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The Conspicuous Absence of Placenta Consumption in Human Postpartum Females: The Fire Hypothesis

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The absence of human placentophagy, the maternal consumption of the afterbirth, is puzzling given its ubiquity and probable adaptive value in other mammals. We propose that human fire use may have led to placentophagy avoidance in our species. In our environment of evolutionary adaptedness, gravid women would likely have been regularly exposed to smoke and ash, which is known to contain harmful substances. Because the placenta filters some toxicants which then accumulate there across pregnancy, maternal placentophagy may have had deleterious consequences for the overall fitness of mother, offspring, or both, leading to its elimination from our species' behavioral repertoire.

KEYWORDS placentophagy, afterbirth, fire, toxins

Placentophagy, the maternal consumption of the afterbirth, is ubiquitous in eutherian mammalian species. Of over 4,000 extant terrestrial mammalian species (Wilson and Reeder 2005), only humans, and camelids (camels, llamas, alpacas, guanacos, and vicunas) have been documented as species in which mothers do not routinely consume the placenta postpartum (Hrdy 2009; Kristal 1980; Vaughan and Tibary 2006). Several hypotheses have been advanced to explain maternal placentophagy in eutherian mammals. These include (1) cleaning the nest site and predator avoidance, (2) a shift toward carnivorousness at parturition, (3) general hunger, and (4) specific hunger (for foodstuffs that contain a particular substance; see Kristal 1980 for a

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review). Different species' biologies and ecologies have evoked different explanations, and as a consequence, none of the existing hypotheses can fully account for the behavior across species (Kristal 1980).

Some research has been conducted to understand the benefits of placentophagy among animals. Data from multiple rodent studies strongly support the hypothesis that placental tissue consumption enhances analgesic effects through a specific endogenous opioid pathway (DiPirro and Kristal 2004; Kristal 1991), and similar hypotheses have been proposed for possible comparable effects in human mothers if they were to engage in the behavior (Apari and Rózsa 2006).

While the analgesic enhancing effect of placentophagy has been well studied in rodent models, and is currently the best established benefit of placentophagy supported by rigorous empirical research, other studies have focused on alternative potential benefits of the maternal behavior, including its effects on lactation. Research on postpartum hormonal changes in female rodents allowed to eat the placenta, compared to those in which placentophagy was prevented, found that placentophagic animals had higher prolactin levels than nonplacentophagic control animals (Blank and Friesen 1980). Similarly, a study using rabbits showed decreased lactation in mothers whose placenta and pups were removed postpartum compared to those in the control group (González-Mariscal et al. 1998). Such studies are not without limitations, however, and the results must be interpreted with caution. For instance, placentophagy suppression in the former study, and pup removal in the latter would have interrupted normal parturitional and postpartum processes, and both of these disruptions would have impacted maternal behavior and endocrine function. Beyond these experimental studies, and in particular outside of rodent models, however, little hypothesis testing research has been conducted on maternal placentophagy aimed at understanding the possible adaptive benefit(s) of the behavior.

Although the ultimate adaptive function of placentophagy is still unclear, that placenta is regularly and enthusiastically consumed by parturient females in the overwhelming majority of mammalian species—in some cases even at the cost of immediate neonatal maternal care—suggests a probable benefit of the consumptive behavior. Among our closest mammalian relatives, placentophagy regularly occurs in all nonhuman primate species—although not necessarily with each individual birth (Stewart 1977). The behavior has been recorded across diverse primate species (see table 1), ranging from baboons (Condit and Smith 1994; Dunbar and Dunbar 1974), to tamarins (Price 1990; Pryce et al. 1988), to great and lesser apes (Galdikas 1982; Hooton 1946; Stewart 1977). While no systematic study focusing on placentophagy in primates has been conducted as of yet, the behavior is often recorded in larger studies of primate birth in both captive and wild environments (see Turner et al. 2010). Despite its near-universal frequency among mammals in general, and primates in particular, placentophagy has

