How a common prescription medication has resulted in a dramatic rise in cases of Polycystic Ovary Syndrome – the leading cause of female infertility

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How a common prescription medication has resulted in a dramatic rise in cases of suffers with Polycystic Ovary Syndrome

Polycystic Ovary Syndrome (PCOS) is becoming increasingly common with latest figures estimating that 1 in 10 females in the UK suffer from the condition[1]. This rise in recent decades has caused a greater degree of research into the condition, with theories speculating links with other health conditions and environmental causes becoming more common. However, without a definitive correlation of the biological mechanisms the currently accepted hypothesis is that it is likely to be the result of genetic factors, though most of the literature will cite the cause as still unknown. Whilst genetic factors cannot be disregarded completely, especially with the acceptance of the condition dating back centuries, it is the transition from it being known as a rare condition to one that has become extremely common over recent decades that to me confirms genetics are not the major underlying cause of the condition. Of course there may be some genetic elements involved that may make one person more susceptible than another, by way of an anatomical or biological variation somewhere along the causation pathway, but it would seem irrational to suggest that genetics are the sole cause when we have seen a rapidly increasing number of cases in recent years[2].

The Production of Oestrogen and Testosterone in the Reproductive Glands

We must begin though with a little basic background knowledge of how both sexes produce testosterone and oestrogen in their respective reproductive organs; note that both sexes do produce both hormones, females do produce some testosterone as males do produce some oestrogen, albeit each in far lower quantities than their respective primary sex hormone. In both sexes the process is initiated in the hypothalamus by way of release of a chemical messenger known as gonadotropin-releasing hormone (GnRH), this in turn stimulates the pituitary gland to release both follicle stimulating hormone (FSH) and luteinizing hormone (LH) which in turn cause the respective reproductive organs to produce sex hormones.

Current literature confirms that LH is responsible for the production of testosterone in males and that FSH plays a role in sperm production[3]. It has also been confirmed that in females, the release of FSH is responsible for the onset of the development of the follicle (egg) and that LH is responsible for the maturation of the follicle[4]. The initial stage of development correlates with a rise in levels of oestrogen in the blood and once those levels are sufficiently high there a rapid spike in the release of LH, which in turn is followed by a rise in progesterone; progesterone being the hormone responsible for preparing the lining of the uterus for implantation of the developing egg. One important point worth noting here, which will become relevant later on[+], is that the spike in LH following this elevation in oestrogen signifies that the release of FSH and LH from the pituitary gland do not always correlate with a relative release in proportion to one another and that they can therefore be stimulated independently of each other. Although not required for the purpose of this article, there may be an obvious set of assumptions to make here that, although I have not searched excessively, do not yet appear to have been stated in the scientific literature. That of which, would be the potential similarities between the actions of the hormones when considered regarding the production of sperm in males and the production of the egg in females. I would therefore predict that it will later, if not already, be proven that the role of FSH in males is linked to the stimulation of oestrogen production, correlating with the initial development of the sperm cells, that testosterone then aids their maturity and that progesterone in males is responsible for preparing the environment in which they develop much like is the case with preparing the environment for the follicles to develop in females. It would then also be apparent that it may not be the LH itself that is responsible for the maturation of the egg in a female directly, but that LH stimulates the female to produce testosterone, as is the case in males, in order to aid the development and growth of the egg. Although these assumptions do seem obvious, the following processes regarding the causation of PCOS and indeed female infertility are not dependant on them, they are simply points relating to the fertility mechanisms of both sexes that should be explored further.

Artificial Hormonal Implementation

It is well known that the natural testosterone production in males who take anabolic steroids shuts down due to the high levels of artificial testosterone circulating in the blood which causes a disruption in the adrenal axis. The detection of these high testosterone levels by the hypothalamus results in a corresponding suppression of LH release from the pituitary gland shutting down the production of natural testosterone in the testes as the body has effectively been tricked into thinking that a high level of testosterone has been produced. Whilst in

most cases the adrenal axis will return to its normal state following cessation of usage of exogenous testosterone, in cases of extended usage duration, the recovery process becomes much harder and the complete restoration of the adrenal axis to its prior state becomes less certain. The greater the time span as to which exogenous testosterone is consumed, the greater the likelihood of a permanent suppression of LH production, resulting in a decreasing ability of the testes to produce natural testosterone.

