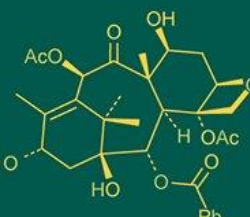
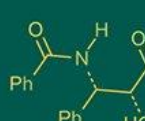


International Journal of Advanced Biochemistry Research



ISSN Print: 2617-4693
ISSN Online: 2617-4707
NAAS Rating (2026): 5.29
IJABR 2026; 10(7): 263-266
www.biochemjournal.com
Received: 02-04-2026
Accepted: 06-05-2026

Hasnaa Yagoub Yousif
BSc, Department of
Biochemistry, College of
Medicine, University of
Basrah, Basrah, Iraq

Salman K Ajlan
Professor. MBChB, MRCP
(End. & Diabetes, UK),
Department of Biochemistry,
College of Medicine, University
of Basrah, Basrah, Iraq

Homeostatic model assessment among patients with impaired fasting glycaemia in Basrah, Iraq

Hasnaa Yagoub Yousif and Salman K Ajlan

DOI: <https://www.doi.org/10.33545/26174693.2026.v10.i7d.8667>

Abstract

Background: Impaired fasting glycaemia (IFG) is a condition, in which fasting plasma glucose (FPG) levels are consistently higher than the normal range, but below the cut-off value for the diagnosis of diabetes. Pancreatic β -cell dysfunction and insulin resistance (IR) have a principal role in the pathogenesis of IFG. Patients with IFG are at high risk of progression to type 2 diabetes (T2D), and developing atherosclerotic cardiovascular disease (ASCVD),

Objective: To determine the degree of IR (via HOMA-IR) and β -cell dysfunction (via HOMA-B) among patients with IFG.

Methods: The study included 143 patients with IFG, 61 males and 82 females, 14-70 years of age, and 150 apparently healthy subjects, 62 males and 88 females, 15-70 years age as a control group. Physiological measurements include weight, height, waist circumference (WC), and blood pressure (BP). Biochemical measurements include FPG, glycated haemoglobin (HbA1c) and insulin. In addition, HOMA-IR and HOMA-B were also determined.

Results: Insulin level was significantly higher among patients with IFG in comparison to control subjects, ($p < 0.000$). There were no significant differences in FPG and HbA1c between IFG and control group, ($p > 0.05$). Concerning HOMA-IR and HOMA-B, patients with IFG showed a significantly higher HOMA-IR and a significantly lower HOMA-B values compared to control subjects, ($P = 0.000$).

Conclusion: Patients with IFG have significantly higher insulin and HOMA-IR and significantly lower HOMA-B values compared with control subjects. This indicates that these patients are a high risk of progressing to T2D and its complications.

Keywords: Impaired fasting glycaemia, insulin resistance, β -cell function

Introduction

Impaired fasting glycaemia (IFG) is a category of impaired glucose regulation, where blood glucose levels during fasting are consistently above the normal range without exceeding the diagnostic cut-off value of diabetes mellitus (DM) [1-3].

β -cell dysfunction and insulin resistance (IR) have an important role in the pathogenesis of IFG [4]. Two conditions of impaired glucose homeostasis intermediate between normality and diabetes have been described. These are the state of IFG and impaired glucose tolerance (IGT). They couldn't be considered as distinct clinical entities but in both, there is an increased risk of development of macro-cardiovascular disease (CVD), but not the micro-vascular complications of type 2 diabetes (T2D). Both conditions increase the future risk of developing diabetes [5].

Initially, in IFG, a compensatory increase in insulin secretion occurs, keeping glucose levels within normal limits. However as the disease advances, insulin secretion is insufficient to maintain glucose homeostasis. People with IFG have a defect with their insulin secretory response to glucose in the early stages [6].

