1990 – 2017年195个国家的膳食风险对健康的影响：2017年全球疾病负担研究的系统分析

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摘要

背景次优饮食是非传染性疾病（NCDs）的重要可预防风险因素;然而，其对非传染性疾病负担的影响尚未得到系统评估。本研究旨在评估195个国家主要食物和营养素的消费量，并量化其次优摄入量对NCD死亡率和发病率的影响。

方法通过比较风险评估方法，我们估计了25岁或以上成年人中每种膳食风险因素（也称为人群归因分数）引起的疾病特异性负担的比例。该分析的主要投入包括每种饮食因素的摄入量，饮食因素对疾病终点的影响大小，以及与最低死亡风险相关的摄入量。然后，通过使用疾病特异性人口归因分数，死亡率和残疾调整生命年（DALYs），我们计算了每种疾病结果可归因于饮食的死亡和DALYs的数量。

结果2017年，1,100万（95%不确定区间[U] 10-12）死亡和2.55亿（234-274）DALYs归因于饮食风险因素。大量摄入钠（300万[1-5]死亡和7000万[34-118] DALYs），全谷物摄入量低（300万[2-4]死亡和8200万[59-109] DALYs），以及水果摄入量低（200万[1-4]死亡和6500万[41-92] DALYs）是全球和许多国家死亡和DALYs的主要膳食风险因素。膳食数据来自混合来源，并非所有国家都可获得，增加了我们估计的统计不确定性。

解释本研究全面描述了次优饮食对NCD死亡率和发病率的潜在影响，强调了改善各国饮食的必要性。我们的研究结果将为实施基于证据的饮食干预提供信息，并为评估其每年对人类健康的影响提供平台。

资助比尔和梅琳达盖茨基金会。

在线发布
2019年4月3日
http://dx.doi.org/10.1016/S0140-6736(19)30041-8

见**在线/评论**
http://dx.doi.org/10.1016/S0140-6736(19)30500-8

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介绍

饮食习惯与慢性非传染性疾病（NCDs）之间的关系已得到广泛研究。[1-5]长期随机对照NCD终点试验对于大多数饮食因素并不可行，但合成其他流行病学证据，包括长期前瞻性观察研究和中期结果的短期试验，为特定饮食因素（如水果，蔬菜，加工肉类和反式脂肪摄入量）与非传染性疾病（缺血性心脏病，糖尿病和结直肠癌）之间潜在的因果关系提供了支持性证据。[2-7]这些研究结果被广泛用于国家和国际饮食指南，旨在预防非传染性疾病。[8,9]然而，由于不同国家饮食消费特征的复杂性，评估次优饮食对健康的影响人口水平是不可能的。

在过去十年中，已经做出量化的努力由特定饮食引起的疾病负担因素。[10-19]这些努力虽然有用但有几个重要的局限性，包括饮食消费的地理代表性数据不足，饮食摄入人口分布的不准确表征，不同膳食评估来源的偏差计算不充分，摄入量的标准化。每天2000千卡，并且不足以说明饮食因素摄入量的人与人之间的差异。

为了解决这些局限性，作为全球疾病，伤害和风险因素研究负担（GBD）2017的一部分，我们系统地收集了来自多个来源的具有地理代表性的膳食数据，其特征是成年人中15种食物和营养素的摄入量的人口分布在195个国家25岁或以上，估计每个饮食因素对NCD死亡率的影响，并量化不良饮食习惯对NCD死亡率的总体影响。我们还评估了饮食与社会经济发展之间的关系，并评估了饮食中疾病负担随时间的变化趋势。此分析取代之前的所有结果

研究背景本研究前的证据

我们系统地搜索了MEDLINE和全球健康数据交换（GHDx），以确定提供15种食品和营养素消费的国家或国家代表性估计的研究。我们仅包括在1980年1月1日至2016年12月31日期间收集的分析报告中包含的195个国家之一的研究报告。研究是如果使用非随机样本或特定子群体进行排除。我们通过使用全球疾病负担，伤害和风险因素研究比较风险评估方法估算每种膳食风险的潜在健康影响。

增加了本研究的价值

本研究提供了全国15种饮食因素消费的综合图景，并量化了潜力每个饮食成分的次优摄入量对195个国家的慢性病死亡率和发病率的影响。

此外，本研究还描述了饮食与发育之间的关系，并评估了1990年至2017年饮食引起的疾病负担趋势。大量摄入钠，全谷物摄入量低，水果摄入量低是主要的饮食风险因素全球和许多国家的死亡和DALYs。

所有可用证据的含义

该研究强调了在全球，区域和国家层面改善饮食的必要性。该研究结果为人口水平干预改善饮食提供了优先考虑。

使用一致的方法和定义，通过全面重新分析1990年至2017年的所有数据，对膳食风险进行GBD。

方法

选择饮食风险因素

我们选择了15个符合GBD危险因素选择标准的饮食风险因素（表）。[10-13]这些标准包括风险因素对疾病负担或政策的重要性;是否有足够的数据来估计风险因素暴露;支持风险因素暴露与疾病终点之间因果关系的流行病学证据的强度，以及数据的可用性，以量化暴露中每单位变化的这种关系的程度;和证据支持所有人群的影响的普遍性。评估每种饮食 - 疾病对的因果关系的流行病学证据强度的过程在别处描述[10-13]并在附录中进行了总结。

人口膳食摄入量

我们对科学文献进行了系统评价，以确定提供国家或国家代表性营养调查，提供每种饮食因素消费数据（附录）。我们还在全球健康数据交换网站上搜索了全国或地方代表性的营养调查和家庭预算调查。此外，对于食品集团，我们使用了Euromonitor的全国销售数据和联合国粮食及农业组织食品平衡表的国家可用性数据。对于营养素，我们使用来自全球营养数据库的国家可用数据。[20]对于钠，我们收集了24小时尿钠的数据，如果有的话。对于反式脂肪，我们使用Euromonitor的销售数据来处理氢化植物油。2017年GBD中使用的所有膳食数据来源列表均可在全球健康数据交换网站上公布。对于每个饮食因素，我们计算了数据代表性指数作为我们确定风险因素暴露数据的国家的比例（表）。

我们的饮食数据来自多个来源，并受到不同类型偏倚的影响。我们将24小时饮食召回视为评估人口水平平均摄入量的黄金标准方法，并据此调整其他来源的膳食数据（附录）。某些类型的膳食数据（即可用性，销售额和家庭数据）仅适用于所有年龄组和男女。为了将这些数据分成标准的特定年龄组和性别特定组，我们首先使用营养调查数据估算了摄入量的全球年龄和性别模式，然后使用这些模式分割可用性，销售额和家庭数据。

我们使用时空高斯过程回归方法估算每个膳食风险因子的平均摄入量，按年龄，性别，国家和年份（附录）。为了改进我们在数据稀疏模型中的估计，我们测试了各种与摄入量具有合理关系的协变量，并包括具有最佳拟合和预期方向系数的协变量（附录）。

饮食风险对疾病终点的影响大小

对于每种饮食 - 疾病对，我们使用已发表的前瞻性观察研究荟萃分析数据来估计死亡率和发病率的相对风险。[21]对于仅有发病率证据的饮食 - 疾病对，我们假设估计的相对风险也适用于死亡率（附录）。考虑到饮食和代谢风险因素的关系以及心血管疾病和2型糖尿病代谢风险相对风险的良好年龄趋势，我们使用代谢风险因素[22]的相对风险的年龄趋势来估计年龄 - 心血管疾病和2型糖尿病的饮食风险的特定相对风险（附录）。为了估计钠对结果的影响，我们首先估计了尿钠与变化之间的关系

有关附录，请参见在线

有**关全球健康数据交换的更多信息，请参阅**http://ghdx.healthdata.org

有关Euromonitor的更多信息，请参阅https://www.euromonitor.com/

有**关食物平衡表的更多信息，请参阅**http://www.fao.org/经济/ ESS / FBS / EN /

对**于全球营养数据库，请参阅**https://nutrition.healthdata.组织/全球营养数据库

有关所有膳食数据来源的列表，请参阅http://ghdx.健康数据输入源

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在收缩压，然后估计收缩压变化与疾病结果之间的关系。[14]

最佳摄入量

我们将最佳摄入水平定义为风险暴露水平，以最大限度地降低所有死亡原因的风险。为了估计每种饮食因素的最佳摄入量，我们首先根据饮食相对风险的荟萃分析中包含的研究，计算与每种疾病终点的最低死亡风险相关的摄入水平。然后，我们使用每种疾病的全球死亡比例作为权重，计算最佳摄入水平作为这些数字的加权平均值。为了反映最佳摄入水平的不确定性，我们假设均匀的不确定性分布高于和低于平均值20%。[13]对于钠，支持选择最佳摄入水平的证据是不确定的。[23,24]因此，我们在不确定性估计抽样中包括了不同最佳摄入水平的均匀分布。

疾病特异性死亡和残疾调整生命年

按年龄，性别，国家和年份分列的疾病特异性死亡和残疾调整生命年（DALYs）的数据来自GBD 2017.在其他地方详细描述了用于估计原因特异性死亡率和DALYs的GBD方法。[25,26]

疾病负担的饮食风险

我们使用GBD比较风险评估方法，按年龄，性别，国家和年份估算每种饮食 - 疾病对的人口归因分数。[10-13]然后，我们通过以下方法估算每个膳食风险因素的死亡人数和DALYs数量。将人口归因分数乘以疾病特异性死亡和DALYs的总数。

为了确定国家的发展连续性，我们使用了社会人口指数（SDI），这是一个根据人均滞后分配收入，15岁或15岁以上人口的平均受教育程度和总生育率计算的总结度量。 25岁以下女性的比率。[12,13]To

	曝光定义	最佳摄入量（最佳摄入量）录取）	数据代表性指数（%）
饮食中水果含量低	平均每日食用水果（新鲜，冷冻，煮熟，罐装或干果，不包括果汁和盐渍或腌制水果）	每天250克（200-300）	94·9
饮食中蔬菜含量低	平均每日食用蔬菜（新鲜，冷冻，煮熟，罐装或干蔬菜，不包括豆类和盐渍或腌制蔬菜，果汁，坚果，种子和淀粉类蔬菜，如土豆或玉米）	每天360克（290-430）	94·9
豆类饮食低	平均每日食用豆类（新鲜，冷冻，煮熟，罐装或干豆类）	每天60克（50-70）	94·9
全谷物饮食低	从早餐谷物，面包，米饭，意大利面，饼干，松饼，玉米饼，煎饼和其他来源平均每日消费全谷物（麸皮，胚芽和胚乳的天然比例）	每天125克（100-150）	94·9
坚果和种子的饮食低	平均每日食用坚果和种子食物	每天21克（16-25）	94·9
饮食中牛奶含量低	平均每日食用的牛奶，包括非脂肪，低脂肪和全脂牛奶，不包括豆浆和其他植物衍生物	每天435克（350-520）	94·9
饮食高红肉	平均每日食用红肉（牛肉，猪肉，羊肉和山羊，但不包括家禽，鱼，蛋和所有加工肉类）	每天23克（18-27）	94·9
加工肉类含量高的食物意味着每天食用通过吸烟，腌制，腌制或添加化学防腐剂保存的肉类		每天2克（0-4）	36·9
饮食含糖量高的饮料	平均每日消费饮料≥50kcal/ 226·8份，包括碳酸饮料，苏打水，能量饮料，果汁饮料，但不包括100%水果和蔬菜汁	每天3克（0-5）	36·9
饮食中纤维含量低	平均每日摄入的纤维来自所有来源，包括水果，蔬菜，谷物，豆类和豆类	每天24克（19-28）	94·9
饮食中钙含量低	每天平均摄入的钙来自所有来源，包括牛奶，酸奶和奶酪	每天1·25克（1·00-1·50）	94·9
海鲜ω-3脂肪酸含量低	平均每日摄入二十碳五烯酸和二十二碳六烯酸	每天250毫克（200-300）	94·9
饮食中多不饱和脂肪酸含量低	平均每日摄入所有来源的ω-6脂肪酸，主要是液体植物油，包括豆油，玉米油和红花油	每日总能量的11%（9-13）	94·9
反式脂肪酸含量高的饮食来自所有来源的反式脂肪的每日摄入量，主要是部分氢化植物油和反刍动物产品		每日总能量的0·5%（0·0-1·0）	36·9
饮食中含钠量高	24小时尿钠，以每天g计	每天3克（1-5）*	26·2

*为了反映现有证据中关于最佳钠摄入量的不确定性，每天1-5克被认为是钠的最佳水平的不确定范围，其中每天少于2-3克是摄入钠的摄入量在随机对照试验中血压水平最低，每天4-5克是观察性研究中与心血管疾病风险最低相关的钠摄入量。