During the initial stage of follicle development in a female the higher levels of oestrogen in the blood correlate with a suppression of FSH production via these same mechanisms, higher oestrogen detected by the hypothalamus results in decreasing levels of FSH being released by the pituitary gland, a process that is essential for the development of the follicle during pregnancy. However, when consuming exogenous oestrogen via birth control medication this same process will take place; as such, the mechanism behind which birth control pills functions is often described as a means of tricking the body into thinking it is pregnant. In such cases we are again disrupting the adrenal axis resulting in a suppression of FSH production and a corresponding suppression of oestrogen produced by natural means in the ovaries. Like the suppression of natural testosterone experienced by males, a greater time span of usage will result in a greater likelihood of longer lasting if not permanent suppression of the natural FSH and thus oestrogen production. As the higher levels of oestrogen during the development stage also stimulate the spiking of LH release, it may also be the case that excessive androgen hormones such as testosterone derivatives are also produced when a female is using the birth control pill due to the higher levels of oestrogen being detected with a far greater frequency than would be the case in a natural environment and therefore, the corresponding rapid releases of LH are likely to also be more frequent. Whilst the levels of LH during exogenous oestrogen consumption will need to be explored further with regards to scientific research, it is clear that the suppression of FSH alone will still alter the FSH:LH ratio in favour of higher levels of LH and it has been shown that this is coherent with PCOS[5]. Therefore, whilst consuming the pill without awareness to these disruptions to the adrenal axis and in cases where the axis fails to return to normal function following cessation of the birth control, the lower FSH:LH ratio will cause the symptoms of PCOS and without the corresponding production of required levels of oestrogens and progesterone, the follicles will be attempting to grown in an unfavourable environment. As such the increase in the levels of the androgen hormones relative to the levels of female sex hormones will result in symptoms such as the growth of the ovaries due to the growth factors these hormones exhibit as well as causing the common issues many female suffers of PCOS experience regarding hair loss as a result of excessive DHT (the androgenic hormone dihydrotestosterone) levels, the hormone responsible for male pattern baldness.

With the increasing usage of the birth control pill since its introduction, particularly with a dramatic increase in usage over the last few decades we should expect, as indeed is the case, that cases of PCOS are also increasing. Therefore, whilst some of the exact mechanisms of which I have been able to make considered assumptions will require further research, it can be said now with absolute certainty that it is the result of the usage of the birth control pill that is the fundamental cause of PCOS and thus the major cause of female infertility.

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[1] https://www.nhs.uk/conditions/polycystic-ovary-syndrome-pcos/

[2] https://www.thelogicalindian.com/amp/health/rising-cases-of-pcos-in-women-30634

[3] https://medlineplus.gov/lab-tests/follicle-stimulating-hormone-fsh-levels-test/

[4] https://crh.ucsf.edu/fertility/fertility_cycle

[+] Note that males experience a spike in oestrogen when the usage of exogenous testosterone is ceased, this would be the result of low testosterone being detected by the hypothalamus and therefore the pituitary is stimulated to produce both LH and FSH with the natural balance, which is heavily in favour of LH. But with the

LH production being supressed it can be expected that the result will be a relatively normal production of FSH alone which in turn will cause spikes in oestrogen production in the male due to the feedback loop where the detection of low testosterone continues. This of course can operate in the reverse manner in females when exogenous oestrogen is removed and LH levels rise due to the hypothalamus continuously detecting low oestrogen and thus stimulating the pituitary to release the natural ratio of a higher FSH to LH release, but with the FSH release being suppressed the LH alone will continue to be released in far higher amounts than would otherwise be the case with a healthy adrenal axis.

[5] https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7520057/