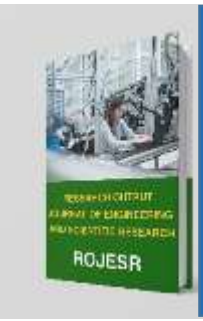

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Smart Nanomaterials for Glucose-Responsive Drug Delivery Systems in Diabetic Patients with Obesity

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ABSTRACT

Glucose-responsive drug delivery systems aim to emulate pancreatic feedback by sensing hyperglycemia and releasing therapeutics proportionally, thereby improving time in range while mitigating hypoglycemia. In people with diabetes and coexisting obesity, the value proposition is amplified by altered pharmacokinetics in expanded adipose depots, variable subcutaneous perfusion, chronic low-grade inflammation, and behavioral burdens associated with frequent injections and complex dosing. Smart nanomaterials like lipid, polymer, hydrogel, and hybrid architectures endowed with enzymatic, chemical, or physical glucose-sensing motifs offer the spatial and temporal control required for adaptive delivery of insulin and adjunct agents. This review synthesizes design principles for responsive nanoplatforms; compares enzymatic (glucose oxidase/dehydrogenase), boronic acid–diol, and lectin strategies; examines device-route integration via microneedles, subcutaneous depots, oral and pulmonary formulations; and discusses peroxide management, antifouling strategies, and closed-loop–like kinetics. We evaluate safety, manufacturability, and regulatory pathways tailored to chronic metabolic indications and obesity-related physiology, and propose clinical trial frameworks that pair continuous glucose monitoring with imaging and mechanistic biomarkers. Finally, we highlight human factors such as usability, adherence, equity, and compatibility with incretins and SGLT2 inhibitors that will determine real-world impact. Smart nanomaterials can decouple efficacy from risk by matching dose to demand, offering patient-centered control that complements contemporary pharmacotherapy.

Keywords: glucose-responsive; smart nanomaterials; insulin delivery; microneedles; boronic acid; glucose oxidase; obesity; type 2 diabetes; closed-loop; stimuli-responsive

1. INTRODUCTION

Diabetes management hinges on aligning insulin supply with dynamic glucose demand. Despite advances in analog insulins, incretin-based agents, and algorithmic pump therapy, many people struggle to maintain glucose within target ranges while avoiding hypoglycemia, particularly those living with obesity[1–3]. Obesity introduces physiologic and behavioral challenges that blunt therapeutic precision. Expanded and heterogeneous subcutaneous adipose tissue slows and variably absorbs injected drugs; chronic, low-grade inflammation alters insulin signaling and vascular reactivity; and comorbidities such as fatty liver disease and sleep-disordered breathing complicate pharmacology and glycemic rhythms[4, 5]. Meanwhile, the daily burden of injections, dose calculations, and hypoglycemia prevention discourages adherence [6]. These realities motivate delivery systems that sense glucose and autonomously adapt output, shifting decision-making from the patient to the material.

Smart nanomaterials offer such autonomy by coupling a sensor to an actuator within nanoscale architectures that can be formulated as injectable depots, microneedle patches, oral capsules, or inhalable aerosols[7]. The sensor detects hyperglycemia through enzymatic reactions that generate pH or redox changes, through reversible chemical binding to glucose diols, or via lectins that modulate crosslinks when glucose competes for binding. The actuator translates these cues into increased permeability, bond cleavage, or cargo desorption, thereby releasing insulin or complementary agents such as amylin analogs, GLP-1 receptor agonists, or hepatoselective sensitizers[7]. The same constructs can throttle release as glucose normalizes, creating negative

feedback akin to islet physiology. Nanoscale design is central: high surface area accelerates mass transfer; responsive linkers shorten response time; and anti-fouling coronas preserve sensitivity in complex biological fluids[8].

The obesity context heightens engineering demands. Thickened subcutaneous layers and variable dermal perfusion can delay access to interstitial glucose and drug egress. Inflammatory milieus increase reactive oxygen species and protease activity, potentially interfering with sensors but also providing orthogonal triggers for context-aware dosing[5, 9–11]. Skin and tissue mechanics differ, affecting microneedle insertion force and depot dispersion. Moreover, pharmacotherapy for obesity GLP-1/GIP receptor agonists, sometimes at high doses, changes gastric emptying and appetite, causing meal-timing variability that stresses prandial control. Smart systems must therefore be robust to fluctuating glucose trajectories, temperature, motion, and local tissue chemistry, while maintaining biocompatibility over months of repeated use.

