Archives of Diabetes Mechanisms Associated with Type 2 Diabetes Occurrences: Structural Equation Confirmatory Model

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Research Article

Mechanisms Associated with Type 2 Diabetes Occurrences: Structural Equation Confirmatory Model

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Abstract

Introduction: The worldwide prevalence of diabetes, in people over 18 years of age, ranged from 4.7% in 1980 to 8.5% in 2014. Diabetes was the direct cause of 1.6 million deaths, and 1.1 million premature deaths were attributable to hyperglycemia. The study's objective was to identify mechanisms of diabetes production that could serve as a basis for its control.

Methods: The analysis conducted through a 4-phase system of multiple structural equations modeling (SEM), includes specification, identification, estimation, and evaluation. We used a specified and over-identified model, along with the least-square estimates method of robust regression proceduresto address the disruptive violations in the assumed multivariate normality.

Results: Physical activity showed low factor loads (AVE 12.6%), lipids and blood pressure showed high loads, which explained 50.2% and 50.4% of the variability in their coefficients. The effect of lipids on glycosylated hemoglobin was significant: an increase of one point in lipids predicted a 0.608 unit increase in the blood glucose level. There was also statistical significance in the path from physical activity to lipids; an increase in physical activity decreased 0.318 units on the lipid profile.

Conclusions: Physical activity lowers blood pressure, lipid profile, and obesity, factors directly related to glycemic control.

Keywords: Diabetes production mechanism, Structural equations model, Confirmatory statistical model

Introduction

Diabetes is the epidemic disease of the 21st century [1]. The WHO estimates that high blood glucose is the third most important risk factor for 6% of deaths worldwide [2]. According to the International Diabetes Federation (IDF), in 2015, diabetes prevalence was 8.8% and 12% of the world's health expenditure was applied to this disease. Over 415 million people suffer from it and approximately five million die from this cause, which is equivalent to one death every six seconds [3].

Currently, 80% of the disease burden is in developing countries [4–6]. By 2015, the prevalence was 12.9% in North America and the Caribbean region, the age-adjusted prevalence was 9.6% in Central and South America [3] and in Colombia, it varied between 7.1% and 8.5%. The average global cost attributed to the disease is $2.7 million and it is the fifth leading cause of death in Colombia, with a rate of 15 deaths per 100,000 people [7]. At such a high cost, the disease is a major challenge to health systems and an obstacle to economic equity and sustainable development [8,9].

Risk factors associated with this disease have traditionally been studied at an individual level and most of the methods used to confirm theoretical hypotheses based on the analysis of empirical data offer a simple structural model, which is a complex and multifactorial world, provides partial knowledge that is difficult to understand and apply in public health [10,11]. This research aimed to identify whether the causal mechanisms of the main theoretically established risk factors had an empirical correlation with the disease burden. The empirical adequacy of the theorization of the causal mechanism of type 2 diabetes was evaluated, through a structural equation model, with globally accepted risk factors, measured in a transversal way in people living in the city of Santiago de Cali, Colombia.

Methods

Design and data source

AII records from blood glucose tests measuring total cholesterol and lipid profiles were selected from a database containing information on risk measurement factors for no communicable diseases in the city of Santiago de Cali, during 2013. We excluded those with a prior diagnosis of diabetes, a height measurement of 1.35m or less, weight at 35 kg or less, systolic pressure measuring less than or equal to 50 or greater than 220 mm/Hg, diastolic pressure measuring less than or equal to 20 or greater than 180 mm/Hg, triglycerides lower than 80 or greater than 1500 mg/dL, and those with missing data on the variables of main interest in the modeling. In the end, information was obtained for 26 variables and 423 records.

Variables and evaluated effects

The WHO (2)defined a theoretical framework focused on globally accepted factors with data available to estimate exposure in the population on which pathways or mechanisms for its reduction are known. This was the starting point for a specification model that could preserve classification factors (distal, intermediate, and direct) as well as a priori factorial structure identifying the person's socioeconomic status as a behavioral conditioner, reflected in diet and a sedentary lifestyle (Figure 1). (Lifestyles including alcohol and tobacco consumption, physical activity, and dietary changes, reported being associated with the occurrence of metabolic syndrome, influence increases in blood glucose levels as a predictor of diabetes. Pathways that would directly or indirectly affect blood glucose levels were posed and lifestyles were assumed usual pattern behaviors in the respondents [12–17].

The response variable, type 2 diabetes, was defined based on the blood glucose level, measured by the glycated hemoglobin test. Three categories were stipulated: a) norm glycemic: individuals with blood glucose levels lower than 5.6%, b) pre-diabetic, individuals with blood glucose levels between 5.7% and 6.4%, and e) diabetic, those with blood glucose levels higher than 6.5%.

