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Review

Evolutionary Perspectives on Male Homosexuality: A Review

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Abstract

This review provides a comprehensive coverage of the leading evolutionary hypotheses to date on male homosexuality: the sexual antagonism model, the tipping-point model, and the kin selection hypothesis. It does so by first, surveying the most prominent findings on the biological causes of male homosexuality; second, discussing the effects of male homosexuality on individual fitness; and third, outlining the currently contending evolutionary theories on male homosexuality and critically evaluating each against current, pertinent empirical evidence. This review reveals that male homosexuality is a complex, multifaceted phenomenon influenced by an interplay of genomic and environmental factors that may have had unique evolutionary trajectories. Thus, there is likely more than one evolutionary mechanism at play responsible for the maintenance of gay alleles in the human population. Current research largely supports the notion that the alleles responsible for male homosexuality bestow fitness benefits in heterosexual carriers. The tipping-point model and sexual antagonism model, but not the kin selection hypothesis, are in line with such evidence. Future research into the genomic underpinnings of sexual orientation in homosexual males and its genetic equivalents in heterosexual males and females may allow for further evaluation of these hypotheses.

Keywords: Human Evolution, Evolutionary Psychology, Mating Preferences, Sexual Orientation, Male Homosexuality

1 Introduction & Background

Homosexuality refers to sexual attraction, romantic attraction, or sexual behaviour toward members of the same sex[1]. As a sexual orientation, it is the enduring pattern of sexual or romantic attraction and behaviour toward members of the same sex[1]. According to research, human sexual orientation exists along a continuum that ranges from exclusive homosexuality to exclusive heterosexuality and includes many forms of bisexuality; it does not exist as a mere heterosexual–homosexual dichotomy[2, 3, 4].

Homosexual behaviour has been documented in more than 500 different species of animals: in various primate species and in every major animal group (mammals, birds, reptiles, amphibians, fish, insects, and invertebrates)[5, 6, 7, 8]. This includes courtship, affection, sexual activity, pair bonding, and parenting all observed in multiple settings: in the wild, in captivity, and in the laboratory[5, 6, 7, 8]. Homosexuality has also been documented in human societies over several millennia and archaeological evidence (petroglyphs, ancient paintings, tomb figurations, etc.) has confirmed its occurrence prehistorically[9, 10, 11]. Since human sexuality varies along a continuum, reliably measuring the prevalence of homosexuality in the human population is challenging for researchers. Moreover, due to the widespread heterosexist discrimination found in many societies, many homosexual individuals do not openly identify as such[1]. The documented prevalence of homosexuality has been found to vary largely over time and geographic region[12]. According to some surveys, 2–11% of people in the West have had some form of same-sex sexual contact in their life[13]. This percentage rises to 16–20% when both same-sex behaviours and same-sex attractions are considered.In a 2006 Australian study, 20% of respondents anonymously reported some homosexual feelings, although only 2–3% identified themselves as exclusively homosexual[14]. In the scientific community, the consensus is that approximately 2–9% of females and 1–10% of males in the West are exclusively homosexual[12, 15]. Thus, homosexuality represents a small but significant sexual minority phenotype in humans.

The persistence of homosexuality throughout the evolutionary history of primates and other animals has been coined an "evolutionary paradox"[16]. Several competing evolutionary hypotheses have attempted to shed light on this Darwinian puzzle, however, no consensus has yet been reached on a single, prevailing evolutionary account of the matter[17]. Because most research in the scientific literature pertains to male homosexuality and the empirical studies investigating lesbianism are sparse, the evolution of lesbianism remains understudied. Consequently, only male homosexuality will be examined in this review.

This review is divided into three main sections: "Biological Links", "Measures of Maladaptiveness", and "Evolutionary Perspectives". The first section surveys the most notable research on the nature and causes of male homosexuality from a psychobiological perspective. This provides us with a backdrop of knowledge against which we can appropriately assess evolutionary explanations later on. The second section discusses the evolutionary maladaptiveness of male homosexuality and its effects on individual fitness. The third section surveys the most prominent evolutionary theories to date on male homosexuality and critically evaluates each against recent empirical evidence.

1.1 Biological Links

Scientists believe that male homosexuality is the result of an interplay of biological and intrauterine environmental factors, and that it is shaped very early in life[12, 18, 19, 20]. Scientists generally do not believe that one's sexual orientation is a matter of choice[12, 18, 19, 20].

