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Pre-diabetes in Young Adults: Current Evidence and Future Risks

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Abstract
Pre-diabetes has emerged as a major public health concern among young adults worldwide. Traditionally considered a condition affecting middle-aged and older individuals, pre-diabetes is increasingly diagnosed in individuals aged 18–40 years. The rising prevalence is attributed to sedentary lifestyles, obesity, unhealthy dietary patterns, sleep disturbances, and genetic susceptibility. Pre-diabetes is characterized by impaired fasting glucose, impaired glucose tolerance, or elevated glycated hemoglobin levels below the threshold for diabetes mellitus. Growing evidence suggests that pre-diabetes is not a benign condition; rather, it is associated with increased risks of type 2 diabetes mellitus (T2DM), cardiovascular disease, chronic kidney disease, and early microvascular dysfunction. This review summarizes current evidence regarding the epidemiology, risk factors, pathophysiology, and future health implications of pre-diabetes in young adults. Early identification and timely intervention may significantly reduce long-term morbidity and healthcare burden.

Keywords: Pre-diabetes, Young Adults, Type 2 Diabetes Mellitus, Insulin Resistance, Cardiovascular Risk

Introduction

Pre-diabetes represents an intermediate metabolic state between normoglycemia and diabetes mellitus. In 2010, the American Diabetes Association (ADA) recommended the addition of Hemoglobin A1c (HbA1c) as a diagnostic test for diabetes, along with Oral Glucose Tolerance Test (OGTT)[1]. According to the ADA, the definition of pre-diabetes is based on any one of the three diagnostic criteria: Impaired fasting plasma glucose of 100–125 mg/dL, HbA1c of 5.7–6.4%, or impaired glucose tolerance following an oral glucose tolerance test[15]. Impaired Glucose Tolerance (IGT) is defined as two-hour glucose between 140–199 mg/dl during a 75-g oral glucose tolerance test (OGTT)[15]. Studies conducted on the prevalence of pre-diabetes in China, Malaysia, Sri Lanka, Australia and the US show a prevalence percent of 35.7% among 170,287 participants[2], 11.62% among 103,063 participants[3], 13.7% among

1,137,925 participants[4], 24.0% among 3180 young adults[5], respectively. The pooled prevalence of pre-diabetes has increased dramatically, from 7.64% to 14.27% globally during the past two decades. Increase in prevalence is markedly more among young adults, where a pooled prevalence of 8.84%[6] has been found. Recent epidemiological studies indicate that low and middle-income countries are experiencing a disproportionately high burden of pre-diabetes due to urbanization, obesity, and lifestyle changes.

TABLE 1. ***Taken from: Lawal Y, Bello F, Kaoje YS. Pre-diabetes Deserves More Attention[11]***

TABLE 1 Differing Diagnostic Criteria for Prediabetes and Diabetes

	FPG, mmol/L		2-Hour Post-Load Plasma Glucose, mmol/L		A1C, %	
Guidelines	IFG	Diabetes	IGT	Diabetes	Prediabetes	Diabetes
WHO	6.1-6.9	≥7.0	7.8-11.0	≥11.1	5.7-6.4	≥6.5
ADA	5.6-6.9	≥7.0	7.8-11.0	≥11.1	5.7-6.4	≥6.5
CPG	6.1-6.9	≥7.0	7.8-11.0	≥11.1	6.0-6.4	≥6.5

Pathophysiology

Pre-diabetes represents an early stage in the natural history of T2DM, due to the interaction between insulin resistance and progressive pancreatic β-cell dysfunction. The pathophysiology is complex, and is as follows:-

