THE UNIVERSITY OF THE WEST INDIES
AT ST. AUGUSTINE, TRINIDAD AND TOBAGO

A Research Paper

Submitted in partial requirements

for HUEC 3012

of

The University of the West Indies

Title: Differences in the Intake of Foods high in Fats, Sugars and low in Antioxidants in Woman with Polycystic Ovary Syndrome and a Control Group

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Year Submitted: 2010

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ACKNOWLEDGEMENT

The completion of this project was made possible by Dr. Nichols, the Head of Department for Agricultural Economics & Extensions. Thank you to all of my family and friends who aided me in gathering data for this project.

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Abstract

The main objective of this study was to test the hypothesis that Women with Polycystic Ovary Syndrome (PCOS) have a higher intake of foods high in fats and sugars and a lower intake of foods high in antioxidants than women without Polycystic Ovary Syndrome. The study design was that of a case control study. Seventy three (73) women between the ages of 16 – 40 years were recruited to participate in the study. Of the 73 participants 43 represented women who had been clinically diagnosed with PCOS, and 33 participants represented normal women who had no medical history of PCOS. The findings of the study revealed that women with PCOS did have a higher intake of foods high in fats and sugars, and a lower intake of foods high in antioxidants than women without PCOS. The PCOS group had a higher rate of consumption high glycemic index foods than the non-PCOS group. More women with PCOS consumed fried foods, sugary snacks, and flour based products like pasta, roti and baked goods. It was also shown that women with PCOS had higher BMI's and waist circumference measurements, with a strong correlation between BMI and waist circumference ($p = 0.03$). When compared with the controls it was found that women with PCOS did consume more fats and sugar and lower levels of antioxidants than women without the syndrome, and there was also a tendency to foods rated as high glycemic index scale.

Introduction

Prevalence of Polycystic Ovarian Syndrome

Throughout the world Polycystic Ovarian Syndrome, or PCOS as it is commonly known, is described as a common and perplexing endocrine disorder of women in their reproductive years with a prevalence of up to 10% (Bhagel et al, 2010). Worldwide between 5% - 10% of all women have some degree of Polycystic Ovarian Syndrome frequently co-existing with obesity and diabetes. As much as 90% of women with irregular menses are affected by PCOS. This syndrome has been found to have a significant impact on the quality of life of women not only in the reproductive years but also in the ensuing years leading into menopause. (Dunaif et al, 2001; Elsenbruch et al, 2003).

In the United States PCOS showed a prevalence of between 4% - 12% (4) as many as 5 million American women are affected, whereas a prevalence of 6.5% - 8% was noted in various European studies (Azziz et al, 2004; Knochenhauer et al, 1998). Accurate prevalence figures are difficult to source due to the lack of consensus regarding diagnosis (Hart et al. 2004). There has been no substantial or conclusive evidence to date to confirm that factors such as ethnicity, race or locality influence prevalence, mainly due to the lack of research and exploration into such areas.

It has been reported by the IVF Fertility Center in Trinidad that as much as 1 out of every 4 young women have been diagnosed with Polycystic Ovaries (Trinidad & Tobago IVF Fertility Centre, 2010), as opposed to the estimated 1 out of every 15 ratio expressed on a worldwide basis.(Legro et al. 2001). In a recent interview Dr. Catherine Minto-Bain (a doctor with the IVF Fertility Center) stated in an article that though presently there exists no statistics to suggest the prevalence of PCOS amongst Trinidadian women she believes the levels to be high. (Binto, 2010). The figures reflecting prevalence may in actuality be much higher as several studies have revealed that many women are unaware they are affected by the syndrome.

Defining Polycystic Ovarian Syndrome

Polycystic Ovarian Syndrome was first described by two American gynecologists, Irving F. Stein, Sr., and Michael L. Leventhal, in the year 1953 and was referred to as the Stein-Leventhal syndrome. Their research suggested an association between polycystic ovaries, signs of hirsutism and amenorrhea. (Stein et al, 1935)

Several other names such as functional ovarian hypernadrogenism, ovarian hyperthecosis and sclerocystic ovary syndrome have been proposed over the years but very rarely used. The medical overview of Polycystic Ovarian Syndrome is ever evolving as the underlying pathophysiology of the syndrome is to date not entirely understood. (Azziz et al, 2004). The NIH/NICHD consensus workshop held in 1990 recognized a patient as having PCOS if the all of the following diagnostic criteria were present; hyperandrogenemia (clinical or biochemical) and infrequent/irregular ovulation (oligo-ovulation), excluding all other causes of polycystic ovaries. During a meeting held in Rotterdam in 2003 this consensus was revised and Polycystic Ovaries was added to the list of diagnostic criteria, however, a patient only has to present with two of the three criteria to be diagnosed with the syndrome. (Rotterdam, 2004). Worldwide Polycystic Ovarian Syndrome is viewed in several ways; it has been referred to as:

- The most common endocrinopathy plaguing women of reproductive age
- The most common metabolic abnormality present in women of reproductive age
- A very common reproductive order amongst women of reproductive age

All of the above have credibility as PCOS is noted as a collection of various symptoms, signs and endocrine disturbances, which reinforces the point that it is heterogeneous in nature (Balen et al. 1995).

Pathogenesis of the Syndrome

Polycystic Ovarian Syndrome acquired its name from the presence of multiple ‘poly’ cysts (immature follicles) inside the ovaries that are present on examination by ultrasound. Several physiological inconsistencies have been identified as possible underlying mechanisms for the syndrome:

- Elevated luteinizing hormone (LH) levels due to increased secretion by the pituitary gland as a result of increased secretion of the gonadotrophin releasing hormone (GRH)
- Increased androgen production by the ovaries
- Increased levels of insulin resulting in hyperinsulinemia and insulin resistance

Increased luteinizing hormone levels coupled with hyperinsulinemia effectively means an increase in androgen production by the ovaries (namely the ovarian theca cells). It is highly unlikely that there is any one single cause for the onset of PCOS. The pathophysiology of the disease could be a combination of both environmental factors and genetic predisposition (Dunaif et al. 2001). There has been recent development in genetic research that suggests PCOS could be the result of a complex genetic trait disorder (Frank et al. 2001)

Symptoms of Polycystic Ovary Syndrome

Not all women affected by the syndrome exhibit the clinical and biochemical features that define PCOS. (Hart et al, 2004). Manifestations of the symptoms associated with the syndrome are so varied that a person may present one, all or a combination of any of the following symptoms; acne, hirsutism, hair loss (male pattern baldness), dandruff, skin tags, acanthosis nigricans , anovulation/oligo-ovulation, ovarian cysts, obesity, infertility, insulin resistance, hyperinsulinemia, high blood pressure, high cholesterol and/or hypertension. It should be duly noted that not all women exhibiting Polycystic Ovaries present with the features that define the syndrome, however, polycystic ovaries is the defining feature of PCOS in ceratin populations namely the UK and the vast majority of Europe (Balen, 1999). Features that accompany PCOS, such as obesity, hyperinsulinemia and hyperandrogenaemia can add increased risk of future co-morbidities such as diabetes, cardio vascular disease, metabolic syndrome and various types of cancer (Rajkowha et al. 2000; Balen, 2001). Other long term health consequences include pregnancy and seizure disorders.

Effect of Diet on Polycystic Ovary Syndrome

Several of the symptoms and comorbities associated with PCOS, discussed above, are affected by dietary intake both during the onset and developmental phases. Increase in body weight and tendency to obesity both play active roles in the development and worsening of the syndrome. Diet also has a massive impact on the management of chronic non-communicable disorders, of which, several of these disorders serve as comorbidites to PCOS.

Dietary intake monitoring and regulation is crucial in ensuring some measure of control is exercised in maintaining healthy lifestyles that will promote regulation and balance of hormone production, blood sugar levels, blood pressure levels and blood lipid levels. Trends in eating habits have evolved considerably over the last two decades, especially as a result of the Nutrition Transition. (Popkin, 1994) This occurrence is defined by increased intake of fats and simple carbohydrates while there is significant decrease in intake of fruits and vegetables. The resulting situation is one in which there is a massive shift from foods rich in antioxidants, vitamins and minerals to those foods high in fats and simple sugars. It has been reported that due to decreased intake of fruits and vegetables an estimated 2.7 million deaths occur yearly worldwide (Fitzroy Henry 2006). Such dietary behavior is a huge risk factor for overweight and obesity, and other chronic diseases such as CVD and diabetes. It is possible that this dietary shift and its resultant increase in prevalence of obesity and other chronic diseases have also served to increase the risk factors associated with the prevalence of PCOS.

Impact of Polycystic Ovary Syndrome on Quality of Life

Physiological Impact

PCOS has had an increasingly immense impact over the years on the health related quality of life of women affected by the syndrome, both in their reproductive years and following well into menopause. In a recent study, conducted by Ching etal in Australia it was found that the reported, via a health questionnaire (SF-36) HRQL for women with PCOS was lower than that of both women without PCOS

and women with obesity. The aforementioned finding has been found to be consistent in several other studies measuring HRQL of women with PCOS.

