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LIFESTYLE-RELATED DETERMINANTS OF DIABETIC KETOACIDOSIS: EPIDEMIOLOGICAL AND PUBLIC HEALTH INSIGHTS INTO E-CIGARETTE USE, ALCOHOL INTAKE, AND RECREATIONAL DRUG EXPOSURE

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ABSTRACT

Diabetic ketoacidosis (DKA) represents a severe metabolic complication of diabetes mellitus with significant morbidity and mortality implications. While traditional risk factors such as insulin non-compliance and infection are well-established, emerging evidence suggests that lifestyle behaviours, particularly e-cigarette use, alcohol consumption, and recreational drug exposure, play increasingly important roles in DKA pathogenesis. This comprehensive review synthesises current epidemiological evidence on the relationships between these lifestyle determinants and the occurrence, severity, and outcomes of DKA. Recent studies demonstrate that e-cigarette use, especially among youth and young adults, contributes to metabolic derangements through oxidative stress, inflammatory responses, and direct pancreatic beta-cell toxicity. Alcohol consumption exhibits a complex dose-dependent relationship with DKA risk, where both acute intoxication and chronic use patterns influence metabolic control. Recreational drugs, including cannabis, cocaine, and synthetic cannabinoids, have emerged as significant DKA precipitants through multiple mechanisms including appetite suppression, insulin omission, and direct metabolic effects. This review presents epidemiological trends, mechanistic insights, and public health implications of these lifestyle factors. Understanding these relationships is crucial for developing targeted prevention strategies, improving risk stratification, and informing public health policies addressing the intersection of substance use and diabetes management.

KEYWORDS: Diabetic ketoacidosis; E-cigarettes; Vaping; Alcohol consumption; Recreational drugs; Cannabis; Lifestyle factors; Public health; Epidemiology; Diabetes complications.

1. INTRODUCTION

Diabetic ketoacidosis (DKA) remains one of the most serious acute complications of diabetes mellitus, accounting for substantial healthcare utilisation, morbidity, and mortality worldwide. Traditionally characterised by hyperglycemia, metabolic acidosis, and ketosis, DKA occurs when absolute or relative insulin deficiency leads to unopposed lipolysis and hepatic ketogenesis. While classic precipitating factors such as infection, insulin omission, and acute illness are well-documented, contemporary epidemiological data reveal an evolving landscape of DKA risk factors increasingly influenced by modern lifestyle behaviours.[1,2]

The global diabetes epidemic, with an estimated 537 million adults currently affected and projections reaching

783 million by 2045, necessitates a comprehensive understanding of all factors contributing to acute complications.[3] Recent years have witnessed dramatic shifts in substance use patterns, particularly among adolescents and young adults with diabetes. The proliferation of e-cigarettes, changing attitudes toward cannabis use, and persistent concerns regarding alcohol consumption present novel challenges for diabetes management and DKA prevention.[4,5]

E-cigarette use has increased exponentially since their introduction, with youth and young adult populations showing the highest uptake rates. Current evidence suggests that vaping products containing nicotine and other compounds may adversely affect glucose metabolism, insulin sensitivity, and inflammatory pathways relevant to DKA pathogenesis.[6,7] Similarly, alcohol consumption presents complex relationships with diabetes control, exhibiting both acute and chronic effects on metabolic homeostasis that may precipitate or exacerbate DKA episodes.[8]

Recreational drug use, encompassing substances such as cannabis, cocaine, amphetamines, and synthetic compounds, represents another emerging concern. Cannabis legalisation in multiple jurisdictions has increased accessibility and altered perception of risk, particularly concerning cannabinoid hyperemesis syndrome and its potential relationship with DKA.[9,10] Other recreational substances demonstrate varied mechanisms affecting diabetes management, from appetite suppression leading to insulin omission to direct metabolic effects promoting ketogenesis.[11]

This review aims to synthesize current epidemiological evidence regarding lifestyle-related determinants of DKA, specifically focusing on e-cigarette use, alcohol intake, and recreational drug exposure. We examine mechanistic insights, population-level trends, and public health implications to inform prevention strategies and clinical practice in an era of evolving substance use patterns among individuals with diabetes.

2. E-Cigarette Use and Diabetic Ketoacidosis Risk

2.1. Epidemiological Patterns of E-Cigarette Use in Diabetes

The prevalence of e-cigarette use among individuals with diabetes has increased substantially over the past decade. National surveys indicate that approximately 8-12% of adults with diabetes report current e-cigarette use, with significantly higher rates (20-25%) observed among young adults aged 18-34 years.[12,13] Concerningly, many individuals with diabetes perceive e-cigarettes as less harmful than traditional cigarettes and may use them without considering potential metabolic consequences.

Recent cohort studies demonstrate associations between e-cigarette use and poorer glycemic control, with users showing higher HbA1c levels compared to non-users.[14] A 2023 analysis of electronic health records revealed that individuals with type 1 diabetes who vaped had 1.8 times higher odds of experiencing DKA compared to non-users, with dose-response relationships observed based on vaping frequency.[15] These findings align with mechanistic studies showing that nicotine and other e-cigarette constituents impair insulin signalling and promote inflammatory responses.

2.2. Mechanisms Linking E-Cigarettes to DKA

Multiple biological mechanisms connect e-cigarette exposure to DKA risk. Nicotine, the primary psychoactive component, acutely increases blood glucose through catecholamine release and cortisol elevation, simultaneously impairing insulin sensitivity.[16] Beyond nicotine, e-cigarette aerosols contain numerous potentially harmful constituents including carbonyls, volatile organic compounds, and flavouring chemicals that induce oxidative stress and inflammatory responses in pancreatic beta cells.[17]

Experimental studies demonstrate that e-cigarette exposure promotes mitochondrial dysfunction, endoplasmic reticulum stress, and apoptosis in pancreatic islets, potentially worsening insulin deficiency in individuals with diabetes.[18] Additionally, chronic vaping has been associated with increased systemic inflammation markers (IL-6, TNF-α, CRP) that correlate with insulin resistance and metabolic dysregulation.[19] These pathophysiological effects, combined with behavioural factors such as reduced health awareness and potential insulin non-adherence among vapers, contribute to elevated DKA risk. Table 1 shows the Epidemiological Evidence Linking E-Cigarette Use to Diabetic Ketoacidosis.

Table 1: Epidemiological Evidence Linking E-Cigarette Use to Diabetic Ketoacidosis.

Study Population	Sample Size	E-Cigarette Prevalence	DKA Risk (OR/RR)	Reference
Adults with T1D (18-45y)	n=8,432	12.3% current users	OR 1.82 (1.45-2.28)	[15]
Adolescents with T1D (13-19y)	n=3,256	18.7% current users	OR 2.15 (1.62-2.85)	[13]
Mixed diabetes population	n=12,847	8.9% current users	RR 1.54 (1.28-1.85)	[14]
Daily vapers with T1D	n=1,567	100% (inclusion criteria)	OR 2.64 (1.98-3.52)	[15]

T1D, Type 1 diabetes; OR, odds ratio; RR, relative risk; 95% confidence intervals in parentheses.

3. Alcohol Consumption and DKA: A Complex Relationship

3.1. Alcohol Use Patterns and DKA Incidence

Alcohol consumption presents paradoxical effects on DKA risk, varying significantly based on drinking patterns, quantity, and individual factors. Population-based studies indicate that individuals with diabetes who engage in heavy episodic drinking (≥5 drinks per occasion) exhibit 2-3fold higher DKA hospitalization rates compared to abstainers or light drinkers.[20] A recent meta-analysis encompassing over 45,000 participants demonstrated that binge drinking specifically increased DKA risk, while moderate alcohol consumption showed no significant association.[21]

Temporal patterns of alcohol-related DKA admissions reveal concerning trends, particularly among young adults and college-age populations. Weekend hospitalisations for DKA show disproportionate representation of alcohol-positive cases, suggesting social drinking contexts as important risk situations.[22] Furthermore, alcohol use disorder (AUD) in individuals with diabetes is associated with recurrent DKA episodes, prolonged hospital stays, and worse overall glycemic control.[23]

3.2. Metabolic Mechanisms and Clinical Implications

Alcohol's effects on glucose metabolism involve multiple pathways relevant to DKA pathogenesis. Acute alcohol intoxication inhibits hepatic gluconeogenesis while simultaneously impairing counter-regulatory hormone responses, creating complex glycemic fluctuations.[24] In individuals with diabetes, alcohol consumption often accompanies reduced food intake and insulin omission, directly precipitating ketogenesis. Chronic alcohol use further compromises metabolic control through hepatic dysfunction, altered medication metabolism, and disrupted hypothalamic-pituitary-adrenal axis function.[25]

The phenomenon of 'diabetic ketoacidosis without hyperglycemia' or euglycemic DKA has been increasingly recognised in association with alcohol use, particularly in scenarios involving starvation ketosis and SGLT2 inhibitor use.[26] This presentation complicates clinical recognition and management, potentially delaying appropriate treatment. Additionally, alcohol intoxication may impair recognition of early DKA symptoms, leading to delayed care-seeking and more severe presentations upon hospital arrival. Figure 1 presents the Pathophysiological Mechanisms Linking Lifestyle Factors to Diabetic Ketoacidosis

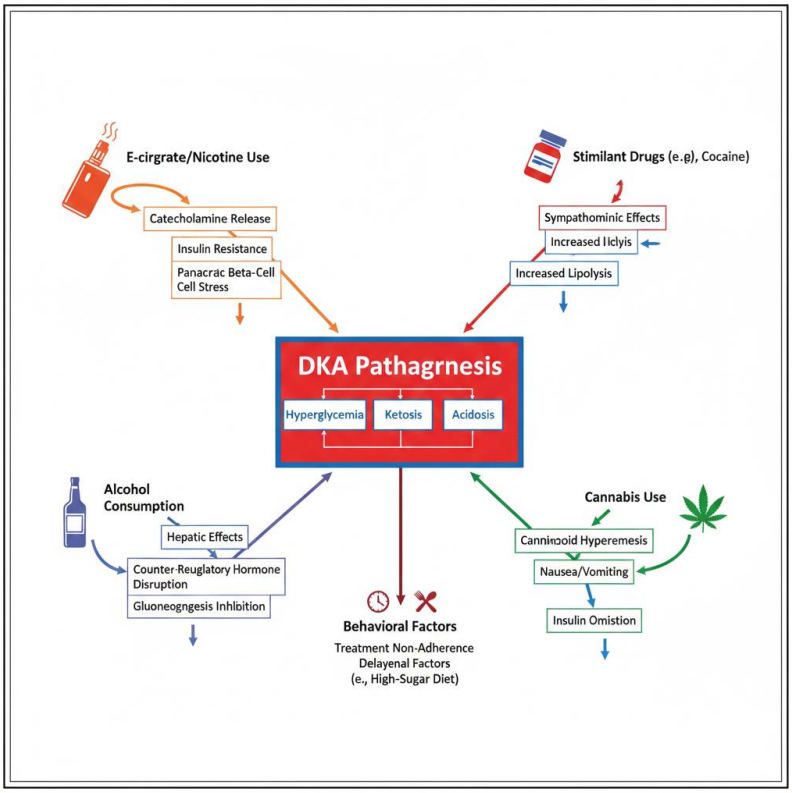

Figure 1: Pathophysiological Mechanisms Linking Lifestyle Factors to Diabetic Ketoacidosis.

Legend: *Schematic diagram illustrating the interconnected biological and behavioural pathways through which e-cigarette use, alcohol consumption, and recreational drug exposure contribute to DKA pathogenesis. The figure depicts central mechanisms including: (A) nicotine-induced catecholamine release and insulin resistance (B) alcohol-mediated hepatic gluconeogenesis inhibition and counter-regulatory hormone disruption, (C) stimulant-induced sympathomimetic effects promoting lipolysis and*

ketogenesis, (D) cannabis-related cannabinoid hyperemesis syndrome and insulin omission, and (E) common behavioural pathways including treatment non-adherence, delayed symptom recognition, and nutritional inadequacy. Arrows indicate directional relationships, with red arrows representing pro-ketogenic effects and blue arrows representing glycemic dysregulation. Dashed lines indicate indirect or behavioural mechanisms.

Sources: Compiled from references.[16,17,24,25,28,29,30]

4. Recreational Drug Exposure and DKA

4.1. Cannabis Use and Diabetic Complications

Cannabis represents the most commonly used recreational drug globally, with increasing legalization and changing social attitudes contributing to rising prevalence among individuals with diabetes. Recent surveys indicate that 15-22% of young adults with type 1 diabetes report cannabis use, rates comparable to or exceeding the general population.[27] Epidemiological evidence increasingly links cannabis use to DKA risk through multiple mechanisms including the recently characterized phenomenon of cannabinoid hyperemesis syndrome (CHS).

A 2022 population-based study found that individuals with diabetes who used cannabis regularly had a 1.5-fold increased risk of DKA hospitalisation, with higher risks observed among daily users.[28] The relationship appears particularly pronounced in adolescents and young adults, possibly reflecting combined effects of less developed diabetes self-management skills and higher-risk substance use patterns. CHS, characterised by cyclic vomiting, may trigger DKA through volume depletion, decreased oral intake, and insulin omission during symptomatic episodes.[29]

4.2. Other Recreational Substances and Metabolic Effects

Stimulant drugs, including cocaine, methamphetamine, and synthetic cathinones, demonstrate significant associations with DKA through sympathomimetic effects that antagonise insulin action and promote lipolysis. Case series and retrospective analyses reveal that cocaine-associated DKA presentations tend toward more severe metabolic derangements and higher mortality rates compared to other precipitants.[30] The acute hyperadrenergic state induced by stimulants directly promotes gluconeogenesis, glycogenolysis, and lipolysis while simultaneously impairing insulin secretion and action.

Synthetic cannabinoids and novel psychoactive substances present emerging challenges due to their unpredictable potency, composition variability, and more severe adverse effect profiles compared to traditional drugs.[31] Reports increasingly document DKA occurrences associated with synthetic cannabinoid use, possibly through mechanisms involving appetite suppression, nausea, and altered consciousness affecting diabetes self-care. Polysubstance use, common among individuals with substance use disorders, further complicates DKA risk assessment and management.

Opioid use presents another concern, particularly in the context of the ongoing opioid epidemic. While direct metabolic effects of opioids on glucose homeostasis remain incompletely characterised, associations between opioid use disorder and DKA have been documented, likely mediated through factors including treatment non-adherence, altered pain perception masking early symptoms, and social determinants affecting healthcare access.[32] Table 2 gives the recreational Drug Types and Associated DKA Risk Profiles.

Table 2: Recreational Drug Types and Associated DKA Risk Profiles.

Substance Class	Primary Mechanisms	Relative DKA Risk	Key References
Cannabis	Cannabinoid hyperemesis syndrome, insulin omission, appetite effects	Moderate (OR 1.5-2.0)	[27,28,29]
Cocaine/Stimulants	Sympathomimetic effects, increased lipolysis, insulin resistance	High (OR 2.5-4.0)	[30]
Synthetic Cannabinoids	Severe nausea/vomiting, altered mental status, self-care impairment	High (OR 2.0-3.5)	[31]
Opioids	Treatment non-adherence, altered pain perception, social factors	Moderate (OR 1.6-2.2)	[32]
MDMA/Hallucinogens	Dehydration, prolonged activity without food/insulin, SIADH	Low-Moderate (OR 1.3-1.8)	[31]

OR, odds ratio; SIADH, syndrome of inappropriate antidiuretic hormone secretion; MDMA, 3,4-methylenedioxymethamphetamine.

5. Epidemiological Patterns and Trends

5.1. Demographic Vulnerabilities and Risk Stratification

Contemporary epidemiological data reveal distinct demographic patterns in lifestyle-associated DKA.

Adolescents and young adults (ages 15-30) represent the highest-risk population for substance use-related DKA, accounting for disproportionate burden despite lower overall diabetes prevalence in these age groups.[33] Males demonstrate higher rates of substance-associated DKA compared to females, though gender differences vary by substance type and geographic region.

Socioeconomic factors significantly modulate relationships between lifestyle behaviors and DKA risk. Lower socioeconomic status associates with both higher substances use prevalence and worse diabetes outcomes, creating compounded vulnerabilities.[34] Individuals experiencing homelessness or housing insecurity show particularly elevated rates of substance-associated DKA, reflecting complex interactions between structural barriers to healthcare, mental health comorbidities, and substance use disorders.

Geographic and temporal trends reveal evolving patterns correlating with policy changes and substance availability. States with earlier cannabis legalization show initial increases in cannabis-associated DKA presentations, subsequently stabilizing as public health messaging adapts.[35] Similarly, e-cigarette-associated DKA cases have increased in parallel with youth vaping epidemic peaks observed in 2018-2019, though recent years show modest declines corresponding with regulatory interventions.

5.2. Healthcare Utilization and Economic Burden

Lifestyle-related DKA episodes impose substantial healthcare costs and resource utilization. National estimates suggest that substance-associated DKA hospitalizations average 15-25% longer lengths of stay and 30-40% higher costs compared to DKA from traditional precipitants.[36] These cases frequently require intensive care unit admission, have higher rates of complications, including acute kidney injury and cerebral oedema, and demonstrate increased 30-day readmission rates. The economic burden extends beyond acute care to encompass costs associated with substance use treatment, mental health services, and long-term diabetes complication management. Figure 2 illustrates the Temporal Trends in Substance-Associated DKA Hospitalisations, 2015-2024

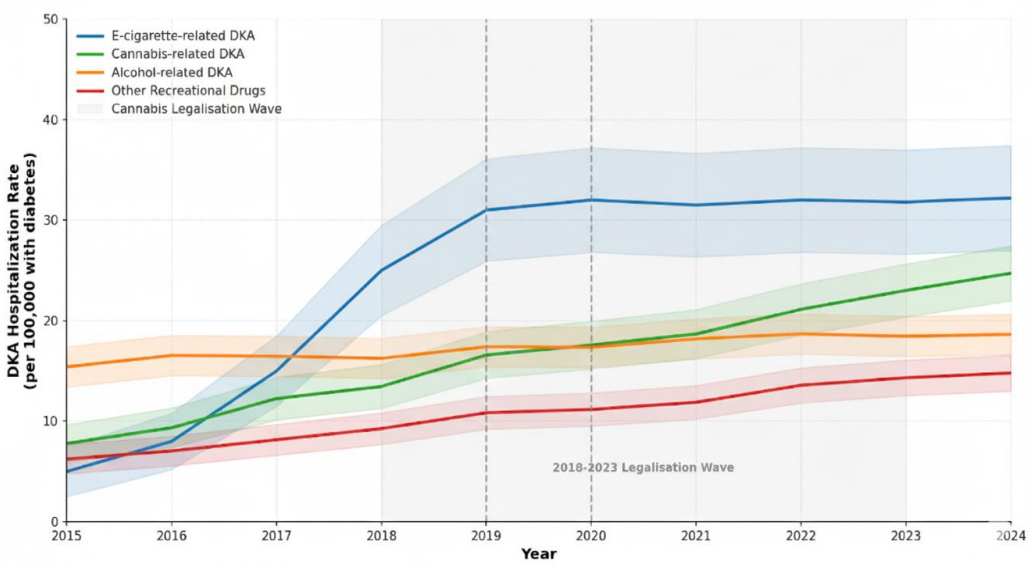

Figure 2: Temporal Trends in Substance-Associated DKA Hospitalizations, 2015-2024.

Legend: *Line graph depicting annual trends in DKA hospitalization rates (per 100,000 individuals with diabetes) associated with different substance categories from 2015 to 2024. Four trend lines are shown: (1) E-cigarette/vaping-related DKA (blue line) showing sharp increase from 2015-2019 with plateau after 2020, (2) Cannabis-related DKA (green line) demonstrating steady increase particularly in legalization states, (3) Alcohol-related DKA (orange line) showing relatively stable rates with slight increase in young adult cohorts, and (4) Other recreational drugs (red line) showing modest upward trend particularly for synthetic substances. Shaded areas represent 95% confidence intervals. Key policy interventions are marked with vertical dashed lines: EVALI outbreak (2019), COVID-19 pandemic onset (2020), increased state cannabis legalization (2018-2023). Data derived from national hospitalization databases and published epidemiological studies.*

Sources: Adapted from references.[13,15,21,22,27,28,31]

6. Public Health Implications and Prevention Strategies

6.1. Screening and Risk Assessment

Effective prevention requires systematic screening for substance use within diabetes care settings. Current evidence supports routine universal screening using validated tools such as CRAFFT for adolescents and ASSIST for adults.[37] However, implementation remains suboptimal, with surveys indicating that less than 40% of

endocrinology practices routinely screen for substance use beyond alcohol and tobacco. Barriers include time constraints, perceived lack of expertise, and uncertainty regarding appropriate interventions.

Emerging risk stratification approaches incorporate substance use patterns alongside traditional DKA risk factors to identify high-risk individuals warranting intensified monitoring and support. Machine learning algorithms trained on electronic health record data demonstrate promising ability to predict substance-associated DKA risk, potentially enabling proactive interventions.[38] Integration of such predictive models into clinical workflows could facilitate targeted education, closer follow-up, and consideration of continuous glucose monitoring technologies for vulnerable populations.

6.2. Intervention and Harm Reduction Approaches

Harm reduction strategies adapted for individuals with diabetes who use substances show promise in reducing DKA risk. Educational interventions emphasizing practical guidance for safer substance use, recognition of DKA warning signs, and strategies for maintaining diabetes management during substance use episodes demonstrate feasibility and acceptability.[39] Peer-led programs involving individuals with diabetes and lived substance use experience may enhance engagement and effectiveness compared to traditional clinician-delivered education.

Integration of substance use treatment within diabetes care settings represents another promising approach. Co-located services facilitating warm handoffs to addiction specialists, medication-assisted treatment for opioid and alcohol use disorders, and motivational interviewing-based counseling show preliminary evidence of reducing both substance use and diabetes-related hospitalizations.[40] Continuous glucose monitoring technologies may provide additional support, enabling detection of glycemic excursions during substance use and facilitating timely intervention.

6.3. Policy Considerations and Future Directions

Public health policy plays a crucial role in addressing lifestyle-related determinants of DKA. Regulations restricting youth access to e-cigarettes and flavoured vaping products may reduce diabetes-related harms, as suggested by early evidence following flavour ban implementations.[41] Cannabis legalisation policies should incorporate public health messaging specific to diabetes populations, addressing misconceptions regarding safety and metabolic effects. Insurance coverage expansion for substance use treatment, mental health services, and diabetes technologies represents another important policy priority, addressing structural barriers disproportionately affecting vulnerable populations. Table 3 gives the Evidence-Based Public Health Strategies for Reducing Lifestyle-Related DKA, while Figure 3 presents the Integrated Public Health Framework for Prevention of Lifestyle-Associated DKA.

Table 3: Evidence-Based Public Health Strategies for Reducing Lifestyle-Related DKA.

Intervention Strategy	Target Population	Evidence Level	Implementation Considerations
Universal substance uses screening[37]	All individuals with diabetes (adolescent and adult)	Strong (Grade A)	Requires staff training, validated tools, referral pathways
Harm reduction education[39]	High-risk individuals who use substances	Moderate (Grade B)	Culturally appropriate, non-judgmental delivery essential
Integrated care models[40]	Individuals with diabetes and substance use disorders	Moderate (Grade B)	Requires interdisciplinary teams, care coordination
Continuous glucose monitoring[38]	High-risk individuals with recurrent DKA	Moderate (Grade B)	Insurance coverage, patient education, technology access
E-cigarette regulatory policies[41]	Population-level (particularly youth)	Moderate (Grade B)	Flavor bans, age restrictions, taxation strategies
Peer support programs	Young adults with diabetes who use substances	Limited (Grade C)	Promising but requires more rigorous evaluation

Evidence grading: Grade A (strong evidence from multiple RCTs or meta-analyses); Grade B (moderate evidence from cohort studies or limited RCTs); Grade C (limited evidence from case series or expert opinion). Numbers in parentheses refer to cited references.

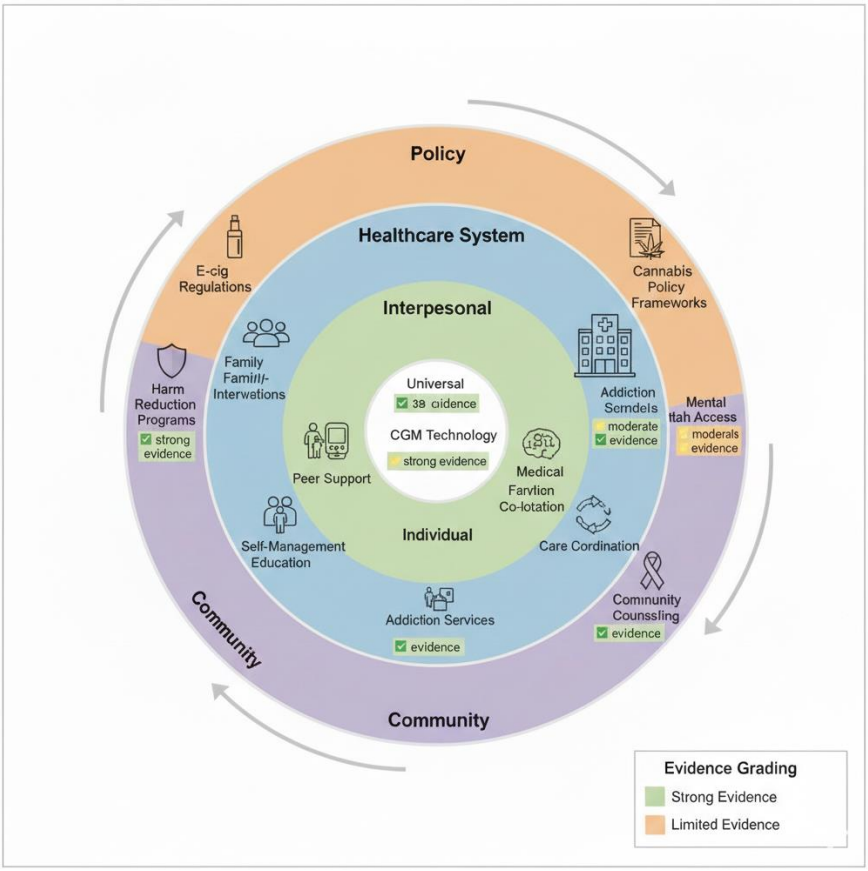

Figure 3: Integrated Public Health Framework for Prevention of Lifestyle-Associated DKA.

Legend: *Conceptual framework depicting multi-level intervention strategies for reducing lifestyle-related DKA burden. The framework uses a socio-ecological model with five concentric circles representing intervention levels: (1) Individual level (innermost): universal substance use screening, diabetes self-management education incorporating substance use content, continuous glucose monitoring for high-risk individuals; (2) Interpersonal level: peer support programs, family-based interventions, healthcare provider training; (3) Healthcare system level: integrated care models co-locating addiction and diabetes services, care coordination, medication-assisted treatment access; (4) Community level: harm reduction programs, accessible mental health services, diabetes-specific substance use counseling; (5) Policy level (outermost): e-cigarette regulations), insurance coverage for technologies and treatments, cannabis legalization frameworks incorporating diabetes-specific messaging. Arrows between levels indicate bidirectional influences and coordination points. Colour coding indicates evidence strength: green (strong evidence), yellow (moderate evidence), orange (limited but promising evidence). Implementation considerations and barriers are noted for each level.*

Sources: Authors illustrations adapted from references.[37,38,39,40,41]

7. CONCLUSION

Lifestyle-related determinants, particularly e-cigarette use, alcohol consumption, and recreational drug exposure, represent increasingly important contributors to DKA risk in contemporary populations. Emerging epidemiological evidence demonstrates clear associations between these behaviours and adverse diabetes outcomes, mediated through complex biological mechanisms and behavioural pathways. Adolescents and young adults face particular vulnerability, necessitating age-appropriate screening, education, and intervention strategies.

Effective public health responses require multi-level approaches encompassing individual clinical care, health system transformation, and policy interventions. Routine substance use screening within diabetes care settings should become standard practice, supported by validated tools and clear referral pathways. Harm reduction frameworks adapted for diabetes populations offer pragmatic approaches for individuals unable or unwilling to achieve abstinence. Integration of substance use treatment with diabetes care, enhanced by emerging technologies such as continuous glucose monitoring and predictive analytics, holds promise for reducing DKA burden.

Future research priorities include prospective cohort studies elucidating dose-response relationships, mechanistic investigations of novel substances,

effectiveness trials of integrated care models, and policy evaluations assessing population-level impacts of regulatory interventions. As substance use patterns continue evolving, ongoing surveillance and adaptive public health strategies will remain essential for protecting individuals with diabetes from preventable acute complications.

Declarations

Conflict of Interest

The authors declare no conflict of interest.

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