Research Output Journal of Engineering and Scientific Research 5(1): 53-58, 2026

ROJESR Publications Online ISSN: 1115-9790

https://rojournals.org/roj-engineering-and-scientific-research/ Print ISSN: 1115-6155

https://doi.org/10.59298/ROJESR/2026/5.15358

Smart Nanocarriers for Co-Delivery of Anti-Obesity and Anticancer Agents

Kamanzi Ntakirutimana G.

School of Natural and Applied Sciences Kampala International University Uganda

ABSTRACT

Obesity and cancer intersect through shared inflammatory, endocrine, and metabolic circuits that amplify risk, accelerate progression, and blunt therapeutic response. Treating them as separate diseases leaves substantial biology unaddressed at the adipose–tumor interface. Smart nanocarriers engineered for co-delivery of anti-obesity and anticancer payloads offer a strategy to modulate host metabolism while simultaneously attacking malignant cells and their microenvironments. By tuning size, shape, and surface chemistry, and by embedding logic for stimuli responsiveness, active targeting, and imaging, multifunctional platforms can synchronize exposure of GLP-1 or AMPK-directed agents with chemotherapeutics, kinase inhibitors, immunomodulators, or nucleic acids, all within the pharmacokinetic and tissue constraints of high-BMI hosts. This review maps the rationale and design landscape for dual-action nanotherapy, from lipid and polymeric constructs to biomimetic and hybrid systems; details trigger chemistries and ligand choices that align with obesity-altered vasculature, extracellular matrix, and immune tone; and outlines dosing, safety, and translational principles that convert mechanistic synergy into clinical benefit. The central proposition is that metabolic correction and tumor control are not competing objectives but co-requisites that nanotechnology can deliver in a coordinated, patient-tailored manner.

Keywords: co-delivery; anti-obesity agents; obesity-associated cancer; smart nanocarriers; stimuli-responsive release

INTRODUCTION

Obesity reshapes organismal physiology in ways that directly promote carcinogenesis and resistance to therapy. Hypertrophic adipocytes develop hypoxia and endoplasmic-reticulum stress, recruit inflammatory myeloid cells, and secrete adipokines that bias signaling toward proliferation and angiogenesis[1–4]. Insulin resistance elevates insulin and IGF-1 tone, while lipotoxic species such as ceramides and oxidized lipids sustain NF-κB and STAT3 programs in epithelial and stromal compartments[5–7]. These axes converge in tumor microenvironments that are fibrotic, hypoxic, and immunosuppressed, with dysfunctional vasculature and high interstitial fluid pressure that collectively hinder drug penetration. Conventional anticancer regimens respond by dose escalation or regimen intensification, which often worsens cardiometabolic toxicity in patients already burdened by obesity-related comorbidities[8]. Conversely, lifestyle or pharmacologic weight reduction improves inflammatory and endocrine tone but seldom addresses established malignant clones or their stromal niches.

Smart nanocarriers recast this dilemma as a design problem. Encapsulating synergistic payloads an anti-obesity agent and an anticancer therapy within a single programmable vehicle enables matched pharmacokinetics, co-localized delivery, and controllable sequence or ratio of exposure[9–12]. A liposomal or polymeric particle can, for example, deliver a GLP-1 receptor agonist or adiponectin-mimetic alongside a topoisomerase inhibitor or PI3K–mTOR blocker; a biomimetic shell can guide the construct to inflamed vasculature and macrophage-rich compartments; and embedded triggers can orchestrate the order in which payloads are released, first softening stromal or metabolic barriers and then imposing cytotoxic or immune-activating pressure. Such choreography

is difficult with free combinations because of disparate half-lives, volumes of distribution, and off-target liabilities[9].

The co-delivery concept extends beyond simple colocalization. By biasing endothelial and stromal biology toward normalized perfusion, decreasing adipose lipolysis and lipotoxic signaling, and enhancing adiponectin–AMPK tone, the metabolic payload can increase tumor accessibility and reduce systemic toxicity, thereby widening the therapeutic window for the anticancer component[13–15]. Simultaneously, cytotoxic, targeted, or nucleic-acid therapies shrink tumor burden and deplete pro-inflammatory sources that perpetuate insulin resistance. This reciprocal reinforcement elevates the strategy from additive to potentially supra-additive. The sections that follow delineate design principles for such platforms, summarize leading chassis and cargo pairings, describe logic gates and targeting motifs that align with obesity-specific pathophysiology, and outline pharmacokinetic, safety, and translational frameworks suited to high-BMI populations.

