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Viewpoint

Antiobesity medications in rheumatology. Quo vadis?

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ABSTRACT

Obesity is a well-recognised comorbidity in the context of rheumatic and musculoskeletal diseases (RMDs), adversely affecting disease-related outcomes. Adipose tissue, through immunological mechanisms, induces a low-grade inflammatory state; as a result, there has been growing interest in evaluating the potential role of antiobesity drugs in the management of RMDs. Although they were initially approved for type 2 diabetes mellitus, obesity, and their cardiorenal associations, there is increasing evidence that glucagon-like peptide-1 receptor agonists (GLP-1 RAs) in particular may exert immunomodulatory and anti-inflammatory effects in the setting of RMDs. In this viewpoint, we discuss current data regarding the effects of GLP-1 RAs on several conditions, including osteoarthritis, systemic lupus erythematosus, rheumatoid arthritis, fibromyalgia, and osteoporosis. We also highlight ongoing studies, which appear to be promising. Furthermore, we propose that these drugs could be administered to difficult-to-manage cases or in people at an increased risk of developing RMDs (like obese psoriatic patients) or even as adjunctive therapy, considering also the cost barrier that exists in most countries. Preliminary findings are encouraging; however, as most of the available evidence is limited to a small sample size, large-scale randomised controlled trials are needed to evaluate also the long-term safety of these drugs throughout the spectrum of rheumatic disorders.

OBESITY AND ANTIOBESITY DRUGS

Obesity is defined as excessive fat deposition in different parts of the body or organs. Although it is classically defined as a body mass index (BMI) ≥30 kg/m^2, the term 'clinical obesity' was recently introduced. It refers to obesity-related organ or tissue dysfunction, limitation of daily activities, and includes assessment of body fat (where tools are available) or measurement of other anthropometric characteristics (eg, waist circumference) in addition to BMI [1,2].

For obesity management, in addition to a calorie-restricted diet and regular physical activity, pharmacotherapy can be added not only to achieve but also to maintain weight loss [3].

There are 6 pharmaceutical agents currently available and US Food and Drug Administration approved for weight management, which act by decreasing appetite, enhancing satiety, and delaying gastric emptying: semaglutide and liraglutide (glucagon-like peptide-1 receptor agonists [GLP-1 RAs]), orlistat (gastric and pancreatic lipase inhibitor), phentermine-topiramate (norepinephrine agonist/γ-aminobutyric acid agonist and glutamate antagonist), naltrexone-bupropion (opioid receptor antagonist/dopamine and norepinephrine reuptake inhibitor), and recently, tirzepatide

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(gastric inhibitory polypeptide/glucagon-like peptide-1 [GLP-1] dual agonist) [4].

OBESITY IN RHEUMATIC AND MUSCULOSKELETAL DISEASES

The spectrum of rheumatic and musculoskeletal diseases (RMDs) includes inflammatory and crystal-induced arthritides, connective tissue diseases (eg, systemic lupus erythematosus [SLE]), degenerative joint diseases (eg, osteoarthritis [OA]), and conditions related to bone metabolism (eg, osteoporosis) and chronic pain (eg, fibromyalgia).

It has been demonstrated that there is a complex interplay between obesity and RMDs, especially inflammatory arthritides. Besides, obesity is one of the most common comorbidities in the setting of psoriatic arthritis (PsA) and, to a lesser extent, in rheumatoid arthritis (RA) and axial spondyloarthritis (SpA) [5], with the respective figures being about 40% for PsA and 10% to 15% for RA, SpA and SLE [6,7]. It is also evident that obese people living with inflammatory arthritis exhibit lower odds of achieving or maintaining favourable therapeutic outcomes [8]. The reasons behind this are not entirely clear. It was established that there is an interaction between the immune system and the adipose tissue. Visceral fat can produce proinflammatory cytokines (eg, interleukin [IL]-17 and tumour necrosis factor [TNF]-α) and adipokines (leptin, resistin, visfatin, and adiponectin) that have both anti-inflammatory and proinflammatory properties [9]. Therefore, adipose tissue is not an 'innocent bystander' but an immunologically active player [10]. Hence, obesity is considered a chronic low-grade inflammatory state.

