Relations between Inflammation and Access to care, Treatment, and Diabetes in Older Mexico

Dominik Grätz[1], Rachel Miller-Moudgil[1], Amber Somarriba[1], Brittany Spinner[1], & Tian Walker[1]

[1] University of Oregon

Author Note

List of group members ordered by alphabet.

Abstract

Background. Background goes here. *Methods.* Methods go here. *Results.* Results here. *Conclusions.* Conclusions here.

Keywords: Diabetes, access to care, inflammation, health, Mexico, China

Word count: X (this cannot easily be done automatically, we can also just leave it out)

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```
## # A tibble: 172 x 2
##    `RQ4df$access` `RQ4df$crp2`
##    <fct>          <fct>
##  1 No             high
##  2 No             low
##  3 Yes            low
##  4 No             low
##  5 No             low
##  6 No             low
##  7 No             low
##  8 Yes            low
##  9 Yes            low
## 10 No             low
## # ... with 162 more rows


##
##  Pearson's Chi-squared test with Yates' continuity correction
##
## data:  RQ4df$access and RQ4df$crp2
## X-squared = 0.56544, df = 1, p-value = 0.4521


##             RQ4df$crp2
## RQ4df$access      low     high
##          Yes 67.34302 13.65698
##          No  75.65698 15.34302
```