Any factor that could confuse the relationship that was sought through the study was presented as a variable with direct effects on both the exposure of interest and the dependent variable; this was the case for independent variables such as age, sex, ethnicity, tobacco, and alcohol consumption. Measurement errors were assumed independent of each other, indicators as endogenous and factors as exogenous variables that varied and covaried freely. The standard reflexive measurement of the factors was used, assuming that there were positive intercorrelations between the indicators of the same factor, added to the fact that the exogenous variables contain measurement error terms that had to be manifested at the level of the indicator and not at the level of the latent compound. It is worth noting that each error term represented a unique variance, and it is a proxy indicator of all residual variation sources that were not explained by the model [18].

When variables like socioeconomic status, present a priority effect on other variables, such as diet and physical activity, the latter was considered as endogenous variables that have a disturbance (residual error) designated as "D" to represent all the unmeasured effects of the factor and not only those derived from the measurement error. In turn, physical activity and diet would, directly and indirectly, affect blood sugar levels, mediated through a factor called metabolic syndrome. It is worth noting that priority effects between latent variables and the variations of the disturbances were calculated by controlling the measurement error in the observed variables.

In this research, several alternative models were considered that played an important role in the phase of re-specification and detection bias due to the presence of equivalent models.

Statistical analysis and structural equation models (SEM)

The analysis was conducted in four stages: model specification, identification, estimation, and assessment. A specified and over-identified model was available. The method of robust weighted least squares was used to estimate the SEM, to salve the inconvenience of violating the multivariate normality assumption [19]. STATA/SE V.11.1 and descriptive statistics were used for database debugging, and then the Lavaan package of the R software version 3.3.3 was used for model estimation [20]. Inherent weights to the sampling design were not used in the adjustment of the estimates since the calculation of the results was based on the analysis of a subsample formed from the filters of the inclusion-exclusion criteria, so that these weights were not inherited.

The model fit was assessed in terms of the magnitude and significance of the estimated parameters, and the variance explained by the independent variables of interest. Several statistical measures were used to derive the correspondence of the model with the data: the goodness of absolute fit (Chi-square), the relative fit (CFI), and the parsimonious fit (RMSEA). Since none of these procedures provides the information needed to assess the model, they were used simultaneously. Modification of the indices was used to evaluate and select the specific routes or mechanisms identified by the model with the best fit, a p-value<0.05 was considered statistically significant.

Results

Exploratory analysis

Among the 423 participants, 86 (20.33%) were classified as norm glycemic, 141 (33.33%) as pre-diabetic, and 196 (46.33%) as diabetic. 65.01% were female, age range 12-98 years, the highest proportion of people with type 2 diabetes, 27.04%, were between 40 and 50 years of age. 62.7% of the population earned less than the current legal minimum wage and 51.3% fell within a range of 6-11 years of schooling.

When comparing the group of diabetics and pre-diabetics with norm glycemic individuals, at a significance level of 5%, it is observed that the former were more likely to have high diastolic blood pressure, 7.09% and 16.84% compared to 16.28%, p=0.008, have high LDL cholesterol levels 6.63% and 2.13% vs. 0.0%, p=0.001, consume alcoholic beverage 10.64% and 5.51% vs. 17.44%, p=0.008 and have high total cholesterol levels 20.57% and 18.37% vs. 12.79%, p= 0.006 (Table 1).

SEM and route or mechanism analysis

The estimation process of the first over-identified recursive structural model, with a measurement of 4 factors and 23 indicators, converged to an admissible solution. However, the model failed the Chi-square test whose value was equal to 474.94, with 269 degrees of freedom and a p-value of 0.00. Besides, it presented poor convergent validity, the low explanatory power of the factor on the variability of its constitutive indicators, and an AVE (Average Variance Extracted) less than 0.5 for most of the constructs, which suggests that more factors are required because the variables involved do not measure a common domain. RMSEA (90% CI) equal to 0.045 (0.030-0.052), p-value for RMSEA equal to 0.87.

