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ELEANOR WHITEWAY AND DENIS R. ALEXANDER

Understanding the causes of same-sex attraction

The aim of this paper is to review the current academic literature on the aetiology of same-sex attraction, with a particular focus on its biological causes. Environmental, biological and choice-based aetiologies are discussed, and the empirical evidence for each position is considered. We conclude that, while some aetiologies are better supported than others, no putative cause of same-sex attraction has a sufficient empirical basis to demonstrate its causal role in same-sex attraction. Furthermore, no single cause can explain the variety of forms of same-sex attraction across different genders and cultures. We suggest that same-sex attraction is likely to be caused by a complex interplay of factors, both biological and environmental, and that causal pathways are unique to the individual.[1]

Keywords: Same-sex attraction; homosexuality; genetics; environment; causation

Introduction

The aim of this paper is to review the current academic literature on the aetiology of same-sex attraction (SSA). No perspectives are provided on the behavioural choices an individual could make once their same-sex attraction becomes apparent to them, or on the morality of same-sex sexual behaviour. It is important for Christians to be familiar with the current research on this topic, both because of the Christian calling to be truth-tellers, and since it is important that the theological claims and policy decisions concerning same-sex attraction that are made by church communities are considered in the light of the best contemporary scientific understanding.[2] Currently, there is a great diversity in understanding about the causes of SSA, across Christian denominations. We hope this review will be of help in bringing clarity to this complex issue.

Many hypotheses about the causes of SSA have been proposed, which can be divided into three broad types of causes: environmental, biologi-

1 Issues of space entail that several points of interest are either discussed only briefly or omitted. For an expanded discussion and full references, please see the longer version of this article online at www.scienceandchristianbelief.org, or contact Denis Alexander at dra24@hermes.cam.ac.uk for a copy of this version.

2 A consideration that also applies to secular politics; see Bailey, J.M. 'Anti-gay Uganda claims sexual orientation is a choice', *New Scientist* (2014), http://www.newscientist.com/article/dn25121-antigay-uganda-claims-sexual- orientation-is-a-choice.html (accessed 7th November 2014).

cal and personal choice. Following some comments on the definition and measurement of SSA and related terms, we briefly review the personal choice and environmental explanations proffered for SSA, then consider biological explanations in greater detail. However, in separating the proposed causes, it should not be assumed that a single cause or causal chain is responsible for such a complex phenomenon as SSA. In reality, as will be discussed, it is very likely that many different causes are operating in tandem, and that causes are operating in different ways across the cohort of same-sex attracted individuals, in ways that are likely to be gender- and culture-specific. Although considered under separate headings, all influences are in reality completely integrated within the life of a developing individual, meaning that no one should expect to find 'the' cause of an individual's same-sex attraction.

This paper necessarily deals with averages and generalisations, but it should not be forgotten that questions of sexual orientation and sexual attraction are immensely complex, with exceptions for every generalisation, involving as they do questions of love, morality and society. Every individual, regardless of sexual orientation, is made in God's image, and in considering an array of impersonal data, it is important to keep in mind that behind the statistics are human individuals, each one of whom is loved deeply by God.

Defining and measuring same-sex attraction

Before reviewing the causes of SSA, it is important to clarify what is meant by same-sex attraction, and acknowledge the complexities of definition and meaning in the field of sexual behaviour studies.[3] Sexual attraction refers to erotic desire experienced towards other individuals. Attraction is not a discrete variable, and exists along a continuum, from attraction exclusively toward the opposite sex (OSA) to attraction exclusively toward the same sex, with attraction to both sexes equally in the middle. An individual's sexual attraction status is generally measured on the Kinsey scale, which defines seven points ranging from 0 (exclusively OSA) to 6 (exclusively SSA). Although sexual attraction is continuous, in practice almost all the studies reviewed below that use the Kinsey scale collapse the continuum into discrete categories to increase statistical power: OSA (0-1), bisexual (2-4) and SSA (5-6), or OSA (0-1) and SSA (2-6), which should be remembered when interpreting empirical data.

Accurately measuring same-sex attraction, either demographically or

3 For an expanded discussion, see the longer version of this article online; see also Savin-Williams, R.C. 'Who's gay? Does it matter?', *Current Directions in Psychological Science* (2006) 15(1), 40-44; Gates, G.R. 'How many people are lesbian, gay, bisexual, and transgender?', Los Angeles: The Williams Institute (2011).

in experimental populations, is not straightforward. Firstly, attraction is often conflated with, or extrapolated from, measures of related but distinct concepts including sexual behaviour, sexual fantasy and self-identity. All these facets together contribute to an individual's sexual orientation. These facets do not always, or even frequently, exactly correlate, but interact in multiple ways in different individuals. For example, a person who experiences same-sex attraction may not engage in same-sex behaviour, and someone who self-identifies as straight may well experience some degree of SSA. On average, nearly three times as many people report some degree of SSA than the number of people who self-identify as gay, lesbian or bisexual. Consequently, studies that measure concepts other than attraction should be used to make inferences about same-sex attraction with caution. Having said that, in many of the studies reviewed below, it is a reasonable assumption that same-sex behaviour and/or a homosexual identity accompany same-sex attraction (although the converse is not necessarily true).

Secondly, even where attraction is directly assessed, measuring instruments (usually self-report questionnaires or surveys) can differ enormously. Variables include how questions are phrased, the degree or frequency of attraction deemed sufficient to categorise as SSA, the number of possible options offered, and the degree of anonymity. Different instruments can thus produce significantly differing results.[4] The time period assessed (lifetime SSA v. current or recent SSA) is critical, as sexual attraction can be a dynamic trait. Long-running longitudinal surveys in the US and New Zealand have found that although the majority of individuals, between 80% and 90%, have a stable sexual attraction or sexual self-identity across their lifespan, a sizeable minority experience change in their sexual attraction status over time.[5] Changes occur in both directions (i.e. SSA to OSA and vice versa) across the lifespan (Figure 1), so it is incorrect to assume that individuals who report current OSA never have or never will experience SSA. In absolute terms, more individuals move from exclusive OSA to degrees of SSA than move in the other direction, although in percentage terms, the reverse is true. There is also a significant sex difference, with female sexual attraction status much more labile than in men. These data are important for interpreting the causes of SSA – the variation in when same-sex attraction develops in different people is suggestive that no one cause is sufficient to explain all forms of SSA.

The literature considering the question as to whether SSA individuals can become OSA via various therapeutic and/or prayer and counselling regimes is not considered here. The natural lability of SSA in some indi-

4 Gates, G.R. *op. cit.*, (3).

5 Mock, S.E. & Eibach, R.P. 'Stability and change in sexual orientation identity over a 10-year period in adulthood', *Archives of Sexual Behavior* (2012) 41(3), 641-648; Ott, M., Corliss, H., et al. 'Stability and change in self-reported sexual orientation identity in young people: Application of mobility metrics', *Archives of Sexual Behavior* (2011) 40(3), 519-532.

viduals mentioned above should be kept in mind when considering this literature.

