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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.

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 current best scientific understanding[1]. Currently, there is a great diversity in understanding about the causes of SSA, across Christian denominations[2], and these interpretations have frequently been an important factor in determining a theological position on homosexuality (in parallel with or complementary to scriptural, pastoral and doctrinal reasoning). Thus, 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, biological, 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

[1] 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 7 November 2014).

[2] For more information, many denominations list short summaries of their theological position on homosexuality on their websites.

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[3].

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[4]. 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[5], 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). Alternatively, researchers use reduced three-point or five-point scales. These reduced or collapsed scales can obscure

[3] To support this point, the following four books recount the personal experiences of Christians who experience same-sex attraction across different cultural contexts. We do not endorse all the statements made by these authors, but cite their books to highlight the broad heterogeneity in the experiences of different Christians. Lee, J. *Torn: Rescuing the Gospel from the Gays-v-Christians Debate,* Jericho Books (2012); Butterfield, R.C. *The Secret Thoughts of an Unlikely Convert,* Pittsburgh: Crown and Covenant Publications (2012); Hill, W. *Washed and Waiting – Reflections on Christian Faithfulness and Homosexuality*, Grand Rapids, MA: Zondervan (2010); Keane, C. (ed.) *What Some of You Were,* Matthias Media (2001).

[4] See 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, for a discussion of difficulties and complexity in measuring sexual attraction and its related constructs (freely available online).

[5] Developed by Alfred Kinsey in the 1940s, in Kinsey, A.C., Pomeroy, W.B., & Martin, C.E. *Sexual Behavior in the Human Male*, Bloomington: Indiana University Press (1948). For a detailed discussion of the merits and problems of using Kinsey scales, see Vrangalova, Z. & Savin-Williams, R.C. 'Mostly heterosexual and mostly gay/lesbian: Evidence for new sexual orientation identities', *Archives of Sexual Behavior* (2012) 41(1), 85-101, and Sell, R.L. 'Defining and measuring sexual orientation: A review', *Archives of Sexual Behavior* (1997) 26(6), 643-658.

the true complexities of sexual attraction status, and contribute to uncertainty of measurement.

Accurately measuring same-sex attraction, either demographically or 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, relationship status, romantic attraction, sexual lifestyle and self-identity. All these facets together contribute to an individual's sexual orientation (although the lay public's definitions of sexual orientation are generally not so inclusive[6], and there is no one agreed definition). These facets do not always, or even frequently, exactly correlate, but interact in multiple ways in different individuals[7]. 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[8]. On average, nearly three times as many people report some degree of sexual attraction than the number of people who self-identify as gay, lesbian or bisexual[9]. 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 self-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 they are presented (written, telephone or face-to-face), how questions are phrased[10], the number of possible options offered (3-point, 5-point or 7-point scales, as discussed above), the degree or frequency of attraction deemed sufficient to categorise as SSA, and the degree of anonymity (poorly-anonymised surveys are likely to produce a bias towards greater opposite-sex attraction due to social stigmatisation). As with any survey about sexuality, the data are affected by the extent to which respondents answer accurately. All these factors mean that different demographic surveys of the prevalence of same-sex attraction, same-sex behaviour and non-heterosexual identities produce significantly differing data[11].

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[6] See Savin-Williams *op. cit*., (4).

[7] Savin-Williams, R.C. 'Who's gay? Does it matter?', *Current Directions in Psychological Science* (2006) 15(1), 40-44.

[8] For an example, see the participants in Chandra, A., Mosher, W.D. & Copen, C. *Sexual behavior, sexual attraction, and sexual identity in the United States: Data from the 2006-2008 National Survey of Family Growth*, National Health Statistics Reports: National Center for Health Statistics (2011), where 9% of self-identified heterosexuals had experienced same-sex behaviour, and 4.6% of individuals who reported exclusive OSA had experienced same-sex behaviour.

[9] See Savin-Williams *op. cit*., (4). See also Gates, G.R. 'How many people are lesbian, gay, bisexual, and transgender?', Los Angeles: The Williams Institute (2011).

[10] For an example of how variable question-wording can produce significantly different answers in a matched population, see Hayes, J., Chakraborty, A. et al. 'Prevalence of same-sex behavior and orientation in England: Results from a national survey', *Archives of Sexual Behavior* (2012) 41(3), 631-639.

[11] See Gates, G.R. 'LGBT Demographics: Comparisons among population-based surveys', Los Angeles: The Williams Institute (2014), and Gates *op. cit*., (9), for an in-depth discussion of design differences between global survey instruments.

The time period assessed (lifetime SSA v. current or recent SSA) is critical, as sexual attraction can be a dynamic trait. A small number of long-running (6 - 17 years) longitudinal surveys in the US and New Zealand[12] 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. The greatest stability is among those who report exclusive attraction status (either exclusively OSA or exclusively SSA). Changes occur in both directions (ie. 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. However, it should be emphasised that a change in sexual attraction status as reported in these studies does not necessarily mean a change from exclusive OSA to exclusive SSA – a change from primarily SSA to exclusively SSA is counted as dynamic. 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 individuals mentioned above should be kept in mind when considering this literature[13].

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[12] Longitudinal surveys have been conducted in both adolescent and adult populations enrolled in large-scale health monitoring and development studies such as the National Survey of Mid-Life Development in the United States (MIDUS), the AddHealth cohort and the Dunedin Multidisciplinary Health and Development Study. n = approx. 1000 - 14,000. Adolescents and young adults: Savin-Williams, R.C., Joyner, K. & Rieger, G. 'Prevalence and stability of self-reported sexual orientation identity during young adulthood', *Archives of Sexual Behavior* (2012) 41(1), 103-110; 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; Savin-Williams, R.C. & Ream, G. 'Prevalence and stability of sexual orientation components during adolescence and young adulthood', *Archives of Sexual Behavior* (2007) 36(3), 385-394. Mid-aged adults: Dickson, N., van Roode, T., Cameron, C. & Paul, C. 'Stability and change in same-sex attraction, experience, and identity by sex and age in a New Zealand birth cohort', *Archives of Sexual Behavior* (2013) 42(5), 753-763; 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.

[13] If you are interested in this area, the review paper by Serovich, S.M., Craft, P., et al. 'A systematic review of the research base on sexual reorientation therapies', *Journal of Marital and Family Therapy* (2008) 34(2), 227-238, is a good place to start. Comprehensive references can be obtained from the American Psychological Association. 'Report of the American Psychological Association's Taskforce on Appropriate Therapeutic Responses to Sexual Orientation', Washington, D.C: American Psychological Association (2009). Also relevant are Beckstead, A.L. 'Can we change sexual orientation?', *Archives of Sexual Behavior* (2012) 41(1), 121-134, and Jones, S.L. & Yarhouse, M.A. 'A longitudinal study of attempted religiously mediated sexual orientation change', *Journal of Sex and Marital Therapy* (2011) 37(5), 404-427. Also of interest is Spitzer, R.L. 'Can some gay men and lesbians change their sexual orientation? 200 participants reporting a change from homosexual to heterosexual orientation', *Archives of Sexual Behavior* (2003) 32(5), 403-417, but note that this paper was formally retracted by its author in

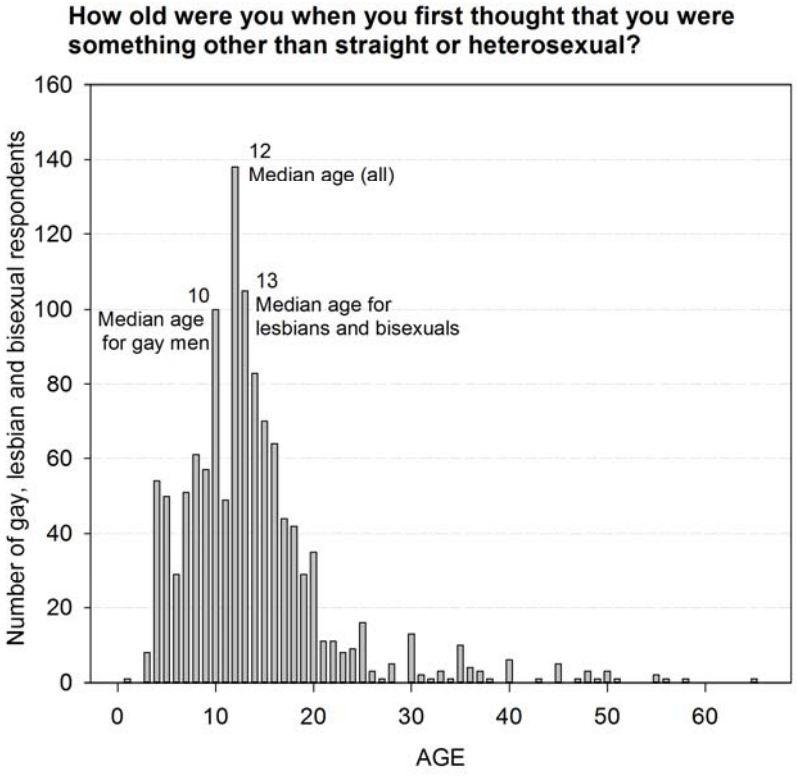

Figure 1. The development of same-sex attraction in a US demographic sample. The majority of SSA individuals develop stable same-sex attractions in early puberty, with a median age of 10 for men and 12 for women. A minority first identify their same-sex attraction much later in life. Adapted from Pew Research Center (with permission).[14]

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[15]. 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[16]). The prevalence of having exclusive same-sex attraction or behaviour is much lower than the prevalence of having any degree of same-sex attraction or behaviour.

