Placentophagia: A Biobehavioral Enigma

(or *De gustibus non disputandum est)*[1]

MARK B. KRISTAL

Department of Psychology, State University of New York at Buffalo, Amherst, NY 14226

KRISTAL, M. B. *Placentophagia: A biohehavioral enigma (or* De gustibus non disputandum est). NEUROSCI. BIOBEHAV. REV. **4**(2) 141-150, 1980. -- Although ingestion of the afterbirth during delivery is a reliable component of parturitional behavior of mothers in most mammalian species, we know almost nothing of the direct causes or consequences of the act. Traditional explanations of placentophagia, such as general or specific hunger, are discussed and evaluated in light of recent experimental results. Next, research is reviewed which has attempted to distinguish between placentophagia as a maternal behavior and placentophagia as an ingestive behavior. Finally, consequences of the behavior, which may also be viewed as ultimate causes in an evolutionary sense, are considered, such as the possibility of beneficial effects on maternal behavior or reproductive competence, on protection against predators, and on immunological protection afforded either the mother or the young.

Placenta   Placentophagia   Afterbirth   Parturition
Mammals   Immunology   Adaptive advantage   Pregnancy
Fetal membranes   Maternal behavior   Ingestion   Amnion

DURING parturition, mothers of almost all mammalian species expel not only a fetus, but also an associated after birth. The afterbirth consists primarily of a placenta, connected to the fetus by an umbilicus, and also includes amnionic and chorionic

membranes and a large quantity of fluid. During delivery, a striking behavior occurs in most nonhuman mammalian species: *the mother consumes the afterbirth.* Although *this placentophagia* does not seem, on the surface, to be critical to the birth process or to the immediate well-being of the infant, the mother purposefully, laboriously, and usually completely, devours the placenta and fetal membranes. Often she stops attending to the newborn during placentophagia, which may last for an hour or more, and resumes infant-directed behaviors only when the afterbirth has been completely eaten. To date we know almost nothing of the causes or consequences of this behavioral phenomenon.

The placenta is a large, highly complex organ, capable of a multiplicity of synthetic, secretory, filtration, analytic, and transport functions [45,71]. It serves as the interface of the maternal and fetal physiological systems.

Despite similarities in function, characteristics of placenta -- specifically chorio-allantoic placenta -- vary widely among mammals. Morphologically, placentas may be disc-shaped (discoid, as in higher primates and rodents); a band around the amnionic sac (zonary, as in most carnivores); a group of from several to over a hundred islands of tissue (cotyledonary or multiplex, as in most ruminants); or diffuse (diffuse or microcotyledonary, as in some nonruminant ungulates). Placenta types also differ histologically and can be distinguished by the number of tissue layers separating the maternal and fetal blood supplies: epitheliochorial placentas are those in which the maternal and fetal circulations are separated by three maternal and three fetal tissue layers (as in ungulates, aquatic mammals, and some prosimians); endotheliochorial, in which one maternal and three fetal tissue layers separate the systems (as in most carnivores); and haemochorial, in which the maternal tissue layers are entirely absent (as in higher primates, rodents, lagomorphs, and insectivores). Finally, afterbirths can be categorized by the portion (fetal or maternal) of the placenta delivered during parturition: deciduate afterbirths are those in which both the fetal and maternal portions of the placenta are delivered (as in humans and rodents); adeciduate afterbirths are those in which only the fetal portion of the placenta is delivered (as in carnivores and ungulates); and contra-deciduate after- births are those in which both the fetal and maternal portions of the placenta remain in the uterus and are resorbed (as in marsupials) [28,45,71].

During labor, the placenta detaches from the wall of the uterus, and, as the fetus begins to emerge, rapidly loses its significance as a life-support system. Expulsion of the placenta occurs from seconds to hours after the delivery of the neonate, depending on the species. Survival of the neonate requires the freeing of the head and face from fetal membranes and detachment from the placenta by severing of the umbilicus. The latter is usually accomplished by biting, tearing, or stretching the cord, but may occur as a result of movement of the mother or infant, without particular attention being directed to the cord. This is frequently the case in species where the latency between emergence of the neonate and expulsion of the placenta is long.

Until recently, no systematic investigation of the biological basis or significance of placentophagia had been under taken. It was clear in Lehrman's 1961 review of the literature on placentophagia [42] that the references prior to that time primarily

documented which species did, and which did not, eat the afterbirth. With few exceptions, the literature consisted of incidental observations, anecdotal references, and untested speculations about the causes of the phenomenon. Nevertheless, the body of literature on the occurrence of placentophagia is quite comprehensive, and provides evidence of group trends in the occurrence of placentophagia among mammals. With certain notable exceptions, such as some semi-aquatic species (of the Order Pinnipedia) [32] and fully aquatic mammals (Order Cetacea), placentophagia has been observed as a routine behavior among most other eutherian (placental) mammalian orders. It has been well documented in Insectivora, Rodentia, Chiroptera, Lagomorpha, Carnivora, Perissodactyla, Artiodactyla (with the camel as a noted exception), and Primates [9,42,66]. Marsupials, which are an order of metatherian (pouched) mammals, resorb rather than deliver the placenta, and there fore cannot engage in placentophagia; they do, however, vigorously lick birth fluids as they are excreted.