```
##             RQ4df$crp2
## RQ4df$access low high
##          Yes  65   16
##          No   78   13
```

FIGURE REFERENCES

Figure 5

Figure 3

Figure 1

Figure 2

TABLE REFERENCES

Table 1 (Regression Table 1)

Table 2 (Regression Table 2)

Table 3 (Descriptives Table)

Introduction

Diabetes and its insidious complications continue to expand as a global health burden at an alarming rate. As of 2021, there were approximately 537 million adults living with diabetes in the world and this number is expected to jump to 783 million by 2045. A disproportionate percentage of these people live in low to middle income countries (LMICs). In light of the COVID-19 pandemic, it is also of great import that we better understand the relationships between diabetes and infectious diseases as diabetes both increased the severity of Covid (in people with elevated a1c levels) and has increased in incidence during the Covid 19 pandemic (Yang et al., 2020) (Rohm, Meier, Olefsky, & Donath, 2022).

Additionally, diabetes is associated with a steep increase in cardiovascular disease risk and is a leading cause of death in many low to middle income countries (LMICs) including Mexico. Although the precise classification of diabetes remains controversial because of the complex nature of its pathogenesis, there are three universally acknowledged subtypes: type 1 diabetes, type 2 diabetes, and gestational diabetes. Diabetes is a progressive disease in that the longer one has it, the more complications ensue. Therefore, it is helpful to conceptualize diabetes as a process that can be stopped, but not reversed. Research that contributes to slowing down or stopping the process can be extremely valuable to global health regardless of its contribution to cure and prevention because of the astronomical rates of diabetes in our world today.

Inflammation is a strong indicator of diabetes development and progression. Inflammation predicts the development of diabetes (Dilys J. Freeman et al., 2002) (Dilys J. Freeman et al., 2001) (Schmidt et al., 1999). Specifically, trials for drugs directed at inflammation among people with type 2 diabetes have indicated that drugs targeted at inflammation may be a therapeutic option for preventing diabetes (Agrawal & Kant, 2014).Retinopathy and focal neuropathy (Said, 2007) have also been linked to inflammatory processes. Additionally, the direct damage caused by high blood glucose leads to more inflammation and creates a nasty feedback loop wherein inflammation causes more insulin resistance which leads to high blood glucose.

Diabetes treatment and inflammation