1.1.1 Genetics

Studies have shown that male homosexuality is not evenly distributed within the population but rather runs in families, generally on the maternal line[14, 21, 22]. Despite numerous attempts, no single "gay gene" has been identified; however, there is evidence for the presence of multiple contributing genetic factors for homosexuality throughout the human genome[12, 23, 18, 22, 24, 25, 26, 27, 28, 29]. Some association has been found with the Xq28 region on the X-chromosome of homosexual males[25, 28, 29]. In addition, a recent study has found a higher proportion of homosexual males with type A blood and with Rh-negative blood than heterosexual males[24]. Since these traits are controlled for by genes located on autosomal chromosomes, this indicates a possible autosomal genetic contribution to the development of a homosexual orientation[24]. However, this may also be the result of a confounding association between blood group and the X-linked androgen receptor gene[30].

Male homosexuality has also been found to be more common in male relatives on the same maternal line[14, 21, 22]. Moreover, identical twins are more likely to share a sexual orientation than fraternal twins[12, 18, 26, 31]. While some research on identical twins has revealed a 50% concordance rate for homosexuality among the siblings, other studies have found a 20% concordance rate[12, 18, 26, 31]. This research indicates that approximately a third of variation in sexual orientation is attributable to genetic differences among the siblings[18, 31]. Given the differences in sexual orientation in many sets of identical twins, researchers conclude that sexual orientation cannot be attributed to genetic factors alone[12, 18, 26, 31]. Hypotheses addressing these differences consider epigenetic, developmental, and environmental modifiers, such as differences in intrauterine blood transfusion and hormone exposure among the siblings[12, 18, 26, 31].

1.1.2 Environmental Factors (Nurture)

There is no scientific evidence that the social environment after birth has an effect on an individual's sexual orientation[12, 19, 20, 32]. Likewise, there are no empirical studies that support attributing a homosexual orientation to early abuse, trauma, family dysfunction, abnormal parenting, or any other adverse life events[12, 32, 33]. Moreover, there is no evidence that the use of psychological interventions (i.e. conversion therapies) can change one's sexual orientation[34, 35].

1.1.3 The Uterine Environment

Research suggests that sexual differentiation of the human brain occurs during fetal development, programming our gender identities and sexual orientations while we are in the womb[20, 36]. Circulating testosterone from the developing testes is said to act as an organizing factor for the developing nerve cells during a brief critical period, promoting the development of permanent male-typical neuronal patterns[20, 36, 37]. Female-typical neuronal development is said to occur in the relative absence of this hormone surge[36, 37]. Research suggests that male sexual orientation is influenced by intrauterine factors that affect fetal testosterone production, thereby influencing the masculinization of the male fetal brain[19, 20, 36, 37]. This is consistent with documented observable differences in the brains of homosexual and heterosexual males[19, 20, 36, 37]. A number of variations in brain structure have been reported between homosexual and heterosexual men, including the size of the suprachiasmatic nucleus and INAH3 neuronal group of the hypothalamus, with homosexual men typically exhibiting sex-atypical dimorphisms[19, 20, 36, 37, 38]. These findings are consistent with the role of the hypothalamus as a regulator in reproductive function[36]. Furthermore, concentrations of intrauterine testosterone may be influenced by maternal consumption of certain drugs, direct injection of the hormone, maternal immune system reactions, and maternal stress[39]. While some scientists speculate that homosexual males may have been exposed to lower androgen levels in the womb, others maintain that genes still undiscovered may play a role in reduced androgen sensitivity responses in male fetuses that grow up to be homosexual[32, 37, 40].

1.1.4 Maternal Stress

Research suggests that if a woman experiences severe emotional stress during her pregnancy, the likelihood of her giving birth to a homosexual son may increase[39, 41, 42, 43]. This

is said to be because circulating maternal stress hormones (e.g. cortisol) cross the placenta and disrupt fetal testosterone levels and their synchronization with neurodevelopmental epochs[41, 42, 43].

1.1.5 Fraternal Birth Order

Boys with older brothers are significantly more likely to be homosexual, with the chance of homosexuality increasing by about 33% with every older brother[18, 19, 37, 42, 44, 45, 46]. In fact, fraternal birth order is now considered to be one of the most reliable, cross-culturally robust epidemiological variables identified in the study of homosexuality[18, 19, 45, 46]. Consistent with this finding is that the finger length ratio between the index and ring fingers (the 2D:4D ratio), a crude measure of prenatal exposure to testosterone, decreases as fraternal birth order increases[18, 40]. To explain the fraternal birth order finding, it has been proposed that male fetuses provoke a maternal immune system reaction that becomes stronger with each successive male fetus[44, 45]. Maternal antibodies, part of this immune reaction, cross the blood–brain barrier and attack the proteins that play a role in the masculinization of the male fetal brain[45].