With such severe symptoms and comorbidities such as cancer, CVD and diabetes (with increased risk of gestational diabetes) the health of many affected women has become compromised with promise of future decline as a result of the syndrome. Cardiovascular disease and cancer, according to the Global Health Council has the highest mortality rate globally in women 44 years and over. Increases in incidence of PCOS could mean increased risk of mortality for the world female population. PCOS is one of the leading causes of infertility which can range between as low as 35% to as high as 94% in various populations due mainly to anovulation (Goldzieher et al. 1963, Franks et al. 1995, Guzick et al. 1998). Prevalence of obesity and overweight can range as high as 50% - 60% in women with PCOS (Wright et al, 2004), which is also a significant risk factor for CVD and diabetes.

Psychological Impact

The greatest impacts of the syndrome have been demonstrated on a physiological, psychological and emotional level. Studies have shown that various symptoms of the syndrome can cause significantly elevated levels of emotional stress (Eggers et al. 2001) and psychological distress (Elsenbruch and Hahn etal). The psychological morbidity of women with PCOS is higher than that of the general population (Barth et al, 1993). One study title cited PCOS as "The Thief of Womanhood", the women in this study affected by PCOS saw themselves as 'freakish', 'abnormal' and 'not proper women' (Kitzinger et al. 2003).

In today's world where body image is an integral part of self perception certain symptoms of the syndrome such as hirsutism, obesity and acne can cause low self esteem and depression. Himelein etal reported that women with PCOS experienced greater body image dissatisfaction and were more prone to depression than women with infertility and women without PCOS or infertility. There is research to suggest that health related quality of life can differ depending on ethnicity (Schmid et al. 2004), however, greater research is required in this area. Women with PCOS had a lower mental health score than patients

with other chronic ailments (Mallon et al. 1999). There have also been reports of increased occurrence of emotional and psychological problems at work, affecting overall productivity (Paulson et al 1988; Downey et al 1989).

Economic Impact

Increasing levels of obesity and chronic diseases have already placed a heavy burden worldwide with 70 billion dollars being spent in 1995 alone to meet costs incurred as a result of obesity (Guilliford et al). It is also estimated that annually TT $10.7 is spent in Trinidad to facilitate admittance of persons with diabetes. Increase in incidence of PCOS and resulting comorbities will only add significantly to the already existing social and economic burden of chronic non-communicable diseases.

As a result of these findings it is necessary to investigate and assess factors that are related to PCOS prevalence. This study will attempt to assess the dietary aspect of the syndrome, i.e. effect of certain dietary components on PCOS status, as dietary intake is also a risk factor for obesity, CVD and diabetes status all of which are associated with PCOS. It will also attempt to investigate how BMI and waist circumference are related to PCOS prevalence. No research was found indicating that studies have been conducted in Trinidad to assess the association between dietary intake and PCOS status, and anthropometric data as relates to PCOS.

The bulk of this research paper will provide a detailed account of the literature review, methodology, results and ensuing discussion. The methodology provides a written account of the structure and method of data collection, whilst identifying the tools to be used in assessing the data collected. All collected data will be organized and processed in the form of tables illustrated in the results section of the paper, which will provide the basis for the discussion. The discussion will address whether the study was successful in achieving the objectives, and to what extent the results obtained conform to and are supported by information presented from research already conducted.

This study will provide information that would justify the basis for more in depth, invasive studies. The information obtained from this study could be very useful in identifying certain key factors that may

be instrumental in increasing the risk of PCOS. At the very least this study should aid in providing us with a clearer picture of the way Trinidadian women with the syndrome should treat with it on a dietary and anthropometric level so as to decrease the prevalence of the symptoms and reduce the risk of associated risk factors and resulting comorbidites. All of the studies reviewed were conducted mainly in the United States and Europe, no research was found that indicated such studies had been conducted in Trinidad.

Literature Review

Polycystic ovarian syndrome over the last twenty years has shown increasing prevalence in the female population worldwide. In years past PCOS was unknown to many as an existing medical condition, however, it has in recent times earned the title of 'Hidden Epidemic'. With increased awareness of this syndrome efforts have been intensified through research to understand the syndrome and its underlying pathogenesis and pathophysiology. There has also been extensive research into dietary intake and various dietary components and the effect/contribution to PCOS development and status. Several research papers have also been published illustrating the relationship that exists between variance in anthropometric data and genetics as relates to PCOS. This study will assess how dietary intake, namely intake foods high in fats, sugars and antioxidants vary in women affected and unaffected by PCOS, whilst also seeking to determine the association between BMI and waist circumference and PCOS in Trinidadian women. The literature review will provide an overview of various other research conducted lending insight into existing information about known relationships between dietary intake/ anthropometric data/genetics and PCOS.

Dietary Intake and PCOS

Nutrition has been cited as "one of the major lifestyle factors related to the development of non-communicable diseases" (Puska, 2002). A cohort study conducted on a University Campus in the United States investigated the difference in dietary intake between 30 women with PCOS and 27 health controls. Age, race and BMI were similar for both groups. Data collection was done via a food questionnaire and a 4 day food record. This study measured fasting sera for glucose and insulin concentrations. There was no significant difference in total consumption of energy, macronutrients, micronutrients and high glycemic index foods between the two groups. However, it was discovered that women with PCOS did intake higher quantities of certain high glycemic index foods. There was no evidence to support that diet composition affected fasting insulin concentration or variances in glucose to insulin ratios. (Douglas et al, 2006).

Several studies have been conducted with regards high glycemic index foods and how intake affects women with PCOS. In a very recent study conducted in Australia, a group of Australian researchers sought to measure the effects of a low glycemic index diet on women with PCOS as compared to the effects of a regular healthy diet. During this study a group of 96 obese and overweight premenopausal women were split into two groups and were followed for a period of 12 months or until they lost 7% of their body weight. One group followed a low glycemic index diet and the other a regular healthy diet, with clinical and biochemical assessments being conducted throughout the study. At the end of the study of the 96 original participants only 46 remained. However, it was observed that women who had followed the low glycemic index diet did experience more beneficial changes in health that those of the control group. The researchers were able to conclude that, "To the best of their knowledge, this study provides the first objective evidence to justify the use of low-GI diets in the management of PCOS". (Marsh et al, 2010).

Several other studies have established that there is definitely a relationship between glycemic index of foods, carbohydrate intake and PCOS. In a study measuring the effects of a low-carbohydrate, ketogenic diet on the polycystic ovary syndrome it was found that women on a low carbohydrate, ketogenic diet showed significant reductions in weight and regularization of hormone levels in obese women with PCOS. (Mavropoulos et al, 2005).

However, there are some studies that have provided conflicting evidence for intake of carbohydrates when compared with the aforementioned studies. One study conducted in the United Kingdom (UK) reported no that the energy intake of carbohydrates was actually lower for the group of women with PCOS than the control group. This study was designed to measure dietary intake, body composition and physical activity levels in women with PCOS and an age and weight matched control group. (Barr et al, 2008). This study was the first study of its kind to be conducted in the UK and the results were consistent with those of a similar study conducted in the US (Wright et al, 2004). Wright et al. conducted a study in the US amongst a group of women with PCOS and an age matched control group, in which dietary intake, physical activity and obesity were measured and compared for both groups. The results of this study also

reflected no significant difference in dietary intake in carbohydrates. (Wright et al, 2004). Barr et al. did however, demonstrate in the results that a significantly higher amount of energy was obtained from fat, and more specifically an overall higher intake of saturated fats for the group of women with PCOS as opposed to the control group.

Several studies have reported increased intake of fats in women with non communicable diseases. Fat intake is an area of concern for women with PCOS, as research has shown that dietary fat is instrumental in the development of obesity (Bray et al, 1998). It has been reported that 50%-60% of women with PCOS classify as obese or overweight (McKittrick 2002), as opposed to the 30% for the general population (Flegal et al 2002). In a study relating to the effects of insulin resistance on various endocrine related factors of PCOS (Nestler et al. 1999) it was observed that prior to the occurrence of symptoms associated with PCOS, patients were reported to have significant gains in weight. This observation supported a claim made in earlier years by Nestler et al. in 1989 that obesity played an active role in the development of PCOS. Due to the strong link between fat and obesity and obesity and PCOS it is necessary to determine how viable fat is as a causal factor.

