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Association Between Plasma Leptin Levels and Self-Reported Screen-Time Among Type 2 Diabetics with Obesity

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ABSTRACT

Background: In the current digital era, increased screen time is associated with sedentary behavior, which can adversely impact metabolic and hormonal health. Leptin, a hormone secreted by adipose tissue, plays a crucial role in energy homeostasis and is implicated in obesity and type 2 diabetes mellitus (T2DM). Emerging evidence suggests a link between screen exposure and leptin regulation, yet this relationship remains underexplored in adult diabetic populations.

Objectives: To evaluate the association between plasma leptin levels and self-reported screen time, and to assess its potential impact on metabolic health among individuals with T2DM and obesity.

Methods: A cross-sectional study was conducted over two years at Pacific Medical College & Hospital, Udaipur. A total of 250 obese participants were enrolled and divided into two groups: 125 with T2DM and 125 without. Demographic, anthropometric, and biochemical data, including fasting blood glucose, HbA1c, and leptin levels, were collected. Screen time was self-reported via structured interviews. Statistical analysis was performed using SPSS, with significance set at $p < 0.05$.

Results: A positive correlation was observed between screen time and plasma leptin levels (Pearson's $r = 0.199$; $p < 0.05$). No significant differences were found in fasting glucose or HbA1c across screen-time categories. Difference in BMI with screen-time was statistically significantly (ANOVA F statistics = 5.809, p value <0.05).

Conclusion: Prolonged screen exposure is significantly associated with increased leptin levels among obese individuals with T2DM. These findings highlight leptin as a sensitive biomarker for sedentary behavior and underscore the importance of lifestyle interventions targeting screen-time reduction in metabolic disease management.

Keywords: *Leptin, Screen time, Type 2 diabetes mellitus, FBS, BMI, Obesity*

1. INTRODUCTION

In the modern digital era, screen time has significantly increased across all age groups due to the widespread use of smartphones, computers, televisions, and tablets. This rise in sedentary behavior is associated with numerous adverse health outcomes, particularly in metabolic and hormonal regulation. One hormone that has gained considerable attention in recent years in relation to obesity and energy homeostasis is leptin.[1] Leptin is a peptide hormone primarily secreted by adipocytes (fat cells) and plays a central role in the regulation of body weight, appetite, and metabolism. Acting on receptors in the hypothalamus, leptin signals satiety and helps modulate energy intake and expenditure. Alterations in leptin levels or leptin resistance are strongly linked with obesity, insulin resistance, and metabolic syndrome.[2]

Increased screen time is frequently associated with physical inactivity, poor dietary patterns, reduced sleep quality, and increased risk of overweight and obesity—all of which may influence leptin secretion and function. Emerging research

suggests a bidirectional relationship between screen time and leptin regulation, where sedentary behavior may disrupt leptin signaling, and abnormal leptin levels may contribute to behavioral patterns such as prolonged screen use and disrupted circadian rhythms.[3] Despite growing evidence, the precise mechanistic link between screen time and leptin dynamics remains underexplored. Understanding this relationship could provide valuable insights into early metabolic dysregulation and inform preventive strategies targeting childhood and adolescent obesity, which are often influenced by lifestyle habits, including screen use.[4]

Leptin, a hormone primarily secreted by adipose tissue, plays a pivotal role in regulating body weight by signaling satiety and influencing energy homeostasis. The discovery of the **leptin gene (ob gene)** by Zhang et al. (1994) and its function as an adiposity signal, as elucidated by Friedman and Halaas (1998), laid the groundwork for understanding the hormonal regulation of obesity and metabolism.[5,6] Several studies have demonstrated that alterations in leptin secretion or signaling are closely associated with obesity and metabolic dysfunctions. Elevated leptin levels, often observed in individuals with high body fat, are commonly associated with leptin resistance—a condition in which the brain does not respond adequately to leptin signals, resulting in continued food intake despite energy sufficiency.[5]

Sleep deprivation, which is commonly linked to excessive screen time, has also been shown to affect leptin levels. In a study by **Spiegel et al. (2004)**, short sleep duration led to a reduction in leptin levels and an increase in ghrelin, thereby promoting increased appetite and potential weight gain.[7]These hormonal fluctuations suggest that screen time could indirectly influence leptin regulation through its impact on sleep and circadian rhythm.

