SANAMED
2014; 9(3): 253–263
ISSN-1452-662X

UDK: 616.379-008.64-054.7(931)
ID: 212448524
Revijalni rad

ETHNICITY AND TYPE 2 DIABETES IN ASIAN INDIAN MIGRANTS IN AUCKLAND, NEW ZEALAND

Jowitt Ljiljana

University of Auckland, Department of Surgery, Auckland, New Zealand

Primljen/Received 11. 07. 2014. god.

Prihvaæen/Accepted 15. 10. 2014. god.

Abstract: *T*he aim of this review was to present ethnic differences in body size and body composition in Asian Indian migrants in New Zealand, associated with metabolic syndrome and type 2 diabetes, through the comparison with other ethnic groups in New Zealand. International databases including *PubMed* and *Google scholar* were consulted, as well as the websites of the *World Health Organization* and *International Diabetes Federation*. About 74 studies out of 128 publications were selected to ensure relevance to the topic of the review. Seven research projects were presented for the body size and body composition of Asian Indian migrants in New Zealand. The prevalence of type 2 diabetes of 8.6% in Asian Indians in New Zealand is still higher than in their homeland, owing to their ethnicity, genetic predisposition, sedentary lifestyle and altered nutrition, and other psychosocial factors related to migration and living conditions like stress at work and depression. For the same body mass index, in comparison with people of other ethnic groups in New Zealand Asian Indians had more total body fat, higher percent body fat, more central fat, less lean mass and appendicular skeletal muscle mass. Central obesity was associated with insulin resistance and low grade systemic inflammation. Considering the evidence that type 2 diabetes develops ten years earlier in Asian Indians than in other populations, further studies are warranted to shed some light on the still incompletely understood metabolic syndrome and "thin-fat" Indian phenotype.

Key words: ethnicity, Asian Indian migrants, body composition, insulin resistance, metabolic syndrome.

INTRODUCTION

Asia has become the major epicenter of a diabetes epidemic, and accounts for 60% of the world"s diabetic population (1). The number of people with diabetes in the region of South Asia is estimated to increase to 120.9 million by 2030 (2). Estimates based on population growth, ageing, and rate of urbanization in Asia show that by the year 2030 India as the largest country in the region will have about 79.4 million people with diabetes. Studies from India showed higher prevalence rates in urban areas compared to rural settings, a finding that has been partly attributed to the adoption of a „Western" lifestyle as in the case of migrant South Asians. Rapid economic development, urbanization, and changes in nutritional status, have led to an explosive increase in prevalence of type 2 diabetes. South Asians who migrated to westernized countries have about four times higher prevalence of diabetes than those living in urban India (3). Factors which contributed to increase in higher prevalence of type 2 diabetes in Asian Indian migrants include ethnicity, genetic predisposition, change in diet and lifestyle, and other psychosocial factors mostly depression and stress at work place related to migration and living conditions (4, 5). From a diet rich in pulses and cereals, which contain large amounts of fruits and vegetables, Asian Indian migrants have changed to a diet high in sugars and saturated and total fats, low in fruits, vegetables and fibre (6).

The prevalence of type 2 diabetes in Indian diaspora was the highest in the United Kingdom (11–33%) followed by Norway (14–28%), United States (18%), Singapore (12.8%) and Canada (10%), whereas the prevalence in native South Asians of Pakistan, India and Bangladesh is 7.6, 7.1, and 6.1% respectively (7). The prevalence of type 2 diabetes of 8.6% in Asian Indians in New Zealand is still higher than in their country of origin.

The number of Asian Indians who migrated to New Zealand, has grown from the 2001 census to 2006 census, from 61, 803 to 104,583 respectively making them, after the Chinese ethnic group, the second largest Asian ethnic group in New Zealand. Between the two censuses, the population in New Zealand has grown by

6.4%, with a 40% increase in the Asian populations (8). About 26% of the New Zealand Asian population comprises Asian Indians. In 2013 the Asian Indian ethnic group was the second largest Asian ethnic group, with 155,178 people (32.9 percent of the Asian ethnic group, up from 29.5 percent in 2006) (9).

Comprehensive epidemiological data about the prevalence of type 2 diabetes were obtained in South Auckland between 1991 and 1995, where a household survey of 100,000 residents was undertaken, recorded a highest prevalence of diabetes of 8.6% in Asian Indians in the 40–49 age groups (10). Between the two New Zealand Health Surveys (NZHSs), in the 2002–03 and 2006–07, a significant rise in obesity from 44% to 55% respectively was recorded in Asian Indians, as well as the highest prevalence of diabetes and coronary heart disease (CHD) (11). A recent HbA1c (glycated haemoglobin (A1c)) screening in South Auckland for undiagnosed diabetes of 50,819 volunteers aged 20+ recorded particularly high rates of HbA1c in Asian Indians (12).

In spite of their population growth, Asian ethnic groups have been largely neglected by New Zealand health and research policies (13), although the research on health issues in Asian Indian migrants has been developed in the recent years.

The aim of this review was to present ethnic differences in body size and body composition in Asian Indian migrants in New Zealand associated with metabolic syndrome and type 2 diabetes, through the comparison with other ethnic groups in New Zealand Presented studies were part of the current health research carried out in Asian Indian migrants at AUT University and University of Auckland, New Zealand. In this review, the name Asian Indians is used to define the ethnic group of South Asian Indians, who migrated to New Zealand from Indian subcontinent, Sri-Lanka, Pakistan, and Bangladesh.

International databases including *PubMed* and *Google scholar* were consulted in a search using the terms “South Asians”, “Asian Indians”, “Asian Indian migrants”, “central obesity”, “metabolic syndrome”, “insulin resistance”, and “body composition”, “obesity”, and their combinations. The websites of the *World Health Organization* and *International Diabetes Federation* were also consulted. The searches provided 128 publications from which 74 were selected and assessed to ensure their relevance to the main topic of the review. Seven research projects, out of 74, were separately presented in terms of body composition and body size relevant to “thin-fat” phenotype of Asian Indian migrants in New Zealand. Manual search was employed to allocate these studies from the database of the AUT University, published by the researchers at the Faculty of Health and Environmental Sciences.

Participants in these studies were clinically healthy, although the most of the participants were overweight, and had no knowledge of presence of the metabolic syndrome. Recruitment of participants for these studies was from the urban Auckland areas, by personal contact, advertisement or through existing networks of recruiters.

