

## The Relation Between Sexual Orientation and Penile Size

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*The relation between sexual orientation and penile dimensions in a large sample of men was studied. Subjects were 5122 men interviewed by the Kinsey Institute for Research in Sex, Gender, and Reproduction from 1938 to 1963. They were dichotomously classified as either homosexual (n = 935) or heterosexual (n = 4187). Penile dimensions were assessed using five measures of penile length and circumference from Kinsey's original protocol. On all five measures, homosexual men reported larger penises than did heterosexual men. Explanations for these differences are discussed, including the possibility that these findings provide additional evidence that variations in prenatal hormonal levels (or other biological mechanisms affecting reproductive structures) affect sexual orientation development.*

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**KEY WORDS:** sexual orientation; penile dimensions; hormones; homosexuality; Kinsey; men.

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### INTRODUCTION

Biological factors, such as prenatal hormonal conditions (e.g., testosterone levels), have been argued to be important in the development of sexual orientation (e.g., Ellis and Ames, 1987). However, one criticism of these approaches (e.g., Byne and Parsons, 1993) is that there is little evidence that homosexual men and women differ from heterosexual men and women in genital morphology, a plausible consequence if biological factors affecting reproduction, such as prenatal hormonal levels, underlie sexual orientation. It is clear, for example, that alterations in typical levels of prenatal hormones can affect genital size and morphology, as evidenced by girls with congenital adrenal hyperplasia (CAH) who have masculinized genitalia

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as a result of exposure to higher-than-typical levels of androgens prenatally or by boys who have demasculinized genitalia as a result of androgen insensitivity syndrome (e.g., Conte and Grumbach, 1995). However, although women affected by CAH have been found to have elevated rates of same-sex attraction (e.g., Zucker *et al.*, 1996), these women make up only a very small percentage of the people with same-sex attraction.

A number of factors may account for why heterosexuals and the vast majority of homosexuals do not seem to differ in genital morphology. One explanation concerns the timing of prenatal hormonal fluctuations responsible for alterations in sexual orientation on the one hand and the timing of possible alterations in genital morphology on the other. For example, there is evidence that the beginning of genital differentiation (e.g., development of the penis) occurs earlier in gestation (i.e., at 8 weeks) than the beginning of the differentiation of brain structures putatively relevant for sexual identity and sexual orientation (Ellis and Ames, 1987). Thus, a small alteration in testosterone levels in one phase of pregnancy may not alter genital development in any detectable way, but it may have effects on brain structures (e.g., hypothalamus) relevant for sexual orientation.

A related explanation is that alterations in typical prenatal hormones do not always affect genital growth. For example, two recent studies of CAH boys, who have been exposed to higher-than-typical levels of prenatal and prepubertal androgens, did not find different lengths of penises in these boys relative to controls, although the samples of the CAH boys were small ( $n = 9$  and  $n = 12$ ) and 2 of the boys in one CAH sample had micropenis (Levy and Husmann, 1996; Sutherland *et al.*, 1996). In addition, researchers administering higher-than-typical dosages of prenatal testosterone did not induce more-than-typical growth in a small sample of human fetal penile specimens (Baskin *et al.*, 1997). This recent research suggests that individual differences in adult phallic size may be largely the result of differences in the inherent genetic structure for organogenesis when stimulated by appropriate hormonal conditions. However, these research studies had, as mentioned, small samples, and it is clear that the mechanisms, including hormonal ones, responsible for the growth (and the cessation of growth) of the genitalia are not fully understood (Baskin *et al.*, 1997).

A third explanation is that this issue has not been adequately studied in large samples. A large sample may be needed to detect small variations in genital size that have occurred because of alterations in typical levels of prenatal hormones or other relevant growth and development mechanisms. One early and relatively unknown study using a reasonably large sample did find that homosexuals differ, on average, from heterosexuals in genital morphology (see Nedoma and Freund, 1961). These investigators found that homosexual men ( $n = 126$ ) had larger penises than a comparison sample of heterosexual men ( $n = 86$ ). More data are needed to establish the reliability of this finding, however. A very large data set that contains information on sexual orientation and genital size in men is the original