ISSN 0973-2063 (online) 0973-8894 (print)

Bioinformation 22(7): 4346-4349 (2026)

BIOINFORMATION
Discovery at the interface of physical and biological sciences

BIOMEDICAL INFORMATICS

www.bioinformation.net

Volume 22(7)

Research Article

Received July 1, 2026; Revised July 31, 2026; Accepted July 31, 2026, Published July 31, 2026

DOI: 10.6026/973206300224346

SJIF 2026 (Scientific Journal Impact Factor for 2026) = 8.478
2022 Impact Factor (2023 Clarivate Inc. release) is 1.9

Edited by A Prashanth
E-mail: phyjunc@gmail.com
Phone: +91 7259404071
Citation: Hariprasath *et al.* Bioinformation 22(7): 4346-4349 (2026)

Assessment of thyroid status in prediabetes

G. Hariprasath [1], B. Jyothirmayi[1], P. Ramana Sai[1] & K. Senthil Kumar[2,*]

[1]Department of Biochemistry, Madha Medical College & Research Institute, Chennai, Tamil Nadu, India; [2]Department of Physiology, Madha Medical College & RI, Tamil Nadu, India; *Corresponding author

Affiliation URL:
https://mmcri.in/

Author contact:
G. Hariprasath - E-mail: gopal.hariprasath@gmail.com; Phone: +91 8838736107
B. Jyothirmayi - E-mail: drjyothi71@gmail.com
P. Ramana Sai - E-mail: parasugani71@gmail.com
K. Senthil Kumar - E-mail: drksk.cool@gmail.com; Phone: +91 9940416604

ISSN 0973-2063 (online) 0973-8894 (print)
Bioinformation 22(7): 4346-4349 (2026)

Abstract:
Prediabetes and thyroid dysfunction are common endocrine disorders that may influence the progression of diabetes and its complications. Therefore, it is of interest to evaluate the thyroid status in 129 prediabetic subjects and 100 healthy controls using fasting plasma glucose, postprandial plasma glucose, glycated hemoglobin and thyroid profile measurements. Glycemic parameters were significantly higher in prediabetic subjects than in controls ($p<0.01$). Serum thyroid-stimulating hormone levels were significantly higher in prediabetic subjects despite remaining within the normal reference range, whereas free triiodothyronine and free thyroxine levels showed no significant differences between groups. Thus, data shows that early thyroid functional alterations in prediabetes and support regular thyroid screening for timely detection of endocrine abnormalities.

Keywords: Thyroid hormones, screening, prediabetes, thyroid dysfunction and diabetes mellitus

Background:
Prediabetes is an intermediate state of hyperglycemia with blood glucose range above normal but below the diabetes threshold **[1]**. The American Diabetes Association defines Prediabetes by the presence of Impaired Fasting glycemia and/or Impaired Glucose Tolerance and/or having $HbA1_C$ levels in the range of 5.7–6.4% **[2]**. Though the etiology of prediabetes is not known, insulin resistance and Beta cell dysfunction were considered to play key role in the pathogenesis of Prediabetes **[3]**. Individuals with prediabetes generally have an increased risk of developing diabetes and its associated complications such as diabetic retinopathy, neuropathy, nephropathy and macrovascular complications **[4].** Thyroid hormone is a major regulator of metabolism and energy expenditure and has been considered as an important factor in the regulation of glucose homeostasis **[5]**. Thyroid dysfunction *i.e.* both hypothyroidism and hyperthyroidism were found to be associated with diabetes mellitus. Subjects with thyroid hormone deficiency are more likely to develop obesity, metabolic syndrome and diabetes than healthy individuals **[6].** Hyperthyroidism also can induce glucose intolerance via lowering insulin secretion and peripheral insulin sensitivity **[7]**. In addition, studies have reported variations of thyroid hormone levels even within the normal reference range to be associated with increased risk of developing Type 2 Diabetes Mellitus **[8]**. Thus, Thyroid dysfunction if undiagnosed would additionally increase the risk for diabetes and its associated complications in subjects with Prediabetes **[9]**. Therefore, it is of interest to assess the thyroid hormone levels in subjects with prediabetes and healthy controls and to determine the prevalence of thyroid dysfunction among prediabetic individuals.