1. **Insulin Resistance:** The primary pathologic abnormality of pre-diabetes, it is a diminished response of insulin tissues such as skeletal muscle, liver and adipose tissue to circulating glucose. The condition is strongly associated with visceral adiposity, chronic low-grade inflammation and deranged adipokine secretion, leading to elevated circulating free fatty acids. Pancreatic β-cells respond to this condition by increasing insulin secretion, leading to hyperinsulinemia and approximately maintains near-normal plasma glucose concentrations[7].
2. **Progressive β-cell dysfunction:** Unresolved, prolonged insulin resistance leads to a loss in ability of pancreatic β-cells to compensate for the increased metabolic demands. β-cell dysfunction can be caused by a wide plethora of reasons, namely; glucotoxicity, lipotoxicity, oxidative stress, endoplasmic reticulum stress, mitochondrial dysfunction and genetic susceptibility. The decline in β-cell function ultimately results in sub-optimal insulin secretion and impaired glucose homeostasis[7].
3. **Impaired Glucose Homeostasis:** Pre-diabetes manifests clinically as impaired fasting glucose (IFG), impaired glucose tolerance (IGT), or both. IFG is due to hepatic insulin resistance, leading to increased hepatic gluconeogenesis and fasting hyperglycemia. Contrasting to IGF, IGT is primarily and predominantly attributable to reduced insulin mediated glucose uptake by skeletal muscle, resulting in elevated postpandrial blood glucose levels[7].
4. **Early Pancreatic and Vascular Changes:** Morphologic alterations in the pancreas such as – a reduction in β-cell mass and early deposition of islet amyloid in genetically susceptible individuals. Concurrently, endothelial dysfunction and low-grade vascular inflammation starts to develop, initiating atherosclerosis even before overt diabetes is diagnosed[7].
5. **Clinical Progression:** If appropriate lifestyle modification and pharmacological intervention is not done, persistent insulin resistance and progressive β-cell failure can cause worsening dysglycemia

and eventually progresses to type two diabetes mellitus. Increased cardiovascular disease, hypertension, dyslipidemia, and other metabolic syndromes[7].

To summarize, the development of pre-diabetes involves insulin resistance, pancreatic β-cell dysfunction, chronic inflammation, and altered adipokine signaling.

Excess visceral adiposity contributes to impaired glucose uptake and increased hepatic glucose production. In genetically susceptible individuals, progressive β-cell exhaustion leads to worsening glycemic control and eventual progression to T2DM.

Young adults with obesity often demonstrate early metabolic abnormalities that may remain clinically silent for years.

Major Risk Factors

Identification of modifiable and non-modifiable risk factors of pre-diabetes is essential for early diagnosis and timely preventive strategies.[10 15 16 18 19]

1. **Obesity, particularly central or visceral obesity:** It is the most important and relatively easily modifiable risk factor for pre-diabetes. Excess adipose tissue causes insulin resistance through increased secretion of pro-inflammatory cytokines, free fatty acids, and adipokines, resulting in impaired insulin signaling in skeletal muscle, liver, and adipose tissue. Visceral adiposity also contributes to chronic low-grade inflammation, oxidative stress, and endothelial dysfunction, all of which accelerate glucose intolerance. Epidemiological studies consistently show that overweight and obese individuals have a significantly greater risk of developing pre-diabetes than those with normal body mass index. Even modest reductions in body weight improves insulin sensitivity and delays disease progression, focusing obesity as the principal target for preventive interventions.[10 15 16 18 19]
2. **Physical inactivity:** another major determinant of pre-diabetes, sedentary lifestyles reduce skeletal muscle glucose uptake, impair insulin-mediated glucose disposal and promote weight gain. Regular physical activity enhances insulin sensitivity, improves mitochondrial function, and facilitates glucose utilization. Evidence from research studies indicate that lifestyle modification programs having at least 150 minutes of moderate to intense physical activity per week significantly reduce the incidence of T2DM among individuals with pre-diabetes. Hence, routine exercise is a boon and a cost-effective preventive strategy.[10 15 16 18]
3. **Dietary habits**: strongly influence the development of pre-diabetes. Imbalanced diets rich in refined carbohydrates, sugary beverages, saturated trans fats and processed foods contribute to obesity, insulin resistance and chronic inflammation. In contrast, dietary patterns having more of whole grains, fruits, vegetables, legumes, nuts and unsaturated fats are proven to improve glycemic control and reduced metabolic risk. Excessive caloric intake increases abdominal and peripheral adiposity and β-cell stress, resulting in rapid progression toward overt diabetes. Hence, nutritional modification remains a cornerstone of both primary prevention and management of pre-diabetes.[10 15 16]
4. **Increasing age:** a well-recognized non-modifiable risk factor for pre-diabetes. Aging causes progressive reductions in insulin sensitivity, decline in pancreatic β-cell function, changes in body composition, and diminished physical activity. Current clinical recommendations advocate routine screening for adults aged 45 years and older because prevalence rises substantially with advancing age. Recent evidence indicates an alarming increase in pre-diabetes among younger adults largely attributable to rising obesity rates, sedentary lifestyles, and unhealthy dietary practices.[15 16]