Pathogenesis of type 2 diabetes

The healthy pancreatic β cell is capable of adapting to changes in insulin action (14). Any decrease in adaptation of the â pancreatic cells to insulin levels, leads to impaired glucose tolerance (IGT), and impaired fasting glucose (IFG) or type 2 diabetes. Impaired pancreatic insulin secretion has been reported in all type 2 diabetic patients in all ethnic groups. Decreased insulin secretion and IFG cause acceleration of endogenous glucose production or hyperglycemia. Increased endogenous glucose production and hepatic insulin resistance represent the driving force for hyperglycemia in type 2 diabetes. The process of conversion from an insulin resistant state to type 2 diabetes is dependent on a relative deficit in â cell insulin secretion capacity. However, type 2 diabetes cannot develop as long as β cell secretory capacity matches the degree of insulin resistance (15).

The role of adipokines in metabolic syndrome and type 2 diabetes

Besides storing fat for excess energy, adipose tissue is an endocrine organ which produces and releases molecules commonly referred to as adipokines (16). Most adipokines in obesity form an important part of an “adipo-insular” axis, dysregulaton which may support β-cell failure and development of type 2 diabetes (17).

Research studies suggest that the adipokines, adiponectin and leptin, regulate functional β cell mass, and are crucial for protection against the development of metabolic syndrome and diabetes (18, 19). Adiponectin is a key regulator of insulin sensitivity and tissue inflammation, with predominant action in the liver, skeletal muscles, and vasculature (20). Prominent roles of adiponectin are to improve hepatic insulin sensitivity, increase fuel oxidation, and reduce vascular inflammation. Circulating levels of adiponectin are inversely proportional to body fat content.

Adipokine leptin plays a major role in regulation of energy intake and energy expenditure, and its levels increase in response to accumulation of long-chain free fatty acids (21). In obesity, leptin resistance causes insulin resistance, hepatic steatosis, type 2 diabetes, and

cardiac dysfunction (22). Circulating plasma levels of free fatty acids (FFAs), ceramides and glucose, promote serine phosphorylation of insulin receptor substrate (IRS-1) present in the skeletal muscle tissue; tumor necrosis factor α (TNF-α), promotes serine/threonine phosphorylation of the insulin receptor and IRS-1 (23). Serine-phosphorylated IRS-1 inhibits insulin receptor tyrosine kinase activity, which inhibits downstream insulin signaling. Defective insulin signaling in both the skeletal muscle tissue and fat tissue seems to play a role in development of type 2 diabetes (24, 25).

Inflammatory cytokine interleukin -6 (IL-6) is produced by adipocytes which may also be responsive to IL-6, owing to the presence of IL-6 receptors in the membrane of abdominal subcutaneous adipocytes (26). It was also observed that IL-6 expression was increased 15 fold in the fat tissue of insulin resistant people, which indicated IL-6 induced insulin resistance in fat cells by which it was produced (27). Insulin resistance leads to increased fat mass and BMI. It was suggested that the IL-6- 174C allele from the common functional gene variant IL-6-174GC, is associated with higher BMI in people with type 2 diabetes, and a role for this gene is in mediating inflammatory insulin resistance (28).

The plasma levels of interleukin -6 (IL-6) increase in obesity, and in combination with other cytokines has cytotoxic effects on β-cells causing β-cells apoptosis, and synergizes with interleukin -1 (IL-1) in this respect (29). The interleukin-1 (IL-1) family of ligands and receptors is primarily associated with acute and chronic inflammation, where IL-1β causes local and systemic inflammatory conditions called autoinflammatory diseases (30, 31). The type of inflammation associated with type 2 diabetes is mediated by IL-1β. A sensitive marker for systemic inflammation is the acute-phase C-reactive protein (CRP) (32, 33). Higher plasma concentrations of CRP suggest a state of low-grade systemic inflammation in obese and/or insulin resistant people.

Excessive insulin resistance and metabolic syndrome in Asian Indians

Apparently, both insulin resistance and reduced insulin secretion are involved in the pathogenesis of type 2 diabetes, and the predominant mechanism seems to be different in various ethnic groups. Excessive insulin resistance in Asian Indian migrants appears to be the predominant mechanism for the excessive prevalence of diabetes in this population (34).

Genetic predisposition, central obesity, and unfavorable lifestyle, including unhealthy diet, sedentary lifestyle, and other psycho-social factors related to migration and living conditions were associated with the prevalence of type 2 diabetes in Asian Indian migrants However, lifestyle changes associated with the process of urbanization/westernization might largely explain an ongoing increase in the prevalence of type 2 diabetes in Asian Indian migrants. Apparently, environmental factors certainly play a major role in diabetes epidemics, which occurs on a background of genetic susceptibility (35).

Asian Indians have different body phenotype from Europeans (36). The major differences are in high body fat, high truncal, subcutaneous and intra-abdominal fat, and low muscle mass. Biochemical parameters include hyperinsulinemia, hyperglycemia, dyslipidemia, hyperleptinemia, low levels of adiponectin and high levels of CRP, procoagulant state and endothelial dysfunction.

Owing to lean appearance and lower mean BMI, central obesity in Asian Indians cannot be clinically observed (37). Central obesity plays a significant role in pathogenesis of insulin resistance and is associated with increased risk of diabetes, hypertension, dyslipidemias and atherosclerosis, and represents the corner-stone of the metabolic syndrome (38). Centrally obese people have the most severe insulin resistance and low plasma levels of cytokine adiponectin, as is commonly seen in Asian Indians (39). Low adiponectin concentrations in Asian Indians with impaired glucose tolerance, are predictive of prospective diabetes. Apparently, high percent of total body fat, central obesity, insulin resistance, hyperinsulinaemia, and low muscle mass, predispose Asian Indians to the development of the metabolic syndrome (40). Moreover, about 25% of the urban Indian population of obese or non-obese adults has non-alcoholic fatty liver disease (NAFLD) with significantly higher insulin resistance than those without NAFLD (41). A proton magnetic resonance spectroscopy study of enzymes involved in gluconeogenesis pathway showed elevated gluconeogenesis in non-diabetic, non-obese Indian people with NAFLD. This finding indicates increased risk for the development of type 2 diabetes in later life.