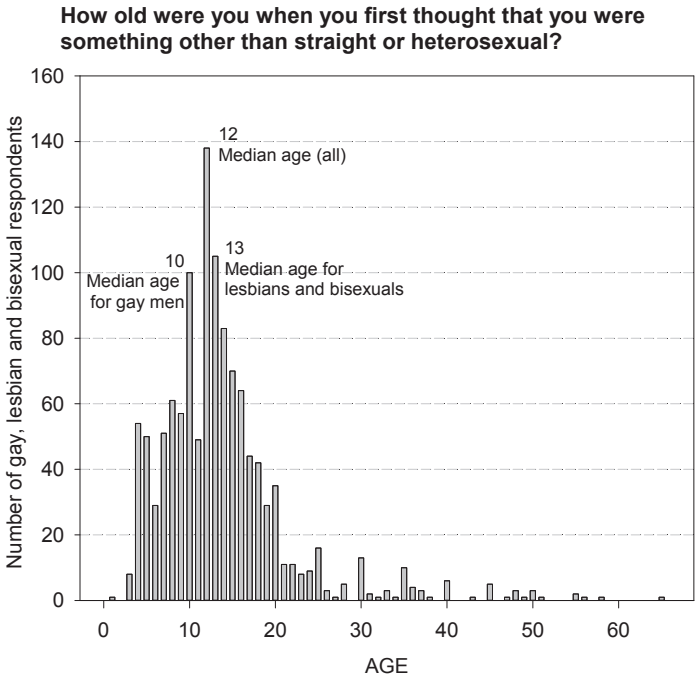

Figure 1. The development of same-sex attraction in a US demographic sample. The majority of SSA individuals develop stable same-sex attractions in early puberty, with a median age of 10 for men and 12 for women. A minority first identify their same-sex attraction much later in life. Adapted from Pew Research Center (with permission).[6]

Because of the difficulties of measurement, estimates of the prevalence of same-sex attraction, in either national or global populations, vary widely. Table 1 lists some recent prevalence estimates for the national UK population for same-sex attraction and behaviour, aggregated from multiple data sources. Across surveys some trends can be identified. Consistently, more women report experiencing some degree of SSA than men. Experiencing some same-sex attraction is more prevalent in the population than undertaking same-sex behaviour, which in turn is generally more prevalent than having a non-heterosexual self-identity (although measures of identity are highly variable across surveys). The prevalence

6 Pew Research Center 'A survey of LGBT Americans: attitudes, experiences and values in changing times', Washington, DC: Pew Research Center (2013).

of having exclusive same-sex attraction or behaviour is much lower than the prevalence of having any degree of same-sex attraction or behaviour.

	Men	Women
Any degree of same-sex attraction across the lifespan	6-8%	9-10%
Exclusive same-sex attraction	2%	<1%
Any same-sex behaviour across the lifespan	5%	2%
Exclusive same-sex behaviour	1%	<1%

Table 1. Summary prevalence rates in the UK for same-sex attraction and behaviour. Data aggregated from multiple data sources. Rates are comparable to other Western societies including the US and France. Very little data exist for developing societies. From Savin-Williams, R.C. (2009).[7]

Choice

Before reviewing in detail putative biological and environmental aetiologies of SSA, it is necessary to briefly consider a third category, that of personal choice. Many theorists would argue that an experience of sexual attraction, by definition, cannot be consciously chosen or willed, as attraction is a fundamental mental state that is not controlled by the conscious mind (the 'standard' model).[8] It is not disputed that individuals can make conscious choices about their sexual behaviour or sexual self-identity, or that same-sex attraction, once acknowledged, could be strengthened or reinforced by behavioural choices, but attraction itself is thought to be inherent, a state of being that 'happens' to someone rather than being the result of a conscious choice.

However, the standard model is not universally accepted, and it has been suggested by a number of groups that SSA is a conscious choice, and that individuals can will themselves to be attracted to persons of the same sex. Proponents of a choice-based aetiology cite several potential reasons why an individual might choose to be attracted to someone of the same sex, including personal politics, restricted opportunity, socio-cultural factors or revolt against cultural norms. For example, the lesbian feminist movement of the 1970s and early 1980s argued that women should choose to be attracted only to other women, as a rejection of heteronormativity and

7 Savin-Williams, R.C. 'How many gays are there? It depends', in Hope, D.A. (ed.) *Contemporary Perspectives on Lesbian, Gay, and Bisexual Identities,* New York: Springer (2009), pp. 5-41.

8 See the longer version of this article online for an expanded discussion.

patriarchal oppression. Much of the rhetoric of the organised Christian Right in the US and Africa portrays homosexuality as a 'lifestyle choice' that can be altered simply by choosing to be attracted to the opposite sex. Within the gay community, a group known as 'queer by choice' argue that their own experience of same-sex attraction is that they made a choice to feel that way.[9]

There is little empirical evidence that directly addresses this hypothesis, leading to often intense media debates,[10] but much of what is known about sexual attraction suggests that personal choice may be a causal factor for only a small, possibly very small, minority of SSA individuals, if it can be a matter of choice at all. No systematic survey has been done, but anecdotal evidence and informal surveys have found that the majority of SSA individuals report that they feel they were 'born gay' or simply became aware of a pre-existing and unconscious attraction. It is noteworthy that the average age at which homosexuals report first thinking they were other than straight is 12 (Figure 1); it seems unreasonable to suggest that adolescents of this age are all making conscious decisions to embrace an attraction that they may have seen derided in the popular press and society at large. Overall, if personal choice cannot completely be ruled out as a causal factor (which would involve denying the voices of those SSA individuals who feel that this does reflect their experience), it is unlikely to be the cause of SSA in the large majority of individuals, and there are, in any case, several other plausible aetiologies of SSA, as reviewed below.

Environmental causes

Psychoanalysis, parenting and phobia

During the middle part of the twentieth century, the most prominent aetiological explanation of SSA was derived from psychoanalysis. Beginning in 1940, neo-Freudian psychoanalyst Sandor Rado and his followers theorised that there was no innate psychological capacity for same-sex attraction (thinking exclusively about male SSA), and that SSA resulted from abnormal parent-son relationships. The classic homosexual male was thought to have an emotionally hostile and distant father and a 'close-binding-intimate' mother, known as the 'triangular system'; the smothering relationship with the mother and the lack of a heterosexual

9 Madwin, G. 'Myths about queer by choice people', (1999), http://www.queerbychoice.com/myths.html (accessed 10th November 2014).

10 Parris, M. 'Who's totally gay? There's no straight answer', *The Times,* 21st April 2013 (accessed at http://www.thetimes.co.uk/tto/opinion/columnists/matthewparris/article3390885.ece, 7th November 2014); Strudwick, P. 'You do not choose to be straight or gay; it chooses you', *The Independent,* 26th January 2012 (accessed at http://blogs.independent.co.uk/2012/01/26/you-do-not-choose-to-be-straight-or- gay-it-chooses-you/, 7th November 2014).

male role model caused the son to develop a phobia of having sexual interactions with women.[11] A small flurry of studies during the 1950s and 1960s reported finding this type of 'triangular' relationship in homosexual populations, most famously in Irving Beiber and colleagues' 1962 study of a population of institutionalised homosexuals in New York.[12] During this period, the triangular system became psychoanalytic orthodoxy, and was incorporated into the first and second editions of the Diagnostic and Statistical Manual (DSM) published (in 1952 and 1968 respectively) by the American Psychiatric Association, which listed homosexuality as a 'sociopathic personality disturbance'.

