Jiang *et al. Lipids in Health and Disease* (2025) 24:62
https://doi.org/10.1186/s12944-025-02468-y

Lipids in Health and Disease

RESEARCH

Open Access

Examining the dietary contributions of lipids to pancreatic cancer burden (1990–2021): incidence trends and future projections

Kexin Jiang[1,2], Zhirong Zhao[3], Mu Yuan[4], Hua Ji[2], Yiwen Zhao[5], Hanyu Ding[1,2], Jiajie Feng[5], Yongjiang Zhou[5] and Ruiwu Dai[1,2*]

Abstract

Background Pancreatic cancer (PC) ranks sixth globally among cancer deaths, imposing a significant burden on healthcare systems worldwide. Although diet is known to be a major risk factor, Although diet is a well-established risk factor for PC, the precise dietary components linked to the disease remain inconclusive, with studies showing varying results across different populations and regions. This study addresses this gap through a comprehensive analysis of PC incidence trends from 1990 to 2021, with a specific focus on associations with age, dietary patterns, and socio-demographic determinants.

Methods The data utilized in this study were obtained from the 2021 Global Burden of Disease (GBD) results database, updated on May 16, 2024. Unlike traditional single-variable correlation analyses, a Bayesian generalized linear model was applied to assess the association between food intake and disease incidence during the period 1990–2021. To account for variations related to year and region, these variables were incorporated as covariates in the model, allowing for a more nuanced and comprehensive analysis of the background factors. Finally, the "BAPC" package was employed to project age-standardized incidence rates of PC through the year 2051.

Results The global incidence of PC increased from 3.90 per 100,000 people (95% CI: 3.69, 4.08) in 1990 to 6.44 per 100,000 (95% CI: 5.86, 6.93) in 2021. The analysis revealed significant associations between PC incidence and the intake of nuts, omega-3 fatty acids, polyunsaturated fatty acids (PUFA), trans fats, dietary sodium, and calcium. In typical countries, higher intake of nuts and PUFA was associated with a reduced incidence of PC, while trans fats were positively correlated with increased incidence. The age-standardized Bayesian Age-Period-Cohort (BAPC) prediction indicates that the incidence rates of PC will show a downward trend after 2021.

Conclusions From 1990 to 2021, the global incidence of PC exhibited a rapid upward trend, suggesting an increasing global healthcare burden. The findings of this study suggest that dietary lipid intake is significantly associated with PC incidence at a global level. This finding underscores the importance of dietary fat composition, particularly in the context of pancreatic cancer prevention, suggesting that individuals should pay attention to the types and sources of fats in their diets to mitigate disease risk.

*Correspondence:
Ruiwu Dai
dairuiwu@swjtu.edu.cn
Full list of author information is available at the end of the article

Keywords Dietary factors, Global burden of disease (GBD), Pancreatic cancer, Sociodemographic index (SDI)

Introduction

Pancreatic cancer (PC) is widely acknowledged as one of the most lethal malignancies. Over the past 30 years, the global incidence of PC has been 58.6 cases per million people, accompanied by a mortality rate of 57.7 cases per million [1, 2]. Of particular concern is the five-year survival rate for PC, which is approximately 10%, the lowest among major cancers [3]. Due to the asymptomatic nature of early-stage PC, it is often diagnosed at advanced stages, underscoring the urgent need for effective preventive measures to reduce PC incidence [4, 5]. In addition to immutable genetic factors, modifiable risk factors for PC include obesity, smoking, alcohol intake and dietary characteristic [6–9]. Addressing these modifiable risk factors is crucial for reducing the global incidence of PC and improving public health. Although it is established that PC incidence is closely related to dietary factors, the specific types of diets associated with PC risk remain inconclusive. A comprehensive analysis of global dietary patterns and PC incidence data is essential to elucidate these relationships and understand how dietary characteristics influence PC trends over time [10–12].

The significant impact of diet on PC risk has garnered substantial attention. A cohort study involving 50,045 adults aged 40 to 75 identified unsaturated fatty acids, particularly monounsaturated fatty acids (MUFAs), as protective dietary factors against PC [13]. Conversely, a meta-analysis of 38 prospective cohort studies found no significant association between omega-3 fatty acid intake and PC incidence [14]. Additionally, numerous reports have examined the effects of dietary factors such as sugar-sweetened beverages, red meat, vegetables, and fruits on PC incidence. Studies have shown that sugar-sweetened beverages may increase PC risk, likely due to their high glycemic index and the insulin surge they trigger [15]. Similarly, high consumption of red meat, especially processed meats, is associated with an increased risk of PC, possibly due to carcinogens like nitrosamines [16, 17]. In contrast, vegetables and fruits, rich in antioxidants and fiber, may reduce PC risk by lowering inflammation and oxidative stress [18].

The current study hypothesizes that comprehensive dietary patterns - particularly lipid-related components - exert a more significant influence on global PC incidence than isolated food types. This hypothesis is driven by two critical gaps in existing literature: First, while prior research has predominantly focused on individual dietary elements, the synergistic effects of combined dietary components remain unexplored, particularly regarding lipid profiles as systemic metabolic regulators. Second, most evidence derives from population-specific cohorts lacking global representativeness, potentially obscuring macro-level dietary-PC relationships shaped by regional food cultures and economic development. Some existing research relies predominantly on retrospective analyses and meta-analyses. Prospective studies are considered more suitable for investigating long-term health trends and assessing the long-term effects of interventions, but their results take longer to generate and are more suited for collecting specific data, which can present challenges in data collection. In contrast, ecological studies provide valuable insights at the population level, integrating broader dietary patterns and offering a unique perspective that complements the individual-level findings of prospective cohort studies. Furthermore, there is a lack of global big data comparisons on the relationship between overall dietary types and PC incidence [19–22]. Therefore, a comprehensive study integrating the impact of various dietary characteristics on PC incidence is essential to address these controversies and inform the development of effective healthcare policies.

The Global Burden of Disease (GBD), Injuries, and Risk Factors Study 2021 leverages the latest epidemiological data and advanced statistical techniques to construct comprehensive health metrics with demographic characteristics, facilitating an in-depth analysis of global disease trends over time. Updated on May 16, 2024, with data from 1990 to 2021 on disease burden, and again on June 5, 2024, with updated dietary risk exposure data. Data were extracted from the GBD 2021 database, and a Bayesian generalized linear mixed model (BGLMM) was developed to examine the association between dietary characteristics and PC incidence at global and country-specific levels, stratified by development status, during the period 1990–2021. This methodology represents a significant improvement over prior studies that primarily utilized Spearman or Pearson correlation tests to examine univariate relationships. By stratifying dietary patterns across different income levels and geographic regions—such as high-income nations, Mediterranean diet-practicing regions, populations with predominantly vegetarian diets, and areas with high fried food consumption—our study provides a nuanced understanding of dietary risk factors at a global scale.

Furthermore, representative countries were categorized into distinct dietary pattern groups, including high-income nations, Mediterranean diet-practicing regions, populations with predominantly vegetarian diets, and regions characterized by high fried food consumption. The association between PC incidence and dominant dietary profiles within these groups was subsequently examined. Global maps were generated to visualize the

Table 1 Trends of pancreatic cancer incidence from 1990 to 2021 by SDI quintiles

	Global		High SDI		High-middle SDI		Middle SDI		Low-middle SDI		Low SDI	
Year	1990	2021	1990	2021	1990	2021	1990	2021	1990	2021	1990	2021
Sex (Number [a])												
Male	110395.6 (104541.9, 116510.2)	273617.1 (250808.5, 299347.6)	48924.4 (47492.8, 50236.6)	110810.2 (103440.4,116614.4)	36647.4 (34070.2, 39388.1)	82216.5 (72057.7, 94434.4)	18275.6 (16222.5, 20589.6)	59272.8 (51107.1, 68782.6)	4757.2 (3956.4, 5546.5)	16924.2 (15572, 18315.6)	1634 (1245.1, 1976.1)	4113.1 (3373.9, 5077.6)
Female	97509.7 (90497.6, 103233)	234915.6 (205148.7, 255434.6)	48597.6 (44692.2, 50831)	106336.1 (90284.5, 115698.6)	29,844 (27832.5, 32030.5)	66080.6 (57506.5, 74467.8)	14107.5 (12451.4, 15846.5)	44303.1 (38489.6, 50372.7)	3559 (2918.7, 4247.5)	14223.7 (12912.1, 15451.2)	1268.2 (943, 1583.6)	3717.2 (2999.5, 4455.2)
Age (Rate[b] and Number)												
Age standardized (Rate)	5.5 (5.2, 5.7)	6 (5.4, 6.4)	8.8 (8.3, 9)	10 (9.1, 10.6)	6.8 (6.4, 7.1)	7.5 (6.7, 8.3)	3.2 (2.9, 3.5)	3.9 (3.4, 4.4)	1.4 (1.2, 1.6)	2.2 (2.0, 2.4)	1.3 (0.9, 1.9)	1.6 (1.3, 1.9)
All ages (Rate)	3.90 (3.69, 4.08)	6.44 (5.86, 6.93)	11.09(10.49,11.45)	19.85(17.71, 21.18)	6.25(6.60,5.91)	11.37(12.61,10.16)	1.88 (2.07,1.71)	4.23(4.76,3.73)	0.72(0.84,0.61)	1.62(1.75,1.51)	0.58(0.45, 0.69)	0.70(0.58, 0.84)
25 to 29	559.8 (516.9, 606.7)	684.1 (618.4, 751.2)	103.6 (99.1, 107.8)	107.0 (101.5, 112.8)	203.8 (183.9, 224.7)	167.2 (145.7, 191.3)	191.7 (172.8, 212.2)	252.4 (218.4, 285.5)	45.9 (38.2, 56.3)	113.2 (98.4, 132.7)	14.3 (10.8, 17.8)	43.7 (33.5, 57.2)
30 to 34	1382.3 (1283.8, 1493.7)	2089.8 (1879.5, 2300.1)	312.3 (302.3, 323.9)	342.7 (325.7, 362.9)	522.4 (477.8, 575.2)	636.1 (546.8, 734.2)	425.8 (385.7, 470.2)	769.7 (661.5, 873.1)	94.2 (78.6, 113)	257.6 (226.5, 294.6)	26 (19.6, 32.2)	82 (64.7, 108.4)
35 to 39	3252 (3003.9, 3503.2)	4526.3 (4113.6, 4959.4)	804 (780.1, 827)	868.3 (831.2, 912.8)	1255 (1130.6, 1394.4)	1440.5 (1249, 1650.1)	955.6 (856.3, 1070.9)	1539.7 (1337.2, 1757.3)	181.1 (152.3, 215)	516.4 (459.2, 584.3)	52.6 (39.6, 64.6)	157.8 (123.6, 205.3)
40 to 44	5457.1 (5088.3, 5860.1)	8371.7 (7620.7, 9125.3)	1694.3 (1639, 1748.5)	1811.4 (1735.5, 1890.9)	1980.5 (1815.8, 2165.9)	2679.6 (2343.8, 3081.2)	1386.2 (1228.4, 1581.2)	2660.7 (2299.1, 3014.1)	299.2 (250.6, 356.6)	940.1 (847.3, 1049.5)	89.8 (67.8, 112.5)	272 (215, 347.8)
45 to 49	8280.6 (7811.2, 8793.6)	15,432 (14064.8, 17004.3)	2916.5 (2832.6, 2999.7)	3704.1 (3552.1, 3853.5)	2809.6 (2572.4, 3052.4)	4958.6 (4302.9, 5716.5)	1885.9 (1688.2, 2104.6)	4717.9 (4044.5, 5468)	494.4 (410.7, 588.2)	1572.4 (1419.9, 1738.9)	163.1 (124.2, 202.1)	463.3 (371.5, 595)
50 to 54	14864.1 (14006.3, 15823.2)	28619.5 (26054.2, 31458.2)	4899 (4742.9, 5030.9)	7569.5 (7268.4, 7875.7)	5690.2 (5309, 6148.7)	9177.1 (7964.9, 10484.4)	3166 (2816, 3580.9)	8495.9 (7312.6, 9770.6)	814.9 (686.6, 985.7)	2637.7 (2395, 2930.4)	274.4 (211.9, 339.9)	711.1 (568, 895.7)
55 to 59	21424.3 (20260.3, 22719.4)	44529.4 (41008.2, 48099)	7738.9 (7530.7, 7928.9)	13,430 (12898.3, 14028.4)	7865.3 (7315, 8448.6)	14230.5 (12542.8, 16106.5)	4296.4 (3842.2, 4798.2)	12015.9 (10410, 13664.4)	1093.7 (908.9, 1306.4)	3825 (3442.6, 4205.2)	399.2 (309.4, 490.4)	983.3 (806.5, 1210.5)
60 to 64	29027.8 (27611, 30403.5)	59148.6 (54940.7, 63511.3)	11577.5 (11232.2, 11866.5)	20990.1 (19940.5, 21957.5)	10590.6 (9989.4, 11206)	18544.4 (16598.9, 20573.2)	4954.5 (4453.2, 5541)	13,657 (12068.7, 15435.6)	1376.8 (1143.5, 1660.8)	4737 (4285.1, 5226.6)	485.6 (374.2, 597.9)	1152.7 (947.6, 1389.6)
65 to 69	32058.7 (30714.8, 33394.2)	75,378 (69573.4, 81385)	14924.9 (14423.8,15343.9)	28124.4 (26449.7, 29722.5)	10,393 (9827.8, 10952.6)	23,868 (21282.3, 27002.6)	4832.7 (4388.3, 5324.6)	16875.4 (14732.8, 19063.1)	1338.7 (1118.8, 1564.2)	5135.3 (4729.7, 5591.2)	520.3 (403.5, 626.8)	1288.1 (1073.4, 1536.6)

