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**Ethnic Inequalities in Health**  
**Why is the prevalence of Type 2 Diabetes higher among**  
**South Asian immigrants?**

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## ABSTRACT

Type 2 diabetes mellitus (T2DM) is a major public health problem with the burden of the disease distributed unevenly. The prevalence of the disease is higher among South Asian immigrants in comparison to the prevalence among the population of the host countries. Studies from Norway, UK and beyond indicate that there is ethnic inequality in the prevalence of T2DM. In order to find out the reason for this high prevalence of T2DM, this thesis, by reviewing relevant literature, looks into different factors which are said to affect the health inequality. Those different factors underlying the high prevalence of T2DM includes genetics, socioeconomic position (SEP), culture, migration, lifestyle, language barrier and access to health care services. The ethnic inequality in health happens due to a complex web of intermingled factors. Genetic factors, SEP, migration, dietary habit, pattern of physical activity, obesity, language barrier and access to health care all contribute to the higher prevalence of T2DM among South Asian immigrants.

Although researches regarding the genetic and biological factors underlying diabetes are far from completed, there is an indication that South Asians are genetically prone to T2DM. In addition to that, a number of studies conducted to find out the correlation between T2DM and SEP indicated that low SEP was related to a higher prevalence of diabetes. Low educational level, low income and deprived areas are associated with higher prevalence of T2DM among South Asian immigrants. Moreover, the dietary habit of South Asian immigrants, their physical inactivity, obesity especially the high waist-to-hip ratio, language barrier and less access to health care contribute to the high prevalence of T2DM among the South Asian immigrants in UK and Norway. On top of that, migration of South Asians from the Indian Subcontinent to western countries by itself appears to be one of the contributing factors in health inequality. Numerous changes in the socio-cultural environment occurs with migration, which in turn leads to shifts in SEP, access to health care and life style, including diet and physical activity, and in due course the high prevalence of T2DM. Genetics and early life conditions affect health outcomes later in life and may interact with the changes taking place after migration.

Key words: ethnic inequality in health, type 2 diabetes mellitus, South Asian immigrants, migration and health, socioeconomic position

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## 1. INTRODUCTION

### 1.1 Background

On June 26, 2011, citing researchers from Imperial College in London and Harvard University in the US, the BBC announced that the number of adults with diabetes in the world has more than doubled since 1980 (BBC 2011). According to World Health Organization (WHO) 346 million people worldwide have diabetes and an estimated 3.4 million people died from consequences of high blood sugar in 2004. WHO projects that diabetes death will double between 2005 and 2030 (WHO 2012a).

On the same date, TV2, a Norwegian commercial TV channel, broadcasted that the number of diabetic patients in Norway has also doubled since 1980 (TV2 2012). According to the Norwegian Health Institute (NHI) there are approximately 135,000 people using diabetes medicines in Norway today. In addition, it is believed that there are many people with diabetes who do not use diabetes medicine and still many more who do not know that they have diabetes. Therefore, it is hardly possible to know the exact number of diabetes cases in Norway (FHI 2012).

Just recently, on August 9, 2012, NRK, the Norwegian Broadcasting Corporation declared what it called “Diabetic Shock among Asians”. According to the news, the current trend of diabetes “epidemic” is especially alarming among immigrants from the Indian sub-continent (Pakistan, Sri Lanka and India) who live in Norway (NRK 2012). The BBC characterized the situation in the UK as “the South Asians time-bomb”(BBC 2010).

Already in 2000, a study from Oslo indicated that among the immigrants from the Indian subcontinent living in Oslo between 20 and 35% were diabetic. That is really alarming when compared to the National average for Norwegian population which is about 3-6% (Jenum et al. 2005; FHI 2012).

According to the Norwegian Institute of Public Health, more than one in four immigrant women from South Asian countries have diabetes. For men, the proportion is one out of

seven. The research indicates that immigrants from South Asian countries like India, Pakistan and Sri Lanka have alarmingly higher diabetes prevalence than ethnic Norwegians and other Western Europeans. Immigrant women from South Asia are particularly vulnerable, according to a survey of 2513 people who were invited to a study in Romsås and Furuset districts in Oslo in 2000 (FHI 2012; Jenum et al. 2005).

The results of the Romsås/ Furuset study show the following incidence of diabetes in the age group of 30-59 years:

27.5 percent of women from South Asia

14.3 percent of men from South Asia

2.9 percent of Western women

5.9 per cent of Western men

Similarly, in UK, studies have shown that the prevalence of type 2 diabetes mellitus (T2DM) among South Asians is significantly greater than in many other ethnic groups. South Asian people who live in the UK are up to six times more likely to have diabetes than the white population. The prevalence of doctor-diagnosed diabetes increases markedly with age, in both men and women. 44.4% of Pakistani women aged 55+ and 25.3% of Pakistani men in the same age group have diabetes. In another survey in inner city Manchester, where around 30% of the population is South Asian, amongst individuals aged 35–79 years, 30% of Pakistani men, and 36% of Pakistani women had diabetes (Mather and Keen 1985; Sproston and Mindell 2006).

Comparing the data of diabetes prevalence from Norway and the UK, one can easily see the heavy burden of the disease on South Asians. Table 1 gives a quick comparison between the results of studies conducted in inner city Manchester, UK and Furuset/Romsås area in Oslo, Norway.

Table 1: T2DM prevalence among South Asians in Oslo and in Manchester

Data source:(Sproston and Mindell 2006; Jenum et al. 2005)

	Oslo	Manchester
South Asian women	27.5%	36%
South Asian men	14.3%	30%
South Asian average	21%	33%
Country average men	4.4 %	4.3%
Country average women	2.9 %	3.4%

Likewise, studies conducted on immigrants from the Indian subcontinent residing in different countries all over the world point out that this immigrant group is more affected than the rest of the populations. The health inequality is crystal clear and quite huge ( Kumar 2008; Radha and Mohan 2007).

What is the reason for this huge gap? What are the factors contributing to these ethnic inequalities in health? This paper, by reviewing relevant literature, seeks to find possible answers to this question.

### 1.2 The Research Questions

My research question is: Why is the prevalence of T2DM so high among South Asians in comparison to the rest of the population? I will investigate if the reason behind the high prevalence of T2DM among South Asians is only genetic or if there are some other factors. At the same time I will examine the roles of socioeconomic position, migration, culture and life style in the high prevalence of T2DM among South Asians.

### 1.3 The Scope of the Paper

Diabetes mellitus is a collection of diseases comprising different chronic abnormalities which is too wide for this thesis to cover. To fit into the size of the thesis, therefore, I am required to cut into size. Consequently, the focus of the thesis is mainly T2DM. More specifically, it is about T2DM among South Asian immigrants or immigrants from the



Indian subcontinent in Norway and the UK. Nevertheless, some relevant studies from other countries are also used.

As a healthcare professional who has been working in a metropolitan hospital in a multicultural city like Oslo, I am familiar with the burden of T2DM on South Asians in the city. The interest to choose the topic for my thesis comes initially from my work experience. The lack of sufficient studies on this area in Norway is my first challenge in the process of writing this thesis. In order to overcome the challenge I had to look for other country, preferably with similar health care system with Norway, where I might find sufficient studies on the area of my topic. I then found out that a lot of studies were conducted in UK on this issue. Consequently I have chosen UK the country with more or less similar health care system which inhabits millions of South Asians.

The terminology *South Asia* which sometimes called the *Indian subcontinent* refers to India, Pakistan, Sri Lanka and Bangladesh. Today, immigrants from South Asia live almost all over the world. In the UK there are over 3 million South Asians and in Norway over 50,000. South Asians in UK comprises Pakistanis, Sri Lankans, Indians and Bangladeshis but South Asians in Norway are mainly from Pakistan, Sri Lanka and India. Since the number of Bangladeshis in Norway is few, no study was conducted on their health status. Therefore, the studies from Norway which is incorporated in this thesis are conducted mainly on Pakistanis, Sri Lankans and Indians.

This thesis contains 8 chapters. The first part, chapter 1, is introduction. Chapter 2 presents theories of health inequality briefly. Chapter 3 portrays a background history of immigrants from the Indian subcontinent, when and how they came to Norway and the UK. Chapter 4 gives a brief account on what diabetes is, how it is diagnosed and complications related to the disease. Chapter 5 deals with the genetics and biological factors underlying T2DM. Chapter 6 describes the impact of socioeconomic position (SEP) on the high prevalence of T2DM among South Asians. Chapter 7 is dedicated to the culture, migration, lifestyle and obesity connected factors. In chapter 8 the thesis discusses the central issues, presents some recommendations and puts conclusion. A complete list of literature used in thesis is presented at the end of the paper under the bibliography section.

#### 1.4 Material and Method

This thesis is a literature review. I used relevant books and articles for this thesis. The sources for data were Medline, Google scholar and other relevant data bases. Key words like diabetes and South Asians, genetics of diabetes, socioeconomic position and diabetes, ethnic inequalities of health, diabetes and migration, diabetes and culture, diabetes and lifestyle are used. In addition to that repeated search was conducted using some free text and key authors by name. The results are further refined in accordance with their relevance. Special attention and focus was given to research conducted in Norway and the UK.

## 2. ETHNIC INEQUALITIES IN HEALTH

According to WHO *health inequalities* can be defined as differences in health status or in the distribution of health determinants between different population groups. For example, differences in mobility between elderly people and younger populations or differences in mortality rates between people from different social classes can be referred to as health inequalities (WHO 2012b).

Similarly, Kwachi et al (2002) describes health inequality as the broad term used to designate differences, variations, and disparities in the health achievements of individuals and groups. A higher incidence of T2DM among South Asian immigrants as compared to the population of the host country can be one straight forward example of health inequality. If T2DM is randomly or equally distributed among all groups of the population then there is no presence of health inequality as to T2DM in that population (Kawachi, Subramanian, and Almeida-Filho 2002)

It is important to distinguish between *inequality* in health and *inequity*. The two terms are sometimes confused, but are not interchangeable; inequity refers to unfair, avoidable differences arising from poor governance, corruption or cultural exclusion while inequality simply refers to the uneven distribution of health or health resources as a result of genetic or other factors or the lack of resources. Some health inequalities are attributable to biological variations or free choice and others are attributable to the external environment and conditions mainly outside the control of the individuals concerned. As in the first case, if the variation is due to biology or free choice, it may be impossible or ethically or ideologically unacceptable to change the health determinants and so the health inequalities are unavoidable. However, as in the second case, if the variation are attributable to external environment and conditions outside the control of the individuals concerned, then the uneven distribution may be unnecessary and avoidable as well as unjust and unfair, so that the resulting health inequalities also lead to inequity in health (WHO 2012b).

The core of the difference between equality and equity is that the identification of health inequities entails normative judgment based upon one's theories of justice, one's theories of society, and one's reasoning underlying the origin of health inequalities. Because

identifying health inequities involves normative judgment, science alone cannot determine which inequalities are also inequitable, nor what proportion of an observed inequality is unjust or unfair (Kawachi, Subramanian, and Almeida-Filho 2002).

Some researchers suggest that most of the ethnic inequalities in health are unjust because they reflect an unfair distribution of the underlying social determinants of health including access to educational opportunities, safe jobs, health care, and the social bases of self respect (Daniels 2001; Woodward and Kawachi 2000). On the other hand, some others would deny any role of social injustice in the creation of health inequalities. Much of this debate revolves around the issues of free will and individual responsibility for self care. Those who emphasize individual responsibility tend to view health inequalities as the outcome of differences in how people make choices (for example, the decision to start smoking, or to adhere to a risk taking hobby), whereas social determinists view the same choices as arising out of constrained and unfair circumstances like targeting of tobacco advertising to low income children (Kawachi, Subramanian, and Almeida-Filho 2002).

The discussion of what is unfair and unjust about health inequities takes us beyond the scope of this thesis into moral and political philosophy, where a long-running argument about equality and justice has generated a large, complex and never ending debate.

Back to the issue at hand, inequality by race and ethnic group, potentially, is a powerful tool for scientific analysis and for social action in the field of health. Answers to questions such as “why, in comparison to the rest of the population as a whole, is diabetes so common among immigrants from the Indian subcontinent?” holds important information about the causes of the disease and benefits all populations because results are likely to be generalizable (Bhopal 2007, 152).

Health status, disease occurrence and mortality patterns in populations are sculpted by factors such as wealth, environmental quality and protection, diet, behavior, occupational and domestic stress, and genetic inheritance. Varying exposures to these and other factors by ethnic group over long timescales generate ethnic differences (Bhopal 2007, 152).

Bhopal (2007) points out 6 major factors which can possibly generate or influence ethnic health inequality. These factors are: socio economic position, culture, migration, lifestyle, access to health care and last but not least genetic or biological factors (Bhopal 2007, 153).

Similarly, Kumar and Viken (2010, 39-40) classify theories which can explain ethnic health disparities into 6 categories. Accordingly, the ethnic health differences can be due to one or more of the following: consequences of migration, result of difference in socio economic position, happened as a result of cultural difference/lifestyle factors, the result of genetic or biological differences, inaccessibility to health services or it can be the result of statistical errors.

This thesis looks into the above mentioned factors in order to investigate- if they can explain the high prevalence of T2DM among immigrants from the Indian Subcontinent.

### 3. IMMIGRANTS FROM THE INDIAN SUBCONTINENT

The 1960s discovery of North Sea oil changed Norway and its society once and for all. As a result of oil export the country developed into one of the wealthiest countries in the world. The booming economy opened new opportunities and created a new demand in the labor market. So that, the country became attractive for immigrants outside the European continent (Kumar and Viken 2010, 42; Statistics Norway 2012).

Immigrants from the Indian subcontinent were among the first non-European immigrant groups to arrive in Norway. In fact, Pakistanis were the first in the group. The first generation Pakistani immigrants who arrived in Norway during 1967 were young men as guest workers under Norway's then-liberal immigration scheme which allowed for unskilled "guest workers" to temporarily settle in Norway. The law was later amended to allow for already arrived guest workers to permanently settle in Norway. Following stricter immigration laws passed in 1976, Pakistan immigration to Norway shifted from the arrival of new immigrants, to family reunifications, in which Pakistani Norwegians could apply for their close relatives and/or spouses to immigrate to Norway (Kumar and Viken 2010, 42; Statistics Norway 2012).

According to the latest figure from Statistic Norway currently there are 31884 Pakistanis, 14017 Sri Lankans and 8484 Indians in Norway. The immigrant population is concentrated in the big cities like Oslo. At the beginning of 2011, Oslo had the largest population of immigrants and Norwegian-born to immigrant parents, both in relative and absolute figures. Of Oslo's 599 200 inhabitants, 170 200 were immigrants or Norwegian-born to immigrant parents, which is 28.4 per cent of the capital's entire population. Immigrants from the Indian subcontinent in Oslo constitute 33463. According to Statistic Norway there are 22034 Pakistanis, 7365 Sri Lankans and 4064 Indians in Oslo (Statistics Norway 2012).

In UK the first mass migration of South Asians happened in the early 1950s when Indians from the Punjab province of both India and Pakistan, and the province of Sylhet which is now in Bangladesh arrived in Britain. This first mass migration can roughly be divided along religious lines into Muslims from Pakistan and mostly Sikhs from India. The people from Sylhet were Bangladeshi Muslims. These immigrants were blue collar workers who lived and worked in factories in inner city areas of the UK. The main concentrations are

still found around the West Midlands, Manchester, Bradford and London. They were not well educated and their knowledge of English was poor. Over the last 50 years there have been at least two generations of South Asians born and educated in the UK. Many still live in poor, socially deprived inner city areas. The education and earnings of this group of immigrants is often low and there are still communication difficulties and the level of understanding of English is commonly poor (Bond 2012).

