Diabetes

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Content

Introduction

With the recent increase in our standards of living, most of the human population will face many diseases caused by obesity and overconsumption, one of which is diabetes. As of now, over 422 million people are suffering from diabetes, with over 1.6 million people dying from it.[1] It is clear that diabetes is a serious issue that needs to be addressed.

Our project, Diabetes, aims to solve this issue, as it aims to discover the factors of diabetes via researching for the prevalence of diabetes and its correlations to other diseases, and present such data in the form of an interactive tableau site to the general public and medical institutions to spread awareness on diabetes and possibly solve it on a global scale.

Our project's focus is to use data science to discover the prevalence and diseases correlated to diabetes, process such data and release it to the public via an interactive tableau site.

Literature Reviews

Predicting risk of type 2 diabetes in England and Wales: prospective derivation and validation of QDScore

Hippisley-Cox, J., Coupland, C., Robson, J., Sheikh, A., & Brindle, P. (2009). Predicting risk of type 2 diabetes in England and Wales: prospective derivation and validation of QDScore. *Bmj, 338*(Mar17 2). doi:10.1136/bmj.b880

This study looks into the risk of type 2 diabetes in England and Wales for people of different ethnic groups and between the age of 25-79. This paper discusses the use of a new risk prediction algorithm for assessing the risk of developing type 2 diabetes among a very large and unselected population derived from family practice, with appropriate weightings for ethnicity and social deprivation.The algorithm (QDScore) is based on variables that are readily available in patients' electronic health records to enable it to be readily cost effective.

An open cohort of patients aged 25-79 years at the study entry date is identified and calculated the crude incident rates of type 2 diabetes according to their age, ethnic group, and deprivation in fifths. Using a Cox proportional hazards model for the data, the authors estimate the coefficients and hazard ratios associated with each potential risk factor.Fractional polynomials is then used to obtain a non-linear risk relation. Interactions between each variable and age and between smoking and deprivation were tested and significant interactions were included in the final model. Multiple imputation were used to replace missing values for smoking status and body mass index. Multiply imputed datasets by using Rubin's rules were also fitted to the model to combine estimates of effects

and standard errors of estimates to allow for the uncertainty caused by missing data.

In the article, the authors concluded that a marked difference is found in the age standardised incidence rates of type 2 diabetes by deprivation, with a more than twofold difference for women when comparing the most deprived fifth with the most affluent fifth. Age standardised rates were also found to be significantly higher for men in every ethnic group compared with the white reference group, except for Chinese men. In women, age standardised incidence rates were higher for every group compared with the white reference group. This data are then combined into the QDScore which becomes a simple method to access the risk of diabetes.

To ensure their conclusion is valid, the authors validated the sample in another sample of separate practices and discovered that the QDScore has good discrimination and explains approximately 50% of the total variation in times to diagnosis of diabetes.The D statistic, which is a measure of discrimination appropriate for survival type data, was higher in the QDScore algorithm than some other researches. However, in order to support the issues raised, interactions between the variables and risk of diabetes were tested while only the significant interactions were included. In other to ensure there is sufficient data to provide a trend, multiple imputations were set to fill in the missing body mass index and other data.

Nonetheless, the author assumed that the patients who were not given insulin before the age of 35 have type 1 diabetes while the others have type 2 diabetes. Thus, this might affect the results as there might be patients having type 2 diabetes being given insulin before 35.

Despite so the authors work gave us a new method to predict the risk of type 2 diabetes in a very large and unselected group, with appropriate weightage for each factor. The algorithm also provides the risk of diabetes

through different variables such as age, ethnic group, social deprivation and Body Mass Index.

Overall, the article enabled me to gain insight on use of data analysis tools in the field of healthcare and how it could be used to assess risk of chronic diseases such as diabetes. The article also provides insights on some methods in data analysis such as using fractional polynomials to model interactions between the variables that are non-linear.

β-Cell Deficit and Increased β-Cell Apoptosis in Humans With Type 2 Diabetes

Butler, A. E., Janson, J., Bonner-Weir, S., Ritzel, R., Rizza, R. A., & Butler, P. C. (2003). β-Cell Deficit and Increased β-Cell Apoptosis in Humans With Type 2 Diabetes. Diabetes, 52, 102-110. Doi: https://doi.org/10.2337/diabetes.52.1.102

The purpose of conducting this research was to find out whether it was possible to reverse diabetes to a degree rather than just palliate glycemia.

There are 3 key questions the authors are addressing. Firstly, whether beta-cell mass increases in humans with obesity as compared to lean nondiabetic humans. Secondly, whether beta-cell mass decreases in humans with type 2 diabetes as compared to age-, sex-, and weight-matched non-diabetic humans. And lastly, what was the mechanism subserving any deficit in beta-cell mass in type 2 diabetes.

The research was conducted by obtaining human pancreatic tissue from Minnesota, and processing pancreatic tissue by staining sections of the tissue by Congo Red and immunochemistry to check for hematoxylin and insulin. Furthermore, sections of tissue were analyzed for replication by immunochemistry for Ki67 and for apoptosis using the TdT-FragEL Kit. This was done in order to determine the weight of the pancreas and hence the beta-cell mass. Density of nuclei per insulin-positive area was also measured to ensure that any changes in the relative beta-cell volume was not due to changes in in the size of individual beta-cells. The tissues were digitally photographed and examined to manually count the number of nuclei present in the insulin-stained area in order to compute the ratio of nuclei per insulin-positive area. Frequency of the beta-cell apoptosis and replication events were divided by the relative beta-cell volume to provide a

comparison of these events relative to beta-cell volume per case as the relative beta-cell volume differed among groups.

The author concluded that relative beta-cell volume and hence the beta-cell mass decreases in both obese and lean humans with type-2 diabetes compared with their non-diabetic age and weight-matched counterparts. The authors also ascribe the mechanism for the decrease in beta-cell mass to an increase in the frequency of beta-cell apoptosis with the rate of new islet information being unaffected. The implication for prevention of type-2 diabetes is that strategies that avoid the increased frequency of beta-cell apoptosis were most rational. Also, in people with established type-2 diabetes, inhibition of the 3- to 10-fold increased rate of apoptosis may lead to restoration of beta-cell mass since islet neogenesis appears intact.

This can be observed where obese humans with IFG and type-2 diabetes had a 40% and 63% deficit in relative beta-cell volume compared with non-diabetic obese subjects. There was also an increased percentage of beta-cell-positive duct cells in obese versus lean cases but no difference between the obese non-diabetic and type-2 diabetic cases or lean type-2 diabetic and non-diabetic cases. When the frequency of apoptosis was expressed as the frequency of beta-cell apoptosis divided by the relative beta-cell volume, then the frequency of apoptosis was a 3-fold increase in obese diabetic cases and a 10-fold increase in lean cases of type-3 diabetes versus their respective control groups. Islet amyloid was present in 81% of obese cases with type-2 diabetes compared to 10% in obese non-diabetic cases, and the extent of birefringence in affected islets was higher in type-2 diabetic cases, both lean and obese, when compared to their non-diabetic counterparts.

The authors assumed that humans' beta-cell mass is dynamic and regulated, similar to mice, with input of beta-cells from new islet information and beta-cell replication within islets and output from beta-cell senescence.

They also assumed that the increase in beta-cell mass in obese non-diabetic humans was due to a response to insulin resistance. Furthermore, they assume that the extent of this increase in the markedly obese humans studied in this region is much lower than the increase observed in mice because the pancreatic weight in obese non-diabetic humans tends to be larger than lean non-diabetic humans.

By conducting this research, the author has furthered the studies in the mechanism of increased beta-cell apoptosis, as well as push research forward in the possibility of reversing diabetes to a degree rather than just palliate glycemia through improving the understanding of the relation between diabetes and beta-cells.

The author has provided me insight on how beta-cells impact diabetes. However, some improvements can be made to this research, such as researching whether results will differ based on region. Also, the of test subjects could be increased to improve reliability. By expanding our research to relation of beta cells and diabetes, we can examine data from other regions and hence try to improve on the current conclusion.

Type 2 Diabetes Mellitus: A Review of Current Trends

Olokoba, A. B., Obateru, O. A., & Olokoba, L. B. (2012). Type 2 Diabetes Mellitus: A Review of Current Trends. Type 2 Diabetes Mellitus: A Review of Current Trends. doi:10.5001/omj.2012.68

This study looks into the trend of Type 2 diabetes mellitus (DM) in which prevalence has been increasing steadily all over the world. The paper discusses the trend of type 2 diabetes being more common throughout the years, its relation to genetics and lifestyle, and methods to control and manage the condition. The paper also highlights the fact that there although is no cure in sight for type 2 diabetes, treatment modalities include lifestyle modifications, treatment of obesity, oral hypoglycemic agents, and insulin sensitizers like metformin, a biguanide that reduces insulin resistance, is still the recommended first line medication especially for obese patients.