TABLE 1 Recorded Placentopahgy in Nonhuman Primate Species

Family	Species	Captive/Wild	References
Cheirogaleidae	Lesser Mouse Lemur (*Microcebus Murinus*)	Captive	in Hayssen, van Tienhoven, and van Tienhoven 1993
Lemuridae	Black lemurs (*Lemur macaco macaco*)	Captive	in Hayssen et al. 1993
	Ringtailed lemurs (*Lemur catta*)	Free-ranging	Sauther 1991
	Ruffed lemurs (*Varecia variegata*)	Captive	in Hayssen et al. 1993
Indriidae	Malagasy prosimians (*Propithecus verreauxi*)	Free-ranging	Richard 1976
	Sifaka (*Propithecus verreauxi coquereli)*	Captive	in Hayssen et al. 1993
Lorisidae	Slender loris (*Loris tardigradus lydekkerianus*)	Captive	in Hayssen et al. 1993; Kadam and Swayamprabha 1980
Galagidae	Lesser Bushbaby (*Galago senegalensis moholi*)	Captive	in Hayssen et al. 1993
Callitrichidae	Common marmoset (*Calthrix jacchus jacclius*)	Captive	in Hayssen et al. 1993; Stevenson and Poole 1976
	Cotton-top tamarin (*Saguinus oedipus*)	Captive	Price 1990
	Red-bellied tamarin (*Saguinus labiatus*)	Captive	Pryce et al. 1988
Callimiconidae	Goeldi's monkey (*Callimico goeldii*)	Captive	in Hayssen et al. 1993
Cebidae	Squirrel monkeys (*Saimiri sciurea*)	Captive	in Hayssen et al. 1993; Hopf 1967; Takeshita 1961
Aotidae	Owl monkey (*Aotus trivirgatus*)	Captive	in Hayssen et al. 1993
Cercopithecidae	Gelada baboon (*Theropitehcus gelada*)	Wild	Dunbar and Dunbar 1974
	Japanese macaque (*Macaca fuscata*)	Wild	Thomsen and Soltis 2000
	Japanese macaque (*Macaca fuscata*)	Free-ranging	Turner et al. 2010
	Japanese macaque (*Mucaca fuscata*)	Captive	Negayama, Negayama, and Kondo 1986
	Java macaque (*Macaca fascicularis*)	Captive	Kemps and Timmermans 1982; Timmermans and Vossen 1996
	Mona monkey (*Cercopithecus mona*)	Captive	Takeshita 1961
	Olive baboon (*Papio anubis*)	Feral	Nash 1974

(*Continued*)

TABLE 1 (Continued)

Family	Species	Captive/Wild	References
	Patas monkeys (*Erythrocebus patas*)	Captive	Hemmalin and Loy 1989
	Proboscis monkeys (*Nasalis larvatus*)	Wild	Gorzitze 1996
	Rhesus monkey (*Macacus rhesus*)	Captive	Adachi, Saito, and Tanioka 1982; Brandt and Mitchell 1973; Tinklepaugh and Hartman 1930
	Stumptail macaques (*Maeaea aretoides*)	Captive	Gouzoules 1974
	Toque macaque (*Macaca sinica*)	Wild	Ratnayeke and Dittus 1989
	Yellow baboon (*Papio cynocephalus*)	Wild	Condit and Smith 1994
Atelidae	Black and gold howler monkeys (*Alouatta caraya*)	Wild	Peker et al. 2009
	Howler Monkeys (*Alouatta seniculu*)	Free-ranging	Sekulic 1982
	Howler monkeys (*Alouatta seniculus*)	Free-ranging	Sekulic 1982
	Mantled howling monkey (*Alouatta palliata*)	Wild	Moreno, Salas, and Glander 1991
	Mexican mantled howler monkeys (*Alouatta palliata*)	Semi-free-ranging	Dias 2005
	Red-handed howler monkey (*Alouatta belzebul*)	Free-ranging	Camargo and Ferrari 2007
Hyolbatidae	Gibbon (*Hyolbates*)	Captive	Hooton 1946
	Muller's Bornean gibbon (*Hylobates lar mulleri*)	Captive	in Hayssen et al. 1993
	Pileated gobbon (*Hylobates lar pileatus*)		in Hayssen et al. 1993
Pongidae	Bonobo (*Pan paniscus*)	Captive	Bloser and Savage-Rumbaugh 1989; in Hayssen et al. 1993
	Chimpanzee (*Pan troglodytes schweinfurthii*)	Free-ranging	Goodall and Athumani 1980: in Hayssen et al. 1993
	Lowland gorilla (*Gorilla gorilla gorilla*)	Captive	Beck 1984; in Hayssen et al. 1993
	Mountain gorilla (*Gorilla gorilla beringei*)	Wild	Stewart 1977; Stewart 1984
	Orangutan (*Pongo poygmaeus*)	Captive	in Hayssen et al. 1993

