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ARTICLE

The tale of EDCs and trans identities*

Maite Arraiza Zabalegui

Faculty of Education, Philosophy, and Anthropology, University of the Basque Country, Donostia, Spain

Email: maite.arraiza@ehu.eus

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Abstract

This paper critically analyses the hypothesis of the aetiological link between EDCs and trans identities from a scientific point of view, evincing its lack of evidence. It also problematizes the hypothesis by drawing from gender studies scholars who have denounced the transsex panic underlying the scientific literature on the effects of EDC on non-human animals, as well as from philosophical, biological, STG studies', and neuroscientific elaborations that address sex-gender identities. It finds that the hypothesis that causally links prenatal exposure to EDCs and trans identities, which fuses biological determinism with a toxic and perturbing element, not only obscures the dynamic processual and relational character of trans identities, but also offers a pathologising understanding of them.

Keywords: EDCs; Trans identities; pathologisation; neurobiology; Gender studies

Introduction

EDCs (endocrine disrupting chemicals) are defined as 'an exogenous chemical, or mixture of chemicals, that interfere with any aspect of hormone action' (Gore et al. 2015: 3). They are present in a large number of products and practices, such as food, computers and electric equipment, cans and bottles, hormone treatments and many other kinds of drugs, clothes, cosmetic and self-care products, pesticides, metal, paper, textiles, or waste and oil products management and incineration (Gore et al. 2015; Rose 2014). Although various EDCs have been banned due to their noxious effects, they are still present in many places of the planet. Some of their substitutes also have been shown to have deleterious effects, and several EDCs are unknown, since they are not revealed by their manufacturers (Blum et al. 2015; Gioia et al. 2014; Schnoor 2014).

Since Rachel Carson published her famous *Silent Spring* (1962), shining the spotlight on the harmful effects of DDT, much has been written about these compounds,

especially the synthetic ones. While EDCs' multiple adverse health effects,[1] such as cancers, diabetes, obesity, thyroid problems, and immune, neurological, and cardiovascular diseases have been reported in non-human and human animals, a considerable part of the scientific literature has focused on their effects on sex-gender and reproduction. The emphasis on sexual matters became crystallized in the title of the Wingspread Conference Statement of 1991: 'Chemically-Induced Alterations of Sexual Development: The Wildlife/Human Connections'. It was at this conference that the term 'endocrine disruptor' was coined.

Recently, a proliferation of scientific works has attempted to causally relate prenatal exposure to EDCs and trans identities. This group of works can be situated in the historical trend of scientific research that legitimizes social hierarchies and inequalities based on sex-gender, sexual orientation, and race. Examples include scientific works on the so-called sex hormones, particularly testosterone and its causal role in a wide range of behaviours – from the type of play and a better athletic performance to a greater aggressiveness in men; or the research linking smaller brain size in black people to lower intelligence.

Parallel to their increasing social prominence, trans identities have gained great scientific attention in the last three decades, with a blossoming of hypotheses and theories aimed at explaining their possible causation, among which is the aetiological hypothesis of prenatal exposure to EDCs and trans identities. The aim of this paper is to critically analyse the scientific literature that postulates this hypothesis, as well as to offer a characterization of it. As I will show, the scientific work linking EDCs to trans identities, on the one hand, is grounded in no scientific evidence. On the other, it fuses biological determinism with an external toxic and disrupting element in its explanation of trans identities, offering a pathologising understanding of them. Biological determinism constrains the possibility and fact of change and drastically diminishes the role of social, cultural, historical, and political elements in the configuration of sex-gender identities. Thus, the hypothesis under analysis not only obscures the dynamic processual and relational character of trans identities, but also situates them out of the ordinary explanatory schema.

This scientific rhetoric, far from contributing to increasing equality and enhancing life conditions, curtails the autonomy and decision capacity of trans people. Pathologisation stigmatizes trans people, creating the breeding ground for discrimination and violence. At a time when trans lives are precarized and under exclusion and attack in multiple ways, not only by the violence that denies them their right to exist and their legitimacy to decide, but also by the physical violence that includes killing, what matters is not only the scientific quality of the scientific accounts, but also their social implications and consequences, namely, which societal sex-gender model they do serve.

The structure of the paper is as follows: In the second section, I examine the critiques made by gender studies scholars of the scientific literature on EDCs and transness in non-human animals, and show an alternative conceptualization of sex. In the third section, I critically analyse the scientific literature that advances the aetiological hypothesis of EDCs and human trans identities, drawing from the aforementioned

[1]On their unequally distributed effects on human bodies and populations due to inequality axis of race, ethnicity, class, sex-gender, etc., see Scott (2015); Ruiz et al. (2018).

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criticisms and feminist elaborations in several fields. In the last section, I present some concluding remarks.

EDCs and transness in non-human animals: From the 'transsex panic' to a relational, open, and dynamic understanding of sex

As already noted, numerous scientific studies have focused their attention on the alterations provoked by EDCs in non-human animals' sex morphology and functions. Examples include organotins induced female masculinization in more than 268 species of gastropods (Titley-O'Neal et al. 2011);[2] 'abnormalities' in sex steroids and gonadal morphology in the alligators of the Lake Apopka (Florida) (Guillette et al. 1994); high incidence of intersexuality, considered as 'alarming', in the *Rutilus rutilus* in the United Kingdom (Jobling et al. 1998: 2503); or reduced size of sex organs in polar bears linked to organohalogens, which 'pose a risk' to these bears (Sonne et al. 2006: 5668).[3]

In view of what this sample highlights, various authors have called attention to the excessive weight given in scientific literature on EDCs to sex-gender[4] related affairs, particularly to 'feminized', 'transgendered', 'intersexed' animals, and 'abnormalities' of various types, in detriment of relevant health and environmental adverse effects. This prominence, as the scientific studies referred to above and sensationalistic headlines of informative science articles show, takes the form of alarm and fear. Di Chiro calls it 'sex panic' (2010: 202) and, more accurately and in tune with the focus of this paper, Ah-King and Hayward (2014: 4) designate it 'transsex panic'. As Kier suggests, 'it is interesting to consider why the idea of transgenderness is being used to represent toxicity and eco-catastrophe … Simply stated, why is "transgender" the signifier…?' (2010: 314).