5. **Family history:** along with genetic predisposition, it significantly influences susceptibility to pre-diabetes. Pre-diabetic young adults with a first-degree relative affected by T2DM have an increased risk of developing insulin resistance and impaired β-cell function. The inherited risk interacts with environmental exposures such as obesity, poor nutrition, and physical inactivity to lead to the final disease outcome. It is very important for individuals with a family history of diabetes to undergo regular metabolic screening and lifestyle counseling.[12]
6. **Metabolic disorders:** frequently coexist with pre-diabetes silently and further increase the disease risk. Hypertension, dyslipidemia, metabolic syndrome, and metabolic dysfunction-associated steatotic liver disease (MASLD) are common in pre-diabetic and T2DM patients. Studies have shown that pre-diabetes itself is a risk factor for early vascular abnormalities and increased cardiovascular risk even before progression to diabetes. Therefore, cardiovascular health screening remains crucial for pre-diabetics.[13-20]
7. **Women with a history of gestational diabetes mellitus (GDM):** another high-risk group, pregnancy-induced insulin resistance may persist after delivery, predisposing affected women to persistent glucose intolerance and eventual T2DM. Clinical guidelines hence recommend lifelong metabolic screening in women with a history of GDM. Additionally, certain ethnicities—including South Asians, African Americans, Hispanic Americans, and Native Americans have disproportionately higher rates of pre-diabetes and T2DM due to a combined effect of genetic susceptibility, body fat distribution, socioeconomic factors and lifestyle factors. Targeted screening and preventive interventions are therefore crucial in such women. [15]

The global burden of pre-diabetes continues to rise at an alarming rate. Current estimates indicate that nearly 720 million adults worldwide were living with pre-diabetes in 2021, and this number may exceed one billion by 2045 if effective preventive measures are not implemented. Also, individuals with pre-diabetes progress to T2DM at an estimated annual rate of approximately 5-10%, although progression varies according to individual risk factors and lifestyle characteristics. Unarguably, numerous clinical trials have demonstrated that intensive lifestyle modification, including dietary improvement, weight reduction, and regular physical activity can substantially reduce this progression, showcasing the reversible nature of pre-diabetes when detected early.[10-19]

To summarize, pre-diabetes develops through the interaction of multiple modifiable and non-modifiable risk factors, with obesity, physical inactivity, unhealthy dietary habits, advancing age, family history, hypertension, dyslipidemia, MASLD, previous gestational diabetes, and genetic susceptibility representing the principal determinants. Early identification of high-risk individuals, coupled with evidence-based lifestyle interventions and regular metabolic monitoring, offers an effective strategy for preventing progression to T2DM and reducing the long-term burden of cardiovascular and metabolic complications.

TABLE 2. A summary of key risk factors associated with pre-diabetes in young adults.

Risk Factor	***Mechanism***
Obesity (more specifically, abdominal obesity)	Increased insulin resistance
Physical inactivity	Reduced glucose utilization
Family history of diabetes	Genetic susceptibility
Hypertension	Shared metabolic pathways

Smoking	Oxidative stress and inflammation
Poor sleep quality	Hormonal dysregulation
Processed-food consumption	Excess caloric intake

Systematic reviews have consistently identified obesity, hypertension, physical inactivity, and family history as the strongest predictors of pre-diabetes.

Clinical Consequences of Pre-Diabetes

Pre-diabetes for long has traditionally been viewed as an intermediate metabolic state preceding T2DM. However, new researches and mounting evidences indicate that it is very much a clinically significant disorder, associated with structural, functional and biochemical abnormalities whose onset starts well before the diagnostic threshold for diabetes is even reached. Rathere than merely being a risk category, pre-diabetes is being increasingly recognized globally as a disease continuum characterized by- Insulin resistance, pancreatic β-cell dysfunction, endothelial insult and injury, chronic inflammation and metabolic dysregulation. These result in early organ damage and long term morbidity. Such alterations show a multi system affect, substantially increasing the risk of cardiovascular diseases, microvascular complications, chronic kidney disease, hepatic dysfunction and progression to overt diabetes[8-15].

