Original Article

Burden of cardiovascular disease in China attributable to unbalanced fatty acid intake from 1990 to 2050

Xiangrong Liu BMed[1,2], Xinshen Zhang MD[1], Lu Liu MD[1], Zhimeng Yu BMed[1], Xin Qin BMed[1], Yetong Ma BMed[1,2], Xueyan Yang BMed[1], Yinghua Liu MD[1]

[1]*Department of Nutrition, the First Medical Center of the Chinese People's Liberation Army General Hospital, Beijing, China*
[2]*People's Liberation Army Postgraduate Medical School, Beijing, China*

Background and Objectives: This study aimed to analyze the trends in death and disability-adjusted life years (DALYs) due to cardiovascular disease (CVD) associated with unbalanced dietary fatty acid intake in China from 1990 to 2021, and to predict the disease burden levels up to 2050. **Methods and Study Design:** Using Global Burden of Disease 2021 data, we examined death and DALYs rates by age, sex, and risk factors. Joinpoint regression assessed temporal trends. Decomposition analysis evaluated contributions of population growth, aging, and epidemiological transitions. Age-period-cohort (APC) modeling estimated cohort and period effects. Bayesian Age-Period-Cohort (BAPC) modeling projected future CVD burden. **Results:** Age-standardized death and DALYs rates for CVD attributable to low seafood n-3 polyunsaturated fatty acids (PUFAs) and high trans fatty acids declined, while those due to insufficient n-6 PUFAs intake increased. Despite fluctuations, overall CVD burden showed a downward trend. Burden was higher in males and older groups. Population growth drove the absolute increase in burden, while aging and epidemiological shifts had variable effects by risk factor. APC modeling revealed significant age, period, and cohort influences. BAPC projections indicate continued decline in CVD burden from fatty acid imbalances through 2050. **Conclusions:** The CVD burden linked to imbalanced fatty acid consumption demonstrated a general decline, with forecasts suggesting a continued decrease by 2050. Older adults and males were highlighted as priority populations for focused interventions. These results can inform the development of targeted prevention and control initiatives.

Key Words: cardiovascular disease, n-3 polyunsaturated fatty acids, trans fatty acids, decomposition analysis, Bayesian Age-Period-Cohort modeling

INTRODUCTION

Unbalanced fatty acid intake referred to a dietary imbalance in the proportions of different types of fatty acids, including intakes of polyunsaturated fatty acids (PUFAs), omega-3 fatty acids, monounsaturated fatty acids (MUFAs), saturated fatty acids (SFAs), and trans fatty acids (TFAs) deviating from optimal levels, leading to an overall disruption of fatty acid homeostasis.[1,2] Unbalanced fatty acid intake is a global health issue, and its adverse effects involved multiple systems including the gut, metabolism, cardiovascular system, immune system, and nervous system. Excessive SFAs intake disrupts intestinal lipid metabolism and induces intestinal homeostasis imbalance, leading to intestinal inflammation and immune dysfunction.[3] Inadequate intake of PUFAs is also associated with cognitive decline.[4] Furthermore, unbalanced fatty acid intake, a significant contributor to the burden of cardiovascular disease (CVD), warrants attention. Overall, high intake of SFAs and TFAs was associated with increased incidence of atherosclerosis and CVD. Replacing part of SFAs or TFAs with MUFAs or PUFAs reduced the associated risk.[5] Among them, the combined effect of high TFAs intake and low n-3 PUFAs intake might have exacerbated the CVD burden, while the role of n-6 PUFAs needed to be evaluated in the context of specific metabolic backgrounds, such as the n-6/n-3 ratio.[6] Further research found that supplementation with n-3 PUFAs reduced the risk of death by lowering triglyceride levels, improving inflammatory status, and enhancing cardiovascular outcomes.[2,7] The pathogenic mechanisms of SFAs primarily promote CVD through pro-inflammatory, pro-oxidative, pro-atherosclerotic, and pro-thrombotic pathways. In contrast, the biological pathways of n-3 PUFAs improve CVD outcomes via anti-inflammatory and antioxidant effects, improved lipid profiles, and enhanced endothelial function. These pathways suggest that reducing SFAs intake and the n-6/n-3 ratio, while increasing n-3 PUFAs consumption, may be key strategies for the prevention and treatment of CVD.

Corresponding Author: Dr Yinghua Liu, Department of Nutrition, the First Medical Center of the Chinese People′s Liberation Army General Hospital, Beijing, China, No. 28 Fuxing Road, Haidian District, Beijing 100853, China
Tel: +86-66-937443; Fax: +86-66-937443
Email: liuyinghua77@163.com
Manuscript received 08 September 2025. Initial review completed 26 September 2025. Revision accepted 07 October 2025.
doi: 10.6133/apjcn.202602_35(1).0010

China, the world's most populous country, was particularly affected by the burden of CVD due to unbalanced fatty acid intake. The long-term inadequate intake of n-3 PUFAs in the Chinese population was significantly associated with the burden of ischemic heart disease (IHD). A study based on Global Burden of Disease (GBD) data using Age-Period-Cohort (APC) analysis indicated that insufficient intake of n-3 PUFAs was a significant risk factor for IHD. Long-term inadequate intake led to an increased burden of IHD, particularly among the elderly population, which was consistent with global patterns.[6] Levels of n-6 PUFAs in red blood cells were positively associated with the risk of coronary artery disease, but specific data for the Chinese population were lacking.[8] Similarly, although no literature directly reported TFAs data for China, it was noted that the mechanism linking western dietary patterns (high in TFAs) to CVD, which was spreading in developing countries, was applicable to China, especially against the backdrop of urbanization and dietary changes.[9,10]

The traditional Chinese dietary pattern, characterized by low seafood consumption, may lead to inadequate intake of PUFAs including n-3 PUFAs, while industrialization may have increased TFA intake. However, previous studies lacked direct data to explore their associations with CVD. This study aimed to conduct a comprehensive and in-depth investigation of the CVD burden in China attributable to unbalanced fatty acid intake from 1990 to 2021, using data from the GBD 2021 study. The disease burden was described in detail through stratification by age and sex. Joinpoint regression analysis, decomposition analysis, and the APC model were used to further investigate the underlying driving factors of the disease burden. At the same time, the Bayesian age-period-cohort (BAPC) model was used to predict potential trends up to 2050. These analytical results helped precisely identify high-risk populations for intervention and provided strong scientific support for current and future prevention and control of chronic diseases, improvement of national nutrition, and formulation of health policies, enabling more efficient allocation of public health resources.

METHODS

Data sources

All data utilized in this study were obtained from the GBD 2021 dataset, available through the GBD Results Tool (http://ghdx.healthdata.org/gbd-results-tool/). The GBD database serves as an extensive global health repository, systematically gathering epidemiological data—such as incidence, prevalence, death, years of life lost, years lived with disability, and disability-adjusted life years (DALYs)—for 371 diseases and injuries, as well as 88 key risk factors, across 204 countries and territories.In the 2021 GBD framework's assessment of risk factors, three were identified as significantly associated with CVD. These included diet low in seafood n-3 PUFAs, diet low in n-6 PUFAs and diet high in TFAs. This study used data from China and adopted two main indicators-death and DALYs- to assess the burden of CVD, both of which are reported with 95% uncertainty intervals (UI).

Statistical analysis

Temporal trends from 1990 to 2021 were analyzed using the estimated annual percentage change (EAPC) in age-standardized rate (ASR), enabling systematic comparisons across age groups and sexes.

Joinpoint regression analysis uses piecewise linear regression to adaptively model trends by fitting a series of connected line segments. The analysis starts with the most basic model—containing no joinpoints—and iteratively evaluates whether the inclusion of one or more joinpoints significantly enhances the model's fit, based on a Monte Carlo permutation test. For each resulting segment, the annual percentage change in age-standardized death rate (ASDR) and age-standardized DALYs is estimated. Furthermore, the average annual percentage change (AAPC) and its associated 95% confidence interval (CI) are calculated to provide a summary measure of the overall trend over the entire study period, from 1990 to 2021.[11]

Decomposition analysis was applied to assess the contributions of population growth, demographic aging, and changes in disease epidemiology to the overall disease burden.[12] This method allows the total burden to be broken down into the effects of specific factors, enabling the identification of the most influential drivers and the direction (increase or decrease) of their impact.

