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FUNCTIONAL AND STRUCTURAL CHANGES IN THE ADRENAL GLAND IN METABOLIC SYNDROME: THE HPA AXIS, ALDOSTERONE, AND THE EMERGING MASAD CONCEPT

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Abstract. *Metabolic syndrome(MetS) affects a substantial proportion of adults worldwide and is defined, according to the harmonized criteria issued jointly by the International Diabetes Federation, the American Heart Association, and other major bodies, by the co-occurrence of at least three of five components: abdominal obesity, elevated triglycerides, reduced HDL-cholesterol, hypertension, and elevated fasting glucose [1]. Its pathophysiology is traditionally framed around insulin resistance and visceral adipose tissue dysfunction. However, endocrine organs beyond the pancreas and adipose tissue also shape the metabolic phenotype, and the adrenal gland occupies a particularly strategic position: it produces glucocorticoids, mineralocorticoids, and catecholamines, all of which directly influence glucose metabolism, sodium handling, vascular tone, and fat distribution.*

Three broad lines of evidence link the adrenal gland to MetS. First, chronic activation of the HPA axis and resulting hypercortisolism share striking phenotypic similarities with MetS, giving rise to the long-standing "functional hypercortisolism" hypothesis [2]. Second, emerging histological and molecular data suggest that the adrenal cortex itself undergoes structural remodeling — steatosis, inflammation, and fibrosis — under conditions of diet-induced metabolic dysfunction, a phenomenon now termed metabolic dysfunction-associated steatotic adrenal disease, or MASAD [8]. Third, aldosterone and the broader renin-angiotensin-aldosterone system have been repositioned from simple regulators of blood pressure to active drivers of insulin resistance and adipose tissue dysfunction [9-15]. A fourth, clinically important thread connects these mechanisms to everyday practice: patients with incidentally discovered adrenal masses, particularly those with subtle autonomous cortisol secretion, show a disproportionately high burden of metabolic abnormalities [17-24].

This review integrates these four threads into a single narrative, moving from functional (hormonal) to structural (morphological) and finally to clinical manifestations, with the goal of presenting the adrenal gland as an active, modifiable component of metabolic syndrome rather than a downstream bystander.

Introduction

Metabolic syndrome is a common cluster of metabolic abnormalities that includes abdominal obesity, elevated triglycerides, reduced HDL-cholesterol, hypertension, and elevated fasting glucose. According to the harmonized criteria developed by the International Diabetes Federation, the American Heart Association, and other major organizations, the presence of at least three of these five components is sufficient for its diagnosis [1].

The condition is traditionally understood in terms of insulin resistance and dysfunction of visceral adipose tissue. However, metabolic regulation involves several endocrine organs, and the adrenal gland is particularly important because it produces hormones that influence glucose metabolism, blood pressure, fluid balance, and fat distribution.

In recent years, evidence has increasingly connected adrenal function with the development of metabolic syndrome. One important area of interest is the hypothalamic-pituitary-adrenal (HPA) axis. Chronic changes in HPA axis activity and cortisol secretion can produce metabolic effects that resemble several features of metabolic syndrome, including central adiposity, insulin resistance, and hypertension. This has led to the concept of functional hypercortisolism, in which alterations in cortisol regulation may contribute to metabolic dysfunction even in the absence of overt Cushing's syndrome [2].

A second area of growing interest is the structure and function of the adrenal cortex itself.

Experimental studies have suggested that prolonged metabolic disturbances may be accompanied by lipid accumulation, inflammation, and other forms of adrenal remodeling. These findings have contributed to the recently proposed concept of metabolic dysfunction-associated steatotic adrenal disease (MASAD) [8]. Although most of the available structural evidence comes from experimental models, the possibility that metabolic dysfunction may directly affect the adrenal gland has opened a new area of investigation.

The third area involves aldosterone and the renin-angiotensin-aldosterone system (RAAS).

Beyond their well-established roles in blood pressure and sodium regulation, aldosterone and related signaling pathways have been implicated in insulin resistance, adipose tissue dysfunction, and cardiovascular complications [9–15]. These mechanisms also have clinical relevance because patients with adrenal incidentalomas, particularly those with autonomous cortisol secretion, frequently present with metabolic abnormalities [17–24]. Thus, the relationship between adrenal disorders and metabolic syndrome may be relevant not only to basic pathophysiology but also to clinical practice.

