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Women's Immune System are Stronger Than Men's in Fighting COVID-19

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Abstract

Covid-19 pandemic affects all over the World. More than 14 million people affected and 6 lakh death occurs due to Covid-19. Men are more likely than women to die of the coronavirus, what factor protect the women in Covid-19 mortality. So scientists are thinking that female sex hormones responsible for that, so in womens mortality rate is less than compare to Men in Covid-19.

Key Words- COVID-19, Immunity, Woman, Men, Female sex hormone, Pandemic etc.

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INTRODUCTION

On March 11, 2020, the World Health Organization declared the coronavirus outbreak a pandemic. Since December 2019, the world has experienced an outbreak of coronavirus disease 2019 (COVID-19).

While men and women have the same susceptibility to COVID-19, men are more prone to higher morbidity and mortality independent of age. This difference can be justified by the cell energy hypothesis. Estrogens (as the main sex steroid of females) are potent stabilizers of ATP production during oxidative stress (e.g. during COVID-19-induced inflammation). Therefore, it seems that women are more capable to maintain the c-ATP of their immune cells during the immune response to COVID-19. With this notion in mind, men are more susceptible to immune dysregulation following COVID-19 infection.

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When it comes to fighting the deadly novel coronavirus which has killed over 2 million people globally, worldwide data suggest that the mortality rate is much higher in men than women.

In an exclusive interview to India Today, Dr Sharon Moalem, a Canadian-born physician and rare disease specialist, explained why women fared better in fighting Covid-19 than men.

"Females have two X chromosomes, whereas males have one X and one Y chromosome. X chromosomes are necessary for survival and contain important genes related to the brain. Y chromosomes, on the other hand, are found only in males and are not crucial for survival. Men are more biologically fragile due to this," Dr Sharon Moalem, who is also the author of 'The Better Half: On the Genetic Superiority of Women' said.

Today, the rapid outbreak of Corona Virus Disease 2019 (COVID-19 or SARS-CoV-2) is the leading health issue. There is a paucity of studies investigating the factors affecting immune response to COVID-19. In addition, there has been no detailed report for this immune response. Given the genomic similarity of 79% with Severe Acute Respiratory Syndrome coronavirus (SARS-CoV), nearly the same reaction to the immune system is expected for COVID-19. In response to SARS-CoV, both innate and adaptive immune systems are involved. SARS-CoV applies several mechanisms to overcome the immune response. First, it inhibits the rapid expression of interferon type 1 (IFN-1). IFN-1 is known as the "initial alarm" upon encounter with the virus that modulates the immune cells to the so-called "antiviral state". Moreover, SARS-CoV interferes with IFN-1 signaling through inhibition of STAT-1 phosphorylation. The third defensive mechanism of SARS-CoV is immune exhaustion through exaggerated and prolonged IFN-1 production by plasmacytoid dendritic cells (pDCs). This process leads to the influx of activated neutrophils and inflammatory monocytes/macrophages, that in turn, results in lung immunopathology (e.g. acute respiratory distress syndrome). Finally, the resulted so-called "cytokine storm" further weakens the immune system through IFN-1-mediated T cell apoptosis. In this article, we aim to provide a new hypothesis to describe how the repletion of cellular adenosine triphosphate (c-ATP) can promote immunity against COVID-19. Thereafter, we justify the current knowledge regarding the characteristics of COVID-19 infection by our hypothesis and give several approaches to improve the c-ATP.

The hypothesis

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Considering the pivotal role of ATP in cellular function, c-ATP depletion can lead to cellular dysfunction. Immune cells are not an exception. In this article, c-ATP is the index of cellular energy.

Evaluation of the Hypothesis

Here, we show how c-ATP repletion can counteract with defensive mechanisms of COVID-19 and promote the immune system to the enhancement pathway.

ATP Facilitates IFN Production

COVID-19 interferes with a rapid rise in IFN-1. Therefore, it deactivates the so-called "initial alarm" of the innate immune system, by unknown mechanisms. This facilitates its replication. Zhang et al. have demonstrated that enhancement in the c-ATP can reverse this process. This occurs by the facilitation of IFN secretion through P38/JNK/ATF-2 signaling pathway. Therefore, ATP-depleted cells are more susceptible to this effect of COVID-19.

