Dietary and lifestyle factors of diabetes in Inuit of Canada

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Contribution of Authors:

Since this study was a part of 2007-2008 IPY HIS and the data was already collected, Ms. Saghar Sefidbakht analyzed the data, and wrote the manuscript. Dr. Grace Egeland as a principal investigator and organizer for the Inuit Health Survey managed the data collection and provided guidance, feedback, and editorial assistance for thesis and the manuscript. Dr. Young also contributed in the Inuit Health Survey and provided some guidance.

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List of Abbreviations:

ADA= American Diabetes Association

BMI= Body Mass Index

BF= Body Fat

CDA=Canadian Diabetes Association

CFGHE= Canada's Food Guide to Healthy Eating

CHO= Carbohydrate

E= Energy

FA= Fatty Acid

FFQ= Food Frequency Questionnaire

FNIRHS =First Nation and Inuit Regional Health Survey

GDM= Gestational Diabetes Mellitus

GI= Glucose Intolerant

HEI= Healthy Eating Index

IFG= Impaired Fasting Glucose

IGT= Impaired Glucose Tolerance

IHS= Inuit Health Survey

IPAQ =International Physical Activity Questionnaire

IPY= International Polar Year

IR HOMA=Insulin Resistance using Homeostasis Model Assessment Index

LA= Linoleic Acid

MET=Metabolic Equivalent

NG= Normoglycemic

NIDDK=National Institute of Diabetes and Digestive and Kidney Diseases

NIDDM= Non Insulin Dependent Diabetes Mellitus

OGTT= Oral Glucose Tolerance Test

PROT= Protein

PUFA= Poly Unsaturated Fatty Acid

RBC= Red Blood Cell

SES= Socioeconomic Status

SFA= Saturated Fatty Acid

TF= Traditional Food

TFA= Trans Fatty Acid

Type 2 DM= Type 2 Diabetes Mellitus

WC= Waist Circumference

1.1.Abstract:

Introduction: Among Inuit, rates of diabetes are currently increasing.

Objectives: To investigate the lifestyle factors associated with newly identified glucose intolerance (GI) among Inuit.

Methods: A cross-sectional study of a subsample of 813 adults with a 2-hr oral glucose tolerance test who participated in the International Polar Year Inuit Health Survey (2007-2008). Those with pre-existing diabetes were excluded. Individual and dietary questionnaires and anthropometric measurements were also collected.

Results:

GI was associated with older age and a higher body mass index, %body fat, and waist circumference. Percent Energy protein and % Energy high-sugar drinks were positively associated with GI. Adjusting for those two aforementioned nutrients, %E traditional food was significantly protective ($P<0.05$). Fiber (g/d) was inversely and cholesterol (mg/d) was positively associated with risk for GI with a borderline significance ($P< 0.10$).

Conclusion: These findings emphasize the need for dietary and lifestyle changes to prevent high rates of GI among Inuit.

1.2. Résumé:

Introduction: Chez les Inuit, le taux de diabète courament à la hausse.

Objectifs: Etudier les facteurs associés au style de vie, chez les Inuit nouvellement diagnostiqués avec l'intolérance au glucose (IG).

Méthodes: Une étude transversale d'un sous-échantillon utilisant un test de glucose oral de tolerance de 2-h sur 813 adultes ayant participé à l "International Polar Year Inuit Health Survey" (2007-2008). Ceux qui ayant un diabète préexistant ont été exclus. Des questionnaires individuels et alimentaires et des mesures anthropométriques ont également été recueillis chez chacun des participants.

Résultats: L'IG a été positivement associée à l'âge, l'indice de masse corporelle, le pourcentage de masse adipeuse, le tour de taille, le pourcentage d'énergie provenant des proteines et de l'énergie provenant des boissons sucrées. Après ajustement pour ces deux types d à liment, la nourriture traditionnelle offer une protection significative contre l' IG de ($P < 0.05$). La consomation de fibres (g/j) est inversement associée et le cholestérol (mg /j) positivement associé au risque d' IG, avec une signification limitée ($P < 0.10$).

Conclusion: Ces résultats soulignent le besoin de changements nutritionels et de mode de vie pour prévenir les taux élevés d' IG chez les Inuit.

2. Background:

2.1. Introduction:

According to the Canadian Diabetes Association (CDA), an estimated 285 million individuals world-wide have diabetes and almost 7 million people develop diabetes each year[1] . According to the American Diabetes Association (ADA), 7.8 % of the population of the USA (23.6 million children and adults) have diabetes [2] . According to CDA in 2010 more than 3 million Canadians were affected by diabetes, and almost 6 million more had pre-diabetes [1].

Diabetes is a disease in which insulin is not produced in sufficient quantity, or the insulin sensitivity of the cells is diminished and body is unable to use blood glucose for energy [3]. In a normal situation when glucose and amino acid levels of blood are elevated after a meal, insulin secretion will be stimulated; which in turn increases glucose uptake, utilization, and storage in cells [4]. The possible risk factors for diabetes are genetics, and environmental factors such as decreased physical activity and obesity.

According to Health Canada the rate of diabetes in Canada's Aboriginal population is 3 to 5 times higher than the general population [5] . According to a 1991 Statistics Canada report, 8.5% of North American Indians on Indian reserves and settlements, 5.3% of North American Indians off-reserve, 5.5% of Métis, and 1.9% of Inuit had diabetes [6]. Also, while type 2 DM has typically affected older adults, children and, in particular, Aboriginal children, are experiencing an increase in the incidence of type 2 DM. Although the rates of diabetes are low

among Inuit, according to Health Canada, an increasing trend in the rates of type-2 DM has also been noted for Inuit [7]

2.2. Classification:

The current thesis focuses on type 2 DM which is one of the 4 types of diabetes which is characterized by a reduction in insulin sensitivity of cells. Type 1diabetes mellitus is due to insufficient production of insulin resulting from destruction of pancreatic *beta*-cells and is considered to be very rare among Inuit [8]The other two types of diabetes include other specific types of diabetes which are due to other causes such as genetic defects in *beta*-cell function or insulin activity, diseases of the exocrine pancreas, and drug or chemical induced diabetes; and gestational diabetes mellitus (GDM) defined as diabetes diagnosed during pregnancy [9]. Glucose tolerance deteriorates during normal pregnancy in all women; 2-7% of which develop GDM [10]. In addition to type 2 DM, two conditions have been associated with increased risk of developing type 2 DM: impaired fasting glucose (IFG) and impaired glucose tolerance (IGT) which are commonly referred to as pre-diabetes. Pre-diabetes is diagnosed when the glucose levels of blood are higher than normal but not yet high enough to diagnose diabetes (i.e. Fasting plasma glucose >= 7 mmol/L or 2-hr , and 2-hr plasma glucose >=11.1 mmol/L) [9].

2.3. Risk factors:

The risk of developing type 2 DM increases with age, obesity, and lack of physical activity [11].

Family history of diabetes is another risk factor for diabetes with a relative risk ranging from 2 to 6 in different studies [12]. Also, minorities and women have been shown to be at greater risk for diabetes than Caucasians and men [13]. Studies have also shown that low income and educational status are associated with an elevated risk of diabetes [14].

Several studies showed that smoking can be a risk factor as well [15, 16]. On the other hand, some studies showed a possible association between moderate alcohol intake and lower risk of type 2 DM [17, 18]. Generally, the role of smoking and alcohol on type 2 DM risk is not well established.

2.3.1. Sex:

More than half of the people affected with diabetes in United States are men (11.2% of men vs. 10.2% of women) [2]. A study based on the data of NHANES III showed that the prevalence rate of diabetes diagnosed by a physician before NHANES III was similar between non-Hispanic white men and women, but it was significantly higher in women for non-Hispanic blacks and Mexican-Americans. The rate of diagnosed and undiagnosed diabetes plus IGT was similar for the whole U.S. population [19]. The highlights from National Diabetes Surveillance System of Canada in 2005-2006 show that 5.5% of all women, and 6.2% of all men had diabetes. Therefore in Canada the prevalence of diabetes is

higher in men with the exception of young adults, where the prevalence of diabetes was higher in young women than young men [20]. Yet in a study of Ontario's native population women appeared to be more at risk of developing obesity, IGT, and type-2 DM, and at an earlier stage of life [21]. A systematic review indicated that glycemic status and risk of type-2 diabetes may differ in men and women due to the differences in endogenous sex hormones [22].

2.3.2. Age:

It is known that with aging glycemic control deteriorates and the prevalence of IFG and IGT increases [23]. Further, studies suggest that each year of aging is associated with an increase in HbA1c [24, 25]: a marker of plasma glucose concentration over the previous 2-3 months [26, 27]. Davidson mentions 5 possible explanation for impaired glucose homeostasis with aging: improper diets, decreased physical activity, reduced lean body mass, reduced insulin secretion, and finally insulin antagonism [23]. Even when studies [28, 29] controlled for weight gain, reduced physical activity, increased drug intake and other influential diseases due to aging that might affect glucose homeostasis in the body, and an independent physiological age related effect on HbA1c has been cited. Highlights from the National Diabetes Surveillance System in Canada show that 22% of Canadians between 75-79 years of age had been diagnosed with diabetes which was almost ten times the prevalence seen in adult Canadians between 35-39 years of age (2.3%) [20]. Based on American Diabetes Association criteria the total

prevalence of diabetes (diagnosed and undiagnosed) rose from 1-2% at ages 20-39 years to 18-20% at ages 60-74 years, and plateaued after 74 years of age [19].

2.3.3. Ethnicity:

According to American Diabetes Association criteria, the NHANES III data, US minority groups have higher risk of diabetes, i.e. the risk of diabetes in Mexican-Americans and non-Hispanic blacks is almost twice higher than that of non-Hispanic whites [19]. Also, minority groups in the U.S. are at higher risk of GDM [30]. In Canada, a study by Rodrigues et al showed that the rate of GDM in the Cree of the Eeyou-Istchee region of eastern James Bay was as high as 12.8%, which is almost twice as high as that of the general population of North America [31]. High-risk ethnicities in Canada are: Aboriginal, African, Hispanic, and Asian ethnicities [20]. One of the reasons which has been mentioned for the higher risk of diabetes in minority groups is the existence of the "thrifty gene" in these racial groups, which is a left over gene from their ancestors, which used to help them survive during famine periods [32].

- **Arctic Indigenous Populations**:

The prevalence of diabetes and obesity have increased dramatically among Alaska natives in the second half of the 20th century [33, 34], the prevalence of diabetes increased by 22% from 15.7/1000 in 1986 to 19.2/1000 in 1993, the vast majority of which were type 2 DM; the incidence was 1.5 per 1000 Alaska natives per year [35]. Epidemiological data on Alaskan overweight and obesity

show that the increase in obesity prevalence parallels the changes in diabetes prevalence in this population [36]. Increased consumption of market foods, reduced physical activity, and some other factors such as stress, depression, smoking, alcohol consumption can be the possible reasons for the increased prevalence of chronic disease in Alaskans, although the relative effects of each of them is unknown [37].

Also a study of Greenland Inuit showed that the prevalence of diabetes was high, and heredity was a major factor, while obesity and diet were important environmental factors [38].

According to public health agency of Canada the rate of diabetes is also high in Canadian Aboriginal populations [20]. In 1997 the First Nation and Inuit Regional Health Survey (FNIRHS) which contained all first nation people on reserves in all provinces of Canada plus Inuit of Labrador, showed that age-adjusted prevalence of diabetes in First Nation men and women was 3.6 and 5.3 times higher than Canadian men and women [39]. For Métis, although there is less data available [39],a study by Bruce [40] suggests that the prevalence of diabetes in Métis (6%) is twice as high as that of the overall population of Canada (3%). Canadian Inuit people are an exception in this matter, since their rate of diabetes (≈1.9% in 1991) is less than the national average [6, 41]; however, there is a concern that in the future this rate will increase [39].

2.3.4. Family history:

Several genetic factors have been reported as being related to risk of diabetes, but their usefulness for estimating the risk of diabetes is limited [42, 43]. Although genetic information related to diabetes might become a useful tool in public health evaluation systems in the future [44], family history is an adequate and easily obtained marker of genetic and environmental factors shared by close relatives [12, 45]. Family history is a well-documented independent risk factor for Type 2 DM [12, 46] with an estimated relative risk (RR) ranging from 2-6 depending on study designs [12]. In epidemiological studies, individuals with positive history of diabetes in their first degree relatives had higher risk of developing diabetes, and that risk was increased if both parents had diabetes [12]. For instance in one study of Pima Indians, they found a 2.3 times and 3.9 times higher risk of developing diabetes in those with one parent affected or both parents affected, respectively [47]. One study showed that Insulin resistance is a poor indicator of Type-2 DM in those who did not have a family history of the disease [46].

2.3.5. Body Mass Index (BMI) and other anthropometric risk factors:

BMI is an independent modifiable risk factor for diabetes [48] as it is related to insulin resistance and related health effects including atherosclerosis, hypertension, hypertriglyceridaemia, glucose intolerance, dyslipidemia, hyperuricemia [11]. Obesity is considered as a major cause of insulin receptor

dysfunction; which is thought to be related to the increased expression of tumor necrosis factor α, and abnormality in the protein tyrosine phosphate of skeletal muscle. Also central obesity can lead to insulin resistance which can increase insulin demand and cause hyperinsulinemia; which in time can lead to hepatic and muscular insulin resistance [49]. Insulin resistance is a responsible factor for hyperglycemia in type-2 DM. The risk factors for insulin resistance are multi-factorial, including: abnormal function of insulin receptors, disturbed insulin signaling pathways, abnormal glucose transportation or glucose metabolism. One of the most important factors for insulin resistance in liver and skeletal muscle is the increased availability of free fatty acids (FFA). This effect is more evident in obese individuals who have abnormal FFA metabolisms. In these individuals, there is an increase in the FFA release from adipose depot to the blood. The increased level of FFA, through a competitive inhibition, impairs the muscle uptake of glucose [50].

Overweight diabetic patients showed an improvement in their glycemic control and reduction of plasma lipids through weight reduction [51]. Therefore, one of the recommended treatments for insulin resistance is reducing weight, lowering plasma lipid levels, increasing physical activity and improving diet which can directly and indirectly affect insulin receptors [52]. Hamman et al found that a 1 kg of weight loss decreased the risk of developing diabetes by 16%; the incidence of diabetes was 44% lower among those who did not achieve the intended weight loss but achieved the physical activity goals [53]. An energy decrease of 500-1000 kcal/d can lead to a weight loss of 1-2 pounds/wk. A moderate physical

activity of 60-75 mins/day or a vigourous physical activity of 35 min/d is needed to maintain long-term weight loss [54]. A study of Canadian Inuit showed that body mass index (BMI), waist circumference (WC), and percent body fat (%BF) predicted insulin resistance using homeostasis model assessment index (IR HOMA) and insulin sensitivity index (ISI 0,120) in women; and in men %BF predicted IR HOMA, and %BF and WC predicted ISI 0,120 [55]. In Canada's Aboriginal population the central (abdominal) obesity with the higher waist to hip ratio is the predominant type of obesity [21, 56]. Central obesity has been shown to be positively associated with risk of diabetes [39]. In Pima Indians even after controlling for BMI there was a significant positive association between waist to thigh ratio (upper body distribution of fat) and diabetes [57].

On the other hand, one study showed that especially in women shorter height and leg length, and lower leg length–to– height ratio (higher sitting height-to-height ratio), were all associated with higher percent of body fat. Also, they found a negative association between leg length–to– height ratio and insulin resistance estimated by HOMA-IR especially in women [58].

2.3.6. Physical activity (PA):

Physical activity even without weight change can increase insulin sensitivity [59]. Studies have shown that exercise can enhance insulin sensitivity in both type 2 DM patients and non-diabetic individuals; the effect of which can remain up to 72-hrs after exercise [60]. Beside its direct influence on insulin sensitivity, PA also can assist in weight loss (particularly of body fat supplies) and weight

management, which can also help in controlling diabetes [61]. Exercise and insulin both have different signaling pathways which activate glucose transport. This may explain why muscle glucose transport increases in response to an acute bout of exercise in individuals with insulin resistance. Regular exercise in humans has a lot of beneficial effects on the skeletal muscle including an increase in the GLUT4 expression [62].

Cross-sectional studies demonstrate that the prevalence of diabetes is higher among those with a sedentary lifestyle [63, 64], even after controlling for BMI, sex, age and waist-to-thigh ratio [64]. In a prospective study, leisure time activities, such as walking, stair climbing, and sports were protective against development of type 2 DM. The incidence of diabetes decreased as the energy expenditure increased from less than 500 to 3500 kcal/week. After adjusting for age, by each 500 kcal increase in energy expenditure the incidence of type 2 DM diminished by 6% (RR: 0.94) [65]. For a healthy body weight Canada's Physical Activity Guide recommends 30 to 60 minutes of moderate physical activity per day for adults and at least 90 minutes per day for children and youth. This activity can be in periods of at least 10 minutes for adults and five minutes for children and youth [66]. For people with diabetes it is recommended to have a moderate – to vigorous-intensity aerobic exercise of at least 150 min/wk (e.g. 30 minutes, 5 days a week) [67]. Moderate physical activity is defined as activity performed at an intensity of 3 to 6 METs (work metabolic rate/resting metabolic rate), which for most healthy adults is equal to brisk walking at 3 to 4 mph [68].

2.3.7. Diet and lifestyle:

A reduction in the intake of saturated fatty acids (SFA), *trans* fatty acids (TFA), and cholesterol is likely to improve lipid profiles and insulin sensitivity [69]. In regards to fat intake and risk of type 2 DM, studies are conflicting. One prospective study of Pima Indian women showed that dietary fat and calorie intake were associated with increased diabetes risk [70]. Similarly, an association between fat intake and diabetes was observed in Mexican Americans [71]. Another study showed a possible long-term (5 yrs) effect of a reduced fat ad libitum diet on maintaining improvements in glycemic status [72]. On the other hand some studies showed no significant association between total fat intake and type 2 DM incidence [73].

In regards to the effect of types of fat on type 2 DM, some studies suggested a protective effect of substituting non-hydrogenated poly unsaturated fatty acid (PUFA) for TFA on Type 2 DM risk [73]. Further, one study showed that the negative effect of PUFA: SFA ratio on type 2 DM was independent of age, sex, family history of diabetes, and other lifestyle factors [74]. Dietary SFA has been more consistently associated with increased risk of type 2DM. Studies have shown that palmitate prevents the activation of insulin receptor substrates which can lead to insulin resistance [75, 76]; also studies in rats showed that palmitate might reduce the levels of adiponectin, [77] - a protein produced by adipocytes which has an insulin sensitizing effect and is inversely associated with obesity [78]. Therefore SFA can lead to insulin resistance through decreasing adiponectin

secretion and insulin signaling pathways [79]. SFA also decreases PPARγ coactivator (PGC)-1α, which can lead to a reduction in FA and glucose oxidation ,so they will accumulate in tissues and blood [79]. Finally SFA can increase the levels of diacylglycerol, ceramide, and protein kinase C in muscles, which can also adversely affect the glucose uptake [79]. For long-chain n-3 fatty acids found in sea foods, there is some controversy; one study showed high consumption of C20-C22 *n*-3 FAs protects against the development of metabolic syndrome and glucose intolerance [80]. The suggested mechanism is that n-3 FAs affect the cell membrane phospholipids in a way that insulin receptors' activity increase which will lead to increased insulin secretion from the ß-cells and augmented insulin sensitivity in peripheral cells [81], it can also stimulate the translation of adiponectin in adipocytes [82]. On the other hand, some studies showed a diabetogenic impact of long-chain n-3 fatty acids on pancreatic ß-cell function; it showed that the enhancement of whole body insulin sensitivity by supplementing a high SFA diet with n-3 FA is less than the suppression in insulin response to glucose [83]. For n-6 fatty acid which is mostly found in safflower, sunflower and corn oils, and its most common form is linoleic acid (LA), there is not much evidence in regards to its association with diabetes [84]. However, a few studies found that n-6 fatty acid intake is negatively associated with type 2 DM [73, 85, 86]. Other studies showed that substituting SFA with n-6 FA improved insulin sensitivity [87, 88]. For cholesterol a positive association with the incidence of diabetes was observed [89].

American Diabetes Association (ADA) suggests a high carbohydrate diet, which is a good source of vitamins, minerals, and fiber, to prevent and treat obesity and type 2 DM [90]. The ADA diet was shown to increase satiety and therefore decrease the total energy intake [91]. A low calorie, high carbohydrate (CHO), low fat diet has been shown to decrease the risk of type 2 DM in high risk individuals [92]. There is also a high mono-unsaturated fatty acid diet, which has shown to improve lipoprotein profiles and glycemic control in both type 1 DM and Type 2 DM [93]. Another popular, but controversial diet, is a high- protein, high-fat, low-CHO diet [91]. While short-term studies have shown that the high-protein diet can improve glucose metabolism, increase satiety and decrease energy intake, other cross-sectional epidemiological studies showed that it may increase the risk of diabetes [91]. In short term studies (6 months) a greater weight loss was observed in among those on a high-protein high-fat low-CHO diet, than the conventional diet group, with restricted fat intake [94, 95]. This effect was not observed after 12 months [94, 96]. A small study [97] showed that although the weight loss was comparable between the 2 diets, a high-CHO diet increased insulin sensitivity and decreased fasting plasma glucose and glycosylated hemoglobin, while the high protein diet was not effective. On the other hand, a few cross-sectional studies showed that high protein diet has a negative association with insulin sensitivity and glucose tolerance [98, 99]. Some prospective studies also showed that this diet can increase the incidence of type 2 DM [100, 101]. A positive association was seen between processed red meat consumption and type 2 DM [100]. It has been suggested that increased amino

acid levels in the plasma can have substrate-mediated (substrates for gluconeogenesis), and hormone-mediated (stimulating endogenous secretion of both insulin and glucagon and changing the ratio of insulin/glucagon ratio) effects on glucose metabolism [102]. An in-vivo study [103] showed that a short-term elevated amino acid level in plasma was followed by an impaired increase in skeletal muscle glucose 6-phosphate, reduced insulin-stimulated glucose disposal from the whole body, and a decrease in skeletal muscle glycogen synthesis rates. The study indicated that elevated amino acid levels can directly inhibit muscle glucose transport and/or phosphorylation, and therefore lead to insulin resistance. Also, it was suggested that amino acid overload can cause S6 kinase 1 over-activation which inhibits insulin induced class-1 phosphoinositide 3-kinase activation, and can lead to insulin resistance through negative feedback [104]. It is also assumed that in type 1 diabetic patients, high protein diet can increase the progression of autoimmune-mediated loss of endogenous insulin secretion [98, 105].

