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RESEARCH ARTICLE

Differential Associations of Serum Cortisol Levels and Hepcidin with Body Mass Index and Waist Circumference in Healthy Young Men

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Abstract

BACKGROUND: Cortisol is a glucocorticoid hormone involved in the physiological response to stress and metabolic regulation, while hepcidin regulates systemic iron homeostasis and is influenced by inflammation and metabolic conditions. Although they are involved in distinct physiological pathways, alterations in both biomarkers may be associated with adiposity through metabolic and inflammatory processes. However, their associations with adiposity measures in healthy young men remain insufficiently understood. Therefore, this study was conducted to assess serum cortisol and hepcidin levels and their correlations with body mass index (BMI) and waist circumference (WC).

METHODS: An analytic observational study with a cross-sectional design was conducted among 51 healthy students aged 18–22 years with no history of acute or chronic infection at the Faculty of Medicine, Diponegoro University. Blood was collected from subjects, and serum cortisol and hepcidin levels were measured using sandwich enzyme-linked immunosorbent assay (ELISA). BMI was calculated from body weight and height, and WC was measured at the umbilical level. Correlation analyses used Spearman or Pearson tests according to data distribution.

RESULTS: Mean serum cortisol was 108.84±39.29 ng/mL and mean hepcidin was 2.39±1.19 ng/mL. Median BMI was 22.14 kg/m² (15.63–37.49), and median WC was 76.00 cm (64.00–113.30). Cortisol levels showed significant positive correlations with BMI (r=0.353, p=0.011) and WC (r=0.386, p=0.005). In contrast, hepcidin levels were not significantly correlated with BMI (r=0.175, *p*=0.216) or WC (r=0.157, *p*=0.266).

CONCLUSION: Serum cortisol was positively correlated with overall and central adiposity, whereas hepcidin was not. Cortisol may therefore be more closely associated with adiposity in healthy young men.

KEYWORDS: cortisol, hepcidin, body mass index, waist circumference

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Introduction

Obesity is increasingly recognized as a chronic low-grade inflammatory and metabolic condition rather than simply an excess accumulation of body fat. Adipose tissue is an active endocrine organ that contributes to systemic metabolic regulation through the secretion of hormones, adipokines, and inflammatory mediators. Dysregulation of these endocrine and inflammatory processes in obesity may contribute to metabolic disturbances and obesity-related complications.(1)

Cortisol, the principal glucocorticoid hormone synthesized in the zona fasciculata of the adrenal cortex, plays an important role in metabolic homeostasis, stress adaptation, immune regulation, and blood pressure control. (2-4) Its secretion is tightly regulated by the hypothalamic–pituitary–adrenal (HPA) axis, a key neuroendocrine

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system activated in response to physical and psychological stress.(5) Dysregulation of HPA axis activity has been associated with metabolic disturbances and alterations in cortisol dynamics in obesity. Studies have demonstrated altered adrenocorticotropic hormone (ACTH)–cortisol responsiveness in obesity and increased cortisol production with increasing body mass index (BMI) and body fat, whereas circulating free cortisol may remain unchanged because of enhanced cortisol clearance.(6-8) These findings suggest that the relationship between cortisol and adiposity may be complex and dependent on the cortisol parameter assessed.

In parallel, hepcidin is a peptide hormone primarily synthesized in the liver and serves as the principal regulator of systemic iron homeostasis by controlling the activity of ferroportin, the major cellular iron exporter.(9,10) Beyond its role in iron metabolism, hepcidin is responsive to inflammatory signaling, particularly through pro-inflammatory cytokines such as interleukin-6 (IL-6), as well as metabolic and adipokine signaling.(11) Adipose tissue has also been shown to express hepcidin, particularly under inflammatory conditions, suggesting a potential relationship between adiposity, inflammation, and iron homeostasis. (12) However, the association between circulating hepcidin levels and adiposity remains insufficiently characterized.