2 Design Principles and Pharmacology of Dual-Action Nanocarriers

Multifunctional carriers succeed when they control four variables: spatial localization, temporal sequencing, dose ratio, and biological addressability. Spatial localization is governed by hydrodynamic size, surface charge, and stealth chemistry. Sizes between roughly seventy and one hundred nanometers balance prolonged circulation with penetration in fibrotic, inflamed tissues typical of obese tumors, while near-neutral or weakly negative zeta potentials minimize nonspecific interactions and complement activation[16]. Dense, non-immunogenic stealth, increasingly based on zwitterionic or poly(2-oxazoline) coronas, counters dyslipidemia-driven protein corona formation and anti-PEG immunity that may be more frequent in metabolic syndrome[17, 18].

Temporal sequencing determines which payload acts first. In many designs, metabolic normalization precedes tumor-directed cytotoxicity. Acid-labile linkers can release a GLP-1 agonist or AMPK activator within the mildly acidic adipose–tumor interstitium, while more stringent triggers enzymes such as matrix metalloproteinases or higher levels of reactive oxygen species, release the anticancer payload after perfusion improves[19, 20]. Alternatively, a short priming burst of mild photothermal heating or stromal enzyme can precede both payloads from a single particle.

Dose ratio matters because the therapeutic window for a GLP-1 agonist or adiponectin-mimetic is narrow when off-target tissues are exposed, and because drug–drug synergy is often ratio dependent[21]. Co-encapsulation enforces a fixed or programmable ratio independent of systemic clearance, stabilizing pharmacodynamics across heterogeneous body compositions. Finally, biological addressability aligns ligands with receptors enriched in obese microenvironments. RGD and related peptides direct endothelial integrins on angiogenic vessels; mannose or folate motifs bias uptake to CD206-positive or FOLR-β-positive macrophages; transferrin and anti-HER2 guide tumor cells; and fatty-acid anchors hitchhike on albumin to reach sites of lipid flux.

3 Lipid-Based Smart Carriers for Metabolic–Oncologic Co-Therapy

Liposomal platforms accommodate hydrophilic and hydrophobic cargos in aqueous cores and bilayers, respectively, and their membranes support pH-, redox-, and heat-triggered release. One design encapsulates a GLP-1 receptor agonist or an adiponectin-mimetic peptide in the aqueous core, stabilizing it against proteolysis, while intercalating a chemotherapeutic such as doxorubicin or irinotecan within the bilayer [22, 23]. The bilayer composition is tuned to destabilize at tumor pH, releasing the cytotoxic agent after the peptide has diffused to nearby adipocytes, endothelium, and macrophages[22, 24, 25]. A related formulation pairs an AMPK activator with a PI3K–mTOR inhibitor; by curtailing anabolism in both tumor and stromal cells, the metabolic payload reduces ATP-intensive resistance programs and may mitigate hyperglycemia associated with PI3K pathway blockade.

Lipid nanoparticles optimized for nucleic-acid delivery enable gene-level co-therapy: siRNA against leptin or STAT3 reduces pro-tumor adipokine signaling and myeloid polarization, while mRNA encodes tumor suppressors or immune agonists. Sequential release is programmed by ionizable lipid chemistry and biodegradable helpers with distinct pKa and hydrolysis rates[24, 26, 27]. Albumin-binding lipids and short-chain fatty-acid anchors increase extravasation at inflamed endothelium, where albumin flux is high in obesity. For regional disease, thermosensitive liposomes triggered by mild hyperthermia can deposit both payloads within peritumoral fat pads, minimizing systemic exposure while exploiting improved local perfusion.

Safety considerations in lipid constructs include complement activation and hepatic retention. Alternative stealth polymers and graded infusion mitigate infusion reactions. In fatty liver disease, careful control of particle size and surface density reduces Kupffer cell uptake; theranostic lipids or co-encapsulated tracers enable real-time verification of tumor accumulation before activating external triggers or delivering subsequent systemic agents.