Another important dietary factor of research interest is the glycemic load (glycemic index* amount of CHO) in the diet. Diets high in glycemic load and low in cereal fiber were shown to be associated with increased risk of diabetes [106]. One study in college students showed that dietary fiber was negatively associated with fasting insulin levels in men and women, and was negatively associated with adiposity measurements in men [107]. Another study showed that an increase in insoluble fiber intake for 3 days increased the whole body insulin sensitivity in overweight and obese women [108]. Studies have shown

that incorporating glycemic load assessments into population-based observational studies is problematic given the methodological problems in characterizing glycemic load associated with mixed meals where protein and fat content can affect the glycemic responses to the type and amount of carbohydrate consumed [109]. However, as Willet et al mention, although the other components of diet might affect the glycemic responses to the carbohydrate load, this variation does not significantly affect the validity of glycemic index calculated for mixed meals [110].

Large prospective cohorts of both men and non-pregnant women showed an association between "Western diet" - (red and processed meat, sweets and refined grains) - with increased risk of type 2 DM [111, 112]. A study in Native Canadians of northwestern Ontario [113] showed that some foods were protective against IGT and diabetes such as vegetables, breakfast foods and hot meal foods that are mostly rich in fiber and low in fat content. On the other hand, high consumption of junk foods, the bread and the butter groups – that have high contents of fat and simple sugar and low fiber- and also fatty methods of food preparation were both associated with increased risk of diabetes [113]. Another study in U.S. men also supported the idea that high glycemic index foods and low cereal fiber consumption were positively associated with risk of type 2 DM [114]. Lifestyle interventions aimed at reducing energy intake and increasing physical activity are inversely associated with insulin resistance and the incidence of diabetes [69]. Also lifestyle changes with the aim of reducing weight and total fat and SFA intake and

increasing physical activity were effective in preventing type 2 DM in high-risk individuals [115]. In Diabetes Prevention Program (DPP) after 2.8 years, those in the lifestyle intervention group had 58% reduced risk for developing diabetes, while those in Metformin intervention group had a 31% reduced risk. Also it was seen that Metformin had almost no effects in postponing diabetes in individuals greater than 60 years of age or people with a BMI<=30 kg/m^2 [116].

Health Canada encourages people to maintain healthy habits which include eating healthy and being active. Healthy eating includes eating the recommended daily types and amounts of food according to Canada's Food Guide to Healthy Eating (CFGHE) [117], and reducing total fat intake (by choosing lower fat products), SFA and TFA consumption, also eating less sugary foods, to limit extra energy intake [118] . The National Cholesterol Education Program recommends a daily intake of total fat equal or less than 25% to 35% of total calories and a daily intake of SFA <7% of total calories [69]. In 1998, the American Diabetes Association called for flexibility in the amount of recommended daily fat for individuals with diabetes, to make it possible to have palatable and low-calorie choices based on cultural context and individual taste [119].

2.3.8. Smoking:

In 2007 a systematic review and meta-analysis on the active smoking and the risk of type 2 DM yielded 25 prospective studies which were published between 1992-2006. From these 25 studies, 24 reported adjusted RRs greater than 1 (ranging

from 0.82-3.74), while the pooled adjusted RR was 1.44 (95% CI, 1.31-1.58) [16]. Some Studies showed that smoking is a risk factor for diabetes, independent of BMI [120]. And some showed a dose-response association between smoking and incidence of diabetes in both men and women [15]; they found that diabetes rate was higher in those who smoked >=2 packs a day than those who never smoked (the increased rate was 45% in men and 74% in women) [15]. Cullen et al suggested a BMI-independent effect through which the risk of diabetes was elevated by smoking [120]. In the 2007 systematic review authors concluded that although the included studies meet several recommended criteria for causation (temporality, dose response association, and biological plausibility), there is still a need for future studies with detailed measurement and adjustment for potential confounding factors such as socioeconomic status, physical activity, and dietary habits [16]. The proposed mechanism for positive effect of smoking on diabetes include decreased insulin sensitivity, abdominal obesity, endothelial dysfunction [121, 122], and antiestrogenic effects of smoking in women [123]; also it is suggested that smoking provokes hyperglycemia, elevated insulin, and hypertension [120, 124].

On the other hand, there are few studies which showed a negative effect between smoking and metabolic syndrome, which was due to the role of smoking in the weight loss [125]. One study showed that among Inuit, non-smokers were heavier than smokers [126]. These findings suggest that in Inuit, smoking may decrease the risk of obesity-related diseases [125]. One study in British towns showed that the risk of diabetes and cardiovascular disease remained the same over the five

years after quitting smoking, which might have been due to the following weight gain. The authors then conclude that although smoking-cessation is associated with weight gain and therefore increased risk of diabetes, in the long-term the benefits will outweigh this disadvantage [127].

2.3.9. Alcohol consumption:

Moderate alcohol consumption has been related to lower risk of type 2 DM [17]. A review of past studies showed that moderate alcohol consumption was associated with lower risk of type 2 DM while high consumption seemed to increase that risk. However, findings were not consistent regarding the association between moderate alcohol consumption and diabetes [128].

Possible mechanisms for the effect of moderate alcohol consumption on lowering risk of type 2 DM include that moderate drinking is associated with lower concentrations of markers of inflammatory and endothelial dysfunction [129, 130], and better insulin sensitivity [131, 132], which could be mediated by an increase in adiponectin levels [133]. Also some studies suggest that moderate alcohol consumption might be related to lower BMI and weight gain in women, but not men [134]. In a cross-sectional study of the severely obese, type 2 DM was significantly less in alcohol consumers than rare- or non-consumers (OR: 0.29; 95% confidence interval, 0.16 to 0.55). A u-shape relationship was found between the amount and frequency of alcohol consumption and fasting glucose, HbA1C, insulin resistance measurement index, and fasting triglyceride. But this effect was attenuated after excluding diabetic patients from the analysis [18].

Further, polymorphism in the alcohol dehydrogenase gene (ADH1C) has been shown to modify the relationship between alcohol intake and risk of type 2 DM [135].

2.3.10. Socioeconomic status [136]:

Socioeconomic status is an important factor in health care and disease prevention [14]. Both educational and income levels are important indicators of socioeconomic status (SES) [14]. In Sweden, low social status was associated with poor glycaemic control [137]. In Canada, the prevalence of self-reported diabetes increased with decreasing educational and income levels in both sexes; the odds ratio for women, but not men, remained significant after adjustment for age, area of residence, body mass index, and physical activity [14]. Another study in Canada showed that the prevalence of diabetes increased with age and was also associated with low income [136].

2.4. Diet and diabetes in Northern Indigenous Peoples:

Arctic traditional food is defined as harvested foods from local environment, and includes fish and marine mammals, game animals, wild greens, and berries [138, 139]. N-3 fatty acids are mostly available in sea foods like fish and sea mammals, where decosahexanoic acid (C22:6 n-3) (DHA) and eicosapentaenoic acid (C20:5n-3) (EPA) are found. However, if sea foods are not available, some vegetables and plant oils can provide short-chain n-3 fatty acids, which is mostly in the form of alpha-linolenic acid (ALA: C18:3 n-3)

[140]; therefore Arctic traditional food is rich in *n*-3 fatty acids (FAs) [141]. Features of traditional diet are similar across Arctic Indigenous populations. The traditional diet of Alaskan Natives is remarkably high in fat, yet its emphasis on marine sources of fatty acids is thought to contribute to the historically low prevalence of chronic diseases observed in this population [80]. On the other hand a study of Canadian Baffin by Kuhnlein et al. showed that although the consumption of animal foods was high, most of the fat and SFA intake came from market foods [142, 143].

Westernization over the past century has made major changes in food sources and intake of numerous nutrients among Alaskan Natives; the benefits of traditional food are not well understood due to a lack of long-term prospective studies [144]. But benefits of traditional food have been described in several studies. Bersamin and his colleagues [144] found that fatty acid composition of the diet differed according to the level of traditional food intake in Alaskans. Traditional food intake was positively associated with higher total fat, EPA, and DHA intake, and negatively associated with linoleic acid (LA) intake. Also analyses of red blood cell fatty acid composition showed a positive association between markers of traditional food consumption and high-density lipoprotein cholesterol concentration and a significant negative association with triglyceride concentration. Although total fat intake was higher among participants consuming more traditional food, it did not put them at a greater risk for chronic disease; they suggest it might be explained by the specific types of fatty acids in the traditional foods. The ratio of PUFA: SFA was higher in those who consumed more

traditional foods and although they had a higher intake of total fat, there was no significant difference in their total SFA intake. Also the study showed no significant relationship between the high-fat traditional diet and weight, % body fat, or BMI [144].

Another study by Bersamin et al showed that Eskimos with highest consumption of traditional foods consumed 10.7% more energy from fat and 18.1% more energy from protein, while those with lowest consumption of traditional food derived 30% more energy from carbohydrates. There was also a significant difference between their micro nutrient intakes; participants with highest level of traditional food consumption consumed significantly more vitamin A, vitamin D, vitamin E, iron, and *n*-3 FAs, but less vitamin C, calcium, and total dietary fiber [145].

Besides containing more *n*-3 FAs, traditional food has other nutrient benefits such as being rich in MUFA, protein, and micronutrients [146]. A study on Arctic Canadian Indigenous Peoples (Yukon, Dene/Metis, Inuit) [147] showed that in adults, traditional animals and fish contributed an average of 17% of dietary energy in Yukon First Nations, to 28% of dietary energy in Inuit; the overall community average ranges varied between 6-40% in the 3 cultural groups of the study (among Inuit adults community averages ranged from 13 to 40%). Mean intake of traditional animals and fish in Dene/Metis children was 4.5% (0.4-15%) of their dietary energy and in Inuit teens the mean intake of traditional food (TF) was 15% of total energy; this younger generation provided >40% of their total energy intake from high-sugar and high-fat foods. Results showed that in both

children and adults, even a single portion of the traditional animals and fish food led to a significant increase ($P<.05$) in intakes of energy, protein, vitamin D, vitamin E, riboflavin, vitamin B-6, iron, zinc, copper, magnesium, manganese, phosphorus, and potassium (the difference for manganese, vitamin D and energy was not significant in children) [147]. A study on one of the Baffin communities showed that although TF was high in fat and the consumption of traditional food was high in the community, market food contributed more fat; it also contributed more energy, carbohydrate, sodium, and calcium. In most age groups, traditional food contributed more vitamin A, protein, phosphorous, iron, magnesium, and zinc [142]. Zinc is a select nutrient which can be found in a wide variety of foods, particularly in association with protein food sources, and since protein consumption is higher in those who consume more traditional foods, it can be expected that zinc consumption will also be higher in traditional food consumers, as it was found in the last 2 studies as well. As Beletate et al mention in their review article, the mineral zinc plays an important role in the synthesis and action of insulin, it seems to enhance the stimulation of insulin action and insulin receptor tyrosine kinase activity; therefore it can play a role in prevention of type 2 DM [11].

In contrast with traditional food, western diet has more TFA, which is created through the transformation of PUFAs from their normal *cis* form to the *trans* form through hydrogenation during high heating process; and high intakes of TFAs have been associated with adverse effects on type 2 DM [148].

A rapid shift away from traditional food and an associated increase in market food has been seen in Indigenous Peoples globally [143].The shift away from traditional diets to western diets joined with reduced physical activity has increased the prevalence of certain chronic diseases, including obesity, cardiovascular disease, and type 2 DM [149, 150]. A similar experience is hypothesized to be occurring among Alaskan Natives. Coincident with a change in dietary patterns, the prevalence of cardiovascular co-morbidities, including obesity has increased and may even be higher among Alaskan Natives than non-Alaskan Natives [151]. A study of Alaska native residents of 20 years old or more, showed that the incidence of type 2 DM in Athabascan Indians was twice as high as that among Yup'ik Eskimos. Athabascan Indians consumed more market food relative to TF than Yup'ik Eskimos, and consumed less indigenous carbohydrate and fat. Those with glucose intolerance had greater market protein intake and less seal oil intake, and were heavier than those with glucose tolerance [152]. Ebbesson et al found a relationship between fatty acid metabolism and glucose tolerance in Eskimos and that participants with impaired glucose tolerance had lower plasma concentrations of certain *n*-3 fatty acids (18:3 and 20:5 *n*-3 FAs) and *n*-6 FAs (18:3, 20:3 and 22:4 *n*-6 FAs) and had higher concentrations of palmitic acid (16:0) and oleic acid (18:1 *n*-9) than those with normal glucose tolerance; which suggests that a deviation from traditional food of fish and marine mammals may be associated with glucose intolerance and insulin resistance [153].

Although as mentioned above traditional food has important advantages (higher *n*-3 FAs, higher PUFA: SFA ratio, and lower TFAs) that might be helpful in preventing DM, there are other nutrient exposures associated with traditional food that could have either beneficial or deleterious effects that should be mentioned as well. Traditional food has high concentrations of selenium (Se) (esp. from marine foods such as whale, seal, and fish). Although Se has an important anti-oxidative effect (through its role in glutathione peroxidase [154]) with potential benefits for protecting individuals against oxidative stress and related health outcomes), it is also known to be harmful at high doses. A study of Greenlandic Inuit showed that whole blood Se concentrations ranged from 178 µg/L in one population to 488 µg/L in the other [155]. Se, in normal doses, is thought to be effective in diabetes due to its anti oxidative characteristics [156]. One study showed a lower serum and RBC levels of Se in diabetic patients (both type1 and 2) vs. non-diabetic participants, and suggest that this reduction in Se level of diabetic patients is due to increased glutathione peroxidase activity [156]. Yet, a recent study has shown that high serum Se levels were positively associated with the prevalence of diabetes [157], also another study suggested that selenium supplementation may have increased type 2 DM [158]. Thereby with a proposed dose-dependent effect of Se on type 2 DM, and since traditional food has high levels of Se, the evaluation of biomarkers of Se intake and risk for type 2 DM is warranted. While typically in those who consume more western and manufactured foods SFA [144] and TFA intake is higher compared to those who consume more traditional foods [148], a healthy western diet has some advantages like

containing more vitamin C, and fiber through fruit, vegetables, and whole grains [145], and has been shown to be inversely associated with diabetes incidence [159] [160].

3. Rationale:

The goal of this thesis is to evaluate the dietary and lifestyle factors associated with newly diagnosed diabetes and pre-diabetes in Inuit residing in 3 jurisdictions in Canada (Inuvialuit Settlement Region, Nunavut Territory, and Nunatsiavut). The thesis will provide Inuit-specific context of the lifestyle, diet quality, and traditional vs. market food consumption characteristics related to emerging diabetes and will help to inform public health agencies and communities involved in the design of public health promotions and interventions.

4. Study hypotheses:

The main hypothesis of this study is that glucose intolerant (GI) Inuit have less reliance on Traditional food (TF) and poorer dietary quality than normoglycemic (NG) Inuit after adjusting for important covariates.

5. Study objectives:

The overall aim of this thesis is to evaluate the dietary, anthropometric, and other lifestyle factors for their association with newly identified GI among Inuit.

More specifically this study will investigate the association of the following dietary factors with GI:

- Extent of traditional food vs. market food intake by using the FFQ.
- Macronutrient intake distribution, high sugar foods and drinks, and high fat foods intake distribution by using the 24-hr recall.
- Diet quality [based on Canada's Food Guide to Healthy Eating (CFGHE) and the Canadian version of the Healthy Eating Index] by using the 24-hr recall.
- Selected micronutrient intake such as zinc (Zn) by using the 24-hr recall.

6. Ethics and research licenses:

Ethical permission from McGill University Institutional Review Board, plus research licenses from Nunavut Research Institute and Aurora Research Institute, as well as written consents from participants were obtained. Nunatsiavut waived requirement for license. Steering committees and Community-University agreements were developed.

7. Study Design:

This study represents analyses of data collected from a cross-sectional International Polar Year Inuit Health Survey (2007-2008). Households were randomly selected from 36 communities representing 3 Inuit jurisdictions: Inuvialuit Settlement Region, Nunavut Territory, and Nunatsiavut. A total of 2,796 households were approached regarding participation in the survey, from which 1,901 (68.0%) households participated. From these households, 2,595

adults 18 years of age or older participated in the survey. From this sample, those who completed the oral glucose tolerance test (OGTT) and did not have previously diagnosed diabetes were included in the thesis analyses (n=813). For detecting previous diagnosis of diabetes, medication charts were used, and those on any medication or treatments for diabetes were excluded. The survey included 2 kinds of activities: land and ship activities. After informing the randomly selected households and signing the participants in a step by step process, and filling the home-based questionnaires, the nurses interviewed the participants for the supplements and medicines used, and clinic appointment was made which was either held on the CCGS Amundsen ship or in a local clinic for the 3 inland communities. The questionnaires conducted included one 24-hr dietary recall, one food frequency questionnaire (FFQ), and individual questionnaires; the questionnaires were completed with the help of bilingual English and Inuit speaking interviewers. For clinical evaluations, height, weight, and blood pressure were measured, also blood samples were collected for tests such as fasting plasma glucose (FPG), and OGTT as described below:

7.1. 24-hr dietary recall:

For 24-hr dietary recalls, trained interviewers obtained a detailed and precise description of all foods and drinks consumed in the last 24 hrs using a 4 stage multiple pass technique and 3 dimensional food model kits to help determine portion sizes. The 4 stage interviewing technique involved: 1. obtaining a quick list of foods eaten and the time of consumption, 2. asking respondents to describe the consumed foods and drinks in more details, and the interviewer must make

sure that they did not forget any snacks drinks or sugar, cream and milk in the coffee or tea, 3. going back to the beginning of the recall and ask the participants to give details about the amounts of each food and drinks they consumed; the models of standardized portions is used, and 4. a final check.

24-hr dietary recalls have some strength compared to FFQ; like food records, 24-hr dietary recalls are open ended, and therefore allow accommodating any food, food combination and food preparation method. This is helpful in comparing nutrient intakes with their recommendations, and also in measuring absolute energy and macronutrient intakes. 24-hr dietary recall also shows the diversity in the study population, and contributes to the flexibility of data analysis, since it provides foods, nutrients and food groups [161]. One study on a group of non-institutionalized elderly subjects compared the internal validity of 24-hr dietary recall and a 7-day dietary record showed that both resulted in approximately equal estimates of the mean intake [162]. Some studies [163, 164] showed that there is a small difference, about 10% [164], between the calculated and the analyzed zinc content of foods. For selenium (Se) intake estimation direct chemical determination is not practical for large study samples. Food composition tables could be an alternative to estimate individual Se intakes based on their diet. But even this method is limited to studies in which the foods are not from different geographic areas with geologically different soils [165]. Only in such studies a fairly good agreement between the calculated and the analyzed Se values would be observed [166]. On the other hand, some studies that compared the individuals' energy intake measured from self-reports with their energy expenditure, showed

that food records might underestimate energy intake, which is mostly due to underreporting. Underreporting among obese [167] and elderly [168] populations is common. It has also been noted in athletic women [169], and men [170]. To evaluate the extent of under-reporting of energy intake, the energy intake/BMR ratio [171, 172] was assessed and evaluated using the Goldberg cut-off methodology [172].

Also to evaluate the dietary quality and compare it between the glucose tolerance groups, the Canadian version of the healthy eating index (HEI) was used [173]. HEI was calculated for each of the 9 diet quality components based on the requirements for age and sex and was then summed to provide a total HEI-score for each participant.

7.2.Food Frequency Questionnaire (FFQ):

FFQs were administered after 24-hr recalls; it measured the usual frequency and servings of the certain foods and drinks consumed over the past year before the survey. Each respondent was asked to report their usual frequency of country and store-bought food consumption and their usual serving sizes. FFQ had 2 parts as follows: Country food questionnaire which contained food items from hunting/fishing/gathering (15 sea mammal, 9 land animal, 6 fish, 4 bird, 3 plant foods); respondents were asked how often they ate these foods over the last year; this was based on seasonal consumption (IN season; OFF season). And abbreviated market food questionnaire which referred to foods purchased in stores (soft drinks (regular or diet), powder drinks (fruit drinks, sport drinks, iced tea),

real fruit juice, milk (fresh or powder), and chips/crisps/cheese puffs); respondents were asked how often they consumed a specific food item during the last month.

7.3. **Individual Questionnaire:**

The individual questionnaires included information on general health, dental health, and medical information (including information on cancer, heart disease, diabetes, high blood pressure, and high blood cholesterol in the respondent or their parents and siblings), smoking, socio economic status, and physical activity; the questions used to measure physical activity were based on the short version of the International Physical Activity Questionnaire (IPAQ) [174]. In 2000, 14 centers from 12 countries evaluated the repeatability and validity of short and long form of IPAQ; a median ρ of about 0.8 in reliability, which was almost the same for short and long form, and a median ρ of 0.3 in validity, which was comparable to most self-reported validation studies, was found [175]. The individual questionnaire used in the current study included questions about how many days in the last week the respondent walked, did vigorous activities, or engaged in moderate activities, and how much time in each day they engaged in each of those activities. The physical activity assessment is described elsewhere [176]:

Briefly, each activity in the IPAQ has a MET (metabolic equivalent) score, which represents multiples of resting metabolic rate, for a standard body weight of 60 kg in a quiet sitting position using the comprehensive compendium of physical activity [177]. The MET score is then multiplied by the minutes spent in that

activity in the past week to give the value for each activity (MET minutes/week) [175]. The values from each activity were then summed to provide a total MET min/week. Based on the 2005 IPAQ protocol, MET values of 3.3, 4, and 8 were considered for walking, moderate activity, and vigorous activity in respect [178] The following formulas were also taken from IPAQ 2005 protocol were used:

- Walking MET-minutes/week= 3.3*walking minutes*walking days
- Moderate MET-minutes/week=4*moderate-intensity activity minutes*moderate intensity days
- Vigorous MET-minutes/week=8*vigorous-intensity activity minutes*vigorous intensity days
- Total physical activity MET-minutes/week = sum of Total (Walking + Moderate + Vigorous) MET-minutes/week scores

IPAQ PA categories are as follows:

-high: a)vigorous-intensity activity on at least 3 days and accumulating at least 1500MET-min/wk OR, b) 7 or more days of any combination of walking, moderate-intensity or vigorous activities achieving a minimum of at least 3000 MET-min/wk.

-moderate: a) 3 or more days of vigorous activity of at least 20 minutes/day OR, b) 5 or more days of moderate-intensity activity or walking of at least 30 minutes/day OR, c) 5 or more days of any combination of walking, moderate, or vigorous activity achieving a minimum of 600 MET-min/wk.

-low: If responses do not meet the criteria for "high" or "moderate" PA.

7.4. Anthropometric measurements:

The anthropometric measurements included: height (using stadiometer, to the closest millimeter) which is used along with body weight to calculate body mass index (kg/m^2), sitting height (using sitting height table, to measure the vertical distance between sitting surface and top of head to the nearest mm), waist circumference which is a reliable index for determining the extent of abdominal obesity that can be related to the risk of metabolic complications (by a flexible tape- midpoint between the top of the hip and the last loose rib), weight and % body fat using bioelectrical impedance analysis [Tanita scale (TBF-300A,Tanita Corp, Arlington Heights, Illinois): by entering sex, body type, height, age, and weight of clothing, gives the body weight, and %BF values]; bioelectrical impedance analysis combines the impedance value with anthropometric data to measure body components but does not specify the location of body fat.

