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Integrated Gene Ontology, Cheminformatics and Molecular Simulation Approaches for Elucidating the *MET/NFKB1/ABCG2* Inhibition by *Rheum emodi* Against Rheumatoid Arthritis

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ABSTRACT This study comprehensively investigated the bioactive compounds of *Rheum emodi* and their potential therapeutic impact on rheumatoid arthritis (RA). Initial screening identified six unique compounds with favourable drug-likeness, which collectively targeted 320 proteins. Network analysis revealed 27 gene overlaps with RA, highlighting five hub genes (MET, NFKB1, ABCG2, PARP1, ABCB1), wherein MET, NFKB1, and PARP1 were significantly upregulated in RA. Pathway enrichment analysis implicated these genes in lipid transport, cell communication, and key signa;ling pathways (NF-kappa B, PI3K-Akt, ABC transporters). Molecular docking and dynamics simulations indicated emodin exhibited strong, stable binding to MET and ABCG2 hubs. In vitro, *R. emodi* extract inhibited SW-982 cell viability (IC_{50} = 21.1/ µg/mL), induced apoptosis and ROS, and reduced cell migration. qRT-PCR confirmed downregulation of MET and NFKB1 post-treatment. Collectively, the findings support multi-target inhibitory actions of *R. emodi* extract in RA, underpinned by gene, pathway, and cellular effects.