4 Polymeric, Hybrid, and Biomimetic Platforms for Programmable Sequencing

Polymeric nanoparticles based on PLGA, PEG-PLA, and related backbones offer programmable degradation and multi-compartment architectures. Core–shell formats place an outer shell loaded with metabolic modulators that erode quickly in acidic interstitium, while an inner core carries chemotherapeutics or kinase inhibitors released over days[28–31]. Charge-reversal layers keep particles neutral in circulation and switch to cationic

in tumors, enhancing endosomal escape for nucleic acids that silence LEPR–STAT3 signaling or aromatase transcripts in hormone-sensitive cancers linked to obesity.

Hybrid systems fuse inorganic photothermal cores with polymer shells that bear both metabolic and anticancer agents. A brief, sub-ablative heating pulse normalizes perfusion and lowers interstitial pressure; in the same session, acid-triggered shell erosion releases an AMPK activator into adipose and endothelium, followed by chemotherapeutic release as matrix metalloproteinases rise. This single-session choreography aligns energy and pharmacology without requiring multiple hospital visits[32].

Biomimetic carriers cloaked with macrophage or adipocyte membranes inherit homing receptors and immune stealth. These shells guide particles to inflamed adipose depots and macrophage-rich niches while avoiding rapid clearance[32]. Within this chassis, a GLP-1 agonist can temper local cytokine production and lipolysis, while a PI3K-γ inhibitor or cytotoxic payload reprograms or depletes protumor myeloid cells. Engineered extracellular vesicles derived from adipose stromal cells offer a related approach: donor cells are edited to overexpress adiponectin variants and to exclude oncogenic microRNAs, generating vesicles that both deliver a metabolic correction and ferry anticancer siRNA or small-molecule cargo. Such vesicles naturally engage adipose–tumor interfaces, leveraging endogenous trafficking codes to enforce co-localization[33, 34].

5 Stimuli-Responsive Logic, Targeting, and Imaging for Precision Co-Delivery

Smart co-delivery depends on reliable triggers that map to microenvironmental features accentuated by obesity. pH-sensitive linkers open in the mildly acidic interstitium surrounding tumors and inflamed adipose, releasing metabolic payloads first[35–37]. Reactive oxygen species, elevated by metaflammation, cleave thioketal or boronic ester linkers to discharge cytotoxic or targeted agents only after oxidative thresholds are reached. Enzyme-responsive peptides keyed to matrix metalloproteinases or lipases enrich release at the adipose–tumor border; thermal triggers from embedded photothermal elements synchronize stromal priming with drug deposition.

Targeting ligands are selected to triangulate endothelial, myeloid, and tumor compartments. RGD-family peptides bind angiogenic integrins; mannose and folate engage macrophages that dominate obese microenvironments; anti-HER2 or anti-EGFR fragments focus delivery to tumor cells; and fatty-acid motifs enable albumin hitchhiking toward regions of lipid flux[25, 27, 32, 37]. Multivalent presentation increases avidity without compromising stealth when ligands are spaced and buffered by flexible linkers.

Imaging converts these mechanisms into measurable therapy. Near-infrared fluorophores and photoacoustic chromophores embedded in the carrier track distribute across adipose, liver, spleen, and tumor. Ratiometric reporters reveal when the metabolic payload has been released, cueing the timing of the cytotoxic wave [27]. In trials, such theranostic readouts support adaptive scheduling and stratification by body composition; in practice, they help clinicians confirm that co-localization, being a prerequisite for synergy, has actually occurred in each patient.

6 Pharmacokinetics, Dosing, Safety, and Translation in High-BMI Populations

High-BMI pharmacology challenges one-size-fits-all dosing. Expanded plasma volume, altered lipoprotein profiles, and fatty liver disease accelerate mononuclear phagocyte uptake and distort nanoparticle exposure[38]. Lean body mass or allometric scaling better predicts distribution than total body weight. Corona control using zwitterionic coatings stabilizes behavior in dyslipidemic matrices; hydrodynamic diameters near eighty to one hundred nanometers reduce hepatic sequestration while maintaining tumor access, though smaller cross-linked micelles may be selected for desmoplastic disease at the cost of shorter half-life[38].

