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Ethnicity and Type 2 Diabetes in Pacific Island Adults in New Zealand

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Abstract

The aim of this review was to present ethnic characteristics of type 2 diabetes in Pacific Island adults in New Zealand. International databases including *PubMed* and *Google scholar* were consulted in a search using the terms "Pacific Island populations", "New Zealand", "ethnicity", "obesity", "metabolic syndrome", "type 2diabetes", "food security, and their combinations, as well as the websites of the *World Health Organization, International Diabetes Federation, and the Ministry of Health New Zealand*.

Pacific Island populations originate from the islands in the Pacific Ocean, and they may have similar genetic and cultural origins to one another. Also, they have some of the highest reported levels of obesity in the world, and New Zealand has rates of obesity that are comparable to most rich nations including USA. In New Zealand, the incidence of type 2diabetes and cardiovascular disease is significantly higher among Pacific Island populations than in Europeans. Pacific Island populations still have comparatively higher rates of obesity (68%) and diagnosed diabetes (13%) than any other ethnic group in New Zealand. Universal anthropometric measures of obesity, which were derived from studies in Europeans, might underestimate the body size and body composition in Pacific Island populations. Hence, there is a need for ethnic specific cut-offs for anthropometric measures of obesity, and better assessment of body composition in these under high risk populations for type 2 diabetes and cardiovascular disease. Considering the high levels of obesity, Pacific Island populations represent an example of the classical thrifty genotype. Type 2 diabetes in Pacific Island populations is based upon their high prevalence of morbid obesity or some other distinct and unknown mechanism. Further research is warranted to explore and clarify ethnic specific pathways, other than obesity, which led to type 2 diabetes in Pacific Island populations in New Zealand.

Keywords

Pacific island population, New Zealand, ethnicity, Obesity, Metabolic syndrome, Type 2 diabetes.

Introduction

The definition of ethnicity by Smith (1986), was accepted by the current New Zealand Official Statistics Ethnicity Standard which defines ethnicity as the ethnic group or groups that people identify with or feel they belong to [1]. Ethnicity is self-perceived in New Zealand, and people in censuses can choose up to six ethnic groups, although in practice most people affiliate with one or two.

In the 2001 census there were 72.8 % NZ European, 14.7 Maori, 2.5 % New Zealanders, 6.5 % of Pacific Island peoples, and 6.6 % of the population were of Asian ethnicity [2]. In the 2006 census changes occurred due to external migration of Asian peoples. The percentage of NZ Europeans was 61.7%, 11.1 % New Zealanders, 14.6% Maori, 6.9 % Pacific Island peoples, and 9.2% Asian population. It is estimated that by the year 2016 the population will be 73 % Europeans, 16 % Maori, and 8 % Pacific Island populations living in New Zealand. Also, Pacific Island populations are projected to increase from 7.2% of the total population in 2006 to 9.6% of the total population in 2026 [3].

Obesity, and in particular central obesity is a major risk factor for many chronic, debilitating and life-threatening diseases such as diabetes and cardiovascular disease (CVD) [4]. Based on a larger number of studies, the world prevalence of diabetes among adults (aged 20 – 79 years) will increase to 7.7% or 439 million adults by 2030 [5]. It is predicted that between 2010 and 2030, there will be a 69% increase in number of adults with diabetes in developing countries, and a 20% increase in developed countries. This indicates a growing burden of diabetes with the largest increase in developing countries.

In New Zealand over the past two decades the prevalence in diabetes has more than doubled, being more common in Maori, Pacific Island and South Asian populations compared with other New Zealanders. In New Zealand, the incidence of type 2diabetes and cardiovascular disease is significantly higher among Pacific Island populations than in Europeans [6].

The aim of this review was to present ethnic characteristics in type 2 diabetes in Pacific Island populations in New Zealand.