The research was conducted by analysing data of the different factors that may be affecting prevalence rates of type 2 diabetes mellitus such as Lifestyle, Genetics, and Medical Conditions, screening and diagnosis, and Pharmacological Agents used to treat diabetes.

The Author concluded that type 2 DM is a metabolic disease that can be prevented through lifestyle modification, diet control, and control of overweight and obesity. Education of the populace is still key to the control of this emerging epidemic. Novel drugs are being developed, yet no cure is available in sight for the disease, despite new insight into the pathophysiology of the disease. Management should be tailored to improve the quality of life of individuals with type 2 DM.

This can be observed when there was significant reduction in the incidence of type 2 DM with a combination of maintenance of body mass index of 25 kg/m^2, eating high fibre and unsaturated fat and diet low in saturated and trans-fats and glycemic index, regular exercise, abstinence from smoking and moderate consumption of alcohol.

The author assumed that increased rate of childhood obesity between the 1960s and 2000 have led to the increase in type 2 DM in children and adolescents only using the weak correlation between them.

By conducting his research, the author has furthered research in the trends of type 2 diabetes with other factors and management of the disease.

The author has provided me insights about basic trends of type 2 diabetes and some of the factors affecting the rates of type 2 diabetes. The author also showed, using data, how the factors affects the prevalence rates of type 2 diabetes.

National, regional, and global trends in fasting plasma glucose and diabetes prevalence since 1980: systematic analysis of health examination surveys and epidemiological studies with 370 country-years and 2·7 million participants

Danaei, G., Finucane, M. M., Lu, Y., Singh, G. M., Cowan, M. J., Paciorek, C. J., . . . Ezzati, M. (2011). National, regional, and global trends in fasting plasma glucose and diabetes prevalence since 1980: Systematic analysis of health examination surveys and epidemiological studies with 370 country-years and 2·7 million participants. *The Lancet, 378*(9785), 31-40. doi:10.1016/s0140-6736(11)60679-x

The purpose of writing this research paper was to estimate trends and associated uncertainties by country and region in glycaemia and diabetes prevalence.This is done so as to understand the effects of diet and lifestyle within populations, assess the performance of interventions, and plan health services.

Hyperglycemia and diabetes are important causes of mortality and morbidity worldwide, through both direct clinical sequelae and increased mortality from cardiovascular and kidney diseases. With rising overweight and obesity, concern has risen about a global diabetes epidemic, with harmful effects on life expectancy and health-care costs. The current studies have hindered the ability to systematically examine trends due to changing diabetes definitions, false data pool representing country, and data pooled together from different years without adjustment for underlying trends. Hence, the authors wanted to carry out a consistent and comparable global analysis of trends in order to remedy this issue.

The authors did the research by obtaining data from health examination surveys and epidemiological studies (370 country-years and 2·7 million participants), then converting systematically between different

glycaemic metrics. For each sex, they used a Bayesian hierarchical model to estimate mean FPG and its uncertainty by age, country, and year, accounting for whether a study was nationally, sub nationally, or community representative.

The authors conclude that glycaemia and diabetes are rising globally, driven both by population growth and ageing and by increasing age-specific prevalences. Genetic factors associated with ethnic origin, fetal and early life nutritional status, diet quality, and physical activity might also affect glycaemic values and trends. Because high BMI is a risk factor for the two metabolic indicators, systolic blood pressure and total cholesterol, these differences probably arise from other determinants, including dietary composition and medical treatment. Effective preventive interventions are needed, and health systems should prepare to detect and manage diabetes and its sequelae. Primary prevention of dysglycemia will need weight control, physical activity, and improved diet quality. Such interventions are difficult to implement within populations and will not affect diabetes incidence in the short term. Therefore, health systems in most countries will inevitably have to develop programmes to improve detection and management of diabetes to slow progression to microvascular and macrovascular complications.

The authors supported this conclusion by presenting data, stating that in 2008, global age-standardised mean FPG was 5.50 mmol/L (95% uncertainty interval 5.37–5.63) for men and 5.42 mmol/L (5.29–5.54) for women, having risen by 0.07 mmol/L and 0.09 mmol/L per decade, respectively. Age-standardised adult diabetes prevalence was 9.8% (8.6–11.2) in men and 9.2% (8.0–10.5) in women in 2008, up from 8.3% (6.5–10.4) and 7.5% (5.8–9.6) in 1980. The number of people with diabetes increased from 153 (127–182) million in 1980, to 347 (314–382) million in 2008. They recorded almost no change in mean FPG in east and southeast Asia and central and eastern Europe. Oceania had the largest rise, and the highest mean FPG (6.09 mmol/L, 5.73–6.49 for men; 6.08 mmol/L,

5.72–6.46 for women) and diabetes prevalence (15.5%, 11.6–20.1 for men; and 15·9%, 12.1–20.5 for women) in 2008. Mean FPG and diabetes prevalence in 2008 were also high in south Asia, Latin America and the Caribbean, and central Asia, north Africa, and the Middle East. Mean FPG in 2008 was lowest in sub Saharan Africa, east and southeast Asia, and high-income Asia-Pacific. In high-income subregions, western Europe had the smallest rise, 0.07 mmol/L per decade for men and 0.03 mmol/L per decade for women; North America had the largest rise, 0.18 mmol/L per decade for men and 0.14 mmol/L per decade for women.

A few studies have examined global patterns of glycaemia and diabetes, but have not estimated past trends for all countries and regions. Other studies have assessed trends in specific countries or regions. A publication also estimated that there were 285 million people with diabetes worldwide in 2010, but some of the data were from specific occupational groups, communities with high obesity prevalence, or health-care facilities and practitioners, registries, and self-reported diabetes. Narrative reviews have stated that diabetes is rising in Asia and Africa, without addressing incomparable age groups in the studies included and other aspects of data comparability. Previous studies also had not distinguished between data that are nationally representative and those that are subnational or from specific communities. The authors final dataset included 370 country-years with 2·7 million participants and thus has improved on the current studies on global analysis of trends on diabetes, Hence, this helps health systems in most countries to develop programmes and improve detection and management of diabetes to slow progression for microvascular and macrovascular complications. They have hence successfully estimated trends and associated uncertainties by country and region to the highest accuracy.

The main limitation of the study is that despite extensive data seeking, many country-years still did not have data, especially in the 1980s and in some low income and middle-income countries. The absence of

data is reflected in wider uncertainty intervals. Hence, we can improve on this by including more recent data from these low and middle income countries to improve the accuracy of results. Further, the authors noted substantial incomparability in metrics of glycaemia in published data. Specifically, they had data for mean postprandial glucose, mean HbA1c, and diabetes prevalence using 18 different definitions. Although they systematically converted between different glycaemia metrics, the conversions led to increases in uncertainty intervals. Hence, we plan to gather more recent data with a more definitive and singular definition, and hence increase the accuracy of the results as it would decrease the uncertainty intervals.

Methods

Firstly, we used data from www.data.gov and www.moh.gov.sg. Since these data sources are from the Singapore Government, it is the most reliable in identifying data related to diabetes in Singapore. However, due to the lack of diabetes related data on these sites, we decided to manually collect these data before processing it in RStudio, followed by obtaining an easy to understand table or graph via Tableau.

As for acquiring datasets on diabetes at a global scale, we used data sources such as www.data.worldbank.org, www.who.int and www.idf.org.

Worldbank was used due to it providing free and open access to a supply of global development data. The datasets that we found on Worldbank also had the transfer to csv file option, which was quite convenient since .csv files could be read by RStudio, thus there was no need to scrape them via Python or to manually fill in Excel.

World Health Organisation's data was used as well since it is a part of the United Nations, and thus would be the most reliable in analysing diabetes and its correlation to other diseases such as Hypertension.

The International Diabetes Foundation was used as its networks to diabetes groups in almost every Nation in the World was extremely useful in identifying the prevalence of diabetes.

Lastly, we extracted data from websites such as www.cdc.gov and www.healthdata.org to attain data on diabetes and its prevalence in certain countries that would be useful in our research, such as those from the United States and China so that we could easily see diabetes' prevalence and correlation to other diseases at a local level in different countries

compared to the simple data sources from global sites. Furthermore, sources such as www.healthdata.org has data sources which provides a large dataset containing data from years 1999-2012, allowing us to compare with the historical data for some possible factors of diabetes and attempt to find a correlation between them.

For these data sources that aren't locally based, we plan to scrape such data in Python whenever possible. Ironically, we found no such need for it due to these data being either easy and not tedious to manually fill it in or having the option to convert to a .csv file. Afterwards, we would then process the relevant datasets in RStudio and get a good graph out of Tableau.

