



**THE IMPORTANCE OF DESTIGMATIZATION IN GENDER
IDENTITY: A CASE REPORT**

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| <p>Article Info <i>Received 10/11/2015</i> <i>Revised 15/11/2015</i> <i>Accepted 20/11/2015</i></p> <p>Key words: Stigmatisation, gender identity, Genetic counselling, psychology.</p> | <p>ABSTRACT Gender identity is predisposed by genetic, hormonal and psychological factors. From the one hand sexual orientation is natural person’s right to express his personality features. From the other hand World Health Organization (WHO) eliminated homosexuality from international disease classification list. However up till now community has not enough knowledge, about mechanisms of sexual orientation development. Question nature versus nurture about homosexuality is already answered by science. Homosexuality is not choice but determination. Here is presented case of misunderstanding about development of homosexuality even in nowadays. Current case describes how homosexual son, was rejected by his parents because of the lack of knowledge about the origins of sexual orientation. Such parent behavior was conditioned by their thinking that homosexuality is depravity. Only after genetic and psychological counseling have parents accepted their son with his orientation. The present case show how important is to provide adequate information to the family and all community about gender identity. In present article also revises literature about hormonal, genetic and neurophysiological factors important for development of homosexuality.</p> |
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INTRODUCTION

Gender identity is predisposed by genetic, hormonal and psychological factors. From the one hand sexual orientation is natural person’s right to express his personality features. From the other hand World health organization (WHO) eliminated homosexuality from international disease classification list. However up till now some individuals have not enough knowledge, about mechanisms of sexual orientation development.

Question nature versus nurture about homosexuality is already answered by science. Homosexuality is not choice but determination. Despite there is no exact one gene that is responsible for development of homosexuality, however it is like lefthandines, that is determined as well.

CASE REPORT

Here is presented case of misunderstanding about development of homosexuality even in nowadays. To genetic counselling was admitted family with son, that has

shown homosexuality features. When the boy discovered that he is homosexual and shared this information with his parents, they rejected and expelled him from the house, despite the fact that parents were educated persons (both were teachers). Such parents behavior was conditioned by their thinking that homosexuality is depravity.

Afterwards all family came to genetic and psychological counselling at Lithuanian health science university hospital. Boys chromosomal analysis was performed and showed normal karyotype (46XY). Psychologist explained about nature of homosexuality. Afterwards relationship between parents and son become closer and mote trustful. After all parents accepted their son and his orientation.

The present case show how important is to provide adequate information to the family and all community about gender identity. In present article hormonal, genetic and neurophysiological factors



important for the development of homosexuality are described [1-5].

DISCUSSION

The discussion of current topic will consist of the summary of articles which discusses hormones influence to brain development as well as human behaviour in general.

G. Dörner starts his article with discussing a scientist Magnus Hirschfeld who can be called the initiator of sex-related studies and who started his researches as early as the end of XIX century [6-7]. He organized the Scientific Humanitarian Committee with aim of decriminalization of sexual variations. Since 1899 he published the Journal for Sex Research. In 1918 Hirschfeld founded the first Institute for Sexology in Berlin and gathered information of about 8000 homosexuals and 2000 transvestites and transsexuals, respectively. The term *transsexualism* was also introduced by Hirschfeld [5].

Hirschfeld was the first to postulate – in contrast to Freud – that genuine homosexuality occurs spontaneously and independently of postnatal psychosocial factors. This postulate was later strongly confirmed by psychologists and sociologists in a Kinsey-Institute-Report [1]. Hirschfeld described behavioural differences between homosexuals and heterosexuals occurring in childhood and observed that many homosexuals display homosexual tendencies as early as before puberty. He also emphasized the importance of Endocrinology for elucidation of the aetogenesis of sexual variations and deviations. In 1912, he postulated endocrine substances which are produced in tests as “Andrin” and in ovaries “Gynaecin” and are able to stimulate the sexual drive. Later on several researches received the Noble Prize for the isolation, characterization and synthesis of such substances. In addition, Hirschfeld postulated that a bisexual “Anlage” (Lay-out) of the brain is responsible for different developments of the direction in sexual drive. Genetic and endocrine factors were presumed to be the reason for such variations. Genuine sexuality was considered to be an inborn, natural, sexual variation [1].

On the one hand, Hirschfeld described an increased familial frequency of homosexuality as well as a high concordance in identical and discordance in nonidentical twins. This thesis of a genetic component in the aetogenesis of homosexuality was then strongly supported by Kallmann (1953) in the USA [13]. On the other hand, Hirschfeld stimulated Eugen Steinach in Vienna to implant tests and ovaries in castrated animals in the opposite sexes and to study their effects on sexual behaviour. Steinach recognized that sex-specific mating and gender role behaviour are influenced, indeed, by endocrine factors of gonads [14]. Following prepubertal castration plus implantation of the opposite gonads he achieved a certain masculinisation of psychosexual behaviour in females and feminization in males. Most of all, Steinach (1912) reported that these effects were the stronger the earlier gonadectomy and transplantation of the opposite gonads were carried out before puberty [14].