Hepatic IR and normal muscle insulin sensitivity are the hallmarks of the IFG. The development of T2D is linked to obesity and IR. β -cell malfunction is thought to be necessary for hyperglycemia to arise. In addition, β -cell malfunction, which develops early in the course of the disease, is an essential aberration in the presence of IR. A different hypothesis has been proposed, in which primary β -cell overstimulation causes insulin hypersecretion, which leads to obesity and IR, and eventually to β -cell exhaustion [7].

Corresponding Author:
Salman K Ajlan
Professor. MBChB, MRCP
(End. & Diabetes, UK),
Department of Biochemistry,
College of Medicine, University
of Basrah, Basrah, Iraq

Patients with prediabetes typically has no distinct signs or symptoms except the sole sign of high blood sugar [8, 9]. Such individuals should be monitored for symptoms such as increased thirst, polyuria, weight loss and lethargy [10].

Patients with of IFG are at a high risk of developing T2D [4, 11]. The annual conversion rate of IFG to T2D ranges from 3-11% [12-14]. Considerable proportion of people with prediabetes will progress to T2D with high risk in the co-existence of overweight or obesity [6]. Also, it has been found that sedentary lifestyle, obesity and hypertriglyceridemia independently increased the risk for T2D and persistence of prediabetes. HbA1c was the strongest single predictor progression of IFG into T2D [8]. People older than 45 years with visceral obesity and have risk factors such as, first-degree relative with diabetes, history of CVD, hypertension and dyslipidemia should be screened regularly [15, 16].

The aim of the present study was to determine the degree of IR and β -cell dysfunction among patients with IFG.

Patients and Methods

This is a case-control study conducted at Department of Biochemistry, University of Basrah, Basrah, Iraq, from January 2022 throughout May 2022. It included 293 participants, 143 patients with IFG, and 150 apparently healthy individuals matched for both age and sex. All participants attended Al-Sadr Teaching Hospital, Basrah, Iraq, either for medical consultation or regular check-up. Patients with IFG, were 61 males and 82 females, 14-70 years of age. IFG is considered with fasting plasma glucose (FPG) level of 100-125 mg/dL and glycated haemoglobin (HbA1c) value of 5.7-6.4% [17, 18]. With regard to control subjects, they were, 62 males and 88 females, 15-70 years of age, who were apparently health subjects with no history of diabetes and confirmed to be in good health state, by careful clinical examination and biochemical laboratory tests. Informed written agreement was obtained from all participants. Also, the study was accepted by the Ethical Committee at the College of Medicine, University of Basrah. Exclusion criteria include pregnant women, diabetes, patients with gastrectomy, history of thyroid dysfunction, renal disease, liver disease and critical cases. The participants were interviewed using a detailed questionnaire included enquiries regarding socio-demographic characteristics such as: age, gender, marital status, educational level, history of systemic diseases, family history of diabetes, smoking habit and alcohol consumption. All participants in the study were underwent body weight, height and waist circumference (WC) measurements with light clothes and no shoes. Body mass index (BMI) was calculated using the formula (Kg/m^2). Systolic blood pressure (SBP) and diastolic blood pressure (DBP) was measured twice with mercury sphygmomanometer with at least 10 minutes between the two measurements.

Determination of FPG was carried out by reference enzymatic methods [19]. The level of HbA1c was measured by ion exchange high performance liquid chromatography (HPLC) method [20]. Interpretation of HbA1c values was as follows [21]: Non-diabetic values of HbA1c are < 5.7% (National Glycohemoglobin Standardization Program "NGSP") which is equivalent to < 39 mmol/mol (International federation of Clinical Chemistry and Laboratory Medicine "IFCC"). Prediabetic values are 5.7-6.4% (NGSP) or 39-47 mmol/mol (IFCC) and diabetic

values were $\geq 6.5\%$ (NGSP) or ≥ 48 mmol/mol (IFCC). Serum insulin level measured by electrochemiluminescence immunoassay "ECLIA" method [22].

Homeostatic model assessment: β -cell function and IR were measured using the following equations [23-25].

$$\text{HOMA-B} = 360 \times \text{fasting insulin } (\mu\text{IU/ml}) / \text{FBG } (\text{mg/dL}) - 63$$

Where,

B: % of β -cell function, FBG: Fasting blood glucose.

