Fried food consumption, genetic risk, and body mass index: gene-diet interaction analysis in three US cohort studies

Qibin Qi,[1 2] Audrey Y Chu,[3] Daniel I Chasman,[3 4] Frank B Hu,[1 5 6] Lu Qi,[1 5] on behalf of the coauthors listed in the full paper on bmj.com

EDITORIAL by Blakemore and Buxton

[1]Department of Nutrition, Harvard School of Public Health, Boston, MA, USA
[2]Department of Epidemiology and Population Health, Albert Einstein College of Medicine, Bronx, NY, USA
[3]Division of Preventive Medicine, Department of Medicine, Brigham and Women's Hospital and Harvard Medical School, Boston, MA, USA
[4]Division of Genetics, Department of Medicine, Brigham and Women's Hospital and Harvard Medical School, Boston, MA, USA
[5]Channing Division of Network Medicine, Department of Medicine, Brigham and Women's Hospital and Harvard Medical School, Boston, MA, USA
[6]Department of Epidemiology, Harvard School of Public Health, Boston, MA, USA
Correspondence to: L Qi, Department of Nutrition, Harvard School of Public Health, 665 Huntington Ave, Boston, MA 02115
nhlqi@channing.harvard.edu

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STUDY QUESTION

Does genetic predisposition interact with the effect of fried food consumption on adiposity?

SUMMARY ANSWER

The association between fried food consumption and adiposity is strengthened by genetic predisposition; and the genetic influences on adiposity are amplified by overconsumption of fried foods.

WHAT IS KNOWN AND WHAT THIS PAPER ADDS

Fried food consumption and genetic variants have been associated with adiposity. The current study shows that these two types of risk factors might interact with each other in relation to body mass index (BMI) and risk of obesity.

Participants and setting

Our study participants include women and men from three prospective cohorts in the United States: the Nurses' Health Study (NHS), Health Professionals Follow-up Study (HPFS), and Women's Genome Health Study (WGHS).

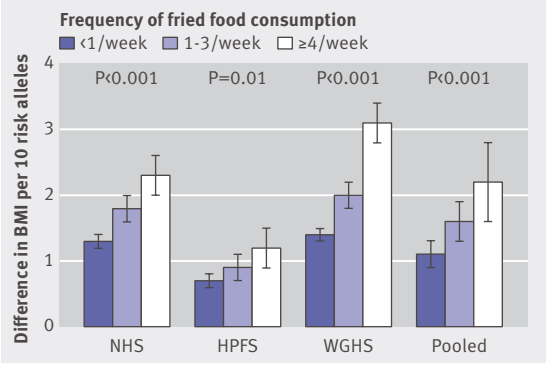
Design, size, and duration

We analyzed interactions between a genetic risk score based on 32 genetic variants associated with BMI (from a published genome-wide association study) and fried food consumption on the effect on BMI and obesity in 16 002 individuals from the NHS and HPFS. We then replicated these analyses in 21 421 individuals from the WGHS.

Main results and the role of chance

Consistent interactions between fried food consumption and the genetic risk score on BMI were identified in both the NHS and HPFS (P≤0.001 for interaction). Among participants in the highest third of the risk score, the differences in BMI between individuals who consumed fried foods four and more times a week and those who consumed less than once a week amounted to 1.0 (SE 0.2) in women and 0.7 (SE 0.2) in men, whereas the corresponding differences were 0.5 (SE 0.2) and 0.4 (SE 0.2) in the lowest third of the risk score. The gene-diet interaction was replicated in the WGHS (P<0.001 for interaction). Viewed differently, the genetic association with adiposity was strengthened with higher consumption of fried foods. In the combined three cohorts, the differences in BMI per 10 risk alleles were 1.1 (SE 0.2), 1.6 (SE 0.3), and 2.2 (SE 0.6) for fried food consumption less than once, once to three times, and four or more times a week (P<0.001 for interaction); and the odds ratios (95% confidence intervals) for obesity per 10 risk alleles were 1.61 (1.40 to 1.87), 2.12 (1.73 to 2.59), and 2.72 (2.12 to 3.48) across the three categories of consumption (P=0.002 for interaction). In addition, the variants in or near genes highly expressed or known to act in the central nervous system showed significant interactions with fried food consumption, with the FTO (fat mass and obesity associated) variant showing the strongest result (P<0.001 for interaction). The reproducible results in three independent cohorts were less likely to be due to chance.

