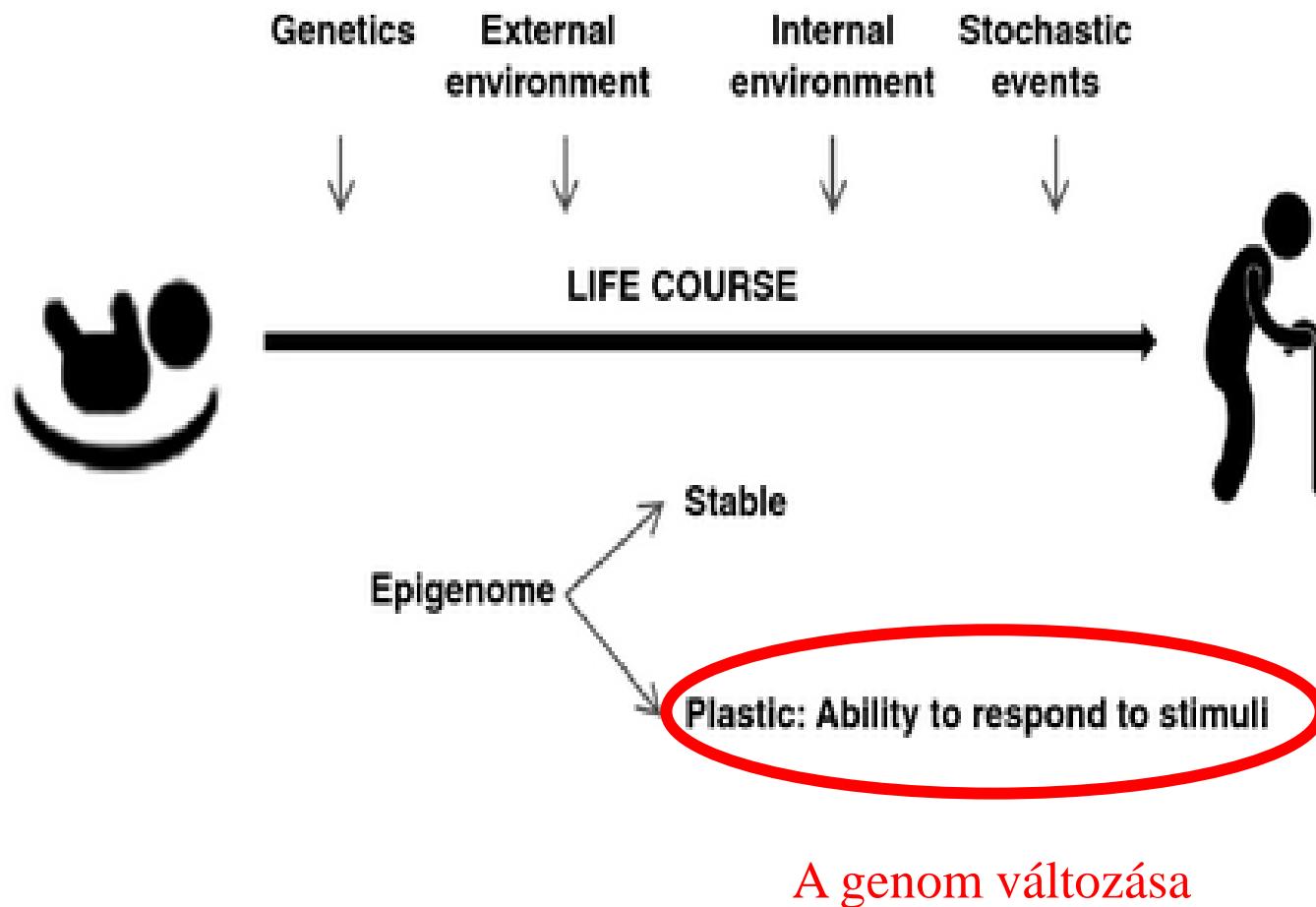


# A genetika tényezők szerepe gyermekek táplálásában

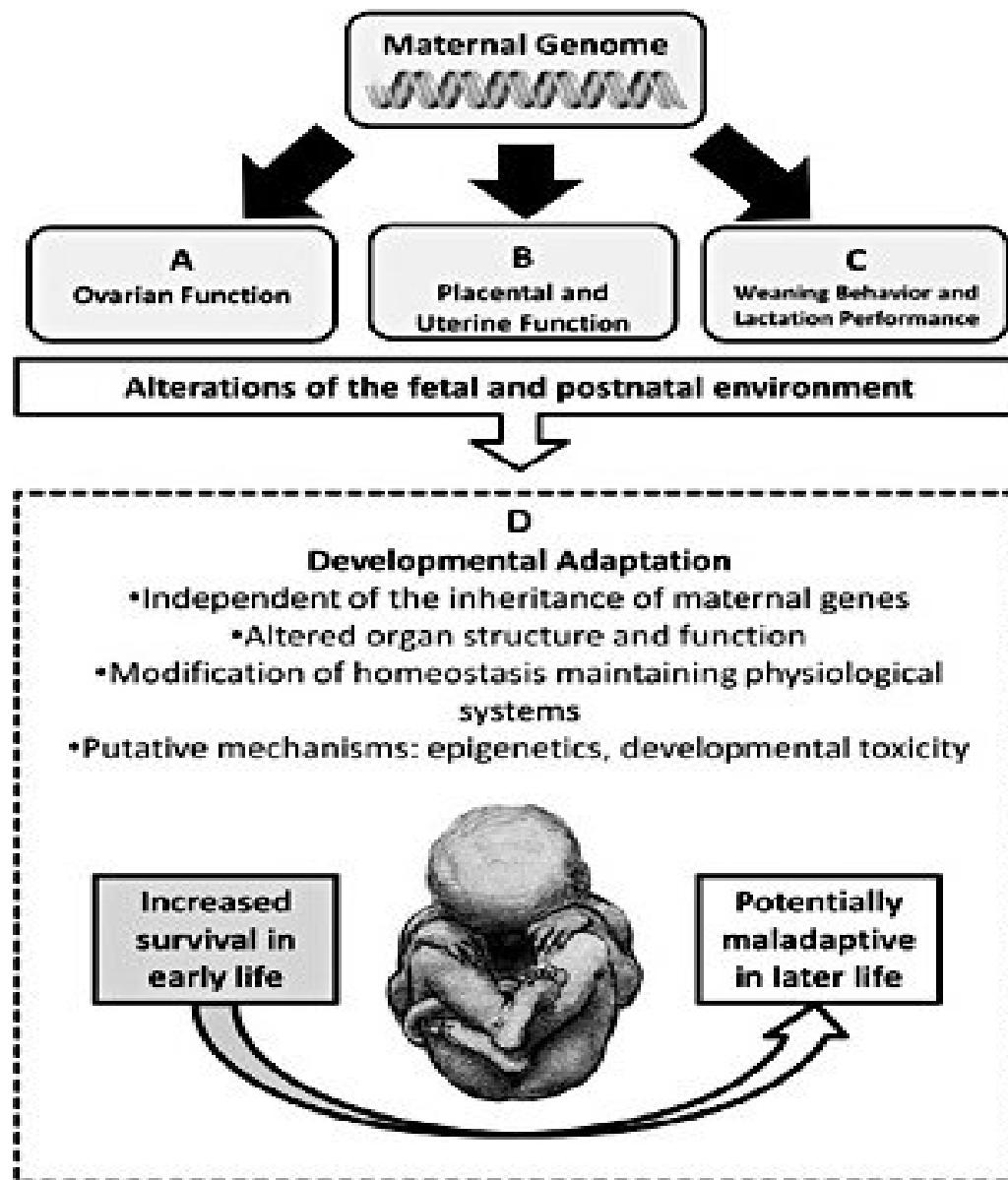
Decsi Tamás

Pécsi Tudományegyetem  
Gyermekgyógyászati Klinika

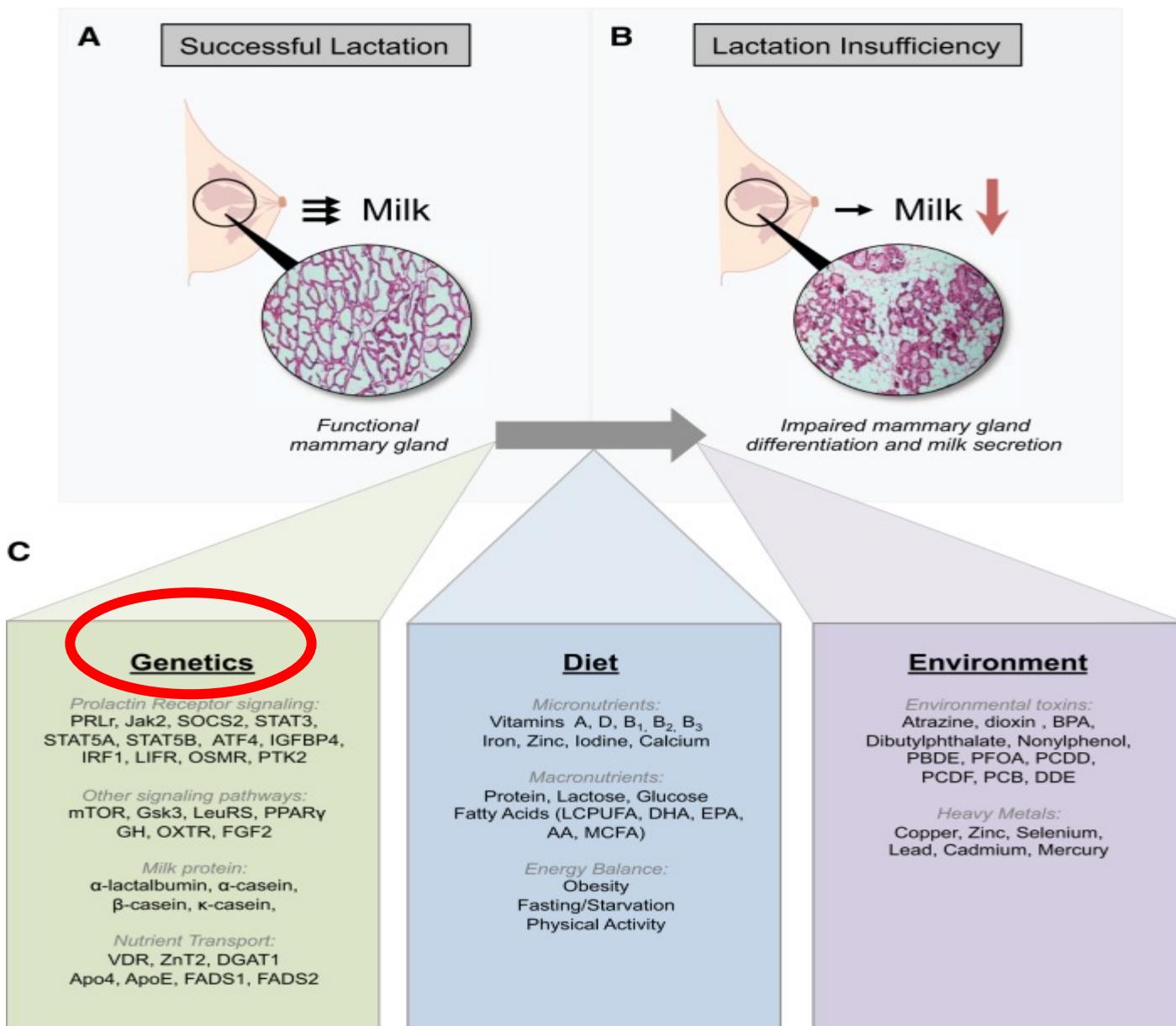
