

## **Current Thinking Regarding the Etiology of Gender Dysphoria**

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Although the origins of being gender dysphoric can not yet be declared outright, there is a growing body of evidence that Gender Identity Disorder (GID) as described in the Diagnostic and Statistical Manual IV (1994) is at least in part, the result of insufficient or inappropriate androgenization of the brain at a critical stage of embryonic development. As a result, the affected individual may be left with between a partial and a full sense of having a cross-sexed gender identity. It is this difference that may be the root cause behind an overwhelming need to transition. Evidence of sexual differentiation of the brain has been documented in at least three different areas of research: physical measurement, case reports involving ablatio penis, and 5-alpha reductase deficiency. A fourth, behavioral theory called autogynephilia, describing how a strong desire to transition from male-to-female develops, is also noted below.

Measurement: Zhou J.-N, et al. (1997) examined the volume of the central subdivision of the bed nucleus of the stria terminalis (BSTc), and found that a female-sized BSTc was found in male-to-female transsexuals. This led them to declare that a female brain structure exists in genetically male transsexuals, supporting the hypothesis that gender identity develops as a result of an interaction between the developing brain and sex hormones.

In a follow-up study KRUIJVER et al. (2000) wanted to know if the reported difference according to gender identity in the central part of the bed nucleus of the stria terminalis (BSTc) was based on a neuronal difference in the BSTc itself or a reflection of a difference in vasoactive

intestinal polypeptide innervation from the amygdala. To do this they looked at 42 subjects to determine the number of somatostatin-expressing neurons in the BSTc in relation to sex, sexual orientation, gender identity, and past or present hormonal status. They found that regardless of sexual orientation, men had almost twice as many somatostatin neurons as women. The number of neurons in the BSTc of male-to-female transsexuals was similar to that of the females, while the neuron number of a female-to-male transsexual was found to be in the male range. Hormone treatment or sex hormone level variations in adulthood did not seem to have influenced BSTc neuron numbers. They go on to declare that their " findings of somatostatin neuronal sex differences in the BSTc and its sex reversal in the transsexual brain clearly support the paradigm that in transsexuals sexual differentiation of the brain and genitals may go into opposite directions and point to a neurobiological basis of gender identity disorder."

Historical reports involving ablatio penis : There are two reported cases in which one twin of identical twins lost his penis in a circumcision accident shortly after birth. In both cases, believing that gender identity was primarily culturally learned, doctors advised the parents to rear the boy who had lost his penis as a girl. (Money, 1975). However, in both cases, despite their female rearing and the introduction of feminizing hormones at puberty, the children eventually rejected their assignment as female and are now living their adult lives as males. Diamond, M., (1982); Diamond, M. & Sigmundson, H.K. (1997)

5-Alpha Reductase Deficiency: This is a situation in which a 46XY genetically male child is born with such significantly undeveloped male genitalia that he is assigned and reared as female. 5 alpha-reductase is

an enzyme that converts testosterone into dihydrotestosterone, an androgen that is needed for the completion of the differentiation of male genitals in utero. Despite the lack of the enzyme, these individuals retain androgen receptivity and the ability to synthesis testosterone. As a consequence, at puberty they develop male secondary sex characteristics and, in a significant number, revert to the masculine gender role.

Imperato-McGinley, J., Peterson, R.E., & Gautier, T., & Sturla, E., (1979).

Autogynephilia: A fourth hypothesis is behavioral based. The name for this condition is autogynephilia; the love of one's self as a woman. The term was introduced into the literature by sexologist Ray Blanchard (1989a, 1989b) and was further developed by physician and sexologist Anne Lawrence (1998). Neither Blanchard nor Lawrence claim that the condition causes gender variant behavior. Instead they show quite clearly that for a majority of genetic males who are gender dysphoric, a life long behavior of sexual self gratifying experiences ( masturbation tied to crossdressing tied to fantasizing of one's self as a woman) may play a strong role in entrenching an overwhelming desire to transition.

It should also be noted here that the treatment regimen for gender dysphoria (HBIGDA, Standards of Care) calls for the introduction of cross-sex hormones as the second phase of a triadic treatment plan. Genetic males receive large doses of estrogens and genetic females receive large doses of androgens (testosterone). In virtually every case in which the individual is truly transgendered, there is a marked sense of relief from anxiety. It is as if there are receptor sites in the brain of these individuals that are starved for the cross-sex hormones their body is otherwise unable to provide for them.

As a consequence I have taken as my working theory that the gender variant condition is physiological in origin, that it is an innately anxious condition and that it must be addressed by physical and/or psychological means if the individual is to attain peace of mind.

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