7.5. Blood sample collection and laboratory analyses:

After asking the participants when they last drank, ate, or smoked, venous blood samples were drawn. The first laboratory assessment was FPG (after at least 7 hr fasting, but not more than 18 hrs) in which a 3mL venous blood sample was collected in gray top tubes and was labeled as G1 which means fasting sample. Samples were put in plastic bags and kept in lab cooler at 4°C, then as soon as possible they were centrifuged to separate plasma from RBC; with a transfer pipette the plasma was transferred to a labeled cryovial. They were then capped

with a red cap and frozen at -80°C and were sent to university of Guelph for analysis. The second laboratory analysis was the oral glucose tolerance test (OGTT) that was used to measure the ability to remove an added glucose load (75 g glucose) from circulation (in 2 hrs). For those who were going to do the OGTT the fasting blood sugar was quickly estimated by using OneTouch® Ultra2TM glucometer (LifeScan, Inc., Milpitas, California) to make sure it's safe to proceed with OGTT. The process of sampling was the same as FPG except that the vacutainer was labeled as G2 for the 2hr PC sample. To avoid hypoglycemia a protein breakfast was provided following the test. Hyperglycemia is a sign of diabetes, patients with BG> 10mmol/L were referred to medical doctors. Since some of the diabetic patients might have normal levels of FPG but are unable to produce enough insulin for prompt metabolism of the ingested CHO, it is important to do both tests to identify diabetic individuals. OGTT was not performed on those who had already been diagnosed with diabetes. The samples were taken 2 hrs after the glucose load was ingested, and were kept in a grey top tube labeled as G2. The same process as fasting blood sample was done on them before being sent to the lab. RBC fatty acids were also measured in another laboratory assessment.

8. Sample Size Estimation and Statistics:

For sample size estimation, it was estimated that for a power of 90% with a significance level of 0.05 for detecting an effect size as small as 1% in %E from fat with a standard deviation (SD) as big as 4%, and for 2 t-tests (one for males,

one for females), a sample as big as 676 would be needed. Therefore the sample of 813 is safe for this study.

Stata10 was used to measure mean, SD, and percentages for demographic and anthropometric characteristics based on sex and GI-status.

For categorical variables, chi-square test was used to detect differences between the GI status groups of each sex. For continuous anthropometric and dietary variables, t-test was used to compare GI-status groups of each sex.

Multivariate logistic regressions models were conducted for each dietary factor separately or in combination with few other dietary factors controlling for important demographic and anthropometric covariates.

9. Definitions:

Diabetes is characterized by casual plasma glucose of >= 200 mg/dl (11.1mmol/l) plus classic symptoms of diabetes such as polyuria, polydipsia, and unexplained weight loss; or fasting plasma glucose (FPG) >= 126mg/dl (7mmol/l) in which plasma glucose is measured after an 8 hour fasting; or with a 2-hr plasma glucose >= 200mg/dl (11.1mmol/l) during an oral glucose tolerance test (OGTT) using a glucose load containing 75-g anhydrous glucose dissolved in water [9].
Pre-diabetes is a stage where hyperglycemia exists but it is not severe enough to meet the criteria of diabetes. Depending on whether it is recognizable through

FPG or OGTT, this stage can be categorized either as IFG (impaired fasting glucose) or IGT (impaired glucose tolerance):

IFG is identified by FPG of 100 mg/dl (5.6mmol/l) to 125 mg/dl (6.9mmol/l). and IGT is identified by 2-h plasma glucose of 140 mg/dl (7.8mmol/l) to 199 mg/dl (11.0mmol/l) [9]. The IFG of >5.6 predicted future diabetes better than previous cutoff of >6.1 [179].

IFG and IGT are called "pre-diabetes"; which is a risk factor for future type 2 DM and CVD [180]. For the purpose of this thesis, Inuit with IGT, IFG, and type 2 DM will be combined into Glucose Intolerant group (GI).

Based on WHO, BMI between 25-29.9 kg/m^2 is defined as overweight and BMI >=30 kg/m^2 is obese [181]. The same definition (obese: BMI>=30) is used in the current study to investigate %obese in the sample population.

The IDF waist circumference cut offs for metabolic syndrome in USA was used for this study (WC>=102 cm in men, and WC> =88cm in women) [182, 183], and it used the American Council on Exercise cut off to identify high %BF (%BF>= 25 for men, and %BF>=31 for women).

10. Manuscript: "Characteristics associated with emergence of glucose intolerance in a low-risk Canadian Inuit population."

Sefidbakht S, Young K, Egeland GM.

Abstract:

Introduction: The rapid shift away from traditional food along with other lifestyle changes including reductions in physical activity among Indigenous Peoples globally has increased the prevalence of obesity-related chronic diseases such as type-2 diabetes mellitus (DM). Among Inuit, rates are still low but increasing.

Objectives: To investigate among Inuit the association between dietary, anthropometric and other lifestyle factors with newly identified glucose intolerance (GI), defined as either type 2 DM, impaired fasting glucose , or impaired glucose tolerance.

Methods: This cross-sectional study was a part of the Canadian International Polar Year Inuit Health Survey (2007-2008). Of the original 2,595 participating adults, those with completed 2-hr oral glucose tolerance test (OGTT) and without pre-existing diabetes were included in the current analyses (n=813). Anthropometric assessments and questionnaires on medication usage and health histories, food frequency and a 24-hr dietary recall were administered.

Results: For men and women, GI was associated with older age and a higher body mass index, %body fat, and waist circumference (WC). In multivariable logistic regression, % E from PROT (OR: 1.04, $p \leq 0.01$), and %E from high-sugar drinks

(OR: 1.03, $p \leq 0.05$) were significantly associated with increased risk of GI; while %E from traditional food was significantly associated with reduced risk for GI (OR: 0.99, $p \leq 0$ .05) in a model with all three aforementioned dietary variables, age, sex, region, and WC.

Fiber (g/d) was inversely and cholesterol (mg/d) positively associated with risk for GI but associations only approached borderline significance ($P < 0.10$).

Conclusion: Adiposity, advancing age, and % E from non-Indigenous protein in the previous day were risk factors for GI. The findings contribute to the emerging literature on the relationship of high processed meat intake and high-sugar drinks with diabetes risk.

Introduction:

Worldwide, almost 285 million people have diabetes. In Canada, 2 millions have diabetes and 6 millions have pre-diabetes [1]. The rate of diabetes is 3-5 times higher in Aboriginal populations than the general population of Canada. The rate is still low among Inuit, but there is evidence that it is increasing [5] [38]. The risk of developing type 2 diabetes mellitus (DM) increases with age, obesity, and lack of physical activity [11], and is higher among those with a family history [12]. Also, minority groups, women [13], and those with a low income and education are at increased risk of diabetes [14]. Role of smoking is still controversial [16, 125], while a negative association between moderate alcohol intake and type 2

DM has been reported [17]. Diet is another important factor in type 2 DM. A reduction in the intake of SFA, *trans* fatty acids (TFA), and cholesterol was shown to improve lipid profiles and insulin sensitivity in an intervention study [69].

While some studies have shown a positive association between fat intake and type 2 DM [70, 72], other studies have shown no significant association [73]. A protective effect of substituting non-hydrogenated PUFA for TFA [73] and SFA [74] has been observed while the role of n-3 fatty acids in diabetes is controversial [78, 81, 83]. Some studies have shown that n-6 fatty acid intake is negatively associated with type 2 DM [73] and others have found that substituting SFA with n-6 FA improved insulin sensitivity [88]. Also a positive association between cholesterol intake and incidence of diabetes has been reported [89]. Cross-sectional studies have shown that a high protein diet is associated with increased risk for insulin resistance and glucose intolerance [98, 99]. One study in Alaska Natives showed a higher intake of non-Indigenous protein among those with glucose intolerance [152]. High carbohydrate, low calorie, low fat diet was associated with decreased risk of type 2 DM in high-risk individuals [92] and the American Diabetes Association now recommends a high carbohydrate diet (45-65% of calories, <130 g/d for adults), which provides vitamins, minerals, and fiber considered important in the prevention and treatment of obesity and type 2 DM [90].

Arctic traditional food (TF) is defined as harvested foods from the local environment, and includes fish and marine mammals, game meat, wild greens, and berries [139].

The traditional diet of Arctic Indigenous Peoples is remarkably high in fat, yet the marine sources of fatty acids are thought to contribute to the historically low prevalence of chronic diseases observed in this population [80]. Also a study of Canadian Inuit found that while the consumption of TF was high, most of the fat and SFA in the diet came from market foods [142, 143].

The goal of the current study is to evaluate the dietary and lifestyle factors associated with newly diagnosed diabetes and pre-diabetes in Inuit of Canada.

Methods:

A cross-sectional Canadian International Polar Year Inuit Health Survey was conducted in 3 jurisdictions (Inuvialuit Settlement Region of Northwest Territory, Nunavut Territory, and Nunatsiavut of Northern Labrador) in 2007 and 2008. Households were randomly selected and self-identifying Inuit adults 18 years of age or older were invited to participate in the survey. A total of 2796 Inuit households were successfully contacted and 1901 (68.0% of households) participated in the survey (n=2,595). Due to survey logistical constraints, approximately a 30% sub-sample of survey participants had a 75g oral glucose tolerance test (OGTT) either on board the Canadian Coast Guard Ship (CCGS) Amundsen which facilitated the research in 33 coastal communities or assessed at clinic sites in 3 land-based surveys for inland communities. Thus, of the original

2,595 adults that participated in the survey, only those with completed 2-hr OGTT and without pre-existing diagnosed diabetes, as identified by medication or dietary treatments, were included in the present analyses (n=813). The exclusion of individuals with pre-existing diabetes was important for the present analyses as a diabetes diagnosis would influence dietary and other lifestyle behaviors and complicate the interpretation of the results evaluating these factors as they relate to the emergence of disease in a low-risk population.

The results from the fasting and the OGTT were used to classify participants as glucose intolerant (GI) or normoglycemic (NG), using American Diabetes Association criteria for impaired glucose tolerance and impaired fasting glucose [180] (IFG: FPG of 5.6-6.9mmol/L; IGT: 2 hr-OGTT of 7.8-11 mmol/L). The ADA criteria (5.6-6.9 mmol.l) were used instead of CDA criteria (6.1-6.9 mmol/l) since studies have shown that the 5.6 mmol/dl is a better cut point for prediction of future diabetes [184]. Fasting plasma glucose (after at least 7 hr fasting, but not more than 18 hrs) in which a 3mL venous blood sample was collected in gray top tubes. Samples were put in plastic bags and kept in lab cooler at 4°C, then as soon as possible they were centrifuged to separate plasma from RBC; with a transfer pipette the plasma was transferred to a labeled cryovial. They were then capped with a red cap frozen at -80°C and were sent to university of Guelph for analysis of plasma glucose assessed by Glucose Hexokinase II (GLUH) method [185]. The samples were again taken 2 hrs after the glucose load (75 g) was ingested. Nurses also conducted anthropometric assessments, collected fasting and 2-hour venous blood samples, and assessed health histories and medication

usage. Trained bilingual Inuit and English language interviewers administered a FFQ and 24-hr dietary recall questionnaires and a day-to-day dietary quality control assessments conducted by dietitians. The FFQ included a traditional food component which contained an extensive list of food items from hunting fishing, and gathering (15 sea mammal, 9 land animal, 6 fish, 4 bird, 3 plant foods); respondents were asked how often they ate these foods over the last year (seasonal consumption: IN season; OFF season), and to estimate portion sizes using food model kits. The second component of the FFQ included an abbreviated market food section which referred to beverages and snacks purchased in stores and eating establishments including: soft drinks, powder drinks, fruit drinks, sport drinks, iced tea, real fruit juice, milk (fresh or powder), and chips/crisps/cheese puffs. Further, respondents were asked how often they consumed a specific food item during the last month.

For analysis, the frequency and amount of intake of each type of TF (sea mammals; land animals, fish, birds and plant foods) were summed and differences in frequency of consumption were evaluated by GI status. A total TF intake variable was generated by summing up the frequency of the five TF types intakes. For the market foods FFQ sugar-sweetened beverages summed soft-drink, powdered sugar drinks, energy drinks, and fruit juices/flavored punches. Participants removed shoes and wore light clothing for the anthropometric assessments which included height (HT), waist circumference (WC), and weight (WT) and % body fat (%BF) using a Tanita foot-to-foot bioelectrical impedance scale (TBF-300A,Tanita Corp, Arlington Heights, Illinois). Body mass index

(BMI) was calculated as kg/m^2 where a BMI between 25-29.9 kg/m^2 was considered overweight and a BMI >=30 kg/m^2 was considered obese according to WHO criteria [181].The International Diabetes Federation (IDF) suggested cut offs for metabolic syndrome in USA were used to define high waist circumference (WC>=102 cm in men, and WC> =88cm in women)[183]; and the American Council on Exercise criteria was used for defining high %body fat (%BF>= 25 for men, and %BF>=31 for women) [186].
A short version of International Physical Activity Questionnaire (IPAQ) was used to calculate MET-min/week [175, 177].

Dietary variables examined included: Canada's Food Guide To Healthy Eating (CFGHE) food groups (4 groups: Grain products, vegetables and fruits, milk products, meat and alternatives [117]), % energy from macronutrients, and selected dietary factors postulated to be related to type 2 DM risk, including fiber [107], cholesterol [89], and zinc [11]). The Canadian Nutrient File was used to estimate the nutrient intakes [187].The Canadian version of HEI [173] was calculated for each individual where a score of 80 or higher indicates a healthy eating pattern.

Statistical analysis:

Chi-square tests were conducted to evaluate differences in demographic characteristics between those with and without GI in analyses stratified by sex. T-tests for differences in mean dietary exposures between those with and without GI

were also evaluated. Furthermore, multivariable logistic regression analyses was conducted, where the outcome was glucose intolerance (yes vs. no) and independent variables considered included dietary factors, and important covariates such as age, sex, WC, and region (representing Inuvialuit Settlement Region, Nunavut and Nunatsiavut). Also, total energy intake was controlled for in the logistic regressions when evaluating the past-day nutrient intakes such as fiber (g/d), zinc (mg/d), and cholesterol (mg/d). Macro-nutrients and TF intakes were evaluated in regression analyses using the nutrient density approach (i.e., as % of energy intake). It is important to note that since a certain amount of a nutrient has less effect on larger and higher energy-consuming people than smaller and lower energy consumers, in epidemiologic studies nutrient intake should be adjusted for total energy intake [161]. One analytic way to adjust for energy intake when investigating the role of a specific nutrient in a disease, which is associated with energy intake, is to control for total energy intake as a second independent variable in the multivariate model. Using nutrient densities (macronutrient intake as a percentage of total calorie intake) is another way to control for the effect of total calorie intake [161].

Further, to evaluate the extent of under-reporting of energy intake by participants and to evaluate whether the extent of under-reporting varied by GI status, the ratio of reported energy intake to basal metabolic rate (EI:BMR) was evaluated where a ratio under 1.52 was considered an indication of under-reporting based upon methodology described elsewhere [171, 172]. Statistical analyses were conducted using STATA version 10.1 (STATA corporation, College Station, Texas). Two-

sided tests were conducted in all analyses and a p-value $\leq$ 0.05 was considered statistically significant.

Results:

Demographic characteristics

A total of 319 males (M) and 494 females (F) completed the 2-hr OGTT. The prevalence of GI was 19.4% in men and 18.0% in women. The mean age of the men and women included in the present analyses was 42.6 yrs (SD: 14.2) and 41.6 yrs (SD: 13.7), respectively. Women were more likely to be obese than men (44 vs. 26%, $p \leq 0.05$), and were more likely to be in high-WC group (63.0 vs. 28.0 %, $p < 0.05$) and high-%BF group (68.4 vs. 40.8%, $p < 0.05$); they also had significantly lower mean MET-minute/wk (3118.7, SD=3591.4) than men (5412.8, SD=5129.7, $p < 0.05$) and smoked fewer number of cigarettes/day (10.5, SD=6.9) than men (12.2, SD=7.8; $p \leq 0.05$).

WC was strongly and positively correlated with other adiposity measurements such as BMI (r: 0.91, $p < 0.05$), and %BF (r: 0.78, $p < 0.05$). Men and women with GI were significantly older than those without GI (for men: 52.2 yrs (SD: 13.6) vs. 40.3 yrs (SD: 13.3), $p<0.01$; and for women 50.7 yrs (SD: 13.4) vs. 39.6 yrs (SD: 13.0), $p \leq 0.01$). As with age, adiposity was strongly associated with risk for GI, where men and women with GI had a higher mean BMI than normoglycemics (for men 29.5 (SD: 5.3) vs. 26.3 (SD: 4.8), $p<0.01$; and for women: 32.8 (SD: 7.1)

vs. 28.6 (SD: 6.8), $p<0.01$). Similarly, the mean %BF (Men: 28.3 (SD=8.4) vs. 22.1 (SD=8.6), $p< 0.05$; women: 40.9 (SD=7.7) vs. 34.7 (SD=9.7), $p< 0.05$), and WC (men: 100.2 (SD=15.5) vs. 90.1 (SD=13.7), $p< 0.05$; women: 103.4 (SD=17.2) vs. 93.1 (SD=16.5), $p< 0.05$) were significantly higher among men and women with GI than among normoglycemics . Physical activity total MET (min/wk) was not significantly different between the GI groups of men (NG: 5439.8 (SD= 5103.7) vs. GI: 5299.7 (SD=5279.3), *p*: 0.85), and women (NG: 3080.0 (SD=3635.1) vs. GI: 3263.4 (SD=3398.9), *p*: 0.67).

In further analyses of the % GI by categories of risk factors, a striking age-gradient in risk for GI was noted for men and women (Table 1). Likewise, a greater risk of GI was noted among those classified with high BMI, %BF, and WC. Those with less than a high school education and those reporting no alcohol drinking had a greater prevalence of GI relative to those with a higher level of education, and those reporting alcohol consumption. Smokers had a lower prevalence of GI than non-smokers. Percent GI was statistically significantly higher in men with a family history of diabetes compared to men without a family history; however differences were not statistically significant for women (Table 1).

In a multivariable logistic regression model entering all the statistically significant demographic characteristics noted above, age and WC remained significantly associated with GI, whereas smoking, alcohol, family history of type 2 DM, and

education were no longer significantly associated with GI Thus, in further multivariable logistic regression analyses considering dietary factors associated with GI, age and WC were entered as co-variates. Further, while region and sex were not significant correlates of GI status, they were included in subsequent multivariable modeling evaluating dietary factors as there are differences in opportunities for TF consumption by region, age, and sex.

Dietary Characteristics- 24hr Recall:

Men reported a total energy intake of 2564.67 kcalories (SD=1552.82) and women reported an intake of 2107.47 kcalories (SD=1045.68). The ratio of EI:BMR indicates that participants were mildly under-reporting energy intake (EI:BMR=1.48 (SD=.93) and 1.47 (SD=.77), in men and women, respectively). However, there were non-statistically significant tendencies for those with GI to under-report energy intake to a greater degree than those without GI (EI: BMR for Men= 1.51vs. 1.35, for NGs and GIs respectively, *p:* .25; for Women= 1.5 vs. 1.33, for NGs and GIs respectively, *p*: .07). For both sexes, *t*-test showed a significantly higher %E from PROT in GIs than NGs. In men, %E from CHO was significantly lower in GIs than NGs. In both sexes, there was no significant difference in the mean %E from fat between the GIs and NGs. The %E from TF was significantly higher in GI women, but not men, than NGs (Table 2).

In analyses of macronutrients separately entered in logistic regression models, the % E from protein (%E-PROT) was significantly and positively related to GI, and %E from high-sugar drinks was borderline significantly and positively associated

with GI adjusting for age, sex, region, and WC, whereas the % E from traditional food (%E-TF), %E from high fat foods, %E from carbohydrate (%E-CHO) and fat (%E-Fat) were not significant correlates of GI (Table 3). When %E-PROT was categorized into %E Indigenous-PROT and %E non-Indigenous-PROT, only %E non-Indigenous-PROT showed a borderline significant positive association with GI (OR: 1.03, *p*: .06); and %E from indigenous PROT showed no significant association with GI (OR: 1.00, *p*: .5)

In an additional logistic regression model with % E-PROT, %E-TF and %E from high-sugar drinks plus the demographic covariates, % E-PROT (OR: 1.04, *p*: .00) and %E from high-sugar drinks (OR:1.03, *p*: .03) were significantly related to higher risk of GI , while %E-TF was a significantly protective factor (OR:0.99, *p*: .05).

In other logistic regression models, with actual nutrients entered separately, adjusting for WC, age, sex, region, and total energy intake, fiber (g/d) was inversely while cholesterol (mg/d) was positively related to risk of GI, but differences only approached borderline statistical significance ($p<0.10$) (Table 3). In an additional model, including fiber (g/d), cholesterol (mg/d), and zinc (mg/d) plus WC, age, sex, region, and total k-calories, the effects of fiber (OR: .973, *p*: .13) and cholesterol (OR: 1.0004, *p*: .16) were attenuated , and zinc showed no significant association with GI (OR: 1.004, *p*: .62).The effect of fiber (g/d) remained borderline significant after controlling for protein (g/d) (OR: .97, *p*: .10).

The findings for dietary variables were not changed in additional analysis controlling for physical activity (total-MET).

Healthy Eating Index (HEI) and Canadian Food Guide:

Between those with and without GI, there was no significant difference in the mean intakes of food groups based on Canada's Food Guide to Healthy Eating (CFGHE) calculated from the 24-hr-recall. Further, the Canadian HEI scores were low in the study population but not significantly different between GI and NG men and women ($p>0.1$). In univariate analyses: for men with GI vs. NG, respectively, the mean HEI was 49.0 (SD=10.5) vs. 50.1 (SD=11.0), and for women the mean HEI was 57.0 (SD=11.6) vs. 55.5 (SD=12.9).

Further in multivariable logistic regression analyses controlling for age, sex, WC, region, and smoking (yes vs. no), there was no significant association between HEI score and glucose intolerance (OR: .99, *p*: .58).

Dietary Characteristics –FFQ:

For the FFQ traditional food items, t-tests showed no significant differences in the frequency of total TF intake per day between GI and NG groups (for men: 1.8 (SD=1.6) vs. 1.8 (SD=2.8) *p*: .98; and for women: 1.2 (SD= 1.6) vs. 1.3 (SD=1.8), *p*: .93).