Safety is bidirectional because payloads influence host metabolism and tumor biology. GLP-1 agonists can cause gastrointestinal effects; AMPK activators interact with mitochondrial metabolism; kinase inhibitors interact with glucose homeostasis[39]. Co-encapsulation allows lower individual doses for equivalent effect, but careful release kinetics are essential to avoid transient hypoglycemia or off-target mitochondrial stress. Hepatic monitoring is heightened in steatosis; renal monitoring attends to polymer metabolite clearance. Complement activation–related pseudoallergy is mitigated by alternative stealth and graded infusion[39].

Translation requires manufacturing discipline that links critical quality attributes, size distribution, ligand density, trigger thresholds, payload ratio, and release kinetics to potency assays reflecting the dual mechanism[40]. These include reductions in circulating leptin: adiponectin ratios, increases in p-AMPK and improved microvascular flow in adipose–tumor interfaces, alongside tumor shrinkage or pathway inhibition markers. Imaging-verified delivery acts as a companion diagnostic to justify dose and schedule[41]. Clinical designs should synchronize nanotherapy with metabolic stabilization through diet, exercise, or pharmacologic agents to maximize microenvironment receptivity, and should prespecify BMI-aware stratification so that efficacy in high-BMI cohorts is measured rather than averaged away.

CONCLUSION

Smart nanocarriers for co-delivery of anti-obesity and anticancer agents transform a clinical trade-off into a coordinated strategy. By correcting metabolic and inflammatory drivers while simultaneously applying tumor-directed pressure, dual-action platforms address the coupled biology that links adipose dysfunction to malignant fitness. Lipid, polymeric, biomimetic, and hybrid constructs supply the structural and chemical diversity needed to tune localization, sequence, and ratio of exposure; stimuli-responsive logic and targeted ligands align action

with the altered vasculature, matrix, and immune tone of obesity; and embedded imaging ensures that design intent is realized in vivo. With BMI-aware dosing, corona-informed stealth, and manufacturing frameworks that tie quality to mechanism, co-delivery can move from conceptual synergy to reproducible benefit, shrinking tumors while rehabilitating the metabolic terrain that gave rise to them.