The ability of cells to absorb insulin can be increased through diet, exercise, and oral pills.Increasing exercise and dieting can cause major decreases in inflammation. Some of the drugs for type 2 diabetes aimed at increasing insulin sensitivity also decrease inflammation (e.g., drugs that cause weight loss). In the opposite direction, insulin can cause severe low blood glucose levels that initiate a stress response causing more inflammation.

Methods

Procedures and Sample

357 participants were included in our analyses. Of those, 115 were assessed to be male (32.20 %), 241 were identified to be female (67.50 %). Sex data for 1 participant was missing (see Table 3). This indicates that females were overrepresented in our sample, assuming binary sexes are represented equally in the population ($\chi^2(1) = 44.60$, $p < 0.0001$). The age in our sample was $M = 68$ years (SD = 8.40 years). Because of missing values in the variables of interest in some of our analyses, subjects were excluded. Thus, N is reported separately for each analysis.

Variables

The original WHO dataset contains more than 1,600 variables, not all of which are relevant to our research questions. Therefore, we have limited our analysis to 10 variables, listed below in Table 1. In our analysis, we exclude participants who reported that they already have a formal diabetes diagnosis from a doctor, since we are interested in HBA1C values for people who do not have a diabetes diagnosis. We also exclude participants age 50 or older, since this group typically has high HbA1c levels regardless of the presence of diabetes, as well as participants with CRP levels above 5, since because such values indicate infection which would confound our analysis.