Association between genetic risk score and BMI according to frequency of fried food consumption



Bias, confounding, and other reasons for caution

Though we carefully adjusted for multiple diet and lifestyle factors (including physical activity, smoking, alcohol intake, intake of sugar sweetened beverages, alternative healthy eating index, and total energy intake), confounding by other unmeasured or unknown factors might exist. Measurement errors in fried food consumption and other diet and lifestyle factors assessed by questionnaires are inevitable, though the questionnaires have been well validated in our cohorts. The information about the specific fried foods that our participants consumed at home or away from home was not collected in our study. Finally, further investigations in randomized clinical trials and experimental settings are warranted to validate the interactions.

Generalisability to other populations

This study was conducted in middle aged and older adults of European ancestry recruited in the US. Whether the results are generalizable to other populations is unknown.

Study funding/potential competing interests

The study was funded in part by the National Institutes of Health. The authors were independent from the funders in all aspects of the study.

Associations between exposure to takeaway food outlets, takeaway food consumption, and body weight in Cambridgeshire, UK: population based, cross sectional study

Thomas Burgoine,[1] Nita G Forouhi,[1] Simon J Griffin,[1 2] Nicholas J Wareham,[1] Pablo Monsivais[1 2]

EDITORIAL by Neckerman

[1]UKCRC Centre for Diet and Activity Research, MRC Epidemiology Unit, University of Cambridge School of Clinical Medicine, Box 285, Institute of Metabolic Science, Cambridge Biomedical Campus, Cambridge CB2 0QQ, UK

[2]Department of Public Health and Primary Care, Institute of Public Health, University of Cambridge, Cambridge, UK

Correspondence to: T Burgoine
tb464@medschl.cam.ac.uk

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STUDY QUESTION Does overall exposure to takeaway food outlets at home, at work, and along commuting routes promote takeaway food consumption and overweight?

SUMMARY ANSWER Greater exposure to takeaway food outlets was associated with higher consumption of takeaway food and higher body weight.

WHAT IS KNOWN AND WHAT THIS PAPER ADDS During the past decade in the United Kingdom, takeaway food outlets have proliferated; but research on whether exposure to these outlets influences food consumption and body weight has only focused on residential neighbourhoods, and has yielded inconsistent findings. Our findings include workplace and commuting exposures and suggest that policy interventions to restrict takeaway food access in order to reduce takeaway food consumption and body weight, a strategy increasingly adopted at the local level in the UK, might be successful.

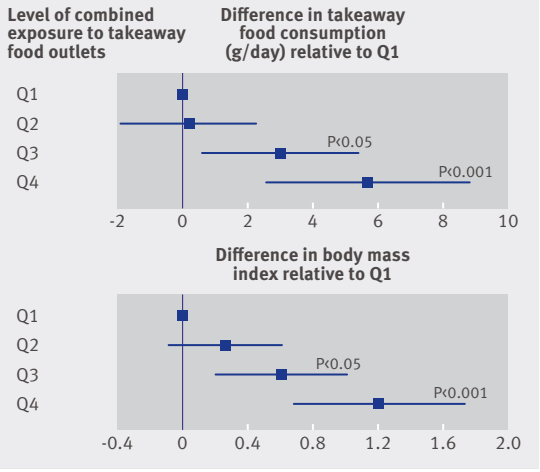
Participants and setting

The study included working adults (n=5442, aged 29-62 years) who participated in the ongoing Fenland Study, Cambridgeshire, UK.

Design

Our cross sectional study used a geographical information system to calculate individual level exposure to takeaway food outlets in three environments (at home, at work, and along commuting routes (the shortest route between home and work)). Participant exposure levels were estimated and the sample was divided into quarters (Q1 being the least exposed and Q4 being the most exposed). Differences in food consumption and body weight were estimated using multiple regression, adjusting for known confounders.

