were not sexually active but were using HCP, keeping in mind that we excluded participants who had any reproductive problems that would have warranted therapeutic use of HCP.

In a separate study, not reported here, conducted at the same time and using the same sample as this study we found support for the compensatory prophylaxis hypothesis in the form of systematic alterations in disgust sensitivity and contamination-related thoughts and behaviors across the menstrual cycle (Fleischman and Fessler, 2006). It is therefore possible that the core features of the compensatory prophylaxis hypothesis are correct, yet such alterations in behavior do not extend to diet, perhaps because the chronology of meatborne illness is such that cyclic alterations in meat consumption would not provide protection from infection. The immune components that are reduced during the luteal phase of the menstrual cycle are mostly those that counteract parasitic or bacterial infection, but foodborne parasites and pathogenic bacteria take variable intervals of time to make their way from the stomach into the gut. Some common meatborne pathogens, such as *Clostridium perfingens*, take only hours to develop clinically, while others may take several days or weeks (Bloom, 2002). Additionally, many parasites and bacteria are opportunistic, waiting for a lapse in immunity before they begin proliferating in the gut. For example, *Toxoplasma gondii*, a common meatborne parasite, takes many months to develop into an infection, and is opportunistic. (Walker, Roberts, Ferguson, Jebbari, and Alexander, 1997). Other common meatborne pathogens, such as *Listeria monocytogenes*, are opportunistic and have variable incubation periods ranging from 2-70 days (St. Georgiev, 2003). Given the long incubation periods characteristic of some of the most common meatborne pathogens that pose a threat to women (see Fessler, 2002), infections from meat may not have exerted selection pressure for cyclical aversion to meat. Instead, such pathogens may have favored a lower attraction to meat in women relative to men, a possibility consistent with the observation that women in many societies consume less meat than men, and, indeed, are more likely to be vegetarians (reviewed in Fessler, 2001).

**Acknowledgements:** Thanks go to Connie Bogard, Praradhana Challapalli, Lindsay Harrington, Sarah Hebble, Sari M Hochberger, Amanda Maggard, Claire McCarthy, Megan Senior, Jennifer Anne Stokes and Ashley Szabo who assisted in developing and conducting this research.
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Thanks to Valentin Boerner, David Buss, Jaime Confer, Judith Easton, Louisa Fleischman, Sarah E. Hill, Joonghwan Jeon, David Lewis, and Carin Perilloux for valuable feedback on an earlier version of this manuscript.

Received 12 June 2007; Revision submitted 24 August 2007; Accepted 27 August 2007

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Footnotes:
1. The $p$ value .005 is the Bonferroni corrected $p$ value when .05 is divided by 10 independent comparisons.