never been recorded among postpartum human female mothers in pre-industrial and natural fertility conditions (Kristal 1980; Soyková-Pachnerová et al. 1954; Young and Benyshek 2010).

The presence of placentophagy in nearly all present-day mammalian species could be the outcome of convergent evolution, however, it is more parsimonious to consider placentophagy as a pleisomorphic mammalian trait, which was lost in the few extant species that do not routinely engage in the behavior. In light of our shared ancestry with apes and nonhuman primates among whom maternal placenta consumption is widespread, it is likely that placentophagy is an ancestral mammalian behavior that was present among human ancestors and was selected against, and subsequently lost, at some point in hominin evolutionary history, sometime after the bifurcation of hominins from the pongid branch. Exactly why, when, and through what process our hominin ancestors ceased engaging in placentophagy is currently unknown. Our investigative model proposes a hypothesis regarding why and how the behavior may have been lost in our species or our direct human ancestors.

Given the ubiquity of placentophagy in the nonhuman mammalian world and its probable adaptive value, placentophagy's conspicuous absence in humans is puzzling. The most comprehensive cross-cultural study to date—surveying up to 300 societies around the world—found no evidence of maternal placentophagy in pre-industrial human societies (Kristal 1980; Trevathan 1987; Young and Benyshek 2010). Beginning in the 1970s, however, various forms of placentophagy have been recorded among a small number of U.S. and Mexican women who promote the practice based on its purported therapeutic benefits (Selander 2009; Janszen 1980; Field 1984; Bastien 2004). This suite of contemporary practices, however, has emerged only very recently, and although it may be helpful in understanding the consequences of placentophagy in our species, it does not represent a behavior that persisted throughout our evolutionary past (see Young and Benyshek 2010, for a review).

Previous attempts to account for the absence of placentophagy in humans have primarily relied on culture-based justifications (e.g., taboo, ritualistic practices). Field (1984) suggests that systems of beliefs, specifically ideas about "clean" and "unclean" foods, might be responsible for the absence of human placentophagy. According to this model, the placenta is systematically associated with birth, which is often conceptualized as an "unclean" event. While there are known "universal" prohibitions that reinforce behavioral norms, such as incest taboos between first degree blood relatives (Brown 1991) and an avoidance of close contact with spoiled and decaying biological matter (Rozin and Fallon 1987), such rare universal prohibitions are restricted to behaviors with demonstrated fitness consequences (e.g., controlling the propagation of deleterious recessive traits, and inhibiting disease transmission). In the vast diversity of the world's cultures, we

do find many examples of cultural beliefs and practices that represent 'exceptions' to common (rather than universal) cultural taboos, such as longstanding and well documented cultural traditions of cannibalism (Harris 1998). The strict lack of a single exception in the cross cultural record would seem to set placentophagy apart from those *exceptional* behaviors that are strongly informed by cultural conventions (e.g., cannibalism), and place it more in line with behaviors that appear to have significant effects on an organism's fitness (e.g., incest).

Other explanations for the behavior's absence argue for the apparent lack of direct benefit that humans would obtain from eating placental tissue. Some authors reason that unlike other animals, human mothers are more likely to have been adequately nourished at parturition, and would no longer need to consume the placenta for its additional nutritional benefits (Friess 2007).