Job Distribution

The following table is the job distribution of this project.

Member	Roles
Denzel Chia Wen Xuan (3S2)	Research for data Compile and shortlist data
Tan Guan Lin (3S1)	Compile and shortlist data
	Data Analysis
David Lim Kang Wei (3S2)	Project Documentation Research for data

Timeline

Our timeline is as follows:

Date	Overview	Outcome
29th Jan	● Introduction to defence science ● Group allocation ● Introduction to literature reviews	● Start of this Defence Science group ● Learnt how to do literature reviews
1st Feb	● Introduction by KDD	---
8th Feb	● Briefing on proposal	● Did a proposal draft
22nd Feb	● Proposal draft evaluation ● Written report briefing	● Made edits to proposal
5th-9th Mar	● Data science sabbatical	● Learnt how to code via RStudio and Tableau ● Learnt about statistics
23rd Mar	● Proposal presentation (rehearsal)	● Improved proposal slides, draft
3rd Apr	● Proposal Evaluation	● Proposal passed
19th Apr	● Update on written report format ● Submission details	● Updated project status ● Started on written report ● Submitted first draft of written report
2nd Jun	● Met with KDD Mentors	● Clearer view on project ● Started data gathering
3rd-24th Jun	● Meetings over Discord to gather and process data	● Finished most of data gathering
27th Jun	● Unveiled KDD competition details	● Edited written report

	● Evaluation of written report	
7th Jul	● Met with KDD Mentors to discuss data obtained	● Learnt how to utilise data ● Will continue data collection and analysis
10th Jul	● Met in school to upload processed datasets to slides	
12th Jul	● Mid-Term Evaluation	● Realised that we need to revamp slides ● Rechanged presentation slides' format
3rd Aug	● Met with mentor on slides and written report	● Improve slides, made improvements to the written report
4th Aug	● Met with KDD mentors	● Learnt about conditional probability (to improve code) ● Improved both written report and slides
8th Aug	● Met with mentor on updated slides and written report	● Removed code idea ● Updated aim, conclusion, summary
17 Aug	● Final Evaluation ● KDD Showdown	

Results and Discussion

In order to find out the causes of diabetes, we decided to search on prevalence of diabetes and its correlation to other diseases around the globe. All the data shown were sorted neatly to be presented via Tableau.

<u>Prevalence Data:</u>

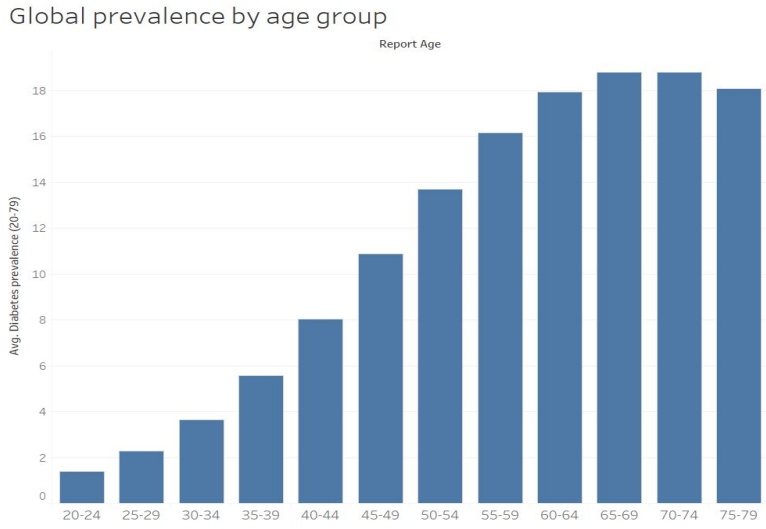


Fig 1.1

Fig 1.1 is a graph showing the prevalence of diabetes by age worldwide[2]. As shown in the figure, the prevalence rates increase as one ages. This could be due to the slower glucose metabolism in adults thus leading to a lower insulin secretion and higher insulin resistance, hence an increased chance for diabetes[10].

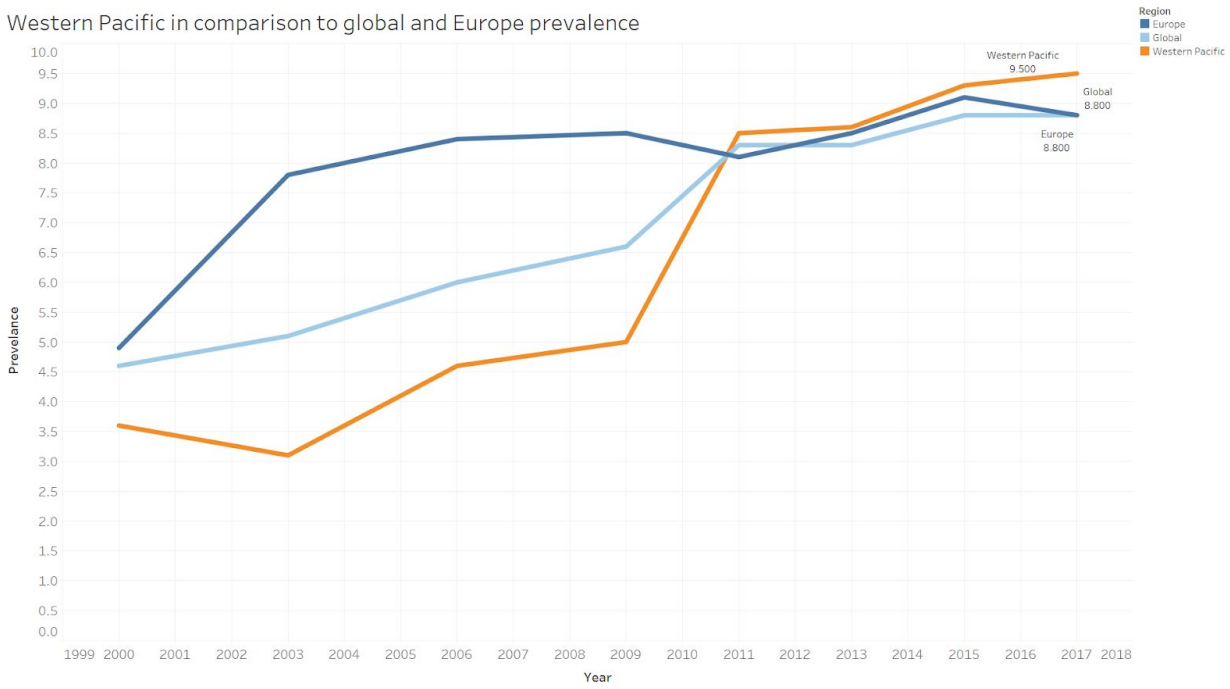


Fig 1.2

Fig 1.2 is a graph showing the prevalence in diabetes in comparison to regions such as Europe and Western Pacific over the years[2]. While both show a general increasing trend like the global trend, the prevalence of diabetes is different. This could have been due to the difference in lifestyle and diets in different regions. As a result, the chance of people getting diabetes would be different, hence the difference in the prevalence of diabetes[12].

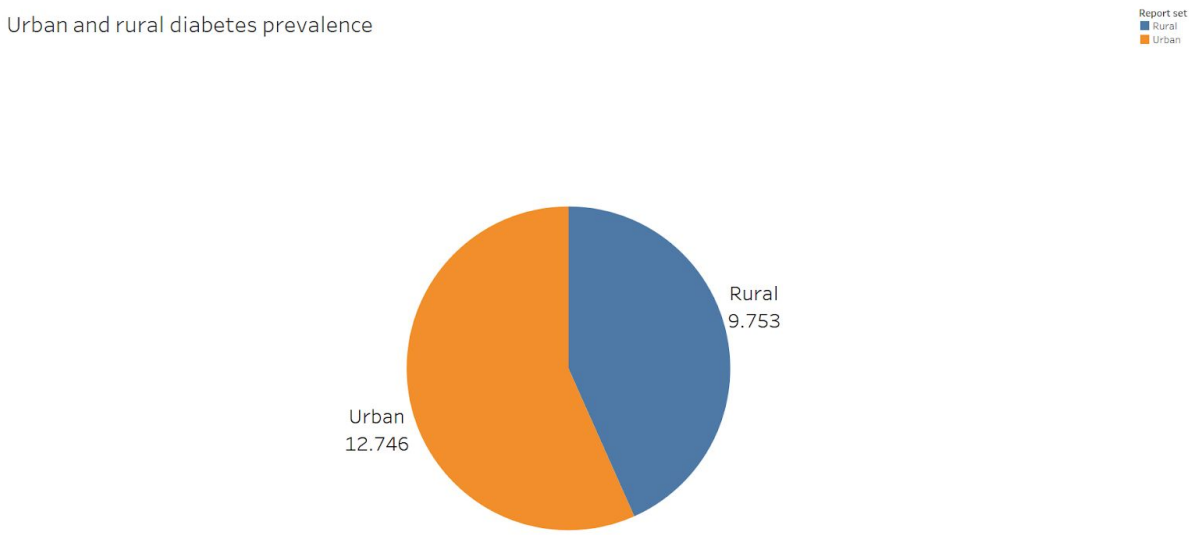


Fig 1.3

While on a global perspective, we decided to obtain data on whether urbanisation is a factor of diabetes. As shown in Fig 1.3, a pie chart showing the prevalence of diabetes in rural and urban regions[2], urban regions tend to have a higher prevalence rate of diabetes than in rural regions. This could be due to urbanisation bringing about affluent lifestyles and changes in one's dietary habits. As a result, one is more likely to be able to afford and consume unhealthy food that may be rich in glucose, thus increasing the chances of getting diabetes, hence the increase in prevalence rate of diabetes in urban regions than in rural regions[11].