The second mass migration occurred in the mid 1970s when, due to political disturbances in East Africa, South Asians living there migrated to the UK. These people were mostly Gujaratis; they were well educated and most had been established businesspeople in East Africa. They settled around Leicester and London and most are Hindus (Bond 2012).

According to the 2011 UK Census, there were approximately 3.3 million South Asians in UK representing around 5.3% of the total population. This is up from around 2.3 million South Asians in the 2001 census. Those of Indian origin comprised 2.5% of the population, people of Pakistani origin comprised 2.0%, and around 0.8% were of Bangladeshi origin (“2011 Census - Ethnicity” 2012).

#### 4. DIABETES MELLITUS

Diabetes mellitus, or simply diabetes, is a group of metabolic diseases in which a person has high blood sugar, either because the body does not produce enough insulin, or because cells do not respond to the insulin that is produced. This high blood sugar produces the classical symptoms of frequent urination, increased thirst and increased hunger (WHO 2012c; Gardner and Shoback 2011).

There are three main types of diabetes mellitus. Type 1 diabetes mellitus results from the body's failure to produce insulin, and presently requires the person to inject insulin or wear an insulin pump. This form was previously referred to as "insulin-dependent diabetes mellitus" (IDDM) or "juvenile diabetes". T2DM results from insulin resistance, a condition in which cells fail to use insulin properly, sometimes combined with an absolute insulin deficiency. This form was previously referred to as non insulin-dependent diabetes mellitus (NIDDM) or "adult-onset diabetes". The third main form, gestational diabetes occurs when pregnant women without a previous diagnosis of diabetes develop a high blood glucose level. It may precede development of T2DM. T2DM which is the main focus of this paper comprises 90% of people with diabetes around the world (WHO 2012c; Gardner and Shoback 2011).

##### 4.1 Diabetes Diagnosis and its Consequences

Diabetes is diagnosed if the (venous) fasting plasma glucose (FPG) value is  $\geq 7.0$  mmol/l (126 mg/dl), or if the casual plasma glucose value is  $\geq 11.1$  mmol/l (200 mg/dl), or if the plasma glucose value 2 hours after a 75g oral load of glucose  $\geq 11.1$  mmol/l (200 mg/dl). In asymptomatic subjects, performing the test on one occasion is not enough to establish the diagnosis (i.e. basis to treat diabetes). This must be confirmed by carrying out at least one further test on a subsequent day (WHO 2012a).

Impaired glucose tolerance (IGT) and impaired fasting glycaemia (IFG) are risk categories for the future development of diabetes and cardiovascular disease (CVD). An individual falling into the IFG category on the fasting result may also have IGT on the 2-h value or, indeed, diabetes. If an individual falls into two different categories, the more severe one applies (WHO 2012c).



Having diabetes can also put patients at a higher risk of developing different diabetes related complications. Diabetes complications can be divided into microvascular and macrovascular complications; and most of the consequences of diabetes result from its macrovascular and microvascular complications. Microvascular complications are complications which happen due to damage to small blood vessels whereas macrovascular complications are complications which occur as a consequence of the damage to larger blood vessels. Microvascular complications include damage to eyes (retinopathy) leading to blindness, to kidneys (nephropathy) leading to renal failure, and to nerves (neuropathy) leading to impotence and diabetic foot disorders including severe infections leading to amputation. Macrovascular complications include cardiovascular diseases such as heart attacks, strokes and insufficiency in blood flow to legs (WHO 2012a).

## 5. GENETIC AND BIOLOGICAL DIFFERENCES

Genetic factors have been widely considered to play a role in the increased risk of T2DM in South Asians, but studies carried out in this regard on South Asian population are relatively few. A complete understanding of the full picture of the genetics and the biological processes underlying T2DM is still an ongoing process and the final result is yet to come. What makes the genetics of T2DM complicated is that there are a lot of genes associated in the process. Radha and Mohan (2007) assert that T2DM is a polygenic disorder with multiple genes located on different chromosomes contributing to its susceptibility. Analysis of the genetic factors is further complicated by the fact that numerous environmental factors interact with genes to produce the disorder. Only a minority of cases of T2DM is caused by single gene defects and one example is maturity onset diabetes of the young (MODY) (Radha and Mohan 2007).

In the study of the genetics of T2DM two major approaches are commonly used – *candidate gene* and *genome-wide association studies*. The candidate gene approach to conducting genetic association studies focuses on associations between genetic variation within pre-specified genes of interest and phenotypes or disease states. This is in contrast to genome-wide association studies, which scan the entire genome for common genetic variation. Candidate genes are most often selected for study based on a priori knowledge of the gene's biological functional impact on the trait or disease in question. A genome-wide association study also known as whole genome association study is an examination of many common genetic variants in different individuals to see if any variant is associated with a trait. A genome-wide association study typically focuses on associations between single-nucleotide polymorphisms and traits like major diseases (Malecki 2005; Baier and Hanson 2004).

### 5.1 Genes Associated with T2DM

In terms of their genetic makeup, T2DM is categorized under two forms namely monogenic forms of diabetes and polygenic forms of diabetes. Significant progress has been made in the characterization of monogenic forms of T2DM using these techniques but the study of the complex polygenic T2DM has, until recently, been much slower. Significant advances in recent years following improved genotyping techniques and the completion of the Human Genome Project have allowed identification of several

susceptibility genes, offering new insights into the pathogenesis of this complex condition (Frayling and McCarthy 2007).

Monogenic forms of diabetes which constitute a very small proportion of T2DM (<5%), are a consequence of rare mutations in a single gene. These mutations substantially change the structure and subsequently the function of a protein. Monogenic forms are characterized by high phenotypic penetrance, early age of diagnosis, and a distinct clinical picture. Genetic background plays a critical role in their pathogenesis, while the environment only slightly modifies the clinical picture (Radha and Mohan 2007).

A polygenic form of diabetes which is also called multifactorial is the genetics of the common variety of T2DM. This form of T2DM is a result of the interaction between the environment and multiple genes. The susceptibility is associated with frequent polymorphisms that create amino acid variants in exons or influence the expression of genes in the regulatory parts (Frayling and McCarthy 2007). Alleles of these polymorphisms are present in both healthy individuals and T2DM patients, although with different frequencies. These sequence variants are associated with just a limited increase in the risk of developing the disease. They can be considered susceptibility variants, but by themselves are not causative factors that unequivocally determine the disease (Radha and Mohan 2007).

Although there have been fewer genetic studies involving South Asians in comparison with the western populations, recent studies using the genom-wide scanning technique have identified genes associated with the high prevalence of T2DM in the South Asian population.

In 2011 an international team of researchers led by Imperial College London (Kooner et al. 2011) has identified six new genetic variants associated with T2DM in South Asians. This new study is the first to focus on genes underlying diabetes amongst people originating from South Asia. The researchers examined the DNA of 18,731 people with T2DM and 39,856 healthy controls. The genomes of the participants were analyzed to look for locations where variations were more common in those with diabetes. The results identified six positions where differences of a single letter in the genetic code were

associated with T2DM, suggesting that nearby genes have a role in the disease (Kooner et al. 2011).

From time to time researchers are coming up with newly discovered genes which say something new about the genetic nature of T2DM. Just recently another team of international scientists from the Diabetes Genetics Replication and Meta-analysis (DIAGRAM) Consortium used a new DNA chip to probe deeper into the genetic variations that commonly occur in the DNA and which may have some connection T2DM. They discovered 10 more DNA regions linked to T2DM, bringing the total known genetic variations associated with T2DM to more than 60 (Consortium 2012).

Among the previous discoveries the Calpain 10 gene is one of the genes said to have association with increased risk of T2DM among South Asians. A haplotype of three important polymorphisms of this gene which is shown to be connected with an increased risk of T2DM in a Mexican American population indicated mixed results in other populations. Studies in South Asians have shown that the original haplotype increase the risk of T2DM. The frequency of this polymorphism is very low and its contribution to the risk of T2DM is, therefore, small (Evans et al. 2001).

Another important gene in this regard is the peroxisome proliferator activator gamma (PPAR gamma) which is an essential regulator of glucose and lipid metabolism. A common Pro12Ala polymorphism of this gene has been shown to be protective in white populations (Altshuler et al. 2000). A study conducted by Radha et al. (2006) on 697 South Asians and 457 Caucasians living in Dallas/Forth Worth, Texas, and 1,619 South Asians living in Chennai, India tried to determine whether the peroxisome proliferator-activated receptor (PPAR)- $\gamma$  Pro12ala polymorphism modulate susceptibility to diabetes in South Asians. Although further replication studies are necessary to test the validity of the described genotype-phenotype relationship, the study supports the hypothesis that the PPAR- $\gamma$  Pro12Ala polymorphism is protective against diabetes in Caucasians but not in South Asians. This polymorphism is present at the same frequency in South Asians with and without diabetes, and its presence was not associated with either improved insulin sensitivity or decreased risk of T2DM (Radha et al. 2006).

The Transcription Factor 7 Like 2 (TCF7L2) is another important gene in the list of T2DM susceptibility genes. First described in an Icelandic population, and replicated in several other populations, including South Asians, this gene has been shown to have the strongest association with the risk of T2DM (Zeggini and McCarthy 2007). The exact role of this gene in the pathogenesis of T2DM, however, remains unknown at present.

Many other genes such as PPAR gamma co-activator 1 alpha (PGC-1), ectoenzyme nucleotide polypeptide (ENPP1), uncoupling protein genes (UCP2 and UCP3), insulin receptor substrate (IRS-2), beta cell potassium channel gene (KCNJ11) and adiponectin gene have also been studied in South Asians and shown to have modest associations with T2DM (V. Radha and Mohan 2007). Small sample sizes, however, make it difficult to interpret these studies or to exclude possible associations.

Moreover, Radha and Mohan (2007) reviewed the studies on genetics of diabetes in Asian Indians. There appears to be certain genes which predispose Indians to diabetes while other genes which afford protection against diabetes and insulin resistance to Caucasians, do not appear to protect Indians (Radha and Mohan 2007).

In addition to that, scientists have studied if the fat burning mechanism of the muscles of South Asians has contributed to the high prevalence of T2DM among the population. Hall et al. (2010) have investigated whether differences in oxidative capacity and capacity for fatty acid utilization in South Asians might contribute to the high prevalence of T2DM. An experimental study was conducted on South Asians and Europeans to understand the fat burning rate of the muscles of South Asians and to explore whether differences between South Asians and white Europeans could explain the increased risk of T2DM. The study compared 20 men of South Asian origin with 20 men of white European descent. The researchers focused on whether there were biochemical differences in the way the two ethnic groups metabolized their fat stores. The participants performed exercise tests following a 12-hour overnight fast to look at fat and carbohydrate metabolism (use of fat or carbohydrate as energy sources during exercise). They measured insulin sensitivity by looking at glucose and insulin responses to an oral glucose tolerance test. The patients' glucose and insulin levels were measured after fasting and after they had been given glucose, to see how well their body responded to and managed glucose levels. The

researchers took a blood sample and a muscle and fat biopsy from each participant's thigh to search for genes that may be involved in fat metabolism or the insulin system (Hall et al. 2010).

These findings suggest that there may be differences in fat metabolism during exercise between South Asian and European men. These differences were associated with a reduced sensitivity to insulin, which may contribute to the higher risk of type 2 diabetes in the South Asian population. However, this was preliminary research carried out in a very small number of people – only 20 people were included in each group. The results ideally need to be confirmed in a larger number of people. In particular, a larger study is needed to investigate whether there are ethnic differences in the activity of the genes and proteins involved in fat metabolism and insulin signaling (Hall et al. 2010).

## 5.2 Genes Associated with Obesity

Obesity is one important risk factor for T2DM. Recent findings suggest that genes play important role in obesity. The indirect scientific evidence for a genetic basis for obesity comes from a variety of studies. Mostly, this evidence includes studies of resemblance and differences among family members, twins, and adoptees. Another source of evidence includes studies that have found some genes at higher frequencies among the obese i.e. genome-wide association studies. These investigations suggest that a sizable portion of the weight variation in adults is due to genetic factors. Two important genes are identified in this regard namely *fat-mass and obesity-Associated gene (FTO)* and *Melanocortin 4 Receptor (MC4R) gene* (Frayling et al. 2007; Chambers et al. 2008).

Common variants of the fat-mass and obesity-associated gene (FTO) were shown to be associated with obesity in European populations (Frayling et al. 2007). Individuals with these variants were on average 3kg heavier than those who did not possess them. Those individuals were also at an increased risk of T2DM but this was secondary to the obesity rather than due to the variants of the gene itself. In South Asians, however, the presence of this polymorphism was associated with an increased risk of T2DM independent of body mass index (BMI) (Yajnik et al. 2009; Chambers et al. 2008).

Melanocortin 4 Receptor (MC4R) gene is another important gene identified in a study with South Asians and Europeans living in the UK (Chambers et al. 2008). This study found that the variant of the MC4R gene was associated with increased risk of adiposity and insulin resistance. Individuals with variants of this gene had a waist circumference around 2cm larger and insulin resistance (HOMA-IR) approximately 10 per cent greater than those who do not have the MC4R variation. The increased frequency of the risk allele in South Asians has been proposed as an explanation for the increased levels of T2DM in this group (Chambers et al. 2008).

### 5.3 Genes Associated with Diabetes Complications

Like T2DM itself and obesity, the predisposition to diabetic complications also varies considerably among ethnic groups. Researches indicate that the prevalence of diabetic nephropathy and retinopathy is higher in South Asians than in white populations (Raymond et al. 2009; Mather, Chaturvedi, and Kehely 1998). Risk of these complications, particularly nephropathy, is thought to be genetically determined. Researchers have looked at polymorphisms of the Angiotensin Converting Enzyme (ACE1) gene, aldosterone synthase gene (for nephropathy) (Prasad et al. 2006) and the Vascular Endothelial Growth Factor (VEGF) gene (Uthra et al. 2008) for retinopathy. Findings from these studies, however, have not been conclusive and need to be verified in larger cohorts.

### 5.4 Different Hypothesis Elucidating High Prevalence of T2DM

Parallel to the ongoing comprehensive study of the genetics of the disease, different hypothetical perspectives are also used to elucidate the etiology of the current epidemic of T2DM. In this regard four hypotheses were formulated in the last 50 years or so as to why South Asians are more prone to developing diabetes. The thrifty gene hypothesis, the thrifty phenotype hypothesis, the thrifty epigenomic hypothesis and the drift gene hypothesis constitute theories focusing on in-utero metabolic factors.

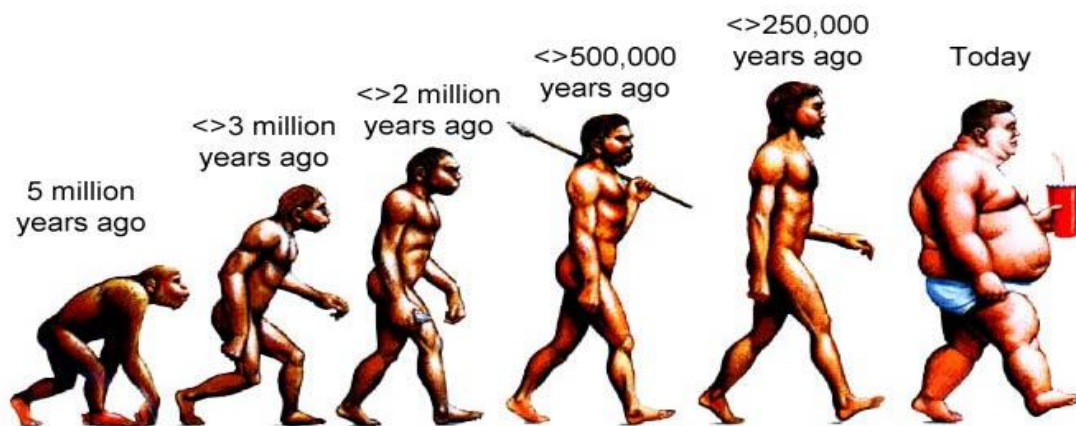


Fig.1. “Are we evolving or devolving?” pictorial representation of evolutionary path way of T2DM and obesity (Source: Google pictures).

