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Anexa 5

STANDARDELE MINIMALE, NECESARE ȘI OBLIGATORII
PENTRU ÎNSCRIERE LA CONCURS
pentru ocuparea postului de Asistent Universitar
perioadă nedeterminată

- Deținerea diplomei de Doctor în domeniul postului[1]/Adeverinței Școala Doctorală
  Adeverință școală doctorală – opis pagina 6
- Deținerea titlului de medic rezident sau a unui titlu medical superior la disciplinele de concurs cu corespondent în Rețeaua Sanitară, în specialitatea postului
  Medic rezident Cardiologie – nu se aplică
- Minimum 2 lucrări în reviste din domeniul postului pentru care candidează (medicină, medicină dentară, farmacie sau științe inginerești, în conformitate cu postul scos la concurs), in extenso, dintre care 1 articol în reviste cotate ISI, prim autor sau autor corespondent, 1 articol în reviste BDI, prim autor.

  Lucrări în revistă cotată ISI[2]

  1. autor corespondent: 10.3390/life15081184, opis pagina 42-63
  2. autor corespondent: 10.3390/pharmaceutics14091906, opis pagina 64-88

  Lucrări în revistă indexată BDI[3]

  3. –
  4. –

Pentru posturile de asistent, altele decât cele din domeniul sănătate și științe inginerești (educație fizică, psihologie, limbi moderne), echivalarea standardelor minimale se face conform art.29, al.(9) din Metodologie.

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[1] *Nume diplomă, serie, număr*
[2] *Autori, titlu articol, nume revistă, volum, număr, paginație, factor de impact al revistei pentru anul publicării articolului, link*
[3] *Autori, titlu articol, nume revistă, volum, număr, paginație, numele bazei internaționale de date, link*

life

MDPI

Article

Atorvastatin Confers Renoprotection and Modulates Inflammation in Diabetic Rats on a High-Fat Diet

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Abstract

Objective: Uncovering the renoprotective and anti-inflammatory effects of atorvastatin treatment in diabetic-and-obese rats by employing traditional renal function indicators (urea and creatinine) and four prototypical cytokines (IL-1β, il-6, IL-17α, TNFα). Method: Twenty-eight male Wistar rats, aged 6 months, 350–400 g, were randomized into four groups. The first group, G-I, the denominated control, were fed standard chow over the whole course of the experiments. The rodents in G-II were exposed to a High-Fat Diet. The last two groups were exposed to Streptozotocin peritoneal injection (35 mg/kg of body weight). A short biochemical assessment was performed before diabetes model induction to ensure appropriate glucose metabolism before experiments. Following model induction, only rodents in group G-IV were gradually introduced to the same High-Fat Diet as received by G-II. Model confirmation 10 days after injections marked the start of statin treatment in group G-IV, by daily gavage of atorvastatin 20 mg/kg of body weight/day for 21 days. At the end of the experiments, the biochemical profile of interest comprised typical renal retention byproducts (urea and creatinine) and the inflammatory profile described using plasma levels of TNFα, IL-17α, IL-6, and IL-1β. Results: Treatment with Atorvastatin was associated with a statistically significant improvement in renal function in G-IV compared to untreated diabetic rodents in G-III. Changes in inflammatory activity showed partial association with statin therapy, TNFα and IL-17α mirroring the trend in urea and creatinine values. Conclusions: Our results indicate that atorvastatin treatment yields a myriad of pleiotropic activities, among which renal protection was clearly demonstrated in this model of diabetic-and-obese rodents. The statin impact on inflammation regulation may not be as clear-cut, but the potential synergy of renal function preservation and partial tapering of inflammatory activity requires further research in severely metabolically challenged models.

Keywords: statin; inflammation; diabetes mellitus; obesity; pleiotropy; renoprotective

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pharmaceutics

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Review

The Impact of Angiotensin-Converting Enzyme-2/Angiotensin 1-7 Axis in Establishing Severe COVID-19 Consequences

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Abstract: The COVID-19 pandemic has put a tremendous stress on the medical community over the last two years. Managing the infection proved a lot more difficult after several research communities started to recognize the long-term effects of this disease. The cellular receptor for the virus was identified as angiotensin-converting enzyme-2 (ACE2), a molecule responsible for a wide array of processes, broadly variable amongst different organs. Angiotensin (Ang) 1-7 is the product of Ang II, a decaying reaction catalysed by ACE2. The effects observed after altering the level of ACE2 are essentially related to the variation of Ang 1-7. The renin-angiotensin-aldosterone system (RAAS) is comprised of two main branches, with ACE2 representing a crucial component of the protective part of the complex. The ACE2/Ang (1-7) axis is well represented in the testis, heart, brain, kidney, and intestine. Infection with the novel SARS-CoV-2 virus determines downregulation of ACE2 and interrupts the equilibrium between ACE and ACE2 in these organs. In this review, we highlight the link between the local effects of RAAS and the consequences of COVID-19 infection as they arise from observational studies.

Keywords: renin-angiotensin-aldosterone system; RAAS; angiotensin-converting enzyme-2; ACE2; angiotensin 1-7; Ang (1-7); COVID-19; long-term effects

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1. Introduction

Angiotensin-converting enzyme-2 (ACE2), a type I transmembrane protein expressed in many organs such as the heart, kidneys, lungs (type II alveolar cell), and intestine, plays a central role in the pathology of COVID-19 infection [1]. As it was in the case of SARS-CoV, the coupling of the virus molecule to the ACE2 receptor leads to fusion and downregulation [2,3]. Angiotensin-converting enzyme (ACE) and ACE2 are involved in the function regulation of several organs, metabolic condition, and, ultimately, the renin-angiotensin-aldosterone system (RAAS). Currently, this axis is described as two closely interlocking components: a deleterious arm—ACE—and a protective arm—ACE2 [4]. Despite the beneficial side of ACE2, a higher propensity for severe forms of infection has been previously assigned to higher levels of circulating ACE2 [5]. The vast majority of the effects precipitated by COVID-19 infection on key peripheral organs are linked to derangement of the ACE2/ACE axis. There is an ever-growing number of studies focused on observing the impact of the infection outside the lung. Additionally, the considerable amount of medical records allows for a thorough analysis of risk factors.

In this review, we aim to shed light on the molecular mechanisms underlying the tremendous consequences of COVID-19 infection, while keeping the RAAS at the heart of the discussion.