C-reactive protein and hba1c measured through dried bloodspots (minimally invasive biomarkers) (McDade, Williams, & Snodgrass, 2007)).

Self-report surveys conducted by trained interviewers.

Data are from the World Health Organization's Study on Adult Health and Ageing (SAGE). Our data is from 1 of 5 countries where the data were collected.

Cite R packages here comment 495

Results

Regression 1

Demo of inline code usage. We found that this regression model (Table 1) using 357 participants was not significant $F(2, 354) = 1.06$, $p = 0.35$ with $R^2 = 0.01$. The regression coefficient for age was b_{age} = -0.01 ($SE = 0.01$, $p = 0.36$), and for use of medication $b_{medication} = 0.20$ ($SE = 0.18$, $p = 0.28$).

Regression 2

Demo of inline code usage. We found that this regression model (Table 2) using 357 participants was not significant $F(2, 354) = 2.70$, $p = 0.07$ with $R^2 = 0.01$. The regression coefficient for age was b_{age} = -0.01 ($SE = 0.01$, $p = 0.41$), and for diet and exercise b_{Diet} = -0.24 ($SE = 0.12$, $p < 0.05$).

Chi Square Test

First χ^2 results: ($\chi^2(1) = 0.57$, $p = 0.45$). Second χ^2 results: ($\chi^2(1) = 0.57$, $p = 0.45$).

Discussion

Limitations