An explanation proposed by Barker as “thrifty phenotype” hypothesis, ascribes the epidemic of type 2 diabetes to an unfavorable intrauterine environment (42). Fetal under-nutrition leads to altered metabolic programming in resource-poor developing countries like India (43, 44). An abundance of food supply may lead to increased velocity of weight gain during childhood and catch-up obesity in low-birth weight Indian babies, which has been reported to be important for adult-onset hyperglycemia and cardiovascular risk factors (45).

Intrauterine growth and development of Indian babies is completed before birth, and is the result of or-

chestrated gene expression influenced by the mother (46). Indian mothers are small and thought to be chronically malnourished, with iron, vitamin, and nutrient deficiencies. One third of Indian babies are born with low birth weight (LBW < 2,5 kg). In comparison with European babies, Indian "thin-fat" babies were lighter and smaller, with the smallest abdominal circumference and mid-arm circumference, while the most preserved measurement was the subscapular skinfold thickness, even in the lightest babies (47). The sub-scapular skinfold in Indian babies is better preserved than the triceps skinfold, suggesting a tendency in Indians to central adiposity, even during intrauterine development. It seems that the relatively thin and centrally fat phenotype of Indian adults originates in intrauterine life (48).

The possibility exists that observed abnormalities might have genetic causes (49). It has been reported that a possible role in regulation of insulin receptor signaling is attributed to ectonucleotide pyrophosphatase phosphodiesterase 1 (ENPP1), a widely expressed class II transmembrane glycoprotein, which could interact with the insulin receptor and decrease insulin-induced tyrosine phosphorylation of its intra-cytoplasmic domain (50, 51). Possibly, this physical interaction occurs on the cell surface between gene ENPP1 and the insulin receptor, preventing insulin-induced conformational changes of the extracellular receptor alpha subunit. This failure prevents beta subunit autophosphorylation and tyrosine-kinase activity, which switches off insulin signaling. A reported gene variant ENPP1 121Q, appears to contribute to the excessive insulin resistance and type 2 diabetes in Asian Indian migrants living in Dallas and South Asians in Chennai, and might provide an important genetic marker to identify Asian Indian people at risk with type 2 diabetes (52).

The D1057 D genotype of insulin receptor substrate -2 (IRS-2) gene, makes Asian Indians susceptible to type 2 diabetes by interacting with obesity (53). Similarly, the peroxisome proliferator activated receptor-co-activator-1 α (PGC-1) gene polymorphism Thr394Thr (G-A) is associated with type 2 diabetes among Asian Indians, and with total, central, and subcutaneous body fat (54, 55).

Vitamin D deficiency and insulin resistance in Asian Indians

The association between vitamin D deficiency and insulin resistance is still incompletely understood in Asian Indians. Serum concentrations of vitamin D are largely determined by food intake and ultraviolet exposure in sunny India (56). Prevalent social and cultural practices in India preclude exposure to sunshine. The problem of vitamin D deficiency worsens in women during pregnancy and has important consequences for the newborn, including fetal hypovitaminosis D, neonatal rickets, tetany, and infantile rickets which is associated with infection of the lower respiratory tract, zand is the leading cause of infant mortality (57, 58). Vitamin D has more targets such as heart, stomach, brain, liver, skin, pancreatic β cells, thyroid, parathyroid and adrenal glands and immune cells which contain the nuclear vitamin D receptor (VDR) and the enzyme 1 α-hydroxylase which facilitates conversion of vitamin D into its active form in kidneys (59, 60). This finding indicates actions of vitamin D other than calcium homeostasis and bone metabolism. The presence of the VDR in pancreatic β cells supports the findings that vitamin D affects insulin secretion and/or insulin sensitivity through the insulin receptor gene. In Asian Indian migrants vitamin D deficiency is further associated with elevated parathyroid hormone (PTH). When serum 25-xydroxyvitamin D (25OHD) falls below 15 ng/mL, PTH levels rise sharply (61). Low levels of vitamin D correlate positively with BMD and/or increased markers of bone catabolism when compared with Caucasians (62). In cultured fibroblasts derived from two groups of participants (Asian Indians and Caucasians) an enzyme 25OHD-24-hydroxylase activity (in vitro) was higher in Asian Indians, which raised concerns about increased catabolism of serum vitamin D. Further, skin capacity for vitamin D synthesis seems to be substantial in both ethnic groups, Asian Indians and Caucasians. Serum vitamin D was measured after exposure to UV-radiation (63). To achieve a given level of vitamin D production, Asian Indians needed over twice as much UV-B exposure when compared to Caucasians. However, suboptimal production of vitamin D in Asian Indians might be the result of conjoined effects of possible catabolism of serum vitamin D and dark skin pigmentation which has been found to decrease skin synthesis of vitamin D because longer exposure to UV radiation is needed. In addition, Asian Indian migrants in sunny Auckland have very low BMC and BMD, which might be related to low serum concentrations of vitamin D, which further is associated with insulin resistance.

HEALTH RESEARCH IN ASIAN INDIAN MIGRANTS AT AUT UNIVERSITY

Research studies present ethnic differences in body size and body composition in Asian Indian migrants in NZ

The study by *Rush et al.*, (64) provided comparative analysis of the body composition of European, Maori, Pacific Island, and Asian Indian peoples in urban

Auckland, New Zealand. The method used in this study included anthropometric measurements (height, weight, and BMI kg/m^2) and the whole body composition (fat mass, fat-free soft tissue and bone mineral content (BMC)) was assessed by DEXA. Fat — free mass was calculated as the sum of fat-free soft tissue and BMC. Percentage of body fat (%BF) was calculated as 100 x FM/DEXA weight. This was the first study which made direct comparison between Polynesian and Asian Indian peoples in New Zealand, the two ethnic groups considered to lie at opposite ends of the spectrum in terms of their body size and body composition. At a fixed percentage of body fat corresponding to BMI of 30 kg/m^2 for Pacific Island people BMI values were up to 5 units higher and for Asian Indians up to 6 units lower, a span of 11 BMI units. For the same BMI, body fat in Pacific men and women was 25% and 38% respectively, while in Asian Indian men and women percent body fat was 37% and 47%, respectively. Therefore, Asian Indian people have more total body fat (TBF) than Pacific people, more central fat, less lean mass and appendicular skeletal muscle mass (APSMM), and less bone mineral content (BMC) than other participants in the study of different ethnicities. Use of universal BMI cut-off points underestimate risks associated with the levels of obesity in Polynesian and Asian Indian ethnic groups, although it does show a need for ethnic specific BMI cutoffs for people of both ethnicities. The BMI cutoff point for observed metabolic risk in different Asian populations should be between 22 kg/m^2 and 25 kg/m^2, and for high risk the range from 26 kg/m^2 to 31 kg/m^2 is appropriate (65). It was also observed that with increasing age, body fat in Asian Indian people showed a shift in the fat body distribution to the abdominal area which shows their tendency for the development of central obesity and consequently insulin resistance, while in people of other ethnicities in the study increase in abdominal fat was coupled with increase in total body fat.