Adiposity is commonly assessed using BMI and waist circumference (WC), which provide complementary information on overall and central adiposity, respectively. While BMI is widely used to assess body size, it cannot distinguish fat mass from lean mass, whereas WC provides an indirect measure of central adiposity and may better reflect visceral fat accumulation and its metabolic consequences.(13-15) Although cortisol and hepcidin are linked to metabolic and inflammatory processes, they represent distinct biological pathways that may have different relationships with adiposity. However, limited evidence has examined both biomarkers simultaneously in relation to BMI and WC, particularly among healthy young men. Therefore, this study was conducted to assess serum cortisol and hepcidin levels and determine their correlations with BMI and WC in healthy young men.

Methods

Study Design and Subject Recruitment

A cross-sectional observational analytic study was conducted at the Faculty of Medicine, Diponegoro University, Semarang, Indonesia. Subjects were recruited using consecutive sampling. The final analysis included 51 healthy male medical students with body temperature of 36–37°C at the time of data collection and no history of acute or chronic infection. The study was restricted to male participants to minimize potential biological variability and confounding related to menstrual status and iron homeostasis.

Subjects were excluded if they had a history of adrenal or pituitary disease; conditions related to iron metabolism, including iron-deficiency anemia; were receiving iron supplementation; or had used medications that could affect cortisol concentrations, including oral or implantable estrogen preparations, inhaled corticosteroids (budesonide or fluticasone), injectable corticosteroids, oral corticosteroids (prednisone, methylprednisolone, or dexamethasone), or topical corticosteroids. Subjects reporting herbal medication use or consumption of more than three cups of strong coffee or tea per day were also excluded.

Anthropometric Measurements

Anthropometric measurements were performed using standardized procedures. Body weight was measured using a digital weighing scale (GEA EB-9350, GEA Medical, Tangerang, Indonesia), and body height was measured using a OneMed stature meter. WC was measured using a non-stretchable measuring tape with a measurement precision of 0.5 mm. Measurements were taken with subjects standing upright, at the midpoint between the lower margin of last palpable rib and iliac crest, at the end of a normal expiration.

BMI was calculated as body weight in kilograms divided by height in meters squared (kg/m^2). BMI was categorized according to the Asia-Pacific criteria as underweight (<18.5 kg/m^2), normal weight (18.5–22.9 kg/m^2), overweight (23.0–24.9 kg/m^2), obesity class I (25.0–29.9 kg/m^2), and obesity class II (≥30.0 kg/m^2). Each anthropometric measurement was performed twice, and the mean of the two measurements was used for analysis.

Serum Cortisol Measurement

A total of 3 mL of venous blood was collected in the morning at approximately 8 AM by venipuncture using a syringe and transferred into a plain tube without anticoagulant. The blood samples were then processed to obtain serum for the measurement of cortisol concentrations. Serum cortisol concentrations were measured using a competitive enzyme-linked immunosorbent assay (ELISA) with a Human Cortisol ELISA Kit (Catalogue No. E-EL-0157; Elabscience Biotechnology, Wuhan, China) with a detection range of 6.25–400 ng/mL and a sensitivity of 2.92 ng/mL at the

Iodine Deficiency Disorders (GAKI) Laboratory, Faculty of Medicine, Diponegoro University, Semarang, Indonesia.

Standards, blanks, and samples were added to the microplate wells, followed immediately by the addition of the Biotinylated Detection Antibody working solution. The plate was sealed and incubated for 45 minutes at 37°C. After this incubation, the liquid was aspirated from the wells, and the plate was washed three times to remove unbound components. HRP Conjugate working solution was then added to each well, and the plate was incubated again for 30 minutes at 37°C before undergoing a second, five-cycle wash process to eliminate excess enzyme. The enzymatic reaction was initiated by the addition of Substrate Reagent, followed by a 15-minute incubation at 37°C in the dark, until a blue color developed. Finally, the Stop Solution was added to terminate the reaction, turning the color yellow. The optical density (OD) of each well was measured immediately at 450 nm using a microplate reader. The reference range for morning serum cortisol was 5–25 µg/dL (50–250 ng/mL).