Furthermore, we applied an APC model to investigate the effects of age, period, and birth cohort on death and DALYs resulting from CVD attributable to unbalanced fatty acid intake. Key parameters in the APC model include net drift and local drift. Period effects capture temporal changes that uniformly impact all age groups, likely reflecting shifts in societal, cultural, economic, or environmental conditions. Cohort effects reflect variations among individuals born in the same year. Net drift quantifies the overall log-linear trend across periods and cohorts, representing the total annual percentage change in expected age-adjusted rates over time. Local drift, in contrast, measures the log-linear trend for each specific age group across periods and cohorts, indicating the annual percentage change in expected age-specific rates within that group over time.[13,14]

The BAPC model efficiently disentangles the distinct impacts of age, time period, and birth cohort on health outcomes, enabling a deeper understanding of generational health trends and the effects of public health interventions or policy changes. By employing integrated nested laplace approximations (INLA)—a computationally efficient Bayesian technique—the model avoids the time-consuming simulations required by conventional Markov chain Monte Carlo methods. INLA enables fast and accurate estimation of model parameters and predictions by approximating posterior distributions, making it particularly suitable for large-scale datasets. The BAPC model generates detailed predicted rates across different age groups and calculates ASR, thereby supporting meaningful comparisons of health outcomes across diverse populations.[15,16]

All statistical analyses and data visualizations were performed using R (version 4.4.2) and JD_GBDR (V2.37, jingding Medical Technology Co., Ltd). A p-value less than 0.05 was considered statistically significant. Ethical

approval was not required for this study, as it utilized publicly available data from the GBD database.

RESULTS

CVD burden attributable to unbalanced fatty acids intake

In China, deaths from CVD attributable to diet low in seafood n-3 PUFAs increased from 56,638.05 (95% UI: 11,739.17 to 94,407.26) in 1990 to 72,410.24 (95% UI: 13,118.63 to 133,501.94) in 2021. DALYs declined from 1,592,558.25 (95% UI: 340,659.83 to 2,656,747.45) to 1,428,781.49 (95% UI: 265,229.09 to 2,626,045.22) over the same period. About CVD attributable to diet low in n-6 PUFAs, deaths increased from 55,930.14 (95% UI: -170,764.58 to 212,352.64) in 1990 to 164,339.14 (95% UI: -447,086.45 to 649,796.96) in 2021. DALYs rose from 1,556,556.64 (95% UI: -5,115,429.42 to 5,822,314.84) to 3,388,149.94 (95% UI: -9,860,479.71 to 13,069,419.07). Deaths from CVD attributable to diet high in TFAs increased from 3,514.26 (95% UI: 411.46 to 7,564.10) in 1990 to 5,289.63 (95% UI: 546.88 to 11,916.05) in 2021. DALYs rose from 94,333.06 (95% UI: 10,858.80 to 200,343.06) to 102,189.44 (95% UI: 10,264.42 to 225,839.36) (Table 1).

For diet low in seafood n-3 PUFAs, the ASDR declined from 8.82 (95% UI: 1.80 to 14.80) per 100,000 population in 1990 to 4.12 (95% UI: 0.76 to 7.63) in 2021, with an EAPC of -2.17 (95% CI: -2.66 to -1.67). The age-standardized DALYs rate decreased from 190.45 (95% UI: 40.01 to 315.64) to 74.90 (95% UI: 13.91 to 135.90) per 100,000 population (EAPC: -2.80; 95% CI: -3.23 to -2.38). On the contrary, for diet low in n-6 PUFAs, the ASDR increased from 8.60 (95% UI: -24.94 to 33.04) per 100,000 population in 1990 to 9.06 (95% UI: -24.53 to 35.91) in 2021, with an EAPC of 0.59 (95% CI: 0.27 to 0.90). Correspondingly, for diet high in TFAs, the ASDR decreased from 0.57 (95% UI: 0.06 to 1.23) to 0.30 (95% UI: 0.03 to 0.68) per 100,000 population (EAPC: -2.70; 95% CI: -3.21 to -2.20). The age-standardized DALYs rate decreased from 11.66 (95% UI: 1.33 to 24.33) per 100,000 population to 5.27 (95% UI: 0.54 to 11.67) in 2021 (EAPC: -3.28; 95% CI: -3.76 to -2.79). (Table 2)

Gender and age stratification of CVD burden attributable to unbalanced fatty acids intake

The ASDR and age-standardized DALYs rate for CVD attributable to diet low in seafood n-3 PUFAs, diet low in n-6 PUFAs and diet high in TFAs increased with age (Figure 1). About CVD attributable to diet low in n-6 PUFAs and diet high in TFAs, across all age groups, males had higher the ASDR and age-standardized DALYs rate than females (Figure 1C-F). In contrast, about diet low in seafood n-3 PUFAs, across almost all age groups after 65 years of age, females were greater than males (Figure 1A-B). Similarly, after the age of 65, the number of females was greater than that of males (Figure 1A-B). In a diet low in n-6 PUFAs and a diet high in TFAs, the age at which the number of females exceeded that of males was 90 years (Figure 1C-F).

Joinpoint regression analysis

The Joinpoint regression model was used to analyze the data on CVD burden attributable to unbalanced fatty acids intake from 1990 to 2021, and the APC and AAPC were calculated. For diet low in seafood n-3 PUFAs, from 1990 to 1998, there was a decrease in ASDR (APC = -1.50). Between 1998 and 2004, there was a rapid increase in ASDR (APC = 2.93). From 2004 to 2011, the ASDR trended downward (APC = -3.57). From 2011 to 2021, the rate of decrease accelerated (APC = -5.40) (Figure 2A).The ASDR (AAPC = -0.15) and age-standardized DALYs rates (AAPC = -3.75) for CVD attributable to diet low in seafood n-3 PUFAs showed an decreasing trend over the period (Figure 2A and B). Similarly, for CVD attributable to diet high in TFAs, both the ASDR (AAPC = -0.01) and age-standardized DALYs rates (AAPC = -0.20) decreased over time (Figure 2E and F). However, for CVD attributable to diet low in n-6 PUFAs , the ASDR (AAPC = 0.01) showed an increasing trend (Figure 2C), while the age-standardized DALYs showed an overall decreasing trend (AAPC = -0.44) (Figure 2D).

Decomposition analysis

To evaluate how population growth, aging, and shifts in epidemiology contributed to trends in CVD linked to unbalanced fatty acids intake, we performed a decomposition analysis. Results indicated that population growth and aging were key drivers of the rising burden of CVD attributable to diet low in seafood n-3 PUFAs, diet low in n-6 PUFAs, while epidemiological changes partially offset this increase (Figure 3A-D). Aging was a major factor in reducing the burden of CVD attributable to diet high in TFAs (Figure 3E-F).

APC model analysis

The age effect of CVD burden attributable to unbalanced fatty acids intake exhibited the expected exponential distribution pattern. The period and cohort effects for the three risk factors showed similar trends. The CVD burden attributable to diet low in seafood n-3 PUFAs and diet low in n-6 PUFAs increased between 2000 and 2005, but decreased during all other periods (Figure 4A-D). The CVD burden attributable to a diet high in TFAs consistently decreased across all periods (Figure 4E-F). Among birth cohorts, the CVD burden attributable to a diet low in seafood n-3 PUFAs and a diet high in TFAs continuously improved. In contrast, the CVD burden attributable to a diet low in n-6 PUFAs increased among individuals born before 1940, but decreased thereafter (Figure 4C-D).

BAPC model prediction

Using the BAPC model, we projected deaths and DALYs attributable to diet low in seafood n-3 PUFAs, diet low in n-6 PUFAs and diet high in TFAs for CVD and age specific types to 2050. In China, both ASDR and age-standardized DALYs rate for CVD exhibited a general decline (Figure 5). The results across all age groups also showed a similar downward trend (Figure 6).