Although placentophagia in nonhuman primates has been noted during normal births in prosimians, old and new world monkeys, and in apes (see [9] for review), the question re mains open of whether human groups practice, or practiced, placentophagia. In an early paper on parturition in the rhesus macaque, Tinklepaugh and Hartman [73] described the act of placentophagia in great detail. In doing so, they made a passing, unreferenced remark about placentophagia in humans:

"After licking the afterbirth, she begins the gruelling task -- one common to most if not all sub-human mammals and probably related to human placentophagia -- of consuming this tough fibrous mass. Holding the organ in her hands, she bites and tears at it with her teeth." ([73] p. 89).
Stimulated by this reference to human placentophagia, I undertook a search of the Human Relations Area files at the State University of New York at Buffalo in 1975. The anthropological subcategories examined for each of the 296 cultural groups catalogued were: childbirth, difficult and unusual births, postnatal care, gratification and control of hunger, cannibalism, and nutrition. The fate of the afterbirth could be determined for 70% of the cultures listed; in not one was placentophagia noted. The majority of cultures burned or buried the placenta. In many, a totemism was associated with the afterbirth, and a piece of the placenta or umbilicus was saved as a talisman. In some cultures (Pomo, Toradja, Siwans, Vietnam), portions of placenta are saved for subsequent medicinal application (see also [54]).

In many cultures, however, strong statements against eating placenta were noted, suggesting that these cultures recognized placenta as a substance that could be eaten. Whether these prohibitions or the symbolic substitutions seen in other cultures were designed to counteract a tendency to eat placenta, or to reinforce the distinction between humans and other animals, is unclear. The Navaho treated the placenta as sacred, but also as poisonous; the Kol believed that if the placenta were eaten by the mother, she would die. The Shilliuk, apparently practicing symbolic ingestion of the afterbirth, buried the placenta at the roots of a fruit tree, then during the next season ritualistically ate the fruit or drank tea brewed from the fruit. It remains possible that placentophagia is practiced in some cultures, or may have been before the era of modern anthropological records, as

Ober has pointed out [54]. Tinklepaugh and Hartman may have been aware of some information that has since faded into obscurity.

TRADITIONAL EXPLANATIONS OF PLACENTOPHAGIA

The sharp distinction between the prevalence of placentophagia in nonhuman eutherian mammals and the near, if not total, absence of placentophagia in human cultures raises some interesting questions which are difficult to answer without information regarding the causes and consequences of placentophagia in nonhuman species.

The immediate problem is to understand the factors contributing to placentophagia in those species that engage in the behavior, and to try to understand the consequences of the act to the mother, the offspring, or the social group. Several hypotheses concerning the mechanism for the initiation of placentophagia have existed in animal lore for a long time. One, designed to explain placentophagia in primarily herbivorous species, is that the mother undergoes *a shift in food preference toward carnivorousness* at the time of parturition. Lehrman [42] characterized the behavior of herbivores at parturition as "voraciously carnivorous". The behavior of the cow at parturition has been specifically de scribed elsewhere as carnivorous [29], as has that of the rhesus macaque [73,74]. Another hypothesis is that mothers consume the afterbirth because of *general hunger,* i.e., that anorexia prior to parturition leads to placentophagia as a means of maintaining homeostatic food intake requirements; it has been noted in the literature that the domestic bitch actively avoids food and water during the last 24 hours of pregnancy [31,43], and that the mare frequently shows anorexia during labor [19]. A third hypothesis is that placentophagia is a response to *specific hunger,* i.e., a response to specific nutritional [56,73] or hormonal [77] needs which can be satisfied by consuming the afterbirth. The needs in question are assumed to be a product of metabolic or endocrine changes associated with late pregnancy and parturition. A fourth hypothesis is that mothers eat the afterbirth *to maintain the cleanliness of the nest site and to avoid attracting predators* [19,66,73,77].

The fourth hypothesis (one which should most appropriately be regarded as pertaining to a consequence and not to a direct cause of placentophagia), that of nest hygiene and protection against predators, while ethologically the most appealing hypothesis, is the least tenable and might be rejected on several grounds. (a) Mothers of relatively unchallenged predatory species eat the afterbirth. (b) Mothers of non-nesting species (e.g., ruminants) eat the afterbirth, and in fact, remain at the birth site long after the neonate is able to walk away, in order to finish consuming the placenta [19,29]. (c) Certain arboreal primates that deliver in trees do not drop the afterbirth to the ground, but rather keep it and spend an hour or two eating it [9,73]. Finally, (d) the olfactory cues emanating from the fluids that have saturated the ground might be expected to be effective predator attractants, and these fluids are apparently not cleaned up during placentophagia. Although no empirical evidence has yet been obtained regarding the adaptive advantage conferred under the nest hygiene/antipredation hypothesis, the counter-examples seem at least as effective a set of arguments as those for the hypothesis.

The remaining three hypotheses, either singly or in combination, are insufficient to provide a comprehensive explanation for placentophagia in nonhuman eutherian mammals. Whereas many placentophagic species do show decreased food intake prior to parturition [19,31,43], the rat does not [14,39]. Placentophagia, however, is observed in nearly 100% of normal rat parturitions [59,75]. To test the hypothesis of a shift to carnivorousness at parturition, rhesus monkeys were presented with liver, beef, and pork, prior to, during, and immediately after delivery [73]. In all cases, the meats were refused. More recently, multiparous parturient rats, made aphagic and adipsic with lateral hypothalamic lesions, were presented with bits of rat liver, ground beef, and donor placenta [35]. The rats ate the placenta, but refused the other meats. These results can be interpreted as strong evidence against a general increase in the motivation to eat meat, per se, around the time of parturition.