One study sought to assess dyslipidemia (predictors, incidence and patterns) in women with polycystic ovaries. The control group for this study showed no signs of hyperandrogenism, were eumenorrheic and matched for BMI and age. The results of this study revealed that women with PCOS were found to have twice the incidence of dyslipidemia as compared to the control group, and high levels of triglycerides (TG). It was concluded that low levels of high density lipoprotein-C (HDL-C) was the main cause for the dyslipidemia and BMI had a significant impact on this condition as well.(Rocha et al, 2010). A similar study conducted in the United States also showed high levels of triglycerides in women with PCOS, however, they had higher levels of low density lipoprotein-C, HDL-C. This study comprised of non-Hispanic white women with PCOS and an ethnically matched control group. Each group was further sub-divided into obese and non obese. (Legro et al. 2001). Both studies showed significantly high levels of triglycerides for the women with PCOS. Generally people with high triglyceride levels often times have a high total cholesterol, that is, high levels of LDL ‘bad cholesterol’ and low levels of HDL ‘good

cholesterol'. (Gotto, 1998). This association existing between TG's and its effect on HDL and LDL levels has been found to significantly increase the risk of cardiovascular both in the general population and in women affected with PCOS. (Talbotta et al, 1998, Patel et al, 2004, Grotto, 1998). It is important to note that triglycerides may also independently be a risk factor for cardiovascular disease as shown by the meta analysis of 17 population based prospective studies.(Hokanson and Austin, 1996).

In another related cross sectional study conducted by Roa Barrios et al. in 2009 it was shown that women with PCOS showed had a higher frequency of triglycerides, fasting glucose and waist circumference. The researchers were able to conclude that higher levels of TG/HDL-C ratios were seen in women with PCOS and they also showed a close relationship between these ratios and waist circumference and insulin resistance.

There is a very close association between dyslipidemia and insulin resistance in women with PCOS. (Kalra et al, 2006). The study group consisted of 50 infertile women with PCOS and the researchers sought to investigate the relationship between insulin resistance with dyslipidemia and metabolic syndrome. The results showed that women with insulin resistance showed higher levels of cholesterol, low density lipoproteins and triglycerides with lower levels of HDL's. Prevalence of obesity and LDL's were also significantly higher for this group. (El-Manzy et al, 2010).

Dislipidemia and obesity are also integral risk factors for the development of cardiovascular disease in women with PCOS. In a study investigating overweight, PCOS affected women and the risk of cardiovascular disease it was found that this group had increased risk when compared to healthy controls. Both groups were age and BMI matched. (Meyer et al. 2005).

Another similar study conducted by Glueck et al. showed results consistent with that of the aforementioned study, that is, women with PCOS have increased risk of cardiovascular disease. This results of this study also showed that women with PCOS also had higher BMI and waist circumference than that of the health controls. (Glueck et al, 2009).

Several studies have shown through anthropometric measurement that women with PCOS have a tendency to higher BMI and waist circumference. One such study conducted by Wright et al. on a group

of women with PCOS and a healthy control group to assess dietary intake, physical activity, and obesity showed anthropometric data for both groups. It was reported that the women in the PCOS group had significant different means for BMI and waist-to-hip ratio than those of the control group. No significant difference in dietary intake was reported for this study. (Wright et al. 2003).

In a study conducted by Vrbíková et al. aimed at assessing how prevalent metabolic syndrome was in women with PCOS it was reported that the most frequent features in women with PCOS was increased waist circumference and BMI. There were also reports of increased HDL's and high TG's. They concluded that obesity especially of the abdominal type increased the risk for metabolic syndrome and insulin resistance. Another study reported that women with PCOS have been found to have increased deposition of adipose tissue in abdominal area as well as the waist and upper arms. (Zabuliene et al 2010).

PCOS is such that there is no one causal factor; there may be several underlying causes of the syndrome. Research has also taken the direction of studies into genetic factors that may be causal where PCOS is concerned. Certain variants in a gene known as the FTO (fat mass and obesity associated) gene appear to be correlated with obesity. In a recent study researchers attempted to assess how the FTO gene via the effect it has on adiposity affected the susceptiblity of women with PCOS to type 2 diabetes. The study revealed a significant association between the FTO gene and PCOS. This finding is based on the likelihood that due to the effect the FTO gene has on fat mass in affecting development of obesity it also contributes to the development of PCOS. (Barber et al. 2008).

The FTO gene has also been found to have marked effects on increases in BMI from youth into adulthood. As such the FTO gene may through its effect on obesity and thus PCOS may also be influential in the BMI status of women with PCOS. The FTO gene is only one of the areas of genetic research into PCOS. One study investigated the incidence of PCO in daughters of patients presenting with the syndrome. (Battaglia et al. 2002).

Though the underlying pathogenesis of PCOS is described as heterogeneous in nature, diet is considered an integral part of managing development of the syndrome. Several diets have been proposed for use by women with PCOS so as to improve their health related quality of life (Coffy et al. 2006). Most

of these diets encourage eating habits that entail low intake of high glycemic index foods and low fat consumption. Women are also encouraged to intake sufficient antioxidants so as to aid in the prevention of certain risk factors and effectively chronic diseases that can result in later years due to PCOS. (McKittrick, 2002). At the end of this study I am seeking to assess if dietary intake is significantly different for women with PCOS as compared with women unaffected by PCOS, and how nutrition affects PCOS. Can foods high in sugars and fats and low in antioxidant in women with PCOS predispose them to a future of chronic illnesses and resulting morbities?

Theoretical Framework

This study sought to compare the intake in women with PCOS as opposed to that in non-PCOS. This study was based on comparing intake and assessing differences. The causal agents being environmental, dietary and genetic factors can affect the process and result in one of two outcomes, increased risk of chronic disease or reduced risk of chronic disease.

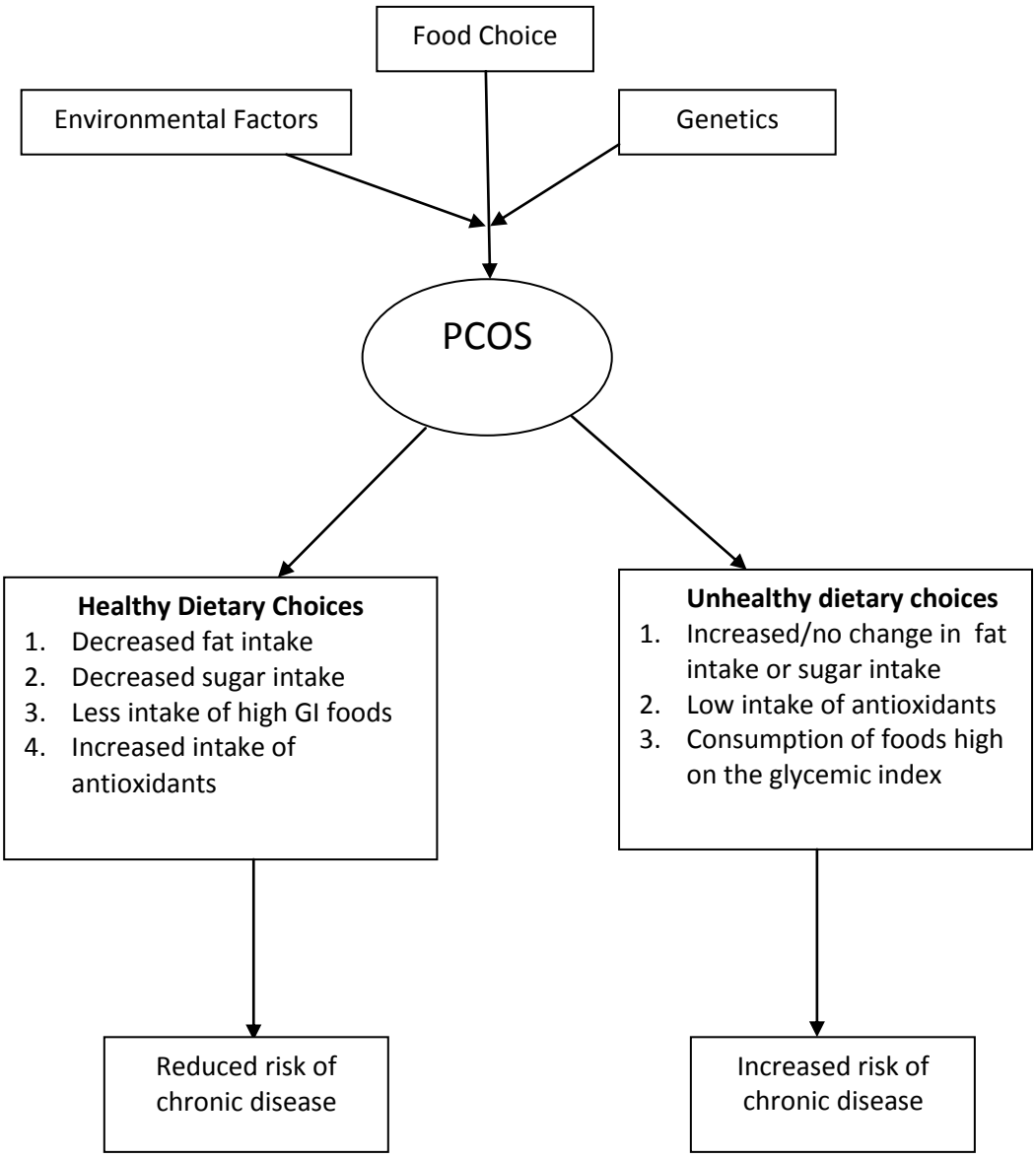

Figure 1: Illustration of Theoretical Framework associated with the Study

Methodology

Aims & Objectives of this Study

The main objective of this study is to compare the intake of foods high in fats, sugars and antioxidants in women with Polycystic Ovarian Syndrome as opposed to women without Polycystic Ovarian Syndrome. This study will also assess how BMI and waist circumference vary with the presence of Polycystic Ovarian Syndrome, while also determining if the incidence of Polycystic Ovarian Syndrome is increased due to genetic predisposition.