Table 1. Numbers of deaths, DALYs of CVD attributable to unbalanced fatty acids intake in 1990 and 2021 in China

	Sex	Number of Deaths (95%UI)		Number of DALYs (95%UI)	
		1990	2021	1990	2021
Diet low in sea-food n-3 PUFAs	Both	56,638.05 (11,739.17, 94,407.26)	72,410.24 (13,118.63, 133,501.94)	1,592,558.25 (340,659.83, 2,656,747.45)	1,428,781.49 (265,229.09, 2,626,045.22)
	Female	29,314.00 (6,321.53, 50,554.72)	40,311.83 (6,856.05, 76,325.35)	761,230.67 (169,519.36, 1,272,330.32)	715,853.06 (124,462.46, 1,356,005.83)
	Male	27,324.05 (5,590.58, 47,861.59)	32,098.42 (6,143.98, 62,802.63)	831,327.58 (176,033.46, 1,460,140.12)	712,928.42 (139,528.61, 1,369,834.08)
Diet low in n-6 PUFAs	Both	55,930.14 (-170,764.58, 212,352.64)	164,339.14 (-447,086.45, 649,796.96)	1,556,556.64 (-5,115,429.42, 5,822,314.84)	3,388,149.94 (-9,860,479.71, 13,069,419.07)
	Female	26,171.61 (-75,656.45, 100,251.74)	70,588.11 (-187,335.00, 278,783.69)	669,240.67 (-2,075,267.31, 2,527,686.95)	1,274,356.07 (-3,734,121.45, 5,052,567.23)
	Male	29,758.53 (-90,480.44, 114,357.33)	93,751.03 (-249,009.50, 374,287.75)	887,315.96 (-2,954,779.51, 3,399,827.62)	2,113,793.87 (-6,108,065.32, 8,386,235.93)
Diet high in TFAs	Both	3,514.26 (411.46, 7,564.10)	5,289.63 (546.88, 11,916.05)	94,333.06 (10,858.80, 200,343.06)	102,189.44 (10,264.42, 225,839.36)
	Female	1,658.01 (159.96, 3,824.94)	2,313.63 (192.02, 5,506.54)	40,891.95 (4,082.77, 92,499.89)	39,025.18 (3,483.37, 92,626.77)
	Male	1,856.25 (211.30, 4,066.81)	2,976.00 (258.57, 7,317.80)	53,441.12 (6,337.54, 117,257.56)	63,164.26 (5,364.93, 149,659.37)

UI, uncertainty intervals; DALYs, disability-adjusted life years; CVD, cardiovascular diseases; PUFAs, polyunsaturated fatty acids; TFAs, trans fatty acids.

Table 2. ASDR and age-standardised DALYs rates of CVD attributable to unbalanced fatty acids intake and their temporal trend from 1990 to 2021 in China

	Sex	Death (95%UI)			DALYs (95%UI)		
		ASR in 1990 (per 100,000 population)	ASR in 2021 (per 100,000 population)	EAPC (1990-2021)	ASR in 1990 (per 100,000 population)	ASR in 2021 (per 100,000 population)	EAPC (1990-2021)
Diet low in sea-food n-3 PUFAs	Both	8.82 (1.80,14.80)	4.12 (0.76, 7.63)	-2.17 (-2.66, -1.67)	190.45 (40.01, 315.64)	74.90 (13.91,135.90)	-2.80 (-3.23, -2.38)
	Female	8.58 (1.83,14.85)	3.99 (0.68, 7.66)	-2.15 (-2.61, -1.68)	181.73 (39.86, 307.06)	68.84 (12.02,129.29)	-2.90 (-3.30, -2.50)
	Male	9.21 (1.80,16.05)	4.29 (0.81, 8.61)	-2.20 (-2.75, -1.66)	199.74 (40.82, 345.62)	81.05 (15.67,154.13)	-2.72 (-3.18,-2.25)
Diet low in n-6 PUFAs	Both	8.60 (-24.94,33.04)	9.06 (-24.53, 35.91)	0.59 (0.27, 0.90)	186.26 (-582.33, 706.47)	172.83 (-505.67, 669.37)	0.09 (-0.15, 0.33)
	Female	7.64 (-20.96,29.15)	6.92 (-18.22, 27.21)	0.09 (-0.23, 0.40)	160.54 (-482.20, 608.13)	121.00 (-355.45, 479.43)	-0.61 (-0.86, -0.36)
	Male	9.98 (-28.07,38.39)	12.08 (-31.78, 47.95)	1.04 (0.70, 1.37)	215.04 (-660.52, 820.42)	232.22 (-660.35, 922.71)	0.60 (0.35, 0.85)
Diet high in TFAs	Both	0.57 (0.06,1.23)	0.30 (0.03, 0.68)	-2.70 (-3.21, -2.20)	11.66 (1.33, 24.33)	5.27 (0.54, 11.67)	-3.28 (-3.76, -2.79)
	Female	0.50 (0.05,1.10)	0.23 (0.02, 0.55)	-3.11 (-3.62, -2.59)	10.07 (1.02, 22.09)	3.73 (0.32, 8.91)	-3.87 (-4.38, -3.37)
	Male	0.67 (0.07,1.46)	0.40 (0.04, 0.95)	-2.33 (-2.83, -1.82)	13.53 (1.52, 29.03)	7.09 (0.62, 16.49)	-2.83 (-3.31, -2.35)

UI, uncertainty intervals; DALYs, disability-adjusted life years; ASR, age-standardized rate; EAPC, estimated annual percentage change; CVD, cardiovascular diseases; PUFAs, polyunsaturated fatty acids; TFAs, trans fatty acids.

