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Preeclampsia Argues Against an Ovulatory Shift in Female Mate Preferences

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Ovulatory Shift

A number of researchers have posited that human females experience shifts in their preferences for various masculine traits around the time of ovulation, which is the point of peak fertility in the menstrual cycle. The tradeoff is purported to be one where the periovulatory female favors a short-term mating bout with a male who exhibits traits of high genetic quality and/or attractiveness, such as highly masculinized features or a high degree of facial symmetry. This shift toward a short-term mating strategy may come either at the cost of a preexisting long-term relationship (cuckoldry) or in lieu of a long-term relationship. Most often, these ideas are conceived in terms of extra-pair copulations where securing access to the genetic material of a high-quality male is achieved while retaining the caregiving and provisioning of the male from the preexisting long-term pair-bond. A corollary of this tradeoff is the assertion that males with attractive, highly masculinized features are unlikely to be reliable caregivers / providers over the long-term either in general to all their mates or in specific to the female who can only secure their attention for short-term mating.

This purported tradeoff goes by many names, including, but not limited to: the “good genes” or “good genes ovulatory shift hypothesis” (Gangestad & Thornhill, 1998; Gangestad et al., 2005), the “dual-mating strategy hypothesis” (Pillsworth & Haselton, 2006), or “strategic pluralism” (Gangestad & Simpson, 2000). Here, for the sake of brevity, I will refer to these overlapping ideas simply as “ovulatory shift.” Frequently, ovulatory shift is invoked to explain concealed ovulation and the seemingly maladaptive receptivity to mating outside of the fertile period seen in human females. In this line of thinking, a female can continue to mate with a pair-bonded male, thereby securing his paternal provisioning for future offspring, while surreptitiously securing an extra-pair copulation with a higher-quality male who will provide superior genetic material, i.e., cuckoldry. I will argue here that a more thorough accounting of preeclampsia’s role in human pregnancy both renders ovulatory shift hypotheses seriously miscalculated and helps potentially explain concealed ovulation in humans.

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First, let us establish that the evidence for ovulatory shift is mixed at best (Buss & Schmitt, 2019). While an exhaustive discussion of this literature is outside the scope of this work, I will attempt to provide an overview. Much confusion arose in 2014 with one meta-review on the evidence for the ovulatory shift hypothesis finding “robust relationship context-dependent cycle shifts in women’s mate preferences” (Gildersleeve et al., 2014) and another, published in the same year, concluding that most of the observed effects are likely due to research artifacts (Wood et al., 2014). Since then, there have been several objections raised on the grounds of statistical rigor and experimental design (Arslan et al., 2018; Shirazi et al., 2019). Since these meta-reviews were conducted, there has been equivocal support found such that both intra-pair and extra-pair desire increases around ovulation (Arslan et al., 2018). There have also been additional failures of replication (Jones et al., 2018), including a longitudinal study using a pre-registered design and concomitant hormone measurements that found no evidence for mate preference shifts across the menstrual cycle (Stern et al., 2021). This work by Stern et al. also contains an up-to-date summary of research around these ideas that interested readers would do well to pursue. Nonetheless, ovulatory shift ideas still enjoy widespread cachet. Recently, the impact of the male partner’s attractiveness or investment in the relationship has each been invoked to add nuance and explain failures in tests of ovulatory shift hypotheses (Gangestad & Dinh, 2022).

Preeclampsia

Before discussing how preeclampsia factors into the trade-offs inherent to a short-term mating strategy, an introduction to the condition is in order. Preeclampsia refers to the chronically elevated levels of blood pressure during pregnancy that occurs prior to the onset of eclampsia, which is hypertension-induced seizures. Seizures during pregnancy have been described since antiquity the world over (Robillard, 2018). Today, preeclampsia affects 2–8% of pregnancies worldwide and is frequently cited as the leading cause of maternal mortality (Magee et al., 2022; Rätsep et al., 2016). Preeclampsia occurs either as early onset, which will be the focus of this discussion, or late-onset. Late-onset preeclampsia seems driven more by maternal factors—such as obesity (Bicocca et al., 2020; Staff & Redman, 2018)—and is thought to have seen an increased incidence in modern times, to the point where it now accounts for 90% of preeclampsia cases in developed countries (Lisonkova et al., 2014; Robillard et al., 2019). Meanwhile, early onset preeclampsia is $\sim 4 \times$ as deadly as late-onset preeclampsia (Lisonkova et al., 2014). Similarly, severe maternal morbidity can be up to $21 \times$ higher for early onset preeclampsia (Lisonkova et al., 2014). There is no cure for either form of preeclampsia other than labor induction or cesarean delivery and even with modern medical care, the rate of perinatal mortality among mothers with preeclampsia is 5–11% in high income countries and as high as 40% in low and middle income countries (Tlaye et al., 2020). We do not know how pre-modern foragers fared with preeclampsia, but contemporary nomads and pastoralists show us that vulnerability to heat and lack of housing seem to increase preeclampsia morbidity (Baharav et al., 2023; Mekonen et al., 2018). Obviously, selection would not have favored any reproductive strategies that increase the risk of preeclampsia unless the countervailing benefits were enormous.

