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Remodeling of Cellular Composition in the Rat Carotid Body's Neurogenic Niche Elicited by Prolonged Intermittent Hypoxia

Patricia Sobrino*

Department of Medical Physiology and Biophysics, University of Seville, Seville, Spain

Abstract

The Carotid Body (CB) stands as a prototypical organ specialized in acute oxygen (O_2) sensing, orchestrating reflexive hyperventilation and heightened cardiac output during instances of hypoxemia. The recurrence of intermittent hypoxia generates a repetitive stimulus that potentially triggers CB overactivation, contributing to the sympathetic hyperactivity observed in sleep apnea sufferers. Despite evidence showcasing the malleability of CB function due to chronic intermittent hypoxia (CIH), the intricate mechanisms underpinning this phenomenon remain partially understood. This study unveils that CIH elicits a modest expansion in CB dimensions and prompts a reconfiguration of cell types within the CB structure. Notably, this involves the mobilization of latent, immature neuroblasts, which embark upon a journey of differentiation, ultimately transforming into mature, O_2-sensing, neuron-like chemoreceptor glomus cells. Through the prospective isolation of distinct cell classes, we demonstrate that as CB neuroblasts mature, there is a concurrent surge in the expression of specific genes associated with acute O_2-sensing in glomus cells. Furthermore, CIH enhances the hypoxia-responsive capacity of mitochondria both in the progressing neuroblasts and the established glomus cells. This novel insight into the mechanisms governing CB-mediated sympathetic overflow offers a fresh perspective and the potential for the development of innovative pharmacological interventions with implications for sleep apnea patients.

Keywords: Cardiovascular • Metabolic alterations • Pathogenesis

Introduction

Obstructive sleep apnea represents a prevalent ailment within the human populace, predisposing individuals to substantial cardiovascular and metabolic disruptions. The activation of the carotid body, the primary arterial oxygen-sensing chemoreceptor, through recurrent episodes of hypoxemia, serves to intensify the carotid body-mediated chemoreflex. This, in turn, contributes significantly to the characteristic sympathetic overflow observed in sleep apnea patients. In rat models, the induction of chronic intermittent hypoxemia prompts a rapid surge in neurogenesis within the carotid body. This swift activation triggers neuroblasts to embark on a journey of proliferation and subsequent maturation into O_2-sensing chemoreceptor glomus cells [1].

Maturation processes experienced by carotid body neuroblasts and glomus cells under the influence of chronic intermittent hypoxia bring about an upregulation of genes associated with acute O_2 sensing. Furthermore, these cells exhibit an enhanced responsiveness to hypoxia at the mitochondrial level. These groundbreaking discoveries introduce new perspectives into the intricate mechanisms driving carotid body-mediated sympathetic hyperactivation. The possibility of pharmacologically modulating the rapid neurogenesis within the carotid body holds potential as a strategy to mitigate the detrimental effects stemming from chronic intermittent hypoxemia in sleep apnea patients.

The carotid body, a small cluster of chemosensory cells located at the bifurcation of the common carotid artery, plays a pivotal role in the body's response to changes in oxygen levels. It is a critical component of the peripheral chemoreflex pathway that helps regulate breathing and cardiovascular function. Chronic Intermittent Hypoxia (CIH), a condition characterized by recurring episodes of low oxygen levels, has been shown to significantly impact the carotid body's structure and function. Recent research has unveiled intriguing insights into how CIH-induced neurogenic changes lead to the rearrangement of cell types within the rat carotid body neurogenic niche [2,3].

***Address for Correspondence:** Patricia Sobrino, Department of Medical Physiology and Biophysics, University of Seville, Seville, Spain, E-mail: gortega1@us.es*

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Description

Cell types in the carotid body neurogenic niche

The carotid body comprises three main cell types: glomus (Type I) cells, sustentacular (Type II) cells, and undifferentiated neural crest-derived precursor cells. Glomus cells, the primary chemoreceptive cells, are responsible for detecting changes in arterial oxygen and carbon dioxide levels. Sustentacular cells, on the other hand, provide structural and metabolic support to the glomus cells and are actively involved in modulating their activity. The undifferentiated precursor cells are an essential component of the CB's regenerative capacity, contributing to the replenishment of glomus and sustentacular cells [4].

CIH-induced rearrangement of cell types

Chronic intermittent hypoxia triggers a series of complex adaptive responses within the carotid body, ultimately leading to a rearrangement of cell types within the neurogenic niche. One of the prominent effects of CIH is the proliferation and differentiation of the undifferentiated precursor cells. Studies have shown that CIH promotes the expansion of this cell population, enhancing the pool of potential glomus and sustentacular cells.

Furthermore, CIH appears to induce a phenotypic shift in sustentacular cells, potentially influencing their role in supporting glomus cell function. Research suggests that sustained exposure to intermittent hypoxia leads to alterations in sustentacular cell gene expression, resulting in changes to their metabolic and secretory properties. This shift may impact the overall functionality of the carotid body and contribute to its adaptive response to chronic oxygen fluctuations [5].

Implications and future directions

The rearrangement of cell types within the rat carotid body neurogenic niche induced by chronic intermittent hypoxia carries significant physiological

implications. The enhanced proliferation of undifferentiated precursor cells may represent a compensatory mechanism aimed at maintaining the CB's sensitivity to oxygen fluctuations. However, the long-term consequences of this increased proliferation on the CB's overall function and regenerative potential warrant further investigation.

Understanding the specific molecular mechanisms that underlie CIH-induced changes in sustentacular cells is another promising avenue of research. Unraveling the signaling pathways and gene regulatory networks involved in this phenotypic shift could shed light on novel therapeutic targets for respiratory and cardiovascular conditions linked to chronic hypoxia [6].

Conclusion

The carotid body's intricate network of cell types is finely tuned to detect and respond to changes in oxygen levels, ensuring the body's adaptation to varying environmental conditions. Chronic intermittent hypoxia disrupts this delicate balance, inducing rearrangement of cell types within the rat carotid body neurogenic niche. The increased proliferation of undifferentiated precursor cells and altered sustentacular cell properties are key features of this adaptive response. As research in this field progresses, a deeper understanding of CIH-induced neurogenic changes could pave the way for innovative strategies to mitigate the adverse effects of chronic hypoxia on respiratory and cardiovascular health.

Acknowledgement

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Conflict of Interest

None.

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