We would argue that placentophagy's disappearance in every human population against a background of mammalian and primate ubiquity suggests the presence of powerful selective pressures that eliminated the behavior in the ancestral human lineage. One possible explanation for the lack of human maternal placentophagy could derive from the deleterious health consequences associated with a universal practice unique to humans—the habitual use of controlled fire.

EXPOSURE TO OPEN HEARTH FIRES?

To explain the human departure from the near-universal mammalian norm of postpartum maternal placentophagy, we suggest here that something in our species' evolutionary past likely changed our interaction with the environment and altered the cost-benefit ratio of human maternal placentophagy. What might this monumental shift in our species' evolutionary past have been? We suggest one of the great leaps in human evolution: the controlled use of fire, especially for transforming foodstuffs (i.e., cooking).

Beyond its invaluable contributions to providing warmth, light, and protection from predators, the controlled use of fire allowed ancestral humans to successfully unlock vast amounts of energy (i.e., calories) that were otherwise inaccessible to earlier hominins and to members of other animal species, thereby extending the reach of ancestral humans' foraging capabilities. Precisely when hominins first began using controlled fire is a matter of current debate. Some leading researchers argue that ancestral humans' controlled use of fire began as early as 1.9 million years ago, based on archaeological sites in Africa (Wrangham 2009), with the earliest firm evidence of its use at 790,000 year B.P. in Israel (Goren-Inbar et al. 2004). Strong evidence of controlled fire use in Europe dates to 400,000 years B.P. at sites in England and Germany (Roebroeks and Villa 2011). Boiling,

heat-softening, roasting, and carbonizing are all techniques mastered cross-culturally that have successfully extended the scope of the human nutritional resource bank. By transforming foods that would otherwise be poorly absorbed and utilized by the body, cooking has allowed humans to benefit from higher energy- and nutrient-dense foods, and has increased the breadth of available resources that can be consumed by detoxifying some, and ridding others of dangerous pathogens. Indeed, so essential is cooking to human evolution and nutrition that Wrangham and colleagues have argued for its recognition as a distinct human "biological trait" (Wrangham and Conklin-Brittain 2003). Partially as a result of the access to greater and more varied dietary resources that cooking facilitated, ancestral humans were able to develop especially large brains and to enjoy the advantages their increased intellectual capacity provided (Wrangham 2009).

Cooking has been, and remains, an essential human trait; all past and extant modern human cultures have relied to varying degrees on cooked foods (Harris 1992; Wrangham 2009). Yet the controlled use of fire and cooking are not gained without costs. Some of these costs are associated with negative transformations of foodstuffs themselves, and include the heat-sensitive destruction of some nutrients (especially vitamins), and the fact that cooking does create some long-term toxic compounds in foods. From an evolutionary perspective, however, the benefits of cooking—especially in terms of available energy—far outweigh its relatively modest costs (Wrangham 2009). But the costs of cooking may extend beyond the direct effects on food. While cooking with fire substantially increases the availability of energy and can eliminate many pathogens and toxins found in raw foods, open fires also increase exposure to specific environmental metals and other toxic substances through smoke inhalation.

Research has shown that woodsmoke contains a suite of harmful substances including noxious gases (e.g., carbon monoxide), toxic chemicals (e.g., formaldehyde), and heavy metals (e.g., mercury; Larson and Koenig 1994). A large body of research points to an array of deleterious health effects associated with smoke inhalation where domestic cook fires are the primary source of woodsmoke exposure. These include increased risks of chronic pulmonary obstructive disease, asthma, cardiovascular disease, respiratory infections, and tuberculosis (see Torres-Duque et al. 2008, for review). While the majority of this research focuses on indoor woodsmoke exposure (*with and without* smoke ventilation) in developing countries, several studies have identified woodsmoke health risks due to outdoor occupational exposure (e.g., forest firefighters), and the effects of residential wood burning in developed countries (see Naeher et al. 2007, for a review). As with cooking, the costs of chronic woodsmoke exposure are now clear. But from an evolutionary perspective, just as with cooking, the enormous benefits derived from the controlled use of fire for immediate human survival and reproduction far outweigh its clear and significant

long-term health costs. The health risks associated with woodsmoke exposure, however, may have been particularly pronounced among women over the course of evolutionary history.