Although the concept of 'specific hunger' is more ambiguous than it appears, it presumably pertains to a need, produced by a special physiological state, for a specific ingestible substance. One should not assume, however, that the smell or taste to which the animal is especially attracted during the period of specific hunger is necessarily that of the deficient substance; during specific hunger the now- attractive smell or taste need only be of a substance that usually occurs in nature in conjunction with the deficient substance, which itself may be sensorily undetectable. There fore the problem of separating what the animal needs from what it is attracted to, becomes critical. At any rate, two particular observations provide the strongest evidence against a simple, straightforward specific-hunger interpretation. First, in both rats [38] and mice [37,41], a substantial proportion of virgin females presented with placenta obtained from donors eat it enthusiastically. Second, of the female rats that do not eat placenta as virgins (but do eat it as they deliver it during parturition), most are still unwilling to eat donor placenta offered to them one or two hours prior to parturition (Kristal, Peters and Graber, unpublished observation). These results suggest that a unique physiological state, such as that which exists at parturition, does not exist even shortly beforehand, and that that state which exists at parturition cannot be a prerequisite for placentophagia since the behavior occurs readily in virgins. Although these observations do not address the issue of specifically attractive components of the afterbirth, they do point out the need for a thorough understanding of the response of non-pregnant females, which are not experiencing the unique physiological state.

PLACENTOPHAGIA IN NONPREGNANT MICE AND RATS

Despite the fact that virtually all female mice and rats enthusiastically eat placenta during delivery, the response of nonpregnant mice and rats to donor placenta presented in a dish is clearly dichotomous. Female mice [41] either immediately approach and eat placenta, or back away, tremble, and occasionally tail-rattle, until the material is removed. The reproductive condition at the time of testing and the genetic background of the mice were both found to affect the proportion of placentophages in the test groups. Among virgins, the proportion of placentophages in a group of BALB/cBy mice was about 0.25, whereas in a C57BL/6By group the proportion of placentophages was 0. The same two strains (different mice) were tested under two other reproductive conditions: (a) tested 10 days

after delivery of their first litter, which was removed within hours of parturition, or (b) tested ten days after ten days of nursing their first litter.

We found that the delivery plus brief pup contact elevated the proportion of placentophages in a BALB/cBy group significantly (to about 0.70), but that the proportion among C57BL/6By mice was still extremely low (about 0.06). On the other hand, parturition plus ten days of nursing experience produced only a slightly higher proportion of placentophages among BALB/cBy mice than did parturition alone (about 0.81), whereas the proportion of placentophages in the C57BL/6By group was greatly increased by the addition of nursing experience (to 0.50). Incidentally, neither strain's placentophages showed a preference for either their own strain's or the other strain's donor placenta. (For this and all placentophagia experiments conducted subsequently by us on both mice and rats, donor placentas used for testing were obtained surgically from CO_2-killed donor females on Day 21 of pregnancy. The placentas were then rapidly frozen, along with a few drops of physiological saline, and maintained at -20° C. Immediately prior to use, the placentas were rapidly warmed to about 39° C and presented for 15 min to subjects that had been without food for two hours and without water for 15 min. All placentophagia-testing of nonpregnant subjects was conducted during the third quarter of the lights-on phase of the day/night cycle, to minimize interference from ingestion due to homeostatic feeding. For rats, the tests were conducted over three consecutive days or until placentophagia occurred [38]. Since the behavior is dichotomous, the data acquired in this now-standard testing procedure are nominal: each subject is rated as either a placentophage or a nonplacentophage. The proportions of placentophages in various groups are then compared statistically.)

In the absence of genetic or cross-fostering studies, observations of strain differences only suggest a genetic basis for the phenotypic difference. Therefore, a follow-up genetic analysis of the observed BALB/cBy-C57BL/6By difference in placentophagia was conducted [37], using the Recombinant Inbred Strain technique [3]. By testing virgins of the BALB/cBy and C57BL/6By strains, the reciprocal F1 female offspring, and virgins of seven inbred recombinant strains derived from BALB/cBy and C57BL/6By progenitors, and typing each group as showing either a BALB-like or C57-like response to placenta, a pattern of allelic distribution could be discerned. The data indicated that the group's characteristic response could be attributable to the action of two genes, both dominant at the C57 allele. When both C57 alleles were present, therefore, the response of the virgin would be most likely to be aversion. Considering the observation that many of the nonplacentophagic virgins emitted tailrattling and backed away from the placenta, we postulated that the mode of action of the two-gene combination (when dominant) is to prevent or inhibit placentophagia by promoting competing avoidance responses (e.g., neophobia). Subsequently, at the first parturition, the factors promoting placentophagia over ride the avoidance and -- at least for many of the animals -- remove placenta from the conceptual category of novel/aversive substances.

Although rats do not tailrattle when agitated, as do mice, the behavioral distinction between eaters and noneaters when presented with donor placenta is still dramatic [38]. Commonly, placentophagic rats begin to eat within the first minute or two of their first

exposure to the dish of placenta. The nonplacentophages frequently withdraw to a far corner of the cage and huddle relatively motionless, turned away from the dish, until the material is removed from the cage. On the second presentation, many of the nonplacentophages become extremely active and often attempt to leap out of the cage as the dish of donor placenta is being put in. We also found that, as in mice, the proportion of placentophages among nonpregnant primiparae is greater than that among virgins, indicating that the attitude toward placenta of some virgin nonplacentophages is reversed by a subsequent delivery.

In virgin rats, the proportion of placentophages in groups of Long-Evans females purchased from the Charles River Breeding Laboratories is usually about 0.45. The proportion among groups of Long-Evans virgins born and raised in our laboratory (daughters of purchased parents) varies from 0.04 to about 0.20 [38]. The reasons for the difference between the purchased and home-grown virgins are unknown, al though we have begun to consider the possibility that stress, such as that of being crated and shipped, produced an altered response to novel situations and makes virgins more likely to eat placenta. More recent findings in our laboratory (Kristal *et al.,* unpublished observations) have provided some corroboration for this hypothesis. Confirmed nonplacentophages (as determined in the three-day test) do not eat donor placenta presented in a fourth test that occurs four to six weeks later, providing the nonplacentophage has led a relatively uneventful life during the interval. But if any one of a variety of stressful experiences occurs during the interval (e.g., a long series of injections, surgery, pregnancy, an automobile ride), *25%* of these nonplacentophages then eat placenta on the fourth test. In short, the proportion of placentophages among stressed home-grown Long-Evans virgins is quite similar to that of purchased virgins. Despite the initial difference in proportion of placentophages in groups of purchased and home-grown virgins, there were no group differences when tested as nonpregnant primiparae (about 60% placentophages in both groups).