Outside the field of psychoanalysis, however, the triangular system had many critics, who highlighted a number of methodological flaws and unproven assumptions inherent to the psychoanalytic model. Sample sizes were always small, generally $n < 50$, and many studies did not use control groups. Sample cohorts were universally drawn from populations either in prison for homosexual offences, or undergoing psychiatric treatment for homosexuality. This introduced a significant source of bias, as probands often presented with multiple co-morbid psychiatric problems. The finding that SSA was associated with mental disorder was thus a reflection of sample bias, not an indication of the underlying cause of the probands' SSA. In 1956, Evelyn Hooker conducted a study with a population of non-criminalised, non-institutionalised gay men in California.[13] She found that these SSA men were otherwise psychologically healthy. When two trained psychiatrists were asked to distinguish between the psychological profiles of these gay men and a group of matched heterosexual controls, they did no better than chance. This result was supported by evidence from large-scale sociological surveys and from cross-cultural studies which suggested that homosexual behaviour was much more widespread and common than had been supposed[14] and that the majority of SSA individuals were psychologically normal and socially well-adjusted.

Beginning in 1966, attempts to replicate the results of Beiber et al. in non-clinical and non-institutionalised populations almost all ended in failure. Only one study supported the triangular system in its entirety, while many others found small or no differences in family relationships between

11 Drescher, J. 'A history of homosexuality and organized psychoanalysis', *Journal of the American Academy of Psychoanalysis and Dynamic Psychiatry* (2008) 36(3), 443-460.

12 Beiber, I., Dain, H.J. et al. *Homosexuality: a psychoanalytic study,* New York: Basic Books (1962).

13 Hooker, E. 'The adjustment of the male overt homosexual', *Journal of Projective Techniques* (1957) 21(1), 18-31.

14 Alfred Kinsey's original study of male homosexuality (n = 12,000) suggested that 10% of American males were point 5 or 6 on the Kinsey scale (see above), which combined attraction, fantasy and behaviour. Population surveys in the intervening 60 years suggest that these numbers were inflated by sampling bias. Kinsey, A.C., Pomeroy, W.B., & Martin, C.E. *Sexual Behaviour in the Human Male*, Bloomington: Indiana University Press (1948).

OSA and SSA men.[15] In the largest study (n = 979 homosexual, 477 heterosexual), no differences were found between homo- and heterosexual men in terms of having either hostile fathers or overbearing mothers.[16] In a 1974 survey, Siegelman found that the SSA men were more likely to have more emotionally hostile and rejecting relationships with their fathers only if they also scored highly for neuroticism,[17] supporting the argument that SSA had been wrongly conflated with other psychiatric problems due to sampling bias.

In conclusion, there is no evidence to suggest that SSA in men is the result of a phobic avoidance of heterosexual relationships caused by abnormal parent-son interactions. Even in studies that seemed to support the theory, sizeable numbers of participants reported perfectly healthy relationships with their parents, and larger, unbiased samples repeatedly found no evidence to support this aetiology. By the 1970s, the case for the triangular model had collapsed, and in 1973 same-sex attraction was removed from the DSM-III. It was also removed from the directories of the American Psychological Association in 1975, the World Health Organisation in 1992 and the Chinese Society of Psychiatry in 2001.

Childhood abuse and experience of trauma

A more recent proposal is the theory that SSA develops as a reaction to experiences of childhood sexual abuse (CSA). Most of the support for this aetiology comes from the growing body of cross-sectional and clinical studies, mainly from the US, which report that both male and female homosexuals experience higher levels, as much as two or threefold higher, of childhood sexual and physical abuse compared to matched heterosexuals.[18] A recent US demographic study (n = 22,071) found significantly elevated sexual, physical and emotional abuse during childhood (up to age 18) in adult homosexual and bisexual men and women.[19]

15 Siegelman, M. 'Parental background of male homosexuals and heterosexuals', *Archives of Sexual Behavior* (1974) 3(1), 3-18.

16 Bell, A.P., Weinberg, M.S. & Hammersmith, S.K. *Sexual preference, its development in men and women,* Bloomington: Indiana University Press (1981). This was one of the very few studies to also examine family relationships and SSA in women; as with men, no difference was found between homo- and heterosexual women.

17 Siegelman, M. *op. cit*., (15).

18 e.g., Balsam, K.F., Rothblum, E.D. & Beauchaine, T.P. 'Victimization over the lifespan: A comparison of lesbian, gay, bisexual, and heterosexual siblings', *Journal of Consulting and Clinical Psychology* (2005) 73(3), 477-487.

19 Andersen, J.P. & Blosnich, J. 'Disparities in adverse childhood experiences among sexual minority and heterosexual adults: Results from a multi-state probability-based sample', *PloS One* (2013) 8(1), e54691. It has been argued by some critics that this correlation results from recall bias, and that adult homosexuals retrospectively use CSA as a post-hoc rationalisation for their stigmatised behaviour, but evidence for this assertion is lacking, and there is nothing to suggest that this is not a genuine correlation.

Unfortunately, cross-sectional studies cannot distinguish the temporal order of correlated events, so the direction of causality is unknown. It is possible that experiencing CSA could be a contributing causal factor in the development of a same-sex attraction. If the abuser is the same sex as their victim, abuse could be interpreted as 'confirmation' that the person is homosexual, while an abuser of the opposite sex could provoke a traumatic avoidance of persons of that sex. However, it is equally possible that the effect is causal in the reverse direction. Almost all studies of CSA categorise childhood as occurring up to age 16 or 18, well after the age at which same-sex attraction first appears in the majority of SSA individuals (see Figure 1). Abuse may be a violent reaction to a teenager's 'coming-out', or in response to gender-atypical behaviour or appearance (see below). Only two studies have used research designs that can assign a direction of causality, and neither is conclusive. One of these studies (n = 800) investigated historical criminal convictions for child neglect and abuse in children aged 11 or under (i.e. pre-pubertal) in a single US city, then measured adult same-sex behaviour in abused children thirty years later – they found that men sexually abused in childhood reported a higher number of lifetime same-sex partners, but no increase in the likelihood of same-sex cohabitation.[20]

Much more work is needed to understand the links between childhood sexual abuse and SSA, and the present data are ambiguous. It remains possible that abuse in childhood could be causally responsible for the development of SSA in a small number of individuals, but it is clear that childhood abuse is neither necessary nor sufficient for the development of SSA, as the majority of SSA individuals do not report any form of abuse.[21]

Socialisation

'Socialisation' refers to the hypothesis that pre-pubertal children are 'blank slates' with regard to sexual attraction, and that attraction is acquired or learned from socio-cultural cues and interactions with parents, siblings, peers, mentors, role models and the wider culture. This theoretical model assumes that SSA develops unconsciously and uncontrollably when an individual is exposed to examples of same-sex attraction and behaviour, presumably via a positive reinforcement mechanism. (It is worth reiterating that this article discusses the aetiology of same-sex attraction only, not behaviour. It is not disputed that sexual behaviour can be strongly

20 Wilson, H. & Widom, C. 'Does physical abuse, sexual abuse, or neglect in childhood increase the likelihood of same-sex sexual relationships and cohabitation? A prospective 30-year follow-up', *Archives of Sexual Behavior* (2010) 39(1), 63-74. An obvious limitation with this study is that only a percentage of abuse cases ever reach the court system, and these cases may well not be representative of all abuse cases involving homosexual individuals.

21 Andersen & Blosnich *op. cit.*, (19).

influenced by peer and parental attitudes, and by legal and moral codes. The socialisation aetiology posits that attraction as well as behaviour is influenced by these factors).

