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REVIEW

Androgens in SARS-CoV-2 Coronavirus Infections

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Summary

Recent molecular biology findings have shown that for the penetration of the SARS-CoV-2 coronavirus into host cells, a key role is played by protease serine 2, the activity of which is dependent on androgens. The important role of androgens is also evidenced by clinical observations that men in some age categories are infected by this novel coronavirus up to two times more frequently than women. In addition, men with androgenic alopecia tend to have more serious clinical courses, while men with androgen deprivation as a result of prostate cancer treatments tend to have milder courses. This is in line with the fact that preadolescent children are only rarely sickened with serious forms of SARS-CoV-2 infections. Even though these observations may be explained by other factors, many authors have hypothesized that lowered androgen levels and blocking their activity using anti-androgen medication may moderate the course of the viral infection in intermediately- to critically-affected cases. Clearly, it would be important for androgen deprivation to block not just gonadal androgens, but also adrenal androgens. On the other hand, low androgen levels are considered to be a risk factor for the course of SARS-CoV-2 infections, either because low androgen levels have a general effect on anabolic-catabolic equilibrium and energy metabolism, or because of the ability of testosterone to modify the immune system. It is not yet clear if infection with this novel coronavirus might induce hypogonadism, leading to undesirable side effects on male fertility.

Key words

COVID-19 • Androgens • Adrenal androgens • Male fertility

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Introduction

The current coronavirus disease COVID-19, caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), has evolved into a global pandemic affecting the worldwide population. Two notably consistent findings are the low rates of prepubertal mortality, and that men are more likely to have severe symptoms and need hospitalization. The genetic predisposition of the host (Vašků 2020) and the activity of the immune system (Pačes *et al.* 2020) both play important roles, but the endocrine system also affects the course of infections.

The first biological requirement for SARS-CoV-2 coronavirus infectivity, which mainly causes acute respiratory syndromes, is the penetration of the virus into the host cells, generally type II pneumocytes. This is a complex process in which endocrine factors are involved.

Hypotheses on the influence of androgens on the course of SARS-CoV-2 infections were first described by Montopoli *et al.* in May 2020 (Montopoli *et al.* 2020) almost simultaneously with the group in the USA (Goren *et al.* 2020a). Montopoli and colleagues arrived at this hypothesis based on data on the different courses of coronavirus infections in men with prostate cancer treated with androgen deprivation therapy or treated with other methods. The role of sex hormones in the course of COVID-19 became a hot topic, and by the end of 2020 over a hundred studies had been published on the influence of androgens on coronavirus infections. In addition to clinical observations, these hypotheses were based on the molecular biology of the penetration of SARS-CoV-2 into host cells.

The basic process of coronavirus infection

A key step in coronavirus infection is the penetration of the virus into the host cells. Briefly, this can be described at the level of molecular biology as follows. The SARS-CoV-2 coronavirus enters the cell through receptors for angiotensin converting enzyme type 2 (ACE-2), which is expressed in pneumocytes. ACE-2 functions to metabolize angiotensin II to angiotensin variants with lower inflammatory activity. Angiotensin II binds to type 2 angiotensin receptors (AT1Rs), leading to the induction of pulmonary vasoconstriction and inflammation modulated by nuclear factor κB (NF-κB). This pathway increases the production of cytokines. Low levels of the conversion enzyme ACE-2 and high levels of angiotensin II result in the increased permeability of pulmonary veins and inflammatory damage to lung tissue (Gurwitz *et al.* 2020). The reason for lung damage during more severe SARS-CoV-2 infection stages is therefore currently thought to be from a cytokine storm resulting from a non-controlled immune response to lung damage (Shi *et al.* 2020, Al-Lami *et al.* 2020). The primary modulator at the beginning of this process is angiotensin II.

For viral attachment to the cell surface, proteolysis made possible by transmembrane protease serine 2 (TMPRSS2) is also necessary (Hoffmann *et al.* 2020). This protease allows the virus to bind to the ACE-2 receptor, and is considered necessary for SARS-CoV-2 coronavirus infection (Wambier and Goren 2020). Transcription of the TMPRSS2 gene is likely dependent on androgen receptors, and androgens are a pro-motor for this gene (Lucas *et al.* 2014). Factors that regulate angiotensin II levels therefore include not just those known from studies on the renin-angiotensin-aldosterone axis, but also androgens.

In addition, androgens also play other roles influencing the course of SARS-CoV-2 infections, such as suppressing the immune system, as well as effects on overall organism resilience and anabolic-catabolic equilibrium, on the cardiovascular system, and on metabolism in general.