Taken together, these findings suggest that the adrenal gland may be more than a passive participant in metabolic syndrome. Rather, changes in adrenal hormone regulation and, potentially, adrenal structure may interact with metabolic dysfunction in a bidirectional relationship. This review brings together the current evidence on these functional, structural, and clinical connections, with particular attention to HPA axis activity, cortisol, adrenal remodeling and MASAD, and aldosterone-RAAS signaling. By examining these mechanisms together, the review aims to clarify the potential contribution of the adrenal gland to the development and progression of metabolic syndrome.

2. The HPA Axis and Cortisol Dynamics in Metabolic Syndrome

2.1 The functional hypercortisolism hypothesis

The clinical features of overt Cushing’s syndrome, including central obesity, hypertension, glucose intolerance, and dyslipidemia, closely resemble many of the metabolic abnormalities seen in metabolic syndrome. This similarity has led to the hypothesis that even relatively small but persistent increases in glucocorticoid exposure may contribute to metabolic dysfunction in

individuals who do not have a classical endocrine disorder. The concept of “functional hypercortisolism” suggests that some patients with metabolic syndrome may have subtle alterations in hypothalamic-pituitary-adrenal (HPA) axis activity without developing the marked cortisol excess characteristic of Cushing’s syndrome [2]. Several factors have been proposed to contribute to this altered HPA axis activity, including chronic psychosocial stress, disturbed sleep, and early-life influences such as low birth weight. These factors have been associated with changes in cortisol secretion and stress responsiveness [2,5].

The potential metabolic effects of prolonged glucocorticoid exposure provide a possible explanation for this association. Glucocorticoids promote fat accumulation, particularly in visceral adipose tissue, partly by affecting adipocyte differentiation and lipid metabolism. At the same time, they increase hepatic gluconeogenesis and can impair insulin signaling in skeletal muscle.

Glucocorticoid excess may also contribute to increased vascular tone and blood pressure.

Together, these effects can produce several of the characteristic features of metabolic syndrome, including visceral obesity, insulin resistance, hyperglycemia, and hypertension [2,5,7].].

2.2 Conflicting clinical evidence

Despite the biological plausibility of the functional hypercortisolism hypothesis, clinical evidence has not been entirely consistent. Some case-control studies have reported evidence of altered HPA axis activity in individuals with metabolic syndrome. For example, patients with metabolic syndrome have shown higher cortisol levels following dexamethasone administration and greater ACTH responses during an oral glucose tolerance test compared with matched controls, despite having similar basal cortisol concentrations [4]. These findings suggest that the main abnormality may not necessarily be an increase in resting cortisol secretion, but rather an altered ability of the HPA axis to respond to physiological or pharmacological challenges and to maintain normal negative feedback.

However, other studies have not found a clear relationship between cortisol levels and metabolic syndrome. A systematic review and meta-analysis of observational studies found no significant overall association between basal cortisol concentrations and metabolic syndrome [3].

The authors therefore suggested that HPA axis dysfunction may not be a consistent feature in all individuals with the syndrome. One possible explanation is the considerable heterogeneity of metabolic syndrome itself. Altered cortisol regulation may be more apparent in particular subgroups, such as individuals with greater central adiposity, hypertension, or insulin resistance, rather than representing a universal characteristic of the condition [3].

2.3 Stress, sensitivity, and glucocorticoid signaling

The metabolic effects of glucocorticoids may depend not only on their concentration in the circulation but also on how strongly individual tissues respond to them. This tissue-level sensitivity is influenced by glucocorticoid receptor expression, the activity of 11β-hydroxysteroid dehydrogenase type 1 (11β-HSD1), and the local conversion of inactive cortisone into active cortisol in tissues such as adipose tissue and the liver [6,7].

In this way, 11β-HSD1 acts as a local amplifier of glucocorticoid action. It converts cortisone into cortisol within adipose tissue, and its activity or expression has been reported to be increased in visceral adipose tissue in obesity [6,7].

Chronic psychosocial stress may further contribute to this process by maintaining HPA axis activity and promoting visceral fat accumulation [5]. This creates a potentially self-reinforcing relationship: prolonged stress may increase glucocorticoid exposure and promote visceral adiposity, while increased adiposity may subsequently affect glucocorticoid metabolism and feedback regulation at both central and peripheral levels [5].