WHAT IS THE POTENTIAL ROLE OF GLP-1 RAs IN THE MANAGEMENT OF RMDs?

GLP-1 RAs demonstrate a wide range of benefits. Beyond their importance in cardiorenal and metabolic outcomes, there is also growing evidence of their immunomodulatory and anti-inflammatory effects in nonmetabolic diseases [11]. Repurposing of these drugs, or at least their introduction as an add-on treatment in RMDs, seems to be a likely scenario in the near future, delineating the connection between different disciplines.

The risk of all-cause mortality and major adverse cardiovascular events in patients with immune-mediated inflammatory diseases and type 2 diabetes mellitus is lower with the administration of GLP-1 RAs compared with dipeptidyl peptidase-4 inhibitor exposure, based on a recently published cohort study [12]. An overview of the accumulating evidence, presented below, of the potential benefit of GLP-1 RAs in RMDs is depicted in the Figure and summarised in the Table.

OA

The negative impact of obesity (mechanical overload and low-grade systemic inflammation) in OA is well established. Although it is classified as a noninflammatory disorder, in the early stages, inflammation of the synovium, mediated by cytokines and prodegradative mediators, can occur [13].

There are studies that highlight the beneficial effects of GLP-1 RAs in the management of OA. Immunohistochemistry revealed that GLP-1 receptor protein is expressed in both the synovial membrane and chondrocytes of human OA knee joints [14]. Based on this evidence, the efficacy of liraglutide on pain perception and inflammatory response in OA was assessed in animal models. A significant dose-dependent increase in paw withdrawal threshold was detected in OA mouse models that received intra-articular injections of liraglutide compared with saline-treated mice; the efficacy persisted until the end of the experiment. Furthermore, it was observed that the IL-1β–induced proinflammatory mediators were diminished, and cartilage breakdown was prevented following liraglutide treatment [14].

Pain management was also evaluated in the STEP (Semaglutide Treatment Effect in People with Obesity) 9 trial, in which