#### 5.4.1 Thrifty Genotype Hypothesis

One of the first theories in the last fifty years or so which made attempt to explain the tendency of certain ethnic groups’ high prevalence of obesity and diabetes is the so called “Thrifty Genotype Hypothesis” proposed by geneticist James Neel in 1962. According to this hypothesis certain genes in human have evolved to maximize metabolic efficiency and food searching behavior. In times of abundance food supply these genes predispose their carriers to diseases caused by excess nutritional intake, such as obesity and T2DM (Neel 1962).

The thrifty genotype hypothesis relates low birth weight of South Asians to adult diabetes. It suggests that low nutrient conditions in-utero result in selective survival of infants who have insulin insensitivity allowing for efficient intake and utilization of nutrients. This genotype is beneficial in a low calorie environment, but may increase vulnerability to diabetes in an environment with an abundance of calories, as is found in most of the developed world like Norway and UK. This fits with the high T2DM prevalence among South Asian immigrants in these countries (McCance et al. 1994; Neel 1962).

The hypothesis suggests that the 'thrifty' genotype would have been advantageous for hunter and gatherer populations because it would allow them to fatten more quickly during times of abundance. Fatter individuals carrying the thrifty genes would thus better survive



times of food scarcity. However, in modern societies with a constant abundance of food, this genotype efficiently prepares individuals for a famine that never comes. The result is widespread chronic obesity and related health problems like T2DM (Neel 1962).

This hypothesis has received various criticisms and consequently several modified or alternative hypotheses have been proposed.

#### 5.4.2 Thrifty Phenotype Hypothesis

The challenges posed to the thrifty gene hypothesis gave rise to another theory called “the thrifty phenotype hypothesis”. The thrifty phenotype hypothesis theorizes that instead of the "thrifty factors" arising from genetic factors, that it is a direct result of the environment within the womb during development that brought about the epidemic. The development of insulin resistance is theorized to be directly related to the body "predicting" a life of starvation for the developing fetus (Watve and Yajnik 2007).

As it is presented in figure 2, the thrifty phenotype hypothesis proposes that the epidemiological associations between poor fetal and infant growth and the subsequent development of T2D and the metabolic syndrome result from the effects of poor nutrition in early life, which produces permanent changes in glucose-insulin metabolism. These changes include reduced capacity for insulin secretion and insulin resistance which, combined with effects of obesity, ageing and physical inactivity, are the most important factors in determining T2DM (Watve and Yajnik 2007).

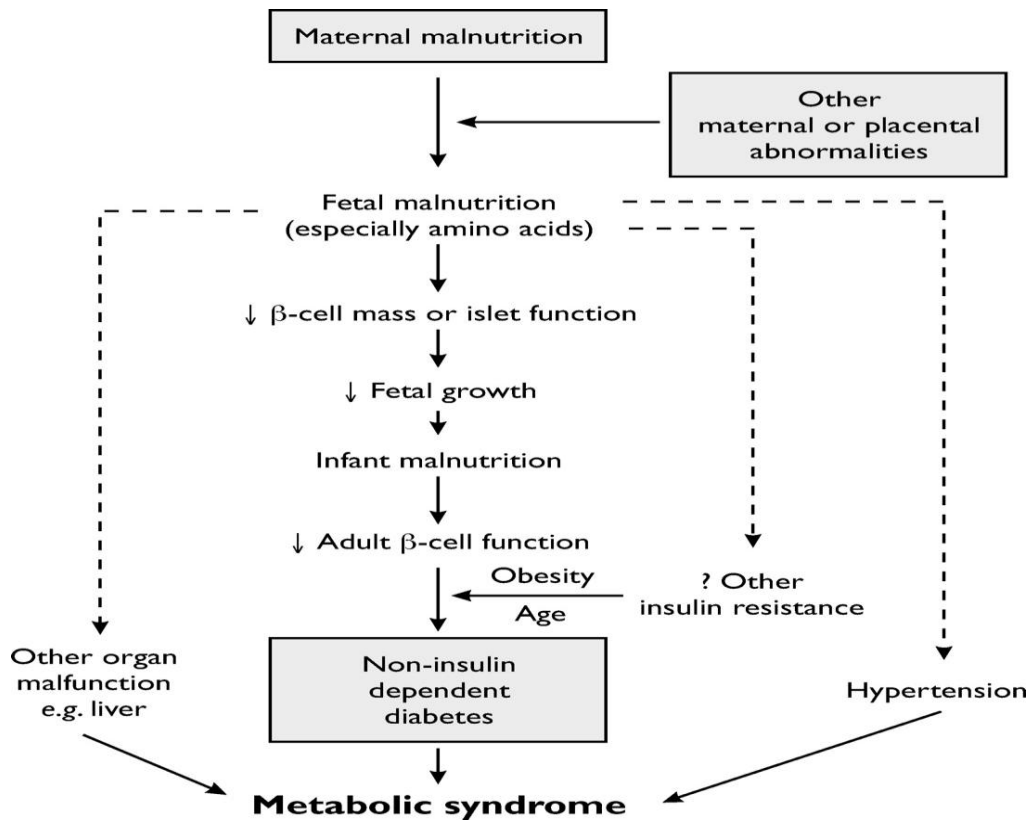


Fig.2: The original diagrammatic representation of the thrifty phenotype hypothesis (Hales and Barker 2001).

Hence, one of the main causes of T2DM has been attributed to poor fetal and infant growth and the subsequent development of the metabolic syndrome.

Since the hypothesis was proposed, many studies world-wide have confirmed the initial epidemiological evidence. Although the relationship with insulin resistance is clear at all ages studied, the relation of insulin secretion is less clear. The relative contribution of genes and environment to these relationships remains a matter of debate (Hales and Barker 2001).

The theory has been modified latter on. An updated version of the diagrammatic representation of the thrifty phenotype hypothesis is shown in Figure 3. Also included in the diagram are more speculative suggestions that changes in the structure and function of blood vessels may play a key role in changing organ growth and function, and that maternal hyperglycaemia may contribute to the type and consequences of fetal malnutrition.

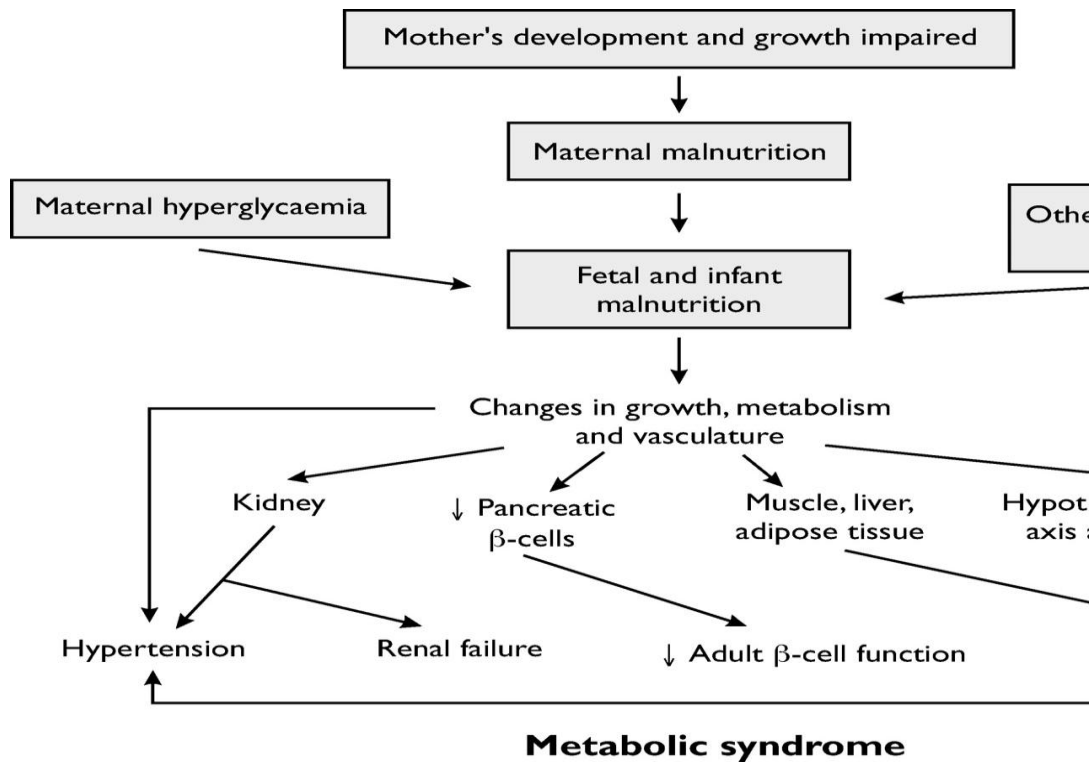


Fig.3: An updated diagram of the thrifty phenotype hypothesis incorporating recent findings and concepts. Also included are new speculative features: maternal hyperglycaemia as predisposing factor and key roles of the vascular, hypothalamic-pituitary-adrenal axis and sympathetic systems (Hales and Barker 2001).

Studies suggest that South Asians tend to have lower birth weight. Hales and Barker hypothesize that lower birth weight relates to T2DM with their thrifty phenotype hypothesis, which attributes the vulnerability to diabetes to environmental factors in the womb rather than genetic characteristics in the fetus. They suggest that nutritional deficiencies in utero, which result in lower birth weight, may also lead to reduced  $\beta$ -cell mass or impaired  $\beta$ -cell function. These atypical  $\beta$ -cells may be unable to produce sufficient insulin throughout the individual's life, resulting in overt diabetes when  $\beta$ -cell insulin secretion is unable to compensate for increased metabolic demand (Hales and Barker 2001).

Other relevant observations arose from metabolism researchers who note that for practically every other species on earth, fat metabolism is well regulated (International Congress on Obesity, Hirsch, and Itallie 1985) and that "most wild animals are in fact very lean" and that they remain lean "even when adequate food is supplied."

#### 5.4.3 Drifty Gene Hypothesis

As an alternative to the thrifty gene hypothesis the British biologist John Speakman presented “Drifty Gene Hypothesis”. The critique of Speakman (2006) to the thrifty gene hypothesis is based on an analysis of the pattern and level of mortality during famines. Despite much anecdotal evidence used to suggest that famines cause substantial mortality, Speakman suggests that where real data are available famines actually involve rather low levels of mortality and there is no evidence that fat people survive famines better than lean people. In fact mortality actually falls mostly on groups such as the very young and very old where differential mortality in relation to body composition is highly unlikely (Speakman 2008; Speakman 2007).

#### 5.4.4 Thrifty Epigenomic Hypothesis

The other alternative hypothesis which was proposed in response to the criticisms of the original thrifty genotype theory for explaining the evolutionary bases of obesity and related diseases was the "Thrifty Epigenomic Hypothesis". The "thrifty epigenomic hypothesis" is a combination of the thrifty phenotype and thrifty genotype hypotheses. While it argues that there is an ancient, canalized (genetically coded) physiological system for being "thrifty", the theory argues that an individual's disease risk is primarily determined by epigenetic events. Slight, epigenetic modifications at many genomic loci (gene regulatory networks) alter the shape of the canal in response to environmental influences and thereby establish a predisposition for complex diseases such as metabolic syndrome. There may be epigenetic inheritance of disease risk (Watve and Yajnik 2007).

Watve and Yajnik (2007) suggested that changing insulin resistance mediates two phenotypic transitions: a transition in reproductive strategy from "r" (large number of ill-nurtured offspring) to "K" (smaller number of carefully nurtured offspring) and a switch from a lifestyle dependent upon muscular strength to one dependent on brain power. Because the environmental conditions that would facilitate each transition are heavily overlapping, the scientists surmise, a common switch could have evolved for the two transitions (Watve and Yajnik 2007).

## 6. SOCIOECONOMIC POSITION AND DIABETES

Socioeconomic position (SEP) refers to the social and economic factors that indicate what positions individuals or groups hold within the structure of a society, such as educational level, income or wealth. Socioeconomic inequalities in health are the differences in opportunities for maintaining good health between people with different SEPs (Krieger, Williams, and Moss 1997).

Similarly, House and Williams (2001) describe SEP as individuals' position in a system of social stratification that differentially allocates the major resources enabling people to achieve health or other desired goals. These resources centrally include education, occupation, income and assets or wealth, which are related to each other and to health in a casual framework as elucidated in a simple model in figure 4 below (House and Williams 2001, 83).

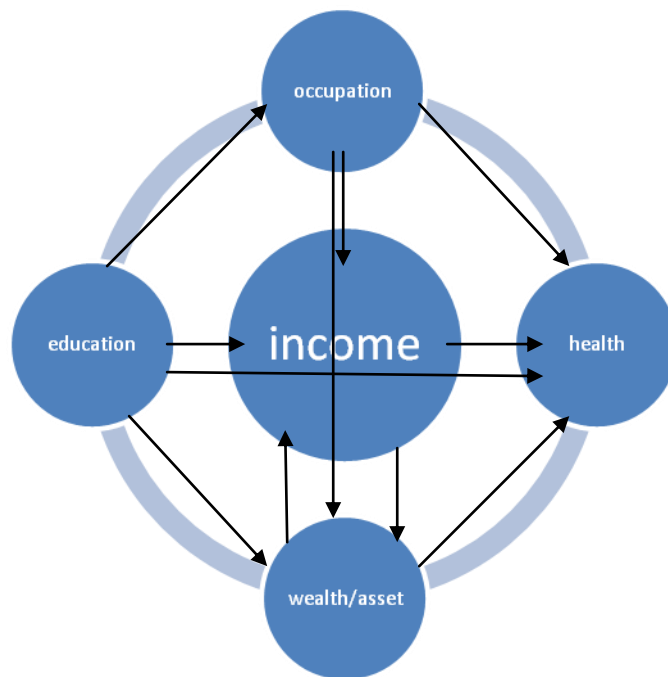


Fig.4 Simple causal model relating major indicators of socioeconomic position to each other and to health (House and Williams 2001, 84).

This model suggests that over the life course, individuals first acquire varying levels and types of education which in turn help them to enter various types of occupations which then yield income, which finally enables them to accumulate assets or wealth. Each subsequent variable in this casual chain is generally most affected by the immediately

prior variable with potential residual effects of earlier variables (House and Williams 2001, 83).

However, Brown et al. (2004) do not agree with the fact that research on the relation between SEP and health has often paid attention only to income, wealth, education, and occupation. They argue that SEP should take into account other wider aspects. Consequently, Brown et al. (2004) presented a much broader model as shown in figure 5 below which illustrated how SEP influences health among persons with diabetes through community factors (e.g. availability of healthy foods, availability of places to exercise), health behaviors (e.g. diet, physical activity), access to healthcare and processes of diabetes care (e.g. measurement of HbA1c, smoking cessation) (Brown et al. 2004).

Path way 1 in fig.5 below illustrates the relation between SEP and health outcomes in persons with diabetes. Research on the relation between SEP and health has often focused on individual characteristics such as income, wealth, education, and occupation. However, SEP encompasses not only current individual socioeconomic status but also social relationships, community-level characteristics, and gradients of SEP at the individual and community levels, and it can be conceptualized and measured over the life course. Use of this broader framework may provide greater insights into the relation between SEP and health. For example, because the progression of both type 1 diabetes and type 2 diabetes can be influenced by behavior over the life course, SEP in childhood may have profound consequences for long-term health, even if SEP changes during adulthood. In addition, neighborhoods or communities may play an instrumental role in the health status of their residents through the availability of health care services, neighborhood characteristics that promote health (e.g., access to stores that sell healthy foods and places to exercise) or disease (e.g., toxic environments), and the prevailing attitudes toward health and health behaviors in those communities (Brown et al. 2004).

