

Is First Same-Sex Attraction a Developmental Milestone?

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Abstract

This paper combines the well-known concept of developmental milestones with standard statistical analysis of their spread in time to gauge the milestone status/genetic influence on the timing of first same-sex attraction (SSA) by comparison with timing of puberty. SSA is not a developmental milestone, nor does its timing have high genetic influence. The relative standard deviation (RSD) of the average age of first SSA is 40%, which is very high compared with the approximately 7% for milestones with very high known genetic influence, such as puberty. As reported in many studies over a period of thirty years, first attraction occurs at a mean age of ten for both sexes, both orientations, and cross-culturally. While it is commonly claimed in the literature that first SSA is a genuine sexually related attraction and biologically preprogrammed, both of these claims are doubtful. First attraction is on average about two years before puberty; hence it is mostly not puberty-driven. The age of ten is possibly connected with peak awareness of social gender differences. Alternatively but much less probably, the age of first SSA is connected with adrenarche (maturing of the adrenal glands). Age of first attraction turns out to be a poor choice to illustrate alleged innateness. Very few individuals have SSA as their earliest memories, which is hence a false stereotype.

Introduction

It is rather common to hear gay people say, “Oh, I’ve always been this way. My earliest memories are of feeling different, and attracted to males” (Hillier, Turner, & Mitchell, 2005). In context this usually means that their earliest memories are of SSA, and it implies that such individuals must have been born with those feelings. This is even claimed to be the case cross-culturally (McLelland, 2000). It is still possible to find academic statements implicitly or explicitly suggesting that one is born gay. For example, LeVay (2010) declares that “I am inclined to place most of the developmental control in the hands of prenatal hormones” (p. 279), and *Born Gay* is even the title of one book (Wilson & Rahman, 2005). By this, the authors mean that SSA is influenced predominantly by prenatal factors.

Clearly, people with SSA are not “born that way”—immediately after birth, such individuals cannot even differentiate between themselves and their mothers, let alone distinguish between the genders. The phrase “born that way” therefore means in this context *predestined*, or bound to develop SSA. If this were true, the development of SSA would be a milestone event, like puberty or gestation, which is biologically programmed to occur in a set developmental sequence. The term *milestone* has been applied to various stages in the “coming out” process of GLB people (Floyd & Bakeman, 2006), and first same-sex sexual attraction is one of the milestones included. As the balance of this paper shows, this term is applied inaccurately, since no evidence of biological programming for SSA has been documented.

Statistics of Developmental Milestones

Developmental milestones are tabulated in the literature for things like fetal growth, motor skills development, social skills, teeth eruption, puberty, and menopause. Failure or delay in reaching a milestone may be an important indicator of an underlying medical problem. As typical with a biological system, there will be a range of ages for a particular milestone derived from surveys of normal individuals. There will be a mean, and then a measure of age-spread, normally confidence intervals or the standard deviation, finally tabulated and used by medical professionals. These are generally larger, the later the milestone.

For example, there is a 3.8y standard deviation on the timing of menopause, but only a 0.023y standard deviation on gestation length (Table 1). Clearly the two measures are not directly or usefully comparable. The standard mathematical measurement that avoids this problem uses the coefficient of variation, or the *relative* standard deviation (RSD). The RSD is the standard deviation divided by the mean—in this case, the mean age. If an RSD exceeds 50%, the event is not a milestone. The RSD is used extensively in this paper for comparisons, and it should be noted that some are close to the 50% cutoff.

RSDs for selected postnatal milestones are given in Table 1. The literature for first same-sex attraction is treated later (see Table 3). RSD is calculated using time since conception. *First heterosexual/homosexual* is first sexual experience/initiation.