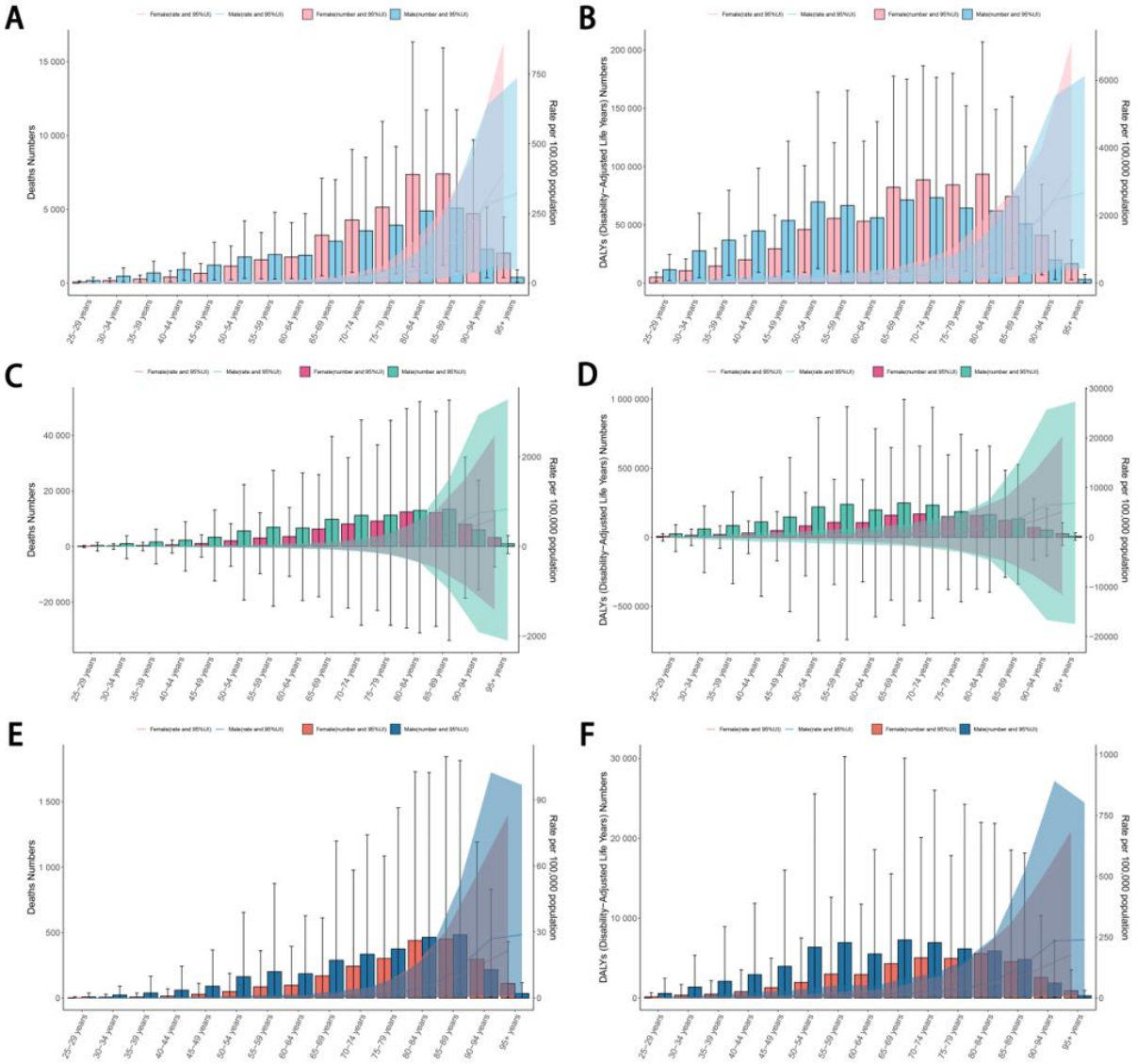

Figure 1. In China, the ASDR and age-standardized DALYs rate per 100,000 people of CVD attributable to diet low in seafood n-3 PUFAs (A-B), diet low in n-6 PUFAs (C-D) and diet high in TFAs (E-F) by age and sex in 2021. ASDR, age-standardized death rate; DALYs, disability-adjusted life years; CVD, cardiovascular diseases; PUFAs, polyunsaturated fatty acids; TFAs, trans fatty acids

DISCUSSION

Based on the latest data from the 2021 GBD study, we systematically analyzed the impact of CVD attributable to unbalanced fatty acid intake on death and DALYs in China between 1990 and 2021, and projected the epidemiological trends to 2050. The study found that the CVD ASDR and age-standardized DALYs rates attributable to a diet low in seafood n-3 PUFAs and high in TFAs both showed a decreasing trend, while those attributable to a diet low in n-6 PUFAs showed an increasing trend. Joinpoint regression further revealed that the CVD burden attributable to unbalanced fatty acid intake fluctuated over time but showed an overall decreasing trend. In addition, the disease burden was particularly pronounced among males and the elderly population. Decomposition analysis indicated that population growth was the main driver of the increase in CVD burden, while epidemiological changes and population aging played different roles across the various risk factors. Using the APC model, we explored the relationship between CVD burden and age, period, and cohort effects. According to the BAPC model, projections for 2050 indicate that the CVD burden attributable to unbalanced fatty acid intake will be further reduced.

Hu JJ et al. found that males were more susceptible to the effects of unbalanced fatty acid intake than females, particularly evident in younger age groups,[1] which was consistent with our findings. This might have been related to males dietary habits, which tended to include higher intake of SFAs and TFAs.[17,18] Gender differences in fat oxidation preference during physical activity were observed, while gut microbiota composition, sex hormone regulation, and exposure to environmental toxins were considered potential biological amplification mechanisms. Furthermore, due to aging, the elderly experienced a decline in fatty acid metabolism capacity, making them more susceptible to energy metabolism disorders, oxidative stress, and inflammatory responses caused by unbalanced fatty acid intake, thereby further increasing the risk of CVD.[19,20] In the Chinese population's diet, intake of n-3

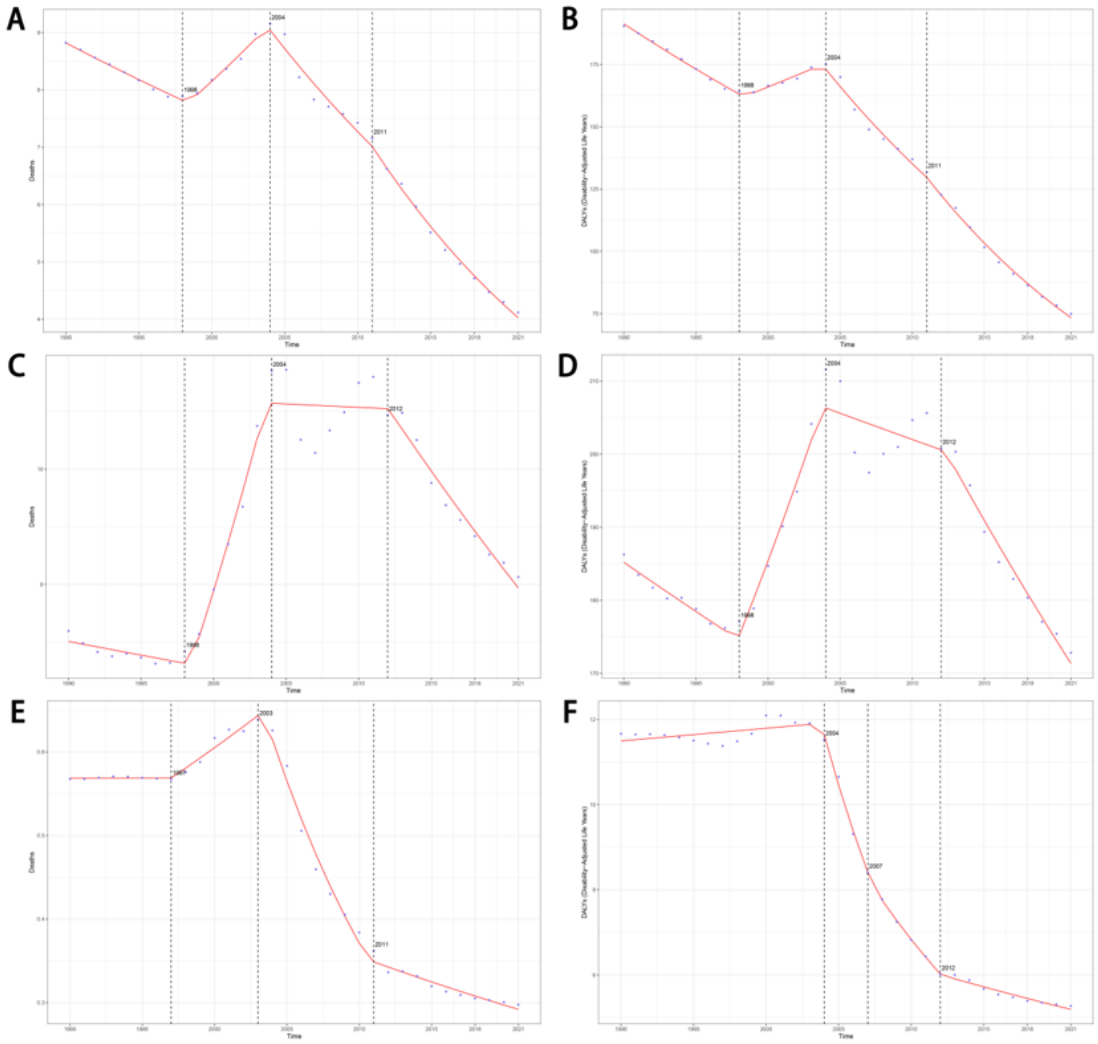

Figure 2. In China, the trend of ASDR and age-standardized DALYs rate for CVD attributable to diet low in seafood n-3 PUFAs (A-B), diet low in n-6 PUFAs (C-D) and diet high in TFAs (E-F) from 1990 to 2021. ASDR, age-standardized death rate; DALYs, disability-adjusted life years; CVD, cardiovascular diseases; PUFAs, polyunsaturated fatty acids; TFAs, trans fatty acids

PUFAs was generally inadequate, while intake of SFAs and TFAs was relatively high.[1,6] This unbalanced fatty acid intake promoted atherosclerosis, dyslipidemia, and myocardial energy metabolism disorders.[17,19] The elderly faced higher risks because their dietary patterns were relatively fixed, making it more difficult to adjust their dietary structure.[21] The CVD effects of n-6 PUFAs were found to be highly dependent on the balance of its ratio to n-3 PUFAs, rather than on its individual action. In modern diets, the n-6/n-3 ratio was significantly elevated, reaching as high as 16:1, which far exceeded the evolutionary adapted ratio of 1–2:1. This elevated ratio promoted a pro-thrombotic and pro-inflammatory state and was associated with an increased risk of CVD.