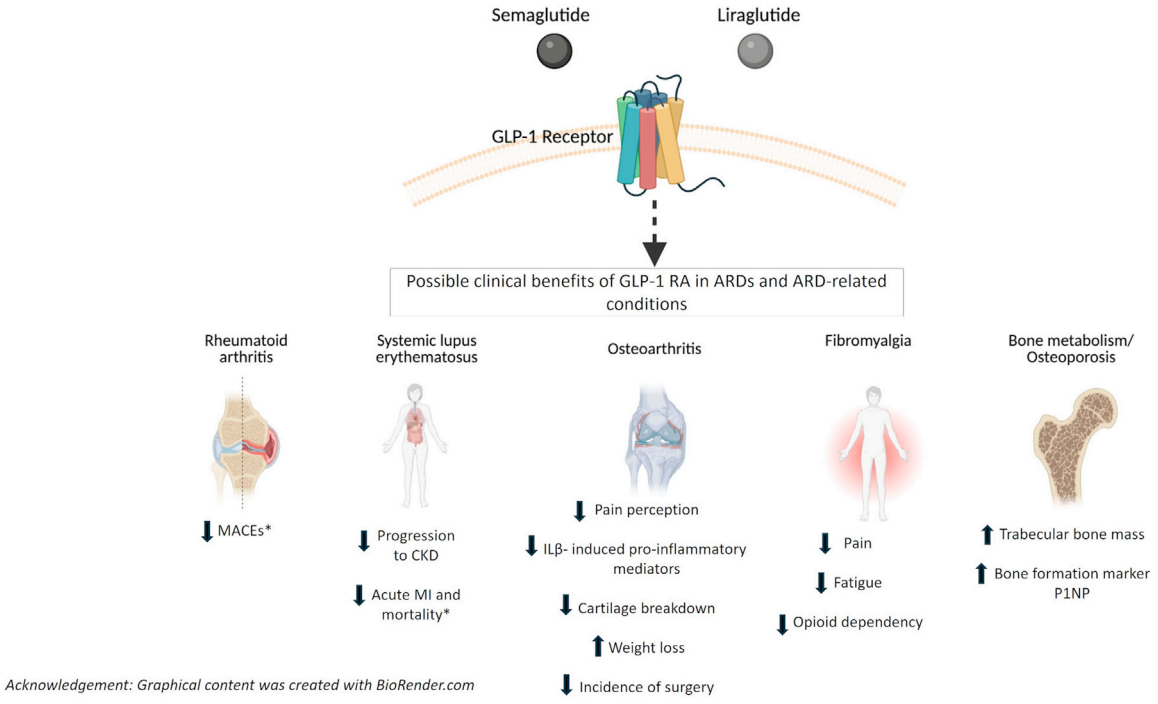

Figure. Potential clinical benefits of glucagon-like peptide-1 receptor agonists (GLP-1 RAs) in autoimmune rheumatic diseases (ARDs) and ARD-related conditions. CKD, chronic kidney disease; ILβ, interleukin-β; MACE, major adverse cardiovascular event; MI, myocardial infarction; P1NP, N-terminal propeptide of type 1 procollagen. *Data were retrieved from abstracts.

Table
Studies on antiobesity drugs in rheumatic diseases

Study group	Intervention	Study objective	Main results
MIA OA mice model	IA liraglutide injections	Anti-inflammatory and analgesic effects of liraglutide	Decrease in pain-related behaviour
Obese individuals (knee OA)	Semaglutide 2.4 mg vs placebo group	Percentage change in weight and WOMAC score	Weight and WOMAC score both improved in the semaglutide group
Diabetic individuals with OA	GLP-1 RA group vs control group	Incidence of knee surgery	Lower incidence of knee surgery in the GLP-1 RA group
Overweight/obese individuals (knee OA)	Liraglutide 3 mg/d vs placebo group	Efficacy and safety of liraglutide	Weight loss, no difference in KOOS
SLE patients	GLP-1 RAs	Outcomes of GLP1-RAs in SLE	Not associated with increased disease flares
SLE nephritis patients	GLP-1 RAs vs SGLT-2 inhibitors	Effect of GLP-1 RAs compared with SGLT-2 inhibitors	Lower risk of CKD, MI, and mortality in GLP-1 RAs patients
RA patients	GLP-1 RAs with/without JAK inhibitor	Role of GLP-1 RAs against CV adverse effects	Lower risk of acute coronary syndromes, DVT, and overall arterial CV events
Fibromyalgia patients	GLP-1 RAs users vs GLP-1 RAs naive	Impact of GLP-1 RAs on fibromyalgia's opioid use, fatigue, and pain	Reduction of symptom burden

CKD, chronic kidney disease; CV, cardiovascular; DVT, deep vein thrombosis; GLP-1 RA, glucagon-like peptide-1 receptor agonist; IA, intra-articular; JAK, Janus kinase; KOOS, Knee injury and Osteoarthritis Outcome Score; MI, myocardial infarction; MIA, monoiodoacetate; OA, osteoarthritis; RA, rheumatoid arthritis; SGLT-2, sodium-glucose cotransporter-2; SLE, systemic lupus erythematosus; WOMAC, Western Ontario and McMaster Universities Osteoarthritis Index.

407 individuals with moderate knee OA were enrolled. They were randomised to receive once-weekly subcutaneous semaglutide (2.4 mg) or placebo, together with a low-calorie diet and adjusted physical activity. The mean change in both body weight from baseline and the Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC) pain score was significantly improved at week 68 in the semaglutide group [15].