Serum Hepcidin Measurement

Similar to cortisol measurement, blood samples for hepcidin measurement were collected in the morning at approximately 8 AM. Serum hepcidin concentrations were measured using a Human Hepc (Hepcidin) ELISA Kit (Catalogue No. E-EL-H0077; Elabscience Biotechnology) at the Iodine Deficiency Disorders (GAKI) Laboratory, Faculty of Medicine, Diponegoro University, Semarang, Indonesia using the same procedure as for cortisol. The reference interval for serum hepcidin documented in the original study protocol was 13.3–54.4 ng/mL.

Statistical Analysis

The distribution of continuous variables was assessed using the Kolmogorov–Smirnov test. Normally distributed continuous variables were presented as mean±standard deviation (SD), whereas non-normally distributed variables were presented as median and interquartile range. Correlation analysis was performed according to data distribution. Spearman's rank correlation test was used for non-normally distributed variables. A p-value<0.05 was considered statistically significant.

Results

Subject Characteristics

The characteristics of the study participants were presented in Table 1. Serum cortisol and hepcidin levels were normally distributed (both p=0.200), with mean values of 108.84±39.29 and 2.39±1.19 ng/mL, respectively, whereas BMI and WC were not normally distributed (p=0.028 and p=0.015), with medians of 22.14 kg/m² and 76.00 cm, respectively.

Positive Correlation between BMI and Serum Cortisol

The association between BMI and serum cortisol levels was assessed using Spearman's rank correlation test. The analysis demonstrated a significant positive correlation between BMI and serum cortisol levels (r=0.353, p=0.011), indicating a weak-to-moderate positive correlation. The scatterplot illustrating the relationship between BMI and serum cortisol levels was presented in Figure 1.

Positive Correlation between WC and Serum Cortisol

The association between WC and serum cortisol levels was assessed using Spearman's rank correlation test. A significant positive correlation was observed between WC and serum cortisol levels (r=0.386, p=0.005), indicating a weak-to-moderate positive correlation. The scatterplot illustrating this relationship was presented in Figure 2.

No Significant Correlation between BMI and Serum Hepcidin

The association between BMI and serum hepcidin levels was assessed using Spearman's rank correlation test.

Table 1. Subjects characteristics.

Variables	Mean±SD	Median (Min-Max)
Age (years)		19 (18–20)
Body mass index (kg/m^2)		22.14 (15.63–37.49)
Waist circumference (cm)		76.00 (64.00–113.30)
Serum cortisol levels (ng/mL)	108.84±39.295	
Serum hepcidin levels (ng/mL)	2.39±1.19	
Weight (kg)	66.89±14.01	
Height (cm)	169.71±5.71	

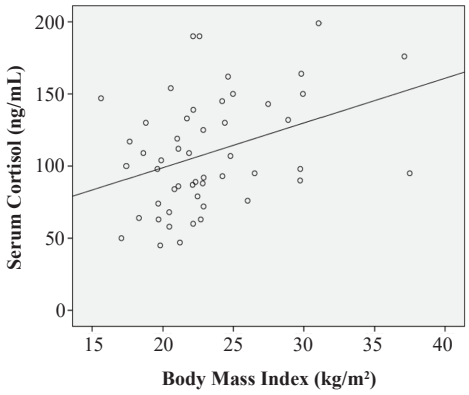


Figure 1. Scatterplot of the correlation between BMI and serum cortisol levels.

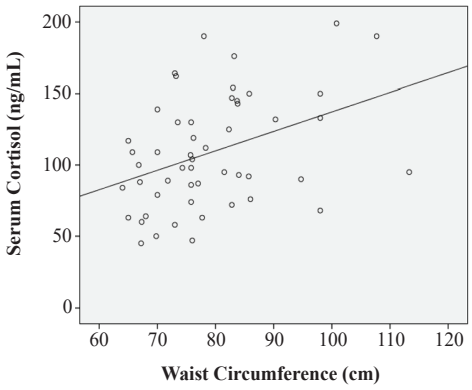


Figure 2. Scatterplot of the correlation between WC and serum cortisol levels.

