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Obesity-Related Chronic Low-Grade Inflammation and Metabolic Risk Stratification: Evidence from Inflammatory Biomarkers and Adipokines

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Abstract

Obesity is a serious health problem because of the various diseases associated with it, such as insulin resistance, type 2 diabetes, cardiovascular disease and metabolic syndrome. Although BMI is commonly used to screen for obesity, it does not show the distribution of fat or how healthy adipose tissue is in different people. This review examines the relationship between obesity-related chronic low-grade inflammation and metabolic risk from a molecular epidemiological perspective. Current evidence suggests that in obese individuals, adipose tissue expansion, adipocyte hypertrophy, tissue hypoxia, oxidative stress, endoplasmic reticulum stress, and immune cell infiltration collectively promote the formation of chronic low-grade inflammation, further affecting insulin sensitivity and systemic metabolic function. Inflammatory biomarkers such as CRP, IL-6, TNF-α, NLR, and SII, as well as adipokines such as leptin, adiponectin, and resistin, can provide molecular evidence for explaining obesity-related metabolic risk. Overall, chronic low-grade inflammation may be an important mediating mechanism linking obesity and metabolic abnormalities, and related biomarkers have potential applications in metabolic risk stratification, early identification, and precision intervention in populations with obesity.

Keywords

obesity, chronic low-grade inflammation, metabolic risk, inflammatory biomarkers, adipokines

1. Introduction

Obesity has become a prominent public health problem worldwide. The prevalence of overweight and obesity continues to increase worldwide. This problem affects not only adults, but also children and adolescents [1,2]. Obesity is closely related to a variety of metabolic abnormalities, including insulin resistance, type 2 diabetes, dyslipidemia, hypertension, metabolic syndrome and cardiovascular diseases. Based on this situation, the identification of obesity-related metabolic risks has become a research problem that needs to be solved in the fields of epidemiology and public health.

BMI is easy to calculate and is widely used to identify overweight and obese people in the general population. BMI alone cannot explain all the differences in metabolic risk among people with obesity. Some individuals with obesity may not show obvious metabolic abnormalities, whereas others may already have

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diabetes or other metabolic diseases. Therefore, in the future, when assessing the risk of obesity, we need to consider a person's metabolic condition, fat distribution and other types of obesity [3–5].

Chronic low-grade inflammation may be an important mechanism linking obesity with metabolic risk. In obesity, adipose tissue dysfunction, immune cell infiltration, and inflammatory mediator release may contribute to metabolic abnormalities. At the molecular level, inflammatory biomarkers and adipokines, such as CRP, IL-6, TNF-α, leptin, adiponectin and resistin may provide molecular evidence for differences in metabolic risk among individuals with obesity [6–8]. This review focuses on the role of obesity-related chronic low-grade inflammation and related biomarkers in metabolic risk stratification.

2. Theoretical Background

2.1 Obesity and Metabolic Risk

Obesity is generally defined as excessive body fat accumulation that may be harmful to health. BMI is a relatively simple calculation that is frequently used in traditional research and clinical practice to identify overweight and obesity at the population level. However, BMI can only reflect the relationship between body weight and height, and does not reveal fat distribution or individual metabolic conditions. Therefore, BMI alone should not be used as the only indicator of the actual health risks faced by individuals with obesity [3]. Obesity is closely related to a variety of metabolic abnormalities, including insulin resistance, type 2 diabetes, dyslipidemia, hypertension, metabolic syndrome and increased risk of cardiovascular disease. Metabolic risk varies among individuals with obesity. Some individuals with obesity may not have obvious metabolic abnormalities temporarily, while others have already developed multiple metabolic problems. This shows that obesity should not be understood as just weight gain but should be regarded as a complex state related to fat distribution, metabolic function and long-term disease risk [4].