# A korai hatások késői következményei



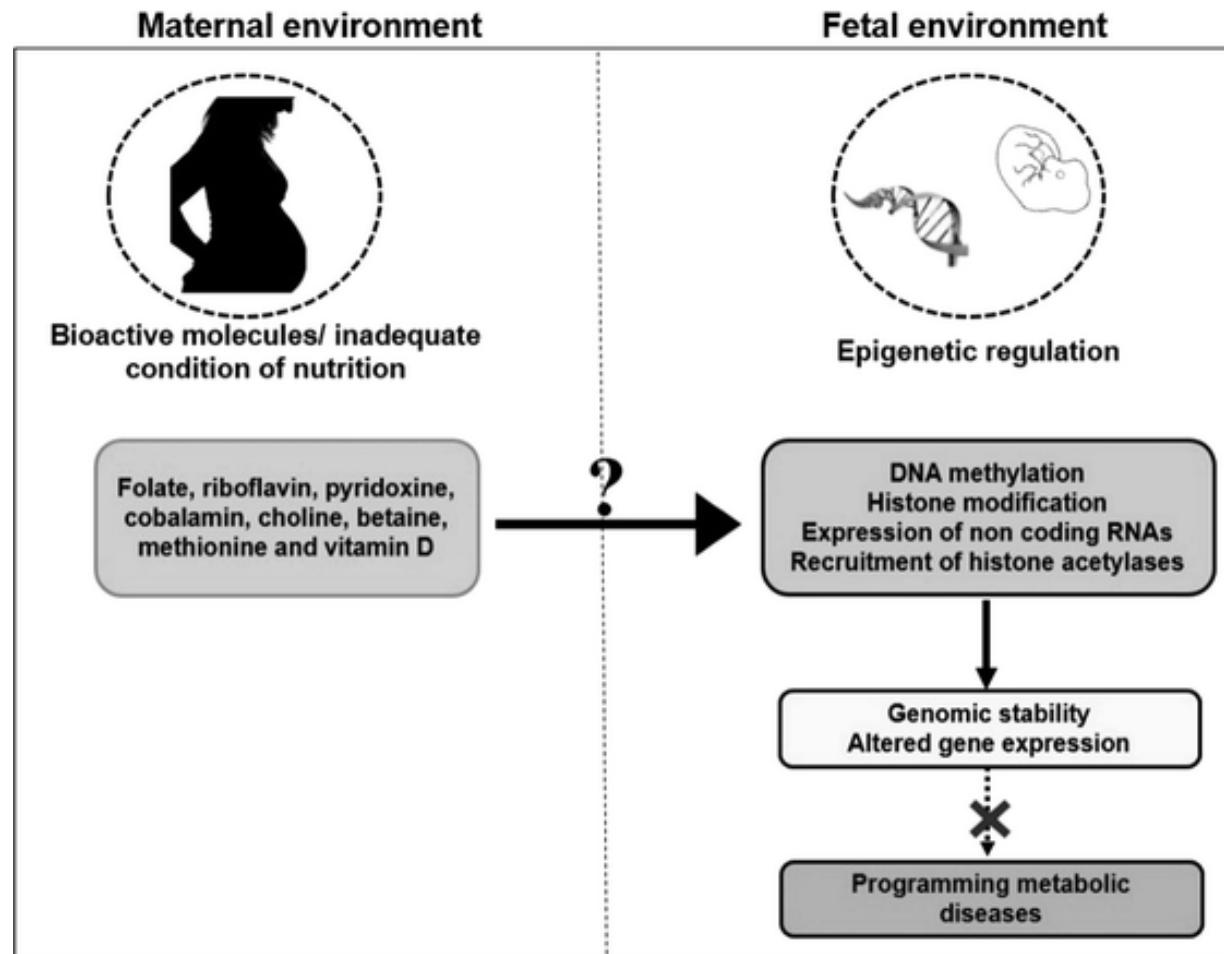
# The Advanced Fetal Programming Hypothesis



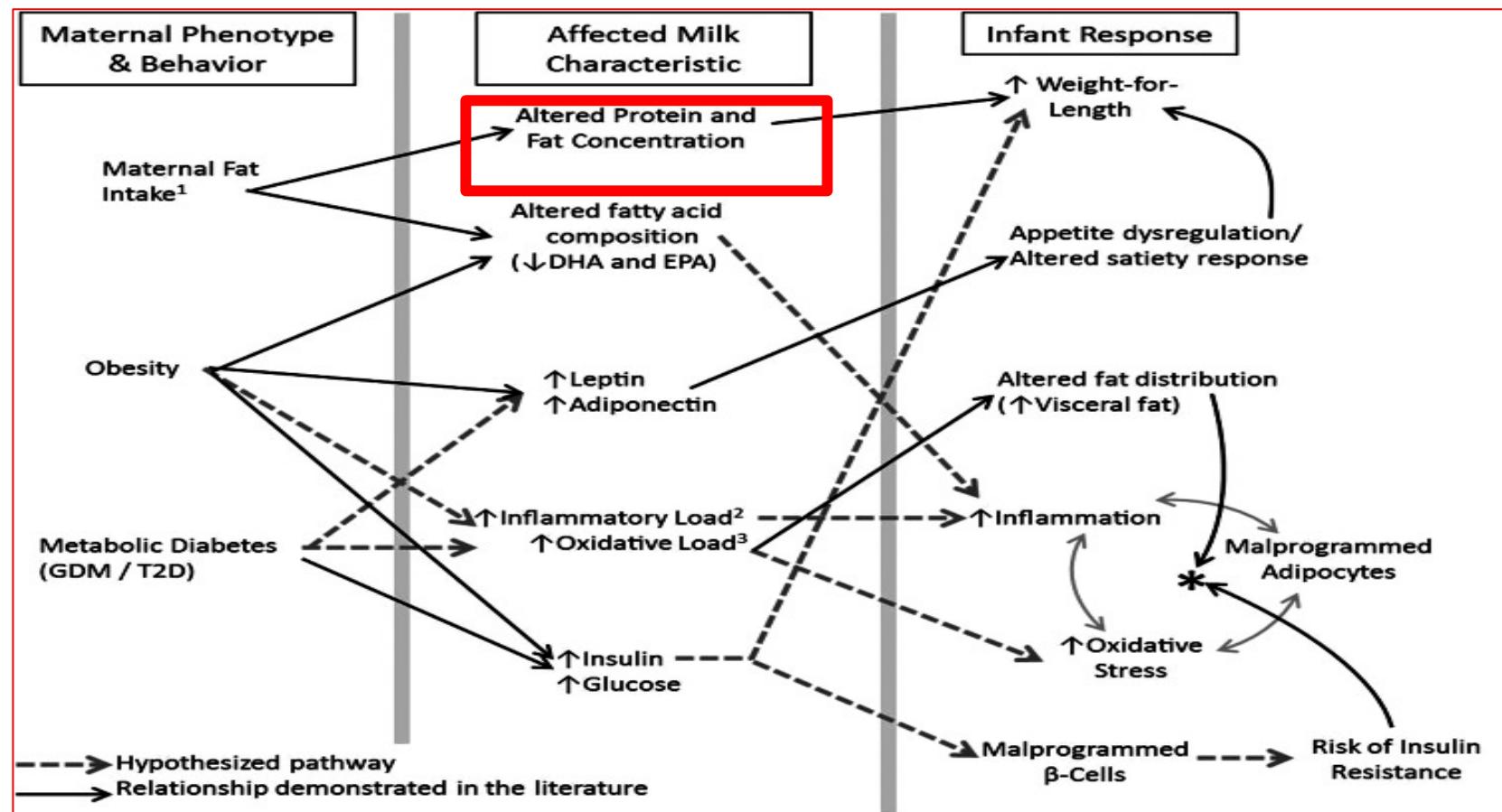
# A szoptatás sikert befolyásoló tényezők



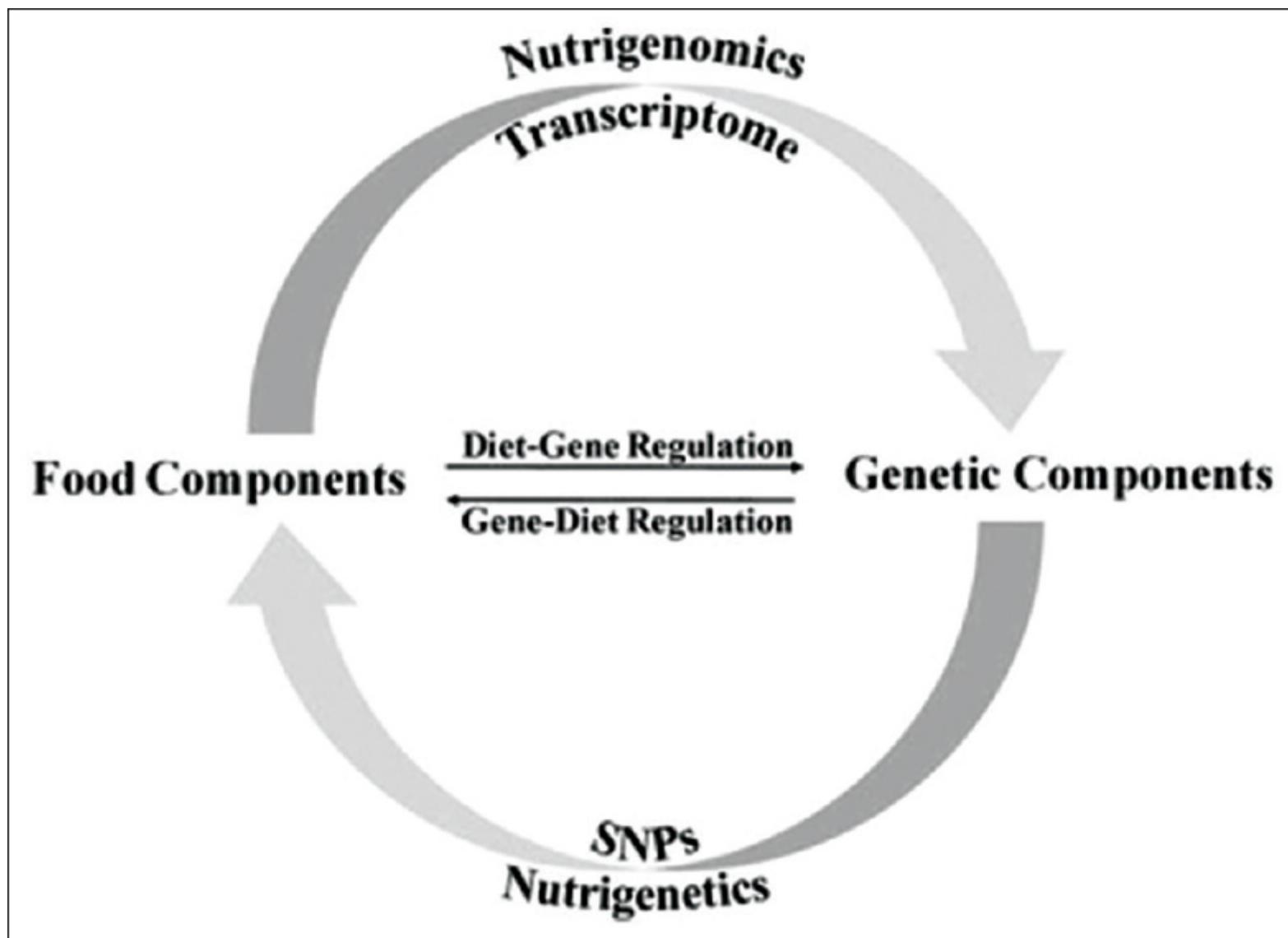
# A biológiaileg aktív molekulák epigenetikai módosulása