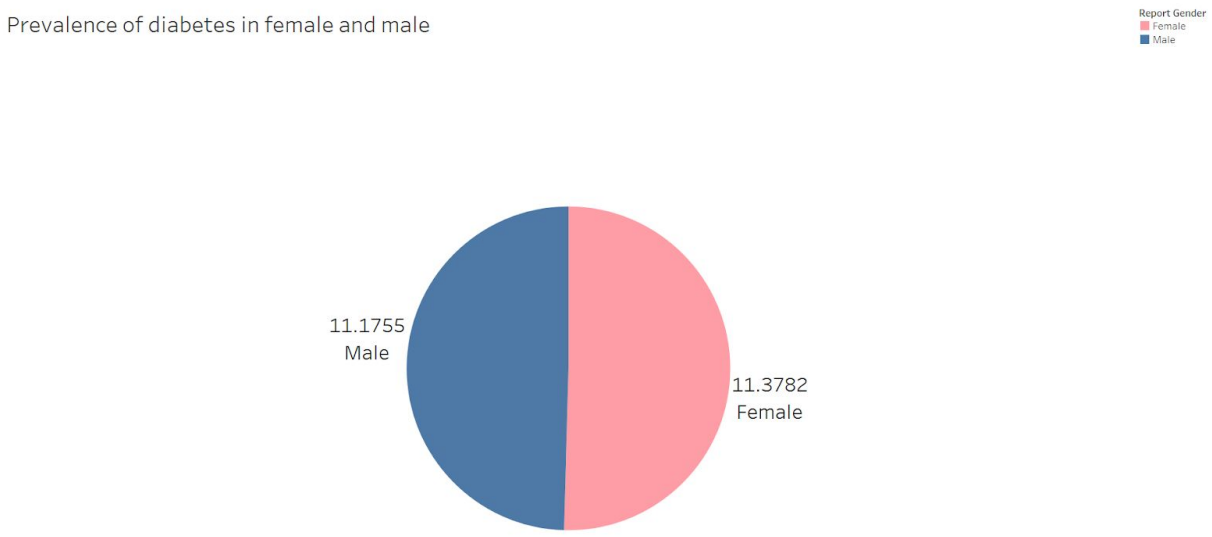


Fig 1.4

Apart from urbanisation, we have also searched data on gender and its relation to diabetes. As shown in Fig 1.4, a pie chart showing the prevalence of diabetes between genders worldwide[2], females tend to have a higher diabetes prevalence than males.

However, this was not always the case. When we searched for data on gender and its relation to diabetes in Singapore, we found out that instead, males tend to have a higher prevalence for diabetes than females per age group in Singapore, as shown in Fig 1.5, a graph showing the prevalence of diabetes in Singaporeans based on gender and age[2]. As a result, with conflicting data, the gender factor and its relation to diabetes remains inconclusive.

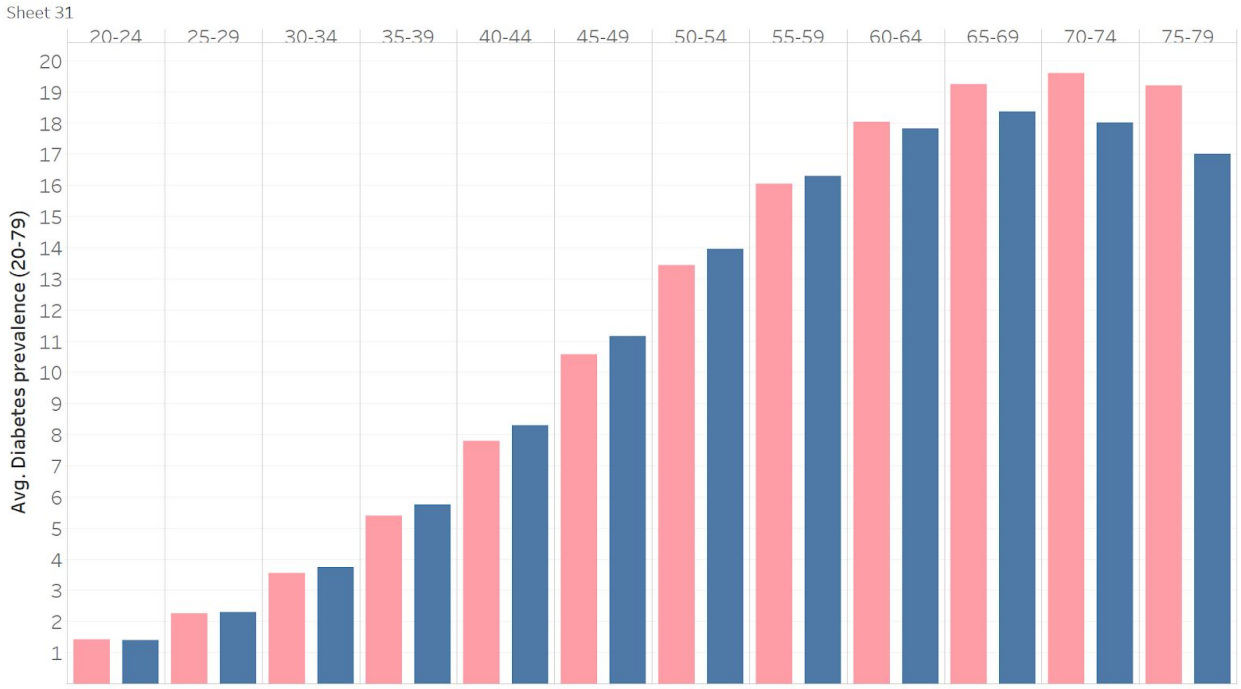


Fig 1.5

Apart from gender, we also decided to compile a data on the ethnicity in Singapore and accoun as to the difference in diabetes prevalences in these ethnic groups. As shown in Fig 1.6, a pie chart showing the prevalence of diabetes between ethnic groups in Singapore[4], this difference could be due to different ethnic groups having different eating habits as well as the “thrifty gene” in certain races. Some ethnic groups have different cultures and eating habits, wherein they eat diets higher in glucose or fat content, thus resulting in a higher chance of getting diabetes[13,14]. Additionally, in the past certain races had a “thrifty gene” developed by their ancestors to help them survive in times of famine[9]. While this has served their ancestors well when food was scarce, this may have led to an increase in their chances for diabetes in Singapore’s society today, where food is more than enough for the population.