Study Population

The study comprised of 73 Trinidadian women chosen via convenient sampling to participate in this case-control study. Of the 73 participants 43 individuals represented the subset of the population clinically diagnosed with Polycystic Ovarian Syndrome whilst the remaining 30 represented the control group (i.e. participant not affected by Polycystic Ovarian Syndrome). The participants ranged in ages between 16 – 40 years with the highest frequency being 38.4% and ranging between 21 – 25 years of age. The study population constituted 39.7% Indo-Trinidadian, 31.6% Mixed Races, 23.3% Afro-Trinidadian, 2.7% Caucasian and 2.7% Chinese. The participants had varying levels of education, profession, location and marital status. Of the 73 participants there was a 100% response to the questionnaires administered, as well as to the 118 questions contained in the questionnaire.

Study Design

A type of observational study design known as a case control study design was used in constructing this study. The primary objective of the study is to assess the difference in intake levels of fats, sugars and antioxidants between women with Polycystic Ovarian Syndrome and women without Polycystic Ovarian Syndrome. Case control studies seek to establish if there is a link between a particular condition or disease (in this study that condition being Polycystic Ovarian Syndrome) and exposure to a particular factor (in this study that being frequency of certain dietary components) in retrospect. The two groups

represented in this study are the subset of the study population (i.e. women with Polycystic Ovarian Syndrome) and the control group (women without Polycystic Ovarian Syndrome). It should be duly noted that the fact that the outcome of each participant in both groups are already known at the time they are selected to participate in the study is what defines the study as retrospective.

This type of study design facilitates a quick and easy way to collect data for assessment, especially when facing stringent time constraints. However, as a result of the retrospective nature of the study design a significant level of bias can be introduced during recall, and incidence cannot be measured as incidence data cannot be generated. Although the study does have its drawbacks the conclusions generated from the study can be very instrumental in justifying more in depth, costly studies.

A questionnaire consisting of 118 questions was used as the data collecting instrument, a copy of which is included in the appendix (see Appendix C). To maintain the anonymity of the respondents each questionnaire was assigned a Patient Identification number. The questionnaire was geared mainly towards collecting dietary intake frequency data. However, a wide variety of other factors perceived to have a marked effect on PCOS status; including anthropometric, socio-demographic, personal medical history and familial medical history was also explored. The main variables in this study were the dependant variable - PCOS Status and the independent variables - Dietary Intake Frequency, BMI and Waist Circumference.

The anthropometric data was measured by the researcher, whilst all other data was self reported, with the frequency of intake of various foods being recalled over a six (6) month period. The dietary intake frequency questions were adapted from the NHANES Food Frequency Questionnaire developed by the United States National Cancer Institute, and adjusted as necessary to cater to the target groups.

Primary Hypotheses

Null Hypothesis: Women with Polycystic Ovarian Syndrome do not have a higher intake of foods high in fats and sugars and a lower intake of foods high in antioxidants than women without Polycystic Ovarian Syndrome.

Alternative Hypothesis: Women with Polycystic Ovarian Syndrome have a higher intake of foods high in fats and sugars and a lower intake of foods high in antioxidants than women without Polycystic Ovarian Syndrome.

Secondary Hypotheses

Null Hypothesis: Women with Polycystic Ovarian Syndrome do not have a higher BMI and a larger Waist Circumference than women without Polycystic Ovarian Syndrome

Alternative Hypothesis: Women with Polycystic Ovarian Syndrome have a higher BMI and a larger Waist Circumference than women without Polycystic Ovarian Syndrome

In this study confounding factors, such as obesity and diabetes, were measured and considered for so as to reduce the bias that could result in the conclusion.

Process of Sampling

Prior to the distribution of questionnaires to the study and control groups the questionnaire was pre-tested on a group of 10 individuals (n=10). This pilot study allowed for the assessment of the information contained in the questionnaire and the ease with which the pre-test group was able to understand and accurately respond to the questions. Following this assessment adjustments were made to rectify any discrepancies that could compromise the data collection process. All individuals selected to be participants in the study had to satisfy the following criteria:

Table 1: Criteria for selection of Participants for the Study and Control Groups

Participant Criteria	Study Group: PCOS	Control Group: NON-PCOS
Female	χ	χ
≤ 40 yrs of age	χ	χ
Clinically diagnosed with PCOS	χ	
No clinical diagnosis of PCOS		χ

The questionnaires were distributed to customers frequenting various business establishments along the East West Corridor, North District and Central District over a three week period. Three establishments were visited in each area each of which the researcher had prior association with the owners. Each establishment was visited once per week on varying days between the hours of 9:00am – 3:00pm. The purpose of the study was explained to the owners and verbal permission obtained to engage the assistance of the incoming customers. The snowball method of sampling was also employed in administering questionnaires to family, friends and associates. Based on the methods of sampling employed it is uncertain as to how accurate a representation the study group is of the affected population.

The questionnaires were administered by the researcher so as to reduce any inaccurate or biased responses due to lack of understanding of a particular question(s) and/or answer(s). Recording of answers by the researcher also ensured that no questions were left unanswered. The participants were asked to base their food, supplement consumption and physical activity responses on a six (6) month recall from the day of the study.

The anthropometric data was measured by the researcher at the time of administration of the questionnaire. Specific tools were used to obtain measurements for each type of anthropometric date (see appendix Table 2).

On approach the potential participants were greeted and apprised of the nature and purpose of the study. Women matching the criteria listed in Table 1 were kindly asked to participate in the study.

Permission was obtained to measure and record the various anthropometric measurements, after which the questionnaire was administered by the researcher and the responses recorded.

To ensure accuracy and uniformity of measurements each measurement was taken in accordance with a specific standardized procedure (see appendix Table 3).

Method of Data Analysis

The data collected comprised of both categorical and quantitative data. Analysis of the data obtained via administration of the questionnaires was analyzed using the Statistical Package for the Social Sciences (SPSS) (Version 17). Data analysis involved performing T-tests, Chi Square Tests and Pearson Correlations. The T-test was used in the analysis of the anthropometric data to illustrate the difference in means for height, weight, BMI and waist circumference, and whether there was significant difference between the two groups. Pearson correlations were also used to aid in the analysis of the anthropometric data to determine if there was any relationship (correlation) between BMI and waist circumference. The chi square test was used in the analysis of all categorical data, with the main focus on dietary intake. The dietary intake responses were re-coded to reflect categories of high and low rather than ordered ranges. Never to one serving per week was defined as low intake, while all other ranges above once per week was defined as high.

Results:

Table 2 shows the demographic characteristics of the both the non PCOS and PCOS groups. The study population comprised of 73 female participants. Of the 73 participants 58.9% of the study population represented women with PCOS and 41.1% represented women without PCOS. The results obtained showed that there was no statistical significance with regards to difference in age, ethnicity, marital status, education or profession between the two groups.

The majority of women comprising the study population were found to be between 21-25 years of age (38.4%), Indo-Trinidadian (39.7%), located along the east-west corridor (41.1%), single (43.8%), educated to a secondary level (45.2%), have jobs classed at a professional level (28.8%) and reported a monthly household income in excess of $8000.00 (41.1%).

Although no statistical significance was noted for age between the two groups; however, the PCOS group had a total of 62.8% ranging between 21-30 years of age.

Although there was no statistically significant difference in profession between the two groups 39.5% of the PCOS group had jobs classed at a professional level while only 13.3% of the non-PCOS group had jobs classed at a professional level. The minority of women in both the non PCOS and PCOS groups were housewives, with frequencies of 6.7% and 7% respectively.

There was a significant difference ($p = < 0.05$) between the non PCOS and PCOS groups in terms of location. The majority of respondents in each group were located along the east-west corridor, with 46.7% in the non PCOS group and 37.2% in the PCOS group. Whilst, 20% non PCOS and 20.9% PCOS were located in north Trinidad, 23% non PCOS and 7% PCOS were located in south Trinidad and 10% non PCOS and 34.9% PCOS were located in central Trinidad.

