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Assessment of Stress and Related Biochemical Alterations in Type 2 Diabetes Mellitus and Obesity Patients

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Diabetes Mellitus, Depression, Stress, Cortisol, Adiponectin.

Abstract:
The metabolic illness known as type 2 diabetes mellitus (T2DM) is still linked to physiological impairment and affects a sizable percentage of the world's population. There are a number of risk factors for developing type 2 diabetes, but obesity and altered basal metabolic rate are among the most prominent. About 80% of those with T2DM are also found to be obese; those with obesity have a higher risk of acquiring depression, and the disease is thought to double that risk. The vast majority of studies show that T2DM affects neuronal physiology via changing cortisol and adiponectin levels in the blood. This research looked at the correlation between metabolic disorders including type 2 diabetes and the stress and depression risk factors cortisol and adiponectin.

1. Introduction

The predicted rate of rise in prevalence in the previous two decades has already exceeded that of high-income nations, making diabetes a growing health care concern primarily impacting low- and middle-income countries. About three-quarters of the world's estimated 1.1 billion individuals living with diabetes are in low- and middle-income nations. There are now over 537 million persons with T2DM, according to the most recent statistics provided by IDF in 2021, and this number is expected to rise to about 783 million by 2045. The estimated number of people worldwide with diabetes in 2025 was 438 million in 2010, about a decade ago. However, the actual number has already exceeded the estimate by roughly 99 million. Ethnicity, age, family history of diabetes, birth weight, obesity, socioeconomic level, and the degree of westernisation are all thought to have a role in the observed variations in prevalence from country to country.[1]

One-quarter of the world's diabetic population resides in Southeast Asia, making diabetes one of the fastest increasing global health issues of the 21st century, as shown by the findings of the most recent edition (10th) of the IDF. India has 74.2 million people with diabetes, followed by Bangladesh with 13.1 million, Sri Lanka with 1.4 million, Nepal with 1.1 million, and Mauritius with 0.25 million. India, China, and the United States continue to have the highest per capita rates of diabetes worldwide. One in six persons worldwide has diabetes, and India is often cited as the epicentre due to its high prevalence . Increased insulin resistance, greater abdominal adiposity, lower adiponectin levels, and higher Creactive protein levels are all examples of the so-called "Asian Indian Phenotype". As a worldwide public health epidemic, type 2 diabetes poses a serious danger to the economics of all countries, but especially those in development. Rapid urbanisation, dietary shifts, and an increase in sedentary lifestyles have contributed to

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a global increase in the prevalence of type 2 diabetes.[2-3]

The association between overweight (BMI and obesity and an elevated risk of incident diabetes is well established clinically and described extensively in the written literature. In order to provide a whole picture, it is crucial to note that coexisting obesity is one of the most strongly related variables with the rising incidence of type 2 diabetes. Patients with type 2 diabetes mellitus often have a BMI more than 25.0. The data also show that the percentage of people who are overweight or obese has risen from 22.5% to 30.0% to 34.6% between 1994 and 1998, 1999 and 2004, and 2005 and 2010, and from 2.7% to 4.9% to 6.4% between 2005 and 2010. Worryingly, the prevalence of obesity among children and adolescents has increased fourfold since 1980 .[4-5]

2. Material and Methods

Clinically diagnosed cases of type 2 diabetes mellitus were the subjects of this cross-sectional investigation. Patients were included if they had been using antidiabetic medication for at least 6 months and had a body mass index (BMI) of less than 30. Patients were included or excluded based on predetermined criteria. Jamia Hamdard Institutional Ethics Committee gave their approval to begin the project. Participants provided written informed consent. Lucknow, is where patients from Altis Hospital were enrolled. After enrolment, patients were evaluated once for symptoms of sadness, stress, and adiponectin levels using blood samples.

The purpose of the research was to determine whether or not people with type 2 diabetes fall into one of two distinct phenotype:

1) The stress reaction of patients as evaluated by questionnaires.

2) Secondly, the levels of stress-related biochemical markers.

SampleSizeEstimation

"In order to detect a significant difference of 20% between groups for cortisol and adiponectin levels, a sample size of 42 subjects per group and control was deemed sufficient. This would yield a total of 126 subjects." 153 participants were needed (51 in each group) to account for the anticipated 20% attrition rate.