There is very little empirical evidence to support the socialisation hypothesis and the literature is small. A single study of SSA men with gay brothers found that 83% experienced SSA before becoming aware of their brother's orientation, suggesting that, in males at least, sexual attraction is not acquired from siblings.[22] A more recent study found that sexual attraction (though not behaviour) is not 'learned' from adolescent peers, and that same-sex attracted adolescents do not predominantly associate in peer groups with other SSA individuals.[23]

A slightly larger number of studies in the US and the UK have investigated whether the offspring of homosexual parents are more likely to be same-sex attracted due to parental imitation. Four small quantitative studies looking exclusively at lesbian-headed families have been carried out.[24] Because lesbian parenting is a relatively recent phenomenon, these studies have been confined to adolescents and young adults (15 – 23 years old). Overall, these studies have concluded that there are no reliable differences in sexual attraction or behaviour among offspring raised by lesbian mothers and offspring raised in mother-father households. However, daughters of lesbian-headed families appear to be significantly more open to possible future same-sex behaviour and more likely to self-identify as other than exclusively heterosexual. It is unknown how far these results are limited by the age of participants – sexuality is known to be particularly fluid during adolescence, making it hard to generalise these results to stable sexual attraction in later life. There is thus no evidence currently to suggest that same-sex attraction is causatively influenced by parental example.[25]

Outside of family and peer networks, no evidence exists about the role that broader cultural factors, like increasing visibility and viability of same-sex relationships in the media or professional sports, might play in the development of SSA. Most studies in this field do not investigate attraction but either behaviour or identity. In these contexts, it is difficult

22 Dawood, K., Pillard, R.C. et al. 'Familial aspects of male homosexuality', *Archives of Sexual Behavior* (2000) 29(2), 155-163.

23 Brakefield, T., Mednick, S. et al. 'Same-sex sexual attraction does not spread in adolescent social networks', *Archives of Sexual Behavior* (2014) 43(2), 335-344.

24 See Gartrell, N.K., Bos, H.M.W., & Goldberg, N.G. 'Adolescents of the US National Longitudinal Lesbian Family Study: Sexual orientation, sexual behavior, and sexual risk exposure', *Archives of Sexual Behavior* (2011) 40(6), 1199-1209.

25 For a discussion, see Kuvalanka, K.A. & Goldberg, A.E. '"Second generation" voices: Queer youth with lesbian/bisexual mothers', *Journal of Youth and Adolescence* (2009) 38(7), 904-919, and Stacey, J. & Biblarz, T.J. '(How) Does the sexual orientation of parents matter?', *American Sociological Review* (2001) 66(2), 159-183.

to distinguish the socialisation hypothesis from the effect of social change. The hypothesis requires that individuals are exposed to mainly positive examples of same-sex relationships in order to develop SSA, but a society where these examples are available is likely to be a safe environment to publicly identify and 'come out' as homosexual, increasing the number of people who will appear as SSA in surveys and studies. It cannot be argued that a positive correlation between the prevalence of SSA and a liberal society is evidence for a causal connection between the two.

In conclusion, the evidence that socialisation via siblings, peers, parents or culture is a cause of SSA is very weak, although there are some dissenting voices to this general conclusion.

Biological causes

Genetics – twin studies

The theory that SSA is caused by a variant gene[26] or variant genes has become a prevalent hypothesis in the past 20 years – 44% of respondents in one US survey mentioned genetics as a cause of SSA.[27] The 'gay gene' hypothesis crops up frequently in media and popular science articles, in song lyrics and at Gay Pride marches. At least two recently-published science fiction books take the existence of a 'gay gene' as a central premise.[28] Yet despite its pop-culture prominence, it is clear that there is no single gene for SSA. Children of homosexual parents do not inherit SSA in the strict Mendelian ratios we would expect if this trait were controlled by a single gene. We would expect identical twins to be always concordant (i.e. the same) for SSA if they were carriers of the same 'gay gene'; in reality, they are never 100% concordant.[29] A more plausible suggestion is that SSA is partially but not exclusively affected by multiple genetic variants.

Support for a genetic aetiology of SSA comes primarily from twin studies. Twin studies facilitate the separation of genetic and environmental factors potentially contributing to SSA by studying sets of twins who share either their genes or their environments to different degrees.[30] Such stud-

26 A gene that exists in two or more different forms in a population.

27 Sheldon, J.P., Pfeffer, C.A. et al. 'Beliefs about the etiology of homosexuality and about the ramifications of discovering its possible genetic origin', *Journal of Homosexuality* (2007) 52(3-4), 111-150.

28 Jude, J. *Gay Gene Rising*, PrideInspired.com (2011); Sones Feinberg, L. *The Gay Gene Discovery*, GLB Publishers (2008).

29 e.g., Alanko, K., Santtila, P. et al. 'Common genetic effects of gender atypical behavior in childhood and sexual orientation in adulthood: A study of Finnish twins', *Archives of Sexual Behavior* (2010) 39(1), 81-92.

30 For a simple discussion of the mathematics of the twin study method, see Plomin, R.D., DeFries, J.C., McClearn, G.E. & McGuffin, P. *Behavioral Genetics (5th edn.),* New York: Worth (2008).

ies are carried out by comparing the similarity between identical twins brought up together with the similarity between fraternal twins also raised together, because fraternal twins share on average only half of their variant genes, while identical twins share them all. This methodology allows quantification of the extent to which the variability in sexual attraction in a particular population (known as the variance) is correlated with the genetic variance that exists within that population; this value is known as the heritability value.[31] Critically, 'heritability' should not be confused with 'inheritance'. Inheritance refers to the transmission and expression of genes in individuals, and children inherit on average 50% of their genes from each parent. Heritability, by contrast, is a population statistic, which does not apply to individuals, and refers only to the proportion of variance in the population that can be ascribed to genetic variation in that population. It is incorrect to say that any individual's SSA is 40%, or 60%, or whatever per cent, caused by their genes. But heritability values do provide a useful way of assessing whether genetic factors are correlated with the variation in SSA at a population level.

Systematic twin studies on SSA and sexual orientation date from the early 1990s, and a substantial number have been carried out. An influential early pair of twin studies[32] found heritabilities of 40–70% for both men and women; these estimates were subsequently reported as 50% in substantial media coverage. However, these studies were potentially heavily affected by bias – they recruited volunteers through gay-friendly publications, making it more likely that concordant pairs of twins would sign up. Follow-up studies have therefore used population-based twin registries from Australia, Sweden, Finland, the US and the UK to generate systematic cohorts with typically several thousand participants. These studies have used a range of different measures of attraction, behaviour and identity, in male and female cohorts.

Overall, twin studies have found significant heritabilities for all the different facets of sexual orientation, including attraction, but with substantial differences in estimated heritability values between studies.[33] In both men and women, heritability ranges between around 15% and around 50%. Studies disagree on whether particular facets of sexual orientation are more heritable in men or in women. Heterogeneity of the measured

31 In brief, twin studies divide the total variance of a population into genetic, shared environmental, and unique environmental components. Components that contribute to the variance can be expressed as a percentage or a fraction. A heritability value of 1, or 100%, would mean that variation in a behaviour in a specified population is influenced entirely by genetic variation, as is the case for genetic diseases like Cystic Fibrosis and Huntington's Disease.

32 Bailey, J.M., Pillard, R.C., Neale, M.C. & Agyei, Y. 'Heritable factors influence sexual orientation in women', *Archives of Sexual Behavior* (1993) 50(3), 217-223; Bailey, J.M. & Pillard, R.C. 'A genetic study of male sexual orientation', *Archives of General Psychiatry* (1991) 48(12), 1089-1096.

33 See longer version of this article online for references.

variable is likely to be a key component in the poor reproducibility of these data (i.e. studies are measuring different constructs with different experimental methodologies). However, what can be emphasised in these studies is that environmental influences are the largest contributing factor to same-sex attraction and related constructs. So however these data are interpreted, variant genes are insufficient as a sole causal explanation of SSA.