Association between combined exposure to takeaway food outlets, takeaway food consumption, and body mass index



Primary outcomes

Self reported consumption of takeaway food (g/day; pizza, chips, burgers, fried food) using food frequency questionnaires, and measured body mass index.

Main results and the role of chance

Exposure to takeaway food outlets was positively and strongly associated with takeaway food consumption and body mass index, with evidence of a dose-response association. In all three domains combined, the group of people most exposed to takeaway food outlets consumed an additional 5.7 g/day of takeaway food (95% confidence interval 2.6 to 8.8; P<0.001), and had an average body mass index 1.21 (0.68 to 1.74; P<0.001) greater, relative to those least exposed.

Bias, confounding, and other reasons for caution

We accounted for confounders including age, sex, household income, highest educational qualification, smoking status and energy expenditure through physical activity (both in the body mass index model only), total energy intake (food consumption model only), car ownership, supermarket access, and commuting route distance. Important sources of potential error and bias included the definition of "neighbourhood" used, which may not have reflected actual exposures; and assumption of travel from home to work along the shortest street network route. Such factors affecting exposure may have attenuated our observed associations. Our cross sectional study design could not account for self selection of exposure to takeaway food outlets based on preference for takeaway food consumption and body weight (reverse causality). Therefore, the study design prevented us from inferring causal associations between the environment, diet, and body mass index.

Generalisability to other populations

The Fenland Study was designed to be representative of the Cambridgeshire region, with sample characteristics congruent with the region's demographic characteristics (educated, employed, and white British). This sample may be less representative of other regions in the UK.

Study funding/potential competing interests See bmj.com.

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The impact of a bodyweight and physical activity intervention (BeWEL) initiated through a national colorectal cancer screening programme: randomised controlled trial

Annie S Anderson,[1] Angela M Craigie,[1] Stephen Caswell,[1] Shaun Treweek,[2] Martine Stead,[3] Maureen Macleod,[1] Fergus Daly,[4] Jill Belch,[5] Jackie Rodger,[6] Alison Kirk,[7] Anne Ludbrook,[8] Petra Rauchhaus,[9] Patricia Norwood,[8] Joyce Thompson,[10] Jane Wardle,[11] Robert J C Steele[1]

[1]Centre for Research into Cancer Prevention and Screening, Cancer Division, Medical Research Institute, Ninewells Medical School, Dundee DD1 9SY, UK
[2]Health Services Research Unit, University of Aberdeen, Aberdeen, UK
[3]Institute for Social Marketing, University of Stirling, Stirling, UK
[4]Frontier Science (Scotland), Kingussie, Inverness-shire, UK
[5]Vascular Diseases Research Unit, Medical Research Institute, Ninewells Medical School, Dundee, UK
[6]Ninewells Hospital and Medical School, Dundee, UK
[7]Physical Activity for Health Research Group, School of Psychological Sciences and Health, University of Strathclyde, Glasgow, UK
[8]Health Economics Research Unit, University of Aberdeen, Aberdeen, UK
[9]Tayside Clinical Trials Unit, Tayside Medical Science Centre, Ninewells Hospital and Medical School, Dundee, UK
[10]Directorate of Public Health, NHS Tayside, Kings Cross Hospital, Dundee, UK
[11]Health Behaviour Research Centre, Institute of Epidemiology and Public Health, University College London, London, UK
Correspondence to: A S Anderson
a.s.anderson@dundee.ac.uk

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STUDY QUESTION

What is the impact of a diet and physical activity intervention programme on bodyweight change in overweight or obese people attending routine screening for colorectal cancer who have had colorectal adenomas removed but are at risk of developing further obesity related conditions?

SUMMARY ANSWER

A 12 month, personalised, behaviourally focused weight loss programme within a national colorectal cancer screening programme was associated with sustained changes in body weight, physical activity, and eating and drinking habits, offering potential for risk reduction of disease in older adults.

WHAT IS KNOWN AND WHAT THIS PAPER ADDS

Excess body weight, low physical activity levels, and inappropriate diet are risk factors for colorectal adenomas and cancer, and the screening setting provides an opportunity to promote and achieve weight loss in older adults who are at risk of a range of obesity related comorbidities. An intervention of 5.25 hours of lifestyle counsellor contact over 12 months delivered on a one to one basis resulted in continuous and significant weight loss, with improvements in blood pressure and blood glucose levels.