REFERENCES

1. Ahmed, B., Konje, J.C.: The epidemiology of obesity in reproduction. Best Pract. Res. Clin. Obstet. Gynaecol. 89, 102342 (2023). https://doi.org/10.1016/j.bpobgyn.2023.102342
2. Ahmed, S.K., Mohammed, R.A.: Obesity: Prevalence, causes, consequences, management, preventive strategies and future research directions. Metab. Open. 27, 100375 (2025). https://doi.org/10.1016/j.metop.2025.100375
3. Alanazi, A., Craven, A., Spirou, S.V., Santos-Martinez, M.J., Medina, C., Gobbo, O.L.: Nanomedicine as a Promising Treatment Approach for Obesity. J. Nanotheranostics. 6, 21 (2025). https://doi.org/10.3390/jnt6030021
4. Aloo, S.-O., Ofosu, F.K., Kim, N.-H., Kilonzi, S.M., Oh, D.-H.: Insights on Dietary Polyphenols as Agents against Metabolic Disorders: Obesity as a Target Disease. Antioxidants. 12, 416 (2023). https://doi.org/10.3390/antiox12020416
5. Abbasi, E., Khodadadi, I.: High-fat diet may increase the risk of insulin resistance by inducing dysbiosis. Metab. Open. 27, 100381 (2025). https://doi.org/10.1016/j.metop.2025.100381
6. AbdlWhab, H.M., Al-Saffar, A., Mahdi, O.A., Alameri, R.B.: The impact of insulin resistance and glycaemic control on insulin-like growth factor-1 in patients with type 2 diabetes: a cross-sectional study. Clin. Diabetes Endocrinol. 10, 36 (2024). https://doi.org/10.1186/s40842-024-00202-8
7. Apostolopoulou, M., Lambadiari, V., Roden, M., Dimitriadis, G.D.: Insulin Resistance in Type 1 Diabetes: Pathophysiological, Clinical, and Therapeutic Relevance. Endocr. Rev. 46, 317–348 (2025). https://doi.org/10.1210/endrev/bnae032
8. Ali, M.M., Parveen, S., Williams, V., Dons, R., Uwaifo, G.I.: Cardiometabolic comorbidities and complications of obesity and chronic kidney disease (CKD). J. Clin. Transl. Endocrinol. 36, 100341 (2024). https://doi.org/10.1016/j.jcte.2024.100341
9. Kapalatiya, H., Madav, Y., Tambe, V.S., Wairkar, S.: Enzyme-responsive smart nanocarriers for targeted chemotherapy: an overview. Drug Deliv. Transl. Res. 12, 1293–1305 (2022). https://doi.org/10.1007/s13346-021-01020-6
10. Karimi, M., Zangabad, P.S., Ghasemi, A., Amiri, M., Bahrami, M., Malekzad, H., Asl, H.G., Mahdieh, Z., Bozorgomid, M., Ghasemi, A., Boyuk, M.R.R.T., Hamblin, M.R.: Temperature-Responsive Smart Nanocarriers for Delivery Of Therapeutic Agents: Applications and Recent Advances. ACS Appl. Mater. Interfaces. 8, 21107–21133 (2016). https://doi.org/10.1021/acsami.6b00371
11. Kaushik, A., Khan, S., Pharasi, N., Mani, S.: Dual pH and ultrasound responsive nanocarriers: A smart approach in cancer theranostics. J. Drug Deliv. Sci. Technol. 95, 105560 (2024). https://doi.org/10.1016/j.jddst.2024.105560
12. Liu, X., He, F., Liu, M.: New opportunities of stimulus-responsive smart nanocarriers in cancer therapy. Nano Mater. Sci. (2024). https://doi.org/10.1016/j.nanoms.2024.10.013
13. Alzaid, F., Fagherazzi, G., Riveline, J.-P., Bahman, F., Al-Rashed, F., Al-Mulla, F., Ahmad, R.: Immune cell–adipose tissue crosstalk in metabolic diseases with a focus on type 1 diabetes. Diabetologia. 68, 1616–1631 (2025). https://doi.org/10.1007/s00125-025-06437-z
14. Auger, C., Kajimura, S.: Adipose Tissue Remodeling in Pathophysiology. Annu. Rev. Pathol. Mech. Dis. 18, 71–93 (2023). https://doi.org/10.1146/annurev-pathol-042220-023633
15. Basso, P.J., Schcolnik-Cabrera, A., Zhu, M., Strachan, E., Clemente-Casares, X., Tsai, S.: Weight Loss-Associated Remodeling of Adipose Tissue Immunometabolism. Obes. Rev. n/a, e13975. https://doi.org/10.1111/obr.13975
16. Chen, W., Cheng, C.-A., Zink, J.I.: Spatial, Temporal, and Dose Control of Drug Delivery using Noninvasive Magnetic Stimulation. ACS Nano. 13, 1292–1308 (2019). https://doi.org/10.1021/acsnano.8b06655
17. Holick, C.T., Klein, T., Mehnert, C., Adermann, F., Anufriev, I., Streiber, M., Harder, L., Traeger, A., Hoeppener, S., Franke, C., Nischang, I., Schubert, S., Schubert, U.S.: Poly(2-ethyl-2-oxazoline) (POx) as Poly(ethylene glycol) (PEG)-Lipid Substitute for Lipid Nanoparticle Formulations. Small Weinh. Bergstr. Ger. 21, 2411354 (2025). https://doi.org/10.1002/smll.202411354
18. van Zyl, D.G., Mendes, L.P., Semper, R.P., Rueckert, C., Baumhof, P.: Poly(2-methyl-2-oxazoline) as a polyethylene glycol alternative for lipid nanoparticle formulation. Front. Drug Deliv. 4, (2024). https://doi.org/10.3389/fddev.2024.1383038
19. Li, R., Sun, X., Li, P., Li, W., Zhao, L., Zhu, L., Zhu, S.: GLP-1-Induced AMPK Activation Inhibits PARP-1 and Promotes LXR-Mediated ABCA1 Expression to Protect Pancreatic β-Cells Against Cholesterol-Induced Toxicity Through Cholesterol Efflux. Front. Cell Dev. Biol. 9, 646113 (2021). https://doi.org/10.3389/fcell.2021.646113