Furthermore, the potential use of GLP-1 RAs as disease-modifying drugs for knee OA was studied by the Shanghai Osteoarthritis Cohort, involving 1807 patients with both knee OA and type 2 diabetes. The group that was exposed to GLP-1 RAs experienced greater weight loss and a lower incidence of knee surgery than the control group. Statistically significant differences were also detected between the 2 groups on the WOMAC total and pain subscale scores [16].

By contrast, in another study in which 156 patients were randomised to receive liraglutide 3 mg/d or placebo, after an 8-week diet, results were less encouraging; only one of the primary endpoints (changes in body weight), but not the other (a change in the Knee injury and Osteoarthritis Outcome Score [KOOS]), was met [17].

SLE

There is some evidence regarding the use of GLP-1 RAs in SLE patients. After presentation of a case of drug-induced lupus secondary to semaglutide administration [18], a retrospective study showed that GLP-1 RAs were not associated with flares of the disease after 6 to 10 months of use, although these patients experienced a significant decrease in BMI [19]. Nevertheless, these findings require careful interpretation due to the small sample size and the absence of a control group. In another recent cohort study of patients with lupus nephritis (n = 1034), the use of GLP-1 RAs correlated with a reduced risk of progression of chronic kidney disease, acute myocardial infarction, and mortality in comparison with patients on sodium-glucose cotransporter-2 (SGLT2) inhibitors (n = 1375). These data are published only as an abstract thus far; therefore, it should be interpreted with caution as some information, like previous treatments administered for lupus nephritis, is lacking [20].

RA

Current data on RA are limited. Recent evidence from Mendelian randomisation analysis revealed that increased GLP-1 receptor gene expression is significantly associated with a reduced risk of several autoimmune diseases. In the case of RA, it was detected that higher GLP-1 receptor gene expression was associated with a 27% lower odds for RA development [21]. Moreover, a recent retrospective cohort analysis of RA patients on Janus kinase (JAK) inhibitors divided into 2 groups, each cohort comprising 2449 patients (one with concurrent GLP-1 RAs use and the other only on JAK inhibitors), showed that those receiving GLP-1 RAs demonstrated an approximately 35% lower risk of acute coronary syndrome ($p < .001$), a 31% lower risk of deep venous thrombosis ($p < .05$), and about a 33% lower incidence of arterial cardiovascular events ($p < .001$) [22]. Certainly, further studies with larger populations are needed to clearly elucidate the role and the effect of GLP-1 RAs in patients with RA.

Fibromyalgia

Despite the limited data (published in abstract form thus far), positive results for fibromyalgia were reported. Analysing data from the TriNetX network, 2 groups were evaluated: group 1, patients diagnosed with fibromyalgia who have used GLP-1 RAs at least twice on different occasions (n = 46,409), and group 2, patients with fibromyalgia without a recorded use of GLP-1 RAs (n = 716,185). It was shown that the use of GLP-1 RAs was connected to significantly reduced pain, fatigue, and opioid dependency [23].

Osteoporosis

The relation between GLP-1 and bone metabolism may elucidate the impact of GLP-1 RAs on bone quality, too. Treatment with liraglutide in healthy obese women, after a weight loss period, showed that those who received the drug demonstrated an increase in bone formation marker N-terminal propeptide of type 1 procollagen (P1NP) by 16% vs the control group ($p < .05$) [24]. In addition, a positive effect of GLP-1 RAs on bone health

was found in ovariectomised mice, which received liraglutide and exenatide, resulting in improvement of trabecular bone mass (but not on cortical bone) in comparison with the saline group [25].

However, plasma P1NP was not affected after semaglutide administration in patients with increased fracture risk; in this group, enhanced bone resorption was recorded, which was attributed to concurrent weight loss [26].

WHEN AND BY WHOM COULD GLP-1 RAS BE USED?