Both groups also showed significant differences in monthly household incomes ($p = < 0.05$). As much as 53.5% of the PCOS group reported incomes of in excess of $8000.00, whilst 23.3% of the non PCOS reported incomes of in excess of $8000.00. The majority (36.7%) of the non PCOS group reported

earnings ranging from $3001.00 to $5000.00, while 9.3% of the PCOS group reported incomes in this bracket. Very small percentages of non PCOS and PCOS reported no steady income i.e. 6.7% and 11.6% respectively and incomes ranging below $2000.00 that is, 6.7% and 2.3% respectively.

Table 2: Demographic Characteristics of the study population by PCOS status

Variables	Frequency (%)		p-value
	Non- PCOS (n=30)	PCOS (n=43)	
Age			
16-20	4(13.3)*	4(9.3)	
21-25	12(40.0)	16(37.2)	
26-30	2(6.7)	11(25.6)	0.248
31-35	7(23.3)	9(20.9)	
36-40	5(16.7)	3(7.0)	
Ethnicity			
Afro-trinidadian	9(30.0)	8(18.6)	
Indo-trinidadian	13(43.3)	16(37.2)	
Caucasian	1(3.3)	1(2.3)	0.490
Chinese	1(3.3)	1(2.3)	
Mixed Races	6(20.0)	17(39.5)	
Location			
North Trinidad	6(20.0)	9(20.9)	**0.041****
South Trinidad	7(23.3)	3(7.0)	

East-West Corridor	14(46.7)	16(37.2)	
Central Trinidad	3(10.0)	15(34.9)	
Status			
Single	17(56.7)	15(34.9)	
Married	9(30.0)	14(32.6)	
Divorced	1(3.3)	0(0.0)	0.107
Separated	1(3.3)	2(4.7)	
Common Law	2(6.7)	12(27.9)	
Education			
Primary	0(0.0)	0(0.0)	
Secondary	15(50.0)	18(41.9)	0.250
College/A'level	3(10.0)	11(25.6)	
University	12(40.0)	14(32.6)	
Profession			
Professional	4(13.3)	17(39.5)	
Technical	8(26.7)	6(14.0)	0.166
Clerical	8(26.7)	9(20.9)	
Student	8(26.7)	8(18.6)	

Housewife	2(6.7)	3(7.0)	
Income			
No steady income	2(6.7)	5(11.6)	
< $2000	2(6.7)	1(2.3)	
$2001 - $3000	4(13.3)	1(2.3)	0.009
$3001 - $5000	11(36.7)	4(9.5)	
$5001 - $8000	4(13.3)	9(20.9)	
> $8000	7(23.3)	23(53.5)	

*Figures in brackets represent percentage whilst other numbers represent frequency of responses by individuals in each category

****Figures demonstrating significant p-values are bolded.** For p-values of p≤ 0.05 there is significance.

Table 3 illustrates the variations in anthropometric measurements measured by the researcher in both the PCOS and non-PCOS groups. Statistical analysis of the anthropometric measurements resulted in significant p-values for weight, BMI and waist circumference, confirming that there were significant differences in both groups for each variable. There was no statistical significance in the difference in height amongst the PCOS and non-PCOS groups, with means ranging between 163.32 ± 7.31 and 163.91 ± 8.21. The p-value was highly significant for BMI, i.e. p = **<0.001.**

The PCOS group measurements in weight reflected a mean weight of 73.54 ± 20.19 while that of the non-PCOS group reflected a mean weight of 61.51 ± 9.56. Mean BMI was 27.00 ± 6.94 and 22.88 ± 3.22 for the PCOS and non-PCOS groups respectively. There was a mean waist circumference of 34.13 ± 5.51 in the PCOS group and 31.65 ± 4.18 in the non-PCOS group. Further analysis of BMI and waist

circumference resulted in a correlation coefficient of 0.793 and p = **<0.001,** with a coefficient of determination of 0.629. Therefore, there was a strong positive correlation between BMI and waist circumference with 62.9% of the variation in BMI explained by variations in waist circumference.

Table 3: Anthropometric Measurements vs PCOS Status

Variables	Frequency	Mean	p-value
Height			
PCOS	43	163.32 ± 7.31	0.746
Non-PCOS	30	163.91 ± 8.21	
Weight			
PCOS	43	73.54 ± 20.19	**0.002***
Non-PCOS	30	61.51 ± 9.56	
BMI			
PCOS	43	27.00 ± 6.94	**<0.001**
Non-PCOS	30	22.88 ± 3.22	
Waist circumference			
PCOS	43	34.13 ± 5.51	**0.032**
Non-PCOS	30	31.65 ± 4.18	

***Figures demonstrating significant p-values are bolded.** For p-values of p≤ 0.05 there is significance.

Table 4 shows how the PCOS status varies with intake of various fruits and vegetables. Statistical analysis revealed that there was no statistically significant difference in intake of the majority of fruits and vegetables.

Only two of variables exhibited significant difference between the two groups. There was a significant difference in the intake of citrus between the two groups, 60.5% of the PCOS group had low levels of intake of citrus, whilst 63.3% of the non-PCOS had high levels of intake of citrus. The same was true for carrots and pumpkin, 53.5% of the PCOS group had a higher frequency of intake than the non-PCOS group of which 70% reported having a lower frequency of intake.

Table 4: PCOS Status vs Frequency of Intake of Fruits & Vegetables

Variable		NON-PCOS (n=30)	PCOS (n=43)	p-value
Citrus	Low	11(36.7)*	26(60.5)	**0.045**
	High	19(63.3)	17(39.5)	
Bananas	Low	17(56.7)	22(51.2)	0.643
	High	13(43.3)	21(48.8)	
Apple/Pear/Mango	Low	18(60.0)	31(72.1)	0.279
	High	12(40.0)	12(27.9)	
Paw Paw/Melon	Low	24(80.0)	33(76.7)	0.741
	High	6(20.0)	10(23.3)	
Prunes	Low	27(90.0)	35(81.4)	0.312
	High	3(10.0)	8(18.6)	

Raisins	Low High	29(96.7) 1(3.3)	39(90.7) 4(9.3)	0.321
Fruit Juice	Low High	10(33.3) 20(66.7)	13(30.2) 30(69.8)	0.779
Fruit Servings per month	Low High	14(46.7) 16(53.3)	15(34.9) 28(65.1)	0.311
Tomatoes	Low High	16(53.3) 14(46.7)	20(46.5) 23(53.5)	0.566
Green Leafy Vegetables	Low High	17(56.7) 13(43.3)	25(58.1) 18(41.9)	0.900
Peas & Beans	Low High	17(56.7) 13(43.3)	15(34.9) 28(65.1)	0.065
Cruciferous Vegetables	Low High	22(73.3) 8(26.7)	37(86.0) 6(14.0)	0.175
Mixed Vegetables	Low High	21(70.0) 9(30.0)	29(67.4) 14(32.6)	0.817
Fresh Salad	Low High	17(56.7) 13(43.3)	19(44.2) 24(55.8)	0.294

Provision	Low High	28(93.3) 2(6.7)	39(90.7) 4(9.3)	0.687
Carrots & Pumpkin	Low High	21(70.0) 9(30.0)	20(46.5) 23(53.5)	**0.047**
Eggplant	Low High	29(96.7) 1(3.3)	40(93.0) 3(7.0)	0.501
Vegetable Servings per month	Low High	11(36.7) 19(63.3)	9(20.9) 34(79.1)	0.138

* Figures in brackets represent percentage whilst other numbers represent frequency of responses by individuals in each category

Figures demonstrating significant p-values are bolded. For p-values of $p \leq 0.05$ there is significance.

Table 5 illustrates how PCOS status varies with frequency of intake of animal products. There was no statistically significant difference between the groups, for none of the variables was $p \leq 0.05$. Women in both groups demonstrated equally low frequencies of intake of whole milk, skimmed milk, ice cream, vegetarian cheese, processed meats, hot dogs, beef, lamb, fish canned meat and sea food. Both groups had high intake frequencies for eggs, butter and chicken. Although there was no implied statistical significance of differences in both groups for cheese it should be noted that 43.3% of the non-PCOS group had a high frequency of intake whereas 60.5% of the PCOS group had a high frequency of intake.