Table 1 (continued)

	Global		High SDI		High-middle SDI		Middle SDI		Low-middle SDI		Low SDI	
Year	**1990**	**2021**	**1990**	**2021**	**1990**	**2021**	**1990**	**2021**	**1990**	**2021**	**1990**	**2021**
70 to 74	28985.5 (27580.7, 30259.8)	81735.8 (75474.3, 88081.5)	14606.5 (13957.9, 15088.6)	35920.2 (33135.3, 37881.6)	8526.3 (8061.5, 9084.2)	24056.5 (21464, 26968.5)	4289.8 (3900.9, 4730.7)	15900.4 (13793, 18062.6)	1107.3 (925.8, 1290.1)	4642.2 (4284.3, 5051.6)	419.5 (323.2, 507.8)	1126.2 (935.9, 1329.8)
75 to 79	28174.2 (26662.4, 29304.5)	66845.1 (59832.2, 71659.2)	15730.2 (14786.2, 16345.9)	32722.6 (29129.3, 34878.3)	8382.2 (7888.4, 8788.8)	18249.1 (16110.3, 20143.4)	3005.4 (2764.8, 3281.9)	11757.4 (10262.6, 13301.1)	750.4 (632.3, 869.4)	3252.1 (3008, 3500.5)	264.6 (205.2, 318.8)	791.2 (659.3, 941.4)
80 to 84	19974.7 (17994.2, 21147.5)	57494.3 (49214.7, 62462.8)	12426.6 (11129.9, 13203.3)	30899.9 (25514.5, 33961.6)	5204 (4706.4, 5532.6)	15768.5 (13611.5, 17360.3)	1752.2 (1597.5, 1896.3)	8179 (7021.1, 9254.7)	436.3 (364, 506.8)	2111.5 (1894.1, 2288.3)	126.3 (97.2, 155.4)	475.6 (397.1, 571.6)
85 to 89	3517.9 (3713.1, 3176.3	39807(31638.4, 44119.1)	7441.5 (6272, 8016.7)	18178 (13884.9, 21339.5)	2732.5 (2400, 2949.2)	9860.3 (8140.1, 11019.2)	847.8 (915.1, 761.5)	5691.2 (4775, 6441.6)	186 (154.5, 215)	948.5 (808, 1035.6)	42.3 (31.4, 53.1)	189.8 (153.6, 231.4)
90 to 94	3248 (2651.4, 3582.2)	18533.1 (14107.1, 20923.1)	2398.6 (1914.6, 2684.1)	12819.8 (9563.8, 14706.1)	596.3 (501.7, 649.4)	3794.4 (3004.5, 4269.9)	193.2 (167.1, 212.3)	1560.1 (1254, 1785.9)	48 (39, 55.4)	298.9 (244.8, 332.9)	1.3 (0.9, 1.9)	45.1 (35.4, 57)
95 plus	594.5 (445.4, 669.2)	4911.2 (3476.5, 5740.7)	454.7 (336.3, 516.4)	3728.2 (2587.8, 4402)	99.4 (78.4, 110.8)	777.8 (581.5, 896.1)	29.6 (23.5, 34.3)	324.5 (242.6, 383)	8.9 (6.9, 10.6)	69.8 (49.8, 81.3)	8.6 (6.1, 11.4)	8.9 (6.6, 12)

[a]Number: The estimated number of new pancreatic cancer cases, with the 95% confidence interval in parentheses

[b]Rate: The number of new pancreatic cancer cases per 100,000 people, with the 95% confidence interval in parentheses

spatial distribution of PC incidence rates and dietary patterns, facilitating the analysis of worldwide disease-diet correlations. Finally, the Bayesian Age-Period-Cohort (BAPC) model was employed to project age-standardized PC incidence rates, estimating potential trends through the year 2051.

Methods and materials

Data sources

The data for this study were sourced from the 2021 Global Burden of Disease results database, updated on May 16, 2024. The GBD database is a publicly accessible resource that systematically estimates the incidence, prevalence, mortality, and risk factors of 369 diseases and injuries across 204 countries and territories from 1990 to 2021. The incidence data for all diseases come from 36,916 data sources, including scientific literature, household survey data, epidemiological monitoring data, disease registry data, clinical informatics, and other sources. The GBD study uses DisMod-MR 2.1 (Disease Modeling Meta-Regression; version 2.1) for modeling incidence. DisMod-MR 2.1 is a Bayesian disease modeling tool officially used by GBD to generate internally consistent estimates of incidence, prevalence, remission, and mortality by sex, location, year, and age group. The calculation of these incidence rates not only relies on direct incidence data but also incorporates related information derived from other disease burden estimates, ensuring the reliability and consistency of the model. The global pancreatic cancer incidence data from 1990 to 2021 can be found in Table 1 [23].

The dietary data for the 15 food items were extracted from the GBD 2021 Dietary Risk Exposure Estimates 1990–2021, which includes daily per capita intake (in grams/day or energy/day) by country, age, and gender. The selection of these 15 dietary factors adheres to the criteria established by the GBD Study for choosing risk factors. These criteria include the significance of the risk factor to disease burden or public health policy, the availability of sufficient data to estimate exposure levels, the strength of epidemiological evidence supporting a causal relationship between the risk factor and disease outcomes, and the availability of data to quantify the relationship between changes in exposure and health outcomes. Additionally, the factors must have evidence supporting their universal impact across different populations [24]. The food items analyzed were milk, nuts, omega-3 fatty acids, PUFA, dietary sodium, red meat, trans fats, vegetables, legumes, calcium, sugar-sweetened beverages, processed meat, fruits, and dietary fiber. All kinds of dietary data were included in the analysis [25].

Socio-demographic Index (SDI) data were obtained from the GBD 2021 database (accessed May 16, 2024), covering the period 1950–2021. Country-specific SDI values for 2021 were integrated with dietary datasets to evaluate variations in dietary patterns stratified by SDI quintiles [26].

Regarding missing data, all dietary data used in this analysis were sourced from the GBD 2021 estimates, which provide comprehensive datasets across a wide range of countries and demographic groups. In cases where specific data were missing for particular countries or years, the GBD model uses a robust interpolation method based on available data from neighboring years or similar regions, ensuring the estimates are internally consistent and complete. Therefore, while some regional variations may exist, the dataset is designed to minimize missing data and provide reliable estimates across all included countries and territories.

Covariates

The SDI is utilized as a comprehensive indicator of a region's economic development level, with higher values indicating more developed economies. The SDI is a geometric mean of three components, each scaled from 0 to 1: the total fertility rate for women under 25 years (TFU25), the average education level for individuals aged 15 and older (EDU15+), and the lag-distributed income per capita (LDI). An SDI value of 0 represents the theoretically lowest level of development related to health, while an SDI value of 1 represents the highest level. Countries and regions were categorized based on their SDI values into five groups: "Low SDI (0-0.466)", "Low-middle SDI (0.467–0.619)", "Middle SDI (0.620–0.712)", "High-middle SDI (0.713–0.810)", and "High SDI (0.811-1)". Each region's SDI value was then merged with the dietary intake data for the 15 food items. Missing values due to discrepancies in country names or case inconsistencies were manually corrected.