1. Early Endocrine Dysfunction

One of the earliest clinical consequences of pre-diabetes is sub-optimal pancreatic β-cell function. Individuals genetically predisposed to diabetes show a significantly diminished first-phase insulin response even though their had normal glucose tolerance. Notably, pre-diabetic offspring of diabetic parents had significantly reduced insulin secretion during the first 10 minutes following intravenous glucose administration, while maintaining normal glucose tolerance tests[9]. This defect was a precedent to measurable hyperglycaemia, highlighting that β-cell dysfunction as one of the earliest detectable abnormalities in pre-diabetes. Many subjects also demonstrated delayed insulin peaks and exaggerated growth hormone responses, suggesting that endocrine dysregulation begins years before clinical diabetes develops[15].

2. Cardiovascular Consequences

Cardiovascular disease (CVD) by far presents as the most clinically important complication associated with pre-diabetes. Even mild elevations in fasting plasma glucose(FPG), through oxidative stress, initiate endothelial dysfunction. Advanced glycation end-product formation, decreased nitric oxide bioavailability, increased vascular inflammation, and activation of pro-thrombotic pathways therefore lead to vascular injury long before diabetes is diagnosed[9 10].

Large epidemiological studies show that individuals with pre-diabetes have significantly higher risks(6-101%) of developing coronary artery disease, myocardial infarction, stroke, heart failure, atrial fibrillation, and cardiovascular mortality compared with normoglycaemic individuals[13 14]. The HOPE study showed that risk of CVD increases by almost 9% for every 1mmol/L of FPG glucose increase[15].

3. Microvascular Dysfunction

Endothelial dysfunction is defined as the loss of vasodilatory, anti-coagulant and anti-inflammatory properties of endothelium and upregulation of pathways that promote vasoconstriction, thrombosis and inflammation in the arterial wall, due to decreased NO availability.

Contrary to earlier beliefs, microvascular injury begins during pre-diabetes rather than after the onset of overt diabetes. Persistent mild hyperglycaemia induces endothelial dysfunction by increasing the expression of several microRNAs, namely; mi,R-320, miR-221, miR-503, miR-125. Result of this induction is capillary basement membrane thickening, impaired vasodilatation, oxidative stress, and extracellular matrix remodelling. These abnormalities impair tissue perfusion and reduce insulin delivery to skeletal muscle, leading to worsening insulin resistance and promoting progression to diabetes[10 15].

Interestingly, even early pathological studies identified structural vascular abnormalities in genetically predisposed individuals before abnormalities of glucose tolerance became apparent. Research studies have highlighted evidence demonstrating increased thickness of glomerular and skeletal muscle capillary basement membranes during the pre-diabetic state, supporting the concept that microvascular disease precedes overt diabetes.

These findings have since been corroborated by modern imaging studies demonstrating retinal, renal, cutaneous, and skeletal muscle microvascular dysfunction in individuals with pre-diabetes. Such alterations are now considered important biomarkers of future cardiovascular disease and diabetic complications[10 15].

4. Renal Consequences

The kidney is one of the earliest organs affected by pre-diabetes. Endothelial dysfunction, glomerular hyperfiltration, oxidative stress, and chronic inflammation can cause progressive renal injury before diabetes onset. Individuals with pre-diabetes have increased prevalence of microalbuminuria, deranged glomerular filtration, and chronic kidney disease compared with normoglycaemic populations[16].

A simple way of assessment of renal microvasculature is to measure urine albumin excretion. Albuminuria is the phenomenon in which urine albumin-to-creatinine ratio (ACR) is >= 30mg/g. Although ADA does not recommend the use of terms such as Microalbuminuria (MAU) and Macroalbuminuria, some studies still apply these terms. Microalbuminuria is ACR>=30-299mg/g and Macroalbuminuria is ACR >300mg/g. MAU is an established risk factor. Patients with MAU have a high risk of acute coronary heart disease, stroke and peripheral vascular disease[16].