Normal value of HOMA-B: 100%.

$$\text{HOMA-IR} = \text{FBG } (\text{mg/dL}) \times \text{fasting insulin } (\mu\text{IU/ml}) / 405$$

Where, IR: Insulin resistance, FBG: Fasting blood glucose.

Normal value of HOMA-IR: ≤ 2.5 .

Statistical Package for Social Science (SPSS) program version 25 was used to analyze the data of this study and the results were expressed as Mean $\pm$ Standard Deviation (SD) and percentage. For continuous data, independent t-test was used to compare two different groups. Pearson correlation was used to find out the correlation coefficient (r-value) among HOMA-B and HOMA-IR with biochemical parameters and other variables in the study population. P-value of less than 0.05 was considered as the lowest limit for significance.

Results

The distribution of patients with IFG and control subjects by socio-demographic and anthropometric variables is shown in Table 1. There were no significant differences between patients with IFG and control subjects with regard to age, sex and BMI, $p > 0.05$. On the other hand, SBP, DBP and WC in either sex were significantly higher among patients with IFG compared to control persons, ($p < 0.05$).

Table 1. Socio-demographic and anthropometric characteristics

Characteristic		IFG (n = 143)	Controls (n = 150)	P-value
Age (years)		40.9 $\pm$ 10.4	40.4 $\pm$ 12.3	> 0.05
Sex	Male	61(42.7%)	65(43.3%)	> 0.05
	Female	82(57.3%)	85(56.7%)	
SBP (mm.Hg)		123.6 $\pm$ 13.2	120.8 $\pm$ 12.4	< 0.05
DBP (mm.Hg)		75.2 $\pm$ 10.8	70.2 $\pm$ 6.7	< 0.05
BMI (Kg/m^2)		35.8 $\pm$ 5.1	32.8 $\pm$ 6.3	> 0.05
WC (cm) Males	≤ 102	104.0 $\pm$ 22.1	96.9 $\pm$ 8.8	< 0.05
	>102			
WC (cm) Females	≤ 88	101.32 $\pm$ 12.71	93.10 $\pm$ 13.51	< 0.05
	>88			

Data are presented as Mean $\pm$ SD

As presented in Table 2, insulin level was significantly higher among patients with IFG in comparison to control subjects, ($p < 0.000$). On the other hand, FPG and HbA1c were non-significantly higher in the IFG group compared to the control one, ($p > 0.05$).

Table 2: FPG, HbA1c and insulin levels

Variables	IFG (n = 143)	Controls (n = 150)	P-value
FPG (mg/dL)	111.1 $\pm$ 7.0	90.8 $\pm$ 6.9	0.207
HbA1c%	5.8 $\pm$ 0.38	5.3 $\pm$ 0.35	0.239
Insulin ($\mu\text{IU/ml}$)	22.2 $\pm$ 15.1	11.5 $\pm$ 7.3	0.000

Data are presented as Mean $\pm$ SD

Table 3 presents HOMA-IR and HOMA-B values among patients with IFG and control persons. Patients with IFG showed significantly higher HOMA-IR and significantly lower HOMA-B values compared to control subjects, ($P = 0.000$).

Table 3: HOMA-IR and HOMA-B values

Variables	IFG (n = 143)	Control (n = 150)	P-value
HOMA-IR	5.3±3.3	2.5±1.7	0.000
HOMA-B	139.7±79.2	163.2±135.1	0.000