Table 1. Postnatal Milestones

<i>Milestone</i>	<i>Reference</i>	<i>RSD%</i>
Gestation length	(Kieler, Axelsson, Nilsson, & Waldenström, 1995)	3.0
First crawling, walking	(Adolph, Vereijken, & Denny, 1998)	7.6
First word, sentence	(Neligan & Prudham, 1969)	5.5, 3.8 (M/F)
Teeth eruption	(Hägg & Taranger, 1985)	8
Puberty	(Kaltiala-Heino, Marttunen, Rantanen, & Rimpela, 2003)	8.6
First heterosex	(Laumann, Gagnon, Michael, & Michaels, 1994)	7.1
First homosex	(Savin-Williams & Diamond, 2000)	33, 27 (M/F)
Hetero-marriage	(Laumann et al., 1994)	6.2
First birth	(Martin et al., 2002)	25
Graying	(Keogh & Walsh, 1965)	26
Balding	(Paik, Yoon, Sim, Kim, & Kim, 2001)	28
Menopause	(de Bruin et al., 2001) (Hayakawa et al., 1992)	7.3
Lifespan	(CDC, 2008)	25 ^a

We notice that same-sex initiation seems to have a much larger RSD than opposite-sex initiation or other milestones. High milestone variability is the result of a combination of genetic influences, family/social influences, and random events. These act to increase

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the RSD, so it might be a natural interpretation to say that many other influences are involved. It is not very surprising that the RSD for age of first birth to a mother is large, because many more factors enter into this than marriage, including deliberate postponement, difficulties conceiving, and so on. It is no surprise that lifespan has a larger RSD because many factors, such as accidents and lifestyle choices, are involved.

However, things might not be so simple. Sometimes a societal stricture or legal requirement may actually decrease the RSD; for example, all Swedish children must start school at age seven, and the RSD of the exact age is only about 4%. Similarly, it might be thought rather strange that age of marriage is so tightly constrained, but there are many social factors that reduce the spread and tend to produce similarity. If all of one's friends are getting married, there is pressure to get married at a similar time. Because graduation from tertiary education is a normal transition point, age at first marriage might also converge then, and the RSD might be small.

The rule of thumb is that most environmental influences act to increase differences and enlarge the RSD, and that probably also applies to first SSA attraction. Since the degree of environmental influence increases a great deal after birth; one of the clearest illustrations of minimum milestone variability (i.e., relatively small RSD) is prenatal development. Such data is available and can be calculated now from MRI scans and ultrasounds, as shown in Table 2.

Table 2. Prenatal Milestones

<i>Milestone</i>	<i>Reference</i>	<i>Mean Years</i>	<i>RSD%</i>
Size of 10 mm fetal sac	(Creighton University Medical Center, 2006)	0.115	4.1
First head rotation	(Creighton University Medical Center, 2006)	0.200	4.6
Singular sulcus development	(Garel et al., 2001)	0.433	2.2
First arm movement	(Kurjak et al., 2006)	0.538	3.8

RSD expressed as time since conception.

The mean of these relative standard deviations (RSDs) is 3.7%, which is less than the lowest *postnatal* milestones in Table 1.

For purely genetic influence, there is evidence from colonies of laboratory mice that the degree of timing-spread might be even lower (Murray et al., 2010). For laboratory mice, with environmental conditions held very constant by researchers, the timing of gestation has a relative standard deviation of about 1.9%. This varies a little depending on the particular mouse strain. This is lower than the prenatal RSDs for humans in Table 2, but for a fuller comparison with humans, more research is needed.

First Attraction Conceptual Difficulties

The concept of first sexual attraction is now discussed in light of the above background. The concept of attraction is more fundamental than sexual identity, because the latter will have a significant social input; similarly, a behavioral criterion is possibly unreliable. The first attraction data under consideration, although apparently more fundamental, do not necessarily involve genuine erotic arousal and may be less clear-cut than one might imagine. The answers obtained to questionnaires designed to gather data

on first attraction depend on how the questions are framed (Rich Savin-William, personal communication, June 2009). The first attraction may consist of admiration, fascination, or hero worship, and may only later become sexualized. It is assumed here that any reported first attraction has at least a sexualized tinge (Herdt, McClintock, Henderson, Lehavot, & Simoni, 2000).

Another criticism of the attraction data is that adult memories of age of first attraction may be imprecise and unreliable. However, it is reassuring that the test-retest reliability of first attraction age is good (Schrimshaw et al., 2006) and little different from those for sexual identity realization and first same-sex encounter, which are likely to be better remembered.