表：1990 - 2017年膳食风险因子暴露定义，最佳水平和数据代表性指数

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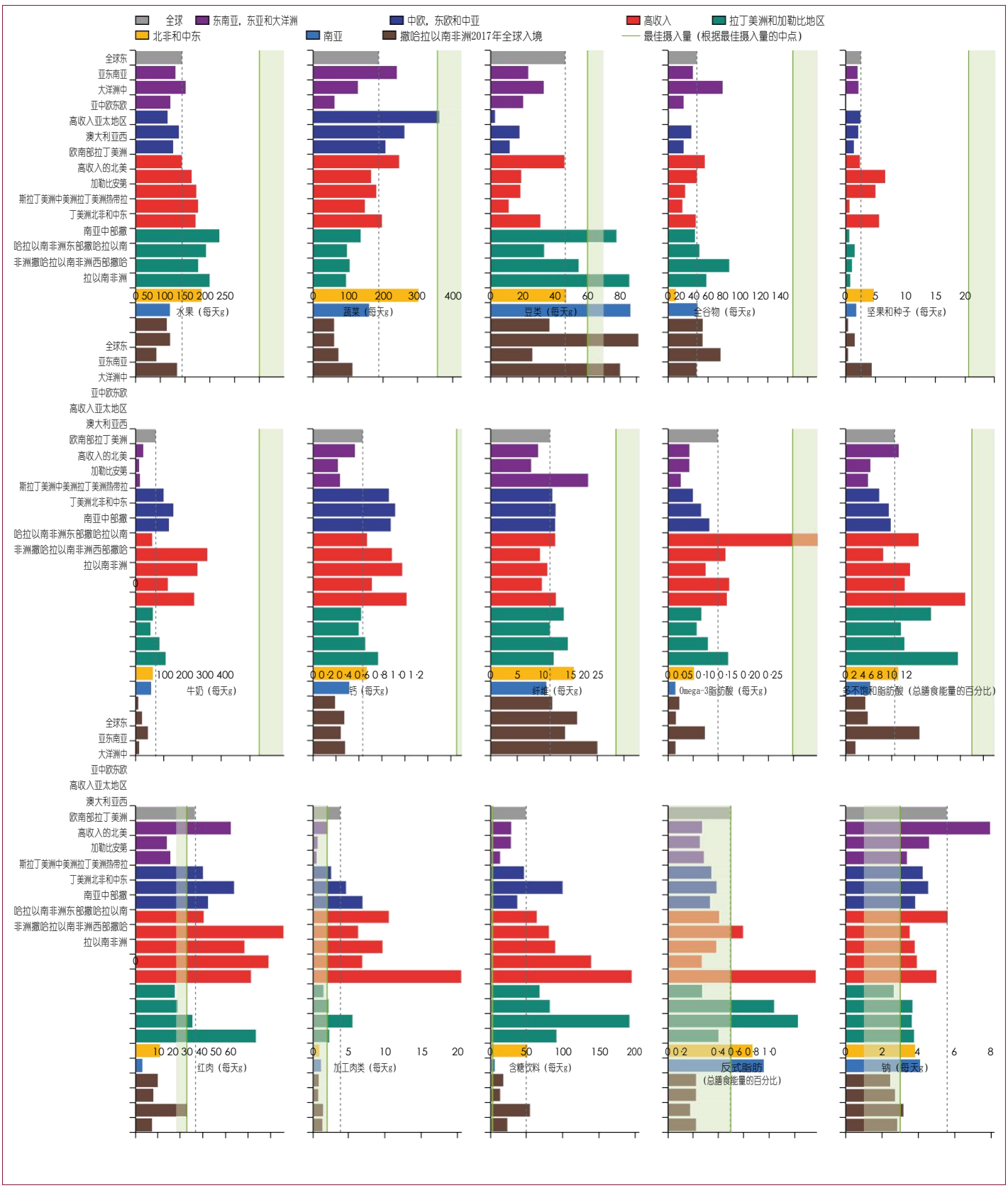

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估计每个饮食因素的摄入量或摄入量的差异，我们将每个饮食因素的当前摄入量与其最佳摄入量的中点进行比较（表）。饮食成分的高摄入量是指摄入水平高于最佳摄入量的中点，低摄入量是指摄入量低于最佳摄入量的中点。

为了结合参数的不确定性（暴露，相对风险，最佳摄入水平和死亡率）以及建模不确定性，我们遵循蒙特卡罗方法。我们在每次迭代时使用每个参数的一次绘制重复所有计算1000次。使用这1000次抽取，我们计算了最终估计的平均值和95%不确定性区间（UI）。

所有统计分析均在Python 3.5版中完成。

资金来源的作用

该研究的资助者在研究设计，数据收集，数据分析，数据解释或撰写报告方面没有任何作用。第一作者和相应的作者可以完全访问研究中的所有数据，并对提交出版的决定负有最终责任。

结果

食用主要食物和营养素

在全球范围内，2017年几乎所有健康食品和营养素的消费都不是最理想的（图1）。在坚果和种子，牛奶和全谷物中观察到当前和最佳摄入量之间的最大差距，平均消耗量为12%（95%UI 12-13；3g [2-3]坚果和
每天种子），16%（16-17;每天71克[70-72]牛奶），
和最佳水平的23%（23-23；29克[29-29]全谷物）（基于舍入前的数据计算的百分比）。与次优的健康食品消费同时，所有不健康食品和营养素的每日摄入量超过了全球最佳水平（图1）。含糖饮料（每天49克）的消费量远高于最佳摄入量。同样，加工肉类的全球消费量（每天4克[4-4]，比最佳量大90%）和钠（每天6克[5-6]，比最佳量大86%）远远高于最佳水平。全球红肉摄入量（每天27克[26-28]）比最佳摄入量高18%。与女性相比，男性健康和不健康食品的摄入量通常较高。健康和不健康食品的摄入量在中年人（50-69岁）中普遍较高，在年轻人（25 -49岁）中最低，少数例外。在年轻人中观察到含糖饮料和豆类的摄入量最高，随着年龄的增长呈下降趋势。

图1：2017年全球和区域一级25岁或以上成年人饮食因素的年龄标准摄入量

在区域一级，2017年所有健康食品的摄入量均低于所有21个GBD区域的最佳水平（图1）。唯一的例外是中亚地区的蔬菜摄入量，亚太地区高收入地区的海鲜omega-3脂肪酸，加勒比地区，拉丁美洲热带地区，南亚地区，撒哈拉以南非洲地区和撒哈拉以南非洲地区的豆类. .在不健康的食品群体中，钠和含糖饮料的消费量高于几乎每个地区的最佳水平。澳大拉西亚，拉丁美洲南部和拉丁美洲热带地区的红肉消费量最高。高收入的北美洲加工肉类摄入量最高，其次是高收入的亚太地区和西欧。在高收入的北美洲，拉丁美洲中部和拉丁美洲的安第斯山脉中观察到最高的反式脂肪摄入量。

饮食对死亡率的总体影响

在全球范围内，2017年，饮食风险造成1100万[95%UI 10-12]死亡（22%[95%UI 21-24]成人死亡总数）和2.55亿（234-274）DALYs（15%）成人中所有DALYs的[14-17];附录）。心血管疾病是饮食相关死亡（1000万[9-10]死亡）和DALYs（2.07亿[192-222] DALYs）的主要原因，其次是癌症（913 090 [743 345-1098 432]死亡和20百万[17-24] DALYs）和2型糖尿病（338 714例死亡[244 995-447 003]和2400万[16-33] DALYs）。超过500万
（95%UI 5-5）饮食相关死亡（占总饮食相关死亡的45%[43-46]）和1.77亿（163-192）与饮食相关的DALYs（占总饮食的70%[68-71]）相关的DALYs）发生在70岁以下的成年人中。

2017年，在21个GBD地区，大洋洲观察到25岁或以上成年人所有与饮食相关的死亡和DALYs的年龄标准化率最高（678 [95%UI 616-746]死亡率）每10万人口有10万人口和17 804 [16 041-19 907] DALYs;附录）。在亚太地区高收入人群中观察到成人（25岁或以上）所有与饮食有关的死亡率最低（每10万人口死亡人数为97 [89-106]），与所有与饮食有关的DALYs的最低比率为在澳大利亚观察到的情况（2182 [1955-2444] DALYs / 10万人口）。与饮食相关的心血管疾病死亡率和DALYs率最高的地区是中亚地区（每10万人死亡人数为613 [566-658]人）
ulation）和大洋洲（14 755 [13 212-16 512] DALYs / 10万人口），而在亚太地区高收入人群中观察到心血管疾病死亡率和DALYs率最低（每10万人中有68 [63-75]人死亡）人口和1443 [1329-1573] DALYs / 10万人口）。东亚的饮食相关癌症死亡率和DALY率最高（每10万人口死亡41例[34-49]，每10万人口死亡878例[736-1023]），北非和中东最低（9例[每10万人中有8-11人死亡

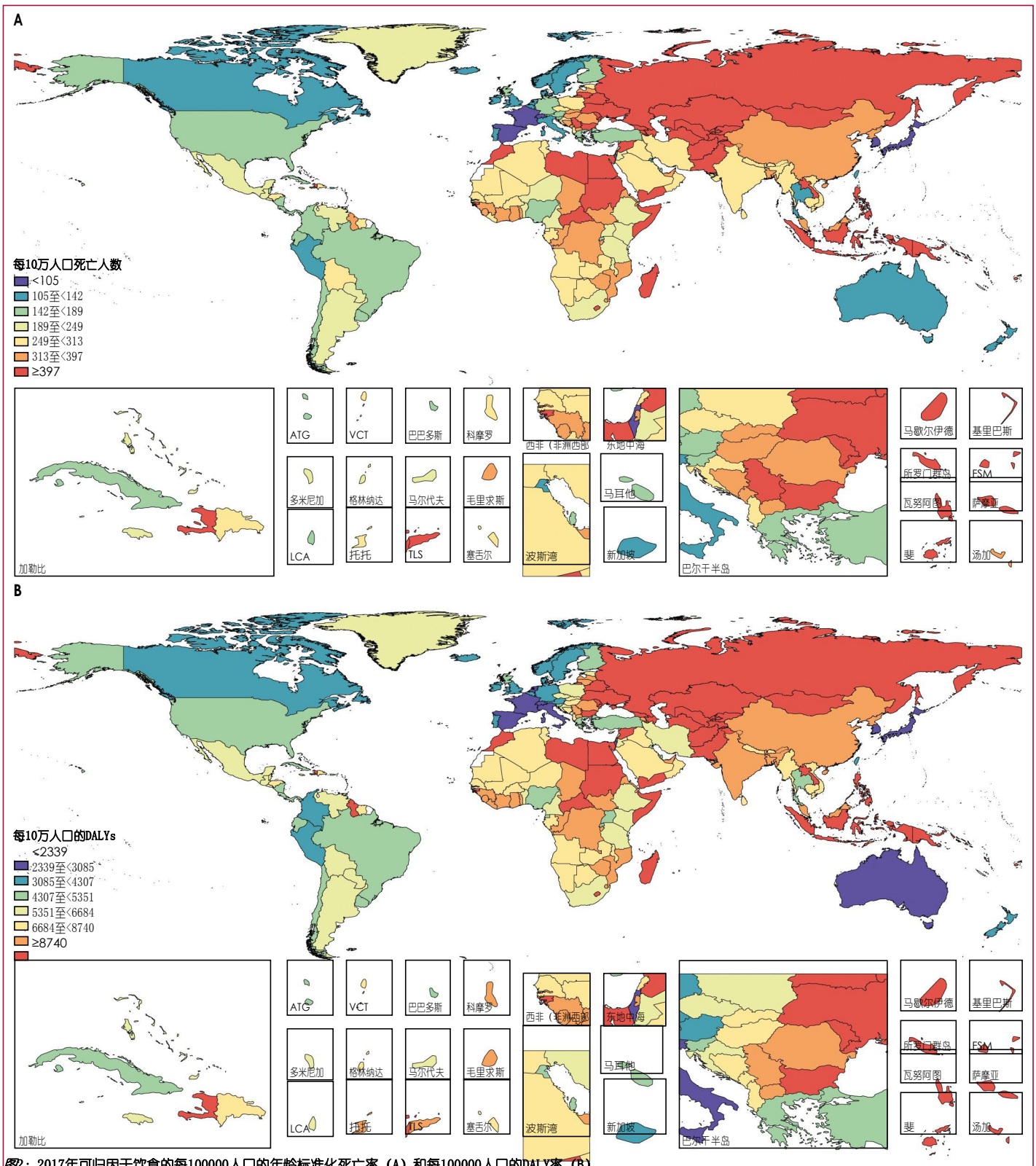

图2：2017年可归因于饮食的每100000人口的年龄标准化死亡率（A）和每100000人口的DALY率（B）

ATG =安提瓜和巴布达。ISL =群岛。FSM =密克罗尼西亚联邦。LCA =圣卢西亚。TLS =东帝汶。TTO =特立尼达和多巴哥。VCT =圣文森特和格林纳丁斯。