Design

A multicentre, 1:1 parallel group, randomised controlled trial of a weight loss intervention versus usual care (ISRCTN 53033856). Participants were randomised using permuted block to a control group (weight loss booklet) or 12 month intervention group (three visits with a counsellor and monthly telephone calls) focusing on diet, activity, and behaviour change, with the provision of weighing scales and personalised caloric restriction.

Participants and setting

We recruited participants aged 50 to 74 years, with a body mass index >25 (weight (kg)/height (m)^2) in four Scottish NHS health boards (Tayside, Forth Valley, Ayrshire and Arran, Greater Glasgow and Clyde). These participants had a diagnosis of adenoma following a positive faecal occult blood test result and colonoscopy as part of the national bowel screening programme.

Primary outcome

Weight change over 12 months.

Main results and the role of chance

Of 997 patients approached, 49% expressed an interest in participation. Following exclusions 329 (74% men) were randomised: 163 to intervention and 166 to control. At 12 months, primary outcome data were available for 148 (91%) participants in the intervention group and 157 (95%) in the control group. Mean weight loss was 3.50 kg (SD 4.91) (95% confidence interval 2.70 to 4.30) compared with 0.78 kg (SD 3.77) (0.19 to 1.38) in the intervention and control groups. The group difference was 2.69 kg (95% confidence interval 1.70 to 3.67 kg). Differences between groups were significant for waist circumference, body mass index, blood pressure, blood glucose level, diet, and physical activity.

Harms

No reported adverse events were considered to be related to trial participation.

Bias, confounding, and other reasons for caution

A weakness of the study was the need for repeat measurements of body weight in the control group, which may have been enough to motivate some degree of weight management.

Generalisability to other populations

Findings should be generalisable to other settings for colorectal cancer screening. However, lifestyle interventions in these settings only reach people who choose to participate in screening programmes.

Study funding/potential competing interests See bmj.com

Changes in anthropometric measures at 12 months by treatment group. Values are means (95% confidence intervals) unless stated otherwise

	Intervention group			Control group			
Measures	No in group	Mean (SD)	Difference to baseline	No in group	Mean (SD)	Difference to baseline	Between group differences*, P value
Body weight (kg):							
Baseline	163	90.2 (14.9)		166	88.4 (14.3)		
12 months	148	87.2 (15.7)	−3.50 (−4.30 to −2.71)	157	88.1 (14.2)	−0.78 (−1.38 to −0.19)	2.69 (1.70 to 3.67), <0.001
% bodyweight loss:							
12 months	148	3.9 (5.4)		157	0.83 (4.10)		3.04 (2.16 to 3.92), <0.001
Waist circumference (cm):							
Baseline	163	104.7 (10.9)		166	103.9 (10.9)		
12 months	145	100.2 (12.0)	−4.91 (−5.79 to −4.03)	157	102.1 (11.1)	−2.16 (−2.85 to −1.47)	2.68 (1.74 to 3.62), <0.001

Use of macrolides in mother and child and risk of infantile hypertrophic pyloric stenosis: nationwide cohort study

Marie Lund,[1] Björn Pasternak,[1] Rie B Davidsen,[1] Bjarke Feenstra,[1] Camilla Krogh,[1] Lars J Diaz,[1] Jan Wohlfahrt,[1] Mads Melbye[1 2]

[1]Department of Epidemiology Research, National Health Surveillance and Research, Statens Serum Institut, Artillerivej 5, 2300 Copenhagen S, Denmark

[2]Department of Medicine, Stanford School of Medicine, Stanford, CA, USA

Correspondence to: M Lund
mxd@ssi.dk

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STUDY QUESTION

Is macrolide use in mother and child from pregnancy onset until 120 days after birth associated with infantile hypertrophic pyloric stenosis (IHPS)?