The modeling consisted of the following steps to locate the source

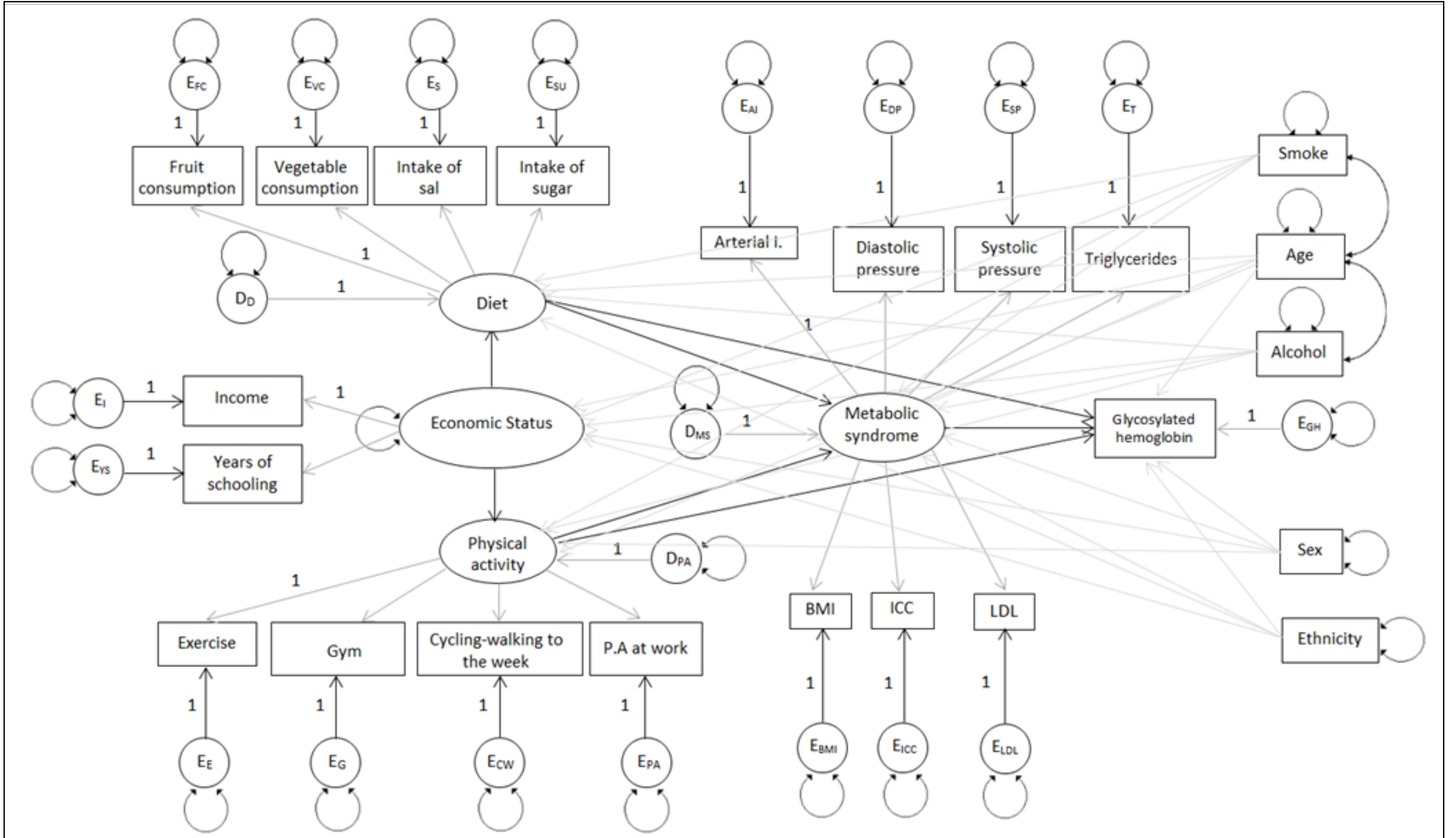

Figure 1: Global Structural Modelling for mechanisms producing type 2 diabetes.

Source: Compiled by author.

Table 1: Population characteristics according to blood glucose level: normal, pre-diabetes, and diabetes.

Characteristics	Normal		Pre-diabetics		Diabetes		Total		p-value Pearson χ^2
	n	%	n	%	n	%	n	%	
Sex									0.844
Male	29	33.72	52	36.88	67	34.18	148	34.98	
Female	57	66.28	89	63.12	129	65.81	275	65.01	
Age									0.465
11 – 20	3	3.49	10	7.09	6	3.06	19	4.49	
21 – 30	14	16.28	29	20.57	21	10.71	64	15.13	
31 – 40	12	13.95	16	11.35	20	10.20	48	11.35	
40 – 50	20	23.26	27	19.15	53	27.04	100	23.64	
51 – 60	18	20.93	25	17.73	43	21.94	86	20.33	
61 – 70	12	13.95	20	14.18	32	16.33	64	15.13	
71 – 80	3	3.49	9	6.38	13	6.63	25	5.91	
>80	4	4.65	5	3.55	8	4.08	17	4.02	
Years of schooling									0.433[a]
0	2	2.33	3	2.13	4	2.04	9	2.13	
1 – 5	27	31.40	27	19.15	55	28.06	109	25.77	
6 – 11	38	44.19	78	55.32	101	51.53	217	51.30	
12 – 16	17	19.77	28	19.86	28	14.29	73	17.26	
17 – 20	2	2.33	5	3.55	8	4.08	15	3.55	
Income									0.683