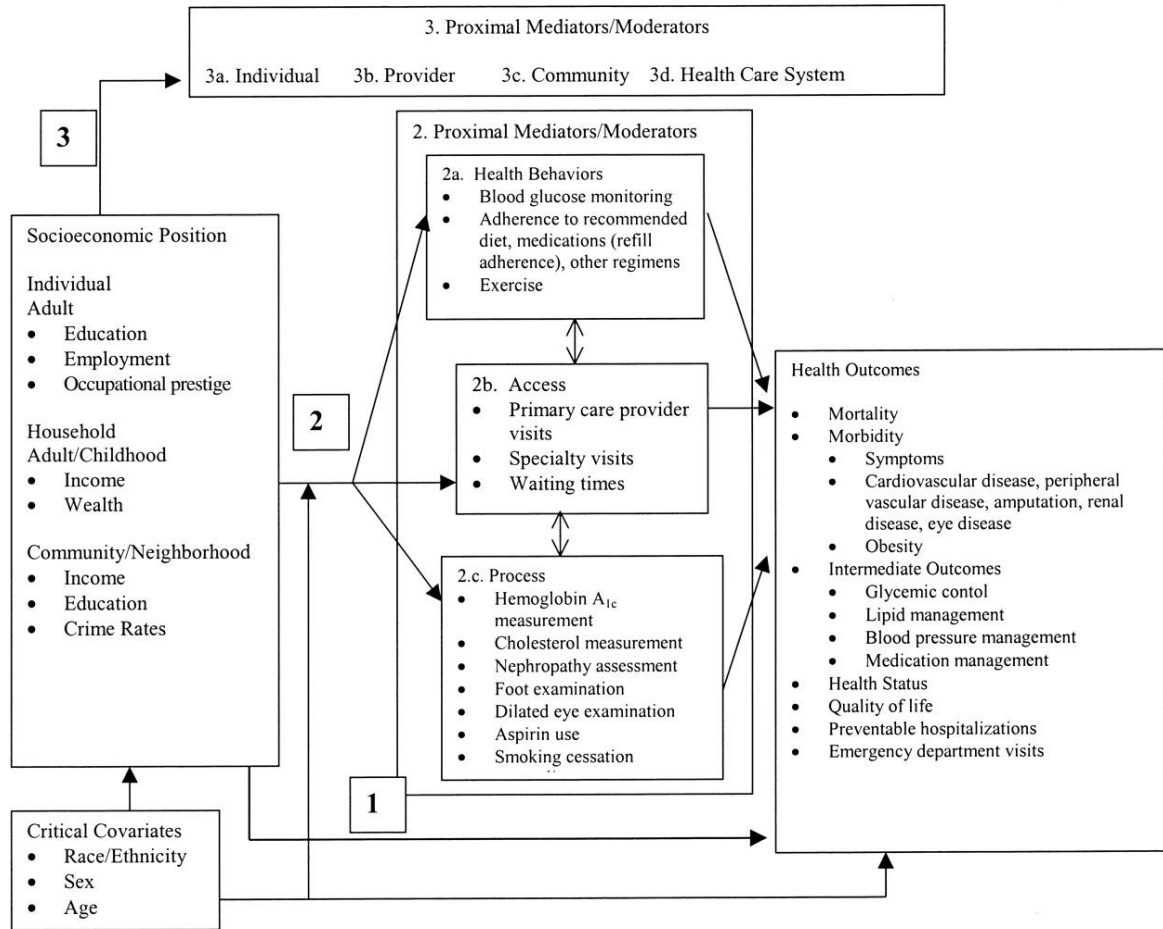


Figure 5: Conceptual framework for the relation between socioeconomic position and health among persons with diabetes mellitus. Numbers and letters refer to pathways mentioned in the text (Brown et al. 2004).

According to Helman (2007) economic factors and social inequality are some of the most important causes of ill health, since poverty may result in poor nutrition, overcrowded living conditions, housing or work sited in areas with greater environment dangers (such as near factories producing toxic chemical), as well as exposure to physical and drug and alcohol abuse. The unequal distribution of wealth and resources and of access to health care facilities – both between countries and within each country itself – can also lead to this situation. In many Western countries these disparities are particularly evident in ethnic or cultural minority groups, whether they are immigrant or native borne (Helman 2007, 5).

Nevertheless, researchers do not always agree on the contribution of SEP to health inequality. The role of SEP in explaining ethnic differences in health is widely contested.

For Wild and McKeigue (1997), for example, SEP has minimal or no contribution in explaining ethnic inequalities in health. They assert that other factors like cultural and genetic elements play larger role. Wild and McKeigue (1997) who compared mortalities for selected groups of immigrants with the national average in England and Wales ruled out the role of SEP in the observed ethnic inequality in health. They concluded that widening differences in mortality ratios for migrants compared with the general population were not simply due to socioeconomic inequalities. The low mortality from all causes for Caribbean immigrants could largely be attributed to low mortality from ischaemic heart disease, which is unexplained. The excess mortality from cerebrovascular and hypertensive diseases in migrants from both West Africa and the Caribbean suggests that genetic factors underlie the susceptibility to hypertension in people of black African descent (Wild and McKeigue 1997).

On the other hand, many researchers like Navarro (1990) and Sheldon & Parker (1992) underline the role of SEP in explaining ethnic differences in health. They emphasize that ethnic differences in health are predominately determined by socio-economic inequalities (Kumar and Viken 2010, 39–40; Sheldon and Parker 1992).

Likewise, several studies in Europe which have analyzed the relationship between socioeconomic inequalities and chronic diseases (Larrañaga et al. 2005; Dalstra et al. 2005), or mortality (Avendaño et al. 2005; Avendano et al. 2006) found an association between health and SEP. Among populations of disadvantaged SEPs the majority of health indicators are worse. The main factors that relate SEP to health and diabetes are general socioeconomic and political context, working and living conditions, health behaviors and psychosocial factors (Solar and Irwin 2007).

A number of specific studies have been conducted to find out the correlation between T2DM and SEP. One of the many studies aimed at finding out the association of SEP and the prevalence of T2DM is a comprehensive study by Espelt et al. (2008) which was conducted in different areas across Europe to determine and quantify SEP inequalities in T2DM. The study concluded that educational attainment and T2DM were inversely related, in terms of both morbidity and mortality rates (Espelt et al. 2008).



The study analyzed data from ten representative national health surveys and 13 mortality registers including Norway. For national health surveys the dependent variable was the presence of T2DM by self-report and for mortality registers it was death from diabetes. Educational level (SEP), age and sex were independent variables, and age-adjusted prevalence ratios (PRs) and risk ratios (RRs) were calculated (Espelt et al. 2008).

The study indicated that low SEP was related to a higher prevalence of T2DM, for example men who attained a level of education equivalent to lower secondary school or less had a PR of 1.6 compared with those who attained tertiary level education, whereas the corresponding value in women was 2.2. Moreover, in all countries, having a disadvantaged SEP is related to a higher rate of mortality from T2DM and a linear relationship is observed. Eastern European countries have higher relative inequalities in mortality by SEP. According to the data, the RR of dying from diabetes for women with low SEP is 3.4, while in men it is 2.0. According to this study A lower level of education is associated with high risk of T2DM (Espelt et al. 2008).

Similarly, Sacerdote et al. (2012) who studied the association of SEP and risk of T2DM in 7 western European countries found out that Lower educational level is a predictor of incident T2DM in European countries. This study demonstrates the inequalities in the risk of T2DM in Western European countries, with an inverse relationship between educational level and risk of T2DM that is only partially explained by variations in BMI (Sacerdote et al. 2012).

Similarly, a team of researchers in Calgary, Canada who studied the association of socio-economic status with T2DM prevalence indicated that low income is associated with a higher prevalence of T2DM (Rabi et al. 2006).

Another study which was conducted to find out the association of SEP and T2DM was the one which was conducted in the Hague (Middelkoop et al. 1999). The Municipal Health Service in Hague has investigated the prevalence of self reported T2DM among the South Asian inhabitants of the city and the relationship between T2DM prevalence and socioeconomic status in this population. In this study a total of 3,131 South Asians over 30 years of age, randomly selected (stratified according to age and sex) from the

municipal register, were included in a postal survey with subsequent telephone interviews with non-respondents (Middelkoop et al. 1999).

The results reveals that Self-reported T2DM prevalence varies from 6.4% in the 31-49 year age group to 37.1% in the over 60s age group. The relationship with age varies across the different socioeconomic strata. There are only a few people with T2DM in the 31-39 year age group in the highest stratum, whereas in the lowest stratum at the same age the prevalence of self-reported diabetes approximates 20%. In the over 60s age group the prevalence of diabetes does not significantly differ between the higher and lower socioeconomic strata (Middelkoop et al. 1999).

Bhopal et al (1999), although not found some important differences while making broad comparisons between all South Asians and Europeans, they indicate that South Asians were still disadvantaged across a wide range of risk factors (Bhopal et al. 1999).

A population based study in UK which looked into the relation between SEP and the age-sex specific prevalence of T2DM confirmed an inverse association between SEP and the prevalence of T2DM in the middle years of life. This finding suggests that exposure to factors that are implicated in the causation of diabetes is more common in deprived areas showing increased prevalence of T2DM in deprived areas (Connolly et al. 2000). Moreover, another study conducted in Scotland indicated that the prevalence of T2DM increases with the degree of deprivation. Among 366 849 Tayside residents in Scotland, 792 and 5474 patients with Type 1 and Type 2 diabetes, respectively, were identified from a diabetes register. (1 – least deprived, 6/7 – most deprived). The prevalence of T2DM, not type 1, was found to be varied by deprivation. People in deprivation category 6 and 7 were 1.6-times more likely to have Type 2 diabetes than those least deprived (Evans et al. 2000).

Similarly, another population-based study in Italy Turin by Gnavi et al.(2008) shows that there are socio-economic inequalities in the prevalence of the disease, particularly in women, and in young people (Gnavi et al. 2008).

In the contrary, one study from Oslo didn't shows a similar relationship between good health and SEP. Syed et al. (2006) conducted a study in order to observe the inequality in health from the perspective of socio-economic factors in relation to ethnic Pakistanis and ethnic Norwegians in Oslo. The socio-economic conditions were found to be inversely related to self-rated health, diabetes and distress for the ethnic Norwegians but not for Pakistanis. The results of the study confirm the overall poor health reported by the ethnic Pakistanis compared to the Norwegians, irrespective of socioeconomic status. The economical gradient is less marked for Pakistanis, and even an opposite trend was observed for distress. For example, individuals with a reported higher education and or higher household income have shown an association with good health among the Norwegians, whereas inconsistent result was noted for the Pakistanis (Syed et al. 2006).

Possible explanations for the disparity in the observed associations of health with education and income for the ethnic Pakistanis and the Norwegians may entail that the ethnic Pakistanis at large belong to the low levels of education and income group. Given the small number of participants from Pakistan belonging to predominantly high education and income strata, it is possible that one does not observe any association of the social gradient with health although the relationship exists. The second possible explanation is that individuals with higher education from the ethnic Pakistani families were not successful in obtaining an employment that may correspond with their educational background. Therefore higher education for the Pakistani's did not result in improved economy and thereby health. Moreover underemployment may interfere with self esteem which may result in stress and depression with an obvious consequence on health. Another factor that could contribute to the lack of positive association between education and health in the Pakistanis is inaccuracy in reporting education. The conclusion was that in studies based on self reporting, the tendency to over report education due to social desirability could not be ignored. Among the SE indices, employment appears to have the maximum impact in explaining higher self reported morbidity among the Pakistanis. This may suggest that being employed, though not necessarily with a high income, has a positive impact on health (Syed et al. 2006).

As it happens in any scientific study there are surprising findings and sometimes conflicting results in the study of the association of prevalence of T2DM and SEP. Nevertheless, in most cases lower socioeconomic status is associated with increased risk of T2DM in an already genetically prone immigrant population of South Asian.

## 7. DIABETES AND CULTURE, MIGRATION AND CHANGE IN LIFESTYLE

In 1871 Taylor defined culture as "that complex whole which includes knowledge, belief, art, morals, law, customs, and many other capabilities and habits acquired by members of society" (Helman 2007, 2).

Similarly, Robert Murphy (1986) offered a more illustrative definition of culture as follows: "Culture means the total body of tradition borne by a society and transmitted from generation to generation. It thus refers to the norms, values, and standards by which people act, and it includes the ways distinctive in each society of ordering the world and rendering it intelligible. Culture is a set of mechanisms for survival, but it provides us also with a definition of reality. It is the matrix into which we are born, it is the anvil upon which our persons and destinies are forged "(Murphy 1988, 14).

From these definitions one can see that culture is a set of guidelines that individuals inherit as members of a particular society, and that tell them how to view the world, how to experience it emotionally, and how to behave in it in relation to other people, to supernatural forces or gods, and to the natural environment. It also provides them with a way of transmitting these guidelines to the next generation by the use of symbols, language, art and rituals. To some extent, culture can be seen as an inherited "lens" through which the individual perceive and understands the world that he inhabits and learns how to live within it. Growing up within any society is a form of enculturation, where by the individual slowly acquires the cultural lens of that society. Without such a shared perception of the world, both the cohesion and continuity of any human group would be impossible (Helman 2007, 2).

Therefore, cultural background has an important influence on many aspects of people's lives, including their beliefs, behaviors, perceptions, emotions, language, religion, rituals, family structure, diet, dress, body image, concept of space and time, and attitudes to illness, pain and other forms of misfortune – all of which may have important implications for health and health care (Helman 2007, 3).

There are a number of factors which can give South Asians their unique sense of cultural identity and belonging which can directly or indirectly affect their health. To understand

the impact of culture on their health in general and on their risk to T2DM in particular, we need to look at a number of factors, including, migration with the changes it entails, physical activity, diet, language barriers, access to health services and attitudes to medical treatment. In the next sections I will discuss these elements one by one.

### 7.1 Migration and T2DM

Migration entails tremendous transitions and changes in the life of immigrants. The changes are multidirectional, both negative and positive and involve moving from low or middle income country to an industrialized country, from rural to urban areas, often rapidly in the course of a few hours or days. Immigrants are often forced to adapt the cultures of the host county while carrying with them their own (Kumar and Viken 2010).

Immigrants from low and middle income countries are likely to end up in phases of the demographic, epidemiological and nutritional transition thus moving from areas of higher fertility and mortality, higher prevalence's of infectious diseases and under nutrition to higher prevalence's of chronic diseases (Popkin 2004). Migration speeds up these transitions that may have already started in the home countries and are close to completion in the host countries. Thus the risk for disease could change with migration as does the prevalence for diseases in particular for conditions influenced by the gene-environmental interaction (Kumar and Viken 2010).

When we talk about ethnic inequalities in health, migration is often mentioned as one of the contributing factors in such a way that migration may alter or accelerate inequalities (Bhopal, 2007). Numerous changes in the socio-cultural environment may occur with migration, which in turn may lead to shifts in socio-economic status, work status, access to health care and life style, including diet and physical activity, and ultimately health. Genetics and early life conditions affect health outcomes later in life and may interact with the changes occurring after migration (Barker 2001; Forsdahl 1977; Eriksson et al. 2003).

It has been long observed that the processes of urbanization or westernization that is associated with migration lead to the availability and abundance of calorie-dense/low-fiber foods and the adoption of sedentary lifestyles. This has consequently led to increased risks

of morbidity and mortality from chronic diet and lifestyle-related diseases like DM2 (Misra and Ganda 2007).