Table 5: PCOS Status vs Frequency of Intake of Animal Products

Variable		NON-PCOS (n=30)	PCOS (n=43)	p-value
Skimmed Milk	Low	19(63.3)*	26(60.5)	0.804
	High	11(36.7)	17(39.5)	
Whole Milk	Low	17(56.7)	31(72.1)	0.172
	High	13(43.3)	12(27.9)	
Ice Cream	Low	21(70.0)	32(74.4)	0.677
	High	9(30.0)	11(25.6)	
Cheese	Low	17(56.7)	17(39.5)	0.149
	High	13(43.3)	26(60.5)	
Vegetarian Cheese	Low	24(80.0)	35(81.4)	0.882
	High	6(20.0)	8(18.6)	
Eggs	Low	13(43.3)	17(39.5)	0.746
	High	17(56.7)	26(60.5)	
Butter	Low	12(40.0)	19(44.2)	0.722
	High	18(60.0)	24(55.8)	
Processed Meats	Low	22(73.3)	22(51.2)	0.057
	High	8(26.7)	21(48.8)	

Hot Dogs	Low High	20(66.7) 10(33.3)	31(72.1) 12(27.9)	0.619
Burger & Meatballs	Low High	23(76.7) 7(23.3)	39(90.7) 4(9.3)	0.099
Chicken	Low High	2(6.7) 28(93.3)	1(2.3) 42(97.7)	0.358
Beef	Low High	26(86.7) 4(13.3)	39(90.7) 4(9.3)	0.588
Lamb	Low High	27(90.0) 3(10.0)	36(83.7) 7(16.3)	0.443
Fish	Low High	24(80.0) 6(20.0)	36(83.7) 7(16.3)	0.683
Canned Meat	Low High	28(93.3) 2(6.7)	38(88.4) 5(11.6)	0.479
Seafood	Low High	28(93.3) 2(6.7)	40(93.0) 3(7.0)	0.959

* Figures in brackets represent percentage whilst other numbers represent frequency of responses by individuals in each category

****Figures demonstrating significant p-values are bolded.** For p-values of $p \leq 0.05$ there is significance

Table 6 illustrates how PCOS status varies with frequency of intake of various staples. There was no evident significant difference between groups for cereals, bread, pasta, potatoes or crackers. Intake of bread, pasta and potatoes was generally high for both the PCOS and non-PCOS groups. For rice however, there was significant difference between the two groups ($p \leq 0.05$), 83.7% of the PCOS group reported having high frequency of intake of rice whereas 60% of the non-PCOS group reported high frequency of intake.

Table 6: PCOS Status vs Frequency of Intake of Staples

Variable		NON-PCOS (n=30)	PCOS (n=43)	p-value
Cereal	Low	15(50.0)*	24(55.8)	0.624
	High	15(50.0)	19(44.2)	
Cooked Cereal	Low	25(83.3)	37(86.0)	0.750
	High	5(16.7)	6(14.0)	
Roti	Low	23(76.7)	27(62.8)	0.209
	High	7(23.3)	16(37.2)	
Bagels Muffins Pita	Low	25(83.3)	41(95.3)	0.086
	High	5(16.7)	2(4.7)	
Rice	Low	12(40.0)	7(16.3)	**0.023**
	High	18(60.0)	36(83.7)	

Pasta	Low High	20(66.7) 10(33.3)	27(62.8) 16(37.2)	0.734
Potato	Low High	18(60.0) 12(40.0)	32(74.4) 11(25.6)	0.192
Crackers	Low High	16(53.3) 14(46.7)	21(48.8) 22(51.2)	0.705

* Figures in brackets represent percentage whilst other numbers represent frequency of responses by individuals in each category

**__Figures demonstrating significant p-values are bolded.__ For p-values of $p \leq 0.05$ there is significance

Table 7 shows how PCOS status varies with intake of various beverages. There was significant statistical difference in the intake of coffee with 58.3% of the non-PCOS and 76.7% of the PCOS group having a high frequency of intake. Although there was no implied statistical significance the frequency of intake of soda was slightly higher in the PCOS group with 69.8% of the PCOS group having high frequency of intake as opposed to the 56.7% in the non-PCOS group.

Table 7: PCOS Status vs Frequency of Intake of Beverages

Variable		NON-PCOS (n=30)	PCOS (n=43)	p-value
Soda	Low	13(43.3)*	13(30.2)	0.250
	High	17(56.7)	30(69.8)	
Coffee	Low	14(46.7)	10(23.3)	**0.036**
	High	16(58.3)	33(76.7)	
Alcohol	Low	25(83.3)	37(86.0)	0.750
	High	5(16.7)	6(14.0)	
Water	Low	4(13.3)	1(2.3)	0.067
	High	26(86.7)	42(97.7)	

* Figures in brackets represent percentage whilst other numbers represent frequency of responses by individuals in each category

Figures demonstrating significant p-values are bolded. For p-values of $p \leq 0.05$ there is significance

Table 8 shows how PCOS status varies with frequency of intake of sweets and baked goods. Intake of sweets and baked goods was generally low for each group. Intake of sweet syrups and jams showed significant difference, with 26.7% of the non-PCOS group and only 2.3% of the PCOS group having high frequency of intake.

Table 8: PCOS Status vs Frequency of Intake of Sweets/Baked Goods

Variable		NON-PCOS (n=30)	PCOS (n=43)	p-value
Chocolate	Low	20(66.7)	27(62.8)*	0.734
	High	10(33.3)	16(37.2)	
Candy	Low	22(73.3)	35(81.4)	0.413
	High	8(26.7)	8(18.6)	
Sweet Syrup/Jams	Low	22(73.3)	42(97.7)	**0.002**
	High	8(26.7)	1(2.3)	
Baked Goods	Low	23(76.7)	31(72.1)	0.661
	High	7(23.3)	12(27.9)	
Cake	Low	26(86.7)	35(81.4)	0.550
	High	4(13.3)	8(18.6)	

* Figures in brackets represent percentage whilst other numbers represent frequency of responses by individuals in each category

Figures demonstrating significant p-values are bolded. For p-values of $p \leq 0.05$ there is significance

Table 9 illustrates how PCOS status varies with intake of fast food and home fried foods. Both groups reflected low frequencies of intake for each variable. There was a statistically significant difference between the two groups for reported intake of burgers. 90.7% of the PCOS group reported low frequency of intake, whilst 70% of the non-PCOS group reported low frequency of intake.

Table 9: PCOS Status vs Frequency of Intake of Fast Food/Home Fried

Variable		NON-PCOS (n=30)	PCOS (n=43)	p-value
Pizza	Low	28(93.3)	37(86.0)	0.327
	High	2(6.7)	6(14.0)	
Fried Foods	Low	23(76.7)*	29(67.4)	0.392
	High	7(23.3)	14(32.6)	
Burgers	Low	21(70.0)	39(90.7)	**0.023**
	High	9(30.0)	4(9.3)	
Home Fried Foods	Low	19(63.3)	23(53.5)	0.402
	High	11(36.7)	20(46.5)	

* Figures in brackets represent percentage whilst other numbers represent frequency of responses by individuals in each category

Figures demonstrating significant p-values are bolded. For p-values of $p \leq 0.05$ there is significance

Figure 2 shows the distribution of the ages at which PCOS onsets. The most frequently occuring ages of onset fell with in the range of 21 – 25 years, this represented 49% of the PCOS group. Of the women affected by PCOS in this study 65% of those women experienced onset of PCOS between the ages of 15 – 25 years of age. Only 16% of the PCOS group experienced onset in later years, 26 – 30 years.

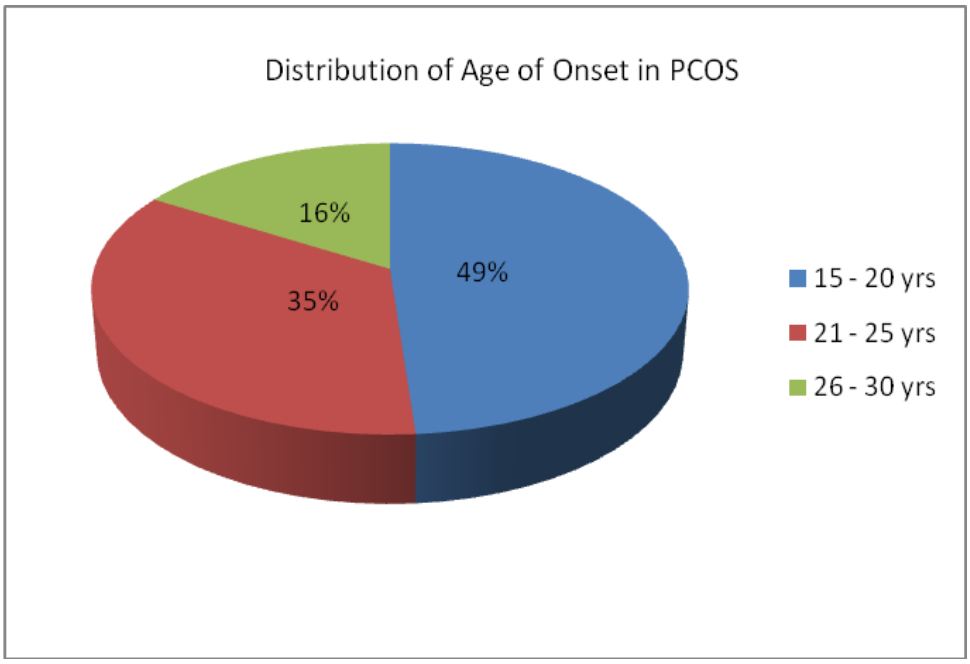

Figure 2: Illustrates the Distribution of Age of Onset in PCOS

Figure 3 shows how the frequency of various chronic diseases and risk factors vary in both the non-PCOS and PCOS groups. A minority of the women in the non-PCOS group reported being affected by chronic diseases and risk factors. For thyroid dysfunction, hypertension, elevated cholesterol levels, diabetes, renal (kidney) disease, cancers/tumors/growths, colonic dysfunction, endometriosis, fibroids, cervical cancer and ovarian cysts there was a reported 0% for each variable for women in the non-PCOS group.