To align with dietary data, the same age range used in the dietary burden data was applied to pancreatic cancer incidence rates, dividing the population into 15 five-year age groups for individuals aged 25 and above: "25–29 years", "30–34 years",... up to "90–94 years", and "95 + years". The study period was from 1990 to 2021, and gender categories included "male", "female", and "both". Locations were selected as "all countries and territories", "Global", and all SDI categories. All these variables were incorporated as covariates in the model establishment.

GBD data processing and modeling process

Bayesian generalized linear model was established using the Markov Chain Monte Carlo (MCMC) method based on Bayesian theory. This model aimed to explore the relationship between pancreatic cancer incidence and the daily per capita intake of 15 food items. Significant variables were selected to observe their intake proportions in different countries and regions, allowing a comprehensive analysis of the relationship between incidence rates and dietary intake variations in various environments. In this model, the observed data were pancreatic cancer incidence rates, and the explanatory variables were the per capita intake of the 15 food items. The fixed effects of the model included the impact of food intake on pancreatic cancer incidence, while year, SDI, age, and gender were incorporated as random effects. This model fully accounted for the influences of food, year, SDI, age, and gender. Further analysis was conducted by grouping data by SDI, year, and age.

The Bayesian Age-Period-Cohort (BAPC) model was further applied to project sex-specific PC burden through 2051. The BAPC framework extends the conventional Age-Period-Cohort (APC) model, incorporating Bayesian inference to estimate age-, period-, and cohort-specific effects on disease rates while accounting for population demographic structures. Essentially, the APC model can be understood as a log-linear Poisson model. The BAPC model applies a second-order random walk with an inverse gamma prior distribution to the age, period, and cohort effects, under the assumption that the effects are similar at adjacent time points. To approximate the marginal posterior distribution, the BAPC model employs the integrated nested Laplace approximation.

Statistical analysis

All data were analyzed using R. Data cleaning and organization were performed with R packages such as "dplyr" and "Tidyr". Model building was conducted using the "MCMCglmm" and "lattice" packages. Data prediction was carried out using the "BAPC" package, while map data processing and visualization were done with the "scatterpie", "rgeos", and "rnaturalearth" packages. Data visualization was achieved using packages including "ggplot2", "magrittr", and "tidyverse".

Results

Trends in global pancreatic cancer incidence from 1990 to 2021

Table 1 reveals the PC incidence rates by age, gender, and SDI subgroups for the years 1990 and 2021. From a temporal perspective, the global incidence rate of PC in 1990 was 3.90 (95% CI: 3.69–4.08) per 100,000 people, increasing to 6.44 (95% CI: 5.86–6.93) per 100,000 people by 2021. Further analysis by SDI subgroups revealed notable differences. In the High SDI group, the PC incidence rate increased from 11.09 (95% CI: 10.49–11.45) in 1990 to 19.85 (95% CI: 17.71–21.18) in 2021. Conversely, the Low SDI group saw a smaller increase from 0.58 (95% CI: 0.45–0.69) to 0.70 (95% CI: 0.58–0.84) over the same period. Notably, the Low-middle SDI group (from 0.72 to 1.62 per 100,000), the Middle SDI group (from 1.88 to 4.23), and the High-middle SDI group (from 6.25 to 11.37) all experienced more than a twofold increase in incidence rates over the past three decades (Fig. 1A). However, when compared to the incidence rates of all ages, the changes in incidence rates after age standardization are not significant, indicating that age composition has a substantial influence on incidence rates. When examined by gender, the incidence number of PC in males (from 110395.6 to 273617.1) was consistently higher than that in females (from 97509.7 to 234915.6) in both 1990 and 2021.

An age-stratified analysis reveals a strong correlation between PC incidence and age. Whether it is 1990 or 2021, the incidence of PC in individuals aged 25–50 remains below 10 per 100,000 people. However, there is a marked increase in incidence rates after the age of 50. Among the very elderly population over 80 years old, the incidence of PC exceeds 50 per 100,000 people (Table 1). When further analyzed by SDI subgroups, similar trends are observed. In all SDI categories, the incidence of PC increases with age. This consistent pattern underscores that the elderly population, particularly those over 80, experience significantly higher incidence rates, often surpassing 50 per 100,000 people (Fig. 1B).

Dietary structure changes from 1990 to 2021

Further analysis of global dietary structure changes requires examination through the lens of the SDI. Table 2 presents the levels of intake for milk, nuts, omega-3 fatty acids, PUFAs, dietary sodium, red meat, trans fat, vegetables, legumes, legumes, calcium, sugar-sweetened beverages, processed meat, fruits, and dietary fiber across different SDI groups in 1990 and 2021. Across both 1990 and 2021, higher intakes of milk, fruits, vegetables, omega-3 fatty acids, PUFA, red meat, and processed meat were observed in High-middle SDI and High SDI groups. Conversely, the intake levels of dietary sodium, legumes, and trans fats were relatively similar across different SDI groups. Notably, despite the promotion of healthy eating habits, the intake of processed meat and trans fats in High-middle SDI and High SDI groups did not show an increasing trend over time.

Temporal trends in the intake of 15 food items across SDI quintiles (1990–2021) were visualized via line charts to facilitate comparative analysis. The Bayesian generalized linear model outputs, including effect estimates and credible intervals, are summarized in Table 2. Six dietary components—nuts, omega-3 fatty acids, PUFA, trans fat, dietary sodium, and calcium—demonstrated statistically significant associations with PC incidence ($P < 0.05$) and are graphically emphasized in Fig. 1C. The intake levels of the remaining foods are shown in Supplementary Fig. 1. Moreover, the intake levels of the six aforementioned food items across different age groups are displayed in Fig. 1D. It is observed that the consumption of omega-3

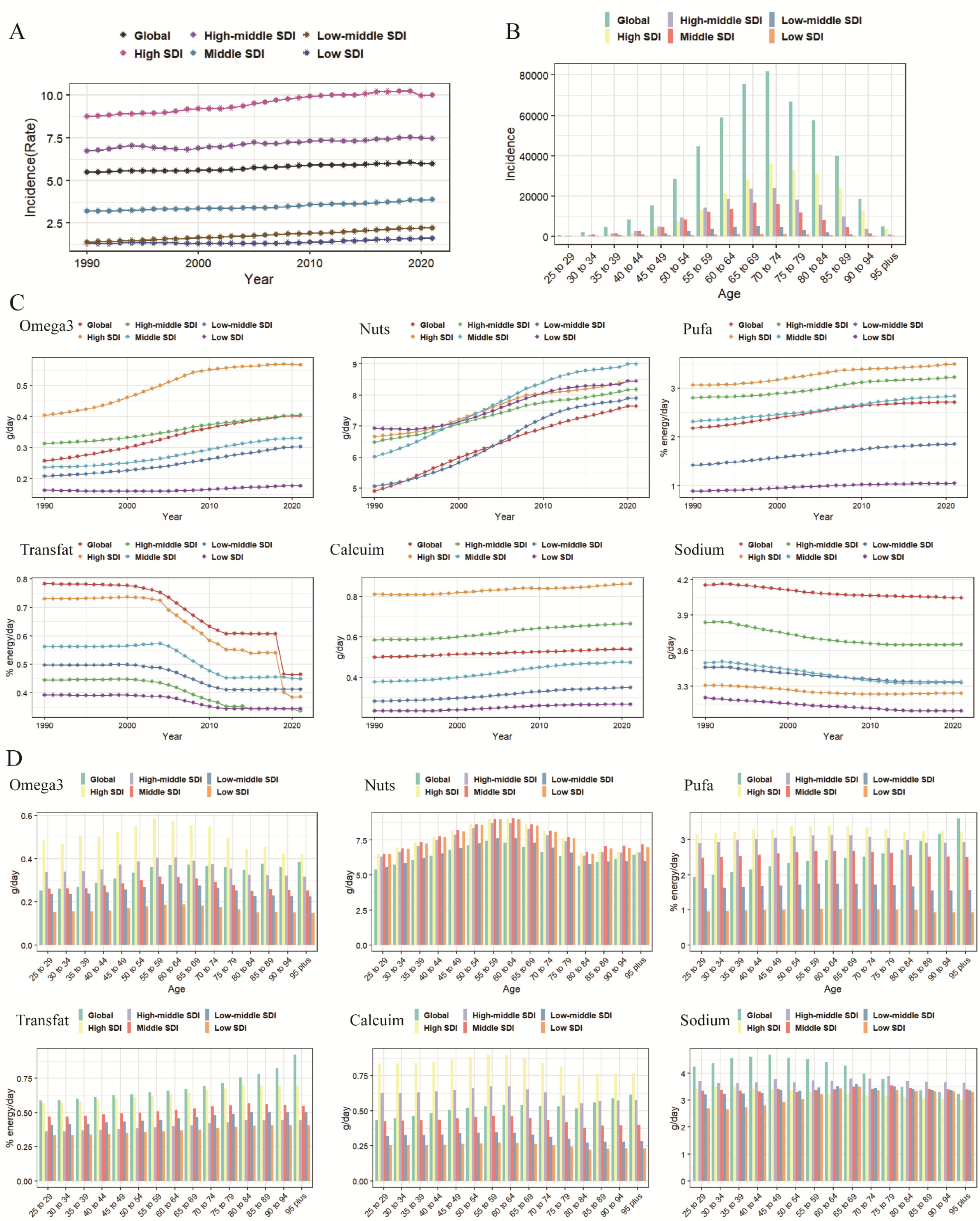

Fig. 1 Incidence rate of pancreatic cancer and changes in food intake based on year and age. **A:** Age-standardized incidence rate of pancreatic cancer in global and five SDI regions from 1990 to 2021 **B:** Incidence number of pancreatic cancer in different age groups, ranging from 25–29 to over 95 years old, for the global and five SDI regions in 2021. **C:** From 1990 to 2021, Trends in the intake of six foods significantly associated with the incidence of pancreatic cancer, as measured by the SDI. **D:** Age-stratified intake levels of six food items for the global population and five SDI regions in 2021

Table 2 Trends in the intake of 15 different foods from 1990 to 2021 and the associated significance *P*-value with the incidence rate of pancreatic cancer