The CVD burden in China attributable to unbalanced fatty acid intake declined rapidly around 2004 and 2011. This may have been related to the period of rapid economic growth in China, during which residents' dietary patterns might have shifted from high SFAs intake toward a more balanced nutritional profile.[22] A study on the burden of IHD in the Chinese population revealed that the IHD burden declined during the 2000s, partly due to public health interventions, including nutritional supplementation, health education, and promotion of dietary diversity, which improved n-3 PUFAs intake and thereby reduced lipid accumulation and inflammation-related CVD risks.[6]

In China, from 1990 to 2021, the CVD burden attributable to high TFAs intake was influenced by epidemiological changes and population growth as risk factors, while population aging acted as a protective factor. Regarding epidemiology, since the 1990s, China has undergone rapid urbanization and dietary westernization, characterized by increased consumption of processed foods, which directly heightened exposure to TFAs. The widespread use of frying in home cooking led to the formation of TFAs, while industrial hydrogenation processes introduced high levels of TFAs into foods, both of which elevated the risk of CVD.[23,24] Another study specifically analyzed the longitudinal changes in dietary quality in China from 1997 to 2006: the dietary pattern characterized by high TFAs and low PUFAs intake significantly increased.[25,26] This shift from a traditional low-TFAs, plant-based diet to a high-fat, high-TFAs diet was a key risk factor for the CVD burden.[27,28] From a regional perspective, provincial-level data in China showed that this change was markedly

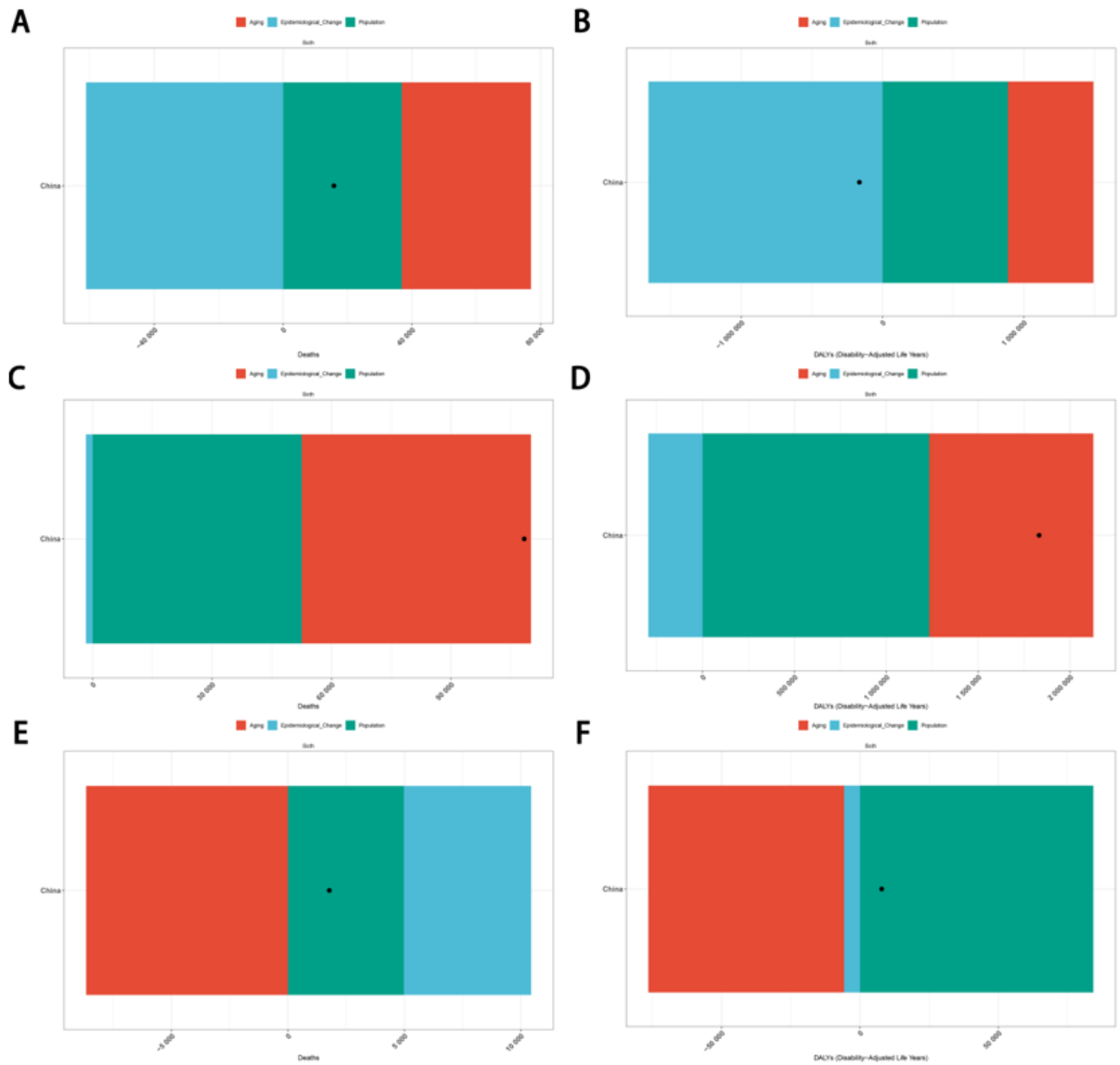

Figure 3. In China, decomposition analysis of the trends in CVD attributable to diet low in seafood n-3 PUFAs (A-B), diet low in n-6 PUFAs (C-D) and diet high in TFAs (E-F) ASDR and age-standardized DALYs rate from 1990 to 2021. CVD, cardiovascular diseases; PUFAs, polyunsaturated fatty acids; TFAs, trans fatty acids; ASDR, age-standardized death rate; DALYs, disability-adjusted life years

different between urban and rural areas, with a higher burden in urban areas, reflecting the risks of dietary inequity.[29] Population growth may be considered a risk factor, as China rapid population expansion has increased the number of people exposed to high-TFAs environments.. A study based on GBD data found that population growth, combined with dietary risk factors, drove up the absolute deaths rate of CVD. The absolute number of CVD deaths attributable to diet increased partly due to overall population growth.[1,24] Furthermore, industrialization and urbanization accompanying population growth enhanced the accessibility of foods high in TFAs, leading more people to consume excessive amounts of TFAs, thereby increasing the risk of myocardial infarction and death.[30,31] Interestingly, aging emerged as a protective factor, and the reasons were multifaceted. First, the dietary patterns of older adults were relatively stable and less influenced by processed foods. Second, the survival effect meant that individuals who lived to old age tended to have healthier lifestyles. Additionally, age-related metabolic changes might have reduced sensitivity to certain dietary fats.[32] Finally, the older generation might have had a survival advantage due to earlier exposure to lower levels of environmental risk factors, such as industrial trifluoromethane. This advantage might also have been related to the fact that individuals who survived into old age inherently possessed greater resilience to risks.[33]

The study also had several limitations. It relied entirely on estimated data from the GBD 2021 database rather than original individual-level data, with the GBD estimates carrying uncertainty influenced by input data quality, model assumptions, and statistical methods. Particularly for a vast and dietarily diverse country like China, national average estimates might have masked important regional, urban-rural, or socioeconomic disparities. Additionally, the study analyzed diet low in n-6 PUFAs as an independent risk factor, but existing evidence suggests the health effects of n-6 PUFAs might be closely related