The threat that these scientific discourses unveil is not related to death *per se*, or to the ability of a species as a whole to reproduce, but to the alteration, by apparently exogenous toxic substances, of a natural order conceptualized as binary, dimorphic, and heteronormative. Importantly, it is the allegedly essential, static, and immutable character of this order that seems to be at stake, making change itself a deeply problematic and threating element. Even in species in which sex change and intersexuality are well documented, their presence apparently needs to be kept to levels deemed acceptable. Thus, a pathologising gaze is projected onto animal bodily and behavioural forms that transcend and subvert this essentialist cisheteronormative framework. The discourses of racial and social justice activists, environmentalists, and feminist biologists, endocrinologists, and historians that aim at studying the deleterious effects of EDCs, raising consciousness and urging political action on the issue, reinforce heteronormativity through their hyperfocusing on sex-gender and sexuality, and appealing to the normal and natural order (Di Chiro 2010: 210-211).[5]

[2]Yet, 42 species don't show female masculinization when exposed to organotins in laboratory or field, and in some species imposex happens 'naturally', without exposure to tin compounds (Titley-O'Neal et al. 2011).

[3]The reports of several laboratory studies on the effects of EDCs include intersexuality and sex reversals in fishes and turtles (Bergeron et al. 1994; Santos et al. 2017); gonadal impairments in anurans (Tamschick et al. 2016); or malformations and feminization of male embryos in Japanese quails (Berg et al. 1999).

[4]For the use of the term 'gender' to refer to fishes' sex, see Konkel (2016).

[5]See, for instance, Hayes et al. (2002) and Langston (2003).

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In this sense, while engaging with the critical voices on the environmental damages brought on by human made EDCs that are driven by big corporations, this paper critically analyses the emphasis placed on transness, as well as the underlying reasons, and seeks for alternative conceptualizations. As Pollock (2016: 190) asks: are all the effects of EDCs necessarily bad? Leaving behind this transsex panic, how might these effects be conceptualized apart from essentialism and (eco)cisheteronormativity?[6]

Following Kier's suggestion of understanding the ability to change sex due to the toxic presence of EDCs as both a response and an adaptation (2010: 310), Ah-King and Hayward (2014) interpret EDCs as elements that take part, along with many others, in the dynamic, relational, and open ongoing process of sexing. This dynamic and relational process of sexing signifies that sex is not given once and for all, neither just in one form for all the species taxa, but it emerges with and responds to the environment in a myriad of ways. Instead of conceiving sex as a 'nature-given dichotomy, or essentially discrete characteristic', sex and the multiple elements that constitute sex are 'better understood as a responsive potential, changing over an individual's lifetime in interaction with environmental factors, as well as over evolutionary time' (Ah-King and Hayward 2014: 6). The authors move away from purity politics and reduce the apocalyptic tone by pointing out that in this moment of history, the environmental toxicity of EDCs has also become part of the dynamic process of sexing.

This notion of sex as a responsive potential is based on the dynamic model of reactive sex, of sex as a norm of reaction,[7] theorized by Ah-King and Nylin, according to which sex attributes, behaviours, and sex determination, even when it is considered genetic, are fundamentally plastic (2010: 234). For Ah-King and Nylin, it is a paradox that variation in sex determination, reproductive strategies, and sex change is well known in biology, yet this variation is still primarily depicted as a two-sex norm, and the rest, as deviations from this norm, alternatives, and sex role-reversals (2010: 236). Contrary to this conceptualization, there is a 'tremendous' variation in sex, sex attributes, and behaviour, as a result of genetic and environmental influences on phenotypes,[8] which make sex 'a particularly illustrative example of … the ubiquitous developmental plasticity of living systems' (Ah-King and Nylin 2010: 244).

From an evolutionary viewpoint, genetic and environmental sex determination systems repeatedly evolve one from the other, and genetic sex determination systems also show evolutionary flexibility and diversity (Ah-King and Nylin 2010: 240). A large range of animal taxa has environmental sex determination; namely, social environment, temperature, or pH influence sex determination. Some species combine different sex determination systems, including simultaneous hermaphroditism, and many change sex during their lifetime, at a certain body size, or in response to ecological and social environment (Ah-King and Hayward 2014: 6; Ah-King and Nylin 2010: 239-240). But even in species with genetic sex determination and large sex differences,

[6]This concept follows what Di Chiro calls 'eco(hetero)normativity' (2010: 202).

[7]A reaction norm is 'the range of phenotypic expressions that one genotype can give rise to, in response to different environmental conditions' (Ah-King and Nylin 2010: 235). This means that a phenotype does not originate from genes, but from the intertwinement of genes and environment, namely, from a reactive phenotype. Ah-King and Nylin extend the concept of reaction norm to species with genetic sex determination and pronounced differences between the sexes.

[8]Importantly, causality is multidirectional, meaning that behaviour, for instance, can also generate changes in genes, morphology, and environment (Ah-King and Nylin 2010: 236).

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an individual's phenotype also depends on environmental influences in development (Ah-King and Nylin 2010: 238).[9]

From this understanding of sex as a potential, a responsiveness, an opening out that is more dynamic than static, Ah-King and Hayward reconceptualise EDCs as elements that have the power to induce sexual changes even in organisms whose sexual possibilities or sex potential are more limited (2014: 6). These EDCs driven sexual transformations are signs of ecological resilience to toxicity, which shows a trans and queer potential. As Kier points out, 'life in many ways is simultaneously fragile, resilient, adaptive, and … transsex exhibits the ability to find ways to transform the possibilities of re/production' (2010: 316).[10]

What about humans? Making *non-sense* of the hypothesis of EDCs as part of the aetiology of trans identities

The anxiety and fear regarding the effects of EDCs on sexual development, morphology, and reproduction extend to human animals.[11] More to the point of this paper, the same transsex panic arises when the possible effects of EDCs on humans are examined. Ernie Hood begins his article 'Are EDCs Blurring Issues of Gender?' (2005) with the following words:

> Although scientists have postulated a wide range of adverse human health effects of exposure to endocrine-disrupting chemicals (EDCs), the nexus of the debate is the concern that prenatal and childhood exposure to EDCs may be responsible for a variety of abnormalities in human sexuality, gender development and behaviors. … Could such exposures even be involved in the etiology of children born with ambiguous gender? (Hood 2005: 671)

Other scientific informative media articles, such as 'The dissolution of gender' (Hedaya 2019) or 'Gender Fluidity and Hormone Disruptors: Hormone-disrupting chemicals may increase gender dysphoria' (Barber 2019), also alert the readers not only about the dangers of EDCs in relation to gender identity, but also about the dangers of transsexuality, gender blur, and gender fluidity. Delving into the question posed by Hood, the professor of psychiatry Robert Hedaya (2019) states: 'It is a reasonable hypothesis that the subjective disturbances of gender identity are the psychological manifestation of altered gene-neuro-humoral signaling caused by the chemical soup we live in'.[12] Is it?

[9]Among species, the appearance of females and males ranges from similar to dimorphic. Dimorphism, namely, the existence of two differentiate forms in a species, is also dependent on ecological factors instead of sex, and most sexual characters overlap between the sexes (Ah-King and Nylin 2010: 241-243).

[10]Although the meaning of the concept 'transsex' of Kier differs from the meaning used in this paper, it works accordingly here. An example of this reworking of reproduction can be seen in the fertilizable eggs of male basses in some rivers in the United States (Ah-King and Hayward 2014: 9; Konkel 2016).

[11]For Ah-King and Hayward (2014: 5) and Di Chiro (2010: 209), what is unveiled here is ultimately the uneasiness produced by a possible questioning of the continuity of patriarchal hegemonic masculinity due to EDCs toxicity. *The Estrogen Effect: Assault on the Male* (Cadbury 1993) and the focus on reduced sperm counts and rising hypospadias and cryptorchidism are among the examples they cite.

[12]Even institutions such as the UPMC Children's Hospital of Pittsburgh echo the theory that links pre- and perinatal action of EDCs to human transgenderness in one of its leaflets.

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Scientific works on the implication of EDCs in trans identities

In order to approach the hypothesis of the causal role of EDCs in human transness, it is necessary to analyse the existing scientific production on this matter, which some of the informative articles already mentioned echo. In this sense, we can distinguish neurobiological theories or hypotheses that include the possible involvement of EDCs in the aetiology of trans identities; literature reviews; and studies on associations between EDCs and behaviours.

One of the theories that postulate this causal role is the neurobiological theory about the origin of gender dysphoria. This theory is inscribed in the organizational-activational (O/A) hypothesis, according to which foetal testicular secretion of testosterone masculinizes the brain in utero, while its absence generates the female brain. This hypothesis holds that sexually dimorphic behaviours and gender identity are the result of the interaction between sex hormones and neurons that organize the brain prenatally. In puberty, hormone levels activate these permanently and irreversibly programmed aspects. As intrauterine sexual differentiation of the brain occurs later than that of the sexual organs, according to the neurobiological theory about the origin of gender dysphoria, these two processes can be influenced independently, creating 'reversals' in sexually dimorphic brain structures and, thus, resulting in gender dysphoria (Savic et al. 2010: 43-44; Swaab and Bao 2013: 2979). Swaab's team claims to have found these reversals in two structures: the central subdivision of the bed nucleus of the stria terminalis (BSTc) and the third interstitial nucleus of the anterior hypothalamus (INAH3).

Among the underlying causes of gender dysphoria would be immunological factors, genetic factors, like chromosomal 'abnormalities' and genetic polymorphisms of oestrogen and androgen receptors and aromatase gene, as well as 'abnormal' prenatal hormone levels that affect the brain (Swaab and Bao 2013: 2983; Swaab et al. 2021: 430).[13] The latter comprises the effects of EDCs, such as antiepileptic drugs or the synthetic oestrogen diethyletilbestrol (DES) taken during pregnancy, the proof of which is found on the DES children's website, which claims that transsexuality occurs in the 35.5% of the cases (Savic et al. 2010: 49; Swaab et al. 2021: 433). This detrimental effect of EDCs on the sexual differentiation of the human foetal brain is also based on studies on bisphenol-B (BPB) induced disruption of sexual differentiation in the zebrafish, as well as on studies associating prenatal exposure to phthalates to less male-typical behaviour in boys, and to pesticides to smaller testicles and penises in boys (Swaab et al. 2021: 433).

Another neurobiological hypothesis that includes the potential causal role of EDCs in its explanation of trans identities is the neurodevelopmental cortical hypothesis, which refines Swaab's team's work. This hypothesis is also inscribed in the O/A hypothesis, but modifies it slightly.[14] In compliance with the neurodevelopmental

[13]Besides these 'abnormalities', this theory includes other pathologising elements such as placing transsexuality in the list of neurological and psychiatric diseases (Swaab and Bao 2013: 2981) or claiming for biological markers for the diagnosis of gender dysphoria (Swaab et al. 2021: 438). The idea of inversion or reversal has also been historically used to name *perverse* and *deviate* sexualities and identities.

[14]It includes some latest modifications of the O/A hypothesis, such as the not only hormonal but also genetic sexual differentiation of the brain; the not-always-necessary hormone activation effect in conducts prenatally organized; as well as the involvement of oestrogens in the masculinisation of the

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cortical hypothesis, there would be a slowdown (or a detention) in the cortical thinning process in cis women, trans women, and trans men, compared to that in cis men, affecting different cortical regions and creating four distinct cortical phenotypes, one for each group: cis men, cis women, trans women, and trans men (Guillamon et al. 2016: 1637). The different speed of decrease in each variant of gender would be programmed (Guillamon 2021a: 135).

These distinct structural and functional phenotypes would be ultimately due to gene polymorphisms of sex hormone receptors and aromatase gene, which would create differences in the efficiency of these receptors during brain sexual differentiation (Guillamon 2021a: 156). And here is where EDCs could play their part in the aetiology of trans identities, prenatally affecting brain sexual differentiation through epigenetic mechanisms that alter genetic expression, such as DNA methylation, and leading to the development of a particular brain phenotype (Guillamon 2021a: 143). This aetiological role of EDCs is sustained again on the foundational effects of DES when it comes to the hypothesis in humans, as well as on bisphenol-A (BPA) induced alterations on sex morphology and behaviour in rodents, and associations of BPA in humans (less fertility, delay of puberty in girls and pubertal advancement in boys, undescended testicles, or lower quality of sperm) (Guillamon 2021a: 111–113).