Since obesity appears to influence both the onset and treatment response across most RMDs, one could consider GLP1-RAs as a useful aid. Given the cost, as in most countries, GLP-1 RAs are currently covered only for type 2 diabetes patients. A relevant question is: which patients would be more suitable to receive these drugs, and whether the latter can act as a disease-modifying treatment in this context. Considering the accumulating data for difficult-to-manage/refractory cases of inflammatory arthritis, these patients could be the right candidates. Also, in an effort to intercept disease, people at high risk for developing RMDs, such as pre-RA or obese psoriasis patients with relevant risk factors, could have some benefit [27,28]. Finally, it is not irrational to hypothesise that these drugs might have immunomodulatory effects that would enhance/modify the action of classic disease-modifying antirheumatic drugs. However, studies involving larger populations are warranted to confirm their long-term safety.

FUTURE STUDIES AND PERSPECTIVES

At this time, 9 antiobesity drugs are undergoing phase 3 clinical trials either by targeting the GLP-1 receptor alone (orforglipron, semaglutide, ecnoglutide, and TG103), in combination with a GLP-1 receptor agonist and amylin (CagriSema, Novo Nordisk), by acting as agonist on both GLP-1 and glucagon receptors (mazdutide and survodutide), or acting as triple agonists (GLP-1, glucose-dependent insulinotropic peptide, and glucagon receptor [retatrutide]) [29]. Despite their effect on weight control, their broader systemic impact is being investigated across a variety of clinical conditions. The concomitant use of ixekizumab with tirzepatide in obese/overweight adults with active PsA or psoriasis is being studied in phase 3 clinical trials to show the efficacy and safety of this drug combination not only for weight management but also for improvement of psoriatic disease (NCT06588296 and NCT06588283). This viewpoint discusses the accumulating evidence that antiobesity drugs have beneficial effects on a variety of RMDs. In addition to their effect on cardiovascular events as mentioned before, GLP-1 RAs are undergoing trials for metabolic liver disease, kidney disease, and Alzheimer's disease [4]. It is evident that the antiobesity drugs expected to enter the market in the coming years may transform the management of several chronic diseases.

Therefore, the answer to 'quo vadis', cannot be easily given. Nonetheless, the preliminary findings seem to be encouraging, opening new avenues for the development of novel therapeutic strategies for the management of RMDs.

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Competing interests

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Niki Kyriazi: Writing – original draft, Investigation, Data curation. **Konstantinos D. Vassilakis:** Writing – original draft, Data curation. **Amalia Bakiri:** Writing – original draft, Data curation. **Alexios Iliopoulos:** Writing – review & editing, Conceptualization. **George E. Fragoulis:** Writing – review & editing, Investigation, Conceptualization.

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REFERENCES

[1] Rubino F, Cummings DE, Eckel RH, Cohen RV, Wilding JPH, Brown WA, et al. Definition and diagnostic criteria of clinical obesity. Lancet Diabetes Endocrinol 2025 Mar;13(3):221–62. doi: 10.1016/S2213-8587(24)00316-4.

[2] Lin X, Li H. Obesity: epidemiology, pathophysiology, and therapeutics. Front Endocrinol (Lausanne) 2021 Sep 6;12:706978. doi: 10.3389/fendo.2021.706978.

[3] Visseren FLJ, Mach F, Smulders YM, Carballo D, Koskinas KC, Bäck M, et al. 2021 ESC guidelines on cardiovascular disease prevention in clinical practice. Eur Heart J 2021;42:3227–37.

[4] Chakhtoura M, Haber R, Ghezzawi M, Rhayem C, Tcheroyan R, Mantzoros CS. Pharmacotherapy of obesity: an update on the available medications and drugs under investigation. EClinicalMedicine 2023 Mar 20;58:101882. doi: 10.1016/j.eclinm.2023.101882.

[5] Williams JC, Hum RM, Rogers K, Maglio C, Alam U, Zhao SS. Metabolic syndrome and psoriatic arthritis: the role of weight loss as a disease-modifying therapy. Ther Adv Musculoskelet Dis 2024 Aug 19;16 1759720X241271886. doi: 10.1177/1759720×241271886.