2012 with serious concerns over its conclusions and validity. Spitzer, R.L. 'Spitzer reassesses his 2003 study of reparative therapy of homosexuality', *Archives of Sexual Behavior* (2012) 41(4), 757.

[14] Pew Research Center 'A survey of LGBT Americans: Attitudes, Experiences and Values in Changing Times', Washington, DC: Pew Research Center (2013).

[15] Main source: Savin-Williams *op. cit*., (4). For the UK, see also Hayes *op. cit*., (10). Recent US demographic data can be found in Gates *op. cit*., (9).

[16] See Gates *op. cit*., (11).

	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).[17]

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 philosopher Arthur Schopenhauer articulated this argument as far back as 1839, in a phrase usually translated as 'Man can do what he wills, but he cannot will what he wills'[18], and the principle has remained substantially unchanged since then. Under this model, sexual attraction is a trait to be discovered within oneself, not formed. This 'standard model'[19], so named because it is the model most commonly held by biologists, sociologists and the general public, underlies many survey instruments of sexual attraction[20] and popular writing about homosexuality[21]. It is not disputed by these theorists that individuals can make conscious choices about their sexual behaviour and 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[22], and it has been suggested by a number of groups over the years 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 hypothesis 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

[17] Savin-Williams *op. cit*., (4).

[18] Schopenhauer, A. 'Über die Freiheit des menschlichen Willens (On the Freedom of the Will)', an essay presented to the Royal Norwegian Society of Sciences in 1839.

[19] See Wilkerson, W.S. 'Is it a choice? Sexual orientation as interpretation', *Journal of Social Philosophy* (2009) 40(1), 97-116.

[20] See Savin-Williams *op. cit*., (4).

[21] For an example, see 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/, 7 November 2014).

[22] See Wilkerson *op. cit*., (19), for a theoretical discussion of what constitutes choice, interpretation and discovery in sexual attraction.

choose to be attracted only to other women, as a rejection of heteronormativity and patriarchal oppression[23]. 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[24]. 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[25]. This narrative has been extremely influential in the US – for the past 30 years between 30% and 40% of respondents to American surveys have cited a choice aetiology when asked about the causes of homosexuality[26]. However, it is worth noting that there is often a political dimension to what the lay public believe the causes of same-sex attraction are, particularly in the US, as the answer can have profound implications for legal policy and social practices[27]. It is also possible that individuals who

[23] Ellis, S.J. & Peel, E. 'Lesbian feminisms: Historical and present possibilities', *Feminism & Psychology* (2011) 21(2), 198-204.

[24] 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, 7 November 2014); Madwin, G. 'Myths about queer by choice people', (1999), http://www.queerbychoice.com/myths.html (accessed 10 November 2014); Whisman, V. *Queer by Choice: Lesbians, Gay Men, and the Politics of Identity,* New York: Routledge (1996).

[25] The Christian Right is an informal coalition of socially conservative Christian denominations and institutions such as Focus on the Family, Moral Majority and the Family Research Council, that first gained a substantial media presence in the conservative revival of the 1980s. See Burack, C. & Josephson, J. 'Origin stories: Same-sex sexuality and Christian Right politics', *Culture and Religion* (2005) 6(3), 369-392; Wilcox, S.A. 'Cultural context and the conventions of science journalism: Drama and contradiction in media coverage of biological ideas about sexuality', *Critical Studies in Media Communication* (2003) 20(3), 225-247.

[26] Data taken from Lewis, G.B. 'Does believing homosexuality is innate increase support for gay rights?', *Policy Studies Journal* (2009) 37(4), 669-693. This paper analysed poll results from 1983 to 2006, which asked the question 'In your opinion, what causes homosexuality? Is it something that people are born with … or is it something that develops because of the way people are brought up … or is it just the way some people prefer to live?'. It should be noted that these data only go up to 2006, and it is possible that views have shifted substantially since that year, as the increased media focus on same-sex marriages has brought the issue to the forefront of public attention. Data from Gallup's annual Values and Beliefs poll show that views on causes of same-sex attraction are continuing to shift in recent years, although this question does not ask about choice in precise terms. See Gallup 'More American see Gay, Lesbian Orientation as Birth Factor', Gallup.com (2013). Available at http://www.gallup.com/poll/162569/americans-gay-lesbian-orientation-birth-factor.aspx (accessed 10 November 2014).

[27] The 'lifestyle choice' narrative has frequently been used to argue against the push by gay rights' activists in the US for legal equality in employment, adoption rights and marriage law, because under US constitutional law it is permissible to exclude defined groups from particular civil and legal rights only if membership of the group is voluntary. A belief that homosexuality is a lifestyle choice has been found to be strongly correlated with opposition to such legal rights for gay individuals. See Smith, S.J., Zanotti, D.C., Axelton, A.M. & Saucier, D.A. 'Individuals' beliefs about the etiology of same-sex sexual orientation', *Journal of Homosexuality* (2011) 58(8), 1110-1131; Haider-Markel, D.P. & Joslyn, M.R. 'Beliefs about the origins of homosexuality and support for gay rights – An empirical test of attribution theory', *Public Opinion Quarterly* (2008) 72(2), 291-310; Wood, P.B. & Bartkowski, J.P. 'Attribution style and public policy attitudes toward gay rights', *Social Science Quarterly* (2004) 85(1), 58-74.

support a choice aetiology are eliding attraction and behaviour[28] – as noted above it is not disputed that sexual behaviour is within a person's conscious control.

There is little empirical evidence that directly addresses this hypothesis, leading to often intense media debates, 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[29]. 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. In earlier decades, following Freud, homosexuality had been widely thought of within the psychological community as an 'immature phase' of sexual development that everyone briefly experienced at some point[30]. However, 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[31]. 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 male role model caused the son to develop a phobia of having sexual interactions with women[32]. The triangular system was entirely theoretical when Rado first proposed it, but a small flurry of studies during the 1950s and 1960s reported finding this type of 'triangular' relationship in homosexual populations, most famously in Irving

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[28] For examples of elision between attraction and behaviour, see some of the responses to a survey conducted in the US in 2007. 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.

[29] e.g., Dahir, M. 'Why are we gay?', *The Advocate,* 17 July 2001, pp. 30-42. See also the books by Christian authors listed in reference (3).

[30] Friedman, R.M. 'The psychoanalytic model of male homosexuality: a historical and theoretical critique', *Psychoanalytic Review* (1986) 73(4), 483-519.

[31] Rado, S. 'A critical examination of the concept of bisexuality', *Psychosomatic Medicine* (1940) 11(4), 459-467.

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

Beiber and colleagues' 1962 study of a population of institutionalised homosexuals in New York[33]. Beiber et al. found that around 60% of the men in their study reported having difficult and unsatisfactory relationships with their fathers (although this left 40% of the sample who did not report any problem with their parental relationships). Other studies also reported that homosexual men were more likely to be emotionally detached from their fathers. 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'[34].

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[35]. 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[36] and from cross-cultural studies[37] which suggested that homosexual behaviour was much more widespread and common than had been supposed[38], and that the majority of SSA individuals were psychologically normal and socially well-adjusted.

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[33] Beiber, I., Dain, H.J., et al. *Homosexuality: a psychoanalytic study,* New York: Basic Books (1962). See also Socarides, C.W. *The Overt Homosexual,* New York: Grune and Stratton (1968). For a review of psychoanalytic studies of the 1950s and 1960s see LaSala, M. 'Out of the darkness: Three waves of family research and the emergence of family therapy for lesbian and gay people', *Clinical Social Work Journal* (2013) 41(3), 267-276.

[34] Drescher *op. cit*., (32).

[35] Hooker, E. 'The adjustment of the male overt homosexual', *Journal of Projective Techniques* (1957) 21(1), 18-31. For a retrospective examination of the social and professional impact of this study, see Hooker, E. 'Reflections of a 40-year exploration. A scientific view on homosexuality', *The American Psychologist* (1993) 48(4), 450-453.

[36] Kinsey, A.C., Pomeroy, W.B. & Martin, C.E. *Sexual Behavior in the Human Female*, Bloomington: Indiana University Press (1953); Kinsey *op. cit*., (5).

[37] Ford, C.S. & Beach, F.A. *Patterns of Sexual Behavior,* New York: Harper and Brothers (1951).

[38] Kinsey's original study of male homosexuality suggested that 10% of American males (n = approx. 12,000) 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 somewhat inflated by sampling bias, but the 10% statistic had a big social impact at the time.

Beginning in 1966, attempts to replicate the results of Beiber et al. and similar studies in larger non-clinical and non-institutional populations almost all ended in failure. Only one study supported the triangular system in its entirety[39], while many others found small or no differences in family relationships between OSA and SSA men[40]. 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[41]. In a 1974 survey, Siegelman found that SSA men were more likely to have more emotionally hostile and rejecting relationships with their fathers only if they also scored highly for neuroticism[42], 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[43]. It was also removed from the directories of 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

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[39] Evans, R.B. 'Childhood parental relationships of homosexual men', *Journal of Consulting and Clinical Psychology* (1969) 33(2), 129-135.

[40] e.g., Robinson, B.E., Skeen, P., Hobson, C.F. & Herrman, M. 'Gay men's and women's perceptions of early family life and their relationships with parents', *Family Relations* (1982) 31(1), 79-83; Siegelman, M. 'Parental background of homosexual and heterosexual men: A cross-national replication', *Archives of Sexual Behavior* (1981) 10(6), 505-513; Hooker, E. 'Parental relations and male homosexuality in patient and nonpatient samples', *Journal of Consulting and Clinical Psychology* (1969) 33(2), 140-142; Greenblatt, D. 'Semantic differential analysis of the "Triangular System" hypothesis in "adjusted" male homosexuals', Los Angeles: University of California Press (1966). Other studies of the period are reviewed in Friedman *op. cit*., (30).