In the human evolutionary past, given what we know about gender specialization in the EEA (Bowlby 1960), hominin females were probably exposed to greater concentrations of smoke and ash from open fires, and on more occasions than males, as they are still in the rural developing world today (Ezzati 2001; von Schirnding 2001) where domestic wood fires are common (Torres-Duque et al. 2008). Indeed, a large number of environmental health studies have shown that morbidity and mortality associated with biomass smoke exposure is highest among women, newborns and young children (Po, Fitzgerald, and Carlesten 2011; Pope et al. 2010). Such differential exposure for women—and its concomitant deleterious health consequences—is due to a common, cross-cultural sexual division of labor that makes cooking, fire-tending, and other domestic tasks primarily women's work in developing countries (Wickeramsinghe 2001)—a pattern that, based on observations of contemporary hunter-gatherers (Marlowe 2010; Lee and Daly 1999) probably has had a long evolutionary history. Throughout human evolution then, ancestral human females would likely have been subjected to the greatest direct exposure to the smoke and ash of fire on a regular and daily basis.

Exposure to controlled fire-released toxicants for a gravid female would have had a far greater health impact than that for other categories of individuals. Behavioral and environmental risks extend not only to pregnant women themselves but to the fetus that they are carrying (Fessler, Eng, and Navarette 2005; Flaxman and Sherman 2000; Messinger and Lester 2008). The placenta plays the central role in the dynamic exchange between the physical environment, the mother and the fetus. A primary function of the placenta is to facilitate the exchange of nutrients and gases between mother and fetus. Another function of the organ is to act as a barrier to prevent the transfer of harmful substances (teratogenic and others) to the developing fetus. The placenta does not provide complete protection from toxicants, pathogens and other contaminants, however. Some developmentally harmful substances pass through the placental barrier rather easily (e.g., ethanol), while others are near-completely filtered out and remain locked in placental tissues until they are eventually eliminated from the body after placental "delivery." Thus, as a consequence of its functioning, harmful substances can accumulate in placental tissue (Myllynen, Pasanen, and Pelkonen 2005). When environmental exposure to toxic substances is low, accumulation is similarly low. But as environmental exposure increases, so too does the bioaccumulation of deleterious substances in the afterbirth. An implication of one of the great revolutions in human history—the controlled use of fire—could be that once fire use by humans became ubiquitous in our evolutionary past, placentophagy may have substantially increased

maternal exposure to specific bioaccumulated environmental metals and other harmful substances, and could have significantly reduced the reproductive fitness of the postpartum females who engaged in the behavior. As a result, we hypothesize that natural selection may have eliminated maternal placentophagy among humans or our direct human ancestors.

Previous research has shown that metals, which are released into the environment through natural processes, accumulate in vegetation during growth and can become concentrated in plants over time (Greger 2004). Although we have no direct measurements of heavy metal concentrations in prehistoric wood samples, measurements taken from ice core samples document fluctuating atmospheric circulation of natural lead, copper, zinc and cadmium as far back as 155,000 years ago, indicating that these metals were present in the environment and available for uptake by plants (Boutron et al. 1993; Hong et al. 1996). Using evidence that predates glacial records we can infer that heavy metals such as cadmium were largely available in the EEA of archaic hominin species in Africa throughout both the Pliocene, and the Pleistocene. A primary natural source of cadmium deposition in the earth's crust is through volcanic activity (Faroon et al. 2008). The Rift Valley and more generally the whole of East Africa show evidence of tremendous volcanic activity at a time when archaic humans were evolving into more modern hominin species, spanning the era during which the controlled use of fire would have emerged in our ancestors (Baker et al. 1971; Chorowicz 2005; Dawson 1992; Logatchev, Beloussov, and Milanovsky 1972).