	Global		High SDI		High-middle SDI		Middle SDI		Low-middle SDI		Low SDI		eff.samp	BGLMM modle *P* value
year	**1990**	**2021**	**1990**	**2021**	**1990**	**2021**	**1990**	**2021**	**1990**	**2021**	**1990**	**2021**		
Milk (g/day)	85.77 (84.27–87.37)	73.64 (71.43–75.99)	185.94 (168.54-204.86)	199.46 (170.31-232.22)	124.61 (115.8-134.07)	130.29 (112.54-150.29)	73.01 (68.66–77.63)	87.64 (76.81–99.62)	48.81 (45.86–51.97)	55.62 (48.35–63.67)	27.98 (25.96–30.22)	31.88 (27.35–36.91)	-0.54	< 0.001 ***
Nuts (g/day)	4.71 (4.43–5.05)	7.47 (6.95–8.06)	6.53 (5.39–8.01)	8.42 (6.06–11.47)	6.35 (5.11–7.97)	8.07 (5.73–11.08)	5.87 (4.66–7.46)	8.75 (6.34–11.93)	4.91 (4.15–5.88)	7.68 (5.65–10.26)	6.83 (5.83–8.09)	8.27 (5.99–11.2)	-13.52	< 0.001 ***
Omega3 (g/day)	0.23 (0.22–0.25)	0.34 (0.31–0.37)	0.4 (0.32–0.49)	0.56 (0.45–0.7)	0.31 (0.25–0.38)	0.4 (0.31–0.5)	0.23 (0.19–0.28)	0.32 (0.26–0.4)	0.2 (0.17–0.24)	0.3 (0.23–0.37)	0.16 (0.13–0.19)	0.17 (0.14–0.21)	561.93	< 0.001 ***
Red meat (g/day)	29.02 (27.14–31.39)	31.77 (28.88–35.25)	56.01 (50.41–63.07)	56.12 (41.87–74.14)	37.99 (34.1-42.91)	40.83 (30.46–53.87)	25.55 (23.4-28.21)	30.19 (23.24–38.83)	18.24 (16.9-19.84)	18.85 (14.28–24.55)	15.11 (13.7-16.88)	15.42 (11.44–20.45)	3.03	0.004 **
Sodium (g/day)	4.66 (4.38–4.97)	4.36 (4.12–4.61)	3.48 (3.01–4.01)	3.44 (2.98–3.96)	3.76 (3.22–4.37)	3.64 (3.12–4.22)	3.42 (2.97–3.92)	3.32 (2.89–3.8)	3.33 (2.91–3.8)	3.25 (2.85–3.71)	2.93 (2.51–3.41)	2.72 (2.33–3.16)	-9.05	< 0.001 ***
Transfat(%energy/day)	0.68 (0.63–0.74)	0.46 (0.43–0.51)	0.67 (0.52–0.85)	0.35 (0.28–0.45)	0.41 (0.32–0.52)	0.31 (0.24–0.39)	0.52 (0.42–0.65)	0.42 (0.33–0.52)	0.46 (0.36–0.58)	0.38 (0.3–0.48)	0.36 (0.28–0.46)	0.32 (0.24–0.4)	-172.57	< 0.001 ***
Vegetable (g/day)	146.43 (144.15-148.76)	205.33 (192.8-219.31)	170.99 (155.11-189.38)	193.26 (158.82-233.65)	181.6 (168.1-196.82)	214.74 (177.71-257.46)	108.76 (102.98-114.99)	164.95 (141.13-192.15)	65.79 (61.33–70.73)	99.56 (84.38-117.14)	40.74 (37.86–43.99)	48.95 (40.15–59.15)	2.77	< 0.001 ***
Wholegrains (g/day)	27.02 (26.52–27.53)	27.82 (26.85–28.84)	20.35 (18.93–21.92)	19.28 (16.26–22.74)	11.5 (10.22–13.03)	13.74 (11.36–16.59)	20.67 (19.56–21.91)	23.04 (20.12–26.37)	25.65 (24.38–27.02)	27.16 (23.97–30.7)	22.74 (21.39–24.23)	24.41 (20.97–28.29)	-1.01	< 0.001 ***
Pufa (%energy/day)	1.82 (1.8–1.84)	2.32 (2.26–2.39)	3.03 (2.83–3.25)	3.46 (3.06–3.89)	2.8 (2.63–2.98)	3.19 (2.84–3.58)	2.3 (2.19–2.41)	2.8 (2.53–3.1)	1.4 (1.33–1.49)	1.85 (1.65–2.07)	0.88 (0.83–0.94)	1.04 (0.93–1.18)	-14.06	< 0.001 ***
Legumes (g/day)	28.52 (27.94–29.14)	38.03 (35.97–40.37)	20.86 (18.11–24.03)	23.77 (18.92–29.56)	23.09 (20.29–26.33)	26.55 (21.07–33.06)	27.19 (24.67–30.01)	36.73 (30.16–44.48)	25.35 (23.28–27.65)	33.25 (27.39–40.17)	35.76 (32.79–39.05)	39.61 (31.58–49.1)	3.73	< 0.001 ***
Calcium (g/day)	0.45 (0.45–0.46)	0.5 (0.5–0.51)	0.83 (0.8–0.85)	0.88 (0.84–0.92)	0.6 (0.58–0.61)	0.68 (0.65–0.71)	0.39 (0.38–0.39)	0.48 (0.46–0.5)	0.3 (0.29–0.3)	0.37 (0.35–0.38)	0.24 (0.24–0.25)	0.28 (0.26–0.29)	-24.28	0.002 **
Sugar-sweeted beverages (g/day)	45.31 (39.29–53.39)	69.12 (60.8-79.51)	114.73 (56.34-215.87)	152.83 (78.1-281.03)	82.95 (39.82-158.74)	121.83 (61.42-225.62)	50.32 (23.93–98.94)	79.69 (39.96-151.53)	21.7 (9.54–44.97)	36.27 (16.26–74.91)	12.65 (4.94–27.62)	18.43 (6.95–40.65)	-0.61	< 0.001 ***
Processed meat (g/day)	14.68 (13.61–15.85)	11.77 (10.97–12.65)	33.95 (25.38–44.7)	38.84 (29.44–50.5)	15.68 (11.91–20.45)	16.58 (12.48–21.74)	6.47 (4.7–8.74)	7.68 (5.65–10.24)	5.84 (4.06–8.17)	6.56 (4.56–9.19)	8.04 (5.47–11.47)	8.72 (5.92–12.4)	0.11	0.084

Table 2 (continued)

year	Global		High SDI		High-middle SDI		Middle SDI		Low-middle SDI		Low SDI		eff.samp	BGLMM modle *P* value
	1990	2021	1990	2021	1990	2021	1990	2021	1990	2021	1990	2021		
Fruit (g/day)	77.78 (74.34–81.8)	106.96 (100.41-114.27)	126.83 (111.2-145.96)	140.3 (108.34-179.89)	128.12 (114.98-143.97)	145.79 (113.37-185.45)	117.28 (108.01-127.95)	144.11 (116.03-178.05)	68.79 (62.95–75.67)	84.14 (66.27-105.49)	85.88 (78.89–94.08)	84.61 (65.62-107.76)	-0.41	< 0.001 ***
Fiber (g/day)	13.41 (12.93–13.98)	15.78 (14.99–16.7)	14.85 (13.7-16.16)	15.78 (13.47–18.41)	15.99 (14.88–17.26)	17.18 (14.68–20.03)	14.68 (13.98–15.47)	16.68 (14.62–18.98)	14.74 (14.05–15.49)	17.3 (15.02–19.82)	17.23 (16.49–18.03)	19.07 (16.54–21.89)	7.71	0.058

fatty acids and trans fats slightly increases among older age groups, while the intake of nuts is higher among young and middle-aged individuals. No significant age-related differences were noted for the remaining food items.

Relationship between dietary pattern and incidence rate of PC

Figure 2 illustrates the global dietary structures and the incidence of PC in 2021. In terms of new cases, ten countries and regions among 204 surveyed reported at least 10,000 new instances of PC, with China, the United States, Japan, Germany, and the Russian Federation ranking in the top five. These five nations accounted for 53% of the global incidence of PC. Seventy-one countries had incidence rates above the global average, with Greenland (15.21 per 100,000 people), Monaco (13.27 per 100,000), Uruguay (12.55 per 100,000), Finland (12.15 per 100,000), and the Czech Republic (11.73 per 100,000) among the 18 countries where the incidence exceeds 10 per 100,000, mostly in high SDI countries. Regarding dietary structure, the intake of nuts was generally high across most countries, yet the levels of unsaturated fats and trans fats varied significantly among different nations. Figure 3 displays the intake levels of diets across various countries, with the country having the highest intake serving as a reference point and the rest scaled between 0 and 1. The average intake values for 15 types of food across all countries in 2021 can be found in Supplementary Table 1.

Temporal trends in dietary characteristics and PC incidence rate in representative countries

In this study, to analyze the impact of dietary patterns on pancreatic cancer incidence across various countries, Countries representative of global dietary diversity—including the United States, China, Japan, Iceland, India, Israel, the Marshall Islands, and Mexico—were selected for analysis. Comparative visualizations between dietary patterns and PC incidence rates were subsequently generated. Bar graphs were created using age-standardized incidence rates of pancreatic cancer from 1990 to 2021, and line graphs were plotted for six types of diets that have a significant impact on pancreatic cancer, after calculating age-standardized values. In Fig. 4, it is evident that nut consumption in these eight representative countries has significantly increased over the past three decades, yet the incidence trends of PC vary. Japan shows a rapid increase, with the incidence rate per 100,000 rising by over 50% in thirty years. The USA, China, India, and the Republic of the Marshall Islands exhibit slight increases, with growth not exceeding 20%. No significant changes were observed in Israel and Iceland, where the rates have remained stable for nearly three decades. Over