1.2 Measures of Maladaptiveness

1.2.1 History as a Psychological Disorder

In 1952, when the American Psychiatric Association (APA) published its first Diagnostic and Statistical Manual of Mental Disorders (DSM), homosexuality was classified as a psychological disorder[47]. This classification was later scrutinized when research failed to provide an empirical basis for its support[48, 49, 50]. The APA concluded that this classification reflected untested assumptions based on once-prevalent social norms and removed homosexuality from the DSM, stating that it implies no impairment to general, social, or vocational abilities[47, 48, 49, 50, 51]. Thereafter, the APA urged mental health professionals to act as leaders in helping combat the stigma of mental illness that has long been attributed to homosexual orientations[48]. Today, scientists and mental health professionals agree that homosexuality poses no intrinsic obstacle to leading a healthy, happy, or productive life in the full array of social institutions[18, 32, 48, 49].

1.2.2 Mental Health

Male homosexual youth continue to be at an increased risk of compromised mental health than their heterosexual peers[52, 53, 54]. A recent US study interviewing a community sample of gay youth between the ages of 16 to 20 found that approximately 18% of gay participants met the diagnostic criteria for major depression, 11.3% for PTSD in the past 12 months, and 31% for suicidal ideation[53]. When comparing these findings to national mental health diagnosis rates for the general population, the difference is stark: The rates for these diagnoses and behaviours among youth are 8.2%, 3.9%, and 4.1%, respectively[55, 56]. Gay youth have also been found to be at an increased risk of substance abuse, bullying, and psychiatric comorbidity[54]. In all, researchers agree that the compromised mental health of gay youth is the cause of social ostracism, isolation from family and peers, internalization of negative societal stereotypes, and/or limited support structures in place for them in society[52, 53, 54].

1.2.3 Reproduction

From an evolutionary standpoint, the fundamental maladaptiveness of homosexuality is evident. Homosexual individuals, most typically, do not have children of their own. Although modern methods such as *in vitro* fertilization and artificial insemination are now being used by same-sex couples to produce biological children, these methods have only been developed in the past century and could not have been responsible for the passing on of gay alleles throughout human history[12].

1.3 Evolutionary Perspectives

1.3.1 Homosexual Individuals as "Helpers-in-the-Nest"

In the *Origin of Species*, Darwin described how entire family groups or bloodlines (not just individuals) can compete for selection[57]. The *kin selection hypothesis* of male homosexuality, popularized by Wilson in 1975, posits that homosexual individuals can compensate for their lack of biological children by maximizing the reproductive success of their family members. Thus, rather than reproducing themselves (i.e., *direct fitness*), homosexual individuals enhance the reproductive success of those who share their genetic code (i.e., *indirect* or *inclusive fitness*)[58, 59]. According to this model, although the alleles that predispose individuals to a homosexual orientation do not get passed on through the reproduction of homosexual individuals themselves, they may still get passed on to the next generation by their relatives[58, 59].

Theoretically, there are many ways that homosexual individuals can be said to increase the reproductive success of their family members, such as by contributing resources (eg., food, shelter, etc.), performing "uncle-like" activities (eg., taking care of offspring), and helping family members in times of stress (eg., providing defense, supervision, resources, or care)[59, 60, 61]. The kin selection hypothesis views homosexual individuals as essentially "helpers-in-the-nest"[58]. This hypothesis also argues that homosexual individuals may contribute substantially to the emotional wellbeing and overall cohesion of their family[60, 61]. This idea is consistent with studies showing lower levels of hostility and higher levels of emotional intelligence, cooperation, and empathy in homo-

sexual men[15]. Evolutionarily speaking, the ability of family members to bond with and cooperate cohesively within their familial group may have determined in many cases whether the group survived or perished[60].