2.2 Chronic Low-Grade Inflammation

Chronic low-grade inflammation is an important theoretical basis for understanding obesity-related metabolic abnormalities. Unlike the obvious inflammation caused by acute infection or trauma, chronic low-grade inflammation is usually a long-term, mild, and persistent inflammatory state with a low level of inflammation, but it may have a long-term impact on metabolic function. In the obese state, changes such as adipose tissue expansion, adipocyte hypertrophy, tissue hypoxia, oxidative stress, and endoplasmic reticulum stress promote inflammatory responses and gradually transform adipose tissue into pro-inflammatory tissue [6,7].

White adipose tissue is not only an energy storage tissue but also has endocrine and immune regulatory functions. In the obese state, dysfunctional adipocytes and infiltrating immune cells can release pro-inflammatory mediators such as TNF-α, IL-6, and MCP-1. These inflammatory factors may interfere with insulin signaling, reduce insulin sensitivity, and further participate in the formation of type 2 diabetes and other metabolic abnormalities. Therefore, chronic low-grade inflammation can be regarded as an important mediating mechanism connecting obesity and metabolic risk [7,8].

2.3 Molecular Epidemiological Perspective

From a molecular epidemiological perspective, obesity can be considered a major exposure factor, chronic low-grade inflammation is an important intermediate mechanism, and metabolic abnormalities are the main health outcome. Within this framework, inflammatory biomarkers and adipokines can serve as molecular evidence to explain why obese individuals experience varying degrees of metabolic risk.

Specifically, inflammatory biomarkers such as CRP, IL-6 and TNF-α can reflect the inflammatory state related to obesity, while adipokines such as leptin, adiponectin and resistin can reflect the changes in endocrine function of adipose tissue. Combining and analyzing such molecular indicators with traditional indicators such as BMI, waist circumference, blood sugar, blood lipids, blood pressure and insulin resistance can more comprehensively clarify the intrinsic relationship between obesity and metabolic abnormalities.

Based on this logic, the theoretical framework of this article can be summarized as follows: obesity can cause adipose tissue expansion and abnormal function, further induce chronic low-grade inflammation, disrupt

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the secretion balance of adipokines, and finally increase the risk of insulin resistance, metabolic syndrome and other metabolic diseases. In this process, inflammatory biomarkers and adipokines are not only important indicators for observing the changes of obesity-related molecules but also provide a basis for metabolic risk stratification and early identification of individuals with obesity.

3. Literature Review

3.1 The Relationship Between Obesity and Chronic Low-Grade Inflammation

Obesity-associated chronic low-grade inflammation is frequently referred to as metabolic inflammation or metaflammation. Unlike acute inflammation, metaflammation is a persistent and low-intensity inflammatory state driven by overnutrition, adipose tissue expansion, and immune-metabolic dysregulation. This concept can help explain why obesity is associated with many other metabolic diseases, such as insulin resistance, type 2 diabetes, cardiovascular disease, dyslipidaemia, hypertension and non-alcoholic fatty liver disease [6,9]. Therefore, obesity-related inflammation should be regarded as a process involving dysfunction of adipose tissue, activation of immune cells and impaired resolution of inflammation [9,10].

White adipose tissue is a common location for the development of obesity-related inflammation. Although traditionally viewed mainly as an energy storage tissue, white adipose tissue also has endocrine and immune-regulatory functions. Adipokines and cytokines are produced to regulate metabolism and inflammation in the body. As people gain weight and accumulate more fat, white adipose tissue changes in function and structure. Dysfunctional adipocytes release pro-inflammatory cytokines and can thus harm not only the local adipose tissue but also the metabolic function of distant organs [7].

Although many mechanisms are involved in this inflammation, they are interconnected and should not be viewed in isolation. Adipocyte hypertrophy and an increase in free fatty acids in obesity may cause tissue hypoxia, oxidative stress, endoplasmic reticulum stress and cell senescence. These stress responses may activate inflammatory signaling pathways such as JNK and NF-κB, promoting the production of inflammatory mediators and interfering with insulin signaling [6,7]. At the same time, impaired resolution of inflammation may help explain why low-grade inflammation persists in obesity rather than being fully resolved [10].

