LIFE Science Hub
JOURNAL OF SCIENCE
EXPANDING KNOWLEDGE ABOUT NUTRITION AND HEALTH

ORIGINAL RESEARCH
ISSN 2845-7501

# Insulin and fatty acid desaturases in human monocytes

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## 1. ABSTRACT

The analyzed study evaluated whether insulin can modify the expression of genes involved in fatty acid metabolism in THP-1 monocytes, a human cell line used as an experimental model. In the available material, which consists only of the abstract, the authors report that insulin increased the expression of SCD, FADS1, and FADS2 in a time- and dose-dependent manner. Quantitative values, insulin concentrations, and exposure times are not reported in the available material.The publication contributes to the narrative of "The Choice to Live in Health or Illness" by Dr. A. Colonnese and R. Panzironi, according to which insulin can activate a lipid remodeling program associated with SREBP-1c in immune cells. Independent evidence confirms that insulin can increase SREBP-1c transcription in human cells and that, in monocytes during differentiation, SREBP-1c, FADS1, and FADS2 increase in the same direction. However, a study on primary monocytes did not detect variations in metabolic genes after 24 hours of insulin exposure. The link is therefore supported primarily at a mechanistic and cellular level, rather than as a demonstration in humans of an increase in arachidonic acid or inflammation.

ONLINE ARTICLE

ARTICLE INFORMATION

**Published:** November 23, 2026
**Updated:** November 23, 2026

**DOI** 10.5281/zenodo.24451721

MISSION

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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 research question was whether insulin can act directly on monocytes, cells of the innate immune system, by increasing the expression of enzymes that modify fatty acids. The hypothesis was that insulin signaling induces SCD, FADS1, and FADS2, as well as SREBP-1c, a transcription factor—that is, a protein that regulates the activation of other genes.The relevance to the book's section concerns a specific point: insulin could increase the activity of genes involved in the production and modification of fatty acids within immune cells. Specifically, FADS2 encodes delta-6-desaturase and FADS1 encodes delta-5-desaturase, two enzymes that participate in the transformation of omega-6 precursors along the pathway that includes DGLA and arachidonic acid.The study can document a gene expression response in a cell model exposed to insulin. However, it cannot establish that the same effect occurs in the circulating monocytes of people with hyperinsulinemia, that it actually increases the cellular amount of arachidonic acid, or that it triggers a clinically relevant inflammatory response.

## 3. MATERIALS AND METHODS OF THE ORIGINAL STUDY

The available material consists only of the abstract. The study used THP-1 monocytes, a human cell line cultured in the laboratory, and evaluated the response to insulin exposure. The molecular targets indicated were SCD, FADS1, FADS2, and SREBP-1c.The authors report that the response was evaluated as a function of time and insulin dose. The number of experiments, the concentrations used, the duration of the incubations, the culture conditions, the control group, the laboratory technique employed, the statistical variables, the significance values, and the analysis criteria are not reported in the available material.Since this involves a cell line rather than monocytes freshly isolated from the blood of participants, the model allows for the study of a possible biological mechanism under controlled conditions, but it does not fully reproduce the complexity of the human organism.

## 4. RESULTS

The main result reported in the abstract is that insulin induced the expression of SCD, FADS1, and FADS2 in THP-1 monocytes. The increase is described as time- and dose-dependent: in other words, the response varied based on both the duration of exposure and the amount of insulin used.For the reader, this means that insulin did not act solely as a hormone involved in glucose regulation, but also modified the activity of genes that help determine which fatty acids are available within the cell. SCD acts primarily on the formation of monounsaturated fatty acids; FADS2 and FADS1, on the other hand, participate in the desaturation of polyunsaturated fatty acids.The abstract does not report absolute values, percentage variations, confidence intervals, P-values, or doses. Although SREBP-1c is included among the genes evaluated, the available material does not provide a separate quantitative result on its expression. Therefore, it is not possible to state, based on the abstract alone, either the extent of any SREBP-1c induction or that SREBP-1c is the proven cause of the increase in FADS1 and FADS2.

## 5. INTERPRETATION

In simple terms, the publication indicates that monocytes can perceive insulin and modify their lipid program. This is consistent with the idea that, following an insulin signal, an immune cell can increase its capacity to process fatty acids.The segment of the narrative chain most directly supported is: insulin increased expression of FADS1 and FADS2 in THP-1 monocytes. The link with SREBP-1c is biologically plausible because this transcription factor regulates several genes involved in lipogenesis, i.e., the synthesis and modification of lipids. However, the available material does not demonstrate that insulin increased SREBP-1c with quantitative data, nor that SREBP-1c causally mediated the effect on the two desaturases.The conversion of DGLA into arachidonic acid requires the activity of delta-5-desaturase, encoded by FADS1. An increase in gene expression may increase the cell's potential capacity to perform this transformation, but it is not equivalent to proof that the production of arachidonic acid has increased. Indeed, direct measurements of cellular lipids, eicosanoids, or inflammatory markers are missing

from the available material.

