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COMMENTARY

# The Genetic Inactivation of the Vomero-Nasal Organ in Primates Allows the Evolution of Same-Sex Sexual Behavior But Does Not Explain Homosexual Orientation in Humans

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A variety of mammals, including primates, communicate through pheromones, which are volatile chemical signals produced by glands and detected through the vomero-nasal organ (VNO). This manner of communication is effective and fundamental for eliciting innate responses to locate sexual partners and inducing sexual behavior. Pfau, Jordan, and Breedlove (2019) hypothesized that progressive degeneration of a single-gene coding for pheromones receptors in the VNO of mammals may have triggered a cascade of functional and behavioral consequences that facilitated the development of new modes of sexual communication, including same-sex sexual behavior in primates and humans. This hypothesis is compelling, testable, and heuristic.

In their Target Article, Pfau et al. (2019) suggest that, during primate evolution, inactivation of the transient receptor potential cation channel 2 (TRPC2) gene caused a shift from strictly pheromonal-driven sexual behavior toward a more flexible sexual response that allowed for occasional same-sex sexual behavior. In other words, a more flexible sexual response to different and varied stimuli might have allowed the use of sex, including same-sex sexual activity, in both sexes, in contexts beyond reproduction such as dominance displays, reconciliation, and appeasement (de Waal, 1989).

The main evidence in support of Pfau et al.'s (2019) hypothesis comes from knock-out (KO) TRPC2 mice. Pfau et al. found that this experimental strain of KO mice exhibits delay development and altered intraspecific interactions such as sex discrimination and male–male aggression (Leypold et al., 2002; Stowers et al., 2002). Most importantly, compared to wild mice strains, adult male and female KO TRPC2 mice were observed to engage in unprecedented levels of same-sex sexual behavior including mounting and pelvic thrusting.

Comparing KO TRPC2 mice and Old World monkeys, Pfau et al. (2019) furnish evidence that catarrhine primates, which lack a VNO, also have a nonfunctional TRPC2 gene. They argued that like KO TRPC2 mice, catarrhine primates exhibit reduced aggressions and delayed development. Based on these comparisons, they proposed that this TRPC2 single-gene inactivation might explain not only the cause of same-sex sexual behavior in nonhuman primates, but also the cause of sexual orientation in humans. Further, they proposed that the inactivation of the TRPC2 gene promoted a series of behavioral and social transformation that are common in domesticated animals and self-domesticated humans (Hare, Wobber, & Wrangham, 2012).

The primate data do not fit Pfau et al's (2019) hypothesis perfectly, however, and as the authors admit, there are some prosimians (e.g., brown lemurs, *Lemur fulvus*, and sifakas, *Propitecus verreuxi*; Bagemihl, 1999), as well as some New World monkeys (e.g., common marmoset, *Callitrix jacchus*, and Geoffroy's tamarin, *Saguinus geoffroyi*) that have intact TRPC2 genes and show same-sex sexual behavior (e.g., Manson, Perry, & Parish, 1997; Rothe, 1975). Conversely, there are a number of Old World monkeys such as gibbons and olive colobus (*Procolobus verus*) that lack a functional TRPC2 gene, but have never been shown, in field observations, to exhibit same-sex sexual behavior in either sexes. However, these are minor exceptions, and in general Pfau et al.'s hypothesis is supported by the primate data.

Pfau et al.'s (2019) hypothesis could also be tested in other mammals known to have same-sex sexual behavior. For example, dogs (*Canis* spp.) are known to have same-sex sexual behavior both in females (Beach, Rogers, & LeBoeuf, 1968) and in males (Dagg, 1984), and at the same time they

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are macrosmatic with a keen olfaction. Other possible animal models include domesticated cattle and wild bison, since same-sex sexual behavior has been extensively reported for both (Jezierski, Koziorowski, Goszczyński, & Sieradzka, 1989; Lott, 1983). What about their vomero-nasal receptor activity? Contrary to my expectation, dogs and cattle have a markedly degenerated VNO, and most of the genes coding for receptors in the VNO have completely degenerated and are inactive, while their keen olfaction is due only to the primary olfactory epithelium (Young & Trask, 2007). This evidence from domestic mammals is, thus, consistent with the hypothesis that a relation exists between vomero-nasal pheromone receptor activity and same-sex sexual behavior. However, there is evidence suggesting that in cattle the TRPC2 gene is present and active contrary to Old World monkeys (Grus, Shi, Zhang, & Zhang, 2005).