The second type of work to be analysed here is scientific literature reviews. Saleem and Rizvi (2017) deploy a multifactorial aetiology of transgender identities. This aetiology includes prenatal neuroanatomical factors; genetic factors; associations with autism spectrum disorder, schizophrenia, other psychiatric disorders, and childhood maltreatment; and the role of EDCs.[15] The hypothesis about the role of EDCs is supported by a study showing polychlorinated biphenyls (PCBs) induced impairment of the female rat hypothalamus and Bejerot et al.'s letter to the editor (2011), in which they hypothesise a causal link between prenatal exposure to phthalates and the increase of autism spectrum disorder and its comorbidity with gender identity disorder (Saleem and Rizvi 2017: 5). Nonetheless, the authors note the necessity for more systematic research in this regard.

The same letter to the editor is the only piece regarding humans that Cocchetti et al. (2023) cite in their review. Adding a battery of experimental studies on prenatal exposure to EDCs induced impairments of brain sexual dimorphism, reduced sexual dimorphism in behaviours, and reversals of these behaviours in rodents,[16] Cocchetti et al. (2023: 328) embrace the hypothesis of a possible aetiological link between prenatal exposure to EDCs and gender dysphoria. Still, its basis on data from rodents leads

brain (Guillamon 2021a: 37-41, 79). Hence, hormone activation would not affect gender identity in children without identity problems or in those with gender dysphoria after puberty, but it would affect those whose gender dysphoria fades after puberty (Guillamon et al. 2016: 1637).

[15]Despite presenting numerous data on discrimination, precarization, and violence against transgender people, and noticing 'transgender variants' along history and cultures, 'gender dysphoria' is catalogued as a 'disorder' and a 'neurodevelopmental disorder', and employed as synonym of trans identities.

[16]Laboratory studies on the impact of EDCs on sexual differentiation and function in alligators, turtles, the zebrafish, or the Japanese quail are also shown; as well as reports of micropenis, hypospadias, cryptorchidism, and cervical canal malformations in humans linked to prenatal exposure to DES and other EDCs.

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the authors to contend the need for more investigations 'to establish *EDCs' interference* with sexual differentiation of the brain in *determining… gender identity*' (2023: 328, emphasis added).

Finally, even if the main topic of their review is the increase of intersex cases associated with the risk posed by 'gender altering chemicals', Rich et al. also establish a causal link between EDCs and gender dysphoria, since intersex individuals may experience it: 'EDCs can *interfere* with the complex biochemical pathways of the brain … affecting *normal* behavioral or *gender development*' (2016: 165, emphasis added).[17]

Regarding scientific studies, many have been conducted on associations between pre- and perinatal exposure to EDCs and different behavioural phenomena. After analysing various reviews (see Kahn et al. 2020; Özel and Rüegg 2023; Palanza et al. 2021; Salazar et al. 2021) and around 40 individual studies of the last two decades, I have not found any study that links this exposure to trans identities.[18] Yet, five of them examine associations between standard exposure to EDCs and play behaviour, aggressiveness, and cognition. Since part of the literature analysed refers to some of them to suggest an aetiological link between EDCs and trans identities, what follows is a summary of their findings.

Three of these studies address children's play behaviour. Swan et al. (2010) and Percy et al. (2016) find that prenatal exposure to phthalates was associated with less male-typical behaviour in boys. In the first study, this association was found in four of the nine phthalate metabolites measured, and was statistically significant only in two of them.[19] In the study of Percy et al. (2016: 7), only when the measures of children's play behaviour were dichotomized ($<25^{th}$ percentile vs. all others), statistically significant associations were found, and regarding only two of the nine metabolites.[20] Since no children showed gender dysphoria, the authors link this exposure to subtle changes in the gender spectrum still typical for each sex.

Winneke et al. (2014) find that prenatal exposure to dioxins and PCBs was associated with a more feminine play behaviour in boys and a less feminine behaviour in girls. But the association with the femininity score was only significant in boys, not girls; and only with the measures in maternal milk, not blood.[21] Making a step further in

[17]While they only cite one scientific study linking EDCs to human intersexuality, studies on a myriad of other species pile up. Of note is also that Rich et al. (2016: 163-164) acknowledge the difficulty in determining whether the increase of intersexuality is due to EDCs, the decrease in early surgical intervention, expanded inclusivity, reduced mortality, or a documentation artefact.

[18]These behavioural phenomena are mainly autism; attention-deficit, externalizing behaviours (aggression, hyperactivity, and conduct problems), and internalizing behaviours (depression, somatisation, and anxiety); and cognitive issues, mostly analysing QI.

[19]No such association was found in girls. Amazingly, the parental attitude towards sex-(a)typical toy choices and play, employed to assess children's play behaviour, was close to neutral (see Swan et al. 2010: 262). For the problematicity of using this type of method in research, see Jordan-Young (2010: 252).

[20]When the measures of children's play behaviour were analysed as continuous variables, no association was found with prenatal exposure to phthalates. For the problematicity of categorizing continuous variables in quantiles, a widespread practice in epidemiological research, see Bennette and Vickers (2012). On the other hand, one of the associations was with a more typical play behaviour in girls.

[21]Regarding the masculinity score, significant negative associations in girls were found with milk. In blood, no significant association with masculinity was found in girls, nor in boys. Besides, Winneke et al. (2014: 296) find 'more behavioral femininity in boys and less femininity in girls, but also more masculinity in boys and less masculinity in girls', an 'apparent contradiction'.

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establishing causality, the authors conclude that 'the overall evidence that PCBs and dioxins modify sexually dimorphic behaviour in children is "sufficient"' (2014: 297).