ATP Facilitates IFN Signaling

Following IFN-1 secretion, fundamental changes occur in the immune cells that transform them into the so-called "antiviral state". One of the signaling pathways that take part in this process is the JAK/STAT pathway. JAKs are ATP-dependent enzymes that are bound to the cytoplasmic regions of cytokine receptors. Following attachment of IFN-1 to the cytokine receptor, JAK activates the STAT through trans-phosphorylation. Obviously, c-ATP depletion interferes with this process and further impairs transformation to "antiviral state".

ATP Prevents the Cytokine Storm

Following deactivation of "initial alarm", COVID-19 easily proliferates in-situ. Among the passive host-cells, there are exceptions that can react to the COVID-19, the pDCs. They detect the virus by toll-like receptor 7 (TLR-7). Upon attachment to viral nucleic acids, TLR7 induces profound IFN-1 expression. This response recruits other immune cells and causes massive local inflammation. At first glance, this robust immune response is beneficial for the elimination of COVID-19. However, two factors prevent it. First, impairment of IFN-1 signaling results in impairment of immune cell transformation to the "antiviral state". Therefore, they are not so effective in eliminating existing viruses. Second, persistence profound inflammatory responses may lead to immune exhaustion . The depletion of c-ATP can potentially enhance these detrimental processes in the following ways. In 2016, Rebbapragada et al. demonstrated the effect of ATP in the function of TLR7 by controlling the endo-lysosomal PH. They showed that ATP-

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depletion can increase the endo-lysosomal PH and improve the efficacy of TLR7. Therefore, ATP-depletion can potentially enhance profound IFN-1 secretion in this phase. Secondly, ATP-depletion can potentially prone the recruited immune cells to earlier exhaustion against COVID-19. Therefore, one may conclude that ATP-repletion can prevent the so-called "cytokine storm" and improve the cellular energy to better counteract with COVID-19.

ATP Prevents T-cell Apoptosis

Channappanavar etal. demonstrated that COVID-19 can promote T-cells to IFN-induced apoptosis, resulting in reduced numbers of virus-specific CD8 and CD4 T-cells. From the perspective of cellular energy, this process potentially occurs through IFN-mediated T-cell activation that results in c-ATP depletion. In line with this hypothesis, Perl et al. have shown that following IFN-γ stimulation, mitochondrial hyperpolarization and ATP depletion occurs in T-cells that results in apoptosis. Therefore, ATP-repletion can potentially prevent T-cell apoptosis following "cytokine storm".

Empirical Data

In the following section, we use our hypothesis to demonstrate why specific groups of people are more susceptible to be infected with COVID-19 and why they have a worse prognosis.

Elderly population The case-fatality rate of COVID-19 is the highest (14.8%) in elderly-population. In contrast, children have the lowest risk for both infection and mortality rates. This difference can be demonstrated from the cellular energy aspect. Aging may potentially attenuate the respiratory capacity of mitochondria. This condition may be either due to impairment of peroxisome proliferator-activated receptor-gamma coactivator-1α (PGC-1α) or age-related accumulation of mitochondrial DNA mutations. Moreover, aging can wane the ability of immune cells to secrete IFN following viral infection. As noted earlier, this may be due to ATP-depletion. Therefore, one can conclude that a gradual decline in prognosis with age may rely on a gradual decrease in c-ATP.

Tobacco smokers The risk of long-lasting and serious COVID-19 infection is more among tobacco smokers. Apart from a direct effect on lung parenchyma and a decrease in pulmonary capacity, tobacco smoke can potentially induce immune dysfunction through a decrease in the ATP content of immune cells. This can be due to nicotine-induced mitochondrial dysfunction. The resultant ATP-depletion increases the risk of immune dysregulation

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by COVID-19 (refer to the aforementioned defensive mechanisms of COVID-19).

Serious Chronic Medical Conditions

Recent reports have highlighted some chronic illnesses that increase the mortality of COVID-19. They include underlying conditions such as hypertension, diabetes, coronary heart disease, chronic obstructive lung disease, cancer, and chronic kidney disease. Apart from a decline in cardiovascular reserve, the effect of these chronic conditions on the prognosis of COVID-19 can be justified by our hypothesis.

CONCLUSION

Human cells need nutrients (including glucose, free fatty acids, essential amino acids, and O2) to maintain their c-ATP level. The aforementioned illnesses impede the regular distribution of the nutrients secondary to compromising the function and structure of small and large vessels. Therefore, the human cells (including in-situ immune cells) confront ATP-depletion and results in further immune dysregulation (as mentioned above).

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