

REVIEWS

Exploring Biological Theories of Homosexuality: A Focus on the X-linkage Theory and the Fraternal Birth Order Effect

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This article examines two independent theories, the X-linkage theory and the fraternal birth order effect, which suggest that homosexuality is shaped by complex biological processes, including genetic markers and maternal immune responses. Attention is given to the main arguments for and against these models, integrating both established and recent research. To supplement these theories, the role of epigenetics is noted, examining how prenatal and environmental factors, such as maternal stress, may impact homosexuality. By shedding light on the interplay between genetic predisposition and environmental influences, this paper calls for a deeper, more integrated understanding of homosexuality. Understanding these theories is crucial for advancing scientific inquiry into sexual orientation and informing contemporary societal discussions about identity, acceptance, and discriminatory laws. This study ultimately stresses the importance of continued research to fully elucidate the mechanisms involved in the development of homosexuality.

INTRODUCTION

Despite significant advances in homosexual rights across the world, many still do not view homosexuality as a normal or natural part of the human condition.¹ Historically, homosexuality has been regarded as “unnatural” or “perverse.”¹ In the late 19th century, medical professionals considered homosexuality a mental illness that could be diagnosed and treated.¹ While the removal of homosexuality from the third edition of the *Diagnostic and Statistical Manual of Mental Disorders* in 1973 marked a shift in the medical community’s understanding, the perception that homosexuality is a choice continues to persist.¹ As perceptions of homosexuality have changed, there has been a growing focus on understanding its etiology, including possible biological influences.

Understanding the biological basis of homosexuality is not just important within a historical context; it carries profound implications for modern society. Biological theories can challenge discriminatory policies that assume homosexuality is a choice, as these policies often rely on the belief that sexual orientation can be controlled or changed.² Recognizing homosexuality as a natural variation of human sexuality could be instrumental in influencing

legal frameworks and societal attitudes, promoting the idea that all sexual orientations deserve equal rights and recognition.^{2,3} This shift in understanding could ultimately impact numerous discriminatory policies, including but not limited to those regarding same-sex marriage, health care, adoption rights, and workplace equality, significantly changing the lives of countless individuals.³

An increasing number of studies indicate that sexual orientation is not a choice but is rather a multifaceted interaction of genetic, immunological, and neurodevelopmental factors.⁴ To better understand these processes, this study discusses 2 theories that seek to explain the complex origins of homosexuality. While these theories offer valuable insights, they should not be considered definitive or complete explanations, as other models, such as the prenatal androgen theory, have also been proposed and may contribute to its development.⁵ Nevertheless, this article focuses on the X-linkage theory and the fraternal birth order effect as central to the development of homosexuality because although hormones may play a role, the prenatal androgen theory limits their influence to specific windows of fetal development, indicating a more secondary role compared with genetic and neurodevelopmental factors.⁵ Additionally, although the X-linkage theory and the fraternal birth order effect are independent of one another, their interaction cannot be ruled out.

It remains important to recognize that although the term homosexual refers to all forms of same-sex attraction, gay and lesbian specifically describe male and female same-sex attraction, respectively. In this paper, the term homosexual is used for consistency with existing literature, and the focus remains on male homosexuality to align with the emphasis of past research in relation to these theories.

METHODS

The aim of this article was to examine 2 prominent theories explaining homosexuality, offering a concise overview of their key arguments and rebuttals, while incorporating the most recent studies to reflect a contemporaneous understanding of the topic.

To do so, a systematic search of Ovid Medline was conducted using specific search terms including *fraternal birth order effect*, *X-linkage theory*, *homosexuality genetics*, and *sexual orientation*. For inclusion, we selected studies published since 1993, with a focus on those that provided substantial evidence for or against the theories, as well as those that contributed to the development of the discourse. This timeframe was chosen to encompass both foundational studies, such as the one that originally proposed the X-linkage theory, and more recent research. Priority was placed on peer-reviewed articles, randomized clinical trials, and studies with larger sample sizes, excluding studies that lacked empirical data or did not directly

contribute to the understanding of the theories under review. Rather than providing an exhaustive list of all published literature, existing research was synthesized to contextualize and critically assess the supporting evidence and counter arguments for each theory. Additionally, a targeted search on epigenetic mechanisms was conducted using terms such as *epigenetics and homosexuality*, *prenatal environmental factors and sexual orientation*, and *maternal stress and epigenetics* to address emerging questions and complexities within ongoing discussions regarding these theories.