Concerning hyperactivity and aggression, Braun et al. (2009) observe a positive association between BPA urine concentrations in pregnant women and girls' behaviour. However, they note the difficulty of labelling these effects 'as feminizing or masculinizing without knowing whether these end points are sexually dimorphic' (2009: 1950).

Rauh et al. (2012) find some deformations, reductions, and enlargements in the brain of children prenatally exposed to the pesticide chlorpyrifos (CPF), but only in the high exposure group (upper tertile of CPF concentrations). It 'also displayed disruption of normal sexual dimorphisms in brain structure' and 'reversed' sex differences (Rauh et al. 2012: 7875). The researchers suspect that the impaired scores in working memory and full-scale IQ linked to prenatal CPF exposure in the cohort from which these children were drawn derived from some of these brain 'abnormalities' (2012: 7875).

The 'science' relating trans identities to EDCs: Biological determinism meets a theory about the abnormal

In general, the scientific literature that affirms or suggests an aetiological link between EDCs and trans identities presents three main and interrelated problematic elements: it conceptualises brains and behaviours as sexually dimorphic; it bestows a biological deterministic account of these dimorphisms, as well as of trans identities, and sex-gender identities in general; and it offers an interpretation of trans identities as anomalies that are explained to a great extent by an exogenous disrupting element.

Regarding the first element, both neurobiological hypotheses on trans identities embrace brain sexual dimorphism, either for the whole brain, namely, 'the female and the male brain' (Swaab and Bao 2013: 2979; Swaab et al. 2021: 427), or regionally. While in the case of the neurobiological theory about the origin of gender dysphoria of note are the BSTc and the INAH3, the neurodevelopmental cortical hypothesis also refers to sexually dimorphic brain regions, including cortical thickness, and spatial and verbal abilities (see Guillamon 2021a: 130;[22] Rametti et al. 2011: 199, 202). Thus, for the first theory, in contrast to the mentioned 'cisgender brains', the 'transgender brain' (Swaab 2021: 435) arises when reversals occur in the mentioned sexually dimorphic regions. For the neurodevelopmental cortical hypothesis, two of the four distinct brain phenotypes correspond to trans women and trans men.[23]

The literature reviews also embrace the idea of the sexually dimorphic brain, of gender dysphoria as the result of an opposite brain and genital sexual differentiation, including the reversal of the INAH3 and the BSTc, the implication of the mentioned

[22]Concerning cortical thickness, Guillamon specifies that sexual dimorphism means that cis women present greater cortical thickness in some regions. Even if he acknowledges that it is more correct to reserve the term 'sexual dimorphism' for qualitative differences, declaring the presence of the Y chromosome in all brain cells as the only qualitative brain difference, he also applies the term as two differentiate forms for females and males, not only to morphology and physiology, but also to conduct in humans (see Guillamon 2021a: 36). It is noteworthy that genetic brain sexual dimorphisms permeate the book (see Guillamon 2021a: 40, 59, 85).

[23]'The profile of all the patterns ($m > f$; $f > m$; $m = f$) in all brain regions for all the possible measures determines a male or female, or transgender brain' (Guillamon 2021a: 42, my translation).

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gene polymorphisms or the O/A hypothesis (see Cocchetti et al. 2023: 323; Saleem and Rizvi 2017: 3). The scientific studies, which mainly depart from the O/A hypothesis, affirm sexual dimorphism in relation to play behaviour and the brain (see Percy et al. 2016: 2; Rauh et al. 2012: 7875;[24] Swan et al. 2010: 260; Winneke et al. 2014: 292).

The framework of brain sexual dimorphism seems to be problematic and has been disputed by Daphna Joel and her collaborators, who embrace brain mosaicism. These researchers analysed a great number of brains, arriving at two main findings. The first is that brain regions and features are not sexually dimorphic, because there is overlap, mainly extensive, in all the measures that show sex-gender differences between females and males (Joel 2021: 165, 170; Joel et al. 2015: 15471). This includes the BSTc, the INAH3 – one of the regions with the greatest sex-gender differences and thus with less overlap – and cortical thickness. Moreover, it needs to be taken into account that sex-gender only accounts for around 1% of brain differences (Eliot et al. 2021: 689). The second finding is that brains, in general, are not sexually dimorphic because there is high variability, namely, cis men present features more common in cis women, cis women present features more common in cis men, and both present features that are common in both (Joel 2021: 166; Joel et al. 2015: 15468). Therefore, brains overall do not belong in two distinct classes: female brain/male brain. Brains are better characterized in one highly heterogeneous population, since each brain presents its own unique mosaic of regional and functional differences (Joel 2021; Joel et al. 2015).

The point is not that sex-gender individual and group differences do not exist. They do, and there are several of them. The point is that these individual differences do not consistently add up until two distinct types of brains are created. Relevantly, they are present in people with diverse sex-gender identities. Likewise, as we will see, these differences do not belong just to the biological domain nor are they innate, being brain plasticity fundamental in this regard. All of this leads to the use of the expression 'sex-gender differences' instead of 'sexual dimorphism'.

Brain mosaicism also allows us to problematize the concept of the trans brain in its different versions. If regional brain sexual dimorphism is refuted, there is no reversal of such dimorphism. Regarding the four brain phenotypes, each brain, not each group, shows a unique mosaic. Thus, the different brain pattern for each of the four groups, even in the mosaic form, would be problematic, since not only there is regional overlap, but also group sex-gender differences in specific brain features – and here Joel et al. also include cortical thickness– do not add up to create distinct types of brains or distinct brain phenotypes (Joel et al. 2018). Although Joel et al. discuss mainly cis women and men, the same logic can be applied to trans women and men.

This substantial overlap has also been observed in most social, cognitive, and personality variables, including spatial visualization and verbal fluency – one of the verbal skills that show the largest sex-gender differences – so group differences between women and men are small. Even in characteristics such as physical aggression or mental rotation, there is a non-trivial overlap (Hyde 2014; Rippon 2014).

If, in the best of cases, the problem of sexual dimorphism regarding brains and behaviours is a matter of linguistic expression, it brings with it an erroneous

[24]'Normal' sexual dimorphism means for Rauh et al. (2012: 7873-7874) female-larger-than-male or male-larger-than-female brain sex differences. They also assess the reversal and disruption of this dimorphism.