The aim of the study by *Rush et al.*, (66) was to characterize ethnic differences in the relationships between total body fatness and body size and body fat distribution in women from five ethnic groups in New Zealand and South Africa (SA). The objective of the study was to investigate differences in body composition, especially the relationship between BMI kg/m^2 and %BF among female participants. The study participants were 721 women aged 18–60 years from five ethnic groups in New Zealand (173 European, 76 Maori, 84 Pacific, and 93 Asian Indian) and South Africa (SA 201 black and 94 European). The method used included anthropometry (measurements of height, weight, waist and hip circumference), and TBF, central and peripheral body fat, BMC and ASMM were derived from dual X-ray absorptiometry in the Department of Surgery, University of Auckland, New Zealand and the Department of Human Biology, University of Cape Town in South Africa. BMI was derived from height and weight. It was reported that for the same BMI of 30 kg/m^2, the Pacific women had the lowest body fat (~38%BF) while Asian Indian women had the greatest body fat content (~48%BF). Pacific women had the highest levels of fat free mass (FFM) and APSMM, while Asian Indian women had the lowest FFM and APSMM. More importantly, New Zealand Asian Indian women had the greatest central fat mass, followed by the NZ Maori, NZ European and NZ Pacific women who had the least. Also, DEXA derived peripheral or appendicular fat mass (AFM) was the highest in SA black women and Asian Indian women, which can be explained by their greater total body fatness. In addition, the whole body BMC was lower in Asian Indian women followed by Pacific and Maori women. This finding suggests that vitamin D deficiency in Asian Indians in New Zealand is associated with impaired glucose tolerance in the population under a high risk of developing type 2 diabetes.

The study by *Rush et al.*, (67) recruited a total of 114 healthy male volunteers (64 European, 31 Pacific Island, and 19 Asian Indian) aged 17–30 years. Height and weight were measured, BMI was calculated, while %BF, FFM, BMC, bone mineral density (BMD), abdominal fat, thigh fat, and APSMM were obtained from total body DEXA scans. For the fixed BMI, Asian Indian men had significantly more body fat than Pacific Island and European men. These ethnic differences were explained by differences in body build and muscularity in particular. Compared with European men of similar weight and height, Asian Indian men had significantly less skeletal muscle mass, while Pacific Island men had significantly more. Examination of body fat distribution has shown that Asian Indian men have more central fat than European or Pacific Island men. BMC and BMD were lower in Asian Indian than in European and Pacific Island men.

The levels of body fatness, physical activity, and nutritional behavior in 52 Asian Indian men and 62 Asian Indian women, aged 44–91 years (mean 67,5 ± 7.6ys) in the study by *Kolt et al.*, (68). The study draws attention to the different levels of fatness in Asian Indian men and women. The measurements of the BF by the bioelectrical impedance (BIA) have ranged from 13.2% to 58.8% (mean = 41.1, SD = 9.1) for both of the sexes. Asian Indian men (34.6%, SD = 6.8) had significantly less body fat than their female (45.7%, SD = 6.8) counterparts. Cutoff points of greater than 25% body fat for Asian Indian men, and 30% body fat for Asian Indian women, put them under the increased risk of

type 2 diabetes and CHD associated with excess body fat. The use of WC ethnic specific cutoff points in Asian Indian men and women (greater than 90 cm for men and 80 cm for women), have shown that 82% of men and 90% of women had significantly high levels of central obesity, which predispose them to insulin resistance and risk of type 2 diabetes.

Ethnic differences in body size and body composition in Asian Indian children in NZ

The study by *Duncan et al.*, (69), investigated demographic and lifestyle risk factors for excess body fatness in a multiethnic group of 1229 healthy children aged 5–11 years, which consisted of 46.8% European, 33.1% Polynesian, 15.9% Asian, and 4.1% from other ethnicities. The Asian group comprised of Asian Indian children (38.3%), Chinese (21.9%), and Korean (13.8%), Filipino (9.7%), Sri Lankan (4.1%), and other Asian (12.2%) children. The study draws attention to the body composition of Asian Indian children, which in this study is presented only through the %BF, measured using hand-to-foot BIA. Over-fat children were defined as those with a %BF ≥ 25% (boys) and %BF ≥ 30% (girls). Asian children had more excess body fat than European children. Asian Indian and Sri Lankan children comprised the majority of Asian children (42.4%), who at the age of eight had more fat tissue than their European counterparts of the same age, which already put them under the high risk of developing insulin resistance in adolescence or later life.

Genetics and/or lifestyle changes

Living predominantly sedentary lifestyle with low level of physical activity, predispose Asian Indian population in New Zealand to type 2 diabetes and CHD. It appears that early intervention programs are more successful when initiated at an early stage of metabolic syndrome. It is well known that metabolic syndrome is not a diagnosis (70). It is rather a pre-morbid condition that can be reversed at an early stage. The impact of a group diet and physical activity on body composition, lipid profile and insulin resistance in Asian Indian migrants was assessed in the study by *Rush et al.*, (71). Study participants were Asian Indian men and women (aged ? 50 y), recruited from urban Auckland. Anthropometric measures of obesity for total body (BMI) and central fat (WC), and fasting blood tests for serum glucose, insulin and lipids, and blood pressure, were obtained one month prior to the commencement of the intervention program, and were repeated after a five-month period, following the intervention of altered diet and exercise. Significant decrease in body weight, total and central body fat, resulted in decrease in blood pressure in Asian Indian men, while these changes were not significant in women. Lipid profiles in both men and women improved, such as increased blood level of high density lipoprotein (HDL), decreased low density lipoprotein (LDL), and total cholesterol/HDL ratio, without changes in serum glucose, insulin resistance and triglycerides. Apparently, the intervention program was a good indicator that change was possible but limited. However, in the early stages of the disease, insulin resistance is compensated by an increase in pancreatic β- cell mass and function, that often delays diagnosis of type 2 diabetes for a period of years (72).