Clinical relevance depends on kinetics that fit daily life. Postprandial spikes require minute-scale responsiveness and sufficient capacity to blunt excursions without overshoot[12]. Overnight protection calls for stable basal output with damped sensitivity to avoid nocturnal hypoglycemia. Materials must remain responsive despite protein adsorption, fibrotic encapsulation, or enzyme drift. For microneedle patches, thin diffusion barriers, high-density arrays, and breathable backings support rapid equilibration with interstitial glucose; for subcutaneous depots, core–shell geometries and percolating pore networks shorten lag times[13]. Oral and pulmonary routes face their own barriers, including gastric acidity and alveolar macrophage clearance, but they can provide a rapid onset with high acceptance if engineered correctly.

Safety and manufacturability frame translation. Enzymatic systems that rely on glucose oxidase generate hydrogen peroxide and acid; peroxide scavengers and buffering domains are required to protect tissue[13]. Boronic acid chemistries must be tuned to physiologic pH and avoid nonspecific diol binding; lectin-based platforms must mitigate immunogenicity and leakage. All carriers, like lipid, polymer, or hybrid, must minimize complement activation and anti-polymer antibodies, remain stable during shipping and storage, and integrate with user-friendly devices[14]. Regulatory pathways will judge not only glycemic endpoints but also the durability of responsiveness, biostability, and compatibility with concomitant medications and sensors, such as continuous glucose monitors[14].

This review structures the field around six themes: design blueprints for glucose-responsive nanomaterials; enzymatic transduction and peroxide management; boronic acid and lectin chemistries; device-route integration for people with obesity; safety, manufacturing, and regulation; and clinical translation with human factors. Across sections, we emphasize how smart nanomaterials can complement, rather than replace, pharmacology and digital tools, helping patients achieve tighter control with less cognitive load and fewer adverse events. The central proposition is that material intelligence encoded in nanoscale architecture can bring closed-loop logic into simple, unobtrusive formats suited to the realities of living with diabetes and obesity.

2. Design Blueprints for Glucose-Responsive Smart Nanomaterials

At the heart of glucose-responsive delivery is a coupling between a sensing module and a release module within a stable, biocompatible chassis[15]. Nanoparticle and nano–micro hybrid systems employ three archetypes. In enzyme-driven designs, glucose oxidase or dehydrogenase converts glucose into chemical gradients like pH shifts, redox flux that actuate pH-labile or oxidant-cleavable linkers. In affinity-driven designs, phenylboronic acid moieties reversibly bind glucose diols; rising glucose disrupts crosslinks or displaces polymer–polymer interactions, increasing mesh permeability[15]. In lectin-mediated designs, glucose competes with polymer-bound saccharides for lectin binding, loosening networks and releasing cargo. All three can be embedded in lipid, polymeric, or hydrogel matrices[15].

Responsive kinetics are sculpted by geometry. Nanoscale carriers with porous shells minimize diffusion distances and shorten lag, while core–shell architectures segregate sensor and cargo to reduce autocatalytic degradation[16]. Layer-by-layer assemblies allow orthogonal triggers: an outer boronic layer for rapid prandial response and an inner acetal-linked insulin depot for basal support. Degradable crosslinkers such as thioketals, disulfides, orthoesters, add redox and pH sensitivity that aligns with inflamed or hypoxic microenvironments often seen in obesity. Antifouling coronas formed by zwitterionic polymers or polysarcosine preserve responsiveness by resisting protein adsorption and cellular encapsulation[17].

Signal fidelity demands noise control. Temperature and ionic strength modulate boronate equilibria; pH buffering domains smooth enzyme-induced acidification; catalase or peroxidase mimics quench hydrogen peroxide to protect both tissues and enzymes[18]. Glucose selectivity is improved by tuning boronate pKa toward physiologic values and by placing boronic groups in sterically constrained pockets that discriminate against other diols. For lectin-based systems, immobilization and shielding reduce immunogenicity and prevent protein leaching[18].