There was also no significant difference in the amount of total traditional food intake (g/d) between GI and NG groups (for men: 531.7 (SD=543.6) vs. 663.8

(SD=1424), *p*: .49); and for women: 306.4 (SD=465.9) vs. 399.2 (SD=804.9), *p*: .3)

Controlling for age, WC, sex, and region no significant association was found between the frequency (OR: .95, *p*: .47) and amount (OR: .99, *p*: .22) of the consumption of traditional and market food items and glucose intolerance.

Discussion:

A high rate of obesity was observed in the current study population, much higher than rates noted for the general Canadian population (13% in men and 15% in women) [188]. Further, obesity's strong association with risk of GI in the current study suggests that the population is at considerable risk for the emergence of type 2 DM particularly as obese individuals advance in age. Further, the findings relating a lower educational level and a family history of type 2 DM with increased risk of GI are consistent with the existing literature [12, 14]. For smoking, the percent GI in nonsmokers was higher than in smokers but the difference disappeared in multivariable logistic regression controlling for other covariates.

WC was the chosen anthropometric covariate entered in the logistic regression models as it had a strong positive correlation with other adiposity measurements such as BMI, and %BF. Studies among Canadian Inuit show that the role of WC in insulin resistance is more obvious than BMI and %BF [55].The other confounders that were controlled for in the regression model were region, age, and sex; all of which could have an important effect on both glucose tolerance and

diet. Both age and WC were significantly different between GIs, and NGs of each sex group. Education was not included in the multivariable model due to concerns of over-adjusting as food choices can vary by educational status, also it did not show a significant association after controlling for other characteristics (Table2).

Based upon the 24-hr-recall data, %E PROT, and intakes of cholesterol were significant and borderline significant risk factors for GI, while fiber showed a tendency for a protective effect. Given the limitation of relying upon one 24-hr recall which would bias results toward the null, the significant associations observed between these key dietary variables and GI was notable. This result was consistent with the results from North American Aboriginal population [189]. When we separated %E from Indigenous PROT from non-Indigenous PROT we found that only non-indigenous PROT was positively associated with GI.

The existing literature on protein intake and glucose metabolism suggests that in the short-term a high protein intake may improve glucose metabolism [190], whereas in cross-sectional studies, high protein intake has been associated with glucose intolerance [98, 99] and consumption of processed meats is associated with type 2 DM risk [101, 152, 191]. In the Mannisto et al study, the positive association of processed meat intake with the risk of type 2 DM was better explained by the sodium content of processed meat than SFA, protein, cholesterol, haeme Fe, Mg and nitrate; the results were not changed by obesity [191].

On the other hand, it has been suggested that increased amino acid levels in the plasma can have effects on glucose metabolism through substrate-mediated gluconeogenesis, and to a lesser extent, through hormone-mediated effects which involve stimulating insulin and glucagon and changing the insulin/glucagon ratio [102]. An in-vivo study showed that short-term elevated amino acid in plasma was followed by impaired increase in skeletal muscle glucose 6-phosphate, reduced insulin-stimulated glucose disposal from the whole body, and decreased skeletal muscle glycogen synthesis. The study indicated that elevated amino acid levels directly inhibit muscle glucose transport and/or phosphorylation [103]. Also higher fasting glucose production, decreased suppression of hepatic glucose production, and increased gluconeogenesis, associated with a high protein diet, all can potentially contribute to the increased post absorptive demand on ß-cells, which can lead to faster development of ß-cell damage [98]. An amino acid overload was suggested to interfere with insulin signaling and lead to insulin resistance through effects related to activation of the mammalian target of rapamycin (mTOR) and ribosomal protein S6 kinase-1 in which S6K1 is over-activated, and can inhibit IRS1 activity and insulin induced class-1 Pl3K activation (negative feedback), which can lead to insulin resistance [104]. While more work is needed to elucidate mechanisms, the type of amino acids appears to be important [192]; therefore the differences in amino acid content of market food protein and traditional food protein may explain the difference observed in their associations with GI. Unfortunately little or no work has characterized the relative composition of amino acids in traditional food protein sources. However, studies

have shown that the type of protein has an important effect on glucose tolerance. In 2000 Lavigne et al. compared the fasting plasma glucose and insulin levels in rats fed isoenergetic diets containing casein, soy protein, or cod protein. They found that cod and soy protein-fed rats had higher disposal rates of glucose and therefore better insulin sensitivity than those fed with casein [193]. They tried to explain the cellular mechanism and found that cod protein regulates the Pl 3-kinase/Akt pathway and increases the translocation of GLUT4 to the T-tubules in obese high-fat-fed rats [194]. In 2007, Ouellet et al. compared the insulin sensitivity by using a hyperinsulinemic-euglycemic clamp in 19 insulin resistant humans with isocaloric diets containing cod-protein or lean beef, pork, veal, eggs, milk, and milk products (BPVEM) for 4 weeks. Both diets had similar levels of fibers and monounsaturated, polyunsaturated (including n-3), and saturated fatty acids. They found that Cod-protein fed subjects had better insulin sensitivity [192]. It is suggested that the improving effect of Cod-protein is probably due to its amino acid contents [192]. Cod protein has lower levels of branched-chain amino acids (valine, leucine, and isoleucine) and higher levels of arginine [192]. A decrease in insulin-stimulated uptake of glucose was observed in the forearm muscle which could be due to the activation of the mammalian target of rapamycin/S6K1 pathway [195]. Arginine as a substrate for nitric oxide has a role in vasodilatation that may explain the better glucose disposal [196]. Also animal studies have shown that taurine, which is higher in white fish than in pork and beef [197], might increase insulin sensitivity in insulin resistant animals by lowering protein tyrosine phosphatase and increasing protein tyrosine kinase

[198]. In Ouellet et al study they mention that although they tried to keep the n-3 PUFA content of both diets equal, by adding fish oil to the BPVEM diet, the n-3 PUFA used in the form of fish was more available than the n-3 PUFA from fish oil. Therefore, the n-3 PUFA content of fish might contribute to the observed protective effect of cod protein [192].

Also as we know traditional Arctic protein sources are associated with other nutrients important in regards to diabetes, such as Se [155], and n-3 fatty acids [141]; evaluation of which was beyond the scope of the current paper.

In a 20 year cohort a positive association was found between the cholesterol, MUFA and SFA intake, 20 yrs before diagnosis, with incidence of diabetes [199]. However as it was mentioned in other studies it is difficult to explain how cholesterol affects diabetes [89]. Diets high in glycemic load (glycemic index* amount of CHO) and low in cereal fiber was shown to be associated with increased risk of diabetes [106]. One study in college students showed that dietary fiber was negatively associated with fasting insulin levels in men and women, and was negatively associated with adiposity measurements in men [107]. Another study showed that an increase in insoluble fiber intake for 3 days increased the whole body insulin sensitivity in overweight and obese women [108].
The findings in the current report that % E from TF was associated with a lower risk for GI is compatible with an earlier report from Alaska which investigated the association between fatty acid metabolism and type-2 DM [153] and another

report from Greenland's Inuit population which showed that the intake of fruits and seal meat was negatively associated with the risk of diabetes [38]. Also in Alaskan natives higher consumption of seal oil and salmon was associated with lower risk of glucose intolerance [200].

Our study is fairly novel in showing a significant positive association between high-sugar drinks and GI, The Canadian soft drink industry uses high-fructose corn syrup 20 times more than it uses sugar as the sweetening agent [201]. There is emerging literature which shows that high fructose intake can lower the circulating insulin and leptin which can lead to weight gain and metabolic syndrome [202].

The non-significant association between the HEI score and GI status found in logistic regression is consistent with the literature which showed that the association between adherences to American guidelines, evaluated by HEI, and the risk of chronic diseases was small [203].

Limitations:

While the study population showed a mild degree of under-reporting of energy intake overall, there were tendencies for a greater degree of under-reporting among those with GI than among those without GI. As obese individuals were more likely to have GI and as obese individuals are more likely to under-report E intake [167], the tendency for under-reporting of E intake in the current study is consistent with the literature. However, as Livingstone mentions, if lower intakes

are caused by underreporting of the whole diet, micronutrient density (%E) should not be different between under-reporters and non under-reporters [204]. However, the tendency for under-reporting of E intake among those with GI illustrates the difficulty of evaluating the contribution of total energy intake to GI.

Another limitation of the dietary assessment is the reliance upon one 24-hr recall. While the methodology can provide a valuable assessment of eating patterns in a population, in smaller sub-group analyses, true differences in habitual diet between those with and without GI may be obscured as one 24-hr-recall cannot estimate usual intake [205]. Also, the estimates of dietary intakes are problematic because of variability in nutrient composition of mixed dishes and baked goods and incomplete, outdated, or substituted information from US data contained in the Canadian Nutrient File. Further, the BMR for heavier individuals may be slightly over-estimated due to a higher adipose tissue mass [206].

In addition, the traditional FFQ was based on the past year intake. FFQ like other dietary methods has random and systematic errors and its validity depends on different demographic characteristics of the participants [207].

A HEI of 51-80 shows that the diet needs improvement [173]; which is the case in this population. No significant differences in HEI scores were found between NGs and GIs of each sex group. This might be due to the limitation of not having the usual intakes, which itself is due to the lack of multiple 24-hr recalls. Having only one 24-hr recall provides large standard deviations which can increase the

risk of misclassifications. Also as Bersamin et.al [208] mention, a lower HEI does not necessarily mean that the nutrient intake is also lower; the contribution of traditional diet (ex. Fish roe as a source of Ca) to the diet quality might be missed or underestimated in HEI. Therefore the moderately low HEI in this study might either be due to a low quality diet, or high traditional food consumption. Another limitation of this study which needs to be considered is that the information for the table added salt was not provided by the questionnaires which could have led to an underestimated sodium intake and an overestimated HEI score.

Conclusion:

The findings of the current research point at the fact that lower diet quality, characterized with high intakes of cholesterol and non-indigenous protein and low intakes of fiber, and higher adiposity measurements are associated with glucose intolerance in Inuit. This can help individuals, communities and public health professionals in their efforts to prevent diabetes in Inuit communities. Weight management through physical activity and enhancements in diet quality are needed.

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Tables:

Table 1: Percent Glucose Intolerant by Demographic Characteristics and Sex: IPY Inuit Health Survey 2007-2008.

		Men		Women	
		(N)	**%GI**	**(N)**	**%GI**
Region	Nunavut	224	18.3	337	18.69
	Inuvialuit	50	30	94	14.89
	Nunatsiavut	45	13.33	63	19.05
Age	<30	66	4.55*	109	4.59*
	30-40	72	12.5	108	13.89
	>40-60	136	22.06	223	20.18
	>60	45	44.44	54	44.44
Smoking	Yes	208	15.38*	339	12.68*
	No	107	26.17	153	28.76
BMI	<30	235	14.47*	275	10.91*
	>=30	83	33.73	214	26.17
%BF	Low	186	11.29*	153	7.84*
	High	128	32.03	333	22.22
WC	Low	226	11.95*	177	9.6*
	High	88	37.5	303	22.11
Alcohol	Yes	202	14.85*	279	12.19*
	No	96	26.04	191	25.13
Education	< high school	60	28.33*	83	31.33*
	>=high school	250	16.4	406	15.02
family history of diabetes	Yes	36	27.78*	91	19.78
	No	199	13.57	284	15.49

**P < 0.05, Chi-square tests for difference in the %GI by characteristics in analyses separately conducted for men and women*

Table 2: 24-hr recall-based dietary intakes of GIs and NGs according to their sexes: IPY Inuit Health Survey 2007-2008.

		Male		P	Female		P
		NG	GI		NG	GI	
High sugar beverages (%E)		3.7 ± 7.5	4.1±8	0.7	4.5±8.7	5.6±10.4	0.32
High fat foods (%E)		36.8±21.5	35.9±25.1	0.78	37.4±20.9	36.6±19.7	0.74
High sugar foods (%E)		30.1±18.1	29.3±17.8	0.75	30.7±19.2	30.2±20.5	0.84
Zinc (mg/d)		19.1±16.5	20.1±14.3	0.67	15.0±14.4	15.9±10.9	0.6
Fiber (g/d)		11.2±9.5	7.7±6.3	0.01	10.4±7.3	8.7±6.7	0.05
cholesterol (mg/d)		467.8± 400.4	493.9± 384,7	0.65	348±315.1	377.9±274.3	0.42
% energy from:	CHO	45.0±15.8	38.1±18.3	<0.01	47.8±15.5	44.4±15.6	0.07
	Fat	32.3±11.2	33.5±11.6	0.47	32.5±11.8	32±10.6	0.72
	Pro.	21.2±10.9	27.1±13.7	<0.01	19±9	23.1±10.4	<0.01
% E from TF		18±22.4	22.7±25.5	0.17	14.8±19.6	19.7±23	0.05
% E from SFA		10.6±4.1	11.3±4.4	0.31	10.3±4.2	10.2±4.2	0.98
%E from MUFA		13±5.3	14.1±6	0.15	12.5±5.2	12.7±5.4	0.78

% E from PUFA	5.5±2.6	5.7 ±3.2	0.64	5.7±3.1	5.58±3	0.68

Table 3: Demographic and Dietary Factors evaluated for their association with glucose intolerance in multiple logistic regression models [1,2,3,4] : IPY Inuit Health Survey 2007-2008.

Models		Beta Coef	SE	P	OR	95%CI
Model1						
	Age	.05	.01	.00	1.05	1.02-1.07
	WC	.04	.01	.00	1.04	1.03-1.06
	Region					
	Nunavut				referent	
	Inuvialuit	-.19	.31	.54	.82	.44-1.53
	Nunatsiavut	-.18	.35	.61	.83	.42-1.67
	Sex (Female Vs. Male)	-.07	.26	.79	.93	.56-1.55
	Smoking (Yes vs. No)	-.05	.27	.84	.95	.56-1.6
	Alcohol (Yes vs. No)	-.33	.27	.20	.71	.42-1.2
	Education (>=HS vs. Lower)	.07	.37	.85	1.07	.52-2.21
	Family history (Yes vs. No)	.1	.29	.73	1.1	.63-1.93
Model2						
	%E from TF	-.0005	.005	.91	.99	.99-1.01
	%E from Fat	-.0017	.009	.85	.99	.98-1.02
	%E from CHO	-.007	.007	.3	.99	.98-1.01
	% E from PROT	.0203	.01	.04	1.02	1.001-1.04
	% E from high-sugar drinks	.021	.012	.07	1.021	.998-1.04
	%E from high fat foods	.001	.005	.76	1.001	.992-1.01

Model3						
	Fiber (g/d)	-.03	.02	.08	.97	.94-1.004
	Zinc (mg/d)	.008	.008	.29	1.01	.99-1.02
	Chol (mg/d)	.0006	.0003	.06	1.0006	.99-1.001
Model4						
	HEI	-.004	.009	.60	.99	.98-1.01

[1]*Model 1 includes all demographic characteristics listed.*

[2]*Model 2 includes each dietary variable separately with age, sex, WC, and region*

[3]*Model 3 includes each nutrient separately with age, sex, waist circumference, region, and total kilocalories*

[4]*Model 4 includes HEI (healthy eating index, Canadian version [173]) with age, sex, WC, and region.*

11. Conclusion:

In summary the findings of this study provided us with some perspectives of the diet quality and lifestyle characteristics associated with newly identified glucose intolerance among Inuit residing in the Canadian Arctic. Obesity and advancing age were the two primary risk factors for GI in the current thesis investigation. One paradoxical finding was that a greater percent of women were obese and had high %BF than men, yet men and women had similar rates of GI. One possible explanation is that for the same BMI women carry more fat than men. Further research is needed to compare the hip circumferences, which is a good indicator of subcutaneous fat that is associated with protective effects against IR and GI. The data from the 24-hr recall found that glucose intolerance was positively associated with cholesterol and %E from non-indigenous PROT, and was negatively associated with fiber intake. This suggests that GIs had higher intakes of processed meat and lower intake of fiber containing fruits, vegetables and grains. The overall dietary factors associated with GI were related to the characteristics of an unhealthy Western diet. Although the total HEI was not significantly different between the GI groups, the findings support our hypothesis that glucose intolerant Inuit have lower diet quality and lower reliance on TF intake. Future research is needed to evaluate the Toenail Se and Serum n-3FA levels and type of AA in TF.

There is a need for education emphasizing dietary and other behavioral changes. The dietary education for decreasing the intake of high-sugar beverages should be combined with strategies to increase the accessibility to healthy market foods

(such as fruits and vegetables). Dietary education promoting a higher TF intake should be followed by educational programs about TF production.

Finally, the thesis contributes to the newly emerging literature suggesting that excessive processed meat consumption may have deleterious effects on insulin resistance and raises concerns regarding the potential long-term consequences of the popular high-protein diet.

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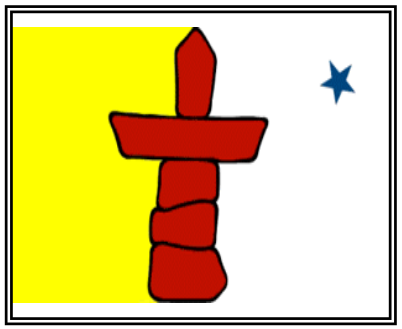
Appendixes



cspn3is2 `Nnstz
STUDY NO.

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INT. NO.

Qanuipitali? How about us, how are we?

*iE/s9lxb3X5gk8 xWd`y6*  *wMz !: wkw5 ieq8*  xW3h3`g2 _ W/E3ymJ5 xWd`y5	**Inuit Health Survey 2007** *FOOD FREQUENCY QUESTIONNAIRE* *PART 1: COUNTRY FOOD* *PART 2: MARKET FOOD* Interviewer-Completed Questionnaire

Centre for Indigenous People' Nutrition and Environment
Macdonald Campus of McGill University
21,111 Lakeshore Rd
Ste-Anne-de-Bellevue, (QC) H9X 3V9

W/E3bsizb s9lz

yea/6

Completion Date: ___/____/2007
m d y

Starting Time: ____/____
h m

wMz !: wkw5 ieq8

Part 1: Country Food

NINw3ymJ6 s9lw5 s9l6 WNhxDy6 be6 czf5 Legend: # = number, D=day, W=week, M=month, S=season	**xCisMs3g6 iE/sMscV** Was it eaten in the past year?			**cktQsXl2X2X** Frequency		**cltQ iElx`ha=5** Usual serving			Office use only
wkw5 ieq8 Country Food	**`w** Yes or **`xZ** No GNINw3IAH (circle)	**cz`fiz** Season	✓	#	**s9lz xCAz bez czf5** D / W / M / S	c5txt3gt5 # of servings	cktQ xqJu4 serving model	Thickness	sdmw8i z Weight
wm3usb5 i3J`t5 / Sea mammals									
1. eMlZ2 iez Gk`bui6, `symJ6 s}?`li8 dx6H Beluga meat (fresh, cooked or frozen)	`w `xZ Y N	IN season							
		OFF season							
2. eMlZ2 iez i4f Beluga, dried	w `xZ Y N	IN season							
		OFF season							
3. m`b6 s3hc3gi G`symqg6 s}?`li8 `symJ6H Beluga muktuk with blubber (raw or boiled)	w `xZ Y N	IN season							
		OFF season							
4. m`b6 s3hcCi G`symqg6 s}?`li8 `symJ6H Beluga muktuk without blubber (raw or boiled)	w `xZ Y N	IN season							
		OFF season							
5. eMlZ2 s3hz G`symqg6 s}?`li8 `symJ6H Beluga blubber (raw or cooked)	w `xZ Y N	IN season							
		OFF season							
6. eMlZ2 s3h3z s}?`li8 uyC6 Beluga oil or misirak	w `xZ Y N	IN season							
		OFF season							
7. `gZo2 eMlZ2 iez Gk`bui6, `symJ6 s}?`li8 dx6H Narwhal meat (fresh, cooked or frozen)	w `xZ Y N	IN season							
		OFF season							

NlNw3ymJ6 s9lw5 s9l6 WNhxDy6 be6 czf5 Legend: # = number, D=day, W=week, M=month, S=season	**xCisMs3g6 iE/sMscV** Was it eaten in the past year?			**cktQsXl2X2X** Frequency		**cltQ iElx`ha=5** Usual serving			Office use only
wkw5 ieq8 Country Food	**`w** Yes or **`xZ** No GNlNw3lAH (circle)	**cz`fiz** Season	✓	#	**s9lz xCAz bez czf5** D / W / M / S	c5txt3gt5 # of servings	cktQ xqJu4 serving model	Thickness	sdmw8iz Weight
8. gZo2 s3hz G`symqg6 s}?`li8 `symJ6H Narwhal blubber (raw or cooked)	w `xZ Y N	IN season							
		OFF season							
9. gZo2 m5`bz s3hc3gi G`symqg6 s}?`li8 `symJ6H Narwhal muktuk with blubber (raw or boiled)	w `xZ Y N	IN season							
		OFF season							
10. gZo2 m5`bz s3hcCi G`symqg6 s}?`li8 `symJ6H Narwhal muktuk without blubber (raw or boiled)	w `xZ Y N	IN season							
		OFF season							
11. N5t6, s3hc3gi G`symJ6 s}?`li8 `symqg6H Ringed seal, blubber (raw or boiled)	w `xZ Y N	IN season							
		OFF season							
12. N5ts2 taz G`symJ6 s}?`li8 `symqgH Ringed seal, liver (raw or cooked)	w `xZ Y N	IN season							
		OFF season							
13. N5ts2 iez G`symqg6, symJ6 s}?`li8 dx6H Ringed seal, meat (raw, cooked or frozen)	w `xZ Y N	IN season							
		OFF season							
14. xw=s2 s3hz G`symqg6, `symJ6, wAN6H Walrus blubber (raw, cooked, aged)	w `xZ Y N	IN season							
		OFF season							
15. xw=s2 iez G`symqg6, `symJ6, wAN6H Walrus meat (raw, boiled, aged)	w `xZ Y N	IN season							
		OFF season							
kNusb5 i3J`t5 / Land animals									