Data are presented as Mean ± SD

Discussion

The prevalence of IFG and IGT continuously rising worldwide, and could reach a figure of 8.4% by 2045 [26]. The risk of progression of people with prediabetes to T2D is appreciable particularly in the absence of intervention [4, 27]. It has been found that, and 23.0% of individuals with prediabetes progressed to T2D, an 4.6% annual rate of progression of 4, 6% [8]. People with undiagnosed and untreated prediabetes are at risk of sustained hyperglycaemia which may results in serious complications [28, 29]. In patients with IFG, the demand for an increase in insulin exceeds the limited capacity of pancreatic β -cells to produce insulin. This results in a decrease in insulin secretion which in turn aggravated by the toxic effect of free fatty acids, and hence hyperglycemia ultimately occurs [30]. Diabetes is a chronic metabolic disorder representing a global public health problem with dramatically increasing prevalence [31, 32]. T2D is the most common type of diabetes, its prevalence is dramatically increasing, and quickly became an epidemic around the world [26, 33, 34]. In Basrah, Southern Iraq, diabetes received considerable interest, and several studies have been conducted in this regard. In addition, the frequency of diabetes in Basrah is high, where a high proportion of patients have undiagnosed diabetes [35-37].

Concerning FBG, HbA1c and insulin levels, insulin level was significantly higher among patients with IFG in comparison to control subjects, (Table 2). This indicates a state of hyperinsulinaemia in association with IFG, which represents a compensatory phenomenon to overcome IR to maintain blood glucose concentration within normal limits [6]. This finding also indicates a state of IR which happened mainly due to a receptor defect and positive feedback between glucose concentration, which is the principal stimulus for insulin release, and β -cell producing insulin. The development of diabetes due to severe IR may be retarded unless the capacity to secrete additional amounts of insulin to compensate for IR is impaired. In addition, Individuals with IR suffer from several abnormalities including impaired glucose tolerance, dyslipidemia, endothelial dysfunction, and elevated inflammatory markers [38]. HOMA-IR index was used to determine the level of IR, whereas the HOMA-B index was used to determine the level of β -cell function and insulin secretion. Also, HOMA-IR is linked to some risk factors for the occurrence complications due to diabetes, including CVD and decreasing renal function [39]. With regard to HOMA-IR and HOMA-B, patients with IFG showed a significantly higher HOMA-IR and significantly lower HOMA-B values compared to control subjects, (Tables 3). β -cells fail to compensate for the predominant IR, therefore prediabetes ensues, and diabetes may ultimately develop. As glucose

levels are elevated, β -cell function deteriorates (low HOMA- β), with decreasing sensitivity to glucose, thus exacerbating hyperglycemia. The pancreatic islet cell mass has been reported to be reduced in size in patients with diabetes [40]. In addition, the observed hyperinsulinaemia may be attributed to the associated visceral obesity with the consequent resistance to the effect of insulin.

In conclusion, patients with IFG have significantly higher insulin and HOMA-IR, and significantly lower HOMA-B values in comparison to with control persons. This implies that patients with IFG a considerable risk of progressing to T2D and, hence, to its diverse complications.

