

WEIGHT REBOUND AFTER GLP-1 DISCONTINUATION IN INDIA: A HIGH-RISK
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ABSTRACT

Background: Glucagon-like peptide-1 receptor agonists (GLP-1 RAs) and dual GLP-1/GIP agonists have transformed obesity and diabetes care, but rapid weight regain and partial reversal of cardiometabolic gains follow discontinuation. In India, rebound occurs on a high-risk substrate of central adiposity, thin-fat and sarcopenic-obesity phenotypes and dense metabolic obesity at lower BMI, so even modest off-drug weight or waist increases may accelerate glycaemic and vascular deterioration. **Methods:** This narrative review integrates randomised trials and real-world cohorts of GLP-1/GLP-1–GIP tapering or withdrawal, metabolic/bariatric surgery data, and Indian epidemiological, body-composition and pharmacogenomic studies. We searched major databases to March 2026 for studies on incretin discontinuation, weight rebound, Indian obesity phenotypes, sarcopenic and abdominal obesity and incretin-pathway variants (TCF7L2, ADIPOQ, ARRB1), and reviewed National Family Health Survey-5 (NFHS-5) and Indian Council of Medical Research–India Diabetes Study (ICMR–INDIAB) datasets. **Results:** Across STEP-1, SURPASS-1 and SURMOUNT-CN, approximately two-thirds of prior weight loss is regained within 6–12 months of withdrawal, with glycated haemoglobin (HbA1c) and weight drifting within weeks. Indian data show high prevalence of abdominal obesity and sarcopenic obesity in type 2 diabetes, and rare metabolically healthy obesity. Thin-fat and sarcopenic phenotypes, with TCF7L2/ADIPOQ/ARRB1-linked vulnerability, suggest fat regain and lean-mass loss may precipitate disproportionate relapse. Metabolic/bariatric surgery and incretin therapy are bidirectional partners: incretins optimise peri-operative risk and weight courses, while surgery provides durable support when drugs are tapered. **Conclusion:** In India, post-GLP-1 weight rebound should be managed as a high-risk cardiometabolic transition. A planned, phenotype-directed strategy of avoiding abrupt withdrawal, leveraging bariatric–incretin synergy, waist-first and HbA1c-first surveillance, and maintaining lean mass can compress relapse and sustain organ protection.

KEYWORDS: GLP-1/GIP agonists, Weight rebound, Obesity in India, Asian Indian phenotype, Sarcopenic obesity, Bariatric surgery.

INTRODUCTION

Glucagon-like peptide-1 receptor agonists (GLP-1RAs) have emerged as a pivotal therapeutic agent for the management of type 2 diabetes mellitus (T2DM) and obesity, demonstrating robust efficacy in glycaemic control, weight reduction, cardiovascular risk mitigation, and renal protection. GLP-1, an incretin hormone, plays a central role in glucose homeostasis and appetite regulation by enhancing glucose-dependent insulin secretion, improving insulin sensitivity, and delaying

gastric emptying.^[1] Alongside GLP-1, glucose-dependent insulintropic polypeptide (GIP) exerts complementary, tissue-specific metabolic effects; both incretins modulate central satiety pathways and stimulate insulin secretion from pancreatic β -cells, while exhibiting divergent glucagon dynamics, GIP being glucagonotropic during hypoglycaemia and GLP-1 glucagonostatic during hyperglycaemia. At the adipocyte level, GIP promotes lipogenesis, whereas GLP-1 indirectly facilitates lipolysis, collectively supporting adipocyte function,

reducing ectopic fat deposition, and enhancing adiponectin secretion. These integrated mechanisms underpin their role in maintaining metabolic homeostasis and mitigating cardiometabolic risk.^[2]

Weight regain following discontinuation of GLP-1RAs and dual GLP-1/GIP agonists is increasingly recognised as a biologically mediated consequence of withdrawal of sustained neurohormonal appetite and metabolic regulation.^[3-5] Notably, the clinical impact of this rebound is population-dependent.