An alternative explanation of lower incidences of placentophagia among home-grown virgins was that inadvertent genetic selection for nonplacentophagia had occurred in our rat laboratory. This was checked by means of a simple selection experiment [38]. Known placentophagic and nonplacentophagic purchased virgins were bred to the same few males: the female offspring of these matings were then tested when sexually mature. No correlation was found between the classification of the mother in the placentophagia test and the proportion of placentophages among her daughters.

We examined the influence of estrous cycle stage on the virgin's response to donor placenta [38]. The only effect found was in relation to the stage of the cycle in which placentophages were least likely to eat placenta for the *first* time. Placentophages were equally likely to try placenta for the first time in estrus, diestrus, and late proestrus, but would not eat placenta for the first time during proestrus. If, however, the rat had eaten placenta first in a stage other than proestrus, she would subsequently readily eat placenta during proestrus. Therefore, proestrus was not associated with a general suppression of placentophagia, but rather with an unwillingness to ingest this particular substance when it was novel.

We have attempted to correlate the dichotomous response of virgin rats toward placenta with differences along other behavior dimensions. One major but perhaps not obvious advantage of finding such a correlated difference is that we would then have a prediction of the response toward placenta without having to expose the rat to placenta. Among the tests we have conducted were open field activity, rapidity in acquiring a conditioned taste aversion, ease of induction of maternal behavior by continuous exposure to pups, response toward palatable food after taste adulteration with quinine, and the relationship between estrogen dose and sexual receptivity (lordosis). Disappointingly, placentophages and nonplacentophages do not appear to differ on any of these tests.

THE EFFECT OF PREGNANCY AND OF SPECIFIC HORMONES ON PLACENTOPHAGIA

The striking fact that even those rats that avoid placenta as virgins become enthusiastic placenta-eaters during their first delivery suggested to us that endocrine or other physiological changes during pregnancy may shift the attitude toward placenta in much the same way that they facilitate the rapid onset of pup-oriented maternal behaviors (retrieval of young to the nest, pup-licking, crouching over pups) in the immediate-prepartum period ([60,68] also see [61] for re view). Peters, Steuer, Whitney, Nishita and I have been conducting a series of studies to determine when during pregnancy, and how abruptly, the response of the noneaters is reversed; the effect of the termination of pregnancy on the reversal of attitude; and incidentally, the effect of the duration of pregnancy and of pregnancy termination on the onset of maternal responsiveness in our Long-Evans rats. To determine the incidence of placentophagia during pregnancy among females that had been nonplacentophagic virgins, we gave such noneaters one standard placentophagia test on a particular day of pregnancy. Each female was tested only once. However, it should be noted that this single test constitutes the fourth placentophagia test which occurs four to six weeks after the 3-day screening series described on the pre ceding page. Since a major -- potentially stressful -- event, mating and the onset of pregnancy, occurred in the interval between the third and fourth placentophagia test, we would expect, as mentioned earlier, that the baseline proportion of placentophages in each pregnancy-day group would be about 0.25. After having tested at least 12 and as many as 24 rats per day on Days 7, 9,11, and 13 through 22 of pregnancy, it appears the incidence of placentophagia is markedly higher than baseline on Days 13, 19, and 22, but on none of those days is the proportion of the group in excess of 0.50. This pattern does not correlate with serum hormone levels over pregnancy of any of the major reproductive hormones (see [67] for review). In a separate test, resolute nonplacentophages, those that still refused placenta after a delayed fourth placentophagia test, were tested for their response toward placenta during the last day of pregnancy. As late as three to five hours before delivery, 85-90% of the group still refused even to approach placenta. Needless to say, all were enthusiastic eaters of both their own and of donor placenta at delivery. It appears, therefore, that the sudden and dramatic attractiveness of placenta to rats that previously found it aversive, must occur only in the final moments before delivery, or as the placenta emerges during delivery. This raises the possibility that the termination (rather than duration) of pregnancy, and the endocrine changes produced thereby, may be effective in inducing at traction to placenta, just as it influences the onset of positive

responses toward the pups themselves [58,60]. Our pilot work has indicated that groups of nonplacentophages with pregnancies terminated by Caesarean section on Day 21 show very high incidences of placentophagia, whereas those with pregnancies terminated on Day 19 do not.

It is not yet clear to what extent the lateness or absence of onset of placentophagia in prepartum rats is attributable to delay or absence of the mechanism involved in triggering placentophagia, or, on the other hand a general lack of maternal responsiveness on the part of Long-Evans rats. The parallel examination of the prepartum onset of maternal responsiveness in our Long-Evans rats has begun to show that rats of this strain exhibit significantly less prepartum maternal behavior than either Sprague-Dawley [60] or Wistar [68] rats.

Although the finding that virgin rats do not eat placenta for the first time in proestrus [38] suggested that estrogen may be involved in placentophagia (according to [76] estrogen levels are high during proestrus), two studies we conducted involving manipulation of estrogen levels have pro vided only slight evidence of estrogen involvement. First, we have found (Kristal and Peters, unpublished observations) that when estradiol benzoate is injected after ovariectomy+hysterectomy in doses large enough to facilitate the onset of maternal responsivenes to foster pups [65] in Long-Evans virgins (300 μ g/kg; no effect on either maternal responsiveness or placentophagia was observed with smaller doses), an inhibition of placentophagia occurs. Ordinarily, if given a 15-min exposure to fresh donor placenta every 12 hours, intact nonplacentophages eventually (10-15 days) overcome their apparent aversion to placenta. The ovariectomized/hysterectomized virgins injected with a maternal behavior-facilitating dose of estrogen, showed a continuing unwillingness to eat donor placenta which lasted well beyond the effect of estrogen on vaginal cornification. Since food intake was unaffected beyond the first couple of days, a suppression of placentophagia due to the anorectic effect of estrogen is highly unlikely.

The second estrogen manipulation involved the application of estradiol benzoate directly to the ventromedial hypothalamus (VMH) through chronically implanted cannulae (Kristal and Steuer, unpublished observations). The subjects were either virgin placentophages or nonplacentophages; all subjects were ovariectomized and hysterectomized. Each of the relatively few virgins tested was tested for sexual receptivity, the latency to exhibit maternal responsiveness induced by constant exposure to pups (maternal sensitization), and placentophagia. Although all the subjects showed heightened sexual receptivity after two days with estrogen implants, no clear-cut effects on placentophagia could be observed at this time. When each female became maternal (usually after the effects of estrogen on sexual receptivity could not longer be detected), she was again tested for placentophagia. All but one rat ate placenta at this time. Nine days after the estrogen implants, each rat was again tested for placentophagia. All ate, indicating that prolonged estrogen exposure in the VMH does not lead to a suppression of placentophagia. More research, particularly involving other brain sites, is needed in order to determine whether, and how, estrogen influences placentophagia.

The possibility that prolactin is involved in placentophagia at parturition, and incidentally, in nonpregnant rats, was investigated by Chapin [13] in my laboratory. She was able to demonstrate complete independence of placentophagia and prolactin levels in regard to both the initiation of placentophagia in nonplacentophages, and the incorporation of placentophagia into the permanent behavior repertoire of the female. The possibility that prolactin is effective, but only in the presence of other factors (hormonal or neural), has not yet been examined.

That resolute nonplacentophages are not attracted to placenta even shortly before delivery, and that they do not respond to any of the specific hormone treatments we have tried, but may respond to pregnancy-termination produced by Caesarean section, has led us to begin to consider another possible mechanism. The neural/mechanical stimulation of the uterus and cervix which always occurs during labor and delivery (and is unavoidable during Caesarean section) may be critical to the initiation of placentophagia and influential in the onset of other maternal behaviors.

The proximal portions of the uterine horns (in bicornuate uteri) and the cervix are innervated by the pelvic nerve, and the distal portions of the horns apparently by a branch of the hypogastric nerve [1,8,12,18,22]. Pelvic nerve function has been implicated in the processes leading up to and affecting delivery, in that pelvic neurectomy prevents the onset of delivery [12,33,70]. The substrate for the parturition block following pelvic neurectomy seems to be a spinal reflex arc involving effectors in uterine tissue [44]. Pelvic nerve afferent activity has also been shown to produce changes in hypothalamic neural activity [4] and is considered to be the afferent arm of the Ferguson reflex, in which oxytocin re lease is elicited by cervico/uterine stimulation [44]. Activity in the periphery of this system might be modifiable by the action of reproductive hormones, such as estrogen, on uterine sensory receptors. This might be directly analogous to the action of estrogen on pudendal nerve receptors [34]. Direct afferent influences on the hypothalamus arising in the uterus and cervix may account for facilitation of the onset of maternal behavior, in pseudopregnant rats, observed after distention of the uterus [21], and may account for the need to remove the uterus in order to facilitate the onset of maternal behavior with estrogen injections in ovariectomized virgin rats [65]. Thus, uterine stimulation, produced in the prepartum period by growth and movement of the near-term fetuses, may be responsible for both the onset of delivery of certain objects (neonates and afterbirths), and the onset of behaviors directed toward these objects (maternal behavior and placentophagia). The advantage is obvious of having one set of events, such as uterine stimulation, trigger two consequent phenomena (delivery and behavioral responsiveness) as a mechanism to insure proper temporal sequencing.

The effect of uterine sensory stimulation on brain involvement in the onset of placentophagia during delivery might be conceptualized as taking one of two forms. The first, already discussed in relation to the onset of other maternal behaviors, might consist of a mechanism that activates a specific hypothalamic neural substrate which initiates placentophagia either simultaneously with, or as part of a combined system for pup-directed maternal behaviors. The second mechanism, which would be anatomically similar but functionally distinct from the first, would be one in which the sensory

stimulation arising from the uterus and birth canal would act as a nonspecific, stress-inducing stimulus. Stress, such as that produced by pressure applied to the tail of the rat, has been found to result in the initiation or disinhibition of low-probability (in relation to context) oral behaviors ranging from gnawing and feeding to coprophagia and pup retrieval (see [2] for review). Preliminary trials in our laboratory have indicated that tail-pinch also causes virgin nonplacentophages to eat placenta, but this observation alone sheds no light on whether an analogous phenomenon, perhaps enhanced by hormonal changes in the immediately prepartum period, is responsible for placentophagia during delivery.

PLACENTOPHAGIA AND HOMEOSTATIC FEEDING

An important aspect of our understanding of the mechanisms involved in the initiation and control of placentophagia concerns the relationship of placentophagia to feeding and drinking. Although we have been discussing placentophagia as a maternal behavior, it is, after all, also an ingestive behavior. Relatively little is known of the neural control of maternal behavior in the mammal; it is fortunate that a great deal is known about the neural control of feeding and drinking. Lateral hypothalamic (LH) lesions produced through indwelling electrodes 24 hours prior to parturition eliminated feeding, drinking, and placentophagia in females giving birth for the first time; unlike feeding and drinking, response to placenta of rats with previous parturitional experience, whether pregnant or not during the test, was unaffected by the LH lesions [35].