The usefulness of data from these twin studies is somewhat limited by their very large confidence intervals, which often include 0% heritability as the lowest boundary.[34] Twin studies are also limited by their reliance on a set of assumptions which some theorists argue are invalid.[35] It is possible to obtain a significant heritability value if a trait is not influenced by genes at all, because the twin method assumes that identical and fraternal twins are equally exposed to their shared environment. Consequently, any non-genetic factor which causes identical twins to be more similar to each other than fraternal twins will yield a positive heritability value. For example, the fetal environment will be experienced more similarly by identical twins sharing the same placenta (about two-thirds of all identical twins) than by twins with individual placentas (one-third of identical twins and all fraternal twins). This has particular significance for hormonal theories of SSA, discussed below.

In conclusion, a straightforward reading of twin studies appears to support a genetic aetiology of same-sex attraction, yet the data are actually far from conclusive.[36] Heritability values are inconsistent among studies, with large confidence intervals, and may be spurious if the assumptions of the twin study method are invalid. Even if the reported significant heritability values are accurate, they still indicate a larger role for environmental factors than for genes in the aetiology of SSA (it is important to note that twin

34 95% confidence intervals are the range within which a result is 95% likely to be the true result. Thus, figures where the range includes 0 are suspect.

35 For example, twin zygosity is almost always assumed based on physical appearance, which is generally but not always a sufficient indicator. The assumption that identical twins share 100% of their DNA and non-identical twins share 50% is not always true for either MZ or DZ twins. In addition both identical and non-identical twins are epigenetically different at birth. For critical assessments of the methodology of heritability studies, see Visscher, P.M., Hill, W.G. & Wray, N.R. 'Heritability in the genomics era – concepts and misconceptions', *Nature Reviews Genetics* (2008) 9(4), 255-266; Richardson, K. & Norgate, S. 'The equal environments assumption of classical twin studies may not hold', *British Journal of Educational Psychology* (2005) 75, 339-350.

36 An important criticism of a genetic aetiology of SSA is that evolutionary theory predicts that genes causing SSA would rapidly be filtered out of a population by the mechanism of natural selection, as homosexual individuals typically have far fewer children (this argument does not consider the very recent rise in surrogacy and in vitro fertilisation options for same-sex couples, but concerns children born from heterosexual relationships conducted by SSA individuals). However, there are theoretical mechanisms by which genes predisposing individuals to same-sex attraction could be maintained in a population, notably kin selection and sexually antagonistic selection. For a discussion, see the longer version of this article online.

studies define any non-genetic but nonetheless biological factors, such as hormones, as 'environmental', so this finding does not rule out non-genetic biological aetiologies).

Genetics – specific genes

A small number of molecular genetic studies have investigated whether any specific genetic variants are associated with SSA; although no single genetic mutation is causally responsible for SSA by itself, multiple variants, potentially several hundreds or thousands, could be influencing SSA in combination. Research in this area has been contradictory, and beset by problems of reliability, replication and validity.

The first molecular genetic study on SSA was published by Dean Hamer and colleagues in 1993[37] – the paper attracted widespread press and public attention and can be credited with bringing the idea of a 'gay gene' into public discourse. Hamer et al. analysed 22 genetic markers on the X chromosome in 40 pairs of brothers, both of whom were same-sex attracted. Five markers in a region known as Xq28[38] were present in 33 of the pairs (83%), suggesting that a gene or genes in this region could be contributing to the brothers' shared SSA. The authors were careful to acknowledge that the study had several limitations, including a small sample size, a sample that was overwhelmingly white, and a very stringent definition of SSA. Furthermore, the region of interest identified is several million base pairs in size and contains over a hundred genes, any one of which could be the gene of interest. Hamer et al. were also careful not to discount potential environmental factors or to argue that the putative gene was causal in all instances of male SSA (seven of the pairs were discordant for at least one of the five markers). Thus, this first paper did not pinpoint any specific genetic variants contributing to SSA. Nevertheless, Xq28 rapidly came to be known in press reports as 'the gay gene',[39] despite the fact that it is a spatial signifier, not a gene name, and despite the fact that the study results were preliminary, unreplicated and far from conclusive. Hamer's subsequent book entitled *The Science of Desire: The search for the gay gene and the biology of behaviour*[40] only added to the confusion.

37 Hamer, D.H., Hu, S. et al. 'A linkage between DNA markers on the X- chromosome and male sexual orientation', *Science* (1993) 261(5119), 321-327.

38 Conventions of chromosomal mapping nomenclature can be found at http://www.nature.com/scitable/topicpage/chromosome-mapping-idiograms-302.

39 Kitzinger, J. 'Constructing and deconstructing the "gay gene": Media reporting of genetics, sexual diversity and "deviance"', in Ellison, G. & Goodman, A. *The Nature of Difference: Science, Society and Human Biology,* London: Taylor & Francis (2005), pp. 99-117.

40 Hamer, D.H. & Copeland, P. *The Science of Desire: The Search for the Gay Gene and the Biology of Behaviour,* New York: Simon & Schuster (1994).

A small number of studies have tried to replicate Hamer et al.'s original findings for the X chromosome, but these efforts have mainly ended in failure. Although the original research group managed to replicate their results in an independent cohort (n = 33 pairs), two other studies using the same methodology with larger samples failed to do so.[41] One recent linkage study (n = 409 pairs of brothers) has reported finding a nearly-significant association with male SSA and a marker in the Xq28 region.[42] In addition, pedigree studies, which measure SSA across families, have found inconsistent support for the hypothesis that male homosexuals in one family are related to each other through the maternal line, thereby sharing a genetic variant located on a shared X chromosome, as would be expected if one or more X-linked genes contributed to the trait.[43] The reason(s) for these various contradictory data are currently unknown.

Four studies have conducted genome-wide scans in order to locate genes related to sexual orientation on other chromosomes; however, none of these have produced any strongly significant or replicated findings. Mustanski et al. reported nearly-significant correlation between sexual orientation and the 7q36 region in a medium-sized study, but this result was not replicated by another group.[44] Sanders et al. reported a significant association between the 8q12 region and male SSA.[45] In addition, the personal DNA testing company 23andMe conducted a genome-wide association study (GWAS) using its customer database (n = 7887 men, 5570 women), but found no significant correlation between having a homosexual identity and any genetic marker.[46] However, such a null result is not surprising as a GWAS requires a very large sample size to produce significant results. In addition, three studies in male populations have looked at markers in

41 Mustanski, B.S., DuPree, M.G. et al. 'A genomewide scan of male sexual orientation', *Human Genetics* (2005) 116(4), 272-278; Rice, G., Anderson, C., Risch, N. & Ebers, G. 'Male homosexuality: Absence of linkage to microsatellite markers at Xq28', *Science* (1999) 284(5414), 665-667.

42 Sanders, A.R., Martin, E.R. et al. 'Genome-wide scan demonstrates significant linkage for male sexual orientation', *Psychological Medicine* (2014), available online at http://journals.cambridge.org/action/displayAbstract?fromPage=online&aid=9385646&fileId=S0033291714002451, (accessed 16th November 2014). Note that this result was not significant at a genome-wide level, although it is suggestive.

43 VanderLaan, D.P., Forrester, D.L., Petterson, L.J. & Vasey, P.L. 'The prevalence of fa'afafine relatives among Samoan gynephilic men and fa'afafine', *Archives of Sexual Behavior* (2013) 42(3), 353-359; Schwartz, G., Kim, R.M. et al. 'Biodemographic and physical correlates of sexual orientation in men', *Archives of Sexual Behavior* (2010) 39(1), 93-109.