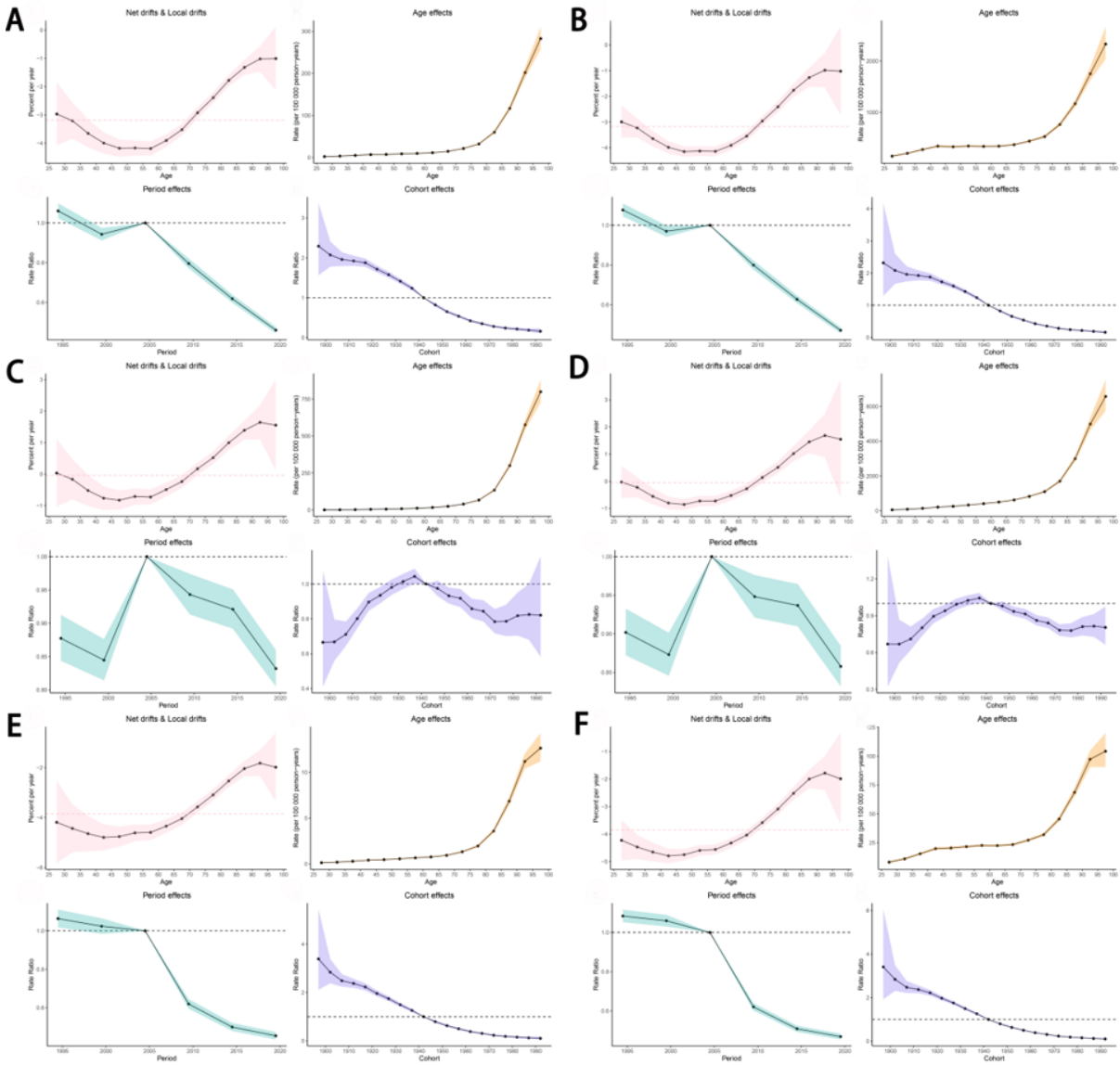

Figure 4. In China, Age-Period-Cohort analysis of CVD burden attributable to diet low in seafood n-3 PUFAs (A-B), diet low in n-6 PUFAs (C-D) and diet high in TFAs (E-F). CVD, cardiovascular diseases; PUFAs, polyunsaturated fatty acids; TFAs, trans fatty acids

to the n-6/n-3 ratio, so analyzing inadequate intake alone might not have fully captured their complex biological effects, especially within the Chinese population where relevant research evidence was relatively weak. Furthermore, the study focused on describing macro-level trends and attributable burdens without delving deeply into the specific biological mechanisms by which fatty acid intake influenced CVD or sufficiently analyzing the combined effects of substitution or synergy among different fatty acids on the overall disease burden.

Conclusion

From 1990 to 2021, the CVD burden in China attributable to unbalanced fatty acid intake showed an overall decreasing trend, and future projections indicate a further reduction by 2050. Elderly and male populations were identified as key groups requiring targeted interventions. Population growth consistently exacerbated the disease burden, while epidemiological changes and population aging played varying roles across different risk factors. These findings highlight the importance of continuously optimizing dietary fat composition. It is recommended that public health agencies and healthcare professionals, while promoting increased n-3 PUFAs intake and reduced TFAs consumption, develop precise nutritional intervention strategies for high-risk populations to more effectively alleviate the burden of CVD.

ACKNOWLEDGEMENTS

We appreciate the works by the Global Burden of Disease study 2021 collaborators.

CONFLICT OF INTEREST AND FUNDING DISCLOSURES

The authors declare no conflict of interest.

This research was funded by the Research Project on High-Quality Development of Clinical Nutrition Services (No.2025-1-Z-01).