We were not able to look at pills and insulin separately and some their effects have the potential to cancel each other out. The sex variable was established through interviewer discernment.

References

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Tables and Figures

Table 1

Effect of Medication on CRP, controlling for Age.

Predictor	b	95% CI	$t(354)$	p
Intercept	2.00	[1.06, 2.94]	4.18	$< .001$
Age	-0.01	[−0.02, 0.01]	-0.91	.362
Medication	0.20	[−0.17, 0.56]	1.07	.284

Note. Model fit: $F(2, 354) = 1.06$, $p = 0.35$, $R^2 = 0.01$.

Table 2

Effect of Diet and Exercise on CRP, controlling for Age.

Predictor	b	95% CI	$t(354)$	p
Intercept	2.10	[1.17, 3.04]	4.43	$< .001$
Age	-0.01	[−0.02, 0.01]	-0.83	.408
Diet and Exercise	-0.25	[−0.47, −0.02]	-2.11	.036

Note. Model fit: $F(2, 354) = 2.71$, $p = 0.07$, $R^2 = 0.02$.

Table 3

Descriptive statistics.

		Mexico
N_{total}		357
Sex		
	male	115 (32.20 %)
	female	241 (67.50 %)
	unknown	1 (0.30 %)
Age		68 ($SD = 8.40$)

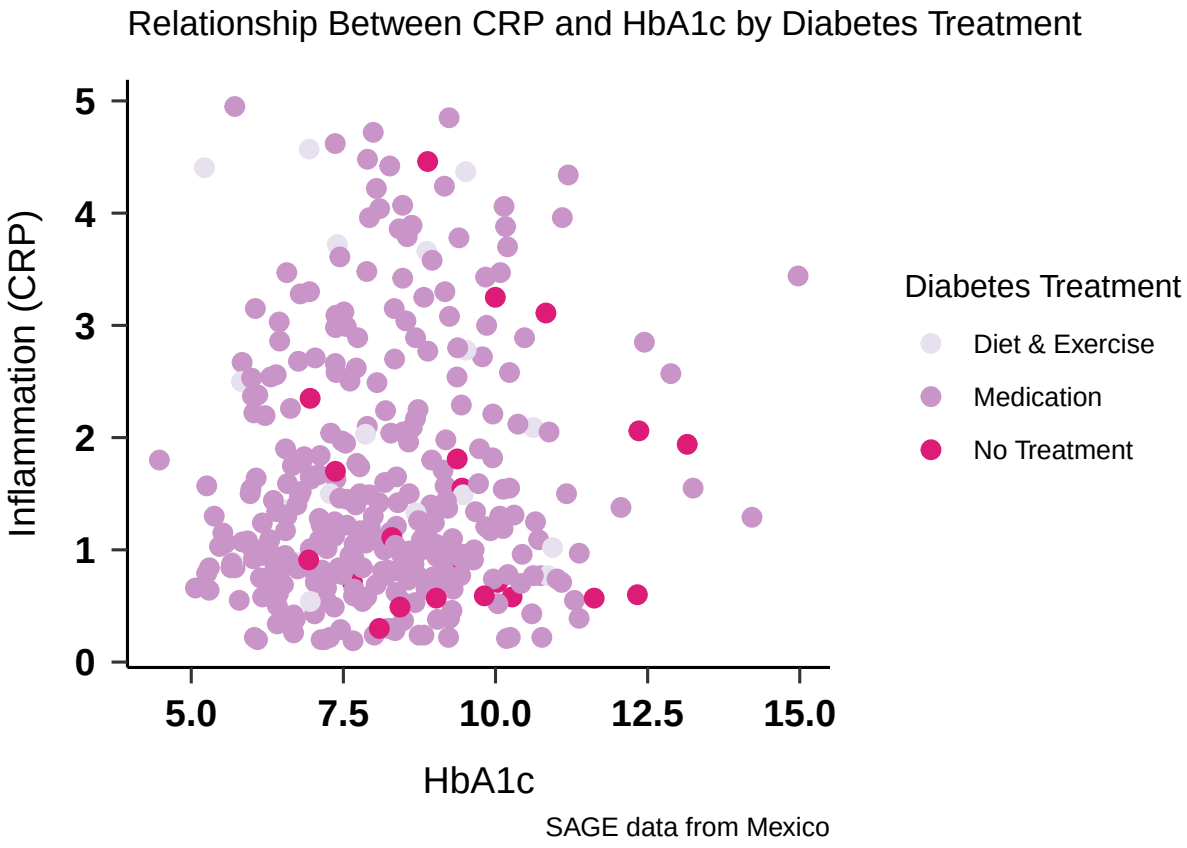
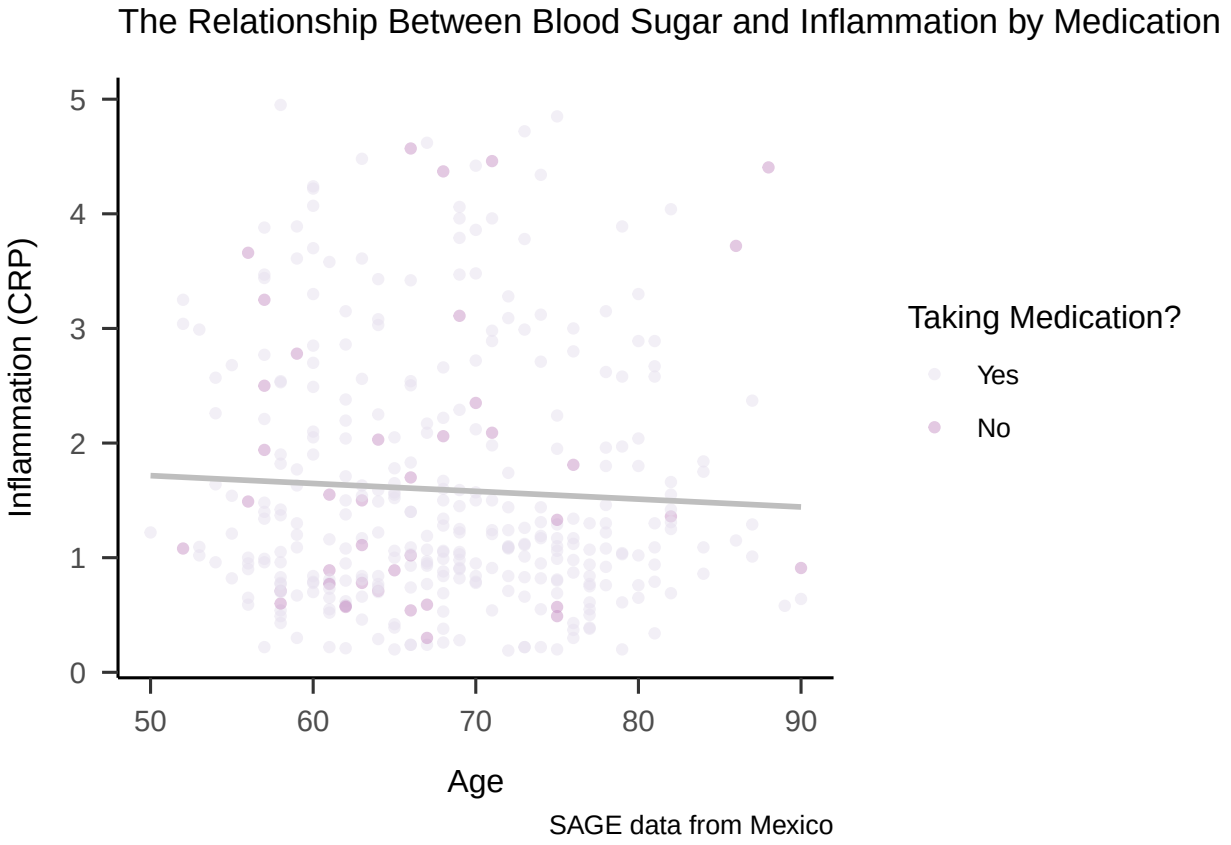


Figure 1. Here goes the caption.



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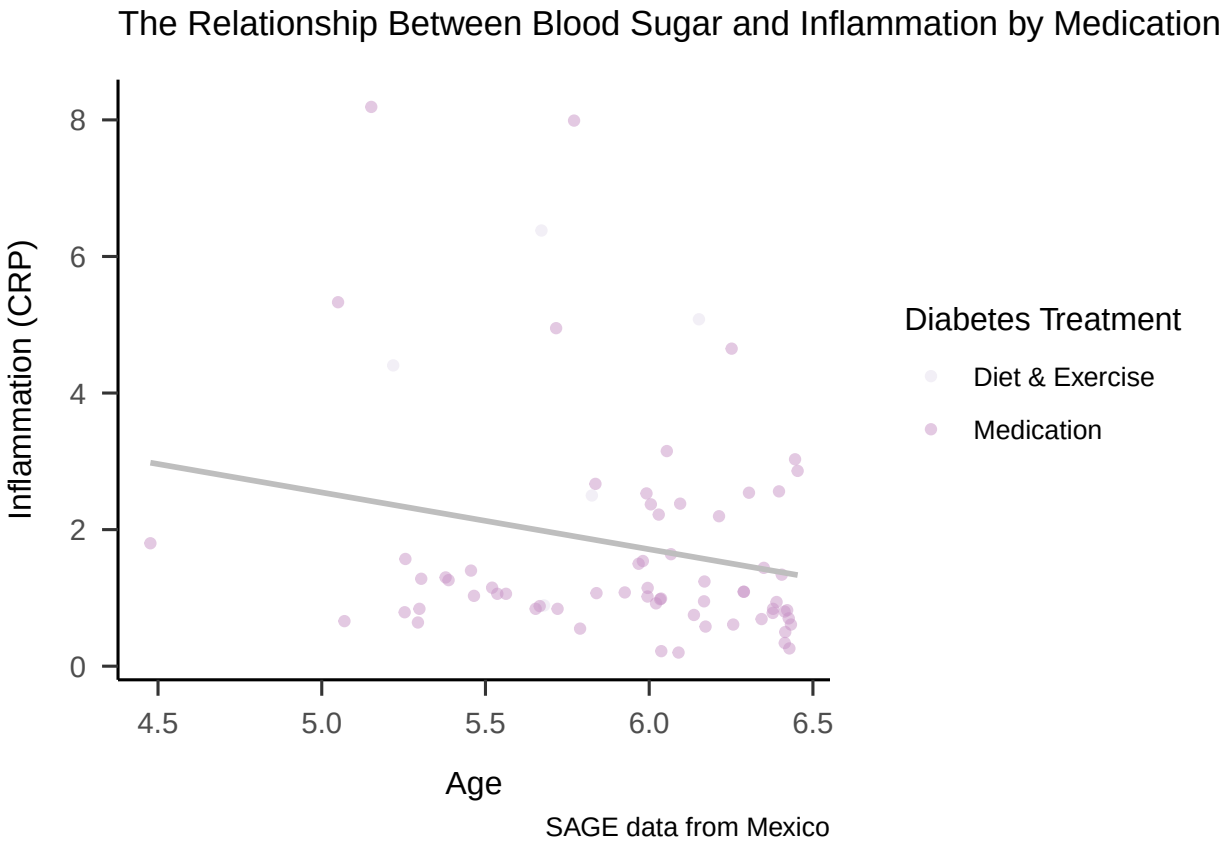

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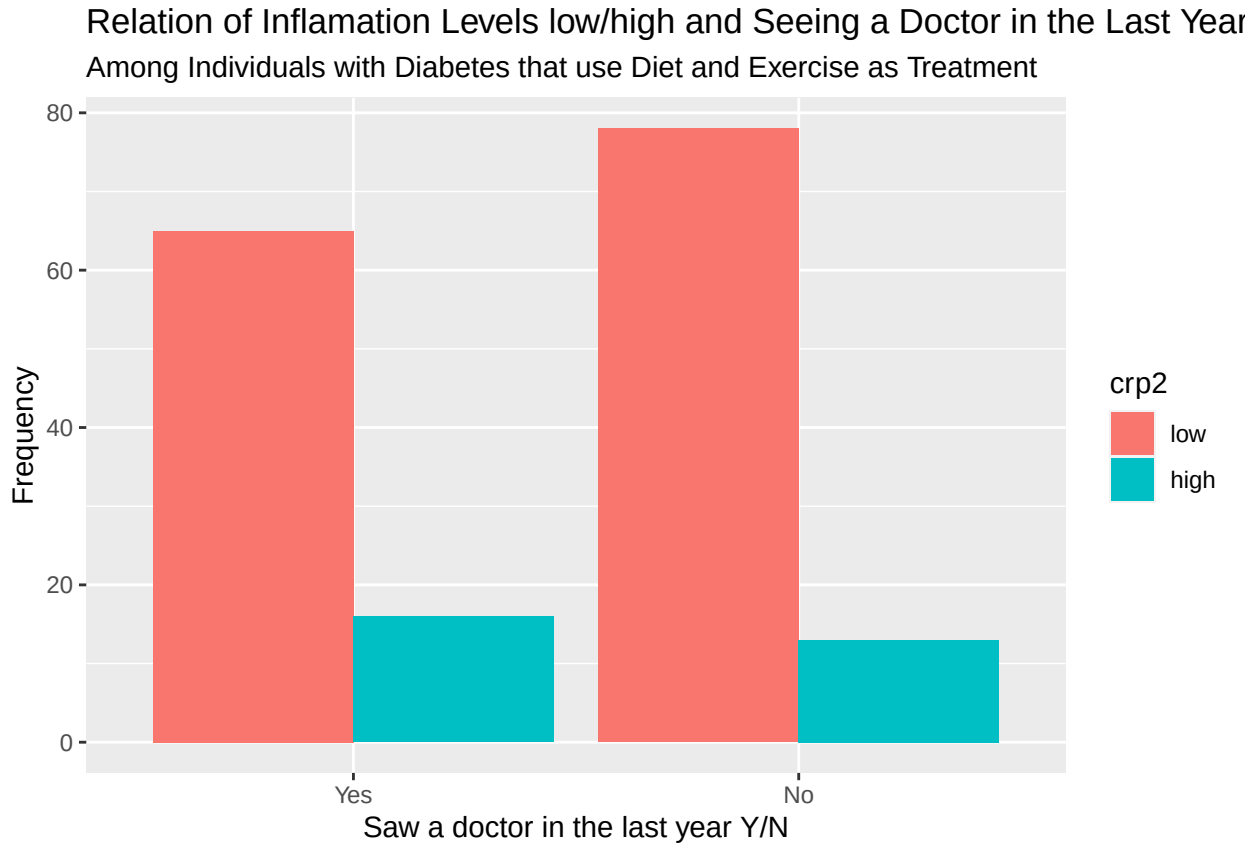

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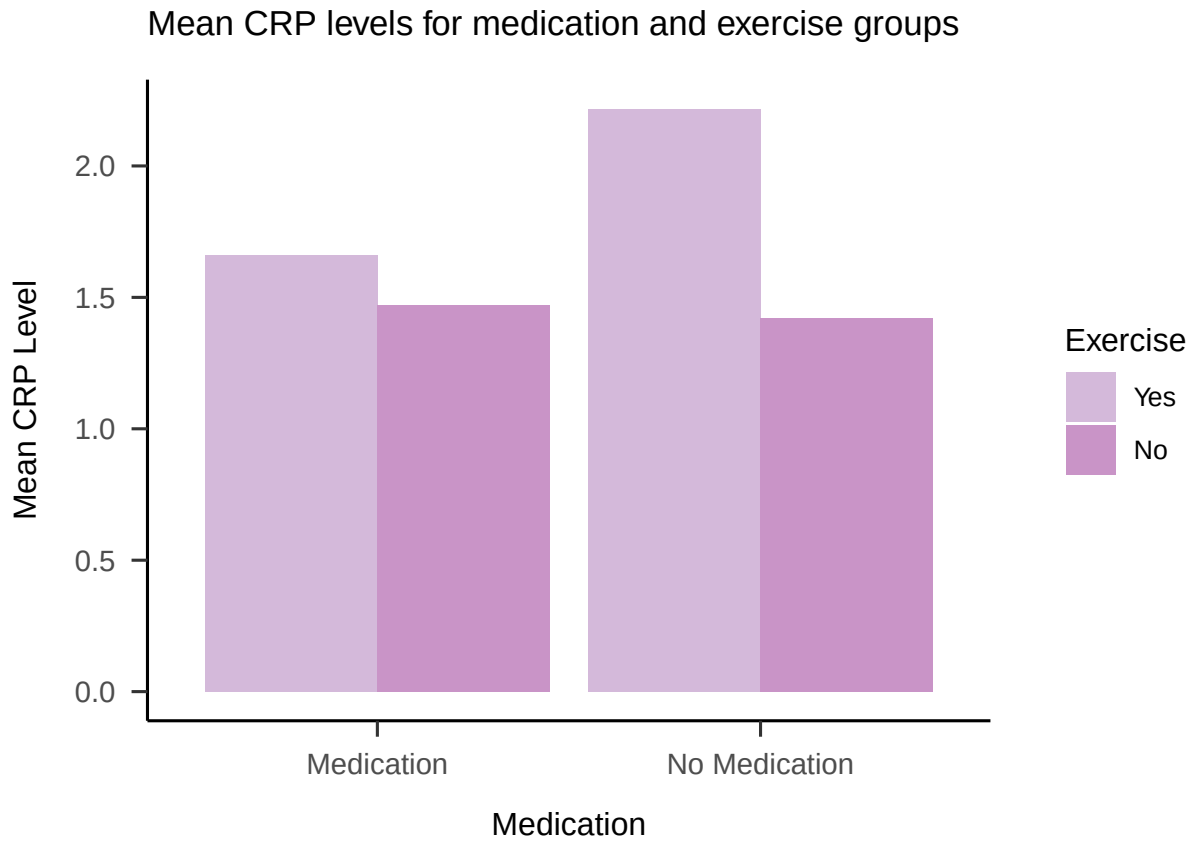

Figure 5. Here goes the caption.