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和每100 000人口的203 [169-243] DALYs）。
大洋洲（每10万人口死亡人数60 [44-78]）
2426 [1737-3198]每10万人口的DALYs）与饮食相关的糖尿病死亡和DALYs的年龄标准化率最高，亚太地区的高收入率最低（每10万人中有2 [2-3]人死亡，290 [202-395] DALYs / 10万人口）。2017年，在大洋洲（死亡率的60%[95%UI 56-63]）和东亚（DALYs的64%[60-68]）观察到饮食相关死亡和心血管疾病DALYs的年龄标准化比例最高），来自东亚癌症的人（15%[13-18]的死亡和15%[12-17]的DALYs）和那些来自高收入北美的2型糖尿病患者（41%[34-48]）死亡率和DALYs的50%[42-58]；附录）。来自这些原因的死亡率和DALYs的年龄标准化比例最低的是西欧（撒哈拉以南非洲地区42%[38-45]的死亡率和44%[41-47]的DALYs）（5%[4- 6]死亡和4%[4-5] DALYs）和东南亚（29%[20-38]死亡和35%[25-46] DALYs）。
2017年，在全球20个人口最多的国家中，
埃及的所有饮食相关死亡年龄标准化率最高（552 [95%UI 490-620]死亡率）
10万人口）和DALYs（11 837 [10 525-13 268]每10万人口的DALYs）和日本所有与饮食有关的死亡率（每10万人口死亡人数为97 [89-106]）和DALYs率最低（2300 [2099-2513]每10万人口的DALYs；图2）。中国的饮食相关心血管疾病死亡年龄标准化率最高（每10万人中有299 [275-324]人死亡）
和埃及的DALY率最高（每10万人口10 811 [9577-12 209] DALYs）。中国的饮食相关癌症死亡率和DALYs率最高（每10万人口死亡42例[34-49]，每10万人口死亡889例[744-1036]），墨西哥饮食相关类型2死亡率最高糖尿病死亡和DALYs（35 [28-44]
每10万人口死亡人数和每10万人口1605 [1231-2034] DALYs）。日本的饮食相关心血管疾病死亡率和DALYs率最低（每10万人口死亡人数为69 [63-75]人和1507人）
[1389-1639] DALYs / 10万人口）和糖尿病死亡人数和DALYs（每10万人口一例死亡[1-1]，每10万人口死亡234例[161-321]）。埃及的饮食相关癌症死亡率和DALYs率最低（每10万人口中有5 [4-6]人死亡）
120 [96-146]每10万人口的DALYs；附录）。在埃及观察到25岁或以上成年人所有与饮食有关的死亡（30%[27-33]）和DALYs（23%[21-25]）的年龄标准化比例最高，且所有比例最低在尼日利亚观察到同一年龄组的饮食相关死亡（11%[9-12]）和DALYs（7%[6-8]）（附录）。2017年在巴基斯坦观察到与饮食相关的心血管疾病死亡率和DALYs比例最高（60%[95%UI 57-64]死亡率和66%[62-69] DALYs），中国癌症死亡率和DALYs（美国有16%[13-18]的死亡和15%[13-17]的DALYs）和2型糖尿病死亡和DALYs（41%）
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死亡率[34-49]和DALYs的50%[43-58]）。土耳其的心血管疾病死亡率和DALYs比例最低（42%[38-47]死亡率和44%[40-49] DALYs），癌症死亡率和DALYs在埃及（4%[3-4]死亡率和DALYs的3%[3-4]，以及孟加拉国的2型糖尿病死亡和DALYs（25%[17-34]的死亡率和34%[23-45]的DALYs）。

饮食中各个成分对死亡率的影响

少数饮食风险对健康结果有很大影响。2017年，超过一半的饮食相关死亡和三分之二的饮食相关DALY可归因于高钠摄入量（300万[95%UI 1-5]死亡和7000万[34-118] DALYs），全谷物摄入量低（300万[2-4]死亡和8,200万[59-109] DALYs），水果摄入量低（200万[1-4]死亡和6500万[41-92] DALYs；数字3）。全谷物摄入量低是男性和女性DALYs的主要膳食风险因素，也是女性死亡率的主要膳食危险因素。钠在男性死亡率方面排名第一，其次是全谷物和水果。全谷物摄入量低是年轻人（25 -50岁）死亡和DALYs的主要风险，钠在老年人（≥70岁）中排名第一。
2017年，在21个GBD地区，饮食整体低
谷物是死亡（16个地区）和DALYs（17个地区；图4）最常见的主要膳食危险因素。高钠饮食是东亚和高收入亚太地区死亡和DALYs的主要膳食风险因素（附录）。在撒哈拉以南非洲南部，水果含量低，拉丁美洲中部地区，坚果和种子含量低的饮食是导致2017年死亡和DALYs比例最高的饮食风险因素。
在中国，日本和泰国，大量摄入钠是导致死亡和DALYs的主要饮食风险。在美国，印度，巴西，巴基斯坦，尼日利亚，俄罗斯，埃及，德国，伊朗和土耳其，低谷物摄入量是导致死亡和DALYs的主要膳食风险因素。在孟加拉国，低水果摄入量是与死亡和DALY相关的主要饮食风险。在墨西哥，坚果和种子摄入量低，与饮食相关的死亡和DALYs排名第一。红肉，加工肉，反式脂肪和含糖饮料的高消费量在大多数高人口国家的死亡和DALY的膳食风险排名中排在最后（附录）。

饮食与SDI的关系

总体而言，2017年，在中低SDI国家中观察到所有与饮食相关的死亡和DALYs的年龄标准化率最高（344 [95%UI 319-369]死亡率
10万人口和7797 [7265-8386] DALYs / 10万人口）和高中SDI国家（347 [324-369]人均死亡人数为每10万人和6998人）
[6534-7454] DALY超过100,000人口；附录）。

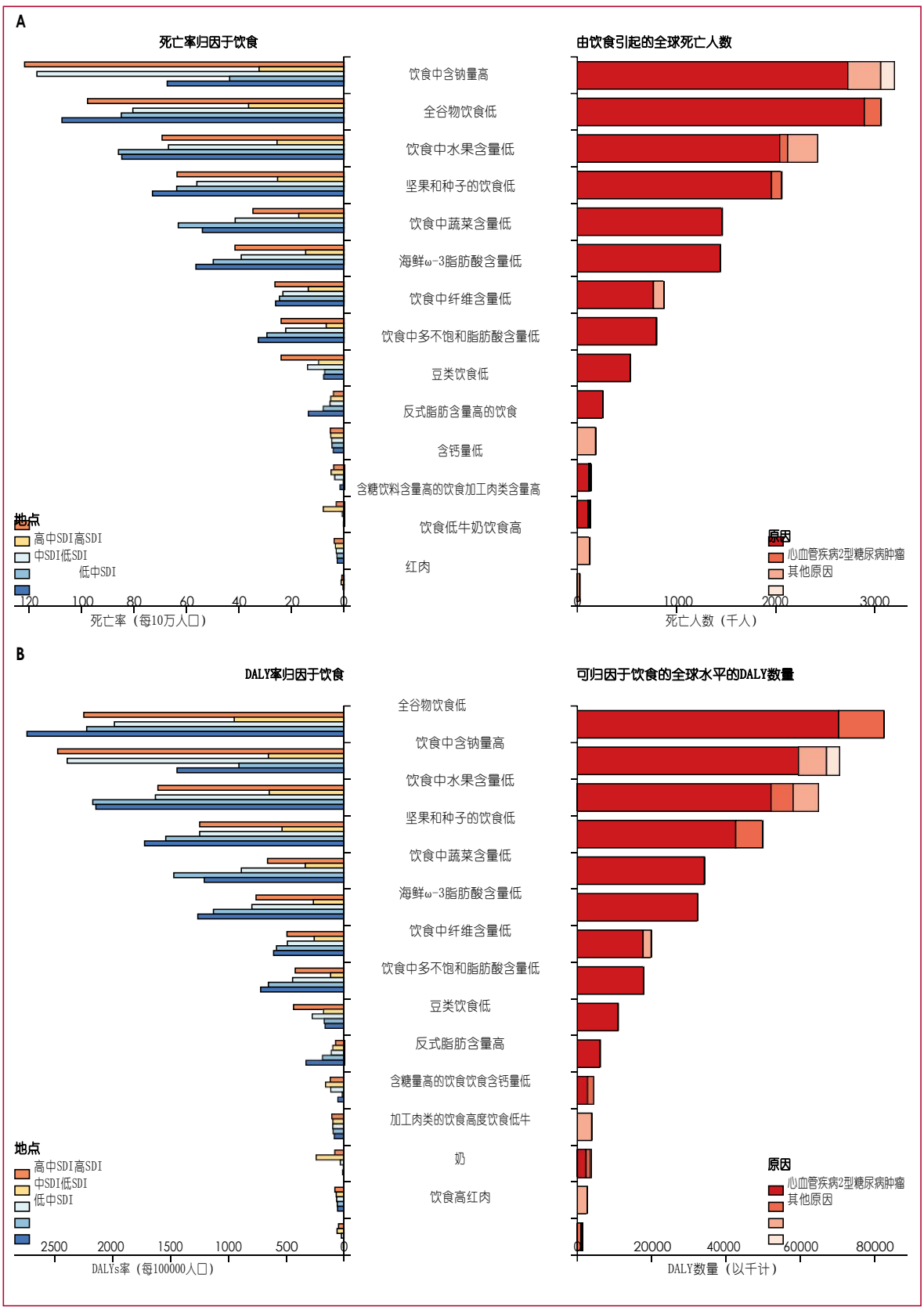

图3：死亡人数和DALYs和年龄标准化死亡率2017年全球和SDI水平的个人饮食风险导致的比率和DALY率（每10万人口）

DALY =伤残调整生命年。SDI =社会人口指数。

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图4：2017年全球和区域层面个人饮食风险导致的死亡率和DALY的年龄标准化比例
DALYs =残疾调整生命年。

在高SDI国家中观察到饮食风险的最低负担（每100 000人口死亡139 [129-148]人，每10万人口死亡3032 [2802-3265]）。中低SDI的年龄标准化饮食相关死亡率和DALYs率最高心血管疾病（每10万人中有311 [288-335]人死亡，6685 [6228-7161] DALYs 10万人口）和糖尿病（14 [10-18]人死亡每10万人口和每10万人口681 [477-914] DALYs）。高中SDI最高

年龄标准化的与饮食相关的癌症死亡率（每10万人口中有29［24-34］人死亡）
630［529-731］DALYs / 10万人口）。年龄标准化的饮食相关死亡率和心血管疾病DALYs的最低标准化率（每100 000人口有113［104-122］人死亡和2156［2005-2306］DALYs
每10万人口）和糖尿病（5［4-6］人死亡）
在高SDI国家中观察到每10万人口和每10万人444［324-587］DALYs），在低SDI国家中观察到最低的癌症死亡率（每10万人口中有15［12-17］人死亡）
和每10万人口中的324［268-376］DALYs）。在高中SDI国家中观察到与所有原因相关的饮食相关死亡和DALYs比例最高（29％[95％UI 27-31]死亡率和19％[17-21] DALYs），最低比例在低SDI国家中观察到与饮食相关的死亡（16％[15-17]死亡），在高SDI国家中观察到DALYs的比例最低（DALYs的10％[9-11]；附录）。饮食风险造成心血管疾病死亡率的55％[51-59]和SDI中期国家DALYs的60％[56-63]，心血管疾病死亡率的46％[42-49]和49％[46-52]心血管疾病DALYs在高SDI国家。中等SDI国家的癌症死亡比例最高（12％[10-14]）和DALYs（11％[9-13]），高SDI国家死亡率最低（8％[7-9]　）和DALYs（7％[6-9]）。在高SDI国家中观察到由饮食引起的糖尿病负担最高（死亡率为35％[28-43]，DALYs为46％[38-55]），在低SDI国家中观察到最低的归因负担（31％[22-39]死亡和39％[29-50] DALYs）。
高中，中等SDI国家参加了此次会议
高消费钠导致的死亡和DALYs风险最大，而高中低SDI国家的全谷物饮食风险最高（图3）。在低SDI国家，低水果摄入量是导致死亡的主要膳食风险，全谷物摄入量低是DALYs的主要膳食风险。除SDI低以外，各级SDI的国家都有相同的四种主要饮食风险：高钠，低全谷，低果，低坚果和种子。低SDI国家的四大主要饮食风险是低谷物，低果实，坚果和种子低，蔬菜含量低。

营养转变对饮食风险的影响

自1990年以来，由于饮食风险导致的死亡人数（800万[95％UI 7-8]死亡）和DALYs（184［172-197］DALYs）显着增加至1,100万（10-12）人死亡和2.55亿（ 234-274）2017年的DALYs（附录）。这一增长的主要原因是人口增长和人口老龄化。在消除了人口增长和人口老龄化的影响后，年龄标准化的归因死亡率和DALY率在1990年之间显着下降
和2017年;从406（381-430）死亡人数10万人口
每10万人中有275人（258-292人）死亡，并且
从8536（8063-9013）DALYs每10万人口到
6080（5685-6472）DALYs / 10万人口。这种减少似乎主要是由于背景死亡率的下降，因为在同一时期，与饮食风险相关的死亡和DALYs比例保持相对稳定。