The interaction between lifestyle factors—particularly **screen time** and **sedentary behavior**—and leptin dynamics has gained increasing research interest. **Rey-López et al. (2008)** and **Robinson et al. (2017)** highlighted that sedentary behaviors, including prolonged screen exposure, contribute significantly to the development of childhood and adolescent obesity.[8,9] These behaviors are often accompanied by poor dietary habits, reduced physical activity, and disrupted sleep—all of which may alter leptin secretion and impair its physiological functions.

Furthermore, **Chaput and Tremblay (2007)** reported that cognitive sedentary activities, such as screen use or computer work, might increase energy intake beyond physiological needs, potentially disturbing leptin balance.[10] **Taveras et al. (2005)** and **Belot and James (2011)** emphasized that family and environmental contexts, including mealtime habits and screen time during meals, are important determinants of both leptin levels and overall metabolic health in adolescents.[11,12]

Emerging studies have begun to explore leptin's role in behavioral regulation and its feedback relationship with screen time. **Shroff et al. (2014)** found a significant correlation between altered adipokine profiles (including leptin) and cardiovascular risk markers in children with high sedentary time.[13] Additionally, **Thivel et al. (2013)** suggested that modern sedentary lifestyles, characterized by increased screen engagement, not only contribute to overconsumption of calories but also lead to hormonal disturbances that reinforce sedentary patterns and metabolic dysregulation.[14]

2. OBJECTIVES

- To evaluate the association between plasma leptin levels and self-reported screen time, and to assess its potential impact on metabolic health among individuals with type 2 diabetes mellitus.
- To investigate the correlation between prolonged screen exposure and alterations in leptin regulation among individuals with type 2 diabetes mellitus.

3. MATERIALS & METHODS

This was a cross-sectional study over the duration of 2 years in biochemistry department at Pacific Medical College & Hospital, Udaipur (Rajasthan). Calculated estimated sample size was 250 patients by using convenient sampling, a kind of non-probability sampling. Study population was divided in 2 groups. Group 1 included 125 cases of obesity with T2DM and group 2 included 125 cases of obesity without T2DM. Patient was selected on the basis of history, physical examination & preliminary lab investigations. The presence of T2DM was confirmed by consulting physician. All the case subjects selected for study will be without insulin therapy. Patients with history of any of the following diseases were excluded from the study:

a. Diabetes Mellitus type 1
b. AIDS.
c. Cardiac disorders
d. Liver diseases.
e. Patient with effusion & shock.
f. Pregnant women.
g. Lactating mother.

h. Any type of acute or chronic illness

A standardized pre-structured questionnaire with consent was filled by direct interview of admitted patients including the demographic, anthropometric and laboratory data of patients. Ethical clearance from ethical committee has been obtained prior to beginning of study. Blood sample was collected by vein puncture under aseptic precaution. Serum was separated from the sample and analyzed for Blood sugar, HBA1c, Lipid profile and leptin levels. Data was entered in excel sheet and statistical analysis was done using software SPSS (statistical package for social studies). Appropriate tests were applied and p value <.05 was considered as level of significance.

4. RESULTS

Table 1. Demography, anthropometry, laboratory values in diabetics & non-diabetics obese.

Variables	Obese with Type 2 Diabetes Mellitus (n=125)	Obese without diabetes (n=125)	Test statistics	p value
Age (frequency and percentage)				
31-40 years	38 (30.4)	38 (30.4)	Chi square 1.769	.778
41-50 years	35 (28.0)	34 (27.2)		
51-60 years	27 (21.6)	21 (16.8)		
61-70 years	13 (10.4)	15 (12.0)		
71-80 years	12 (9.6)	17 (13.6)		
Sex (frequency and percentage)				
Male	57 (45.6)	56 (44.8)	Chi square 0.016	.899
Female	68 (54.4)	69 (55.2)		
BMI (frequency and average value in kg/m^2)				
25.0 to 29.9	5 (29.5)	11 (28.5)	***t = 4.01***	**.0013**
30.0 to 34.9	83 (33.4)	97 (32.5)	***t = 4.90***	**.0001**
35.0 to 39.9	37 (36.2)	17 (35.5)	***t = 2.19***	**.0326**
FBS (mg/dl) Mean ± SD	174.06 ± 10.52	91.99 ± 9.75	***t = 63.99***	**.0001**
HBA1c (gm%) Mean ± SD	7.98 ± 0.59	5.2 ± 0.47	***t = 41.07***	**.0001**
Leptin (ng/ml) Mean ± SD	42.60 ± 10.52	35.52 ± 10.52	***t = 2.80***	**.0054**