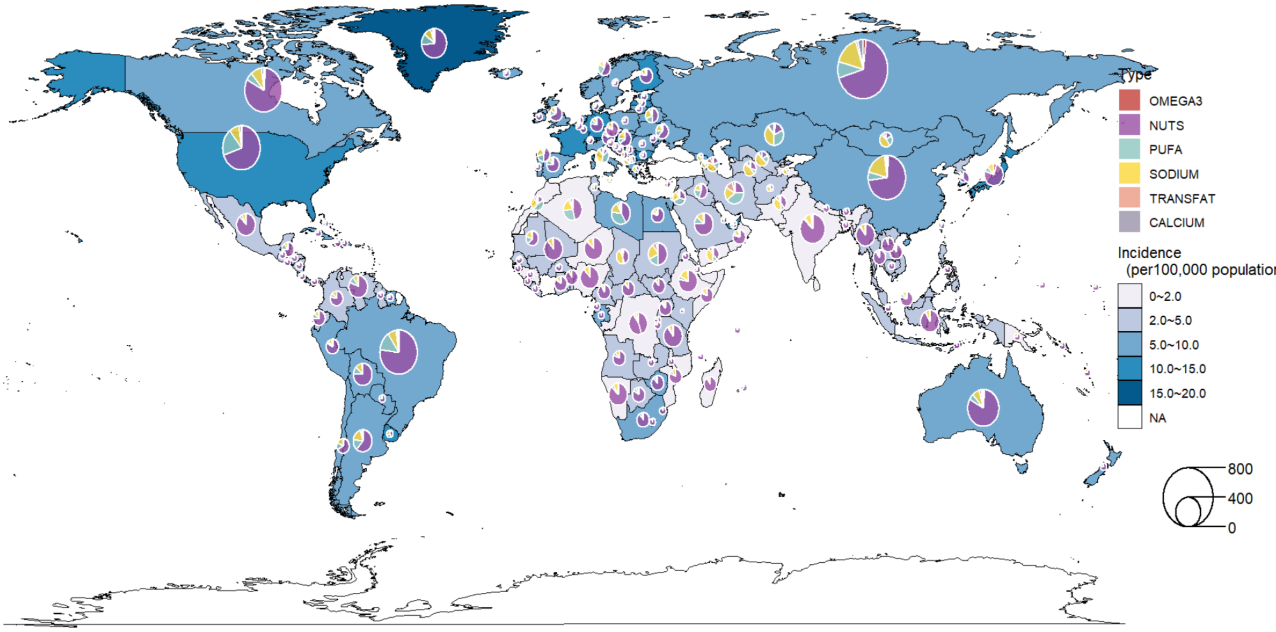

Fig. 2 Incidence of pancreatic cancer and intake of six associated foods worldwide in 2021. The color of each country on the map indicates its age-standardized incidence rate of pancreatic cancer, while the pie chart within each country's section shows the relative intake of six specific foods

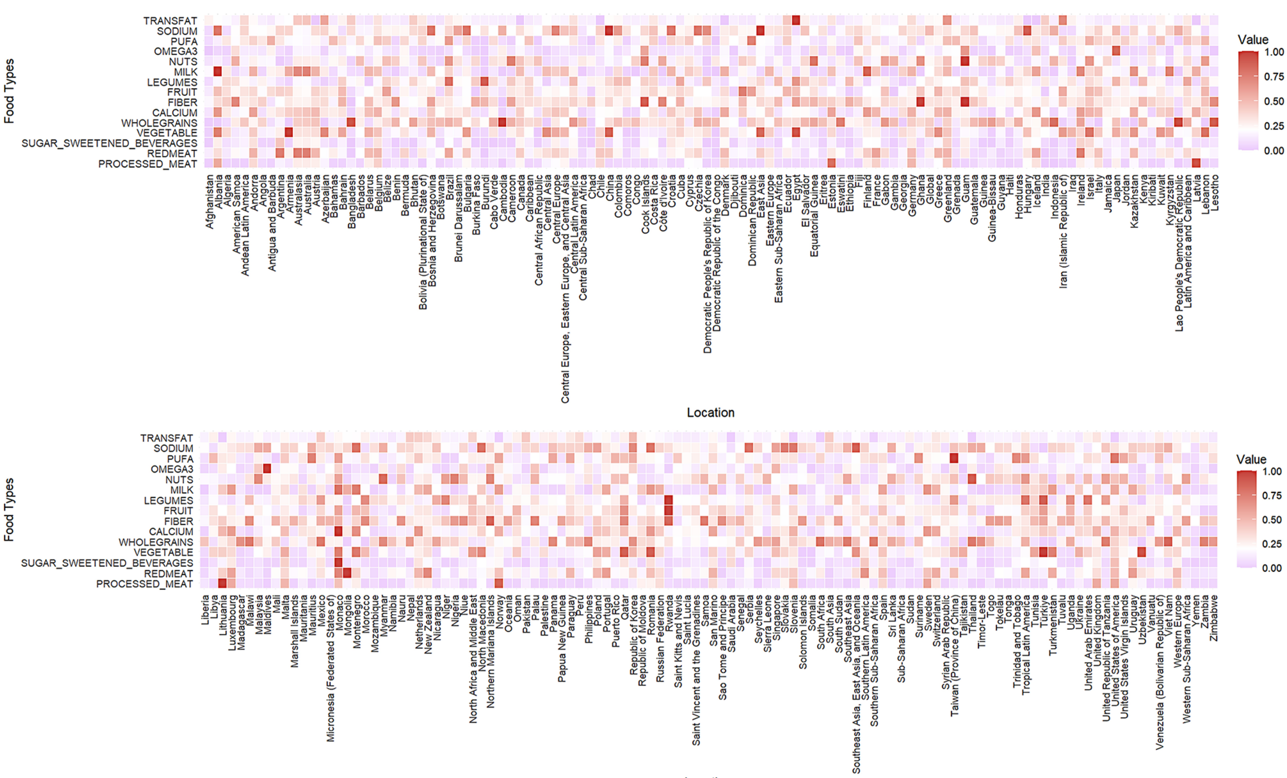

Fig. 3 Intake of all 15 types of food in different countries and regions in 2021, standardized to a scale of 0–1

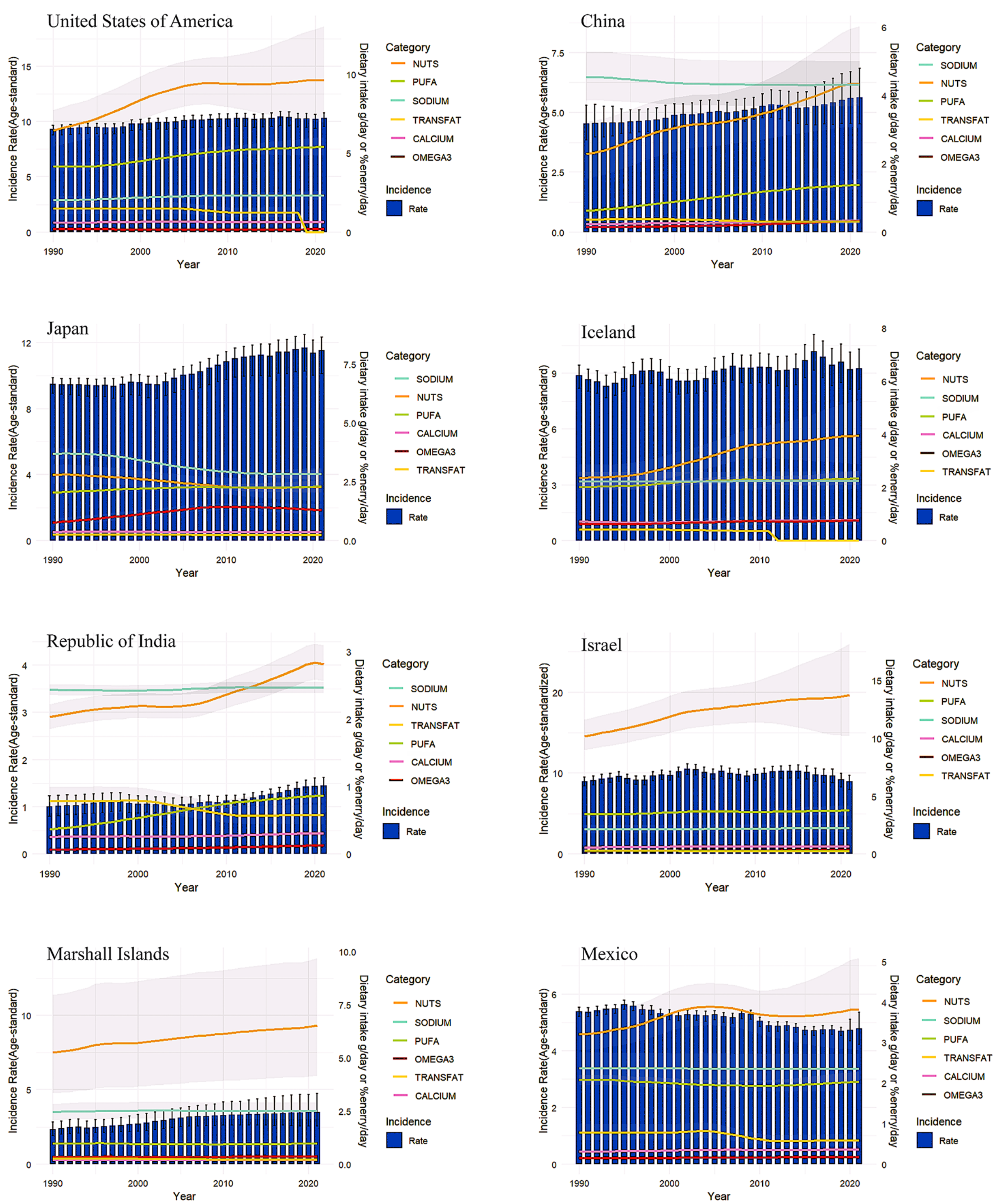

Fig. 4 Biaxial chart showing incidence rates and intake curves for six food types from 1990 to 2021 in eight countries with representative dietary structures. The X-axis represents years, the left Y-axis represents age-standardized incidence rates, and the right Y-axis represents different dietary intake (g/day or % energy/day). The 95% confidence interval for the incidence rate is denoted by error bars, while the 95% confidence interval for food intake is depicted by a gray band range