Infiltrating immune cells further amplify inflammation in adipose tissue. Macrophages are among the key immune cells involved in this process. Macrophages in white adipose tissue are relatively numerous in obesity and insulin resistance, and they release more pro-inflammatory factors. This process is associated with adipocyte dysfunction, glucose intolerance and insulin resistance [7]. Besides macrophages, T cells and other immune cells can also be involved in the inflammatory network of adipose tissue, suggesting that obesity-related inflammation is likely the result of interactions between adipocytes and immune cells [11].

Overall, the development of obesity-related chronic low-grade inflammation can be summarized as a continuous action pathway: adipose tissue expansion first causes adipocyte dysfunction and cell stress and then promotes immune cell infiltration, inflammatory mediator release, and causes a decrease in insulin sensitivity. Under the mediation of cytokines such as TNF-α, IL-6, IL-18 and MCP-1, chronic inflammation of adipose tissue constitutes a key relationship between obesity and insulin resistance and type 2 diabetes [7,8]. Therefore, chronic low-grade inflammation provides an important mechanism for explaining the increased metabolic risk of obese individuals [6–8].

3.2 Inflammatory Biomarkers and Metabolic Risk

Inflammatory biomarkers are important molecular evidence for understanding obesity-related chronic low-grade inflammation and metabolic risk. Compared with traditional anthropometric indicators such as BMI and waist circumference, inflammatory biomarkers can reflect the low-grade inflammatory state in obese individuals from the perspective of immune response and molecular regulation. Common obesity-related inflammatory biomarkers include general inflammatory factors, such as C-reactive protein, IL-6 and TNF-α, as well as composite immune-inflammatory indices such as the neutrophil-to-lymphocyte ratio and the systemic immune-inflammation index. These markers may be linked to obesity, chronic inflammation and other metabolic disorders [12].

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High-sensitivity C-reactive protein (hs-CRP) is a commonly used systemic inflammatory marker in obesity research. Adipose tissue dysfunction and the release of pro-inflammatory factors in obesity can stimulate the liver to produce more CRP, thereby reflecting the level of chronic low-grade inflammation in the body. Compared with some cytokines, CRP has relatively good stability and is widely used in clinical practice; therefore, it is often employed to assess the extent of systemic inflammation in obese individuals. However, CRP alone cannot directly explain the source of inflammation, nor can it explain the complete mechanism linking obesity to metabolic disorders. Therefore, in metabolic risk assessment, CRP is more suitable as an indicator of systemic inflammation levels rather than the sole basis for judging metabolic risk [13].

IL-6 and TNF-α are important pro-inflammatory cytokines linking obesity, inflammatory response, and metabolic abnormalities. In an obese state, adipocytes and infiltrating immune cells can release inflammatory factors such as IL-6 and TNF-α, which may participate in the formation of insulin resistance and other metabolic abnormalities by affecting insulin signaling pathways, adipose tissue function, and glucose metabolism. Unlike CRP, which mainly reflects systemic inflammation levels, IL-6 and TNF-α are better able to reflect the mechanistic changes of obesity-related inflammation. Therefore, these two indicators are often used to observe whether the inflammatory state has improved after obesity intervention [14].

In addition to classic inflammatory factors, composite immune-inflammatory indicators derived from routine blood tests have also received increasing attention. The systemic immune-inflammation index, which combines platelet, neutrophil, and lymphocyte information, may reflect systemic inflammatory and immune status. Studies based on NHANES data have reported associations between SII and adult obesity, abdominal obesity, and metabolic syndrome, suggesting its potential value in population-based risk assessment [15–17]. However, indicators such as NLR, PLR, SII, and SIRI are easily influenced by infection, age, lifestyle, chronic disease, and medication use. Therefore, their stability and predictive value in obesity-related metabolic risk assessment still require further validation [18].

