Gut microbiota-derived trimethylamine-N-oxide: a bridge between dietary fatty acid and cardiovascular disease?

ABSTRACT

Cardiovascular disease (CVD) is a serious disease that endangers human health and is one of the leading causes of death. Recent studies have reported that gut microbiota plays an important role in the development of CVD, especially its metabolite trimethylamine-N-oxide (TMAO). Dietary precursors, such as choline, L-carnitine, phosphatidylcholine and betaine were metabolized to trimethylamine (TMA) under the action of gut microbiota, and subsequently oxidized by hepatic flavin monooxygenases (FMOs) to form TMAO. Dietary fat is one of three major nutrients in food, has been found to have a positive or negative effect on the development of CVD. Multiple clinical and experimental evidences suggested that dietary fatty acids (FAs) can affect TMAO production through gut microbiota and/or FMO3 enzyme activity. This article summarizes the existing gut microbiota-mediated reduction of TMA, discusses the molecular mechanism of dietary FAs in the pathobiology of CVD from the view of TMAO. Therefore, this review provides new insight into the association of dietary FAs and CVD, paving the way for dietary FAs therapy for CVD.

Keyword: Cardiovascular disease (CVD); Gut microbiota; Trimethylamine-N-oxide (TMAO); Flavin monooxygenases (FMOs); Dietary fatty acids (FAs)