We recently examined [49] whether (a) the rats in the 1973 study might not have been all placentophages to start with, and (b) whether exposure to donor placenta in a dish would have provided a sufficient experience to prevent the elimination of placentophagia in females with LH damage. We found that inadvertent selection for placentophages would not have changed the results of the Kristal 1973 study, and that unlike the protection afforded by previous parturition, previous experience with a dish of placenta *did not* protect against elimination of placentophagia by LH lesions. It was concluded in the 1973 study that the lateral hypothalamus controls placentophagia up to and including the first parturition, but that after acquisition of parturitional experience, placentophagia is removed from lateral hypothalamic influence, and the possibility was raised that the previous diencephalic behavior becomes corticalized [72]. Several previous studies in the literature have also reported an emancipation from primary hormonal and neural control mechanisms of a variety of behaviors as a consequence of experience [10,11,16,17,46,47,48,63,64]. If acquisition and cortical storage of parturitional experience does account for the "emancipation" of various perinatal behaviors from previous controlling influences, it would appear that learning is a critical and rapidly occurring phenomenon during parturition. We have already ruled out the possibility of facilitated general learning during the perinatal period [36], but it remains possible that there is enhancement, based on motivational or sensory changes, of specific learned associations involving stimuli related specifically to the parturitional environment. Regardless of the hypothesized mechanism of emancipation, it seems clear that placentophagia ceases to be controlled by the neural substrate of homeostatic feeding after the acquisition of parturitional experience.

We have used the technique of taste aversion conditioning [20] to examine the extent to which placentophagia in rats is influenced by manipulations that affect other ingestive behaviors. Engwall and Kristal [16] examined the effects of an aversion to placenta induced (a) in nonpregnant nulliparae, on placentophagia and maternal behavior when they subsequently gave birth; (b) in nonpregnant primiparae, on placentophagia and maternal behavior when they subsequently gave birth; and (c) in parturient primiparae, on placentophagia and maternal behavior when they subsequently gave birth again. Taste aversion conditioning eliminated placentophagia during parturition in all three groups. Previous parturitional experience with placenta reduced retention (but not acquisition) of the aversion, and, under certain circumstances, enabled the females to discriminate between frozen thawed/warmed donor placenta and fresh placenta they delivered themselves. The aversion expressed during parturition was not found to produce any obvious impairment of maternal behavior or pup survival; the observation of maternal behavior was superficial, however, and a more detailed analysis of the maternal behavior of placenta-avoiders at delivery is needed. A subsequent test was con ducted of the discrimination made by the female rats on the basis of the fresh-vs-frozen and the own-vs-other dimensions (Carlson and Kristal, unpublished observation). We found that the rats discriminated fresh placenta from that which had been frozen but not their own from that of a donor. Furthermore, the rats generalized the aversion from frozen to fresh placenta, but not vice-versa.

The conditioned rats that avoided placenta during delivery and still manifested apparently normal pup-directed caretaking behavior [16] may have done so because their response toward amnionic fluid was normal. Presently, we are testing, by means of conditioned taste aversion, (a) whether rats can discriminate placenta from amnionic fluid, and (b) whether a conditioned aversion to amnionic fluid or to both amnionic fluid and placenta will affect maternal behavior at delivery. Preliminary results suggest that rats do discriminate between placenta and amnionic fluid, and that a conditioned aversion to amnionic fluid alone results in placentophagia and relatively normal pup care at delivery.

The palatability, attractiveness, and caloric content of placenta are involved in our consideration of placentophagia as an ingestive behavior. To nonpregnant placentophages, placenta is certainly a highly attractive substance. To parturient rats it is even more so, since during delivery it is much easier to take pups away from the mother than to take placenta away from her. We (Kristal and Nishita, unpublished observations presented at the Eastern Psychological Association Meetings, April 1980) examined the effect of presenting virgin placentophages with unrestricted access either to placenta or to other attractive substances (chocolate chip cookies or a mash of baby cereal, milk, and saccharin) for four hours during the middle of the day. None of the rats was food deprived and all had had all food and water intake, fecal output, and estrous cycle stage monitored for two weeks prior to presentation. The rats presented with placenta ate 40-60 placentas during the four-hour test (approximately 0.57 gm/placenta). The amount of material eaten by the rats presented with placenta was considerably more than that eaten by either of the other two groups, but when intake was converted to caloric intake, the groups were less different owing to the low caloric density of the placenta (0.67 total kcal/gm). However, intake of rat chow during the night following the test indicated that

unlike the rats in the other groups, the rats presented with placenta did not show a compensatory reduction in intake of rat chow. Furthermore, the placenta-tested rats returned to normal day/night baseline eating patterns rapidly, whereas the rats of the other groups did not.

PLACENTOPHAGIA AND MEDIAL PREOPTIC MECHANISMS OF MATERNAL BEHAVIOR

Recently, Noonan and I [50] examined the dissociability of placentophagia from other perinatal behaviors such as pup retrieval, nest building and pup licking, with lesions of the medial preoptic area (MPO) of the hypothalamus. Numan reported elimination of ongoing maternal behavior in lactating rats with lesions of the MPO, and a disturbance in the ability to initiate maternal behavior after MPO lesions in virgin rats [51,52]. The effect of MPO lesions on the ability to *initiate* maternal behavior in *parturient* rats, however had not been examined. We produced lesions through indwelling electrodes 24 hours prepartum in pregnant rats and 24 hours before placenta-presentation in virgins. The lesions had no negative effect on placentophagia in virgins. Of the pregnant rats receiving MPO lesions prepartum, about half manifested a disruption only of nest-building (disturbed nests were not rebuilt) with the rest showing a deficit (in the form of delayed onset) of nest-building, placentophagia, pup-retrieval, and pup-licking. By Day 5 postpartum, only those rats that had manifested the disruption only of nest-building still showed a deficit. This study indicates, as do the studies on the relationship between placentophagia and feeding, that depending on circumstances placentophagia can have characteristics of other pup-oriented (maternal) behaviors or of other ingestive behaviors.