NINw3ymJ6 s9lw5 s9l6 WNhxDy6 be6 czf5 Legend: # = number, D=day, W=week, M=month, S=season	**xCisMs3g6 iE/sMscV** Was it eaten in the past year?			**cktQsXl2X2X** Frequency		**cltQ iElx`ha=5** Usual serving			Office use only
wkw5 ieq8 Country Food	**`w** Yes or **`xZ** No GNlNw3lAH (circle)	**cz`fiz** Season	✓	#	**s9lz xCAz bez czf5** D / W / M / S	c5txt3gt5 # of servings	cktQ xqJu4 serving model	Thickness	sdmw8iz Weight
16. g5`g2 iez G`symqg6, yCymJ6, `symJ6, `sJoxEymJ6, i4f, wAN6H Caribou meat (raw, baked, cooked, boiled, aged)	w `xZ Y N	IN season							
		OFF season							
17. g5`g2 iez Gi4fH Caribou meat (dried)	w `xZ Y N	IN season							
		OFF season							
18. g5`g2 taz G`symqg6, yCymJ6, `symJ6H Caribou liver (raw, baked, cooked)	w `xZ Y N	IN season							
		OFF season							
19. g5`g2 `smtz G`symqg6, `sJoxEymJ6H Caribou heart (raw, boiled)	w `xZ Y N	IN season							
		OFF season							
20. g5`g2 scz G`symqg6, `symJ6H Caribou tongue (raw, cooked)	w `xZ Y N	IN season							
		OFF season							
21. g5`g2 xexDz GenDxz, wlq8H Caribou stomach (walls, contents)	w `xZ Y N	IN season							
		OFF season							
22. g5g2 b3gz G`symqg6, `sJoxEymJ6H Caribou kidney (raw, boiled)	w `xZ Y N	IN season							
		OFF season							
23. N`k2 iez G`symqg6, `symJ6H Polar bear, meat (raw, boiled)	w `xZ Y N	IN season							
		OFF season							

NlNw3ymJ6 s9lw5 s9l6 WNhxDy6 be6 czf5 Legend: # = number, D=day, W=week, M=month, S=season	**xCisMs3g6 iE/sMscV** Was it eaten in the past year?			**cktQsXl2X2X** Frequency		**cltQ iElx`ha=5** Usual serving			Office use only
wkw5 ieq8 Country Food	**`w** Yes or **`xZ** No GNlNw3lAH (circle)	**cz`fiz** Season	✓	#	**s9lz xCAz bez czf5** D / W / M / S	c5txt3gt5 # of servings	cktQ xqJu4 serving model	Thickness	sdmw8iz Weight
24. svos2 iez G`symqg6, `symJ6H Rabbit meat (raw, cooked)	w `xZ Y N	IN season							
		OFF season							
wcl4 / Fish									
25. wcl`W, iez G`symqg6, `sJoxEymJ6, dx6, ifH Arctic char, meat (raw, boiled, frozen, dried)	w `xZ Y N	IN season							
		OFF season							
26. Nb3N6 Halibut	w `xZ Y N	IN season							
		OFF season							
27. coCo4 Turbot	w `xZ Y N	IN season							
		OFF season							
28. frs/5 Mussels	w `xZ Y N	IN season							
		OFF season							
29. sh5 Gx7`jmJ5H Clams *Circle serving given: with shells OR without shells*	w `xZ Y N	IN season							
		OFF season							
30. raX5 Shrimp	w `xZ Y N	IN season							
		OFF season							
t7ux8 / Birds									
31. xe`Q5 Ptarmigan	w `xZ Y N	IN season							
		OFF season							

NINw3ymJ6 s9lw5 s9l6 WNhxDy6 be6 czf5 Legend: # = number, D=day, W=week, M=month, S=season	**xCisMs3g6 iE/sMscV** Was it eaten in the past year?			**cktQsXl2X2X** Frequency		**cltQ iElx`ha=5** Usual serving			Office use only
wkw5 ieq8 Country Food	**`w** Yes or **`xZ** No GNlNw3lAH (circle)	**cz`fiz** Season	✓	#	**s9lz xCAz bez czf5** D / W / M / S	c5txt3gt5 # of servings	cktQ xqJu4 serving model	Thickness	sdmw8iz Weight
32. i3`o5 Canada goose	w `xZ Y N	IN season							
		OFF season							
33. u`t5 Eider duck	w `xZ Y N	IN season							
		OFF season							
34. m8iq8 i3`o5 x7m u`t5 Eggs of goose or eiderduck	w `xZ Y N	IN season							
		OFF season							
WD3g5 / Plants									
35. rAbq3N8, X3z8, r7u8Nw8, xyq9l ki?5bs`h5 Blueberries, crowberries, cranberries, other picked berries	w `xZ Y N	IN season							
		OFF season							
36. crN3g5 WD3g5 Sour leaves	w `xZ Y N	IN season							
		OFF season							
37. fx8`i5 Welk (seaweed)	w `xZ Y N	IN season							
		OFF season							
38. In general, what is your preferred way of eating country food? 1- Raw (frozen, dried? 2- Cooked (boiled, fried, dried?									

wMz @: is=xn5 i`e5

Part 2: Market Food

Legend: # = number, D=day, W=week, M=month NlNw3ymJ6 s9lw5 s9l6 be6	**xCisMs3g6 iE/sMscV** Was it eaten in the past month?	**cktQsXl2X2X** Frequency		**cltQ iElx`ha=5** Usual serving			Office use only
is=xn5 i`e5 x7m wuCnw5 Market Foods and Beverages	` **w** Yes or **`xZ** No GNlNw3lAH (circle)	#	**s9lz xCAz bez** D/W/M	c5txt3gt5 # of servings	cktQ xqJu4 serving model	Thickness	sdmw8i z Weight
wuCn5 / Beverages							
38. wuZ5 Soft drinks ***NlNw3lA iDx3X5bw5*** */Circle your usual choice:* wuZgw8N6 s}?`l nl5tnsto4 Regular or Diet	w `xZ Y N						
39. Xi3g5 wuCnw5 WcystlA nNym5bstQJ5 S3ymJ5 wuZn5, `t5 nNym5bstQJ5, GwuZn5 Xi3g5 x7m S3ym5bstQJ5 wuZn5H Powdered drinks including fruit drinks/ Sports drinks, iced tea, (Tang, punch, Kool-Aid, Sunny D, Gatorade)	w `xZ Y N						
40. wMymq5g5 wuZn5 GxoA3`ug5 s}?`li8 dx5H Real fruit juice (bottled or frozen)	w `xZ Y N						
41. w7j4 GwuCn6 s}?`li8 Xi3g6H Milk (fresh or powdered) ***NlNw3lA iDx3X5bw5*** */Circle your usual choice:* w7j9ME4 whole, 2%, 1%, Xi3g6 skim	w `xZ Y N						
rhgw8Nw8 / Miscellaneous							
42. Xbwg y2{, `yy`o5 Chips/crisps/cheese puffs	w `xZ Y N						

d/8N`u4U

Ending Time: ____/____
h m

xW3h3bst9lA scsy6 xg3b6 GNlNw3lAH / Interview language (circle):

wk5tg5 / Inuktitut

c9l`Ntg5 / English

wk5tg5 x7m c9l`Ntg5 / Inuktitut and English

d/8N`u4

Thank you!

ADULT INFORMED CONSENT – INFORMATION SHEET

Title of Research Project: Inuit Health Survey *"Qanuippitali: How about us, how are we?"*

Funded by Government of Canada Program for International Polar Year, Canadian Institutes for Health Research and Northern Contaminants Program

Steering Committee members:

Name	**Organization**	**Telephone #**
Dr. Grace Egeland	Principal Investigator, McGill University/CINE	(514) 398-8642
Dr. Isaac Sobol	Co-Principal Investigator, Government of Nunavut, DHSS	(867) 975-5700
Dr. Geraldine Osborne	Co-Principal Investigator, Government of Nunavut, DHSS	(867) 979-5700
Dr. Kue Young	Co-Principal Investigator, University of Toronto	(416) 978-6459
Lynda Gunn	Nunavut Association of Municipalities(NAM)	(867) 979-3111
Laakkuluk Williamson	Nunavut Tunngavik Incorporated (NTI)	(867) 975-4900

ᐃᓐᓇᕐᓂᑦ ᖃᐅᔨᑎᑦᓯᔪᑎ ᐊᖏᕈᑎᐅᑉ ᒥᒃᓵᓄᑦ - ᑐᑭᓯᒋᐊᕈᑎᐅᓪᓗᓂ

ᖃᐅᔨᓴᖅᑕᐅᔪᑎᐅᑉ ᑕᐃᔫᓯᖓ: ᐃᓄᐃᑦ ᐃᓄᑦᓯᐊᕐᓂᕐᒧᑦ ᖃᐅᔨᓴᖅᑕᐅᓂᖓᑦ - *ᖃᓄᐃᑉᐱᑕᓕ? ᐅᕙᑦᑎᓪᓗ, ᖃᓄᐃᑉᐱᑕ?*

ᑮᓇᐅᔭᖅᑲᖅᑎᑕᐅᓪᓗᑎ ᒐᕙᒪᒃᑯᖏᓐᓂᑦ ᑲᓇᑕᐅᑉ ᓯᓚᕐᔪᐊᕐᒥ ᐅᑭᐅᖅᑕᖅᑐᓂᕐᒧᑦ ᐊᕐᕋᒍᖅᑲᖅᑎᓪᓗᒍ ᖃᐅᔨᓴᕈᑎᖅᑲᖅᑎᓪᓗᒋᑦ ᐃᓅᓯᓕᕆᓂᕐᒧᑦ/ᐋᓐᓂᐊᖅᑲᖅᓇᖏᑦᑐᓕᕆᓂᕐᒧᓪᓗ

ᐸᕐᓇᐃᔨᑦ ᑲᑎᒪᔨᕋᓛᖏᑦ:

ᐊᑎᖓ:	**ᓇᑭᙶᕐᓂᖓ**	**ᐅᖄᓚᐅᑎᖓ:**
ᑑᑦᑕᖅ ᒍᐃᔅ ᐃᕐᓛᓐ	ᐊᖏᔪᖅᑳ ᖃᐅᔨᓴᖅᑎᓂ, ᓯᓚᑦᑐᓴᕐᕕᖓᓂ ᒪᒋᐅᓪ	(514) 398-8642
ᑑᑦᑕᖅ ᐊᐃᓴᒃ ᓱᐳᓪ	ᐊᖏᔪᖅᑳᖑᖅᑲᑎᖓ ᖃᐅᔨᓴᖅᑎᐅᑉ, ᓄᓇᕗᒻᒪᒃᑯᖏᓐᓂᑦ ᐋᓐᓂᐊᖅᑲᖅᓇᖏᑦᑐᓕᕆᔨᒃᑯᓐᓂᑦ ᐃᓄᓕᕆᔨᒃᑯᓐᓂᑦ	(867) 975-5700
ᑑᑦᑕᖅ ᔨᐅᕆᑏᓐᐋᕗᐊᓐ	ᐊᖏᔪᖅᑳᖑᖅᑲᑎᖓ ᖃᐅᔨᓴᖅᑎᐅᑉ, ᓄᓇᕗᒻᒪᒃᑯᖏᓐᓂᑦ ᐋᓐᓂᐊᖅᑲᖅᓇᖏᑦᑐᓕᕆᔨᒃᑯᓐᓂᑦ ᐃᓄᓕᕆᔨᒃᑯᓐᓂᑦ	(867) 975-5700
ᑑᑦᑕᖅ ᑯ ᔭᖕ	ᐊᖏᔪᖅᑳᖑᖅᑲᑎᖓ ᖃᐅᔨᓴᖅᑎᐅᑉ, ᓯᓚᑦᑐᓴᕐᕕᖓᓂ ᑐᕌᓐᑐ	(416) 978-6459
ᓖᓐᑕ ᒐᓐ	ᓄᓇᕗᒻᒥ ᑲᑐᔾᔨᖃᑎᒌᖕᒋᑦ ᓄᓇᓕᕆᔨᒃᑯᖏᓐᓄᑦ	(867) 979-3111
ᓛᑯᓗᒃ ᐅᐃᓕᐊᒻᓴᓐ	ᓄᓇᕗᑦ ᑐᓐᖓᕕᒃᑯᓐᓂᑦ	(867) 975-4900

1. Description of the Survey:

- You are invited to participate in a comprehensive Nunavut-wide Inuit Health Survey taking place this summer.

- Your participation in the Survey is voluntary. You may withdraw from the study, at any time without any consequence. And, you will continue to have access to the health care you are entitled to at any health care institution or health clinic you attend.

- Your participation in the Inuit Health Survey indicates that:
  - you are interested in knowing the state of your personal health; And
  - you are also interested in making health and wellness better for all Inuit in Nunavut.

We would like you to feel empowered by this process and for you to know that we are honored to have you participate.

2. How the survey came about:

- In 2004, a Health Survey called “Qanuippitaa? How are we?” took place in Nunavik. A similar survey Qanuippitali? How about us, how are we? took place last fall in 18 communities in Nunavut. This summer the health survey will take place in Inuvialuit, Nunavut and Nunatsiavut.

- The information collected in Nunavut this summer through the Inuit Health Survey, *“Qanuippitali? How about us, how are we?”* will help compare health indicators for different regions in which Inuit live in the Circumpolar North. As well, the study will allow us to learn how Inuit adapt and thrive in the face of change.

3. What is the Purpose of the Survey?

- To gain a better understanding of the health status of Inuit in Nunavut and the factors that contribute to Inuit resiliency.
- To capture the many aspects of life that play a role in Inuit health and wellness including the extent of food availability, household crowding and social support and how these factors effect the well-being of Inuit.
- To look at factors that deal with suicidal thoughts, alcohol, drug abuse, history of violence and sexual abuse.
- To look at dietary habits, environmental exposures, and physical activity.
- To look at the commonness of a type of infection that is related to stomach ulcers and other types of infection that come from animals and marine mammals.
- To find out if different types of chemicals and fat in blood can tell us about risks for heart disease and diabetes.

1. **ᑭᓲᒪᖕᒐᖅ ᖃᐅᔨᓴᖅᑕᐅᒍᑎ:**

- ᐃᓚᐅᔪᓐᓇᕐᑯᓪᓗᑎᑦ ᓄᓇᕗᒻᒥᐅᓄᑦ ᖃᐅᔨᓴᖅᑕᐅᒍᑎᖃᖅᑐᖃᖅᑎᓪᓗᒍ ᐃᓄᐃᑦ ᐃᓅᓯᖏᓐᓂᒃ ᖃᓄᐃᒻᒪᖔᑕ ᐊᐅᓚᐅᓂᐊᖅᑐᒥ.
- ᐃᓚᐅᒍᒪᓐᓂᕈᑦᑎᑦ ᐃᓚᐅᕆᐊᑐᖏᑦᑐᑎᑦ. ᐃᓚᐅᔪᒪᒍᓐᓃᕐᓂᕈᑦᑎᑦ ᖃᖕᒐᑐᐃᓐᓇᖅᓯᐊᒃᑯᑦ ᐃᓚᐅᖏᒍᓐᓇᖅᑐᑎᑦ. ᖃᓄᐃᒍᑎᕆᔾᔭᖏᑕᐃᑦ ᐋᓐᓂᐊᖅᑲᕐᓇᖏᑦᑐᓕᕆᓂᕐᒧᑦ ᐱᔨᑦᓯᑕᐅᒍᑎᑎᑦ.
- ᐃᓚᐅᓂᕆᔭᖅᑕᐃᑦ ᑕᐃᒪ ᐃᓄᐃᑦ ᐃᓅᓯᖏᓐᓂᒃ ᖃᐅᔨᓴᖅᑕᐅᓂᕐᒧᑦ:
- ᑐᑭᓯᒍᒪᓂᖅᑲᕋᑦ ᑭᓯᐊᓂ ᖃᓄᐃᒻᒪᖔᖅᐱᑦ
- ᖃᓄᐃᑦᓴᖏᓐᓇᕙᓪᓗ ᐃᓅᓯᖃᑦᓯᐊᕐᓂᕐᒥᑦ ᐋᓐᓂᐊᖅᑲᕐᓇᖏᑦᑐᓕᕆᓂᕐᒥᑦ ᐃᓄᓐᓄᑦ ᓄᓇᕗᒻᒥ ᐱᕋᓪᓚᕐᑯᓪᓗᒍ.

ᐃᓚᐅᓂᕆᔭᐃᑦ ᐊᑦᓯᐊᓗᒃ ᐅᐱᒋᔭᕙᑦ ᐱᕙᓪᓚᖁᑎᐅᕐᑯᔭᕋᓗᐊᕙᓪᓗ.

2. **ᖃᓄᖅ ᖃᐅᔨᓴᕐᓂᖅ ᓴᖅᑭᓚᐅᕐᒪᖔᑦ:ᖃᓄᖅ ᐱᕆᐊᕐᓂᕐᒪᖔᖅ ᖃᐅᔨᓴᕈᑎᐅᓂᐊᖅᑐᖅ:**

- 2004-ᖑᑎᓪᓗᒍ, ᐊᓐᓂᐊᖅᑲᕐᓇᖏᑦᑐᓕᕆᓂᕐᒧᑦ ᖃᐅᔨᓴᕐᓂᖅ ᑕᐃᔭᖅ "ᖃᓄᐃᑉᐱᑖ?" ᐱᕆᐊᖅᑐᒥᓂᖅ ᓄᓇᕕᒻᒥ. ᐊᔾᔨᒐᓚᖕᒐ ᖃᓄᐃᑉᐱᑕᓕ? ᐅᕙᒍᓪᓕ, ᖃᓄᐃᑉᐱᑕᓕ? ᖃᐅᔨᓴᕐᓂᖅ ᐅᑭᐅᓵᖑᓚᐅᖅᓯᒪᔪᖅ ᐊᕿᓂ ᐱᕆᐊᓚᐅᖅᓯᒪᔪᖅ ᖁᓕᑦ ᓯᑕᒪᐅᔪᖅᑐᓪᓗ ᓄᓇᓕᓐᓂ ᓄᓇᕗᒻᒥ. ᐊᐅᔭᐅᓂᐊᖅᑐᖅ ᐋᓐᓂᐊᖅᑲᕐᓇᙱᑦᑐᓕᕆᓂᕐᒧᑦ ᖃᐅᔨᓴᕐᓂᖅ ᐱᕆᐊᕈᓖᖅᑐᖅ ᐃᓄᕕᐊᓗᒻᒥ, ᓄᓇᕗᒻᒥ ᓄᓇᑦᓯᐊᕗᒻᒥᓗ.

- ᖃᐅᔨᓴᖅᑕᐅᒍᑕᐅᔪᑦ ᓄᐊᑕᐅᔪᑦ ᓄᓇᕗᒻᒥ ᐊᐅᔭᐅᓚᖕᒐᔪᖅ ᐃᓄᐃᑦ ᐋᓐᓂᐊᖅᑲᕐᓇᖏᑦᑐᓕᕆᓂᕐᒧᑦ ᖃᐅᔨᓴᖅᑕᐅᓂᖏᓐᓂ, "ᖃᓄᐃᑉᐱᑕᓕ? ᐅᕙᒍᓪᓕ, ᖃᓄᐃᑉᐱᑕᓕ?" ᖃᐅᔨᓴᕈᑕᐅᓂᐊᖅᑐᑦ ᖃᓄᖅ ᐊᔾᔨᒌᙱᓐᓂᖃᐅᖅᑐᑦ ᓄᓇᓖᑦ ᐊᕕᑦᑐᖅᓯᒪᔪᓂ ᐃᓄᐃᑦ ᓇᔪᖅᑕᖏᓐᓂ ᐅᑭᐅᖅᑕᖅᑐᓕᒫᒥ. ᐊᒪᑦᑕᐅᖅ, ᖃᐅᔨᓴᕐᓂᖅ ᐅᕙᑦᑎᓐᓂ ᐃᓕᑉᐹᓪᓕᑎᑦᓯᓂᐊᕐᒥᔪᑦ ᖃᓄᖅ ᐃᓄᐃᑦ ᐃᓕᑦᑐᕆᓐᓇᕐᓂᖏᓐᓂᒃ ᐃᓅᓯᐊᕈᓐᓇᕐᓂᖏᓐᓂᓪᓗ ᐊᓯᔾᔨᕙᓪᓕᐊᔪᒥ.

3. **ᑭᓲᕙ ᐱᔾᔪᑎᐅᓗᐊᖅᑐᖅ ᖃᐅᔨᓴᕈᑎ?**

- ᑐᑭᓯᐊᔭᐅᕐᑯᓪᓗᒋᑦ ᐋᓐᓂᐊᖅᑲᕐᓇᖏᑦᑐᓕᕆᓂᕐᒧᑦ ᐃᓄᐃᑦ ᖃᓄᖅ ᐊᓯᔾᔨᕝᕙᓪᓕᐊᒐᓗᐊᖅᑎᓪᓗᒋᑦ ᐃᓅᓯᑦ ᐱᔪᓐᓇᓚᐅᕐᒪᖔᑕ.
- ᐃᓅᓯᕆᔭᖏᓐᓂᒃ ᐃᓄᐃᑦ ᐃᓅᓯᖃᑦᓯᐊᕐᓂᖏᑦ ᑲᒪᑦᓯᐊᕐᓂᖏᑦ ᓂᕿᖏᑦ, ᐃᓪᓗᕿᑦᓴᕋᓗᐊᖅᑐᑎᒃ ᐃᑲᔪᖃᑎᒌᑦᓯᐊᕈᓐᓇᕐᓂᖏᑦ ᖃᓅᒻᒪᖔᑕ ᐃᓄᐃᑦ ᐃᓅᓯᖏᓐᓂᒃ.
- ᖃᐅᔨᒐᓱᐊᕐᓗᑎᓪᓗ ᑭᓱᓄᑦ ᐃᒻᒥᓂᕐᓂᕐᒧᑦ ᐃᓱᒪᖃᓕᖅᑲᑦᑕᕐᒪᖔᑕ, ᐊᑐᖅᑕᐅᓂᖏᓪᓗ ᐃᒥᐊᓗᒃ, ᐊᖕᒑᔭᕐᓇᖅᑐᑦ, ᓂᖕᒐᕐᓂᑦ ᐊᒻᒪᓗ ᖁᓄᔪᕐᓂᐊᕐᓃᑦ.
- ᖃᐅᔨᕆᐊᕐᓗᑎᓪᓗ ᓂᕿᑭᔭᐅᕙᑦᑐᓂᒃ, ᐊᕙᑎᕆᕙᑦᑕᖏᓐᓂᓪᓗ ᐊᒻᒪᓗ ᐃᓅᓯᖏᓐᓂᒃ ᓱᐃᑏᕿᒍᑎᐅᖃᑦᑕᖅᑐᓂᒃ.
- ᖃᐅᔨᒐᓱᐊᕐᓗᑎᓪᓗ ᑭᓱᓄᑦ ᐊᕿᐊᕈᓪᓗᓕᖅᑲᑦᑕᕐᒪᖔᑕ ᐊᒻᒪᓗ ᐊᓯᖏᓐᓂᒃ ᐊᑲᐅᖏᔾᔪᑎᖃᖃᑦᑕᕐᒪᖔᑕ ᐆᒪᔪᕐᓂᑦ ᐊᒻᒪᓗ ᐳᐃᔾᔪᓐᓂᑦ.
- ᖃᐅᔨᒐᓱᐊᕐᓗᑎᓪᓗ ᐅᖅᓱᖃᓗᐊᖃᑦᑕᕐᓂᖏᑦ ᐊᐅᖕᒐᒍᑦ ᐆᒪᓯᓇᓕᓴᐃᒪᖔᑕ ᐅᕝᕙᓘᓐᓃᑦ ᓱᑲᖃᓗᐊᕈᑎᐅᖃᑦᑕᕐᒪᖔᑕ.