There was a statistically significant difference in the occurence of thyroid dysfunction ($p \leq 0.05$) between both goups.

It was reported that 18.6% of the women in the PCOS group reported being affected, while there was no reported occurrence in women of the non-PCOS group. Diabetes was found to affect 25.6% of the women in the PCOS group while there were no reported cases of diabetes in the non-PCOS group, with a p-value of ($p \leq 0.05$) indicating statistical significance. There was a strong statistically significant difference between both groups for presence of ovarian cysts ($p < 0.001$). For women in the PCOS group 55.8% reported having ovarian cysts as opposed to the non-PCOS group in which there were no reported cases. Although the difference in both groups was not significant for endometriosis and cancer/tumors/growths, there was a reported 9.3% and 11.6% in women if the PCOS group, while there were no reported cases in the non-PCOS group.

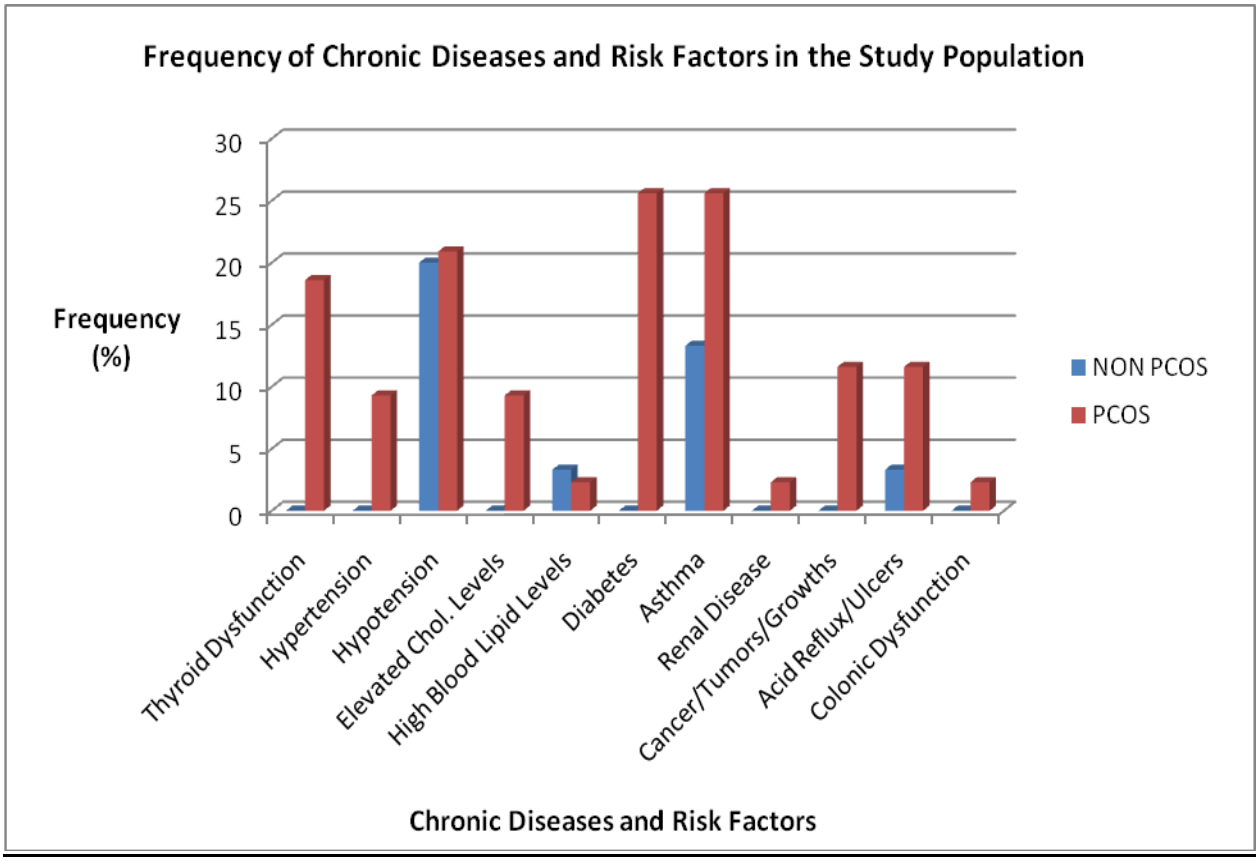

Figure 3: Illustrates the Frequency of People in the PCOS and non-PCOS Groups with Chronic Dieases and Risk Factors

Figure 4 illustrates the frequency of familial medical history of non-communicable diseases in both the non-PCOS and PCOS groups. Of the PCOS group 83.4% reported having a familial history of PCOS, as opposed to 16.7% reported for the non-PCOS group. Further analysis revealed a p-value of ($p < 0.001$) indicating that there was a significant statistical difference between both groups for familial history of PCOS.

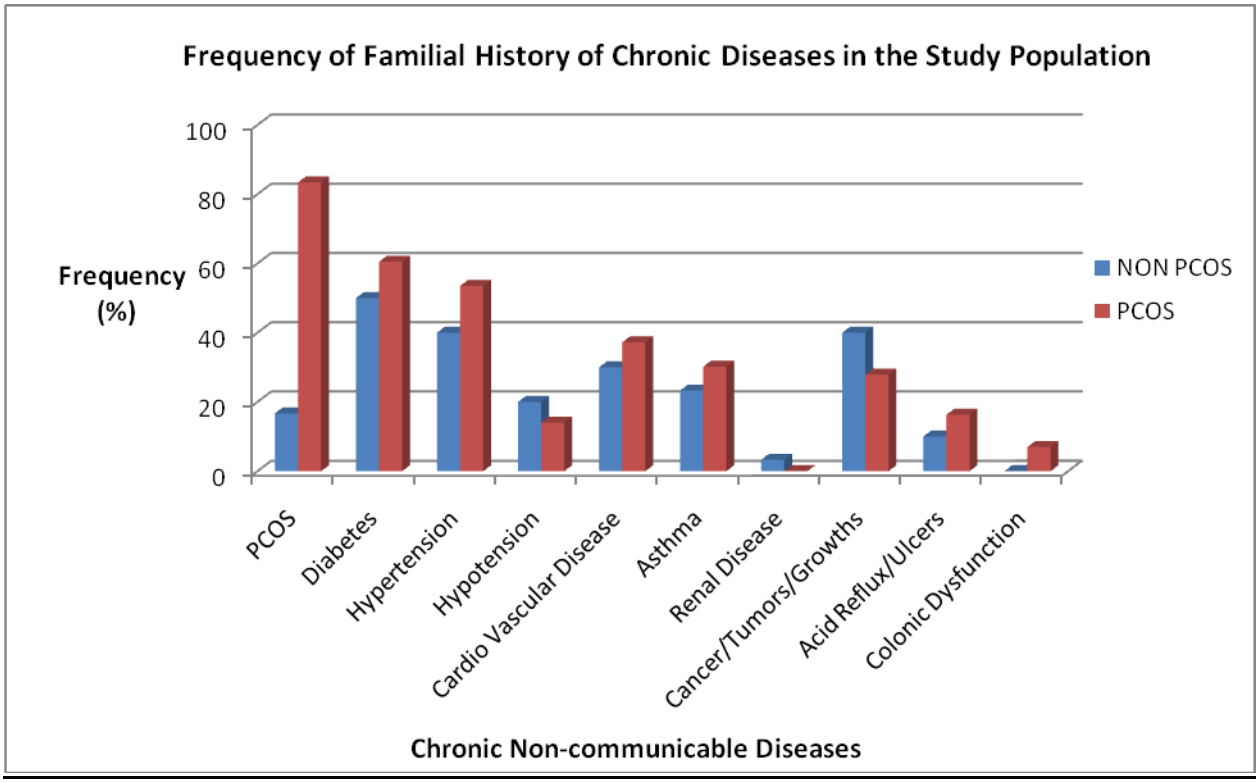

Figure 4: Illustrates the Frequency of People in the PCOS and non-PCOS Groups with a Familial History of Chronic Dieases

Discussion

Polycystic ovarian syndrome (PCOS) is currently afflicting 1 out of every 10 women worldwide and the underlying causes of the syndrome are still not entirely understood. This primary purpose of this paper is to assess how fat, sugar and antioxidant intake vary in women with PCOS in comparison to that of women without PCOS. This paper also sought to identify differences in anthropometric data between the two groups. The findings of this study revealed that there exists some degree of difference in dietary intake between women with PCOS and women without PCOS; there was also significant evidence to suggest that there is strong variation in BMI and waist circumference between the two groups.