Research on the genes implicated in pheromone detection and the evolution of the VNO of mammals (Moriya-Ito, Hayakawa, Suzuki, Hagino-Yamagishi, & Nikaido, 2018) suggests that there are a multitude of genes classified in two super-families, which are expressed in the VNO, and some of them in the main olfactory epithelium as well. Both the vomero-nasal receptor genes type-1 (V1Rs) with single exon and type-2 (V2Rs) with multiple exons are seven-transmembrane G protein-coupled receptors (Nei, Niimura, & Nozawa, 2008). Both vomero-nasal receptor families have closely related homologs in the vertebrate taste system: V1Rs are closely related to T2R bitter taste receptors (Chandrashekar et al., 2000), and V2Rs are closely related to T1R sweet and umami taste receptors (Hoon et al., 1999).

Grus, Shi, Zhang, and Zhang (2005) found that these genes originated in fishes, are extremely variable among mammals, and evolved through duplication, deletion, and inactivation. Vomero-nasal receptor coding genes belong to the super-family of genes with the highest numerical variability across species. The proportion of intact V1Rs relates to multiple aspects of VNO anatomy, including its relative size (Garrett & Steiper, 2014). The large variation of V1Rs in mammals may be an adaptation to a broad range of environments, and the comparison of V1Rs repertoires is critical for inferring the importance of the VNO to each species (Garrett & Steiper, 2014).

Moriya-ito et al. (2018) reconstructed the phylogeny of V1R genes in primates, almost all code for receptor proteins that are present in the VNO. Moriya-ito et al. suggested that, in general, V1Rs underwent positive selection, grew in number, and are expressed in the VNO of prosimians. This correlates with the socioecology of many prosimians, which are nocturnal, solitary, and communicate extensively with pheromones, including for mating (Dixson, 1995). It has been also found that in Old and New World primates V1Rs show a generalized trend toward degeneration (Grus et al., 2005; Yodler & Larsen, 2014) and this regressive selection happened not only in primates, but also in whales and bats (Grus et al., 2005). Recently, Yoder and Larsen (2014) showed a reduction in the number of intact V1Rs in anthropoids for which the VNO was reduced or vestigial (Smith et al., 2002, 2011), which suggested a correlation between the progressive inactivation of V1Rs and reduction of the VNO. Young and Trask (2007) also found that V2Rs families have completely degenerated in humans, chimpanzees, macaques, cattle, and dogs. Each now possesses 9–20 pseudogenes, but no intact V2Rs.

Rather than hypothesizing a single-gene inactivation as in Pfau et al.'s (2019) Target Article, is it possible that general regression of vomero-nasal functions is causally associated with release from strict sexual response to pheromones and, by extension, an increase in same-sex sexual behavior? It is noteworthy that visual sexual signals, like the genital skin swellings, are very widespread among Old World monkeys and apes, but not in prosimians and New World monkeys (Dixson, 1983). It has been proposed that sexual swelling might visually signal receptivity in widely dispersed social animals like chimpanzee, macaques, and baboons, but could also be interpreted as evidence of the substitution of pheromone signals with visual ones.

Now we come to my main concern with the argument forwarded by Pfau et al. (2019): TRPC2 inactivation seems like just one of the many genes involved in pheromone communication in the VNO, which underwent regression partly in New Word monkeys and completely in Old World monkeys, as in other diurnal mammals. Why, then, suggest a specific gene, TRPC2, as the driver of the whole behavioral transformation? Instead, why not hypothesize that these behavioral transformations resulted from the combined inactivation and degeneration of a variety of genes involved in pheromone detection and in the development and function of the VNO. It could be argued, alternatively to Pfau et al.'s Target Article, that the general regression of these genes allowed the progressive shift from chemical communication toward visual communication, including same-sex sexual behavior as seems to have happen in New and Old World primates, but also in sea mammals, bats, cattle, and dogs (Young & Trask, 2007) .