[41] 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.

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

[43] For a history of how the APA came to its 1973 decision, and the various political and scientific factors that played into it, see Morgan, K.S. & Nerison, R.M. 'Homosexuality and psychopolitics: an historical overview', *Psychotherapy* (1993) 30(1), 133-140.

heterosexuals[44]. 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[45].

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[46]. It is also possible that feeling shame or guilt over abuse could encourage further stigmatised behaviour. 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 the age of 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'[47], or in response to gender-atypical behaviour or appearance; a man who places strong emphasis on traditional masculinity might abuse a teenage boy who they perceived as 'girly' or effeminate (see section below on sex and gender atypicality and its relationship to same-sex attraction). Abuse could also occur in response to the strong threat open homosexuality often poses to social and community norms. An additional possibility is that likelihood of abuse and likelihood of SSA are both influenced by a separate underlying factor that is currently unknown.
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 (ie. 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[48]. The second study (n=33,771) utilised a methodology known as instrumental variable

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[44] e.g., Stoddard, J.P., Dibble, S.L. & Fineman, N. 'Sexual and physical abuse: A comparison between lesbians and their heterosexual sisters', *Journal of Homosexuality* (2009) 56(4), 407-420; Balsam, K.F., Rothblum, E.D. & Beauchaine, T.P. 'Victimization over the life span: A comparison of lesbian, gay, bisexual, and heterosexual siblings', *Journal of Consulting and Clinical Psychology* (2005) 73(3), 477-487. For a review of this literature, see 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.

[45] 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.

[46] For a discussion of possible causal pathways, see Gartner, R.B. 'Sexual victimisation of boys by men: Meanings and consequences', *Journal of Gay and Lesbian Psychotherapy* (1999) 3, 1-33.

[47] For example, homosexual women who publicly come out may experience 'corrective rape', where abuse by males is carried out ostensibly to reverse a homosexual orientation. If this occurs before age 18 it will appear on surveys of childhood sexual abuse.

[48] Wilson *op. cit*., (44). 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.

analysis[49], and reported that in both male and female cohorts abuse occurred prior to the development of SSA; however, this study suffered from retrospective reporting (possible recall bias), has not been replicated, and its methodology is controversial[50].

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[51].

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 (as yet entirely) theoretical model assumes that SSA develops unconsciously and uncontrollably when an individual is exposed to positive 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 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 unreplicated 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[52]. 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[53].

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

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[49] Roberts, A., Glymour, M. & Koenen, K. 'Does maltreatment in childhood affect sexual orientation in adulthood?', *Archives of Sexual Behavior* (2013) 42(2), 161-171.

[50] See Bailey, D. & Bailey, J.M. 'Poor instruments lead to poor inferences: Comment on Roberts, Glymour, and Koenen (2013)', *Archives of Sexual Behavior* (2013) 42(8), 1649-1652, for a dissenting commentary.

[51] Andersen *op. cit*., (45).

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

[53] 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. This study utilised a method of network visualisation to generate maps of peer-to-peer networks using data from Waves I and II of the US AddHealth cohort, dating from the 1990s. They found significant peer influences on sexual behaviour, but no influences on sexual attraction.

to parental imitation. Many of these studies have been qualitative and narrative[54]. In addition, four small quantitative studies looking exclusively at lesbian-headed families have been carried out[55]. Because lesbian parenting is a relatively recent phenomenon, these studies have been confined to adolescents and young adults (15 – 23 years old)[56], and have used a variety of measures of sexual and romantic attraction and sexual behaviour. 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 (the same is not true of sons of lesbian mothers). It is unknown how far these results are limited by the age of the 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[57].

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[58]. In these contexts, it is difficult 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

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[54] e.g., 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. For a review of early work, see Stacey, J. & Biblarz, T.J. '(How) Does the sexual orientation of parents matter?', *American Sociological Review* (2001) 66(2), 159-183.

[55] 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; Golombok, S. & Badger, S. 'Children raised in mother-headed families from infancy: A follow-up of children of lesbian and single heterosexual mothers, at early adulthood', *Human Reproduction* (2010) 25(1), 150-157; Wainright, J.L., Russell, S.T. & Patterson, C.J. 'Psychosocial adjustment, school outcomes, and romantic relationships of adolescents with same-sex parents', *Child Development* (2004) 75(6), 1886-1898; Golombok, S. & Tasker, F. 'Do parents influence the sexual orientation of their children? Findings from a longitudinal study of lesbian families', *Developmental Psychology* (1996) 32(1), 3-11. See also Regnerus, M. 'How different are the adult children of parents who have same-sex relationships? Findings from the New Family Structures Study', *Social Science Research* (2012) 41(4), 752-770, but note that the methodology of this study was highly controversial.

[56] Offspring have been derived from both earlier heterosexual relationships, and planned IVF treatments. Sample sizes are always $n < 100$.

[57] For a discussion, see Bos, H. & Sandfort, T.G.M. 'Children's gender identity in lesbian and heterosexual two-parent families', *Sex Roles* (2010) 62(1-2), 114-126; Istar Lev, A. 'How Queer! – The development of gender identity and sexual orientation in LBGTQ-headed families', *Family Process* (2010) 49(3), 268-290; Biblarz, T.J. & Stacey, J. 'How does the gender of parents matter?', *Journal of Marriage and Family* (2010) 72(1), 3-22; Stacey *op. cit*., (54).

[58] e.g., Gomillion, S.C. & Giuliano, T.A. 'The influence of media role models on gay, lesbian, and bisexual identity', *Journal of Homosexuality* (2011) 58(3), 330-354.

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.

Maternal stress and prenatal influences

Another possible aetiology of SSA is that it is determined prenatally by various factors acting upon a woman during her pregnancy. A developing foetus is highly sensitive to perturbations in the uterine chemical and hormonal environment, and it is known that factors like maternal drug use, elevated levels of stress in the mother and poor maternal nutrition during pregnancy can have lifelong effects on the offspring, and sometimes even intergenerational effects in addition. These effects can be physical, such as an increased risk of diabetes in children whose mothers were malnourished during pregnancy[59], behavioural, such as elevated risk for hyperactivity and antisocial behaviour in children whose mothers smoked during pregnancy[60], or psychological, such as impacts on cognitive capacity in children whose mothers were highly stressed[61]. It is therefore plausible to suggest that such maternal influences could affect sexual attraction status in the fetus, although this will not be manifest until later in life.

The hypothesis that children of highly stressed mothers are more likely to be same-sex attracted is based on the fact that highly stressed mothers have elevated levels of circulating cortisol, which can pass through the placental barrier and affect the fetal brain[62]. This hypothesis first came to prominence in the 1980s when an East German biologist, Gunter Dörner, and colleagues published two studies that investigated sexual attraction status in individuals whose mothers were pregnant during the Second World War[63]. Dörner et al. suggested that wartime mothers had a higher incidence of homosexual sons than did women who were pregnant in the peacetime years immediately after the war. However, the Dörner studies were criticised for merely inferring high levels of stress from the mothers' proximity to war, rather than measuring stress directly and

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[59] Lumey, L.H., Stein, A.D. & Susser, E. 'Prenatal famine and adult health', in Fielding, J.E., Brownson, R.C. & Green, L.W. *Annual Review of Public Health, Vol 32.* (2009), pp. 237-262.

[60] e.g., Knopik, V.S. 'Maternal smoking during pregnancy and child outcomes: Real or spurious effect?', *Developmental Neuropsychology* (2009) 34(1), 1-36.

[61] e.g., Tuovinen, S. Raikkonen, K. et al. 'Hypertensive disorders in pregnancy and cognitive decline in the offspring up to old age', *Neurology* (2012) 79(15), 1578-1582; Van den Bergh, B.R.H., Mulder, E.J.H., Mennes, M. & Glover, V. 'Antenatal maternal anxiety and stress and the neurobehavioural development of the fetus and child: links and possible mechanisms. A review', *Neuroscience and Biobehavioral Reviews* (2005) 29(2), 237-258.

[62] e.g., Weinstock, M. 'The long-term behavioural consequences of prenatal stress', *Neuroscience and Biobehavioural Reviews* (2008) 32(6), 1073-1086.

[63] Dörner, G., Schenk, B., Schmiedel, B. & Ahrens, L. 'Stressful events in prenatal life of bi- and homosexual men', *Experimental and Clinical Endocrinology and Diabetes* (1983) 81(1), 83-87; Dörner, G., Geier, T. et al. 'Prenatal stress as possible aetiogenetic factor of homosexuality in human males', *Endokrinologie* (1980) 75(3), 365-368;

taking account of individual circumstances. They also inferred homosexuality from the fact that the men were being treated for venereal disease, rather than using more sensitive and precise measures of SSA. A replication study conducted in Germany failed to replicate the findings[64]. Three more studies on the association of maternal stress and SSA have since been carried out, using a measure of life-event stress[65]. One found no correlation[66]; the remaining two (carried out by the same research group) found that SSA in males was positively correlated with higher maternal stress levels when the stress was experienced in a specific trimester, but failed to agree whether the first or second trimester was the critical period[67].

In conclusion, it seems likely that there is no effect of maternal stress on the likelihood of SSA. This is also the case for maternal smoking, alcohol use and malnutrition, which have each only been investigated in single, unreplicated studies[68].