44 Ramagopalan, S.V., Dyment, D.A. et al. 'A genome-wide scan of male sexual orientation', *Journal of Human Genetics* (2010) 55(2), 131-132; Mustanski et al. *op. cit*., (41).

45 Sanders et al. *op. cit*., (42).

46 Drabant, E.M., Kiefer, A.K. et al. 'Genome-wide association study of sexual orientation in a large, web-based cohort', *23andme* (2012). It is worth noting that these results have not been published in the peer-reviewed literature, and measured only self-identification of homosexuality, rather than SSA.

specific genes, known as a candidate gene study. Two studies[47] on genes involved in sexual differentiation, aromatase cytochrome P450 and the androgen receptor gene, yielded no significant results, but a recent study conducted in China found significant correlation between a variant of the sonic hedgehog (SHH) gene, which is involved in morphological patterning, and self-identified homosexuality.[48] However, these results have not as yet been replicated and are limited to a single population, and it would be premature to generalise these findings.

In conclusion, no single variant gene, or even genetic region, has been consistently associated with SSA in either men or women.

The fraternal birth order effect

As far back as the 1930s, it was hypothesised that SSA in males might be related to sibling number or to the order in which children are born. The few small studies to investigate this hypothesis during the twentieth century produced inconclusive results, but since the 1990s a Canadian research group has carried out a series of studies into the effect of birth order on sexual attraction in multiple cohorts. These have consistently found that younger brothers in a family of boys are significantly more likely to be SSA than their older brothers, independent of socioeconomic status, maternal age and overall family size.[49] The total number of siblings is not significant, nor is the effect seen in women; consequently, this phenomenon is termed the fraternal birth order (FBO) effect. These results have been replicated in independent cohorts.

The data are best explained by a theory known as the maternal immune hypothesis. It is known that the likelihood of an individual being SSA increases as the number of older brothers increases (i.e. third sons are more likely to be SSA than second sons). The increasing severity of the phenotype is analogous to an immune response following infection. This led researchers to suggest that male fetal cells carrying Y-chromosome encoded antigens (known as H-Y antigens) cross the placental barrier into the mother's bloodstream, provoking an immune response in the mother (because she carries no Y chromosome). In this theory, the antibodies to the Y-chromosome specific antigen(s) then pass back into the fetus and

47 DuPree, M.G., Mustanski, B.S. et al. 'A candidate gene study of CYP19 (Aromatase) and male sexual orientation', *Behavior Genetics* (2004) 34(3), 243- 250; Macke, J.P., Hu, N. et al. 'Sequence variation in the androgen receptor gene is not a common determinant of male sexual orientation', *American Journal of Human Genetics* (1993) 53(4), 844-852.

48 Wang, B., Zhou, S. et al. 'Association analysis between the Tag SNP for Sonic Hedgehog rs9333613 polymorphism and male sexual orientation', *Journal of Andrology* (2012) 33(5), 951-954.

49 For a review, see Bogaert, A.F. & Skorska, M. 'Sexual orientation, fraternal birth order, and the maternal immune hypothesis: A review', *Frontiers in Neuroendocrinology* (2011) 32(2), 247-254. See the longer version of this article online for more references.

affect either the developing fetal brain and/or the fetal genome and/or fetal epigenome. With each successive male pregnancy, the number and binding efficiency of maternal antibodies increases until it crosses a threshold sufficient to influence the development of SSA. Support for the maternal immune hypothesis comes from the fact that having older sisters (who would not provoke such an immune reaction in the mother in this scenario) does not increase the likelihood of SSA in younger brothers while, at the same time, miscarried male fetuses do increase the likelihood of SSA in subsequent male pregnancies, suggesting a prenatal effect.[50] Furthermore, a study of boys raised with adoptive siblings found that the number of non-biological older brothers had no effect on SSA rates, while the number of biological older brothers did, again suggesting the effect is prenatal.[51]

However, there are significant problems with this hypothesis. It has never been directly shown that male foetuses can provoke a relevant maternal immune response (although it is known that organs from male donors transplanted into female recipients can cause such a reaction). The mode of action of any putative maternal antibody is also unknown – the uncertainty surrounding other genetic, hormonal and neurological causal pathways to SSA makes elucidation of a presumed mechanism difficult. It is also not clear why the FBO effect is not a universal effect, as the vast majority of younger sons are opposite-sex attracted. It is possible that birth order acts as a risk factor in combination with another causal mechanism (environmental or biological), but how such interaction might work remains unknown.

It should be emphasised that the FBO effect is not sufficient to explain male SSA. Numerous studies have shown that some first-born males report being SSA. Theoretical attempts to calculate the percentage of the homosexual population who could attribute their SSA to the FBO effect have yielded estimates of between 15% and 30%, which is non-negligible, but also represents a minority of homosexuals. In conclusion, more data are needed, especially data relating to possible molecular mechanisms, before the maternal immune hypothesis can be accepted as valid.

Sex and gender atypicality

Humans are sexually dimorphic both physiologically (different sexes) and behaviourally (different genders). Since at least the 1860s it has been hypothesised that SSA arises as a result of deviation from the sex or gender developmental norm – in other words, that SSA males are feminised, and

50 Ellis, L. & Blanchard, R. 'Birth order, sibling sex ratio, and maternal miscarriages in homosexual and heterosexual men and women', *Personality and Individual Differences* (2001) 30(4), 543-552.

51 Bogaert, A.F. 'Biological versus nonbiological older brothers and men's sexual orientation', *PNAS* (2006) 103(28), 10771-10774.

SSA females are masculinised, and thus display a sexual behaviour at variance with their chromosomal sex. This 'intersex'[52] hypothesis is still widely referenced today by the general public; popular stereotypes in both Western and non-Western countries suggest that adult homosexuals are atypical in their physicality, mannerisms and social behaviour, as well as in their sexual behaviour.

Evidence that homosexual men and women are physically sex-atypical has been limited and contradictory. On the one hand, there is no suggestion that any more than a tiny minority of SSA adults are genitally atypical, and no consistent evidence that they differ in height, weight or other physical attributes. On the other hand, a US research team has demonstrated in several culturally diverse groups that an individual's sexual orientation can be accurately assessed at a rate above chance based purely on their physical appearance, speech and movement displayed in short (10s) video clips.[53] This assessment of sexual orientation was partially mediated by a judgement of atypicality (i.e. more sex-atypical persons were judged to be homosexual, often correctly), suggesting that some physical differences may exist between SSA and OSA individuals. However, the authors were careful to point out that the prediction rate was not 100%, and sex atypicality is not a necessary trait associated with same-sex attraction.

On the face of it, much stronger support for the intersex hypothesis comes from observations that SSA individuals tend to be gender-atypical in their behaviour. Studies by Richard Lippa and others[54] have argued that adult homosexuals, both male and female, are more likely to be gender-atypical for a range of personality traits. They are also more likely to be judged by others as more gender-atypical in their behaviour, and to self-identify as more gender-atypical on a variety of measures of masculinity-femininity.[55] The link is even stronger in childhood; numerous studies since the 1960s have demonstrated a robust correlation between gender-atypical behaviour in pre-pubertal childhood and higher likelihood of adult SSA.[56] Indeed, this is one of the strongest findings in the field

52 The term 'intersex' is used very broadly in the literature to describe a range of conditions where elements of male and female are present in one individual, including disorders of sex development like hermaphroditism or chromosomal aneuploides, and behavioural conditions such as Gender Identity Disorder.