LITERATURE REVIEW

X-Linkage Theory

Hamer et al⁶ were the first to present evidence for the X-linkage theory, demonstrating a significant association between certain markers on the X chromosome and male homosexuality. Subsequent studies focused extensively on the Xq28 region of the X chromosome, citing it as a potential locus for genes related to male homosexuality.⁷ In 2015, Sanders et al⁸ reported a genetic linkage study of 409 independent pairs of homosexual brothers (908 analyzed individuals in 384 families) and confirmed the association between Xq28 and male homosexuality. In 2021, Sanders et al⁹ jointly analyzed the Molecular Genetic Study of Sexual Orientation dataset (409 sibling pairs from 384 families) and the study by Hamer et al⁶ (155 sibling pairs from 145 families), repeatedly showing an association between male homosexuality and chromosome Xq28. More recent research, such as a 2021 genome-wide association study, analyzed 1478 homosexual males and 3313 heterosexual males from the Han Chinese population and identified a new locus, *FMR1NB*, on chromosome X, distinct from the Xq28 region.¹⁰ While these findings introduce new genetic loci that may contribute to homosexuality, they do not negate the continued significance of the X chromosome in these studies. Instead, they suggest a more complex genetic landscape for homosexuality, where multiple loci, including those on the X chromosome, may interact in ways that influence sexual orientation. To date, these appear to be the most current and significant genome-wide association studies.

Arguments Against the X-Linkage Theory

As scientists have sought to clarify this model, arguments have arisen, particularly around the concordance of sexual orientation among monozygotic twins.¹¹ Researchers have argued that if sexual orientation were strictly determined by genes on the X chromosome, identical twins who share 100% of their genetic material should always have the same sexual orientation.¹¹ However, research has found discordant twin pairs, where one twin identifies as heterosexual and the other as homosexual.¹¹ Other twin investigations have shown concordance rates of around 65.8% or lower, far from the 100% expected if X-linked genes were the sole determinant.¹²

Similarly, if male same-sex attraction were primarily influenced by an X-linked gene, it would be expected to follow a clear maternal inheritance pattern, but many analyses have failed to identify any consistent or reliable pattern.^{13,14} Studies have also cited additional loci on autosomal chromosomes that seem to be associated with homosexuality, showing that it is not limited to the X chromosome.^{9,10,14} In fact, while research from Sanders et al⁹ and Hu et al¹⁰ demonstrated associations with chromosome X, they also identified relevant loci on chromosomes 8 and 19, respectively. Other genome-wide investigations by Ganna et al¹⁵ did not identify an X chromosome locus and have instead found associations with only autosomal loci, such as chromosomes 4, 7, 11, 12, and 15.

Fraternal Birth Order Effect

As research sought to understand patterns of male homosexuality and its potential links to familial and birth order factors, researchers began to note that homosexual males were more likely to be later-born than first-born and had a higher number of older brothers than older sisters.¹⁶ This observation gave rise to the fraternal birth order effect, first reported by Blanchard and Bogaert.¹⁶ Its prevailing hypothesis suggests that some mothers develop placenta-permeable antibodies against a Y-linked minor histocompatibility antigen in males, which affects the brain of the fetus and leads to same-sex attraction.¹⁶ The primary male-specific antigens involved in this process are neuroligin 4 Y-linked (NLGN4Y), a cell adhesion molecule crucial for synaptic plasticity and the formation of excitatory synapses in brain regions related to cognition and social behavior, and protocadherin 11 Y-linked (PCDH11Y), a protein that regulates the establishment and maintenance of synaptic connections, influencing neuronal connectivity in areas responsible for social interaction and communication.¹⁷⁻¹⁹ Disruption of these antigens is thought to lead to altered brain development, potentially influencing social behavior and sexual orientation. With each subsequent male pregnancy, the maternal immune response is hypothesized to intensify, thereby increasing the likelihood of same-sex attraction in later-born sons.^{16,17} The fraternal birth order effect has been documented worldwide, including North America, South America, Europe, Asia, and Oceania.²⁰ Several repeat studies by Blanchard and fellow researchers, including Purcell et al,²¹ Cantor et al,²² Bogaert,²³ and VanderLaan and Vasey,²⁴ have consistently found evidence supporting the fraternal birth order effect. Many recent studies, including those by researchers not affiliated with Blanchard, such as Ablaza et al²⁰ and Fořt et al,²⁵ also support the existence of the fraternal birth order effect.