The analysis showed no significant correlation between BMI and serum hepcidin levels (r=0.175, *p*=0.216). The scatterplot illustrating the relationship between BMI and serum hepcidin levels was presented in Figure 3.

No Significant Correlation between WC and Serum Hepcidin

The association between WC and serum hepcidin levels was assessed using Spearman's rank correlation test. The analysis showed no significant correlation between WC and serum hepcidin levels (r=0.157, *p*=0.266). The scatterplot illustrating the relationship between WC and serum hepcidin levels was presented in Figure 4. These results suggest that serum hepcidin may be influenced by factors beyond adiposity alone.

Discussion

In this study, all subjects were young male medical students aged 18–22 years. This relatively narrow age range also reduced variability related to aging. The median BMI and WC were 22.14 kg/m² and 76.00 cm, respectively, indicating that the study population was generally within normal anthropometric ranges, although the observed ranges included participants with higher BMI and WC. These characteristics provide a relatively homogeneous population for examining the associations between adiposity measures and circulating cortisol and hepcidin levels.

The present study demonstrated significant positive correlations between serum cortisol levels and both BMI and WC. These findings are consistent with previous studies reporting associations between cortisol and adiposity or body fat distribution.(16-20) Cortisol plays an important role in the HPA axis and contributes to energy metabolism, glucose regulation, and stress adaptation. Alterations in cortisol production, clearance, and tissue metabolism may influence fat accumulation and metabolic regulation.(21,22) Several studies have reported that cortisol levels or cortisol-related measures are associated with greater adiposity, particularly in individuals with metabolic syndrome or central obesity.(23,24) However, other studies have reported

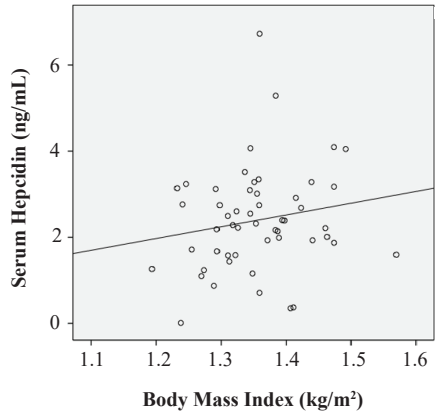


Figure 3. Scatterplot of the correlation between BMI and serum hepcidin levels.

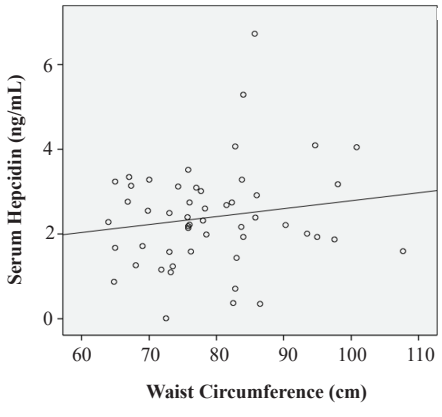


Figure 4. Scatterplot of the correlation between WC and serum hepcidin levels.

reduced or unchanged circulating cortisol concentrations in individuals with obesity.(25,26) These discrepancies may be related to differences in study populations, the type of cortisol measurement used, sampling conditions, and the complex regulation of cortisol metabolism at the tissue level. In particular, circulating serum cortisol may not fully reflect local glucocorticoid activity in adipose tissue. The enzyme 11β-hydroxysteroid dehydrogenase type 1 (11β-HSD1), which converts inactive cortisone to active cortisol, has been proposed as one mechanism linking adipose tissue metabolism with local glucocorticoid exposure.(27) However, 11β-HSD1 expression or activity was not directly assessed in the present study; therefore, this mechanism should be considered as a possible explanation based on previous evidence rather than a finding of this study.