According to the AusDiab study of 10,000 paricipants, MAU prevalence for IGT is 9.9%, in IFG 8.3% and more than double in diabetic patients. About 30% of the participants who were newly diagnosed with diabetes had some degree of kidney damage. It highlighted a very important fact that hyperglycemia can cause kidney damage even before glucose levels reach diabetic thresholds[16.]

Research has consistently shown that pre-diabetes was linked to an increased risk of renal injury[16].

Hence, recognition of the early renal abnormalities is clinically significant because intervention may prevent irreversible nephron loss and delay progression toward diabetic nephropathy.

5. Neurological Consequences

Peripheral neuropathy is major future outcome of pre-diabetes. Small-fibre neuropathy may develop even in the absence of overt diabetes due to chronic metabolic stress, oxidative injury, mitochondrial dysfunction and impaired microvascular perfusion of peripheral nerves. Patients frequently report numbness, burning sensations, paraesthesia, or reduced vibration perception before meeting diagnostic criteria for diabetes[17].

Diabetic Retinopathy (DR) is the leading cause of blindness in inworking-age populations with diabetes. Retinopathy in pre-diabetic patients is uncommon, with venular dilatation being the main finding. Some recent studies analysed retinal vascular changes using optical coherence tomography angiography (OCTA) in pre-diabetic individuals. Findings of OCTA were- the size of focal avascular zone and the macular vessel diameter, were larger in the pre-diabetic patients. The vessel area density in superficial macular area decreased in pre-diabetes. The central foveal thickness was significantly decreased in pre-diabetes. The perfusion density and vascular length density decreased in pre-diabetics. On the other hand, some studies identified some neuroretinal changes regarding the thickness of macula and the peripapillary retinal nerve fiber layer using both OCT and fundus fluorescein angiography in pre-diabetics[16 17].

Acquired color vision impairment, signs of retinopathy, cataracts, and corneal surface disorders were ophthalmological complications also reported among people with prediabetes[11].

Additionally, pre-diabetes has also been associated with cerebral microvascular dysfunction and an increased long-term risk of vascular dementia and cognitive decline. Chronic inflammation and endothelial dysfunction impair cerebral blood flow, contributing to subtle neurological deficits that may precede overt diabetes by several years.

6. Hepatic and Metabolic Consequences

Pre-diabetes is closely associated with widespread metabolic abnormalities beyond glucose dysregulation. Insulin resistance promotes increased hepatic glucose production, enhanced lipolysis, elevated circulating free fatty acids, hypertriglyceridaemia, reduced HDL cholesterol, and formation of small dense LDL particles. These metabolic disturbances predispose individuals to metabolic dysfunction-associated steatotic liver disease (MASLD), formerly known as non-alcoholic fatty liver disease. MASLD is a spectrum, ranging from fatty liver to nonalcoholic steatohepatitis (NASH) to liver cirrhosis[11].

Altered intestinal microbiome is commonly seen in pre-diabetes. Studies have demonstrated intestinal dysbiosis, with marked depletion of the genus Clostridium and bacterium Akkermansia muciniphila. The condition was found similar to Inflammatory Bowel Disease (IBD)[11].

Persistent ectopic lipid accumulation further aggravates insulin resistance, establishing a vicious cycle that accelerates progression toward overt diabetes and cardiovascular disease.

7. Progression to Type 2 Diabetes Mellitus

Perhaps the most significant clinical consequence of pre-diabetes is its progression to T2DM. Although regression to normoglycaemia is possible, especially following intensive lifestyle modification, a substantial proportion of individuals progress to diabetes over time. The risk depends upon the diagnostic definition employed and the presence of additional risk factors such as obesity, family history, sedentary lifestyle, and metabolic syndrome. Epidemiological studies estimate annual progression rates of approximately 5–10%, with considerably higher risks among individuals exhibiting both impaired fasting glucose and impaired glucose tolerance[8 15].

In line with the study done by of Boden et al. suggest that endocrine abnormalities may be detectable years before alterations in glucose homeostasis become clinically evident, emphasizing that the pathological process underlying diabetes begins well before conventional diagnostic criteria are fulfilled. Measurement of early insulin responses may therefore identify high-risk individuals even when glucose tolerance remains normal[9].