All the information collected in the Survey will help:

- plan for future health care service delivery and health promotion efforts;
- help to develop Inuit-specific prevention and intervention strategies to delay or prevent diabetes and heart disease; And
- look at possible ways to help people deal with the rapid changes that are occurring in Arctic communities.

4. Am I being asked to participate just this one time?

- There are two components to the Survey:

  FIRST, you are being asked to participate in a one-time Health Survey. Your participation will involve:
    - a Food Security questionnaire through a home visit; and
    - a clinic appointment on the ship when it arrives in your community.

  SECOND, if you agree to participate in the one-time Health survey, you will also be asked for:
    - a follow-up within a 7 year period where you will become part of the International Inuit Health Study; For those participating in the follow-up, a nurse will review your medical record to verify if you have a chronic disease now and to review if any chronic disease developments have occurred within a 7 year follow-up.

5. What is the purpose of the International Inuit Health Study?

- It is to compare results with similar surveys in other Inuit regions in Circumpolar countries; and
- Because some diseases that appear over time are not common, it will be beneficial to join data from different regions and health surveys in order to look at possible factors that may be leading to diseases or conditions.

6. How was my home selected? How was I selected?

- Your home was selected randomly from a list of homes in your community and your participation is voluntary.

ᑐᑭᓯᒃᑲᓐᓂᕐᓂᐊᕐᒪᑕ ᖃᐅᔨᓴᕐᓂᒃᑯᑦ:

- ᓯᕗᓂᒃᓴᒧᑦ ᐋᓐᓂᐊᖅᑲᓐᓇᖏᑦᑐᓕᕆᓂᕐᒧᑦ ᐱᔨᑦᑎᕈᑎᐅᕙᑦᑐᓄᑦ ᐊᒻᒪᓗ ᐋᓐᓂᐊᖅᑲᓐᓇᖏᑦᑐᓕᕆᓂᕐᒧᑦ ᑐᑭᓯᒋᐊᕈᑎᒃᓴᓂᒃ ᐃᓅᓯᖅᑳᓯᐊᕋᓱᕐᓂᕐᒧᑦ ᑕᑯᑦᓴᐅᑎᒃᓯᒃᑲᓐᓂᕐᓗᑎᒃ.
- ᐋᖅᑭᑦᓱᐃᒍᒪᓪᓗᑎᓪᓗ ᐃᓄᓐᓄᑦ ᑐᕌᖓᔪᓂᒃ ᖃᓄᐃᖏᑦᓯᐊᕐᓂᕐᒧᑦ ᐃᓅᓯᖅᑳᓯᐊᕐᓂᕐᒧᑦ ᑲᒪᑦᓯᐊᕐᓂᒧᑦ ᐸᕐᓇᒍᒪᓪᓗᑎᒃ ᐅᒻᒪᓯᓇᑦᑕᐃᓕᕐᒧᑦ ᐊᒻᒪᓗ ᓱᑲᖅᑲᓗᐊᑦᑕᐃᓕᓂᕐᒧᑦ.
- ᐊᓯᔾᔨᖅᐸᓪᓕᐊᒍᑎᐅᔪᑦ ᓱᒃᑲᓕᔪᐊᓘᒻᒪᑕ ᐅᑭᐅᖅᑕᖅᑐᒥ ᐃᑲᔪᕈᑎᒃᓴᓂᒃ ᖃᓄᖅᑑᕈᒪᒻᒪᑕ.

4. **ᐊᐱᕆᔾᔪᑎᐅᓴᕗᖓ ᐊᑕᐅᓯᐊᑐᐃᓐᓇᕐᓗᖓ?**

- ᒪᕈᓕᖅᑳᖓᔪᑦ ᖃᐅᔨᓴᐅᑎᐅᓂᐊᖅᑐᑦ:

ᓯᕗᓪᓕᖅ: ᐊᑕᐅᓯᐊᑐᐃᓐᓇᕐᓗᑎᑦ ᖃᐅᔨᓴᖅᑕᐅᓂᖃᕐᓂᐊᖅᑐᑎᑦ. ᐃᓚᐅᓗᑎᑦ ᓂᕿᒋᖃᑦᑕᖅᑕᕝᑦ ᒥᒃᓵᓄᑦ ᐊᖏᕋᓐᓂ ᐊᒻᒪᓗ ᖃᐅᔨᓴᖅᑐᐅᓗᑎᑦ ᐅᒥᐊᕐᔪᐊᒥ ᑎᑭᑉᐸᑦ ᓄᓇᑦᓯᓐᓄᑦ.

ᐊᐃᑉᐸᖓ: ᐊᖏᖅᑐᕝᓂᐅᒍᕝᑦ ᓯᕗᓪᓕᕐᒥ ᐊᑕᐅᓯᐊᖅᑕᐅᒍᒪᓗᑎᑦ ᑕᐃᒪ ᖃᐅᔨᓴᖅᑕᐅᒃᑲᓐᓂᒍᒪᒐᔭᖅᑐᑎᑦ ᐊᕐᕋᒍᐃᑦ 7 ᐊᓂᒍᓚᐅᖅᑎᓪᓗᒋᑦ. <u>**ᓯᓚᕐᔪᐊᓕᒫᒥ ᐃᓄᐃᑦ ᐃᓅᓯᖏᓐᓂᒃ ᖃᐅᔨᓴᖅᑕᐅᓂᖃᓕᕐᒥᐸᑕ**</u>;
ᑕᐃᒃᑯᐊ ᖃᐅᔨᓴᖅᑕᐅᒃᑲᓐᓂᕈᒪᒐᔭᖅᑐᑦ ᕿᒥᕐᕈᔭᐅᑦᓯᐊᕆᐊᖃᕋᔭᖅᑐᑦ ᖃᓄᐃᒍᑎᒋᓯᒪᓕᖅᑕᖏᑦ ᐋᓐᓂᐊᓯᐅᖅᑎᒧᑦ. ᐋᓐᓂᐊᖃᓕᖅᑐᒥᓂᐅᓐᓂᕈᓂ ᑖᒃᑯᐊ ᐊᕐᕌᒍᐃᑦ 7 ᐊᑐᓚᐅᖅᑕᖏᑦ ᖃᓄᐃᑉᐹᓕᖅᓯᒪᒻᒪᖕᖔᖅ ᖃᐅᔨᓴᖅᑕᐅᒃᑲᓐᓂᕈᒪᒐᔭᖅᑐᖅ.

5. **ᑭᓱᒥᒃ ᐱᔾᔪᑎᖃᕐᒪᑦ ᓯᓚᕐᔪᐊᓕᒫᒥ ᐃᓄᐃᑦ ᐃᓅᓯᖏᓐᓂᒃ ᖃᐅᔨᓴᖅᑕᐅᓂᖃᓕᕐᒥᐸᑕ?**

- ᖃᐅᔨᓴᐅᑎᒥᓂᑦ ᐊᓯᖏᓐᓄᑦ ᐃᓄᐃᑦ ᓄᓇᖃᖅᑲᕐᕕᒋᔭᖏᓐᓂᒃ ᐅᑭᐅᖅᑕᖅᑐᓕᒫᒥ ᖃᓄᑎᒋ ᐊᔾᔨᒌᖏᑎᒋᒻᒪᖔᑕ ᐅᔾᔨᕐᓱᖅᑕᐅᓗᑎᒃ.
- ᖃᓄᐃᒍᑎᐅᕙᑦᑐᑦ ᐊᔾᔨᒌᖏᒻᒪᑕ ᑎᑎᕋᖅᑕᐅᕙᓪᓕᐊᓯᒪᐸᑕ ᖃᐅᔨᓴᖅᑕᐅᓂᒃᑯᑦ ᐊᑲᐅᓂᖅᑲᓐᓂᐅᓴᒻᒪᑕ ᐋᓐᓂᐊᖅᑲᓐᓇᖏᑦᑐᓕᕆᓂᕐᒧᑦ ᖃᓄᖅ ᑭᓯᓂᓪᓗ ᐱᔾᔪᑎᖃᖅᐸᓪᓚᐃᓂᖏᓐᓂᒃ ᖃᐅᔨᓴᕈᑎᐅᒪᑕ.

6. **ᖃᓄᖅ ᐊᖏᕋᕋ ᓂᕈᐊᖅᑕᐅᓚᐅᕐᒪᑦ? ᖃᓄᖅ ᓂᕈᐊᖅᑕᐅᓚᐅᖅᐳᖓ?**

- ᓂᕈᐊᖅᑕᐅᑐᐃᓐᓇᓚᐅᖅᑐᑎᑦ ᓄᓇᓕᓐᓂ ᐊᖏᕋᕆᔭᐅᔪᑦ ᓂᕈᐊᕝᕕᐅᑐᐃᓐᓇᓚᐅᖅᑐᑦ ᐃᓚᐅᒍᒪᒍᕝᑦ ᐃᓱᒪᖅᓱᖅᑐᑎᑦ.

7. What will I be asked to do?

The first part of the Health Survey takes place at your home:

- In a face-to-face interview, you will be asked to answer a questionnaire about household crowding and food security;
- Answer questions about your own use of medicine and vitamins;
- Schedule a clinic appointment which will take place on board the Coast Guard Ship Amundsen when it arrives in your community; and
- You will be asked to fast overnight (for at least 8 hours) which means no drinks or food after midnight and no drinks or food before you come to the ship. Water will be allowed.

8. What will I be asked to do on the day I visit the Coast Guard Ship?

On the ship: you will be asked to complete 2 parts:

FIRST:

- answer a questionnaire about general health, dental health, tobacco use, nutritional habits, and physical activity; and
- answer a Community and Personal Wellness questionnaire to find out about:
  - your social support, wellness & how you cope;
  - alcohol and drug use; as well as
  - life experiences such as violence, abuse & suicide

The questionnaire contains some very sensitive and personal questions and you can choose to answer this questionnaire in a face-to-face interview or on your own. You can also choose not to answer any questions that make you feel uncomfortable. Once filled out, the forms will be placed in a sealed envelope.

7. ᖃᓄᐃᓕᐅᖁᔭᐅᓂᐊᕋᒪ?

ᓯᕗᓕᖅ ᖃᐅᔨᓴᖅᑕᐅᓂᕆᓂᐊᖅᑕᐃᑦ ᐊᖏᕋᓐᓂ ᐊᐱᖅᓱᖅᑕᐅᒐᔭᖅᑐᑎᑦ:

- ᐊᐱᖅᓱᖅᑕᐅᒐᔭᖅᑐᑎᑦ ᐃᓕᕐᓱᓐᓂ ᐊᐱᖅᑯᑎᖃᐅᕚᖅᑐᑦ ᐃᓪᓗᓯ ᐃᓄᒋᐊᕐᕕᐅᖃᑦᑕᕐᒪᖔᖅ ᐊᒻᒪᓗ ᓂᕿᒋᖃᑦᑕᖅᑕᓯ ᓇᒦᖃᑦᑕᒻᒪᖔᑕ.
- ᖃᓄᐃᑦᑐᓂᓪᓗ ᐃᓅᓕᓴᐅᑎᖃᖃᑦᑕᕐᒪᖔᑉᐱᑦ ᐊᒻᒪᓗ ᐊᓯᙱᓐᓂᒃ ᐃᓅᓯᕐᒧᑦ ᐊᑐᖅᑐᓂᒃ.
- ᐅᒥᐊᕐᔪᐊᒥ ᖃᐅᔨᓴᖅᑕᐅᓂᖃᕋᔭᕐᒥᔪᑎᑦ.
- ᓂᕆᑦᑕᐃᓕᖁᔭᐅᒐᔭᖅᑐᑎᑦ ᐃᕋᓂᒃ 8ᓂᒃ ᖃᐅᔨᓴᖅᑕᐅᓚᐅᕐᑎᓐᓂᓐᓂᒃ ᐅᒥᐊᕐᔪᒥ. ᐃᒥᕈᓐᓇᕋᔭᕋᓗᐊᖅᑐᑎᑦ.

8. ᖃᓄᐃᓕᐅᖁᔭᐅᒐᔭᖅᐳᖓ ᐅᒥᐊᕐᔪᐊᒥ?

ᒪᕐᕈᐃᓕᖅᑲᖓᓂᐊᖅᑐᑦ ᖃᐅᔨᓴᖅᑕᐅᓃᑦ ᐅᒥᐊᕐᔪᐊᒥ.

ᓯᕗᓪᓕᖅ:

- ᑭᐅᖃᑦᑕᕐᓗᒋᑦ ᐊᐱᖅᑯᑎᐅᔪᑦ ᖃᓄᐃᖃᑦᑕᕐᒪᖔᖅᐱᑦ, ᑭᒍᑎᑎᑦ, ᓯᒃᑲᓕᐊᖅᑐᖃᑦᑕᕐᒪᖔᖅᐱᑦ, ᓂᕿᒋᕙᑦᑕᑎᑦ ᐊᒻᒪᓗ ᑎᒥᒧᑦ ᐃᖃᐃᓕᓴᕈᑎᒋᕙᑦᑕᑎᑦ.
- ᓄᓇᓕᕐᓱᓐᓂᒃ ᐊᒻᒪᓗ ᓇᒥᓂᖅ ᐃᓄᓯᕆᕙᑦᑕᐃᑦ ᐊᐱᖅᑯᑎᐅᓖᕐᒥᔪᑦ: ᖃᓄᖅ ᐃᑲᔪᖃᑦᑕᕐᒪᖔᖅᐱᑦ, ᑲᒪᒋᑦᓯᐊᖃᑦᑕᕐᒪᖓᖅᐱᑦ, ᖃᓄᕐᓗ ᕿᓄᐃᔮᖃᑦᑕᕐᒪᖔᖅᐱᑦ;
- ᐃᒥᐊᓗᖃᑦᑕᕐᒪᖔᖅᐱᑦ, ᐊᖓᔭᕐᓇᖅᑐᓂᓪᓗ ᐱᖃᑦᑕᕐᒪᖔᖅᐱᑦ.
- ᐃᓅᓯᕆᓯᒪᔭᓐᓂᒃ ᓂᓐᖓᐅᒪᓂᖃᓚᐅᖅᓯᒪᒻᒪᖔᖅᐱᑦ, ᐋᓐᓂᕈᑎᖃᖅᓯᒪᒻᒪᖔᖅᐱᑦ, ᐃᒻᒥᓂᕋᓱᐊᕐᓂᑰᒻᒪᖔᖅᐱᑦ.

ᐊᐱᖅᑯᑎᐅᔪᑦ ᐃᓅᓯᕐᓄᙵᓗᐊᖅᑑᒐᓗᐊᑦ ᑭᓱᐊᓂ ᑭᐅᒍᒪᒍᕕᑦ ᐃᓱᒪᖅᓱᖅᑐᑎᑦ. ᑭᐅᒋᐊᑐᙱᒻᒥᔪᑦ ᐃᓚᙵᑦ ᐊᑲᐅᖏᙱᑐᐊᕈᕕᑦ. ᑕᐃᒃᑯᐊ ᑭᐅᔾᔪᑎᑎᑦ ᑐᖅᑯᖅᑕᐅᓯᒪᕐᓯᐊᕐᓂᐊᖅᑐᑦ.

SECOND:

- The nurse will:
  - take your blood pressure and pulse;
  - take a finger prick blood test to see if your blood sugar level is normal;
  - if your blood sugar is not normal, we will not proceed with this test;
  - if your blood sugar level is normal we will take no more than 45 mL of blood from a vein in your arm (approximately 3 tablespoons);
  - you will then be given a sweet drink and 2 hours later, another small sample of blood (approximately 5 mL or 1 teaspoon) will be taken;
  - take a few toenail clippings to analyze for the metal selenium: Selenium helps fight cancer and because it is found in country food, the analysis of your toenail clippings will let us see how much country food you eat;
  - ask you questions about your medical history; and
  - measure your height, sitting height, weight, waist measurement and body fat.

<u>For both men and women, 40 years and older, additional tests will be done such as:</u>

- Monitor your heart rate by wearing a holter monitor, a small instrument that records your heart rate for about 2 hours.
  - Some irregularities are not important but others are and you will be notified if you need follow-up evaluation.
- Using an ultrasound, an instrument that is used outside of your body, we will look at the artery in your neck to see how easily the blood is flowing to your brain.
  - <u>If</u> the artery is too narrow you will be notified of the need to see a doctor for follow-up because you may be at risk of a stroke. And,
- Abdominal ultrasound to assess body fat.

- **<u>Women</u>** will also be asked to have their bone mineral density measured using a low dose x-ray of the forearm and heel that will look at your bone health.

- Transportation to and from the ship will be provided.

9. What will you test in my blood?

<u>We will test:</u>

- Your risk of heart disease by testing for different types of chemicals and fat in your blood;
- Glucose and insulin which will tell us about your risk of diabetes;
- Vitamin D and proteins related to bone health;
- Effects of diet to see if you are getting enough healthy food to meet your body’s needs;
- Effects of a bacteria that can cause stomach ulcers such as *H.pylori*;
- Effects of types of infection that spread from animals to humans; And
- Effects of exposures to environmental contaminants.

ᐊᐃᑉᐸᖓ:

ᐋᓐᓂᐊᓯᖅᐅᑎᒧᑦ:

- ᑎᓪᓕᕐᓂᑦ ᖃᐅᔨᓴᖅᑕᐅᒐᔭᖅᑐᖅ
- ᐊᐅᓚᖅᑕᐅᒐᔭᖅᑐᑎᑦ ᐊᓑᒃᑯᑦ ᖃᐅᔨᓴᖅᑕᐅᓗᓂ ᐊᐅᑦ ᓱᑲᖓ ᓈᒻᒪᒃᑲᓗᐊᕐᒪᖕᒋᖅ.
- ᓱᑲᕐᑲᓗᐊᕆᔭᐅᒍᓇᑦ ᑲᔪᓯᒍᓐᓇᕋᔭᖕᒋᑦᑐᖅ ᖃᐅᔨᓴᖅᑕᐅᓃᑦ.
- ᓱᑲᐃᑦ ᓈᒻᒪᑉᐸᑦ ᐊᐅᓚᖅᑕᐅᓂᐊᖅᑐᑎᑦ ᑕᓕᕐᓂᑦ ᐊᐅᓚᖅᑕᐅᒍᑎᑦ ᐊᓗᑎᖅᐸᐃᑦ ᐱᖓᓱᒐᔭᖅᑐᑦ.
- ᐃᒥᖅᑎᑕᐅᓂᐊᖅᑐᑎᑦ ᓯᕐᓇᖅᑐᒥᒃ ᐊᒻᒪᓗ ᐃᑲᕋᕝ ᒪᕆᒄ ᐊᓂᒍᖅᐸᑎᒃ ᐊᐅᓚᖅᑕᐅᓕᕐᒥᓗᑎᑦ ᐊᓘᑎᕋᕐᒥᒃ.
- ᐳᑐᒃᑯᑦ ᑯᑭᖅᑕᐅᒐᓖᓪᓗᑎᑦ ᖃᐅᔨᓴᖅᑕᐅᓂᐊᕐᒪᑦ ᐋᓇᐅᒪᔾᔪᑎᖃᑦᓯᐊᕐᒪᖕᒋᖅᐱᑦ ᖃᐅᔨᔭᐅᒍᓇᕐᒪᑦ ᓂᕿᑐᐃᓇᖅᑐᖃᑦᑕᑦᓯᐊᕈᕚᑦ.
- ᐊᐱᖅᓱᖅᑕᐅᓗᑎᓪᓗ ᖃᓄᐃᖃᑦᑕᕐᒪᖕᒋᖅᐱᑦ ᐋᓐᓂᐊᖃᑦᑕᕐᓂᑯᒻᒪᖕᒋᖅᐱᑦ.
- ᖃᓄᓕᒫᖅ ᐆᑦᑐᖅᑕᐅᓗᑎᑦ ᑕᑭᓃᑦ, ᐅᖁᒪᐃᓐᓃᑦ ᐊᒻᒪᓗ ᑎᒥᑉᐱᑦ ᐅᖅᓱᖓ.

ᐊᖑᑎᓄᑦ ᐊᕐᓇᓄᓪᓗ ᐊᕿᒍᖃᓕᖅᑐᓄᑦ 40 ᐅᖓᑖᓂᓗ ᖃᐅᔨᓴᖅᑕᐅᒍᑎᖃᕐᓂᐊᕐᒥᔪᑦ ᐅᑯᓂᖓ:

- ᐆᒪᑎᑦ ᖃᐅᔨᓴᐅᑎᒧᑦ ᖃᐅᔨᓴᖅᑕᐅᓗᓂ ᐃᑲᕋᓐᓄᑦ ᒪᕈᓐᓄᑦ.
- ᖃᐅᔨᓚᐅᓲᖑᒻᒪᑕ ᖃᐅᔨᓴᖅᑕᐅᒃᑲᓐᓂᕆᐊᖃᕐᓂᕈᕙᑦ ᖃᐅᔨᑎᑕᐅᒐᔭᖅᑐᑎᑦ.
- ᖃᐅᔨᓴᐅᑎᒃᑯᑦ ᑏᕙᕋᕵᒃᑯᑦ ᖃᐅᔨᓴᖅᑕᐅᓂᐊᖅᑐᖅ ᑎᒥᓃᑦᑐᑦ ᖁᖓᓯᓐᓂᑦ ᑕᖃᐸᐅᔭᖅ ᖃᓇᓴᕐᓄᑦ ᐊᐅᒃ ᐃᖏᕋᕋᓯᐊᕋᓗᐊᕐᒪᖕᒋᖅ.
- ᖃᐅᔨᔭᐅᒍᕙᑦ ᑕᖃᓪᓗᑦᑐᑎᑦ ᖃᐅᔨᒃᑲᑕᐅᓵᐅᑎᑭᒐᔭᖅᑐᑎᑦ ᔫᑦᑕᒧᑦ ᑕᑯᔭᒃᓴᐅᓕᕐᓗᑎᑦ ᖃᐅᔨᓴᖅᑕᐅᓯᐊᕐᓕᕐᓗᑎᓗ.
- ᐊᑯᐊᑦ ᖃᐅᔨᓴᖅᑕᐅᓂᐊᖅᑐᖅ ᖃᓄᖅ ᐅᖅᓱᖃᖅᑎᑭᒻᒪᖕᒋᖅ.
- **ᐊᕐᓇᐃᑦ** ᖃᐅᔨᓴᖅᑕᐅᓂᐊᕐᒥᔪᑦ ᓴᐅᓂᖕᒋᑦ ᑕᕐᕋᖅᑐᖅᑕᐅᓗᑎᒃ ᐊᒐᖓᒍᑦ ᐊᒻᒪᓗ ᑭᒻᒥᖓᒍᑦ

ᐅᒥᐊᕐᔪᐊᒨᕈᔭᐅᒐᔭᖅᑐᑦ.