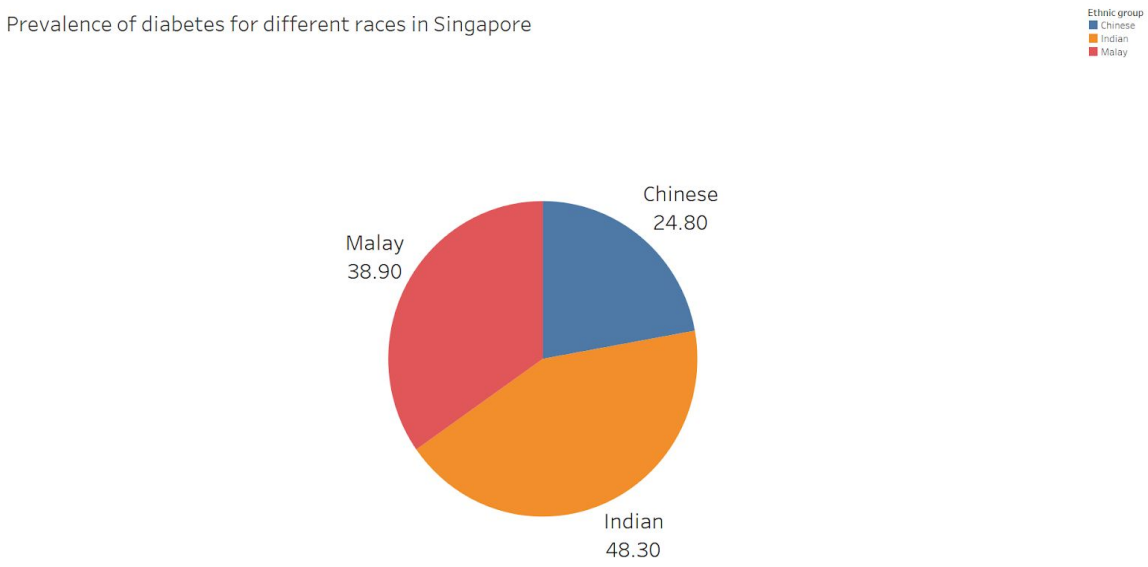

Prevalence of diabetes for different races in Singapore
Ethnic group
Chinese
Indian
Malay
Chinese
24.80
Malay
38.90
Indian
48.30

Fig 1.6

Correlation Data:

Before the discussion of the data that was obtained and processed, here are some basic definitions of the risk factors that we processed, along with the definition of diabetes:

Name	Defined As...
Diabetes Mellitus	≥126 mg/dl of sugar concentration in blood
Hypertension	Blood pressure ≥140 mmHG
Obese	Body having ≥30.0 kg/m^2
Physical Activity	Physical activity of <60 min/wk (week)
Total Cholesterol	Sum of cholesterol in blood ≥200 mg/dl
LDL Cholesterol	Amount of LDL Cholesterol in blood ≥130 mg/dl
HDL Cholesterol	Amount of HDL Cholesterol in blood <40 mg/dl
Triglycerides	Amount of triglycerides in blood ≥150 mg/dl

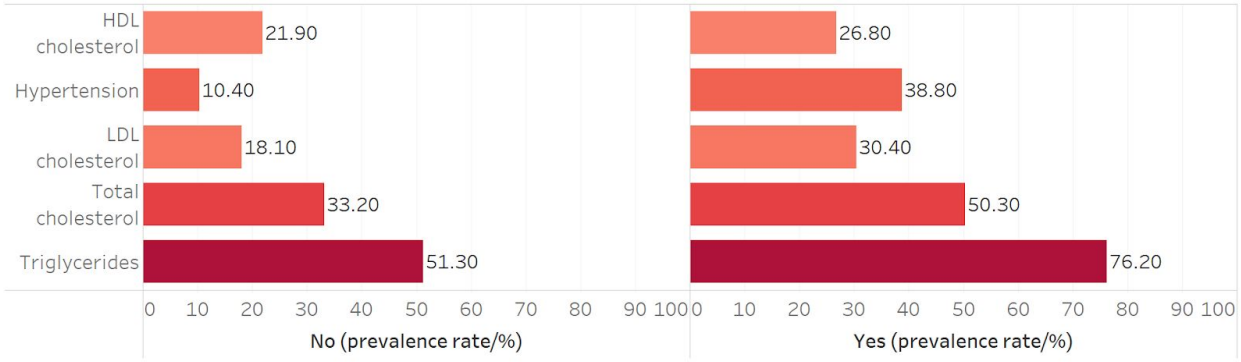
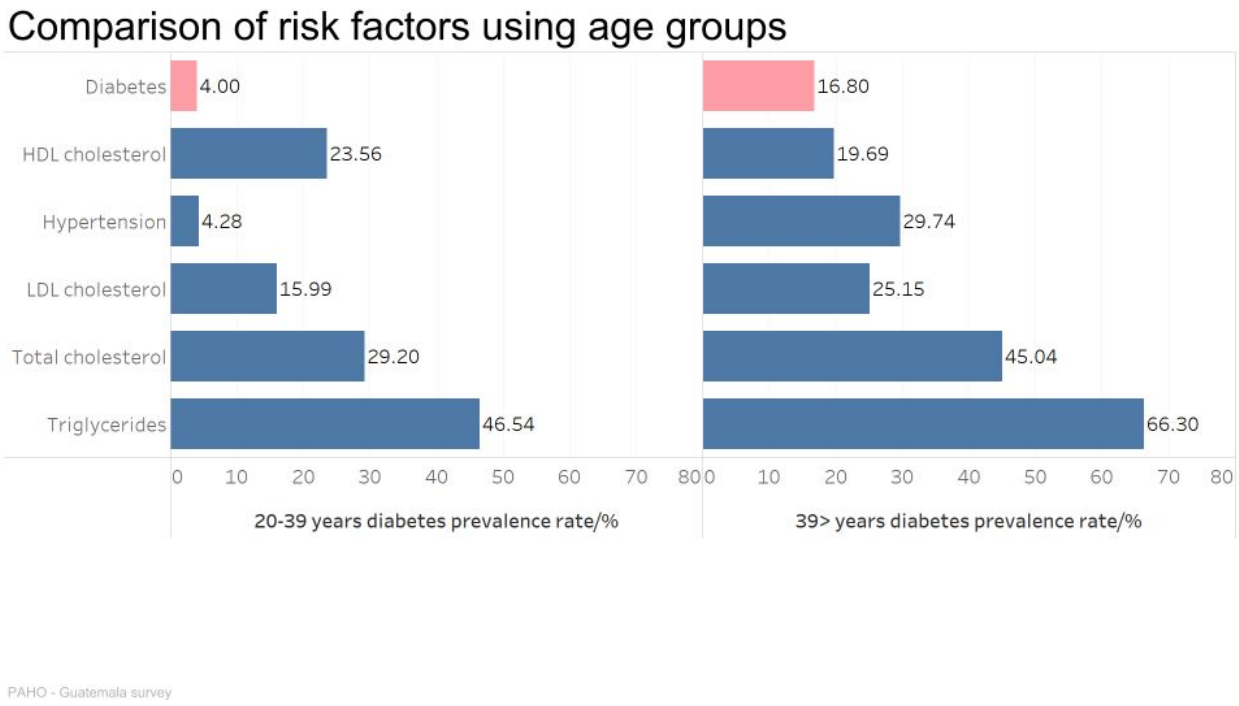


Fig 2.1

Fig 2.1 is a bar graph showing the comparison of risk factors between those with and without diabetes[3]. In it, the five risk factors that we processed were compiled into two bars each, one with people with diabetes, and the other with people without diabetes based on the prevalence rate. As shown, diseases such as hypertension have a very high percentage in its prevalence rate for those with diabetes, with an increase of 28.4%, hence it may be the disease that is highly correlated to diabetes. Additionally, diseases such as LDL cholesterol and triglycerides also reveal an increase between those with and without diabetes of 12.3% and 24.9%, thus it may be linked with diabetes to a certain extent. On the flipside, risk factors such as physical activity and diseases such as HDL cholesterol have little to no increase between the prevalence rates of those with and without diabetes with low increase in prevalence rates of 4.9%, thus they have little to no correlation with diabetes.

Fig 2.2



In addition, we decided to compare our selected risk factors with age groups. As shown in Fig 2.2, a bar graph showing the comparison of risk factors via age groups[3], we used diabetes as a control to determine whether if any of our selected risk factors were related to diabetes based on other factors, such as age. In Fig 2.2, between age groups there was a drop in prevalence of HDL cholesterol from 23.56% to 19.69% while that of diabetes saw a rose from the 20-39 age group to the >39 age group from 4.00% to 16.80%, thus further showing that HDL cholesterol is not related to diabetes. On the flipside, risk factors such as hypertension, LDL cholesterol, total cholesterol and triglycerides saw increases from 4.28% to 29.74%, 15.99% to 25.15%, 29.20% to 45.04% and 46.54% to 66.30% respectively. With all of them showing a rise in age groups just as diabetes, it shows that these risk factors can be concluded to be correlated to diabetes.

So, why are diseases such as hypertension, LDL cholesterol and triglycerides are linked with diabetes to a certain extent? Based on our research, hypertension is the result of inflammation and oxidative stress,

which is a low-grade inflammatory process that occurs in both diabetes and hypertension, and a lack of insulin resistance, which results in the insulin not being able to play its pivotal role in the body thus resulting in the development of hypertension and diabetes mellitus. As both diseases share the common risk factors, it is obvious for one to see that both diseases would tend to co-exist and therefore, are correlated[6].

As for LDL cholesterol, diabetes tend to raise cholesterol levels, especially LDL cholesterols (Low-density Lipoproteins), along with triglycerides. This condition is known as diabetic dyslipidemia. Additionally, such an increase in cholesterol levels can lead to the buildup of fatty plaques in the arteries leading to atherosclerosis and possibly coronary heart disease[7].

For triglycerides, not only is it correlated to diabetes due to diabetic dyslipidemia as mentioned, it is also correlated to diabetes in another way. Triglycerides are made up of one molecule of sugar and 3 molecules of fatty acid and are produced by the body to store glucose long-term. Hence, since triglycerides are made up of carbohydrates and fats, an increase in triglycerides would mean an increase in uptake of carbohydrates and fats. Since insulin is needed in order to decrease these high levels of carbohydrates, an increase in triglycerides would show that there are some signs of insulin resistance, which is a precursor to type 2 diabetes mellitus, thus both diseases are correlated[8]. This strong relation to diabetes is also why triglycerides are used as a sign of diabetes by doctors.

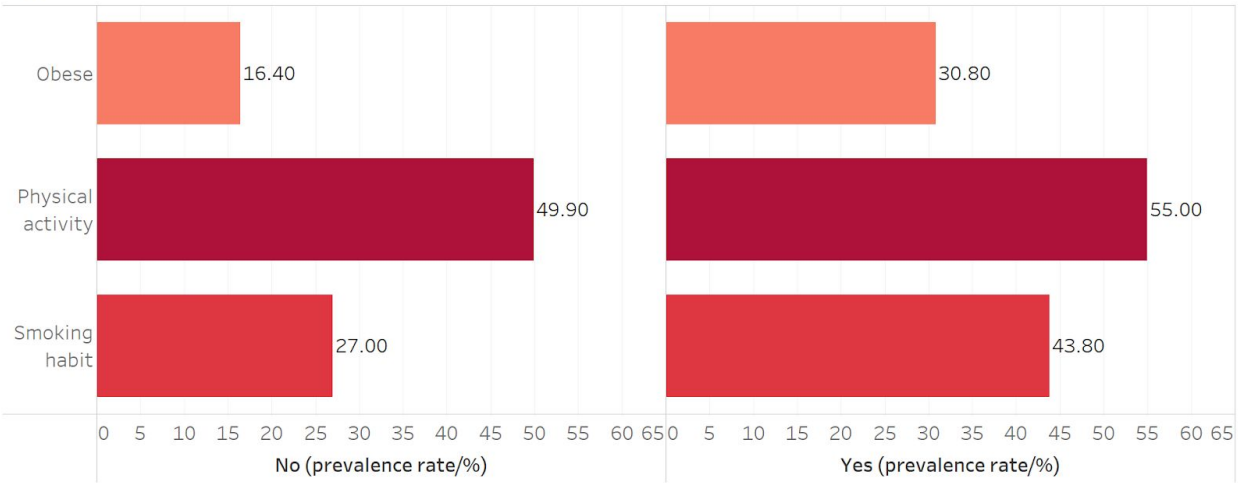


Fig 2.3

Apart from analysing the correlation of other diseases to diabetes, we also analysed lifestyle factors that may be correlated to diabetes, such as low physical activity, obesity and smoking habit. As shown in Fig 2.3, a graph showing the comparison of lifestyle factors between those who have and those who do not have diabetes[3], it was shown that the prevalence rate of people with obesity or spend less time on physical activity have a slight increase in prevalence, with obesity increasing by 14.40% and physical activity increasing by 5.10%, hence they may not be correlated. Smoking habit however showed an increase of 16.80%, thus it may be correlated with diabetes.

Conclusion

Overall, as shown from the prevalence data collected, it can be inferred that diabetes can be caused from age, lifestyle, urbanisation, dietary habits as well as ethnicity, yet the factor of gender and its relation to the prevalence of diabetes remains inconclusive due to contradicting data, which exists as a limitation.

We have also inferred that diabetes is correlated to diseases such as hypertension, atherosclerosis, coronary heart disease and increase in LDL cholesterol and triglycerides alongside lifestyle factors such as smoking habit. However, in all of these correlated diseases, the increase in LDL cholesterol and triglycerides would be the most harmful. As mentioned earlier, the increase in LDL cholesterol and triglycerides can lead to atherosclerosis and coronary heart diseases, which may lead to myocardial infarction, also known as heart disease, which can kill.

Additionally, having processed this data, our group has also uploaded them on a tableau site for people to view them and interact with our data.

Bibliographies

(1) Diabetes programme. (2018, June 21). Retrieved from http://www.who.int/diabetes/en/

(2) IDF Diabetes Atlas. (n.d.). Retrieved from https://www.idf.org/e-library/epidemiology-research/diabetes-atlas/134-idf-diabetes-atlas-8th-edition.html

(3) (n.d.). Retrieved from https://www.paho.org/hq/index.php?option=com_content&view=article&id=3038:2010-survey-diabetes-hypertension-chronic-disease-risk-factors-villa-nueva-guatemala-2007&catid=1415:integrated-disease-management&Itemid=1353&lang=en

(4) Information Paper on Diabetes in Singapore (Issue brief). (n.d.).

(5) Chentli, F., Azzoug, S., & Mahgoun, S. (2015). Retrieved from https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4673801/

(6) Cheung, B. M., & Li, C. (2012). Diabetes and Hypertension: Is There a Common Metabolic Pathway? Current Atherosclerosis Reports, 14(2), 160-166. doi:10.1007/s11883-012-0227-2

(7) Cholesterol Abnormalities & Diabetes. (n.d.). Retrieved from http://www.heart.org/HEARTORG/Conditions/More/Diabetes/WhyDiabetesMatters/Cholesterol-Abnormalities-Diabetes_UCM_313868_Article.jsp#.W0VOgdIzY2w

(8) How Triglycerides Affect Your Risk of Diabetes. (n.d.). Retrieved from https://www.webmd.com/cholesterol-management/diabetes#1

(9) Thrifty gene hypothesis. (2018, July 03). Retrieved from https://en.wikipedia.org/wiki/Thrifty_gene_hypothesis

(10) Suastika, K., Dwipayana, P., Siswadi, M., & Tuty, R. (2012). Age is an Important Risk Factor for Type 2 Diabetes Mellitus and Cardiovascular Diseases. *Glucose Tolerance*. doi:10.5772/52397

(11) Gassasse, Z., Smith, D., Finer, S., & Gallo, V. (2017, October 01). Association between urbanisation and type 2 diabetes: An ecological study. Retrieved from https://gh.bmj.com/content/2/4/e000473

(12) Why are Asians at Higher Risk? (n.d.). Retrieved from http://asiandiabetesprevention.org/what-is-diabetes/why-are-asians-higher-risk

(13) Orenstein, B. W. (2015, September 18). How Ethnicity Affects Type 2 Diabetes Risk. Retrieved from https://www.everydayhealth.com/hs/type-2-diabetes-management/ethnicity-diabetes-risk/

(14) Vann, M. R. (2016, June 02). How Ethnicity Affects Type 2 Diabetes Risk. Retrieved from https://www.everydayhealth.com/type-2-diabetes/ethnicity-and-your-diabetes-risk

Appendix

The following are the data which we have processed, but were either limited due to lack of data or were not used in the written report.

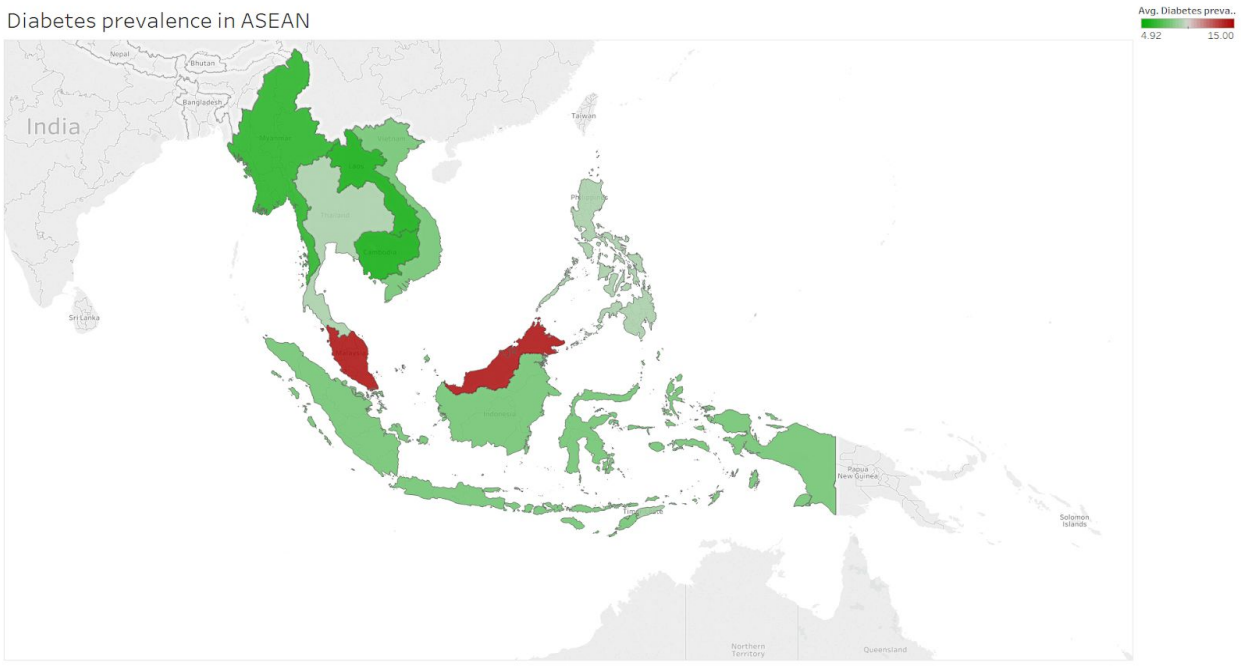


Fig 3.1[2]