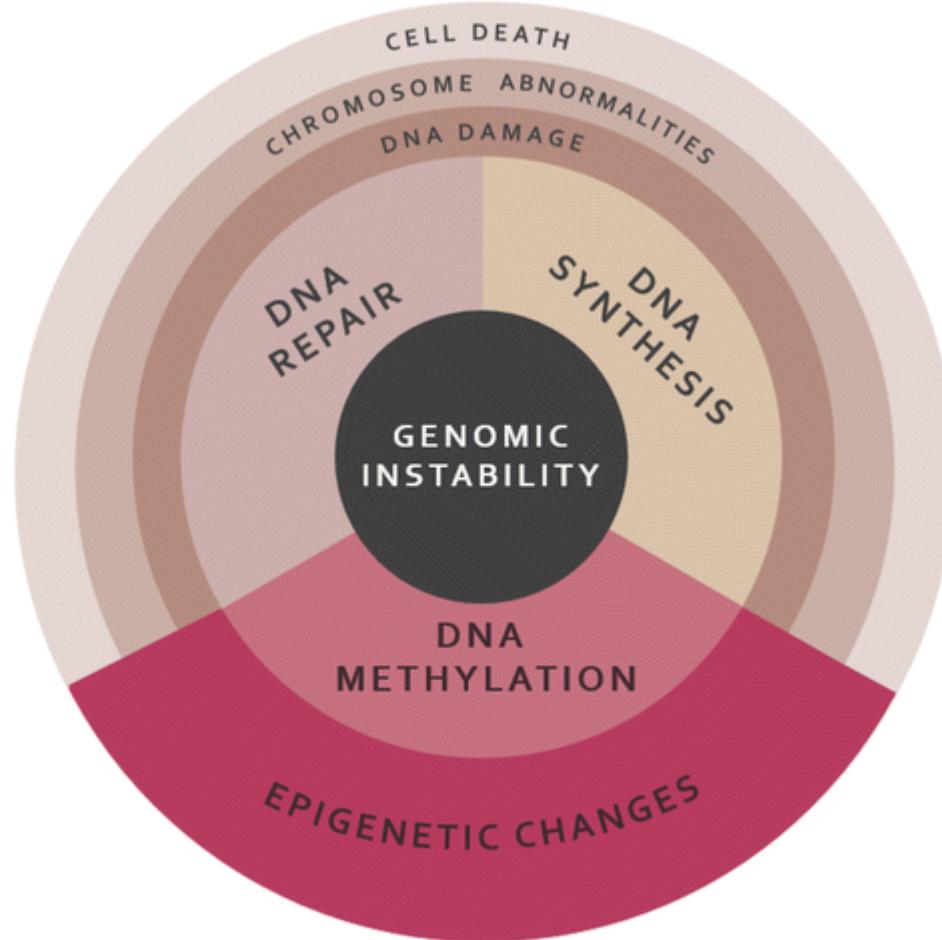
# A női tej szerepe a programozódás jelenségében