Overall, inflammatory biomarkers provide an important molecular basis for assessing obesity-related metabolic risks. CRP can reflect the level of systemic chronic inflammation. IL-6 and TNF-α are more closely related to the mechanism of adipose tissue inflammation and insulin resistance. Composite indicators such as NLR, SII and SIRI are more suitable for immuno-inflammatory assessment in large sample population research, and it is also more convenient to operate. However, a single inflammatory marker is not enough to fully explain the difference in metabolic risk between obese individuals. Follow-up studies can combine multiple inflammatory indicators, and combine the analysis of adipokines, insulin resistance indicators and obesity phenotypes to improve the explanatory effectiveness of metabolic risk stratification in individuals with obesity [12–18].

3.3 Adipokines and Obesity-Related Inflammation

Adipokines are important molecular mediators linking adipose tissue dysfunction, chronic low-grade inflammation, and metabolic risk. Adipose tissue not only stores energy but also functions as an endocrine organ by secreting various adipokines involved in immune, inflammatory, and metabolic regulation. In obesity, adipose tissue expansion and adipocyte dysfunction can alter adipokine secretion patterns, increasing pro-inflammatory adipokines while decreasing protective or anti-inflammatory adipokines. This adipokine imbalance may further aggravate chronic low-grade inflammation and contribute to insulin resistance, abnormal glucose metabolism, and other metabolic abnormalities [19].

Leptin is a type of adipokine associated with inflammation in obesity. Leptin normally helps regulate appetite, energy expenditure and body weight in the body. Leptin levels are generally high in obesity, but leptin resistance can reduce the body's response to leptin. Therefore, high leptin levels may be due to an increase in fat tissue or to immune activation and inflammation [20].

Adiponectin is generally known to be anti-inflammatory and improve insulin sensitivity. It is generally lower in people with obesity, and thus may have a reduced protective effect on glucose and lipid metabolism and inflammation. Low levels of adiponectin are probably due to dysfunctional adipose tissue and increased cardiometabolic risk [21].

Resistin is also linked to obesity, inflammation and insulin resistance, but its actual function is not well known. Some evidence suggests that resistin can lead to insulin resistance and type 2 diabetes through

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inflammation, but this has not been consistently observed in all populations and studies. Therefore, resistin is to be regarded as a complementary adipokine rather than an indispensable one [22].

Overall, adipokines help explain adipose tissue dysfunction and metabolic risk in individuals with obesity. Leptin may reflect increased fat mass and pro-inflammatory activity, adiponectin may indicate reduced anti-inflammatory and insulin-sensitizing protection, and resistin may provide complementary information related to inflammation and insulin resistance [19–22].

4. Discussion & Synthesis

4.1 Key Findings

Based on the existing research results, the metabolic risk related to obesity cannot be fully explained by excessive fat accumulation or a single indicator of BMI. Chronic low-grade inflammation provides an important mechanism for understanding why obese individuals have different degrees of metabolic risk. In obesity, adipose tissue expansion, fat cell dysfunction, cell stress response activation and immune cell infiltration will promote the release of inflammatory mediators and interfere with insulin signal transmission. These pathological processes can eventually induce insulin resistance, type 2 diabetes, metabolic syndrome and other cardiac metabolic abnormalities [6–8].

Inflammatory biomarkers and adipokines provide molecular evidence for this process. Together, these markers suggest that obesity-related metabolic risk is associated not only with body size but also with inflammatory activity, adipose tissue function and immune-metabolic regulation [12–22].

4.2 Controversies and Divergence

Although most studies support an association between obesity, chronic low-grade inflammation, and metabolic risk, findings are not completely consistent across the literature. One important reason is the heterogeneity of study populations. Age, sex, ethnicity, obesity severity, disease status, lifestyle factors, and medication use may all influence inflammatory biomarker levels. Therefore, the relationship between inflammation-related biomarkers and metabolic risk may vary across different populations.