First Attraction Literature Data

Kinsey, Pomeroy, and Martin (1948) and Kinsey, Pomeroy, Martin, and Gebhard (1953)—the first to investigate sexuality on a really large scale—give lots of sexual data with age, but ironically none on first attraction. They accumulated data on first arousal instead, and by this they explicitly meant physiological arousal, not just attraction. In a review of the literature, Herdt and colleagues (2000) cite the first published calculation of a first-attraction age (ten years) as a long time after the work of Kinsey et al. (Saghir & Robins, 1973).

Since Saghir and Robins (1973), there have been many subsequent studies that measured first attraction (see Table 3). Some studies give only an estimate of the age, while others also give the standard deviation of the age, or enough information so that a standard deviation may be calculated.

Table 3. Mean Ages for First Same-Sex Attraction

<i>Reference</i>	<i>Mean First Attraction</i>	<i>Comment</i>
Remafedi, Farrow, & Deisher (1991)	10	Both sexes combined
Savin-Williams (1995)	9.6±3.6, 10.1±3.7	Male/female
Bailey & Oberschneider (1997)	10.4	
D'Augelli, Hershberger, & Pilkington (1998)	10±4	Both sexes combined
Savin-Williams (1998)	7.5±3 10.5±6	Male/female
D'Augelli et al.(2005)	10±3.4	Both sexes combined
Schrimshaw et al. (2006)	10.9±3.8	Both sexes combined
Floyd & Bakeman (2006)	11.4±4.8 15.3±6.9	Male/Female
McCabe, Hughes, Bostwick, Morales, & Boyd (2012); McCabe et al. (2012)	10	
Grossman (2008)	12.9± ca. 7, and 9.8±3.5	Two estimates: men only
Corliss, Cochran, Mays, Greenland, & Seeman, (2009)	16±8	Women only. May include attractions other than first.

The mean and standard deviation of the measurements for age at first same-sex attraction for the twelve studies listed in Table 3 is 10.0±4.0 years for both sexes.

The Whitam and Mathy (1986) study of males and the Whitam, Daskalos, Sobolewski, and Padilla (1998) study of females give cross-cultural data that is consistent

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with the studies cited in Table 3. In the Witam et al. studies (see Tables 4 and 5), standard deviations for first same-sex attraction were calculated from age ranges provided rather than from year-by-year data. Note that the measured ages of first opposite-sex attraction (OSA) also is included. Comments about the comparison between age of first SSA and OSA are offered in the technical appendix.

Table 4. Age for First SSA for Males

	<i>Brazil</i>	<i>Guatemala</i>	<i>Philippines</i>	<i>USA</i>
SSA	10.6±5.5	8.2±4.9	11.4±3.4	10.9±4.5
OSA	11.6±2.9	9.1±4.2	11.8±3.3	10.3±4.8

(Whitam & Mathy, 1986) Values are years, and errors are one standard deviation

Table 5. Age for First SSA for Females

	<i>Brazil</i>	<i>Peru</i>	<i>Philippines</i>	<i>USA</i>
SSA	14.8±6.9	14.7±7.2	15.2±6.1	13.7±7.3
OSA	12.5±2.8	12.4±3.7	15.1±3.2	9.9±3.6

(Whitam et al., 1998) Values are years and errors are one standard deviation.

Overall, the RSDs are similar to the data in Table 3—that is, the standard deviations are a large fraction of the ages rather than a small fraction.

In their review of the literature, Herdt et al. (2000) describe data from various primitive and sophisticated cultures and estimate that the first attraction (for both SSA and OSA) occurs at age ten. This is interpreted as evidence of a biological origin for first

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attraction. The title of their paper is the memorable phrase: “The Magical Age of 10.”