# Gén-táplálék kölcsönhatás

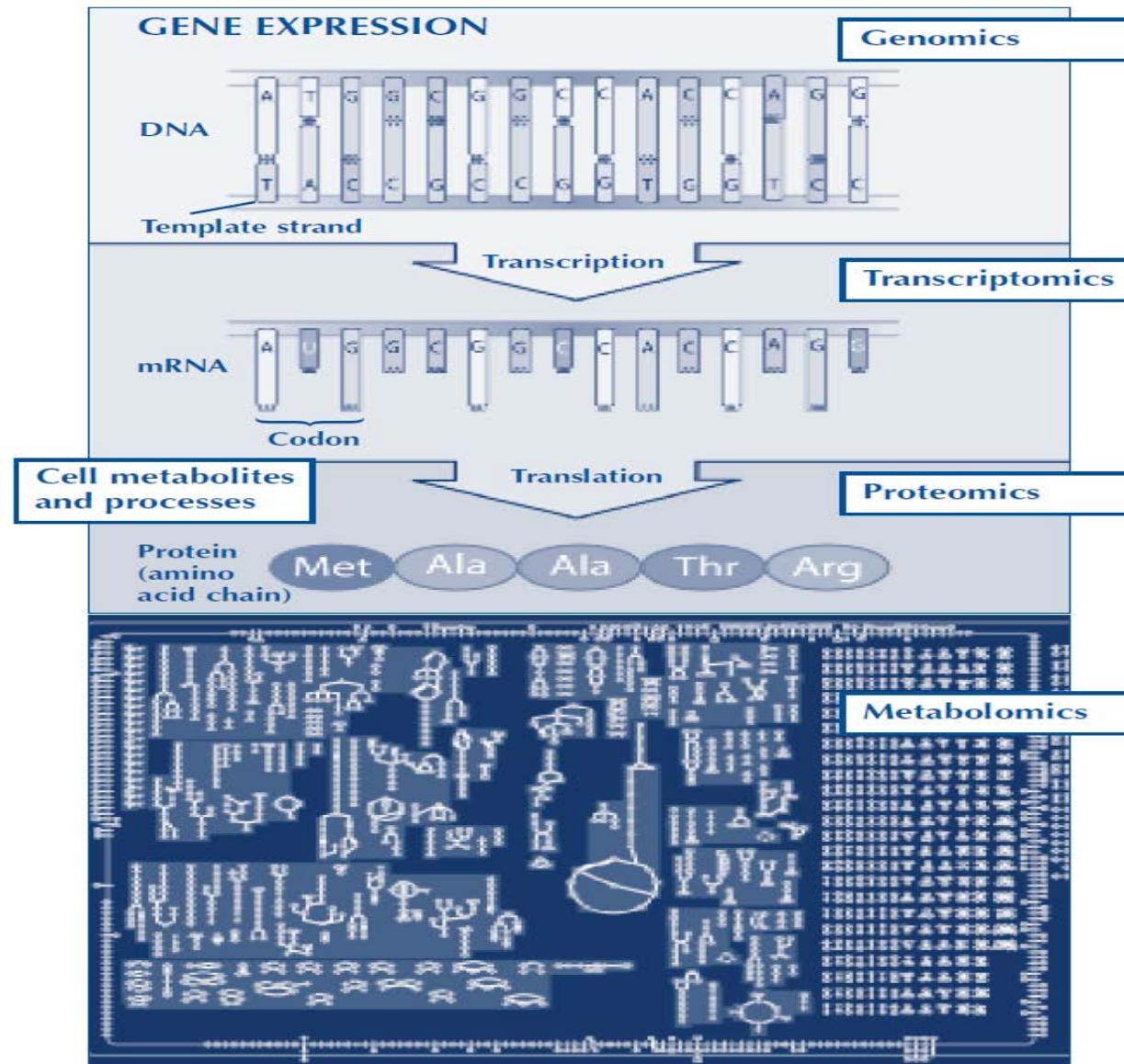


# A genom változásához hozzájáruló tényezők

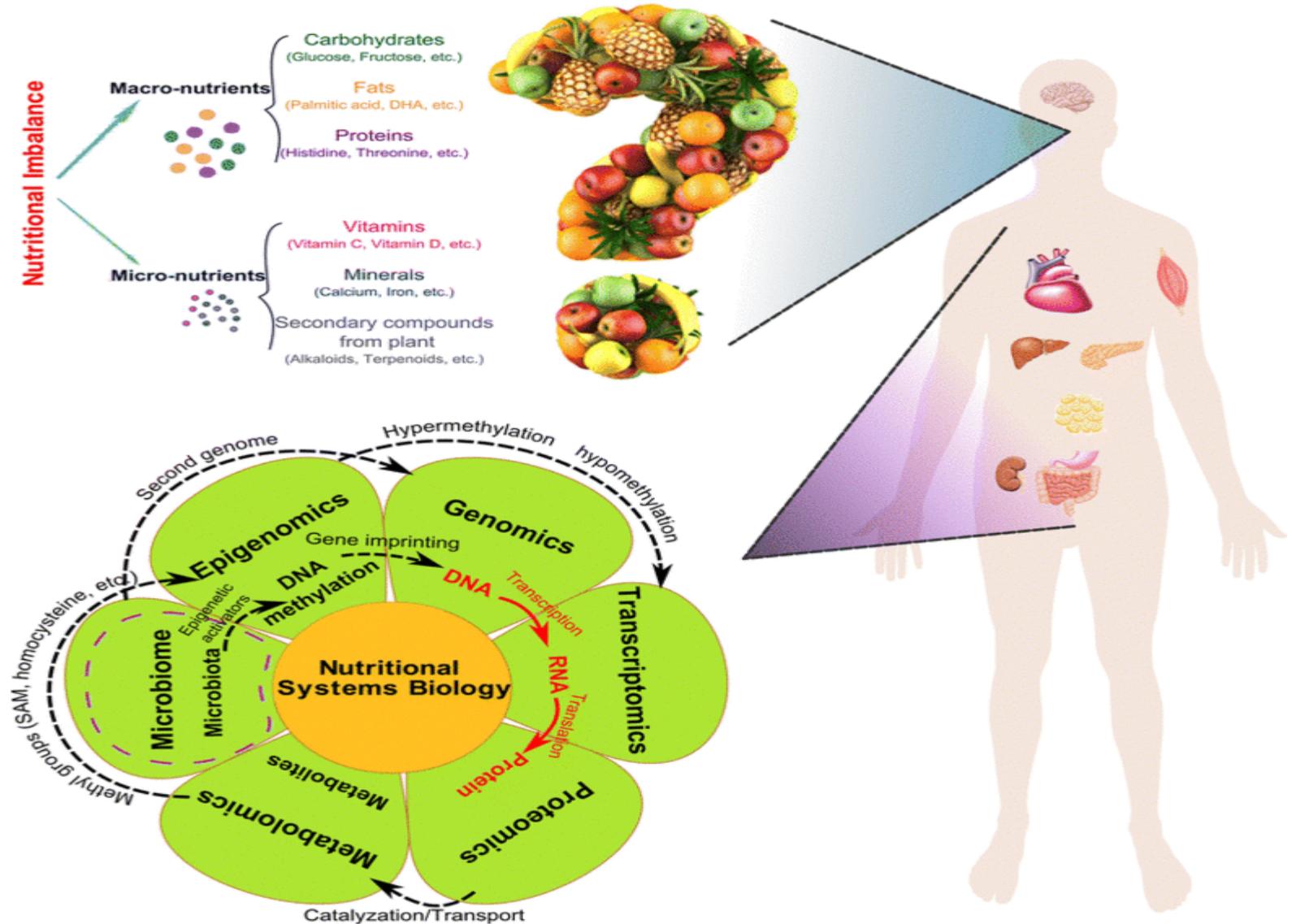


# A táplálkozás és a gének egymásra hatását vizsgáló módszerek

FIGURE 3. The steps in gene expression and the 'omics' descriptor

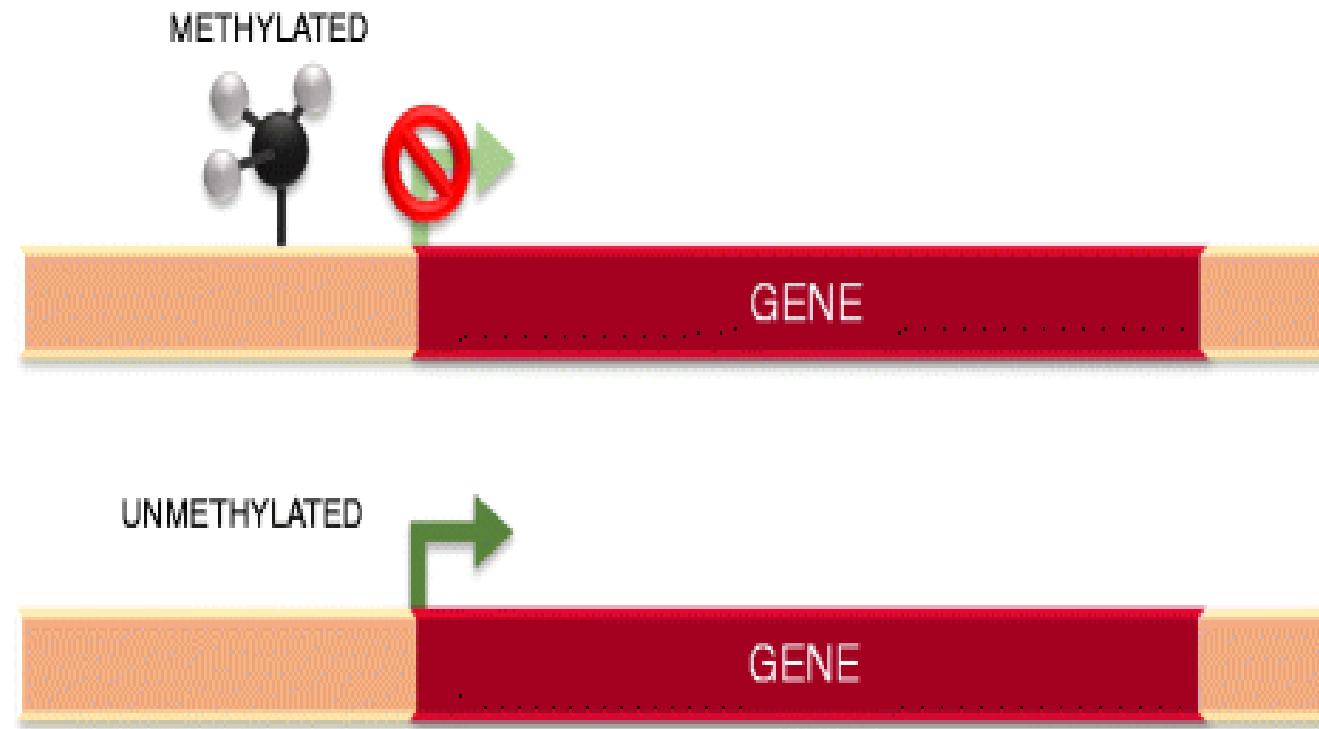


# A táplálkozás és a gének egymásra hatását vizsgáló módszerek

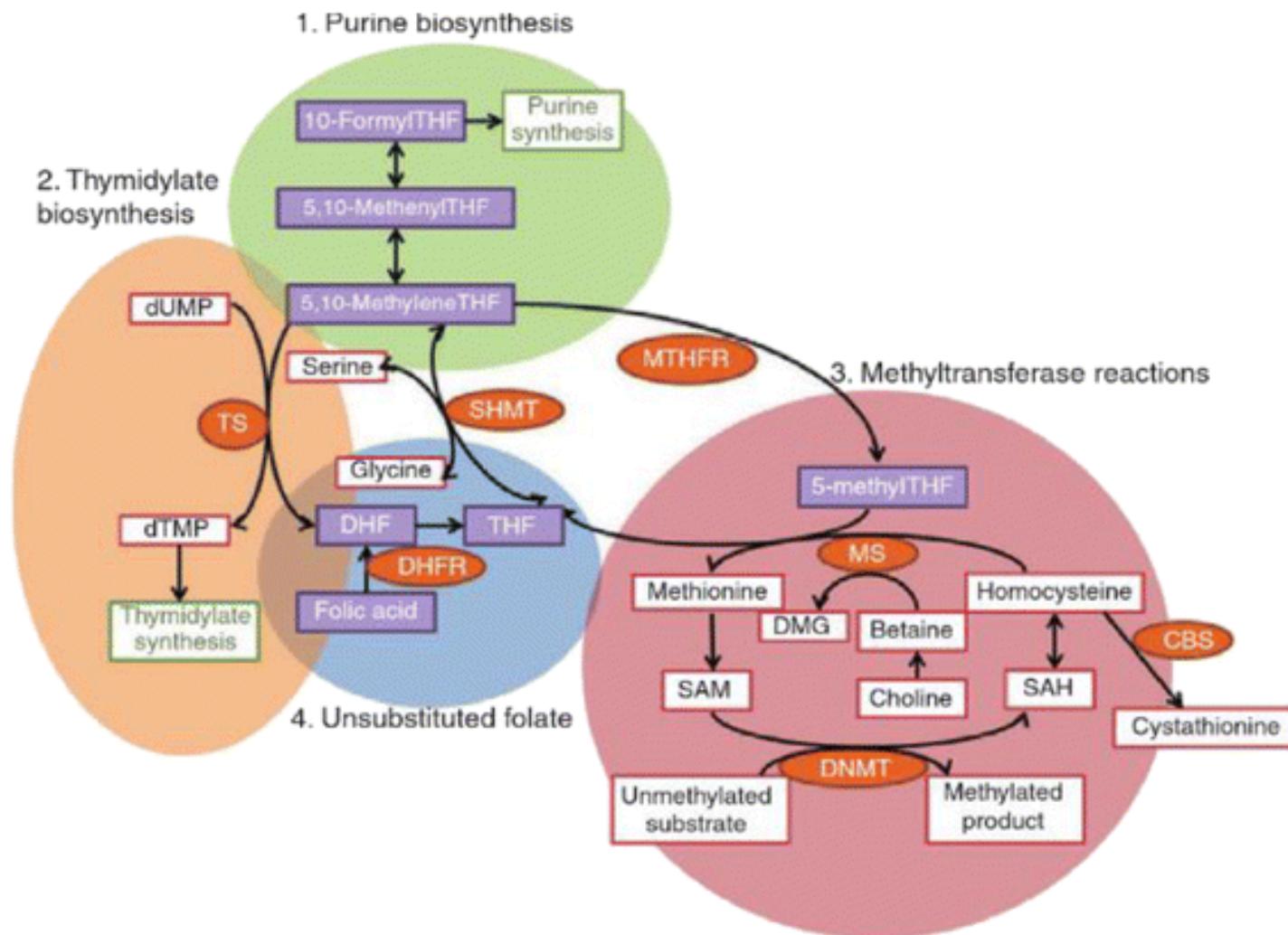


Zhao Y et al, Studying the relationship between genetics and nutrition in the improvement of human health.  
Genes & Nutrition 10:31 2015

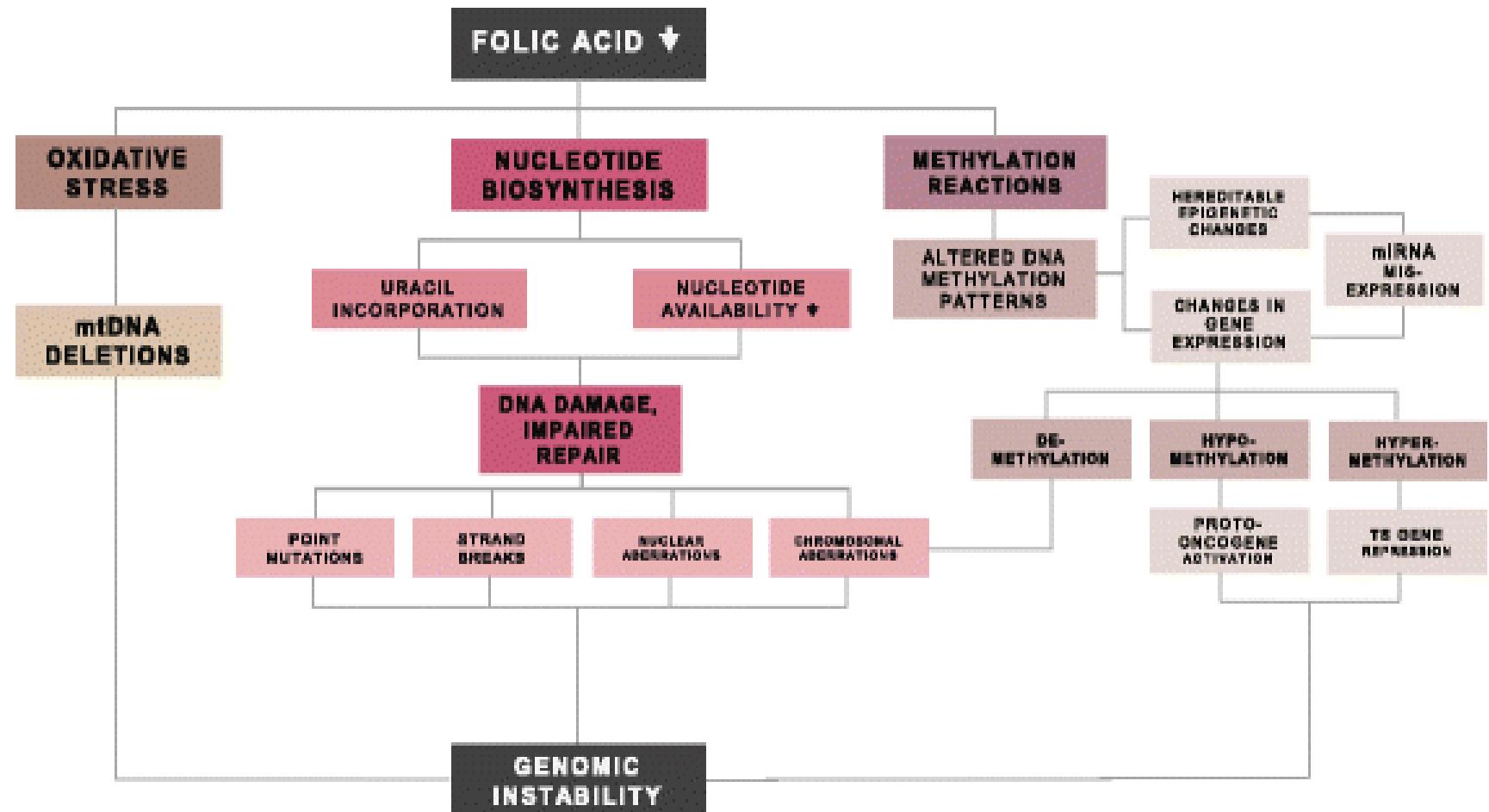
# A metiláció hatása a gén expresszivitására