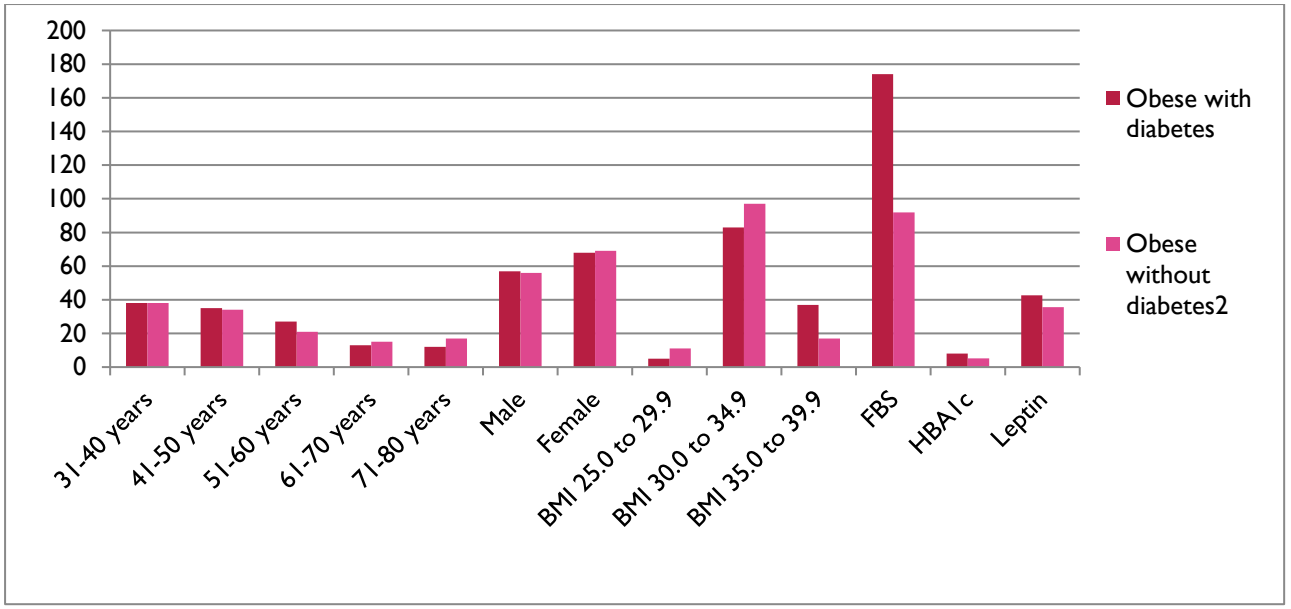

Figure 1 Demography, anthropometry, laboratory values in diabetics & non-diabetics

Table 1 summarised the demographic, anthropometric and laboratory data in diabetics & non-diabetics obese population of our study. Majority of study population belong to 4th decade with female preponderance showing no significant difference for age and sex by Chi square. Both groups were found to have statistically significant difference for BMI, Fasting blood sugar, HBA1c and Plasma leptin levels (single t test, p value <.05).