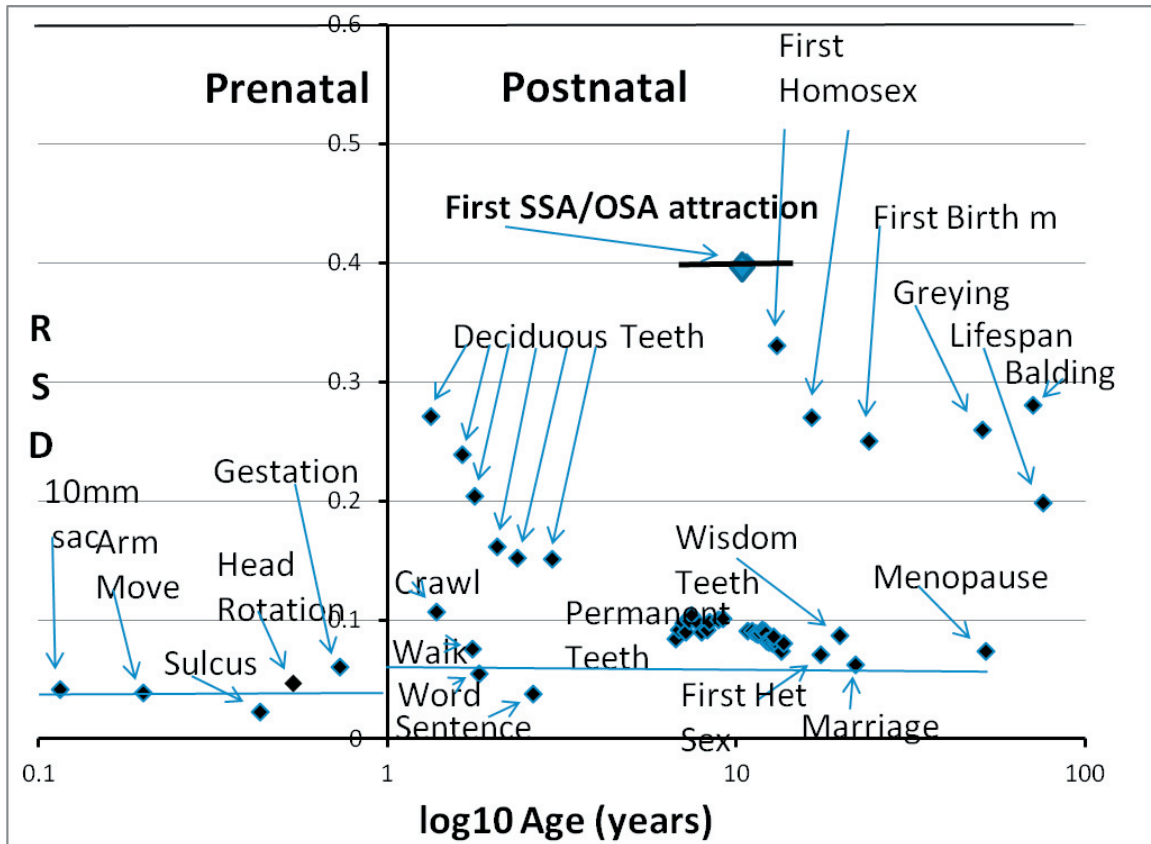
Significantly, the age tabulated in their work does not correlate with the measured age of puberty. This is problematic as evidence for the biological origin of first attraction.

At least in the United States, in more than thirty years of studies—from Saghir and Robins (1973) to Corliss et al. (2009)—measured age of first attraction has changed little. While the age of puberty in the West has decreased considerably over several decades (Katiala-Heino et al., 2003; Kinsey et al., 1948, 1953), in some of the primitive cultures, normal puberty occurs as late as age 19. Herdt et al. (2000) claim that since the age of first attraction is not changing, this must mean that first SSA (and OSA) are biologically programmed and occur independent of puberty and culture. In effect, they assert that age of first attraction is much more tightly biologically constrained than the age of puberty itself, which is very unlikely. The data in the present paper refute this interpretation because the spread in the timing of first attraction is much too large when compared with the age of puberty.

Comparison of Developmental Milestones with First SSA and OSA and First SS and OS Sexual Initiation

The RSD for all of the previously tabulated data on developmental milestones and first SSA (see Tables 1–5) are compared below in Figure 1. The larger the RSD, the wider the spread in the data.

Figure 1. Developmental Milestone RSDs Combined with RSD for First SSA



In the figure, *First Homosex* and *First Het Sex* are data points for first intercourse/initiation for homosexual and heterosexual respectively.

The highest horizontal thick line for *First SSA/OSA Attraction* emphasizes the 40% relative standard deviation, compared with other lesser relative standard deviations elsewhere in Figure 1; the enlarged diamond is merely for emphasis. This figure shows visually the point in this paper that most biological events are more tightly clustered in age than first attraction. For example, menopause occurs over a restricted age range, but graying of hair is much more variable in age. Lines indicating approximate lowest RSDs for prenatal and postnatal developmental events are included. The OSA first attraction RSD point, which is the same as for SSA, was derived from Tables 4 and 5.