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conception of them as having two different forms: one for females and the other for males. Indeed, Eliot et al. (2021: 690) highlight that 'the issue is more than semantic', since '[t]he term "dimorphism" has potent heuristic value, reinforcing the belief of two categorically distinct organs' that have evolved to produce two psychologically distinct types of people designed to carry out different social tasks. And too often, this binary categorization entails a logical inference: a conceptualization in the form of reversal, abnormalities, or out of the ordinary explicative schema in the case of trans persons.

The second problem, intimately related to the first, is that trans identities, and sex-gender identities in general, as well as behaviours are prenatally or shortly thereafter determined basically by the genetic and hormonal organization of the brain, that is, they are biologically determined. The neurobiological theory about the origin of gender dysphoria explicitly denies the influence of postnatal social factors in the emergence of trans and sex-gender identities (see Swaab and Bao 2013: 2997; Swaab et al. 2021: 438). For the neurodevelopmental cortical hypothesis, identities similarly derive from the prenatal organization of the female or male brain (congruent or not with the sex assigned at birth) (Guillamon 2021a: 156-157).[25] This biologically deterministic view is also mainly shared by the analysed reviews regarding trans identities[26] and by the scientific studies on behaviours.

What this account entails is the disregard of the active role that social, cultural, discursive, and historical factors play in the emergence and development of trans and sex-gender identities, as well as behaviours, and the neglect of their dynamic processual character. The trans depathologisation framework, on the contrary, brings with it, among many other things, an understanding of transsexuality as a culturally and historically specific construction, and a critique of the colonial character of Western psychiatric classifications for rendering invisible the diversity of sex-gender-sexuality expressions worldwide (Suess et al. 2014: 74-75). The works of Magnus Hirschfeld and Harry Benjamin, as of many trans activists, are crucial for understanding this historically and socially situated emergence and development of transsexuality. Following Gertjee Mak (2012: 157-158), this phenomenon has to do with two main social constructions that emerged in the beginning of the 20th century: the concept of gender, which was the result of a sexual internalisation process that began at the end of the 19th century and became, by the hands of Robert Stoller in the 1960s, the notion 'gender identity'; and surgical procedures followed by hormonal technologies, which allowed for the configuration of sex-gendered bodies in previously unknown ways.[27]

For Anne Fausto-Sterling (2020: 272, 303), who describes sex-gender identity as a cultural phenomenon woven into the body, multiple entangled dimensions (historical, cultural, social, biological) and multiple entangled events related to these intertwined dimensions take part in the emergence and development of identities. This makes

[25]For Guillamon (2021b), the experienced gender identity is unmodifiable.

[26]Yet, there are some nuances. Saleem and Rizvi (2017: 3), for instance, include childhood maltreatment in their multifactorial aetiology. Cocchetti et al. mention, almost anecdotally, that 'other factors – such as social and familiar environment, as well as hormonal changes during puberty – may play a role' (2013: 323).

[27]This does not mean that non binary people and transvestism didn't exist before. Indeed, societies with what we now call three, four, and even five sex-genders are well documented throughout history. See, for instance, Herdt (1996).

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sex-gender identities subjective but fundamentally intersubjective. Specifically, she analyses how gender norms and expectations are embodied, through dyadic and other interactions, as well as through colours, toys, clothes, etc., from the age of three months, and how gender-related knowledge, activities, and ultimately sex-gender identities emerge (see Fausto-Sterling 2020: 298-313).[28] Several works from disciplines such as feminist neuroscience, psychology, social neuroendocrinology, and science studies likewise evince the influence of gender imperatives, roles, and stereotypes on hormones (see Fine 2017; van Anders et al. 2015), brains (see Rippon et al. 2014),[29] and behaviours and abilities (see Hyde 2014; Jordan-Young 2010).[30] The same works also show that hormones and brains change throughout life due to an array of factors, pointing to brain plasticity as a crucial element.[31]

This dynamicity is similarly observed regarding behaviour. Studies indicate that the mentioned differences in mental rotation and other spatial abilities are not present in early infancy, are smaller in children, and disappear when trained women and men are studied (Eliot et al. 2021: 685-686). Likewise, differences in aggressiveness between men and women disappear in contexts in which the sex-gender identity of the subjects is unknown (Lightdale and Prentice 1994).

Fausto-Sterling's (2020) characterization of trans and overall sex-gender identities as dynamic processes, as previously outlined, deeply problematizes not only the view of the prenatally determined trans identities, but also the unmodifiable nature of identity once experienced. Even if most children exhibit a sex-gender identity around the age of three, identity develops in a lifelong dynamic process, with more or less stability or fluidity, depending on the cases.[32]

These two elements – entangled multidimensionality and processual dynamicity – that characterize trans and, in general, sex-gender identities, have implications for the hypothesis that places EDCs into the aetiology of trans identities. If EDCs played a role in their emergence and development, it would be very difficult to disentangle it. Besides, as the Endocrine Society emphasizes in its 'Second Scientific Statement

[28]She also analyses how play behaviour has very much to do with these gendered dyadic interactions and gender norms. Similarly, Jordan-Young (2010: 218-252) examines how gender norms and expectations influence children's play behaviour, and presents studies showing results that do not support the hypothesis of the innate dimorphic type of play and toy preference.

[29]Among these works is also the conceptualization of the gender dysphoria experienced by *some* trans persons as a manifestation, at least in part, of the harm that a cisexist, binary, and genitalocentric society does to the neural representation of the self in the cortex (see Walsh and Einstein 2020).

[30]Jordan-Young (2010) includes gender into the notion of norm of reaction, as Ah-King and Nylin (2010: 238) do with culture in humans and other non-human animals, and applies it to physical traits, as well as to behaviours, preferences, and abilities. She uses the concept 'gender NORs' (norms of reaction). The fusing of experience and heredity implies a reworking of the definition of sex, since the way in which sex works depends on gender, as well as other cultural aspects (Jordan-Young 2010: 286).

