

IS THERE A ‘FIXED BIOLOGICAL BASIS FOR GENDER IDENTITY’?

There is a short and easily overlooked passage in the Endocrine Society’s recent Scientific Statement on “Considering Sex as a Biologic Variable in Basic and Clinical Studies” (Bhargava et al. 2021) which raises some rather interesting questions.

The passage in question is to be found some way into a section of the paper headed “Biological Basis of Diversity in Sexual/Gender Development and Orientation” and reads as follows:

“Although gender is strongly influenced by environmental and cultural forces, it is unknown if the choice to function in society in male, female, or other role(s) is also affected by biological factors (89-91). A general issue is that the association of sex, gender, or sexual orientation with specific brain structures, or with other biological variables, does not establish whether the biological variables are causes or consequences or noncausal correlates of the behavioral characteristics or function of the individuals studied.”

In simple terms, what the Endocrine Society is stating here is that the assertion that gender identity is in some way innate and directly influenced and/or determined by biological factors is, at least for the time being, unproven based on current evidence, a view for which the paper provides three supporting references, numbers 89-91 inclusive.

Of particular interest here is the second of those three references (90), a 2015 review article by Aruna Saraswat, MD; Megan Weinand, BA, BS; Joshua D. Safer, MD from the journal “Endocrine Practice” entitled “Evidence Supporting the Biological Nature of Gender Identity” (Saraswat et al. 2015).

This particular paper is of interest for two reasons. First and foremost because it advances a conclusion that seems to be markedly at odds with the position set out by the Endocrine Society, namely:

“Current data suggest a biological etiology for transgender identity. Studies of DSD patients and neuroanatomical studies provide the strongest evidence for the organic basis of transgender identity. Sample sizes of the majority of studies to date on this subject are small, and conclusions must be interpreted with caution. Further research is required to assign specific biological mechanisms for gender identity.”

The second feature of this paper that makes it of particular interest is the background and identity of its corresponding author, Joshua D Safer MD, for reasons that should be obvious from this extract from his official biography on the website at the Mount Sinai Hospital in New York City.

“Joshua D. Safer, MD, FACP, FACE is the Executive Director of the Mount Sinai Center for Transgender Medicine and Surgery (CTMS) and Professor of Medicine at the Icahn School of Medicine at Mount Sinai.

Dr. Safer was previously the founding Medical Director of the Center for Transgender Medicine and Surgery at Boston Medical Center and Boston University School of Medicine. Dr. Safer earned his medical degree from the University of Wisconsin. He completed his internal medicine residency at Mount Sinai Beth Israel and his endocrinology fellowship at Beth Israel Deaconess Medical Center in Boston.

Dr. Safer was the inaugural president of the United States Professional Association for Transgender Health (USPATH). He also serves on the Global Education Initiative committee for the World Professional Association for Transgender Health (WPATH) and on the Standards of Care revision committee for WPATH. Dr. Safer was a co-author of the Endocrine Society guidelines for the medical care of transgender patients and has been a scientific co-chair for WPATH's international meetings.”

I think it would be fair to say that Dr. Safer and his co-authors - one of whom is, somewhat ironically, ‘deadnamed’ in the official citation for this paper – are pretty well invested in the idea that gender identity has biological foundations as is evident from the rather confident, if not overconfident, manner in which they introduce their review paper.

“Gender identity is a fundamental human attribute which has a profound impact on personal well-being. Transgender individuals are those whose lived and identified gender identity differs from their natal sex. Various etiologies for transgender identity have been proposed. Misconceptions which assume that gender identity can be altered still exist. However, clinical experience with treatment of transgender persons over the years has clearly demonstrated that the best outcomes with these individuals are achieved with their requested hormone therapy and surgical sexual transition, as opposed to psychiatric intervention alone (1). In this manuscript we will review the data which support a fixed, biological basis for gender identity.”

So, what we have here certainly appears to be a significant mismatch in interpretation between the views set out in this review paper and those set out by the Endocrine Society’s position paper.

[From this point on I will be referring to this paper using the name of its lead author, Aruna Saraswat, not least because it is unclear as to the exact extent of Safer’s own contribution to the paper or even if that contribution extends much beyond lending his name and professional status/reputation to it.]

Now I don’t propose here to work through the Saraswat paper in its entirety. Rather I want to highlight a couple of issues that I think call into serious question the reliability of Saraswat’s arguments and conclusions.