In contrast to cortisol, no significant correlation was observed between serum hepcidin levels and BMI or WC. These findings are consistent with studies suggesting that adiposity alone may not be a major determinant of circulating hepcidin levels.(12,28) Hepcidin is primarily regulated by iron homeostasis, inflammation, and erythropoietic activity, rather than by anthropometric measures alone. (29) Although adipose tissue is associated with a state of low-grade inflammation, the relationship between adiposity and circulating hepcidin may be mediated by several factors, including IL-6, leptin, and iron status.(30,31) Some studies have reported increased hepcidin concentrations in individuals with obesity, particularly in the presence of metabolic syndrome or chronic inflammation.(32) In contrast, other studies have reported minimal or no association between adiposity and circulating hepcidin levels.(28,33) These differences suggest that the relationship between adiposity and hepcidin is complex and may depend on the presence of inflammatory activation and alterations in iron homeostasis.

The absence of a significant association between hepcidin and BMI or WC in the present study may therefore reflect the multifactorial regulation of hepcidin. Although all participants were classified as healthy according to the study eligibility criteria, factors such as iron status, dietary intake, inflammatory activity, and adipose tissue distribution were not comprehensively assessed in the present analysis. Differences in cytokine expression and other biological factors may also contribute to variation in hepcidin concentrations among individuals with similar anthropometric measurements.(34,35)

The contrasting associations observed for cortisol and hepcidin may reflect their different physiological roles. Cortisol is primarily involved in neuroendocrine stress responses and metabolic regulation, whereas hepcidin is a key regulator of systemic iron homeostasis and is strongly influenced by inflammatory and hematologic signals. The significant correlations between cortisol and both BMI and WC suggest an association between circulating cortisol and adiposity measures in this population. In contrast, the absence of significant correlations between hepcidin and the same anthropometric measures suggests that circulating hepcidin may be influenced by factors beyond adiposity alone.

This study included only young male medical students, which limits the generalizability of the findings to women, older adults, and other populations. The restriction to males was intended to reduce potential variability related to menstrual status and iron homeostasis, but it also limits the applicability of the findings to females. Moreover, potential determinants of hepcidin, including iron status and inflammatory markers such as IL-6 and TNF-α, were not measured in the present study. Dietary factors and other potential determinants of cortisol and hepcidin concentrations were also not comprehensively assessed. In addition, although cortisol samples were collected in the morning, the available records did not document fasting status, which may represent another source of variability. Future studies should include larger and more diverse populations, including female participants and different age groups, and should use longitudinal designs to clarify temporal relationships between adiposity and circulating biomarkers. Further investigations should also incorporate iron-related parameters and inflammatory markers, such as serum iron, ferritin, transferrin saturation, IL-6, and tumor necrosis factor-alpha (TNF-α), as well as more detailed assessments of adipose tissue distribution. Assessment of 11β-HSD1 expression or activity may also help clarify the potential mechanisms underlying the observed association between cortisol and adiposity.

Conclusion

In healthy young men, serum cortisol levels showed significant positive correlations with both BMI and WC. In contrast, serum hepcidin levels were not significantly correlated with BMI or WC. These findings indicate that, within this study population, cortisol was associated with measures of overall and central adiposity, whereas circulating hepcidin was not. Further studies incorporating inflammatory and iron-related parameters, as well as longitudinal designs and more diverse populations,

are needed to clarify the mechanisms underlying these associations.

Authors Contribution

MK, NSW, FY, DR, and PA contributed equally to this work. All authors were involved in the conceptualization, methodology, investigation, data collection, data analysis, interpretation of results, and manuscript preparation. All authors reviewed, revised, and approved the final version of the manuscript and agreed to be accountable for all aspects of the work.

Ethical Statement

The study was approved by the Health Research Ethics Committee, Faculty of Medicine, Diponegoro University/ Dr. Kariadi General Hospital, under Ethical Approval No. 475/EC/FK-RSDK/2016. Written informed consent was obtained from all participants before study participation.

Conflict of Interest

The authors have no conflicts of interest to declare.

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