讨论

我们对195个国家的膳食消费模式进行了系统评估，全面了解了人口不良饮食习惯对健康的影响。我们发现改善饮食可能会阻止全球每五个人中就有一人死亡。我们的研究结果表明，与许多其他风险因素不同，饮食风险影响人们，无论其年龄，性别和居住地的社会人口学发展如何。虽然不同国家的个体饮食因素的影响各不相同，但三种饮食因素（全谷物，水果和钠）的非最佳摄入量占死亡人数的50％以及可归因于饮食的DALYs的66％。

我们的研究结果表明，次优饮食造成的死亡人数超过全球任何其他风险，包括吸烟，[11,12]突出了改善各国人类饮食的迫切需要。虽然钠，糖和脂肪是过去二十年饮食政策辩论的主要焦点，[27,28]我们的评估显示，死亡的主要饮食风险因素是钠含量高，全谷物含量低，果实含量低坚果和种子含量低，蔬菜含量低，ω-3脂肪酸含量低;每个人占全球死亡人数的2％以上。这一发现表明，饮食政策侧重于促进当前摄入量低于最佳水平的饮食成分的摄入可能比仅针对糖和脂肪的政策产生更大的影响，强调需要采取全面的食物系统干预措施来促进这些食品的生产，分配和消费跨国。

过去十年，一系列的有效性
已经系统地评估了人口水平的饮食干预措施，并确定了几个有希望的干预措施。[29-31]这些措施包括大众媒体宣传活动，食品和菜单标签，食品定价策略（补贴和税收），学校采购政策和工地健康计划。这些干预措施的成本效益分析表明，针对特定饮食因素（如钠）可能不仅具有成本效益，而且可以节省成本。[32-35]然而，通过人口水平干预改善饮食面临着几项重大挑战。首先，大多数这些饮食干预措施的观察效果远低于在全球范围内实现最佳饮食所需的水平。[29,30]其次，几乎没有证据表明这些干预措施对几个重要措施的有效性。

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饮食因素（即坚果，全麦，海鲜，红肉和加工肉）。第三，膳食干预的成本效益分析通常基于一系列简化假设，并未考虑消费者的反应（例如替代效应），食品工业（例如食品重组和定价策略）等。现实世界中的利益相关者。[32-35]第四，尽管实施其中一些政策（例如反式禁令）的公众和政治意愿日益增强，但很少有国家成功地采用和实施这些政策。[36,37]第五，许多这些政策仅针对消费者，而不是整个食品系统中存在的各种相互关联的因素，如食品生产，加工和分销。事实上，这些因素可能会影响饮食消费，因此将其纳入改善饮食非常重要。[38,39]因此，鉴于饮食引起的疾病负担的严重程度以及现有干预措施的局限性，新食品系统的发展迫切需要干预。

我们的结果表明需要进行广泛的改变全球，区域和国家各级食品系统的各个部门，以改善饮食。如果做得不好，农业实践的变化可能会引起人们对气候变化，生物多样性丧失，土地和土壤退化以及淡水枯竭的潜在环境影响的担忧。[40-43]过去十年中出现了越来越多的证据表明将不健康的动物性食物（例如，红肉和加工肉类）的饮食转移到健康的植物性食物（例如，水果，蔬菜和全谷物）可能与较低的温室气体排放相关，因此可能更加环境可持续。[40-43]评估从动物饮食到植物性饮食转变的其他环境影响的少数研究也表明，这种转变可能与较低的土地利用和水足迹有关。[41]但是，由于这些研究中的方法和研究问题以及对各国膳食消费模式的可靠估计的稀缺性，对环境影响的综合评估o迄今为止，还没有可能实现全球最佳饮食。GBD估计每年195个国家的主要食物和营养素的膳食消费量。这些数据提供了一个独特的机会，以一致和可比的方式量化全球，区域和国家层面当前膳食消费模式的环境负担。此外，这些数据可能用于评估各种食物系统干预措施对人类健康和环境的影响。[42]

我们的研究也证明了全国的差距关于世界不同地区关键食物和营养素摄入量的代表性个人水平数据，强调了建立关键膳食危险因素国家监测和监测系统的重要性。[17,18]例如，尽管许多国家收集关于水果和蔬菜摄入量的数据，关于钠等特定营养素摄入量的数据很少。粮农组织/世卫组织全球个人食物消费数据工具[44]旨在解决这一问题，但仍存在若干重要差距。在缺乏可靠的生物标志物或更准确的膳食评估方法的情况下，24小时饮食回忆或饮食记录仍然是膳食评估的黄金标准方法。然而，验证研究的证据表明，由于回忆偏差或潜在的社会期望，它对食物和营养素的评估不是很可靠。[45,46]这一证据强调了开发和验证创新饮食评估方法的必要性。在过去的十年中，已经开发出新的方法;然而，它们尚未被广泛使用，其有效性尚未得到系统评估。[47]此外，准确估算营养素（如纤维，钙和多不饱和脂肪酸）仍然是一项重大挑战。许多国家没有当地的食物成分表，并依赖其他国家食物成分表中的数据（例如，美国农业部食品成分表）。此外，混合菜肴的配方以及食品的配方，特别是其脂肪，糖和钠的含量，因国家和时间的不同而不同，这使得对营养素的真实摄入量的估计更具挑战性。

我们对流行病学证据的系统评估显示了现有饮食相对风险的几个重要限制。膳食危险因素对疾病终点的影响大小主要来自前瞻性观察研究的荟萃分析。虽然许多这些饮食相对风险已针对主要混杂因素（例如，年龄，性别，吸烟和身体活动）进行了调整，但不能排除残留混杂的可能性。为了消除能量摄入的影响作为潜在的混淆因素并解决饮食评估工具中的测量误差，大多数队列已经在他们的统计模型中调整了总能量摄入量。这种能量调节意味着饮食成分被定义为饮食份额的风险，而不是绝对暴露水平。换句话说，食物和常量营养素摄入量的增加应该通过减少其他饮食因素的摄入量来补偿，以保持总能量摄入量不变。因此，饮食中每种成分的相对变化风险取决于其被替代的其他成分。然而，通过队列研究的荟萃分析估计的相对风险通常不指定替代类型。饮食因素（如全谷物）的定义也因研究而异。此外，鉴于健康饮食因素的摄入量通常彼此正相关且与有害饮食因素呈负相关，个体饮食因素的影响大小可能被高估。许多用于估计相对风险的观察性研究没有纠正膳食测量误差的风险评估，有些已经调整了

因果路径的因素。虽然许多队列研究已经收集了饮食数据，但只有少数人发表了他们的评估结果，这增加了发表偏倚的可能性。这些限制突出了协作努力收集和协调来自队列研究的所有可用饮食数据的需要，并对每个饮食 – 疾病对进行汇总分析，并在调整同一组混杂因素后量化效应大小。

在解释和使用我们的研究结果时，还应考虑其他潜在的限制。我们没有评估其他形式的营养不良（即营养不足和肥胖）的影响。支持饮食风险和疾病终点之间因果关系的流行病学证据主要来自观察性研究，证据强度通常弱于支持其他既定风险因素（如烟草使用和高收缩血液）之间因果关系的证据强度压力）和慢性病。此外，证据的强度因食物和营养素而异。膳食数据来自混合来源，并非适用于所有国家。这些因素增加了我们对饮食风险暴露的估计的统计不确定性。对于钠，我们没有包括来自尿液样本的数据，这导致钠的数据代表性指数低于其他饮食风险。在估算饮食的NCD负担时，我们假设饮食因素的分布在每个分析单位（即国家，年龄和性别组）中是独立的，这可能导致低估或高估饮食的综合影响。因素。为了量化饮食因素相关性的影响，我们使用来自美国国家健康和营养检查调查的个体水平数据，并估计饮食风险的总体负担（即，联合人群可归因部分），考虑和不考虑其相关性。联合人口归因分数的绝对差异平均小于2%。此外，由于一些饮食风险因素导致的死亡可能不是相互排斥的，这可能导致高估饮食引起的疾病负担。

总之，我们发现饮食习惯不良与一系列慢性病相关，可能是全球所有国家NCD死亡率的主要原因。这一发现强调了协调全球努力提高人类饮食质量的迫切需要。鉴于饮食行为的复杂性和对饮食的广泛影响，改善饮食需要整个食物系统中各种行为者的积极合作，以及针对食品系统的多个部门的政策。

GBD 2017饮食合作者

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AAf准备了初稿。KAF和KMC构建了数字和表格。AAf和PJS开发了饮食风险模型。CJLM提供了全面指导。JSS管理该项目。AAf和ECM根据其他作者的评论和评论者的反馈最终确定了稿件。所有其他作者提供了数据，审查了结果，或审查并撰写了论文。

利益声明

JMG报告联合利华的拨款。LJ报告了Mills Scientific Council的个人费用。SL报告Amgen的个人费用，
Berlin-Chemie, Merck Sharp & Dohme (MSD), Novo Nordisk, Sanofi-Aventis, Synlab, Unilever和Upfield，以及Preventicus的非财务支持。SL还是德国营养学会科学委员会的成员，也是德国营养学会基于证据的脂肪摄入和预防营养相关疾病指南的合着者。WMä报告来自Siemens Diagnostics, Aegerion Pharmaceuticals, Amgen, AstraZeneca, Danone Research, Pfizer, BASF, Numares AG和Berlin-Chemie的赠款和个人费用；Hoffmann LaRoche, MSD, Sanofi和Synageva的个人费用；来自雅培诊断的资助；并与Synlab Holding Deutschland GmbH合作。WMe目前是联合国人口基金秘鲁国家办事处 – 人口基金的人口与发展方案分析员，该机构不一定赞同这项研究。RMi报告了美国国立卫生研究院，比尔和梅林达盖茨基金会和联合利华的赠款；和世界银行和邦吉的个人费用。DM报告来自美国国立卫生研究院和比尔及梅林达盖茨基金会的研究经费；来自GOED, DSM的个人费用，
营养影响，波洛克通讯，Bunge, Indigo农业，Amarin, Acasti Pharma和美国的测试厨房；Elysium Health（有股票期权），Omada Health和DayTwo的科学顾问委员会角色；来自UpToDate的章节版税。此外，DM被列为授予Tufts University（Somerville, MA, USA；未经许可）的专利US8889739和US9987243的共同发明人，用于
反式棕榈油酸预防和治疗胰岛素抵抗，2型糖尿病和相关病症，以及降低代谢风险因素。CDR报告乳品管理学院的个人费用。AES报告来自IEM，诺华，施维雅和雅培的个人费用。
AGT报告澳大利亚国家健康与医学研究委员会的拨款。所有其他作者均未声明任何竞争利益。

致谢

HB的工作得到了伊朗Sari的Mazandaran医学科学大学的资助。AB由加拿大公共卫生署支持。JCF通过项目UID / Multi / 50016/2013承认FundaçãoparaaCiênciaeaTecnologia的资金支持。TL得到芬兰科学院的支持（拨款号码287488和号码319200）。SL的工作由德国联邦教育和研究部（nutriCARD；拨款协议号01EA1411A）资助。TM承认德国耶拿，哈勒和莱比锡的营养和心血管健康能力集群（nutriCARD）提供了额外的机构支持。AES支持
南非医学研究委员会和国家研究基金会的南非研究主席倡议。
AGT承认国家健康与医学研究委员会（澳大利亚）提供的奖学金。ST的工作得到了教育和欧洲文化基金会（IPEP），Sara Borrell博士后计划（参考编号CD15 / 00019，来自西班牙Salud Carlos III研究所）和Fondos Europeo de Desarrollo Regional（FEDER）的支持。TW承认拉贾拉塔大学医学系医学系的学术支持，
斯里兰卡；世界神经病学联合会；和世界神经康复联合会。JHW得到了新南威尔士大学（澳大利亚）的Scientia奖学金的支持。我们感谢
Pauline Kim和Adrienne Chew负责编辑协助。

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Health effects of dietary risks in 195 countries, 1990–2017: a systematic analysis for the Global Burden of Disease Study 2017

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Summary

Background Suboptimal diet is an important preventable risk factor for non-communicable diseases (NCDs); however, its impact on the burden of NCDs has not been systematically evaluated. This study aimed to evaluate the consumption of major foods and nutrients across 195 countries and to quantify the impact of their suboptimal intake on NCD mortality and morbidity.

Methods By use of a comparative risk assessment approach, we estimated the proportion of disease-specific burden attributable to each dietary risk factor (also referred to as population attributable fraction) among adults aged 25 years or older. The main inputs to this analysis included the intake of each dietary factor, the effect size of the dietary factor on disease endpoint, and the level of intake associated with the lowest risk of mortality. Then, by use of disease-specific population attributable fractions, mortality, and disability-adjusted life-years (DALYs), we calculated the number of deaths and DALYs attributable to diet for each disease outcome.

Findings In 2017, 11 million (95% uncertainty interval [UI] 10–12) deaths and 255 million (234–274) DALYs were attributable to dietary risk factors. High intake of sodium (3 million [1–5] deaths and 70 million [34–118] DALYs), low intake of whole grains (3 million [2–4] deaths and 82 million [59–109] DALYs), and low intake of fruits (2 million [1–4] deaths and 65 million [41–92] DALYs) were the leading dietary risk factors for deaths and DALYs globally and in many countries. Dietary data were from mixed sources and were not available for all countries, increasing the statistical uncertainty of our estimates.

