LETTER TO THE EDITOR

Growing Epidemic of Nonalcoholic Fatty Liver Disease (NAFLD) in India: Time to Revisit the Diet

Sharmila Dudani[1], Kavita Tomar[2], Aditi Rawat[3]

Journal of Medical Academics (2023): 10.5005/jp-journals-11003-0126

Sir,

A rising prevalence of nonalcoholic fatty liver disease (NAFLD) in India is adding to the already high total burden of noncommunicable diseases. NAFLD is defined as the presence of macrovesicular steatosis in >5% of the hepatocytes. It encompasses simple steatosis, nonalcoholic steatohepatitis (NASH), and perisinusoidal fibrosis leading to cirrhosis. The global prevalence of NAFLD is estimated to be 25%, whereas, in adult Indians, it is reported between 6.7 and 55.1%.[1] Prevalence is higher in hospital-based studies (40.8%) and urban population (40.0%) vs community-based studies (28.2%) and rural population (29.2%). With the increasing prevalence of hypertension, metabolic syndrome, obesity, type 2 diabetes mellitus (T2DM), polycystic ovarian disease, and hypothyroidism in India, the prevalence of NAFLD is expected to rise.

Most of the patients with NAFLD have simple steatosis; 10–30% of patients develop NASH, which is associated with hepatocellular injury and inflammation. Approximately 20–30% of patients with NASH ultimately develop cirrhosis which carries a significant risk of developing hepatocellular carcinoma.[2] NAFLD is usually asymptomatic, and diagnosis is usually incidental with abnormal liver enzymes and the presence of steatosis on imaging. It has been shown that almost 80% of subjects with central obesity and T2DM have evidence of NAFLD on imaging. The prevalence of metabolic syndrome and prediabetes increases with the rise in hepatic fat content and plays a role in the impairment of hepatic, muscle, and adipose sensitivity to insulin in obese adolescents. NAFLD is challenging as it does not have any standardized, widely accepted medical treatments, and thus dietary changes and lifestyle modifications are critical in the prevention and progression of the disease. The pathophysiology of NAFLD is complex, involving oxidative stress with the generation of reactive oxygen species (ROS) and inflammation.[3]

Dietary habits have changed dramatically in the last 50 years in Western countries, and a similar change has been mirrored in the Indian diet as well. Indian diets are predominantly plant-based vegetarian with low intake of animal fats. Though the dietary allowance for fat is standardized and conforms to global standards, the content of n-6 and n-3 fatty acids is often overlooked. n-6 and n-3 polyunsaturated fatty acids (PUFA) are essential fatty acids as they are not synthesized by the body and need to be provided in the diet. These two classes of PUFA are metabolically and functionally distinct and not interchangeable. The most common n-6 PUFA is linoleic acid (LA; 18:3 n-6), derived mainly from seed oils (safflower and sunflower). The most common n-3 PUFA is α-linolenic acid (ALNA; 18:3 n-3) found in green leafy vegetables (fenugreek and mustard) and certain nuts like walnuts. Long chain n-3 PUFA, namely eicosapentaenoic acid (EPA; 20:5 n-3) and docosahexaenoic acid (DHA; 22:6 n-3) are also deficient as intake of fatty fish (salmon and mackerel) is limited in most of the population. The optimal n-6 to n-3 ratio is 1:1, and a ratio of upto 4:1 is acceptable. ALNA can be converted to long-chain n-3 PUFA, namely EPA and DHA, in the body by the enzyme Δ-6 desaturase. However, this conversion is impaired in the presence of a high n-6:n-3 ratio. The increasing use of n-6 PUFA as a cooking medium in Indian diets, replacing the traditional fats, namely ghee (clarified butter), coconut oil, butter, and mustard oil, has led to a very high n-6:n-3 ratio, that is 100:1 with an adverse metabolic profile. There is growing evidence that the intake of n-6 PUFA plays a central role in the pathogenesis of NAFLD. The intrahepatic fat in subjects with steatohepatitis is composed of an increased amount of n-6 PUFA.[4] Furthermore, a progressive increase in the n-6:n-3 ratio is observed from healthy controls to NAFLD to subjects with NASH.[3] Oxidized LA metabolites are associated with increased production of ROS and proinflammatory cytokines like tumor necrosis factor α. Studies have shown the beneficial effects of n-3 PUFA as they are anti-inflammatory, decrease *de novo* lipogenesis in hepatocytes, and reduce levels of serum triglyceride, along with improved insulin sensitivity.[3] The phantom of high cholesterol as a cause of heart disease has led to the disappearance of saturated fats from the Indian diet and replaced by n-6 PUFA-enriched seed oils. This has actually had a deleterious outcome on cardiac events, as evidenced by the Minnesota coronary experiment and the Sydney diet heart experiment, and possibly may be the cause behind not only NAFLD but the bludgeoning rise of obesity, metabolic syndrome, and cancer as well.[3]

There are limited observational studies on associations between n-3 and n-6 PUFA intake and NAFLD risk. Cross-sectional studies conducted in Israeli, Chinese, and United States populations observed a negative association between n-3 PUFA intake and NAFLD.[3]

It is time to revisit our dietary recommendations and national guidelines in view of the growing epidemic rise of

[1–3]Department of Pathology, Army College of Medical Sciences, Delhi, India

Corresponding Author: Sharmila Dudani, Department of Pathology, Army College of Medical Sciences, Delhi, India, Phone: +91 9811778156, e-mail: drsdudani@hotmail.com

How to cite this article: Dudani S, Tomar K, Rawat A. Growing Epidemic of Nonalcoholic Fatty Liver Disease (NAFLD) in India: Time to Revisit the Diet. J Med Acad 2023;6(1):40–41.

Source of support: Nil

Conflict of interest: None

noncommunicable diseases, including NAFLD. It may be prudent to trust the wisdom of our ancestors and include the age-old culinary practices as the traditional Indian diet had a low content of n-6 PUFA and an optimal n-6:n-3 ratio.

References

1. Shalimar, Elhence A, Bansal B, et al. Prevalence of non-alcoholic fatty liver disease in India: a systematic review and meta-analysis. J Clin Exp Hepatol 2022;12(3):818–829. DOI: 10.1016/j.jceh.2021.11.010
2. Cui J, Li L, Ren L, et al. Dietary n-3 and n-6 fatty acid intakes and NAFLD: A cross-sectional study in the United States. Asia Pac J Clin Nutr 2021;30(1):87–98. DOI: 10.6133/apjcn.202103_30(1).0011
3. Santoro N, Caprio S, Feldstein AE. Oxidized metabolites of linoleic acid as biomarkers of liver injury in nonalcoholic steatohepatitis. Clin Lipidol 2013;8(4):411–418. DOI: 10.2217/clp.13.39
4. Puri P, Baillie RA, Wiest MM, et al. A lipidomic analysis of nonalcoholic fatty liver disease. Hepatology 2007;46(4):1081–1090. DOI: 10.1002/hep.21763