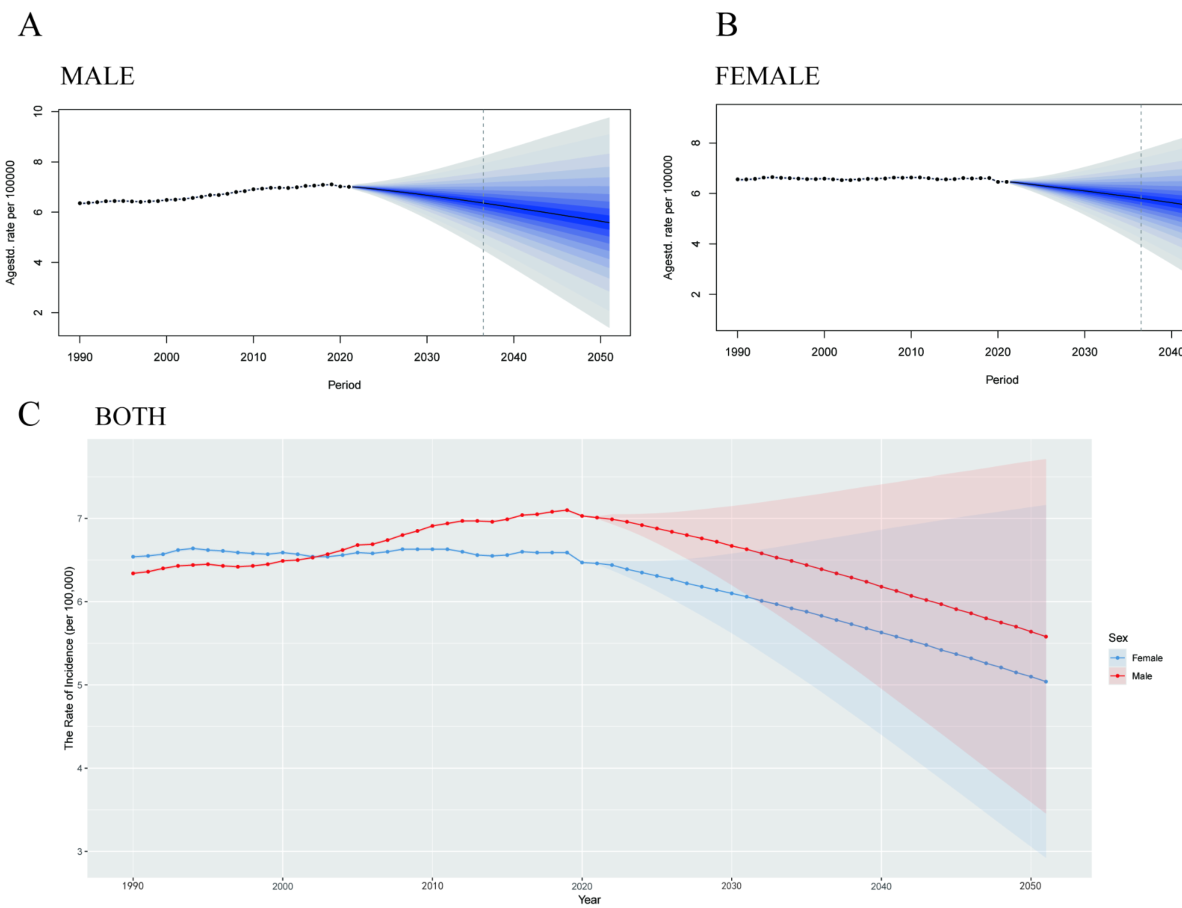

Fig. 5 Age-standardized incidence rate projections for pancreatic cancer over the next 30 years. A-B: Projected incidence trends for males and females. C: Combined line chart of projected incidence rates for both sexes

time, the intake of Omega-3 and PUFA has increased in most countries, while trans fat intake has decreased.

Bayesian age-period-cohort model prediction

Directly comparing incidence rates across different time periods or regions can be biased due to factors like population aging or changes in population structure. By using age-standardized incidence rates (ASR), these factors can be accounted for, allowing for more accurate and fair comparisons over time and across regions. To project future trends, a BAPC model was developed, estimating age-standardized PC incidence rates for the period 2021–2050. Figure 5 presents the BAPC predictions of age-standardized pancreatic cancer incidence rates, separated by sex (male and female). The trends indicate that from 2000 to 2020, the incidence of PC gradually increased, peaking around 2020. Following this peak, a steady decline in incidence is predicted, continuing through 2051.

Although the overall number of PC cases may increase in some countries due to population aging, the age-standardized data suggest that, after 2020, the incidence rates adjusted for age are expected to decline. This indicates that the actual incidence within each age group is decreasing, likely due to improvements in public health interventions, changes in diet and lifestyle, and other factors. Age-standardized incidence rates were projected using a reference population aged ≥ 45 years, with results documented in Supplementary Fig. 2. Notably, although male PC incidence rates consistently exceeded female rates throughout the study period, both sexes demonstrated parallel temporal trends—peaking in the early 2020s followed by a gradual decline. This suggests that, despite the gender differences, the main influencing factors for PC (e.g., environmental and lifestyle factors) may be similarly impactful for both sexes.

Discussion

According to estimates by the International Agency for Research on Cancer (IARC), 2022 witnessed nearly 20 million new cancer cases globally, with about one in five men or women expected to develop cancer during their lifetime [27]. PC rank twelfth in incidence but sixth in mortality, accounting for nearly 5% of all global cancer deaths, posing a significant barrier to increasing life expectancy. Studies indicate that trends in the incidence and mortality rates of pancreatic cancer partially reflect known risk factors: smoking, obesity, diabetes, and heavy alcohol consumption [28–30]. Additionally, dietary strategies remain a highly debated and focused area of research. It is commonly believed that high intake of animal-based foods and low consumption of calcium supplements, whole grains, and dietary fiber are risk factors for various cancers, though this perspective has not been widely accepted in the context of pancreatic tumors [31–33].

It is important to note that merely tracking the number of cases to understand the epidemiological characteristics of a disease can be affected by changes in the population's age structure, which can hinder effective comparisons between nations or regions. Age-standardized rates were therefore employed to estimate the prevalence of PC [34–36]. The analysis identified a previously unreported phenomenon, indicating that fatty foods have a significant impact on the incidence of PC. Based on posterior mean estimates from the BGLMM, nuts, omega-3 fatty acids, PUFAs, trans fats, calcium, and sodium were identified as the dietary components most strongly associated with PC incidence (Table 2). Notably, most of these components are lipids, suggesting that changes in dietary fat composition may impact the incidence of PC. Furthermore, populations in countries or regions with a medium or higher SDI tend to have greater access to animal products. This, combined with better diagnosis levels, contributes to higher incidence rates of PC.

Extensive research has established a close link between diet and the onset and progression of pancreatic cancer [37–41]. Studies by Evan et al. have shown that the ratio of unsaturated to saturated fatty acids in tumors correlates strongly with cancer development, and mismatches between diet-induced tumor fatty acid desaturation activity and the availability of specific fatty acid types directly impact tumor growth [42]. Research indicates that a lipid nuclear receptor known as peroxisome proliferator-activated receptor delta (PPARδ) is significantly upregulated in pancreatic intraepithelial neoplasia (PanIN) tissues in both humans and mice. A high-fat diet can activate PPARδ in pancreatic epithelial cells, significantly accelerating the progression from PanIN to pancreatic ductal adenocarcinoma (PDAC) [43]. Furthermore, the link between red meat consumption and increased risk of various cancers has been substantiated by numerous epidemiological studies. A case-control study network conducted in Italy and Switzerland from 1991 to 2009 identified a direct correlation between daily fats derived from meat consumption and PC [44], with fats derived from meat posing a higher risk of inducing pancreatic cancer than those from dairy products [45].

High-fat diets and Western dietary patterns are characterized by a high content of omega-6 polyunsaturated fatty acids (ω-6 PUFAs) and a low content of omega-3 polyunsaturated fatty acids (ω-3 PUFAs). In contrast, low-fat diets, such as the traditional Japanese diet, feature lower levels of ω-6 PUFAs and higher levels of ω-3 PUFAs. Compared to the Chinese diet, the American diet has a higher intake of ω-6 PUFAs, which may associated with a higher incidence of PC. Previous studies have highlighted arachidonic acid (AA), a ω-6 PUFA, has been shown to stimulate the growth of COX-2-positive pancreatic cancer cells, with the mechanism being mediated by COX-2 generated prostaglandin E2 (PGE2) binding to EP2 and EP4 receptors [46]. In contrast, preclinical studies and animal experiments have demonstrated that diets rich inω-3 PUFAs may be beneficial for the prevention of pancreatic cancer [47, 48]. The mechanisms underlying this phenomenon are not fully understood, but several potential explanations have been proposed. One study, through in vivo and in vitro experiments controlling ω-3 PUFA intake, demonstrated a significant association between increased levels of ω-3 PUFAs and a marked reduction in the phosphorylation and activation of AKT and its downstream targets, pFOXO3a and pBAD. This study highlighted the specific roles of eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) in this process [49]. Additionally, another study found that diets high in saturated fats accelerate early-stage pancreatic carcinogenesis, partly by increasing gut permeability and inducing metabolic endotoxemia. This process activates the TLR4 receptors and proliferation signals in the pancreas. In contrast, diets high in ω-3 PUFAs inhibit the upregulation of the TLR4/NF-κB/NOX1 signaling pathway, a phenomenon that may be beneficial for restoring colonic redox homeostasis and barrier function, alleviating endotoxemia, and preventing the development of pancreatic cancer [50].

From 1990 to 2005, the incidence rates of PC in Japan remained relatively stable; however, after 2005, there has been a consistent yearly increase. Japan, a country with high fish consumption, has experienced a gradual increase in ω-3 PUFAs intake from 1990 to 2010, after which the trend plateaued. A large-scale prospective study in Japan demonstrated that higher consumption of ω-3 PUFAs and DHA, particularly among groups with differing fish consumption patterns, was associated with a lower risk of pancreatic cancer [51]. A significant association was observed between ω-3 PUFA intake and PC incidence ($P < 0.001$). However, due to the heterogeneity in dietary patterns and the co-consumption of multiple food types, it remains unclear whether ω-3 PUFAs exert a direct causal effect on PC risk. Given the typical consumption of mixed diets—comprising proteins, carbohydrates, and fats—the independent effect of ω-3 PUFAs may be confounded by interactions with other dietary components. Although a significant association was observed, it remains uncertain whether ω-3 PUFAs alone drive the observed changes in PC incidence. Nevertheless, evidence suggests that ω-3 PUFAs may play a beneficial role in the treatment and prognosis of PC [52].

There are substantial differences in disease incidence and dietary intake across countries, which could be attributed to variations in research methodologies, such as the use of food frequency questionnaires, differences in cooking practices, and other lifestyle factors (e.g., exercise) that were not accounted for. These methodological discrepancies could result in incomplete or inconsistent data collection regarding food intake. For example, while several studies have suggested a protective role of nut consumption against pancreatic cancer, the effects of different types of nuts should be given more attention. Among the various types of nuts, tree nuts have shown a more significant ability to reduce cancer risk [53, 54]. Additionally, the roasting process may lead to the loss of some beneficial components in nuts, particularly polyunsaturated fatty acids and sensitive antioxidants, such as vitamin E. In contrast, raw nuts retain more of their nutritional value, especially in terms of omega-3 fatty acids and plant sterols.

According to the recently published Global Burden of Disease Study 2021, there is a rapid increase in the incidence of PC worldwide. This trend suggests that PC will impose an increasingly substantial burden globally in the future. The analysis revealed a notable surge in the incidence of PC per 100,000 people, particularly in regions classified as high-middle, middle, and low-middle SDI. Conversely, in low SDI areas, where healthcare resources are limited, there is likely a significant underdiagnosis of PC cases. Age stratification shows that regardless of the SDI category, the incidence of PC is generally lower in individuals under 50, while rates are considerably higher in those over 50. There are notable disparities in the incidence among those aged 80 and above across different SDI levels. For this oldest age group, except in low SDI countries, detection rates are relatively high, yet the incidence of PC sharply declines in the very elderly populations of low SDI regions. Moreover, compared to 1990, the growth rates of PC in 2021 were higher in high-middle, middle, and low-middle SDI areas. Age-standardized incidence rates (ASR) were applied in the prediction model to account for variations in population age structure. Following adjustment for age-specific population distributions, a gradual decline in PC incidence was observed. This suggests that, although the total number of cases may increase, the incidence of the disease is relatively decreasing within specific age groups. This trend may reflect the impact of public health interventions, improved lifestyle choices, and more effective diagnosis and treatment methods.