53 Rieger, G., Linsenmeier, J.W. et al. 'Dissecting 'Gaydar': Accuracy and the role of masculinity-femininity', *Archives of Sexual Behavior* (2010) 39(1), 124-140.

54 e.g., Lippa, R.A. 'Sex differences and sexual orientation differences in personality: Findings from the BBC Internet Survey', *Archives of Sexual Behavior* (2008) 37(1), 173-187.

55 Although an Italian study reported that male homosexuals were more likely to view themselves as androgynous than as feminine. Zoccali, R., Muscatello, M.R. et al. 'Gender role identity in a sample of Italian male homosexuals', *Journal of Homosexuality* (2008) 55(2), 265-273.

56 For a review, see Zucker, K.J., Mitchell, J. et al. 'The recalled childhood gender identity/gender role questionnaire: Psychometric properties', *Sex Roles* (2006) 54(7-8), 469-483.

of sexual orientation research, with no contrary findings reported in the past two decades for either male or female cohorts. The majority of studies have been retrospective designs, which are often thought to be subject to recall bias – it is suggested that adult homosexuals may falsely remember higher levels of gender-atypicality given their later knowledge of their same-sex sexual attraction. However, prospective and non-recall studies have produced similar positive findings.[57]

Nevertheless, these behavioural data need to be interpreted cautiously. In adults, correlations between sexual attraction status and gender atypicality are generally only weakly significant. The frequent use of small non-typical clinical populations, particularly for children and adolescents, weakens the generalisability of the findings. The link between the most extreme form of gender atypicality, gender dysphoria, and SSA is still unclear; it might be expected that gender dysphoria would always be accompanied by same-sex attraction (in terms of the chromosomal sex of the individual), but cases of opposite-sex attracted gender-dysphoric individuals have been documented. Moreover, unlike physical differences, gender presentation is bound up with cultural norms, peer pressures and unconscious social expectations and interactions, making it difficult to draw a direct connection between gender atypicality and a general causative biological difference between SSA and OSA individuals. For example, observational studies in several non-Western cultures have described communities of SSA males who are highly feminised in appearance and behaviour; it has been suggested that in these cultures, atypical sexual behaviour is seen as non-deviant and tolerated only if accompanied by other gender-atypical forms of behaviour, forcing SSA individuals to adopt atypical gender presentations.

Overall, although the evidence is somewhat inconclusive and needs further study to tease out potentially confounding factors, it appears that sexual attraction and sex/gender atypicality are correlated in at least a substantial cohort of SSA individuals, and thus it is plausible that atypical masculinisation or feminisation is causal for same-sex attraction in some individuals. In principle, the underlying cause of this correlation could be either psychosocial or biological, or indeed both. The psychosocial explanation is lacking in empirical support and is not further discussed here. Biological causation has been primarily investigated in two interrelated areas: sex hormones and neurology, as discussed below. As with other aetiologies, causal models based on gender atypicality are neither necessary nor sufficient to explain all SSA, as many SSA adults are gender-typical throughout their lives, and the majority of gender-atypical children go on to develop a heterosexual orientation.

57 e.g., Steensma, T.D., van der Ende, J., Verhulst, F.C. & Cohen-Kettenis, P.T. 'Gender variance in childhood and sexual orientation in adulthood: A prospective study', *Journal of Sexual Medicine* (2013) 10(11), 2723-2733.

Sex/gender atypicality – hormones

The development of primary and secondary sexual characteristics is controlled by hormones known as androgens, such as testosterone, dihydrotestosterone and androstenedione, which are produced by the adrenal gland and the sex organs. Both men and women produce androgens, but men typically have a much higher androgen concentration, which is ultimately caused by the presence of the SRY gene located on the Y chromosome. In the vast majority of adults, androgen concentration does not differ between adults who are OSA or SSA. SSA males are not testosterone-deficient, nor are SSA females testosterone-enhanced. It is therefore not possible to 'treat' SSA with hormones in adulthood, even if it were ethical, and historical uses of hormone treatments were shown to be ineffective.

It is frequently hypothesised that exposure to atypical concentrations of androgens in the womb during fetal development is causal for both atypical sex and gender characteristics and same-sex attraction in childhood and adulthood. However, attempts to demonstrate a correlation between fetal hormone concentration and any form of sex/gender atypicality have been hampered by difficulties of measurement.[58] It is impractical to directly assess fetal blood hormone concentrations routinely (fetal blood-sampling is high-risk and invasive), so a variety of proxy methods have been used to measure fetal androgen exposure, some more valid and reliable than others. These include direct measures of androgen concentration in amniotic fluid and maternal blood serum, and using the 2D:4D finger ratio (the ratio between the length of the second and fourth finger, a sexually dimorphic trait that appears by the end of the first trimester). These studies have been marked by inconsistency. While a sizeable number report significant correlations between estimated androgen concentration and measures of SSA and other forms of gender atypicality, an equal number report no differences between SSA and OSA individuals, or even findings in the inverse direction.[59]

The most useful data on fetal hormone concentration have been derived from 'natural experiment' studies – rare disease conditions in which androgen concentration is altered by mutations in androgenic hormone or hormone receptor genes. Women with Congenital Adrenal Hyperplasia (CAH), approximately 1 in 5000 – 15,000 live births, have deficiencies in cortisol production which causes androgen concentration to be significantly higher than typical for females. Around a dozen studies across multiple age ranges have found that women with CAH are significantly more likely

58 For comprehensive reviews of this literature, see Berenbaum, S.A. & Beltz, A.M. 'Sexual differentiation of human behavior: Effects of prenatal and pubertal organizational hormones', *Frontiers in Neuroendocrinology* (2011) 32(2), 183-200, and Hines, M. 'Prenatal endocrine influences on sexual orientation and on sexually differentiated childhood behavior', *Frontiers in Neuroendocrinology* (2011) 32(2), 170-182.

59 For an expanded discussion and references, see the longer version of this article online.

to report experiencing same-sex attraction or fantasy than matched controls.[60] The rate of lifetime SSA is much higher in CAH women, between 15% and 40%, compared to the heterosexual control rate of around 10%; although at the same time this means that well over half of all CAH-affected women are OSA. In addition, more severe forms of CAH (which raise androgen concentration higher) are associated with higher rates of SSA than less severe forms. This finding is highly reliable, although almost all these studies have been conducted in Caucasian populations and may not be generalisable. CAH is also associated with atypicality in gendered behaviours in both childhood and adulthood, including toy choice, drawing, interest in rough sports and occupational preferences.

The inverse condition to CAH is Androgen Insensitivity Syndrome (AIS). Complete AIS occurs when the androgen receptor gene is mutated, meaning that although androgen levels are high, they cannot be detected by the body. Various other mutations also lead to reduced ability to detect androgens, causing partial or mild AIS. In complete AIS, male genitalia and secondary sexual characteristics fail to develop, and XY individuals are raised as females, with diagnosis not occurring until puberty. Only two studies of AIS and sexual orientation in adulthood have been performed; both reported that the vast majority of AIS women are attracted to men, meaning that in terms of their chromosomal sex they are same-sex attracted.

