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Original article

Impact of classical risk factors of type 2 diabetes among Asian Indian, Chinese and Japanese populations

L. He [a,*], J. Tuomilehto [c,d,e], Q. Qiao [a], S. Söderberg [j], M. Daimon [k,l], J. Chambers [f,g,h,i], J. Pitkäniemi [a,b], for the DECODA study group

[a] *Department of Public Health, University of Helsinki, P.O. Box 41, 00014 Helsinki, Finland*
[b] *Finnish Cancer Registry, Institute for Statistical and Epidemiological Cancer Research, Helsinki, Finland*
[c] *Centre for Vascular Prevention, Danube-University Krems, 3500 Krems, Austria*
[d] *Chronic Disease Prevention Unit, National Institute for Health and Welfare, 00271 Helsinki, Finland*
[e] *Diabetes Research Group, King Abdulaziz University, 21589 Jeddah, Saudi Arabia*
[f] *Department of Epidemiology and Biostatistics, Imperial College London, London W2 1NY, United Kingdom*
[g] *Imperial College Healthcare NHS Trust, Hammersmith Hospital, London W12 0HS, United Kingdom*
[h] *Royal Brompton and Harefield Hospitals NHS Trust, London SW3 6NP, United Kingdom*
[i] *Ealing Hospital NHS Trust, Southall, Middlesex UB1 3HW, United Kingdom*
[j] *Department of Public Health and Clinical Medicine, Cardiology, Umeå University, Umeå, Sweden*
[k] *Department of Neurology, Metabolism, Endocrinology and Diabetology, Faculty of Medicine, Yamagata University, Yamagata, Japan*
[l] *Department of Endocrinology and Metabolism, Hirosaki University Graduate School of Medicine, Hirosaki, Japan*

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Abstract

Aims. – This review investigated the population impact of major modifiable type 2 diabetes (T2D) risk factors, with special focus on native Asian Indians, to estimate population attributable risks (PARs) and compare them with estimates from Chinese and Japanese populations.

Methods. – Information was obtained on risk factors in 21,041 Asian Indian, 17,774 Chinese and 17,986 Japanese populations from multiple, large, cross-sectional studies (the DECODA project) of T2D. Crude and adjusted PARs were estimated for the major T2D risk factors.

Results. – Age had the highest crude and adjusted PARs among Asian Indians and Chinese in contrast to waist–hip ratio among Japanese. After adjusting for age, the PAR for body mass index (BMI) in Asian Indians (41.4% [95% CI: 37.2%; 45.4%]) was second only to triglycerides (46.4% [95% CI: 39.5%; 52.8%]) compared with 35.8% [95% CI: 29.9%; 41.4%] in Japanese and 38.4% [95% CI: 33.5%; 43.2%] in Chinese people. The PAR for BMI adjusted for age, LDL and triglycerides (39.7% [95% CI: 31.6%; 47.2%]) was higher than for any other factor in Asian Indians, and was much higher than in the Chinese (16.8% [95% CI: 3.0%; 30.9%]) and Japanese (30.4% [95% CI: 17.5%; 42.2%]) populations.

Conclusion. – This review provides estimates of the association between major risk factors and prevalences of T2D among Asian populations by examining their PARs from large population-based samples. From a public-health point of view, the importance of BMI in Asian Indians is especially highlighted in comparison to the other Asian populations. Given these results and other recent findings on the causality link between BMI and T2D, it can be postulated that obesity may be involved in the aetiology of T2D through interaction with ethnic-specific genetic factors, although ethnicity itself is not a direct risk factor for T2D as people of all ethnic backgrounds develop diabetes.

Keywords: Asian populations; Population attributable risk; Prevalence; Risk factors; Type 2 diabetes mellitus

1. Introduction

Type 2 diabetes mellitus (T2D) is among the most common chronic metabolic diseases, affecting nearly 3% of the population worldwide [1]. Recent estimates suggest that T2D will affect at least 550 million people by 2030, more than previously

* Corresponding author. Tel.: +358 29 412 54 66.
E-mail address: liang.he@helsinki.fi (L. He).

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proposed [2], and become an increasing epidemic in Asian countries [3]. In 2000, the highest worldwide country-specific estimate of T2D, 31.7 million cases, was found in India [1,4]. Native Asian Indians, in particular, show an ethnic susceptibility to T2D and a high disease prevalence [4–6].