Biological explanations

Genetics – twin studies

The theory that SSA is caused by a variant gene[69] or variant genes has become a prevalent hypothesis in the past 20 years[70] – 44% of respondents in one US survey mentioned genetics as one cause of SSA among others while 19% of respondents cited

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[64] Schmidt, G. & Clement, U. 'Does peace prevent homosexuality?', *Journal of Homosexuality* (1995) 28(3-4), 269-275.

[65] Participants are asked to recall the incidence of stressful life-events such as bereavements, job changes, house moves and health problems. The frequency of such events is used as a rough proxy for stress level across the lifespan. This measure does not take into account individual personality differences in approaches to dealing with stress.

[66] Bailey, J.M., Willerman, L. & Parks, C. 'A test of the maternal stress theory of human male homosexuality', *Archives of Sexual Behavior* (1991) 20(3), 277-293.

[67] Ellis, L. & Cole-Harding, S. 'The effects of prenatal stress, and of prenatal alcohol and nicotine exposure, on human sexual orientation', *Physiology and Behavior* (2001) 74(12), 213-226, and Ellis, L., Peckham, W., Ames, M.A. & Burke, D. 'Sexual orientation of human offspring may be altered by severe maternal stress during pregnancy', *Journal of Sex Research* (1988) 25(1), 152-157.

[68] Rooij, S.R., Painter, R.C., Swaab, D.F. & Roseboom, T.J. 'Sexual orientation and gender identity after prenatal exposure to the Dutch famine', *Archives of Sexual Behavior* (2009) 38(3), 411-416; Ellis *op. cit*., (67).

[69] A gene that exists in two or more different forms in the population. Variant forms are known as alleles. The most common form of genetic variant is a Single Nucleotide Polymorphism (SNP, pronounced Snip) where one base is substituted for another. A SNP occurs on average in every 1 in 1000 bases, resulting in around 3 million SNPs per person in the 3.2 billion base pair human genome. Variants can also arise as a result of the deletion or the addition to the genome of larger stretches of nucleotide base-pairs, or even duplications of whole genes (Copy Number Variants, CNVs).

[70] At least in Western countries. There is anecdotal if not systematic evidence that environmental explanations are more prevalent among the lay public than biological explanations in non-Western countries; see Boxill, I., Martin, J. et al. *National survey of attitudes and perceptions of Jamaicans towards same sex relationships*, Mona: University of the West Indies (2011), for an example.

genetics as the exclusive cause of SSA[71]. The 'gay gene' hypothesis crops up frequently in media and popular science articles, in song lyrics and at Gay Pride marches[72]. At least two recently-published science fiction books take the existence of a 'gay gene' as a central premise[73]. 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[74]. We would expect identical twins to be always concordant (ie. the same) for SSA if they were carriers of the same 'gay gene'; in reality, they are never 100% concordant[75]. Moreover, as discussed above, same-sex attraction appears in myriad different forms (the Kinsey scale continuum), and to suggest that a single gene controls all these forms of SSA is implausible, to say the least. 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 environment to different degrees[76]. Such studies 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 their variant genes, while identical twins share them all[77]. This methodology allows

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[71] Sheldon *op. cit*., (28).

[72] For examples, see O'Riordan, K. 'The life of the gay gene: From hypothetical genetic marker to social reality', *Journal of Sex Research* (2012) 49(4), 362-368, and 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 and Francis (2005), pp. 99-117.

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

[74] The incidence of a trait controlled by a single gene can be easily predicted in the next generation, because each person carries two alleles (A and a) which can be present in three combinations (AA, Aa, and aa). Two parents who are both Aa will have on average ¼ AA, ½ Aa, and ¼ aa children, because the two allele types combine at random.

[75] The concordance rate of identical twins varies depending on the variable being measured (attraction, behaviour, etc.). However, in no study are identical twins 100% concordant. Where correlation is measured, identical twins are almost always less than 0.50 correlated, for both male and female twins. However, most studies also find that identical twins are significantly more highly correlated than non-identical twins; see 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; Langstrom, N., Rahman, Q., Carlstrom, E. & Lichtenstein, P. 'Genetic and environmental effects on same-sex sexual behavior: A population study of twins in Sweden', *Archives of Sexual Behavior* (2010) 39(1), 75-80; Santtila, P., Sandnabba, N.K. et al. 'Potential for homosexual response is prevalent and genetic', *Biological Psychology* (2008) 77(1), 102-105. For a discussion of earlier studies, see Hershberger, S.L. 'A twin registry study of male and female sexual orientation', *Journal of Sex Research* (1997) 34(2), 212-222.

[76] 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).

[77] The easiest method by which to separate genetic and environmental factors is to investigate identical twins who were adopted apart at birth – any differences between them can be attributed to their different environments, as they are genetically identical. However, this is obviously a very small

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[78]. 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 other sexual orientation measures date from the early 1990s, and a substantial number have been carried out. An influential early pair of twin studies[79] found heritabilities for overall sexual orientation 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 among studies[80]. 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 variable is likely to be a key component in the poor reproducibility of these data (ie.

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population. Because SSA is rare, the numbers of SSA identical twins who were adopted apart is so small that it does not allow for meaningful study.

[78] In brief, twin studies divide the total variance of a population into genetic, shared environment, and unique environment components. Because the variance describes the total value of all the variability within a population, the components that contribute to the variance can be expressed either as a percentage of the total or as 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.

[79] 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.

[80] e.g., Burri, A., Cherkas, L., Spector, T. & Rahman, Q. 'Genetic and environmental influences on female sexual orientation, childhood gender typicality and adult gender identity', *PloS One* (2011) 6(7), e21982; Alanko *op. cit*.,(75); Langstrom *op. cit*., (75); Santtila *op. cit*., (75); Kendler, K.S., Thornton, L.M., Gilman, S.E. & Kessler, R.C. 'Sexual orientation in a US national sample of twin and nontwin sibling pairs', *American Journal of Psychiatry* (2000) 157(11), 1843-1846.

studies are measuring different constructs with different experimental methodologies)[81]. 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[82]. Twin studies are also limited by their reliance on a set of assumptions which some theorists argue are invalid[83]. 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 foetal 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, as it could suggest that the significant genetic influence seen in twin studies is an artefact of a shared placental environment, not genes at all[84]. Identical twins also report feeling emotionally closer to their co-twin than fraternal twins do, due to their strong physical resemblance[85].

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. Heritability values are inconsistent among studies, with large confidence intervals including 0, 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

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[81] Compare, for instance, Bailey, J.M., Dunne, M.P. & Martin, N.G. 'Genetic and environmental influences on sexual orientation and its correlates in an Australian twin sample', *Journal of Personality and Social Psychology* (2000) 78(3), 524-536, and Kirk, K.M., Bailey, J.M., Dunne, M.P. & Martin, N.G. 'Measurement models for sexual orientation in a community twin sample', *Behavior Genetics* (2000) 30(4), 345-356. These two papers were written by the same researchers, using the same sample, but by constructing their measurement of same-sex attraction slightly differently, they produced markedly different results.

[82] 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.

[83] 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.

[84] Twins who share a placenta are more similar to each other for a range of different personality and cognitive characteristics; see Jacobs, N., Van Gestel, S. et al. 'Heritability estimates of intelligence in twins: Effect of chorion type', *Behavior Genetics* (2001) 31(2), 209-217, for an example.

[85] e.g., Neyer, F.J. 'Twin relationships in old age: A developmental perspective', *Journal of Social and Personal Relationships* (2002) 19, 155-177.

role for environmental factors than for genes in the aetiology of SSA. It is important to note that twin 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[86] - the paper attracted widespread press and public attention and can be credited with bringing the idea of a 'gay gene' into public discourse[87]. Hamer et al. analysed 22 genetic markers[88] on the X chromosome in 40 pairs of brothers, both of whom were same-sex attracted. Five markers in a region known as Xq28[89] 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. There was also possible ascertainment bias[90]. 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', despite the fact that it is a spatial signifier, not a gene name[91], 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*[92] only added to the confusion. The eagerness of the press and public to embrace the 'gay gene' aetiology can perhaps be explained by a number of socio-cultural factors existing in early 1990s

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[86] 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.

[87] Kitzinger *op. cit*., (72)

[88] This type of analysis is known as a linkage study, which involves looking for correlations between the presence of specific DNA nucleotides in particular positions in the genome (the marker) and the trait of interest across multiple family members.

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

[90] The sample collected was a convenience sample, not a demographic sample. It is possible that the characteristics of this sample differed from those of the general population.

[91] O'Riordan *op. cit*., (72).

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

America. Recent successes in identifying causal variants for single-gene disorders, such as the gene involved in Cystic Fibrosis in 1988, meant that the public were primed for single-gene explanations for behaviour as well as medical conditions. Moreover, this narrative was simple to understand and easy to explain. The 'gay gene' explanation was also widely promoted by sections of the gay rights movement at the time, as it was seen as a way of combating what they saw as discriminatory laws[93].

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)[94], two other studies using the same methodology with larger samples failed to do so.[95] 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[96]. 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[97]. 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 SSA or sexual orientation on other chromosomes; however, none of these have produced any strongly significant findings. Mustanski et al. reported nearly-significant correlation between SSA and the 7q36 region in a medium-sized study, but this result

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[93] See O'Riordan *op. cit*., (72).

[94] Hu, S., Pattatucci, A.M.L. et al. 'Linkage between sexual orientation and chromosome Xq28 in males but not in females', *Nature Genetics* (1995) 11(3), 248-256.

[95] 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. Mustanski's study (n = 146 families) was published by the original Hamer research group and half of its data were from the original data set, but they still found no significant result.

[96] 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.