≤ $589.500b	53	61.63	85	60.28	127	64.80	265	62.65	
> $589.500	33	38.37	56	39.72	69	35.20	158	37.35	
Body-mass index									0.394[a]
Underweight (≤18.5)	0	0.00	3	2.13	4	2.04	7	1.65	
Normal (18.5 - 24.9)	39	45.35	64	45.39	71	36.22	174	41.13	
Overweight (25.0 - 29.9)	33	38.37	46	32.62	83	42.35	162	38.30	
Obesity (≥ 30)	14	16.28	28	19.86	38	19.39	80	18.91	
Waist-to-hip ratio									
Men									0.769
≤1	10	34.48	21	40.38	23	34.33	54	36.49	
> 1	19	65.52	31	59.62	44	65.67	94	63.51	
Women									0.638 [a]
≤0.8	1	1.75	4	4.49	3	2.33	8	2.91	
>0.8	56	98.25	85	95.51	126	97.67	267	97.09	
Triglycerides (mg/dL)									0.490
≤150	46	53.49	70	49.65	98	50.00	214	50.59	
151-199	14	16.28	37	26.24	43	21.94	94	22.22	
200 – 499	26	30.23	34	24.11	55	28.06	115	27.19	
Blood pressure (mm/Hg)									
Systolic pressure									0.08
≤ 130	78	90.70	130	92.20	166	84.69	374	88.42	
>130	8	9.30	11	7.80	30	15.31	49	11.58	
Diastolic pressure									0.025
≤ 85	72	83.72	131	92.91	163	83.16	366	86.52	
>85	14	16.28	10	7.09	33	16.84	57	13.48	
Cholesterol HDL (mg/dL)									0.073
<40	35	40.70	72	51.06	100	51.02	207	48.94	
40 – 60	39	45.35	40	28.37	69	35.20	148	34.99	
>60	12	13.95	29	20.57	27	13.78	68	16.08	
Cholesterol LDL (mg/dL)									0.001
<100	46	53.49	52	36.88	59	30.10	157	37.12	
100 – 129	21	24.42	38	26.95	48	24.49	107	25.30	
130 – 159	17	19.77	39	27.66	51	26.02	107	25.30	
160 – 189	2	2.33	9	6.38	25	12.76	36	8.51	
> 190	0	0.00	3	2.13	13	6.63	16	3.78	
Total Cholesterol (mg/dL)									0.006
<200	63	73.26	78	55.32	99	50.51	240	56.74	
200 – 239	12	13.95	34	24.11	61	31.12	107	25.30	
≥ 240	11	12.79	29	20.57	36	18.37	76	17.97	
Smokes									0.985
Si	6	6.98	10	7.09	13	6.63	29	6.86	
No	80	93.02	131	92.91	183	93.37	394	93.14	
Alcohol Consumption									0.008

Si	15	17.44	15	10.64	11	5.61	41	9.69	
No	71	82.56	126	89.36	185	94.39	382	90.31	
Exercise									0.118
Si	23	26.74	45	31.91	76	38.78	144	34.04	
No	63	73.26	96	68.09	120	61.22	279	65.96	

[a]Fisher's exact test, [b]legal minimum wage (2013)

of poor fit: first, the measurement model was proposed (Figure 2) and once an acceptable model was reached, the structural part was evaluated. The factorial model was reformulated eliminating those indicators that did not have a significant factorial load and that presented notable measurement problems, configuring them into potential factors of mismatch in the model, such as the waist-to-hip ratio, salt intake, sugar intake, intense physical activity at work and level of economic income.

The first outlined model (Figure 2) presented a convergent and admissible solution, and it fitted the data well with an x^2 value of 67.87 and an associated p-value of 0.255. The values of other adjustment statistics are favorable (RMSEA=0.017, CFI= 0.98). More convergent validity is evident in this specification and correlations between factors ranging from -0.162 to 0.312, i.e. moderate correlations indicating discriminant validity. The re-specification required examination of modification indices that suggested error covariance between weight and size indicators belonging to the obesity factor, and LDL vs. triglycerides of the lipid profile factor. Under the mentioned arguments and considering the pertinent modifications, a second structural model was configured (Figure 3).