Table 2. Association between self-reported screen-time with BMI and biochemical markers among obese with type 2 diabetes melitus

Variables	Self reported Screen-time (hours/day)				ANOVA statistics
	≤ 2 hours	>2 to 4 hours	>4 to 8 hours	>8 hours	
BMI (kg/m^2)	34.0 ± 1.95	34.11 ±1.97	33.9 ± 1.81	36.32 ± 2.63	F = 5.809 ***p = 0.0009***
FBS (mg/dl)	174.83 ±12.35	174.38 ± 9.29	173.69 ±10.51	173.25 ±7.68	F = 0.095 *p = 0.9626*
HBA1c (gm%)	8.05 ±0.5667	8.00 ±0.6928	7.91 ±0.5682	8.33 ±0.4761	F = 2.231 *p = 0.0880*
Leptin (ng/ml)	11.19 ± 0.313	24.07 ± 6.382	42.74 ± 7.168	61.26 ±12.991	F = 153.48 ***p = 0.000***

Table 2 shows association between self-reported screen-time with BMI and biochemical markers. As per WHO report[15] which recommends limiting recreational screen time to **less than or equal to 2 hours/day** for children and adolescents to avoid negative health outcomes, we have taken 'less than or equal to 2 hours screen time' as a reference group. No significant difference was observed in Fasting Blood Sugar (FBS) and glycated Haemoglobin (HBA1c) for screen time. There is statistically significant difference was found in Body mass index (ANOVA F statistics = 5.809, p value <0.05) and the plasma leptin level (ANOVA F statistics = 153.48, p value <0.05) with increasing screen time.

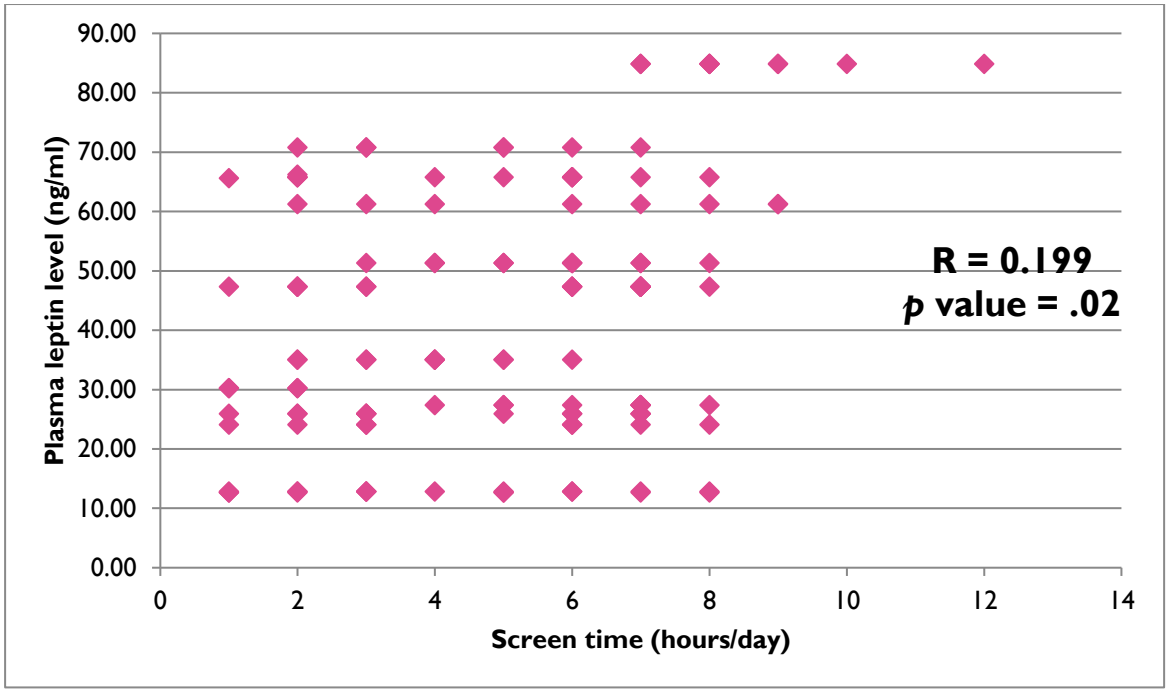

Figure 2 Scatter plot showing relation between screen-time and plasma leptin level

Figure 2 is a scatter plot illustrating correlation between screen-time and plasma leptin level. Pearson's correlation coefficient (R) between screen time and leptin level was 0.199 with $p < 0.05$ showing positive correlation.