Burning vegetation that has accumulated toxic metals releases these substances into the environment through smoke and ash (Faroon et al. 2008; Stefanidou, Athanaselis, and Spiliopoulu 2008), which would be inhaled and ingested with close contact to the fire source. As an example, a study investigating copper exposure in a modern Bedouin population found an increased concentration of the heavy metal near their hearths, an increase that was partly attributed to the release of metals during combustion of vegetation used as fuel for the cooking fire (Grattan, Huxley, and Pyatt 2003). Worldwide, the most commonly used biomass fuel for domestic cooking and heating fires is wood (Torres-Duque et al. 2008). In their review of hard and soft woodsmoke emissions, Larson and Koenig (1994) identify over 70 chemical constituents of woodsmoke, including cadmium, magnesium, and iron, and fifteen other metals. Unlike many other metals, cadmium, inorganic mercury, and trivalent chromium are trapped by the placental barrier in pregnant mothers and accumulate across pregnancy (Clarkson, Nordberg, and Sagar 1985; Iyengar and Rapp 2001). Precisely which of these or other environmental metals or harmful substances, alone or in combination, may have significantly impacted the reproductive fitness of human mothers that consumed their placentas in our evolutionary past awaits further research. At present, there is any number of fire-released environmental metals/toxic substances that could have had such effects (see Clarkson et al. 1985). For illustrative

purposes, however, we can consider how one of these environmental metals—cadmium—might reduce reproductive fitness among human mothers exposed to high doses of the metal, through several distinct pathways.

Cadmium is a naturally occurring toxic metal that is most readily absorbed through inhalation and ingestion, and once it has entered the body, has the capacity to damage a number of tissues and organs (Faroon et al. 2008). An acute oral exposure to cadmium can irritate the gastrointestinal tract, causing nausea, vomiting, diarrhea, abdominal pain, and cramping, and can also have more serious effects. Experimental animal research, in addition to research with human cell cultures and case studies of exposed populations, indicates that cadmium accumulates in the liver and kidneys, and also in the reproductive organs, including the ovaries (Abadin, Hibbs, and Pohl 1997; Henson and Chedrese 2004; Iyengar and Rapp 2001; Shiverick and Salafia 1999). The kidneys are the primary organ targeted by cadmium, and exposure through both chronic inhalation and acute ingestion can lead to accumulation in the kidneys and renal damage or failure (Elinder 1992; Faroon et al. 2008). Although there is no direct human evidence for liver damage resulting from chronic inhalation or acute oral exposure to cadmium, in experimental rodent models, acute single doses given orally were related to hepatic necrosis (Andersen, Nielsen, and Svendsen 1988; Faroon et al. 2008).

Several studies provide evidence that cadmium functions as an endocrine disruptor and that even low dose exposure impacts the production and function of reproductive hormones (Henson and Chedrese 2004). Research examining the effects of low and high doses of cadmium in ovarian cells from pigs suggests that low amounts of the metal seem to increase progesterone production, while a high dose may inhibit production of the hormone, which is necessary to maintain pregnancy (Smida et al. 2004). Rodent studies examining the estrogenic effects of cadmium on uterine and mammary tissue indicate that the metal functions as an endocrine disruptor for estrogen, enhancing estrogen-like activity in nonpregnant, ovariectomized rats (Johnson et al. 2003). The results of another study in which acute doses of cadmium were administered to normally cycling rats found a relationship between exposure and decreased estrogen production (Piasek and Laskey 1994). Both of these effects—the upregulated and decreased estrogenic activity—impair reproductive ability by altering endocrine and ovarian function.