[97] Studies with a positive finding: Rahman, Q., Collins, A. et al. 'Maternal inheritance and familial fecundity factors in male homosexuality', *Archives of Sexual Behavior* (2008) 37(6), 962-969; Camperio-Ciani, A., Corna, F. & Capiluppi, C. 'Evidence for maternally inherited factors favouring male homosexuality and promoting female fecundity', *Proceedings of the Royal Society B: Biological Sciences* (2004) 271(1554), 2217-2221. Studies with a negative finding: 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; Bailey, J.M., Pillard, J.C. et al. 'A family history study of male sexual orientation using three independent samples', *Behavior Genetics* (1999) 29(2), 79-86.

was not replicated by another group[98]. Sanders et al. reported a significant association between the 8q12 region and male SSA, but this has not been identified in any other research[99]. In addition, the personal DNA testing company 23andMe conducted a genome-wide association study (GWAS)[100] using its customer database (n = 7887 men, 5570 women), but found no significant correlation between having a homosexual identity and any genetic marker[101]. However, such a null result is not surprising as a GWAS requires a very large sample size to produce significant results[102]. In addition, three studies in male populations have looked at markers in specific genes, known as a candidate gene study. Two studies[103] 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[104]. 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.

Genetics – the evolutionary paradox

An important criticism of the hypothesis of a genetic aetiology of same-sex attraction is that evolutionary theory predicts that genes that are deterministically causal for SSA would rapidly be filtered out of a population by natural selection. This is because same-sex attraction hugely reduces reproductive fitness – same-sex attracted individuals who also engage in same-sex behaviour have far fewer offspring than OSA individuals in contemporary Western society[105]. Yet historical and ethnographic data suggest that

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[98] 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 2005, *op.cit*.(95).

[99] Sanders *op. cit*., (96).

[100] A GWAS looks at millions of genetic markers across the genome in large cohorts, and compares differences in marker frequency between those members of the cohort who have the trait of interest (cases) and those who do not (controls).

[101] 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.

[102] For a discussion, see Rietveld, C.A., Medland, S.E. et al. 'GWAS of 126,559 individuals identifies genetic variants associated with educational attainment', *Science* (2013) 340(6139), 1467-1471, which used over 125,000 participants to perform a GWAS.

[103] 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.

[104] 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.

[105] e.g., Iemmola, F. & Camperio-Ciani, A. 'New evidence of genetic factors influencing sexual orientation in men: Female fecundity increase in the maternal line', *Archives of Sexual Behavior* (2009) 38(3), 393-399. However, it is worth noting that increased use of IVF technology may well change this situation in the

homosexual behaviour has occurred at persistent low levels across times and cultures[106], creating something of an evolutionary paradox if genes are indeed causally responsible for same-sex attraction. Some critics have used this paradox to argue that genes are therefore not causally associated with the development of SSA. However, this is too simplistic an interpretation. In the first place, genetic variants that contribute to a trait via mechanisms such as gene x environment interaction are not filtered out of the population in the same way, as an environmental component is necessary for a trait to be phenotypically relevant[107]. In the second place, it is possible to maintain deleterious genes (ie. genes that cause a reduction in reproductive fitness) within a particular population if in some circumstances those same genes provide an advantage. Theoretical modelling suggests that for SSA, two evolutionary mechanisms could plausibly maintain genes contributing to SSA within a population: sexually antagonistic selection and kin selection[108], although it should be noted that these theories only apply to same-sex attraction in males, not females.

The sexually antagonistic selection hypothesis is predicated on the theory that genetic factors that are associated with same-sex attraction when in males are associated with increased fertility when in females. In other words, the same genetic variants produce different outcomes across the sexes. In men, the outcome is deleterious for their fitness, but in women, the outcome is advantageous. Thus, there is antagonism between the sexes – the genetic variants will be maintained in the population due to their advantageous effects in women, even though this produces the occasional homosexual son or brother. If the sexually antagonistic selection theory is true, we would expect that the close female relatives (mothers, sisters, and cousins) of SSA men would have larger families, a result of their increased fertility. This has been tested in a small number of cohorts, with inconsistent results. Some studies have found that homosexual men have more brothers and sisters[109], or that female relatives (grandmothers, mothers, sisters,

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near future, as the number of lesbian and gay parents with biological children is increasing. See http://williamsinstitute.law.ucla.edu/research/census-lgbt-demographics-studies/infographic-msas-may-2013/ (accessed 10 November 2014).

[106] Ford *op. cit*., (37).

[107] Gene x environment interaction occurs when a behavioural or physical (phenotypic) outcome is only seen when an individual possesses both a particular genetic mutation and a particular environmental circumstance. One or the other by itself is not enough. The gene may be said to act as a 'predisposing' factor to the phenotypic outcome, which will only appear if the environment is also right. In the case of SSA, a plausible hypothesis is that certain genes may predispose individuals to become SSA if they are sexually abused in childhood. Gene x environment interaction has not been explored in the field of sexuality studies, but in other areas, such as predisposition to violence, it is relatively well investigated.

[108] Barthes, J., Godelle, B. & Raymond, M. 'Human social stratification and hypergyny: Toward an understanding of male homosexual preference', *Evolution and Human Behavior* (2013) 34(3), 155-163; Gavrilets, S. & Rice, W.R. 'Genetic models of homosexuality: generating testable predictions', *Proceedings of the Royal Society B: Biological Sciences* (2006) 273(1605), 3031-3038.

[109] VanderLaan, D.P. & Vasey, P.L. 'Male sexual orientation in independent Samoa: Evidence for fraternal birth order and maternal fecundity effects', *Archives of Sexual Behavior* (2011) 40(3), 495-503; Rieger, G., Blanchard, R. et al. 'Further data concerning Blanchard's (2011) "Fertility in the mother of firstborn homosexual and heterosexual men"', *Archives of Sexual Behavior* (2012) 41(3), 529-531.

aunts) of homosexual men have more children[110]. However, other studies have found no association between fertility and SSA[111]. In addition, none of these studies sufficiently controlled for socio-cultural factors that might be influencing birth rates, such as religious beliefs or family income. Only one study controlled for the possible bias effect of the fraternal birth order (see below) when sampling via a proband cohort of homosexual men. These results are thus currently inconclusive, although suggestive when considered together with the data from twin studies indicating a possible genetic influence on SSA in men.

Kin selection theory also suggests that genetic variants associated with same-sex attraction increase the fitness of close relatives of homosexual men. The reasoning is that, because homosexual men do not usually have offspring of their own, they are able to provide extra-parental care to relatives such as nephews and nieces. The recipients of these extra resources, which could be emotional or material, thus have increased fitness. If nephews and nieces are carrying the same genetic variants as their uncle (they share approximately 25% of their genetic information), their increased fitness means that the genes are more likely to be passed on to the next generation. The kin selection hypothesis thus predicts that homosexual men should altruistically contribute more resources to their kin than childless heterosexual men do. This prediction has been tested in a few small-scale studies. Overall, studies within Western countries have found no evidence for increased kin support in homosexual males[112]; however, these studies often do not take into account societal factors. For example, some SSA men in Western countries have experienced ostracism from their families due to their sexual orientation. Similarly, most Western cultures are individualistic, with little emphasis on broad family support and are also geographically disparate. A research group working with a community in Samoa known as 'fa'afafine'[113] has found that in this community where family ties are highly valued, fa'afafine males do contribute substantially to their close kin[114]. This could be a

[110] VanderLaan, D.P., Forrester, D.L., Petterson, L.J. & Vasey, P.L. 'Offspring production among the extended relatives of Samoan men and fa'afafine', *PloS One* (2012) 7(4), e36088; Camperio-Ciani, A., Cermelli, P. & Zanzotto, G. 'Sexually antagonistic selection in human male homosexuality', *PloS One* (2008) 3(6), e2282; King, M., Green, J. et al. 'Family size in white gay and heterosexual men', *Archives of Sexual Behavior* (2005) 34(1), 117-122.

[111] Blanchard, R. 'Fertility in the mothers of firstborn homosexual and heterosexual men', *Archives of Sexual Behavior* (2012) 41(3), 551-556. Some studies found positive correlations only among maternal relatives, not among paternal relatives: Camperio-Ciani, A. & Pellizzari, E. 'Fecundity of paternal and maternal non-parental female relatives of homosexual and heterosexual men', *PloS One* (2012) 7(12), e51088; Camperio-Ciani *op. cit*., (97).

[112] Abild, M.L., VanderLaan, D.P. & Vasey, P.L. 'No evidence for treating friends' children like kin in Canadian androphilic men', *Journal of Sex Research* (2013) 50(7), 697-703; Vasey, P.L. & VanderLaan, D.P. 'Sexual orientation in men and avuncularity in Japan: Implications for the kin selection hypothesis', *Archives of Sexual Behavior* (2012) 41(1), 209-215; Rahman, Q. & Hull, M.S. 'An empirical test of the kin selection hypothesis for male homosexuality', *Archives of Sexual Behavior* (2005) 34(4), 461-467.

[113] Fa'afafine are defined as homosexual men who appear and behave in stereotypically female ways, including wearing women's clothing. Relationships are generally conducted within the fa'afafine group.

[114] VanderLaan, D.P. & Vasey, P.L. 'Birth order and avuncular tendencies in Samoan man and fa'afafine', *Archives of Sexual Behavior* (2013) 42(3), 371-379; VanderLaan, D.P. & Vasey, P.L. 'Relationship status and elevated avuncularity in Samoan fa'afafine', *Personal Relationships* (2012) 19(2), 326-339; Vasey, P.L. &

phenomenon that is specific to this one community, or to similar social systems, however, and not generalisable to the broader same-sex attracted population. It is worth emphasising that a finding of elevated kin support by homosexual males in tight-knit social systems does not necessarily mean that their SSA is influenced by genetic variation.

