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Gene regulation of mammalian desaturases - Review of the evidence

Dott. A. Conte[1]

1 Nutritional Biologist, Italy

1. ABSTRACT

The analyzed study examined the regulation of desaturases, enzymes that modify fatty acids and contribute to the composition of cellular lipids. It is a mechanistic review publication, of which only the abstract is available: it describes how insulin, highly unsaturated fatty acids, and SREBP-1c participate in the control of the expression of multiple desaturases. However, in the abstract, SREBP-1c is explicitly indicated as a mediator of the regulation of SCD and delta-6-desaturase, while the control of delta-5-desaturase is not directly demonstrated.In section 5.3.2 of "The Choice to Live in Health or Illness" by Dr. A. Colonnese and R. Panzironi, the publication contributes to the narrative that insulin and SREBP-1c coordinate desaturases in immune cells. Independent studies in a human monocytic cell line have observed an increase in the expression of FADS1, the gene for delta-5-desaturase, following exposure to insulin and activation of SREBP-1. However, the evidence remains predominantly cellular and dependent on the biological context; it does not demonstrate that chronic hyperinsulinemia in humans automatically leads to an increase in arachidonic acid or inflammation.

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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 question addressed was how desaturase expression is regulated in mammals. Desaturases are enzymes that insert specific bonds into fatty acids; in this way, they help define which fats will be available in cell membranes and energy stores. The general hypothesis was that enzymes with different functions share certain regulatory signals, including insulin and SREBP-1c (a transcription factor that increases the activity of genes involved in lipid synthesis).The relevance to section 5.3.2 of "The Choice to Live in Health or Illness" by Dr. A. Colonnese and R. Panzironi therefore concerns the common regulatory framework: insulin can promote desaturase expression, and SREBP-1c can act as a transcriptional mediator. However, the study's specific contribution to the narrative is not the direct demonstration, in monocytes, of the entire insulin SREBP-1c delta-5-desaturase arachidonic acid sequence. The available abstract does not, in fact, report direct experiments on delta-5-desaturase in monocytes nor clinical data on hyperinsulinemia.

3. MATERIALS AND METHODS OF THE ORIGINAL STUDY

The available material is only an abstract; the full text is not accessible. The publication appears to be a review article on the gene regulation mechanisms of desaturases in mammals, rather than a single clinical or cellular experiment with a new cohort described in the abstract.The available material does not report sample size, population, inclusion or exclusion criteria, enrollment period, insulin dosages, control groups, quantitative endpoints, statistical analyses, probability values, or follow-up. The abstract distinguishes stearoyl-CoA desaturase, primarily associated with the production of oleic acid and triglycerides, from delta-6 and delta-5-desaturases, which are involved in the formation of highly unsaturated fatty acids destined primarily for membrane phospholipids.

4. RESULTS

The key finding reported is that stearoyl-CoA desaturase, delta-6-desaturase, and delta-5-desaturase share certain regulatory characteristics: their expression is insulin-dependent, suppressed by highly unsaturated fatty acids, and can be induced by peroxisome proliferators. Peroxisomes are cellular structures involved, among other things, in the degradation of certain lipids.The abstract attributes a key role to SREBP-1c in the transcriptional activation of stearoyl-CoA desaturase and delta-6-desaturase by insulin, as well as in their inhibition by highly unsaturated fatty acids. As for delta-5-desaturase, the abstract describes it as an essential enzyme for the synthesis of arachidonic acid and other highly unsaturated fatty acids, but it does not provide a direct experimental result demonstrating the activation of its gene by SREBP-1c.In practical terms, the study supports the biological context in which insulin and SREBP-1c can coordinate multiple enzymes of lipid synthesis. However, it does not allow for quantifying the increase in delta-5-desaturase, establishing its magnitude in human monocytes, or inferring an increase in eicosanoids, molecules derived from arachidonic acid that can participate in inflammation processes.

5. INTERPRETATION

To understand this passage from the book, it is necessary to distinguish the role of each element in the chain. Insulin is a signal that informs cells about nutrient availability. SREBP-1c translates part of this signal into the activation of genes that change the production and composition of lipids. FADS1, the delta-5-desaturase gene, encodes an enzyme that converts DGLA (dihomo-gamma-linolenic acid) into arachidonic acid.The publication subject to the Evidence Review primarily documents the general insulin/SREBP-1c segment coordinated regulation of desaturases. Its formulation is consistent with the hypothesis that delta-5-desaturase also participates in this program, but the specific SREBP-1c FADS1 link is not directly demonstrated in the available material. The most prudent interpretation is therefore that the study provides the biological foundation for the narrative, while confirmation of the specific step requires targeted independent research.Furthermore, even when FADS1 expression increases in cultured cells, this indicates a change in potential genetic activity. It does not automatically prove that enzyme activity, arachidonic acid production, eicosanoid synthesis, or inflammation increase to the same extent in the human body.