References

1. Rao SS, Disraeli P, McGregor T. Impaired glucose tolerance and impaired fasting glucose. *Am Fam Physician*. 2004;69:1961-1968.
2. Tabak AG, Herder C, Rathmann W, Brunner EJ, Kivimäki M. Prediabetes: a high-risk state for diabetes development. *Lancet*. 2012;379:2279-2290.
3. American Diabetes Association. Classification and diagnosis of diabetes: standards of medical care in diabetes-2019. *Diabetes Care*. 2019;42(Suppl 1):S13-S28.
4. Abdul-Ghani MA, Tripathy D, DeFronzo RA. Contributions of β -cell dysfunction and insulin resistance to the pathogenesis of impaired glucose tolerance and impaired fasting glucose. *Diabetes Care*. 2006;29:1130-1138.
5. American Diabetes Association. Diagnosis and classification of diabetes mellitus. *Diabetes Care*. 2010;33(Suppl 1):S62-S69.
6. Nathan DM, Davidson MB, DeFronzo RA, Heine RJ, Henry RR, Pratley R, *et al*. Impaired fasting glucose and impaired glucose tolerance: implications for care. *Diabetes Care*. 2007;30:753-759.
7. Esser N, Utzschneider KM, Kahn SE. Early beta cell dysfunction vs insulin hypersecretion as the primary event in the pathogenesis of dysglycaemia. *Diabetologia*. 2020;63:2007-2021.
8. Bennasar-Veny M, Fresneda S, López-González A, Busquets-Cortés C, Aguiló A, Yáñez AM. Lifestyle and progression to type 2 diabetes in a cohort of workers with prediabetes. *Nutrients*. 2020;12:1538.
9. Alvares S, Coffey R, Algotar AM. Prediabetes. Treasure Island (FL): StatPearls Publishing; 2022.
10. Poltavskiy E, Kim DJ, Bang H. Comparison of screening scores for diabetes and prediabetes. *Diabetes Res Clin Pract*. 2016;118:146-153.
11. DeFronzo RA. From the triumvirate to the ominous octet: a new paradigm for the treatment of type 2 diabetes mellitus. *Clin Diabetol*. 2009;10:101-128.
12. Gerstein HC, Santaguida P, Raina P, Morrison KM, Balion C, Hunt D, *et al*. Annual incidence and relative risk of diabetes in people with various categories of dysglycemia: a systematic overview and meta-analysis of prospective studies. *Diabetes Res Clin Pract*. 2007;78:305-312.
13. Vilanova MB, Falguera M, Marsal JR, Rubinat E, Alcubierre N, Castelblanco E, *et al*. Prevalence, clinical features and risk assessment of pre-diabetes in Spain: the prospective Mollerussa cohort study. *BMJ Open*. 2017;7:e015158.

