P-ISSN: 2664-8822
E-ISSN: 2664-8830
Impact Factor: RJIF 5.44
IJDR 2025; 7(1): 45-53
www.diabetesjournal.in
Received: 09-08-2025
Accepted: 22-09-2025

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Comprehensive review on the role of Dietary Acid Load in health and its effect on different types of Diabetes

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DOI: https://www.doi.org/10.33545/26648822.2025.v7.i1a.26

Abstract
Dietary acid load (DAL), primarily influenced by the intake of acid and base forming foods, has emerged as a significant dietary factor impacting metabolic health. This review provides a comprehensive analysis of the role of DAL in overall health, with a particular focus on its relationship with different types of diabetes, including type 1 diabetes (T1D), type 2 diabetes (T2D), and gestational diabetes mellitus (GDM). In this review we explore the physiological mechanisms through which a high acid-forming diet may contribute to insulin resistance, impaired β-cell function, systemic inflammation, and altered renal function key factors in the pathogenesis of diabetes. We also examine the associations reported in epidemiological and clinical studies, highlighting inconsistencies, potential confounders, and the need for standardized assessment methods such as net endogenous acid production (NEAP) and potential renal acid load (PRAL). Additionally, we discuss the implications of dietary patterns, including western diets and plant-based diets, on DAL and metabolic outcomes. Understanding the impact of DAL offers a promising avenue for dietary interventions aimed at diabetes prevention and management. Future research directions are proposed to further elucidate causal relationships and guide nutritional recommendations.

Keywords: Dietary acid load, types of diabetes, net endogenous acid production, potential renal acid load

Introduction
Maintaining acid-base balance is critical for optimal physiological function, as even minor disruptions in systemic pH can influence metabolic processes, enzymatic activity, and overall cellular homeostasis. An increased dietary acid load (DAL), reflecting a shift toward a net acid-producing dietary pattern, has been implicated in the development of numerous chronic conditions, including cardiometabolic diseases, obesity, metabolic dysfunction-associated steatotic liver disease (MASLD), chronic kidney disease (CKD), endothelial dysfunction, and hypertension. The body's ability to regulate acid-base homeostasis is influenced by both exogenous factors, such as dietary intake, and endogenous buffering and excretion mechanisms, which tend to decline with age and deteriorating renal function [1, 2].

Dietary patterns play a pivotal role in determining net acid or alkali production. Diets high in animal proteins, refined grains, and processed foods characteristic of the western dietary pattern tend to increase net acid load due to their higher content of sulfur-containing amino acids and phosphorus. In contrast, plant-based diets rich in fruits, vegetables, legumes, and whole grains typically result in a lower acid load or even a n*et al*kaline effect, largely due to their high content of potassium, magnesium, and bicarbonate precursors [3, 4].

Diabetes mellitus encompasses a heterogeneous group of metabolic disorders characterized by chronic hyperglycemia resulting from impaired insulin secretion, insulin resistance, or a combination of both. The disease is broadly classified into type 1 diabetes (T1D), type 2 diabetes (T2D), and gestational diabetes mellitus (GDM), each with distinct etiologies and pathophysiological mechanisms [5]. T1D is primarily an autoimmune condition leading to the destruction of pancreatic β-cells, while T2D arises from a combination of insulin resistance and relative insulin deficiency, often associated with obesity, sedentary lifestyle, and genetic

predisposition. GDM develops during pregnancy and poses risks to both maternal and fetal health.

The long-term complications of diabetes ranging from cardiovascular disease and neuropathy to nephropathy and retinopathy are well-documented and significantly impact morbidity and mortality. Emerging evidence suggests that DAL may influence insulin sensitivity, glucose metabolism, and β-cell function, thereby contributing to the onset and progression of diabetes, particularly T2D. The underlying mechanisms are thought to involve low-grade metabolic acidosis, inflammation, altered mineral metabolism, and hormonal imbalances [6, 20].

Although the association between DAL and T2D has been more extensively studied, its potential role in T1D and GDM remains less clear and warrants further investigation. Given the modifiable nature of dietary acid load through nutritional interventions, understanding its role in diabetes pathogenesis presents a valuable opportunity for preventative and therapeutic strategies. This review aims to synthesize current evidence on the health impacts of dietary acid load, with a specific focus on its influence across different types of diabetes. By integrating findings from clinical, epidemiological, and mechanistic studies, we seek to highlight the potential of DAL as a target for nutritional interventions in diabetes prevention and management [7].

Types of Diabetes

Diabetes mellitus is a complex metabolic disorder characterized by chronic hyperglycemia resulting from defects in insulin secretion, insulin action, or both. It is broadly categorized into type 1 diabetes (T1D), type 2 diabetes (T2D), and gestational diabetes mellitus (GDM), each with distinct pathophysiological mechanisms and risk profiles. Types of diabetes, causes, symptoms, suitable remedies are tabulated in (Table 1)**.** Emerging evidence suggests that dietary factors, including dietary acid load (DAL), may modulate risk and progression across these diabetes types by influencing insulin sensitivity, acid-base balance, and systemic inflammation [7, 22]. Understanding the distinct yet interconnected pathways in each form of diabetes is crucial for tailoring dietary interventions.

Table 1: Different types of diabetes, route causes, symptoms and remedies

Type of Diabetes	Causes	Symptoms	Remedies / Management
Type 1 Diabetes	Autoimmune destruction of pancreatic beta cells. Often genetic.	- Excessive thirst & urination- Weight loss- Fatigue- Blurred vision	- Insulin therapy- Carb counting- Regular exercise- Blood glucose monitoring
Type 2 Diabetes	Insulin resistance & impaired insulin production; often due to lifestyle factors and genetics.	- Fatigue- Frequent urination- Increased hunger- Slow wound healing	- Diet & exercise- Oral meds (e.g., Metformin)- Insulin (if needed)- Weight loss
Gestational Diabetes	Hormonal changes during pregnancy causing insulin resistance.	- Usually asymptomatic- Sometimes increased thirst & fatigue	- Healthy eating- Monitoring blood sugar- Exercise- Insulin if necessary- Usually resolves after childbirth
Type 3c Diabetes (Pancreatogenic Diabetes)	Damage to the pancreas due to pancreatitis, pancreatic surgery, or cancer.	- Symptoms similar to Type 1 or 2- Often digestive issues	- Insulin- Pancreatic enzyme replacement- Low-fat diet- Blood sugar monitoring
LADA (Latent Autoimmune Diabetes in Adults)	Slow-onset autoimmune diabetes in adults; misdiagnosed as Type 2.	- Gradual symptoms- Similar to Type 2 at first- Weight loss over time	- Starts with oral meds- Progresses to insulin- Antibody testing for diagnosis
MODY (Maturity-Onset Diabetes of the Young)	Single gene mutations affecting insulin production; hereditary.	- Mild hyperglycaemia- Often no symptoms- Diagnosed in teens/young adults	- Diet- Some subtypes need sulfonylureas- Genetic testing helpful- Rarely needs insulin
Neonatal Diabetes	Genetic defect in insulin production; appears in first 6 months of life.	- Dehydration- Weight loss- High blood sugar in infants	- Insulin or sulfonylureas depending on genetic cause- Genetic testing required
Brittle Diabetes	Unstable and hard-to-control blood glucose, often in Type 1 patients.	- Extreme blood sugar fluctuations- Frequent hospitalization	- Intensive insulin therapy- Continuous glucose monitoring (CGM)- Psychological support
Steroid-Induced Diabetes	Prolonged use of corticosteroids (e.g., for asthma, arthritis) that raise blood sugar levels.	- Same as Type 2 symptoms- High blood sugar during steroid use	- Adjust or stop steroids if possible- Blood sugar control with meds or insulin- Diet and exercise
Cystic Fibrosis-Related Diabetes (CFRD)	Pancreatic damage from cystic fibrosis leads to impaired insulin production.	- Combination of Type 1 and 2 symptoms- Unexplained weight loss- Decline in lung function	- Insulin therapy- High-calorie, high-protein diet- Pancreatic enzyme supplements- Regular glucose monitoring
Wolfram Syndrome (DIDMOAD)	Rare genetic disorder causing diabetes, vision loss, and neurological issues.	- Diabetes in childhood- Vision/hearing loss- Urinary problems- Neurodegeneration	- Insulin for diabetes- Supportive care for neurological and sensory symptoms- Genetic counselling

Effect of Dal on Diabetes

Effect of high and low levels of DAL on different types of diabetes are provided in (Table 1). A high Dietary Acid Load (DAL) has been linked to an increased risk of developing Type 2 Diabetes (T2DM) and insulin resistance. This association is likely due to the connection between high DAL and chronic low-grade metabolic acidosis, which can interfere with normal metabolic processes. Foods that contribute to a high DAL include meat, cheese, and grains, whereas fruits and vegetables have an alkaline (base-producing) effect that helps reduce acid load. Although more population-based studies are needed to establish a causal relationship, current research suggests that lowering dietary acid load may be a useful strategy for preventing and managing diabetes and its complications.

Table 2: Effect of high and low DAL on different types of diabetes

Type of Diabetes	Effect of High DAL	Effect of Low DAL / Alkaline Diet
Type 1 Diabetes	- High DAL may worsen insulin sensitivity- May increase inflammation and oxidative stress	- May reduce inflammation- Supports kidney health- Promotes better blood glucose control
Type 2 Diabetes	- Strong link to insulin resistance- Higher DAL is associated with increased risk of Type 2 diabetes- Can worsen metabolic syndrome	- Improves insulin sensitivity- Helps lower HbA1c- Supports weight loss and better metabolic function
Gestational Diabetes	- High DAL may increase gestational glucose intolerance- May affect fetal development indirectly	- May support better glucose control during pregnancy- Could reduce pregnancy complications
Type 3c Diabetes	- Pancreas already damaged — high DAL can stress kidneys and metabolism further	- Alkaline diet may ease burden on kidneys and pancreas- Promotes digestive health
LADA	- High DAL may accelerate autoimmune activity and beta-cell loss	- Anti-inflammatory effect of alkaline foods may slow progression
MODY	- Not directly linked, but high DAL may impair metabolic health further	- Balanced, alkaline diet supports overall glycaemic control
Neonatal Diabetes	- Limited data- Infant nutrition focused more on genetic management	- Diet is managed carefully with medical input; DAL not a primary factor
Brittle Diabetes	- High DAL can worsen blood sugar fluctuations due to poor metabolic buffering	- Alkaline foods help stabilize metabolism and reduce swings
Steroid-Induced Diabetes	- Steroids already cause insulin resistance; high DAL adds inflammatory load	- Alkaline diet may mitigate some inflammatory and metabolic stress
Cystic Fibrosis Diabetes	- Added metabolic stress from high DAL can worsen nutritional and glucose balance	- Nutrient-dense, alkaline foods help reduce oxidative stress and inflammation
Wolfram Syndrome	- High DAL can exacerbate neurological symptoms via metabolic acidosis	- Alkaline foods may support neuroprotection and reduce oxidative damage

The higher acid-forming potential of a diet, could be associated with diabetes. No study has been done to investigate the association between DAL and polyneuropathy in patients with diabetes. This study aimed to examine the linkage between the DAL and Diabetic Sensory-motor Polyneuropathy (DSPN) in a case-control study. Diabetes mellitus (DM) and cardiovascular disease (CVD) are among the biggest causes of death and health expenses worldwide. A higher dietary acid load (DAL) is associated with chronic low-grade metabolic acidosis, and may increase the risk of insulin resistance (IR), DM, hypertension, and CVD mortality. However, the association between DAL and IR still lacks population-based studies to confirm laboratory findings.

Measurement of Dal

Dietary acid load is a measure of the acid load derived from diet, considering both the potential renal acid load (PRAL) from food components like protein, potassium, phosphorus, calcium, and magnesium, and the organic acids from foods, which are metabolized to bicarbonate and thus have an alkalinizing effect. Current western diets are characterized by a high DAL, due to large amounts of animal protein and processed foods. Human acid-base equilibrium, largely determined by the concentration of hydrogen ions in blood plasma, is a tightly regulated physiological system that aims to maintain blood pH between 7.35 and 7.45. Human acid-base balance requires that net endogenous acid production (NEAP) equates to net acid excretion (NAE). The kidneys and lungs are the primary organs maintaining acid-base balance; the lungs excrete volatile carbon dioxide, while the kidneys excrete non-volatile acids generated by metabolic processes. The amount of non-volatile acid produced by human metabolism is described as endogenous acid production (EAP). The kidneys also reabsorb filtered bicarbonate to buffer endogenous acid and maintain acid-base balance. DAL primarily determines NEAP, stemming from the production of hydrogen ions upon the intake of protein-rich foods, especially animal proteins that are abundant in phosphorus and sulphur. In comparison, fruits and vegetables are high in citrate, which metabolizes to bicarbonate [8].

Dietary acid load can be assessed using the Net Endogenous Acid Production or the PRAL. To accurately measure NEAP, renal net acid excretion is measured using biochemical analysis of urine. PRAL is calculated based on dietary intake. DAL is a measure of how the total acid-producing versus alkaline-producing compounds in your diet affect the body's acid-base balance. Dietary acid loads of commonly used food groups, patterns (proteins, phosphorus, Potassium, Magnesium and Calcium) based on PRAL estimation i.e. Estimated PRAL in 100 g of food presented in (Table 3). Chronic high DAL from a typical western diet can lead to a low-grade metabolic acidosis associated with negative health outcomes. DAL is most often estimated using the PRAL score, which measures the potential of foods to produce acid or alkali [9, 10]. The score is based on the amounts of key nutrients in your diet.

Acid-forming components: High intake of protein and phosphorus contributes to a positive, or acidic, score. Foods like meat, eggs, and most grains are considered acid-forming.

Alkaline-forming components: A high intake of potassium, magnesium, and calcium results in a negative, or alkaline, score. Fruits, vegetables, and legumes are known to have an alkalizing effect.

Dietary acid load: Dietary acid load was estimated using three indicators. The net endogenous acid production (NEAP), the potential renal acid load (PRAL), and dietary acid load (DAL). The following formulas were used

- NEAP (mEq/day) = 54.5 × [protein intake (g/d) /K intake (*mEq*/d)] − 10.2,
- PRAL (mEq/day) = 0.49 × protein (g/day) + 0.037 × phosphorus (mg/day) − 0.021 × potassium (mg/day) − 0.026 × magnesium (mg/day) − 0.013 × calcium (mg/day)
- DAL (mEq/day) = PRAL + (body surface area [m^2] × 41[mEq/day]/1.73 m^2),
- Body surface area was calculated as: 0.024265 × Height $(cm)^{0.3964}$ × Weight (kg).

Table 3: DAL of commonly used food groups and patterns based on PRAL; Estimated PRAL in 100 g of food

Food	Protein	Phosphorus	Potassium	Magnesium	Calcium	DAL	PRAL/100g
Drinks							
Lemon soda	0.05	0	1	1	2	N	0
Coffee ground	0.3	3	50	4	2	B	−0.9
Infusion of fruits	0	0	167	0	0	B	−3.5
Green tea	0	0	15	1	0	B	−0.3
Mineral water	0	0	2	11	33	B	−0.8
Cola Cola	0.00	9	5	0	1	A	0.2
Beer	0.46	14	27	6	4	A	0
Orange juice	0.7	17	200	11	11	B	−3.7
Red Wine	0.07	23	127	12	8	B	−2.2
White wine	0.07	18	71	10	9	B	−1.2
Oil and fatty substances							
Butter	3.21	85	135	10	115	A	0.1
Margarine	0.23	7	51	1	6	B	−0.8
Olive oil	0	0	1	0	1	N	0
Sunflower seed oil	0	0	0	0	0	N	0
Oil seeds or oil-bearing plants							
Hazelnuts	14.95	290	680	163	114	B	−1.9
Peanuts	25.8	376	705	168	92	A	6.2
Almonds	20.96	471	713	279	268	A	2
Nuts	24.06	513	523	201	61	A	13.8
Animal originated Products							
Mussels	23.8	285	268	37	33	A	15.2
Salmon	26.48	305	436	36	11	A	14
Sardines in oil	24.62	490	397	39	382	A	15.9
Prawns	23.98	237	259	39	70	A	13.2
Beef	25.25	179	271	19	23	A	12.5
Chicken	29.8	214	245	27	14	A	16.5
Pork sausage	15.27	130	283	15	12	A	5.8
Pork Meat	26.83	263	352	22	14	A	14.7
Whole egg	13.61	215	152	13	162	A	9
Egg white	10.9	15	163	11	7	A	2.1
Yolk	15.86	390	109	5	129	A	18.1
Cereals and legumes							
White rice	2.69	43	35	12	10	A	1.7
White spaghetti	0.66	14	117	11	21	B	−2.2
Potatoes	2.1	75	544	27	10	B	−8.5
Corn tortilla	6.6	232.5	152.4	66	161.7	A	4.8
Flour tortilla	9.66	210	100	21	205	A	7.2
Beans	9.31	100	646	54	52	B	−7.4
Lentils	9.02	180	369	36	19	A	2.1
Chickpea	8.86	168	291	48	49	A	2.6
Soy	12.35	158	539	60	145	B	−2.9
Bean	7.6	125	268	43	36	A	1.1
Dairy products							
Fresh cheese	18.09	385	129	24	566	A	12.4
Cream	2.44	76	125	10	101	B	−0.2
Gouda	24.94	546	121	29	700	A	20
Parmesan	28.42	627	180	34	853	A	21.4
Whole milk	3.15	84	132	10	113	A	0.2
Skim milk	3.37	101	156	11	122	A	0.2
Greek Yogurt	10.19	135	141	11	110	A	5.3
Vegetables and Fruits							
Broccoli	2.38	67	293	21	40	B	−3.6
Raw carrot	0.93	35	320	12	33	B	−5.7
Cauliflower	1.84	32	142	9	16	B	−1.3
Asparagus	2.4	54	224	14	23	B	−2.2
Celery	0.69	24	260	11	40	B	−5.0
Cucumber	0.65	24	147	13	16	B	−2.4
Raw spinach	2.86	49	558	79	99	B	−11.8
Tomato	0.88	24	237	11	10	B	−4.1
Kale	2.92	55	348	33	254	B	−8.0
Lettuce	1.23	30	247	14	33	B	−4.3
Mushrooms	2.5	120	448	9	18	B	−4.2
Onions	1.1	29	146	10	23	B	−2.0
Apple	0.26	11	107	5	6	B	−1.9

Banana	1.09	22	358	27	5	B	−6.9
Grapes	0.72	20	191	7	10	B	−3.2
Kiwi	1.14	34	312	17	34	B	−5.6
Lemon	1.1	16	138	8	26	B	−2.3
Mango	0.82	14	168	10	11	B	−3.0
Orange	0.94	14	181	10	40	B	−3.6
Strawberries	0.67	24	153	13	16	B	−2.5
Watermelon	0.61	11	112	10	7	B	−2.0
Cherry	0.4	11	146	18	12	B	−3.1

Note: Values captured based on USDA Food Composition, **Databases:** A- Acid load; B- Base load; N- Neutral

DAL and Insulin Resistance

Several cohort studies have demonstrated a strong association between high DAL and insulin resistance, particularly in individuals without pre-existing diabetes or metabolic disorders. For instance, a large cross-sectional study with the participants found that higher fasting plasma lactate, a marker of low-grade metabolic acidosis, correlated positively with insulin resistance, even after adjusting for obesity [11, 12]. Obese individuals, in particular, showed higher lactate levels than their lean counterparts, suggesting that obesity may exacerbate acidosis, further impairing insulin sensitivity. Mechanistically, lower extracellular pH may reduce insulin receptor phosphorylation and downstream signaling, as observed in *in-vitro* models, where a more acidic environment inhibited insulin activity. One hypothesis is that insulin resistance may be a physiological adaptation, facilitating muscle protein breakdown to produce ammonium, which buffers the acid load. While evidence supports the link between high DAL and insulin resistance, the exact mechanisms remain unclear, and disentangling the effects of diet, obesity, and acid load on insulin sensitivity remains a complex challenge. Nevertheless, current western diets, which are typically high in acid-producing foods, are thought to contribute to both obesity and insulin resistance, amplifying metabolic risk.

DAL in Metabolic Disorders

Dietary acid load refers to the net acid-producing potential of the diet and is commonly assessed using validated indices such as PRAL and NEAP. These measures estimate the burden placed on renal and systemic buffering systems to maintain acid-base homeostasis, based on the intake of acid-forming nutrients primarily animal proteins rich in sulphur-containing amino acids and base-forming nutrients, such as potassium, magnesium, and calcium, typically found in fruits and vegetables. A persistently high DAL, often stemming from western-style dietary patterns characterized by excessive consumption of meats, processed foods, and refined grains, alongside inadequate intake of plant-based foods, has been increasingly associated with adverse metabolic outcomes. Emerging evidence suggests that a high dietary acid load may play a significant role in the etiology and progression of metabolic disorders, particularly those involving insulin resistance and impaired glucose metabolism (Figure 1). One proposed mechanism involves the induction of chronic low-grade metabolic acidosis, which may disrupt intracellular signalling pathways critical to insulin sensitivity. Acidosis can impair insulin receptor activity, reduce glucose transporter (GLUT4) expression, and diminish glucose uptake in peripheral tissues, thereby promoting hyperglycemia. Additionally, acidosis has been shown to stimulate cortisol secretion a glucocorticoid hormone known to antagonize insulin action, further compounding insulin resistance [13].

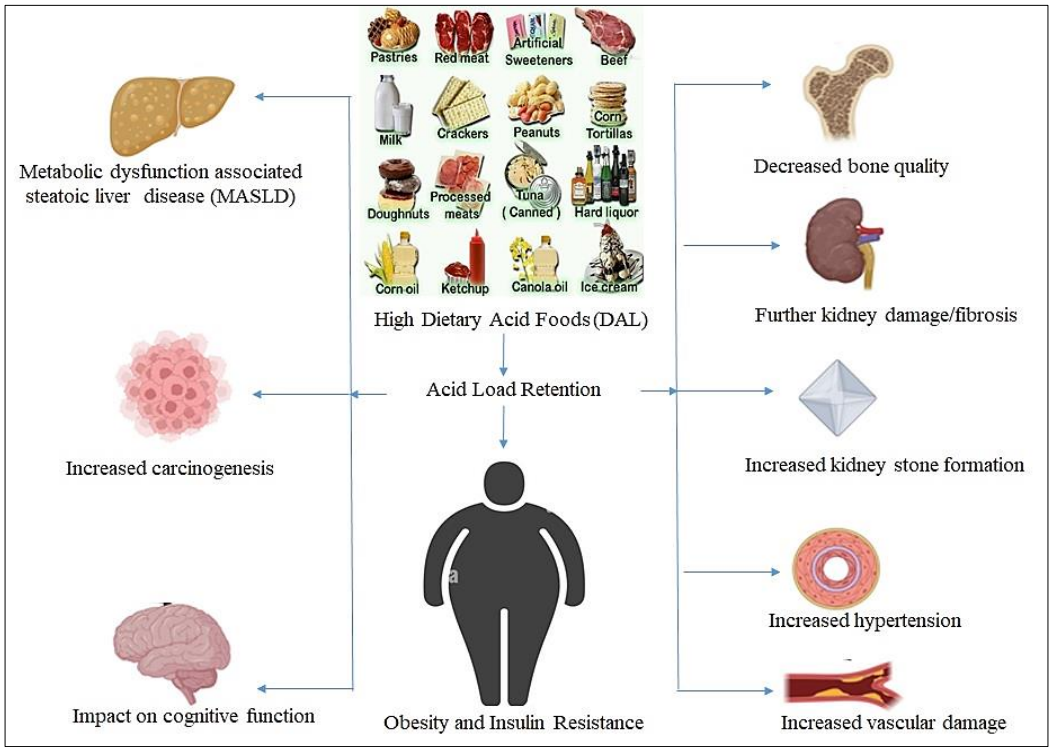

Fig 1: Illustration of the consequences of a high dietary acid load

Beyond its effects on insulin signalling, a high DAL has also been linked to pancreatic β-cell dysfunction. Chronic acidogenic stress may impair β-cell viability and function by promoting oxidative stress, inflammatory cytokine production, and mitochondrial dysfunction factors known to compromise insulin secretion. In individuals with pre-existing metabolic syndrome or obesity, elevated DAL appears to exacerbate systemic inflammation, oxidative stress, and dyslipidaemia, thus amplifying the overall cardiometabolic risk profile. Notably, DAL may also interact with other components of metabolic health, including blood pressure regulation and adipokine secretion. Studies have reported associations between high DAL and increased risk of hypertension, possibly due to alterations in renal sodium handling and vascular tone under acidic conditions. Moreover, acidogenic diets may alter adipocyte function, reducing levels of adiponectin a hormone with anti-inflammatory and insulin-sensitizing properties and increasing pro-inflammatory markers like TNF-α and IL-6. Given these multifaceted mechanisms, dietary acid-base balance is emerging as a potentially modifiable factor in the prevention and management of metabolic disorders, including type 2 diabetes mellitus and its related comorbidities.

Dietary Patterns of Foods in T1D, T2D and Gestational Diabetes Mellitus Incidence and Management

Dietary patterns rich in plant-based foods comprising fruits, vegetables, legumes, whole grains, nuts, and seeds are increasingly recognized for their potential in reducing the risk of developing Type 1 Diabetes, Type 2 Diabetes and GDM, as well as in managing these conditions once diagnosed. While the pathogenesis of T1D is primarily autoimmune, and largely driven by genetic predisposition, emerging evidence suggests that early dietary influences, particularly the consumption of plant-based foods, may modulate the immune response and help delay or prevent the onset of the disease. In contrast, T2D, a metabolic disorder primarily driven by insulin resistance, is closely linked to lifestyle factors, including poor dietary habits. In both types of diabetes, plant-based diets offer substantial health benefits, due to their nutrient density, anti-inflammatory properties, and positive effects on insulin sensitivity [14].

Plant-Based Diets and T1D Incidence

In Type 1 Diabetes, the role of diet in disease onset and progression is less well defined compared to T2D. However, research suggests that certain dietary factors, such as early-life exposure to plant-based foods, could influence the risk of developing T1D. Epidemiological studies have indicated that diets rich in fruits, vegetables, and whole grains during infancy and early childhood may help modulate immune function, potentially reducing the risk of autoimmune diseases like T1D. The high fiber content of plant foods is believed to promote a healthy gut microbiota, which plays a pivotal role in immune system regulation. An imbalance in gut microbiota composition (dysbiosis) has been associated with increased immune activation and inflammation, which are implicated in autoimmune diseases, including T1D. Thus, diets rich in plant-based fiber, antioxidants, and polyphenols may contribute to immune tolerance and reduce the inflammatory pathways that trigger β-cell destruction. Additionally, plant-based diets may help mitigate environmental factors that contribute to the onset of T1D, such as oxidative stress and chronic low-grade inflammation. The anti-inflammatory and antioxidant-rich profile of plant foods is thought to protect pancreatic β-cells from autoimmune attack and damage. However, more research is needed to clearly establish the precise role of plant-based diets in T1D prevention, as the disease's multifactorial etiology means that genetics and immune system function are also key contributors [15].

Plant-Based Diets and T2D Incidence

In contrast to T1D, Type 2 Diabetes is more strongly associated with lifestyle factors, particularly poor dietary habits, sedentary behavior, and obesity. In this context, plant-based dietary patterns have shown considerable promise in both preventing and managing T2D. A growing body of evidence supports the idea that plant-based diets can improve insulin sensitivity, promote weight loss, and reduce overall diabetes risk. These diets, characterized by high intake of non-starchy vegetables, whole grains, legumes, and nuts, are naturally low in saturated fat and rich in fiber, vitamins, minerals, and phytochemicals nutrients that collectively enhance metabolic health. For example, fiber, particularly soluble fiber found in legumes, oats, and certain fruits and vegetables, has a profound effect on glucose metabolism. Fiber slows down carbohydrate digestion and absorption, leading to more gradual increases in blood sugar levels, thereby improving postprandial glycemic control. This effect is particularly beneficial for individuals with T2D, who often struggle with blood sugar spikes following meals. Furthermore, plant-based diets are associated with a lower glycemic index, which helps stabilize blood sugar levels and improve long-term glycemic control. Moreover, plant-based foods are rich in polyphenols, bioactive compounds with potent anti-inflammatory and antioxidant properties [16]. Chronic low-grade inflammation is a hallmark of insulin resistance and is closely linked to T2D pathogenesis. Diets rich in plant foods, such as berries, leafy greens, and cruciferous vegetables, provide an abundance of antioxidants that counteract oxidative stress and reduce inflammatory markers, which may, in turn, improve insulin sensitivity and reduce the risk of T2D.

In clinical trials, plant-based diets have shown significant benefits in reducing key risk factors for T2D, including excess body weight, blood pressure, and dyslipidemia. For instance, randomized controlled trials have demonstrated that individuals with T2D following a plant-based diet experience improved fasting blood glucose levels, enhanced insulin sensitivity, and reduced HbA1c levels compared to those on standard western diets [17]. Additionally, these dietary patterns often lead to reductions in body fat, which is a critical factor in reversing insulin resistance.

Plant-Based Diets and Gestational Diabetes Mellitus Incidence

Plant-based diets have been associated with a reduced incidence of gestational diabetes mellitus, a common pregnancy complication characterized by impaired glucose tolerance. Diets that emphasize whole plant foods such as vegetables, fruits, whole grains, legumes, nuts, and seeds are rich in dietary fiber, antioxidants, and phytochemicals, which can improve insulin sensitivity and reduce systemic inflammation, both key factors in the development of GDM. Studies have shown that women who follow plant-based or

predominantly vegetarian dietary patterns before and during pregnancy have a significantly lower risk of developing GDM compared to those consuming diets high in red and processed meats, refined grains, and saturated fats. The lower glycemic load and anti-inflammatory properties of plant-based diets likely contribute to better glucose regulation during pregnancy [18]. While more research is needed to understand the specific mechanisms, current evidence supports the potential of plant-based eating patterns as a preventive approach to reducing GDM incidence.

Managing Diabetes with Plant-Based Diets

For individuals already diagnosed with T1D or T2D or GDM, plant-based diets can play a crucial role in managing the condition and preventing complications. In T2D, and GDM the combination of low-fat, high-fiber foods can help individuals achieve and maintain a healthy weight, a key component of diabetes management. Additionally, plant-based diets, rich in micronutrients like magnesium, potassium, and folate, support cardiovascular health, which is particularly important for people with T2D who are at increased risk of heart disease (Table 4).

In T1D, while insulin therapy remains essential, dietary adjustments focusing on plant-based foods can help improve overall metabolic control. Since plant foods generally have a low glycemic index, they can be beneficial for maintaining stable blood glucose levels throughout the day. Furthermore, consuming a diet high in plant-based foods can help reduce the burden of diabetes-related complications, such as retinopathy, nephropathy, and neuropathy, by mitigating inflammation and oxidative stress [19, 20].

However, individuals with T1D and T2D must be mindful of specific nutrient needs when adopting a plant-based diet. For example, vitamin B12, iron, calcium, and omega-3 fatty acids are nutrients that may be more challenging to obtain from a plant-based diet, especially for those following a vegan regimen. Thus, careful planning and, when necessary, supplementation is essential to ensure nutritional adequacy.

Table 4: Characteristics of dietary patterns emphasizing the consumption of plant foods

Dietary pattern	Foods
#DASH diet	Emphasizes fruits, vegetables, and low-fat dairy products, and includes whole grains, poultry, fish, and nuts. Reduced consumption of saturated fat, red meat, sweets, and sodium.
Macrobiotic diet	Emphasizes locally grown whole-grain cereals, pulses (legumes), vegetables, seaweed, fermented soy products, and fruit, combined into meals according to the ancient Chinese principle of balance known as yin and yang.
Mediterranean diet	Characterized by a high intake of olive oil as the principal source of dietary fat, fruit, nuts, vegetables, and cereals; a moderate intake of fish and poultry; a low intake of dairy products, red meat, processed meats, and sweets; and a moderate intake of wine with meals.
Pescatarian diet	Plant-based diet including fish or other seafood, but not the flesh of other animals.
Vegan diet	Plant-based diet avoiding all animal foods such as meat (including fish, shellfish, and insects), dairy, eggs, and honey, as well as products such as leather, and those that are tested on animals.
Vegetarian diet	Plant-based diet avoiding all animal flesh-based foods and animal-derived products. Some modified versions allow eggs (ovo), dairy products (lacto), or a combination of both.

#**DASH** - Dietary Approaches to Stop Hypertension.

Future Research Directions and Nutritional Recommendations

Future research on DAL and its impact on the pathophysiology and clinical progression of various forms of diabetes should prioritize well-designed, longitudinal cohort studies and randomized controlled trials. These future studies are essential to establish causal links between high DAL and the incidence or severity of type 1, type 2, and gestational diabetes mellitus. A deeper mechanistic understanding is required to elucidate how diet-induced acid-base imbalances influence insulin resistance, pancreatic β-cell dysfunction, low-grade systemic inflammation, and metabolic acidosis, all of which are implicated in diabetic pathogenesis. Special attention should be given to the role of specific acidogenic nutrients such as sulfur-containing amino acids from animal protein and the buffering potential of alkaline-forming foods rich in potassium, magnesium, and bicarbonate precursors. Furthermore, inter-individual variability in response to DAL should be investigated in the context of genetic polymorphisms, gut microbiota composition, renal function, and other metabolic determinants. Developing standardized, validated tools for quantifying DAL, such as net endogenous acid production and PRAL, across diverse dietary patterns and populations will enhance the reliability and comparability of research findings. Nutritional recommendations derived from this research should advocate for dietary patterns with a lower acid load favouring plant-based foods, whole grains, legumes, fruits, and vegetables, while limiting the consumption of highly acidogenic foods like red meats and processed products. Incorporating DAL considerations into clinical nutrition guidelines and public health policies may offer a novel, evidence-based strategy for diabetes prevention and management at both individual and population levels.

Conclusion

In summary, this comprehensive review highlights the emerging role of DAL as a modifiable nutritional factor influencing metabolic health and the development of various forms of diabetes, including T1D, T2D, and GDM. A growing body of evidence suggests that diets high in acid-forming foods characteristic of Western dietary patterns may contribute to insulin resistance, β-cell dysfunction, systemic inflammation, and other metabolic disturbances central to the pathogenesis of diabetes. Conversely, dietary patterns rich in alkaline-forming plant-based foods appear to confer protective effects, supporting improved glycemic control and reduced diabetes risk. This review underscores the importance of validated DAL assessment tools, such as PRAL and NEAP, for accurately evaluating acid-base dietary contributions across diverse populations and study settings. Despite promising findings in literature, inconsistencies and the presence of confounding variables necessitate further well-designed longitudinal studies and mechanistic research. Incorporating DAL into clinical

nutrition practice and public health strategies may offer a novel, cost-effective approach for diabetes prevention and management. As our understanding of DAL continues to evolve, it holds significant potential to shape future dietary guidelines aimed at improving metabolic outcomes and reducing the global burden of diabetes.

Conflict of Interest
Not available.

Financial Support
Not available.

References

1. Wieers MLAJ, Cobb BB, Visser WJ, Attaye I. Dietary acid load in health and disease. Pflugers Arch European Journal of Physiology. 2024;476:427-443. DOI:10.1007/s00424-024-02910-7
2. Padillaa IAO, Escobar GL, Garcíac CAG, Castellanos FER. Dietary acid load: Mechanisms and evidence of its health repercussions Carga ácida de la dieta; mecanismos y evidencia de sus repercusiones en la salud. Nephrology (English Edition). 2019;39(4):343-354. DOI:10.1016/j.nefro.2018.10.005
3. Seifi N, Rahimi H, Koochakpoor G, Zarei A, Darban RA, *et al*. Higher dietary acid load is associated with an increased risk of metabolic syndrome. Scientific Reports. 2023;13:22154. DOI:10.1038/s41598-023-48429-2
4. Murciano A, Malavolti M, Tait SF, Paduano S, Vinceti M, Filippini T. Plant-based diets and risk of type 2 diabetes: systematic review and dose-response meta-analysis. British Journal of Nutrition. 2025;12:20. DOI:10.1017/s0007114525104157
5. Abshirini M, Bagheri F, Mahaki B, Siassi F, Koohdani F, Safabakhsh M, *et al*. The dietary acid load is higher in subjects with prediabetes who are at greater risk of diabetes: a case-control study. Diabetology & Metabolic Syndrome. 2019;11:52. DOI:10.1186/s13098-019-0447-5
6. Ghorbani M, Moradi N, Behzai B, Shahani T, Mousavi MS, Fathi S. Investigating the relationship between dietary acid load and risk of diabetes; a meta-analysis. Journal of Preventive Epidemiology. 2021;6(1):e16. DOI:10.34172/jpe.2021.16
7. Salvadó JS, Tomás NB, Papandreou C, Bulló B. Dietary Patterns Emphasizing the Consumption of Plant Foods in the Management of Type 2 Diabetes: A Narrative Review. Advances in Nutrition. 2019;10(4):S320-S331. DOI:10.1093/advances/nmy102
8. Kinoshitaa K, Osukaa Y, Yoshiuraa K, Hori N, Georga VF, Satake S, *et al*. High dietary acid load increases the risk of disability in women aged 75 years and older: A community-based cohort study. The Journal of Frailty & Aging. 2025;14(1):100004. DOI:10.1016/j.tjfa.2024.100004
9. Remer T, Manz F. Potential renal acid load of foods and its influence on urine pH. J Am Diet Assoc. 1995;95:791-797. DOI:10.1016/S0002-8223(95)00219-7
10. Remer T, Manz F. Estimation of the renal net acid excretion by adults consuming diets containing variable amounts of protein. Am J Clin Nutr. 1994;59:1356-1361. DOI:10.1093/ajcn/59.6.1356
11. Smeha L, Fassula AS, Franco Moreno YM, Gonzalez-Chica DA, Nunes EA. Dietary acid load is positively associated with insulin resistance: a population-based study. Clin Nutr ESPEN. 2022;49:341-347. DOI:10.1016/j.clnesp.2022.03.025
12. Vetrani C, Bozzetto L, Pepa GD, Rivellese AA, Annuzzi G. Fruitarian Diet and Blood Glucose Control in Type 1 Diabetes: A Case Report. Frontiers in Nutrition. 2022;9:752832. DOI:10.3389/fnut.2022.752832
13. Storz MA, Ronco AL, Hannibal L. Observational and clinical evidence that plant-based nutrition reduces dietary acid load. J Nutr Sci. 2022;11(e93):1-15. DOI:10.1017/jns.2022.93
14. Silva JCD, Anghinoni ICAB, Gomes MB. Plant-Based Food for the Prevention of Type 2 Diabetes: Scoping Review. Nutrients. 2024;16(11):1671. DOI:10.3390/nu16111671
15. Bakhshi H, Naghshi N, Fotros D, Pasand M, Hekmatdoos A, Salavatizadeh M, *et al*. Association between dietary acid load and risk of metabolic dysfunction-associated steatotic liver disease in patients with type 2 diabetes. Frontiers in Nutrition. 2025;12:1-8. DOI:10.3389/fnut.2025.1494617
16. Chen GC, Chai JC, Xing J, Moon JY, Shan Z, Yu B, *et al*. Healthful eating patterns, serum metabolite profile and risk of diabetes in a population-based prospective study of US Hispanics/Latinos. Diabetologia. 2022;65(7):1133-1144. DOI:10.1007/s00125-022-05690-w
17. Elshoff D, Mehta P, Ziouzenkova O. Chronic Kidney Disease Diets for Kidney Failure Prevention: Insights from the IL-11 Paradigm. Nutrients. 2024;16(9):1342. DOI:10.3390/nu16091342
18. Jong JCKD, Li Y, Chen M, Curhan GC, Mattei J, Malik VS, *et al*. Diet-dependent acid load and type 2 diabetes: pooled results from three prospective cohort studies. Diabetologia. 2017;60(2):270-279. DOI:10.1007/s00125-016-4153-7
19. Vidal AP, Serra JM, Saez EL, Monleón SD, Martin FC, Ramírez AP, Suárez JT, Moreno JF. Hereditary renal hypouricaemia type 1 and 2 in three Spanish children. Review of published paediatric cases. Nefrología. 2019;39(4):355-361. DOI:10.1016/j.nefro.2018.08.010
20. Palem CR, Gannu R, Yamsani SK, Yamsani VV, Yamsani MR. Development of bioadhesive buccal tablets for felodipine and pioglitazone in combined dosage form: *in vitro*, ex vivo, and *in vivo* characterization. Drug Delivery. 2011;18(5):344-352.
21. Shiva F, Nourimajd S, Asadi S, Rasaei N, Hasanzadeh M, Qorbani M, *et al*. Association of dietary acid-base load and diabetic sensorimotor polyneuropathy in patients with type 2 diabetes mellitus: A case-control study. Clin Nutr ESPEN. 2022;50:118-123. DOI:10.1016/j.clnesp.2022.06.012
22. Palem CR, Dudhipala N, Battu SK, Goda S, Repka MA, Yamsani MR. Combined dosage form of pioglitazone and felodipine as mucoadhesive pellets via hot melt extrusion for improved buccal delivery with application of quality by design approach. J Drug Deliv Sci Technol. 2015;30:209-219.

How to Cite This Article
Palem CR, Gumudevelli S, Rendla R, Srinija G, Gumudevell S. Comprehensive review on the role of Dietary Acid Load in health and its effect on different types of Diabetes. International Journal of Diabetes Research. 2025; 7(1): 45-53.