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Hypertension and Obesity in Adolescents: Insights into the RAAS Pathways

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Introduction

The Renin-Angiotensin-Aldosterone System (RAAS) plays a pivotal role in regulating blood pressure and fluid balance, with both classical and alternative pathways contributing to its physiological effects. This review examines the involvement of RAAS pathways in the pathogenesis of hypertension and obesity in adolescents. Classical RAAS activation involves the conversion of angiotensinogen to Angiotensin II (Ang II), leading to vasoconstriction and aldosterone release, while alternative pathways, such as the Angiotensin-Converting Enzyme 2 (ACE2)/angiotensin-(1-7)/Mas receptor axis, counterbalance these effects by promoting vasodilation and natriuresis. In obese adolescents, dysregulation of RAAS pathways contributes to hypertension through mechanisms including adipocyte-derived angiotensinogen, insulin resistance and sympathetic nervous system activation. Understanding the interplay between classical and alternative RAAS pathways is crucial for developing targeted interventions to manage hypertension and obesity-related complications in adolescents.

The Renin-Angiotensin-Aldosterone System (RAAS) is a key regulatory pathway involved in blood pressure regulation and fluid balance. Its dysregulation is implicated in the pathogenesis of hypertension, a prevalent cardiovascular disorder with significant health implications, particularly in adolescents. Additionally, obesity, which has reached epidemic proportions worldwide, is closely associated with hypertension and further exacerbates cardiovascular risks in this population. Understanding the intricate interplay between classical and alternative pathways of the RAAS in the context of hypertension and obesity in adolescents is essential for developing effective therapeutic strategies to mitigate cardiovascular complications. The classical pathway of the RAAS involves the conversion of angiotensinogen to Angiotensin II (Ang II) via the actions of renin and Angiotensin-Converting Enzyme (ACE). Ang II exerts vasoconstrictive effects and stimulates aldosterone release, leading to sodium retention and increased blood pressure. In obese adolescents, adipocyte-derived angiotensinogen contributes to RAAS activation, perpetuating hypertension and promoting cardiovascular remodelling. Furthermore, insulin resistance, a hallmark of obesity, enhances RAAS activity, exacerbating hypertension through mechanisms involving oxidative stress and endothelial dysfunction [1,2].

Description

Emerging evidence highlights the role of alternative RAAS pathways, including the Angiotensin-Converting Enzyme 2 (ACE2)/angiotensin-(1-7)/Mas receptor axis, in counterbalancing the effects of the classical pathway. ACE2 cleaves Ang II to generate angiotensin-(1-7), which promotes vasodilation and natriuresis through Mas receptor activation. Dysregulation of the ACE2/angiotensin-(1-7)/Mas receptor axis has been implicated in the pathogenesis of hypertension and obesity-related complications in adolescents. Understanding the balance between classical and alternative RAAS pathways is crucial for elucidating the pathophysiology of hypertension and obesity and identifying novel therapeutic targets. This literature review aims to synthesize current knowledge on the involvement of RAAS pathways in hypertension and obesity in adolescents, with a focus on the underlying mechanisms and potential therapeutic implications. By elucidating the complex interplay between RAAS activation, hypertension and obesity, this review provides insights into the development of targeted interventions to mitigate cardiovascular risks in this vulnerable population [3].

Recent research has shed light on the intricate mechanisms underlying RAAS dysregulation in hypertension and obesity among adolescents. Studies have demonstrated that RAAS activation contributes not only to elevated blood pressure but also to the development of target organ damage, including cardiac hypertrophy, vascular remodelling and renal dysfunction. Moreover, the interaction between RAAS and other signalling pathways, such as the sympathetic nervous system and inflammatory cascades, further exacerbates cardiovascular risks in adolescents with hypertension and obesity. Interventions targeting RAAS pathways have emerged as promising strategies for managing hypertension and obesity-related complications in adolescents. Pharmacological agents that block RAAS components, such as ACE inhibitors, Angiotensin Receptor Blockers (ARBs) and mineralocorticoid receptor antagonists, have demonstrated efficacy in reducing blood pressure and improving cardiovascular outcomes in this population. Furthermore, lifestyle modifications, including weight management, physical activity and dietary sodium restriction, have been shown to attenuate RAAS activation and mitigate hypertension in obese adolescents. However, several challenges remain in translating these findings into clinical practice. Identifying biomarkers of RAAS dysregulation and personalized approaches to treatment are essential for optimizing therapeutic outcomes and minimizing adverse effects. Moreover, long-term studies are needed to assess the efficacy and safety of RAAS-targeted interventions in adolescents and elucidate their impact on cardiovascular morbidity and mortality [4].

The discussion of the Renin-Angiotensin-Aldosterone System (RAAS) pathways in the context of hypertension and obesity in adolescents encompasses a multifaceted exploration of the underlying mechanisms, therapeutic implications, challenges and future directions. Firstly, the discussion delves into the intricate interplay between classical and alternative RAAS pathways. Classical RAAS activation, characterized by angiotensin II-mediated vasoconstriction and aldosterone-induced sodium retention, contributes to elevated blood pressure in hypertensive and obese adolescents. Conversely, alternative RAAS pathways, such as the ACE2/angiotensin-(1-7)/Mas receptor axis exert vasodilatory and natriuretic effects, serving as a counterbalance to mitigate hypertension and its associated cardiovascular complications. Moreover, the discussion highlights the role of RAAS dysregulation in promoting target organ damage, including cardiac hypertrophy, vascular remodelling and renal dysfunction. Understanding the pathophysiological mechanisms underlying RAAS-mediated cardiovascular complications is crucial for developing targeted interventions to prevent disease progression and improve long-term outcomes in adolescents [5,6].

Conclusion

In conclusion, the discussion underscores the pivotal role of the RAAS

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in the pathogenesis of hypertension and obesity-related cardiovascular complications in adolescents. By elucidating the complex interplay between classical and alternative RAAS pathways, this review provides insights into the development of targeted interventions to mitigate cardiovascular risks in this vulnerable population. Moving forward, further research is warranted to unravel the underlying mechanisms of RAAS dysregulation, identify novel therapeutic targets and optimize treatment strategies for managing hypertension and obesity-related complications in adolescents. By addressing these challenges and embracing interdisciplinary approaches, we can advance our understanding of RAAS-mediated cardiovascular disease and improve the health outcomes of adolescents worldwide.

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