Variables related to diet and socioeconomic status were excluded from the model because of their poor convergent validity, low reliability of the constituent indicators, more than 90% of the variability explained by measurement error, and increased overall model mismatch. Covariates such as sex, age, tobacco, and alcohol consumption were also tested. However, they did not present statistical significance. A substantial improvement in adjustment indicators was evidenced compared to the first model. The χ^2 statistic value was 44.58, with an associated p-value of 0.447, so the exact fit hypothesis is not rejected (Table 2).

The RMSEA = 0.006 with a confidence interval that has not a 5% value, indicates that the hypothesis of a close fit is not rejected, this conclusion is corroborated by the p-value=0.009. The relative adjustment index CFI, went from 0.729 in Model 1, to 0.999 in Model II, a value very close to 1.0, which indicates the best adjustment. Estimates of factorial loads, variances, and covariance's of Model II are shown in Table 3. The standardized loads for the lipid profile and blood pressure constructs are high, with a range of variation of 0.530 - 0.928 and 0.608 - 0.800 respectively.

Although the physical activity construct presents very low factorial loads in its indicators, AVE = 12.6%, the panorama is different for the lipid profile and blood pressure constructs, which present high factorial loads, so that they can explain, on average, 50.2% and 50.4% of the variability of their constituent indicators, respectively. AII error variances are significant, except the one associated with the arterial index (0.238 which rejects the hypothesis that this index is measured with little error. AII error covariance's are significant and no significance was found in the association between physical activity and obesity. This correlation is not very large, but its presence helps to "clean" some local adjustment problems in some parts of the measurement model (Table 3).

The lipid profile effect on glycosylated hemoglobin is significant, with a value of 0.608. An increase of one point in the lipid factor, consisting of LDL, triglycerides, and arterial index, predicts an increase in the blood glucose level of 0.608 units in its original metric. Statistical significance was also found in the trajectory coefficient directed from the physical activity towards the lipid profile; so that an increase in physical activity generates a decrease of 0.318 in the lipid profile. Similarly, an increase in the lipid profile causes an increase of 0.768 units in the blood pressure factor, a relationship found to be statistically significant (Table 4).