5. DISCUSSION

This study aimed to evaluate the association between plasma leptin levels and self-reported screen time, and to assess its potential impact on metabolic health among individuals with type 2 diabetes mellitus (T2DM) and obesity. Our results demonstrated a statistically significant positive correlation between screen time and plasma leptin levels, with Pearson's coefficient ($r = 0.199$) indicating a strong positive relationship. These findings suggest that increased screen exposure may be associated with disrupted leptin regulation in obese individuals with T2DM.

Leptin, secreted primarily by adipose tissue, plays a central role in energy homeostasis by signaling satiety and regulating appetite via hypothalamic pathways.[1,5] The observed higher leptin levels with prolonged screen time is particularly noteworthy, as it may reflect alterations in either leptin secretion or sensitivity. This finding aligns with existing literature suggesting that sedentary behavior and circadian rhythm disruption—both commonly associated with extended screen use—can interfere with hormonal balance, including leptin dynamics.[7,10]

Although both diabetic and non-diabetic obese groups showed elevated mean leptin levels, the leptin concentration increased significantly with increasing screen time, regardless of glycemic status. Importantly, difference in BMI with screen-time was statistically significantly (ANOVA F statistics = 5.809, p value <0.05) while fasting blood sugar and HbA1c did not show significant variation across screen time categories, further emphasizing the specific sensitivity of leptin to behavioral and environmental factors like screen exposure.

Our results support the hypothesis of a bidirectional interaction between screen time and leptin regulation. On one hand, excessive screen use may increase leptin secretion or impair its signaling via mechanisms such as reduced physical activity, altered sleep patterns, and increased sympathetic activity.[7,10,14] On the other hand, dysregulated leptin signaling—particularly leptin resistance—may influence behavioral patterns by weakening satiety cues, thereby promoting sedentary habits and increased screen engagement.[13,14]

Previous studies have primarily focused on pediatric or adolescent populations, where similar associations between sedentary behaviors and leptin levels have been reported.[3,8,13] However, our study extends this understanding to adults with metabolic co-morbidities such as T2DM, where hormonal regulation is already compromised. This highlights the need for lifestyle interventions that incorporate screen-time management as a potential strategy for improving metabolic outcomes in high-risk populations.

6. SUMMARY

This cross-sectional study investigated the association between plasma leptin levels and self-reported screen time among individuals with obesity, with and without type 2 diabetes mellitus. A total of 250 participants were enrolled, divided equally between diabetic and non-diabetic obese groups. While both groups had elevated leptin levels, a significant positive correlation was observed between screen time and plasma leptin levels. No significant differences were found in fasting blood glucose, or HbA1c across varying durations of screen time, highlighting leptin as a potentially sensitive biomarker influenced by sedentary digital behavior. These findings support the emerging hypothesis that prolonged screen exposure may disrupt leptin regulation and contribute to metabolic dysregulation in obese individuals, especially those with T2DM.

7. CONCLUSION

The study demonstrates a strong positive association between screen time and plasma leptin levels in obese individuals with type 2 diabetes, suggesting that extended screen exposure may play a role in hormonal imbalance and metabolic dysfunction. Although causality cannot be inferred due to the cross-sectional design, the findings emphasize the importance of monitoring screen time as part of lifestyle interventions in metabolic disease management. Further longitudinal studies are needed to establish causal relationships and explore whether reducing screen time can improve leptin sensitivity and overall metabolic outcomes in this high-risk population.

8. LIMITATIONS

Our study has several limitations. First, the use of self-reported screen time may be subject to recall and reporting biases. Second, the cross-sectional nature of the study limits causal inference; it is unclear whether increased screen time leads to decreased leptin levels or if altered leptin function contributes to behavioral tendencies. Third, we did not control for confounders such as dietary intake, sleep duration, or levels of physical activity, all of which could influence leptin levels independently. Lastly, the study population was limited to a single tertiary care center, which may affect generalizability.

Despite these limitations, our findings suggest that screen time may play a significant role in leptin dysregulation among obese individuals with T2DM. This opens avenues for further longitudinal or interventional studies to assess whether reducing screen time can reduces leptin resistance and overall metabolic health. Future research should also aim to explore mechanistic pathways linking digital exposure to hormonal changes and evaluate the effects of screen-time interventions in clinical practice.

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