The role of inflammation in metabolic syndrome

The relationships between markers of insulin resistance and inflammation, resting energy expenditure (REE), and body composition were examined by professor *Rush et al.*, (73). The participants in the study were 79 (38F, 41M; age 30–49 years) healthy adult Asian Indian migrants from urban Auckland. Total and regional body composition, including regional FM and ASMM were determined by DEXA. Beta-cell function (HOMA B %) (74) and insulin sensitivity (HOMA S %) were derived, using homeostatic model assessment. The REE was measured using indirect calorimetry, and fasting blood samples were taken for the measurement of serum glucose, insulin, and cytokines interleukin (IL)-6, tumour necrosis factor (TNF)-α, and CRP. The association of inflammation and metabolic syndrome is particularly relevant to Asian Indians, owing to their high propensity to insulin resistance and central obesity. Apparently, Asian Indian men had more central body fat distribution than women, and their REE rate was highly associated with plasma circulating cytokine IL-6 concentrations. Further, in both sexes IL-6 concentrations were associated positively with % BF and insulin resistance, and inversely with APSMM and insulin sensitivity. The study showed that the relationship between body fat distribution and insulin sensitivity were strongly sex dependent, where male Asian Indians had a greater propensity for the development of the metabolic syndrome than their female counterparts.

CONCLUSION

The research projects which were presented have recognized the major ethnic characteristics of the typical “thin-fat” Indian phenotype in the population at high risk for development of the metabolic syndrome and type 2 diabetes. In comparison with other ethnic groups in New Zealand Asian Indians had more total

Table1. *Summary of findings in selected studies*

Study	Findings
Rush et al., 2009 "Body size, body composition and fat distribution: comparative analysis of European, Maori, Pacific Island and Asian Indian adults"	Participants: 933 (454 men and 479 women) of European, Maori, Pacific Island, and Asian Indian ethnicity, aged 17–80 years ✓ For the same BMI Asian Indian men and women (37% and 47% respectively) have more total body fat than Pacific men and women (25% and 35% respectively). ✓ Asian Indian people have more central fat, less lean mass and ASMM, and less BMC than other participants in the study ✓ With increasing age, body fat in Indian people showed a shift in the body fat distribution to the abdominal area (increase in central fat and insulin resistance)
Rush et al., 2007 "BMI, fat and muscle differences in urban women of five ethnicities from two countries"	Participants:173NZ European, 76 Maori, 84 Pacific, 93 Asian Indian, and South African (201 South African black and 94 South African white) women, aged 18–60years ✓ For the same BMI of 30 kg/m^2 Asian Indian women had the greatest body fat content (~48%BF) while Pacific Island women had the least (~38%BF) ✓ Asian Indian women had the lowest FFM and ASMM AFM was the highest in South African and Asian Indian women (general fatness) ✓ Asian Indian women had the greatest central fat mass followed by Maori, NZ European, and Pacific Island women ✓ The whole body BMC was lower in Asian Indian women followed by Pacific and Maori women. Low BMC indicates vitamin D deficiency and its association with insulin resistance
Rush et al., 2004 "Body size, body composition and fat distribution: a comparison of young New Zealand men of European, Pacific Island and Asian Indian ethnicities"	Participants: 64 Europeans, 31 Pacific Island, and 19 Asian Indian healthy men, aged 17–30 years ✓ For the fixed BMI Asian Indian men had significantly more body fat than Pacific Island and European men ✓ Asian Indian men when compared with their European counterparts had significantly less skeletal muscle mass, while Pacific Island men had significantly more ✓ Asian Indian men had more central fat than European or Pacific Island men ✓ BMC and BMD were lower in Asian Indian men than in European and Pacific Island people
Kolt et al., 2007 "Body fatness, physical activity, and nutritional behaviors in Asian Indian immigrants to New Zealand"	Participants:52 Asian Indian men and 62 Asian Indian women, aged 44–91 years ✓ The %BF have ranged from 13.2% to 58.8% for both of the sexes ✓ About 82% of Asian Indian men(25% BF) and 90% of women (30%BF) had significantly high levels of central fat which predisposed them to high risk of type 2 diabetes
Duncan et al., 2008 "Risk factors foe excess body fatness in New Zealand children"	Participants: 1229 healthy children aged 5–11 years ✓ Over-fat children were defined as those with $ 25%BF ✓ Asian Indian and Sri Lankan children at the age of eight had more fat tissue than their European counterparts at the same age

Continued on the next page

Rush et al., 2007 "Reduction of abdominal fat and chronic disease factors by lifestyle change in migrant Asian Indians older than 50 years"	Participants: 41 Asian Indians (21 men and 20 women) aged > 50 years ✓ Decrease in body weight, total and central body fat, and decrease in blood pressure ✓ Lipid profiles in both Asian men and women improved (HDL, LDL, and total cholesterol/HDL ratio. ✓ Serum glucose, insulin resistance and triglycerides have not changed ✓ Asian Indian men had higher â cell function (HOMA-B%) and lower ✓ Insulin sensitivity (HOMA-S%)
Rush et al., 2007 "Interleukin-6, tumor necrosis factor-alpha and insulin relationships to body composition, metabolism and resting energy expenditure in a migrant Asian Indian population"	Participants: 79 healthy Asian Indians (38 women and 41 men), aged 30–49 years ✓ Asian Indian men had more central body fat than women ✓ Interleukin-6 was associated with REE in Asian Indian men ✓ In both sexes interleukin-6 was positively associated with: %BF and insulin resistance and inversely associated with ASMM and insulin sensitivity ✓ Asian Indian men have a greater propensity for the development of the metabolic syndrome than their female counterparts

body fat, percent body fat, and central fat, less lean mass and appendicular skeletal muscle mass. Higher plasma circulating levels of inflammatory marker CRP indicated the state of low-grade systemic inflammation in obese and insulin resistant people. CRP reflected higher plasma circulating levels of inflammatory cytokine IL-6 in Asian Indian men and women, which were accompanied by an increase in %BF and insulin resistance. It has been notified that male Asian Indians have a greater propensity for the development of the metabolic syndrome than their female counterparts, owing to the presence of more central fat. BMC and BMD were the lowest in Asian Indian migrants, which might be associated with low serum concentrations of vitamin D, which is further associated with insulin resistance. Further research is warranted to clarify metabolic syndrome and associated comorbidities in Asian Indians.