Compared to other ethnicities, Asian Indians have a distinct susceptibility to develop T2DM and other obesity-related metabolic disorders at a lower BMI. The 'Asian Indian Phenotype', marked by high levels of abdominal fat, insulin resistance, and dyslipidaemia with low HDL cholesterol and high serum triglycerides even with normal BMI, is believed to be a primary factor underlying this heightened risk.^[6]

The rising prevalence of diabetes among South Asians is multifactorial, driven by both biological predisposition and lifestyle transitions. Increased visceral adiposity, insulin resistance, impaired β -cell function, and genetic susceptibility collectively contribute to elevated disease risk. Concurrently, rapid urbanization has led to reduced physical activity, higher consumption of energy-dense processed foods, and increased psychosocial stress, further exacerbating metabolic dysfunction.^[7] Together, these factors create a milieu in which weight regain may translate more rapidly into glycaemic deterioration and cardiovascular risk.

Central adiposity, rather than BMI alone, is the dominant determinant of metabolic risk in Indian populations. NFHS-5 data indicate a high burden of abdominal obesity ($\approx 40\%$ in women and $\approx 12\%$ in men), with peak prevalence among women aged 30-49 years and higher rates in urban setting.^[8] The South Asian "thin-fat" phenotype further accentuates this risk, characterised by increased visceral fat and central adiposity despite relatively low BMI,^[9] and associated with insulin resistance, dyslipidaemia, and heightened cardiovascular risk.^[10] Genetic susceptibility, including polymorphisms in TCF7L2 and lipoprotein lipase, further contributes to this predisposition.^[11] In addition, compared with white European populations, South Asians exhibit higher body fat percentage, greater hepatic fat accumulation, larger subcutaneous abdominal adipocytes, and reduced skeletal muscle mass, reflecting a limited capacity for safe subcutaneous fat storage and a propensity for ectopic fat deposition.^[12]

Globally, the prevalence of overweight and obesity has risen markedly, with adults having BMI ≥ 25 kg/m² increasing from 28.8% to 43% in men and from 29.8% to 44% in women between 1980 and 2022. In India, prevalence has escalated from 6.2% in 1990 to 29.5% in 2022, paralleling a growing burden of metabolic

syndrome and cardiometabolic comorbidities, particularly in urban populations. In this context, weight regain following pharmacological weight loss occurs against a backdrop of pre-existing metabolic stress, potentially amplifying its clinical consequences.^[13]

LITERATURE STRATEGY

This narrative review synthesised evidence on weight regain after discontinuation of GLP-1 receptor agonists and dual GLP-1/GIP agonists, with a focus on Indian obesity and cardiometabolic phenotypes. We used a narrative approach to integrate heterogeneous data from randomised trials, real-world cohorts, epidemiological surveys and mechanistic studies relevant to Indian practice.

We searched PubMed and Google Scholar up to March 2026 using combinations of terms related to GLP-1/incretin therapy, discontinuation or withdrawal, weight rebound, South Asian/Indian obesity, thin-fat and sarcopenic obesity phenotypes, abdominal obesity, metabolically healthy obesity, and incretin-relevant genetic variants (e.g. TCF7L2, ADIPOQ, ARRB1). Reference lists of key trials and reviews, including STEP, SURPASS and SURMOUNT programmes, were also screened.

Eligible sources included human studies reporting weight and/or cardiometabolic outcomes after GLP-1/GLP-1–GIP dose reduction or cessation, and Indian studies describing obesity phenotypes, sarcopenia, abdominal obesity, metabolic health subtypes and incretin-pathway genetics. Priority was given to randomised trials, large observational cohorts and national Indian datasets, but informative smaller studies were retained. Non-English, animal studies and abstracts without adequate data were excluded.

Findings were grouped into four domains: (i) global patterns of weight and cardiometabolic relapse after GLP-1/GLP-1–GIP withdrawal; (ii) Indian obesity and sarcopenic phenotypes; (iii) genetic and pathophysiological vulnerability relevant to incretin biology; and (iv) India-adapted frameworks for tapering, monitoring and re-initiating therapy. Formal quality scoring or meta-analysis was not performed; instead, study design, size and applicability to Indian populations were considered qualitatively when formulating practice recommendations.