Misra and Ganda (2007) reviewed the impact of migration on the incidence and prevalence of obesity and T2DM in different ethnic groups and populations across the world. The prevalence of obesity and T2DM among the migrant populations of South East Asians in different countries in comparison to the prevalence in their respective countries of origin was among the reviewed. The review portrays the effect of migration on the prevalence of T2DM on the immigrant population. It has been indicated that migration causes changes in the affluence and lifestyle of the immigrants which results in high prevalence of obesity, insulin resistance and T2DM among the immigrant population of South Asians (Misra and Ganda 2007).

The prevalence of the metabolic syndrome was found to be highest in South Asians in the United Kingdom in a cross-sectional study (Tillin et al. 2005). The average values of Body Mass Index (BMI), blood pressure, lipids, blood glucose, and insulin resistance of the migrant Asian Indians tended to be higher than those of urban- or rural-based sedentees in India (Misra and Vikram 2004; Bhatnagar et al. 1995; Patel et al. 2006). For example, migrant Asian Indians living in the United Kingdom were more obese, had higher levels of blood pressure, total cholesterol, and blood glucose, and were more insulin resistant than their siblings living in Punjab, India (Bhatnagar et al. 1995).

Similarly, in a comparison of Gujaratis (originating from the state of Gujarat in India) in Britain with non-migrant Gujaratis in India, the former had higher mean values of BMI, blood pressure, lipids, non-esterified fatty acids, and C-reactive protein and a higher dietary intake of calorie and fat; however, the prevalence of T2DM was similarly high in both samples (Patel et al. 2006). Importantly, the prevalence rate of T2DM in migrant Asian Indians has been found to be consistently higher than in other ethnic groups (McKeigue et al. 1992; Anand et al. 2000).

Important observations from the Diabetes Epidemiology Collaborative analysis of Diagnostic Criteria in Europe (DECODA) study (including 11 cohort studies and comprising 24 335 subjects) on the age- and sex-specific prevalence of T2DM and

impaired glucose regulation in four Asian countries, i.e., India, China, Singapore, and Japan, showed that Asian Indians have the highest prevalence of T2DM (McBean et al. 2004).

Interestingly, a retrospective analysis showed that, during 1993–2001, the greatest increase in the prevalence of T2DM (68%) in the elderly in the United States was seen in Asian populations (McBean et al. 2004).

Furthermore, one unpublished study showed stepwise increases in prevalence of T2DM from rural India (8.4%), urban India (13.6%), and in Asian Indians settled in the United States (17.4%) (Misra, unpublished data).

## 7.2 Migration and Change in Dietary Habit

Diet and nutrition are widely believed to play an important part in the development of T2DM. Knowing the dietary habit of South Asian immigrants and the pattern of the change in dietary habit after migration, therefore, lends a crucial clue in the study of the high prevalence of T2DM among this immigrant group.

Wandel et al. (2008) who studied changes in food habits after migration among South Asians settled in Oslo reported relatively large dietary changes after migration. According to their study both Pakistani and the Sri Lankan immigrant groups had a bi-cultural eating pattern with a heavy reliance on traditional dishes for dinner. Furthermore, both groups showed an increase in oil and meat intake and a decrease in the intake of beans and lentils. However, there were also some differences between the groups. The Pakistani ethnic group had adopted the Norwegian diet to a lesser extent than the Sri Lankans, despite having lived longer in Norway, and the pattern of change of the different types of fat was different in that the Pakistanis focused more on oils and the Sri Lankans on butter and margarine in addition to oil. The results also showed that socio-economic factors and aspects related to integration were able to modify the changes that occurred after migration (Wandel et al. 2008).



One of the important results of the study of Wandel et al. (2008) is the increase in consumption of table/ cooking fat and fat rich foods such as meat and meat products, milk and milk products, snacks and fat rich deserts, biscuits and cakes, results which are in agreement with a very high total fat consumption shown in other studies. According (Wandel et al. 2008) this explains a very high rate of obesity and high prevalence of T2DM among these ethnic groups. As a concluding remark on their study, Wandel et al. (2008) underlined what they called "a point of concern" which is the decline in the use of beans and lentils, beneficial foods which may be useful in dietary efforts to reduce the risk of diabetes (Wandel et al. 2008).

In UK, relatively many studies have been conducted to learn the lifestyle of British South Asians. The question of exactly which aspects of the British South Asian lifestyle predispose to glucose intolerance and dyslipidaemia remains, however, largely unanswered.

In one study (Burden et al. 1994) where the glycaemic and insulinaemic effects of South Asian and a European meal were compared, the South Asian diet induced higher and more prolonged rises in plasma glucose and free insulin levels than the European diet. The Asian meal contained 1,439 kcal (14% protein, 45% carbohydrate, 41% fat, 28g dietary fibre), and the European meal 1,370 kcal (18% protein, 25% carbohydrate, 57% fat, 11g dietary fibre). Fifteen normal volunteers with mixed ethnic origin were tested with each meal after an overnight fast, and on consecutive days in random order. There were no differences at fasting or after one hour between either meal. Two hours after the Asian meal, subjects had a higher degree of glycaemia, and a higher degree of insulinaemia (Burden et al. 1994).

Another study which examined the food intake of 173 South Asian and European men aged 40-69 years in London looked into whether there is an association between the diet and the high rates of coronary disease, non-insulin-dependent diabetes, central obesity and insulin resistance among South Asians. The study could not explain the high coronary risk in South Asian people by any unfavorable characteristic of South Asian diets. However, there was increased concentrations of serum insulin at 2 h post glucose which was associated positively with the carbohydrate intake of the diet (Sevak, McKeigue, and

Marmot 1994). It must be remembered, however, that the dietary preferences of South Asian ethnic groups are very diverse (McKeigue et al. 1985) and generalizations should not be made. Ghee (clarified butter), for example, is in common use in groups originating from northern India and Bangladesh and has been shown to contain atherogenic constituents (Jacobson 1987), but ghee is rarely used by people of South Indian origin, whose risk of atherosclerotic disease is equally high (Balarajan et al. 1984). A nutritional analysis of common Punjabi and Gujarati composite dishes from different households, as calculated from the records of weighed ingredients and portion sizes, showed considerable variation in the fat and energy content of different recipes (Kassam-Khamis, Judd, and Thomas 2000) suggesting the use of 'traditional' recipes for nutritional analysis will be inaccurate.

Lawton et al. (2008) conducted qualitative interview with 23 South Asians to look at the eating pattern of Pakistanis and Indians with T2DM, their perceptions of the barriers and facilitators to dietary change, and the social and cultural factors informing their accounts. Despite considerable diversity in the dietary advice received, respondents offered similar accounts of their food and eating practices following diagnosis. Most had continued to consume South Asian foods, especially in the evenings, despite their perceived concerns that these foods could be 'dangerous' and detrimental to their diabetes control. Respondents described such foods as 'strength-giving', and highlighted a cultural expectation to participate in acts of commensality with family/community members. Male respondents often reported limited input into food preparation. Many respondents attempted to balance the perceived risks of eating South Asian foodstuffs against those of alienating themselves from their culture and community by eating such foods in smaller amounts (Lawton et al. 2008).

As part of the Coventry study of diabetes which was carried out in the Foleshill ward of the city, 612 subjects with different ethnic backgrounds undergoing oral glucose tolerance tests provided information on this aspect of lifestyle. According to the study there were no significant differences in the dietary customs of those with normal glucose tolerance, impaired glucose tolerance and newly diagnosed diabetes. Subjects of South Asian origin ate significantly fewer meals per day than European subjects. Evening meal times were 2–3 h later among South Asians. Europeans ate less fruit but more vegetables and more

brown rice than South Asians. Gujeratis ate more rice, fried snacks and white flour. Moslems were least likely to be vegetarians, to drink alcohol and to use home-made ghee and yoghurt, and Punjabi Sikhs and Hindus ate dhal more frequently than Pakistani Moslems, Gujerati Moslems or Hindus. Most South Asians ate Indian sweets and ‘Western’ snacks (Simmons and Williams 1997).

### 7.3 Migration and Language Problem

Language has particularly a significant role to play in the process of migration and societal integration. It constitutes both the medium of everyday communication and a resource, in particular in the context of effective health care. Carballo and Siem (2006) assert that language represents an added problem for migrants, especially when they have health problems. People may have difficulty finding the words to express their symptoms and be unable to talk comfortably with healthcare providers. This is especially true if migrants come from cultures in which people do not easily discuss personal problems and – in the case of women – are not used to being examined by someone with whom they are unfamiliar. Language difficulties and embarrassment at not being able to communicate effectively can lead to migrants choosing to ignore their problems. This can lead also to impatience on the part of healthcare personnel faced with migrants they cannot understand and who appear not to pay attention to what they are being asked or told. In such situations, health problems are easily overlooked or misdiagnosed. When the health problems of migrants are correctly diagnosed, problems related to cultural background and beliefs may persist. There may be different attitudes to chronic disorders and long-term care which can pose problems of adherence and effective follow-up. Particularly in the case of diabetes, it is important that people with the condition, their family and friends, and the healthcare team share a common understanding of the nature of the condition and its effective management (Carballo and Siem 2012).

Similarly, in the study of Wandel et al. (2007) it was concluded that good command of the Norwegian language and educational achievement had beneficial effects on the change in table/cooking fats after migration and on the choice of foods rich in fat and sugar. On the other hand, those who scored high on the index of integration into the Norwegian society were more likely to consume foods rich in fat, and those who were engaged in income

generating work were more likely to consume foods rich in sugar, when all the other socio-economic and integration factors were accounted for. Some of these changes may have substantial health implications (Wandel et al. 2008).

Likewise, language barrier is one big factor which is believed to have contributed to the high prevalence of T2DM among South Asians in UK. South Asians living in the UK speak different languages and understanding of English is predominantly low in the first-generation immigrants. Many patients do not speak or read English, interpreters are still not widely available in healthcare except by prior arrangement, and cultural and religious beliefs make it difficult for patients to attend clinics at certain times or on certain days, to speak openly to members of the opposite sex, or for women to travel alone to clinic appointments (Hawthorne 2001). Adherence by South Asians to medical and lifestyle advice has often been questioned. It has been suggested that interactions with patients should not be viewed simply as opportunities to reinforce ideas but to combine the experiences of patients and healthcare professionals to ensure good healthcare delivery (Bissell, May, and Noyce 2004). Analysis of in-depth questionnaires completed by Pakistan-born persons in Denmark suggested that health workers would themselves benefit by learning more about positive aspects of culture and religion and the way they impact on the day-to-day care of patients (Fagerli, Lien, and Wandel 2005).

Hawthorne and Tomlinson (1999) conducted a study on 201 patients to examine the influence of sex, educational status and place of care on knowledge and self-management of diabetes, and glycaemic control. The subjects were found to have good knowledge of diabetic diet (average scores 72%), and claimed to perform regular glucose measurements (66%), but they were not good at applying their knowledge to problems in their daily life. Only 24% knew how to manage persistent hyperglycaemia. The study indicated that women were worse than men at this and were less likely to understand why glucose levels should be monitored, and had poorer glycaemic control overall. Fifty-four patients were completely illiterate. They had similar knowledge scores to readers but were less able to handle problem scenarios. Forty-five of these patients were women, and multiple regression analysis showed they were more likely to have the poorest glycaemic control. (Hawthorne and Tomlinson 1999). In another study by the same group it was noted that Pakistani women with diabetes, despite knowing less about the condition initially,

improved their knowledge levels through health education to catch up with the men within six months (Hawthorne 2001).

Similarly, structured interviews were carried out with 14 people invited to a peer educational programme to examine the understanding and beliefs of people with diabetes from the Bangladeshi community living in the UK. The result showed that majority of participants did not know what caused diabetes. Knowledge of the management of diabetes was linked to controlling sugar intake and a number of participants reported eating bitter foods such as bitter melon to control their diabetes. There was little access to information as many participants did not speak English and did not have a Bengali-speaking doctor. The majority of participants felt that education classes should teach them what the doctor thought was important and that these classes would best be advertised by word of mouth. Therefore, participants were quite passive about their own self management and relied very strongly on the doctor's views and recommendations (Choudhury, Brophy, and Williams 2009).

#### 7.4 Physical Activity and Diabetes

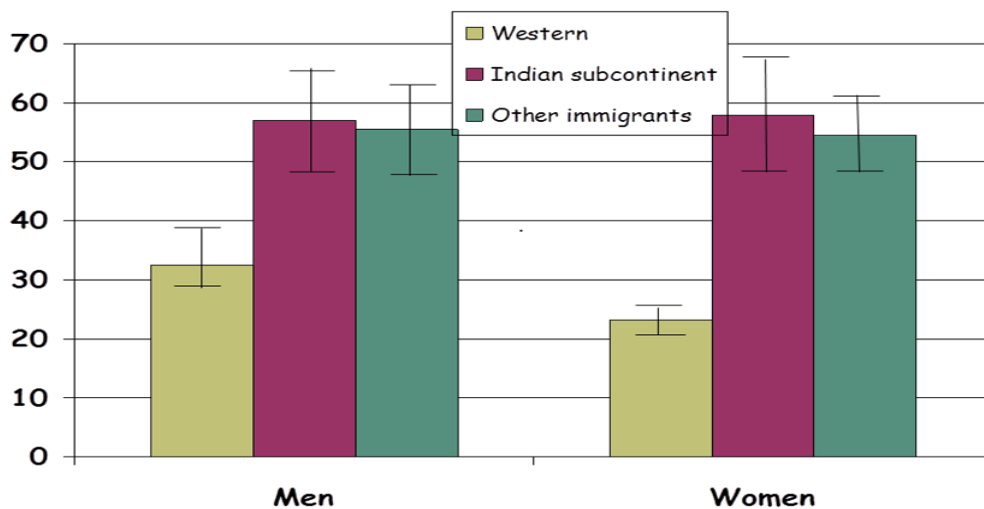
It is a well known fact that lack of physical activity is one of the main causes of T2DM and one of the independent risk factors (WHO 2012a; Dowse et al. 1991). A number of researches have been conducted to investigate the pattern of physical activity among immigrants from the Indian sub continent in different western countries including Norway.

Lauritzen and Holmboe-Ottessen (2005) studied the physical activity pattern of South Asians in Oslo. The study included Norwegian and immigrants from South Asia (Pakistan, India, Bangladesh and Sri Lanka). Participants received a postal invitation with a questionnaire and thereafter attended a physical examination with several measurements and blood sample. The results indicated that South Asians are less physically active than Norwegians. 55% of the men and 50% of the women were doing light exercise less than one hour per week or not at all. Corresponding figure for Norwegians was 17% for men and 15% for women. The study also revealed that the women were less active than the men. The researchers made an index for physical activity from 1-10. 1-2 means inactive, 3-4 some active and so on. The mean score for men was 3.8 and for women 3.6 (Norwegians total mean was 5.8). The study found out that low physical activity was related to high hip-waist-ratio in women, and high blood glucose among those without

known diabetes. The study concluded that physical inactivity may very well be one of the reasons why the South Asians in Oslo have a high prevalence of diabetes (Lauritzen and Holmboe-Ottesen 2005).

Moreover, Kumar et al (2008) pointed out that South Asians in Oslo used their leisure time in a more sedentary way than Norwegians and other ethnic groups in Oslo. According to the study they use their free time for deskbound activities like reading and watching TV as it is indicated in Fig.6 below (Kumar et al. 2008).

### Sedentary\* during leisure time (%)



\* "Yes, mainly sedentary activity (reading, watching TV etc)", 95% CI

Fig.6: Pattern of sedentary activity like reading and watching TV during leisure time by ethnicity in Oslo (Kumar et al. 2008).