Hirschfeld had convinced Iwan Bloch, who was the founder of interdisciplinary sexology, that genuine homosexuality should be recognized as an inborn phenomenon and separated from pseudohomosexuality, i.e. occasional homosexual activities. Subsequently, Bloch postulated at the beginning of this century a chemical factor to exist in the fetus that may affect sexual orientation in later life. In 1936, Vera Dantchakoff succeeded indeed, in demonstrating that prenatal administration of the male sex hormone testosterone in female guinea pigs resulted in masculinisation of their postpubertal sexual behaviour [6,7].

Eugen Steinach also described the positive oestrogen feedback, i.e., the increased release of luteinizing hormone (LH) from the pituitary in female animals following oestrogen administration. Furthermore, he discovered the so-called “sex centre” in the brain which is affected by sex hormones and responsible for the secretion of gonadotrophins in the hypophysis. This discovery of the “hypothalamohypophysial-gonadal system” was later regarded as a decisive event for the foundation of Neuroendocrinology [14].

The following theses postulated by Dörner later had a big or even fatal influence on World Health Organization’s decision in 1990, when they removed homosexuality from the international classification of diseases. Dörner have come up with these following findings on “Sexual Endocrinology”, which were obtained by him and fellow researches in extensive animal experiments and clinical studies:

1. Different regions of the brain are responsible for male and female sexual behaviour.
2. Variations of sex-specific sex hormone levels, when occurring during critical periods of brain development, lead to permanent structural and/or biochemical changes of these brain regions, which are associated with lifelong variations sexual orientation (bi- or homosexuality). In this case the development of male and female bi- or homosexuality is favoured by a deficiency of male sex hormones (androgens) in males and their excess in females, respectively. Thus, experimental models of male and female homosexuality were developed in rats. Castration performed shortly after birth in males followed by tests implantation or androgen administration in adulthood led to predominantly heterotypical sexual behaviour, i.e., to male homosexuality. On the other hand, androgen treatment that was started in females before birth gave rise to female homosexuality in later life. A clear-cut positive oestrogen feedback on LH secretion was only evocable in heterosexual female and homosexual male animals.
3. A positive feedback was also evocable in homosexual men following a single oestrogen injection, in contrast to heterosexual men. This finding suggested that homosexual men possess, at least in part a predominantly female-differentiated brain. These data were then strongly confirmed by other authors [1].