Interpretation This study provides a comprehensive picture of the potential impact of suboptimal diet on NCD mortality and morbidity, highlighting the need for improving diet across nations. Our findings will inform implementation of evidence-based dietary interventions and provide a platform for evaluation of their impact on human health annually.

Funding Bill & Melinda Gates Foundation.

Published **Online**
April 3, 2019
http://dx.doi.org/10.1016/S0140-6736(19)30041-8

See **Online/Comment**
http://dx.doi.org/10.1016/S0140-6736(19)30500-8

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Introduction

The relationship between dietary habits and chronic non-communicable diseases (NCDs) has been extensively investigated.[1–5] Long-term randomised trials with NCD endpoints have not been feasible for most dietary factors, but synthesis of other lines of epidemiological evidence, including long-term prospective observational studies and short-term trials of intermediate outcomes, have provided supporting evidence for potential causal relationships between specific dietary factors (eg, fruits, vegetables, processed meat, and trans fat intake) and NCDs (ischaemic heart disease, diabetes, and colorectal cancer).[2–7] These findings have been widely used to inform national and international dietary guidelines aimed at preventing NCDs.[8,9] However, because of the complexities of characterising dietary consumption across different nations, assessment of the health effects of suboptimal diet at the population level has not been possible.

In the past decade, efforts have been made to quantify the burden of disease attributable to specific dietary factors.[10–19] These efforts, although useful, had several important limitations, including insufficient geographically representative data on dietary consumption, inaccurate characterisation of population distribution of dietary intake, insufficient accounting for biases of different sources of dietary assessment, standardisation of the intake to 2000 kcal per day, and insufficient accounting for within-person variation of intake of dietary factors.

To address these limitations, as part of the Global Burden of Diseases, Injuries, and Risk Factors Study (GBD) 2017, we systematically collected geographically representative dietary data from multiple sources, characterised the population distribution of intake for 15 foods and nutrients among adults aged 25 years or older across 195 countries, estimated the effect of each individual dietary factor on NCD mortality, and quantified the overall impact of poor dietary habits on NCD mortality. We also evaluated the relationship between diet and socioeconomic development, and assessed the trends in disease burden of diet over time. This analysis supersedes all previous results from

Research in context

Evidence before this study

We systematically searched MEDLINE and the Global Health Data Exchange (GHDx) to identify studies providing nationally or subnationally representative estimates of consumption of 15 foods and nutrients. We included only studies reporting data collected between Jan 1, 1980, and Dec 31, 2016, in one of the 195 countries included in this analysis. Studies were excluded if done with non-random samples or among specific subpopulations. We estimated the potential health effects of each dietary risk by use of the Global Burden of Diseases, Injuries, and Risk Factors Study comparative risk assessment approach.

Added value of this study

This study provides a comprehensive picture of consumption of 15 dietary factors across nations and quantifies the potential impact of suboptimal intake of each diet component on chronic disease mortality and morbidity among 195 countries. Additionally, this study characterises the relationship between diet and development and evaluates the trends in the burden of disease attributable to diet from 1990 to 2017. High intake of sodium, low intake of whole grains, and low intake of fruits were the leading dietary risk factors for deaths and DALYs globally and in many countries.

Implications of all the available evidence

This study highlights the need for improving diet at the global, regional, and national level. The findings inform priorities for population-level interventions to improve diet.

GBD with respect to dietary risks by comprehensively reanalysing all data from 1990 to 2017, using consistent methods and definitions.

Methods

Selection of dietary risk factors

We selected 15 dietary risk factors (table) that met GBD selection criteria for risk factors.[10–13] These criteria include the importance of the risk factor to either disease burden or policy; the availability of sufficient data to estimate risk factor exposure; the strength of the epidemiological evidence supporting a causal relationship between risk factor exposure and disease endpoints, and availability of data to quantify the magnitude of this relationship per unit of change in the exposure; and evidence supporting the generalisability of the effects to all populations. The process of evaluation of the strength of epidemiological evidence for the causal relationship of each diet–disease pair is described elsewhere[10–13] and summarised in the appendix.

See Online for appendix

Dietary intake at the population level

We did a systematic review of the scientific literature to identify nationally or subnationally representative nutrition surveys providing data on consumption of each dietary factor (appendix). We also searched the Global Health Data Exchange website for nationally or subnationally representative nutrition surveys and household budget surveys. Additionally, for food groups, we used national sales data from Euromonitor and national availability data from United Nations Food and Agriculture Organization food balance sheets. For nutrients, we used data on their national availability from the Global Nutrient Database.[20] For sodium, we collected data on 24 h urinary sodium, where available. For trans fat, we used sales data from Euromonitor on hydrogenated vegetable oil. The list of all dietary data sources used in GBD 2017 is publicly available at the Global Health Data Exchange website. For each dietary factor, we computed a data representativeness index as the fraction of countries for which we identified any data on the risk factor exposure (table).

For more on the **Global Health Data Exchange** see http://ghdx.healthdata.org

For more on **Euromonitor** see https://www.euromonitor.com/

For more on **food balance sheets** see http://www.fao.org/economic/ess/fbs/en/

For the **Global Nutrient Database** see https://nutrition.healthdata.org/global-nutrient-database

For the list of all **dietary data sources** see http://ghdx.healthdata.org/gbd-2017/data-input-sources

Our dietary data were from multiple sources and were affected by different types of biases. We considered 24 h diet recall as the gold standard method for assessing mean intake at the population level and adjusted dietary data from other sources accordingly (appendix). Some types of dietary data (ie, availability, sales, and household data) were only available for all-age groups and both sexes. To split these data into standard age-specific and sex-specific groups, we first estimated the global age and sex patterns of intake using data from nutrition surveys and then used those patterns to split the availability, sales, and household data.

We used the spatiotemporal Gaussian process regression method to estimate the mean intake of each dietary risk factor by age, sex, country, and year (appendix). To improve our estimates in data-sparse models, we tested a wide range of covariates with plausible relationships with intake and included the covariates with best fit and coefficients in the expected direction (appendix).

Effect size of dietary risks on disease endpoints

For each diet–disease pair, we used data from published meta-analyses of prospective observational studies to estimate the relative risk of mortality and morbidity.[21] For diet–disease pairs for which evidence was only available on morbidity, we assumed that the estimated relative risks were also applied to mortality (appendix). Considering the relationship of diet and metabolic risk factors and the well established age trend of the relative risks of metabolic risks for cardiovascular disease and type 2 diabetes, we used the age trend of the relative risks of metabolic risk factors[22] to estimate the age-specific relative risk of dietary risks for cardiovascular disease and type 2 diabetes (appendix). To estimate the impact of sodium on outcomes, we first estimated the relationship between urinary sodium and change

in systolic blood pressure, and then estimated the relationship between change in systolic blood pressure and disease outcomes.[14]

Optimal level of intake

We defined the optimal level of intake as the level of risk exposure that minimises the risk from all causes of death. To estimate the optimal intake for each dietary factor, we first calculated the level of intake associated with the lowest risk of mortality from each disease endpoint based on the studies included in the meta-analyses of the dietary relative risks. Then, we calculated the optimal level of intake as the weighted mean of these numbers using the global proportion of deaths from each disease as the weight. To reflect the uncertainty of optimal level of intake, we assumed a uniform uncertainty distribution of 20% above and below the mean.[13] For sodium, the evidence supporting the selection of the optimal level of intake was uncertain.[23,24] Therefore, we included a uniform distribution of different optimal levels of intake in the uncertainty estimation sampling.

Disease-specific deaths and disability-adjusted life-years

Data on disease-specific deaths and disability-adjusted life-years (DALYs) by age, sex, country, and year were obtained from GBD 2017. The GBD approach to estimating cause-specific mortality and DALYs has been described in detail elsewhere.[25,26]

Disease burden of dietary risks

We used the GBD comparative risk assessment approach to estimate the population attributable fraction for each diet–disease pair by age, sex, country, and year.[10–13] Then, we estimated the number of deaths and DALYs attributable to each dietary risk factor by multiplying the population attributable fraction by the total number of disease-specific deaths and DALYs.

To position countries on the development continuum, we used the Socio-demographic Index (SDI), which is a summary measure calculated on the basis of lag-distributed income per capita, mean educational attainment of individuals aged 15 years or older, and total fertility rate among women younger than 25 years.[12,13] To

	Exposure definition	Optimal level of intake (optimal range of intake)	Data representativeness index (%)
Diet low in fruits	Mean daily consumption of fruits (fresh, frozen, cooked, canned, or dried fruits, excluding fruit juices and salted or pickled fruits)	250 g (200–300) per day	94·9
Diet low in vegetables	Mean daily consumption of vegetables (fresh, frozen, cooked, canned, or dried vegetables, excluding legumes and salted or pickled vegetables, juices, nuts, seeds, and starchy vegetables such as potatoes or corn)	360 g (290–430) per day	94·9
Diet low in legumes	Mean daily consumption of legumes (fresh, frozen, cooked, canned, or dried legumes)	60 g (50–70) per day	94·9
Diet low in whole grains	Mean daily consumption of whole grains (bran, germ, and endosperm in their natural proportion) from breakfast cereals, bread, rice, pasta, biscuits, muffins, tortillas, pancakes, and other sources	125 g (100–150) per day	94·9
Diet low in nuts and seeds	Mean daily consumption of nut and seed foods	21 g (16–25) per day	94·9
Diet low in milk	Mean daily consumption of milk including non-fat, low-fat, and full-fat milk, excluding soy milk and other plant derivatives	435 g (350–520) per day	94·9
Diet high in red meat	Mean daily consumption of red meat (beef, pork, lamb, and goat, but excluding poultry, fish, eggs, and all processed meats)	23 g (18–27) per day	94·9
Diet high in processed meat	Mean daily consumption of meat preserved by smoking, curing, salting, or addition of chemical preservatives	2 g (0–4) per day	36·9
Diet high in sugar-sweetened beverages	Mean daily consumption of beverages with ≥50 kcal per 226·8 serving, including carbonated beverages, sodas, energy drinks, fruit drinks, but excluding 100% fruit and vegetable juices	3 g (0–5) per day	36·9
Diet low in fibre	Mean daily intake of fibre from all sources including fruits, vegetables, grains, legumes, and pulses	24 g (19–28) per day	94·9
Diet low in calcium	Mean daily intake of calcium from all sources, including milk, yogurt, and cheese	1·25 g (1·00–1·50) per day	94·9
Diet low in seafood omega-3 fatty acids	Mean daily intake of eicosapentaenoic acid and docosahexaenoic acid	250 mg (200–300) per day	94·9
Diet low in polyunsaturated fatty acids	Mean daily intake of omega-6 fatty acids from all sources, mainly liquid vegetable oils, including soybean oil, corn oil, and safflower oil	11% (9–13) of total daily energy	94·9
Diet high in trans fatty acids	Mean daily intake of trans fat from all sources, mainly partially hydrogenated vegetable oils and ruminant products	0·5% (0·0–1·0) of total daily energy	36·9
Diet high in sodium	24 h urinary sodium measured in g per day	3 g (1–5) per day*	26·2

*To reflect the uncertainty in existing evidence on optimal level of intake for sodium, 1–5 g per day was considered as the uncertainty range for the optimal level of sodium where less than 2·3 g per day is the intake level of sodium associated with the lowest level of blood pressure in randomised controlled trials and 4–5 g per day is the level of sodium intake associated with the lowest risk of cardiovascular disease in observational studies.

Table: **Dietary risk factor exposure definitions, optimal level, and data representativeness index, 1990–2017**


Global
Southeast Asia, east Asia, and Oceania
Central Europe, eastern Europe, and central Asia
High income
Latin America and the Caribbean
North Africa and the Middle East
South Asia
Sub-Saharan Africa
Global intake in 2017
Optimal level of intake (according to the midpoint of the optimal range of intake)
Global
East Asia
Southeast Asia
Oceania
Central Asia
Central Europe
Eastern Europe
High-income Asia Pacific
Australasia
Western Europe
Southern Latin America
High-income North America
Caribbean
Andean Latin America
Central Latin America
Tropical Latin America
North Africa and Middle East
South Asia
Central sub-Saharan Africa
Eastern sub-Saharan Africa
Southern sub-Saharan Africa
Western sub-Saharan Africa
0 50 100 150 200 250
Fruits (g per day)
0 100 200 300 400
Vegetables (g per day)
0 20 40 60 80
Legumes (g per day)
0 20 40 60 80 100 120 140
Whole grains (g per day)
0 5 10 15 20
Nuts and seeds (g per day)
0 100 200 300 400
Milk (g per day)
0 0·2 0·4 0·6 0·8 1·0 1·2
Calcium (g per day)
0 5 10 15 20 25
Fibre (g per day)
0 0·05 0·10 0·15 0·20 0·25
Omega-3 fatty acids (g per day)
0 2 4 6 8 10 12
PUFA (percentage of total dietary energy)
0 10 20 30 40 50 60
Red meat (g per day)
0 5 10 15 20
Processed meat (g per day)
0 50 100 150 200
Sugar-sweetened beverages (g per day)
0 0·2 0·4 0·6 0·8 1·0
Trans fats (percentage of total dietary energy)
0 2 4 6 8
Sodium (g per day)

estimate gaps in intake or excess of intake of individual components of diet, we compared the current intake of each dietary factor with the midpoint of its optimal range of intake (table). High intake of a dietary component refers to an intake level higher than the midpoint of the optimal range of intake, and low intake refers to an intake level lower than the midpoint of the optimal range of intake.