Voluntary modern cigarette smoking provides us with a contemporary analogue for the biological mechanism by which involuntary ancient woodsmoke inhalation may have impacted placental accumulation of toxins and maternal health. While admittedly not a perfect comparison, environmental health studies have shown that domestic biomass fire smoke and tobacco smoke share many of the same toxic pollutants. Studies have also established that biomass fire smoke, and both active and passive tobacco

smokes, have similar effects on the health of neonates and infants of mothers exposed to both types of smoke (Tielsch et al. 2009; Mishra, Retherford, and Smith 2005). Given the current lack of detailed data regarding the effects of woodsmoke inhalation on placental accumulation of heavy metals, inhalation of smoke from tobacco is the most useful comparison available to demonstrate these effects through the action of similar mechanisms in the body. Because plants uptake metals from the soil during growth, tobacco leaves contain cadmium which is inhaled through cigarette smoke, and analysis of the placental tissue of mothers who smoke compared to that of mothers who do not reveals elevated levels of cadmium in the placentas of smoking mothers, indicating that inhalation of cigarette smoke increases the concentration of cadmium in the placentas of these women (Piasek et al. 2001; Shiverick and Salafia 1999). Additionally, the levels of metallothionein, a protein in the body that binds to cadmium and other metals and whose production is induced by exposure to these metals, is higher in the placentas of mothers who smoke than in non-smoking mothers, and has been suggested as a mechanism of protection for the developing fetus against exposure to the toxic metal (Ronco et al. 2005). This suggests that exposure to cadmium released through smoke increases the amount of the metal captured by the placenta through increased production of metallothionein. Given what we know about cadmium exposure in experimental contexts, if mothers in the human EEA were systematically exposed to fire smoke during pregnancy, the chronic inhalation of smoke containing low doses of toxicants could have led to the accumulation in mothers' placentas of amounts of cadmium that were well above levels that would be safe to ingest in a single, acute dose if those placentas were eaten postpartum. Although the U.S. DHHS Agency for Toxic Substance and Disease Registry *Toxicological Report for Cadmium* (Faroon et al. 2008) does not include a recommendation for an acute oral dose of cadmium that poses minimal risk to humans, there are sufficient data to determine intermediate and chronic oral doses—0.5 μg Cd/kg/day (exposure between 15 and 364 days) and 0.1 μg Cd/kg/day (exposure over 365 days) respectively.

Since cadmium has a long half-life of about 20 years (Telisman, Azaric, and Prpic-Majic 1986), multiple births in which the placenta is eaten, in addition to accumulation from chronic smoke exposure, would cause increasingly higher accumulations of the metal before the mother's body would have been able to excrete significant amounts of the toxicant. Additionally, research analyzing placental cadmium content in women who smoke shows that increased age and parity are related to higher concentrations of cadmium in the placentas of smokers (Kuhnert, Kuhnert, and Zarlingo 1988), suggesting that mothers in the EEA who were exposed to higher levels of smoke over time and across multiple births may have been at higher risk of environmental metal accumulation in their placentas. Under such circumstances, consuming this now heavy-metal-laden organ could

then have caused damage to the mother's health, and the health of her offspring through breastfeeding, since breast milk contains approximately 10% of maternal blood levels of cadmium (Radisch, Luck, and Nau 1987), and eventually threaten the long-term reproductive fitness of mother, offspring, or both. Although the act of placentophagy itself is short lived, if the organ does indeed contain a high concentration of toxicants, the consumptive behavior, and the concomitant ingestion of an acute dose of these toxicants, could have longstanding health consequences. In addition to directly impacting mother's and offspring overall fitness, because placentophagy (and the resulting ingestion of an acute dose of toxicants) occurs during a time in the life cycle that is closely tied to reproductive success (the minutes and hours shortly after parturition), negative outcomes associated with the behavior would compound the fitness consequences of the behavior.

Thus, the acute exposure to cadmium (or other fire-released environmental metal[s]) and its harmful side effects, via maternal placentophagy could have a fitness-reducing effect for mothers and offspring through at least three distinct pathways: (1) by reducing the mother's health shortly after ingestion of the placenta and impairing her survival or ability to care for her infant, (2) by reducing the mother's long term reproductive health, and (3) by negatively impacting the health and reproductive success, or both, of developing offspring through exposure to dangerous levels of environmental metals via breast milk.

