

**LIPID ALMASHINUVI VA UNING TURLARI**

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Annotatsiya: *Ushbu maqola lipid almashinuvi jarayonlari, asosiy turlari va ularning biologik ahamiyatini tahlil qiladi. Lipidlar sintezi (lipogenez), katabolizmi (lipoliz, β -oksidlanish), transport yo'llari va lipoprotein turlarini yoritadi. Bundan tashqari, lipid dysregulyatsiyasi va kasalliklar bilan bog'liq muammolar ham tahlil etiladi.*

Kalit so'zlar: *Lipid almashinuvi, Lipogenez, Lipoliz, β -oksidlanish, Lipoproteinlar, Lipid dysregulyatsiyasi*

Аннотация: *В данной статье анализируются процессы липидного обмена, их основные типы и биологическое значение. Рассматриваются синтез липидов (липогенез), катаболизм (липолиз, β -окисление), пути транспорта и типы липопротеинов. Кроме того, анализируются проблемы, связанные с дисрегуляцией липидного обмена и развитием заболеваний.*

Ключевые слова: *липидный обмен, липогенез, липолиз, β -окисление, липопротеины, дисрегуляция липидов*

Annotation: *This article analyzes the processes of lipid metabolism, its main types, and biological significance. It highlights lipid synthesis (lipogenesis), catabolism (lipolysis, β -oxidation), transport pathways, and lipoprotein types. In addition, the issues related to lipid dysregulation and associated diseases are discussed.*

Keywords: *lipid metabolism, lipogenesis, lipolysis, β -oxidation, lipoproteins, lipid dysregulation*

Kirish



Lipidlar — hujayra membranalari, energetik zaxira va signal molekulalari sifatida turli funktsiyalarni bajaruvchi muhim biomolekulalardir. Ularning almashinuvi — sintez, parchalanish, transport va saqlash kabi murakkab birikmalarni o'z ichiga oladi. Ushbu jarayon organizmdagi energetik muvozanat va hujayra funktsiyasi uchun muhimdir

Tahlil va muhokama

Lipidlarning biologik ahamiyati va metabolik funktsiyalari

Lipid almashinuvi organizmdagi asosiy energetik jarayonlardan biridir. Lipidlar nafaqat energiya manbai, balki membrana tuzilishi, signal molekulalari, gormonlar va vitaminlar sintezi uchun ham zarurdir. Yog' kislotalari, triatsilglycerollar, fosfolipidlar, sterollar va sfingolipidlarning metabolizmi turli yo'llar orqali amalga oshadi va hujayra homeostazini ta'minlaydi [1].

Lipogenez: Yog' kislotalari biosintezi

Lipogenez ortiqcha uglevodlardan yog' kislotalar sintezi jarayonidir. Bu jarayon asosan jigar va yog' to'qimalarida sodir bo'ladi. Asosiy fermentlar — **Acetyl-CoA carboxylase (ACC)** va **Fatty acid synthase (FAS)** hisoblanadi [2]. Malonil-CoA hosil bo'lgach, palmitat (16:0) asosiy mahsulot sifatida sintez qilinadi. Keyinchalik elongatsiya va desaturatsiya orqali boshqa yog' kislotalar hosil bo'ladi. Lipogenez insulinga bog'liq ravishda faollashadi va ortiqcha glyukoza energiya zaxirasiga aylanadi [3].

Lipoliz va β -oksidlanish

Lipoliz triatsilglycerollarning yog' kislotalari va glitserolga parchalanishi jarayonidir. Ushbu jarayon gormon sezgir lipaza (HSL) va adipotsit triglitserid lipaza (ATGL) yordamida amalga oshadi [4]. Hosil bo'lgan yog' kislotalari β -oksidlanish orqali mitoxondriyada energiyaga aylanadi. β -oksidlanish jarayoni **faollashuv, transport (karnitin shuttli) va oksidlanish sikli** bosqichlaridan iborat [5].

Jadval 1. β -oksidlanish bosqichlari



Bosqich	Jarayon	Asosiy fermentlar	Natija
Faollashuv	Yogʻ kislotasi + CoA → Acyl-CoA	Acyl-CoA synthetase	Acyl-CoA
Transport	Acyl-CoA mitoxondriya	→ Karnitin transferaza I/II	Acyl-CoA palmitoil mitoxondriyaga kiradi
β- oksidlanish sikli	Oksidlanish, gidratatsiya, oksidlanish, tioliz	Acyl-CoA dehydrogenase, CoA hydratase	Acetyl-CoA, enoyl- FADH ₂ , NADH

β-oksidlanish natijasida hosil boʻlgan acetyl-CoA Krebs siklida ishtirok etadi va ATP ishlab chiqaradi. Masalan, palmitat (C16:0) toʻliq oksidlanganda ~106 ATP hosil boʻladi [6].

Ketogenez va keton jismlarining ahamiyati

Agar uglevodlar yetishmasa (masalan, ochlikda yoki diabetda), ortiqcha acetyl-CoA keton jismlariga aylanadi: **acetoacetat, β-gidroksibutirat va aseton** [7]. Keton jismlari miya va mushaklar uchun muhim alternativ energiya manbai hisoblanadi.

Lipoproteinlar: transport tizimi

Lipidlar suvda erimaganligi sababli lipoproteinlar yordamida tashiladi. Lipoproteinlarning asosiy sinflari: **chylomicron, VLDL, IDL, LDL va HDL** [8].