The Mediterranean dietary pattern, typically representative of traditional eating habits in countries around the Mediterranean Sea, derives approximately 40–50% of its energy from carbohydrates (predominantly complex types, such as whole grain bread), 10–20% from proteins (mainly fish), and 30–40% from fats (primarily ω-3 unsaturated fatty acids). Epidemiological studies on cancer have noted that high adherence to the Mediterranean diet can extend lifespan and reduce the risk of various cancers, including breast, stomach, and liver cancer [55]. However, conclusive evidence regarding its impact on pancreatic cancer incidence remains elusive. A significant association between elevated intake of PUFAs and nuts and PC incidence was identified in this analysis; however, confirmation through large-scale prospective cohort studies is warranted.

The age-standardized incidence rates of pancreatic cancer and population projections was used to forecast the incidence of pancreatic cancer in both males and females from 2021 to 2051. The results indicated a downward trend. Although age-standardization has accounted for the influence of population structure, future changes in overall population size and age distribution may still significantly affect the predicted outcomes. As the population ages, after excluding the influence of high incidence rates in the elderly, the total number of new cases could decrease. On the other hand, Currently, the global population is still expanding, and the rising population base may lead to an overall stable trend in disease prevalence, despite an increase in incidence rates, due to shifts in the age distribution. This effect becomes especially pronounced when using age-standardized incidence rates. If crude incidence rates were applied, projections would indicate a sustained upward trend, particularly in countries experiencing or anticipating substantial population aging. Additionally, the impact of the COVID-19 pandemic must also be considered. After 2019, there was a slight decline in the incidence of pancreatic cancer, which could be partially attributed to the higher mortality rates among the elderly caused by the pandemic. This demographic shift may have reduced the number of elderly individuals at risk of developing pancreatic cancer.

Study strengths and limitations

This study offers several methodological and conceptual advancements in understanding the global dietary determinants of PC. First, leveraging the GBD 2021 dataset with updates through June 2024, we employed a BGLMM to analyze associations between dietary characteristics and PC incidence. This approach addresses the limitations of traditional univariate correlation analyses by simultaneously adjusting for demographic confounders, spatial heterogeneity, and temporal trends. Second, socioeconomic-driven stratification by development status and geographic dietary clusters (e.g., Mediterranean regions) reveals macro-scale dietary risk patterns. Third, the BAPC modeling projects temporal trends in PC incidence through 2051, while geospatial visualization delineates spatial patterns in 2021. This dual analytical framework overcomes the unidimensional focus of prior ecological studies. Finally, our use of the most recent GBD dietary exposure metrics ensures alignment with contemporary global nutritional transitions, enhancing the relevance of findings for current public health policy discussions.

Several limitations should be acknowledged. First, the granularity of GBD data—collected at national or regional levels—precludes subnational analyses (e.g., state- or county-level assessments). In populous countries, this may obscure critical heterogeneity, as individual states often exhibit demographic and dietary diversity comparable to sovereign nations. Second, while Bayesian regression tools standardize estimations across heterogeneous diagnostic settings, broader confidence intervals persist in regions with underdeveloped cancer surveillance systems due to incomplete incidence ascertainment. Third, the ecological study design inherently limits causal inference regarding individual-level dietary exposures, though this approach remains optimal for detecting population-level associations influenced by food culture and economic development.

Future research should aim to minimize the impact of these limitations by conducting multi-center cohort studies with large population samples. This can be supplemented with more sophisticated statistical methods, such as stratified regression models, random forests, and other machine learning techniques. Additionally, comparing local or national survey data with global public health databases could help create more refined dietary-disease relationship models. Experimental studies that explore different dietary patterns, such as animal models with high-fat diets, or randomized controlled trials, could further enhance the reliability of the findings.

Our analysis identifies modifiable dietary targets with direct clinical implications: the protective association of nut consumption and unsaturated fatty acids and the hazardous effects of trans fats. These findings advocate for integrating dietary into existing PC risk stratification models, to prioritize high-risk individuals (e.g., those with obesity, metabolic syndrome, or familial PC history) for intensive surveillance programs. For clinical practice, our data support recommending Mediterranean-style dietary patterns—characterized by olive oil-based lipid substitution and nut supplementation—as an effective adjunct in PC prevention protocols.

Conclusion

In summary, a rapid increase in the incidence of PC has been observed globally, with rates exceeding 10 per 100,000 in many countries. Coupled with the growing risk of aging populations, this suggests that PC may pose an increasing burden on global healthcare systems in the future. The findings suggest that the intake of nuts and unsaturated fatty acids may be associated with a reduced incidence of PC, whereas increased consumption of trans fats may contribute to a potential rise in pancreatic cancer rates. These findings advocate for integrating dietary biomarkers into existing PC risk stratification models for intensive surveillance programs. Therefore, prevention of PC, screening of high-risk populations, and appropriate dietary recommendations represent a new and crucial perspective for the future management and prevention of PC, which is vital for reducing the healthcare burden associated with this disease.

Abbreviations

PDAC	Pancreatic ductal adenocarcinoma
PC	Pancreatic cancer
GBD	Global Burden of Disease
SDI	Socio-demographic Index
BGLMM	Bayesian generalized linear mixed model
TFU25	Total fertility rate for women under 25 years
EDU15+	Average education level for individuals aged 15 and older
LDI	Lag-distributed income per capita
MCMC	Markov Chain Monte Carlo
PUFA	Polyunsaturated fatty acid
IARC	International Agency for Research on Cancer

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12944-025-02468-y .

Supplementary Material 1

Supplementary Material 2

Supplementary Material 3

Supplementary Material 4

Supplementary Material 5

Acknowledgements

Not applicable.

Author contributions

All authors have made contributions in this study. Kexin Jiang analyzed preliminary data and completed the manuscript writing, Zhirong Zhao led the content design of the article and further revised the article, Mu Yuan provided guidance on professional knowledge in the research direction and experimental design. Hua Ji completed the main visual analysis. Yiwen Zhao completed data filtering and cleaning. Hanyu Ding and Feng Jiajie participated in the screening of all references. Yongjiang Zhou processed the data. Ruiwu Dai conducted the final manuscript editing and research design.

Funding

This work was supported by the Hospital Management Project of the General Hospital of Western Theater Command in Chengdu, Sichuan Province [grant number 2024-YGLC-A01].

Data availability
No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate
Not applicable.

Competing interests
The authors declare no competing interests.

Author details
[1]Department of General Surgery Center, College of Medicine, The General Hospital of Western Theater Command, Southwest Jiaotong University, Chengdu 610031, Sichuan, China
[2]General Surgery Center, The General Hospital of Western Theater Command, Chengdu 610083, Sichuan, China
[3]Department of General Surgery, Affiliated Jinling Hospital, Medical School of Nanjing University, Nanjing, Jiangsu, China
[4]The General Hospital of Western Theater Command, Chengdu, Sichuan 610083, China
[5]Department of General Surgery, Affiliated Hospital of Southwest Medical University, The General Hospital of Western Theater Command, Chengdu 610083, Sichuan, China

Received: 30 September 2024 / Accepted: 4 February 2025
Published online: 21 February 2025

