Dokumenttyp: journal article

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Standl, M; Lattka, E; Stach, B; Koletzko, S; Bauer, CP; von Berg, A; Berdel, D; Krämer, U; Schaaf, B; Röder, S; Herbarth, O; Buyken, A; Drogies, T; Thiery, J; Koletzko, B; Heinrich, J; GINIplus Study Group; LISAplus Study Group; Heinrich, J; Wichmann, HE; Sausenthaler, S; Zutavern, A; Chen, CM; Schnappinger, M; Rzehak, P; Berdel, D; von Berg, A; Beckmann, C; Groß, I; Koletzko, S; Reinhardt, D; Krauss-Etschmann, S; Bauer, CP; Brockow, I; Grübl, A; Hoffmann, U; Krämer, U; Link, E; Cramer, C; Behrendt, H; Heinrich, J; Wichmann, HE; Sausenthaler, S; Chen, CM; Schnappinger, M; Borte, M; Diez, U; von Berg, A; Beckmann, C; Groß, I; Schaad, B; Lehmann, I; Bauer, M; Gräbsch, C; Röder, S; Schilde, M; Herbarth, O; Dick, C; Magnus, J; Krämer, U; Link, E; Cramer, C; Bauer, CP; Hoffmann, U; Behrendt, H; Grosch, J; Martin, F

Titel des Beitrags:
FADS1 FADS2 gene cluster, PUFA intake and blood lipids in children: results from the GINIplus and LISAplus studies.

Abstract:
Elevated cholesterol levels in children can be a risk factor for cardiovascular diseases in later life. In adults, it has been shown that blood lipid levels are strongly influenced by polymorphisms in the fatty acid desaturase (FADS) gene cluster in addition to nutritional and other exogenous and endogenous determinants. Our aim was to investigate whether lipid levels are determined by the FADS genotype already in children and whether this association interacts with dietary intake of n-3 fatty acids. The analysis was based on data of 2006 children from two German prospective birth cohort studies. Total cholesterol, HDL,
LDL and triglycerides were measured at 10 years of age. Six single nucleotide polymorphisms (SNPs) of the FADS gene cluster were genotyped. Dietary n-3 fatty acid intake was assessed by food frequency questionnaire. Linear regression modeling was used to assess the association between lipid levels, n-3 fatty acid intake and FADS genotype. Individuals carrying the homozygous minor allele had lower levels of total cholesterol [means ratio (MR) ranging from 0.96 (p = 0.0093) to 0.98 (p = 0.2949), depending on SNPs] and LDL [MR between 0.94 (p = 0.0179) and 0.97 (p = 0.2963)] compared to homozygous major allele carriers. Carriers of the heterozygous allele showed lower HDL levels [? between -0.04 (p = 0.0074) to -0.01 (p = 0.3318)] and higher triglyceride levels [MR ranging from 1.06 (p = 0.0065) to 1.07 (p = 0.0028)] compared to homozygous major allele carriers. A higher n-3 PUFA intake was associated with higher concentrations of total cholesterol, LDL, HDL and lower triglyceride levels, but these associations did not interact with the FADS1 FADS2 genotype. Total cholesterol, HDL, LDL and triglyceride concentrations may be influenced by the FADS1 FADS2 genotype already in 10 year old children. Genetically determined blood lipid levels during childhood might differentially predispose individuals to the development of cardiovascular diseases later in life.

Zeitschriftenartikel / Abkürzung:
PLoS ONE

Jahr:
2012

Band:
7

Heft / Issue:
5

Seiten:
e37780

Sprache:
eng

Pubmed:

TUM Einrichtung:
Kinderklinik und Poliklinik; r Molekulare Allergologie und Umweltforschung

Occurences:
- Einrichtungen > Fakultäten > Fakultät für Medizin > Kliniken und Institute > Molekulare Allergologie > Molekulare Allergologie (Prof. Schmidt-Weber) > 2012
- Einrichtungen > Fakultäten > Fakultät für Medizin > Kliniken und Institute > Klinik und Poliklinik für Kinderheilkunde und Jugendmedizin > 2012

entries: