

# Association between diabetes markers and cholesterol

Rabindra Nath Das\*



## Introduction

It is commonly known that the heart disease (HD) and diabetes mellitus (DM) are highly correlated with each other, and generally they co-exist [1-3]. DM is a well identified causal factor for cardiovascular disease (CVD), and it is a chronic progressive disease [4-6]. It was first illustrated over 37 years ago in the Framingham Heart Study that an increased risks of congestive heart failure in patients with diabetes [7]. Generally, the diabetes markers (according to American Diabetes Association (ADA 2013)) are the Haemoglobin A1c (HbA1c  $\geq 7.0$  mmol/L), fasting (no caloric intake for at least 8 hours) plasma glucose (FPG  $\geq 7.0$  mmol/L), 2-hour plasma ( $\geq 11.1$  mmol/L) during an oral glucose tolerance test, and random plasma glucose ( $\geq 11.1$  mmol/L). But for the cardiac disease diagnosis, there is no such well-known marker. In practice, total cholesterol (TC) is used as a heart disease marker, because TC is the main cause of atherosclerosis and CVD [8]. Note that TC is the total of very low-density lipoprotein (VLDL-C), low-density lipoprotein (LDL-C), and high-density lipoprotein (HDL-C). TC does not include Triglycerides, and it is below 200 (mg/dL) is known as normal.

The following queries (or hypotheses) arise as researcher problems. Is there any association between a diabetes marker and TC or its component? What are the effects of TC and its components on a diabetes marker? How are the TC and its components related with the diabetes markers? These answers are little known in the literature. The current report examines these answers based on some real data sets.

## Data set 1

The considered data set collection method and its description are displayed [1]. It includes 366 sample units, and 20 factors/covariates [1]. This data set can be found at <http://biostat.mc.banderbilt.edu/wiki/Main/DataSets?CGISESSID=10713f6d891653ddcbb7ddb9c9cfb79>. For present study, the factors/covariates are reproduced herein. The variables/factors of the present data set are total cholesterol (TC), stabilized glucose (STB.GL), glycosolated hemoglobin (GLYHB or HbA1c), gender (male=1, female=2), age, sample unit's weight (Weight), sample unit's height (Height), HDL-C, lipid ratio=cholesterol/HDL-C (Ratio), body mass index (BMI), 1st diastolic blood pressure (BP) (BP.1d), 1<sup>st</sup> systolic BP (BP.1s), location (1=Buckingham, 2=Louisa) (Location), frame (1=small, 2=medium, 3=large) (Frame), sample unit's waist measurement (Waist), sample unit's hip measurement (Hip), index of fat distribution (=waist/hips ratio) (IFD), 2nd diastolic BP (BP.2d), 2nd systolic BP (BP.2s), postprandial time when labs were drawn (TIM.PN).

The above data set 1 contains glycosolated hemoglobin (HbA1c) as the only diabetes marker. In addition, it contains TC, HDL-C and lipid ratio (cholesterol/HDL-C). Analysis of HbA1c is given in Das [1]. From this analysis, it is noted that the mean HbA1c is directly related with TC ( $P=0.002$ ). It indicates that as TC increases, HbA1c increases. Mean HbA1c is directly correlated with HDL-C ( $P=0.082$ ), indicating that as HDL-C increases, HbA1c increases. The mean HbA1c is inversely correlated with the joint interaction effect of TC & HDL-C

Department of Statistics, University of Burdwan, Burdwan, West Bengal, India

\*Author for correspondence: rabin.bwn@gmail.com

( $P=0.032$ ), indicating that HbA1c increases as the joint effect of TC & HDL-C decreases. Analysis of TC also supports that mean TC is directly correlated with HbA1c ( $P=0.053$ ) [1].

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### Data set 2

The present considered data set is included from Efron et al. [9] which contain 442 subjects with 11 factors /covariates, and it can be found at the site: <http://www4.stat.ncsu.edu/~boos/var.select/diabetes.html>. For the present study, the factors/covariates are displayed as follow. The factors/covariates are total cholesterol (TC), HDL-C, LDL-C, triglyceride (TG), serum concentration of lamorigine (LTG), blood serum glucose (GLU), gender (male=0, female=1), average BP, body mass index, age, a quantitative measure of DM disease progression (QDMDP). The determinants of blood serum glucose and QDMDP have been derived [10,11].