[31]Plasticity is a life-long brain feature, even if it is more striking in early development. Plasticity means that brains are dynamic, that they develop and change due to and entangled with the environment, social interactions, behaviours, and experiences, including gender norms, stereotypes, and expectations, which shape and reshape brains.

[32]While some people change their identity and/or the category used to name it, and some alternate, with diverse frequencies, between different identities, frequently the identity category remains constant throughout life. But in all the cases, changes in anatomy, physiology, subjectivity, experiences, and even identity occur.

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on Endocrine-Disrupting Chemicals', all the scientific works on EDCs and behavioural outcomes are always by definition correlational (Gore et al. 2015: 92), which means that no direct causality can be established – something that part of the scientific literature analysed also acknowledges (see Cocchetti et al. 2023: 328; Guillamon 2021a: 111). The dynamic processual character of sex-gender identities further problematizes the possibility of studying associations between EDCs exposure and trans identities, because it would require almost life-long longitudinal studies.

The third main troubling element of the scientific account that postulates an aetiological link between EDCs and trans identities is that it conceptualizes these identities, more or less explicitly, as an 'anomaly', an 'alteration', a 'disturbance', even a 'disorder', brought about, to a great extent, by an external perturbing and noxious element. This is the same logic that underlies the scientific work regarding the effects of EDCs on non-human animals, denounced by Ah-King and Hayward (2014), Di Chiro (2010), and Kier (2010). Indeed, the endocrine disruptor thesis that acquired the status of a scientific-environmental theory since the Wingspread Conference situates the idea of 'abnormal' or 'disruptor' at its centre (Di Chiro 2010: 205). This theory is not about the genetic or biological abnormality, but about the abnormality and deviance as the outcomes of perturbing 'natural' developmental processes (Di Chiro 2010: 205).

Thus, EDCs would alter and disrupt the normal, natural sexual differentiation of the brain, prompting reversals in sexually dimorphic brain structures and contributing to the development of two of the four distinct brain phenotypes.[33] In this way, by interfering in the duties of genes and hormones, these environmental exogenous toxic substances would disrupt and disturb the normal or usual identity formation, resulting in gender dysphoria or trans identities. This narrative is seen in both neurobiological theories on trans identities, as well as in the reviews of Cocchetti et al. (2023: 328), Rich et al. (2016: 165), and Saleem and Rizvi (2017: 3, 5).

However, the degree of pathologisation in this scientific literature varies. While it is explicit in the first of the neurobiological theories through the synonymy between trans identities and 'gender dysphoria', their causal link to 'abnormalities', or the notion of 'reversal', the neurodevelopmental cortical hypothesis does not show this pathologising narrative. Nevertheless, the concept of the transgender brain, and the distinction between cis and trans brains as distinct 'types' of brains implies a neurobiological foundation of social categories that reveals pathologising inheritances. In the same vein, the view that social or chemical environmental effects would be detectable in the transgender minority, since there is no scientific proof that cisgender binary people depend on them (Guillamon 2021a: 143), posits trans persons out of ordinary explicative schema.

The literature review of Saleem and Rizvi (2017) adds pathologising elements to the narrative of the neurobiological theory about the origin of gender dysphoria, such as associations with autism spectrum disorder, schizophrenia, and other psychiatric disorders. Rich et al. (2016: 164-165) name diverse 'abnormalities' and describe gender dysphoria as a not 'normal' or 'appropriate' gender development.

[33]Epigenetics is the element that enables this bridge. Curiously, even if Guillamon (2021a: XVII) affirms that gender identity is a consequence of genetic, epigenetic, and hormonal mechanisms, when it comes to their own hypothesis to account for the elements that participate in epigenetics, social and not biological environmental factors are generally excluded, except for EDCs.

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This pathologising narrative and language are present in some of the scientific studies on associations between EDCs and behavioural outcomes as well. Even if Percy et al. deploy a notion of gender as fluid, continuous, and on a spectrum, they refer to childhood trans identities as gender dysphoria, pointing that the examined children's behaviours did not show 'deviation from normality' (2016: 4). Winneke et al. postulate that 'endocrine disruptors (EDCs) may *alter* the *normal* sexual structuring of the brain, with resulting in behavioural *sequelae*' (2014: 292, emphases added). Similarly, Rauh et al. (2012) discuss 'disruption' and 'reversal' of 'normal' brain sexual dimorphisms associated to EDCs exposure.

Thus, the scientific literature analysed reinforces not only a binary and normative depiction of sex-gender identities, but also a pathologising view of trans identities, even when it is not clearly stated.

Apart from the three main elements depicted, there is one more problematic element that the scientific literature on EDCs and trans identities shares: the inference and extrapolation of results from non-human animal experiments to human animals. However, as Donna Haraway insists, the 'differences matter', in species, ecologies, economies, lives (2016: 29), as well as in EDCs, doses, and exposure modes. Many, even scientific studies linking EDCs to sexual impairments in non-human animals, caution about extrapolating results between different species, doses, and routes of exposure (see Braun et al. 2009: 1950; Tamschick et al. 2016: 289).[34] In this regard, due to the physiological differences between experimental animals and human animals, the Endocrine Society acknowledges the need of determining whether the observed EDCs' effects in animal models, as well as the known mechanisms underlying these effects and effects in in vitro systems also occur in humans (Gore et al. 2015: 102). It also points out that the extrapolation is even more difficult in the case of behaviours (Gore et al. 2015: 92). Trans identities not only further problematize this extrapolation, but imply an unjustified inference, since there is a relevant difference between human and non-human animals in this regard: as far as we know, gender identity is not something that non-human animals 'have'.[35]

In the 'differences matter' framework, DES deserves special attention, besides being regularly present in the literature analysed, for being considered foundational regarding the hypothesis in humans. DES was administered to millions of Western women from the 1940s to 1970s to prevent miscarriage and other complications during pregnancy.[36] Not only was its inefficacy in this matter demonstrated very early, but it was also discovered a strong association with early-onset clear-cell adenocarcinoma of the

[34]The effects of EDCs are also organ-, tissue-, and cell-specific (Gore et al., 2015, 103). Moreover, there are 'cocktail effects', since most organisms are exposed to mixtures of EDCs. This means that EDCs, when in combination, can act in an additive, synergistic, or agonistic way (Santos et al. 2017). EDCs can have effects at low- and high-dose exposures or/and at middle range-dose exposures, and they may vary in type and degree; hormone receptor kinetics is important as well (Gore et al. 2015: 11-12). The route of exposure includes ingestion, inhalation, dermal absorption, and direct injection to target organs.