The first of these takes us back to the passage from the Endocrine Society’s position paper that I quoted earlier, specifically the observation that:

“A general issue is that the association of sex, gender, or sexual orientation with specific brain structures, or with other biological variables, does not establish whether the biological variables are causes or consequences or noncausal correlates of the behavioral characteristics or function of the individuals studied.”

While the Saraswat paper notes caveats to the interpretation of a number of the studies incorporated in its review of extant literature, nowhere is this issue of causality and the problems inherent in establishing it from current evidence addressed in the paper. Rather, the overall tone of the paper and the presentation of evidence suggests that causality is being assumed rather than questioned, investigated and established based on supporting evidence.

A more specific concern arises in respect of the presentation and interpretation of the first substantive reference provided in the paper, a 2005 paper by psychologist Dr. Heino Mayer-Bahlberg of Columbia University, which was published in the journal “Archives of Sexual Behaviour” under the title “Gender Identity Outcome in Female-Raised 46,XY Persons with Penile Agenesis, Cloacal Exstrophy of the Bladder, or Penile Ablation”.

The Mayer-Bahlberg review paper pulls together evidence from several studies of natal males with severe congenital conditions affecting the development of the external genitalia, a number of whom were surgically assigned as female in childhood and raised, at least initially, as females. I should stress at this point that the congenital conditions included in the Meyer-Bahlberg review are not amongst the subset of Congenital Conditions of Sex Development (CCSD) that can be correctly characterised in clinical, though certainly not in social, terms as ‘Intersex’. There was, and is, no uncertainty whatsoever about the natal sex of the individuals included in these studies. They were biologically male – except where specific reference is made to comparative results from studies of natal females with similar conditions.

As to why they were surgically assigned to the female sex in childhood, Mayer-Bahlberg offers this account:

“Clinical policies of gender assignment in new-borns with ambiguous genitalia are dependent on (1) the clinicians’ theoretical assumptions concerning the determinants of gender, (2) the relative importance attached to outcomes such as gender dysphoria/gender change, fertility, sexual functioning, sexual orientation, and general quality of life, and (3) the medical treatment options available at the time (e.g., sex hormone treatment and specific techniques of genital surgery). The assumptions concerning the determinants of gender are partly based on the results of outcome research on intersex persons, in whom one or more of the biological mechanisms of sexual differentiation are compromised, particularly the prenatal sex-hormonal milieu, which is known to affect both body and brain.”

However, a more cynical view of such practices emphasises the role of surgical expediency in such decisions, as exemplified by the observation that “it’s easier to dig a hole than build a pole”, but that’s a debate for another occasion.

So, getting back to the Mayer-Bahlberg paper, this is how it is initially presented by Saraswat in her paper.

“A seminal study by Meyer-Bahlburg et al involving outcomes of XY individuals raised as females due to severe non-hormonal, anatomic abnormalities of sex development has provided the most convincing evidence that gender identity is fixed (2). These congenital abnormalities include penile agenesis, cloacal exstrophy, and penile ablation.”

So, according to Saraswat, this particular study provides “**the most convincing evidence that gender identity is fixed**”. Keep that statement firmly in mind as you read what follows, as Saraswat’s paper then adds:

“For many years, female gender assignment along with surgical feminization was the dominant approach for these patients. In this study, it was observed that 78% of all female-assigned 46 XY patients were living as females While the majority of these patients did not initiate a gender change to male, none of the 15 male-raised 46 XY patients initiated a gender change to female. Thus, risk of questioning gender identity was higher in those patients raised as females than in those raised as males among 46 XY subjects with one of these conditions.”

So, the most convincing evidence that gender identity is ‘fixed’ [by biological factors] is a study in which almost four-fifths of the natal males that were surgically assigned and raised against their natal sex retained a female identity at the point at which the studies in which they were included conducted their last follow-up review of their personal development.

As a demonstration of biological fixity, a 22% rate of reversion to a male identity hardly seems that convincing.

Now let’s look at how Meyer-Bahlberg presents his results by, to begin with, adding a little more detail to the reported results.

“Table VI summarizes the data within and across syndromes. As the bottom section shows, of the female-assigned patients of childhood age, 69% (including those with possible gender dysphoria) or 62% (excluding those) were living as females, of those of adolescent age 91% (including those with possible gender dysphoria) or 68% (excluding those), of those of adult age 65% or 47%, and of those whose age could not be categorized 100%, altogether across all ages 78% or 65%.

So, the number of female assigned/raised males who retained a female identity without signs of possible gender dysphoria does appear to fall markedly in adulthood, by which time they are highly likely to have been informed of, or become aware of their clinical history, but even in this group slightly under half retained a female identity as Meyer-Bahlberg notes in introducing his discussion of these results.