Although they seemingly provide the strongest evidence that hormone concentration is correlated with sexual attraction status, particularly in women, natural experiment studies of this kind are limited by the fact that it is very hard to disentangle hormonal effects from social effects on gender presentation and sexual behaviour in CAH- and AIS-affected populations. CAH women are physiologically distinct from other women, being on average shorter and heavier, and usually have partially formed male genital tissues that are corrected with surgery and hormone treatment at birth. CAH women thus look 'unfeminine', which could prompt feelings of greater masculinity, as could social expectations that CAH women are 'butch'. Furthermore, CAH women tend to find heterosexual sex difficult or unpleasant due to the effects of surgery, which could increase homosexual fantasy, the most commonly measured variable in CAH studies. AIS individuals are even harder to assess. Although their chromosomal sex is male, complete AIS sufferers are phenotypically indistinguishable from XX women, have female genitalia, and are raised with societal expec-

60 e.g., Frisen, L., Nordenstrom, A. et al. 'Gender role behavior, sexuality, and psychosocial adaptation in women with congenital adrenal hyperplasia due to CYP21A2 deficiency', *Journal of Clinical Endocrinology and Metabolism* (2009) 94(9), 3432-3439; Meyer-Bahlburg, H.L., Dolezal, C., Baker, S. & New, M. 'Sexual orientation in women with classical or nonclassical congenital adrenal hyperplasia as a function of degree of prenatal androgen excess', *Archives of Sexual Behavior* (2008) 37(1), 85-99.

tations of women. Labelling these women as same-sex attracted when they do not think of themselves as male is problematic.

Overall, the evidence that atypical prenatal hormone exposure underlies either SSA or gender-atypical behaviour is limited by the difficulty of assessing fetal androgen concentration. Results using proxy measures are unreliable and inconsistent, and rare disease conditions, whilst seeming to support the hypothesis, are not clear-cut due to possible social effects. It is important to emphasise as well that all these studies are purely correlational, not mechanistic; the way(s) in which androgenic hormones potentially affect the development of sexual characteristics and behaviours, presumably via a combination of genetic, epigenetic and/or neurological effects, are still unclear. It is thus difficult to draw firm conclusions from these data, although it does seem that any hormonal effect on SSA is more readily observable in women than in men.

Sex/gender atypicality – neurology

The human brain is sexually dimorphic in structure, functionality and disease susceptibility. It has been hypothesised since the early 1990s that SSA individuals may have brains that are sex-atypical, leading to attraction to the same sex, and a fairly small number of studies have investigated structural and functional differences, primarily in men.

Beginning in 1990, three now infamous studies using the technique of post-mortem brain dissection announced that homosexual men had significantly different volume or area measurement (compared to heterosexual men) in three specific regions of the brain: the anterior commissure, suprachiasmatic nucleus and a hypothalamic area known as INAH3.[61] In two of these studies, the homosexual men were shown to be comparable to a control group of heterosexual women, that is, they were female-typical. However, these findings have not stood up over time, as they had a significant number of limitations. All three studies used homosexual probands who had died of AIDS. Although attempts were made to control for possible impacts of the disease on brain structure, not enough attention was paid to the possible effects of pharmaceutical treatments or lifestyle factors like drug use. Same-sex attraction was not verified with the probands directly, but taken from hospital records. Studies that have attempted to replicate these results have failed to find any significant differences in these brain regions between SSA and OSA men.[62]

61 Allen, L.S. & Gorski, R.A. 'Sexual orientation and the size of the anterior commissure in the human brain', *PNAS* (1992) 89(15), 7199-7202; LeVay, S. 'A difference in hypothalamic structure between heterosexual and homosexual men', *Science* (1991) 253(5023), 1034-1037; Swaab, D.F. & Hofman, M.A. 'An enlarged suprachiasmatic nucleus in homosexual men', *Brain Research* (1990) 537(1-2), 141- 148.

62 e.g., Byne, W., Tobet, S. et al. 'The interstitial nuclei of the human anterior hypothala-

More recently, magnetic resonance imaging (MRI) has been used to map structure in living brain tissue in two studies. One study reported a significant difference in the volume of the isthmus region of the corpus callosum between homo- and heterosexual males.[63] The other study measured grey matter (GM) density in heterosexual and homosexual men and women, and found only one strongly significant difference: homosexual women had reduced (male-typical) GM density in the left perirhinal cortex.[64] This area is associated with olfactory processing, leading the authors to speculate that olfactory cues may be relevant to the development of SSA in women, but this remains speculative. These results have yet to be replicated and should be treated as preliminary.

In conclusion, the neural mechanisms involved in sexual attraction remain very uncertain, and it is incorrect to say that same-sex attracted men have female brains, and vice versa. No consistent and reliable differences have been found that distinguish SSA and OSA individuals. Neurological studies are limited by small sample sizes, lack of replication and inconsistency in how sexual attraction is measured. The regions of the brain involved in sexual attraction are not clearly defined (in contrast to traits like speech and visual acuity), so any work involves some degree of informed guesswork. The ultimate causes of sexual differentiation of the brain also require further elucidation; both genes and hormones are known to influence neural development, but (as discussed above) no specific genes have been associated with SSA that might work via altering neural development, and the relationship between androgenic hormones and SSA is still unclear.

A further crucial problem with all neurological studies is that they cannot distinguish between innate and learned effects. Because the brain is plastic and new neural circuits are continuously forming in response to experience, it is not unreasonable to suggest that the experience of feeling SSA, practising same-sex behaviour and associating with different groups of peers will have a neurological impact. Therefore, it is impossible to conclude that such differences are causal for SSA, as the exact reverse might be the case.

Conclusion

As reviewed here, there is a wide variety of hypothesised causes for same-sex attraction. Some causal models have received more empirical support

mus: An investigation of variation with sex, sexual orientation, and HIV status', *Hormones and Behavior* (2001) 40(2), 86-92.

63 Witelson, S.F., Kigar, D.L. et al. 'Corpus callosum anatomy in right-handed homosexual and heterosexual men', *Archives of Sexual Behavior* (2008) 37(6), 857-863.

64 Ponseti, J., Siebner, H.R. et al. 'Homosexual women have less grey matter in perirhinal cortex than heterosexual women', *PloS One* (2007) 2(8), e762.

than others, but no cause has yet gained sufficient support to provide a compelling explanation. In men, genetic influences on same-sex attraction cannot be ruled out, although any genetic variants that do influence SSA will be of small effect, and there is no single 'gay gene'. The fraternal birth order effect is well supported, but there is no firm evidence for an immunological explanation for this observation. In women, almost the only positive evidence comes from hormonal studies, suggesting that exposure to elevated androgen levels can lead to developmental changes causing an attraction to women, but these data need to be treated with caution. Certain causal pathways, such as those resulting from childhood abuse, may be significant for a small percentage of the SSA cohort. There is no positive evidence that same-sex attraction in either men or women is caused by socialisation effects, by personal choice, by poor parenting, or by having a brain of the 'wrong sex'.

Our primary conclusion is that no one causal mechanism is both necessary and sufficient to explain the whole gamut of human sexual attraction. Sexual attraction is a highly complex trait, and it seems likely that across the variety of human sexes and cultures, different influences are more important at different times. Not all homosexual men will be carrying the same variant genes. Not all homosexual women are masculinised. The social and cultural environment in which people live is constantly changing, including their friends and partners, together with their own motivations and aspirations, creating a complex system in which biological make-up is integrated with multiple environmental, social and cultural factors. Thus, there is no point in looking for *the* cause of same-sex attraction – it does not exist. This negative conclusion is important, because both Christians and others sometimes assume that the aetiology of SSA is known and straightforward. It is not.

Nell Whiteway is Research Associate and Denis Alexander is Emeritus Director of The Faraday Institute for Science and Religion, St Edmund's College, Cambridge, CB3 0BN, UK.