3. Structural Remodeling of the Adrenal Gland: The MASAD Concept

3.1 A new experimental model

Until recently, most research on adrenal involvement in metabolic syndrome focused on hormonal and functional changes, while much less attention was given to possible structural changes within the adrenal gland itself. This issue was explored in a 2026 experimental study in which adult rabbits were fed a high-fat diet (HFD) for 12 weeks to develop a metabolic syndrome-like phenotype. The adrenal glands were then examined using histological, immunohistochemical, and gene expression analyses [8]. As expected, the HFD-fed animals developed several features of metabolic dysfunction, including obesity, insulin resistance, and hypertension. In addition, their adrenal glands were enlarged and showed increased lipid accumulation, macrophage infiltration, inflammatory changes, and fibrosis compared with animals receiving a regular diet [8].

Based on these findings, the investigators proposed the term metabolic dysfunction-associated steatotic adrenal disease (MASAD) to describe the observed pattern of diet-associated adrenal changes. The concept was intentionally compared with metabolic dysfunction-associated steatotic liver disease (MASLD), in which metabolic dysfunction is associated with abnormal lipid accumulation and subsequent tissue injury [8,16]. Although these findings provide an interesting potential link between systemic metabolic dysfunction and adrenal structural changes, further research is needed to determine whether similar changes occur in humans and whether they have direct clinical significance.

3.2 A steroidogenic shift and RAAS-independent hypertension

The structural changes observed in MASAD were accompanied by significant changes in adrenal steroid production. Despite the presence of hypertension, the HFD-fed rabbits had lower aldosterone levels and reduced expression of genes involved in the renin-angiotensin system.

These findings suggest that classical RAAS overactivity was unlikely to be the main cause of hypertension in this model [8]. Instead, the expression of several key steroidogenic enzymes was reduced, while the levels of some steroid intermediates increased. In particular, plasma progesterone and 11-deoxycorticosterone (11-DOC), a biologically active mineralocorticoid precursor, were elevated [8].

The expression of CYP21A2 also differed between the adrenal cortical zones, with lower expression in the zona glomerulosa and higher expression in the zona fasciculata. This finding suggests that steroid synthesis may be altered differently across the adrenal cortex as structural

changes develop [8]. Notably, 11-DOC levels were positively correlated with all individual features of metabolic syndrome assessed in the study.

This raises the possibility that 11-DOC may contribute to the association between adrenal structural changes and hypertension through a mechanism that is independent of classical RAAS activation [8]. However, whether this mechanism also occurs in humans remains to be established.

3.3 The translational gap

These findings suggest that the adrenal gland may be a target of metabolic injury, rather than simply an organ that responds to metabolic stress through changes in hormone secretion. In this respect, the adrenal gland may share some features with other metabolically affected organs, such as the liver. However, the current evidence is still limited because the structural findings come from a single animal model. At present, there is no direct histological evidence confirming similar adrenal steatosis, inflammation, or fibrosis in humans with obesity or metabolic syndrome.

Human evidence is also largely indirect, based mainly on hormonal measurements and imaging studies rather than direct examination of adrenal tissue. Adrenal biopsy is rarely performed unless there is a specific adrenal mass requiring investigation, which makes direct assessment of structural changes difficult. Therefore, an important question for future research is whether similar remodeling occurs in the human adrenal gland and, if so, whether these changes can be reversed when metabolic health improves.

4. Aldosterone and the Renin-Angiotensin-Aldosterone System

4.1 Adipose tissue as a driver of aldosterone excess

The relationship between aldosterone and metabolic syndrome has become more complex than the traditional view of aldosterone as a hormone released mainly in response to classical RAAS activation. Increasing evidence suggests that adipose tissue may also contribute directly to increased aldosterone production in obesity. One important study showed that leptin, an adipocyte-derived hormone, can act on cells of the adrenal zona glomerulosa and increase the expression of aldosterone synthase (CYP11B2) through a calcium-dependent pathway. This mechanism was associated with endothelial dysfunction and cardiac fibrosis and appeared to occur independently of the classical renin-angiotensin system [9].