Inclusion criteria

All patients who met the inclusion criteria were asked to participate.

- Patients of either sex with diabetes who are older than 18 but younger than 65
- Patients who have signed an informed consent form are eligible to participate in the trial.
- Hb1Ac 6.5%, which is the cutoff for people with type 2 diabetes as determined by the American Diabetes Association
- Patients whose diabetes has been under control for the preceding six months while using an oral hypoglycemic agent
- Patients whose BMI is 30.0 or higher

Exclusion criteria

Here are the conditions that won't be met:

- Patients who are current or former smokers
- Patients suffering from alcoholism or drug addiction
- Patients with any other severe mental disorder, including but not limited to schizophrenia and mental retardation
- Patients whose hypertension is not under control (BP > 180/105 mm Hg).
- Patients using any kind of psychoactive medication
- Patients having a history of psychiatric illness, patients with a current diagnosis of a mental or behavioural problem, and patients with significant cognitive impairment
- Women who are pregnant or nursing
- Patients who declined to or were unable to provide informed written permission.

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Clinical data

Demographicandclinicaldatawasrecordedbasedonaco mmonformat.Thedocumented characteristics were, height, sex, age, weight, diabetes duration. Body massindexforeachsubjectwascalculated.Forcases,HbA 1candfastingbloodglucoselevelswerenoted.Healthchec kupreportsof thehealthyparticipantswereobtained.

Evaluation of Adiponectin in Serum

Participants were asked to fast overnight before having 5 mL of blood drawn in the morning (between 6.00 and 8.00 am). Following the instructions in the ELISA handbook, the serum was isolated from the blood.

Measurement of Cortisol in Serum

In order to ensure reliable and consistent cortisol measurement, samples were taken first thing in the morning (between 6:00 and 8:00 a.m.) "since cortisol levels in serum are impacted by episodic release of cortisol and the resultant diurnal fluctuation. While the patient was fasting, a blood sample of around 5 mL was taken. Serum cortisol levels were measured using chemiluminescence after being isolated from whole blood in accordance with ELISA protocol."

Stress evaluation

Patients' levels of stress were measured using SCQquestionnaire. Due to the wide range of reactions to stress in the human population, around 10% of captives emerge from their experience psychologically stronger. While some people may have little trouble with stress, others may be confronted with tremendous emotional hardship. The SCQ is useful for evaluating aspects of management that contribute to success.

Depression evaluation

The widely used Patient Health Questionnaire (PHQ-9) was used to evaluate the severity of depression and depressive symptoms. (PHQ-9 is a self-report form of the standard diagnostic tool for common mental illnesses. It's the nine DSM-IV categories that make up the depression module. Scores on the PHQ-9 range from 0 to 3, with 0 indicating never and 3 indicating almost daily.

Analytical Statistics

Using the biochemical indicators (cortisol and adiponectin), we were able to identify two groups within the diabetes population. "K-Means cluster analysis revealed two additional subgroups within the T2DM population based on the questionnaire-based scores (PHQ-9 and SCQ) variables. ANOVA was carried out on the control (A) and diabetic (B) groups, as well as the two clusters within the diabetic group, respectively, after cluster identification. Tukey test-based comparisons were regarded to indicate a statistically significant difference between groups if p 0.05 was found. The accuracy of the discovered clusters was then assessed by comparing the clusters based solely on biochemical parameters with those based solely on questionnaire-based scores. The R-Project's R, at version 3.5.3, was used for the cluster analysis."

3. Results

A total of 158 participants, including 105 people with type 2 diabetes who were overweight.

The study included 53 healthy individuals as controls. Patients from HAH Centenary Hospital's outpatient clinic provided the subjects for this study. Fifty-three (N=53) normally-developed adults, with a mean age of 45.08 years, participated in the current investigation. Mean age was 47.03 years for the group of 105 diabetes patients that were included (25% men and 75% women).