Sarcopenic Obesity in India: The Hidden Accelerator of Rebound

Sarcopenic obesity is a condition characterised by the simultaneous presence of excessive adiposity and low muscle mass and/or function that is estimated to affect 10–20 % of older adults.^[14] Emerging Indian data suggest a substantial burden of sarcopenia and sarcopenic obesity among adults with T2DM.^[15] Cross-sectional analyses in Indian T2DM cohorts demonstrate

high prevalence of low muscle mass and reduced muscle strength, even in middle-aged individuals.^[16]

Genetic and Phenotypic Vulnerability: Why Indian Bodies Are Primed for Post-GLP-1 Rebound

GLP-1 receptor agonists preferentially benefit hyperphagic and hedonic-eating phenotypes by engaging central appetite and reward circuits, thereby lowering energy intake, body weight, and glycaemia.^[17,18] A recent systematic review and meta-analysis confirms that GLP-1 RAs significantly reduce visceral adipose tissue (standardized mean difference -0.59) and hepatic fat (weighted mean difference -3.09) versus comparators, underscoring their depot-selective action on high-risk ectopic fat.^[19] These pharmacologic effects map closely onto South Asian “thin-fat” phenotypes, where imaging studies show higher liver and central fat at lower BMI than white Europeans and earlier onset of type 2 diabetes, implying that even modest post-cessation gains in waist or weight can translate into a disproportionate HbA1c rise.^[20–22] In India, this vulnerability is superimposed on a very high prevalence of abdominal obesity, as reported in the NFHS-5-based analyses report.^[8]

Genetically, Asian Indians carry a high burden of incretin-relevant risk variants. TCF7L2 polymorphisms, particularly rs7903146—are strongly associated with type 2 diabetes, impaired β -cell function, and adverse post-prandial lipid–glycaemic profiles, with Indian data linking the risk allele to higher glycaemia, higher HOMA-IR, lower adiponectin, and increased TCF7L2 expression in visceral adipose tissue that correlates with post-prandial triglycerides and glucose.^[23] Additional Indian studies implicate ADIPOQ variants in type 2 diabetes, obesity, and hypoadiponectinaemia, reinforcing a background of adipose dysfunction and low adiponectin in South Indians.^[9] Emerging pharmacogenomic work further suggests that β -arrestin-1-dependent GLP-1 receptor trafficking and biased signaling (ARRB1 and related loci) contribute to interindividual heterogeneity in GLP-1RA response, providing a plausible axis through which some Indian patients may experience particularly rapid weight and waist rebound once therapy is withdrawn.^[24] Collectively, the convergence of thin-fat and sarcopenic phenotypes, high abdominal-obesity prevalence, and TCF7L2/ADIPOQ/ARRB1-linked vulnerability supports an India-specific pharmacogenomics agenda: prospective discontinuation cohorts stratified by incretin-pathway genotypes, tracking depot-specific relapse (waist, VAT, liver fat) alongside HbA1c and lean mass, and using lower, India-adapted thresholds, such as a 0.5–1.0 percent HbA1c rise from nadir or re-crossing NFHS-5 waist cut-offs—to trigger timely re-initiation of GLP-1-based therapy.^[8,21]

Large, randomised trials and real-world studies demonstrate that GLP-1 receptor agonists produce consistent mean weight reduction at the population level,

accompanied by marked interindividual variability in treatment response. Despite established efficacy, approximately 10% of patients do not achieve clinically meaningful weight loss (<5%), indicating heterogeneity in pharmacodynamic and behavioural response. Evidence from real-world cohorts aligns with trial findings, while also indicating that treatment persistence and adherence significantly influence observed effectiveness, contributing to lower weight loss outcomes compared with controlled clinical settings.^[25]

Across multiple analyses, early weight loss response is the strongest predictor of long-term success with GLP-1-based therapies. Post-hoc analyses from STEP and SURMOUNT trials show that early non-responders are less likely to reach clinically meaningful outcomes unless therapy is prolonged.^[26,27] Additional predictors or variables include geriatric age group, female sex, absence of dyslipidaemia, baseline body mass index, sedentary lifestyle, while emerging data suggest a role for genetic variability in GLP1R and GIPR locus in explaining response heterogeneity.^[28,29] Together, these data support a chronic disease management and early-assessment algorithm, where response should be reassessed to guide continuation, intensification, or therapeutic switching.

How many obese Indians have comorbidities?