Proximately, preeclampsia is caused by defective implantation of the placenta during the second phase of trophoblastic invasion. This limits the supply of nutrients the fetus receives,

and hence, maternal blood pressure is increased in an attempt to overcome the insufficient placental perfusion. It should be noted that while all anthropoid primates have highly invasive placentas, humans are unique in the degree of invasiveness as well as in risking preeclampsia (Rosenberg & Trevathan, 2007). We will return later to the question of the ultimate causes of preeclampsia, but first, let us consider the outcomes for both mother and offspring born after surviving a gestation with preeclampsia.

Consequences of Preeclampsia

The risks for a mother who survives preeclampsia do not necessarily end upon delivery of the baby. Such mothers are at increased cardiovascular risk during the decades after delivery, including a ~ 50% increased risk of ischemic heart disease, five-fold increased risk of hypertension, three-fold increased risk of cardiovascular death, and approximately double the risk of heart failure and stroke (Frost et al., 2021). Importantly, these risks are calculated after adjustment for factors such as body mass index and smoking, which increase risk for both preeclampsia and cardiovascular disease.

For offspring, the risk profile that follows in the wake of preeclampsia is both wide and deep. Occurring during the formative months of gestation, preeclampsia exerts lifelong consequences on offspring health across a number of domains. Newborns delivered after preeclampsia face fetal growth restriction and increased risk of prematurity (Magee et al., 2022). The risk for cerebral palsy is nearly doubled across all cases of preeclampsia, a figure which rises to $3.4 \times$ in cases of early onset preeclampsia (a distinction we will return to later) (Mann et al., 2011). Children of preeclamptic pregnancies also face a 30% increased risk for attention-deficit/hyperactivity disorder and a 32–50% increased risk of autism spectrum disorder (Gumusoglu et al., 2020; Maher et al., 2018; Walker et al., 2015). As adults, offspring of preeclamptic pregnancies are at greater risk for anxiety, depression, schizophrenia, and psychotic symptoms (Gumusoglu et al., 2020). Throughout life, these offspring show lower scores on intelligence tests and reduced cognitive functioning (Gumusoglu et al., 2020). Finally, offspring of preeclamptic pregnancies experience a four-fold increased risk of high blood pressure and nearly doubled risk for stroke in later life (Davis et al., 2012).

Again, we are left with the conclusion that any reproductive strategy which increases the risk of preeclampsia would incur substantial penalties on future reproductive fitness. Next, we will see how an ovulatory shift toward a short-term mating strategy would do exactly that.

Preeclampsia, Cohabitation, and Sperm Exposure

Even a transient switch to a short-term mating strategy would increase the risk for preeclampsia. While previous studies had revealed increased risk for preeclampsia in first-time couplings, the association between sexual cohabitation and preeclampsia was first observed almost 30 years ago, by Robillard et al. (1994; see also Robillard et al., 2011), who found that “conception within the first four months of sexual cohabitation of the couple presented a major risk of hypertensive disorders of pregnancy (40–50% incidence), and this risk declined linearly to become very low in women with at least one year of

sexual cohabitation before conception.” More recent findings have reduced that incidence downward substantially, but the protective effect of cohabitation remains. According to the most up-to-date meta-review, published in 2020, first-time mothers in particular see a 37% reduction in preeclampsia risk when they had prior exposure to the father’s semen; this is particularly the case when mothers and father sexually cohabited for at least 12 months prior to the pregnancy (Di Mascio et al., 2020). More recent work from China found that mothers who had 3 or fewer months of sperm exposure faced 10 times the risk for preeclampsia than mothers who had 12 or more months of sperm exposure (Zhu et al., 2021). Similarly, mothers with the highest levels of sperm exposure face a 70% reduced risk of preeclampsia compared to women in the lowest quarter of exposure (Saftlas et al., 2014). For these reasons, preeclampsia is sometimes referred to as a “couple’s disease.” However, studies of barrier contraceptive methods have not found that blockade of semen exposure induces preeclampsia risk (Ness et al., 2004). Readers should also be aware that studies on this topic have not always completely accounted for confounding factors such as maternal body mass index and blood pressure. While some have found that accounting for confounders eliminates the association between sexual cohabitation and preeclampsia (Andraweera et al., 2018), others have found that the association persists after accounting for confounders (Saftlas et al., 2014; Zhu et al., 2021).