The kin selection hypothesis was tested by Vasey, Pocock, and VanderLaan on the Pacific island of Samoa in 2007. In this island, Samoans live in a highly primitive and traditional society reminiscent of the human ancestral past, and Samoan homosexual and transgendered males are socially accepted by the majority of Samoans[61], see also[62, 63]. Vasey et al.'s 2007 study found that gay Samoan men were significantly more willing to help their kin than were straight, childless men, providing the first ever evidence in support of the kin selection hypothesis. However, a later study by Vasey and VanderLaan[64] found that homosexual men in Japan were no more generous or attentive towards their nephews and nieces than were childless, heterosexual men and women. More evidence against the kin selection hypothesis later surfaced in several studies across the United Kingdom, United States, and the West, with homosexual individuals not found to provide more care or resources to family members than their heterosexual counterparts[16, 64, 65]. This remained true regardless of the types of measures used, whether these measures were subjective (e.g., feeling of closeness to the family) or objective (e.g., frequency of contact with the family, distance residing from relatives, etc.)[16, 64, 65]. However, researchers currently disagree on whether these results implicating a lack of support for the kin selection hypothesis could be the cause of data being gathered in modern, industrialized societies (e.g. the UK, USA, and Japan), which are less remnant of the human ancestral environment and are characterized by fervent social intolerance towards homosexuals[62, 63, 66].

1.3.2 Additional Functions of "Gay Alleles"

This explanation posits that the group of alleles that code for a homosexual orientation in gay males also confer strong reproductive advantages in heterosexual individuals, resulting in the persistence of gay alleles in the gene pool as their successful heterosexual carriers pass them down[67, 68, 69].

1.3.3 Coding for Femininity in Males

The *tipping-point model of male homosexuality*, popularized by Edward Miller, posits that the group of alleles that code for a homosexual orientation in gay men confer strong fitness benefits in heterosexual men by coding in them a certain level of psychological femininity[68]. According to Miller, if only a few of these alleles are inherited by males, their reproductive success is enhanced via the expression of attractive, albeit feminine traits such as kindness, empathy, and sensitivity[68]. However, if too many of these alleles are inherited by males, a tipping-point is reached, at which even their mate preferences become feminized[68].

Miller came up with a simplified version of his theory to better illustrate it. He asks the reader to imagine that there are five different genes that each help code for an individual's place along a masculine–feminine continuum. Each of these five genes have two respective alleles: one that pulls the individual to the masculine side of the continuum and one that pulls the individual to the feminine side of the continuum. According to his simplified model, if a man inherits all five of the "feminine-pulling alleles", he will be homosexual and if he inherits less than five, he will not. Homosexuality would continue to persist in the human population if a strong reproductive advantage is conferred on individuals possessing some copies of these feminine-pulling alleles. According to Miller, a low dose of these feminine-pulling alleles significantly enhances a heterosexual male carrier's reproductive success. But in the less common, spontaneous occasion that a significantly large dose of these feminine-pulling alleles is inherited, the male carrier's sexual orientation is altered and his fitness adversely affected. Nonetheless, these alleles would continue to persist in the population if they confer an overall reproductive advantage on their male carriers[68].

Consistent with the tipping-point hypothesis, homosexual men are reported to be more sensitive, kind, and empathetic than heterosexual men, which have been characteristically deemed to be feminine attributes[70]. Furthermore, studies have found that a higher level of psychological femininity in straight men is associated with a greater number of female partners, suggesting that psychological femininity is attractive to women[71, 72]. This could be because psychological femininity indicates a nurturing disposition which could help rear offspring. In another study, researchers predicted that if the tipping-point model of male homosexuality were correct, then heterosexual men with a homosexual male twin should have more attractive feminine-pulling alleles and thus more opposite-sex partners than members of heterosexual twin pairs[15]. The findings of this large community-based twin study (N = 4904) supported this prediction; heterosexual males with a homosexual male twin had significantly more children, significantly more opposite-sex partners, and were significantly younger at their first age of intercourse than members of heterosexual male twin pairs ($p<0.001$)[15]. The results of these and similar studies have made the tipping-point model one of the leading evolutionary theories on male homosexuality to date[67].

1.3.4 Coding for Femininity in Males & Females

Another possibility is that the alleles responsible for male homosexuality code for psychologically or physically feminizing traits in both men and women[21, 67]. The *sexual antago-*

nism model suggests that an allele that is detrimental to the fitness of one sex could be maintained in the population so long as it is beneficial to the fitness of the other sex[21]. An allele that makes its bearer attracted to men and more feminine provides an obvious reproductive advantage to women, but an obvious reproductive disadvantage to men[21]. This allele would code for same-sex attraction if it appears in a male's genome but would maintain a net evolutionary benefit if this occurs rarely[21].

