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Sexual Function of SARS-CoV-2 in Male Fertility

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Description

The scientific community has become more interested in learning more about the potential effects of severe acute respiratory syndrome-Coronavirus 2 (SARS-CoV-2) on human health. Numerous investigations have revealed that SARS-CoV-2 is either directly or indirectly responsible for the damage of the male reproductive system. There is some evidence to suggest that the virus may contribute to male infertility. In order to summarise the connection between the male urogenital tract, male fertility, and the gonadal hormone profile, this review will do just that. One of the organs with the highest expression of the ACE 2-receptor, which enables the virus to enter human cells, is the testis. One suspected clinical symptom of COVID-19 is orchitis, and testicular injury has been discovered during autopsies of COVID-19 individuals who passed away. The blood-testis barrier may be compromised by SARS-CoV-2 infection, favouring testicular injury and the generation of anti-sperm autoantibodies. SARS-CoV-2 has been found in semen in several investigations, and patients with COVID-19 had significantly changed sperm parameters compared to controls. Finally, decreased follicle-stimulating (FSH)/LH and testosterone/LH ratios, greater luteinizing hormone (LH) levels, and lower testosterone levels all point to primary testicular injury. In conclusion, more research is required to determine the precise pathways through which SARS-CoV-2 impacts the male reproductive system and fertility, as well as to determine whether or not these effects are permanent.

The pathogenic coronavirus known as Severe Acute Respiratory Syndrome-CoV-2 (SARS-CoV-2) was the culprit behind the COVID-19 pandemic of the acute respiratory illness. Epidemiological data from the World Health Organization (WHO) show that 583,238,204 COVID-19 cases have been reported globally, with 6,422,235 fatalities and 553,818,416 patients who have recovered.

Multiple organs and systems may be impacted by SARS-CoV2 [1]. The illness may even affect the gonadal function, which could reduce fertility. In this context, SARS-molecular CoV-2's traits point to its presence in the male reproductive system, which in combination with hormonal pathways may result in infertility. However, there are few and conflicting data about the presence of SARS-CoV-2 in the seminal fluid of infected individuals and its effects on sperm parameters [2].

We list all potential ways that the virus might impact the male reproductive system and, consequently, male fertility in this review.

Testis and Angiotensin-Converting Enzyme Receptor 2

It is well established that SARS-CoV-2 enters human cells through the angiotensin-converting enzyme (ACE) 2 receptor [3]. As a result of a potential sex-dependent sensitivity that could arise from a higher ACE2 expression in males, it is also known that COVID-19 appears to have a higher death rate in men [4]. The kidney, thyroid, heart, adipose tissue, small intestine, and testis have the highest levels of ACE2 expression, according to research by Meng-Yuan Li and colleagues [5]. According to these studies, SARS-CoV-2 can harm human tissues other than the lungs, such as the testis. A study using immunohistochemistry revealed that Leydig cells and seminiferous tubules both express ACE2. Wang and colleagues demonstrated the presence of ACE2 in spermatogonia, Leydig and Sertoli cells, and this finding has been validated. Based on a scRNA-seq investigation of human testicular cells, Shen and colleagues demonstrated that Sertoli cells are the most ACE2 mRNA positive cluster. Leydig cells and spermatogonia stem cells have both been discovered to have the ACE2 mRNA. The same study found that male infertile patients' testes had a greater positive rate of ACE2 than male fertile men's. According to these results, COVID-19 may be more likely to affect infertile males than fertile ones, and SARS-CoV-2 may cause male infertility via an ACE2-related route. This study even found an age-related variation in ACE2 mRNA expression, with a larger positive rate in middle-aged males compared to young men.

The renin-angiotensin-aldosterone system (RAAS), which includes the ACE2, regulates steroidogenesis, spermatogenesis, epididymal contractility, and sperm function in the male reproductive system. For instance, sperm motility appears to be influenced by angiotensin II. By cleaving the C-terminal amino acid of angiotensin II, the ACE2 catalyses the production of angiotensin 1-7, indicating that it offers a negative feedback regulation on the RAAS. The fact that spermatogonia from SARS-CoV-2 patients who tested positive for ACE2 appear to have impaired spermatogenesis is an intriguing observation in this regard.

These findings also point to SARS-ability CoV-2's to enter the cells of the male reproductive tract because ACE2 is present. Through a likely ACE2-related mechanism, this may disrupt spermatogenesis and steroidogenesis.

According to the research, SARS-CoV-2 may contribute to male infertility. However, more research is required to determine how the virus impacts testicular function and whether or not this effect is reversible. The ability of SARS-CoV-2 to infect testicles is well recognised. The presence of SARS-CoV2 RNA in the testes and sperm of COVID-19 patients serves as proof of this. In this area, more research is required to determine the exact location of the virus, whether it only exists in suspension or binds to spermatozoa, whether it is capable of replication, and how long it could remain in semen. Orchitis and testicular injury may be brought on by SARS-CoV-2 through both direct and indirect routes. Another piece of evidence supporting primary testicular injury involving the Leydig cells is the fact that patients with COVID-19 had hypotestosteronemia and high levels of LH. A larger percentage of compromised sperm parameters, including morphology, motility, and concentration, has been seen in COVID-19 individuals, according to research.

Conflict of Interest

None.

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