Materials and methodology:
This Cross-sectional study was conducted in Department of Biochemistry, Madha Medical College & Research Institute, Chennai. The study involved 129 Prediabetic subjects and 100 ages matched healthy controls. Individuals with Prediabetes (subjects with impaired fasting plasma glucose (FPG) 100–125 mg/dL or Post Prandial blood plasma glucose 140-199 mg/dl or Glycated hemoglobin level in the range of 5.7% to 6.4 %) were selected as cases in this study. The study was approved by the institutional ethics committee and informed consent was obtained from all the study subjects who themselves voluntarily participated in this study. Subjects with previous history of thyroid diseases, on steroid medications, pregnancy, with liver and kidney diseases were all excluded from the study. Fasting plasma glucose, Post Prandial plasma glucose, HbA1c and Thyroid profile were all measured in these subjects.

Statistical analysis:
Statistical analysis was carried out using SPSS 20.0 and values were expressed in mean ± standard deviation. Statistical difference between the control and Prediabetic groups were carried out using Student "t" test and p value of < 0.05 was considered to be statistically significant. The prevalence of Thyroid dysfunction between the Prediabetes subjects and controls was compared using chi-square test and p value of <0.05 was considered to be statistically significant.

Results:
(**Table 1**) shows the mean Age, Fasting plasma glucose, postprandial plasma glucose and HbA1c levels in healthy controls and Prediabetic subjects. (**Table 2**) shows the comparison of Thyroid profile among controls and Prediabetic subjects. (**Table 3**) shows the prevalence of type of thyroid dysfunction among controls and Prediabetic subjects. The controls and Prediabetic subjects were of same age group and no significant difference was observed between the groups. Fasting plasma glucose, Postprandial plasma glucose and HbA1c levels were all significantly increased in the Prediabetic group compared to the control group ($p<0.01$). There was no significant difference in mean levels of FT_3 and FT_4 between the control group and the Prediabetic group. However, a significant elevation of Serum TSH level ($p<0.05$) that was within the reference range is observed in Prediabetic subjects when compared to controls. The prevalence of Thyroid dysfunction was higher in the Prediabetic group (6.2%) on comparison with the control group (4%), but this difference was not statistically significant ($p>0.05$). In the Prediabetic group among those with Thyroid dysfunction majority of them had subclinical hypothyroidism (4.7%) with the rest being overt hypothyroid (1.5%).

Table 1: Comparison of age, fasting plasma glucose, post prandial plasma glucose and HBA1C values of controls and prediabetes subjects

Parameters	Controls (N=100)	Prediabetes (N=129)	p-value
Age (years)	34.58±3.65	35.22±4.2	0.23
BMI	21.76±2.84	22.73±2.45	p<0.01
Fasting Plasma Glucose (mg/dL)	87.64±11.56	115.33±7.35	p<0.01
Post Prandial Plasma Glucose (mg/dL)	98.2±14.65	147.85±16.75	p<0.01
HbA1c	5.02±0.45	6.09±0.29	p<0.01

Table 2: Comparison of mean of thyroid profile in healthy controls and prediabetes subjects

Parameters	Controls (N=100)	Prediabetic subjects (N=129)	p-value
FT_3	3.1±0.47	2.98±0.53	0.07
FT_{4p}	1.30±0.32	1.22±0.38	0.09
TSH	3.18±1.56	3.6±1.37	p<0.05

Table 3: Prevalence of type of thyroid dysfunction among the prediabetes subjects compared to controls