The above data set 2 contains diabetes markers as random blood glucose (GLU), and QDMDP. In addition, it contains TC, HDL-C, LDL-C, triglyceride (TG), serum concentration of lamorigine (LTG). Analysis of GLU is given in Das [10]. From the analysis [10], it is observed that the mean random blood glucose (GLU) is positively partially associated with TC ( $P=0.1683$ ), implying that the mean GLU increases as the TC increases. The mean GLU is positively associated with TG ( $P=0.0594$ ), indicating that the mean GLU increases as the TG increases. In addition, the mean GLU is directly correlated with LTG ( $P=0.0003$ ), indicating that the mean GLU increases as the LTG increases. The variance of GLU is directly insignificantly correlated with HDL-C ( $P=0.2642$ ).

Analysis of QDMDP is given in Das [11]. From the analysis [11], it is observed that the mean QDMDP is inversely significantly associated with TC ( $P<0.0001$ ), implying that the mean QDMDP increases as the TC decreases. The mean QDMDP is directly correlated with LDL-C ( $P<0.0001$ ), implying that the mean QDMDP increases as the LDL-C increases. The mean QDMDP is directly associated with LTG ( $P<0.0001$ ), implying that the mean QDMDP increases as the LTG increases. The variance of QDMDP is inversely associated with TC ( $P=0.0038$ ), implying that the variance of QDMDP increases as TC decreases. Note that the mean and variance of QDMDP have the identical association (negative) with TC. The

variance of QDMDP is directly correlated with LDL-C ( $P=0.0041$ ), implying that the variance of QDMDP increases as LDL-C increases. Again the mean and variance of QDMDP have the identical association (positive) with LDL-C. Also the variance of QDMDP is inversely associated with TG ( $P=0.0705$ ), indicating that the variance of QDMDP increases as TG decreases.

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### Data set 3

The considered data set is taken from Das [3], which has 64 subjects with 21 factors /variables. For the present study, the factors/variables are given herein. The factors/variables are gender, age, height, weight, body mass index, lifestyle, obesity, smoking habit (no=0, yes=1), dietary habits like eating in outside (no=0, yes=1), TC, fasting plasma glucose level (PGL), LDL-C, HDL-C, serum triglyceride (TG), fasting serum insulin (FI), family BP (no=0, yes=1) (BP), types of oil consumption, family history of DM (no=0, yes=1) (DM), history of past illness (no=0, yes=1), history of any drug intake (no=0, yes=1), hypertension with CVD (no=0, yes=1) (CHD).

The above data set contains fasting plasma glucose level (PGL) as a diabetes marker. It also contains TC, HDL-C, LDL-C and TG. The analysis of PGL is given in Das [3], which shows that the mean fasting plasma glucose level (PGL) is directly partially associated with TC ( $P=0.11$ ), indicating that the mean PGL increases as TC increases. The mean PGL is also directly associated with LDL-C ( $P=0.01$ ), indicating that the mean PGL increases as LDL-C increases. In addition, the variance of PGL is inversely associated with serum triglyceride (TG) ( $P<0.01$ ), indicating that the PGL variance increases as TG decreases.

The above three data sets have been analyzed using both the joint Log-normal and gamma models. These three data sets include three diabetes markers such as HbA1c, random blood glucose, and fasting plasma glucose level. In addition, the data set 2 contains a quantitative measure of DM disease progression which is not clearly described in Efron et al. [9]. From these three data sets it is clearly observed that each diabetes marker is always associated with TC, and its components. Moreover, these diabetes markers are also associated with serum triglyceride and serum concentration of lamorigine. Medical practitioners, diabetes patients and all individuals should care on

diabetes markers, total cholesterol and its components, and along with serum triglyceride, serum concentration of lamorigine.

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### Conflict of interest

The author confirms that this article content has no conflict of interest.

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