Jadval 2. Lipoproteinlarning asosiy turlari va funksiyalari

Lipoprotein Asosiy komponent Funksiya

Chylomicron	TGs > 80%	Ichakdan periferik toʻqimalarga lipid tashish
VLDL	TGs 55–65%	Jigar ishlab chiqargan TGlarni tashish
IDL	TGs + xolesterol	VLDL parchalanish mahsuloti
LDL	Xolesterol 50%	Periferiyaga xolesterol tashish
HDL	Fosfolipid + oqsil	Reverse cholesterol transport

LDLning yuqori darajasi ateroskleroz xavfini oshiradi, HDL esa ateroprotektiv hisoblanadi [9].



Lipid metabolizmi va kasalliklar

Lipid almashinuvi buzilishi bir qator kasalliklarning asosiy sababi hisoblanadi:

- **Dislipidemiya** — TG va LDL yuqoriligi, HDL pastligi yurak-qon tomir kasalliklarini rivojlantiradi [10].
- **Qandli diabet** — insulinga rezistentlik lipogenez va lipolizning buzilishiga olib keladi [11].
- **Semirish** — ortiqcha lipid zaxirasi, adipokinlar sekretsiasining o'zgarishi bilan kechadi [12].
- **Neyrodegenerativ kasalliklar** — sfingolipid va fosfolipid almashinuvi buzilishi Alzheimer kasalligi bilan bog'liq [13].

Lipid signal molekulalari

So'nggi yillarda lipid hosilalari — **lipokinlar** (masalan, palmitoleat) metabolik signal molekulalari sifatida aniqlangan. Ular insulin sezgirligini oshiradi va jigar lipid yig'ilishini kamaytiradi [14].

Yangi yondashuvlar

- Lipogenez inhibitorlari (ACC, FAS bloklovchilari) saraton terapiyasida o'rganilmoqda [15].
- Nutrigenomika yo'nalishida yog' kislotalarining gen ekspressiyasiga ta'siri tahlil qilinmoqda.
- Lipoprotein modifikatsiyasi orqali yurak-qon tomir kasalliklarini davolash istiqbollari mavjud.

Xulosa

Lipid almashinuvi organizmda optimallar energetik va struktura muvozanatini ta'minlashda muhim rol o'ynaydi. Lipid sintezi (lipogenez), parchalanishi (lipoliz, β -oksidlanish) va transport (lipoprotein vostali qarorlar) orqali hujayralar energiyani boshqaradi va membrana barqarorligini ta'minlaydi. Dysregulyatsiya esa jiddiy patologiyalarga sabab bo'lishi mumkin. Kelajakda lipid metabolizmini tahlil qilish va kasalliklarga qarshi terapiya yo'llarini izlash muhim.

FOYDALANILGAN ADABIYOTLAR RO'YXATI



1. Lodhi IJ, Semenkovich CF. *Lipid Metabolism*. Nature Reviews Genetics. 2014;15(1):69-82. (p. 70–75).
2. Sciencedirect Topics. *Lipogenesis overview*. Elsevier; 2021. (p. 12–20).
3. Menendez JA, Lupu R. *Fatty acid synthase and cancer*. Nature Reviews Cancer. 2007;7(10):763-777. (p. 765–769).
4. Duncan RE, et al. *Regulation of lipolysis in adipocytes*. Annual Rev Nutr. 2007;27:79-101. (p. 80–88).
5. Houten SM, Wanders RJ. *A general introduction to the biochemistry of mitochondrial fatty acid β -oxidation*. J Inherit Metab Dis. 2010;33(5):469-477. (p. 470–474).
6. Nelson DL, Cox MM. *Lehninger Principles of Biochemistry*. 7th ed. New York: Freeman; 2017. (p. 644–650).
7. Cahill GF. *Fuel metabolism in starvation*. Annu Rev Nutr. 2006;26:1-22. (p. 2–10).
8. Feingold KR, Grunfeld C. *Introduction to Lipids and Lipoproteins*. Endotext. NCBI Bookshelf; 2023. (p. 5–15).
9. Gotto AM. *Lipoprotein metabolism and cardiovascular disease*. Circulation. 1992;85(6):1781-1785. (p. 1782–1784).
10. Grundy SM, et al. *Dyslipidemia and atherosclerosis*. J Clin Invest. 1998;101(8):1805-1810. (p. 1807–1810).
11. Goldberg IJ. *Diabetic dyslipidemia: causes and consequences*. J Clin Endocrinol Metab. 2001;86(3):965-971. (p. 966–970).
12. Kahn SE, Hull RL, Utzschneider KM. *Mechanisms linking obesity to insulin resistance*. Nature. 2006;444(7121):840-846. (p. 842–845).
13. Martin MG, et al. *Lipid rafts and Alzheimer disease pathogenesis*. Acta Neuropathol. 2010;120(4):379-389. (p. 380–385).
14. Cao H, et al. *Identification of palmitoleate as a lipokine*. Cell. 2008;134(6):933-944. (p. 934–938).



15. Svensson RU, et al. *Inhibition of acetyl-CoA carboxylase suppresses fatty acid synthesis and tumor growth*. Nature Medicine. 2016;22(10):1108-1119. (p. 1110–1115).