There is a significant amount of evidence for this theory. Numerous studies have found significantly greater fecundity in the female matrilineal relatives of homosexual men (i.e. their mothers, aunts and grandmothers) as compared to heterosexual men[21, 73, 74, 75]. Some other studies have also found that the female relatives of homosexual males have significantly fewer abortions and gestational complications than the female relatives of heterosexual males[12, 74]. Moreover, homosexual men have been found to have an excess of matrilineal but not patrilineal male homosexual relatives as compared to heterosexual men[21, 73]. According to researchers, even a modest increase in the reproductive capacity of females carrying these gay alleles could easily account for their maintenance at high levels in the population[21, 76].

2 Review & Discussion

As previously mentioned, significant maternal stress during pregnancy can disrupt fetal testosterone production and increase the likelihood of giving birth to a homosexual son[39]. As such, in the case of highly stressful environments, a family would benefit from having help in providing resources, shelter, and protection to its members. Additionally, because homosexual individuals do not have offspring of their own, this would prevent the family from becoming overburdened with more children in the future and would allow for the sole allocation of resources towards existing family members. Thus, the kin selection hypothesis is consistent with the maternal stress finding and may argue that homosexuality is activated epigenetically by environmental triggers linked to resource feedback, environmental stress, and the general need for help-in-the-nest.

The kin selection hypothesis is also logically consistent with the fraternal birth order finding[19]. If a family is already flush with children, epigenetic switches that alter the sexual orientations of subsequent fetuses and prevent them from adding more offspring to the family would be evolutionarily favorable[19]. Thus, homosexuality may be nature's way of ensuring that families do not have an unmanageable number of mouths to feed. A family flush with children would also benefit from added help-in-the-nest. Moreover, avoiding familial problems arising from competition for mates or for the allocation of resources towards one's own offspring could improve a family's overall health, cohesion, and success. This could be of vital importance in times of stress, when resources are scarce and mates not ample. Nonetheless, if homosexuality is indeed the result of an epigenetic switch that codes for a needed helper-in-the-nest who does not have offspring of his or her own, why does fraternal birth order, in particular, act to trigger such a switch but not birth order more generally? Moreover, no such correlation between birth order and homosexuality has been found for females[19]. The kin selection hypothesis does not address this fundamental disparity.

Ultimately, the kin selection hypothesis suggests that homosexuality is a switch in reproductive strategy: a trade-off between mating effort and alloparenting effort (i.e., parenting offspring other than one's own). However, why would individuals intended to be alloparents be anything but asexual? In insects of the order Hymenoptera (e.g., bees, wasps, and ants), individuals that alloparent are asexual[77]. These individuals do not expend any time, effort, or resources on courtship or on pair bonding with members of the same or opposite sex[77]. The kin selection hypothesis could explain the asexuality in these bugs destined to be alloparents but does not seem to account for homosexuality in humans.

In all, little empirical evidence has been found in support of the kin selection hypothesis. Researchers have concluded that homosexual individuals generally do not provide more care or resources to family members than heterosexual individuals[16, 65, 64]. Moreover, the kin selection hypothesis' feasibility can be questioned from an evolutionary standpoint. Because individuals share at most 25 percent of their genes with their nephews and nieces, they must compensate for every child they do not have themselves with the birth and success of at least two nephews or nieces. This is an inefficient method of passing on one's genetic material from one generation to the next.

As previously mentioned, the tipping-point model of male homosexuality is supported by a variety of evidence. One purported finding that supports this hypothesis is that heterosexual individuals in homosexual–heterosexual twin pairs tend to be younger at their first age of intercourse and tend to have a greater number of opposite-sex partners than members of heterosexual twin pairs[15]. However, there may be an alternate explanation for this large-scale finding. Throughout their life course, twins try to assert their individuality and unlikeness from each another[78]. Thus, heterosexual individuals in heterosexual–homosexual twin pairs may experience an added pressure to act in a more heterosexual way as compared to their twin because it is a distinguish-

ing factor between the pair. Therefore, twin studies that support the tipping-point hypothesis should be regarded with scrutiny. Alternative possible study procedures may involve evaluating the psychological femininity and reproductive success of the fathers and male relatives of homosexual and heterosexual men, not just their twins. Men carrying a greater number of feminine-pulling alleles should be more likely to produce homosexual sons, have a higher level of psychological femininity, and have had a greater number of sexual partners in their lifetime. Another potential area for future research involves investigating the applicability of the tipping-point model to female homosexuality, i.e., whether lesbianism is caused by way of "masculine-pulling alleles." Future studies can investigate whether men find masculine traits attractive in women—analogous to how females find feminine traits attractive in men—to determine whether the tipping-point model is relevant to lesbianism.