CONSEQUENCES OF PLACENTOPHAGIA

The consequences, if any, of placentophagia on maternal responsiveness and thereby on pup survival are likely subtle, since they are as yet undocumented. The main assumption underlying speculations about the consequences of placentophagia is that the effect would most likely be directly on maternal behavior, or would produce physiological changes in the mother that would be reflected in changes in maternal behavior or reproductive competence (lactation, postpartum estrus, fertilizability, etc.). We have found that removal of placentas manually during delivery is extremely disruptive of the birth process, sometimes causing the rat to delay expulsion of the next pup for hours. The effects on maternal behavior are therefore severely confounded. However, as mentioned above [16], cursory examination of maternal behavior after delivery in which placentas were not eaten because of a previously induced taste aversion did not reveal major deficits in caretaking behaviors directed toward the pups. Furthermore, deprivation of placentophagia by Caesarean section did not eliminate maternal behavior or even produce a severe decrement in it [47]. It should be noted, though, that these tests were conducted on rats at a time when a variety of physiological influences, neural and hormonal, were likely contributing to the rapid onset of maternal responsiveness. When these influences were eliminated by examining the effect of placenta and amnionic fluid on the pups' skin on the induction rate of maternal responsiveness in virgin rats (sensitization rate [57]), a significant facilitation of the onset of maternal behavior was observed [40]. The possibility exists, as Birch hypothesized [7], that the attractiveness of substances on the

skin of the neonates induces immediate contact between the adult and the pups. We have suggested [40] that the effectiveness of that immediate contact in eliciting the full constellation of maternal caretaking responses would then be a function of how closely the physiological state of the adult resembles the optimal condition which is found in the parturient female. If this process does operate during delivery, it might also affect the intensity, as well as the speed of onset, of mother-infant bonding. A strong bond based primarily on olfactory cues might not only raise the intensity of infant-directed caretaking behavior, but also enhance identifiability and localizability of the mother's own infant. Although such enhanced capabilities would confer a powerful advantage in a number of ecological settings, they would likely go unnoticed in the laboratory, where a mother and her infants are kept in close quarters and are usually maintained as an isolated unit until weaning.

There has been documentation of both beneficial and detrimental effects of eating placenta in the lactational period. Foster pups with placentas attached, presented to 10-day lactating rats, that had delivered their own litters normally, had an increased survival rate and higher body weights at weaning, than pups presented without placentas attached; maternal behavior was not directly observed [15]. Zarrow, Grota and Denenberg [78] assumed that the enhanced survival rate and body weight of pups with placentas attached, in the Denenberg, *et al.,* [15] study, was due to the hormones present in the placenta, which was presumably consumed by the foster-mother, such as progesterone, estrogen and prolactin (placental lactogen). The highest survival rate and the greatest weight gain of newborn pups with out placentas were exhibited by pups assigned to 10-day lactating mothers injected with prolactin. Progesterone in creased survival rate but not body weight, and estrogen and prolactin had no greater effect than that of prolactin alone. Furthermore, progesterone reaches a peak concentration in plasma on or about Day 4 postpartum; prevention of placentophagia or removal of the litter at parturition reduced the magnitude of the progesterone peak [27].

Ingestion of placenta during delivery, because of the high estrogen and lactogen content, may affect lactation or milk composition [77] in some species. In a somewhat unrigorous study on humans, reported in 1954, disguised freeze- dried human placenta was fed to postpartum women experiencing some lactational difficulty [69]. Apparently, 86% of the subjects showed improved milk production and flow, whereas among control subjects, fed beef treated identically to the placenta, only 33% showed improvement. Further more, the authors classified more than a third of the placenta-improved subjects, but none of the beef-improved subjects, as having an "extremely strong" positive reaction. The authors also reported that a parallel study was being conducted on cows, and that the preliminary data also appeared to support the positive effect of placentophagia on milk production.

An apparently negative effect of placentophagia, or at least of the presence of placenta and amnionic fluid, on the survival-to-weaning of foster rat pups, has been noted [23,24,25,26]. However, several factors were varied over the series of studies, making it difficult to separate out effects of placenta and amnionic fluid, the duration of lactation prior to testing, and the mode of delivery of the foster pups and the mother's own litter.

Grota suggested [23] that placentophagia may make a difference in litter survival when litters provide less than an optimum level of stimulation to the mother. In a test of this hypothesis [25], he found that the presence of placenta did not affect the acceptance or survival of foster pups that had been delivered normally, but de creased the survival probability of (presumably less stimulating) foster pups that had been delivered surgically. Curiously, a negative effect on survival was also produced by the presence of liver rather than placenta, and could be obtained whether these substances were ingested by the lactating foster mothers, or whether they were injected subcutaneously as homogenates.

The possibility that the hormonal content of placenta might affect characteristics of the postpartum estrus in rats, and thereby have immediate reproductive consequences, has been investigated. Sachs [62] found that relative to rats allowed to engage in placentophagia at delivery, those that were placenta-deprived showed no differences in the latency to onset of the postpartum estrus, or in copulation, fertilization, or implantation during the postpartum estrus.