To incorporate the uncertainty of parameters (exposure, relative risk, optimal level of intake, and mortality) as well as modelling uncertainty, we followed a Monte Carlo approach. We repeated all calculations 1000 times using one draw of each parameter at each iteration. Using these 1000 draws, we calculated the mean and 95% uncertainty interval (UI) for the final estimates.

All statistical analyses were done in Python, version 3.5.

Role of the funding source

The funder of the study had no role in study design, data collection, data analysis, data interpretation, or writing of the report. The first author and the corresponding author had full access to all the data in the study and had final responsibility for the decision to submit for publication.

Results

Consumption of major foods and nutrients

Globally, consumption of nearly all healthy foods and nutrients was suboptimal in 2017 (figure 1). The largest gaps between current and optimal intake were observed for nuts and seeds, milk, and whole grains, with mean consumption at 12% (95% UI 12–13; 3 g [2–3] of nuts and seeds per day), 16% (16–17; 71 g [70–72] of milk per day), and 23% (23–23; 29 g [29–29] of whole grains per day) of the optimal levels (percentages calculated on the basis of data before rounding). In parallel with suboptimal healthy food consumption, daily intake of all unhealthy foods and nutrients exceeded the optimal level globally (figure 1). The consumption of sugar-sweetened beverages (49 g per day) was far higher than the optimal intake. Similarly, global consumption of processed meat (4 g [4–4] per day, 90% greater than the optimal amount) and sodium (6 g [5–6] per day, 86% greater than the optimal amount) were far above the optimal levels. The global intake of red meat (27 g [26–28] per day) was 18% greater than the optimal intake. Men generally had a higher intake of both healthy and unhealthy foods than did women. Intake of both healthy and unhealthy foods was generally higher among middle-aged adults (50–69 years) and lowest among young adults (25–49 years) with a few exceptions. The highest intake of sugar-sweetened beverages and legumes were observed among young adults and showed a decreasing trend with age.

Figure 1: **Age-standardised intake of dietary factors among adults aged 25 years or older at the global and regional level in 2017**

At the regional level, in 2017, the intake of all healthy foods was lower than the optimal level in all 21 GBD regions (figure 1). The only exceptions were the intake of vegetables in central Asia, seafood omega-3 fatty acids in high-income Asia Pacific, and legumes in the Caribbean, tropical Latin America, south Asia, western sub-Saharan Africa, and eastern sub-Saharan Africa. Among unhealthy food groups, consumption of sodium and sugar-sweetened beverages were higher than the optimal level in nearly every region. Red meat consumption was highest in Australasia, southern Latin America, and tropical Latin America. High-income North America had the highest processed meat intake followed by high-income Asia Pacific and western Europe. The highest intake of trans fats was observed in high-income North America, central Latin America, and Andean Latin America.

Overall impact of diet on mortality

Globally, in 2017, dietary risks were responsible for 11 million [95% UI 10–12] deaths (22% [95% UI 21–24] of all deaths among adults) and 255 million (234–274) DALYs (15% [14–17] of all DALYs among adults; appendix). Cardiovascular disease was the leading cause of diet-related deaths (10 million [9–10] deaths) and DALYs (207 million [192–222] DALYs), followed by cancers (913 090 [743 345–1 098 432] deaths and 20 million [17–24] DALYs) and type 2 diabetes (338 714 deaths [244 995–447 003] and 24 million [16–33] DALYs). More than 5 million (95% UI 5–5) diet-related deaths (45% [43–46] of total diet-related deaths) and 177 million (163–192) diet-related DALYs (70% [68–71] of total diet-related DALYs) occurred among adults aged younger than 70 years.

Across the 21 GBD regions, in 2017, the highest age-standardised rates of all diet-related deaths and DALYs among adults aged 25 years or older were observed in Oceania (678 [95% UI 616–746] deaths per 100 000 population and 17 804 [16 041–19 907] DALYs per 100 000 population; appendix). The lowest rates of all diet-related deaths among adults (aged 25 years or older) were observed in high-income Asia Pacific (97 [89–106] deaths per 100 000 population) and the lowest rates of all diet-related DALYs were observed in Australasia (2182 [1955–2444] DALYs per 100 000 population). The regions with the highest rates of diet-related cardiovascular disease deaths and DALYs were central Asia (613 [566–658] deaths per 100 000 population) and Oceania (14 755 [13 212–16 512] DALYs per 100 000 population), whereas the lowest rate of cardiovascular disease deaths and DALYs were observed in high-income Asia Pacific (68 [63–75] deaths per 100 000 population and 1443 [1329–1573] DALYs per 100 000 population). Diet-related cancer death and DALY rates were highest in east Asia (41 [34–49] deaths per 100 000 population and 878 [736–1023] DALYs per 100 000 population) and lowest in north Africa and the Middle East (nine [8–11] deaths per 100 000 population

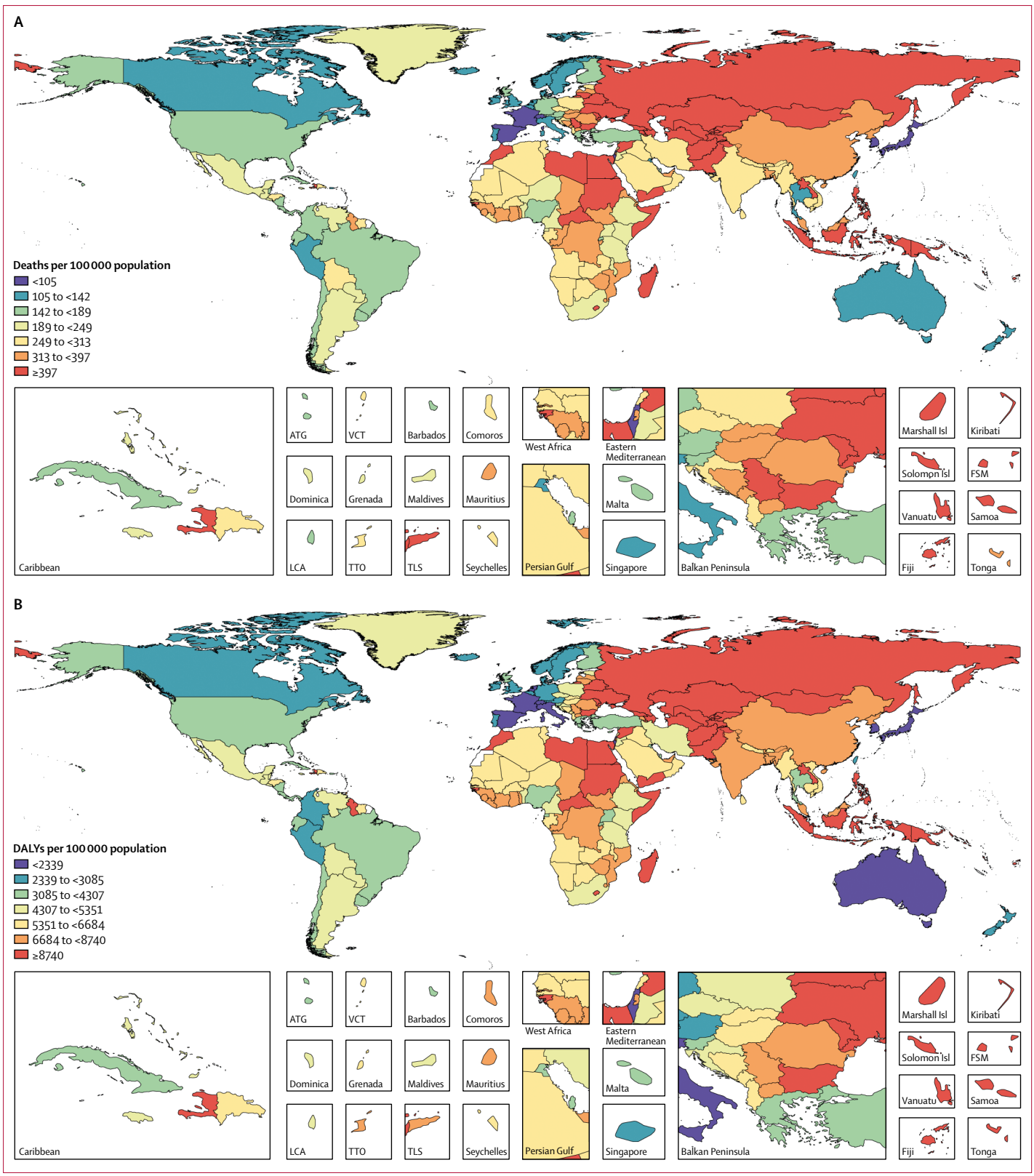

Figure 2: **Age-standardised mortality rate per 100 000 population (A) and DALY rate per 100 000 population (B) attributable to diet in 2017**
ATG=Antigua and Barbuda. Isl=Islands. FSM=Federated States of Micronesia. LCA=Saint Lucia. TLS=Timor-Leste. TTO=Trinidad and Tobago. VCT=Saint Vincent and the Grenadines.

and 203 [169–243] DALYs per 100 000 population). Oceania (60 [44–78] deaths per 100 000 population and 2426 [1737–3198] DALYs per 100 000 population) had the highest age-standardised rate of diet-related diabetes deaths and DALYs, and high-income Asia Pacific had the lowest rates (two [2–3] deaths per 100 000 population and 290 [202–395] DALYs per 100 000 population). In 2017, the highest age-standardised proportions of diet-related deaths and DALYs from cardiovascular disease were observed in Oceania (60% [95% UI 56–63] of deaths) and east Asia (64% [60–68] of DALYs), those from cancer in east Asia (15% [13–18] of deaths and 15% [12–17] of DALYs), and those from type 2 diabetes in high-income North America (41% [34–48] of deaths and 50% [42–58] of DALYs; appendix). The lowest age-standardised proportions of deaths and DALYs from these causes were in western Europe (42% [38–45] of deaths and 44% [41–47] of DALYs), western sub-Saharan Africa (5% [4–6] of deaths and 4% [4–5] DALYs), and southeast Asia (29% [20–38] of deaths and 35% [25–46] of DALYs).

In 2017, among the world's 20 most populous countries, Egypt had the highest age-standardised rate of all diet-related deaths (552 [95% UI 490–620] deaths per 100 000 population) and DALYs (11 837 [10 525–13 268] DALYs per 100 000 population) and Japan had the lowest rate of all diet-related deaths (97 [89–106)] deaths per 100 000 population) and DALYs (2300 [2099–2513] DALYs per 100 000 population; figure 2). China had the highest age-standardised rates of diet-related cardiovascular disease deaths (299 [275–324] deaths per 100 000 population) and Egypt had the highest DALY rates (10 811 [9577–12 209] DALYs per 100 000 population). China had highest rates of diet-related cancer deaths and DALYs (42 [34–49] deaths per 100 000 population and 889 [744–1036] DALYs per 100 000 population), and Mexico had the highest rates of diet-related type 2 diabetes deaths and DALYs (35 [28–44] deaths per 100 000 population and 1605 [1231–2034] DALYs per 100 000 population). Japan had the lowest rate of diet-related cardiovascular disease deaths and DALYs (69 [63–75] deaths per 100 000 population and 1507 [1389–1639] DALYs per 100 000 population) and diabetes deaths and DALYs (one [1–1] death per 100 000 population and 234 [161–321] DALYs per 100 000 population). Egypt had the lowest rate of diet-related cancer deaths and DALYs (five [4–6] deaths per 100 000 population and 120 [96–146] DALYs per 100 000 population; appendix). The highest age-standardised proportion of all diet-related deaths (30% [27–33]) and DALYs (23% [21–25]) in adults aged 25 years or older were observed in Egypt, and the lowest proportion of all diet-related deaths (11% [9–12]) and DALYs (7% [6–8]) in the same age group were observed in Nigeria (appendix). The highest proportions of diet-related cardiovascular disease deaths and DALYs in 2017 were observed in Pakistan (60% [95% UI 57–64] of deaths and 66% [62–69] of DALYs), cancer deaths and DALYs in China (16% [13–18] of deaths and 15% [13–17] of DALYs), and type 2 diabetes deaths and DALYs in the USA (41% [34–49] of deaths and 50% [43–58] of DALYs). The lowest proportions of cardiovascular disease deaths and DALYs were seen in Turkey (42% [38–47] of deaths and 44% [40–49] of DALYs), cancer deaths and DALYs in Egypt (4% [3–4] of deaths and 3% [3–4] of DALYs), and type 2 diabetes deaths and DALYs in Bangladesh (25% [17–34] of deaths and 34% [23–45] of DALYs).