Method

International databases including *PubMed* and *Google scholar* were consulted in a search using the terms "Pacific Island populations", "New Zealand", "ethnicity", "obesity", "metabolic syndrome", "type 2diabetes", "food security:, and their combinations. The websites of the *World Health Organization,* International *Diabetes Federation, and the Ministry of Health New Zealand* were also consulted. The

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searches provided over 100 publications and 71 publications were selected and assessed to ensure their relevance to the main topic of the review.

Origins of Obesity in Pacific Populations

It has been observed that Polynesian population compared to Europeans have some anatomical differences in the hip region and lower limbs [7]. These include bigger leg bones, with a tendency to bowing particularly marked bowing of the femoral bone, to accommodate strong, massive, and bulky muscles on the back of the thigh. The impression is that stronger legs (bones enveloped in big and strong muscles), bigger bones and large hip seem to support weak and large waist. Such large bodies, well-muscled, are suited to life in a thermolabile oceanic environment. This is the so-called Polynesian phenotype which is linked to the metabolic disorders of gout and type 2 diabetes mellitus [8]. The Polynesian phenotype originates from the islands in the Pacific Ocean (Samoa, the Cook Islands, Tonga, Niue, Tokelau, Papua New Guinea, Vanuatu, Kiribati, Fiji, Solomon Islands, Nauru, and French Polynesia). Therefore, Pacific Island populations may have similar genetic and cultural origins to one another. They were subjects to strong selection for a large and muscular body, when humans' colonisation occurred in that region.

Obesity has been long regarded as a symbol of high social status and prosperity in Pacific Island populations [9]. Change in the lifestyle from traditional to a more modernized way of life increased the prevalence of obesity and type 2 diabetes in Pacific Island populations. Studies in Kiribati (Micronesians) and Vanuatu (Melanesians) have shown that rural people derive a greater proportion of energy from carbohydrates and less from fat and protein than urban people, while outer atolls of Kiribati relied heavily on coconuts as a source of energy which provide more fat than in the urban diet [10]. Moreover, the results of study in Nauruans showed total energy intakes of 115% to 135% greater than recommended for maintenance of a healthy weight, with sugar intakes about twice the recommended levels and lower fibre intakes of 30% than recommended levels [11,12]. Unhealthy diet, very high alcohol intake in men, and sedentary lifestyle led to a positive energy balance which resulted in obesity. In Pacific Island populations' obesity was seen at an early age. For example, the prevalence of obesity found in Western Samoa (BMI ≥ 30kg/m^2) in 25 to 34-year olds was 26% in men and 47% in women. Such high levels of obesity in Pacific Island populations could be explained by a "thrifty gene" that helped survival in the past and results in conservation of energy, and became detrimental in conditions of plentiful of food which supported fat deposition that led to obesity and insulin resistance [13]. The "thrifty genotype" hypothesis also explains well current increase in prevalence of obesity in New Zealanders of Polynesian descent, particularly when considered in the context of changes in diet, physical activity, and other unidentified factors in a new affluent culture and country.

Visceral Obesity

It has been widely reported that it is not just the amount of fat, but the distribution of fat which determines the disease risk associated with obesity [14,15]. Central or visceral obesity is represented as an unfavourable distribution of body fat owing to its strong association with insulin resistance, metabolic syndrome, and type 2 diabetes and cardiovascular disease. Visceral fat plays a significant role in pathogenesis of insulin resistance and metabolic syndrome; metabolic syndrome is not a diagnosis; it is rather a pre-morbid condition and can be reversed at an early stage [16]. The currently accepted definition from the International Diabetes Federation is the one which includes central obesity plus any two of four additional factors: raised triglycerides level: ≥1.7mmol/L, reduced HDL-cholesterol: <1.03mmol/L in males and <1.29mmol/L in females, raised blood pressure (systolic BP ≥130 or diastolic BP ≥85mmHg, and raised fasting plasma glucose (FPG) ≥ 5.6mmol/L [17].

Visceral fat is considered to be more metabolically active than subcutaneous fat, causing dysmetabolism of fatty acids and increased influx of free fatty acids into the splanchnic circulation [18,19]. Besides storing fat, adipose tissue releases molecules commonly referred to as adipokines [20]. In obesity, adipokines form an important part of an "adipo-insular" axis, dysregulaton which may support β- cell failure and development of type 2 diabetes[21].