20. Schooling, C.M., Yang, G., Soliman, G.A., Leung, G.M.: A Hypothesis That Glucagon-like Peptide-1 Receptor Agonists Exert Immediate and Multifaceted Effects by Activating Adenosine Monophosphate-Activate Protein Kinase (AMPK). Life. 15, 253 (2025). https://doi.org/10.3390/life15020253
21. Ceasovschih, A., Asaftei, A., Lupo, M.G., Kotlyarov, S., Bartušková, H., Balta, A., Sorodoc, V., Sorodoc, L., Banach, M.: Glucagon-like peptide-1 receptor agonists and muscle mass effects. Pharmacol. Res. 220, 107927 (2025). https://doi.org/10.1016/j.phrs.2025.107927
22. Uti, D.E., Alum, E.U., Atangwho, I.J., Ugwu, O.P.-C., Egbung, G.E., Aja, P.M.: Lipid-based nano-carriers for the delivery of anti-obesity natural compounds: advances in targeted delivery and precision therapeutics. J. Nanobiotechnology. 23, 336 (2025). https://doi.org/10.1186/s12951-025-03412-z
23. Uti, D.E., Omang, W.A., Alum, E.U., Ugwu, O.P.-C., Wokoma, M.A., Oplekwu, R.I., Atangwho, I.J., Egbung, G.E.: Combined Hyaluronic Acid Nanobioconjugates Impair CD44-Signaling for Effective Treatment Against Obesity: A Review of Comparison with Other Actors. Int. J. Nanomedicine. 20, 10101–10126 (2025). https://doi.org/10.2147/IJN.S529250
24. Fan, S., Han, H., Yan, Z., Lu, Y., He, B., Zhang, Q.: Lipid-based nanoparticles for cancer immunotherapy. Med. Rev. 3, 230–269. https://doi.org/10.1515/mr-2023-0020
25. Kumar, R., Dkhar, D.S., Kumari, R., Divya, Mahapatra, S., Srivastava, A., Dubey, V.K., Chandra, P.: Ligand conjugated lipid-based nanocarriers for cancer theranostics. Biotechnol. Bioeng. 119, 3022–3043 (2022). https://doi.org/10.1002/bit.28205
26. Borges, A., de Freitas, V., Mateus, N., Fernandes, I., Oliveira, J.: Solid Lipid Nanoparticles as Carriers of Natural Phenolic Compounds. Antioxidants. 9, 998 (2020). https://doi.org/10.3390/antiox9100998
27. Gabel, M., Knauss, A., Fischer, D., Neurath, M.F., Weigmann, B.: Surface Design Options in Polymer- and Lipid-Based siRNA Nanoparticles Using Antibodies. Int. J. Mol. Sci. 23, 13929 (2022). https://doi.org/10.3390/ijms232213929
28. El-Hammadi, M.M., Arias, J.L.: Recent Advances in the Surface Functionalization of PLGA-Based Nanomedicines. Nanomaterials. 12, 354 (2022). https://doi.org/10.3390/nano12030354
29. Ghandforoushan, P., Hanaee, J., Aghazadeh, Z., Samiei, M., Navali, A.M., Khatibi, A., Davaran, S.: Enhancing the function of PLGA-collagen scaffold by incorporating TGF-β1-loaded PLGA-PEG-PLGA nanoparticles for cartilage tissue engineering using human dental pulp stem cells. Drug Deliv. Transl. Res. 12, 2960–2978 (2022). https://doi.org/10.1007/s13346-022-01161-2
30. Long, Q., Liu, Z., Shao, Q., Shi, H., Huang, S., Jiang, C., Qian, B., Zhong, Y., He, X., Xiang, X., Yang, Y., Li, B., Yan, X., Zhao, Q., Wei, X., Santos, H.A., Ye, X.: Autologous Skin Fibroblast-Based PLGA Nanoparticles for Treating Multiorgan Fibrosis. Adv. Sci. 9, 2200856 (2022). https://doi.org/10.1002/advs.202200856