Overall, as with any attempt to provide an evolutionary explanation for a behavioural trait, it is rather easy to generate adaptationist 'explanations' for the trait in question. The late Stephen Jay Gould's critique of such adaptationist 'just-so stories' should be kept in mind when considering such claims.

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 the order in which children are born. The few small studies to investigate this hypothesis during the twentieth century produced inconclusive results[115], 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[116]. 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[117].

It is possible that the FBO effect is sociological; for example, younger sons may receive less parental care and therefore be more likely to engage in a stigmatised behaviour. Daryl Bem hypothesised that younger brothers were more likely to eroticise masculinity as a result of feeling separate from their older brothers[118]. However, these explanations have not been empirically tested, and must be considered in the light of scant evidence for other socialisation hypotheses, discussed above.

VanderLaan, D.P. 'Avuncular tendencies and the evolution of male androphilia in Samoan fa'afafine', *Archives of Sexual Behavior* (2010) 39(4), 821-830.

[115] See Blanchard, R. 'Birth order and sibling sex ratio in homosexual versus heterosexual males and females', *Annual Review of Sex Research* (1997) 8(1), 27-67, for a review of early work on birth order.

[116] 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; Bogaert, A.F. 'Number of older brothers and sexual orientation: New tests and the attraction/behavior distinction in two national probability samples', *Journal of Personality and Social Psychology* (2003) 84(3), 644-652; Blanchard, R. 'Birth order and sibling sex ratio in homosexual versus heterosexual males and females', *Annual Review of Sex Research* (1997) 8(1), 27-67; Blanchard, R. & Bogaert, A.F. 'Homosexuality in men and number of older brothers', *American Journal of Psychiatry* (1996) 153(1), 27-31. This is just a selection of their many papers on the topic.

[117] VanderLaan *op. cit*., (109); Schwartz *op. cit*., (97).

[118] Bem, D.J. 'Exotic becomes erotic: A developmental theory of sexual orientation', *Psychological Review* (1996) 103(2), 320-335.

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 (ie. third sons are more likely to be SSA than second sons)[119]. 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[120]) 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 affect either the developing fetal brain and/or the fetal genome and/or fetal epigenome[121]. 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[122]. 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 [123] while, at the same time, miscarried male fetuses do increase the likelihood of SSA in subsequent male pregnancies[124], suggesting a prenatal effect. 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 brother did, again suggesting the effect is prenatal[125].

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[126]). 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. More replication is needed to confirm the results of the single study in an adopted population. 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.

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[119] Blanchard *op. cit*., (116).

[120] Antigens are small compounds foreign to the body, which are bound by antibodies as part of the body's immune response. It is known that male cells produce a class of antigenic compounds collectively known as Y-linked minor histocompatability antigens, or H-Y antigens, which are produced from the Y chromosome.

[121] The epigenome refers to the chemical groups which sit 'above' ('epi') the genome and help regulate gene transcription.

[122] For a more detailed review of the maternal immune hypothesis, see Bogaert *op. cit*., (116).

[123] Blanchard *op. cit*., (116).

[124] 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.

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

[126] Tan, JC., Wadia, P.P. et al. 'H-Y antibody development associated with acute rejection in female patients with male kidney transplants', *Transplantation* (2008) 86(1), 75-81.

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[127]. 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[128]. 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 SSA females are masculinised, and thus display a sexual behaviour at variance with their chromosomal sex. This 'intersex'[129] hypothesis, as it is sometimes known, has a long history. One of its original proponents was the German biologist Karl Ulrichs, generally regarded as the father of the scientific study of homosexuality, who argued that SSA males contained a 'female essence'[130]. The theory remained very influential up until the turn of the twentieth century, when it was mostly replaced by Sigmund Freud's theory of homosexuality as immaturity, but has undergone a revival in recent decades. Today, the intersex hypothesis is widely referenced by the general public[131]; 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[132], and no consistent evidence that they

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[127] e.g., Blanchard *op. cit*., (111).

[128] Blanchard, R. & Bogaert, A.F. 'Proportion of homosexual men who owe their sexual orientation to fraternal birth order: An estimate based on two national probability samples', *American Journal of Human Biology* (2004) 16(2), 151-157; Cantor, J., Blanchard, R., Paterson, A. & Bogaert, A.F. 'How many gay men owe their sexual orientation to fraternal birth order?', *Archives of Sexual Behavior* (2002) 31(1), 63-71.

[129] The term 'intersex' is used very broadly to describe a range of conditions where elements of male and female are present in one individual. This includes disorders of sex development like hermaphroditism, genital malformation, chromosomal aneuploidies and genetic conditions. It also includes behavioural conditions such as Gender Identity Disorder and trans-sexualism. Use of the term here should not be taken to necessarily imply that same-sex attracted individuals are hermaphroditic or otherwise physically intersex.

[130] Ulrichs coined this group 'the third sex' or 'urnings'. He never defined the biological nature of the female essence that he believed was responsible for the third sex. For a more detailed discussion of early theories of homosexuality among German biologists and sexologists, see Drescher *op. cit*., (32); Sandfort, T.M. 'Sexual orientation and gender: Stereotypes and beyond', *Archives of Sexual Behavior* (2005) 34(6), 595-611, and Kennedy, H.C. 'The "third sex" theory of Karl Heinrich Ulrichs', *Journal of Homosexuality* (1980) 6(1-2), 103-111.

[131] For an example, see the responses to Sheldon *op. cit*., (28).

[132] Genitalia that are either unformed or contain elements of both male and female genitals can be caused by a variety of conditions, including chromosomal aneuploides like Turner Syndrome and Klinefelter

differ in height, weight or other physical attributes[133]. 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[134]. This assessment of sexual orientation was partially mediated by a judgement of atypicality (ie. more sex-atypical persons were judged to be homosexual, often correctly), suggesting that some physical differences may exist between some SSA and OSA individuals. However, the authors were careful to point out that the prediction rate was not 100%[135], 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[136] and others[137] 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-

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Syndrome, or a range of rare genetic conditions that affect sexual development. All these conditions are rare, much rarer than same-sex attraction, although precise estimates differ depending on exactly which disease condition is the causal factor. See also Bogaert, A.F. & Hershberger, S.L. 'The relation between sexual orientation and penile size', *Archives of Sexual Behavior* (1999) 28(3), 213-221, a single study that examined penile size in a demographic population of SSA men and found no correlation between genital size and homosexuality. Note that this study was carried out on data collected by the Kinsey Institute across a decades-long timescale, and therefore could not be directly verified by the researchers.

[133] e.g., Bogaert, A.F. & Liu, J. 'Physical size and sexual orientation: Analysis of the Chinese Health and Family Life Survey', *Archives of Sexual Behavior* (2013) 42(8), 1555-1559; Bogaert, A.F. 'Physical development and sexual orientation in men and women: An analysis of NATSAL-2000', *Archives of Sexual Behavior* (2010) 39(1), 110-116; Bogaert, A.F. & Friesen, C. 'Sexual orientation and height, weight, and age of puberty: new tests from a British national probability sample', *Biological Psychology* (2002) 59(2), 135-145. Note that all these papers have been carried out by one research group, and independent work is not available.

[134] 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; for the follow-up cross-cultural study from the same authors, see Valentova, J., Rieger, G. et al. 'Judgments of sexual orientation and masculinity-femininity based on thin slices of behavior: A cross-cultural comparison', *Archives of Sexual Behavior* (2011) 40(6), 1145-1152.

[135] Additionally, people's judgements of others are not neutral. In an independent study, it was found that female raters who self-identified as homosexual were significantly different in their judgements of the homosexuality of targets seen on video clips when compared to heterosexual raters; see Ruben, M.A., Hill, K.M, & Hall, J.A. 'How women's sexual orientation guides accuracy of interpersonal judgements of other women', *Cognition and Emotion* (2014) 28(8), 1-10. Other factors such as rater prejudices, implicit bias, or personality factors could also obscure the true relationship between sex atypicality and same-sex attraction. See Rieger 2010 *op. cit*., (134), for a discussion.

[136] 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; Lippa, R.A. 'Sexual orientation and personality', *Annual Review of Sex Research* (2005) 16, 119-153; Lippa, R.A. 'Gender-related traits of heterosexual and homosexual men and women', *Archives of Sexual Behavior* (2002) 31(1), 83-98.

[137] Hershberger, S.L. & Bogaert, A.F. 'Male and female sexual orientation differences in gambling', *Personality and Individual Differences* (2005) 38(6), 1401-1411.

femininity[138]. 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[139]. Indeed, this is one of the strongest findings in the field 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[140]. However, prospective and non-recall studies have produced similar positive findings[141].

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 weakens the generalisability of the findings, particularly for children and adolescents; studies with non-clinical populations produce notably lower estimates of same-sex attraction than those which use cohorts who are being treated for various forms of gender dysphoria[142]. For example, in their prospective demographic study Steensma et al.[143] reported that around 12% of gender-atypical children went on to develop SSA, which is higher than the rate among gender-typical children, but substantially lower than the 70% estimate produced by Wallien et al.[144] in their prospective study of children with Gender Identity Disorder. The link between 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

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[138] e.g., Rieger, G., Linsenmeier, J.A.W., Gygax, L. & Bailey, J.M. 'Sexual orientation and childhood gender nonconformity: Evidence from home videos', *Developmental Psychology* (2008) 44(1), 46-58; for a general dicussion, see Lippa *op. cit*., (136). For a more critical stance, see 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, which found that adult male homosexuals were more likely to view themselves as androgynous rather than feminine.