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Table1: Age, sex, and racial breakdown of participants Age, sex, and racial breakdown of participants

		Weight (kg)	Age (years)	RBS (mg/dl)	Height (cm)	Pulse (beatsper minute)	SBP(mmHg)	DBP(mmHg)
Control (N=53)	**SEM**	1.36	1.11	3.2	1.12	1.18	2.1	1.19
	Mean	69.95	45.08	108.92	152.45	95.79	136.26	88.34
Diabetes/Obese (N=105)	**SEM**	1	0.82	8.36	0.75	0.94	1.76	0.89
	Mean	76.17	47.3	204.9	151.42	94.03	135.59	87.22
Diabetes/Obese - C1 (N=61)	**SEM**	1	1.16	10.02	1.03	1.05	2.3	1.23
	Mean	76.7	46.57	215.05	152.38	93.66	134.7	88.05
Diabetes/Obese-C2 (N =44)	**SEM**	1.56	1.1	13.94	1.08	1.72	2.75	1.25
	Mean	75.43	48.32	191.9	150.1	94.55	136.82	86.07

C1, patients with diabetes/obesity who are not depressed; C2, patients with diabetes/obesity who are depressed. DBP, diastolic blood pressure; SBP, systolic blood pressure; RBS, random blood sugar; M, male; F, female; SEM, standard error of the mean.

The PHQ-9 and the SCQ were given to every single person who took part in the research. Participants' PHQ-9 results allowed us to divide them into two groups: those with a depressed phenotype and those without. The results of the SCQ were used to divide the sample into two groups: those who were more vulnerable to the effects of stress and those who were better able to bounce back from it. which is crucial to the idea of the detected clusters being distinct from each other while sharing comparable HbA1c and BMI values.

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Table2: K-means cluster analysis: divide respondents into two groups according to their PHQ-9 and SCQ scores

		N	Average	STD	SEM	Maximum	Minimum	Median	p-value
PHQ-9 (Score)	**Diabetes/ Obese**	105	9	4.58	0.447	16	1	9	< 0.001
	Diabetes/ Obese - C1	46	4.5	2.05	0.302	8	1	4	
	Diabetes/ Obese - C2	59	12.5	2.4	0.312	16	9	13	
SCQ (Score)	**Diabetes/ Obese**	105	2.6	0.73	0.071	3.8	1.3	2.6	< 0.001
	Diabetes/ Obese - C1	46	3.1	0.35	0.052	3.8	2.5	3.1	
	Diabetes/ Obese -C2	59	2.2	0.67	0.087	3.7	1.3	2.1	

Glycated haemoglobin (HbA1c); C1, patients with diabetes/obesity who do not experience depression; C2, patients with diabetes/obesity who do experience depression; The Patient Health Questionnaire (PHQ-9) The Stress Coping Questionnaire (SCQ); Statistical Variation Dispersion; Standardised Mean Difference, SEM

Clusters of diabetics with similar levels of adiponectin and cortisol were found using biochemical estimates. Individual cluster analysis was performed first, with individuals being placed into groups according to their cortisol (p 0.001) and adiponectin levels (p = 0.001), and then a combined cluster analysis was performed using both biochemical data .

Table3: Using K-Means, divide subjects into two groups according to their cortisol and adiponectin levels.

		N	Average	STD	SEM	Maximum	Minimum	Median	p-value
Cortisol (µg/dL)	**Diabetes / Obese**	105	11	4.3	0.42	22.7	0.6	10.6	< 0.001
	Diabetes/ Obese - C1	62	8.3	2.55	0.323	11.4	0.6	9.1	
	Diabetes/ Obese - C2	43	15	3.03	0.462	22.7	11.8	13.7	
Adiponectin	**Diabetes/ Obese**	105	2.8	0.88	0.086	6.3	1.3	2.7	0.001

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(µg/mL)	**Diabetes/ Obese - C1**	62	3.2	0.79	0.1	6.3	2.2	3.1	
	Diabetes/ Obese - C2	43	2	0.38	0.058	2.7	1.3	2.1	

C1, healthy diabetics/obese individuals; C2, depressed diabetics/obese individuals; STD, Standard deviation; Mean discrepancy, or SEM "In addition, the cluster symmetry/similarity was evaluated using biochemical parameter analysis and compared with questionnaire answers, where it was discovered that the two independent studies had an accuracy of similar cluster formation of 85%. Final clusters were determined using questionnaire response and biochemical parameter estimates together, taking into account the identical clusters . C1 diabetes affects 61 (58%) of the 105 individuals while C2 diabetes affects 44 (42%)."

Table 4: Changes in a number of factors

Variables	**BMI (kg/m2)**	**HbA1c (%) ***	**Cortisol (µg/dL)**	**Adiponectin (µg/mL)**	**PHQ-9 (Score)**	**SCQ (Score)**
Control (N=53)	26.1±2.2	4.6±1.0	10.3±3.5	7.2±2.0	6.0±1.6	2.7±0.5
Diabetes/Obes e (N=105)	33.1±3.1$^{\#}$	8.6±1.5$^{\#}$	11.0±4.3	2.8±0.9$^{\#}$	9.0±4.6$^{\#}$	2.6±0.7
Diabetes/Obes e - C1 (N=61)	33.0±3.1$^{\#}$	8.6±1.5$^{\#}$	8.2±2.6$^{\#\$}$	3.2±0.8$^{\#\$}$	5.8±2.9$^{\$}$	3.1±0.4$^{\#\$}$
Diabetes/Obes e - C2 (N=44)	33.2±3.1$^{\#}$	8.6±1.6$^{\#}$	14.9±3.1$^{\#\$\&}$	2.1±0.4$^{\#\$\&}$	13.4±2.0$^{\#\$\&}$	1.8±0.4$^{\#\$\&}$

The percentage differences between the clusters and the control population and their parent diabetic population for cortisol and adiponectin levels were significant. Diabetes-C2 (45%) had significantly higher serum cortisol levels compared to controls, while the diabetes parent pool (7% of subjects) showed no change, diabetes/obese-C1 (-20%) showed significantly lower, and diabetes-C2 (45%) had significantly higher values compared to controls. It's important to highlight that the changes in both groups were statistically distinct regardless of the direction of the shifts. Serum cortisol levels of the diabetic pool were not different from those of the control population, but there was a clear differentiation between diabetes/obese-C1 and diabetes/obese-C2 in terms of cortisol levels, with C1 showing lower values and C2 showing higher ones, indicating that C1 can be considered stress-resilient. Due to increased cortisol readings associated with a greater stress perception, the diabetes/obese-C2 group might be considered stress susceptible.

Lower values of adiponectin are associated with stress perception, and the fact that reductions in adiponectin were smaller in diabetes/obese-C1 (-56%) than in diabetes/obese-C2 (-71%), with an absolute 15% more reduction in diabetes/obese-C2 (stress vulnerable based on cortisol values), bolsters the case. It is important to highlight that the decreased adiponectin levels seen in the diabetes group when compared to the control group are consistent with the existing literature. Adiponectin levels are much lower in the stress-vulnerable group (diabetes/obese-C2) compared to those of the stress-resilient group (diabetes/obese-C1), as shown by the clusters of diabetic pools.

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Table 5: Analysis of variance (ANOVA) findings for various factors

	BMI		**HbA1c**		**PHQ-9**		**SCQ**		**Cortisol**)		**Adiponectin**)	
Source of Variation	Between Groups	Within Groups	Between Groups	Within Groups	Between Groups	Within Groups	Between Groups	Within Groups	Between Groups	Within Groups	Between Groups	Within Groups
SS	2093.1	2299.18	651.41	457.58	1869.16	2997.8	1869.16	2997.8	1153.25	3365.67	872.26	326.4
df	3	259	3	227	3	259	3	259	3	259	3	259
MS	697.7	8.88	217.14	2.01	623.05	11.58	623.05	11.5745	384.42	12.99	290.76	1.26
F	78.6	-	107.72	-	53.83	-	53.83	-	29.58	-	230.71	-
P-value	<0.001	-	<0.001	-	<0.001	-	<0.001	-	<0.001	-	<0.001	-
F crit	2.64	-	2.64	-	2.64	-	2.64	-	2.63	-	2.63	-

Tukey post hoc tests compared the control (A) and diabetes (B) groups, as well as the two clusters (C1 and C2) discovered from the diabetic group, and "findings for individual groups were judged significantly different if p 0.05. To assess the reliability of the clusters, those based on biochemical markers were compared to those based on questionnaire scores (PHQ-9 and SCQ). R-Project, R, version 3.5.3 was used for the cluster analysis."

Table 6. Body Mass Index (Single Factor) ANOVA

Summary				
Groups	**SampleCount**	**Sum**	**Average**	**Variance**
GroupA	53	1381.3	26.06	4.82
GroupB	105	3474.71	33.09	9.86
Group C1	61	2012.42	32.99	9.40

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Group C2	44	1462.29	33.23	10.69

TUKEYHSDTest			
Grouppair	**TukeyHSD,p value**	**TukeyHSDQ Statistics**	**Tukey HSDInference**
AvsB	0.001	19.80	p<0.01
AvsC1	0.001	17.51	p<0.01
AvsC2	0.001	16.69	p<0.01
BvsC1	0.900	0.30	Insignificant
BvsC2	0.900	0.37	Insignificant
C1vsC2	0.900	0.58	Insignificant

Table7. One-Way Analysis of Variance for HbA1c

Summary				
Groups	**SampleCount**	**Sum**	**Average**	**Variance**
GroupA	53	243.8	4.60	0.97
GroupB	89	764.8	8.59	2.31
Group C1	52	444.9	8.56	2.23
Group C2	37	319.9	8.65	2.50

TUKEYHSDTest			
Grouppair	**TukeyHSD,p value**	**TukeyHSDQ Statistics**	**Tukey HSDInference**
AvsB	0.001	22.93	p<0.01

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AvsC1	0.001	20.19	p<0.01
AvsC2	0.001	18.81	p<0.01
BvsC1	0.9	0.21	Insignificant
BvsC2	0.9	0.27	Insignificant
C1vsC2	0.9	0.42	Insignificant

Table8. Multi-Way ANOVA (Single Factor): PHQ-9

Summary				
Groups	**SampleCount**	**Sum**	**Average**	**Variance**
GroupA	53	318	6.00	2.46
GroupB	105	941	8.96	21.00
Group C1	61	351	5.75	8.56
Group C2	44	590	13.41	4.01

TUKEYHSDTest			
Grouppair	**TukeyHSD,p value**	**TukeyHSDQ Statistics**	**Tukey HSDInference**
AvsB	0.001	7.31	p<0.01
AvsC1	0.9	0.54	Insignificant
AvsC2	0.001	15.10	p<0.01
BvsC1	0.001	8.28	p<0.01
BvsC2	0.900	0.37	p<0.01
C1vsC2	0.900	0.58	p<0.01

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Table 9. Single-Factor (Single-Analyst) Analysis of Variance

Summary				
Groups	**SampleCount**	**Sum**	**Average**	**Variance**
GroupA	53	243.8	4.60	0.97
GroupB	89	764.8	8.59	2.31
Group C1	52	444.9	8.56	2.23
Group C2	37	319.9	8.65	2.50
TUKEYHSD Test				
Group pair	**Tukey HSD,p value**	**Tukey HSDQ Statistics**	**Tukey HSD Inference**	
AvsB	0.462	2.07	Insignificant	
AvsC1	0.001	5.32	p<0.01	
AvsC2	0.001	10.87	p<0.01	
BvsC1	0.001	8.37	p<0.01	
BvsC2	0.001	10.40	p<0.01	
C1vsC2	0.001	16.25	p<0.01	

Table 10. One-Way Analysis of Variance for Cortisol

Summary				
Groups	**SampleCount**	**Sum**	**Average**	**Variance**
GroupA	53	544.82	10.28	12.34

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GroupB	105	1158.60	11.03	18.52
Group C1	61	503.20	8.25	6.51
Group C2	44	655.40	14.90	9.46

TUKEYHSDTest			
Grouppair	**TukeyHSD,p value**	**TukeyHSDQ Statistics**	**Tukey HSDInference**
AvsB	0.588	1.76	Insignificant
AvsC1	0.015	4.24	p<0.05
AvsC2	0.001	8.88	p<0.01
BvsC1	0.001	6.79	p<0.01
BvsC2	0.001	8.43	p<0.01
C1vsC2	0.001	13.18	p<0.01