[6] Kumthekar A, Ogdie A. Obesity and psoriatic arthritis: a narrative review. Rheumatol Ther 2020;7:447–56. doi: 10.1007/s40744-020-00215-6.

[7] Gremese E, Tolusso B, Gigante MR, Ferraccioli G. Obesity as a risk and severity factor in rheumatic diseases (autoimmune chronic inflammatory diseases). Front Immunol 2014 Nov 11;5:576. doi: 10.3389/fimmu.2014.00576.

[8] di Minno MN, Peluso R, Iervolino S, Lupoli R, Russolillo A, Scarpa R, et al. Obesity and the prediction of minimal disease activity: a prospective study in psoriatic arthritis. Arthritis Care Res (Hoboken) 2013 Jan;65(1):141–7. doi: 10.1002/acr.21711.

[9] Nikiphorou E, Fragoulis GE. Inflammation, obesity and rheumatic disease: common mechanistic links. A narrative review. Ther Adv Musculoskelet Dis 2018 Jun 27;10(8):157–67. doi: 10.1177/1759720×18783894.

[10] Versini M, Jeandel PY, Rosenthal E, Shoenfeld Y. Obesity in autoimmune diseases: not a passive bystander. Autoimmun Rev 2014 Sep;13(9):981–1000. doi: 10.1016/j.autrev.2014.07.001.

[11] Nassar M, Nassar O, Abosheaishaa H, Misra A. Comparative outcomes of systemic diseases in people with type 2 diabetes, or obesity alone treated with and without GLP-1 receptor agonists: a retrospective cohort study from the Global Collaborative Network: author list. J Endocrinol Invest 2025 Feb;48 (2):483–97. doi: 10.1007/s40618-024-02466-4.

[12] Karacabeyli D, Lacaille D, Lu N, McCormick N, Xie H, Choi HK, et al. Mortality and major adverse cardiovascular events after glucagon-like peptide-1 receptor agonist initiation in patients with immune-mediated inflammatory