Ablaza et al²⁰ used population-level data (n = 9073496) from the Netherlands to address key limitations of previous studies, including small sample sizes and selective sampling, showing that an increase in older brothers independently raises the likelihood of homosexuality. Their regression model, which controlled for various factors, revealed that adding a younger sister

to a family was linked to a 13.8% decrease in the probability of entering a same-sex relationship (odds ratio [OR] = 0.87, $P < .001$), while moving one place down the birth order without modifying the number of younger and older brothers augmented the probability by 7.9% (OR = 1.08, $P < .001$).²⁰ Most notably, replacing an older sister with an older brother was associated with a 12.5% increase in the likelihood of entering a same-sex relationship (OR = 1.13, $P < .001$).²⁰ Drawing on data from an online questionnaire that included Czech and Slovak participants, Fořt et al²⁵ analyzed a sample of 693 gay men, 843 straight men, 265 lesbian women, and 331 straight women. Using a parameterized logistic regression model, Fořt et al²⁵ confirm the fraternal birth order effect in gay men ([OR] = 1.35 [95% CI, 1.14, 1.60], $P < 0.001$).

Arguments Against the Fraternal Birth Order Effect

Not all research has been able to replicate that the likelihood of male homosexuality increases with the number of older biological brothers, as some studies have failed to show a significant association between the number of older brothers and male homosexuality.²⁶⁻²⁸ Most recently, Vilsmeier et al²⁸ critically reassessed the fraternal birth order effect, building on statements made in their 2021 preprint publication, arguing that previous studies relied on incorrect statistical assumptions.^{28,29} Their meta-analysis (N = 2 778 998) indicated that the association between older brothers and homosexual orientation was “small, inconsistent, and not specific to men.”²⁸ Blanchard and Skorska²⁹ openly critiqued the preprint publication by Vilsmeier et al,^{30,31} suggesting that the discrepancies reported by Vilsmeier et al may stem from their reliance on the “unreliable dataset” from Frisch and Hviid.^{29,32} In doing so, Blanchard and Skorska disqualified the study by Vilsmeier et al²⁸ before it was even published, as it was based on data from their earlier 2021 preprint. To date and to our knowledge, Vilsmeier has not directly addressed Blanchard’s criticisms, raising significant implications for the ongoing discourse surrounding the fraternal birth order effect, particularly regarding the robustness and credibility of the study by Vilsmeier et al.²⁸ Vilsmeier’s lack of response may subsequently call for a reexamination of previous studies relying on the same dataset, potentially leading to further critiques or revisions of arguments against the fraternal birth order effect. In addition to these critiques, researchers, including proponents of the fraternal birth order effect, have further reported that the model does not seem to apply to female homosexuality, raising questions about the sex-specific nature of the effect and its generalizability.^{33,34} This conclusion is directly contradicted by Ablaza et al²⁰ and Fořt et al,²⁵ whose data found strong evidence of a fraternal birth order effect in both sexes.