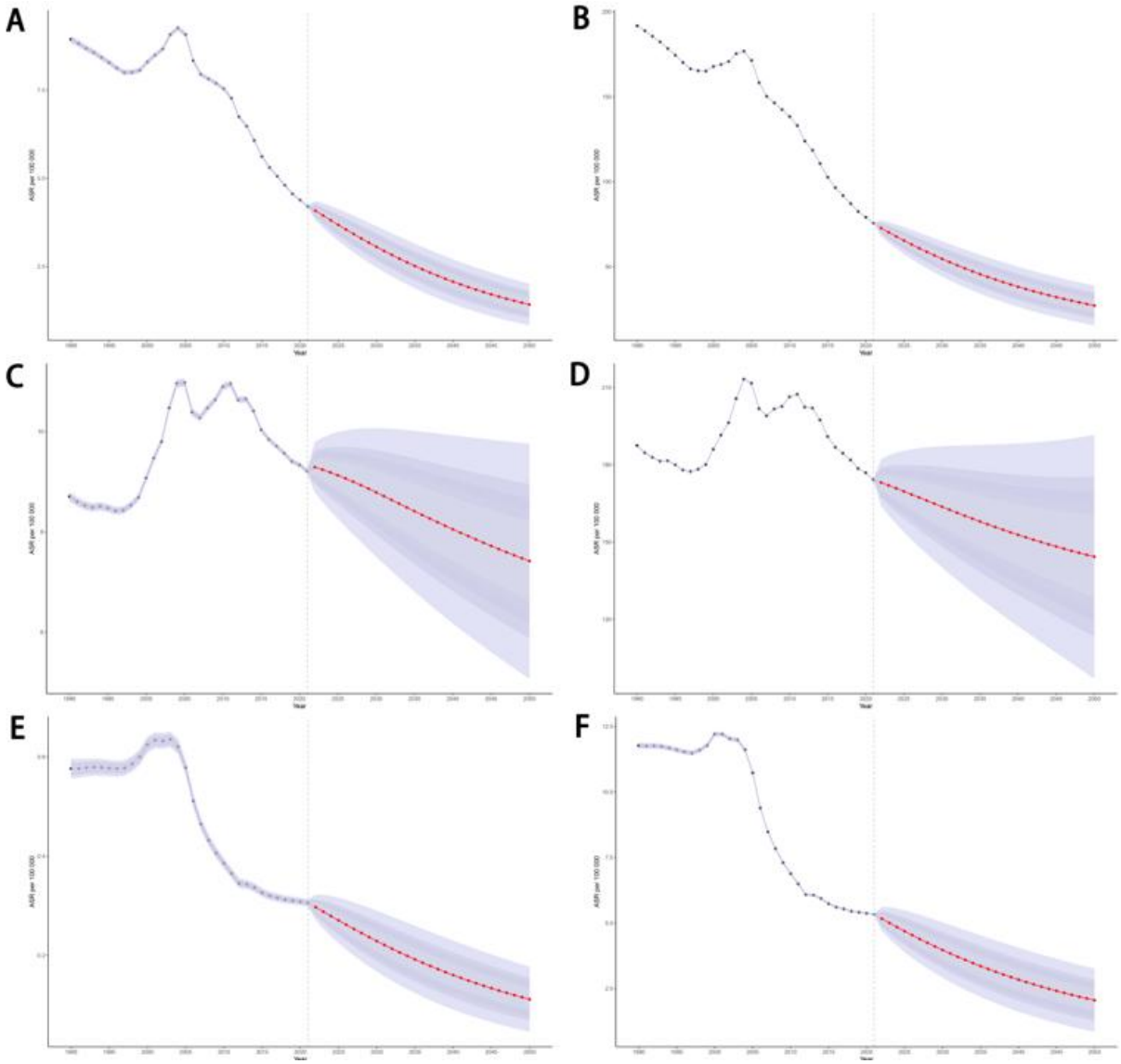

Figure 5. In China, projections of ASDR and age-standardized DALYs rate per 100,000 population of CVD attributable to diet low in seafood n-3 PUFAs (A-B), diet low in n-6 PUFAs (C-D) and diet high in TFAs (E-F) by 2050. ASDR, age-standardized death rate; DALYs, disability-adjusted life years; CVD, cardiovascular diseases; PUFAs, polyunsaturated fatty acids; TFAs, trans fatty acids

REFERENCES

1. Hu JJ, Dong YM, Ding R, Yang JC, Odkhuu E, Zhang L et al. Health burden of unbalanced fatty acids intake from 1990 to 2019: A global analysis. Med. 2023;4(11):778-796.e3. doi: 10.1016/j.medj.2023.08.002.
2. Ivey KL, Nguyen XT, Li R, Furtado J, Cho K, Gaziano JM et al. Association of dietary fatty acids with the risk of atherosclerotic cardiovascular disease in a prospective cohort of United States veterans. Am J Clin Nutr. 2023;118:763-72. doi: 10.1016/j.ajcnut.2023.07.014.
3. Fang W, Liu Y, Chen Q, Xu D, Liu Q, Cao X et al. Palmitic acid induces intestinal lipid metabolism disorder, endoplasmic reticulum stress and inflammation by affecting phosphatidylethanolamine content in large yellow croaker Larimichthys crocea. Front Immunol. 2022;13:984508. doi: 10.3389/fimmu.2022.984508.
4. T V, Muthu A. A perspective of microalga-derived omega-3 fatty acids: scale up and engineering challenges. Crit Rev Food Sci Nutr. 2025:1-15. doi: 10.1080/10408398.2025.2494060.
5. Du HX, Yue SY, Niu D, Liu C, Zhang LG, Chen J et al. Gut Microflora Modulates Th17/Treg Cell Differentiation in Experimental Autoimmune Prostatitis via the Short-Chain Fatty Acid Propionate. Front Immunol. 2022;13:915218. doi: 10.3389/fimmu.2022.915218.
6. Luo L. Ischemic heart disease burden attributable to inadequate omega-3 fatty acid intake in Chinese adults, 1990-2021: an Age-Period-Cohort analysis of the Global Burden of Disease study. Front Nutr. 2025;12:1590278. doi:10.3389/fnut.2025.1590278
7. Zeng F, Heier C, Yu Q, Pang H, Huang F, Zhao Z et al. Human transmembrane protein 68 links triacylglycerol synthesis to membrane lipid homeostasis. FEBS J. 2025;292:2935-52. doi: 10.1111/febs.70044.
8. Ren XL, Liu Y, Chu WJ, Li ZW, Zhang SS, Zhou ZL et al. Blood levels of omega-6 fatty acids and coronary heart disease: a systematic review and metaanalysis of observational epidemiology. Crit Rev Food Sci Nutr. 2023;63:7983-95. doi: 10.1080/10408398.2022.2056867.
9. Zhang Q, Zhang L, Chen C, Li P, Lu B. The gut microbiota-artery axis: A bridge between dietary lipids and atheroscle-

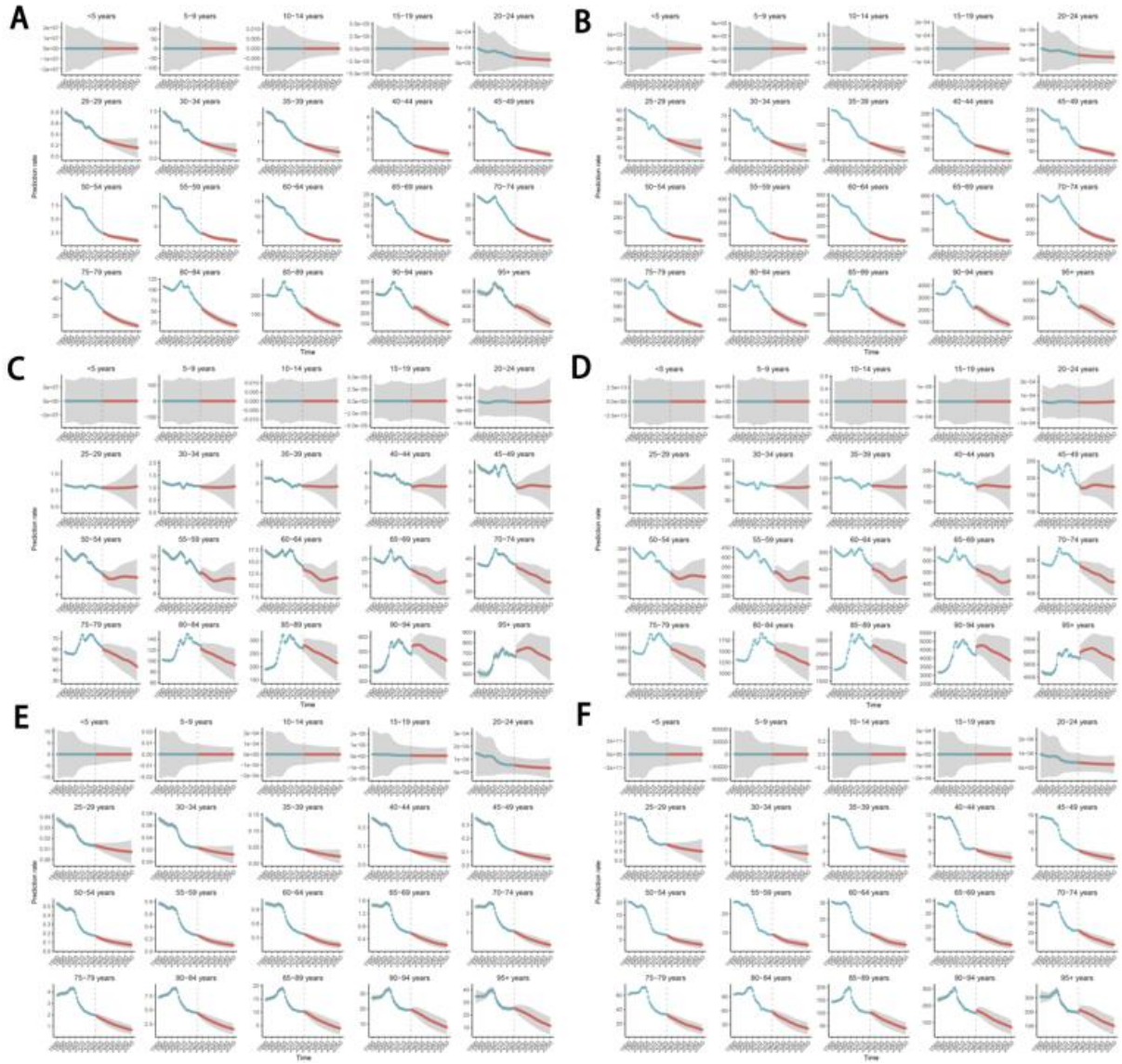

Figure 6. In China, age specific projections of ASDR and age-standardized DALYs rate per 100,000 population of CVD attributable to diet low in seafood n-3 PUFAs (A-B), diet low in n-6 PUFAs (C-D) and diet high in TFAs (E-F) by 2050. ASDR, age-standardized death rate; DALYs, disability-adjusted life years; CVD, cardiovascular diseases; PUFAs, polyunsaturated fatty acids; TFAs, trans fatty acids