diseases and type 2 diabetes: a population-based study. PLoS One 2024 Aug 8;19(8):e0308533. doi: 10.1371/journal.pone.0308533.
[13] Sellam J, Berenbaum F. The role of synovitis in pathophysiology and clinical symptoms of osteoarthritis. Nat Rev Rheumatol 2010 Nov;6(11):625–35. doi: 10.1038/nrrheum.2010.159.
[14] Meurot C, Martin C, Sudre L, Breton J, Bougault C, Rattenbach R, et al. Liraglutide, a glucagon-like peptide 1 receptor agonist, exerts analgesic, anti-inflammatory and anti-degradative actions in osteoarthritis. Sci Rep 2022 Jan 28;12(1):1567. doi: 10.1038/s41598-022-05323-7.
[15] Bliddal H, Bays H, Czernichow S, Uddén Hemmingsson J, Hjelmesæth J, Hoffmann Morville T, et al. Once-weekly semaglutide in persons with obesity and knee osteoarthritis. N Engl J Med 2024 Oct 31;391(17):1573–83. doi: 10.1056/NEJMoa2403664.
[16] Zhu H, Zhou L, Wang Q, Cai Q, Yang F, Jin H, et al. Glucagon-like peptide-1 receptor agonists as a disease-modifying therapy for knee osteoarthritis mediated by weight loss: findings from the Shanghai Osteoarthritis Cohort. Ann Rheum Dis 2023 Sep;82(9):1218–26. doi: 10.1136/ard-2023-223845.
[17] Gudbergsen H, Overgaard A, Henriksen M, Wæhrens EE, Bliddal H, Christensen R, et al. Liraglutide after diet-induced weight loss for pain and weight control in knee osteoarthritis: a randomized controlled trial. Am J Clin Nutr 2021 Feb 2;113(2):314–23. doi: 10.1093/ajcn/nqaa328.
[18] Castellanos V, Workneh H, Malik A, Mehta B. Semaglutide-induced lupus erythematosus with multiorgan involvement. Cureus 2024 Mar 1;16(3): e55324. doi: 10.7759/cureus.55324.
[19] Carlucci PM, Cohen B, Saxena A, Belmont HM, Masson M, Gold HT, et al. A retrospective evaluation of glucagon-like peptide-1 receptor agonists in systemic lupus erythematosus patients. Rheumatology (Oxford) 2025 May 1;64 (5):3085–9. doi: 10.1093/rheumatology/keae547.
[20] Grzybowski K, Ali R, Akbari H, Tan I. OP0078 Comparing the impact of GLP-1 agonists and SGLT-2 inhibitors on outcomes in lupus nephritis: a retrospective cohort study. Ann Rheum Dis 2025;84(Suppl 1):68.
[21] Yang Y, Liu W, Zhang Z, Zhang Y, Wang X, Wang J, et al. Exploring glucagon-like peptide-1 receptor agonists as potential disease-modifying agents in autoimmune diseases. J Autoimmun 2025 May;153:103414. doi: 10.1016/j.jaut.2025.103414.
[22] Beltagy A, Eshak N, Chhaya S. OP0069 GLP1 agonists mitigate the risk of cardiovascular events in rheumatoid arthritis patients treated with JAK inhibitors. Ann Rheum Dis 2025;84(Suppl 1):61–2.
[23] Eshak N, Beltagy A, Bazigh I, Chaisrimaneepan N, Yingchoncharoen P, Kalya S, et al. OP0281 Exploring GLP-1 agonists effects in fibromyalgia patients: a propensity matched analysis using TriNetX database. Ann Rheum Dis 2025;84(Suppl 1):232.
[24] Iepsen EW, Lundgren JR, Hartmann B, Pedersen O, Hansen T, Jørgensen NR, et al. GLP-1 receptor agonist treatment increases bone formation and prevents bone loss in weight-reduced obese women. J Clin Endocrinol Metab 2015 Aug;100(8):2909–17. doi: 10.1210/jc.2015-1176.
[25] Pereira M, Jeyabalan J, Jørgensen CS, Hopkinson M, Al-Jazzar A, Roux JP, et al. Chronic administration of glucagon-like peptide-1 receptor agonists improves trabecular bone mass and architecture in ovariectomised mice. Bone 2015 Dec;81:459–67. doi: 10.1016/j.bone.2015.08.006.
[26] Hansen MS, Wölfel EM, Jeromdesella S, Møller JK, Ejersted C, Jørgensen NR, et al. Once-weekly semaglutide versus placebo in adults with increased fracture risk: a randomised, double-blinded, two-centre, phase 2 trial. EClinicalMedicine 2024 May 3;72:102624. doi: 10.1016/j.eclinm.2024.102624.
[27] Deane KD, Holers VM, Emery P, Mankia K, El-Gabalawy H, Sparks JA, et al. Therapeutic interception in individuals at risk of rheumatoid arthritis to prevent clinically impactful disease. Ann Rheum Dis 2025 Jan;84(1):14–28. doi: 10.1136/ard-2023-224211.
[28] Zabotti A, De Marco G, Gossec L, Baraliakos X, Aletaha D, Iagnocco A, et al. EULAR points to consider for the definition of clinical and imaging features suspicious for progression from psoriasis to psoriatic arthritis. Ann Rheum Dis 2023 Sep;82(9):1162–70. doi: 10.1136/ard-2023-224148.
[29] Kokkorakis M, Chakhtoura M, Rhayem C, Al Rifai J, Ghezzawi M, Valenzuela-Vallejo L, et al. Emerging pharmacotherapies for obesity: a systematic review. Pharmacol Rev 2025 Jan;77(1):100002. doi: 10.1124/pharmrev.123.001045.