Observations of the negative role of androgens on SARS-CoV-2 coronavirus infections

Risks of higher androgen levels

The first study to hypothesize that androgens may play a negative role in more severe cases of SARS-CoV-2 infections was a report (Montopoli *et al.* 2020) on observations of differences in patients with prostate cancer treated by androgen deprivation and those treated with other methods. This observation was supported by other authors (John and Kestner 2020, Cattrini *et al.* 2020, McCoy *et al.* 2020), though still other authors reported no such affect (Klein *et al.* 2021).

Worldwide statistics show that males are more likely to be victims of SARS-CoV-2 coronavirus infections (Giagulli *et al.* 2020, Cattrini *et al.* 2020, Wadman 2020) and that pre-pubertal children are possible carriers of the infection, but their course is largely asymptomatic. While these facts may be explained by reasons other than the hormonal environment (Al-Lami *et al.* 2020, Karlberg *et al.* 2004, Wenham *et al.* 2020), other findings indicate that androgens have a negative role in the course of coronavirus infection (Wadman 2020), not only in men but also in women with signs of hyperandrogenemia, such as those with polycystic ovary syndrome (Cadegieni *et al.* 2020b). Clinical observations have also shown a higher proportion of men with androgenic alopecia (AGA) among infected men than in the general population. AGA is thought to result from the action of dihydrotestosterone, the most potent androgen, on the hair follicles of the scalp (Wambier *et al.* 2020a,b,c, Goren *et al.* 2020a,b, Goren *et al.* 2021).

A relatively common cause of hyperandrogenization in younger individuals is anabolic steroid abuse. However, there is still a lack of information in the literature about the effect of anabolic-androgenic doping on the course of COVID-19. Another indicator of androgenization, in this case temporally localized to fetal development, is thought to be the index finger to ring finger ratio. A high mean 2D:4D ratio indicates prenatal low testosterone/high estradiol ratio. On the basis of a study on more than 180 000 men and women, Manning and Fink (2020) speculated that this ratio is associated with high case fatality rates and male mortality in coronavirus infection. However, a robust Bayesian analysis (Jones *et al.* 2021) of the data could not confirm this hypothesis.

In assessing the effect of androgens on SARS-CoV-2 infections, the involvement of adrenal androgens has not yet been taken into account, as they represent a small proportion of total androgens (Stárka *et al.* 2020b) and may change during the course of the disease, especially when dexamethasone or other corticosteroids are used.

Risks of low androgen levels

Hypotheses on the risk of higher androgen levels on the course of SARS are not supported, however, by other observations on the course of the disease and its mortality. On the contrary low total or free testosterone was reported as a risk. Less positive course of the disease was recorded in androgen deficient patients with testosterone levels below 5 nmol/l (Rowland and O´Brien Bergin 2020), in patients who needed testosterone replacement therapy (Rambhatla *et al.* 2021, La Vignera *et al.* 2020), or in association with the relatively frequent low testosterone in patients with COVID-19 pneumonia or other lung diseases (Pozzilli and Lenzi 2020).

It has been assumed that testosterone, in comparison to estrogen, may predispose men to widespread COVID-19 infections. Low serum testosterone levels, which may be supposed to characterize the hormonal milieu in seriously ill individuals, may predispose men, especially elderly men, to poor prognosis or death (Giagulli *et al.* 2021).

Decreased testosterone levels in critically ill men negatively affect endothelial cell function, lead to immune response disorders and less effective removal of the virus from the body, and contribute to systemic inflammation. Low testosterone could be a prognostic indicator of an adverse course of COVID-19 (Hussein *et al.* 2020).

Low testosterone levels also predicted clinical adverse outcomes in a study on a consecutive series of 31 male patients affected by SARS-CoV-2 pneumonia and recovered in the respiratory intensive care unit (Rasterlli *et al.* 2021). The study demonstrated that lower baseline levels of total testosterone and free testosterone levels predicted poor prognoses and mortality in SARS-CoV-2-infected men. Significantly lower testosterone levels and higher LH and prolactin were also found in patients with COVID-19 pneumonia by other authors (Okçelik 2021, Kadihasanoglu *et al.* 2021). On the contrary, a study in Asia (Xu *et al.* 2021) concluded that in males infected with SARS-CoV-2, most sex-related hormones (testosterone, FSH and LH levels) remain within the normal reference ranges after recovery, and no significant associations were observed between testosterone level and disease duration or severity. At present, there is insufficient evidence to show that SARS-CoV-2 causes hypogonadism and sterility, but the potential risk should not be ignored. Papadopoulos *et al.* (2021) came to a similar conclusion: it is not known whether low testosterone levels in aging hypogonadal males create a permissive environment for severe responses to COVID-19 infection or if the virus inhibits androgen formation.