14. Ezquerro-Lázaro I, Cea-Soriano L, Giráldez-García C, Ruiz A, Franch-Nadal J, Díez-Espino J, *et al.* Lifestyle factors do not explain the difference in diabetes progression according to type of prediabetes: results from a Spanish prospective cohort of prediabetic patients. *Diabetes Res Clin Pract.* 2019;153:66-75.
15. American Diabetes Association. Classification and diagnosis of diabetes: standards of medical care in diabetes-2019. *Diabetes Care.* 2019;42(Suppl 1):S13-S28.
16. Handelsman Y, Bloomgarden ZT, Grunberger G, Umpierrez G, Zimmerman RS, Bailey TS, *et al.* American Association of Clinical Endocrinologists and American College of Endocrinology-clinical practice guidelines for developing a diabetes mellitus comprehensive care plan 2015-executive summary. *Endocr Pract.* 2015;21(Suppl 1):1-87.
17. The Expert Committee on the Diagnosis and Classification of Diabetes Mellitus. Follow-up report on the diagnosis of diabetes mellitus. *Diabetes Care.* 2003;26:3160-3167.
18. American Diabetes Association. Diagnosis and classification of diabetes mellitus. *Diabetes Care.* 2014;37(Suppl 1):S81-S90.
19. Tietz NW, editor. *Clinical Guide to Laboratory Tests.* 4th ed. Philadelphia: WB Saunders; 2006. p. 444-451.
20. Davis JE, McDonald JM, Jarrett L. A high-performance liquid chromatography method for hemoglobin A1c. *Diabetes.* 1978;27:102-107.
21. American Diabetes Association. Classification and diagnosis of diabetes: standards of medical care in diabetes-2020. *Diabetes Care.* 2020;43(Suppl 1):S14-S31.
22. Ochocińska A, Śnitko R, Czekuć-Kryśkiewicz E, Kępka A, Szalecki M, Janas RM. Evaluation of the immunoradiometric and electrochemiluminescence method for the measurement of serum insulin in children. *J Immunoassay Immunochem.* 2016;37:243-250.
23. Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC. Homeostasis model assessment: insulin resistance and β -cell function from fasting plasma glucose and insulin concentrations in man. *Diabetologia.* 1985;28:412-419.
24. Song Y, Manson JE, Tinker L, Howard BV, Kuller LH, Nathan L, *et al.* Insulin sensitivity and insulin secretion determined by homeostasis model assessment (HOMA) and risk of diabetes in a multiethnic cohort of women: the Women's Health Initiative Observational Study. *Diabetes Care.* 2007;30:1747-1752.
25. Singh B, Saxena A. Surrogate markers of insulin resistance: a review. *World J Diabetes.* 2010;1:36-47.
26. Saeedi P, Petersohn I, Salpea P, Malanda B, Karuranga S, Unwin N, *et al.* Global and regional diabetes prevalence estimates for 2019 and projections for 2030 and 2045: results for the International Diabetes Federation Diabetes Atlas. *Diabetes Res Clin Pract.* 2019;157:107843.
27. Alemán-Vega G, Garrido-Elustondo S, del Cura-González I, Sarría-Santamera A. Is a maintained glycaemia between 110 and 125 mg/dL a risk factor in the development of diabetes? *Aten Primaria.* 2017;49:557.
28. Koo BK, Lee CH, Yang BR, Hwang SS, Choi NK. The incidence and prevalence of diabetes and related atherosclerotic complications in Korea: a national health insurance database study. *PLoS One.* 2014;9:e110650.
29. Ursini F, Russo E, D'Angelo S, Arturi F, Hribal ML, D'Antona L, *et al.* Prevalence of undiagnosed diabetes in rheumatoid arthritis: an OGTT study. *Medicine (Baltimore).* 2016;95:e2552.
30. LeRoith D. Pathophysiology of the metabolic syndrome: implications for the cardiometabolic risks associated with type 2 diabetes. *Am J Med Sci.* 2012;343:13-16.
31. Lin Y, Sun Z. Current views on type 2 diabetes. *J Endocrinol.* 2010;204:1.
32. NCD Risk Factor Collaboration (NCD-RisC). Worldwide trends in diabetes since 1980: a pooled analysis of 751 population-based studies with 4.4 million participants. *Lancet.* 2016;387:1513-1530.
33. Guariguata L, Whiting DR, Hambleton I, Beagley J, Linnenkamp U, Shaw JE. Global estimates of diabetes prevalence for 2013 and projections for 2035. *Diabetes Res Clin Pract.* 2014;103:137-149.
34. Ozougwu JC, Obimba KC, Belonwu CD, Unakalamba CB. The pathogenesis and pathophysiology of type 1 and type 2 diabetes mellitus. *J Physiol Pathophysiol.* 2013;4:46-57.
35. Mansour A, Al-Jazairi M. Predictors of incident diabetes mellitus in Basrah, Iraq. *Ann Nutr Metab.* 2007;51:277-280.
36. Mansour AA, Al-Maliky AA, Kasem B, Jabar A, Mosbeh KA. Prevalence of diagnosed and undiagnosed diabetes mellitus in adults aged 19 years and older in Basrah, Iraq. *Diabetes Metab Syndr Obes.* 2014;7:139-144.
37. Abusaib M, Ahmed M, Nwayyir HA, Alidrisi HA, Al-Abbood M, Al-Bayati A, *et al.* Iraqi experts consensus on the management of type 2 diabetes/prediabetes in adults. *Clin Med Insights Endocrinol Diabetes.* 2020;13:1-11.
38. Makrecka-Kuka M, Liepinsh E, Murray AJ, Lemieux H, Dambrova M, Tepp K, *et al.* Altered mitochondrial metabolism in the insulin-resistant heart. *Acta Physiol (Oxf).* 2020;228:e13430.
39. Li Y, Zhang L, Gu Y, Hao C, Zhu T. Insulin resistance as a predictor of cardiovascular disease in patients on peritoneal dialysis. *Perit Dial Int.* 2013;33:411-418.
40. Child A, Larkin EJ, Fontaine MJ. Co-encapsulation of ECM proteins to enhance pancreatic islet cell function. In: *Transplantation, Bioengineering, and Regeneration of the Endocrine Pancreas.* Elsevier; 2020;2:307-313.