Capacity and safety must be balanced. High loading supports prandial needs, but risks burst release; crystallinity control in lipid cores and tortuous diffusion paths in polymer matrices suppress initial bursts without slowing responsiveness excessively[19]. Mechanical properties matter for patches and depots: soft, conformal gels reduce foreign-body response; resilient matrices resist fracture under motion. Integration with sensors either

embedded optical reporters that change fluorescence upon linker cleavage or impedance-based cues provides a window into in situ performance, enabling adaptive dosing in clinical studies[19].

Finally, manufacturability defines viability. Microfluidic mixing yields tight size distributions for lipid nanoparticles; controlled radical polymerization and click chemistry furnish reproducible functional densities for boronate and redox groups[20]. Stability programs must assess enzyme activity, linker integrity, and antifouling performance under thermal and mechanical stress. Thoughtful blueprinting at this level positions smart nanomaterials to deliver the speed, specificity, and durability that daily glycemic control in obesity demands[20].

3. Enzymatic Transduction: Glucose Oxidase/Dehydrogenase Systems and Peroxide Management

Enzymatic platforms translate glucose concentration into actionable chemical signals with high gain. Glucose oxidase (GOx) catalyzes the oxidation of β-D-glucose to gluconic acid, consuming oxygen and generating hydrogen peroxide. The resulting acidification and oxidative flux provide dual handles to trigger release from pH-labile linkers and oxidant-cleavable bonds[21]. However, peroxide is cytotoxic and can inactivate GOx over time, while oxygen dependence introduces drift in hypoxic or poorly perfused tissues, a common scenario in hypertrophic adipose.

Engineering strategies mitigate these liabilities. Co-encapsulation of catalase decomposes peroxide into water and oxygen, simultaneously detoxifying and recycling oxygen to sustain GOx activity[22]. Packaging enzymes within nanodomains that restrict diffusion of large proteins but allow glucose transport protects activity from proteases while keeping the response fast. Buffering microenvironments using histidine-rich polymers or bicarbonate reservoirs limit local pH excursions that might cause inflammation or enzyme denaturation. Oxygen-generating additives such as calcium peroxide or perfluorocarbon emulsions can support responsiveness under low oxygen tension but must be carefully dosed to avoid gas formation or oxidative stress[22].

Glucose dehydrogenase (GDH) offers an alternative transduction route independent of oxygen, using NAD(P)+ or PQQ cofactors[23]. In materials, redox mediators immobilized in the matrix shuttle electrons from GDH to generate localized redox changes that actuate redox-cleavable linkers. GDH systems circumvent peroxide toxicity but introduce cofactor stability challenges; tethered mediators and cofactor-regenerating chemistries stabilize function for weeks.

Kinetics and set points are tuned by enzyme loading, domain size, and mass transport. High enzyme densities accelerate response but may exaggerate local acidity; smaller domains shorten diffusion distances, but risk burst release[23]. For prandial control, thin responsive layers surrounding insulin-rich cores yield minute-scale onset; for basal coverage, thicker, buffered shells damp sensitivity and extend duration. In microneedle patches, distributing micro-reactors along needle shafts increases surface area exposed to interstitial fluid, reducing lag and improving reproducibility across variable dermal perfusion in obesity[23].

Reliability across days to weeks requires protecting enzymes from denaturation and immune recognition. Covalent immobilization, glycosylation-mimetic coatings, and encapsulation in hydrophilic cages extend activity. Antifouling outer layers limit protein adsorption that could throttle glucose access[24]. Importantly, peroxide and acid byproducts should remain below thresholds that irritate skin or provoke fibrosis; preclinical telemetry of local temperature and impedance can detect early tissue responses.

In sum, enzymatic transduction delivers robust, tunable glucose sensitivity but demands careful peroxide management, oxygen balance, and enzyme stabilization needs that smart nanomaterials can satisfy through compartmentalization, scavenging, and mass-transfer engineering.

4. Affinity Chemistries: Phenylboronic Acid, Lectin Systems, and Hybrid Approaches

Affinity-based systems sense glucose via reversible binding rather than catalysis, avoiding peroxide generation and oxygen dependence. Phenylboronic acid (PBA) moieties form cyclic esters with cis-diols on glucose; increased glucose competitively disrupts crosslinks or polymer–polymer associations, opening meshes and releasing cargo[25]. The equilibrium is pH sensitive because boronate anions are the binding-competent species; thus, tuning pKa into the physiological range with electron-withdrawing substituents or ortho-diol intramolecular stabilization improves responsiveness at neutral pH. Embedding PBA in hydrophilic, antifouling backbones minimizes nonspecific interactions with serum glycoproteins and other saccharides[25].