## 6. INDEPENDENT CONFIRMING EVIDENCE

A first piece of the narrative is the effect of insulin on SREBP-1c. Dif and colleagues studied the human SREBP-1c promoter, which is the DNA region that controls gene initiation, in HEK-293 cells and human muscle cells. Insulin increased promoter activity by approximately 1.8-fold in the main construct; the mutation of specific regulatory sequences abolished the stimulatory effect. The authors also observed that insulin increased the binding of SREBP-1 to the promoter. This confirms, in human cells but not in monocytes, that insulin can stimulate the SREBP-1c transcriptional program [1].A second piece concerns the link between the SREBP-1c program and desaturases in monocytes. Ecker and colleagues analyzed primary human monocytes from healthy volunteers during differentiation induced by M-CSF, a signal different from insulin that promotes transformation into macrophages. After four days, compared to the initial monocytes, SREBP-1c increased by an average of 8.38-fold, FADS1 by 6.29-fold, and FADS2 by 12.93-fold. The study does not prove that insulin is responsible for these increases, but it shows a consistent co-regulation between SREBP-1c and the two desaturases in primary human immune cells [2].Taken together, these independent results reconstruct a convergent but incomplete chain: insulin can increase the transcriptional activity of SREBP-1c in human cells [1], while an SREBP-1c-associated program in primary monocytes is accompanied by an increase in FADS1 and FADS2 [2]. The essential limitation is that the studies use different models, signals, and cell types. Neither study alone demonstrates that insulin increases the enzymatic activity of desaturases in the monocytes of people with hyperinsulinemia through SREBP-1c.

## 7. CONFLICTING EVIDENCE

Directly relevant evidence suggests that the results from the THP-1 line should not be automatically extended to primary monocytes. Ratter and colleagues exposed primary human monocytes to insulin under acute conditions and for 24 hours. Acute exposure activated the PI3K/AKT/mTOR pathway, a sequence of intracellular signals that links insulin to many metabolic responses. However, after 24 hours of chronic exposure, the authors did not detect any changes in the expression of insulin-regulated metabolic genes in non-immune cells [3].This result does not directly refute the increase in FADS1 and FADS2 observed in the analyzed study, because the genes evaluated, the doses, the experimental timelines, and the cell model do not necessarily coincide. However, it shows that the transcriptional response of monocytes to insulin depends on the experimental context and that a prolonged insulin signal does not inevitably lead to an increase in metabolic genes. No directly conflicting evidence sufficiently relevant to the expression of FADS1 and FADS2 in human monocytes exposed to insulin has been identified; this does not prove the absence of contrary results.

## 8. QUADRO COMPARATIVO E SINTESI DELLE EVIDENZE

The table distinguishes the result of the publication subject to the Evidence Review from independent verifications of the individual steps of the narrative.

### Quadro comparativo

| Reference | Study type | Population or model | Main result | Direction of evidence | Strength of evidence |
|---|---|---|---|---|---|
| Original study | Cellular | THP-1 monocytes | Insulin increases the expression of SCD, FADS1, and FADS2 in a time- and dose-dependent manner; quantitative data not reported in the available material. | direct confirmation | preliminary |
| [1] | Cellular and human experimental | HEK-293 cells, human muscle cells, and samples during hyperinsulinemic clamp | Insulin increases SREBP-1c promoter activity and SREBP-1 binding to the promoter. | indirect confirmation | moderate |
| [2] | Cellular on primary human cells | Monocytes from healthy volunteers differentiated with M-CSF | During differentiation, SREBP-1c, FADS1, and FADS2 increase in the same direction. | biologically consistent evidence | moderate |
| [3] | Cellular on primary human cells | Primary human monocytes exposed acutely or for 24 hours to insulin | Acute insulin activates PI3K/AKT/mTOR, but 24-hour exposure does not modify insulin-regulated metabolic genes in non-immune cells. | discordant evidence | moderate |

## 9. SINTESI EDITORIALE E CONCLUSIONI

### CONCLUSIONS

The study analyzed directly supports the claim that insulin can increase the expression of FADS1 and FADS2 in THP-1 monocytes. This result is consistent with the book's narrative regarding the lipid remodeling of immune cells in response to insulin signaling.Independent evidence separately confirms that insulin can activate SREBP-1c in human cells and that SREBP-1c, FADS1, and FADS2 can increase together in primary monocytes during differentiation. The overall link is therefore supported at a mechanistic level. However, it has not been proven that hyperinsulinemia increases arachidonic acid or inflammatory mediators in humans through this specific sequence in monocytes.

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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