9. **ᑭᓱᒥᒃ ᖃᐅᔨᓴᕈᑎᖃᕐᓂᐊᕋᕋᓯ ᐊᐅᓚᓂ?**

<u>ᖃᐅᔨᓴᕐᓂᐊᖅᑐᒍᑦ:</u>

- ᐆᒪᑎᓯ ᖃᓄᐃᒍᑎᒃᓴᖃᖕᒋᒃᑲᓗᐊᕐᒪᖕᒋᑕ ᐊᔾᔨᒌᖕᒋᑦᑐᓂᒃ ᐊᒻᒪᓗ ᐅᖅᓱᖃᕐᓂᓯ ᐊᐅᒃᓯᒍᑦ ᖃᐅᔨᓴᖅᑕᐅᓂᐊᕐᒥᔪᑦ.
- ᓱᑲᕐᑲᓗᐊᕐᒪᖕᒋᖅᐱᑦ ᖃᐅᔨᓴᖅᑕᐅᓂᐊᕐᒥᔪᑎᑦ.
- ᕙᐊᑐᒪᓐ D ᐊᒻᒪᓗ ᓴᐅᓂᑎᑦ ᖃᓄᐃᙵᕆᑲᓗᐊᒪᖕᒋᑖ
- ᓂᕆᕐᑲᑦᑕᖅᑕᑎᑦ ᐊᑲᐅᓂᖅᑲᕐᒪᖕᒋᑕ ᑎᒥᓐᓄᑦ.
- ᐊᕐᑭᐊᕈᓗᕈᑎᐅᖅᑲᑦᑕᖅᑐᑦ ᖃᓄᐃᑦᑑᒪᖕᒋᑕ.
- ᐊᑲᐅᒍᓐᓃᕈᑎᐅᖅᑲᑦᑕᖅᑐᑦ ᐆᒪᔪᓐᖓᖅᑐᑦ
- ᐊᐧᑎᒥᑦ ᓱᕈᕈᑎᐅᖅᑲᑦᑕᖅᑐᑦ.

10. When will I get my results?

- Some results will be given to you immediately when you are evaluated by the nurse;
- For most of the blood tests, you will have your own results back within 5 months;
- The results will be sent to you in a letter with explanations of your laboratory and clinical tests, along with some advice and healthy tips. The letter will be written in plain English and Inuktitut.
- Abnormal results will be given to your local health centre for medical follow-up.

11. What is not being tested?

You are **not** being tested for:

- Cancer risk of any type;
- Sexually transmitted infections or other types of infections, such as tuberculosis (T.B.);
- Drug use;
- Hearing, vision; and respiratory health.

12. How long will the Survey take?

- The home visit will take approximately 45 minutes.
- The visit to the Coast Guard Ship will take approximately 3 hours.

13. What are some potential harms, injuries, discomforts or inconveniences?

- A needle will be inserted into your arm to draw blood. This may cause you some discomfort; and you may experience some tenderness or have a small bruise in the area where the needle was inserted:
    - An infection can happen where the blood is taken, however, this is very rare, and the nurse can easily treat the infection.
    - If you feel faint at any time trained nurses will be there to help you.
- If you feel weak, tired, shaky or hungry after drinking the sweet drink, a nurse will be available to help you. All participants who will receive the oral glucose tolerance test will be asked to eat and drink before leaving the ship.
- Some of the questions in the Community and Personal Wellness questionnaire are very sensitive and personal, and may make you feel uncomfortable. You don't have to answer all questions.

10. ᖃᖓᒃᑯᑦ ᑐᓴᖅᑎᑕᐅᒐᔾᖅᑯᖓ?

- ᐃᓚᖏᑦ ᑐᓴᕐᓴᐅᑎᒋᒐᔾᖅᑕᑎᒃ ᐋᓐᓂᐊᓯᐅᖅᑎᒥᑦ
- ᐊᐅᒻᒧᑦ ᖃᐅᔨᓴᐅᑕᐅᒍᑎᓕᒫᒐᓚᓐᓈᖓᔪᓄᑦ, ᓇᒻᒥᓂᖅ ᖃᐅᔨᓴᖅᑕᐅᒍᑎᒥᓂᑦ ᐱᒐᔾᖅᑕᑎᑦ ᐊᓂᒍᖅᓯᒪᓕᖅᐸᑕ.ᑎᑎᖅᑲᐅᓯᐊᕋᔾᖅᑐᑎᑦ ᓇᓗᓇᐃᔾᖅᓯᒪᔪᓂᒃ ᖃᓄᐃᓚᐅᕐᒪᖔᖅ. ᑎᑎᖅᑲᖅ ᖃᑦᑐᓈᑎᑐᑦ ᑐᑭᓯᓇᖅᓯᑎᑕᐅᓯᒪᔪᓂ ᐊᒻᒪᓗ ᐃᓄᒃᑎᑑᖓᓗᓂ. ᑎᑎᕋᕝᕙᐅᓯᒪᓂᐊᖅᑐᑎᑦ ᐋᓐᓂᐊᖃᕐᓇᖏᑦᑐᓕᕆᓂᕐᒧᑦ ᖃᓄᐃᖏᑦᑎᐊᕋᓱᓂᕐᒨᖓᔪᓂᒃ ᐊᒻᒪᓗ ᑐᑭᓯᒋᐊᕈᑎᓂᒃ ᖃᐅᔨᓴᐅᑎᐅᓚᐅᖅᑐᓂᒃ.
- ᑕᐃᒃᑯᐊ ᖃᓄᐃᑦᑐᒥᓃᑦ ᓄᓇᓕᑦᓯᓐᓄᑦ ᐋᓐᓂᐊᕐᕕᖓᓄᑦ ᖃᐅᔨᓴᖅᑕᐅᒃᑲᓐᓂᕈᐊᕐᑲᕋᔾᖅᑐᑦ.

11. ᑭᓱᑦ ᖃᐅᔨᓴᖅᑕᐅᒍᑎᐅᖏᓚᑦ?

ᖃᐅᔨᓴᖅᑕᐅᒍᑎᖃᖏᑦᑐᑎᑦ ᐅᑯᓄᖓ:
- ᐋᓐᓂᐊᕐᑲᕐᒪᖔᖅᐱᑦ
- ᕿᐱᕆᓯᒪᒻᒪᖔᖅᐱᑦ ᖃᓄᐃᑦᑐᐃᓐᓇᕐᓂᒃ, ᐳᕙᑦᑐᒻᒪᖔᖅᐱᑦᑑᓐᓃᑦ
- ᐊᖓᔾᕐᓇᖅᑐᖅᑐᖃᑦᑕᕐᓃᑦ
- ᑑᓴᑦᑎᐊᕐᒪᖔᖅᐱᑦ, ᑕᐅᑐᕆᐊᖏᒻᒪᖔᖅᐱᑦ, ᐊᓂᕐᓂᑭᖃᑦᑕᕐᒪᖔᖅᐱᑦᑑᓐᓃᑦ.

12. ᖃᓄᑎᒋ ᖃᐅᔨᓴᖅᑕᐅᓂᖃᕐᓂᐊᖅᐳᖓ?

- ᐊᖏᕐᕋᕆᔭᓐᓂᒃ ᐊᐱᖅᓱᖅᑕᐅᓃᑦ ᐃᑲᕐᕋᓕᒫᖖᒍᖏᑦᑐᖅ.
- ᐅᒥᐊᕐᔪᐊᒥ ᖃᐅᔨᓴᖅᑕᐅᓃᑦ ᐃᑲᕐᕋᓄᑦ ᐱᖓᓱᓄᑲᔾᖅᑐᖅ.

13. ᑭᓱᒐᔾᖅᐸᑦ ᖃᐅᔨᓴᖅᑕᐅᓂᓐᓄᑦ ᐊᑲᐅᖏᓕᐅᑲᐃᓐᓇᕋᔾᖅᑐᖅ?

- ᐊᐅᖏᑕᐅᒍᑎᑦ. ᐊᑲᐅᖏᑲᐃᓐᓇᕋᔾᖅᑐᖅ ᐊᒻᒪᓗ ᑎᓪᓗᔭᒐᒑᑎᕐᓯᒍᓐᓇᖅᑐᖅ.
- ᐊᑲᐅᖏᓕᐅᑐᐃᓐᓇᕆᐊᖃᖅᑐᑎᑦ ᑲᐳᑎᒥ ᑕᐃᒪᐃᓐᓂᕈᕙᑦ ᐋᓐᓂᐊᓯᐅᖅᑎᒧᑦ ᑲᒪᒋᔭᐅᒍᓐᓇᕋᔾᖅᑐᖅ.
- ᖃᐅᔾᒪᖏᓐᓂᐅᓴᔩᔭᕈᕙᑦ ᐋᓐᓂᐊᓯᐅᖅᑎᑕᖃᑖᖅᑐᖅ ᐊᑐᐃᓐᓇᐅᓂᐊᖅᑐᓂᑦ ᐃᑲᔪᕈᓐᓇᖅᑐᓂᒃ.
- ᑕᖃᑲᐃᓐᓇᕋᔾᖅᑐᕐᓴᐅᕗᑎᑦ, ᓴᔪᒐᓚᓪᓗᑎᓪᓘᓐᓃᑦ ᑳᓗᐊᒧᑦ ᓯᕐᓇᖅᑐᓕᒻᒥᑦ ᐃᒥᒐᕈᕙᑦ, ᐋᓐᓂᐊᓯᖅᐅᑎ ᐊᑐᐃᓐᓇᐅᕝᓯᐊᖅᑐᖅ.
- ᑕᐃᑲᓃᓕᕈᕙᑦ ᑐᒧᐊᑎᑕᐅᖅᑐᑎᑦ ᓰᓯᓕᒻᒥᑦ ᐊᒻᒪᓗ ᐃᒥᒐᕐᓴᔭᒥᒃ ᖃᓄᐃᖁᓇᒃ. ᖃᐅᔨᓴᖅᑕᐅᓃᑦ ᐃᑲᕐᕋᓄᑦ ᐱᖓᓱᓅᓂᐊᕐᒪᑦ ᐊᒻᒪᓗ ᓱᑲᑯᓪᓕᑦ ᐃᒥᖅᑎᑕᐅᓂᐊᕐᒥᔪᑎᑦ ᖃᐅᔨᓴᐅᑎᐅᓂᐊᖅᑐᒥᒃ ᐅᒥᐊᕐᔪᐊᒥᑎᓪᓗᑎᒃ.
- ᐊᐱᖅᑯᑎᐅᔪᑦ ᓄᓇᕝᓯ ᒥᒃᓴᓄᑦ ᐅᕝᕙᓘᓐᓃᑦ ᐃᓅᓯᕗᑦ ᐊᑦᓱᐱᓇᓗᐊᖅᐸᑦᑕ ᑭᐅᒋᐊᑐᖏᑕᑎᑦ.

14. What are the Potential Benefits of the Survey?

- The health survey gives you the opportunity to contribute to a greater understanding of the overall health of Inuit in Nunavut.
- Your participation in this survey will also give you a better understanding of your nutritional status, and your diabetes and heart disease risks.
- You will also get helpful information about what you can do to reduce or prevent health problems. And,
- You will be given a brochure with information on where to go for help in Nunavut if you need to talk about mental wellness, with a 1 800 hot line phone number.

15. Where will my blood go and how will it be stored?

- The blood samples will be sent to the Centre for Indigenous Peoples’ Nutrition and Environment (CINE) at McGill University in Montreal for analysis. Some blood analysis will take place at Laval University in Quebec City and any remaining samples will be returned to Montreal.
- Your blood samples will be identified by a study number only. Your name, birth date or name of your community will not appear on the blood samples.
- Your blood samples will be destroyed within **2** years following data collection if you do not wish to join the 7-year follow-up evaluation.

- **IF** you agree to participate in the International Inuit Health Survey, we will contact you in 7 years for a follow-up and *some* of the blood drawn today will be stored for **10** years. The blood sample will be identified by a study number only. No names, birth dates or names of communities will be used on the blood sample tubes.
- The blood samples will only be used by the research team for looking at markers of heart disease, diabetes, cancer, environmental exposures, and nutritional status. For women over 40, we will also look at bone health. No other research uses of the blood samples are allowed. For example, no genetic tests will be conducted without your permission.
- No third party agencies or companies are allowed access to the samples without the permission of the steering committee and yourself.

14. ᖃᓄᖅ ᐃᑲᔪᕐᓂᕐᑳᕐᓂᐊᕐᒪᑕ ᖃᐅᔨᓴᖅᑕᐅᒍᑎᒃᑲ?

- ᖃᐅᔨᓴᖅᑕᐅᒍᑎᑎᑦ ᑐᑭᓯᕚᓪᓕᖅᑎᕐᓯᓂᐊᖅᑐᑦ ᖃᓄᖅ ᓄᓇᕗᒻᒥ ᐋᓐᓂᐊᕐᑳᕐᓇᖏᑦᑐᓕᕆᓂᕐᒧᑦ ᐃᑲᔫᒃᑲᓐᓂᕈᓐᓇᕐᒪᖔᑦᑕ.
- ᑐᑭᓯᕚᓕᕈᑎᐅᓗᓂᓗ ᓂᕆᔭᐅᕙᑦᑐᑦ ᖃᓄᐃᒍᑎᐅᕙᒻᒪᖔᑕ ᓱᑲᕐᑲᓗᐊᕐᓂᒧᑦ ᐅᕝᕙᓘᓐᓃᑦ ᐆᒪᓯᕆᕐᑲᑦᑕᕐᓂᕐᒧᑦ.
- ᐃᑲᔪᖅᑕᐅᕙᔭᕐᒥᔪᑎᑦ ᖃᓄᖅ ᐃᓕᓐᓂᑦ ᑲᒪᒋᒃᑲᓐᓂᕈᓐᓇᕐᒪᖔᖅᐱᑦ ᐃᓅᓯᕐᑲᕐᓯᐊᕐᑯᓪᓗᑎᑦ ᖃᓄᐃᖏᕐᑯᓪᓗᑎᑦ.
- ᑐᓂᓯᔭᐅᕙᔭᖅᑐᑎᑦ ᑐᑭᓯᒋᐊᕈᑎᒥᒃ ᓇᒧᑦ ᐃᑲᔪᖅᑕᐅᒍᓐᓇᕐᒪᖔᖅᐱᑦ ᐃᓅᓯᓕᕆᓂᕐᒧᑦ ᐊᒻᒪᓗ ᐅᖄᓚᐅᑎᒥᒃ ᐊᑭᕐᑲᖏᑦᑐᒥᒃ ᐅᖄᓚᕝᕕᕐᑲᑦᑕᕈᓐᓇᖅᑕᓐᓂᒃ.

15. ᓇᒧᖓᖅᑕᐅᓂᐅᓴᕗᑦ ᐊᐅᖏᑕᐅᒍᑎᒥᓃᒃᑲ?

- ᐊᐅᖏᖅᑕᐅᒍᑎᒥᓂᐅᔪᑦ ᐱᓯᒪᔭᐅᓂᐊᖅᑐᑦ ᓄᓇᖃᖅᑳᖅᓯᒪᔪᓄᑦ ᓂᕆᖃᑦᑕᖅᑕᖏᓐᓂᒃ ᖃᐅᔨᓴᕈᑎᐅᔪᓂᑦ ᑕᐃᑲᓂ ᓯᓚᑦᑐᖅᓴᕐᕕᒻᒥ ᒪᑭᐅᒥ ᒫᑐᕆᐊᒥ.
- ᐊᐅᖏᖅᑕᐅᒍᑎᒥᓂᐅᔪᑦ ᓈᓴᐅᑎᕐᑲᑐᐃᓐᓇᕐᓂᐊᖅᑐᑦ. ᐊᑎᖓ ᓄᓇᖓᓗ ᑎᑎᕋᖅᑕᐅᓯᒪᙱᔭᖏᑦᑐᑦ.
- ᖃᐅᔨᓴᕈᑎᐅᔪᑦ ᖃᕆᓴᐅᔭᒧᑦ ᐃᓕᔭᐅᕐᓴᐅᑎᒋᓂᐅᓴᔪᑦ ᑕᐃᓐᓇᓗ ᖃᕆᓴᐅᔭᖅ ᑕᒪᑦᓯᒧᖓᑐᐃᓐᓇᖅ ᑐᕐᖓᓪᓗᓂ. ᓈᓴᐅᑏᓐᓇᕐᒥᒃ ᐊᑐᕋᔭᖅᑐᑦ.
- ᐊᐅᖏᖅᑕᐅᒍᑎᒥᓂᑦᑎ ᐊᕐᕌᒍᒃ ᒪᕐᑮᒃ ᐊᓂᒍᖅᐸᑎᒃ ᐃᒋᑕᐅᕙᔭᖅᑐᑦ ᑭᓯᐊᓂ ᐃᓚᐅᕐᑲᑕᐅᔪᒪᓐᓂᕈᕝᕕᑦ ᐊᕐᕋᒍᑦ 7 ᐊᓂᒍᖅᐸᑕ ᖃᐅᔨᓴᖅᑕᐅᒃᑲᓐᓂᕐᓗᑎᑦ.

- ᖃᐅᓴᔨᓴᖅᑕᐅᒃᑲᓐᓂᑰᕈᕝᕕᑦ ᓯᓚᕐᔪᐊᓕᒫᒥ ᐃᓄᐃᑦ ᐃᓅᓯᖏᓐᓂᒃ ᖃᐅᔨᓴᖅᑕᐅᓂᖃᓕᕐᒥᑉᐸᑕ; 7 ᐊᕐᕋᒍᐃᑦ ᐊᑐᓕᖅᐸᑕ ᖃᐅᔨᕆᐊᖅᑕᐅᒃᑲᓐᓂᕋᔭᖅᑐᑎᑦ. ᐊᐅᖏᖅᑕᐅᒍᑎᑦ ᐊᕐᕌᒍᓄᑦ ᖁᓕᓄᑦ ᐱᓯᒪᔭᐅᕙᔭᖅᑐᖅ. ᓈᓴᐅᑎᓕᖅᑕᐅᓯᒪᑐᐃᓐᓇᕐᓗᓂ. ᐊᑎᖃᕋᔭᖏᑦᑐᖅ ᓇᓗᓇᐃᔭᖅᑕᐅᓯᒪᙱᔭᖏᑦᑐᖅ.
- ᐊᐅᖏᑕᐅᒍᑎᒥᓂᑦ ᖃᐅᔨᓴᖅᑎᓄᑦ ᖃᐅᔨᓴᖅᑕᐅᕐᑲᑦᑕᕋᔭᖅᑐᑦ ᐆᒻᒪᑎᓪᓗᒻᒪᖔᑕ, ᓱᑲᕐᑲᓗᐊᕐᒪᖔᑕ ᐋᓐᓂᐊᕐᑲᕐᒪᖔᑕ, ᐊᕙᑎᒧᑦ ᓱᔾᔭᓯᒪᒻᒪᖔᑕ ᐊᒻᒪᓗ ᓂᕆᕙᑦᑕᖏᓐᓄᑦ. ᐊᓯᖏᓐᓄᑦ ᖃᐅᔨᓴᖅᑕᐅᕙᔭᖏᑦᑐᑦ ᐊᑐᖅᑕᐅᒍᓐᓇᕋᔭᖏᑦᑐᑦ. ᖃᐅᔨᓴᖅᑕᐅᒃᑲᓐᓂᕈᓐᓇᖏᑦᑐᑦ ᐊᖏᓚᐅᖅᑎᓐᓇᑎᑦ. ᐊᕐᓇᐃᑦ 40 ᐅᕙᑖᓂᑐᑦ ᖃᐅᔨᓴᖅᑕᐅᕙᔭᖅᒥᔪᑦ ᓴᐅᓂᖏᑦ
- ᐊᓯᖏᓐᓄᑦ ᖃᐅᔨᓴᖅᑎᓄᑦ ᖃᐅᔨᓴᖅᑕᐅᒍᓐᓇᔾᔭᖏᑦᑐᑦ ᐸᕐᓇᐃᔮᓄᑦ ᑲᑎᒪᔩᕋᑖᓂᑦ ᐊᖏᖅᑕᐅᓗᑎ ᑭᓯᐊᓂ ᐊᒻᒪᓗ ᐃᓕᓐᓄᑦ.

16. ***Medical Records and Access to Information***

- **IF** you decide to take part in <u>the International Inuit Study</u>: you will also be asked for permission for a nurse to review your medical chart to verify heart disease, diabetes and related chronic medical conditions. This information will be coded with your study ID number.
- No other personal information will be shared with any community member, organizations or other agencies.
- Only summary findings for all participants grouped together will be shared with regional and national Inuit organizations and public health agencies.

17. Is all information Confidential? YES.

- All interviewers, nurses and technicians have signed a confidentiality agreement and the collected data is confidential.
- You will be given a unique study number to keep your identity and your community's identity confidential. Information from the Community and Personal Wellness questionnaire will be entered into a computer program on a computer with no connection to the internet or other computers. Only your study number will be given to those analyzing the data.
- The names and addresses of participants will be kept on a "master list" in a locked safe at McGill University so your results can be returned to you.
- Your personal clinical blood tests and other clinical measurement results will also be given to Health and Social Services so they can update your medical file with the information and arrange for follow-up services if needed. (No personal questionnaire data will be shared with Health and Social Services).
- If you are at a high risk of suicide, the interviewer is required to refer you for help.
- Also, while we have no questions about minors in the questionnaire, if you provide information that a minor is or has been abused, the interviewer is required to report this to Social Services, otherwise, all information you provide is confidential.

18. Can I stop my participation in the middle of my clinic visit?

- Yes, you may withdraw from the study at any time
- You can choose not to continue even after you have agreed to participate.
- You can choose not to answer questions that you don't feel like answering.
- If you agree to be a part of the International Inuit Health Survey, you are free to withdraw from it at any time.

16. ᐋᓐᓂᐊᕕᒨᕈᑎᒥᓅᑦ ᐊᒻᒪᓗ ᑐᑭᓯᒋᐊᕈᑏᑦ?