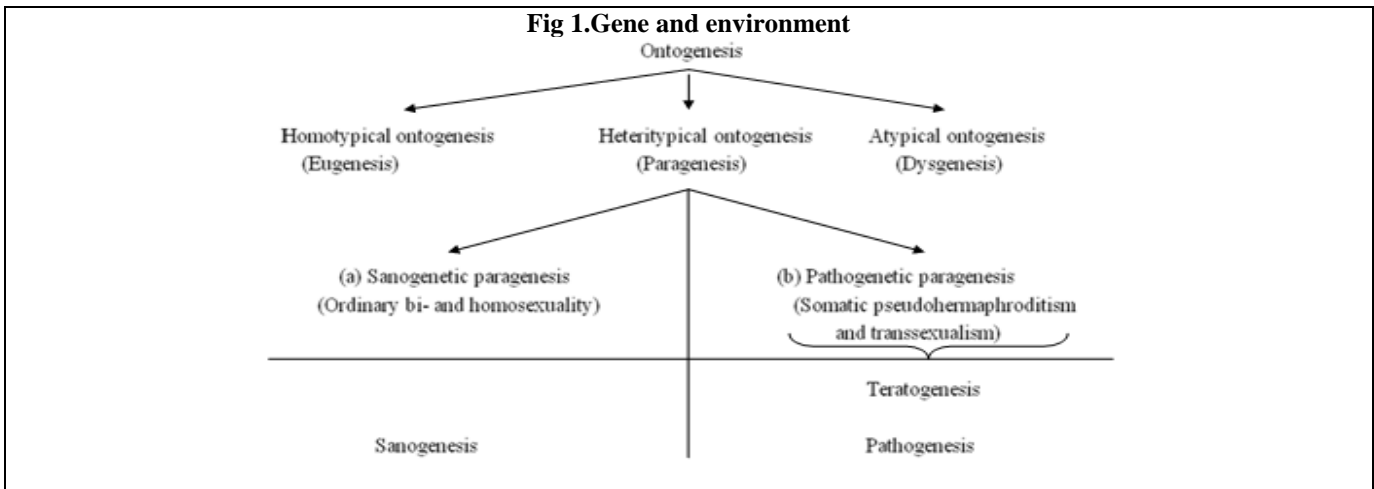


4. Stressful situation in prenatal life as well as specific androgen deficiency in males and androgen excess in females. Such alterations are responsible for variations of sexual brain differentiation. Thus, in homosexual women a slight, heterozygous form of 21-hydroxylase deficiency was diagnosed, which can lead to increased production of adrenal androgens and hence to more or less heterotypical brain development. Various heterozygous genetic alterations display a high prevalence in all populations and are generally not considered as a disease. Therefore, there is also no reason for the WHO to keep homosexuality in

genetic alterations were recognized as possible causes of the international classification of diseases [7].

The effects of sex hormones on brain development are mediated, at least in part by neurotransmitters. Moreover neurotransmitters were recognized to be gene- and environment-dependent organizers of the brain. Therefore, the effects of genes, sex hormones and of the psychosocial environment on sexual differentiation, maturation and function of the brain do not represent alternative but complementary factors; the more so as all of them are mediated by neurotransmitters.

Fig 1. Gene and environment



The latter scheme which explains typical and atypical ontogenesis was used to explain why and how the bi- and homosexuality should no longer be considered as pathology. Here scientist explains that the main reason for misunderstanding and change is the fact that in previous publications the total heterotypical ontogenesis – including ordinary bi- and homosexuality without complications – was united with atypical ontogenesis or dysgenesis to teratogenesis [10]. Therefore he explains how this point of view should be corrected:

In the hormones- and neurotransmitter-dependant ontogenesis, we can distinguish between a homotypical, heterotypical and atypical ontogenesis (Fig. 1). In the case of homotypical ontogenesis or eugenesis a development occurs that is typical for the own genetic sex due to corresponding homotypical hormone and neurotransmitter level. Hence, a homotypical somatic development and homotypical sexual behaviour, i.e., heterosexuality is observed. In the case of heterotypical ontogenesis or paragenesis, on the other hand, a development occurs that is typical – at least in part for the opposite genetic sex due to corresponding heterotypical hormone and neurotransmitter levels. Hence, in dependence of the beginning and the degree or such heterotypical hormone and/or neurotransmitter levels somatic pseudohermaphroditism or more or less heterotypical sexual behaviour i.e. homo- or bisexuality, are found. Finally, in the case of atypical ontogenesis or dysgenesis,

atypical structures and/or functions for both sexes i.e., genuine malformations and/or malfunctions are developed. Such dysgenesis can be mediated by atypical, i.e., abnormal concentrations of hormones and/or neurotransmitters during critical differentiation and maturations periods, especially of the brain [8-11].

The heterotypical ontogenesis or paragenesis can be divided into paragenesis without severe complications and need of therapy. Simple or ordinary homosexuality *per se* is a heterotypical ontogenesis without severe complications and without need of therapy, whereas somatic pseudohermaphroditism or transsexualism are heterotypical developments with severe complications need of therapy.

Structural paragenesis (somatic pseudohermaphroditism) as well as functional paragenesis with severe complications (transsexualism) can be united with dysgenesis to teratogenesis. Hence, teratogenesis is the development of malformations (teratomorphogenesis) or malfunctions (teratophysio- or teratopsychogenesis). Due to this subdivision of ontogenesis, teratogenesis is always a process of pathogenesis, which should be prevented as far as possible. In contrast, heterotypical ontogenesis without complications and need of therapy – i.e., the development of ordinary bi- or homosexuality – is no process of pathogenesis, but of sanogenesis [12].

Dörner's extensive experimental and clinical studies of the past two decades suggest that genuine bi- or



homosexuality are natural, biopsychosocial, gene- and/or environment-dependent sanogenetic and not pathogenetic developmental processes of the brain which are mediated by hormones and/or neurotransmitters. They are caused by heterotypical concentrations of sex hormones and/or neurotransmitters during sexual brain differentiation, which is timed in the human predominantly in prenatal life. This perception promoted already – at least in part – the decriminalization and dediscrimination of bi- and homosexuality and renders also possible a scientifically based depathologization of these sexual variations. Therefore, simple or ordinary homosexuality should no longer be considered as a disease and is also no indication for a curative or preventive therapy.

The aim of these researches lead by many scientists was to achieve a complete decriminalization, dediscrimination, depathologization and general social acceptance of heterotypical sexual orientations.