[35]Far from anthropocentric and exceptionalist views, this issue does not make humans ontologically distinct *fundamentally* or in *essence* from non-human animals. The notion of gender identity is also problematic regarding humans, but this matter exceeds the aim and scope of this paper.

[36]The doses of this potent oestrogen ingested by these women were particularly high (see Gore et al. 2015: 8; Hilakivi-Clarke et al. 2013: 28). For a comparison with the tolerable daily intake of, or the estimated daily exposure to, other prototypical EDCs, see EFSA CEP Panel (2023); Gore et al. (2015: 5).

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vagina, an increased risk for infertility, breast cancer, spontaneous abortion, preterm delivery, ectopic pregnancy, and cervical intraepithelial neoplasia in DES exposed daughters, as well as of cryptorchidism in DES sons (Harris and Waring 2012; Hilakivi-Clarke 2013: 28; Hoover et al. 2011). Potential associations are also being reported in the third generation (Gore et al. 2015: 8; Harris and Waring 2012: 111).

Regarding a possible association between DES exposure and trans identities, the first element that attracts attention is the weakness of the evidence. The only reference offered by the neurobiological theory about the origin of gender dysphoria to assess a 35% of transsexuality in DES sons is Scott P. Kerlin's study (2005), 'Prenatal exposure to diethylstilbestrol (DES) in males and gender-related disorders: results from a 5-year study', found on the website of DES sons. In this study, which involves 500 individuals with confirmed (60% of the sample) and suspected prenatal DES exposure, more than 150 identified themselves as either 'transsexual' (90), 'transgender' (48), or 'gender dysphoric' (17) (Kerlin 2005: 9). However, this paper was presented in a symposium, has not been published in any scientific journal, and cannot thus be considered a formal scientific work.

Aware of this fact, Swaab et al. (2021: 433) contend that a formal study is warranted. Indeed, Troisi et al. (2020) assess the associations of DES exposure with sexual orientation and gender identity in women and men who participated in the US National Cancer Institute DES combined cohort follow-up study. This is the first scientific study analysing DES exposure in relation to sexual orientation and gender identity, and its findings regarding trans identities sharply contrast with the data collected by Kerlin. From the 2,220 women and 933 men exposed, and the 1,086 women and 915 men unexposed, '[o]nly two women, both DES exposed, and three men (two exposed and one unexposed) reported gender identity that did not conform with the sex they were assigned at birth' (Troisi et al. 2020: 452).[37] These were too few people reporting a gender identity different from that assigned 'to analyze potential effects of prenatal DES exposure, but this suggests that any effect would be small' (Troisi et al. 2020: 452).

Conclusion

In this paper, I have analysed the scientific literature that deploys the hypothesis of an aetiological link between EDCs and trans identities, conformed by neurobiological theories on trans identities and scientific literature reviews. As previously pointed out by gender studies scholars regarding the scientific body of literature on the effects of EDCs on non-human animals' sex, the departure point of the aetiological hypothesis under analysis is a 'transsex panic', which is intimately related to its conception of trans identities.

Gender studies scholars who seek to reconceptualise the effects of EDCs from an open, dynamic, and relational or entangled view of sex converge in this characterization with several works from philosophy, biology, science, technology, and gender studies, and feminist neuroscience that address sex-gender identities. In sharp contrast with these elaborations, the aetiological hypothesis linking EDCs to trans identities is inscribed in a biologically deterministic account, which understands trans

[37]Five DES-exposed and other five unexposed persons did not answer the question on gender identity.

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identities and sex-gender identities in general as mainly prenatally determined by genes and hormones. This account ignores the active role that historical, social, cultural, and discursive factors play in the entangled trans identities, as well as their life-long dynamic processual character.

But the hypothesis under analysis involves something else that makes these two pieces, EDCs and biological determinism, fit together: the pathological understanding of trans identities. Here is where biological determinism and this theory of the abnormal go hand in hand: these toxic and disrupting substances would interfere by 'altering' the 'normal' or 'natural' sexual differentiation of the brain, resulting in 'reversals' of brain sexual dimorphisms or contributing to different brain phenotypes in trans persons. However, not only has the same pathologising logic been denounced by gender studies scholars examining the scientific literature on EDCs and transness in non-human animals, but the conception of brains as sexually dimorphic and the notion of group brain phenotypes have also been problematized by brain mosaicism.

Besides the fact that no direct causal relationship can be established in studies analysing prenatal exposure to EDCs and behavioural outcomes, this paper did not identify any scientific study associating EDCs to trans identities, for the only scientific study on this matter was not able to identify any association. The scientific literature that advances the hypothesis of this aetiological link mainly grounds it on extrapolations from studies with laboratory animals. Not only do 'differences matter' regarding species, but also tissues, EDCs, doses, routes of exposure, as well as other myriad of elements. There is an important difference in this respect: the human social construct of gender identity.

The lack of any kind of scientific evidence in this regard situates the hypothesis that causally links EDCs to trans identities closer to a tale than to a scientific hypothesis. To borrow the words of Jordan-Young (which, although were directed at hormones, acquire even more strength regarding ECDs), this does not mean that we know without the shadow of doubt that EDCs 'don't have any such effects (… you can't prove a negative). But it is definitely the case that such effects are not proven' (2010: 236). In the case of EDCs, their role as co-originators of trans identities is far from proven.

> So shall I end with the usual line that 'more research will be necessary' to sort this all out? Yes and no. Surely we need more research, but … I think it would be an extremely poor investment, both scientifically and socially, to continue pouring resources into trying to divide the indivisible. (Jordan-Young 2010: 236)

Furthermore, there is no need for elements that add more pathologisation. If EDCs were actually to take part in the emergence and development of sex-gender identities, it would be time to conceptualize not only their effects, but also trans identities themselves out of this pathologising, 'out of the normal order of things' schema.

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