“The main findings can be summarized as follows:(1) The majority of 46,XY individuals with presumably normal-male prenatal hormonal milieu, non-hormonal anatomic abnormalities of the genitals, and female gender assignment at birth or in early childhood, have not changed gender to male and, for those living as females, gender dysphoria or an explicit wish to change gender have rarely been reported or suspected. This is so across all age categories, although there is a modest trend of an increasing number of such individuals switching to male with advancing

age. Even by adulthood, however, at least about half maintained their female gender, including patients who were fully aware of their medical history.”

He then goes on to add:

“These data do not support a theory of full biological determination of gender identity development by prenatal hormones and/or genetic factors, and one must conclude that gender assignment and the concomitant social factors have a major influence on gender outcome. On the other hand, a number of female-raised individuals did change gender to male and others developed a possible gender dysphoria, which indicates that gender assignment does not dictate gender outcome either. Thus, in the conditions described here, female gender assignment of 46,XY infants and young children carries a risk of later patient-initiated gender change to male that is considerably higher than the risk of patient-initiated gender change to female in male-raised patients, no example of which has been reported in any of the publications we have examined. The risk is also much higher than the rate of gender change (transsexualism) in the general population (maximum population estimate for transsexualism in a Western country has been reported as 1:10,000 in men and 1:30,000 in women (Bakker et al., 1993).”

Remember, this is ostensibly (according to Saraswat) the most convincing evidence that gender identity is fixed (i.e. innate/biologically determined) and yet Meyer-Bahlberg states clearly that the data in these studies do not support such a conclusion and that “gender assignment and the concomitant social factors have a major influence on gender outcome”. It should be noted that gender assignment, here, means not only surgical assignment but includes also the social and legal ‘transition’ of gender identity necessary to raise a natal male as a female, insofar as this was possible at the time.

Meyer-Bahlberg’s findings come with a considerable number of caveats, not least the small size of the included studies in which a total of just 77 natal males assigned and raised as female were included, due to the rarity of the conditions under examination, and the poor quality of assessment methods used in most reports, which means that it is entirely possible that the incidence of both gender change and possible gender dysphoria could be either under or over-reported, as much of this data was collected second-hand from parents.

“The crucial data on gender dysphoria and gender change were mostly based on unsystematic follow-up communications with the parents and not on evaluations of the patients themselves. Most of the gender changes listed in this study were reported at variable periods of time after the systematic research protocol had been administered. The assessments were not done in masked fashion nor were masked co-raters or other forms of independent verification used. It is also remarkable that most of the cases of gender change to male in Table V come from this report as well as from one other report by the same first author (Reiner & Kropp, 2004), which makes future replication by independent investigators all the more important.”

The unnamed first author paper referred to here (Reiner & Gearhart, 2004) makes these pertinent observations on the interpretation of its data:

“Follow-up interviews were not systematic in that, except for the four questions listed in the Methods section, the content and order of the questions varied among the subjects and between follow-up sessions. Although genetic females with cloacal exstrophy were observed clinically, none were included in the study. Spontaneous sex reassignment in children is very rare, however. Finally, the assessment of phenotypic sex itself may have altered parenting: at various times after the initial study assessment, the parents of four subjects assigned to female sex revealed the subjects' birth status to the subjects, contrary to clinic teaching.

Parental attitude toward child rearing based on sex assignment and parental effectiveness are imponderable variables. Thus, conclusive and retrospective determination of factors that were specific to individual parents is not possible. Parents provided assurances that they raised their genetic males as girls as well as possible, given behavior that was often seen as being more typical of boys.”

Before going on to add, by way of a rescue argument:

“That 12 subjects had genetically female sisters who did not have atypical sexual behaviors offers some evidence that parents did use female sex-of-rearing practices. The scope of the subject matter and topics addressed by the questionnaires, the consistency of parents' and subjects' responses over a period of years, and longitudinal persistence of the subjects' declarations of their sexual identity suggest the reliability of outcome data.”

Can we really assume, just from the presence of female siblings, who apparently did not any sex-atypical behaviours in many of these the households, that parents were consistent in using ‘female sex-of-rearing’ practices with children that were known to have been natal males and who frequently exhibited male-typical patterns of behaviour?

I’m really not sure that we can.