Impact of individual components of diet on mortality

A small number of dietary risks had a large impact on health outcomes. In 2017, more than half of diet-related deaths and two-thirds of diet-related DALYs were attributable to high intake of sodium (3 million [95% UI 1–5] deaths and 70 million [34–118] DALYs), low intake of whole grains (3 million [2–4] deaths and 82 million [59–109] DALYs), and low intake of fruits (2 million [1–4] deaths and 65 million [41–92] DALYs; figure 3). Low intake of whole grains was the leading dietary risk factor for DALYs among men and women and the leading dietary risk factor for mortality among women. Sodium ranked first for mortality among men followed by whole grains and fruit. Low intake of whole grains was the leading risk for deaths and DALYs among young adults (aged 25–50 years) and sodium ranked first among older adults (≥70 years).

In 2017, across the 21 GBD regions, a diet low in whole grains was the most common leading dietary risk factor for deaths (in 16 regions) and DALYs (in 17 regions; figure 4). A diet high in sodium was the leading dietary risk factor for deaths and DALYs in east Asia and high-income Asia Pacific regions (appendix). In southern sub-Saharan Africa, a diet low in fruits and in central Latin America a diet low in nuts and seeds were the dietary risk factors responsible for the greatest proportion of deaths and DALYs in 2017.

High intake of sodium was the leading dietary risk for deaths and DALYs in China, Japan, and Thailand. Low intake of whole grains was the leading dietary risk factor for deaths and DALYs in the USA, India, Brazil, Pakistan, Nigeria, Russia, Egypt, Germany, Iran, and Turkey. In Bangladesh, low intake of fruits was the leading dietary risk associated with deaths and DALYs. In Mexico, low intake of nuts and seeds ranked first for diet-related deaths and DALYs. High consumption of red meat, processed meat, trans fat, and sugar-sweetened beverages were towards the bottom in ranking of dietary risks for deaths and DALYs for most high-population countries (appendix).

Relationship between diet and SDI

Overall, in 2017, the highest age-standardised rates of all diet-related deaths and DALYs were observed in low-middle SDI countries (344 [95% UI 319–369] deaths per 100 000 population and 7797 [7265–8386] DALYs per 100 000 population) and high-middle SDI countries (347 [324–369] deaths per 100 000 population and 6998 [6534–7454] DALYs per 100 000 population; appendix).

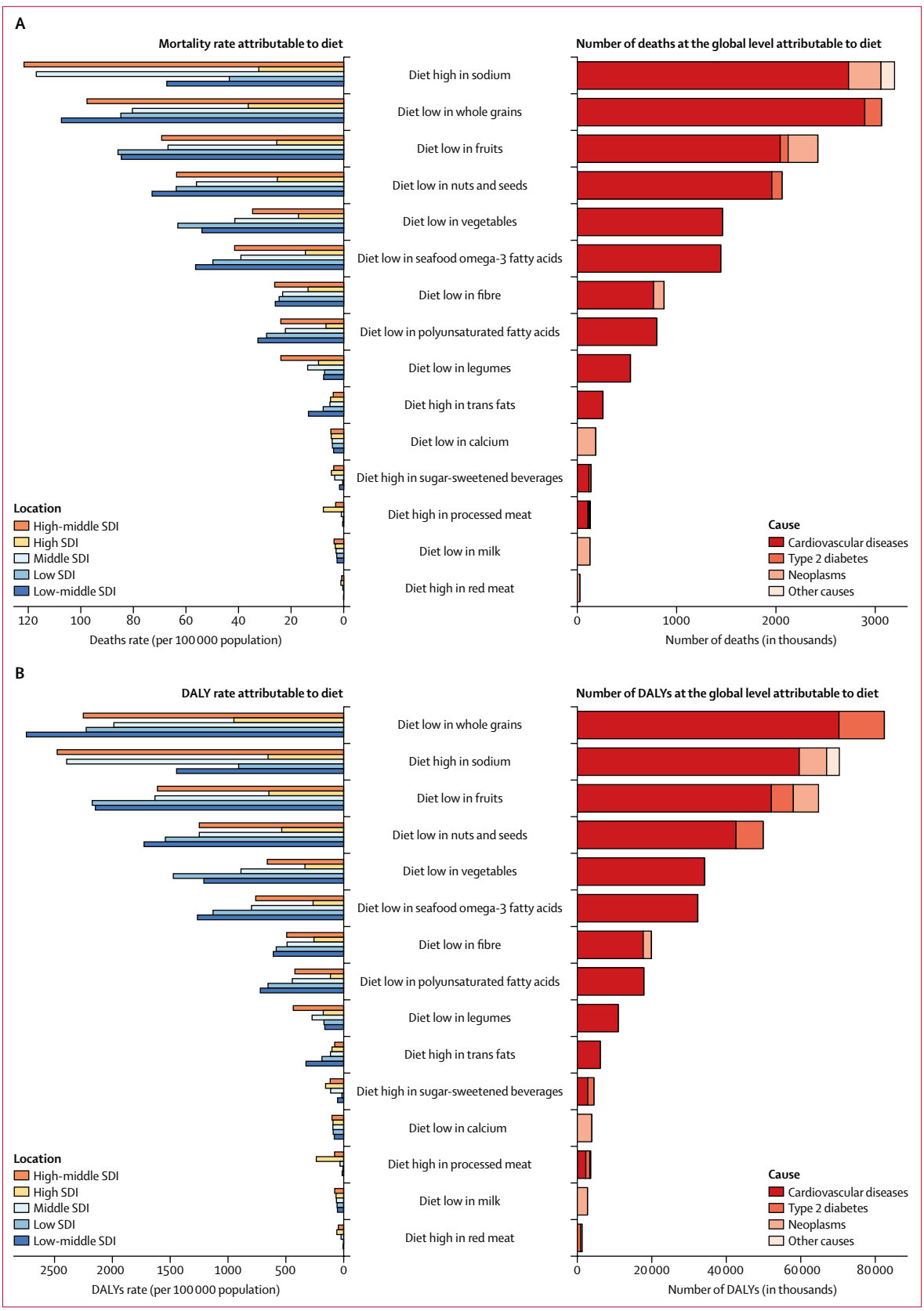

Figure 3: **Number of deaths and DALYs and age-standardised mortality rate and DALY rate (per 100 000 population) attributable to individual dietary risks at the global and SDI level in 2017**
DALY=disability-adjusted life-year. SDI=Socio-demographic Index.

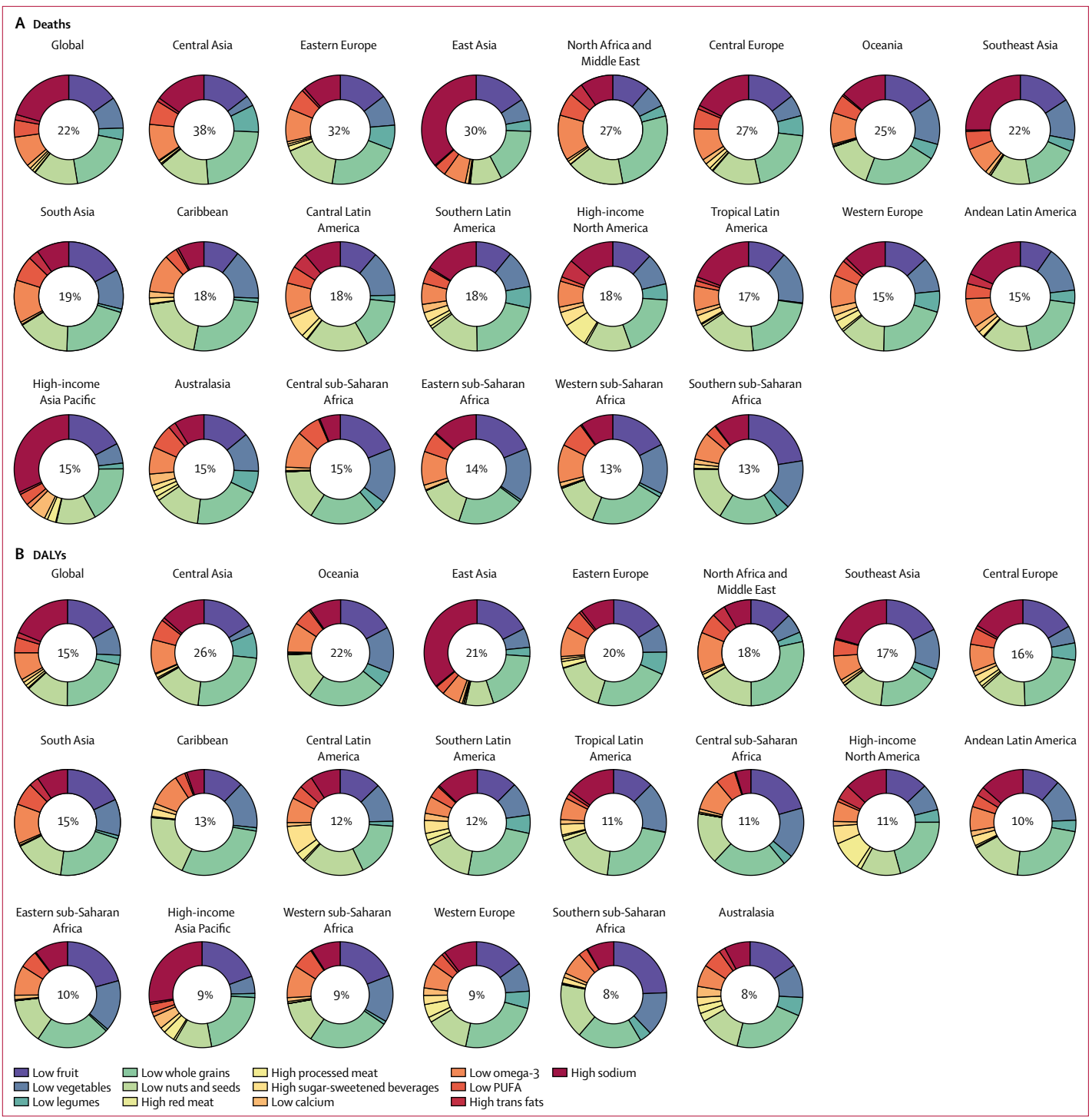

Figure 4: **Age-standardised proportions of deaths and DALYs attributable to individual dietary risks at the global and regional level in 2017**
DALYs=disability-adjusted life-years.

The lowest burden of exposure to dietary risk was observed in high SDI countries (139 [129–148] deaths per 100 000 population and 3032 [2802–3265] DALYs per 100 000 population). Low-middle SDI had the highest age-standardised rates of diet-related deaths and DALYs for cardiovascular disease (311 [288–335] deaths per 100 000 population and 6685 [6228–7161] DALYs per 100 000 population) and diabetes (14 [10–18] deaths per 100 000 population and 681 [477–914] DALYs per 100 000 population). High-middle SDI had the highest

age-standardised rates of diet-related mortality for cancer (29 [24–34] deaths per 100 000 population and 630 [529–731] DALYs per 100 000 population). The lowest age-standardised rate of diet-related deaths and DALYs for cardiovascular disease (113 [104–122] deaths per 100 000 population and 2156 [2005–2306] DALYs per 100 000 population) and diabetes (five [4–6] deaths per 100 000 population and 444 [324–587] DALYs per 100 000 population) was observed in high SDI countries and lowest mortality rate for cancer was observed in low SDI countries (15 [12–17] deaths per 100 000 population and 324 [268–376] DALYs per 100 000 population). The highest proportions of diet-related deaths and DALYs for all causes were observed in high-middle SDI countries (29% [95% UI 27–31] of deaths and 19% [17–21] of DALYs), the lowest proportion of diet-related deaths was observed in low SDI countries (16% [15–17] of deaths), and the lowest proportion of DALYs was observed in high SDI countries (10% [9–11] of DALYs; appendix). Dietary risks were responsible for 55% [51–59] of cardiovascular disease deaths and 60% [56–63] of DALYs in middle SDI countries, and 46% [42–49] of cardiovascular disease deaths and 49% [46–52] of cardiovascular disease DALYs in high SDI countries. Middle SDI countries had the highest proportion of cancer deaths (12% [10–14]) and DALYs (11% [9–13]) and high SDI countries had the lowest proportion of attributable cancer deaths (8% [7–9]) and DALYs (7% [6–9]). The highest burden of diabetes attributable to diet was observed in high SDI countries (35% [28–43] of deaths and 46% [38–55] of DALYs) and lowest attributable burden was observed in the low SDI countries (31% [22–39] of deaths and 39% [29–50] of DALYs).

High-middle and middle SDI countries were at the greatest risk of deaths and DALYs from high consumption of sodium, whereas high and low-middle SDI countries had the greatest risk caused by a diet low in whole grains (figure 3). In low SDI countries, low intake of fruit was the leading dietary risk for deaths and low intake of whole grains was the leading dietary risk for DALYs. Countries at all levels of SDI other than low SDI had the same four leading dietary risks: high sodium, low whole grains, low fruit, and low nuts and seeds. The four leading dietary risks for low SDI countries were a diet in low whole grains, low in fruit, low in nuts and seeds, and low in vegetables.

