Research in obesity

Body mass index (BMI) Obesity is a medical condition defined as an accumulation of an excess of body fat that leads to many health problems and reduced life expectancy. Obesity usually associates with hypertension, diabetes, cardiovascular diseases (CVDs) and dyslipidemia (Grundy, 2004). Obesity is commonly measured by the Body mass index (BMI), which is calculated as the weight in kilograms divided by the square of the height in meters. Normal BMI is considered between 18.5 to 24.9. However, Overweight, obesity and extreme obesity are classified as the following: the BMI for overweight is 25.0 or greater and less than 30.0, obesity BMI is 30.0 or greater and lastly extreme obesity is BMI 40.0 or greater (Mendy, Vargas, Cannon-Smith, & Payton, 2017).

The prevalence and incidence of obesity are increasing rapidly to epidemic proportions in industrial areas as well as worldwide. It has increased three folds between 1975 and 2016 and more than 1.9 billion adults were overweight in 2016 (WHO, 2017). The cause of obesity Obesity results from energy imbalance between energy intake and energy usage at which both environmental and genetic factors play a role in its onset. Environmental risk factors include physical inactivity, consumption of fast foods, sugary food, and inadequate sleep. In addition, genetic factors contribute to the onset of obesity. Obesity could be due to monogenic; mutation in a single gene, or polygenic; mutations in more than one gene. Although those genes do not directly cause obesity, they contribute to its onset and development in parallel with other genetic alterations (O'Rahilly, Farooqi, Yeo, & Challis, 2003). Many studies that have provided data about obesity risk factors were focused on western countries more than any other areas. The western studies' conclusions do not necessarily apply to other population due to a variety of genetic and environmental factors.

Recent progress via genome-wide association studies (GWAS) has identified many additional genetic factors that appear to inflate the risk of disorders in some individuals (Zeggini et al., 2008). Studying different populations with different backgrounds such as Qatari population which has a small population size and high consanguinity will clearly provide a better understanding of obesity risk factors world widely. Consanguinity has reached 54% in Qatar, and obesity prevalence is more than 70%. The high incidence and prevalence of obesity have been due to lifestyle changes after discovering oil in the gulf area and increased wealth (Tomei et al., 2015). Development and building of cities have occurred rapidly which has changed the old lifestyle of traveling, to a sedentary lifestyle, which contributed to increased obesity. Recent GWAS studies have shown the relationship between genes and obesity, but it is not well known if those genetic markers influence similar risks in people of different descent (Larson et al., 2007).

Distribution of diseases related to alleles between different population is uneven especially for the recessive disorder such as monogenic obesity mutation in Leptin Receptor (Hindorff et al., 2009). The risk of a genetic marker identified by GWAS might vary according to the population being studied cause populations with different ancestry have a variability of allelic frequency (Meyre et al., 2009). Genetics of obesity Research in the field of genetics in the past two decades has shown that genetics play a role in obesity and early evidence came from twin studies. Twin studies aimed to determine the genetic component of obesity through producing

heritability estimates for obesity and obesity-related traits (Schousboe et al., 2003).

Twin studies are valuable because they allow for the comparison of the obesity concordance between monozygotic and dizygotic twins, where both twin pairs also share to some extent similar environment. Additional evidence of genetic contribution to obesity also came from adoption studies (Grant, 2014){Grant, 2014, the genetic of obesity}. Genetic studies of twins showed that identical twins have 70 to 90% of similarity of fat mass, while fraternal twins have 35 to 45% indicating that genetic does contribute to obesity (MacDougald, Hwang, Fan, & Lane, 1995).