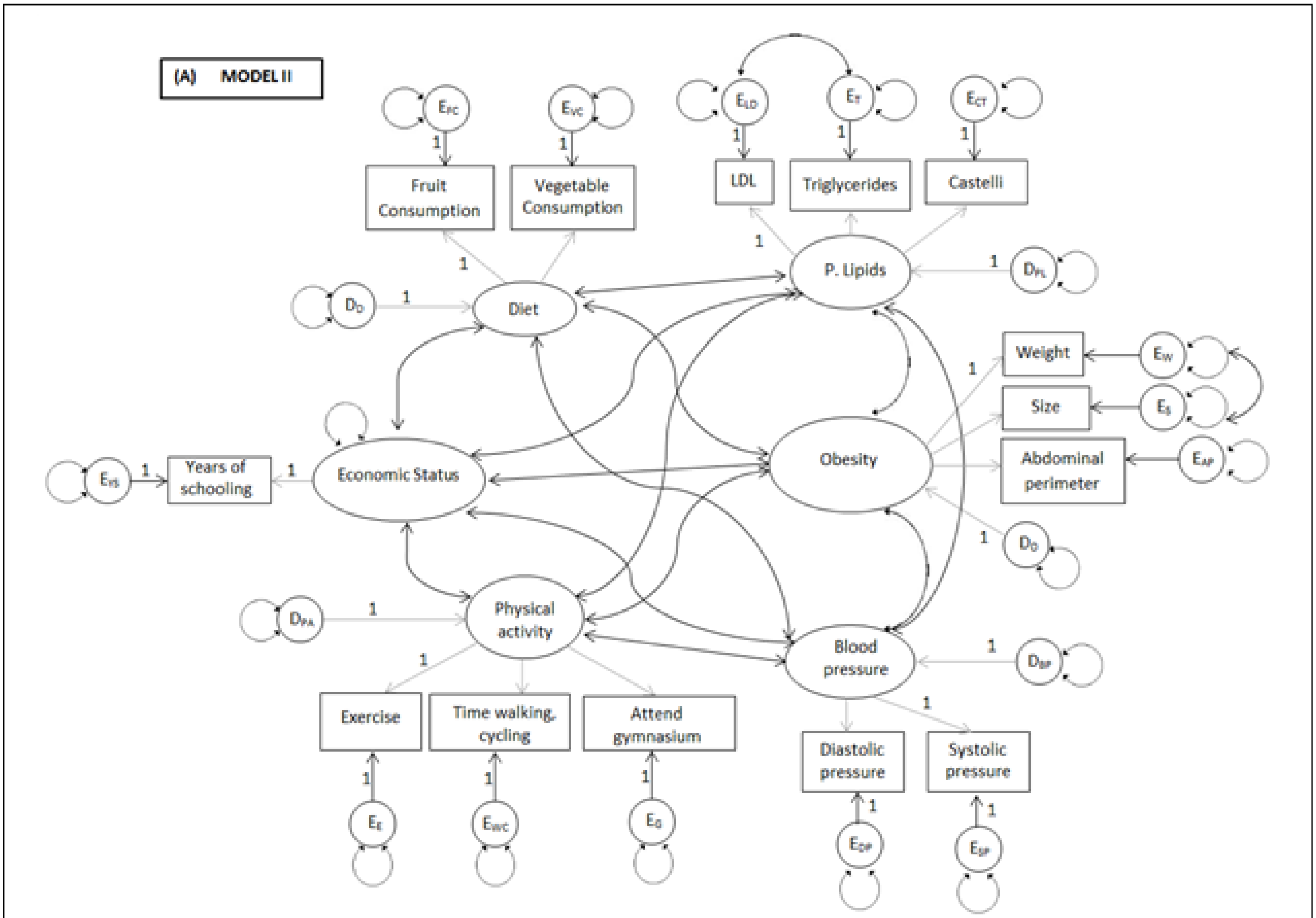

Figure 2: Model I. Re-specified Measurement Model.

Table 2: Comparison of the adjustment statistics in Model I vs. Model II.

Index	Model I (Figure 2)	Model II (Figure 3)
χ^2_M	474.944	44.588
df_M	269	44
P	0.000	0.447
RMSEA (90% IC)	0.045 (0.039 – 0.052)	0.006 (0.000 – 0.035)
P-value RMSEA ≤ 0.05	0.872	0.999
CFI	0.729	0.999

Obesity's indirect effect on blood glucose levels, mediated by the lipid profile, is equal to 0.466. The rationale for this derivation is as follows: even though obesity does not seem to have a direct effect on blood glucose level, it has a direct effect on lipid profile (0.768), and part of this effect (0.608) is transmitted to blood glucose levels. The 0.466 result indicates that it is expected for sugar blood levels to increase in 0.466 units due to obesity increase in one point, mediated through the lipid profile. The total effect of physical activity on blood glucose level is -0.344, i.e. a decrease of 0.344 units is observed in blood glucose level due to physical activity increase in one unit, through all causal relationships direct and indirect, between these two variables (Table 4).