Similarly, in UK, research has indicated that on diagnosis of diabetes, British South Asians are less likely to be physically active than any other ethnic groups. A survey by Williams and colleagues using interview reports of exercise levels showed a lower rate of vigorous exercise in British South Asian men compared to indigenous white males but no difference in women (Williams, Bhopal, and Hunt 1994). Dhawan and colleagues, in a case control study of British South Asians and Indian Asians, showed that twice as many of the former took no physical exercise (Dhawan et al. 1994). Researchers looking at levels of physical activity among South Asians noted some awareness of its importance but a lack of putting it into practice (Lawton et al. 2008). The reasons included cultural norms, social expectations, time constraints and health problems.

A study in which researchers interviewed South Asian women about their understanding of the importance of physical activity noted that the respondents emphasized the cultural importance of being active day to day, rather than the 'western' concept of organized exercise (Sriskantharajah and Kai 2007). However, women's principal motivations and attitudes towards physical activity, i.e. losing weight, socializing and maintaining independence, were culturally similar to 'majority' populations. The exercise options are also different. One study showed that overweight Bangladeshi women from East London favored swimming while the least popular activity was running (Khanam and Costarelli 2008). The great majority of the subjects (96 per cent) reported that they were only willing to take up exercise if they were referred to the gym by their GP as an alternative, or additional, treatment for their complaints. They would not exercise voluntarily (Khanam and Costarelli 2008).

### 7.5 Obesity and T2DM

Obesity which is defined as a body weight  $> 120\%$  of the ideal body weight or a body mass index (BMI)  $> 30\text{kg}/\text{m}^2$  is one of the major environmental risk factors for T2DM (WHO 2012). Studies indicate that the tremendous increase in the rates of T2DM can be attributed, primarily, to the dramatic rise in obesity worldwide (Zimmet et al., 2001). In addition to the general obesity, the waist to hip ratio (WHR) which shows the distribution of body fat around the waist is also blamed to have an impact on the risk of T2DM. WHR is a reflection of abdominal (central) obesity, which is more strongly associated with T2DM than the standard measures of obesity, such as those based on body mass index (WHO 2012).

Studies from North America indicate that obesity and T2DM may be more prevalent among non-Western immigrants than among the host population in Western countries. It has been pointed out that migrating from a less to a more affluent country may lead to weight gain, which is often directly related to the length of stay in the new country (Goel et al. 2004; Anand et al. 2000; Himmelgreen et al. 2004).

## Prevalence of abdominal obesity HUBRO + Innvandrere-HUBRO. Age-adjusted (Waist/hip ratio $\geq 0,85$ in women)

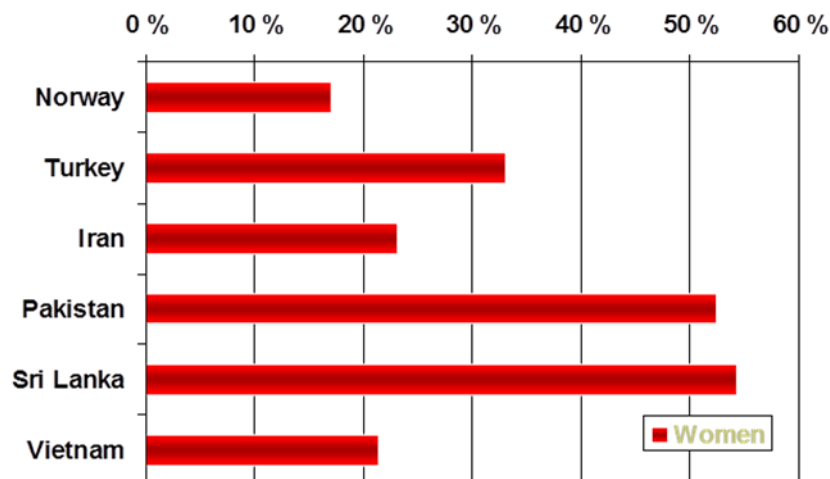


Fig.7: Prevalence of abdominal obesity among women by ethnicity in Oslo. Note the score of Pakistanis and Sri Lankans (Kumar et al. 2008).

The Oslo immigrants' health profile which was the result of the population based health study of the immigrants of Oslo indicated that general obesity was a challenge among South Asians. Moreover, Råberg et al. (2010) concluded in their study that unrecognized overweight exist among South Asians in Norway. As it is shown in figure 7 above, the study revealed that over 50% of the studied South Asian immigrants were obese (BMI  $>30$ ). This was far higher than any of the other ethnic/gender groups. Abdominal obesity waist hip ratio  $> 1$  for men and  $>0.9$  for women was most frequently seen in women from Sri Lanka and Pakistan, which fits with their higher prevalence of diabetes (Kumar 2008, 5).



## 8. DISSCUSSION CONCLUSION AND RECOMMENDATION

### 8.1 Main Findings

The prevalence of T2DM is increasing worldwide and it will double itself if necessary action is not taken timely. The burden of the disease is not evenly distributed among all population groups. South Asians have a high risk of diabetes, particularly when they migrate away from the Indian Subcontinent.

In both Norway, UK and other countries mentioned in this thesis the ethnic inequalities in health in general and inequalities in the prevalence of T2DM in particular are widely documented. The exceptional high prevalence of T2DM among South Asians has been the point of attention for researchers. South Asian immigrants bear a greater load of the problem than the rest of the population.

Such ethnic disparities happen due to a complex web of intermingled factors. Ethnic inequality in the prevalence of T2DM between South Asians and the rest of the population is no exception.

Understanding the possible reasons for this ethnic inequality calls for assessing the factors which are said to affect the disease. Many researchers indicate that such ethnic inequality occurs due to genetic and biological differences, difference in socioeconomic position, cultural and lifestyle differences, migration and difference in access to health care services.

The main task of this thesis has been looking into these factors in order to see if it holds true and explains the high prevalence of T2DM among South Asians and at the same time find out an answer to the research question: “why, in comparison to the rest of population as a whole, is diabetes so common in South Asian immigrants?”

Consequently, attempt is made to look into each one of these factors which are said to affect the prevalence of T2DM. The first factor I looked into was the genetics and biological factor.

Although researches on the genetics of T2DM are still ongoing, researchers agree that genes have an important role in the development of the disease. Theories and hypothesis developed over the past fifty years indicate that there is a biological and genetic factors underlying the high prevalence of T2DM among South Asians. From time to time scientific research is revealing specific genes which are said to be partly responsible for the susceptibility of people from South Asia for T2DM.

Together with the ongoing comprehensive studies of the genetic of T2DM Different hypothesis were developed to explain the high prevalence of T2DM. The Thrifty genotype hypothesis and the thrifty phenotype hypothesis were two of these theories that captured a wide spectrum of attention and critics. In the center of both thrifty phenotype and thrifty genotype hypotheses is a low birth weight due to insufficient nutrition. Studies indicate that low birth weight is associated with predisposition to different metabolic disorders. It has been indicated that South Asians have a lower birth weight than Caucasians which predispose South Asian infants to metabolic disorders including T2DM. Both of these hypotheses suggest further studies need to be conducted to examine whether South Asians are receiving insufficient nutrients in-utero and how this correlates with susceptibility to diabetes.

Like T2DM, both obesity which is a known to be forerunner of T2DM, and diabetes complications seem to be genetically governed. Consequently, there is an indication that South Asians are genetically prone to obesity and diabetes complications. That is, however, just one part of the big story.

There exist broad, complex and intermingled factors underlying the high prevalence of T2DM among South Asian immigrants. Besides genes and biology, a number of factors contribute to the problem. SEP, migration, culture and the lifestyle of South Asians are all play their role for the high prevalence of T2DM among South Asian immigrants. Figure 8 is my graphic presentation of this complex web of intermingled factors underlying the high prevalence of T2DM. The thickness of the arrows which could represent the relative importance of each factor appears to be equal in figure 8; but in reality one factor might be relatively more important than the other. Identifying the relative importance of all these factors definitely calls for a further research.

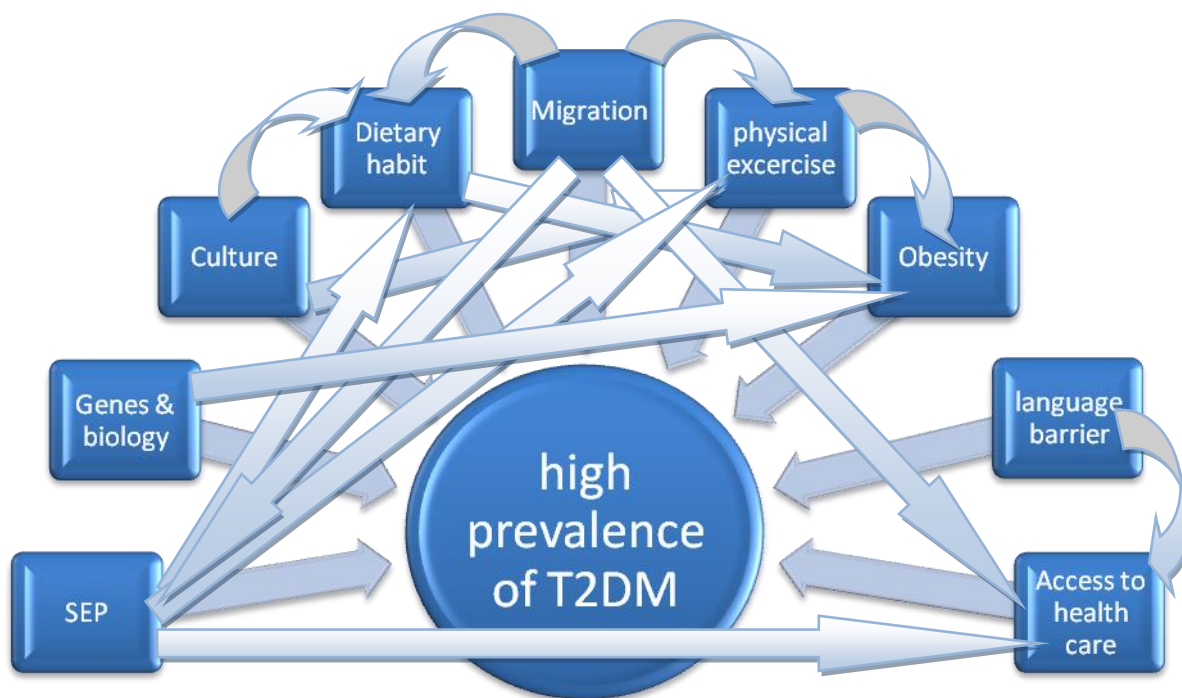


Fig. 8. A model depicting a web of intermingled factors underlying the high prevalence of T2DM among south Asian immigrants.

Although the role of SEP in explaining health inequality is contested, many studies indicate that there is association between high prevalence of T2DM and low SEP. Low educational level, low income and deprived areas are associated with higher prevalence of T2DM among South Asian immigrants.

South Asians are culturally, linguistically and religiously diverse population whose behavior is strongly influenced by their respective cultural values. Regrettably, some aspects of this culture have resulted in an increase in the risk of T2DM in an already biologically vulnerable population. Cultural foods of South Asians, for instance, are parts of the unique cultural identities of the population. The South Asian diet is rich in fats and sugar. It has been indicated both in Norway and UK that the dietary preference of South Asians has contributed to their high prevalence of obesity and T2DM. Migration obviously accelerated the problem because it entailed a significant change in dietary habit of South Asian immigrants mostly on the negative direction.

In addition to that, migration may cause numerous changes in the socio-cultural environment to occur, which in turn may lead to shift in socio-economic status, work status, access to health care and life style, including diet and physical activity, and ultimately health. Genetics and early life conditions affect health outcomes later in life and may interact with the changes occurring after migration (Barker 2001; Forsdahl 1977; Eriksson et al. 2003).

The low level of integration, lack of access and negative attitude towards health care and language barrier have all their contributions to the high level of T2DM prevalence among the South Asian immigrants.

While working with this thesis I came to learn that T2DM is obviously a complex multifactorial disease, not only, as some research report, the result of genetic susceptibilities triggered by an individual's behaviors but also influenced by social, environmental, psychological, and cultural factors.

## 8.2 Policy Recommendations

A better understanding of the mechanisms by which T2DM develops and the factors contributing to the high prevalence of the disease among South Asian immigrants will enable more effective prevention strategies and treatments to be developed, ultimately helping to reduce the incidence and burden of the disease in this high risk group. Knowing the high risk area helps local authorities and health service providers tackle poor health by directing resources and efforts where they are most needed. Moreover, having understood the contributing factors for the high prevalence of T2DM among South Asian immigrants, policy makers need to see:

- proactive policies which look to reduce T2DM rather than policies which simply focus on treatment of T2DM and its complications
- proactive policies which look to reduce obesity rather than policies that simply treat the effects of it
- proactive policies which look to reduce physical inactivity among South Asian immigrants rather than treating the consequences of physical inactivity

In conclusion, one cannot change the genetic makeup of people but one can certainly change other factors which trigger the genes. Improved efforts toward the primary prevention and optimal management of T2DM are necessary to reduce the burden of diabetes and its complications among South Asian immigrants.

### 8.3 Limitations and Further Research

South Asians are individuals whose ethnic roots originate from the Indian subcontinent, a large geographic area that includes India, Pakistan, Sri Lanka, and Bangladesh. Collectively, South Asians represent one fifth of the global population. It is important to recognize that the term “South Asian” refers to a large heterogeneous population, with important differences in diet, culture, and lifestyle among different South Asian populations and religions. Generalizing study results, therefore, are sometimes misleading. This can be mentioned as one limitation.

Future studies of ethnic inequalities in health should be, therefore, large, separating Indian, Sri Lankans, Pakistani and Bangladeshi populations, studying men and women separately, and tracking changes over period of time.

Researches to understand the cultural factors responsible for poor compliance to lifestyle advice and medication is one of the things which needs to be done in order to improve our understanding of the reason for the higher prevalence of T2DM among the South Asian immigrants.

Finally, it has been indicated that low SEP is a risk factor for T2DM; but the extent to which SEP can explain ethnic differences in the prevalence of T2DM between migrants and the host population is still under discussion and calls for further research work.