Even if we accept that the outcome data in this study is reliable, and I see no reason not to, that data offers no real insight into the potential role of parent knowledge of their child’s actual birth status as a confounding factor in this kind of research. All we have to go on are the assurances of parents that their natal male children were raised as girls ‘as well as possible’, and the parents in this study may very well have believed that to be true but it doesn’t remove the possibility that their knowledge of their sex-reassigned child’s actual birth status, couple with the observations of their child’s sex-atypical behaviour for their assigned may have influenced their parenting behaviour in ways which serve to influence their child’s sense of their own gender identity towards that of their natal sex rather than that of their assigned sex.

Nor, indeed, can we dismiss the possibility that development of the gender identities of the children in this study may have been shaped, influenced and maybe even directed over time by a growing awareness of marked differences between their own tastes, preferences and behaviours and those of their natal female siblings and a parallel recognition of similarities with male peers of the same age. Indeed, there is good reason to think, based on animal studies, that the writ of determinative biological influence is highly unlikely to extend to anything so obviously complex as gender identity.

“Developmental psychobiologists since Lehrman have documented innumerable cases in which evolved developmental outcomes require a rich and highly specific developmental environment. In rhesus macaques, for example, the recognition of emotional expressions in conspecifics and the ability to cooperate in agonistic interaction depend on infant social interaction for their development (Mason, 1985).” (Griffiths, 2002)

Meyer-Bahlberg makes one further set of observations that seem particularly striking in the context of current debates and controversies surrounding paediatric transgender healthcare.

“By contrast, it seems that most, if not all, individuals with these conditions raised female showed marked masculinization of gender-role behavior where such data are available and most of those old enough were noted to report a sexual attraction to women or bisexuality (Bradley et al., 1998). (Data on sexual orientation are even more limited in number and assessment quality than data on overall gender role and gender change and were, therefore, not summarized in the Results section.) Thus, in these three syndromes, gender-differentiated behavior (including sexual orientation) appeared to be masculinized in most cases, while core gender identity development appeared much more heterogeneous.

The marked masculinization of behavior seen in these female-assigned 46,XY persons with one of the three non-hormonal conditions of genital abnormalities should not be interpreted as a sign of masculinized core gender identity. Unfortunately, the fact that DSM-IV lists behavioral criteria for GID in non-intersex children of preschool or elementary school age has sometimes led to the misinterpretation of such behaviors as components of gender identity itself. However, as we have learned from 46,XX girls with CAH, marked masculinization of gender-differentiated behavior is not synonymous with GID, gender uncertainty, or a persistent desire to change gender.”

[References omitted for brevity/clarity]

The note of caution sounded here, I think, speaks for itself.

Reading Mayer-Bahlberg’s paper in conjunction with Saraswat’s brief commentary on it one can scarcely believe them to be the same paper. There is, to my mind, no reasonable reading of Mayer-Bahlberg’s paper that would support the contention that it offers “the most convincing evidence that gender identity is fixed” outside the realms of wilful blindness, unrestrained confirmation bias or a frank admission that the evidential support for the assertion that gender identity is fixed is neither convincing or compelling at all, the latter being conspicuous, of course, by its absence.

To reiterate this last point, we are looking here at the very first substantive reference in a paper containing forty references overall and Saraswat woefully mischaracterises Meyer-Bahlberg’s finding to such an extent that one can have little or no confidence in her assessment of the evidence contained in any of those other citations let alone in her conclusion that:

“Current data suggest a biological etiology for transgender identity”

Little wonder, then, that the Endocrine Society cites Saraswat's paper as evidence which supports the view that the role of biological factors in influencing "the choice to function in society in male, female, or other role(s)" is currently unknown.

Unity – June 2021

POSTSCRIPT

Having cited Paul Griffiths' paper "What is Innateness?", which I unreservedly recommend you read in full, it would be, I think, most of remiss of me not to note the following observation from that paper.

"The innateness concept continues to promote the conflation of different biological properties in the ways that brought it into disrepute in animal behaviour studies fifty years ago. Innateness allows writers to move illicitly from the view that a trait has an adaptive history to the view that it is insensitive to environmental influences in development."

Or, to put it another way, innateness allows trans-activists to move illicitly from the view that biological differences between human males and female may exert a measure of influence over certain aspects of their behaviour (e.g. physical aggression) to the view that gender identity is insensitive to environmental influences in development.

Just think about that for a moment. Innateness, a concept that sits at the very core of contemporary trans-ideology and the affirmation-only approach to treating children with apparent variant gender identities was brought into disrepute in animal behaviour studies as long ago as the 1950's.

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