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In Figure 1 the values for the relative standard deviation statistic are much higher for deciduous (baby) teeth than for permanent teeth. This is reasonable, because it is less important that deciduous teeth erupt at fixed times.

It is interesting that even walking and first verbal production seem restricted in time to a surprising extent. In contrast, events like balding and lifespan are much more heavily influenced by the environment. Any genetic heterogeneity is included in the table/figure data and could increase some RSD results.

The same-sex milestones have much larger RSDs than the heterosexual ones. It would be tempting to say that this is the result of societal pressures interfering with SSA, and making the ages at which milestones occur more variable, but this is not correct because the OSA first attraction RSD is similar to the SSA first attraction RSD (and very different from the other OSA milestones). This means either that the concept of “first attraction” is quite unsuitable as a measure of sexual orientation, or similar influences are impacting both.

Comparison of Relative Genetic Influence of Specific Developmental Milestones

Table 6 shows the genetic influence on milestone timing, where known.

Table 6. Percentage Genetic Influence from Twin Studies

<i>Milestone</i>	<i>Reference</i>	<i>% Genetic Influence</i>
Gestation length	(Clausson, Lichtenstein, & Cnattingius, 2000)	31 ^a
First crawling, walking	Not found	
First word, sentence	Not found	
Teeth eruption timing	(Townsend, Hughes, Luciano, Bockmann, & Brook, 2009)	94
Puberty timing	(Silventoinen, Haukka, Dunkel, Tynelius, & Rasmussen, 2008)	91
First heterosex	(Dunne et al., 1997)	72, 49 (M/F)
Marriage	(Trumbetta, Markowitz, & Gottesman, 2007)	27 ^d
Graying	Not found	
Balding	(Rexbye et al., 2005)	79 ^b
Menopause timing	(de Bruin et al., 2001)	86
Lifespan	(Hjelmborg et al., 2006)	26 ^c

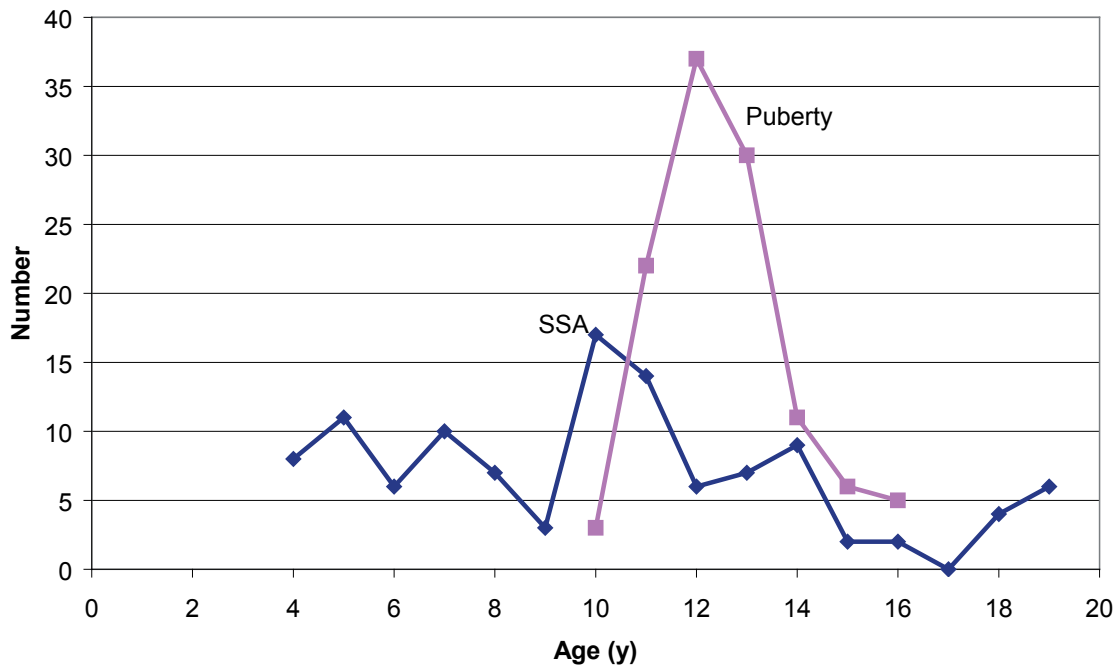
(a) Mother gene influence only—there is also a contribution from the fetus. (b) To a mean baldness criterion rather than age. (c) For 96-year-olds. (Similar results for 2 individual decades previous.) (d). Maximum from ages 20–40, but is RSD on marital status, not RSD on age.