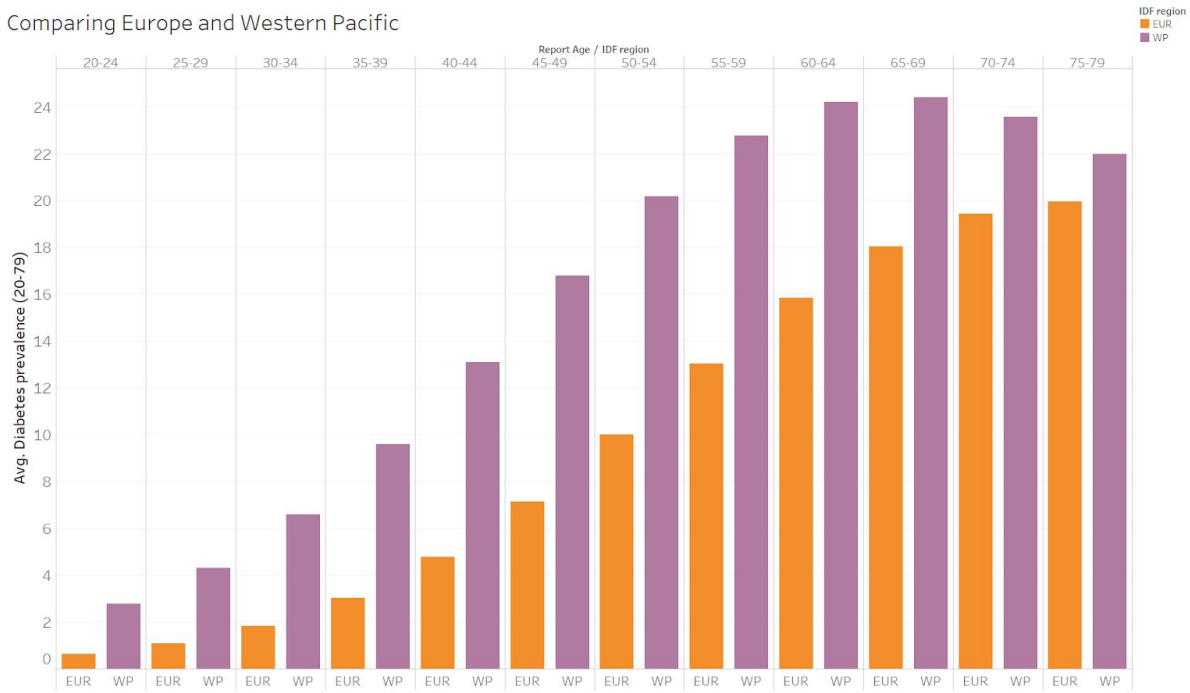


Fig 3.2[2]

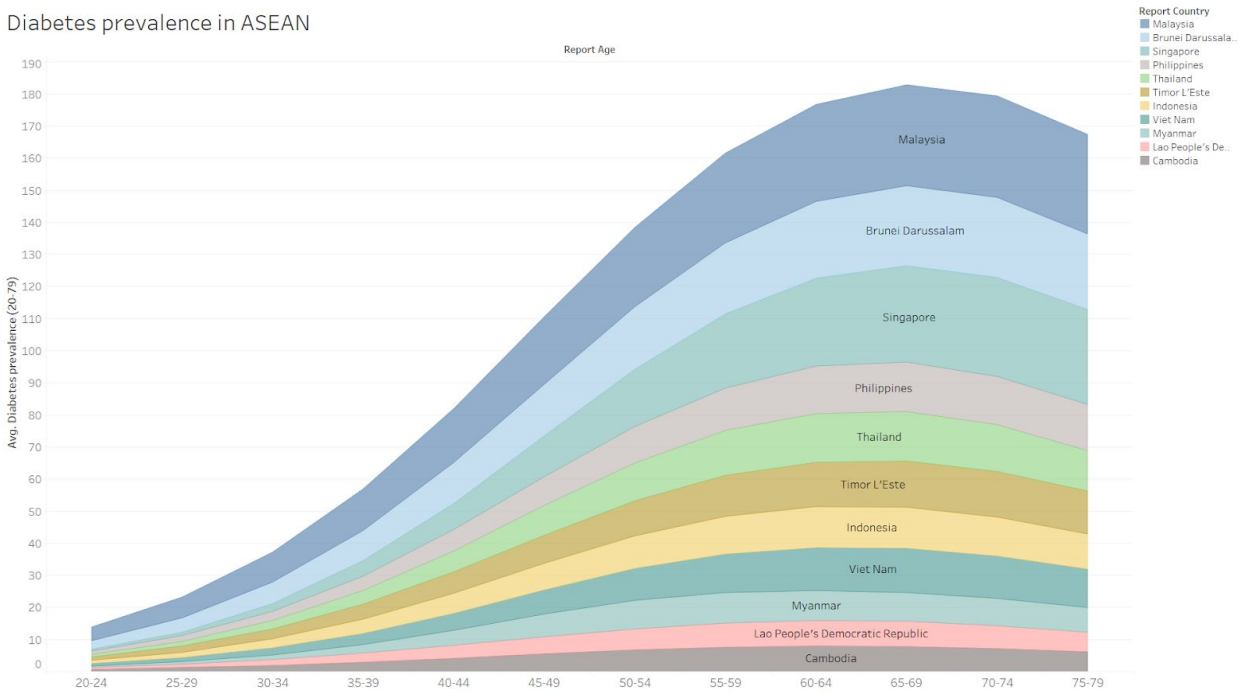


Fig 3.3[2]

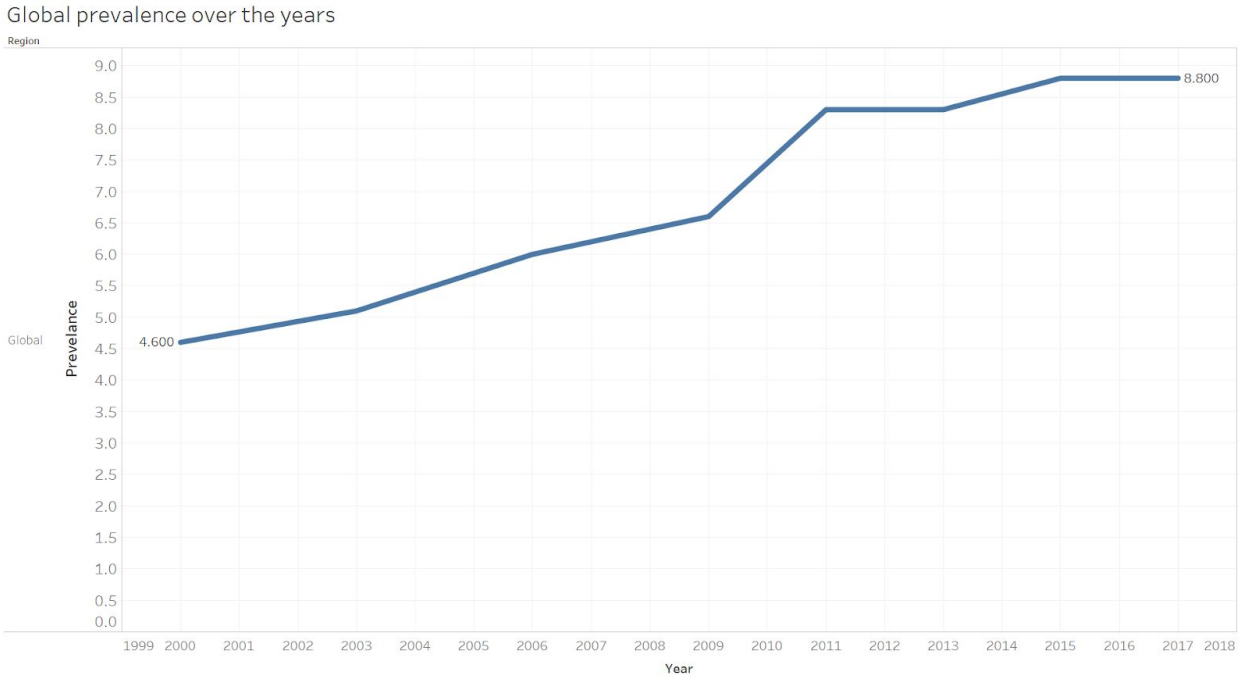


Fig 3.4[2]