Another cause of the difference is the definition of obesity and metabolic health. Some studies have only used BMI, and others have used waist circumference, body fat percentage, visceral fat or metabolic syndrome criteria. Since BMI cannot distinguish between fat and muscle, different definitions of obesity will lead to various research results. This issue is particularly noticeable in the study of metabolically healthy obesity and metabolically unhealthy obesity; although the levels of inflammatory markers are different, the definitions of MHO and MUO are not always the same [5,23,24].

Inflammatory markers are also easily altered by other reasons. Infection, diet, exercise, smoking, medication use, chronic diseases and lifestyle can all affect biomarker levels. If the above reasons are not corrected, the link between obesity and inflammation will be strengthened or weakened. Thus, although the present evidence shows that inflammatory biomarkers are related to obesity and metabolic syndrome, their stability and clinical applicability have not been confirmed yet [12,18].

4.3 Research Gaps

Many problems have not been solved yet. Many studies have only examined one inflammatory index, such as CRP, IL-6, TNF-α or SII, and none of them can explain the metabolic risks associated with obesity alone. Chronic low-grade inflammation, adipokine dysregulation, abnormal fat distribution and insulin resistance are all associated with the development of metabolic disorders. Therefore, only one type of inflammatory marker cannot fully explain why there are various levels of metabolic risk among individuals with obesity.

Second, many studies have not shown a clear causal relationship. Cross-sectional studies have shown that inflammatory markers are correlated with obesity and metabolic syndrome, but it is not known whether excessive inflammation in these cases is a result of obesity or an early manifestation of other metabolic disorders. Therefore, it is still unknown whether inflammatory markers can be used to predict metabolic deterioration or cardiometabolic risk in individuals with obesity.

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Thirdly, inflammatory markers have not been widely used to stratify obesity phenotypes. Although the difference between MHO and MUO indicates different metabolic risks for individuals with obesity, it has not been determined yet which inflammatory markers or adipokines are most suitable for distinguishing between low-risk and high-risk individuals with obesity. Different studies have not reached a complete consensus on the definitions of MHO and MUO, and thus their use in the stratification of risk has not been standardized [5,23].

4.4 Future Directions

In the future, many inflammatory markers and adipokines should be examined at the same time, not just one. CRP, IL-6, TNF-α, NLR, SII, leptin, adiponectin and resistin are all biomarkers of inflammation and metabolic risk in individuals with obesity, so they can be used together to provide a more comprehensive picture.

Longer follow-up periods are also needed. Longitudinal studies can determine whether inflammatory markers predict the future development of insulin resistance, type 2 diabetes, metabolic syndrome and cardiovascular disease in obese individuals and also find out if MHO will change to MUO with rising levels of inflammation. Therefore, this can help clarify whether inflammatory markers have practical value in early risk identification.

Comparison of different groups of people is also needed. Future studies should compare the various inflammatory indicators in different age groups, sexes, ethnic groups, fat distributions and obesity phenotypes. Only after verifying that the above indicators are stable in all populations will researchers know whether they can be used to stratify obesity-related metabolic risks. In the future, building on traditional anthropometric indices, metabolic factors, inflammation indicators and adipokines may help identify high-risk individuals with obesity earlier and more precisely.

5. Conclusion

This review has examined obesity-related chronic low-grade inflammation and metabolic risk. Based on current evidence, obesity is not only the condition of excessive fat accumulation and a high BMI, but also a serious metabolic disorder that includes adipose tissue dysfunction, immune system activation and the release of inflammatory factors. Chronic low-grade inflammation may therefore be one of the reasons that obesity leads to insulin resistance, type 2 diabetes, metabolic syndrome and other cardiometabolic diseases.

Inflammatory markers and adipokines are also associated with increased risk in obesity. CRP, IL-6, TNF-α, NLR, SII, leptin, adiponectin and resistin are all related to metabolic disorders, and some show variations in people with obesity. However, their predictive value and clinical application still require further validation through longitudinal studies. In summary, many inflammatory factors and adipokines are associated with obesity, and thus may be used to predict metabolic risk in individuals with obesity.

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Conflicts of Interest

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