References
1. Gualtieri P, Cianci R, Frank G, Pizzocaro E, De Santis GL, Giannattasio S et al. Pancreatic ductal Adenocarcinoma and Nutrition: exploring the role of Diet and Gut Health. Nutrients. 2023;15(20).
2. Nucci D, Nardi M, Cinnirella A, Campagnoli E, Maffeo M, Perrone PM et al. Adherence to Mediterranean Diet and Risk of Pancreatic Cancer: systematic review and Meta-analysis. Int J Environ Res Public Health. 2023;20(3).
3. Siegel RL, Giaquinto AN, Jemal A. Cancer statistics, 2024. Cancer J Clin. 2024;74(1):12–49.
4. Blackford AL, Canto MI, Dbouk M, Hruban RH, Katona BW, Chak A et al. Pancreatic Cancer surveillance and survival of high-risk individuals. JAMA Oncol. 2024.
5. Conroy T, Pfeiffer P, Vilgrain V, Lamarca A, Seufferlein T, O'Reilly EM, et al. Pancreatic cancer: ESMO Clinical Practice Guideline for diagnosis, treatment and follow-up. Annals Oncology: Official J Eur Soc Med Oncol. 2023;34(11):987–1002.
6. Stoffel EM, Brand RE, Goggins M. Pancreatic Cancer: changing Epidemiology and New approaches to Risk Assessment, early detection, and Prevention. Gastroenterology. 2023;164(5):752–65.
7. Rumgay H, Shield K, Charvat H, Ferrari P, Sornpaisarn B, Obot I, et al. Global burden of cancer in 2020 attributable to alcohol consumption: a population-based study. Lancet Oncol. 2021;22(8):1071–80.
8. Sun X, Teper Y, Sinnet-Smith J, Markarian M, Hines OJ, Li G, et al. Stress and obesity signaling converge on CREB phosphorylation to promote pancreatic cancer. Molecular cancer research: MCR; 2024.
9. Midha S, Chawla S, Garg PK. Modifiable and non-modifiable risk factors for pancreatic cancer: a review. Cancer Lett. 2016;381(1):269–77.
10. Zheng J, Merchant AT, Wirth MD, Zhang J, Antwi SO, Shoaibi A, et al. Inflammatory potential of diet and risk of pancreatic cancer in the prostate, lung, colorectal and ovarian (PLCO) Cancer Screening Trial. Int J Cancer. 2018;142(12):2461–70.
11. Hoyt M, Song Y, Gao S, O'Palka J, Zhang J. Associations between two Dietary Quality scores and Pancreatic Cancer Risk in a US National prospective cohort study. J Am Nutr Association. 2024;43(4):345–55.
12. Julián-Serrano S, Reedy J, Robien K, Stolzenberg-Solomon R. Adherence to 5 Diet Quality indices and Pancreatic Cancer Risk in a large US prospective cohort. Am J Epidemiol. 2022;191(9):1584–600.
13. Ghamarzad Shishavan N, Masoudi S, Mohamadkhani A, Sepanlou SG, Sharafkhah M, Poustchi H, et al. Dietary intake of fatty acids and risk of pancreatic cancer: Golestan cohort study. Nutr J. 2021;20(1):69.
14. MacLean CH, Newberry SJ, Mojica WA, Khanna P, Issa AM, Suttorp MJ, et al. Effects of omega-3 fatty acids on cancer risk: a systematic review. JAMA. 2006;295(4):403–15.
15. Turati F, Rossi M, Mattioli V, Bravi F, Negri E, La Vecchia C. Diabetes risk reduction diet and the risk of pancreatic cancer. Eur J Nutr. 2022;61(1):309–16.
16. Sivasubramanian BP, Dave M, Panchal V, Saifa-Bonsu J, Konka S, Noei F, et al. Comprehensive Review of Red Meat Consumption and the risk of Cancer. Cureus. 2023;15(9):e45324.
17. Zhao Z, Yin Z, Pu Z, Zhao Q. Association between Consumption of Red and processed meat and pancreatic Cancer risk: a systematic review and Meta-analysis. Clin Gastroenterol Hepatology: Official Clin Pract J Am Gastroenterological Association. 2017;15(4):486–e9310.
18. Pericleous M, Rossi RE, Mandair D, Whyand T, Caplin ME. Nutrition and pancreatic cancer. Anticancer Res. 2014;34(1):9–21.
19. Navarrete-Muñoz EM, Wark PA, Romaguera D, Bhoo-Pathy N, Michaud D, Molina-Montes E, et al. Sweet-Beverage consumption and risk of pancreatic cancer in the European prospective investigation into Cancer and Nutrition (EPIC). Am J Clin Nutr. 2016;104(3):760–8.
20. Thiébaut AC, Jiao L, Silverman DT, Cross AJ, Thompson FE, Subar AF, et al. Dietary fatty acids and pancreatic cancer in the NIH-AARP diet and health study. J Natl Cancer Inst. 2009;101(14):1001–11.
21. Stan SD, Singh SV, Brand RE. Chemoprevention strategies for pancreatic cancer. Nat Reviews Gastroenterol Hepatol. 2010;7(6):347–56.
22. Bravi F, Polesel J, Bosetti C, Talamini R, Negri E, Dal Maso L, et al. Dietary intake of selected micronutrients and the risk of pancreatic cancer: an Italian case-control study. Annals Oncology: Official J Eur Soc Med Oncol. 2011;22(1):202–6.
23. Global incidence. Prevalence, years lived with disability (YLDs), disability-adjusted life-years (DALYs), and healthy life expectancy (HALE) for 371 diseases and injuries in 204 countries and territories and 811 subnational locations, 1990–2021: a systematic analysis for the global burden of Disease Study 2021. Lancet (London, England). 2024;403(10440):2133–61.
24. Health effects of dietary risks in 195 countries, 1990–2017: a systematic analysis for the Global Burden of Disease Study 2017. Lancet (London, England). 2019;393(10184):1958-72.
25. Global burden and strength of evidence for. 88 risk factors in 204 countries and 811 subnational locations, 1990–2021: a systematic analysis for the global burden of Disease Study 2021. Lancet (London, England). 2024;403(10440):2162–203.
26. Global Burden of Disease Collaborative Network. 2022 [cited https://vizhub.healthdata.org/gbd-results/.]
27. Bray F, Laversanne M, Sung H, Ferlay J, Siegel RL, Soerjomataram I, et al. Global cancer statistics 2022: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. Cancer J Clin. 2024;74(3):229–63.
28. Zavala VA, Bracci PM, Carethers JM, Carvajal-Carmona L, Coggins NB, Cruz-Correa MR, et al. Cancer health disparities in racial/ethnic minorities in the United States. Br J Cancer. 2021;124(2):315–32.
29. Nduma BN, Ambe S, Ekhator C, Fonkem E. Geographical distribution of pancreatic Cancer in the state of Mississippi by Incidence and Mortality from 2003 to 2019. Cureus. 2022;14(11):e31605.
30. Chen CH, Tsai MK, Lee JH, Lin RT, Hsu CY, Wen C, et al. Sugar-Sweetened beverages is an independent risk from pancreatic Cancer: based on half a million Asian cohort followed for 25 years. Front Oncol. 2022;12:835901.
31. Kim S, Song S, Kim YS, Yang SY, Lee JE. The association between predicted inflammatory status and colorectal adenoma. Sci Rep. 2020;10(1):2433.
32. Abdullah MMH, Hughes J, Grafenauer S. Whole grain intakes are Associated with Healthcare cost Savings following reductions in risk of Colorectal Cancer and Total Cancer Mortality in Australia: a cost-of-illness model. Nutrients. 2021;13(9).
33. Clinton SK, Giovannucci EL, Hursting SD, The World Cancer Research Fund/American Institute for Cancer Research Third. Expert Report on Diet, Nutrition, Physical Activity, and Cancer: impact and future directions. J Nutr. 2020;150(4):663–71.
34. Hu W, Fang L, Zhang H, Ni R, Pan G. Changing trends in the air pollution-related disease burden from 1990 to 2019 and its predicted level in 25 years. Environ Sci Pollut Res Int. 2023;30(1):1761–73.
35. Sun C, Liu Y, Huang Y, Li B, Rang W. Colorectal Cancer incidence and mortality trends and analysis of risk factors in China from 2005 to 2015. Int J Gen Med. 2021;14:9965–76.
36. Yu W, Jiang J, Xie L, Li B, Luo H, Fu Y, et al. Mortality trends in Colorectal Cancer in China during 2000–2015: a Joinpoint regression and Age-Period-Cohort Analysis. Prev Chronic Dis. 2018;15:E156.

37. Salvadori G, Longo VD. Diet comparison suggests a lipid imbalance can slow tumour growth. Nature. 2021;599(7884):206–7.
38. Méndez-Lucas A, Yuneva M. Dinner is served, sir: fighting cancer with the right diet. Cell. 2021;184(26):6226–8.
39. Yang L, TeSlaa T, Ng S, Nofal M, Wang L, Lan T, et al. Ketogenic diet and chemotherapy combine to disrupt pancreatic cancer metabolism and growth. Med (New York NY). 2022;3(2):119–36.
40. Kim SH, Lee SM, Jeung HC, Lee IJ, Park JS, Song M et al. The Effect of Nutrition Intervention with oral nutritional supplements on pancreatic and bile Duct Cancer patients undergoing chemotherapy. Nutrients. 2019;11(5).
41. Cayssials V, Buckland G, Crous-Bou M, Bonet C, Weiderpass E, Skie G, et al. Inflammatory potential of diet and pancreatic cancer risk in the EPIC study. Eur J Nutr. 2022;61(5):2313–20.
42. Lien EC, Westermark AM, Zhang Y, Yuan C, Li Z, Lau AN, et al. Low glycaemic diets alter lipid metabolism to influence tumour growth. Nature. 2021;599(7884):302–7.
43. Liu Y, Deguchi Y, Wei D, Liu F, Moussalli MJ, Deguchi E, et al. Rapid acceleration of KRAS-mutant pancreatic carcinogenesis via remodeling of tumor immune microenvironment by PPARδ. Nat Commun. 2022;13(1):2665.
44. Di Maso M, Talamini R, Bosetti C, Montella M, Zucchetto A, Libra M, et al. Red meat and cancer risk in a network of case-control studies focusing on cooking practices. Annals Oncology: Official J Eur Soc Med Oncol. 2013;24(12):3107–12.
45. Nöthlings U, Wilkens LR, Murphy SP, Hankin JH, Henderson BE, Kolonel LN. Meat and fat intake as risk factors for pancreatic cancer: the multiethnic cohort study. J Natl Cancer Inst. 2005;97(19):1458–65.
46. Funahashi H, Satake M, Hasan S, Sawai H, Newman RA, Reber HA, et al. Opposing effects of n-6 and n-3 polyunsaturated fatty acids on pancreatic cancer growth. Pancreas. 2008;36(4):353–62.
47. Mohammed A, Janakiram NB, Brewer M, Duff A, Lightfoot S, Brush RS, et al. Endogenous n-3 polyunsaturated fatty acids delay progression of pancreatic ductal adenocarcinoma in Fat-1-p48(Cre/+)-LSL-Kras(G12D/+) mice. New York, NY: Neoplasia; 2012;14(12):1249–59.
48. Yu M, Liu H, Duan Y, Zhang D, Li S, Wang F. Four types of fatty acids exert differential impact on pancreatic cancer growth. Cancer Lett. 2015;360(2):187–94.
49. Ding Y, Mullapudi B, Torres C, Mascariñas E, Mancinelli G, Diaz AM et al. Omega-3 fatty acids prevent early pancreatic carcinogenesis via repression of the AKT pathway. Nutrients. 2018;10(9).
50. Ead AS, Wirkus J, Matsukuma K, Mackenzie GG. A high-fat diet induces changes in mesenteric adipose tissue accelerating early-stage pancreatic carcinogenesis in mice. J Nutr Biochem. 2024;131:109690.
51. Hidaka A, Shimazu T, Sawada N, Yamaji T, Iwasaki M, Sasazuki S, et al. Fish, n-3 PUFA consumption, and pancreatic cancer risk in Japanese: a large, population-based, prospective cohort study. Am J Clin Nutr. 2015;102(6):1490–7.
52. Isherwood J, Arshad A, Chung WY, Runau F, Cooke J, Pollard C, et al. Myeloid derived suppressor cells are reduced and T regulatory cells stabilised in patients with advanced pancreatic cancer treated with gemcitabine and intravenous omega 3. Annals Translational Med. 2020;8(5):172.
53. Stevens-Barrón JC, Wall-Medrano A, Álvarez-Parrilla E, Olivas-Armendáriz I, Astiazaran-García H, Robles-Zepeda RE et al. Synergistic interactions between Tocol and Phenolic extracts from different tree nut species against Human Cancer Cell lines. Molecules. 2022;27(10).
54. Long J, Ji Z, Yuan P, Long T, Liu K, Li J, et al. Nut consumption and risk of Cancer: a Meta-analysis of prospective studies. Cancer Epidemiol Biomarkers Prevention: Publication Am Association Cancer Res Cosponsored Am Soc Prev Oncol. 2020;29(3):565–73.
55. Schwingshackl L, Schwedhelm C, Galbete C, Hoffmann G. Adherence to Mediterranean Diet and Risk of Cancer: an updated systematic review and Meta-analysis. Nutrients. 2017;9(10).

Publisher's note