Researches about sexual deviations did not end up in the XX century, there are still variety of different researches carried out which considers issue and causes of homosexuality. One of the examples of such studies was to show the influence of early androgen exposure. Considered article summarizes the large body of experimental research in non-human animals that documents the influences of testosterone on mammalian neurobehavioral development. These studies form the basis for predicting that testosterone influences human neurobehavioral development

This study highlights the importance of the testosterone during early development in further sexual differentiation of the mammalian brain and that it has enduring influences on behavior. Testosterone exerts these influences at times when the testes are active, as evidenced by higher concentrations of testosterone in developing male than in developing female animals. In humans, testosterone is elevated in males from about weeks 8 to 24 of gestation and then again during early postnatal development (so called “mini-puberty”). Individuals exposed to atypical concentrations of testosterone or other androgenic hormones prenatally, for example, because of genetic conditions or because their mothers were prescribed hormones during pregnancy, have been consistently found to show increased male-typical juvenile play behavior, alterations in sexual orientation and gender identity and increased tendencies to engage in physically aggressive behaviour [1, 7].

Thousands of studies of non-human mammals have documented the contribution of gonadal steroids, particularly the testicular hormone, testosterone, to sexual differentiation of the brain and of behavior. Female animals treated with testosterone prenatally or neonatally subsequently show increased male-typical behaviour and decreased female-typical behavior. Similarly, male animals that have their testes removed early in development later show increased female-typical behaviour and reduced male-typical behavior. These effects have been seen for sexual behaviors and for non-reproductive behaviors that

differ by sex, such as aggression and juvenile rough-and-tumble play. They also have been seen in numerous species ranging from rodents to nonhuman primates. The effects of early androgen exposure on both the brain and behavior are often described as organizational and early androgen exposure is thought to produce enduring changes in behavior by altering the development and organization of underlying brain circuits.

Given those proper experiments with humans, where individuals are randomly assigned to be treated with testosterone or placebo early in life, are not ethically acceptable this article discusses two congenital conditions where testosterone levels had some serious influence for further behavioral development.

The strongest evidence that prenatal androgen exposure contributes to human gender development comes from studies of children’s gender-typed play. Girls exposed to elevated androgens prenatally, because they have the genetic condition, congenital adrenal hyperplasia (CAH), show increased male-typical toy, playmate, and activity preferences, a finding replicated in over a dozen studies from several independent research groups in several different countries, including the United States, the United Kingdom, Sweden, and Germany. There is another case that supports this condition and is not congenital. For example: healthy children whose mothers were prescribed androgenic progestins during pregnancy show increased male-typical play, and that those whose mothers were prescribed antiandrogenic hormones show reduced male-typical play, also suggests that androgen exposure, rather than other aspects of the CAH condition, are responsible for the behavioral differences seen in girls with CAH [10].

Another data from individuals with another genetic condition, complete androgen insensitivity syndrome (CAIS), also suggest a contribution of early androgen exposure to maletypical play behavior. These XY individuals have normally functioning testes but are born with feminine-appearing external genitalia, because their cells are unable to respond to the androgens produced by their testes. Adults with CAIS recall engaging in reduced male-typical, and increased female-typical, play behavior in childhood, and these effects have been replicated by an independent research group for children with CAIS as well. Thus, several lines of evidence converge on the conclusion that prenatal exposure to androgenic hormones increases male-typical play behavior in children. Women exposed to elevated androgens prenatally because of CAH also are less likely to be exclusively or almost exclusively heterosexual than are other women, a finding that has again been independently replicated cross-nationally. Individuals with CAIS almost always have a sexual orientation toward men, consistent with their lack of effective androgen exposure, and this finding also has been reported by more than one independent research group. In regard to gender identity or one’s sense of self as male or female, exposure to high levels of androgens prenatally has been linked to an increased likelihood of developing a male gender identity,



despite being reared as a female. A change to living as a man has been observed in about 1% to 3% of women with CAH, as well as in women with other genetic conditions causing exposure to elevated concentrations of androgens during early development, and again has been seen cross-nationally. There also is evidence from more than one research group that individuals with CAIS almost always develop a female gender identity, consistent with their lack of effective androgen exposure.

Evidence linking early androgen exposure to behaviours other than sex-typed childhood play, sexual orientation, and gender identity like empathy, autism, strategic abilities, performance of special tasks, mental rotation tasks or other cognitive abilities is not as strong and have produced largely inconsistent results to provide similarly conclusive evidence and still need to be independently replicated.

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CONCLUSION

In conclusion spreading of knowledge about the development of homosexuality helps a lot in destigmatization of sexual orientation in general population.

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CONFLICT OF INTEREST STATEMENT

Results presented in this paper are original, not published before and paper is not sent to the publication in another Journal.

STATEMENT OF HUMAN AND ANIMAL RIGHTS

All procedures performed in human participants were in accordance with the ethical standards of the institutional research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. This article does not contain any studies with animals performed by any of the authors.