## BIBLIOGRAPHY

- “2011 Census - Ethnicity.” 2012. <http://www.ons.gov.uk/ons/interactive/census-map-2-1---ethnicity/index.html?mode=clean>.
- Altshuler, David, Joel N. Hirschhorn, Mia Klannemark, Cecilia M. Lindgren, Marie-Claude Vohl, James Nemesh, Charles R. Lane, et al. 2000. “The Common PPAR $\gamma$  Pro12Ala Polymorphism Is Associated with Decreased Risk of Type 2 Diabetes.” *Nature Genetics* 26 (1) (September 1): 76–80. doi:10.1038/79216.
- Anand, S S, S Yusuf, V Vuksan, S Devanesen, K K Teo, P A Montague, L Kelemen, et al. 2000. “Differences in Risk Factors, Atherosclerosis, and Cardiovascular Disease Between Ethnic Groups in Canada: The Study of Health Assessment and Risk in Ethnic Groups (SHARE).” *Lancet* 356 (9226) (July 22): 279–284.
- Avendano, M, A E Kunst, M Huisman, F V Lenthe, M Bopp, E Regidor, M Glickman, et al. 2006. “Socioeconomic Status and Ischaemic Heart Disease Mortality in 10 Western European Populations During the 1990s.” *Heart* 92 (4) (April): 461–467. doi:10.1136/hrt.2005.065532.
- Avendaño, M, A E Kunst, F van Lenthe, V Bos, G Costa, T Valkonen, M Cardano, et al. 2005. “Trends in Socioeconomic Disparities in Stroke Mortality in Six European Countries Between 1981-1985 and 1991-1995.” *American Journal of Epidemiology* 161 (1) (January 1): 52–61. doi:10.1093/aje/kwi011.
- Baier, Leslie J., and Robert L. Hanson. 2004. “Genetic Studies of the Etiology of Type 2 Diabetes in Pima Indians Hunting for Pieces to a Complicated Puzzle.” *Diabetes* 53 (5) (May 1): 1181–1186. doi:10.2337/diabetes.53.5.1181.
- Balarajan, R, L Bulusu, A M Adelstein, and V Shukla. 1984. “Patterns of Mortality Among Migrants to England and Wales from the Indian Subcontinent.” *British Medical Journal (Clinical Research Ed.)* 289 (6453) (November 3): 1185–1187.
- Banerji, M A, N Faridi, R Atluri, R L Chaiken, and H E Lebovitz. 1999. “Body Composition, Visceral Fat, Leptin, and Insulin Resistance in Asian Indian Men.” *The Journal of Clinical Endocrinology and Metabolism* 84 (1) (January): 137–144.
- Barker, D J. 2001. “A New Model for the Origins of Chronic Disease.” *Medicine, Health Care, and Philosophy* 4 (1): 31–35.
- BBC. 2010. “Diabetes a ‘South Asian Time-bomb’.” *BBC*, February 23, sec. West Midlands. [http://news.bbc.co.uk/2/hi/uk\\_news/england/west\\_midlands/8524407.stm](http://news.bbc.co.uk/2/hi/uk_news/england/west_midlands/8524407.stm).
- . 2011. “Diabetes Rate ‘Doubles’ Worldwide.” *BBC*, June 26, sec. Health. <http://www.bbc.co.uk/news/uk-13917263>.
- Bhatnagar, D, I S Anand, P N Durrington, D J Patel, G S Wander, M I Mackness, F Creed, B Tomenson, Y Chandrashekar, and M Winterbotham. 1995. “Coronary Risk Factors in People from the Indian Subcontinent Living in West London and Their Siblings in India.” *Lancet* 345 (8947) (February 18): 405–409.
- Bhopal, R., N. Unwin, M. White, J. Yallop, L. Walker, K G M M Alberti, J. Harland, et al. 1999. “Heterogeneity of Coronary Heart Disease Risk Factors in Indian, Pakistani, Bangladeshi, and European Origin Populations: Cross Sectional Study.” *BMJ* 319 (7204) (July 24): 215–220. doi:10.1136/bmj.319.7204.215.
- Bhopal, Raj S. 2007. *Ethnicity, Race, and Health in Multicultural Societies: Foundations for Better Epidemiology, Public Health, and Health Care: Foundations for Better Epidemiology, Public Health, and Health Care*. Oxford University Press.
- Bissell, Paul, Carl R May, and Peter R Noyce. 2004. “From Compliance to Concordance: Barriers to Accomplishing a Re-framed Model of Health Care Interactions.” *Social Science & Medicine* 58 (4) (February): 851–862. doi:10.1016/S0277-9536(03)00259-4.

- Bond, XiaoWei. 2012. "Asians in Britain". Text. Accessed December 22. <http://www.bl.uk/reshelp/findhelpsubject/history/history/asiansinbritain/asiansinbritain.html>.
- Brown, Arleen F., Susan L. Ettner, John Piette, Morris Weinberger, Edward Gregg, Martin F. Shapiro, Andrew J. Karter, et al. 2004. "Socioeconomic Position and Health Among Persons with Diabetes Mellitus: A Conceptual Framework and Review of the Literature." *Epidemiologic Reviews* 26 (1) (July 1): 63–77. doi:10.1093/epirev/mxh002.
- Burden, Ml, A Samanta, D Spalding, and Ac Burden. 1994. "A Comparison of the Glycaemic and Insulinaemic Effects of an Asian and a European Meal." *Practical Diabetes International* 11 (5): 208–211. doi:10.1002/pdi.1960110508.
- Carballo, Manuel, and Frederik Siem. 2012. "Migration and Diabetes: The Emerging Challenge." Accessed December 17. <http://www.idf.org/diabetesvoice/articles/migration-and-diabetes-the-emerging-challenge>.
- Chambers, John C., Paul Elliott, Delilah Zabaneh, Weihua Zhang, Yun Li, Philippe Froguel, David Balding, James Scott, and Jaspal S. Kooner. 2008. "Common Genetic Variation Near MC4R Is Associated with Waist Circumference and Insulin Resistance." *Nature Genetics* 40 (6): 716–718. doi:10.1038/ng.156.
- Choudhury, S M, S Brophy, and R Williams. 2009. "Understanding and Beliefs of Diabetes in the UK Bangladeshi Population." *Diabetic Medicine: a Journal of the British Diabetic Association* 26 (6) (June): 636–640. doi:10.1111/j.1464-5491.2009.02741.x.
- Connolly, V., N. Unwin, P. Sherriff, R. Bilous, and W. Kelly. 2000. "Diabetes Prevalence and Socioeconomic Status: a Population Based Study Showing Increased Prevalence of Type 2 Diabetes Mellitus in Deprived Areas." *Journal of Epidemiology and Community Health* 54 (3) (March 1): 173–177. doi:10.1136/jech.54.3.173.
- Consortium, the DiAbetes Genetics Replication And Meta-analysis (DIAGRAM). 2012. "Large-scale Association Analysis Provides Insights into the Genetic Architecture and Pathophysiology of Type 2 Diabetes." *Nature Genetics* 44 (9): 981–990. doi:10.1038/ng.2383.
- Dalstra, J. a. A., A. E. Kunst, C. Borrell, E. Breeze, E. Cambois, G. Costa, J. J. M. Geurts, et al. 2005. "Socioeconomic Differences in the Prevalence of Common Chronic Diseases: An Overview of Eight European Countries." *International Journal of Epidemiology* 34 (2) (April 1): 316–326. doi:10.1093/ije/dyh386.
- Daniels, Norman. 2001. *Is Inequality Bad For Our Health?* Beacon Press.
- Dhawan, J., C. L. Bray, R. Warburton, D. S. Ghambhir, and J. Morris. 1994. "Insulin Resistance, High Prevalence of Diabetes, and Cardiovascular Risk in Immigrant Asians. Genetic or Environmental Effect?" *British Heart Journal* 72 (5) (November 1): 413–421. doi:10.1136/hrt.72.5.413.
- Dowse, Gary K., Paul Z. Zimmet, Hassam Gareeboo, K. George M. M. Alberti, Jaakko Tuomilehto, Caroline F. Finch, Pierrot Chitson, and Harish Tulsidas. 1991. "Abdominal Obesity and Physical Inactivity as Risk Factors for NIDDM and Impaired Glucose Tolerance in Indian, Creole, and Chinese Mauritians." *Diabetes Care* 14 (4) (April 1): 271–282. doi:10.2337/diacare.14.4.271.
- Eriksson, Dr J. G., T. Forsén, J. Tuomilehto, C. Osmond, and D. J. P. Barker. 2003. "Early Adiposity Rebound in Childhood and Risk of Type 2 Diabetes in Adult Life." *Diabetologia* 46 (2) (February 1): 190–194. doi:10.1007/s00125-002-1012-5.
- Espelt, A., C. Borrell, A. J. Roskam, M. Rodríguez-Sanz, I. Stirbu, A. Dalmau-Bueno, E. Regidor, et al. 2008. "Socioeconomic Inequalities in Diabetes Mellitus Across Europe

- at the Beginning of the 21st Century.” *Diabetologia* 51 (11) (November 1): 1971–1979. doi:10.1007/s00125-008-1146-1.
- Evans, J. M. M., R. W. Newton, D. A. Ruta, T. M. MacDonald, and A. D. Morris. 2000. “Socio-economic Status, Obesity and Prevalence of Type 1 and Type 2 Diabetes Mellitus.” *Diabetic Medicine* 17 (6): 478–480. doi:10.1046/j.1464-5491.2000.00309.x.
- Evans, Julie C., Timothy M. Frayling, Paul G. Cassell, Philip J. Saker, Graham A. Hitman, Mark Walker, Jonathan C. Levy, et al. 2001. “Studies of Association Between the Gene for Calpain-10 and Type 2 Diabetes Mellitus in the United Kingdom.” *American Journal of Human Genetics* 69 (3) (September): 544–552.
- Fagerli, Rønnaug Aa., Marianne E. Lien, and Margareta Wandel. 2005. “Experience of Dietary Advice Among Pakistani-born Persons with Type 2 Diabetes in Oslo.” *Appetite* 45 (3) (December): 295–304. doi:10.1016/j.appet.2005.07.003.
- FHI. 2012. “Mer Om Type 2 Diabetes - Folkehelseinstituttet.” *Folkehelseinstituttet*. Accessed December 15. [http://www.fhi.no/eway/default.aspx?pid=233&trg=MainLeft\\_5648&MainArea\\_5661=5648:0:15,2917:1:0:0:::0:0&MainLeft\\_5648=5544:28142::1:5647:41:::0:0](http://www.fhi.no/eway/default.aspx?pid=233&trg=MainLeft_5648&MainArea_5661=5648:0:15,2917:1:0:0:::0:0&MainLeft_5648=5544:28142::1:5647:41:::0:0).
- Forsdahl, A. 1977. “Are Poor Living Conditions in Childhood and Adolescence an Important Risk Factor for Arteriosclerotic Heart Disease?” *British Journal of Preventive & Social Medicine* 31 (2) (June 1): 91–95. doi:10.1136/jech.31.2.91.
- Frayling, T. M., and M. I. McCarthy. 2007. “Genetic Studies of Diabetes Following the Advent of the Genome-wide Association Study: Where Do We Go from Here?” *Diabetologia* 50 (11) (November 1): 2229–2233. doi:10.1007/s00125-007-0825-7.
- Frayling, Timothy M., Nicholas J. Timpson, Michael N. Weedon, Eleftheria Zeggini, Rachel M. Freathy, Cecilia M. Lindgren, John R. B. Perry, et al. 2007. “A Common Variant in the FTO Gene Is Associated with Body Mass Index and Predisposes to Childhood and Adult Obesity.” *Science* 316 (5826) (May 11): 889–894. doi:10.1126/science.1141634.
- Gardner, David, and Dolores Shoback. 2011. *Greenspan’s Basic and Clinical Endocrinology, Ninth Edition*. 9th ed. McGraw-Hill Medical.
- Gnavi, Roberto, Ludmila Karaghiosoff, Giuseppe Costa, Franco Merletti, and Graziella Bruno. 2008. “Socio-economic Differences in the Prevalence of Diabetes in Italy: The Population-based Turin Study.” *Nutrition, Metabolism and Cardiovascular Diseases* 18 (10) (December): 678–682. doi:10.1016/j.numecd.2007.11.004.
- Goel, M. S., E. P. McCarthy, R. S. Phillips, and C. C. Wee. 2004. “Obesity Among US Immigrant Subgroups by Duration of Residence.” *JAMA: The Journal of the American Medical Association* 292 (23): 2860–2867.
- Hales, C. Nicholas, and David J. P. Barker. 2001. “The Thrifty Phenotype Hypothesis Type 2 Diabetes.” *British Medical Bulletin* 60 (1) (November 1): 5–20. doi:10.1093/bmb/60.1.5.
- Hall, Lesley M. L., Colin N. Moran, Gillian R. Milne, John Wilson, Niall G. MacFarlane, Nita G. Forouhi, Narayanan Hariharan, Ian P. Salt, Naveed Sattar, and Jason M. R. Gill. 2010. “Fat Oxidation, Fitness and Skeletal Muscle Expression of Oxidative/Lipid Metabolism Genes in South Asians: Implications for Insulin Resistance?” *PLoS ONE* 5 (12): e14197. doi:10.1371/journal.pone.0014197.
- Hawthorne, K. 2001. “Effect of Culturally Appropriate Health Education on Glycaemic Control and Knowledge of Diabetes in British Pakistani Women with Type 2 Diabetes Mellitus.” *Health Education Research* 16 (3) (June 1): 373–381. doi:10.1093/her/16.3.373.



- Hawthorne, K., and S. Tomlinson. 1999. "Pakistani Moslems with Type 2 Diabetes Mellitus: Effect of Sex, Literacy Skills, Known Diabetic Complications and Place of Care on Diabetic Knowledge, Reported Self-monitoring Management and Glycaemic Control." *Diabetic Medicine* 16 (7): 591–597. doi:10.1046/j.1464-5491.1999.00102.x.
- Helman, Cecil. 2007. *Culture, health, and illness*. London; New York, NY: Hodder Arnold ; Distributed in the United States of America by Oxford University Press.
- Himmelgreen, D. A., R. Pérez-Escamilla, D. Martinez, A. Bretnall, B. Eells, Y. Peng, and A. Bermúdez. 2004. "The Longer You Stay, the Bigger You Get: Length of Time and Language Use in the US Are Associated with Obesity in Puerto Rican Women." *American Journal of Physical Anthropology* 125 (1): 90–96.
- House, JS, and DR Williams. 2001. "Understanding and Reducing Socioeconomic and Racial/Ethnic Disparities in Health." In *Promoting Health: Intervention Strategies from Social and Behavioral Research*. National Academies Press.
- International Congress on Obesity, Jules Hirsch, and Theodore B Van Itallie. 1985. "Recent advances in obesity research IV : proceedings of the 4th International Congress on Obesity 5-8 October, 1983 New York, USA." In Libbey.
- Jacobson, MarcS. 1987. "CHOLESTEROL OXIDES IN INDIAN GHEE: POSSIBLE CAUSE OF UNEXPLAINED HIGH RISK OF ATHEROSCLEROSIS IN INDIAN IMMIGRANT POPULATIONS." *The Lancet* 330 (8560) (September 19): 656–658. doi:10.1016/S0140-6736(87)92443-3.
- Jenum, A. K., I. Holme, S. Graff-Iversen, and K. I. Birkeland. 2005. "Ethnicity and Sex Are Strong Determinants of Diabetes in an Urban Western Society: Implications for Prevention." *Diabetologia* 48 (3) (March 1): 435–439. doi:10.1007/s00125-005-1668-8.
- Kassam-Khamis, T., P. A. Judd, and J. E. Thomas. 2000. "Frequency of Consumption and Nutrient Composition of Composite Dishes Commonly Consumed in the UK by South Asian Muslims Originating from Bangladesh, Pakistan and East Africa (Ismailis)." *Journal of Human Nutrition and Dietetics* 13 (3): 185–196. doi:10.1046/j.1365-277x.2000.00230.x.
- Kawachi, I., S. V. Subramanian, and N. Almeida-Filho. 2002. "A Glossary for Health Inequalities." *Journal of Epidemiology and Community Health* 56 (9) (September 1): 647–652. doi:10.1136/jech.56.9.647.
- Khanam, Salma, and Vassiliki Costarelli. 2008. "Attitudes Towards Health and Exercise of Overweight Women." *The Journal of the Royal Society for the Promotion of Health* 128 (1) (January): 26–30.
- Kooner, Jaspal S., Danish Saleheen, Xueling Sim, Joban Sehmi, Weihua Zhang, Philippe Frossard, Latonya F. Been, et al. 2011. "Genome-wide Association Study in Individuals of South Asian Ancestry Identifies Six New Type 2 Diabetes Susceptibility Loci." *Nature Genetics* 43 (10): 984–989. doi:10.1038/ng.921.
- Krieger, N., D. R. Williams, and N. E. Moss. 1997. "Measuring Social Class in US Public Health Research: Concepts, Methodologies, and Guidelines." *Annual Review of Public Health* 18 (1): 341–378. doi:10.1146/annurev.publhealth.18.1.341.
- Kumar, B N, and B. Viken. 2010. *Folkehelse i et migrasjonsperspektiv*. Bergen: Fagbokforl.
- Kumar, BN. 2008. "The Oslo Immigrant Health Profile." <http://158.36.43.132/dokumenter/920ab22ad5.pdf>.
- Kumar, BN, L Grøtvedt, HE Meyer, AJ Sogaard, and BH Strand. 2008. *The Oslo immigrant health profile*. Oslo: Folkehelseinstituttet.
- Larrañaga, I., J. M. Arteagoitia, J. L. Rodriguez, F. Gonzalez, S. Esnaola, J. A. Piniés, and the Sentinel Practice Network of the Basque Country. 2005. "Socio-economic Inequalities in the Prevalence of Type 2 Diabetes, Cardiovascular Risk Factors and Chronic