Abbreviations

AFM — appendicular fat mass
APSMM — appendicular skeletal muscle mass
BMI — body mass index
CRP — C-reactive protein
FFM — fat free mass
HbA1c — glycated haemoglobin (A1c)
HOMA B % — beta cell function
HOMA S % — insulin sensitivity
IL-6 — interleukin-6
IL-1 — interleukin-1
IRS-1 — insulin receptor substrate-1
NAFLD — non-alcoholic fatty liver disease
REE — resting energy expenditure
TBF — total body fat
TNF-α — tumour necrosis factor alpha

Sažetak

ETNIČKA PRIPADNOST I DIABETES TIP 2 KOD AZIJSKIH INDIJSKIH MIGRANATA U OKLANDU, NOVI ZELAND

Jowitt Ljiljana

University of Auckland, Department of Surgery, Auckland, New Zealand

Cilj ovog revijalnog rada je bio da se prikažu etničke razlike u veličini i sastavu tela kod Azijskih Indijskih migranata na Novom Zelandu, u vezi sa metaboličkim sindromom i tip 2 dijabetesom, kroz poređenja sa drugim etničkim grupama na Novom Zelandu. Međunarodne baze podataka, uključujući i PubMed i Google Scholar su konsultovani, kao i sajtovi Svetske Zdravstvene Organizacije i Međunarodne Dijabetes Federacije. Oko 74 studija od 128 publikacija su izabrane kao relevantne za temu revijalnog rada. Sedam istraživačkih projekata su prikazali veličinu i sastav tela u Azijskih Indijskih migranata na Novom Zelandu.

Prevalenca tipa 2 dijabetesa od 8,6% kod Azijskih Indusa na Novom Zelandu je još uvek veća nego u njihovoj domovini, zbog njihove etničke pripadnosti, genetske predispozicije, sedelačkog načina života i izmenjene ishrane, i drugih psihosocijalnih faktora koji su vezani za migracije i uslove života kao i stres na radnom mestu i depresiju. Za isti indeks telesne mase, u poređenju sa ljudima iz drugih etničkih grupa na Novom Zelandu, Azijski Indusi imaju više ukupne telesne masti, veci procenat masti u telu, više centralne masti, manje mišićne mase i skeletne mišićne mase. Centralna gojaznost je povezana sa insulinskom rezistencijom i niskim stepenom sistemske inflamacije. S obzirom na dokaze da se tip 2 dijabetesa razvija deset godina ranije u Azijskih Indusa nego u drugim populacijama, dalja istraživanja su neophodna da se razjasni još uvek nekompletno shvaćen metabolički sindrom i „tanki–debeli“ indijski fenotip.

REFERENCES

1. Chan JC, Malik V, Jia W, Kadowaki T, Yajnik CS, Yoon KH, et al. Diabetes in Asia:epidemiology, risk factors, and patophysiology. JAMA. 2009; 301 (20): 2129–40.

2. Jain R, von Hurst PR, Stonehouse W, Love DR, Higgins CM, Coad J. Association of vitamin D receptor gene polymorphisms with insulin resistance and response to vitamin D. Metabolism. 2012; 61(3): 293–301.

3. Zimmet P, Dowse G, Finch C, Serjeantson S, King H. The epidemiology and natural history of NIDDM–lessons from the South Pacific. Diabetes Metab Rev. 1990; 6(2): 91–124.

4. Diaz VA, Mainous AG 3rd, Baker R, Carnemolla M, Majeed A. How does ethnicity affect the association between obesity and diabetes? Diabetic Med. 2007; 24(11): 1199–204.

5. Sachan A, Gupta R, Das V, Agarwal A, Awasthi PK, Bhatia V. High prevalence of vitamin D deficiency among pregnant women and their newborns in northern India. Am J Clin Nutr. 2005; 81(5): 1060–4.

6. Joshi P, Islam S, Pais P, Reddy S, Dorairaj P, Kazmi K, et al. Risk factors for early myocardial infarction in South Asians compared with individuals in other countries. Jama. 2007; 297(3): 286–94.

7. Garduño Diaz SD, Khokhar S. Prevalence, risk factors and complications associated with type 2 diabetes in migrant South Asians. Diabetes Metab Res Rev. 2012; 28(1): 6–24.

8. Bristow S, Rowan J, Rush E. Obesity and gestational diabetes mellitus: breaking the cycle. N Z Med J. 2009; 122(1306): 12–9.

9. Statistics New Zeland 2014. 2013 Census QuickStats about culture and identity.

10. Simmons D, Harry T, Gatland B. Prevalence of known diabetes in different ethnic groups in inner urban South Auckland. N Z Med J. 1999; 112(1094): 316–9.

11. Abbott M, Young W. Asian Health Chart Book 2006:foundation for a new health agenda in New Zeland. N Z Med J. 2006; 119(1244): U2278.

12. Ellison TL, Elliott R, Moyes SA. HbA1c screening for undiagnosed diabetes in New Zealand. Diabetes Metab Res Rev. 2005;21(1): 65–70.

13. Duncan E, Schofield G, Duncan S, Kolt G, Rush E. Ethnicity and body fatness in New Zealanders. NZ Med J. 2004; 117(1195): U913.

14. Stumvoll M, Goldstein BJ, van Haeften TW. Pathogenesis of type 2 diabetes. Endocr Res. 2007; 32(1–2): 19–37.

15. Aguirre V, Werner ED, Giraud J, Lee YH, Shoelson SE, White MF. Phosphorylation of Ser307 in insulin receptor substrate-1 blocks interactions with the insulin receptor and inhibits insulin action. J Biol Chem. 2002; 277(2): 1531–7.

16. Deng Y, Scherer PE. Adipokines as novel biomarkers and regulators of the metabolic syndrome. Ann N Y Acad Sci. 2010; 1212(1): E1–E19.

17. Wellen KE, Hotamisligil GS. Inflammation, stress, and diabetes. J Clin Invest. 2005; 115(5): 1111–9.

18. Weyer C, Funahashi T, Tanaka S, Hotta K, Matsuzawa Y, Pratley RE, et al. Hypoadiponectinemia in obesity and type 2 diabetes: close association with insulin resistance and hyperinsulinemia. J Clin Endocrinol Metab. 2001; 86(5): 1930–5.