Geometry again controls behavior. Dense PBA crosslinking creates steep, switch-like responses but risks hysteresis; sparse, well-distributed motifs yield linear release with broader dynamic ranges suited to basal regulation. Multivalent presentation enhances apparent affinity without overshooting specificity. Hybrid systems pair PBA sensing with pH- or redox-labile linkers, transforming modest affinity shifts into decisive permeability changes. For prandial spikes, thin PBA-rich skins over insulin cores provide rapid, glucose-titrated bursts; for basal coverage, interpenetrating networks with lower PBA density smooth output[25].

Lectin-based designs use glucose-binding proteins such as concanavalin A (ConA) to crosslink saccharide-bearing polymers; glucose competes for binding, reducing crosslink density and releasing the drug[26]. While mechanistically elegant, lectins raise immunogenicity and stability concerns. Immobilization within inert cages, PEG-alternatives to shield epitopes, and covalent anchoring reduce leaching and immune exposure[26]. Even so, clinical translation likely favors PBA or hybrid systems unless lectin safety can be unequivocally established.

Selectivity and biocompatibility are the central challenges. PBA can interact with other diols; positioning motifs within steric pockets and using copolymer environments that prefer glucose size and hydration profiles improve discrimination[27]. Materials must avoid binding to glycosylated cell-surface proteins in a way that alters cell signaling. For patches, sweat and skin lipids complicate performance; hydrophilic overcoats stabilize hydration and reduce interference. For subcutaneous depots, antifouling shells resist protein corona formation that could mask PBA groups and shift set points[27].

Hybrid approaches combine enzymatic and affinity cues for redundancy and range. An outer PBA layer provides a rapid, low-amplitude response to modest excursions, while an inner GOx domain engages during larger spikes, delivering extra capacity with peroxide safely quenched[27]. Such layered intelligence better matches the varied glucose trajectories of patients with obesity, whose meals, gastric emptying, and activity patterns can produce diverse excursions. Properly tuned, affinity chemistries deliver silent, oxygen-independent sensing that complements enzymatic strength and broadens applicability across tissues and routes.

5. Route and Device Integration: Microneedles, Subcutaneous Depots, Oral and Pulmonary Options

Material intelligence must be paired with delivery formats that fit daily life and obesity-related physiology. Microneedle patches place responsive nanomaterials at the dermal–epidermal interface, where interstitial glucose mirrors blood with minimal lag[28]. Dissolving or swelling microneedles deposit nanoparticle-laden microgels that maintain intimate contact with interstitial fluid, enabling minute-scale response. Patch designs favor breathable backings, soft adhesives that accommodate skin folds, and needle geometries that overcome thicker stratum corneum in some individuals. Integrating optical reporters that brighten as release occurs creates an unobtrusive readout for dosing validation during trials[28].

Subcutaneous depots provide a larger capacity and longer duration. Core–shell particles suspended in shear-thinning vehicles form in situ depots after injection, with responsive shells throttling egress. In obesity, heterogeneity in adipose perfusion and lymphatic drainage can increase variability; positioning injections in regions with reliable blood flow and using ultrasound guidance during development can reduce noise[29]. Depot viscoelasticity should resist motion-induced deformation, and antifibrotic excipients can limit encapsulation that would otherwise slow glucose access.

Oral systems are attractive for acceptance but face gastric acidity, proteases, and mucus barriers. Enteric-coated capsules release nanoparticles in the intestine, where PBA-based sensing avoids peroxide toxicity. For insulin, lymphotropic lipid compositions bias uptake into lacteals, providing systemic exposure; materials must balance mucus penetration with sufficient epithelial residence to sense luminal glucose[30]. Because portal-first delivery could better mimic physiology, hybrid strategies that route a fraction to the portal vein via enterocyte transporters may be explored in conjunction with glucose-responsive release to limit hypoglycemia.

Pulmonary delivery offers rapid onset through a thin alveolar barrier. Aerosolizable microcarriers that deaggregate into responsive nanoparticles in the lung can tame prandial spikes without needles[30]. Safety focuses on alveolar macrophage load, surfactant interactions, and chronic inflammation risk; inert, biodegradable matrices and low excipient burdens are essential. For patients using CPAP for sleep apnea, compatibility with humidified airflow should be considered.

