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Research Article

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The Danger Within: Covid-19 Affinity for ACE2 receptors in Adipose Tissue and Testes. The Protective Effects of Estradiol, Fitness and Weight Management

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Abstract

The imminent danger of the Covid-19 pandemic has accelerated research in pharmaceuticals designed to interfere with the virus' entry into the body via ACE2 receptors, or the viral RNA replication that often overwhelms immune defences. The scope of this review was to elucidate the main human vulnerabilities like certain organs' enrichment in ACE2 receptors increasing viral affinity to males, the aged and certain pre-existing conditions including diabetes, CVD and pulmonary diseases, that deteriorate with increasing obesity, inflammation and toxicity. The current perspective focuses on the primary components of dysregulated health predisposing individuals to Covid-19, including hormonal imbalance,increased lipids and lipoproteins, thyroid dysfunction, degraded fitness, and age-related testosterone decline accompanied by cortisol increase that provokes stress eating behaviours and weight accumulation.We examined the molecular dynamics illustrating the action of new therapeutics necessary for Covid-19 patients; theestradiol advantage hypothesis; alternative therapies including hormone replacement procedures andmesenchymal stem cells; plus preventive and protective interventions. Obesity increases the probability of Covid-19 infection due to its abundance of ACE2 receptors.Physical activitymay decrease Covid-19 vulnerability, due to the diminished ACE-2 expression in the muscle. There are a number the fat managementsolutions featuring lasers and radiofrequency which, however do not enhance fitness. Seven recently published clinical studies with a total of 95 subjects, 73 males and 22 females, demonstrated visceral fat reduction combined with increasedskeletal muscle mass. A metanalysis performed on their data revealeda statistically significant decrease in several variables including BMI, lipids, lipoproteins, toxicityand inflammation as measured by CRP, Creatinine and Bilirubin, andoptimally healthier levels ofCortisol, Testosterone, Free T3,IGF-1, Insulin, and the appetite controlling hormones Leptin and Ghrelin.

Introduction

Coronavirus is an enveloped viral conglomerate, synthesized by 30,000 nucleotides, and expressed into a wide variety of diseases that vary from influenza, to the severe acute respiratory syndrome (SARS), the Middle East respiratory syndrome (MERS), and its current evolved version of SARS-Cov-2 or coronavirus disease 2019 (Covid-19) that has currently infected over thirty-four million individuals globally, with over a million, and constantly rising, mortality rates [1].

The Covid-19 Affinity for ACE2 Receptors

The Covid-19 two main genes ORF1a and ORF1b encodesixteen non-structural proteins, and four structural proteins: the spike (S), divided into S1 / S2 subtypes, membrane (M) and envelope (E) proteins on the viral surface, and the nucleocapsid (N) proteins, associated with the viral RNA. The S glycoproteins reflect the characteristic viral morphology surrounded by "coronas" the Greek word for crowns. S1 subunit recognizes and binds to angiotensin-converting enzyme 2 (ACE2) receptors, and S2 releases the fusion peptide to secure the connection [2,3]. ACE2 is a membrane-bound enzyme. ADisintegrin andMetalloprotease 17 (ADAM17) is able to cleave ACE2and cast it into the blood and body fluids, rendering the S1 /ACE2 fusion less likely [4].

The S1/ACE2 affinity has been documented for over 15 years [5,6,7]. The M and E proteins are in charge of the viral assembly and encapsulation of genetic material respectively [8,9]. The N proteins are intertwined with the viral genome and are involved in replicating and transcribing the viral RNA, eventually overwhelming the human biomolecular network. Due to the imminent threat of the pandemic most research has focused on therapeutics rather than prevention. A series of studies postulate that theophylline and pyrimidone can prevent the replicating ability of the N protein, by blocking contact of the protein's N-terminus with RNA, thus inhibiting viral multiplication [10]. Covid-19 does not respond to most nucleotide analogues (NA), designed to interfere with viral replication,due to the Covid-19 inherent Exonuclease (ExoN) domain that compromises NAs; however, it appears to be responsive to the new NA drug Remdesivir, that featuresthe active metabolite, GS441524 [11].Another therapeutic research target is drugs intended to obstruct the Covid-19 entry into the human system associated with TMPRSS2 inhibitors, such as camostatmesylate,ACE2 receptor blockers, or calmodulin antagonists [12,13]. Nevertheless, caution should be taken with ACE inhibitors often used to treat diabetes and heart disease. ACE inhibitors prevent the conversion of angiotensin I into Angiotensin II, a peptide in the renin-angiotensin system