While the causes of T2D are still not completely understood, it is generally believed that T2D results from both genetic and environmental factors. It has long been demonstrated that families with diabetic patients have a significant excess of diabetic relatives compared with families without diabetic patients [7]. More important, genetic factors play a crucial role in the pathogenesis of T2D in the Indian population. A recent study reported that Asian Indians are excessively insulin-resistant compared with Caucasians [5]. In another study of a Western Indian population, a pedigree analysis suggested that a family history of T2D results in a predisposition in successive generations along with a decrease in age at onset of T2D [8]. More recently, an excess maternal transmission of T2D was identified among Asian Indians [9]. On the other hand, many environmental factors, such as diet, lifestyle and body mass index (BMI), are also reportedly associated with the risk of T2D in Indians [6] as well as in other Asian populations such as the Chinese [10]. A large-scale study based on data from the Diabetes Epidemiology: Collaborative Analysis of Diagnostic Criteria in Asia (DECODA) project, organized by the National Institute for Health and Welfare (THL) in Finland and ongoing since 1997, reported that central obesity, measured as the waist-to-stature ratio (WSR), is more strongly associated with T2D than BMI in most Asian populations [11].

When measuring disease-exposure associations from a public-health perspective, some risk factors with a high relative risk may not be playing a predominant role at the population level because few people are exposed to them. Thus, an alternative public-health focus is to quantify the contribution of the population attributable risk (PAR) fraction to prevalence, which may then help to guide interventions. PAR analysis in an Australian cross-sectional study demonstrated that overweight and obesity are associated with diabetes [12]. However, there has been little quantitative measurement at a population level of the impact of the major T2D risk factors in Asian populations.

Thus, given our awareness of the ethnic specificity of T2D morbidity, our aim was to evaluate the impact of known T2D risk factors in the native Asian Indian population, and to compare it with those in other Asian populations by quantifying their attributable contributions to T2D based on DECODA data for PAR [13], which reflects the rate of prevalence reduction in a population by eliminating exposure. This was then compared with the PARs from different populations to identify the most influential population-specific risk factor. In particular, increasing evidence implicates the potential causal role of obesity in the development of T2D [14–16], and the population-level impact of obesity on T2D is a growing concern in public health that has yet to be measured in Asian populations. It is worth noting that, as the data used in the present analysis are cross-sectional, it is only possible to evaluate an association rather than defining a causal inference.

2. Materials and methods

2.1. Data description

Recruitment of the DECODA cohorts has been described in detail in previous publications [17,18]. Our present analysis included a total of 21,041 native Asian Indians, of which 10,052 (47.8%) were male, and 17,774 Chinese and 17,986 Japanese sample populations. Indian subjects living in Fiji ($n = 1068$) and Mauritius ($n = 3282$) were also included for comparison of T2D incidence with native Indians. For the native Indian population, the focus was on population-based cross-sectional surveys for T2D performed during 1993–2000 and 2006. These data were collected from six studies, including Chennai 94 [19], Chennai 97 [20,21], Chennai 2006, Dombivli [22], CURES [23], and the Indian National Urban Diabetes Survey (NUDS) [6], which investigated T2D and impaired glucose regulation using a standard 2-h 75-g oral glucose tolerance test (OGTT).

Individual data were collected and sent to the THL in Helsinki, Finland. Fasting blood samples were collected after overnight fasting for measures of plasma glucose and lipids. The kinase oxidase method was applied for glucose measurements, and the enzymatic method used for triglycerides, and total and high-density lipoprotein (HDL) cholesterol. Low-density lipoprotein (LDL) cholesterol was calculated using the Friedewald formula. Blood pressure was measured on the right arm after 5–10 min of sitting using a standard mercury sphygmomanometer. Waist circumference was measured at the midpoint between the lowermost rib margin and the iliac crest. Smoking status was defined as a categorical variable with three levels: non-smoker; ex-smoker; and current-smoker. A large number of subjects in the Dombivli and NUDS studies were missing data for some variables, including LDL, HDL and triglycerides. The risk factors included in our present study were age, height, BMI, waist–hip ratio (WHR), systolic/diastolic blood pressure (SBP/DBP), LDL, HDL, triglycerides and smoking status. The basic data used for our analyses are presented in Table 1.

The definition of T2D used in our analysis comprised two parts. Previously diagnosed T2D was defined by self-reports of a prior history of T2D or a history of taking drugs for T2D. Newly diagnosed T2D was based on the World Health Organization (WHO) criteria published in 2006: fasting plasma glucose (FPG) $\geq$ 7.0 mmol/L or 2-h plasma glucose $\geq$ 11.1 mmol/L [24]. Any subject whose T2D status or history could not be deduced was excluded from the entire analysis.

2.2. Methods for statistical analysis

The prevalence of T2D was estimated by dividing the total population recruited from the DECODA studies by the number of subjects newly or previously diagnosed with T2D. The 95% confidence intervals (CIs) for this proportion were constructed based on binomial probability, using the Clopper–Pearson method [25].

Analyses of PAR were divided into two stages. First, competing models containing different potential risk factors associated with T2D were assessed to select the factors present in the top