[139] For comprehensive reviews of this literature, 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, and Bailey, J.M. & Zucker, K.J. 'Childhood sex-typed behavior and sexual orientation – a conceptual analysis and quantitative review', *Developmental Psychology* (1995) 31(1), 43-55.

[140] See Gottschalk, L. 'Same-sex sexuality and childhood gender nonconformity: A spurious connection', *Journal of Gender Studies* (2003) 12(1), 35-50.

[141] 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. See also Rieger *op. cit*., (138), which got around the problem of recall bias by using home videos that could not be falsified.

[142] Gender dysphoria describes the psychological conviction that your gender does not match your sex. The psychiatric condition as described in the DSM is Gender Identity Disorder (GID).

[143] Steensma *op. cit*., (141).

[144] Wallien, M.S.C. & Cohen-Kettenis, P.T. 'Psychosexual outcome of gender-dysphoric children', *Journal of the American Academy of Child and Adolescent Development* (2008) 47(12), 1413-1423. See also Drummond, K.D., Bradley, S.J., Peterson-Badali, M. & Zucker, K.J. 'A follow-up study of girls with gender identity disorder', *Developmental Psychology* (2008) 44(1), 34-45, which estimated that 32% of children with GID went on to develop a homosexual orientation.

gender-dysphoric individuals have been documented, as have cases where children who were strongly gender dysphoric developed later heterosexual orientations[145].

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. Observational studies in several non-Western cultures have described communities of SSA males who are highly feminised in appearance and behaviour[146]; 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. This could potentially be forcing SSA individuals to adopt atypical gender presentations in order to avoid persecution; for example, in Iran homosexuality is almost universally associated with trans-sexualism, because the harsh laws on homosexuality necessitate individuals who are attracted to their own (chromosomal) sex to then undergo sex-reassignment surgery if they wish to practise that sexual behaviour[147]. Thus, any biologically-driven association between gender-atypicality and same-sex attraction is completely masked by the influence of the cultural environment.

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[148]. 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.

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

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[145] Wallien *op. cit*., (144).

[146] VanderLaan *op. cit*., (97); Lawrence, A.A. 'Societal individualism predicts prevalence of nonhomosexual orientation in male-to-female transsexualism', *Archives of Sexual Behavior* (2010) 39(2), 573-583.

[147] Lawrence *op. cit*., (146).

[148] For example, it has been hypothesised that children who are more gender-atypical are likely to be excluded from friendships with their own sex, leading them to eroticise same-sex affection. See Lippa *op. cit*., (136).

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[149]. 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[150].

It is frequently hypothesised that exposure to atypical concentrations of androgens during fetal development[151] 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 blood hormone concentration and any form of sex/gender atypicality or sexual orientation have been hampered by difficulties of measurement[152]. 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[153]. Atypical levels of testosterone in amniotic fluid and maternal blood serum have been correlated with gender atypicality in toy choice/play style in childhood[154], but null results have also been reported[155]. No study has assessed SSA directly (due to the long time lag

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[149] Balthazart, J. 'Minireview: Hormones and human sexual orientation', *Endocrinology* (2011) 152(8), 2937-2947; Zitzmann, M. & Nieschlag, E. 'Testosterone levels in healthy men and the relation to behavioural and physical characteristics: facts and constructs', *European Journal of Endocrinology* (2001) 144(3), 183-197.

[150] For a discussion of historical treatments, see Banks, A. & Gartrell, N.K. 'Hormones and sexual orientation: A questionable link', *Journal of Homosexuality* (1995) 28(3-4), 247-268.

[151] The other developmental phase during which androgens are important for sexual development is puberty. The impact of hormone concentration on sexual attraction during puberty is not further discussed in this article, but 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.

[152] In humans, anyway. In the interests of length, this article does not discuss evidence derived from animal studies, but experimental studies which have manipulated hormone concentrations have been carried out in mice, rats and ferrets. In general, these studies have found that in lower mammals, hormones have significant effects on mating choice. For a recent overview, see Henley, C.L., Nunez, A.A, & Clemens, L.G. 'Hormones of choice: The neuroendocrinology of partner preference in animals', *Frontiers of Neuroendocrinology* (2011) 32(2), 146-154. However, care should be exercised in extrapolating from animal studies to humans, as the measures of sexual behaviour and the experimental set-ups used in animal studies are substantially different from real-world sexual attraction in humans.

[153] Several methods are not further discussed here, as results have been inconsistent and sample sizes very small or consisting primarily of case reports. This includes studies of clinical cases of penis ablatio or cloacal exstrophy, exposure to external hormones during fetal development, and oto-acoustic sex differences. For comprehensive reviews see Hines, M. 'Prenatal endocrine influences on sexual orientation and on sexually differentiated childhood behavior', *Frontiers in Neuroendocrinology* (2011) 32(2), 170-182; Berenbaum *op. cit*., (151); Balthazart *op. cit*., (149).

[154] Amniotic fluid sampling: Auyeung, B., Baron-Cohen, S., et al. 'Fetal testosterone predicts sexually differentiated childhood behavior in girls and in boys', *Psychological Science* (2009) 20(2), 144-148. Maternal blood sampling: Hines, M., Johnston, K.J. et al. 'Prenatal stress and gender role behavior in girls and boys: A longitudinal, population study', *Hormones and Behavior* (2002) 42(2), 126-134. Note that Hines found a positive association only in girls.

[155] Constantinescu, M. & Hines, M. 'Relating prenatal testosterone exposure to postnatal behavior in typically developing children: Methods and findings', *Child Development Perspectives* (2012) 6(4), 407-

between fetal measurements and the development of sexual attraction). It is not clear that hormone concentration in amniotic fluid or maternal blood serum is an accurate marker for foetal blood hormone concentration, so these results are limited[156].

Several studies have used the 2D:4D finger ratio - the ratio between the length of the second and fourth finger – which is a sexually dimorphic trait that appears by the end of the first trimester, and is a biomarker for hormone concentration[157]. Over 20 studies have assessed 2D:4D ratios in adults and children with measures of sexual attraction, sexual orientation and various forms of behavioural atypicality, but results have been inconsistent. A sizeable number of studies have reported the predicted finding, that atypicality in finger ratio is associated with a higher likelihood of SSA or gender atypicality, but often only in women, not men[158]. Equally, a smaller number have found the exact inverse, that 2D:4D ratio is only correlated with gender atypicality in men but not women[159]. Yet other studies have found no association at all[160], or even the inverse finding[161]. This inconsistency may be attributable to the imprecise nature of 2D:4D ratio as a biomarker – it is known that digit ratio is affected by genetic factors and by ethnicity, not just hormone concentration[162].

413; van de Beek, C., Goozen, S.M., Buitelaar, J. & Cohen-Kattenis, P.T. 'Prenatal sex hormones (maternal and amniotic fluid) and gender-related play behavior in 13-month-old infants', *Archives of Sexual Behavior* (2009) 38(1), 6-15.

[156] For a discussion, see Kuijper, E.A.M., Ket, J.C.F., Caanen, M.R. & Lambalk, C.B. 'Reproductive hormone concentrations in pregnancy and neonates: a systematic review', *Reproductive BioMedicine Online* (2013) 27(1), 33-63.

[157] Men typically have lower ratios (i.e. more equal finger lengths) than women. See Galis, F., ten Broek, C.A., Dongen, S. & Wijnaendts, L.D. 'Sexual dimorphism in the prenatal digit ratio (2D:4D)', *Archives of Sexual Behavior* (2010) 39(1), 57-62.

[158] Sexual orientation or sexual attraction: Hiraishi, K., Sasaki, S., Shikishima, C. & Ando, J. 'The second to fourth digit ratio (2D:4D) in a Japanese twin sample: Heritability, prenatal hormone transfer, and association with sexual orientation', *Archives of Sexual Behavior* (2012) 41(3) 711-724; Grimbos, T., Dawood, K. et al. 'Sexual orientation and the second to fourth finger length ratio: A meta-analysis in men and women', *Behavioral Neuroscience* (2010) 124(2), 278-287; McFadden, D., Loehlin, J. et al. 'A reanalysis of five studies on sexual orientation and the relative length of the 2nd and 4th fingers (the 2D:4D ratio)', *Archives of Sexual Behavior* (2005) 34(3), 341-356; Williams, T.J., Pepitone, M.E. et al. 'Finger-length ratios and sexual orientation', *Nature* (2000) 404(6777), 455-456.

[159] Sexual orientation or sexual attraction: Manning, J., Churchill, A. & Peters, M. 'The effects of sex, ethnicity, and sexual orientation on self-measured digit ratio (2D:4D)', *Archives of Sexual Behavior* (2007) 36(2), 223-233; Lippa, R.A. 'Are 2D:4D finger-length ratios related to sexual orientation? Yes for men, no for women', *Journal of Personality and Social Psychology* (2003) 85(1), 179-188. Behavioural atypicality: Honekopp, J. & Thierfelder, C. 'Relationships between digit ratio (2D:4D) and sex-typed play behavior in pre-school children', *Personality and Individual Differences* (2009) 47(7), 706-710.

[160] Behavioural atypicality: Voracek, M., Pietschnig, J., Nader, I.W. & Stieger, S. 'Digit ratio (2D:4D) and sex-role orientation: Further evidence and meta-analysis', *Personality and Individual Differences* (2011) 51(4), 417-422; Rammsayer, T.H. & Troche, S.J. 'Sexual dimorphism in second-to-fourth digit ratio and its relation to gender-role orientation in males and females', *Personality and Individual Differences* (2007) 42(6), 911-920.

