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Inflammation, metainflammation, and immunometabolic disorders - Evidence review

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1. ABSTRACT

The analyzed publication examines the reciprocal dialogue between metabolism and the immune system, defined as immunometabolism. The available text is only an abstract and does not report a single clinical or animal experiment, but proposes a conceptual synthesis: the balance between energy use, nutrient availability, and immune response is important for health and can be altered in obesity, diabetes, and other chronic diseases.In section 6.1 of “The Choice to Live in Health or Illness” by Dr. A. Colonnese and R. Panzironi, the publication contributes to the narrative that, in obesity and type 2 diabetes, adipose tissue can harbor macrophages with more pro-inflammatory characteristics, promoting chronic low-grade inflammation and metabolic dysfunction. Independent studies confirm the increase and functional change of macrophages in the fat of obese animals and show compatible signals in humans as well. However, human studies do not uniformly demonstrate that the number of macrophages is an independent cause of insulin resistance.

ONLINE ARTICLE

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MISSION

LIFE Science Hub – Journal of Science was founded with the aim of making scientific knowledge accessible without giving up methodological rigour, quality of sources and authoritativeness of content. The journal promotes the dissemination of knowledge in the health, nutrition, medical and longevity sciences, building a bridge between the world of research and society.

Through articles, reviews, in-depth analyses and expert contributions, the Journal champions evidence-based medicine and science, fostering a correct interpretation of scientific data and countering misinformation. Every piece of content is developed with the aim of translating the complexity of research into understandable, useful and applicable information, while keeping intact the principles of accuracy, transparency and scientific independence.

Free from political, industrial and commercial influence, the journal stands as an authoritative and independent editorial space, dedicated to promoting scientific culture, professional development and the growth of health awareness among the public.

2. ANALYZED STUDY

The central question is whether metabolism and immunity should be considered separate systems or components of the same biological network. The hypothesis illustrated is that the control of energy and nutrients influences immune defenses and, conversely, that immune activation may contribute to chronic metabolic disorders.Within the scope of the book's section, the relevance primarily concerns the concept of metainflammation: a persistent, low-grade inflammation associated with an excess of nutrients and metabolic dysfunction. The publication subject to the Evidence Review is therefore used as a scientific framework to link obesity, adipose tissue alterations, immune response, and insulin resistance.Since the available material contains only the abstract, it is not possible to attribute specific experimental data on macrophages, adipose tissue, type 2 diabetes, or insulin resistance to the study. The available text can directly support the existence of a close conceptual relationship between metabolism and immunity; it cannot, on its own, prove that pro-inflammatory macrophages cause metabolic dysfunction in humans.

3. MATERIALS AND METHODS OF THE ORIGINAL STUDY

The available material is only the abstract of the publication. The abstract does not report a participant population, sample size, inclusion or exclusion criteria, observation period, comparison groups, interventions, laboratory measurements, endpoints, or statistical analyses.The available text has a conceptual and descriptive structure: it presents metabolism as the system that regulates energy, nutrients, and the synthesis or degradation of molecules, and the immune system as the system that distinguishes what belongs to the body from what can harm it. It then discusses the relevance of their interactions for obesity, type 2 diabetes, and other chronic non-communicable diseases.As the full text is not available, further methodological details are not reported in the available material.

4. RESULTS

The abstract does not present quantitative results, P-values, confidence intervals, risk measures, or comparisons between groups. It also fails to report experimental data that would allow for an estimation of how frequently adipose tissue macrophages adopt a pro-inflammatory profile in obesity or type 2 diabetes.The main scientific content reported is that the proper balance between metabolism and immune response is important for cellular and organismal survival. The abstract further states that there are evolutionarily conserved interactions between these two systems and that this balance has implications for obesity, diabetes, and other chronic diseases.In plain terms, the message is that immune cells do not defend the body independently of nutritional conditions, and that tissues involved in metabolism are not biologically detached from inflammation. However, from the available material, it is not possible to derive a quantitative demonstration of the specific passage regarding adipose macrophages.

5. INTERPRETATION

The publication's contribution to the book's narrative is primarily biological and conceptual. Adipose tissue is not merely an energy store; it is an active tissue that communicates with immune cells, hormones, and other organs. When this communication changes persistently, a local inflammatory environment can emerge, capable of interfering with the normal response to insulin.The segment of the chain effectively supported by the available material can be expressed as follows: metabolic imbalance alteration of the interactions between metabolism and immunity greater relevance of inflammation in chronic diseases. The next step, namely the increase in adipose macrophages with pro-inflammatory characteristics and their possible contribution to insulin resistance, requires specific data from independent studies.It is important to distinguish between levels of evidence. The association between obesity, adipose tissue inflammation, and metabolic alterations is biologically plausible and widely studied. Demonstrating that a single population of macrophages is the autonomous cause of human metabolic dysfunction is more difficult, because adipocyte size, fat distribution, sex, diet, and other factors can simultaneously influence both inflammation and insulin sensitivity.