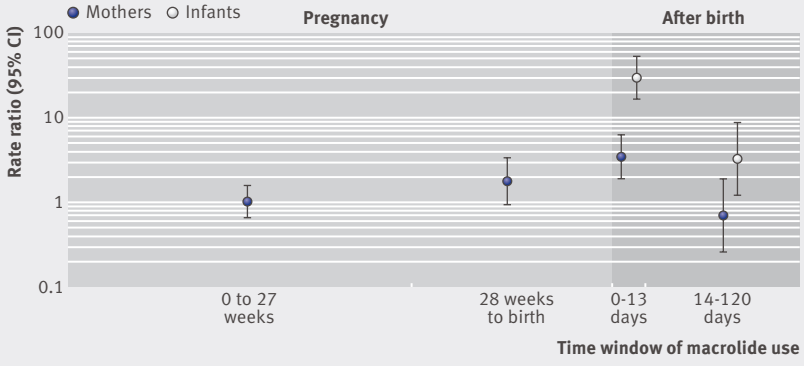
SUMMARY ANSWER

Macrolide treatment of young infants was strongly associated with IHPS. Maternal macrolide use during the first two weeks after birth was also associated with an increased risk of IHPS and there was a possible association with use during late pregnancy.

WHAT IS KNOWN AND WHAT THIS PAPER ADDS

Macrolide use in infants during the first two weeks after birth has been associated with an increased risk of IHPS, but it is unclear if the risk is also increased with later use and with maternal use during late pregnancy and lactation. Macrolide treatment of young infants and their mothers within the first two weeks of birth, and possibly during late pregnancy, was associated with IHPS.

Rate ratios of infantile hypertrophic pyloric stenosis (IHPS) according to maternal use of macrolides and use in infants during pregnancy and up to 120 days after birth (n=999 378 singletons)



	No of IHPS cases	Person years	Rate ratio (95% CI)
Maternal use during pregnancy			
0-27 weeks	20	7569	1.02 (0.65 to 1.59)
28 weeks to birth	10	2286	1.77 (0.95 to 3.31)
None	847	315 569	1 (reference)
Maternal use after birth			
0-13 days	11	1072	3.49 (1.92 to 6.34)
14-120 days	4	2845	0.70 (0.26 to 1.90)
None	834	309 002	1 (reference)
Use in infants after birth			
0-13 days	12	123	29.8 (16.4 to 54.1)
14-120 days	4	867	3.24 (1.20 to 8.74)
None	833	311 928	1 (reference)

Participants and setting

We identified a nationwide cohort of 999 378 liveborn singletons and their mothers and used linked individual level information on filled macrolide prescriptions, surgery for IHPS, and potential confounders.

Design, size, and duration

A register based cohort study in Denmark from 1996 to 2011. The association between macrolide use and IHPS was assessed for six categories of use: maternal use during pregnancy (gestational weeks 0-27 and weeks 28 to birth), maternal use after birth (days 0-13 and 14-120), and use in infants after birth (days 0-13 and 14-120).

Main results and the role of chance

880 infants developed IHPS (0.9 cases per 1000 births). Compared with infants with no use of macrolides, the adjusted rate ratio for IHPS in infants with use of macrolides during days 0-13 after birth was 29.8 (95% confidence interval 16.4 to 54.1) and during days 14-120 was 3.24 (1.20 to 8.74). The rate ratio for maternal use of macrolides for days 0-13 after birth was 3.49 (1.92 to 6.34) and for days 14-120 was 0.70 (0.26 to 1.90). The rate ratios for maternal use of macrolides during pregnancy were 1.02 (0.65 to 1.59) for weeks 0-27 and 1.77 (0.95 to 3.31) for weeks 28 to birth.

Bias, confounding, and other reasons for caution

Potential biases were from confounding by indication, macrolides administered during admission to hospital, and lack of information on breastfeeding status. These were dealt with in sensitivity analyses, which did not change the main conclusions of the study.

Generalisability to other populations

The study took place in a Western population with public healthcare.

Study funding/potential competing interests

All researchers are independent of the study funders: the University of Copenhagen, the Danish Medical Research Council, and the Oak Foundation.

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- See supplementary data for this research paper
- Letter: Beware statins with macrolides (*BMJ* 2011;342:d2703)
- Letter: Pneumonia: Macrolides or amoxicillin for community acquired pneumonia? (*BMJ* 2006;332:1213.3)