19. Oral E, Simha V, Ruiz E, Andewelt A, Premkumar A, Nell P, et al. Leptin-replacement therapy for lipodystrophy. New Engl J Med. 2002; 346(8): 570–8.

20. Whitehead JP, Richards AA, Hickman IJ, Macdonald GA, Prins JJ. Adiponectin–a key adipokine in the metabolic syndrome. Diabetes Obes Metab. 2006; 8(3): 264–80.

21. Donath MY, Shoelson SE. Type 2 diabetes as an inflammatory disease. Nat Rev Immunol. 2011; 11(2): 98–107.

22. Panunti B, Jawa AA, Fonseca VA. Mechanisms and therapeutic targets in type diabetes mellitus. Drug Discov Today Dis Mech. 2004; 1(2): 151–7.

23. Peraldi P, Hotamisligil GS, Buurman WA, White MF, Spiegelman BM. Tumor necrosis factor (TNF)-α inhibits insulin signaling through stimulation of the p55 TNF receptor and activation of sphingomyelinase. J Biol Chem. 1996; 271(22): 13018–22.

24. Caro JF, Sinha MK, Raju SM, Ittoop O, Pories WJ, Flickinger EG, et al. Insulin receptor kinase in human skeletal muscle from obese subjects with and without noninsulin dependent diabetes. J Clin Invest. 1987; 79(5): 1330–7.

25. Olefsky JM. The insulin receptor: its role in insulin resistance of obesity and diabetes. Diabetes. 1976; 25(12): 1154–62.

26. Bastard JP, Maachi M, Van Nhieu JT, Jardel C, Bruskert E, Grimaldi A, et al. Adipose tissue IL-6 content correlates with resistance to insulin activation of glucose uptake both in vivo and in vitro. J Clin Endocrinol Metab. 2002; 87(5): 2084–9.

27. Rotter V, Nagaev I, Smith U. Interleukin-6 (IL-6) induces insulin resistance in 3T3-L1 adipocytes and is, like IL-8 and tumor necrosis factor-α, overexpressed in human fat cells from insulin-resistant subjects. J Biol Chem. 2003; 278(46): 45777–84.

28. Stephens JW, Hurel SJ, Cooper JA, Acharya J, Miller GJ, Humphries SE. A common functional variant in the interleukin-6 gene is associated with increased body mass index in subjects with type 2 diabetes mellitus. Mol Genet Metab. 2004; 82(2): 180–6.

29. Eizirik DL, Sandler S, Welsh N, Cetkovic-Cvrlje M, Nieman A, Geller DA, et al. Cytokines suppress human islet function irrespective of their effects on nitric oxide generation. J Clin Invest. 1994; 93(5): 1968–74.

30. Dinarello CA. Immunological and inflammatory functions of the interleukin-1 family. Annu Rev Immunol. 2009; 27: 519–50.

31. Dinarello CA. A clinical perspective of IL 1β as the gatekeeper of inflammation. Eur J Immunol. 2011; 41(5): 1203–17.

32. Visser M, Bouter LM, McQuillan GM, Wener MH, Harris TB. Elevated C-reactive protein levels in overweight and obese adults. JAMA. 1999; 282(22): 2131–5.

33. Heilbronn LK, Noakes M, Clifton PM. Energy restriction and weight loss on very-low-fat diets reduce C-reactive protein concentrations in obese, healthy women. Arterioscler Thromb Vasc Biol. 2001; 21(6): 968–70.

34. Abate N, Chandalia M. Ethnicity, type 2 diabetes & migrant Asian Indians. Indian J Med Res. 2007; 125(3): 251–8.

35. Mohan V, Sudha V, Radhika G, Radha V, Rema M, Deepa R. Gene-environment interactions and the diabetes epidemic in India. Forum Nutr. 2007; 60: 118–26.

36. Misra A, Khurana L. Obesity-related non-communicable diseases: South Asians vs White Caucasians. Int J Obes (Lond). 2011; 35(2): 167–87.

37. Misra A, Vikram NK. Clinical and pathophysiological consequences of abdominal adiposity and abdominal adipose tissue depots. Nutrition. 2003; 19(5): 457–66.

38. Després JP. Health consequences of visceral obesity. Ann Med. 2001; 33(8): 534–41.

39. Snehalatha C, Mukesh B, Simon M, Viswanathan V, Haffner SM, Ramachandran A. Plasma adiponectin is an independent predictor of type 2 diabetes in Asian indians. Diabetes Care. 2003; 26(12): 3226–9.

40. Yajnik CS, Lubree HG, Rege SS, Naik SS, Deshpande JA, Deshpande SS, et al. Adiposity and hyperinsulinemia in Indians are present at birth. J Clin Endocrinol Metab. 2002; 87(12): 5575–80.

41. Krishnaveni GV, Veena SR, Winder NR, Hill JC, Noonan K, Boucher BJ, et al. Maternal vitamin D status during pregnancy and body composition and cardiovascular risk markers in Indian children: the Mysore Parthenon Study. Am J Clin Nutr. 2011; 93(3): 628–35.

42. Barker DJP, ed. Mothers, babies and health in later life. 2nd ed. Edinburgh: Churchill Livingstone; 1998.

43. Prentice AM. Intrauterine factors, adiposity, and hyperinsulinaemia. BMJ. 2003; 327(7420): 880–1.

44. Prentice AM, Moore SE. Early programming of adult diseases in resource poor countries. Arch Dis Child. 2005; 90(4): 429–32.

45. Sachdev HS, Fall CH, Osmond C, Lakshmy R, Biswas SKD, Leary SD, et al. Anthropometric indicators of body composition in young adults: relation to size at birth and serial measurements of body mass index in childhood in the New Delhi birth cohort. Am J Clin Nutr. 2005; 82(2): 456–66.

46. Yajnik CS. Early life origins of insulin resistance and type 2 diabetes in India and other Asian countries. J Nutr. 2004; 134(1): 205–10.

47. Yajnik CS, Fall CHD, Coyaji KJ, Hirve SS, Rao S, Barker DJ, et al. Neonatal anthropometry: the thin-fat Indian baby. The Pune Maternal Nutrition Study. Int J Obes Relat Metab Disord. 2003; 27(2): 173–80.

48. Yajnik C. Interactions of perturbations in intrauterine growth and growth during childhood on the risk of adult-onset disease. Proc Nutr Soc. 2000; 59(2): 257–65.