# A folsav sejten belüli hatásai

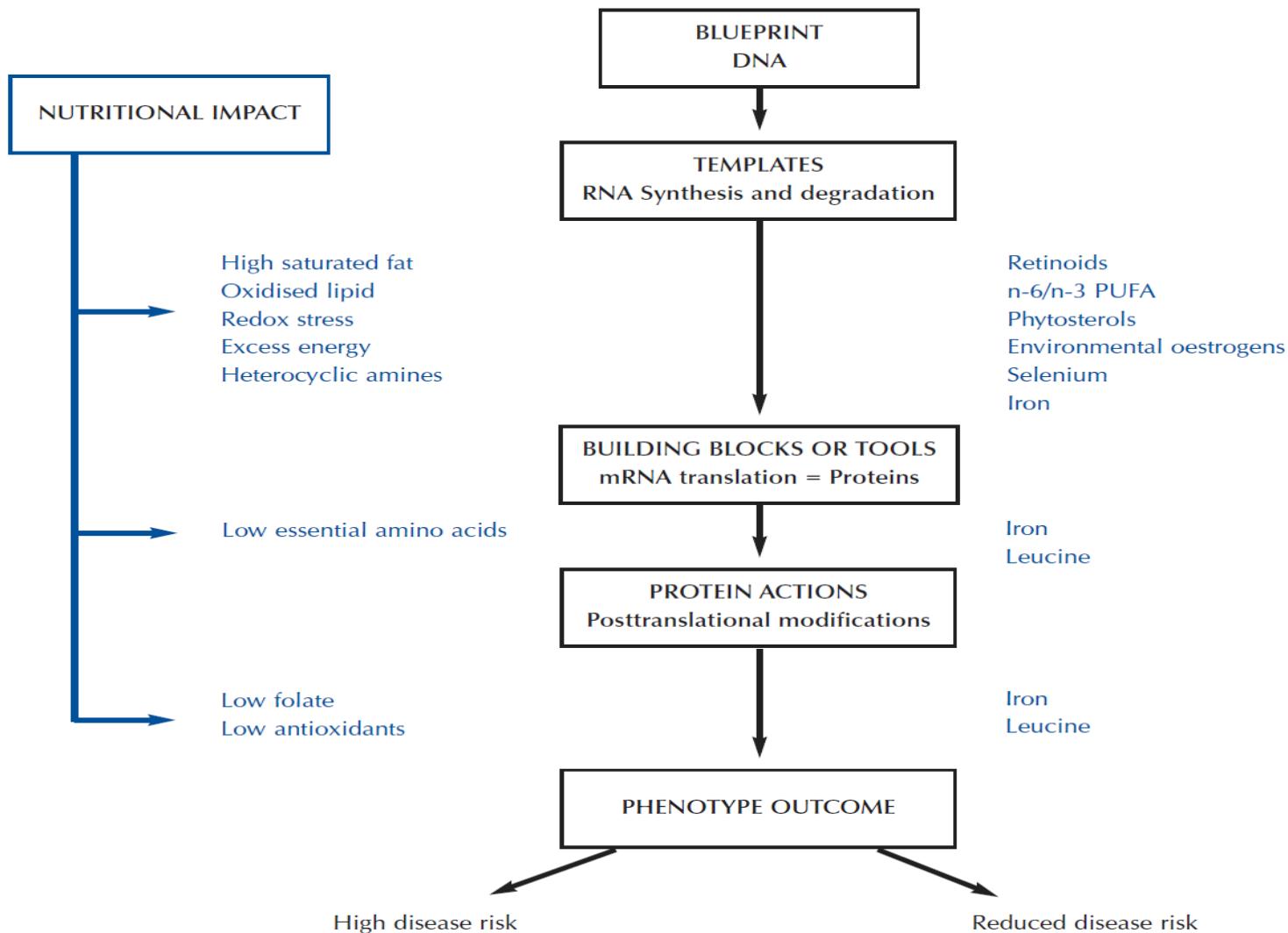


# Egy táplálékösszetevő, a folsav potenciális szerepe a genom változékonyságában



# A fehérjék expresszóját befolyásoló táplálkozási hatások

**FIGURE 7. Pathway of protein expression showing where regulation occurs by nutrients**



Showed in blue are where nutrients can impact

Source: Clarke, S.D. (2001). The Human Genome and Nutrition. In Bowman, B.A. and Russell, R.M. ed. Present Knowledge in Nutrition, 8th ed., ILSI Press, Washington, DC. Reproduced with permission from ILSI Press.

# A fehérjék expresszóját befolyásoló táplálkozási hatások támadásponjai

**Points in the pathway of protein expression regulated by dietary constituents**

<i>Targeted Site</i>	<i>Examples of Nutrient Regulator</i>
Gene transcription	Fatty acids, glucose, cholesterol, retinoids, vitamin D
mRNA stability	Fatty acid, glucose, selenium, iron
mRNA processing	Polyunsaturated fatty acids, glucose
mRNA translation	Iron, amino acids
Post-translational modification	Vitamins and minerals

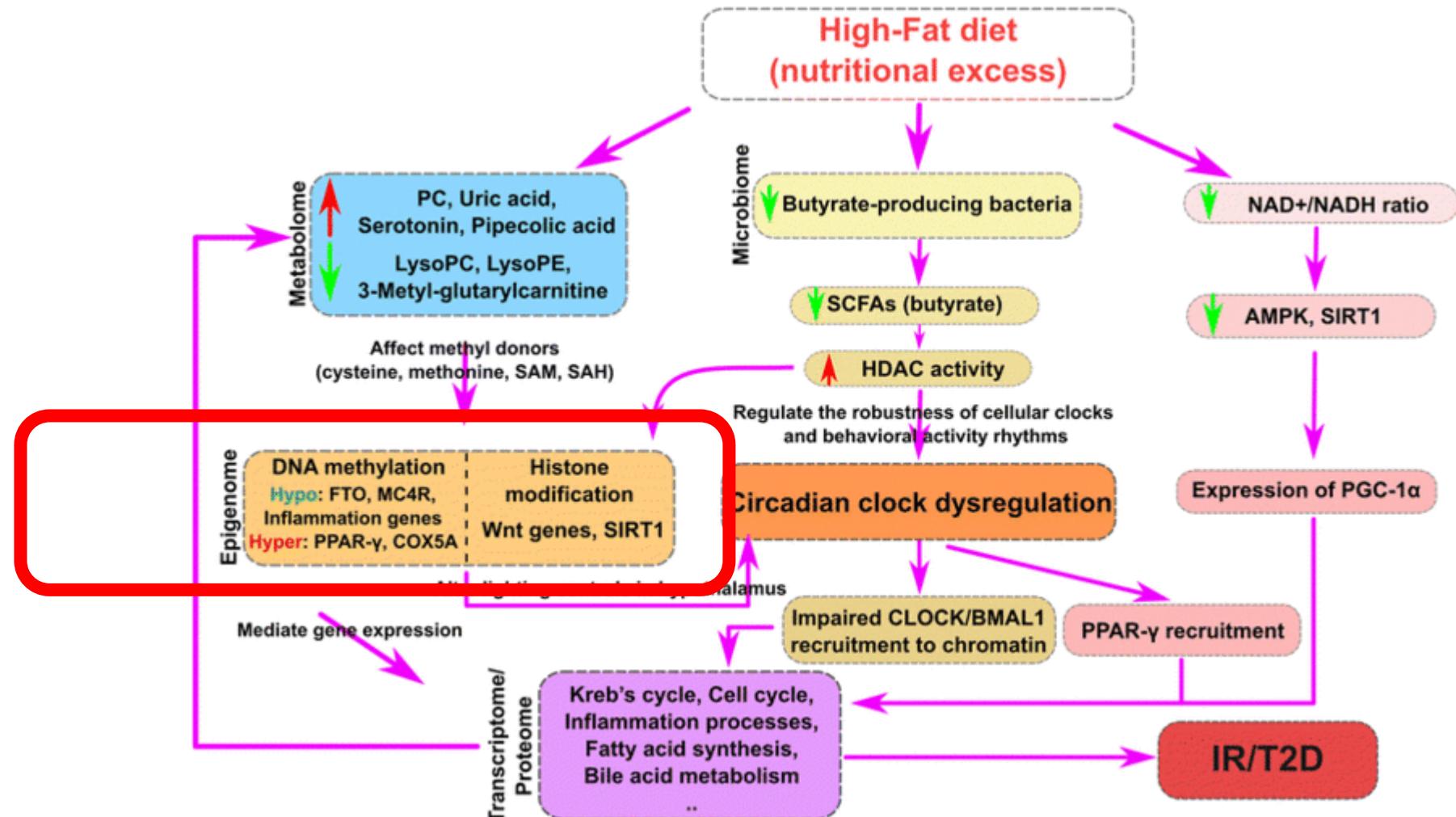
# Táplálékokkal összefüggő genetikai polimorfizmusok potenciális egészségügyi következményei

Examples of known cellular processes and genetic polymorphism with direct consequences for nutrition

<i>Cellular Process</i>	<i>Gene with known polymorphisms</i>	<i>Nutrition/health Impact</i>
Folate metabolism	Methylene tetrahydrofolate reductase, cystathione beta-synthase, methionine synthase, glutamate carboxy-peptidase III	Risk of neural tube defect, Down's syndrome, CVD and cancer
Iron homeostasis	Hereditary haemochromatosis, linked gene HFE and transferrin receptor	Effect on iron requirements, anaemia, and iron overload
Bone health	Vitamin D receptor, oestrogen receptor, type I collagen	Effect on bone metabolism, osteoporosis, mediation of calcium and phosphorus translocation
Lipid metabolism	Apolipoprotein (AIV, B, C3, E), low density lipoprotein receptor, lipoprotein lipase	Effect on blood cholesterol and other cardiovascular risk factors
Immune function	HLA (MHC), tumour necrosis factor $\alpha$ and other cytokines	Susceptibility to various food allergies (such as coeliac disease) and modified susceptibility to cancer through diet

Source: Derived from Elliott, R. and Ong, T.J. (2002). Nutritional genomics, *BMJ* 324, 1438-42, with permission from BMJ Publishing Group.

# A táplálék magas zsírtartalma és a diabetes kialakulása: biokémiai és **genetikai** mechanizmusok



Zhao Y et al, Studying the relationship between genetics and nutrition in the improvement of human health.  
Genes & Nutrition 10:31, 2015

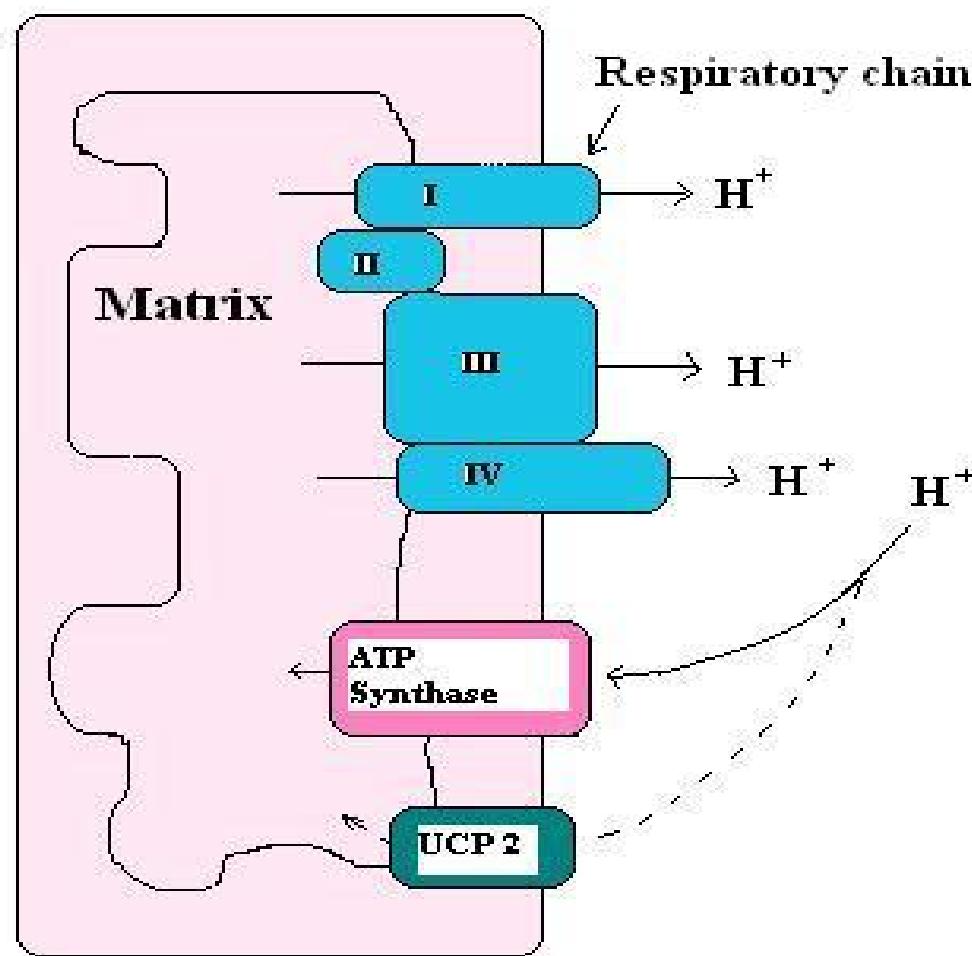
Bokor Sz, Csernus K, Erhardt É, Burus I, Molnár D,  
Decsi T:

Association of n-6 long-chain polyunsaturated fatty acids to —866G/A genotypes of the human uncoupling protein 2 gene in obese children

*Acta Paediatrica*, 96: 1350-1354, 2007.