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We now compare the RSD on points with a known high genetic influence (more than 50%) from Table 6, such as teeth eruption, puberty, first heterosexual intercourse, balding, and menopause. The mean and standard error of the mean for RSD of these selected milestones are 0.120 ± 0.031 . This is very statistically different from the 0.40 for RSD of first attraction ($P < 0.001$) so presumably both SSA and OSA first attraction do not have a predominant genetic component.

For a more specific example, the data for first SSA and puberty for males—derived and redrawn from Hamer, Hu, Magnuson, Hu, and Pattatucci, (1993)—are particularly clear, because they are given separately for each year of age rather than as summary statistics. In Figure 2, note that the data for first SSA are very spread out, compared with the data for puberty.

Figure 2. Male First SSA Attraction (Hamer et al, 1993) The numbers are per year.



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From Figure 2, SSA age-occurrence is not like the genetically influenced shape of puberty. The two means and standard deviations are respectively 10.0 ± 4.1 years, and 12.5 ± 1.4 years; very different at the $p < 0.001$ level by either a t-test or the Levene test for homogeneity of variance.

Using the known very strong genetic influence on puberty timing, the likely genetic influence on first SSA is calculated in the technical appendix. However, the conclusion from the comparison as seen in the figures is that the genetic influence is low and other influences predominate. There is no support for the idea that first attraction is an innate, or inevitable, developmental milestone.

The Possible Involvement of Adrenarche (Full Adrenal Maturity)

Herdt et al. (2000) speculate that the “magical age of 10” may be due to adrenarche, which is a biological milestone. Adrenarche is the first achievement of full adrenal maturity, the point at which androgenic hormones are produced to mature levels and which has been observed to occur also at age ten (Auchus, 2011). Adrenal maturity occurs independent of puberty. It is possible to have puberty without adrenarche (as in the case of adrenal failure), and adrenarche without puberty (as in Turner’s syndrome), and sexual attraction will still develop in either case (this example is for OSA). Auchus mentions that adrenarche is not an abrupt and signaled process occurring in mid childhood but rather a continuous process since birth. It therefore is not only independent of puberty but a different type of process and very spread out over time.

One possibility is that first attraction might be due to some prolonged genetic influence connected with hormones from the adrenal gland, which theoretically might explain the spread-out nature of first attraction. However, this seems very unlikely, given the example of girls with congenital adrenal hyperplasia and OSA (Meyer-Bahlburg, Dolezal, Baker, Ehrhardt, & New, 2006). Such girls have grossly excessive androgen production from well before birth, but not excessive attraction to the opposite sex; in

fact, they have less attraction to the opposite sex. A small but increased proportion of girls with this condition is attracted to the same sex. These girls are not born precociously attracted to the opposite sex; rather they become attracted to the opposite sex in a way similar to those exposed to normal hormone levels. This suggests adrenarche is likely to be a quite minor influence on first attraction.

A Social Hypothesis

An alternative hypothesis is that social environmental factors strongly influence the development of first same- (and opposite-) sex attraction. As described (Whitehead & Whitehead, 2010), the age of ten also coincides with an approximate peak in the differential social gender-development of each sex. For several years after birth, boys and girls have been following the diverging psychological trajectories appropriate to their sex (or for SSA children, often not following them). Having developed social gender characteristics that differ from the opposite sex, boys and girls commonly begin to be interested in those differences, and even attracted to those who are different. This is essentially the “exotic becomes erotic” idea of Bem for OSA, as well as SSA (Bem, 1996). Some of the spread in age at first attraction could simply derive from the variability in time required for encountering a person who is perceived as attractive. Twin studies have shown that romantic opposite-sex attraction has zero genetic influence for adults (Zietsch, Verweij, Heath, & Martin, 2011). Work using a quite large sample of adolescent twins found the same for same-sex attraction in teenagers, i.e., no genetic influence (Bearman & Brueckner, 2002). There seems little doubt that a similar survey for first attraction prepuberty would have a similar result.