Subsequent studies have provided further evidence for a leptin-aldosterone pathway linking adipose tissue to adrenal aldosterone production, with some findings suggesting that the strength of this response may differ between males and females [10]. In addition, increased visceral and perirenal adipose tissue has been associated with idiopathic hyperaldosteronism and low-renin hypertension. These associations may involve both local adipose-derived signaling molecules and mechanical effects of excess perirenal fat on the renal vasculature [14]

4.2 Aldosterone-induced insulin resistance

Beyond its established role in sodium retention and blood pressure regulation, aldosterone may also contribute directly to insulin resistance. Excess aldosterone has been shown to reduce the expression of insulin receptor substrate-1 (IRS-1) in vascular smooth muscle cells, which can impair nitric oxide-mediated vasodilation and promote vascular remodeling and stiffness [11,14].

Activation of the mineralocorticoid receptor (MR) in adipose tissue and skeletal muscle may further interfere with glucose uptake. In parallel, increased reactive oxygen species (ROS) associated with RAAS activation can contribute to insulin resistance in cardiovascular and metabolic tissues through oxidative stress [12,13].

Together, these findings suggest that the effects of aldosterone extend beyond its classical actions on fluid balance and blood pressure. Its direct effects on metabolic and vascular tissues may help explain the close relationship between aldosterone excess, insulin resistance, hypertension, and other features of metabolic syndrome [11,15]

4.3 The cardiovascular-kidney-metabolic (CKM) framework

Taken together, these findings have contributed to a broader clinical perspective known as cardiovascular-kidney-metabolic (CKM) syndrome, which emphasizes the close interaction between metabolic, renal, and cardiovascular disorders. Within this framework, aldosterone and hyperaldosteronism have been proposed as important links between adipose tissue dysfunction, renal abnormalities, and cardiovascular remodeling [14]. Rather than acting only as a regulator of sodium balance and blood pressure, aldosterone may therefore serve as an important signaling link between the adrenal gland and several organs involved in metabolic syndrome.

5. Adrenal Incidentaloma and Subclinical Cushing's Syndrome

5.1 Prevalence and hormonal patterns

Adrenal incidentalomas are adrenal masses that are discovered unexpectedly during imaging performed for reasons unrelated to suspected adrenal disease. They are found in approximately 4% of imaging studies, and although most are hormonally inactive, a clinically relevant proportion show some degree of autonomous cortisol secretion without the typical features of overt Cushing’s syndrome [17]. This condition, historically referred to as subclinical Cushing’s syndrome (SCS), has been reported in approximately 6–19% of patients with adrenal incidentalomas, although the reported prevalence varies depending on the diagnostic criteria and cortisol thresholds used [18,19].

5.2 Metabolic consequences

Multiple cohort studies have reported a higher burden of metabolic abnormalities among patients with SCS compared with those who have non-functioning adrenal adenomas. These abnormalities include higher body mass index, waist-to-hip ratio, fasting insulin, HOMA-IR, total and LDL cholesterol, triglycerides, and blood pressure, together with lower HDL cholesterol and adiponectin levels [19–21]. Even adrenal incidentalomas considered hormonally “silent” have been associated with higher rates of hypertension, type 2 diabetes, and metabolic syndrome compared with the general population. This raises the possibility that current biochemical criteria may not fully reflect the metabolic effects of relatively low levels of autonomous cortisol secretion [18].

The potential clinical significance of this association extends beyond individual metabolic parameters. Long-term follow-up studies have reported higher rates of cardiovascular events and mortality among patients with SCS compared with those with non-secreting adrenal incidentalomas [22].

These findings suggest that even relatively mild autonomous cortisol secretion may have clinically relevant metabolic and cardiovascular effects, although the extent of this risk and the mechanisms involved require further investigation.

5.3 Effect of adrenalectomy

Some of the more direct evidence linking adrenal cortisol secretion with metabolic abnormalities comes from studies examining outcomes after adrenalectomy. In patients with adrenal incidentalomas and SCS who underwent laparoscopic adrenalectomy, cortisol secretion generally returned toward normal, and improvements in hypertension, diabetes, and obesity were reported in a substantial proportion of patients during follow-up [23,24]. These findings suggest that reducing autonomous cortisol secretion may improve several metabolic abnormalities in selected patients.