6. INDEPENDENT CONFIRMING EVIDENCE

Particularly relevant confirmation comes from a study on THP-1 cells, a human cell line with monocytic characteristics. Exposure to insulin increased the expression of SCD, FADS1, and FADS2, along with SREBP-1c, in a time- and dose-dependent manner. FADS1 is the gene that encodes delta-5-desaturase. This result directly confirms the passage in the paragraph stating that, in cells of the monocyte compartment, insulin can be associated with an increase in delta-5-desaturase expression in parallel with SREBP-1c [1]. The limitation is significant: this involves a laboratory-cultured cell line, not primary monocytes exposed to chronic hyperinsulinemia in the body.A second study, also using the THP-1 line, observed that simvastatin early on increased delta-5-desaturase mRNA through SREBP-1 and not through PPAR-alpha (a nuclear receptor that regulates numerous genes of lipid metabolism). This research does not evaluate insulin, but it reinforces an intermediate node of the proposed chain: the activation of SREBP-1 can increase FADS1 expression in monocytic cells [2]. Since the experimental stimulus is a drug and not insulin, it confirms the SREBP-1 FADS1 mechanism, not the entire insulin sequence.Taken together, these two independent studies make the idea that an insulin signal can be accompanied by an increase in SREBP-1c and FADS1 in the monocytic model biologically consistent. However, neither demonstrates that the increased genetic expression necessarily produces more arachidonic acid or a greater inflammatory response in people with hyperinsulinemia.

7. CONFLICTING EVIDENCE

Important contextual evidence shows that the insulin–SREBP-1c axis response is not uniform across tissues. In a study using a hyperinsulinemic-euglycemic clamp—a procedure that maintains stable glycemia while increasing insulin—an acute rise in insulin increased SREBP-1 mRNA in both rat liver and rat and human adipose tissue. However, the post-translational activation of SREBP-1c, i.e., its transformation into the form capable of acting in the nucleus, increased in rat liver but decreased in adipose tissue [3].This evidence does not directly contradict the result in THP-1 cells, because it concerns different tissues and does not measure FADS1 in monocytes. However, it shows that an increase in SREBP-1 mRNA does not always equate to an increase in its final activity and that the response to insulin depends on the cell type. No sufficiently relevant directly conflicting evidence has been identified that demonstrates the absence of insulin-induced FADS1 in THP-1 cells; this does not prove the absence of contrary results.

8. QUADRO COMPARATIVO E SINTESI DELLE EVIDENZE

The table distinguishes the contribution of the publication under review from the independent research used to confirm or define the book's narrative passage.

Quadro comparativo

Reference	Study type	Population or model	Main result	Direction of evidence	Strength of evidence
Original study	Mechanistic summary	Literature on mammalian desaturases; details not reported in the available abstract	Describes shared regulation of desaturases by insulin and the role of SREBP-1c for stearoyl-CoA and delta-6-desaturase	biologically consistent evidence	preliminary
[1]	Cellular	THP-1 cells, human monocytic line	Insulin increases the expression of SREBP-1c, FADS1, and FADS2 in a time- and dose-dependent manner	direct confirmation	moderate
[2]	Cellular	THP-1 cells	The early increase in delta-5-desaturase mRNA induced by simvastatin depends on SREBP-1, not PPAR-alpha	indirect confirmation	moderate
[3]	Physiological intervention in humans and animals	Human and rat adipose tissue; rat liver	Insulin increases SREBP-1 mRNA, but SREBP-1c activation varies by tissue	discordant evidence	moderate

9. SINTESI EDITORIALE E CONCLUSIONI

CONCLUSIONS

The analyzed study supports a general biological principle: insulin and SREBP-1c participate in the regulation of desaturases that shape cellular fatty acid composition. Regarding the specific passage on delta-5-desaturase in monocytes, independent evidence is consistent and shows an increase in FADS1 in a human monocytic cell line exposed to insulin or conditions that activate SREBP-1.The narrative is therefore supported at a mechanistic level. It remains necessary to distinguish the increased expression of a gene in cell culture from effects demonstrated in the organism: it is not yet possible to conclude that hyperinsulinemia directly and generally causes increased production of arachidonic acid or clinically relevant inflammation.

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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