The absence of obvious effects of placentophagia deprivation on infant survival has tempted researchers to fall back on explanations involving less immediate adaptive value, such as those cited earlier (e.g., nest hygiene), or the assumption that placentophagia is significant only in cases where other conditions for infant survival are marginal [62,77]. An apparently overlooked possibility is that placentophagia directly affects the maternal immune-response sys tem, or indirectly that of the neonate via substances transmitted in the milk [30]. The placenta is a "privileged graft": the fetal placenta (and indeed, the fetus), is immunoincompatible with the mother by virtue of antigens inherited from the father, yet the fetal placenta and fetus are obviously not rejected under normal circumstances [6,30]. The mother's immunological system is, however, permanently altered by gestation, due to the migration of fetal erythrocytes across the placenta or during delivery. In certain cases [6], the first pregnancy may immunize the mother against fetal antigens present in a subsequent pregnancy. These cases of mother- fetus immunological rejection, such as graft-vs-host disease, or, in humans, erythroblastosis fetalis (RH-incompatibility), can result either in death of the fetus or neonate, or in severe physical and mental impairment. The disorders are brought about by the formation of antibodies to the fetal antigens in the antigen-deficient mother. That placenta contains factors which, if ingested during delivery, would prevent the mother from forming the antibodies, becomes an intriguing candidate for the elusive adaptive advantage. We may not have to look far for the active substances; even estrogen and progesterone, found in abundance in the placenta, suppress the immunological processes involved in tissue rejection [6].

In addition to the possible milk-born immunological benefits to the suckling neonate, and the possible protection to the mother from becoming immunized against fetal antigens, there is a third route by which placentophagia may be immunologically beneficial. Rather than prevent the mother from becoming immunized, ingestion of placenta may actually effect an immunization -- that of the mother against placental cells remaining *in utero*

after delivery, and which may, if not rejected, eventually pose a threat to the mother's health (e.g., choriocarcinoma) [5,53].

A methodological problem exists which may have operated against finding more obvious beneficial effects of placentophagia on immunological processes directly affecting the mother which would result in decreased fetal mortality, better pup survival and development, and enhanced sub sequent reproductive efficiency. Most laboratory mammals, particularly rodents, arrive in the laboratory in a fairly high state of inbredness, and if anything, become more inbred if they are then encouraged to reproduce for generations within that laboratory. In this artificial situation, the hypothesized adaptive advantage of placentophagia on maternal immunological protection would be likely to be rendered superfluous.

CONCLUSION

Placentophagia is an unusual and intriguing phenomenon. It has been characterized both as an ingestive behavior, which it obviously is, and as a perinatal maternal behavior, or at least as a behavior of maternal females during the perinatal period. Research so far has indicated that in terms of control mechanisms for the behavior, there are some similarities between placentophagia and feeding, and some similarities between placentophagia and infant-directed caretaking behaviors. Certainly, the intense attraction to the afterbirth manifested by mothers during delivery can not he predicted from the behavior of virgins toward the sub stances, since, at least in rodents, the majority of virgins not only do not eat placenta, but behave as if it were strongly aversive. Therefore, the attractiveness of placenta, per se, cannot simply be the explanation for enthusiastic ingestion of the material when it becomes available (which under nor mal circumstances is only during delivery).

Why most mammalian mothers eat the afterbirth during delivery is still a mystery...in fact, a double mystery. We are not sure either of the immediate causes, although presumably the material becomes very attractive in smell and taste at that time; nor are we sure of the consequences of the behavior. The consequences, of course, if beneficial, must have affected evolution in such a way as to provide for the internal substrate that accounts for the immediate causes, and thereby must be considered the ultimate cause of the behavior. For instance, because of an adaptive advantage conferred on mothers that ate placenta during delivery, or on their offspring, selection pressure would eventually increase the frequency of parturitional placenta-eaters in the population. Thus, the beneficial consequences of ingesting the afterbirth will have produced populations of animals that find placenta extremely palatable. To the individual, the immediate causes of eating the afterbirth during the first delivery must necessarily be that it looks good, tastes good, smells good, or feels good. The animal itself is certainly unaware of adaptive consequences of the act.

I should note in passing that it is reasonable to ask why mothers do not eat the neonate as well as the afterbirth. It may well be, particularly in mammals that deliver altricial young, that the predominant tendency during delivery is to ingest everything that emerges, and

that certain external stimuli prevent the ingestion of the neonates, under normal circumstances, while certain internal stimuli help to produce more appropriate behaviors toward these infants [55]. Why mothers of altricial young don't usually eat the young involves an entirely different set of questions which are vigorously being investigated in several laboratories.

We have proceeded on the assumption that placentophagia has a single, fundamental, biological advantage to all mammals, such as an effect on immunology or on mother-infant bonding. The reason for this assumption is that it is found reliably in vastly different mammalian groups: in herbivores as well as in carnivores and omnivores; in arboreal as well as terrestrial species; in species that deliver in the open, among conspecifics, as well as those that deliver in dens, in solitude. Postulating a basic and common function for placentophagia for all mammals seems more reasonable and parsimonious than hypothesizing that different groups of mammals benefit in different ways from placentophagia. It may well be though, that for some species the important benefit is the replenishment of a nutrient, for some the benefit is that it reduces predation, for others that it lessens the likelihood that the neonate will suffocate because of membranes covering the face, and for still others, that the behavior confers an immunological advantage to the mother or young. Perhaps placentophagia produces all these effects in all species, and the physiology and ecology of a species determines which of the many benefits are most advantageous to that particular group. In the absence of empirical evidence, however, one cannot really choose between the two alternatives: that of a single, basic, common advantage of placentophagia, and that of multiple possible advantages of placentophagia. So, for the present, *de gustibus....*

FOOTNOTE

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