However, the available evidence should be interpreted carefully, as surgical outcome studies are generally observational and patient selection may influence the results. Nevertheless, the improvement in metabolic parameters following adrenalectomy provides important human evidence that autonomous adrenal cortisol secretion may contribute to metabolic dysfunction and may represent a modifiable factor in some patients with SCS [23,24].

6. Integrating the Evidence: A Unified Model

Taken together, the available evidence suggests that the adrenal gland may contribute to metabolic syndrome through several interconnected mechanisms. At the functional level, chronic psychosocial and metabolic stress may alter HPA axis activity in susceptible individuals, resulting in a pattern of altered cortisol regulation that may be more apparent during dynamic testing than in basal cortisol measurements [2–7]. Prolonged metabolic stress may also be accompanied by structural changes in the adrenal cortex. In the experimental MASAD model, these changes included lipid accumulation, macrophage infiltration, and fibrosis, together with alterations in steroidogenic activity and increased levels of mineralocorticoid intermediates such as 11-DOC [8].

At the same time, dysfunctional and expanding adipose tissue may influence adrenal aldosterone production through leptin and other adipose-derived signaling pathways. Increased aldosterone activity can then affect vascular and metabolic tissues, potentially contributing to insulin resistance and hypertension [9–15]. Clinically, the relationship between adrenal hormone activity and metabolic dysfunction is particularly evident in patients with adrenal incidentalomas and autonomous cortisol secretion, who frequently have a greater burden of metabolic and cardiovascular abnormalities. Improvements observed after adrenalectomy in selected patients provide further evidence that autonomous adrenal hormone secretion may contribute to these abnormalities [17–24].

Overall, these findings suggest that changes in HPA axis activity, adrenal structure, aldosterone signaling, and autonomous cortisol secretion should not necessarily be viewed as completely separate phenomena. Instead, they may represent interconnected aspects of adrenal involvement in metabolic disease, although the extent to which these mechanisms form a single continuous process remains uncertain.

Further human studies are needed to clarify how these pathways interact and whether structural and functional adrenal changes can be modified through metabolic or targeted treatment.

7. Limitations and Future Directions

Several limitations should be considered when interpreting the current evidence. First, the structural evidence underlying the concept of MASAD comes from a single rabbit model and has not yet been confirmed in humans. Further studies are needed to determine whether similar adrenal changes occur in patients with obesity or metabolic syndrome, potentially using imaging methods to assess adrenal fat accumulation and, where available, histological correlation [8].

Second, clinical studies examining cortisol dynamics in metabolic syndrome are heterogeneous in terms of diagnostic criteria, cortisol assays, and population characteristics. These differences may partly explain the inconsistent findings between studies reporting altered HPA axis activity and meta-analyses that have found no significant association with basal cortisol levels [3,4]. Third, most studies of adrenal incidentalomas are cross-sectional or retrospective, which limits the ability to establish causal relationships between autonomous adrenal hormone secretion and metabolic abnormalities. Although improvements following adrenalectomy provide supportive evidence, these studies are also subject to limitations related to study design and patient selection [23,24].

Future research should therefore focus on longitudinal human studies that evaluate multiple aspects of adrenal involvement within the same population, including dynamic HPA axis testing, imaging-based assessment of adrenal fat, and aldosterone/RAAS profiling. It will also be important to determine whether improvement in metabolic health through interventions such as weight loss or bariatric surgery is accompanied by changes in adrenal structure and function.

Similarly, studies of mineralocorticoid receptor antagonism may help clarify the contribution of aldosterone signaling to metabolic and cardiovascular abnormalities. Such approaches could provide a better understanding of whether adrenal abnormalities are reversible consequences of metabolic dysfunction or contribute independently to its progression.

8. Conclusion

Overall, the available evidence suggests that the adrenal gland may have an important role in the pathophysiology of metabolic syndrome rather than functioning only as a secondary endocrine target. Findings related to HPA axis regulation, adrenal structural changes described in MASAD, aldosterone signaling, and the metabolic abnormalities observed in patients with adrenal incidentalomas all point to a possible interaction between adrenal function and metabolic dysfunction.

However, the strength and clinical relevance of these associations remain uncertain, particularly because much of the structural evidence comes from experimental models and the human clinical literature is heterogeneous.

Further studies are needed to confirm these mechanisms in humans and to determine whether adrenal structural and functional abnormalities can be modified through metabolic or targeted treatment.

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