Association of Obesity and Insulin Resistance

Insulin resistance is strongly associated with obesity which is a significant factor that contributes in the emergence of metabolic disease like type 2 diabetes [15]. Association of insulin resistance with obesity is related to the covalent modification of the IRS-1(Insulin receptor substrate -1) proteins by serine phosphorylation. Circulating plasma levels of free fatty acids, ceramides and glucose, promote serine phosphorylation of IRS-1present in the muscle tissue. Furthermore, plasma circulating cytokines like tumor necrosis factor alpha (TNF-α) also promotes serine/threonine phosphorylation of IRS-1, and inhibits insulin signalling. Fat cells in obese people are large, which make them resistant to the ability of insulin to inhibit lipolysis in visceral and deep subcutaneous fat tissues. Inability of insulin to suppress lipolysis, results in elevated levels of plasma circulating NEFA and alcohol glycerol. This in turn aggravates insulin resistance in the liver and skeletal muscle tissue.

The Prevalence of Obesity and Type 2 Diabetes s in Pacific Island Populations in NZ

Type 2 diabetes occurs earlier in Pacific Island populations, about 10 years before Europeans [22]. Between 2002 and 2004 an annual average incidence rate for type 2diabetes was 370 for Pacific population, 218 for Maori, and 79 for other. The 2006/07 NZ Health Survey recorded the prevalence of type 2 diabetes as European 4.3 %, Maori 5.8 %, Pacific Island peoples 10.1 %, and Asian Indians 6.5%. For Pacific Island adults obesity rates have not changed since 2006/07 [23]. It is estimated that by 2020, 18% of Pacific Island populations will be diagnosed with type 2 diabetes as opposed to 4% of New Zealanders of European origin. It has been reported that during the New Zealand Health Survey, which became a continuous survey in 2011, one in eight Pacific Island adults (13%) reported being diagnosed with type 2diabetes, meaning that the rate of type 2 diabetes in Pacific Island adults was 3.6 times that of non-Pacific adults [24]. Obesity and type 2diabetes are major challenges for Pacific Island adults, and they still have comparatively higher rates of obesity (68%) and diagnosed diabetes (13%) than any other ethnic group in New Zealand. It has also been notified that Pacific Island populations have some of the highest reported levels of obesity in the world, and Australia and New Zealand have rates of obesity that are comparable to most rich nations including USA [25-27].

Ethnic Characteristics of Type 2 Diabetes in Pacific Island Populations

Using the current BMI thresholds for diabetes screening, which have been derived from data on populations of European descent, may lead to inadequate screening in high-risk ethnic group of Pacific Island populations [28]. Several studies have found that Pacific Island adults tend to be leaner (i.e. have a lower %BF, and higher fat-free mass) than New Zealand Europeans of the same body size [29,30]. Also, a BMI of 27kg/m^2 may well be obese for Asian Indian, overweight for European, and adequate for a large muscular frame of a Pacific Islander [31].

It has been shown that the patterns of body fat distribution differ by ethnicity, as well as the amount of fat-free mass vary between Pacific Island populations and other ethnic groups [32]. For example, a wide disparity was seen in the %BF and BMI relationship between Pacific Island and Asian Indian populations in New Zealand. At a fixed percentage of %BF corresponding to a BMI of 30kg/m^2 for Europeans (29% for men, 43% for women), BMI values for Pacific Island peoples were up to 5 units higher and in Asian Indian people up to 6 units lower, a span of 11 BMI units. Pacific Island and Asian Indian men had 25% and 37% of body fat respectively for the BMI

of 30kg/m^2, while in women of both ethnicities, fatness levels were higher than in men, 38% and 48% respectively. Pacific Island women, when compared with women of other ethnicities (Europeans, Maori, South Africans, and Asian Indians), had the lowest %BF, the highest levels of fat free mass (FFM) and appendicular skeletal muscle mass (ASMM) [33]. Ethnic specific muscularity and ethnic specific fat distribution in Pacific Island and Asian populations contribute to the ethnic differences in the relationship between %BF and BMI. Using the universal BMI cut-offs, derived from data on Europeans, the level of fatness in Pacific Island populations might be underestimated owing to differences in body size and body composition.