ICMR–INDIAB data show dense clustering of cardiometabolic abnormalities in Indian adults, with diabetes 11.4%, hypertension 35.5%, generalized obesity 28.6%, abdominal obesity 39.5%, and dyslipidaemia 81.2% in the same national.^[30] In the ICMR–INDIAB-23 subtype analysis (113,043 adults), Metabolically Healthy Obesity (MHO) is only 1.8%, whereas MOO and MONO account for 28.3% and 43.3%, respectively, confirming that most Indians with obesity are metabolically unhealthy.^[6] NFHS-5 further shows waist-defined abdominal obesity with higher prevalence in urban, wealthier population strata and strong links to hypertension and other risk factors.^[8,31] Collectively, these datasets show that “metabolically healthy” obesity is biologically rare in India and that obesity should be treated as a high-probability marker of cardiometabolic disease rather than a benign anthropometric variant.

Why is rebound clinically sharper in comorbid Indian phenotypes after GLP-1/GLP-1–GIP withdrawal

Global discontinuation datasets consistently show that stopping incretin-based anti-obesity therapy is followed by early and clinically meaningful reversal of weight and cardiometabolic gains, and these drifts are likely to be more hazardous in obese Indians with established comorbidities. In STEP-1, participants regained roughly two-thirds of prior weight loss within 1 year of stopping once-weekly semaglutide 2.4 mg, with parallel deterioration of blood pressure, lipids, and glycaemic indices.^[5] In SURPASS-1, even a prespecified 4-week off-drug window was sufficient to detect upward trajectories in HbA1c and body weight after tirzepatide

cessation^[32], and real-world follow-up of SURMOUNT-CN documented progressive weight and waist regain over 26 weeks post-cessation despite residual benefit versus baseline.^[33] These global patterns intersect with the South-Asian “thin-fat” phenotype—greater visceral and hepatic fat and lower BMI thresholds for diabetes—which is well characterised and not fully explained by overall adiposity alone.^[21,22] In India, NFHS-5 data,^[8] together with ICMR–INDIAB-23 classification demonstrating that metabolically obese obese (MOO) individuals have very high odds of type 2 diabetes (OR 12.89) and coronary artery disease (OR 1.92),^[6] indicate that even modest off-drug increases in weight or waist are likely to precipitate earlier HbA1c rebound, faster re-emergence of dyslipidaemia, and higher short-term vascular risk in comorbid patients than in those without cardiometabolic disease.

India-adapted monitoring and escalation for high-risk discontinuation

Given this phenotype- and comorbidity-linked vulnerability, an India-adapted framework for GLP-1/GLP-1–GIP withdrawal should treat centrally obese individuals with T2DM, hypertension, or dyslipidaemia as a priority high-risk group, with tighter surveillance and lower thresholds for maintenance or re-initiation. Year-1 off-therapy surveillance can reasonably include 12–16-weekly assessments of HbA1c, waist circumference, and a simple

body-composition proxy (e.g., BIA-estimated fat mass), with 8–12-weekly reviews in those with diabetes, vascular disease, or high baseline visceral adiposity—timed to capture the early 4-week drifts seen in SURPASS-1 and the 6–12-month reversals seen in STEP-1.^[5,32] Action thresholds for re-escalation or re-initiation of pharmacotherapy could include an HbA1c rise of ≥ 0.5 –1.0 percentage points from on-treatment nadir or breach of individualized glycaemic targets, and/or re-crossing NFHS-5 sex-specific abdominal-obesity cut-offs or a ≥ 5 cm increase in waist from end-of-treatment values, using waist as a practical surrogate of visceral-fat relapse in India.^[6,8] Given the high sarcopenia and sarcopenic obesity prevalence rates, routine muscle-function screening (e.g., hand-grip strength, simple performance tests) alongside BIA can help detect preferential fat-over-lean regain and trigger resistance-training-centred, protein-forward countermeasures.^[15,34] In combination, this pragmatic grid operationalizes global discontinuation signals into an Indian, waist-first, comorbidity-focused algorithm in which obese individuals with T2DM or cardiometabolic disease are maintained longer on therapy, monitored more frequently after cessation, and prioritized for early re-initiation to compress the window of glycaemic and cardiometabolic relapse. An India-adapted, structured strategy to minimize weight regain following GLP-1 receptor agonist discontinuation is summarized in Table 1.

Table 1: A planned Indian programme to prevent weight regain after stopping.