Although the known effects of cadmium (among other environmental metals) and its ability to accumulate in placental tissue across pregnancy makes it a good candidate to explain the disappearance of placentophagy in humans, we would reiterate that it is also possible that other environmental metals or harmful substances released in the smoke and ash of open hearth fires, or interactions between multiple substances, might be responsible for the absence of placentophagy in pre-industrial and natural fertility populations. All potentially toxic substances that accumulate in the placenta should therefore be investigated.

ALTERNATIVE HYPOTHESES

The lack of human maternal placentophagy may alternatively be explained using a different approach, and if this is the case, other hypotheses should be explored. One possible avenue for exploration is that the avoidance of placentophagy could be a response to visual or olfactory cues given by the organ, a signal of potentially pathogen-laden foodstuffs (Rozin and Fallon 1987). Ideas about cleanliness and the proper treatment of the placenta as a product of the vulnerable period of childbirth as reasons for the disappearance of placentophagy should also be investigated (Young and Benyshek 2010).

Alternatively, genetic drift might explain the disappearance of placentophagy in the human lineage. If, at some point in our evolutionary past, maternal placentophagy was a neutral behavioral trait—neither enhancing nor reducing reproductive fitness—and was practiced by some females but not all, as we see in other nonhuman primates, the behavior could have eventually disappeared due to drift (e.g., a population bottleneck). Hypothetically, for the genetic basis of the behavior to be lost for the entire species in such a way, genetic drift should have occurred in a sufficiently small and early human ancestral population. Drift occurring only in select human populations (rather than a species-wide founding population) could reduce the overall frequency of the genetic basis for the behavior, but had the trait been neutral, the behavior would have persisted in some populations, although this does not appear to be the case.

Finally, alternative explanations for the apparent absence of human placentophagy may be obtained by investigating the absence of the behavior in other mammals. Given that camelids are the only other terrestrial mammals among whom placentophagy is not commonly observed, the development of evolutionary models that might provide testable hypotheses regarding its similar absence may provide more insights into its absence among humans.

CONCLUSIONS AND TESTING OF THE MODEL

The model outlined here presents just one of many possible reasons why placentophagy might have disappeared from our species' behavioral repertoire, or that of our direct human ancestors, over the course of hominin evolution. In order to understand whether placental accumulation of toxic metals could have been responsible for this shift, rigorous scientific studies must be performed. Such research should include the analysis of heavy metals released in woodsmoke among foraging groups living in geographical regions that best represent the EEA for the emergence of hominins' controlled use of fire. Additional analysis of the accumulation of toxic substances in the placentas of contemporary mothers in these environments who are regularly exposed to fire smoke would give us a basis for understanding whether harmful substances released by smoke could have reached concentrations that could lead to detrimental fitness outcomes if ingested.

Finally, we know that the amount of environmental metals and other deleterious substances released through biomass combustion is proportional to the amount taken up by the plant during growth and the rate of toxicant biomass fixation varies by species. Identifying the specific wood fuel sources that were used in ancient environments and determining the heavy metal concentration and combustion emission profile of that fuel sources are also important lines of investigation.

Research aimed at a better understanding of the absence of human postpartum maternal placentophagy from an evolutionary perspective is important for several reasons. First, it promises to provide for a more complete understanding of human evolution, both in terms of our shared mammalian and primate evolutionary history, and those features of the hominin lineage that are distinctive. Secondly, recent research informed by life history theory has highlighted the importance of maternal health and nutrition during pregnancy and lactation for a host of offspring health and reproductive outcomes, and their implications for evolutionary processes (Gluckman and Hanson 2004; Kuzawa and Quinn 2009). Investigating the absence of human maternal placentophagy in the context of the Developmental Origins of Health and Disease may provide important new insights into the behavior, both in terms of its evolutionary history among hominins, and potential health considerations for the small but growing number of contemporary women in the developed world that have recently begun to engage in the practice.

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