It has been suggested that at any given body size Pacific Island populations are significantly leaner than Europeans, and that ethnic Pacific Island populations' standards for defining obesity should be developed [34]. These recommendations were adopted for the 1997 National Nutritional Survey, and these adopted BMI ethnic specific categories were higher than for New Zealand Europeans and others. The adopted BMI threshold for being overweight in New Zealand Pacific Island populations was ≥26 kg/m^2, and for obesity ≥32 kg/m^2. Ethnic specific BMI cut-offs for Pacific Island populations were used in the research studies from 1997 onwards. There is insufficient evidence to recommend ethnic specific cut-offs for anthropometric measures of central obesity, WC and WHR in Pacific Island populations [35].

Based on ethnic-specific BMI cut-off (>32kg/m^2), 45% of men and 66% of women were obese in the large study sample of 1175 (467 men, 708 women) Pacific Island adults with the mean age of 42 years for men and 41 years for women [36,37]. The age standardised mean BMI for Pacific Island men was 32.6kg/m^2, while women had a higher age standardised mean BMI of 34.8kg/m^2 than men, or higher prevalence of obesity than men. About 12% of study participants reported having type 2 diabetes. Decrease in BMI and WC in the 60 plus age group was notified, as well as increases in blood lipids levels, except for the HDL, and blood glucose. Both systolic and diastolic blood pressures increased, although it was expected that owing to decrease in BMI and WC, blood pressures should be lower. In younger participants in the study aged 25 -44, estimates of obesity were higher and overweight lower (44.8% vs. 27.4%), with a higher obesity in females (61.5% vs. 34%) when compared with the figures from the National Nutritional Survey in 97'.Interestingly, serum cholesterol levels and HDL cholesterol ratio, triglycerides, and systolic and diastolic pressures were lower in obese younger men than in older, and correlated well with BMI. In women the same risk factors correlated well with WC, which indicated that Pacific island women were more centrally obese than Pacific Island men. These relationships between BMI and WC, and metabolic risk factors associated with obesity put the younger Pacific Island peoples aged 25-44 years at increased risk for type 2 diabetes and CVD. Sadly, the levels of obesity in Pacific Island populations are increasing gradually and steadily and might be explained by ethnicity and unhealthy diet. Despite the fact that Pacific Island populations tend to have a higher proportion of lean mass to fat mass than New Zealand Europeans at given BMI, as an ethnic group they maintain a greater absolute fat mass [38]. However, a given degree of over-fatness might have worse implications for Pacific populations owing to their greater predisposition to type 2 diabetes and CVD compared with Europeans. It has been reported that Pacific Island populations represent an example of the classical thrifty genotype. Compared with the extent of hyperinsulinaemia in South Asian populations [39] and the comparable degree of insulinaemia after adjustment for obesity, Pacific Island populations, as a group, are neither hyperinsulinemic nor insulin resistant using HOMA [40,41]. Type 2 diabetes is probably based upon their high prevalence of obesity or some other distinct mechanism.