49. Abate N, Carulli L, Cabo-Chan Jr A, Chandalia M, Snell PG, Grundy SM. Genetic polymorphism PC-1 K121Q and ethnic susceptibility to insulin resistance. J Clin Endocrinol Metab. 2003; 88(12): 5927–34.

50. Goding JW, Grobben B, Slegers H. Physiological and pathophysiological functions of the ecto-nucleotide pyrophosphatase/phosphodiesterase family. Biochim Biophys Acta. 2003; 1638(1): 1–19.

51. Maddux BA, Goldfine ID. Membrane glycoprotein PC-1 inhibition of insulin receptor function occurs via direct interaction with the receptor alpha-subunit. Diabetes. 2000; 49(1): 13–9.

52. Abate N, Chandalia M, Satija P, Adams-Huet B, Grundy SM, Sandeep S, et al. ENPP1/PC-1 K121Q polymorphism and genetic susceptibility to type 2 diabetes. Diabetes. 2005; 54(4): 1207–13.

53. Bodhini D, Radha V, Deepa R, Ghosh S, Majumder PP, Rao MR, et al. The G1057D polymorphism of IRS-2 gene and its relationship with obesity in conferring susceptibility to type 2 diabetes in Asian Indians. Int J Obes (Lond). 2007; 31(1): 97–102.

54. Vimaleswaran KS, Loos RJ. Progress in the genetics of common obesity and type 2 diabetes. Expert Rev Mol Med. 2010; 12:e7.

55. Vimaleswaran K, Radha V, Ghosh S, Majumder PP, Deepa R, Babu H, et al. Peroxisome proliferator-activated receptor-gamma co-activator-1alpha (PGC-1alpha) gene polymorphisms and their relationship to Type 2 diabetes in Asian Indians. Diabet Med. 2005; 22(11): 1516–21.

56. Rajeswari J, Balasubramanian K, Bhatia V, Sharma VP, Agarwal AK. Aetiology and clinical profile of osteomalacia in adolescent girls in northern India. Natl Med J India. 2003; 16(3): 139–42.

57. Delvin EE, Salle BL, Glorieux FH, Adeleine P, David LS. Vitamin D supplementation during pregnancy: effect on neonatal calcium homeostasis. J Pediatr. 1986; 109(2): 328–34.

58. Muhe L, Lulseged S, Mason KE, Simoes EA. Case-control study of the role of nutritional rickets in the risk of developing pneumonia in Ethiopian children. Lancet. 1997; 349(9068): 1801–4.

59. Chiu KC, Chu A, Go VLW, Saad MF. Hypovitaminosis D is associated with insulin resistance and â cell dysfunction. Am J Clin Nutr. 2004; 79(5): 820–5.

60. Holick MF. Vitamin D: the underappreciated D-lightful hormone that is important for skeletal and cellular health. Curr Opin Endocrinol Diabetes. 2002; 9(1): 87–98.

61. McCarty MF. Poor vitamin D status may contribute to high risk for insulin resistance, obesity, and cardiovascular disease in Asian Indians. Med Hypotheses. 2009; 72(6): 647–51.

62. Awumey EM, Mitra DA, Hollis BW, Kumar R, Bell NH. Vitamin D Metabolism Is Altered in Asian Indians in the Southern United States: A Clinical Research Center Study 1. J Clin Endocrinol Metab. 1998; 83(1): 169–73.

63. Lo CW, Paris PW, Holick MF. Indian and Pakistani immigrants have the same capacity as Caucasians to produce vitamin D in response to ultraviolet irradiation. Am J Clin Nutr. 1986; 44(5): 683–5.

64. Rush EC, Freitas I, Plank LD. Body size, body composition and fat distribution: comparative analysis of European, Maori, Pacific Island and Asian Indian adults. Br J Nutr. 2009; 102(4): 632–41.

65. WHO Expert Consultation. Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies. Lancet. 2004; 363(9403): 157–63.

66. Rush EC, Goedecke JH, Jennings C, Micklesfield L, Dugas L, Lambert EV, et al. BMI, fat and muscle differences in urban women of five ethnicities from two countries. Int J Obes (Lond). 2007; 31(8): 1232–9.

67. Rush E, Plank L, Chandu V, Laulu M, Simmons D, Swinburn B, et al. Body size, body composition, and fat distribution: a comparison of young New Zealand men of European, Pacific Island, and Asian Indian ethnicities. N Z Med J. 2004. 117(1207): U1203.

68. Kolt GS, Schofield GM, Rush EC, Oliver M, Chadha NK. Body fatness, physical activity, and nutritional behaviours in Asian Indian immigrants to New Zealand. Asia Pac J Clin Nutr. 2007; 16(4): 663–70.

69. Duncan JS, Schofield G, Duncan EK, Rush EC. Risk factors for excess body fatness in New Zealand children. Asia Pac J Clin Nutr. 2008; 17(1): 138–47.

70. McAuley KA, Williams SM, Mann JI, Walker RJ, Lewis-Barned NJ, Templa LA, et al. Diagnosing insulin resistance in the general population. Diabetes Care. 2001; 24(3): 460–4.

71. Rush EC, Chandu V, Plank LD. Reduction of abdominal fat and chronic disease factors by lifestyle change in migrant Asian Indians older than 50 years. Asia Pac J Clin Nutr. 2007; 16(4): 671–6.

72. Jonas JC, Bensellam M, Duprez J, Elouil H, Guiot Y, Pascal SM. Glucose regulation of islet stress responses and â cell failure in type 2 diabetes. Diabetes, obesity and metabolism. 2009; 11 Suppl 4: 65–81.

73. Rush EC, Plank LD, Yajnik CS. Interleukin 6, tumour necrosis factor alpha and insulin relationships to body composition, metabolism and resting energy expenditure in a migrant Asian Indian population. Clin Endocrinol (Oxf). 2007; 66(5): 684–90.

74. Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC. Homeostasis model assessment: insulin resistance and â-cell function from fasting plasma glucose and insulin concentrations in man. Diabetologia. 1985; 28(7): 412–9.

Correspondence to /Autor za korespondenciju
MD Ljiljana Jowitt MA
Faculty of Health and Environmental Sciences
Interprofessional Health Studies
North Shore Campus, AF 416
90 Akoranga Drive, North Shore Campus,
Auckland 0627
New Zealand
Work phone number: +649 921999 x 8096;
Work fax number: +649 9219721
Mobile phone number: +649226515962
Email address: ljowitt@aut.ac.nz