

Central Nervous System Dysfunction in Obesity induced Hypertension

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Abstract The activation of the sympathetic nervous system is a major mechanism underlying both human and experimental models of obesity related hypertension. While insulin and the adipokine leptin have long been thought to contribute to obesity related neurogenic mechanisms, the evidence is now very strong that they play a major role, shown particularly in animal studies using selective receptor antagonists. There is not just maintenance of leptin's sympatho-excitatory actions as previously suggested but considerable amplification particularly in renal sympathetic nervous activity. Importantly, these changes are not dependent on short term elevation or reduction in plasma leptin or insulin but require some weeks to develop indicating a slow "neural adaptivity" within hypothalamic signalling. These effects can be carried across generations even when offspring are raised on a normal diet. A better understanding of the underlying mechanism should be a high research priority given the prevalence of obesity not just in the current population but also for future generations.

Keywords hypertension, obesity, insulin, leptin, central nervous system, sympathetic nervous system, hypothalamus, high fat diet, aversive stress, blood pressure.

Introduction

The increase in the prevalence of obesity and the sequelae of hypertension, dyslipidemia, insulin resistance, diabetes, and cardiovascular disease, affecting both developed and developing countries has become a major worldwide burden of disease [1]. Analysis of the Framingham study suggests that over two thirds of newly diagnosed cases of hypertension are related to being overweight or obese [2]. The importance of determining the underlying mechanisms of obesity induced hypertension has therefore grown significantly in recent years. This is evidenced by the 3 fold increase in research articles on obesity hypertension in PubMed over the last

10 years while areas such as renovascular or essential hypertension have remained static. While there are many factors contributing to the increase in blood pressure in obese and overweight patients, increasingly, there is recognition of the important contribution of the sympathetic nervous system (SNS) [3–8]. The hypothalamus, well recognised for its role in regulating fluid balance, blood volume and sodium homeostasis is also the major site for the confluence of neural and hormonal signalling. Perhaps the most relevant of the peripheral signalling molecules are the adipokines leptin and adiponectin, the hormone insulin and gut hormones such as ghrelin. All of these hormones appear to relay metabolic information to the central nervous system (CNS) via specific receptors particularly within the arcuate nucleus of the hypothalamus. Indeed there is now a wealth of knowledge about the hypothalamus with respect to the afferent and efferent neural connections, the nature of the neurons within the subregions and the influence of a multitude of blood borne signalling molecules. The hypothalamus is also a key region for regulating the hormonal and neural response to acute emotional stress. Recent evidence suggests that there may well be overlap between the circuits processing the autonomic responses to stress with those responding to metabolic signalling. Thus, uncovering the cause of CNS dysfunction in obesity is imperative in order to understand the development of obesity-related hypertension. The purpose of this review is to document the progress that has been made in recent years and point the way for future research.

Contribution of the SNS to Obesity Related Hypertension

The landmark early studies from Landsberg and colleagues documented the very strong influence of eating on the activity of the SNS [9]. In a major hypothesis paper, Bray proposed a model that suggested that obesity resulted in

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1. Understanding the eBook Central Nervous System Dysfunction Manual Test
 - The Rise of Digital Reading Central Nervous System Dysfunction Manual Test
 - Advantages of eBooks Over Traditional Books
2. Identifying Central Nervous System Dysfunction Manual Test
 - Exploring Different Genres
 - Considering Fiction vs. Non-Fiction
 - Determining Your Reading Goals
3. Choosing the Right eBook Platform
 - Popular eBook Platforms
 - Features to Look for in an Central Nervous System Dysfunction Manual Test
 - User-Friendly Interface
4. Exploring eBook Recommendations from Central Nervous System Dysfunction Manual Test
 - Personalized Recommendations
 - Central Nervous System Dysfunction Manual Test User Reviews and Ratings
 - Central Nervous System Dysfunction Manual Test and Bestseller Lists
5. Accessing Central Nervous System Dysfunction Manual Test Free and Paid eBooks
 - Central Nervous System Dysfunction Manual Test Public Domain eBooks

- Central Nervous System Dysfunction Manual Test eBook Subscription Services
- Central Nervous System Dysfunction Manual Test Budget-Friendly Options

6. Navigating Central Nervous System Dysfunction Manual Test eBook Formats

- ePub, PDF, MOBI, and More
- Central Nervous System Dysfunction Manual Test Compatibility with Devices
- Central Nervous System Dysfunction Manual Test Enhanced eBook Features

7. Enhancing Your Reading Experience

- Adjustable Fonts and Text Sizes of Central Nervous System Dysfunction Manual Test
- Highlighting and Note-Taking Central Nervous System Dysfunction Manual Test
- Interactive Elements Central Nervous System Dysfunction Manual Test

8. Staying Engaged with Central Nervous System Dysfunction Manual Test

- Joining Online Reading Communities
- Participating in Virtual Book Clubs
- Following Authors and Publishers Central Nervous System Dysfunction Manual Test

9. Balancing eBooks and Physical Books Central Nervous System Dysfunction Manual Test

- Benefits of a Digital Library
- Creating a Diverse Reading Collection Central Nervous System Dysfunction Manual Test

10. Overcoming Reading Challenges

- Dealing with Digital Eye Strain
- Minimizing Distractions
- Managing Screen Time

11. Cultivating a Reading Routine Central Nervous System Dysfunction Manual Test

- Setting Reading Goals Central Nervous System Dysfunction Manual Test
- Carving Out Dedicated Reading Time

12. Sourcing Reliable Information of Central Nervous System Dysfunction Manual Test

- Fact-Checking eBook Content of Central Nervous System Dysfunction Manual Test
- Distinguishing Credible Sources

13. Promoting Lifelong Learning

- Utilizing eBooks for Skill Development
- Exploring Educational eBooks

14. Embracing eBook Trends

- Integration of Multimedia Elements
- Interactive and Gamified eBooks

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