rosis?. Prog Lipid Res. 2023;89:101209. doi:10.1016/j.plipres.2022.101209
10. Custers, Emma EM, Kiliaan, Amanda J. Dietary lipids from body to brain. Prog Lipid Res. 2022;85:101144. doi:10.1016/j.plipres.2021.101144
11. Fan Y, Jiang Y, Li X, Li X, Li Y, Wu H et al. Burden of Lung Cancer Attributable to Occupational Carcinogens from 1990 to 2019 and Projections until 2044 in China. Cancers (Basel). 2022;14:3883. doi: 10.3390/cancers14163883.
12. Bai Z, Wang H, Shen C, An J, Yang Z, Mo X. The global, regional, and national patterns of change in the burden of non-malignant upper gastrointestinal diseases from 1990 to 2019 and the forecast for the next decade. Int J Surg. 2025;111:80-92. doi: 10.1097/JS9.0000000000001902.
13. Yue C, Zhang Q, Sun F, Pan Q. Global, regional and national burden of neuroblastoma and other peripheral nervous system tumors, 1990 to 2021 and predictions to 2035: visualizing epidemiological characteristics based on GBD 2021. Neoplasia. 2025;60:101122. doi:10.1016/j.neo.2025.101122
14. Zou Z, Cini K, Dong B, Ma Y, Ma J, Burgner DP et al. Time Trends in Cardiovascular Disease Mortality Across the BRICS: An Age-Period-Cohort Analysis of Key Nations With Emerging Economies Using the Global Burden of Disease Study 2017. Circulation. 2020;141:790-9. doi: 10.1161/CIRCULATIONAHA.119.042864.
15. Riebler A, Held L. Projecting the future burden of cancer: Bayesian age-period-cohort analysis with integrated nested Laplace approximations. Biom J. 2017;59:531-49. doi: 10.1002/bimj.201500263.
16. Liu Z, Xu K, Jiang Y, Cai N, Fan J, Mao X et al. Global trend of aetiology-based primary liver cancer incidence from 1990 to 2030: a modelling study. Int J Epidemiol. 2021;50:128-42. doi: 10.1093/ije/dyaa196.
17. Khoi CS, Lin TY, Chiang CK. Targeting Insulin Resistance, Reactive Oxygen Species, Inflammation, Programmed Cell Death, ER Stress, and Mitochondrial Dysfunction for the Therapeutic Prevention of Free Fatty Acid-Induced Vascular Endothelial Lipotoxicity. Antioxidants (Basel). 2024;13:1486. doi:10.3390/antiox13121486
18. Jayedi A, Soltani S, Emadi A, Ghods K, Shab-Bidar S. Dietary intake, biomarkers and supplementation of fatty acids and risk of coronary events: a systematic review and dose-

response meta-analysis of randomized controlled trials and prospective observational studies. Crit Rev Food Sci Nutr. 2024;64:12363-82. doi:10.1080/10408398.2023.2251583
19. Liu X, Xu X, Zhang T, Xu L, Tao H, Liu Y et al. Fatty acid metabolism disorders and potential therapeutic traditional Chinese medicines in cardiovascular diseases. Phytother Res. 2023;37:4976-98. doi: 10.1002/ptr.7965.
20. Okamura T, Hashimoto Y, Majima S, Senmaru T, Ushigome E, Nakanishi N et al. Trans Fatty Acid Intake Induces Intestinal Inflammation and Impaired Glucose Tolerance. Front Immunol. 2021;12:669672. doi: 10.3389/fimmu.2021.669672.
21. Shirai Y, Imai T, Abe C, Sezaki A, Miyamoto K, Kawase F et al. The Association Between the Dietary Fatty Acid Fraction and Healthy Life Expectancy: Global Spatiotemporal Epidemiology from 2010 to 2019. J Am Nutr Assoc. 2025;44:591-8. doi: 10.1080/27697061.2025.2472656.
22. Tao Z, Wang Y. The health benefits of dietary short-chain fatty acids in metabolic diseases. Crit Rev Food Sci Nutr. 2025;65:1579-92. doi:10.1080/10408398.2023.2297811
23. Mavlanov U, Czaja TP, Nuriddinov S, Dalimova D, Dragsted LO, Engelsen SB et al. The effects of industrial processing and home cooking practices on trans-fatty acid profiles of vegetable oils. Food Chem. 2025;469:142571. doi: 10.1016/j.foodchem.2024.142571.
24. Wang A, Ao Y, Liu X, Wan X, Zhuang P, Jiao J et al. Potential impact of the time trend of fried food consumption on the cardiovascular disease burden in China. Food Funct. 2025;16:4278-90. doi: 10.1039/d4fo02978j.
25. Liu MW, McNaughton SA, He QQ, Leech R. Longitudinal trajectories of diet quality and subsequent mortality among Chinese adults: results from the China health and nutrition survey 1997-2015. Int J Behav Nutr Phys Act. 2021;18:51. doi: 10.1186/s12966-021-01118-7.
26. Monserrat-Mesquida M, Quetglas-Llabrés MM, Bouzas C, Pastor O, Ugarriza L, Llompart I et al. Plasma Fatty Acid Composition, Oxidative and Inflammatory Status, and Adherence to the Mediterranean Diet of Patients with Non-Alcoholic Fatty Liver Disease. Antioxidants (Basel). 2023;12:1554. doi: 10.3390/antiox12081554.
27. Wendeu-Foyet G, Bellicha A, Chajès V, Huybrechts I, Bard JM, Debras C et al. Different Types of Industry-Produced and Ruminant Trans Fatty Acid Intake and Risk of Type 2 Diabetes: Findings From the NutriNet-Santé Prospective Cohort. Diabetes Care. 2023;46:321-30. doi: 10.2337/dc22-0900.
28. Verneque BJF, Machado AM, de Abreu Silva L, Lopes ACS, Duarte CK. Ruminant and industrial trans-fatty acids consumption and cardiometabolic risk markers: A systematic review. Crit Rev Food Sci Nutr. 2022;62:2050-60. doi:10.1080/10408398.2020.1836471
29. Fang Y, Xia J, Lian Y, Zhang M, Kang Y, Zhao Z et al. The burden of cardiovascular disease attributable to dietary risk factors in the provinces of China, 2002-2018: a nationwide population-based study. Lancet Reg Health West Pac. 2023;37:100784. doi: 10.1016/j.lanwpc.2023.100784.
30. Guo Q, Li T, Qu Y, Liang M, Ha Y, Zhang Y et al. New research development on trans fatty acids in food: Biological effects, analytical methods, formation mechanism, and mitigating measures. Prog Lipid Res. 2023;89:101199. doi: 10.1016/j.plipres.2022.101199.
31. Silva TJ, Barrera-Arellano D, Ribeiro APB. Margarines: Historical approach, technological aspects, nutritional profile, and global trends. Food Res Int. 2021;147:110486. doi:10.1016/j.foodres.2021.110486
32. Zagkos L, Dib MJ, Pinto R, Gill D, Koskeridis F, Drenos F et al. Associations of genetically predicted fatty acid levels across the phenome: A mendelian randomisation study. PLoS Med. 2022;19:e1004141. doi: 10.1371/journal.pmed.1004141.
33. Kuntic M, Kuntic I, Hahad O, Lelieveld J, Münzel T, Daiber A. Impact of air pollution on cardiovascular aging. Mech Ageing Dev. 2023;214:111857. doi:10.1016/j.mad.2023.111857.