Impact of nutrition transition on exposure to dietary risks

Since 1990, the number of deaths (8 million [95% UI 7–8] deaths) and DALYs (184 [172–197] DALYs) attributable to dietary risks significantly increased to 11 million (10–12) deaths and 255 million (234–274) DALYs in 2017 (appendix). The main contributors to this increase were population growth and population ageing. After removing the effect of population growth and population ageing, the age-standardised attributable death and DALY rates showed a significant decrease between 1990 and 2017; from 406 (381–430) deaths per 100 000 population to 275 (258–292) deaths per 100 000 population, and from 8536 (8063–9013) DALYs per 100 000 population to 6080 (5685–6472) DALYs per 100 000 population. This decrease seemed to be driven mostly by decreases in the background mortality rate because, during the same period, the proportion of deaths and DALYs related to dietary risk remained relatively stable.

Discussion

Our systematic evaluation of dietary consumption patterns across 195 countries provides a comprehensive picture of the health effects of poor dietary habits at the population level. We found that improvement of diet could potentially prevent one in every five deaths globally. Our findings show that, unlike many other risk factors, dietary risks affected people regardless of age, sex, and sociodemographic development of their place of residence. Although the impact of individual dietary factors varied across countries, non-optimal intake of three dietary factors (whole grains, fruits, and sodium) accounted for more than 50% of deaths and 66% of DALYs attributable to diet.

Our findings show that suboptimal diet is responsible for more deaths than any other risks globally, including tobacco smoking,[11,12] highlighting the urgent need for improving human diet across nations. Although sodium, sugar, and fat have been the main focus of diet policy debate in the past two decades,[27,28] our assessment shows that the leading dietary risk factors for mortality are diets high in sodium, low in whole grains, low in fruit, low in nuts and seeds, low in vegetables, and low in omega-3 fatty acids; each accounting for more than 2% of global deaths. This finding suggests that dietary policies focusing on promoting the intake of components of diet for which current intake is less than the optimal level might have a greater effect than policies only targeting sugar and fat, highlighting the need for a comprehensive food system interventions to promote the production, distribution, and consumption of these foods across nations.

Over the past decade, the effectiveness of a range of population-level dietary interventions has been systematically evaluated and several promising interventions have been identified.[29–31] These include mass media campaigns, food and menu labeling, food pricing strategies (subsidies and taxation), school procurement policies, and worksite wellness programmes. Cost-effectiveness analyses of these interventions have shown that targeting specific dietary factors (eg, sodium) might not only be cost-effective but cost-saving.[32–35] However, improvement of diet through population-level interventions faces several major challenges. First, the observed effects for most of these dietary interventions are far below the level required to achieve optimal diet globally.[29,30] Second, there is almost no evidence on the effectiveness of these interventions on several important

dietary factors (ie, nuts, whole grains, seafood, red meat, and processed meat). Third, cost-effectiveness analyses of dietary interventions are generally based on a range of simplifying assumptions and do not take into account the reactions of consumers (eg, substitution effect), the food industry (eg, food reformulations and pricing strategies), and other stakeholders in the real world.[32–35] Fourth, despite the growing public and political will for the implementation of some of these policies (eg, trans fat bans), few countries have successfully adopted and implemented them.[36,37] Fifth, many of these policies only target consumers but not the wide range of interconnected factors, such as food production, processing, and distribution, that exist throughout the food system. Indeed, these factors might affect dietary consumption, and it is important to include them to improve diet.[38,39] Therefore, in view of the magnitude of the disease burden attributable to diet and the limitations of the existing interventions, development of novel food system interventions is urgently needed.

Our results show a need for extensive changes in various sectors of the food system at the global, regional, and national levels to improve diet. Changes in agricultural practices, if not done properly, might raise concerns over potential environmental effects on climate change, biodiversity loss, degradation of land and soil, and freshwater depletion.[40–43] A growing body of evidence has emerged in the past decade showing that shifting diet from unhealthy animal-based foods (eg, red meat and processed meat) to healthy plant-based foods (eg, fruits, vegetables, and whole grains) might be associated with lower emission of greenhouse gases and thus might be more environmentally sustainable.[40–43] The few studies evaluating other environmental effects of the shift from animal-based to plant-based diet have also demonstrated that this shift might be associated with lower land use and water footprint.[41] However, because of the variations in the methods and research questions across these studies and scarcity of reliable estimates on dietary consumption patterns across nations, a comprehensive assessment of environmental effects of achieving optimal diet globally has not been possible to date. GBD estimates the dietary consumption of key foods and nutrients across 195 nations annually. These data provide a unique opportunity to quantify the environmental burden of current dietary consumption patterns at global, regional, and national levels in a consistent and comparable way. Additionally, these data could potentially be used to evaluate the effect of various food system interventions on human health and environment.[42]

Our study also demonstrates the gaps in nationally representative individual-level data on intake of key foods and nutrients in different regions of the world, highlighting the importance of establishing national surveillance and monitoring systems for key dietary risk factors.[17,18] For example, although many countries collect data on fruit and vegetable intake, data on intake of specific nutrients such as sodium are scarce. The FAO/WHO Global Individual Food consumption data Tool[44] aims to address this problem, but several important gaps will remain. In the absence of reliable biomarkers or more accurate methods of dietary assessment, the 24 h diet recall or diet record remains the gold standard method of dietary assessment. However, evidence from validation studies suggests that it is not highly reliable for assessment of foods and nutrients due to recall bias or potential social desirability.[45,46] This evidence highlights the need for development and validation of innovative dietary assessment methods. In the past decade, new methods have been developed; however, they have not been widely used and their validity has not been systematically evaluated.[47] Furthermore, accurate estimation of nutrients (eg, fibre, calcium, and polyunsaturated fatty acids), remains a major challenge. Many countries do not have local food composition tables and rely on data from food composition tables from other countries (eg, US Department of Agriculture food composition tables). Additionally, the recipes of mixed dishes as well as formulation of the food products, particularly their content of fat, sugar, and sodium, varies across countries and over time, which makes estimation of the true intake of nutrient more challenging.

Our systematic evaluation of epidemiological evidence shows several important limitations in existing dietary relative risks. The effect sizes of the dietary risk factors on disease endpoints were mostly obtained from meta-analyses of prospective observational studies. Although many of these dietary relative risks have been adjusted for the major confounders (eg, age, sex, smoking, and physical activity), the possibility of residual confounding cannot be excluded. To remove the effect of energy intake as a potential confounder and address measurement error in dietary assessment tools, most cohorts have adjusted for total energy intake in their statistical models. This energy adjustment means that diet components are defined as risks in terms of the share of diet and not as absolute levels of exposure. In other words, an increase in intake of foods and macronutrients should be compensated by a decrease in intake of other dietary factors to hold total energy intake constant. Thus, the relative risk of change in each component of diet depends on the other components for which it is substituted. However, the relative risks estimated from meta-analyses of cohort studies do not generally specify the type of substitution. The definition of dietary factors (eg, whole grains) also varies across studies. Additionally, given the intake of healthy dietary factors are generally positively correlated with each other and inversely correlated with harmful dietary factors, the effect size of the individual dietary factors might be overestimated. Many of the observational studies used for estimation of the relative risks have not corrected risk estimates for dietary measurement error, and some have adjusted for

factors along the causal pathways. Although many cohort studies have collected dietary data, only a few of them have published results of their assessment, which increases the possibility of publication bias. These limitations highlight the need for a collaborative effort to collect and harmonise all available dietary data from cohort studies and to do a pooled analysis for each diet–disease pair and quantify the effect size after adjusting for the same set of confounders.

Other potential limitations should also be considered in interpreting and using the findings of our study. We did not evaluate the effect of other forms of malnutrition (ie, undernutrition and obesity). The epidemiological evidence supporting a causal relationship between dietary risks and disease endpoints were mostly from observational studies, and the strength of evidence was generally weaker than the strength of evidence supporting a causal relationship between other established risks factors (eg, tobacco use and high systolic blood pressure) and chronic diseases. Additionally, the strength of evidence varied across foods and nutrients. Dietary data were from mixed sources and were not available for all countries. These factors increase the statistical uncertainty of our estimates for exposure to dietary risks. For sodium, we did not include data from spot urine sample, which resulted in a lower data representativeness index for sodium than that of other dietary risks. In estimation of the NCD burden of diet, we assumed that the distribution of dietary factors is independent within each unit of analysis (ie, country, age, and sex group), which might have resulted in underestimation or overestimation of the combined effect of dietary factors. To quantify the effect of correlation of dietary factors, we used individual-level data from the US National Health and Nutrition Examination Survey and estimated the overall burden of dietary risks (ie, joint population attributable fractions) with and without taking into account their correlation. The absolute difference in the joint population attributable fractions, on average, was less than 2%. Additionally, deaths due to some dietary risk factors might not be mutually exclusive, which could result in overestimation of the burden of disease attributable to diet.

In summary, we found that poor dietary habits are associated with a range of chronic diseases and can potentially be a major contributor to NCD mortality in all countries worldwide. This finding highlights the urgent need for coordinated global efforts to improve the quality of human diet. Given the complexity of dietary behaviours and the wide range of influences on diet, improving diet requires active collaboration of a variety of actors throughout the food system, along with policies targeting multiple sectors of the food system.

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AAf prepared the first draft. KAF and KMC constructed the figures and tables. AAf and PJS developed models for dietary risks. CJLM provided overall guidance. JSS managed the project. AAf and ECM finalised the manuscript on the basis of comments from other authors and reviewer feedback. All other authors provided data, reviewed results, or reviewed and contributed to the paper.

Declaration of interests

JMG reports grants from Unilever. LJ reports personal fees from Mills Scientific Council. SL reports personal fees from Amgen, Berlin-Chemie, Merck Sharp & Dohme (MSD), Novo Nordisk, Sanofi-Aventis, Synlab, Unilever, and Upfield, and non-financial support from Preventicus. SL is also a member of the Scientific Board of the German Nutrition Society and a coauthor of the evidence-based guideline Fat Intake and Prevention of Nutrition-Related Diseases of the German Nutrition Society. WMä reports grants and personal fees from Siemens Diagnostics, Aegerion Pharmaceuticals, Amgen, AstraZeneca, Danone Research, Pfizer, BASF, Numares AG, and Berlin-Chemie; personal fees from Hoffmann LaRoche, MSD, Sanofi, and Synageva; grants from Abbott Diagnostics; and employment with Synlab Holding Deutschland GmbH. WMe is currently a program analyst for Population and Development at the Peru Country Office of the United Nations Population Fund-UNFPA, an institution that does not necessarily endorse this study. RMi reports grants from the US National Institutes of Health, Bill & Melinda Gates Foundation, and Unilever; and personal fees from World Bank and Bunge. DM reports research funding from the US National Institutes of Health and the, Bill & Melinda Gates Foundation; personal fees from GOED, DSM, Nutrition Impact, Pollock Communications, Bunge, Indigo Agriculture, Amarin, Acasti Pharma, and America's Test Kitchen; scientific advisory board roles with Elysium Health (with stock options), Omada Health, and DayTwo; and chapter royalties from UpToDate. In addition, DM is listed as a co-inventor on patents US8889739 and US9987243 issued to Tufts University (Somerville, MA, USA; unlicensed) for use of trans-palmitoleic acid to prevent and treat insulin resistance, type 2 diabetes, and related conditions, as well as reduce metabolic risk factors. CDR reports personal fees from Dairy Management Institute. AES reports personal fees from IEM, Novartis, Servier, and Abbott. AGT reports grants from National Health and Medical Research Council, Australia. All other authors declare no competing interests.

Acknowledgments

The work of HB was financially supported by Mazandaran University of Medical Sciences, Sari, Iran. AB is supported by the Public Health Agency of Canada. JCF acknowledges funding support from Fundação para a Ciência e a Tecnologia, through project UID/Multi/50016/2013. TL is supported by the Academy of Finland (grants number 287488 and number 319200). The work of SL is funded by the German Federal Ministry of Education and Research (nutriCARD; grant agreement number 01EA1411A). TM acknowledges additional institutional support from the Competence Cluster for Nutrition and Cardiovascular Health (nutriCARD), in Jena, Halle, and Leipzig, Germany. AES is supported by the South African Medical Research Council and the South African Research Chair Initiative by the National Research Foundation. AGT acknowledges fellowship funding from the National Health and Medical Research Council (Australia). ST's work was supported by the Foundation for Education and European Culture (IPEP), the Sara Borrell postdoctoral program (reference number CD15/00019 from the Instituto de Salud Carlos III, Spain) and the Fondos Europeo de Desarrollo Regional (FEDER). TW acknowledges academic support from the Department of Medicine, Faculty of Medicine, University of Rajarata, Sri Lanka; the World Federation of Neurology; and the World Federation for Neuro-Rehabilitation. JHW was supported by a Scientia Fellowship from the University of New South Wales (Australia). We thank Pauline Kim and Adrienne Chew for editing assistance.

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