Device ecosystems shape adoption. Patches should not interfere with continuous glucose monitors worn on the same arm or abdomen; adhesives must tolerate sweat and exercise. Pens and autoinjectors need consistent extrusion of nanoparticle suspensions without clogging. For oral capsules, food-effect robustness improves adherence[31]. Across routes, cold-chain stability, simple instructions, and minimal maintenance encourage sustained use. In all cases, integration with digital tools, such as CGM dashboards, dose diaries, allows patients and clinicians to see material intelligence translate into lived stability[31].

6. Safety, Manufacturability, and Regulatory Pathways in the Context of Obesity

Chronic use in a prevalent condition demands exceptional safety and scalable manufacturing. Materials must limit complement activation, pseudoallergic reactions, and anti-polymer antibodies that can alter pharmacokinetics on re-dosing[32]. PEG substitutes such as zwitterionic polymers or polysarcosine reduce immunogenicity while preserving antifouling. For enzymatic systems, peroxide byproducts must be quenched locally below irritation thresholds; preclinical histology and telemetry at patch or depot sites should exclude fibrosis and nerve irritation[32]. Boronic acid motifs require toxicology tailored to off-target diol interactions, while lectins necessitate rigorous immunogenicity testing and containment of protein leaching.

Manufacturability hinges on reproducible nanoparticle assembly, linker functionalization, and enzyme loading. Microfluidic mixing and continuous nanoprecipitation deliver narrow size distributions; orthogonal click chemistries install responsive groups with high fidelity. Critical quality attributes include particle size/polydispersity, linker density, residual solvent, enzyme activity, peroxide scavenger capacity, and in vitro–in vivo correlation of responsiveness[33]. Stability programs must withstand temperature excursions common in real-world logistics, with protective excipients for enzyme integrity and antifouling coronas to maintain responsiveness after storage. Device compatibility testing, like needle/syringe shear, inhaler aerosolization, and capsule dissolution, prevents performance drift[33].

Regulators will judge not only A1c and time in range but also hypoglycemia incidence, nocturnal safety, and durability of responsiveness. Benchmarked comparators include rapid-acting insulin for prandial performance

and long-acting formulations for basal[34]. For combination products co-delivering insulin with incretins or amylin analogs, interaction studies establish an additive benefit without excessive nausea or hypoglycemia. Radiolabeled microdose studies or MRI-visible tracers can map biodistribution and confirm that sensing domains remain where intended[34]. Human factor evaluations must include individuals with high BMI, variable skin properties, and coexisting conditions such as neuropathy that influence device use.

Obesity-specific considerations include altered skin biomechanics, thicker subcutaneous layers, and pro-inflammatory tissue chemistry that may accelerate enzyme drift or shift boronate equilibria[35]. Designs should be validated across BMI strata, sexes, and skin tones to ensure equitable performance. Environmental and occupational safety plans address solvent use and nanoparticle handling at scale. Cost-of-goods matters in a chronic therapy; process intensification and solvent minimization lower barriers to access, while modular platforms that swap sensing motifs without reworking the chassis streamline lifecycle management[35].

With these safeguards and processes, smart nanomaterials can meet the long horizon of diabetes care, earning regulatory confidence and patient trust.

CONCLUSIONS

Glucose-responsive smart nanomaterials promise to shift diabetes care from fixed regimens to adaptive dosing that mirrors physiologic feedback. By embedding enzymatic, affinity, or hybrid sensing within responsive nanoscale architectures, these systems can deliver insulin and adjunct agents in proportion to glycemic demand, a capability especially valuable in people with obesity whose tissue mechanics and inflammatory milieu complicate conventional delivery. Engineering solutions like peroxide scavenging, antifouling coronas, layered geometries, and device–route integration have matured to provide minute-scale response, stable basal support, and robustness to real-world variability. Translation now rests on rigorous, CGM-centered trials that prove superior stability and safety, manufacturable platforms that maintain responsiveness over months of storage and use, and human factors that reduce burden while ensuring equitable performance. As companions to incretin therapies and lifestyle interventions, glucose-responsive nanomaterials can help patients spend more of life in range with fewer lows and less cognitive load, bringing material intelligence to the frontline of metabolic care.

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