A strength of the current paper is that the standard combination of the concept of genetically influenced developmental milestones and the variation of their age-spread has a large and well-established literature but has never before been applied to SSA. This is a fresh approach to the problem of genetic influence that is normally tackled by twin

studies or family studies. First SSA has such a wide relative standard deviation compared to other clearly genetically influenced milestones that it seems clear the appearance of first SSA is only weakly influenced by genetics. This means that the common belief that people with SSA are “born that way” is not supported by the literature on first attraction.

A possible limitation to this conclusion is that measuring first attraction is commonly done by asking only one question in a retrospective, self-report survey. Also, since the concept of “attraction” is so multifaceted, more research is needed to allow for a fuller exploration of this topic. For the present comparison, puberty was not too different in age from first attraction, but other comparisons with wider age disparities may introduce extra mathematical uncertainty.

Conclusions

Although it is common to hear that first same-sex attraction coincides with earliest memories, numerous surveys show this is a very misleading generalization—half of all reported first attractions are later than age ten. It is doubtful whether this attraction is more than a possible harbinger of possible future sexualized attraction, particularly for SSA. Its very spread-out occurrence in time (about 40% relative standard deviation) makes it nearly impossible that it is predominantly biologically influenced. Human post-natal events that are known to be biologically preprogrammed have a much smaller relative standard deviation of about 7% and prenatal events of about 4%. It is also doubtful that adrenarche—adrenal maturity—is an adequate explanation for this “magical age of 10.” A social explanation based on the development of psychosexual gender differences is more plausible.

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Technical Appendix

In this appendix, data from tables in the body of the paper are used to estimate the genetic and other influences on timing of first attraction. This method is a minor novelty in the literature, but follows from the mathematics used. Comparison of variances is universally employed, but rarely applied to milestones.

The data for timing derived from Figure 2 are 10.0 ± 4.1 years, and 12.5 ± 1.4 years for first SSA and puberty respectively. The variance of these measures, which is the square of the standard deviation of each mean, is used. Therefore, variances of 4.1^2 and 1.4^2 or 16.81 and 1.96, were compared. The genetic contribution to the timing of puberty from Table 6 is 0.91 or $1.96 * 0.91$ or 1.78 (because the genetic contribution to puberty is only 91% instead of 100%). Other things being equal, we compare 1.78 units of variance contribution for timing of puberty with 16.81 units of variance for first SSA. This means that the genetic contribution to first SSA is about 10%. It could possibly be somewhat less, because the mean age of 10 for first SSA is less than the mean age of 12.5 for puberty (see Figure 2). For a general conclusion, it is enough to know that the genetic contribution to the variance of first same-sex attraction timing is weak rather than overwhelming. The result is similar to a previous estimation from twin studies by a quite different method (Whitehead, 2011).

Figure 2 does not give information we could use to repeat the calculation for OSA. Some first attraction ages (standard deviation in parentheses) can be used from Tables 4 and 5, though they are less precise than the Figure 2 data, i.e., OSA(m) 10.3 (47%) OSA(f) 9.9 (36%).

These two results for OSA again indicate a weak effect of genetics, for both males and females and in the order of 10%.

It may surprise readers that the genetic contribution to OSA timing is apparently so low. While it is quite widely assumed that one is “born OSA,” there has been only

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one other quantitative test of this hypothesis (Hershberger, 1997). Hershberger tested the existence—not the timing of first appearance—of OSA contrasted with other sexual orientations and found that the genetic contribution to OSA was 18 to 26%. This is a weak to modest influence and puzzling in light of the general assumption that heterosexual orientation is genetically inherited. But this finding apparently has received no subsequent comment. The present finding is reasonably consistent with Hershberger's work, though for the measurement of the timing of appearance rather than the perceived existence of the orientation itself. This implies that nongenetic factors, such as the role of family and society in developing OSA, are much greater than usually thought and the role of genetics much less (Whitehead & Whitehead, 2010). An alternative interpretation is that “first attraction” is not a reflection of adult sexual orientation and either should not be used, or should be used only with caution.