- ᐃᓚᐅᔪᒪᓐᓂᕈᕕᑦ ᓯᓚᕐᔪᐊᓕᒫᒥ ᐃᓄᐃᑦ ᐃᓅᓯᖏᓐᓂᒃ ᖃᐅᔨᓴᖅᑕᐅᓂᖃᓕᕐᒥᑉᐸᑕ; ᐊᐱᕆᔭᐅᒐᔭᖅᑐᑎᑦ ᐋᓐᓂᐊᓯᐅᖅᑎᒧᑦ ᕿᒥᕐᕈᔭᐅᒍᓐᓇᕐᒪᖖᑕ ᐋᓐᓂᐊᕕᒨᕈᑎᒥᓂᑦ ᖃᐅᔾᔨᐊᕈᑎᐅᒐᔭᕐᒪᑕ ᐆᒻᒪᑎᓗᖏᒃᑲᓗᐊᕐᒪᖖᖅᐱᑦ, ᓱᑲᕐᑲᓗᐊᖏᒃᑲᓗᐊᕐᒪᖖᖅᐱᑦ ᐊᒻᒪᓗ ᐊᓯᖏᓐᓂᒃ ᐊᑲᐅᖏᒍᑎᒋᕙᑦᑕᓐᓂᒃ. ᑎᑎᖅᑕᐅᑐᐃᓐᓇᖁᔭᖅᑐᑦ ᓈᓴᐅᑎᓐᓇᒃᑯᑦ.
- ᑭᓇᐅᓃᑦ ᐊᓯᖏᓐᓄᑦ ᑐᓴᐅᒪᔭᐅᔾᔭᖏᑦᓯᐊᖅᑐᑦ.
- ᑕᐃᒃᑯᐊ ᖃᐅᔨᓴᖅᑕᐅᒍᑎᐅᔪᑦ ᑲᑎᖅᓱᖅᑕᐅᒐᔭᖅᑐᑦ ᑐᓴᖅᑎᑕᐅᓗᑎᒃ ᐃᓄᐃᑦ ᑲᑐᔾᔨᕐᑲᑎᒌᖏᑦ ᐊᒻᒪᓗ ᐋᓐᓂᐊᕐᑲᕐᓇᖏᑦᑐᓕᕆᓂᕐᒧᑦ ᐃᖅᑲᓇᐃᔭᖅᑐᓄᑦ.

17. ᑖᒃᑯᐊ ᑐᓴᖅᑕᐅᔪᑦ ᑕᑯᔭᐅᔾᔭᖏᑦᓯᐊᖅᑑᒐᓗᐊᑦ? ᐃᐋ

- ᑕᒪᕐᒥᑦ ᐊᐱᖅᓱᖅᑎᐅᓂᐊᖅᑐᑦ, ᐋᓐᓂᐊᓱᖅᐅᑎᑦ, ᑲᒪᔨᐅᓂᐊᖅᑐᓪᓗ ᐊᑎᓕᐅᖅᓯᒪᓂᐊᖅᑐᑦ ᐊᖏᕈᑎᒥᒃ ᑐᓴᖅᑎᓯᒋᐊᖃᖕᖏᓐᓂᕐᒥᑦ ᖃᓄᓕᒫᕐᓯᐊᖅ.
- ᑐᓂᓯᔭᐅᒐᔭᖅᑐᑎᑦ ᓇᓗᓇᐃᒃᑯᑎᒋᓂᐊᖅᑕᓐᓂᒃ ᖃᐅᔮᔭᐅᓂᐊᖏᓐᓇᕕᑦ. ᑕᐃᒃᑯᐊ ᐊᐱᖅᑯᑎᐅᔪᑦ ᓄᓇᓕᖕᒑᓂᒃ ᐊᒻᒪᓗ ᐃᓅᓯᖕᒑᓂᒃ ᑎᑎᖅᓯᒪᓂᐊᖅᑐᑦ ᖃᕆᓴᐅᔭᒃᑯᑦ ᐊᒻᒪᓗ ᖃᕆᓴᐅᔭᐃᑦ ᐊᑐᖅᑕᐅᒐᔪᖏᑦᑐᐃᑦ ᑭᓱᑐᐃᓐᓇᕐᒧᑦ. ᓈᓴᐅᑎᖃᑐᐃᓐᓇᖁᔭᖅᑐᑦ ᑕᐃᒃᑯᐊ ᑎᑎᖅᑕᐅᕙᓪᓕᐊᔪᑦ.
- ᑕᐃᒃᑯᐊ ᐊᑎᖏᓐᓂᒃ ᑐᑠᕈᑎᖏᓐᓂᒃ ᑎᑎᖁᖅᓯᒪᔪᑦ ᖀᒃᓯᒪᔪᒥᒃ ᐊᑐᐃᓐᓇᐅᖏᑦᑐᒦᑖᖅᑐᑦ ᒪᕆᐅᑉ ᓯᓚᑦᑐᖅᓴᕝᕕᔾᔪᐊᖓᓂ ᑕᐃᒃᑯᐊ ᖃᐅᔨᓴᕈᑎᐅᓚᐅᖅᑐᑦ ᐃᓕᑦᓯᓐᓄᑦ ᐅᑎᖅᑎᑕᐅᒍᓐᓇᖁᓪᓗᒋᑦ.
- ᐊᐅᖏᖅᑕᐅᒍᑎᒥᓂᑦᑎᑦ ᖃᐅᔨᓴᖅᑕᐅᒍᑎᒥᓂᑦᑎᑦ ᐋᓐᓂᐊᖃᕐᓇᖏᑦᑐᓕᕆᔾᑯᓐᓄᑦ ᐃᓄᓕᕆᔾᑯᓐᓄᑦ ᐋᓐᓂᐊᕕᒨᕈᑎᒋᕙᑦᑕᓐᓄᑦ ᐸᐃᑉᐹᓄᑦ ᑐᓂᔭᐅᒐᔭᖅᑐᑦ ᑭᓯᐊᓂ ᑕᐃᒪᐃᑦᑕᕆᐊᖃᖅᐸᑕ. (ᑕᐃᒃᑯᐊ ᐊᐱᖅᑯᑎᐅᓚᐅᖅᑐᑦ ᑕᐃᑯᖓᖅᑕᐅᒐᔭᖏᑦᑐᑦ.
- ᐃᒻᒥᓂᕐᓂᕐᒧᑦ ᐃᓱᒪᓱᖖᒍᓐᓂᕈᕕᑦ ᖃᐅᔮᔭᐅᓗᑎᑦ ᐊᐱᖅᓱᖅᑎᒧᑦ ᑕᐃᒪ ᐃᑲᔪᖅᑕᐅᒋᐊᕐᑲᕐᓐᓂᒃ ᓇᓗᓇᐃᖅᓯᒋᐊᖅᑲᖁᔭᖅᑐᖅ.
- ᖃᐅᔮᒍᑦᑕᓗ ᐅᑭᐅᑭᓗᐊᖅᑐᒥᒃ ᖃᓄᐃᓕᖅᐅᑕᐅᓯᒪᔪᒥᒃ ᐊᐱᖅᓱᖅᑎ ᐅᖃᕆᐊᖃᖁᔭᖅᑐᖅ ᐃᓄᓕᕆᔾᑯᓐᓄᑦ. ᐊᓯᐊᒍᑦ ᑭᓱᑐᐃᓐᓇᐃᑦ ᑲᙳᓇᖅᑐᑎᑐᑦ ᑲᒪᒋᔭᐅᓂᐊᖅᑐᑦ.

18. ᖃᖓᑐᐃᓐᓇᒃᑯᑦ ᖁᔭᓈᕈᓐᓇᖅᑯᖓ?

- ᖃᖓᑐᐃᓐᓇᕐᓯᐊᒃᑯᑦ ᖁᔭᓈᕈᓐᓇᖅᑐᑎᑦ.
- ᐃᓚᐅᔪᒥᓂᐅᑲᓗᐊᕐᓗᑎᑦ ᖁᔭᓈᕈᓐᓇᖅᑐᑎᑦ.
- ᑭᐅᒋᐊᑐᖏᒻᒥᔪᑎᑦ ᑭᐅᔪᒪᖏᑕᓐᓂᒃ.
- ᐃᓚᐅᒍᒪᔪᒥᓂᐅᓐᓂᕈᕕᑦ ᓯᓚᕐᔪᐊᓕᒫᒥ ᐃᓄᐃᑦ ᐃᓅᓯᖏᓐᓂᒃ ᖃᐅᔨᓴᖅᑕᐅᓂᖃᓕᕐᒥᑉᐸᑕ; ᑖᓯᒍᖓᑦᑕᐅᖅ ᖁᔭᓐᓈᕈᓐᓇᖅᑐᑎᑦ ᖃᖓᑐᐃᓐᓇᕐᓯᐊᖅ.

19. Can I decide not to participate? YES.

- Your decision to be part of the health survey is completely up to you.
- Whether you are part of the study or not, you will continue to have access to the health care you are entitled to at any health care institution or any health clinic you attend.
- There are no negative effects by withdrawing from the Nunavut Inuit Health Survey or from the International Inuit Health Survey.

20. Do I have to spend any money to participate?

- There are no personal costs of participating in the Survey.

21. Who can I contact if I have any questions?

- A member of the health survey team will review the written informed consent with you, and you will be able to keep a copy of it. Should you have any questions, concerns or complaints, a list of contacts and their phone numbers are listed on the consent form.
- **A bilingual staff member is also available to answer your questions: Nick Amautinuar at 867-769-6401**
- You are welcome to call the steering committee members listed on the front sheet at any time before, during or after the survey to request more information, make comments about the survey, complain or withdraw from the study.

19. ᐃᓚᐅᖏᓐᓇᖅᑯᖓ? ᐄ

- ᐃᓱᒪᖅᓱᖅᑐᑎᑦ ᖃᐅᔨᓴᖅᑕᐅᒍᒪᒍᕕᑦ.
- ᐃᓚᐅᖏᒃᑲᓗᐊᕈᕕᑦ ᐋᓐᓂᐊᖃᕐᓇᖏᑦᑐᓕᕆᓂᕐᒧᑦ ᖃᓄᐃᓕᔭᐅᔾᔮᖏᑦᑐᑎᑦ ᐱᔨᑦᓱᖅᑕᐅᒍᓐᓇᖅᑐᑎᑦ ᓱᓕ.
- ᐃᓱᒪᒋᔭᐅᑲᒪᔮᖏᑦᑐᑎᑦ ᐃᓚᐅᒍᓐᓂᕋᓗᐊᕈᕕᑦ ᖃᐅᔨᓴᖅᑕᐅᓂᕐᒧᑦ ᐃᓄᐃᑦ ᐃᓅᓯᖏᑦ ᐊᒻᒪᓗ ᓯᓚᕐᔪᐊᓕᒫᒥ ᐃᓄᐃᑦ ᐃᓅᓯᖏᓐᓂᒃ ᖃᐅᔨᓴᖅᑕᐅᓂᖃᓕᕐᒥᐸᑕ.

20. ᐊᑭᓕᔫᓇᐊᖃᖅᐳᖓ?

- ᐊᑭᓕᒐᒃᓴᖅᑕᖃᔾᔮᖏᑦᑐᖅ.

21. ᑭᓇᒧᑦ ᐊᐱᖅᑯᑎᒃᓴᖃᕈᒪ ᐊᐱᕆᒍᓐᓇᖅᐳᖓ?

- ᐋᓐᓂᐊᖃᕐᓇᖏᑦᑐᓕᕆᓂᕐᒧᑦ ᖃᐅᔨᓴᖃᑕᐅᔪᒧᑦ ᑕᐃᒪ ᐊᑎᓕᐅᖅᓯᒪᔪᒥᒃ ᐊᖏᕈᑎᒥᒃ ᐅᓂᒃᑳᑎᐊᕆᐊᖃᖅᑯᔾᔪᖅ ᖃᓄᐃᓚᐅᕐᒪᖔᖅ. ᐊᐱᖅᑯᑎᒃᓴᖃᕐᓂᕈᕕᑦ ᐃᓱᒫᓗᑎᖃᕈᕕᑦᔫᓐᓃᑦ ᐅᖀᔫᓐᓃᑦ ᐅᓐᓂᕐᓗᒍᒪᒍᕕᑦ ᐅᖄᓚᐅᑎᖏᑦ ᑎᑎᖅᓯᒪᓂᐊᖅᑐᑦ ᐊᖏᕈᑎᐅᑉ ᐃᓗᐊᓂ.
- **ᐊᐱᖅᑯᑎᒃᓴᖃᕈᕕᑦ ᐃᓄᒻᒧᑦ ᐅᖄᓚᕕᖃᕈᓐᓇᖅᑐᑎᑦ: Nick Amautinuar 867-769-6401.**
- ᑐᓐᖓᓱᑦᑎᑎᒻᒥᔪᑦ ᑕᐃᒃᑯᐊ ᐸᕐᓇᐃᔨᑦ ᑲᑎᒪᔩᑦᓵᖏᑦ ᑎᑎᖅᓯᒪᔪᑦ ᓯᕗᓪᓕᖅᐹᒥ ᒪᐸᒐᕐᒥ ᖃᖓᑐᐃᓐᓇᑦᑎᐊᒃᑯᑦ ᖃᐅᔨᓴᖅᑕᐅᒍᑎᒥᓂᖏᑦ ᑐᓴᕈᑎᕆᒍᒪᑐᐊᕈᕕᖏᑦ ᐊᒻᒪᓘᓐᓃᑦ ᖃᐅᔨᓴᐅᑎᐅᔫᑉ ᒥᒃᓵᓄᑦ ᐅᕐᓂᓗᕈᑎᖃᕈᒪᒍᕕᑦ ᐃᓚᐅᒍᒪᖏᒃᑯᕕᑦᔫᓐᓃᑦ.

ᐃᓄᐃᑦ ᐃᓅᓯᖏᑦ ᖃᐅᔨᓴᐅᑎ **ᐃᓐᓇᕐᓂᑦ** ᒪᐃ 31, 2007 ᐊᖏᕈᑎ

ᐃᓄᐃᑦ ᐃᓄᑦᓯᐊᕐᓂᕐᒧᑦ ᖃᐅᔨᓴᖅᑕᐅᓂᖏᑦ - ***ᖃᓄᐃᑉᐱᑕᓕ? ᐅᕙᒍᑦᓕ, ᖃᓄᐃᑉᐱᑕ?***

ᑭᐅᔪᑎᑎᑦ ᐊᒪᓗᑭᑖᓕᖅᑕᑦᑕᕐᓗᒋᑦ:

1. ᑐᑭᓯᑎᑕᐅᓯᒪᕚᖓ ᐱᔾᔪᑎᖏᓐᓂᒃ ᖃᐅᔨᓴᖅᑕᐅᒍᑎᐅᑉ ᐊᒻᒪᓗ ᐱᕚᓕᕈᑎᐅᓂᐊᕐᒪᑕ. ᐄ ᐋᒡᒐ

2. ᑲᙳᓇᖅᑐᑎᑦᑐᑦ ᑲᒪᒋᔭᐅᑦᓯᐊᕐᓂᐊᕐᓂᖏᓐᓂᒃ ᑐᑭᓯᑎᑕᐅᓯᒪᕚᖓ.
ᐊᖏᕈᑎ ᐅᖃᓕᒫᓚᐅᖅᑕᕋ ᐅᕝᕙᓘᓐᓃᑦ ᐅᖃᓕᒫᕐᕕᐅᓪᓗᖓ, ᐊᔾᔨᖓᓂᒃ ᐱᖃᖅᑐᖓ. ᐄ ᐋᒡᒐ

3. ᑐᑭᓯᑎᑕᐅᓯᒪᒋᕚᖓ ᖃᖓᑐᐃᓐᓇᑦᓯᐊᒃᑯᑦ ᖁᔭᓈᕈᓐᓇᖅᑐᖓ. ᐄ ᐋᒡᒐ

ᓯᓚᕐᔪᐊᓕᒫᒥ ᐃᓄᐃᑦ ᐃᓅᓯᖏᓐᓂᒃ ᖃᐅᔨᓴᖅᑕᐅᓂᖃᓕᕐᒥᐸᑕ;

ᐊᖏᖅᑐᖓ ᐃᓚᐅᖃᑕᐅᒍᓐᓇᕈᕆᒪ ᓯᓚᕐᔪᐊᓕᒫᒥ ᐃᓄᐃᑦ ᐃᓅᓯᖏᓐᓂᒃ ᖃᐅᔨᓴᖅᑕᐅᓂᖃᓕᕐᒥᐸᑕ ᐊᒻᒪᓗ ᑐᑭᓯᓪᓗᖓ 7ᓂᒃ ᐊᕿᒍᓂᒃ ᖃᐅᔨᓴᖅᑕᐅᒃᑲᓐᓂᕋᔭᕐᓂᓐᓂᒃ ᐋᓂᐊᓯᖅᑎᒧᑦ ᕿᒥᕐᕈᔭᐅᓗᑎᒃ ᐋᓂᐊᕙᓕᐊᕈᑎᒋᕙᑦᑕᒥᓂᒃᑲ ᐊᒻᒪᓗ ᐋᓂᐊᖃᓕᖅᑐᒥᓂᐅᓐᓂᕈᒪ ᑕᐃᒃᑯᐊᓗ ᖃᓄᖅ ᐋᓂᐊᖃᓕᕐᓂᓐᓂᒃ ᖃᐅᔨᓴᖅᑕᐅᒍᓇᕋᔭᖅᑐᑦ ᐱᕙᓪᓕᐊᓕᖅᑐᒥᓃᑦ.
ᐄ ᐋᒡᒐ

ᐊᖏᖅᑐᖓ ᐊᐅᖏᖅᑕᐅᒍᑎᒥᓂᒃᑲ ᐊᕿᒍᓄᑦ ᖁᓕᓄᑦ ᐱᓯᒪᔭᐅᑌᔭᕐᓂᖏᓐᓂᒃ ᖃᐅᔨᓴᖅᑕᐅᖃᑦᑕᕐᓂᐊᕐᒪᑕ ᐆᒪᑎᓪᓗᓂᕐᒧᑦ, ᓱᑲᖃᓗᕐᐊᕐᓂᕐᒧᑦ, ᐋᓐᓂᐊᖃᕐᓂᕐᒧᑦ, ᐊᓯᖏᓐᓄᓪᓗ ᓱᕈᕈᑕᐅᕙᑦᑐᓄᑦ, ᓂᕆᔭᐅᖏᓐᓄᒃ ᖃᐅᔨᓴᑎᐅᓂᐊᕐᒪᑕ ᑭᓯᐊᓂ ᐊᓯᖏᓐᓅᖏᑦᑐᖅ.
ᐄ ᐋᒡᒐ

ᐊᔾᔨᖓᓂᒃ ᐊᑎᓕᐅᕐᕕᐸᑦ ᑐᓂᔭᐅᓂᐊᖅᑐᑎᑦ.

________________________ ᐊᑎᖓ ᐱᖃᑕᐅᔪᖅ ᖃᐅᔨᓴᖅᑕᐅᓂᕐᒥ

________________________ ᐊᑎᓕᐅᕈᓯᖓ ᖃᐅᔨᓴᖅᑕᐅᑉ

____/_____/_____ ᐊᕐᕌᒍᖓ/ ᑕᖅᑭᖓ/ ᐅᓪᓗᖓ

________________________ ᐊᑎᖓ ᐊᑎᓕᐅᖅᑐᒥᒃ ᑕᐅᑐᑦᑐᖅ

________________________ ᐊᑎᓕᐅᕈᓯᖓ ᑕᐅᑐᑦᑐᒥᓂᐅᑉ

____/_____/_____ ᐊᕐᕌᒍᖓ/ ᑕᖅᑭᖓ/ ᐅᓪᓗᖓ

________________________ ᐊᑎᖓ ᐊᖏᔪᖅᑳᖓᑕ ᖃᐅᔨᓴᖅᑎᐅᑉ ᑎᓕᔭᐅᓯᒪᔪᕐᑰᓐᓃᑦ

________________________ ᐊᑎᓕᐅᕈᓯᖓ ᖃᐅᔨᓴᖅᑎᐅᑉ

_____/_____/_____ ᐊᕐᕌᒍᖓ/ ᑕᖅᑭᖓ/ ᐅᓪᓗᖓ

ᖃᐅᔨᓴᖅᑕᒥᓂᐅᑉ ᑐᕌᕈᑎᖓ ᐅᖄᓚᐅᑎᖓᓗ ᐊᐅᓪᓚᑎᒃᓯᕙᐅᕙᒋᓛᖅᑕᖓ ᖃᐅᔨᓴᖅᑕᐅᒍᑎᒥᓂᖏᓐᓂᒃ:

ᑎᑎᖅᑲᕐᓂᐊᕙᖓ___________ ᐃᓪᓗᖓᑕ ᓈᓴᐅᑎᖓ_____________ ᐅᖄᓚᐅᑎᖓ____________

ᓄᓇᓕᖓᑕ ᐊᑎᖓ: ___________________________________

ᖃᐅᔨᓴᖅᐅᖃᑕᐅᔫᖅ ᐊᑎᖃᖃᓯᑎᒐᔪᑉᐸ ᐊᓯᐊᓂᒃ ᐊᑎᕐᒥ? ᐊᑎᒃᑲᓐᓂᖃᖅᐸᑦ, ᑎᑎᖃᓯᐅᑎᒍᑉ ᐊᑎᙵᖃᓯᐅᑎᖁᑕᖓ:

____

ᑐᓴᐅᒪᔭᐅᒋᐊᓕᒃ ᐊᐅᓚᕐᓯᔪᓄᑦ ᖃᐅᔨᓴᕐᓂᕐᒥᒃ: ᖃᐅᔨᓴᖅᑕᐅᑉ ᑲᒪᒋᔭᐅᔾᔪᑎᖏᓐᓂᒃ ᓇᓗᓇᐃᒃᑯᑕᖅ ᓂᐱᑎᒍᑉ ᐅᕗᖓ

INFORMED CONSENT – Adults

Inuit Health Survey: *"Qanuippitali: How about us, how are we?"*

Please circle your answers:

1. I have read the consent form or have had some one read it to me and I have received a copy of it.

YES NO

2. I have been offered a chance to ask questions and I agree to participate in the Inuit Health Survey. **YES NO**

3. I agree that the medical results and tests (such as blood pressure, blood tests showing health risk) will be provided to Health and Social Services to arrange for follow-up medical evaluation. **YES NO**

<u>International Inuit Health Survey</u>

I agree to participate in the International Inuit Health Survey and understand that I will be asked to participate in a follow-up evaluation within 7 years and that a nurse will review my medical record to verify if I have a chronic disease now and to review if any chronic disease developments have occurred within a 7 year follow-up.

YES NO

I agree that a sample of blood will be kept for 10 years and will be used for analyses of markers of heart disease, diabetes, cancer, effects of contaminants, dietary indicators, and <u>not</u> for other analyses. **YES NO**

*You will receive a signed copy of this form to keep.

___________________________	_____________________________	____/____2008
Name of participant	Signature of participant	dd / mm
___________________________	_____________________________	____/____2008
Name of witness	Signature of witness	dd / mm
_____________________________	_____________________________	____/____2008
Name of Principal Investigator or designated representative	Signature of Investigator	dd / mm

Participant’s address where results are to be sent and phone number:

Box #: _____________   House #: _____________   Phone #: _______________________

Name of community: ______________________________________

Does participant commonly use another name? If so, list other name used:

____

Note to Quality Control Team: Place label from individual checklist below