The only evidence reported in Pfau et al.'s (2019) Target Article against this more general hypothesis is that in Gαi2 KO mice inactivation of this g-protein disrupts aggression, but sexual behavior is not affected (Norlin, Gussing, & Berghard, 2003). Disruption of Gαo, another g-protein crucial for VNO function, also reduces aggression without impacting sexual behavior (Chamero et al., 2011). Norlin et al. (2003) reported unaltered sexual partner preference in their Gαi2 KO mice, but Chamero et al. (2011) do not report on any sexual behavior, so we do not know whether any sexual changes occurred in their Gαo KO mice. Apart from these cases, were KO mice ever produced for all other





Journal : Large 10508 | Article No : 1708 | Pages : 5 | MS Code : 1708 | Dispatch : 15-4-2020

V1Rs and V2Rs that are expressed in the VNO of mammals? What modification in sexuality might such mice show?

Awaiting further evidence, my interpretation, at present, is that circumstantial data best fit the more general hypothesis for a global reduction of gene activity coding in the VNO. We can imagine that, whenever chemical reception and communication receded in favor of the visual communication, selection pressures change. This might have begun when ancestral primates invaded a diurnal niche, increased in social complexity, developed trichromatic vision, and adopted the use body signals (e.g., genital swelling), thereby eliciting the evolution of sexual, rather than chemical, communication signals (Moriya-Ito et al., 2018). Diurnal vision might have enhanced brain size and reduced splanchno-cranial size (Camperio Ciani 1989), thus reinforcing reduction in VNO size. Once sexual behavior was released from the limitation of chemical activation, then sexuality could become much more flexible and could be used by ancestral primates for social communication in a variety of contexts. In extant primates, same-sex sexual behavior might be used to modulate aggression, reconcile conflicts, reinforce dominance rank, and reduce social tension, thereby enriching social complexity. The extreme example is the complex use of sexuality by our closest relative the bonobo (*Pan paniscus*), in which sex is used in the largest variety of social contexts, compared to all other primates (Manson, Perry, & Parish, 1997).

If my interpretation of the evidence is correct, no single gene, such as TRPC2, but rather a whole set of genes (V1Rs, and possibly V2Rs) lost importance. All those genes were implicated, in one way or another, with pheromones detection and communication in the VNO. When the VNO becomes less important for sociosexual communication, its associated genes become less useful and they experience more relaxed selection pressure. The loss of selection pressure allows for mutations to arise, such as stop codons, and generates inactive pseudogenes (Moriya-ito et al., 2018). A relevant exception pertains to those genes that shift function from pheromones detection to oxygen detection. These genes are maintained under stabilizing selective pressure, thus preserving their functionality (Niimura, Matsui, & Touhara, 2014). In sum, I would like to see stronger evidence that the loss of a functional TRPC2 gene played an exclusive role in evolution of same-sex sexuality, as opposed to a general evolutionary transformation in mammals—not just primates—of many vomero-nasal receptor genes, toward amplification or degeneration.

A final critique of the Target Article, but one that is no less relevant, is that Pfau et al.'s (2019) hypothesis does not apply to human homosexuality. Most Old World primates have been observed to engage in same-sex sexual behavior. Very occasionally this involves some homosexual partner preference (Vasey, 2002), but never exclusive homosexual orientation as in humans. In humans, same-sex-sexual behavior lost its social communication function almost completely to become a sexual orientation.