8. Summary of the Clinical Implications

Collectively, current evidence demonstrates that pre-diabetes is a systemic disorder rather than a benign metabolic intermediate. Cardiovascular injury, endothelial dysfunction, renal impairment, neuropathy, hepatic steatosis, and progressive β-cell failure begin during the pre-diabetic stage, often before overt hyperglycaemia becomes apparent. Early identification through appropriate screening, combined with intensive lifestyle modification and selective pharmacological therapy, provides a critical opportunity to prevent irreversible organ damage and substantially reduce future diabetes-related morbidity and mortality.

Evidence also suggests increased risks of:[20 21 22 23]

- Coronary artery disease
- Stroke
- Heart failure
- Chronic kidney disease
- Cognitive decline
- Early endothelial dysfunction [20-23]

An umbrella review of prospective studies demonstrated that individuals with pre-diabetes have significantly higher risks of cardiovascular outcomes and all-cause mortality compared with normoglycemic populations.

TABLE 3. Future Health Risks Associated with Pre diabetes

Outcome	***Relative Risk Trend***
Type 2 Diabetes Mellitus	Very High
Cardiovascular Disease	High
Chronic Kidney Disease	Moderate to High
Dementia	Moderate
All-Cause Mortality	Increased
Microvascular Dysfunction	Increased

Pre-diabetes Progression Pathway

Pre-diabetes develops through a progressive interplay between insulin resistance and pancreatic β-cell dysfunction.

Initial phase: Insulin-sensitive tissues, especially skeletal muscle, adipose tissue, and the liver, become less responsive to insulin because of obesity, physical inactivity, genetic susceptibility and chronic inflammation. Pancreatic β-cells increase insulin secretion as a compensation, mounting to compensatory hyperinsulinemia that initially maintains near-normal blood glucose concentrations.[15 18 19]

Persisting insulin resistance leads to β-cells metabolic stress caused by glucotoxicity, lipotoxicity, oxidative stress, and inflammatory mediators. Over time, β-cell function gradually declines, decreasing insulin secretion relative to the body's metabolic demands. This transition results in impaired fasting glucose (IFG), impaired glucose tolerance (IGT), or both, which constitute the diagnostic states of pre-diabetes. Individuals may remain asymptomatic during this stage.[18 19]

Without adequate timely intervention, persistent hyperglycemia further accelerates β-cell deterioration, ultimately culminating in T2DM. Around 5–10% of individuals with pre-diabetes progress to diabetes annually and this progression is not inevitable. As a result, early identification of pre-diabetes is crucial prevention of disease progression before irreversible β-cell failure and chronic diabetic complications develop. [15 16 17 18 19]

In a short summarized flowchart: Young adults with pre diabetes may remain asymptomatic for years while metabolic damage progresses. The typical progression pathway is illustrated below.

Normoglycemia → Insulin Resistance → Pre diabetes → Type 2 Diabetes → Microvascular and Macro-vascular Complications

Prevention and Management

Lifestyle modification remains the cornerstone of management. Weight reduction, regular physical activity, dietary improvement, smoking cessation, and adequate sleep have demonstrated effectiveness in reducing progression to diabetes. Emerging technologies, including continuous glucose monitoring and artificial intelligence-assisted risk prediction models, may facilitate earlier detection and personalized interventions in high-risk individuals.

Discussion

The growing prevalence of pre diabetes among young adults presents a substantial challenge to healthcare systems. Early metabolic abnormalities may result in decades of cumulative vascular injury before clinical diabetes develop. Current evidence indicates that pre diabetes should be regarded as a disease continuum rather than a benign risk state. Population-based screening programs, targeted lifestyle interventions, and improved awareness among healthcare professionals are essential for reducing future disease burden. Furthermore, longitudinal studies focusing specifically on young adults are needed to clarify long-term outcomes and identify optimal prevention strategies.[20] [21]

Conclusion

Pre diabetes in young adults is an increasingly prevalent condition with significant long-term health implications. It is strongly associated with obesity, sedentary behavior, and other modifiable lifestyle factors. Evidence indicates increased risks of diabetes, cardiovascular disease, chronic kidney disease, and mortality. Early identification and comprehensive lifestyle interventions remain the most effective strategies for preventing disease progression and reducing future healthcare burden.

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