Strategy	Significance and rationale	Core components	Monitoring and follow-up
Awareness of indications and duration	Unsupervised and social-media-driven GLP-1 RA use risks abrupt self-discontinuation and rapid regain, which is problematic in India given high central obesity and cardiometabolic burden ^[35]	Brief structured counselling at initiation on evidence-based obesity/T2DM indications, expected treatment duration, risk of rebound (STEP-1, SURPASS-1), and the need for planned taper. ^[5,32] Provide a simple written “stop plan” (taper schedule, monitoring dates, re-start trigger)	Document indication and phenotype at initiation; reassess benefit–risk at least annually; and perform a planned clinical review before GLP-1 dose reduction or discontinuation. ^[36–38]
Medication taper strategy	After stopping semaglutide or tirzepatide, early HbA1c and weight rebound is seen, with $\approx$ two-thirds of weight loss regained at 1 year in STEP-1. ^[5] In South-Asian thin-fat phenotypes, similar regain may cross glycaemic and vascular thresholds earlier. ^[39]	Prefer stepwise taper (e.g., halving maintenance dose every 4–8 weeks) over abrupt stop. ^[36] Optimise background therapy (metformin, SGLT2 inhibitor, statin, antihypertensives) in line with guideline-based risk management; consider long-term low-dose maintenance in very high-risk patients. ^[37,40]	Measure weight, BMI, waist and HbA1c in intervals as described above ^[8,16]
Nutrition (protein-first, micronutrient)	GLP-1-induced weight loss	Recommend a protein-forward diet	Focused dietary review and micronutrient panel at baseline

rient-aware)	includes lean-mass loss; with low protein and micronutrient intake this may favour fat-predominant regain. ^[41] Micronutrient deficiencies (vitamin B12, iron, vitamin D) are common in Indian obesity and diabetes and can limit adherence to physical-activity plans ^[42]	(~1.0–1.2 g/kg/day) assess vitamin B12, vitamin D, and iron at baseline and periodically, with supplementation as indicated. ^[43–46]	and at least annually (6-monthly in high-risk patients). Short in-person or tele-nutrition follow-ups during taper and the first 6–12 months off treatment to reinforce adherence and adjust plans. ^[5,47,48]
Exercise and physiotherapy (resistance-first)	Sarcopenia and sarcopenic obesity predict functional decline and fat-predominant regain after weight loss. ^[15] Preserving lean mass supports long-term weight and glycaemic control. ^[49]	Prescription of resistance-based training (2–3 sessions/week) combined with ≥150 minutes/week of moderate-intensity aerobic activity is recommended, tailored to comorbidities and access. ^[50]	At follow-up, assess activity and simple functional measures (handgrip strength, chair rise, gait speed), with physiotherapy referral as needed and progressive resistance training to preserve lean mass. ^[51]
Behavioural and psychological management	Mood disorders, disordered eating and low motivation are strong predictors of weight regain after weight-loss interventions but are often under-recognised in obesity care. ^[52]	Screen for depression and disordered eating; deliver brief MI or CBT-based behavioural support and engage family support, where appropriate. ^[53,54]	Use brief validated screens (e.g., PHQ-9, binge-eating screeners) at baseline and key transitions; refer if scores are elevated and set 1–2 specific behavioural goals per visit, reviewed periodically. ^[55–57]

Abbreviations: BMI, body mass index; CBT, cognitive behavioral therapy; GIP, glucose-dependent insulinotropic polypeptide; GLP-1, glucagon-like peptide-1; GLP-1 RA, glucagon-like peptide-1 receptor agonist; HbA1c, glycated haemoglobin; MI, motivational interviewing; PHQ-9, Patient Health Questionnaire-9; SGLT2, sodium-glucose cotransporter-2; T2DM, type 2 diabetes mellitus T2DM.

Metabolic Surgery and Incretin Therapy: A Bidirectional Partnership

Metabolic–bariatric surgery and incretin therapy now function synergistically rather than competitively in advanced obesity care, including in India. Vertical sleeve gastrectomy, Roux-en-Y gastric bypass, one-anastomosis gastric bypass, and biliopancreatic diversion with duodenal switch combine gastric restriction with hormone and bile-acid shifts that improve appetite control and glycaemia.^[58] Randomised and long-term data show that sleeve and Roux-en-Y provide large, durable weight loss and metabolic improvement, with contemporary trials such as By-Band-Sleeve and SM-BOSS reporting broadly comparable 10-year weight outcomes and reflux advantages for Roux-en-Y.^[59,60] One-anastomosis gastric bypass has emerging ≥5-year evidence with ~75–77% excess-weight loss and high