It is important to point out that for the vast majority of cases in the studies cited above, the comparisons being made are between committed relationships of varying lengths. That is, we do not have a reliable estimate for how much preeclampsia risk a pregnancy resulting from a first-time mating bout with a new partner would be at. Sometimes implied, sometimes explicit in ovulatory shift hypotheses, is the idea that a woman would be seeking out high-quality males for relatively brief encounters. Our best estimate for how this could affect preeclampsia risk is likely to be found in the research on assisted reproductive technology, which finds that sperm donation results in a 50–60% increased risk of preeclampsia over baseline rates (Allen et al., 2021; González-Comadran et al., 2014), and this risk is diminished if sperm exposure is repeated as in pregnancies resulting from two or more cycles of intrauterine insemination or in vitro fertilization from the same sperm donor. (Hendin et al., 2023).

I should also note that the lack of cohabitation/semen exposure is believed to contribute only to early onset preeclampsia, where symptoms appear prior to 34 weeks into the pregnancy (Robillard et al., 2022). Recall that early onset preeclampsia has $\sim 4 \times$ greater mortality and $21 \times$ greater severe maternal morbidity (Lisonkova et al., 2014). Note also that the risks for the offspring are higher for early onset preeclampsia. Thus, the risk profile for both mother and child following an eclamptic pregnancy resulting from a short-term mating strategy would likely be greater than the figures cited in the preceding paragraphs. In short, the health consequences of such a pregnancy would be severe.

Broader Implications for Human Evolution

Ovulatory shift is frequently invoked when attempting to explain why humans experience concealed ovulation and mate outside of the fertile period. In this line of reasoning, human females conceal their fertility in order to ensure paternal provisioning from males with

whom they can maintain long-term relationships while discreetly obtaining access to higher-quality males when necessary. However, when considerations of preeclampsia are taken into account, we can see how mating outside the fertile period can be advantageous. Such non-conceptive matings introduce the female immune system to the foreign genetic material of her partner and apparently afford a protective effect against the allograft rejection that is thought to underlie early onset preeclampsia (Robillard et al., 2011, 2022). In short, sexual cohabitation provides immunological introduction between the future mother and father so that when the particularly invasive placenta arrives in the womb, it will be welcomed.

Why then are only humans at risk for preeclampsia? This remains an open question, but there are intriguing hints that the evolution of our: large and metabolically expensive brain, long-term pair-bonding (Schacht & Kramer, 2019), extended sexuality (mating outside the fertile period) and preeclampsia are all inter-related (Martin, 2003; Robillard et al., 2008, 2022). Extreme placental invasiveness to the point of risking preeclampsia may be a crude solution to providing the developing fetal brain with enough of a nutrient supply. Humans also possess a suite of traits that have long puzzled researchers, but which make more sense in light of preeclampsia's sensitivity to pre-conceptive semen exposure, including the aforementioned mating outside of fertile periods, committed long-term pair-bonding, but also relatively large testes size for a pair-bonding species and relatively low rates of conception for a given mating attempt. All of these factors might serve to provide additional "familiarization" of the maternal immune system to a potential father's genetic material.

How then could an ovulatory shift strategy persist in the face of the added risk of preeclampsia and its consequences? While many sexually attractive traits are highly heritable, the competitive advantage of a few points of IQ or cm of height must be weighed cautiously. For instance, while highly heritable, intelligence is twice as predictable from polygenic scores among non-adopted than adopted offspring (Cheesman et al., 2020; Okbay et al., 2022), presumably because some of the impact on offspring intelligence is via genes that increase optimal parenting behaviors. A short-term mating with such an individual would confer less genetic payoff than might be expected had the father been available to aid with childrearing.

In order to carry out a dual-mating strategy successfully, such a female would benefit from repeated mating attempts with the high-quality male outside the fertile period, which would require a complete reorientation of this field of study. One could even say that the more interesting question is why human females do not avoid extra-pair copulations during the periovulatory period given the heightened risk? Indeed, although human males are much more likely to pursue short-term mating strategies, human females still use them (Buss & Schmitt, 2019). With estimates of worldwide non-paternity rates ranging from 1.7% to 3.7% (Buss & Schmitt, 2019), we can conclude that, at least in terms of the reproductive consequences, short-term mating is the exception rather than the rule.

Conclusions

Here, I have shown that how a transient switch to a short-term mating strategy around the time of ovulation, implied by ovulatory shift hypotheses to be advantageous over a

long-term mating strategy with a male of lesser quality, introduces substantial risk in the form of heightened susceptibility to preeclampsia. Conservatively, I estimate the increase in preeclampsia risk to be at least 50% over baseline. A preeclamptic pregnancy then not only introduces substantially increased mortality (again, at least 40% by conservative estimate) but dramatic and long-lasting reductions in health and fitness for the surviving mother and offspring, ranging from poor cardiovascular health to neurodevelopmental burdens.

The ovulatory shift hypotheses already had to contend with decidedly mixed results when their predictions were put to the test. Prior to now, the increased risk for preeclampsia had not been figured into the tradeoff which already included risks to the stability of the long-term relationship. Future work will need to envision a scenario where the benefits of access to a high-quality male outweigh the costs of risking preeclampsia or abandon the hypotheses.

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