[161] McIntyre, M. 'Digit ratios, childhood gender role behavior, and erotic role preferences of gay men', *Archives of Sexual Behavior* (2003) 32(6), 495-497.

[162] Hiraishi *op. cit*., (158).

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[163]. Around a dozen studies across multiple age ranges have found that women with CAH are significantly more likely to report experiencing same-sex attraction or fantasy than matched controls[164]. 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[165], a finding that provides strong support for the fetal hormone hypothesis. 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[166]. 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. AIS occurs in approximately 1 in 20,000 live male births. 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[167].

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[163] For a review, see Hines *op. cit*., (153).

[164] 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 non-classical congenital adrenal hyperplasia as a function of degree of prenatal androgen excess', *Archives of Sexual Behavior* (2008) 37(1), 85-99; Hines, M., Brook, C. & Conway, G.S. 'Androgen and psychosexual development: Core gender identity, sexual orientation, and recalled childhood gender role behavior in women and men with congenital adrenal hyperplasia (CAH)', *Journal of Sex Research* (2004) 41(1), 75-81; Zucker, K.J., Bradley, S.J. et al. 'Psychosexual development of women with congenital adrenal hyperplasia', *Hormones and Behavior* (1996) 30(4), 300-318.

[165] Frisen *op. cit*., (164); Meyer-Bahlburg *op. cit*., (164).

[166] Beltz, A.M., Swanson, J.L. & Berenbaum, S.A. 'Gendered occupational interests: Prenatal androgen effects on psychological orientation to Things versus People', *Hormones and Behavior* (2011) 60(4), 313-317; Meyer-Bahlburg, H.L., Dolezal, C. et al. 'Gender development in women with congenital adrenal hyperplasia as a function of disorder severity', *Archives of Sexual Behavior* (2006) 35(6), 667-684; Pasterski, V.L., Geffer, M.E. et al. 'Prenatal hormones and postnatal socialization by parents as determinants of male-typical toy play in girls with congenital adrenal hyperplasia', *Child Development* (2005) 76(1), 264-278; Berenbaum, S.A. 'Effects of early androgens on sex-typed activities and interests in adolescents with congenital adrenal hyperplasia', *Hormones and Behavior* (1999) 35(1), 102-110.

[167] Hines, M., Ahmed, S.F. & Hughes, I. 'Psychological outcomes and gender-related development in Complete Androgen Insensitivity Syndrome', *Archives of Sexual Behavior* (2003) 32(2), 93-101;

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[168]. 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 indistinct from XX women, have female genitalia, and are raised with societal expectations 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. Many of the studies discussed above assess gendered behaviour, rather than SSA directly. 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 men.

Sex/gender atypicality – neurology

The human brain is sexually dimorphic in structure, functionality and disease susceptibility[169]. Structurally, males have larger brains in relation to overall body mass, as well as differences in regional structures such as the hypothalamus and the corpus callosum, although the exact areas are disputed. The extent and form of functional differences between the sexes is also debated[170]. 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.

Wisniewski, A.B., Migeon, C.J. et al. 'Complete Androgen Insensitivity Syndrome: Long-term medical, surgical, and psychosexual outcome', *Journal of Clinical Endocrinology and Metabolism* (2000) 85(8), 2664-2669.

[168] For a critical discussion, see Jordan-Young, R.M. 'Hormones, context, and 'Brain Gender': A review of evidence from congenital adrenal hyperplasia', *Social Science and Medicine* (2012) 74(11), 1738-1744.

[169] Paus, T. 'Sex differences in the human brain: A developmental perspective', *Progress in Brain Research* (2010) 186, 13-28.

[170] The reaction to a paper on functional connectivity differences between the sexes, Ingalhalikar, M., Smith, A. et al. 'Sex differences in the structural connectome of the human brain', *PNAS* (2014) 111(2), 823-828, is a good example.

Beginning in 1990, three now infamous studies using the technique of post-mortem brain dissection in males were published. Swaab and Hofman[171] reported that the volume of the suprachiasmatic nucleus of the hypothalamus (SCN) was 1.73 times larger in homosexuals than in heterosexuals, with twice as many cells, although interestingly they found no difference between heterosexual women and men in the SCN, and therefore denied that the larger SCN in homosexual males was typical of a 'female brain'. LeVay[172] found that the volume of the third interstitial nucleus of the hypothalamus (INAH3) was approximately twice as large in heterosexual men than in heterosexual women, and that homosexual men were not significantly different from heterosexual women in INAH3 size. Allen and Gorski[173] measured the area of the anterior commisure and reported that the anterior commisure in homosexual men was on average 34% larger than in heterosexual men, which was in fact more extreme than the difference between men and women (extreme female-typical).

However, these findings have not stood up over time, as they have a significant number of limitations. All three studies primarily used homosexual probands who had died of AIDS. Although attempts were made to control for possible impacts of the disease on brain structure by using non-AIDS-affected comparison groups, not enough attention was paid to the possible effects of pharmaceutical treatments or lifestyle factors like drug use[174]. Sample sizes were small and the technically challenging methodology of tracing area or volume in a fixed post-mortem brain is prone to error. LeVay's study in particular was criticised for being methodologically unsound, with only one researcher performing all experiments. 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[175].

More recently, magnetic resonance imaging (MRI) has been used to map structure in living brain tissue in two studies. Witelson et al.[176] reported a significant difference in the volume of the isthmus region in the corpus callosum between homo- and heterosexual men. Ponseti et al.[177] measured global grey matter (GM) density in heterosexual and

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[171] Swaab, D.F. & Hofman, M.A. 'An enlarged suprachiasmatic nucleus in homosexual men', *Brain Research* (1990) 537(1-2), 141-148.

[172] LeVay, S. 'A difference in hypothalamic structure between heterosexual and homosexual men', *Science* (1991) 253(5023), 1034-1037.

[173] 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.

[174] See Mbugua, K. 'Sexual orientation and brain structures: A critical review of recent research', *Current Science* (2003) 84(2), 173-178, for a critical review.

[175] e.g., Byne, W., Tobet, S. et al. 'The interstitial nuclei of the human anterior hypothalamus: An investigation of variation with sex, sexual orientation, and HIV status', *Hormones and Behavior* (2001) 40(2), 86-92; Lasco, M.S., Jordan, T.J. et al. 'A lack of dimorphism of sex or sexual orientation in the human anterior commissure', *Brain Research* (2002) 936(1-2), 95-98.

[176] 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.

[177] 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. Reduced (male-typical) GM volume of the left

homosexual men and women using the technique of voxel-based morphometry (VBM), and found only one strongly significant difference: homosexual women had reduced (male-typical) GM density in the left perirhinal cortex. 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[178].

The development of dynamic brain scanning methods (positron emission tomography and functional MRI) has also allowed potential functional differences between heterosexual and homosexual populations to be measured. As of 2014, two studies have been published, both of which report finding sex-atypical patterns of functionality in homosexual subjects compared to heterosexual subjects, in both men[179] and women[180]. Replication studies are required to verify these findings. A small number of research groups have also examined functional differences via 'cognitive task' experiments , where subjects complete tests of mental attributes like mental rotation, visuospatial processing, navigation, attention, empathy and memory. Whether these mental attributes are sexually dimorphic is disputed. Perhaps unsurprisingly, then, there is little consistency in whether SSA men and women are gender-atypical for these mental attributes. While some studies have reported that there are significant differences between heterosexuals and homosexuals[181] with respect to mental attributes, others report no difference[182].

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, either structural or functional, 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

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cerebellum and left ventral premotor cortex were also observed in homosexual compared to heterosexual women.

[178] Imaging studies suffer particularly from a lack of replication studies, partially because they are costly to run. It should be noted that publication bias (the fact that a finding of a positive association is more likely to be published than a negative finding) may well be affecting the published studies in this area.

[179] Kinnunen, L.H., Moltz, H., Metz, J. & Cooper, M. 'Differential brain activation in exclusively homosexual and heterosexual men produced by the selective serotonin reuptake inhibitor, fluoxetine', *Brain Research* (2004) 1024(1-2), 251-254.

[180] Berglund, H., Lindstrom, P. & Savic, I. 'Brain response to putative pheromones in lesbian women', *PNAS* (2006) 103(21), 8269-8274.

[181] Men: Collaer, M., Reimers, S. & Manning, J. 'Visuospatial performance on an Internet line judgment task and potential hormonal markers: Sex, sexual orientation, and 2D:4D', *Archives of Sexual Behavior* (2007) 36(2), 177-192; Rahman, Q., Wilson, G.D. & Abrahams, S. 'Performance differences between adult heterosexual and homosexual men on the Digit-Symbol Substitution subtest of the WAIS-R', *Journal of Clinical and Experimental Neuropsychology* (2004) 26(1), 141-148. Women: Perry, D., Walder, K., Hendler, T. & Shamay-Tsoory, S.G. 'The gender you are and the gender you like: Sexual preference and empathic neural responses', *Brain Research* (2013) 1534, 66-75.

[182] Rahman, Q. & Wilson, G.D. 'Large sexual-orientation-related differences in performance on mental rotation and judgment of line orientation tasks', *Neuropsychology* (2003) 17(1), 25-31.

require further elucidation; both genes and hormones are known to influence neural development[183], 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 are a wide variety of hypothesised causes for same-sex attraction. Some causal models have received more empirical support 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.

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[183] Wilson, C.A. & Davies, D.C. 'The control of sexual differentiation of the reproductive system and brain', *Reproduction* (2007) 133(2), 331-359.