Type 2 diabetes markers such as fasting insulin, insulin sensitivity, pancreatic β-cell function, and glucoregulation also differ by ethnicity even after controlling for age and adiposity[42-44]. For example, after exposure to intensive physical activities for 16 weeks, two groups (with resistant and aerobic training) of morbidly obese Pacific Island peoples with type 2 diabetes were expected to improve glycosylated haemoglobin (HbA1c), blood lipids, adiponectin, C-reactive protein (CRP) as well as anthropometric and hemodynamic indices of health [45]. An extreme level of obesity was characterised with the BMI at baseline of 43.9 ± 9.5kg/m^2. Aerobic training reduced systolic and diastolic blood pressures and increased triglycerides, LDL and total cholesterol while the level of HDL remained the same. Plasma CRP concentrations in Pacific study participants remained within the optimal range. Interestingly, plasma CRP levels are elevated in obesity and in insulin resistant state, possibly secondary to adipocyte release of interleukin-6 and other cytokines of inflammation [46]. On the contrary, another ethnic group of Asian Indians in Auckland, New Zealand had their markers of inflammation at very high levels and at lower BMI [47]. Further studies are required to explore markers and chemicals of inflammation in obese Pacific Island populations with type 2 diabetes. In these two groups of Pacific Island populations, serum levels of adiponectin remained the same (5.6 to 6.7μg/mL) after a course of vigorous exercise, although they were at the lower end of the desired range (5-25μg/mL), which correlated well with obesity. Adiponectin is abundantly present in the plasma of healthy people in the range from 5-25μg/mL, or 1.9 to 17.0mg/ml [48,49]. The mechanism by which plasma concentrations of adiponectin are reduced in centrally obese people is still not completely understood. It has been reported that adipokine tumour necrosis factor alpha (TNF-α) is a strong inhibitor of adiponectin promoter activity. Plasma concentrations of adiponectin are also decreased in people with hypertension and coronary heart disease, irrespective of insulin resistance [50,51]. It has been suggested that hypoadiponectinemia should be included as a key factor in the metabolic syndrome.

Another adipokine leptin (produced and released by adipocytes), is involved in regulation of appetite, energy expenditure, and modulation of insulin sensitivity [52]. Another role of leptin is to inhibit synthesis and release of neuropeptide Y, which increases serum levels of insulin and corticosteroids in the blood plasma. There was a link between leptin serum concentrations and levels of obesity in people of Western Samoa in the South Pacific; serum concentrations of adipokine leptinin Western Samoan people were proportional to mass of adipose tissue. There was also a link between serum leptin concentrations and total energy expenditure (TEE), resting metabolic rate (RMR) and a level of obesity in obese European and Pacific Island women in New Zealand [53]. Body weight and fat free mass (FFM) were strong predictors of TEE in both ethnic groups, while body composition variables related to % BF and overall levels of fatness, did not correlate well with TEE and RMR in NZ Pacific Island group of obese women. This finding might explain an evolutionary adaptation in NZ Pacific Island women toward metabolic efficiency in fat deposition.

It has been notified that Pacific Island obese women and women with gestational diabetes mellitus in New Zealand have large babies, which indicates a presence of hyperleptinemia at birth [54]. At delivery, umbilical cord samples were collected and analysed for cord leptin. It has been suggested that presence of hyperleptinemia at birth might be due to maternal hyperglycemia and relative hyperinsulinaemia, related to the supply of other fuels during foetal development consistent with fuel mediated teratogenesis [55]. There is sufficient evidence for an endocrine feedback system between fat tissue and pancreatic β cells via the actions of insulin and leptin respectively [56]. Insulin has adipogenic effect and increases production of leptin by fat tissue, while leptin directly inhibits leptin receptors on β cells to decrease insulin production[57]. Increase in fat stores is followed by increase in serum leptin concentrations which reduces insulin serum levels. Conversely, reduction in fat stores leads to increase in insulin production and deposition of additional fat. Increased insulin secretion in mothers with gestational diabetes mellitus is a consequence of hyperglycemia *in utero,* which increases foetal adiposity. In conditions of foetal over nutrition, chronic hyperleptinemia causes inhibition of leptin receptor system making β cells desensitized [58]. Hence, overnutrition -induced leptin resistance during foetal development triggers positive feedback mechanism that leads to hyperinsulinaemia that promotes

further adipogenesis, hyperleptinemia, and insulin resistance with compensatory hyperinsulinism, and adipogenic diabetes during postnatal life.