31. Omidian, H., Wilson, R.L.: PLGA Implants for Controlled Drug Delivery and Regenerative Medicine: Advances, Challenges, and Clinical Potential. Pharmaceuticals. 18, 631 (2025). https://doi.org/10.3390/ph18050631
32. Matlou, G.G., Abrahamse, H.: Hybrid Inorganic-Organic Core-Shell Nanodrug Systems in Targeted Photodynamic Therapy of Cancer. Pharmaceutics. 13, 1773 (2021). https://doi.org/10.3390/pharmaceutics13111773
33. Chen, J., Mei, A., Wei, Y., Li, C., Qian, H., Min, X., Yang, H., Dong, L., Rao, X., Zhong, J.: GLP-1 receptor agonist as a modulator of innate immunity. Front. Immunol. 13, 997578 (2022). https://doi.org/10.3389/fimmu.2022.997578
34. Tong, G., Peng, T., Chen, Y., Sha, L., Dai, H., Xiang, Y., Zou, Z., He, H., Wang, S.: Effects of GLP-1 Receptor Agonists on Biological Behavior of Colorectal Cancer Cells by Regulating PI3K/AKT/mTOR Signaling Pathway. Front. Pharmacol. 13, (2022). https://doi.org/10.3389/fphar.2022.901559
35. Ashrafizadeh, M., Delfi, M., Zarrabi, A., Bigham, A., Sharifi, E., Rabiee, N., Paiva-Santos, A.C., Kumar, A.P., Tan, S.C., Hushmandi, K., Ren, J., Zare, E.N., Makvandi, P.: Stimuli-responsive liposomal nanoformulations in cancer therapy: Pre-clinical & clinical approaches. J. Controlled Release. 351, 50–80 (2022). https://doi.org/10.1016/j.jconrel.2022.08.001
36. Du, J., Lane, L.A., Nie, S.: Stimuli-Responsive Nanoparticles for Targeting the Tumor Microenvironment. J. Control. Release Off. J. Control. Release Soc. 219, 205–214 (2015). https://doi.org/10.1016/j.jconrel.2015.08.050
37. Majumder, J., Minko, T.: Multifunctional and stimuli-responsive nanocarriers for targeted therapeutic delivery. Expert Opin. Drug Deliv. 18, 205–227 (2021). https://doi.org/10.1080/17425247.2021.1828339
38. Stender, S., Kozlitina, J., Nordestgaard, B.G., Tybjærg-Hansen, A., Hobbs, H.H., Cohen, J.C.: Adiposity Amplifies the Genetic Risk of Fatty Liver Disease Conferred by Multiple Loci. Nat. Genet. 49, 842–847 (2017). https://doi.org/10.1038/ng.3855
39. Li, R., Sun, X., Li, P., Li, W., Zhao, L., Zhu, L., Zhu, S.: GLP-1-Induced AMPK Activation Inhibits PARP-1 and Promotes LXR-Mediated ABCA1 Expression to Protect Pancreatic β-Cells Against Cholesterol-

Induced Toxicity Through Cholesterol Efflux. Front. Cell Dev. Biol. 9, 646113 (2021). https://doi.org/10.3389/fcell.2021.646113
40. Taha, M.S., Padmakumar, S., Singh, A., Amiji, M.M.: Critical quality attributes in the development of therapeutic nanomedicines toward clinical translation. Drug Deliv. Transl. Res. 10, 766–790 (2020). https://doi.org/10.1007/s13346-020-00744-1
41. Yadav, P.R., Munni, M.N., Campbell, L., Mostofa, G., Dobson, L., Shittu, M., Pattanayek, S.K., Uddin, Md.J., Das, D.B.: Translation of Polymeric Microneedles for Treatment of Human Diseases: Recent Trends, Progress, and Challenges. Pharmaceutics. 13, 1132 (2021). https://doi.org/10.3390/pharmaceutics13081132

CITE AS: Kamanzi Ntakirutimana G. (2026). Smart Nanocarriers for Co-Delivery of Anti-Obesity and Anticancer Agents. Research Output Journal of Engineering and Scientific Research 5(1): 53-58. https://doi.org/10.59298/ROJESR/2026/5.15358