- Diabetic Complications in the Basque Country, Spain.” *Diabetic Medicine* 22 (8): 1047–1053. doi:10.1111/j.1464-5491.2005.01598.x.
- Lauritzen, Trine, and Holmboe-Ottesen. 2005. “Physical activity and diabetes in the South-Asian population in Oslo” (June 17). <https://www.duo.uio.no/handle/123456789/29777>.
- Lawton, Julia, Naureen Ahmad, Lisa Hanna, Margaret Douglas, Harpreet Bains, and Nina Hallowell. 2008. “‘We Should Change Ourselves, but We Can’t’: Accounts of Food and Eating Practices Amongst British Pakistanis and Indians with Type 2 Diabetes.” *Ethnicity & Health* 13 (4): 305–319. doi:10.1080/13557850701882910.
- Malecki, Maciej T. 2005. “Genetics of Type 2 Diabetes Mellitus.” *Diabetes Research and Clinical Practice* 68 (June): S10–S21. doi:10.1016/j.diabres.2005.03.003.
- Mather, H M, and H Keen. 1985. “The Southall Diabetes Survey: Prevalence of Known Diabetes in Asians and Europeans.” *British Medical Journal (Clinical Research Ed.)* 291 (6502) (October 19): 1081–1084.
- Mather, H. M., N. Chaturvedi, and A.m. Kehely. 1998. “Comparison of Prevalence and Risk Factors for Microalbuminuria in South Asians and Europeans with Type 2 Diabetes Mellitus.” *Diabetic Medicine* 15 (8): 672–677. doi:10.1002/(SICI)1096-9136(199808)15:8<672::AID-DIA648>3.0.CO;2-3.
- McBean, A. Marshall, Shuling Li, David T. Gilbertson, and Allan J. Collins. 2004. “Differences in Diabetes Prevalence, Incidence, and Mortality Among the Elderly of Four Racial/Ethnic Groups: Whites, Blacks, Hispanics, and Asians.” *Diabetes Care* 27 (10) (October 1): 2317–2324. doi:10.2337/diacare.27.10.2317.
- McCance, D R, D J Pettitt, R L Hanson, L T H Jacobsson, W C Knowler, and P H Bennett. 1994. “Birth Weight and Non-insulin Dependent Diabetes: Thrifty Genotype, Thrifty Phenotype, or Surviving Small Baby Genotype?” *BMJ* 308 (6934) (April 9): 942–945. doi:10.1136/bmj.308.6934.942.
- McKeigue, P. M., M. G. Marmot, Y. D. Syndercombe Court, D. E. Cottier, S. Rahman, and R. A. Riemersma. 1988. “Diabetes, Hyperinsulinaemia, and Coronary Risk Factors in Bangladeshis in East London.” *British Heart Journal* 60 (5) (November 1): 390–396. doi:10.1136/hrt.60.5.390.
- McKeigue, P. M., T. Pierpoint, J. E. Ferrie, and M. G. Marmot. 1992. “Relationship of Glucose Intolerance and Hyperinsulinaemia to Body Fat Pattern in South Asians and Europeans.” *Diabetologia* 35 (8) (August 1): 785–791. doi:10.1007/BF00429101.
- McKeigue, P.M., A.M. Adelstein, M.J. Shipley, R.A. Riemersma, M.G. Marmot, S.P. Hunt, S.M. Butler, and P.R. Turner. 1985. “DIET AND RISK FACTORS FOR CORONARY HEART DISEASE IN ASIANS IN NORTHWEST LONDON.” *The Lancet* 326 (8464) (November): 1086–1090. doi:10.1016/S0140-6736(85)90684-1.
- Middelkoop, B. J., S. M. Kesarlal-Sadhoeram, G. N. Ramsaransing, and H. W. Struben. 1999. “Diabetes Mellitus Among South Asian Inhabitants of The Hague: High Prevalence and an Age-specific Socioeconomic Gradient.” *International Journal of Epidemiology* 28 (6) (December 1): 1119–1123. doi:10.1093/ije/28.6.1119.
- Misra, Anoop, and Om P Ganda. 2007. “Migration and Its Impact on Adiposity and Type 2 Diabetes.” *Nutrition (Burbank, Los Angeles County, Calif.)* 23 (9) (September): 696–708. doi:10.1016/j.nut.2007.06.008.
- Misra, Anoop, and Naval K Vikram. 2004. “Insulin Resistance Syndrome (metabolic Syndrome) and Obesity in Asian Indians: Evidence and Implications.” *Nutrition* 20 (5) (May): 482–491. doi:10.1016/j.nut.2004.01.020.
- Murphy, Robert F. 1988. *Cultural and Social Anthropology*. 3rd ed. Pearson.
- Neel, James V. 1962. “Diabetes Mellitus: A ‘Thrifty’ Genotype Rendered Detrimental by ‘Progress’?” *American Journal of Human Genetics* 14 (4) (December): 353.

- NRK. 2012. "Diabetessjokk Blant Asiater - Østlandssendingen - NRK Nyheter." Accessed November 24. <http://www.nrk.no/nyheter/distrikt/ostlandssendingen/1.8257696>.
- Patel, J.V., A. Vyas, J.K. Cruickshank, D. Prabhakaran, E. Hughes, K.S. Reddy, M.I. Mackness, D. Bhatnagar, and P.N. Durrington. 2006. "Impact of Migration on Coronary Heart Disease Risk Factors: Comparison of Gujaratis in Britain and Their Contemporaries in Villages of Origin in India." *Atherosclerosis* 185 (2) (April): 297–306. doi:10.1016/j.atherosclerosis.2005.06.005.
- Popkin, Barry M. 2004. "The Nutrition Transition: An Overview of World Patterns of Change." *Nutrition Reviews* 62: S140–S143. doi:10.1111/j.1753-4887.2004.tb00084.x.
- Prasad, Pushplata, Arun Tiwari, KM Prasanna Kumar, A. C. Ammini, Arvind Gupta, Rajeev Gupta, A. K. Sharma, et al. 2006. "Chronic Renal Insufficiency Among Asian Indians with Type 2 Diabetes: I. Role of RAAS Gene Polymorphisms." *BMC Medical Genetics* 7 (1) (May 3): 42. doi:10.1186/1471-2350-7-42.
- Rabi, Doreen M., Alun L. Edwards, Danielle A. Southern, Lawrence W. Svenson, Peter M. Sargious, Peter Norton, Eric T. Larsen, and William A. Ghali. 2006. "Association of Socio-economic Status with Diabetes Prevalence and Utilization of Diabetes Care Services." *BMC Health Services Research* 6 (1) (October 3): 124. doi:10.1186/1472-6963-6-124.
- Radha, V., and V. Mohan. 2007. "Genetic Predisposition to Type 2 Diabetes Among Asian Indians." *The Indian Journal of Medical Research* 125 (3): 259–74.
- Radha, Venkatesan, Karani S. Vimaleswaran, Hunsur Narayan S. Babu, Nicola Abate, Manisha Chandalia, Pankaj Satija, Scott M. Grundy, et al. 2006. "Role of Genetic Polymorphism Peroxisome Proliferator–Activated Receptor- $\gamma$ 2 Pro12Ala on Ethnic Susceptibility to Diabetes in South-Asian and Caucasian Subjects Evidence for Heterogeneity." *Diabetes Care* 29 (5) (May 1): 1046–1051. doi:10.2337/dc05-1473.
- Raymond, Neil T., Lakshminarayanan Varadhan, Dilini R. Reynold, Kate Bush, Sailesh Sankaranarayanan, Srikanth Bellary, Anthony H. Barnett, Sudhesh Kumar, and J. Paul O'Hare. 2009. "Higher Prevalence of Retinopathy in Diabetic Patients of South Asian Ethnicity Compared With White Europeans in the Community A Cross-sectional Study." *Diabetes Care* 32 (3) (March 1): 410–415. doi:10.2337/dc08-1422.
- Råberg, Marte, Bernadette Kumar, Gerd Holmboe-Ottesen, and Margareta Wandel. 2010. "Overweight and Weight Dissatisfaction Related to Socio-economic Position, Integration and Dietary Indicators Among South Asian Immigrants in Oslo." *Public Health Nutrition* 13 (05): 695–703. doi:10.1017/S1368980009991662.
- Sacerdote, Carlotta, Fulvio Ricceri, Olov Rolandsson, Ileana Baldi, Maria-Dolores Chirlaque, Edith Feskens, Benedetta Bendinelli, et al. 2012. "Lower Educational Level Is a Predictor of Incident Type 2 Diabetes in European Countries: The EPIC-InterAct Study." *International Journal of Epidemiology* 41 (4) (August 1): 1162–1173. doi:10.1093/ije/dys091.
- Sevak, L., P. M. McKeigue, and M. G. Marmot. 1994. "Relationship of Hyperinsulinemia to Dietary Intake in South Asian and European Men." *The American Journal of Clinical Nutrition* 59 (5) (May 1): 1069–1074.
- Sheldon, Trevor A., and Hilda Parker. 1992. "Race and Ethnicity in Health Research." *Journal of Public Health* 14 (2) (June 1): 104–110.
- Simmons, David, and Rhys Williams. 1997. "Dietary Practices Among Europeans and Different South Asian Groups in Coventry." *British Journal of Nutrition* 78 (01): 5–14. doi:10.1079/BJN19970114.
- Solar, Orielle, and Alec Irwin. 2007. "A Conceptual Framework for Action on the Social Determinants of Health". Report Document or other Monograph.

[http://www.who.int/social\\_determinants/resources/csdh\\_framework\\_action\\_05\\_07.pdf](http://www.who.int/social_determinants/resources/csdh_framework_action_05_07.pdf)

- Speakman, J. R. 2008. “Thrifty Genes for Obesity, an Attractive but Flawed Idea, and an Alternative Perspective: The ‘drifty Gene’ Hypothesis.” *International Journal of Obesity* 32 (11) (November 1): 1611–1617. doi:10.1038/ijo.2008.161.
- Speakman, John R. 2007. “A Nonadaptive Scenario Explaining the Genetic Predisposition to Obesity: The ‘Predation Release’ Hypothesis.” *Cell Metabolism* 6 (1) (July 11): 5–12. doi:10.1016/j.cmet.2007.06.004.
- Sproston, K., and J. E. Mindell. 2006. “Health Survey for England 2004. The Health of Minority Ethnic Groups.” [http://www.ic.nhs.uk/webfiles/publications/healthsurvey2004ethnicfull/HealthSurveyforEnglandVol1\\_210406\\_PDF.pdf](http://www.ic.nhs.uk/webfiles/publications/healthsurvey2004ethnicfull/HealthSurveyforEnglandVol1_210406_PDF.pdf).
- Sriskantharajah, Janani, and Joe Kai. 2007. “Promoting Physical Activity Among South Asian Women with Coronary Heart Disease and Diabetes: What Might Help?” *Family Practice* 24 (1) (February 1): 71–76. doi:10.1093/fampra/cml066.
- Statistics Norway. 2012. “Tabell 1 Folkemengde 1. Januar 2011 Og 2012 Og Endringene i 2011, Etter Innvandringskategori Og Landbakgrunn. Absolutte Tall.” Accessed December 15. <http://www.ssb.no/innvbef/tab-2012-04-26-01.html>.
- Syed, Hammad, Odd Dalgard, Akhtar Hussain, Ingvild Dalen, Bjorgulf Claussen, and Nora Ahlberg. 2006. “Inequalities in Health: a Comparative Study Between Ethnic Norwegians and Pakistanis in Oslo, Norway.” *International Journal for Equity in Health* 5 (1) (June 29): 7. doi:10.1186/1475-9276-5-7.
- Tillin, T., N. Forouhi, D. G. Johnston, P. M. McKeigue, N. Chaturvedi, and I. F. Godsland. 2005. “Metabolic Syndrome and Coronary Heart Disease in South Asians, African-Caribbeans and White Europeans: a UK Population-based Cross-sectional Study.” *Diabetologia* 48 (4) (April 1): 649–656. doi:10.1007/s00125-005-1689-3.
- TV2. 2012. “Antall Diabetikere Har Doblet Seg.” *TV 2*. Accessed November 24. <http://www.tv2.no/nyheter/innenriks/helse/antall-diabetikere-har-doblet-seg-3526021.html>.
- Uthra, Satagopan, Rajiv Raman, Bickol N. Mukesh, Samuel A. Rajkumar, Padmaja Kumari R., Pradeep G. Paul, Praveena Lakshmiathy, et al. 2008. “Association of VEGF Gene Polymorphisms with Diabetic Retinopathy in a South Indian Cohort.” *Ophthalmic Genetics* 29 (1) (January): 11–15. doi:10.1080/13816810701663527.
- Wandel, Margareta, Marte Råberg, BN Kumar, and Gerd Holmboe-Ottesen. 2008. “Changes in Food Habits After Migration Among South Asians Settled in Oslo: The Effect of Demographic, Socio-economic and Integration Factors.” *Appetite* 50 (2–3) (March): 376–385. doi:10.1016/j.appet.2007.09.003.
- Watve, Milind G, and Chittaranjan S Yajnik. 2007. “Evolutionary Origins of Insulin Resistance: a Behavioral Switch Hypothesis.” *BMC Evolutionary Biology* 7: 61. doi:10.1186/1471-2148-7-61.
- WHO. 2012a. “WHO | Diabetes.” *WHO*. Accessed November 21. <http://www.who.int/mediacentre/factsheets/fs312/en/>.
- . 2012b. “WHO | Glossary of Terms Used.” *WHO*. Accessed November 27. <http://www.who.int/hia/about/glos/en/index1.html>.
- . 2012c. “WHO | About Diabetes.” *WHO*. Accessed December 12. [http://www.who.int/diabetes/action\\_online/basics/en/index2.html](http://www.who.int/diabetes/action_online/basics/en/index2.html).
- Wild, S, and P McKeigue. 1997. “Cross Sectional Analysis of Mortality by Country of Birth in England and Wales, 1970-92.” *BMJ (Clinical Research Ed.)* 314 (7082) (March 8): 705–710.

- Williams, Rory, Raj Bhopal, and Kate Hunt. 1994. "Coronary Risk in a British Punjabi Population: Comparative Profile of Non-Biochemical Factors." *International Journal of Epidemiology* 23 (1) (February 1): 28–37. doi:10.1093/ije/23.1.28.
- Woodward, Alistair, and Ichiro Kawachi. 2000. "Why Reduce Health Inequalities?" *Journal of Epidemiology and Community Health* 54 (12) (December 1): 923–929. doi:10.1136/jech.54.12.923.
- Yajnik, C. S., C. S. Janipalli, S. Bhaskar, S. R. Kulkarni, R. M. Freathy, S. Prakash, K. R. Mani, et al. 2009. "FTO Gene Variants Are Strongly Associated with Type 2 Diabetes in South Asian Indians." *Diabetologia* 52 (2) (February 1): 247–252. doi:10.1007/s00125-008-1186-6.
- Zeggini, E., and M. I. McCarthy. 2007. "TCF7L2: The Biggest Story in Diabetes Genetics Since HLA?" *Diabetologia* 50 (1) (January 1): 1–4. doi:10.1007/s00125-006-0507-x.