6. INDEPENDENT SUPPORTING EVIDENCE

Initial confirmation concerns the increase of macrophages in adipose tissue during obesity. Weisberg and colleagues studied different murine models of obesity and human adipose tissue samples. In animal models, increased adiposity was associated with an increase in macrophages within the adipose tissue. The authors also found that these macrophages expressed a large amount of tumor necrosis factor-alpha, or TNF-alpha, a cytokine that promotes inflammation, and significant quantities of other inflammatory signals. This finding directly confirms the sequence "obesity greater presence of macrophages in fat inflammatory environment," but it does not prove that this sequence alone causes type 2 diabetes in humans [1].A second study investigated the functional change of macrophages in the fat of obese animals. In mice fed a high-fat diet, Lumeng and colleagues observed a population of macrophages absent in lean mice and characterized by the CD11c marker. In obese mice, signals associated with an M2 profile—conventionally linked to tissue repair and inflammation regulation—decreased, while TNF-alpha and iNOS, characteristics associated with a pro-inflammatory M1 profile, increased. In cultured adipose cells, interleukin-10 attenuated the negative effects of TNF-alpha on the insulin response. This experiment confirms the sequence "experimental obesity prevalence of pro-inflammatory macrophage signals possible interference with insulin signaling," but it concerns mice and cells, not patients [2].Taken together, these independent studies make the proposed narrative consistent: the increase in adipose tissue can be accompanied by a change in its immune population and a greater local production of inflammatory signals. The confirmation is particularly solid at the cellular and animal level; it is more cautious at the clinical level, where the observed relationships may also depend on the amount and location of body fat.

7. CONFLICTING EVIDENCE

A study conducted on 97 adult volunteers has called for a more cautious interpretation of human data. Jia and colleagues measured total macrophages and subpopulations with CD14 and CD206 markers in abdominal subcutaneous fat, along with adipocyte size, body composition, insulin resistance indices, and inflammatory markers. Some simple correlations were present, but after analyses that accounted for adipocyte size and body composition, the number of macrophages did not consistently predict insulin resistance, systemic inflammation, or alterations in triglycerides [3].This result does not deny that macrophages or inflammatory signals may increase in adipose tissue during obesity. Instead, it shows that, in humans, simply counting macrophages in a single fat depot is not necessarily enough to explain metabolic risk. Macrophages themselves can also perform tissue remodeling functions, and the rigid classification into M1 and M2 does not capture their full biological diversity.The conflicting evidence is therefore relevant to the final link in the chain, namely "adipose macrophages metabolic dysfunction." No directly conflicting evidence sufficiently relevant to the increase in macrophages and inflammatory signals in adipose tissue during obesity has been identified; this does not prove the absence of contrary findings.

8. QUADRO COMPARATIVO E SINTESI DELLE EVIDENZE

The table distinguishes the conceptual framework provided by the publication subject to the Evidence Review from independent research that confirms or defines the book's narrative progression.

Quadro comparativo

Reference	Study type	Population or model	Main result	Direction of evidence	Strength of evidence
Original study	Conceptual synthesis; text limited to abstract	Not applicable	Describes the interdependence between metabolism, immunity, and chronic diseases.	biologically consistent evidence	moderate
[1]	Animal experimental with human adipose tissue data	Murine models of obesity and human adipose tissue samples	Obesity is associated with increased macrophage accumulation in adipose tissue and inflammatory signals.	direct confirmation	moderate
[2]	Animal and cellular	Diet-induced obese mice and 3T3-L1 adipocytes	High-fat diet increases pro-inflammatory macrophage markers and reduces M2 markers in fat.	direct confirmation	moderate
[3]	Cross-sectional observational	97 adult volunteers, abdominal subcutaneous adipose tissue	After controlling for adipocyte size and body composition, macrophages did not consistently predict insulin resistance.	discordant evidence	moderate

9. SINTESI EDITORIALE E CONCLUSIONI

CONCLUSIONS

The publication subject to the Evidence Review appropriately supports the general framework that metabolism and the immune system are deeply interconnected. Independent evidence confirms that obesity can increase macrophages in adipose tissue and promote pro-inflammatory signals, especially in animal models.The link connecting this inflammation to metabolic dysfunction is supported at a mechanistic level and is biologically consistent, but it should not be interpreted as a uniform demonstration of causality in humans. The relationship likely depends on the location of the adipose tissue, the size of the adipocytes, body composition, and the functional diversity of immune cells.

Aggiornato al 24/11/2026. Questo contenuto riflette una sintesi divulgativa delle evidenze scientifiche al momento della pubblicazione e non sostituisce il parere medico.

10. TRANSPARENCY AND SCIENTIFIC RESPONSIBILITY

Artificial Intelligence was used solely as a tool for editorial and documentary support. All content, interpretations, bibliographic citations and conclusions have been verified, reviewed and approved by the author, who assumes full scientific responsibility for them.

The manuscript was also subjected to independent peer review prior to publication.

To ensure the greatest transparency, the complete history of the article, including the dates of submission, revision, acceptance and publication, as well as information on the peer

review process, is available on the dedicated Article History page.

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