Table11. Adiponectin One-Way Analysis of Variance

Summary				
Groups	**SampleCount**	**Sum**	**Average**	**Variance**
GroupA	53	381.68	7.20	3.85
GroupB	105	288.95	2.75	0.77
Group C1	61	197.51	3.24	0.63
Group C2	44	91.44	2.08	0.19

TUKEYHSDTest			
Grouppair	**TukeyHSDQ Statistics**	**TukeyHSD, pvalue**	**TukeyHSD Inference**

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AvsB	33.27	0.001	p<0.01
AvsC1	26.59	0.001	p<0.01
AvsC2	31.65	0.001	p<0.01
BvsC1	3.80	0.038	p<0.05
BvsC2	4.73	0.0053	p<0.01
C1vsC2	7.39	0.001	p<0.01

Comparisons of PHQ-9 and SCQ score changes between these clusters and the control group and their parent diabetes group revealed that they were distinct from both groups. Both PHQ-9 and SCQ scores were significantly different between the diabetes parent pool (50%) and the diabetes/obese-C1 (-3%) and the diabetes/obese-C2 (123%) groups, with the former showing significantly higher values compared to the control group and the latter showing significantly lower values. A noteworthy fact for both groups is that the changes were statistically distinct in both directions.

Although diabetics as a whole did not have significantly lower SCQ scores than controls, there was clear differentiation between those in diabetes/obese-C1 and those in diabetes/obese-C2 in the identified clusters. The results of this research show that people with diabetes or obesity (C1) had lower cortisol and greater adiponectin responses than those with type 2 diabetes or obesity (C2). PHQ-9 scores further support the cluster distinction, with the stress-vulnerable population averaging 13. and the stress-resilient population averaging 5.8 .

4. Discussion

The IDF projected that 537 million people throughout the world were living with T2DM in 2021 Comorbidities, especially obesity and depression, contribute significantly to the rising incidence of type 2 diabetes across the world. It is noteworthy that the prevalence of obesity, depression, and type 2 diabetes has all risen at a similar, and increasing, pace. Several research points to the two-way connections between type 2 diabetes and depression, as well as between depression and obesity. Patients with type 2 diabetes and obesity had a 1.63-fold greater risk of depression compared to those with type 2 diabetes alone, according to one of the most influential meta-analyses. Biological processes governing peripheral and neural metabolism converge at HPA axis, which is shared by the illness triad of type 2 diabetes, obesity, and depression. When under stress, the HPA axis is where the action. Alterations in adipokines, which may play a role in mediating the obesity-depression link, have not been well investigated, however.[6-7]

Elevated cortisol release under prolonged stress has been linked to depression. Fluctuating plasma levels of cortisol, a crucial role in metabolic illness, are associated with an increased risk of insulin resistance, hyperglycemic condition, an increase in the number of cases of hypertension, lower levels of HDL-C, higher levels of triglycerides, and abdominal obesity. Type 2 diabetes causes fluctuating cortisol levels in response to emotional distress, increasing the danger of developing metabolic syndrome.[8] In addition to its anti-inflammatory and anti-atherogenic properties, adiponectin has been linked to the regulation of visceral fat accumulation and triglyceride levels .9 The presence of type 2 diabetes also modifies plasma adiponectin levels and related functions, which may raise the risk of developing metabolic diseases. There is a connection between the stress and depression-induced decrease in adiponectin plasma level and the increased risk of metabolic illness.[10]

5. Conclusion

Results may not be indicative of true prevalence of depression in T2DM/obese population. Patients were

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selected using extensive screening procedures and recruited from a Delhi metropolitan area tertiary care centre. If primary and secondary health care settings are studied for dynamic real-world data, the depressive phenotype in the T2DM/Obese pool will be representative of the true burden. Our inability to make definitive conclusions regarding the potential role of diabetes or obesity in depression and their reciprocal effects on the lifespan of glycemic control is due to the exclusion of depressed non-type 2 diabetic or obese controls from our analysis. However, a cross-sectional study design can only investigate the links between diabetes/obesity and depression, while a longitudinal study design is more suited to explore the interdependencies of comorbidities.

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