Type 2 diabetes in Pacific Island populations is also associated with vitamin D deficiency. During the 2008/09 New Zealand Adult Nutrition Survey, vitamin D deficiency was defined as serum values less than 25nmol/L. The annual mean level of serum vitamin D was 57.0nmol/L for obese people, 64.1nmol/L for people who were overweight and, 66.3nmol/L for people who were normal weight or underweight. Pacific Island populations had significantly lower mean levels of vitamin D than the rest of the population in New Zealand, which were 49.6nmol/L and 46.0nmol/L for men and women respectively [59]. Overweight and obesity have been linked to lower serum 25-OHD concentrations as well as to impaired insulin action, glucose metabolism and various other metabolic processes in adipose and lean (muscle) tissue [60]. The relationship between vitamin D3 deficiency and metabolic syndrome is still unclear. There is sufficient evidence that vitamin D plays significant role in glucose homeostasis through its direct action on β cell function [61]. It has also been shown that this relationship only exists in obese individuals. Further evidence suggests that this is more likely due to a combination of factors such as sequestration of vitamin D into fat and lower level of physical activity [62]. However, Pacific Island populations have higher bone mineral density than New Zealand Europeans, and lower fracture rates. Hence, vitamin D deficiency in Pacific Island populations might be explained by ethnicity and by their large body size.

The Impact of Socioeconomic Factors on Health of Pacific Island Populations

The food choice in New Zealand pacific Island population is influenced by affordability, and personal, family, and cultural preferences[63]. Healthier food options are often more expensive than those with high sugar and fat concentrations which are nutritionally more limited. Eating a healthy diet and being physically active might help the Pacific Island populations to maintain a healthy body size. The proportion of Pacific adults who eat at least two servings of fruit every day (46%) is similar to the national level (59%) [64]. However, Pacific adults were less likely to eat three servings of vegetables every day (46%) than the general population (54%), and tend to be less physically active (46%) than the general population (54%) in New Zealand.

Food security is strongly related to current disposable income, and affordability of food has become very important issue for Pacific families [65-68]. Buying cheaper foods due to financial constrains is one aspect of food insecurity in addition to running out of money, skipping meals and experiencing hunger. Often, in order to purchase food, low income families have been found to rely on welfare payments, food vouchers and food banks. Food insecurity in Pacific population is associated with unfavourable food choices which might predispose them to obesity and associated comorbidities. Pacific people tend to have large households, owing to their cultural obligations to extended family that creates extra demands on their income [69]. Income is an important socioeconomic factor that influences the relationship between ethnicity and food security. In 2006, the median annual income of all Pacific Island populations (aged over 15 and over) was $20, 500, compared to $24, 400 for the total population in New Zealand [70]. During the 2013 census the gap between national median personal income and median personal income for Pacific populations increased. It was about $8,800 less than the $28,500 national median income in 2013. About 42% of Pacific populations live in the 10% most deprived areas of the country, with relatively poorer housing and overcrowding, that suggests that the incidence of diabetes is higher for people living in the most deprived areas, compared with people living in the least deprived areas.

Changes in diet and lifestyle were seen to be major factors in the increasing incidence of chronic non-communicable diseases such as diabetes, cardiovascular, and respiratory diseases as the main ones [71]. Pacific Island populations were attracted to New Zealand by the prospect of employment, and were welcomed as a solution to workforce shortages in unskilled and semi-skilled occupations. Unfortunately it did not work to their advantage. Socioeconomic inequalities, low education and unemployment, or stress at work were likely to cause depression and anxiety, and frequently challenged the hypothalamic pituitary axis (HPA) [72,73]. Chronic stress influenced development of visceral obesity through repeated activation of the HPA, resulting in hyper-secretion of cortisol which promotes central fat deposition, where the vicious cycle of type 2 diabetes begins [74,75]. Unhealthy diet and sedentary lifestyle lead to type 2 diabetes through multiple mechanisms besides those resulting from overweight and obesity. The distinct or ethnic specific characteristics of type 2 diabetes in the Pacific Island populations require more research studies to fully explain its complex nature.

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