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Impact of migration on coronary heart disease risk factors: Comparison of Gujaratis in Britain and their contemporaries in villages of origin in India

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Abstract

The causes of the excess coronary heart disease (CHD) risk in South Asian migrants from the Indian subcontinent remain unclear. Comparisons of CHD risk factors amongst South Asian migrants living in Britain with those of the general UK population provide only a partial explanation. We compared Gujaratis in Britain with similar, non-migrant Gujaratis in India, to test the hypothesis that differences in CHD risk factors associated with migration would be more informative. Randomly sampled Gujaratis aged 25–79 years living in Sandwell ($n = 242$) were compared with age-, gender- and caste-matched contemporaries remaining in their villages of origin in Navsari, India ($n = 295$). Lifestyle indices, food intake and physical activity, were assessed with standardised questionnaires and energy expenditure and metabolic parameters measured.

British Gujaratis had higher, mean body mass indices by 6 (4.5–7.4) kg/m^2 mean (95% CI), and greater dietary energy intake, fat intake, blood pressure, fasting serum cholesterol, apolipoprotein B, triglycerides, non-esterified fatty acid (NEFA) and C-reative protein concentrations than Gujaratis in India. Dietary folate and serum folate and Vitamin B_{12} were lower and plasma homocysteine was higher in India. Smoking was less prevalent and high-density lipoprotein cholesterol tended to be higher in Britain. Diabetes prevalence was high in both populations and impaired fasting or 2 h post-glucose challenge plasma glucose was even more prevalent in Gujarat. In India, however, where insulin secretion and NEFA were lower diabetes and impaired glucose tolerance were less frequently accompanied by excess metabolic CVD risk factors.

In conclusion, exposure to increased fat intake and obesity related to migration is likely to explain the disproportionate combination of established and emerging CHD risk factors prevalent in Gujaratis in Britain. Strategies to improve nutrition and to identify and treat cardiovascular risk factors such as dyslipidaemia and hypertension are urgently required.

Keywords: Indian; CHD risk factors; Glucose intolerance; Diet; Physical activity

1. Introduction

Coronary heart disease (CHD) is the leading cause of mortality amongst migrant people of Indian origin. High CHD rates have been consistently reported across the global Indian diaspora, for example, in South Africa [1], Trinidad [2], Singapore [3], Fiji [4], Mauritius [5], California [6] and Canada [7]. Men and women in Britain whose families originate in the Indian sub-continent have an approximately 40% higher CHD morbidity and mortality compared with the British population of European descent [8–13]. Within India,

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CHD prevalence in cities may reach levels similar to that in migrants, but is thought to remain low in rural India [14].

Established risk factors, such as diabetes [14,15], serum cholesterol [12,16], smoking [14], hypertension [1,4] and obesity [4] are major contributors to CHD in Indian populations. However, most previous studies attempting to explain their increase in CHD on migration in these terms compared them with the populations in the locality to which they had moved. While such study designs may help to explain excess CHD risk relative to indigenous people in the new location, they cannot elucidate which of these factors is most adversely affected by migration or to which people of Indian origin may have heightened susceptibility. Thus, among various migrant communities of Indo–Asian origin, only central obesity [17], insulin [17], serum C-peptide [18] and fasting triglycerides [19] were higher in Indians than in Europeans. Serum HDL was lower, but marginally so also was total cholesterol [17]. As a result, doubts were raised as to whether established risk factors could account for the increased CHD amongst Indian migrants [14]. Therefore, in our pilot study, we sought to investigate this question directly by comparing Indian Punjabi migrants to Britain with their siblings still in India's Punjab [20]. Serum cholesterol, obesity and insulin levels were higher among migrants, suggesting that nutritional factors associated with migration were crucial to the increased CHD risk. Likely, candidate factors not then measured included dietary composition, physical activity, nutrionally related serum factors and C-reactive protein (CRP). These are the focus of this larger more definitive study.

We hypothesized that migration would adversely affect established and perhaps reveal novel CHD risk factors, among migrants to Britain compared with contemporaries from an identical cultural and genetic background, still residing in their villages of origin in rural India. We aimed to identify what factors changed on migration, rather than compare migrants with people in their new locality [17,18].

2. Methods

2.1. Participants

In Sandwell, West Midlands, UK, participants from a specific Gujarati community emanating either directly or via East Africa from rural villages around Navsari, a town in Gujarat, India, were randomly selected using surname analysis from a sampling frame. This was constructed from the 1998 electoral register supplemented with Gujarati community directories and local population registers in primary care. Prospective participants were contacted by letter, and then by home visit. In India, sampling frames were constructed from the 1999 electoral roll supplemented with registers for diesel fuel allocation to each household.

Recruitment targets were 300 participants from each country, evenly distributed by gender and 5 age decades (25–74 years). One subject per household was approached. People with serious medical conditions, except cardiovascular disease, were excluded. The local research ethics committee in Britain that and of the All India Institute of Medical Sciences, New Delhi approved the protocol; every participant provided written or witnessed consent.

2.2. Procedures

Participants were invited to attend clinics fasting from 10:00 p.m. the previous evening; trained fieldworkers administered standard, pre-tested questionnaires on lifestyle, medical history, place of origin, daily activity and the Rose angina questionnaire in English or Gujarati. Back translations were checked for original meaning.

Venous blood was collected from all participants. Serum and plasma (collected in citrated and heparin/fluoride containing tubes) were obtained within 1 h by centrifugation and snap-frozen to be stored at $-70\,^{\circ}C$. Participants without previously diagnosed diabetes or a fasting capillary glucose <7 mmol/l had glucose tolerance tests (GTT) beginning between 08:30 and 10:00 a.m. in both sites. A 75 g equivalent glucose load was administered as a fruit-flavoured drink (Maxijoul, SHS Supplies, Liverpool, UK). Venous blood samples were collected fasting and at 30 and 120 min after glucose challenge (based on the WHO criteria [21]). Blood pressure (BP) was measured three times (analysing the mean of the last two), with the validated semi-automatic Omron HEM-705CP (Omron Healthcare Europe, Mannheim, Germany) with appropriate cuff sizes, after >5 min sitting. Hypertension was defined as systolic blood pressure $\geq$140 or diastolic $\geq$90 mmHg or already on treatment [22].

Identical procedures for anthropometric measurements in each site included the Leicester height measure (Seca Ltd., Birmingham, UK), weight (Seca Ltd.), waist and hip (metal tapes). The waist was the narrowest circumference above the umbilicus and below the ribs. The hip circumference was measured over thin clothing as the widest horizontal circumference around the buttocks. These measures were rigorously standardised, with fieldworkers locally revalidated monthly and with regular cross-site visits every 4 months. Participants had a 12-lead ECG (Marquette Mac-PC, Marquette Electronics Inc, GE Healthcare, Slough, UK) analysed using Minnesota coding (Professor P MacFarlane). Only definite CHD diagnosis is reported using major Q wave and QS changes (codes 1–1 and 1–2).

A 4-day food diary for each participant, to include an average weekend, was administered and checked by trained nutritionists. Nutritional content was analysed using a Gujarati foods database developed by AV using Wisp V1.27 Tinuviel Software (Warrington, UK). Diaries were calibrated with 24 h food recalls. To measure everyday physical activity, participants wore on their waists for 48 h '*Caltrac*' accelerometers, (Muscle Dynamics Fitness Network, Torrance, CA, USA) previously validated in similar field conditions [23,24]. The *Caltrac* was calibrated with each participant's age, height and weight. Total energy output was analysed in kcal/24 h.