Dietary Intake

There has always been an existing awareness that there is a link between diet and PCOS, due to the nature of the syndrome and the resulting symptoms and comorbidities. However, there remains the question of the degree to which dietary intake is instrumental in the development and worsening of the condition as PCOS is defined by several underlying conditions and symptoms. (Baghel et al, 2010). There is no single cure or corrective measure by which PCOS can be prevented, treated or controlled. Dietary and lifestyle habits coupled with environmental and genetic factor are all very instrumental in the onset and development of PCOS. (Balen et al, 2001).

Antioxidant Consumption

The findings of this research paper were consistent with those presented by Douglas et al in a study conducted in 2006. Consumption of fruits and vegetables amongst the PCOS and non-PCOS group was found to be relatively similar for most of the foods presented in the questionnaire. Although not pronounced the women in the PCOS group consumed smaller quantities of citrus, apples, pears, bananas and mangoe as compared to that of the non-PCOS. For citrus, cruciferous vegetables and mixed vegetables the intake of non-PCOS women was significantly higher than that of women with PCOS. This indicated that there was a slightly higher intake of certain fruits and vegetables by women without the

syndrome. Fruits and vegetables are considered the main source of micronutrients, and micronutrients are described as antioxidants in the edible form. (Amer. Chem. Soc. 2004). Overall women without PCOS reported a higher intake of vegetables per month. Thus there was an overall higher intake of foods high in antioxidants in women without PCOS. None of the studies referenced in this research paper presented data to support this finding, as the majority of studies reported intake of fruits and vegetables to be relatively the same in both groups, with very minimal variance. (Barr et al. 2008, Douglas et al. 2006). The area of antioxidant intake as relates to PCOS requires significantly more exploration before a firm conclusion can be reached.

Fats, Sugar Consumption

The foods in this study that constituted the fat and sugar components of dietary intake were meats and animal products, staples, sweets/baked goods and fast/home fried foods. These foods are recognized as having high levels of saturated fat, trans fats and high sugar content. (How foods affect triglycerides, 2010). Women with PCOS were found to intake higher quantities of whole milk, cheese, processed meats, eggs, lamb and canned meats. There was also a higher level of intake of foods high in sugar and significantly higher daily usage of raw sugar in women with PCOS than those without. Women with PCOS were found to intake much higher levels of processed meat and significantly higher levels of pizza, fried foods and home fried foods, as was the case in a similar study which showed higher intake of fried foods, namely potato, in women with PCOS. (Douglas et al. 2006). These foods have been found to have extremely high levels of fat. (Hu et al. 2001).

Women with PCOS also showed higher levels of consumption of pumpkin, cereal, roti, and pasta with a significantly higher level of consumption of rice that seen with the non-PCOS group. All of which rank high on the glycemic index. (Glycemic Index of Foods, 2004). There was therefore an overall higher intake of high glycemic index foods in women with PCOS, as was seen in other related studies. (Douglas et al. 2006.)

For women with PCOS there was an overall higher consumption of foods high in fats (namely saturated fats) and sugars, these findings were in keeping with those from studies conducted by Barr et al in 2008 and Wright et al in 2004. Both studies reflected significantly higher amounts of energy obtained from fats, namely saturated fats, in women with PCOS. Such tendencies may be directly related to the occurrence of dyslipidemia in women with PCOS, resulting in high levels of low density lipoproteins, low levels of high density lipoproteins and cholesterol. (Legro et al. 2001, Rocha et al, 2010).

The consumption of alcohol and water in both groups were quite similar with slightly higher intakes of water and soda for women with PCOS. However, results showed a marked higher consumption of coffee in women with PCOS as opposed to women without the condition. There was no evidence in past research to suggest a link between caffeine and PCOS; however, caffeine has been shown to have an effect on hormone levels and diabetes.

BMI and Waist Circumference

Overweight and obesity have been identified as PCOS related, it is still unclear as to whether obesity causes or aids in the development of PCOS. Most of the research suggests that it is both a causative and developmental factor in PCOS. (Hill et al. 2000). Overweight and obesity have a strong correlation with BMI and waist circumference.

The results of this study showed significantly higher weight, BMI and waist circumference in women with PCOS as opposed to the control group. There was a significantly strong correlation between BMI and waist circumference. Women with PCOS were found to have BMI's ranging between 21 – 33 kgs/m^2, therefore a high percentage of the women in the PCOS group classified as overweight or obese. Waist circumference in women with PCOS was found to average 34.13 inches as opposed to non-PCOS women who were found to average 31.65 inches. Women with waist sizes above 30 have a moderate risk of health related disease while those above 35 are at a high risk.

This finding is supported by similar finding by Wright et al. in a study conducted in 2003 which found women with PCOS to have much higher BMI and waist circumference when compared with that of non-

PCOS women, the majority of the women with PCOS also were categorized as overweight or obese. This finding was further supported by a study done to assess deposition of adipose tissue in women with PCOS and it was shown that women with PCOS have much higher levels of abdominal fat than women not affected by the syndrome. (Zabuliene et al 2010). Height was very similar for both the PCOS and non-PCOS group, there is no evidence to suggest that height impacts weight in any way.

Genetic Predisposition

An interesting point of note was the fact that there were very few women with PCOS that had a familial history; however, the mass majority of the women with PCOS reported having relative(s) with the condition. This was an extremely significant finding as it suggest a very strong genetic predisposition. This finding is in keeping with several studies that investigated genetic basis for certain clinical profiles of PCOS. (Legro et al. 1998, Gonzalez et al. 2003)

Strengths and Limitations of the study

The small size of the study sample and the lack of randomness in the selection process were two of the most significant limitations of this study. The small size of the study group resulted in tendencies where there could have been noted significance. Random samples allow for a better representation of the target population, inferences can be more confidently made about the entire population based on the study group. Due to the nature of the selection of the study group this allowed for selection bias. This study patterned after a case-control design and as such the respondents were called on to answer the questionnaire by recalling dietary intake, which left the study open to recall bias. It is possible that inconsistencies in data and resulting inferences were affected by incorrect recall.

However, an attempt was made to consider for confounding factors by measuring and recording all known probable factors so as to reduce bias in inferences made. The researcher administered the questionnaire personally and recorded all data, so as to ensure all questionnaires and questions were answered completely. This also allowed for clarification of any questions or answers that were confusing

to the respondent. In an attempt to eliminate some recall bias the researcher took actual anthropometric measurements at the time of administration of the questionnaire in accordance with standardized procedures so as to reduce measurement bias. These finding may not be an accurate representation of the women in Trinidad with PCOS, however, the results can be used to construct more in depth studies into PCOS in Trinidadian women.

Implications and Recommendations

Though not entirely accurate the results of this study can be used to guide women with PCOS about those dietary components that should be consumed in smaller amounts. It is also indicative of those foods that should be avoided where possible. This study suggests the need for education of women with PCOS to make the dietary choices that will promote health and not advance deterioration and risk of future onset of various chronic diseases. There is a dire need for research into diet and PCOS in the Caribbean due to the lack of research and information available on PCOS currently.

In conclusion, this study has shown that there are significant variations in fat, sugar and antioxidant intake in women with PCOS and women without PCOS. The results obtained from this study supports the hypothesis that women with PCOS do intake higher levels of fats and sugars and lower levels of antioxidants than women without PCOS. It was also very clearly defined in the results that women with PCOS do have higher BMI and waist circumference than women without the syndrome. However, due to the limitations of the study these findings cannot be soundly concluded, hence the need for additional research into diet and PCOS, especially in the Caribbean and Trinidadian women.

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Appendix

Appendix A: Instruments used by researcher to measure Anthropometric Data

Anthropometric Data	Measuring Tool	Units
Height	Height Chart	Centimeters (cms)
Weight	Omron Digital Scale	Kilograms (kgs)
BMI	Formula: $BMI\ (kg/m^2) = \frac{\text{weight (kgs)}}{\text{height } (m^{2})}$	Kgs/m^2
Waist Circumference	Measuring Tape	Inches (In)

Appendix B: Procedures for conducting individual anthropometric measurements

Anthropometric Measurement	Method of Measurement
Height	1. Participants with thick soled shoes were asked to remove them, along with any headwear present 2. Participants were instructed to stand against the height chart on the wall with their heads, shoulders, rear ends and heels touching the wall 3. Ruler was gently pressed down on the top of the head to line up with the reading on the chart and the measurement read
Weight	1. Participants were asked to remove all weighty/bulky objects and shoes 2. Participants were instructed to place feet on foot pads on the scale with head straight and eyes forward 3. When a stable figure was displayed the reading was recorded
BMI	The pre-recorded height and weight readings were used to calculate the BMI using the following formula: $BMI\ (\ kg/m^2\ ) = \frac{\text{weight (kgs)}}{\text{height } (m^{2})}$
Waist Circumference	1. All excess clothing around the waist was removed 2. Participant were asked to stand straight 3. The measurement was taken about the navel