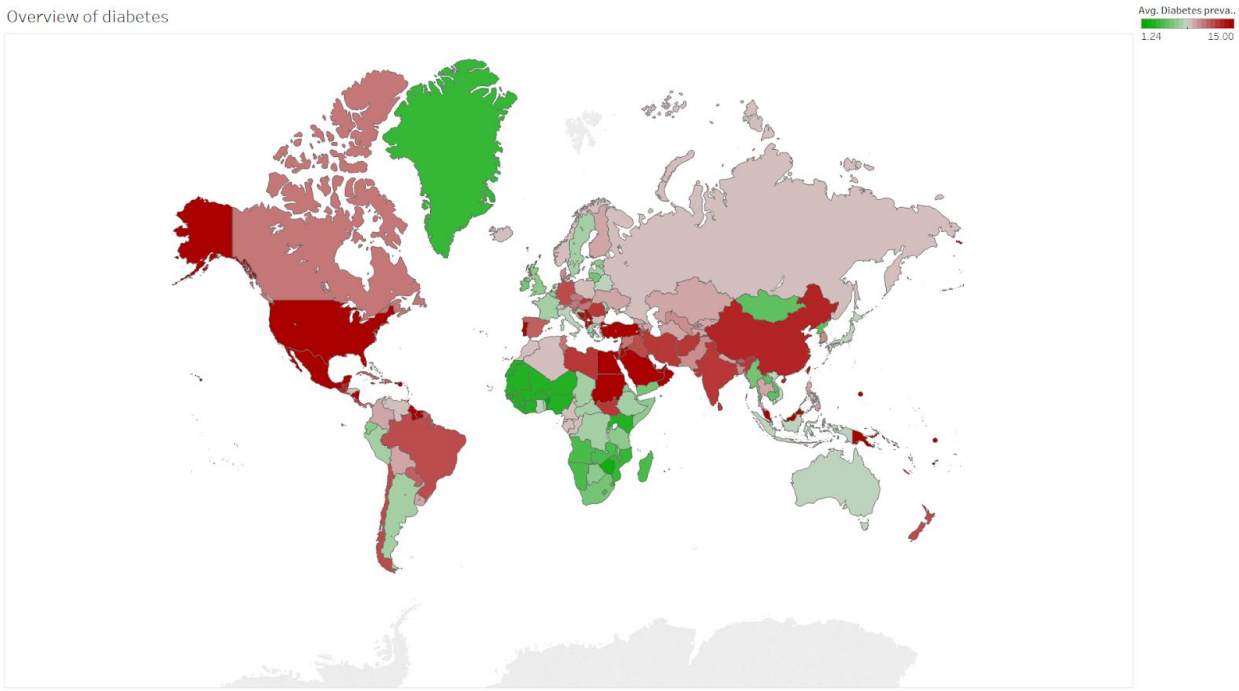


Fig 3.5[2]

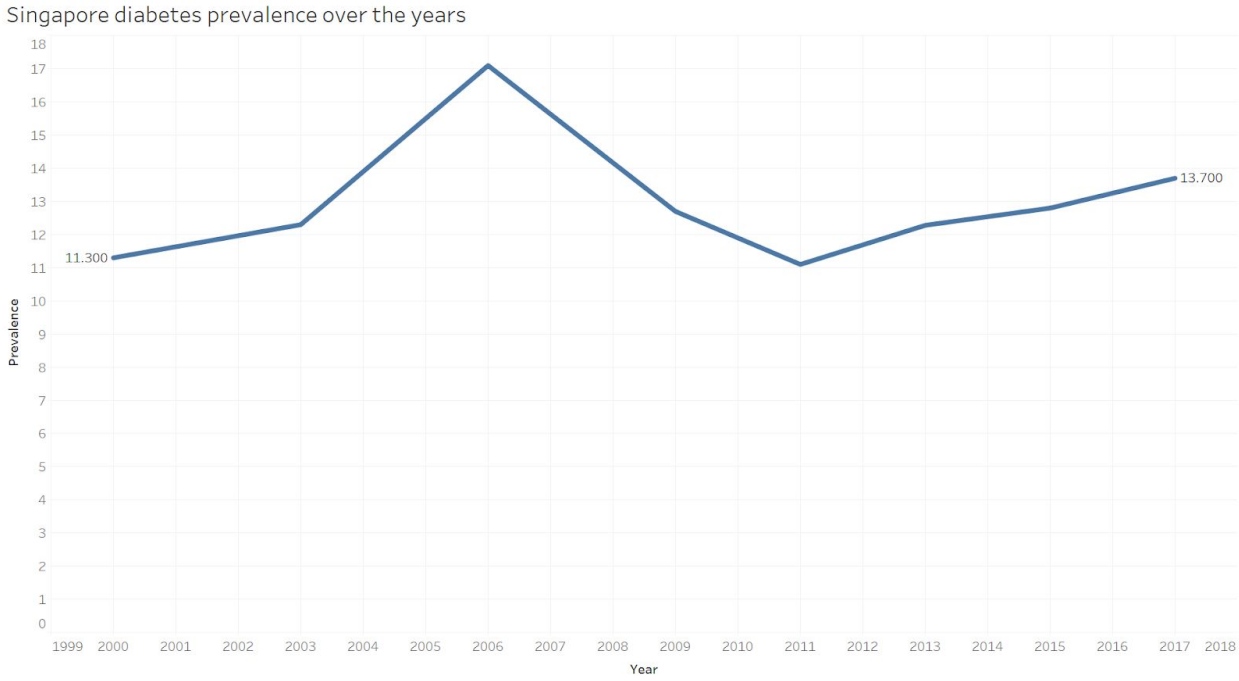


Fig 3.6[2]

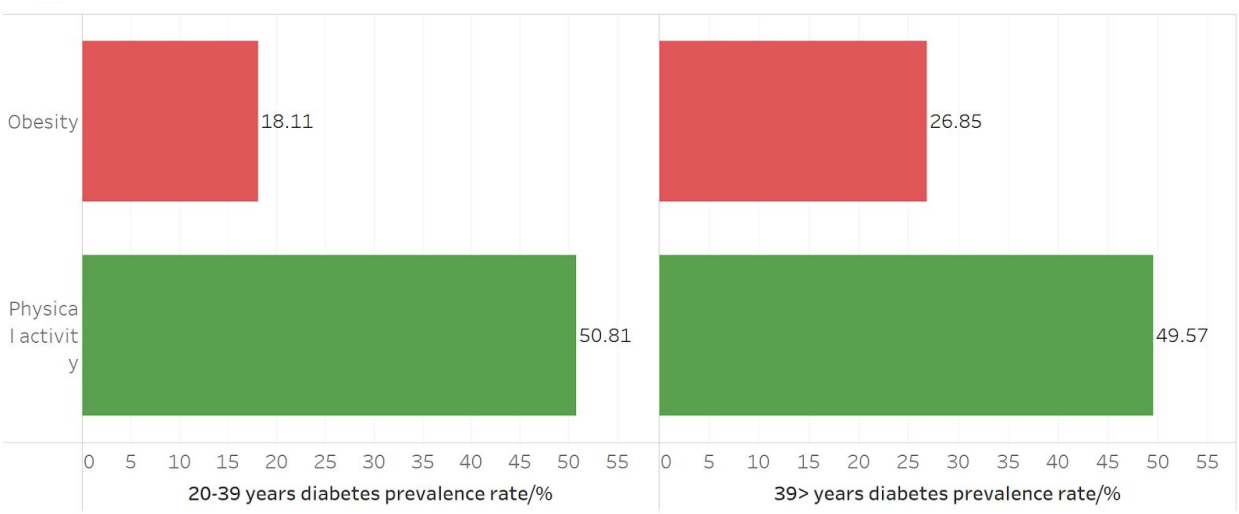

Obesity
Physical activity
18.11
50.81
26.85
49.57
0 5 10 15 20 25 30 35 40 45 50 55
20-39 years diabetes prevalence rate/%
0 5 10 15 20 25 30 35 40 45 50 55
39> years diabetes prevalence rate/%

Fig 3.7[3]