diabetes-remission rates, including Indian randomised data supporting tailored limb lengths without loss of glycaemic benefit.^[61,62] At a population scale, a one-stage meta-analysis of 174,772 individuals with obesity found that metabolic–bariatric surgery was associated with a ~49% reduction in all-cause mortality and a ~6.1-year gain in life expectancy versus usual care.^[63] In parallel, endogenous GLP-1 secretion rises after Roux-en-Y and sleeve and correlates with early postoperative success, while GLP-1 receptor agonists and dual GLP-1/GIP agonists provide a pharmacologic “basal–bolus” incretin signal that can rescue insufficient weight loss or weight regain after surgery.^[64] Systematic reviews and meta-analyses in post-bariatric cohorts show that GLP-1 RAs achieve additional weight reductions of about 7–8 kg from pretreatment baselines and ~4–5 kg versus placebo, with acceptable tolerability and improvements in glycaemic and lipid parameters.^[65–67] Observational US network data indicate that roughly one in six patients initiate GLP-1 pharmacotherapy within several years of sleeve or Roux-en-Y, underlining the real-world convergence of surgery and incretin agents in long-term weight control.^[68]

Bariatric Specialists as Long-Term Stewards of Obesity Care in India

In India's comorbidity-dense, waist-first obesity landscape, bariatric physicians are uniquely placed to integrate surgery and incretin drugs across the full care continuum—from pre-operative optimisation to post-drug or post-surgery relapse management. National ICMR-INDIAB data highlight dense clustering of comorbidities in adults, and show that metabolically healthy obesity is rare, with MOO and MONO subtypes dominating; these patterns mean that truly “low-risk” patients at the point of GLP-1 taper are uncommon.^[6] Furthermore, NFHS-5 analysis reports support waist-based triage and especially tight guardrails for tapering in women.^[8] Against this backdrop, bariatric physicians can adapt post-bariatric discipline to GLP-1/dual GLP-1–GIP discontinuation by: (i) starting with phenotype-anchored risk stratification (waist, sarcopenia/sarcopenic obesity, cardiometabolic load); (ii) favouring taper over abrupt cessation, coupled with a protein-forward, resistance-training-centred behavioural bundle; and (iii) implementing bariatric-style surveillance in Year-1 off therapy—12–16-weekly checks of HbA1c, waist circumference, a body-composition proxy, and key micronutrients (B12, iron, vitamin D), mirroring guidance from state-of-the-art bariatric reviews.^[65] When pre-defined action thresholds are crossed, such as an HbA1c rise as described above, bariatric physicians can re-initiate incretin therapy, step down to more affordable agents, or escalate to metabolic surgery, using the survival and durability advantages of surgery,^[63] together with GLP-1-based medical rescue,^[65,66] to personalise long-term outcomes for Indian patients.

How Indian food patterns drive weight gain and relapse risk

India's rapid nutrition transition, driven by economic growth, has increased consumption of processed foods and refined grains. Diets remain protein-deficient and high in low-quality carbohydrates, saturated fats, and fried foods, with inadequate intake of fruits, vegetables, and fibre. This macronutrient imbalance, combined with sedentary behaviour, is associated with increased risk of incident type 2 diabetes, prediabetes, and generalized and central obesity.^[69–71]

Rising obesity among school-aged adolescents is an emerging public health concern in India. The Obesity Foundation of India attributes this to television advertising promoting nutrient-poor diets, alongside the widespread urban and rural availability of processed foods, reinforcing unhealthy, obesogenic dietary patterns.^[71–73] Promoting a healthy diet and sleep hygiene is crucial to reducing obesity risks.^[74] Reducing low-quality carbohydrates and increasing plant proteins and healthy fats improves cardiometabolic health outcomes.^[70] Variants of vegetarian dietary patterns, including lacto-vegetarian and lacto-ovo vegetarian diets,

have been associated with a $\geq 30\%$ reduction in diabetes risk.^[75]

Furthermore, isocaloric substitution of 5% energy from carbohydrates with protein, especially plant or dairy sources, reduces risk of newly diagnosed T2DM and prediabetes.^[70] Comprehensive health education across ages is essential, including school-based curricula and public campaigns. Policies restricting junk food near schools, alongside strengthening counselling competencies and dietary interventions, promoting fruits, vegetables, and reducing saturated fats, support sustained behaviour change.^[71,76]