The tipping-point model of male homosexuality supports a polymorphic view of male homosexuality (and male sexuality more generally), since it suggests that multiple, "feminizing" alleles are at play and have an additive influence on male sexuality[69]. Therefore, the tipping-point model is consistent with the fluid sexual orientation continuum that scientists agree on today. However, the tipping-point model does not account for the fraternal birth order finding or maternal stress finding of male homosexuality. If male homosexuality is caused by way of feminine-pulling alleles, do intrauterine factors related to fraternal birth order and maternal stress epigenetically activate these alleles? On the one hand, the link between fraternal birth order, maternal stress, and male homosexuality could be viewed as the result of meaningful epigenetic action on these feminine-pulling alleles; making a male fetus more "feminine" after several brothers are born or in times of stress may be evolutionarily favorable. On the other hand, the connection between fraternal birth order, maternal stress, and male homosexuality could be considered arbitrary, a cause of nothing more than chance variations that influence fetal testosterone production and coincidentally affect the development of the male fetal brain. Future research into the early uterine environment's influence on fetal gene expression and its relation to sexual orientation may elucidate the nature of this relationship.

As previously noted, the sexual antagonism model of male homosexuality is supported by a variety of evidence. However, this model cannot yet account for the relatively low frequency of homosexuality in males[21]. According to the principles of sexually antagonistic competition, the alleles that mutually code for homosexuality in men and increased fecundity in women should steadily increase in prevalence in the human population over time, since females that inherit them are met with greater reproductive success[21]. Thus, genotypic ratios within the sexes would become altered[21]. This would result in the steady increase of male homosexuality over time and could hypothetically lead to the eventual "sterilization" of the male sex[21].

However, the maintenance of male homosexuality at a generally fixed ratio and relatively low frequency in the human population for millennia contradicts this assertion[12]. To address this discrepancy, it has been proposed that these sexually antagonistic alleles are commonly expressed in females, but only sporadically expressed in males[21]. However, why there would be such an asymmetry in the expression of these alleles among the sexes remains unclear[21]. In order to move forward with this hypothesis, future genomic research must locate these genetic associations and confirm or disconfirm their asymmetry in expression among the sexes[21].

It is also important to note that, like the tipping-point model of male homosexuality, the sexual antagonism model does not account for the fraternal birth order and maternal stress connection to male homosexuality. Likewise, it may view this connection as either arbitrary or epigenetically meaningful. Future research into the early uterine environment's influence on fetal gene expression in relation to sexual orientation is needed to unravel the nature of this connection.

As previously mentioned, fraternal birth order increases the likelihood of homosexuality[19]. This is believed to be the result of an immune system reaction in the mother that develops after several males are born and interferes with the proteins that have a role in the masculinization of the male fetal brain[44]. It is possible that this immune system reaction has not been sufficiently selected against and hence eliminated by evolution because it can only come into play after several siblings are born, most of whom are heterosexual and go on to have children. Clearly this cannot solely account for the persistence of male homosexuality throughout our evolutionary history, since some individuals are born gay without having any older brothers; however, it may be part of the mechanisms at play.

3 Conclusion

Male homosexuality's persistence in the human population for millennia has been termed an "evolutionary paradox"[16]. There are several competing evolutionary hypotheses that attempt to shed light on this matter, some more supported by evidence than others. Male homosexuality has proven to be a complex, multifaceted phenomenon for both researchers and evolutionary theorists alike[12, 18, 63, 67, 79]. Several coexisting factors may influence the development of homosexuality

in males, whether independently or in conjunction with one another, and each of them may have had unique evolutionary trajectories[12, 18]. Thus, there is likely more than one evolutionary mechanism at play responsible for the persistence of gay alleles in the human population[12, 18].

Current research on male homosexuality primarily supports evolutionary perspectives arguing that "gay alleles" confer strong fitness benefits on heterosexual individuals. The tipping-point model and sexual antagonism model are the two most empirically supported evolutionary theories on male homosexuality to date[18, 21, 67, 69]. Future research into the genomic underpinnings of sexual orientation in homosexual males and its genetic equivalents in heterosexual males and females may allow for further evaluation of these hypotheses. If further research supports both the tipping-point and sexual antagonism models of male homosexuality, it may be that both models account for a unique piece of the evolutionary puzzle and must be merged into a single coherent account of the matter. In all, male homosexuality has proven to be a convoluted evolutionary phenomenon that a substantial amount of additional research is needed to elucidate.

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