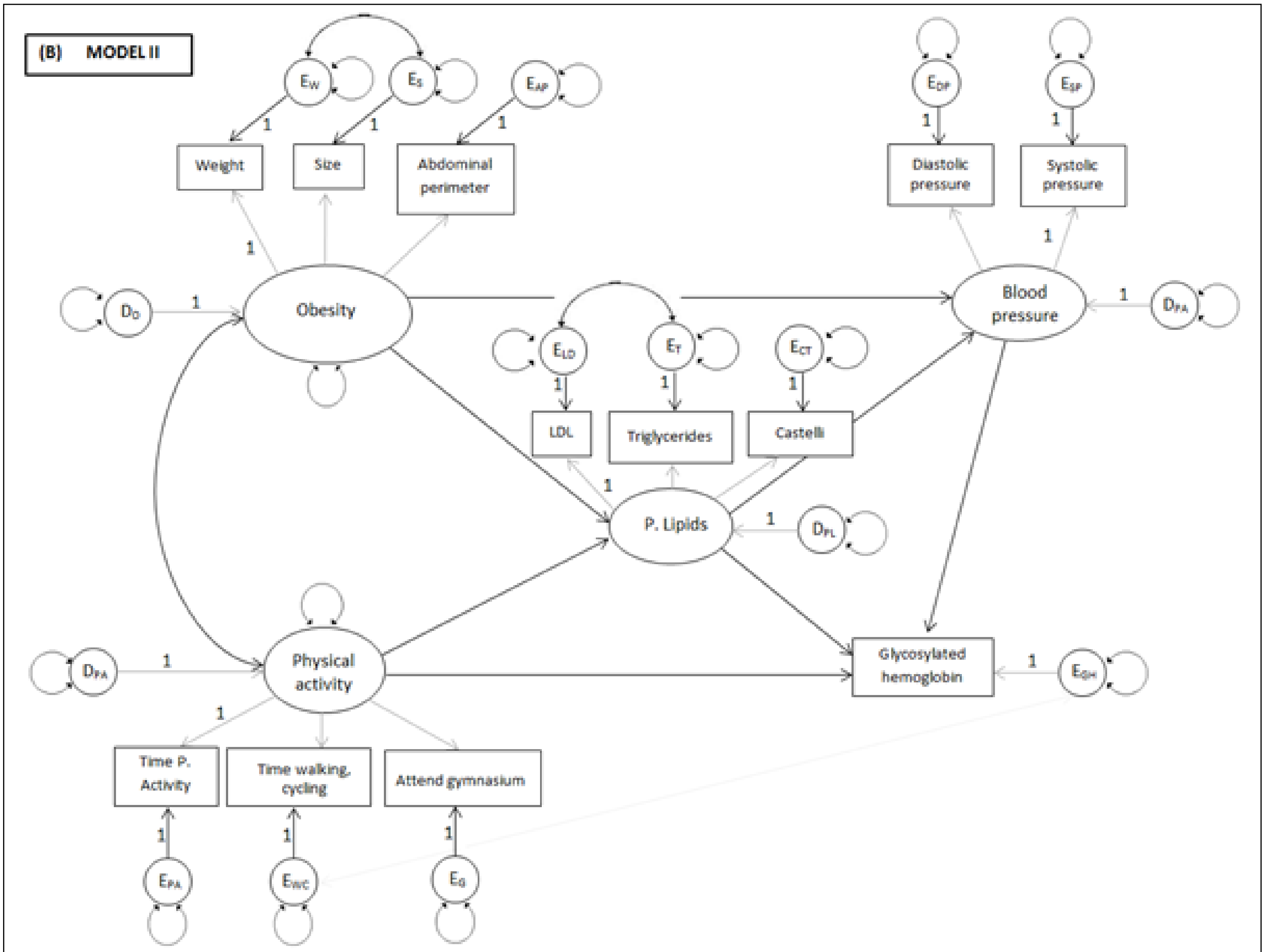

Figure 3: Model II. Re-specification of Causal Mechanisms of Type 2 Diabetes.

Discussion

A model was constructed under the hypothesis proposed in previous studies demonstrating causal associations between sociodemographic, behavioral, anthropometric, and metabolic risk factors, considered responsible for the occurrence of type 2 diabetes [21–25]. The fundamental hypothesis of this model is that the effect of the socioeconomic status on the level of glucose in the blood is indirect and other mediators like diet and physical activity, which in turn affect it directly and indirectly through another factor named metabolic syndrome [26–28], transmit it.

The results of the first stated model showed a poor representation of the constructs for the constitutive indicators in the model of measurement, which represent latent variables that may account, in average, for less than 20% of the variability of its indicators, lack of convergent validity and most of the explained variance is charged to the measurement error variances. In the assessment of the model goodness of fit to data, some discrepancies were found between the predicted and the estimated values (χ^2_M = 474.944, CFL = 0.729, RMSEA = 0.045) which are higher than the expected by chance, for this reason, this model was rejected as the explanation of the chain of causalities of the type 2 diabetes, and the model re-specified.

The resulting model did not take into consideration the socioeco-

Table 3: Estimation of factor loads, variances and covariances of the Model II.

Parameter	Not standardized	Standard error	P-value	Standardized
Factor Loads				
Physical Activity				
Time physical activity	1.000	-	-	0.230
Time cycling or walking	1.452	0.816	0.052	0.291
Attend gymnasium	1.760	0.004	0.007	0.492
Lipid profile				
LDL	1.000	-	-	0.604
Triglycerides	1.452	0.226	0.000	0.530
Arterial I.	1.760	0.275	0.000	0.928
Obesity				
Weight	1.000	-	-	0.718
Size	0.182	0.063	0.004	0.190
Abdominal perimeter	1.191	0.206	0.000	0.833
Blood pressure				
Systolic pressure	1.000	-	-	0.800
Diastolic pressure	0.652	0.194	0.001	0.608
Variances and covariances				
Time physical activity	987.510	462.612	0.033	0.947
Time cycling or walking	1504.679	557.233	0.007	0.915
Attend gymnasium	0.016	0.005	0.002	0.758
LDL	873.743	99.639	0.000	0.635
Triglycerides	2704.319	321.380	0.000	0.719
Arterial I.	250.877	212.427	0.238	0.139
Weight	82.561	16.747	0.000	0.485
Size	78.253	6.127	0.000	0.964
Abdominal perimeter	55.096	22.567	0.015	0.307
Weight ↔ Size	41.230	6.766	0.000	0.513
LDL ↔ Triglycerides	-404.201	141.124	0.004	-0.263
Time walking- cycling ↔ Diabetes	183.510	75.740	0.015	0.089
Physical activity ↔ Obesity	5.041	7.344	0.492	0.072
Factor Variances				
Diabetes	2822.662	410.113	0.000	0.934
Physical activity	55.299	37.386	0.139	1.000
Lipid profile	446.507	97.665	0.000	0.890
Obesity	87.834	17.686	0.000	1.000
Blood pressure	2287.571	750.757	0.002	0.909

Table 4: Estimation of trajectory coefficients for Model.