# UCP 2 function

Mitochondrion

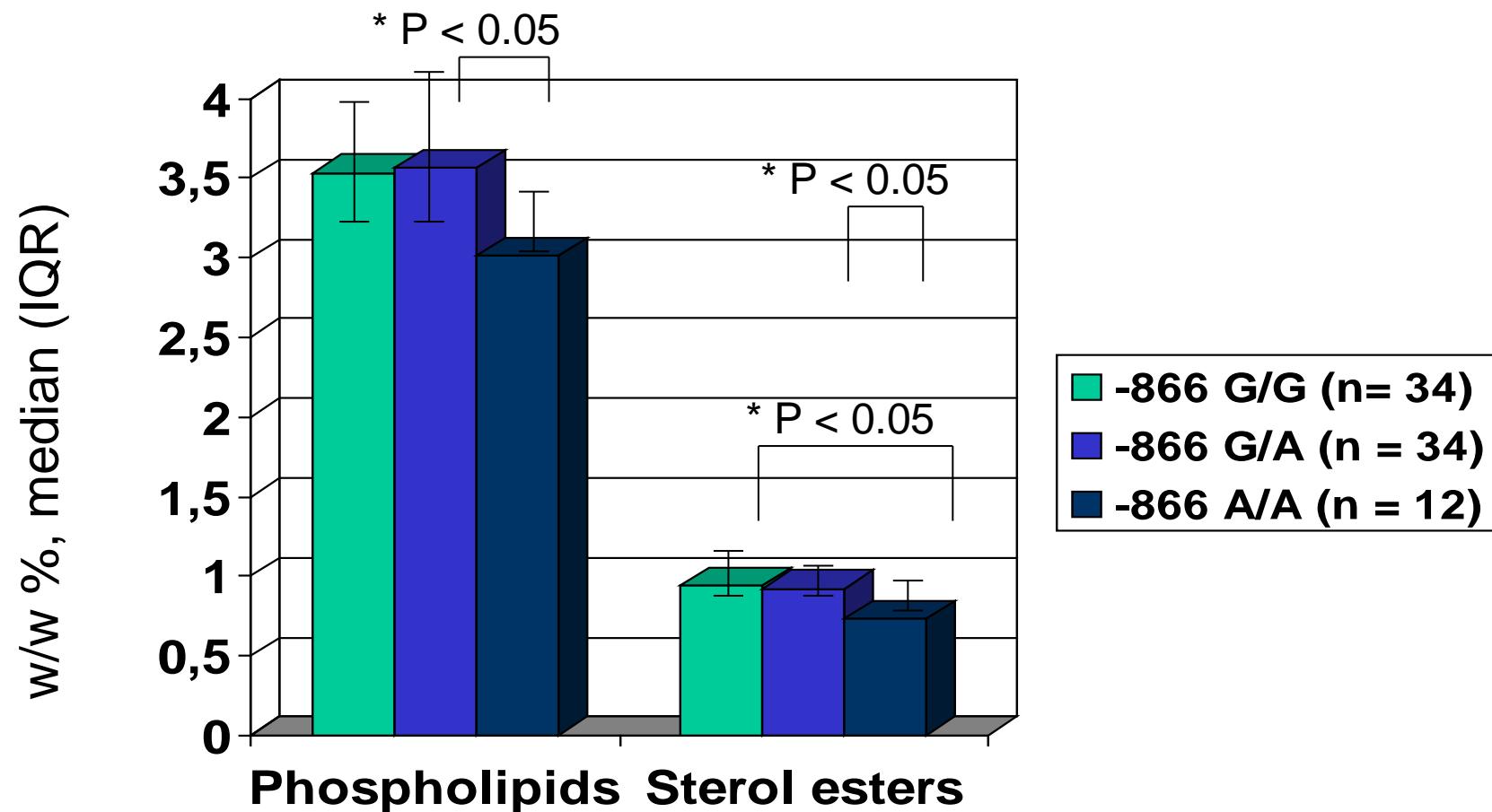


## -866 G/A polymorphism of the UCP2

### A allele variant:

- ◆ Enhanced transcription of the UCP 2 gene
- ◆ Risk of obesity ↓
- ◆ Risk of type 2 diabetes ↑
- ◆ Glucose oxidation ↑
- ◆ Lipid oxidation ↓

# Dihomo-gamma-linolenic acid (C20:3n-6) in plasma phospholipids and sterol esters



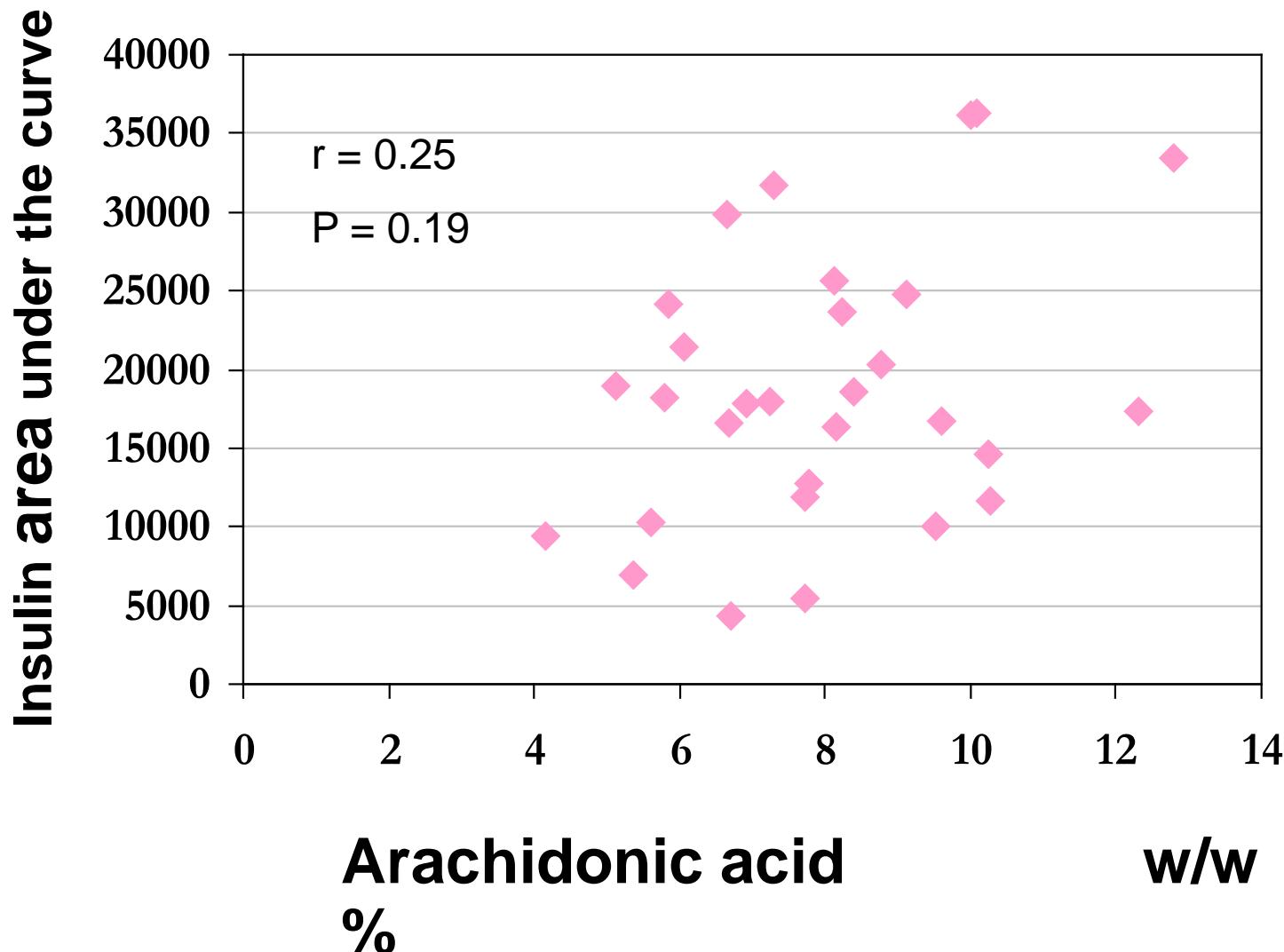
Spearman rank correlation coefficients between DHGLA and AA  
values in plasma sterol esters and insulin concentrations measured  
during OGTT (N = 80)

	DHGLA			AA		
<b>UCP2 genotype:</b>	-866 G/G	-866 G/A	-866 A/A	-866 G/G	-866 G/A	-866 A/A
<b>Insulin 0'</b>	-0.06	0.39	0.2	0.01	-0.06	<b>-0.80**</b>
<b>Insulin 30'</b>	0.31	0.03	0.32	-0.27	0.37	<b>-0.75**</b>
<b>Insulin 60'</b>	<b>0.51**</b>	<b>0.48**</b>	0.42	-0.08	<b>0.43*</b>	<b>-0.75**</b>
<b>Insulin 90'</b>	0.22	0.26	0.28	-0.33	0.17	<b>-0.68*</b>
<b>Insulin 120'</b>	0.34	0.29	0.15	-0.07	0.19	-0.44
<b>Insulin 180'</b>	0.09	0.12	-0.35	-0.03	0.07	-0.59