Thyroid dysfunction	Controls (N=100)	Prediabetic Subjects (N=129)	p-value
Sub clinical hypothyroidism	3 (3%)	6(4.7%)	0.53
Overt hypothyroidism	1 (1%)	2 (1.5%)	0.71
Total	4(4%)	8(6.2%)	0.47

Discussion:

The present study was carried out to assess thyroid hormone levels among subjects with prediabetes and healthy controls to determine the prevalence of thyroid dysfunction in prediabetic subjects. The participants in both the groups were of same age (34.58±3.65 & 35.22±4.2) and no significant difference was observed between them. The mean Fasting plasma glucose, post prandial plasma glucose and HbA1c levels were all significantly elevated in prediabetics compared to controls. This might be due to the presence of insulin resistance and β-cell dysfunction which were considered as key factors in the pathogenesis of prediabetes **[10]**. In our study the overall prevalence of thyroid dysfunction was 6.2% (8 subjects) in prediabetics (129 subjects) compared to 4% (4 subjects) among controls (100 subjects) and no significant differences was observed between the groups (p>0.05). Several studies have documented variable prevalence rates regarding thyroid dysfunction in subjects with prediabetes. A study by Sakyi *et al.* (2023) reported a very high prevalence of thyroid dysfunction (26.4%) among prediabetics than healthy controls (14.9%) **[11]**. Kandel *et al.* (2024) reported the prevalence of thyroid dysfunction among prediabetics and controls to be 12% and 5%, respectively **[12]**. These differences in the prevalence rates regarding thyroid dysfunction among Prediabetic subjects could be due to the diversity of assays used to measure thyroid function tests **[13]** as well as the upper reference range for TSH selected in these studies **[14]**. Lowering the upper limit of normal in case of TSH could have increased the number of individuals diagnosed with subclinical hypothyroidism. In our study as per the kit manufacturer's instructions the upper reference range for TSH was fixed as 5.1μIU/ml. The difference in study design and the difference in duration of prediabetes among the study subjects in could have also contributed to the variations in the prevalence rate of thyroid disorders among subjects with prediabetes **[15]**. Similarly, Du *et al.* reported a higher prevalence of thyroid disorders among individuals with impaired glycemic status than among those with normal glucose metabolism, further supporting the association between thyroid dysfunction and abnormal glucose homeostasis **[22]**. In our study among the Prediabetics, the most common thyroid disorder observed was Subclinical hypothyroidism (4.7%) which was 3% in controls with the rest being overt hypothyroidism (1.5%) which was 1% in controls. No incidence of hyperthyroidism was observed in our study. Regarding thyroid function tests on comparison with controls, we found that FT_3 and FT_4 levels were not significantly different (p>0.05) and though TSH level was significantly high (p<0.05); it was within normal range in prediabetic subjects **[17]**. Sakyi *et al.* (2023) reported that, compared with controls, TSH levels were significantly higher, while no significant difference was observed in FT4 levels among prediabetic subjects **[11]**. However, Iwakura *et al.* (2023) in their study showed that FT3 and FT4 levels were significantly lower and TSH level was significantly higher in prediabetic group than the control group **[13]**. This discrepancy might be due to the difference in study population as well as diversity of assays used to assess thyroid function **[13]**. The marginally elevated TSH that was within normal range in our study indicates an early stage of disruption of thyroid function **[16]** and an increased risk of developing Type 2 diabetes particularly in individuals with prediabetes **[17]**. These findings are consistent with Liu *et al.*, who demonstrated changes in thyroid function markers among patients with prediabetes, supporting the association between prediabetes and early thyroid dysfunction **[23]**. This could be due to interrelationship between insulin resistance and the Hypothalamic–Pituitary–Thyroid (HPT) axis **[18]**. Both Insulin and Thyroid hormones share a complex bidirectional relationship. Insulin exerts a direct effect over thyroid function, impacting synthesis and release of thyroid hormones **[19]** and thyroid hormones in turn increases insulin sensitivity by enhancing insulin signaling pathway and by promoting glucose uptake into the cells **[20]**. Thus, insulin resistance can affect thyroid function and thyroid dysfunction in turn can contribute to insulin resistance and development of Type 2 diabetes **[19]**. The molecular mechanism linking insulin resistance with TSH level is not clear. Available literature suggests that insulin resistance and compensatory hyperinsulinemia in prediabetes might increase Leptin production and Leptin in turn stimulates Hypothalamic Pituitary Thyroid axis to increase TSH secretion by interfering with the negative feedback regulation mechanism of thyroid hormones **[21]**. Some disorders usually manifest or become detectable only after many years which might be the reason for no significant difference observed in FT_3 and FT_4 levels in our study. A onetime cross-sectional study of thyroid status in prediabetes could be too short to capture the real incidence of thyroid dysfunction. Hence a regular screening for thyroid dysfunction can be done in individuals with prediabetes and it will be beneficial in reducing the progression of onset of diabetes as well as in preventing the progression towards development of overt thyroid disorder.

