IMPACT OF DIETARY CHOLINE ON HOMOCYSTEINE METABOLISM IN ATHEROSCLEROSIS PRONE MICE

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Aim. Scientific evidence revealed that a positive correlation exists between cardiovascular risk and plasma levels of TMAO, a product of dietary choline metabolism. This study was aimed at investigating whether dietary choline affects additional metabolic pathways besides that leading to TMAO production.
Methods. Ten-week-old EKO female mice were fed for 16 weeks two standard rodent diets differing for a low (0.09%) or high (1.2%) choline content. Atherosclerosis development was quantified at the aortic sinus, targeted plasma metabolomic and hepatic gene expression were performed. Additionally, in vitro experiments on HepG2 cells were set up to elucidate the mechanism by which choline alters plasma metabolome.
Results. High choline intake was associated with greater atherosclerosis development and increased plasma levels of TMAO. Interestingly, high choline feeding was associated with lower plasma levels of homocysteine and a concomitant increase of its related metabolites, methionine, sarcosine and glycine. Hepatic gene expression of Aldh7a1, Slc44a1, Sardh and Gnmt was increased in EKO mice fed high-choline diet, supporting the metabolic findings. In vitro experiments showed that several pathways are devoted to homocysteine metabolism and can be mutually regulated by acting on enzymes belonging to different synthetic routes.
Conclusions. Our data confirm that an increased dietary intake of choline worsens atherosclerosis burden and leads to increased plasma TMAO levels. Interestingly, choline intake also modulates metabolic processes affecting plasma concentrations of homocysteine as well as methionine, sarcosine, and glycine. These observations offer new insights into the understanding of how choline might influence atherosclerosis development and modify cardiovascular risk.

TYPE 2 DIABETES MELLITUS IS ASSOCIATED WITH INCREASED RISKS OF LOW HANDGRIP STRENGTH AND SARCOPENIA IN ADULTS

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Background. Type 2 diabetes mellitus (T2DM) is steadily increasing worldwide. T2DM has beenidentified as a contributing factor to sarcopenia. Recent evidence, on Asian populations, suggeststhat individuals with diabetes have a higher risk of developing sarcopenia than those withoutdiabetes. However, there has been limited research conducted in European populations. Theprimary objective was to determine the prevalence of sarcopenia and investigate the associationsbetween T2DM and sarcopenia in European adults.
Methods. This retrospective cross-sectional study included 356 individuals aged ≥50 years. Bioelectrical impedance analysis andhandgrip dynamometry were employed to measure appendicular skeletalmuscle mass and handgrip strength (HGS), respectively. Sarcopenia wasdefined according to the criteria outlined in the EWGSOP2 guidelines,which involve the presence of low HGS (< 16 kg for women and < 27 kg formen) combined with low appendicular skeletal muscle mass (< 15 kg inwomen and < 20 kg in men). T2DM was diagnosed based on a fasting bloodglucose concentration of ≥126 mg/dL or the administration ofantidiabetic treatment.
Results. The mean age of the participants was 69±7 years, with 39% being male. The prevalenceof T2DM was 22%, and 12% were treated with hypoglycemic drugs. The overall prevalence ofsarcopenia among European participants was 9%. Participants with T2DM exhibited a significantlyhigher prevalence of low HGS (32% vs. 18%, p-adjusted=0.008), as well as sarcopenia (15% vs. 7%, p-adjusted=0.02) compared to those without T2DM. Multinomial logistic regression analysisrevealed that T2DM was associated with increased odds of having low HGS (OR=2.60; 95% CI=0.99-6.87) and sarcopenia (OR=6.38, 95% CI=1.63-24.9).
Conclusions. Older European adults with diabetes face a significantly higher risk of developinglow HGS and sarcopenia compared to their non-diabetic counterparts. This study confirms thatT2DM is an important influencing factor in the development of sarcopenia.