Research priorities for India

There is an urgent need for state-wise discontinuation registries that track weight, waist circumference, body-fat indices and HbA1c after GLP-1/GLP-1–GIP tapering or withdrawal, aligned with existing state-level metabolic and obesity surveillance in India. Such registries would help characterise real-world relapse trajectories across diverse Indian phenotypes and healthcare settings.^[77–80]

Randomised trials should test sarcopenic-obesity-focused taper bundles that combine higher-protein or HMB-supplemented nutrition with structured resistance training, building on evidence that protein-forward diets and exercise improve body composition and metabolic risk in Indian adults. Pragmatic diet trials using region- and state-specific plate models, informed by recent national dietary profiling and existing plate-model interventions, could evaluate scalable nutrition strategies for post-drug weight maintenance.^[70,81]

Finally, pharmacogenomic studies, such as TCF7L2-stratified and GLP-1 pathway variant-stratified rebound curves, are needed to link incretin-relevant genetic vulnerability to depot-specific relapse and refine thresholds for re-initiating therapy in Indian patients. This agenda would complement ongoing national efforts on metabolic obesity and provide an empirical basis for India-adapted discontinuation algorithms.^[16,23,82–84]

CONCLUSION

India's convergence of central adiposity, thin-fat and sarcopenic-obesity phenotypes means that stopping GLP-1/GLP-1–GIP therapy is a high-risk cardiometabolic transition, not a cosmetic endpoint. A rigorously phenotype-directed discontinuation strategy—eschewing abrupt withdrawal in centrally obese, comorbid patients and explicitly harnessing bariatric–incretin synergy where indicated—can materially blunt neurohormonal and metabolic rebound. Operationalizing this through waist-first and HbA1c-first surveillance, predefined thresholds for re-initiation, and deliberate protection of lean mass via protein-forward nutrition and resistance-dominant exercise transforms incretin cessation from passive de-escalation into an active, protocolized intervention. In doing so, Indian clinicians

can compress the window of relapse, preserve organ protection, and recast GLP-1 discontinuation as a planned, evidence-aligned phase of long-term cardiometabolic disease modification rather than an unstructured off-drug drift.

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AUTHOR CONTRIBUTIONS

Dr. Depti Bellani: Conceptualization, Methodology, Investigation, Data curation, Writing – original draft.

Dr. Shivani Mishra: Investigation, Data curation, Writing – original draft.

Dr. Srishti Priya Maldahiar: Investigation, Writing – original draft.

Dr. Anish Desai: Validation, Writing – review & editing, Supervision.

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ABBREVIATIONS

Abbreviation	Full Form
GLP-1 RA	Glucagon-Like Peptide-1 Receptor Agonist
GLP-1	Glucagon-Like Peptide-1
GIP	Glucose-Dependent Insulinotropic Polypeptide
GLP-1/GIP	Glucagon-Like Peptide-1 / Glucose-Dependent Insulinotropic Polypeptide
BMI	Body Mass Index
T2DM	Type 2 Diabetes Mellitus T2DM
HbA1c	Glycated Hemoglobin
NFHS	National Family Health Survey
ICMR	Indian Council of Medical Research
ICMR–INDIAB	Indian Council of Medical Research–India Diabetes Study
MHO	Metabolically Healthy Obesity
MOO	Metabolically Obese Obesity
MONO	Metabolically Obese Non-Obese
OR	Odds Ratio
VAT	Visceral Adipose Tissue
BIA	Bioelectrical Impedance Analysis
HOMA-IR	Homeostatic Model Assessment of Insulin Resistance
HDL	High-Density Lipoprotein
CBT	Cognitive Behavioral Therapy
MI	Motivational Interviewing
PHQ-9	Patient Health Questionnaire-9
BEDS-7	Binge Eating Disorder Screener-7
SGLT2	Sodium-Glucose Cotransporter-2
B12	Vitamin B12
HMB	β -Hydroxy β -Methylbutyrate
GLP1R	Glucagon-Like Peptide-1 Receptor
GIPR	Glucose-Dependent Insulinotropic Polypeptide Receptor
TCF7L2	Transcription Factor 7-Like 2
ADIPOQ	Adiponectin Gene
ARRB1	Beta-Arrestin 1 Gene

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