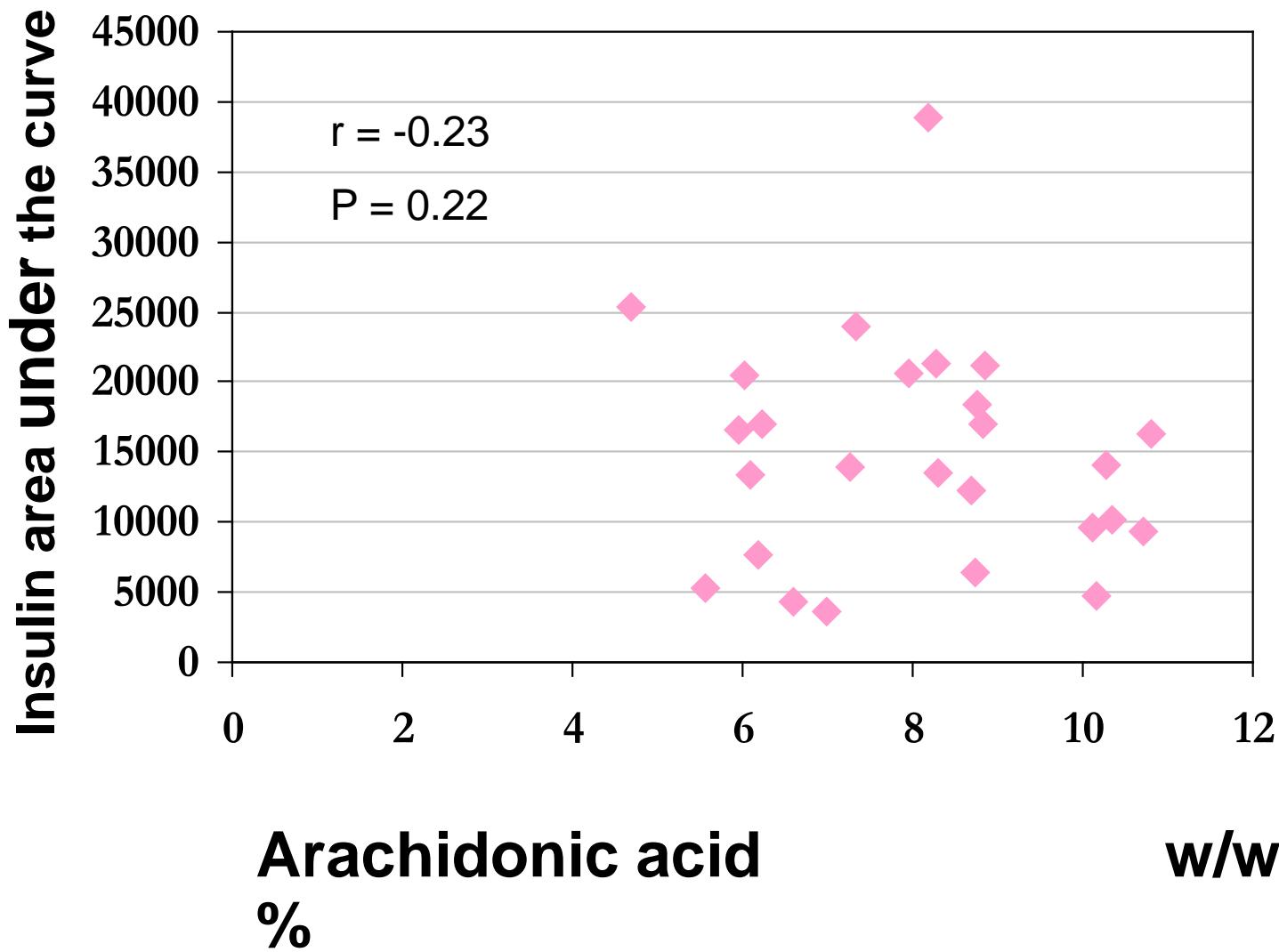
\*P < 0.05; \*\*P < 0.001

Bokor et al, Acta Paediatrica, 96: 1350-1354, 2007

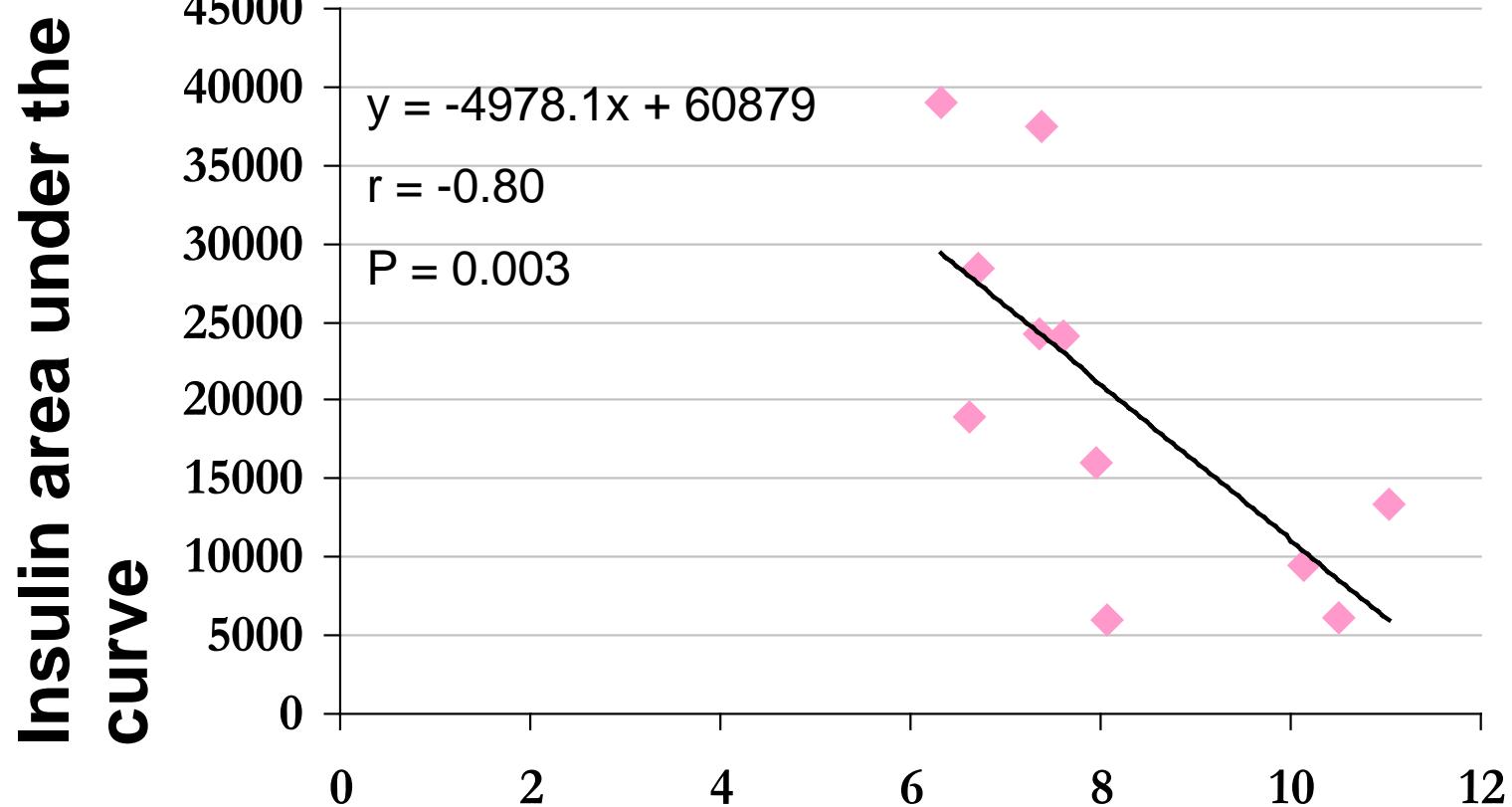
# -866 G/A genotype



# -866 G/G genotype



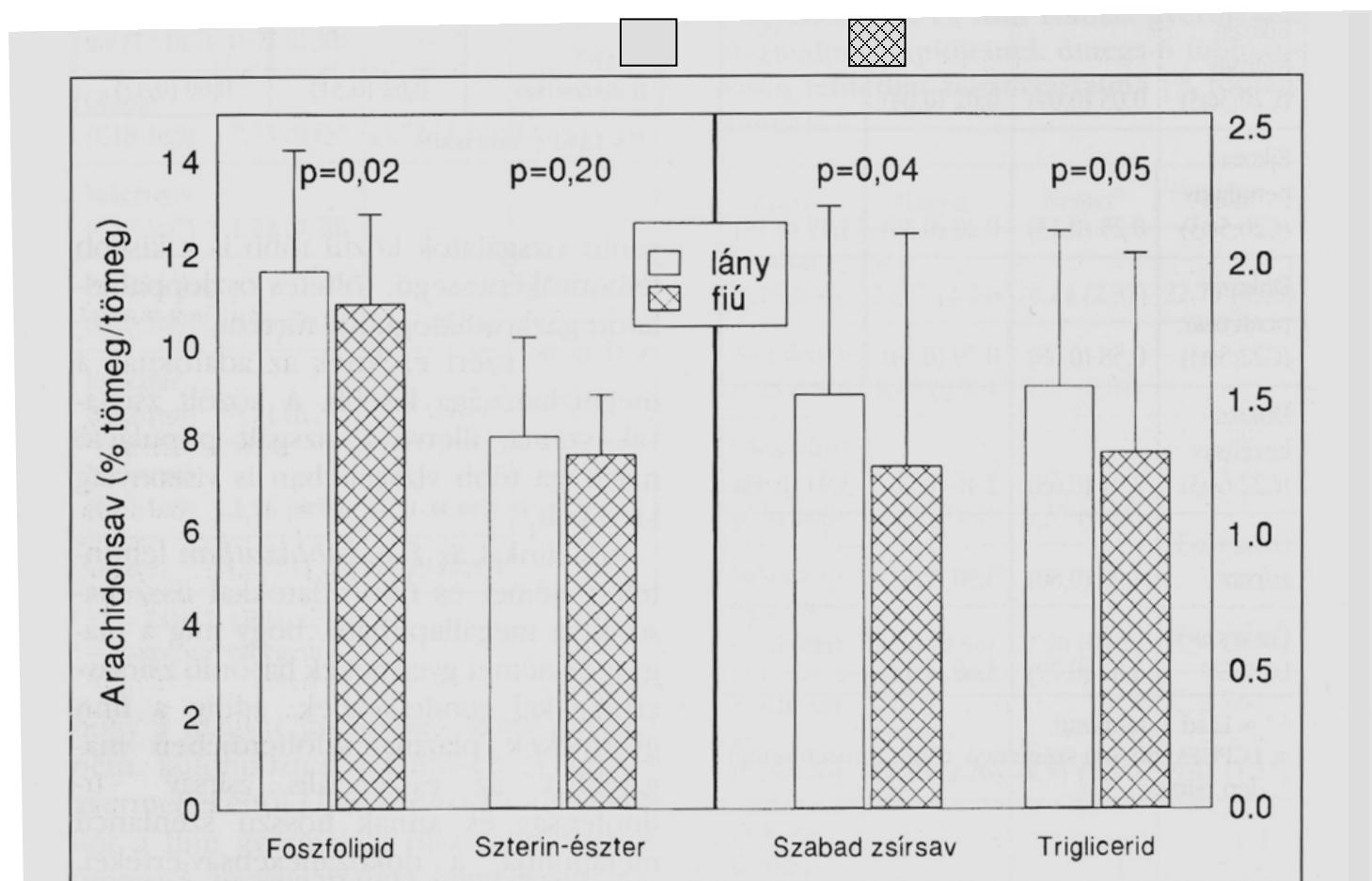
## -866 A/A genotype



**Arachidonic acid  
w/w %**

*Bokor et al, Acta Paediatrica, 96: 1350-1354, 2007*

# Magyar fiúk és lányok zsírsavellátottsága



1. ábra

Magyar iskolás lányok ( $n = 43$ ) és fiúk ( $n = 48$ ) plazmaarachidonsav értékei különböző lipidfrakciókban [% tömeg/tömeg, medián, 3. és 1. kvartális különbsége)

Molnár Sz, Minda H, Burus I, Decsi T: A plazma lipidjeinek zsírsavösszetétele általános iskolás gyermekben Magyarországon Gyermekgyógyászat, 52: 260-265, 2001.

# Pathways of LCPUFA synthesis

## N-6 FATTY ACIDS

**Linoleic acid**  
(C18:2n-6, LA)

$\downarrow$   
 **$\gamma$ -linolenic acid**  
(C18:3n-6, GLA)

$\downarrow$   
**Dihomo- $\gamma$ -linolenic acid**  
(C20:3n-6, DHGLA)

$\downarrow$   
**Arachidonic acid**  
(C20:4n-6, AA)

$\downarrow$   
**Adrenic acid**  
(C22:4n-6)

$\downarrow$   
**Docosapentaenoic acid**  
C22:5n-6

*$\Delta$ -6 desaturation*

*elongation*

*$\Delta$ -5 desaturation*

*elongation*

*elongation,  $\beta$ -oxidation*

## N-3 FATTY ACIDS

**$\alpha$ -linolenic acid**  
(C18:3n-3, ALA)

$\downarrow$   
**Stearidonic acid**  
(C18:4n-3)

$\downarrow$   
**Eicosatetraenoic acid**  
(C20:4n-3)

$\downarrow$   
**Eicosapentaenoic acid**  
(C20:5n-3, EPA)

$\downarrow$   
**Docosapentaenoic acid**  
(C22:5n-3, DPA)

$\downarrow$   
**Docosahexaenoic acid**  
(C22:6n-3, DHA)

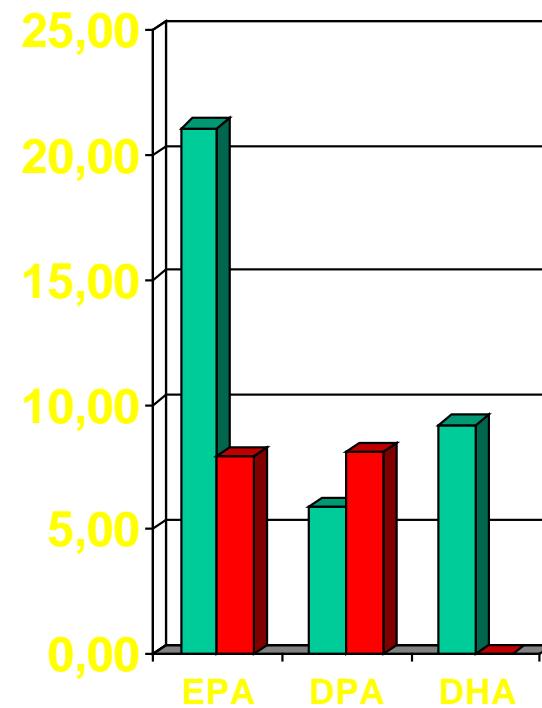
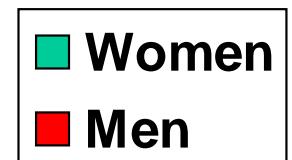
# Estimated net fractional inter-conversion of the tracer [U-<sup>13</sup>C] alpha-linolenic acid