The question has also been raised whether supplementation of low testosterone levels with androgen therapy alters the risk of SARS-coV-2 infections (Rambhatla *et al.* 2021, La Vignera *et al.* 2020). According to the findings by Rambhatla *et al.* 2021, there were no statistically significant differences between hypogonadal men that were not treated, and those on testosterone replacement therapy.

Regarding androgen levels, it should also be pointed out that the majority of patients with SARS-CoV-2 infection in intensive care units are obese men. In overweight and obese men in particular, we generally find lower concentrations of circulating total testosterone, but free and bioavailable testosterone tend to be within reference ranges (Stárka *et al.* 2020a). Indeed these latter two, and not total levels, are relevant to the role of testosterone in the body.

In considering the dual effect of testosterone in pulmonary viral infections, i.e. the adverse effects of both low and normal testosterone concentrations, some authors (Kezele 2020, Hussain *et al.* 2020, Pozzilli *et al.* 2020, Al-Lehmi *et al.* 2020) have concluded that these effects are not mutually exclusive and that they may depend on the stage of the disease. It is certainly justified to imagine different roles of sex hormones in different stages of the immune response *via* Th1 lymphocytes and the body's cellular response to toxins or in the stimulation of antibody production by Th2 lymphocytes.

SARS-CoV-2 coronavirus infections and male fertility

There are a number of hypotheses that suggest that COVID-19 could affect male fertility as an immediate or long-term consequence of the disease. One important finding is that the angiotensin converting enzyme 2 (ACE2) receptor, which aids the entry of SARS-CoV-2 into host cells, is highly expressed in testicular Sertoli and Leydig cells as well as in spermatozoa. Moreover, SARS-CoV-2 infection-induced uncontrolled inflammatory responses may lead to systemic oxidative stress, whose severe disruptive effects on testicular function are well documented (Dutta and Sengupta 2021). Consequences could include spermatogenesis failure, abnormal sperm motility, DNA fragmentation and male infertility, hypogonadism and

erectile dysfunction (Sansone *et al.* 2021, Haghpanah *et al.* 2021). Though these considerations are crucial for male health, clinical studies on this problem are rare. The largest study on 74 men recovered from COVID-19 reported no detection of SARS-CoV-2 in urine, prostatic secretions or semen (Ruan *et al.* 2021).

Hyperandrogenemia in women and COVID-19

Polycystic ovary syndrome (PCOS) is a common female endocrinopathy, with a prevalence ranging from 6 % to 20 % and often accompanied, in addition to hyperandrogenemia, by insulin resistance and obesity, which are risk factors for coronavirus infection. To date, however, there are no data relating SARS-CoV-2 to PCOS in women (Morgante *et al.* 2021). Data relating to SARS-CoV-2 in PCOS women were published by Subramanian *et al.* (2021). They identified 21,292 women with a coded diagnosis of PCO/PCOS and randomly selected 78,310 age and general practice matched control women. The crude COVID-19 incidence was 18.1 and 11.9 per 1,000 person-years among women with and without PCOS, respectively. Age-adjusted Cox regression analysis suggested a 51 % higher risk of COVID-19 among women with PCOS compared to women without PCOS (HR: 1.51 [95 % CI 1.27-1.80], $p<0.001$). Women with PCOS are at an increased risk of COVID-19 infection. On contrary the results of Yale *et al.* (2021) suggest that there is no evidence for an increased risk of COVID-19 infection, hospitalization, or mortality in women with acne vulgaris, PCOS, or hirsutism. Future studies on the course of COVID-19 in PCOS patients will need to take into account their phenotype, and will also need to distinguish the influences of hyperandrogenemia and obesity.

Conclusions

Findings from molecular biology on the mechanisms of SARS-CoV-2 penetration into host cells and clinical experience suggest that sex hormones, and androgens in particular, may play a role in the pathogenesis of this infection. Its course may be modified by both lower as well as higher androgen levels, depending on whether androgen-modified immunity or androgen-promoted viral entry into the cell predominates. Accumulated evidence suggests that there are links between high as well as low levels of testosterone in disease progression supporting the sex hormone role as a double-edged sword (Younis *et al.* 2021). We do yet not know whether the disease stages or other factors are determinant for the testosterone role in the disease course, and we have no evidence on whether coronavirus infections can adversely affect male fertility.

Conflict of Interest

There is no conflict of interest.

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