Direct effects			
	Estimation	Standard error	P-value
Lipid Profile			
Obesity	0.768	0.184	0.448
Physical activity	-0.318	0.227	0.001
Blood Pressure			
Lipid profile	0.768	0.184	0.000
Obesity	-0.318	0.227	0.162
Glycosylated hemoglobin			
Blood pressure	0.034	0.085	0.688
Lipid profile	0.608	0.160	0.000
Physical activity	-0.143	0.419	0.733

nomic status or the diet as factors explaining the relationship with blood glucose level, directly or indirectly. Unlike other models [27,29] indicating significant causal associations between these factors and diabetes, the absence of contribution is possible due to the omission of more important predictive variables or to measurement errors of indicators in the definition of factors of diet and socioeconomic status. This is based on the poor convergent validity evidenced, the indicators low reliability, and the high proportion of variability explained by the measurement error, about 90%. This lack of consistency does not mean that direct effect between them does not exist, what it does point out is that its constitutive indicators are not well defined or measured, which causes confusing causal inferences on how to be useful for clinical or public health purposes.

The second model specified indicates that physical activity, blood pressure, and lipid profile have a direct effect on blood glucose levels. So physical activity causes variations on blood pressure, obesity, and lipid profile that are factors directly responsible for the modification of blood glucose.

The model showed a good overall adjustment (χ^2_M = 44.588, CFI = 0.999, RMSEA = 0.006) its measurement component presented a mixed convergent validity; on the one hand, an explanation of important variability in the lipid profile, blood pressure, and obesity constructs (0.502, 0.504 y 0.415) though it is still a small one for the physical activity factor that explains only 12.6% of the average variability of the constituent indicators.

Physical activity protective effect on blood glucose levels is not direct but is mediated by the lipid profile (-0.193). The lipid profile constituted by the variables LDL, triglycerides, and the arterial index has a positive effect constituted on the levels of sugar in the blood (0.608), as well as it has it on the factor arterial pressure (0.768). No significant associations were found between obesity and physical activity; not between obesity and the lipid profile factor and blood pressure with blood glucose levels.

Blood pressure direct effect on blood glucose levels was confused by the lipid profile and, in part, by obesity represented by abdominal girth, weight, and height. This finding is supported by a previous study that found an increase in insulin resistance affecting glucose metabolism in patients with hypertension, regardless of the level of obesity compared to their controls with normal blood pressure [30].

Physical activity plays an important role in controlling factors associated with diabetes occurrence. increases in the level of physical activity were associated with decreases in dyslipidemia (-0.318) and high blood pressure (-0.244), results consistent with those obtained by Bardenheier et al. [27] in the modeling of diabetes risk factors using SEM.

No relationship was found between socioeconomic status and diet constructs, with factors such as alcohol intake, tobacco consumption, and race/ethnicity recognized in the disease production chain. This could be due to the omission of variables necessary for construct construction, to the lack of precision in measurement instruments, to ambiguity in question formulation resulting in imprecise responses, to the presence of memory and complacency bias, especially concerning behaviors subject to social control.

Among this study limitations, we have: the reliability and consistency of the measurement instrument used, although the instrument was validated in similar contexts; the error or bias in the measurement of the variables; observer errors, in this case of interviewer errors on the field, of individuals who accepted to participate in the interview, for this reason, a generalization of the empirical findings to new environments, contexts or populations is difficult (external validity), and to the set of variables that were not collected and that can affect the construct measurement [31]. Similarly, if the occurrence of diabetes were largely due to the accumulation of the deleterious effect of complex social processes throughout a lifetime, these would be better understood if studied longitudinally. The unique cross-sectional nature of this study, which was not repeated, imposes certain limitations on the causal inference, affecting the external validity of the study.

The main strength of this study lies in the formulation of a model that allows for the simultaneous evaluation of both the formation and the measurement of non-observable variables (theoretical constructs) from the interweaving of causal relationships for the testing of theoretical hypotheses based on the analysis of empirical data, which allows simulating the way these interrelations are presented in a complex, multifactorial and multilevel world [6].

Ethical Aspects

The Institutional Committee of Human Ethics (CIREH) of the Health Faculty of the Universidad Del Valle approved the protocol, in September 2016.

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