Six women and six men

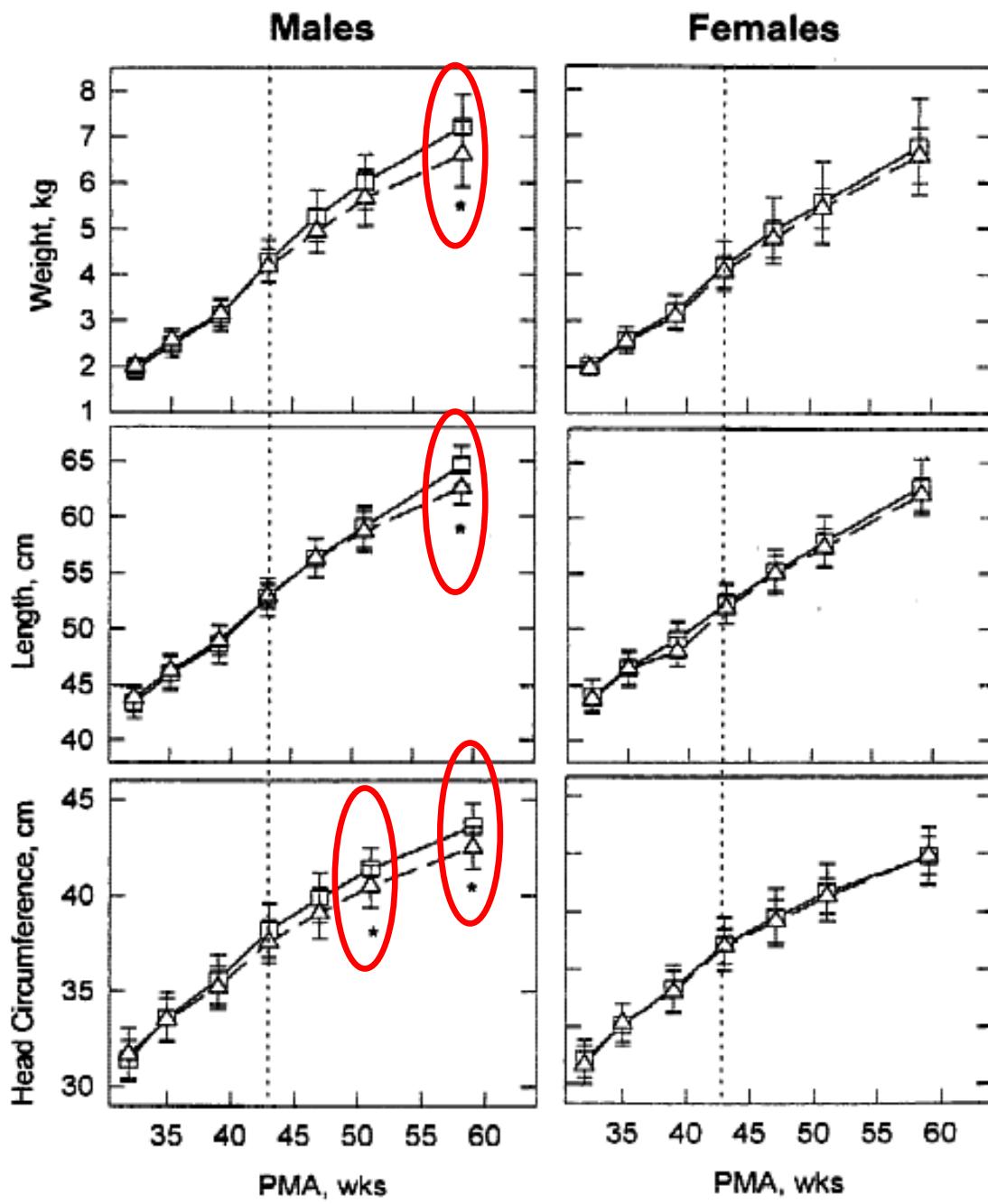
700 mg [U-<sup>13</sup>C] alpha-linolenic acid

blood samples collected 24, 48 and 72 hours and 1, 2 and 3 weeks

data shown from day 21



# Effect of DHA-Containing Formula on Growth of PRETERM Infants to 59 Weeks Postmenstrual Age



- healthy, preterm infants
- fed formula with DHA ( $n = 31$ ,  $\Delta$ )
- or fed formula without DHA ( $n = 32$ ,  $\square$ )
- AA contents were identical in both formulae



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## Early Human Development

journal homepage: [www.elsevier.com/locate/earlhumdev](http://www.elsevier.com/locate/earlhumdev)



# Neonatal fatty acid status and neurodevelopmental outcome at 9 years



Corina de Jong <sup>a,b</sup>, Hedwig K. Kikkert <sup>b</sup>, Jorien Seggers <sup>b</sup>, Gunther Boehm <sup>c</sup>,  
Tamas Decsi <sup>d</sup>, Mijna Hadders-Algra <sup>b,\*</sup>

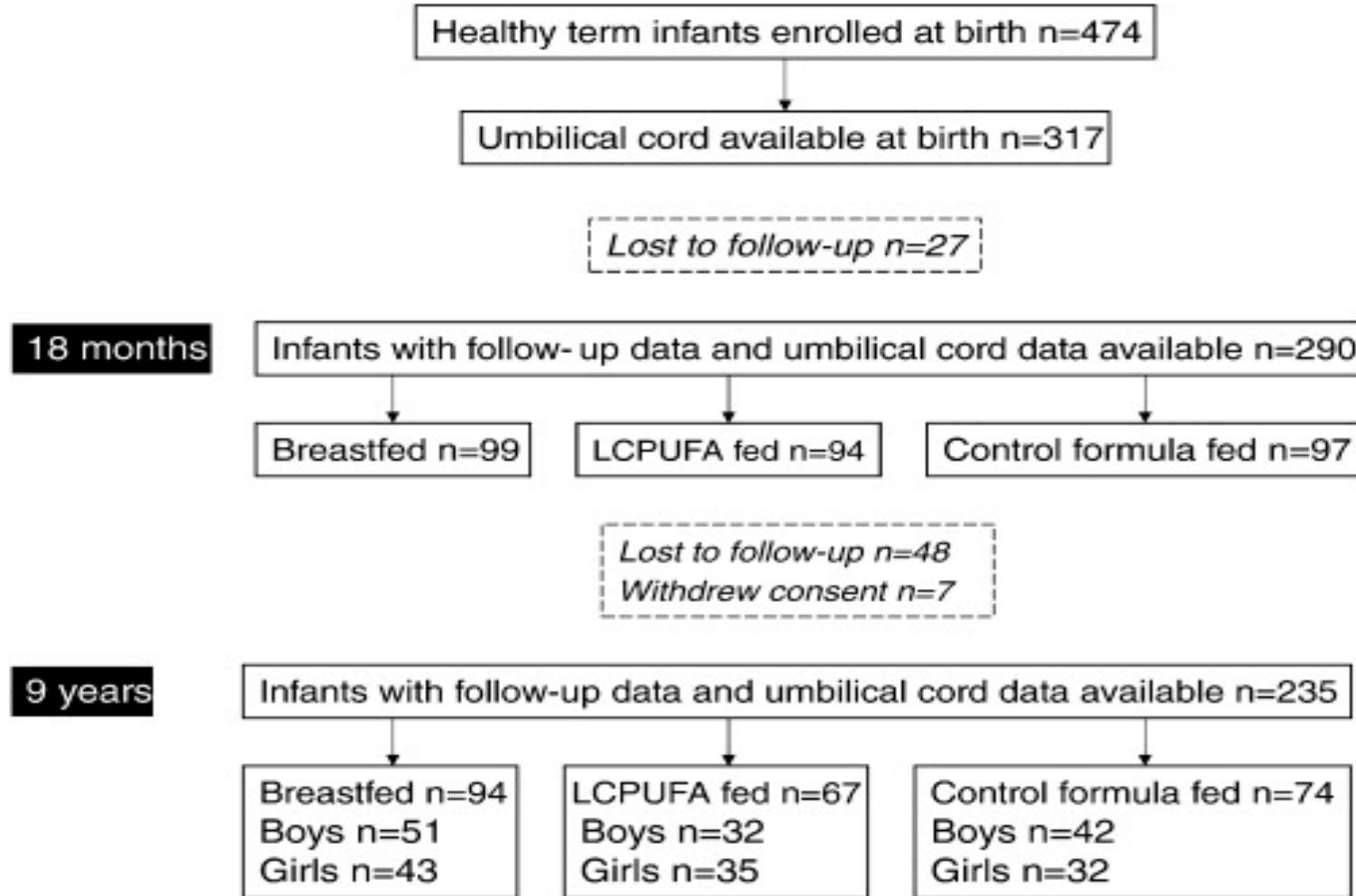
<sup>a</sup> University of Groningen, University Medical Center Groningen, Department of General Practice, Groningen, The Netherlands

<sup>b</sup> University of Groningen, University Medical Center Groningen, Department of Paediatrics, Institute of Developmental Neurology, Groningen, The Netherlands

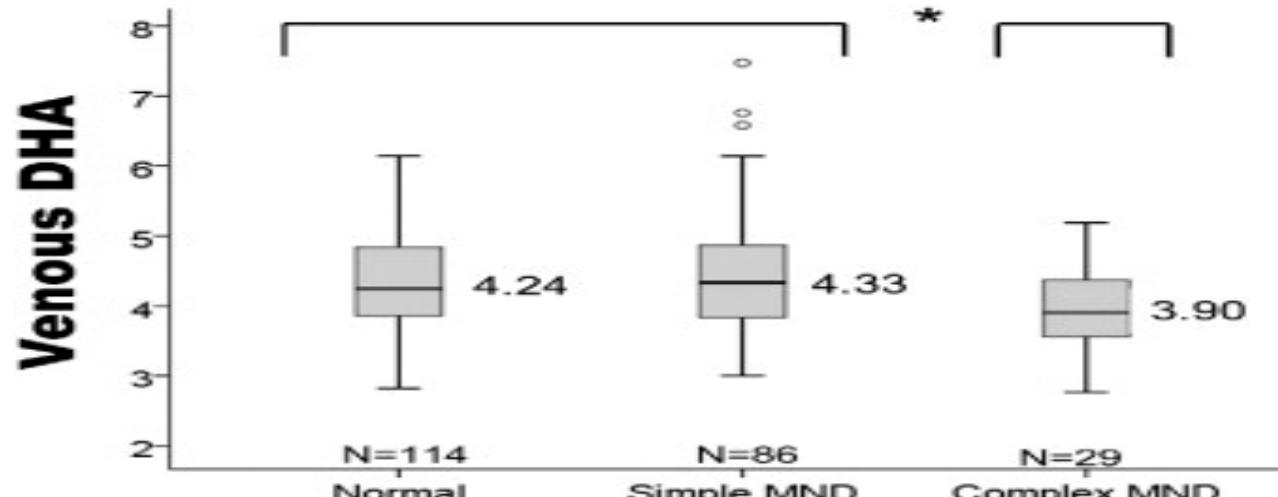
<sup>c</sup> Nutritional Science Consulting, Leipzig, Germany

<sup>d</sup> Department of Paediatrics, University Medical School of Pécs, Pécs, Hungary

# A vizsgálat menete



# DHA újszülöttkorban – neurológiai teszt eredménye 9 éves korban



**Neurological classification**