THE POTENTIAL ROLE OF EPIGENETICS

Epigenetics refers to biochemical modifications that do not involve changes to the underlying DNA sequence, such as DNA methylation, which silences gene expression, and acetylation, which activates gene expression.³⁵ In the context of sexual orientation, these genetic changes appear to offer an important perspective in addressing the complexities of sexual orientation, including the concordance and discordance rates of homosexuality among monozygotic twins, which have been central to the debate surrounding the X-linkage theory.¹¹ Prenatal environmental factors, such as maternal stress, are thought to play a role in understanding this variability because they can influence intrauterine environments and cause epigenetic modifications.³⁶ For instance, if one twin was exposed to higher levels of maternal stress or different prenatal conditions, the intrauterine environment could trigger DNA acetylation in certain genes related to sexual orientation in that twin, while the other twin does not experience the same changes. These changes could help explain why in monozygotic twins who share identical genetic material, one twin may develop a homosexual orientation while the other does not.

Similarly, in the context of the fraternal birth order effect, if a mother experiences stress during her pregnancies, epigenetic modifications could lead to altered immune function.^{36,37} As a woman has more male offspring, she may experience increased psychological and physical stress. Over time, these changes could lead to a heightened immune response, making the mother more likely to produce antibodies against male-specific proteins in subsequent pregnancies.¹⁷ Specific epigenetic modifications influencing sexual differentiation in the human brain remain unidentified, but recent research, including a 2024 study, discuss how epigenetic mechanisms play a crucial role in sex-specific neurobehavioral changes during key developmental periods and in response to environmental factors.³⁸

Studies have further shown that epigenetic changes can be passed down to subsequent generations.³⁹ While research is still in its early stages, epigenetic inheritance offers a potential explanation for why sexual orientation might not only be influenced by an individual's genetic predisposition, but also by the epigenetic environment created by previous generations. It remains important to acknowledge that not all epigenetic changes are stable across generations.⁴⁰ Some epigenetic marks can be reset during gamete formation, while others remain subject to environmental factors.⁴⁰ This complexity and variability across generations may explain why these theories are often criticized for not being universally consistent across all people or generations.

CONCLUSIONS

Large genomic studies have consistently highlighted the X chromosome's role in the genetic basis of homosexuality, lending support to the X-linkage theory.⁸⁻¹⁰ Moreover, recent studies have suggested that this genetic influence is likely multilocal, extending to other regions of the genome in addition to the X chromosome.^{9,10,14} The fraternal birth order effect sustains the biological basis of homosexuality, with accumulating evidence showing that maternal immune responses to male-specific antigens intensify with each consecutive male pregnancy, subsequently influencing the sexual differentiation of the brain in later-born sons.^{16,17,20} While studies such as that of Vilsmeier et al²⁸ have questioned the validity of this effect, expansive research, including that of Ablaza et al,²⁰ provides evidence supporting its presence in both sexes. Epigenetics seem to offer additional insight into these processes, especially in light of monozygotic twin studies, where concordance rates for homosexuality are often less than 100%. The integration of epigenetic factors into existing models could help clarify these inconsistencies. By incorporating the role of environmental influences, such as maternal stress or prenatal conditions, alongside genetic factors, researchers could better understand why some monozygotic twins exhibit different sexual orientations despite sharing the same genetic material. This integration may also provide a more nuanced explanation for the variability observed across studies, offering a more comprehensive understanding of the complex factors that contribute to sexual orientation.

Amid decades of polarized research, including recent studies, the current article emphasizes the urgent need for continued exploration into the complexities of homosexuality. While various theories, including those involving epigenetic influences, offer valuable insights, it is recognized that they may not capture the full spectrum of mechanisms driving the development of homosexuality. In addition to calling for a greater understanding of the molecular mechanisms involved in these theories and epigenetic processes, subsequent avenues of research could examine whether these models are mutually present within the families of homosexual individuals. Furthermore, despite global findings supporting the existence of the fraternal birth order effect, research remains limited in terms of cross-cultural comparisons.²⁰ Exploring the effect size and significance across Western and non-Western populations could reveal regional variations, offering insights into cultural, genetic, or environmental factors that may be at play. Finally, in light of conflicting findings, particularly those from Ablaza et al²⁰ and Fořt et al,²⁵ a deeper investigation into whether the fraternal birth order effect plays a role in female homosexuality is necessary to resolve inconsistencies in existing literature.

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Conflicts of Interest

None reported.

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