Conclusion:

Prediabetic individuals exhibited significantly higher glycemic parameters and mildly elevated serum thyroid-stimulating hormone levels within the normal reference range compared with healthy controls. These findings suggest early thyroid functional alterations during the prediabetic stage. Regular

thyroid function screening may facilitate early detection of abnormalities and support timely preventive interventions.

References:

[1] https://pubmed.ncbi.nlm.nih.gov/29083606/
[2] https://pubmed.ncbi.nlm.nih.gov/38078589/
[3] Dagogo-Jack S. *Diabetes*. 2025 **74**:2155. [PMID: 41115173]
[4] Schlesinger S *et al. Diabetologia*. 2022 **65**:275. [PMID: 34718834]
[5] Sabatino L & Vassalle C. *Biomolecules*. 2025 **15**:361 [PMID: 40149897]
[6] Sarabhai T & Kostev K. *Endocr Connect*. 2025 **14**:e240554 [PMID: 39831884]
[7] Eom YS *et al. Diabetes Metab J*. 2022 **46**:239. [PMID: 35385635]
[8] Mohammed Hussein SM & AbdElmageed RM. *Cureus*. 2021 **13**:e20697 [PMID: 35106234]
[9] Roa Dueñas OH *et al. J Clin Endocrinol Metab*. 2022 **107**:1789. [PMID: 35137143]
[10] Vignesh Babu K *et al. Cureus*. 2025 **17**:e79855 [PMID: 40166517]
[11] Sakyi SA *et al. Endocrinol Diabetes Metab*. 2023 **6**:e447 [PMID: 37621219]
[12] Kandel L *et al. JNMA J Nepal Med Assoc*. 2024 **62**:691. [PMID: 40655891]
[13] Iwakura H *et al. BMC Endocr Disord*. 2023 **23**:146 [PMID: 37430240]
[14] Meamar R *et al. Acta Biomed*. 2021 **92**:e2021283 [PMID: 34738602]
[15] Aswani HS *et al. Int J Mol Sci*. 2025 **26**:2170 [PMID: 40076791]
[16] Jun JE *et al. Sci Rep*. 2024 **14**:15169 [PMID: 38956266]
[17] Hu S *et al. BMC Public Health*. 2025 **25**:1220 [PMID: 40165126]
[18] Wei Y *et al. Front Endocrinol (Lausanne)*. 2024 **15**:1366830 [PMID: 39175570]
[19] Rong F *et al. BMC Med*. 2021 **19**:257 [PMID: 34670571]
[20] https://pubmed.ncbi.nlm.nih.gov/29763182/
[21] Li J & Ma W. *Am J Transl Res*. 2025 **17**:9353. [PMID: 41552333]
[22] Du W *et al. Biotechnol Biotechnol Equip*. 2019 **33**:1244. [DOI: 10.1080/13102818.2019.1656106]
[23] Liu B *et al. Front Endocrinol (Lausanne)*. 2021 **12**:657114. [PMID: 34017311]