I am skeptical that the olfactory and pheromonal processes posited by Pfau et al. (2019) caused a homosexual "orientation" in humans. The evolutionary dilemma of human homosexual orientation has little to do with same-sex sexual behavior, which is only one ingredient of the phenotype. Homosexuality in humans is characterized by a novel and specific phenotype: an individual exclusively attracted sexually and romantically to same-sex individuals. This is the evolutionary dilemma, exclusive same-sex sexual attraction, which inhibits reproduction and reduces fertility. How could such a phenotype evolve and how could it be maintained in the population at a constant, albeit low, frequency? If this phenotype has a genetic basis, then it should become extinct rapidly, which we know does not happen (Camperio Ciani, Battaglia, & Zanzotto, 2015). What are the fitness advantages of exclusive same-sex sexual behavior in our species? Occasional same-sex sexual behavior does not exclude heterosexual sex and reproduction. On the contrary, it might provide a selective benefit to individuals, in reducing aggressiveness by using sexual pleasure to facilitate appeasement, but even if this is true, it is generally not the case in humans. In animals, same-sex sexual behavior enriches communication, sociality, and ultimately benefits individuals, so there is no evolutionary dilemma here. This is the ultimate reason that same-sex sexual behavior evolved in many social organisms (Bagemihl, 1999). That said, these social uses of same-sex sexual behavior do not produce exclusive homosexuality.

Pfau et al., in the Target Article, contend that the absence of reports on possible homosexual orientation in primates, including bonobos, might be ascribed to the possibility that researchers not have detected it yet. They suggest that the absence of evidence is not evidence of absence, but in this case, it is improbable. Vasey (2002) reported facultative (i.e., nonexclusive) homosexual *preference* for a few animals, including the domestic rams and few other ungulates, as well as female Japanese macaque (*Macaca fuscata*). These females occasionally show a preference for same-sex sexual partners over opposite-sex alternatives, but nevertheless they mate heterosexually and they all reproduce, according to my direct experience (Camperio Ciani, 1997; Corradino, 1990). Moreover, most primatologist will acknowledge that primates are difficult to locate in the wild, especially forest dwelling ones; however, once located, sexual behavior becomes overt (both visually and vocally) and very conspicuous in most species, if not all. Sexual interactions in wild primates are much easier to observe than in our species. Infinite hours of observation, including in the wild, focusing on sexual behavior in males and females have been undertaken by ethologists. With such a large sample, it would have been easy to locate individuals that engage in exclusive same-sex sexual behavior,







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but no one has. I have been observing several species of macaques in North Africa and South-East Asia and commonly observe same-sex behavior among both females and males, but never once have I observed a single-subject mating exclusively with same-sex partners (Camperio Ciani 1986; Camperio Ciani, Mouna, & Arhou, 2000; Camperio Ciani et al., 2005). There is only one species in which some males exhibit a homosexual orientation, but this is in sheep, not in primates, and is restricted to domesticates, that have been artificially selected. Domestic rams can thus furnish information on the neurophysiology and endocrinology of homosexuality, but they cannot furnish information about how natural selection might have produced such a phenotype (Roselli, Larkin, Schrunk, & Stormshak, 2004).

Pfau et al. (2019) suggested that if homosexual individuals occur in small groups, as might be the case for many primates, there might be no possibility of finding a homosexual partner with the same orientation. This, they suggest, might help account for the lack of observations of exclusive same-sex sexual partner preference in primates. This speculation is untenable. First, many primates, including baboons, macaques, and vervets, live in large multi-male multi-female groups. If occasional same-sex behavior is already present within a species—as is the case for many primates—an exclusively homosexual individual, should one exist, could find several same-sex partners with whom they could engage in sex, even if those partners were not exclusively homosexual themselves. This in fact happens also in our own species, where exclusive homosexual individuals can find occasional partners who are heterosexual (Whitam, 1992).

In conclusion, the hypothesis that the decline of pheromonal communication allowed for the evolution of social complexity, including same-sex sexuality, is compelling. In my view, the idea that a single gene, and not a whole set of genes, promoted this shift needs further testing. Regardless, Pfau et al.'s (2019) hypothesis while heuristic for the evolution of same-sex sexuality in nonhuman primates, fall short of explaining the evolution of an exclusively homosexual phenotype as seen in humans.

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