Publications Template

#	Research Title	Field	Abstract	Year of Publication Publishing	Publishing Link "URL"
1	**Chloroquine modulates the sulforaphane anti-obesity mechanisms in a high-fat diet model: Role of JAK-2/ STAT-3/ SOCS-3 pathway**	Pharmacology and Toxicology	The phytochemical sulforaphane (SFN) has been studied for its potential anti-obesity effect, but neither its molecular targets nor its interaction with the antimalarial drug chloroquine (CQ) has been fully delineated. Therefore, high-fat diet (HFD) obese rats were randomly allocated into one of five groups and were left untreated or gavaged orally with SFN (0.5 or 1 mg/kg), CQ (5 mg/kg), or their combination (0.5/5 mg/kg) for six successive weeks to assess their potential interaction and the enrolled mechanisms. SFN effectively reduced the HFD-induced weight gain, blood glucose, and serum leptin levels, and improved lipid profile.	2022	https://www.scopus.com/record/display.uri?eid=2-s2.0-85131557108&origin=resultslist&sort=plf-f&src=s&sid=4378001eb0e69f1e6271d2a878a1b0b4&sot=b&sdt=b&s=TITLE-ABS-KEY%28Chloroquine+modulates+the+sulforaphane+anti-obesity+mechanisms+in+a+high-fat+diet+model%3A+Role+of+JAK-2%2F+STAT-3%2F+SOCS-3+pathway%29&sl=141&sessionSearchId=4378001eb0e69f1e6271d2a878a1b0b4
2	**Effect of Sulforaphane on Vascular Calcification in Adenine-Induced Chronic Kidney Disease**	Pharmacology and Toxicology	Vascular calcification (VC) is a convoluted process that leads to pathological accumulation of calcium phosphate crystals in the intima and media layers of the vessel wall that worsens the course of atherosclerosis,	-	In progress

			diabetes, and chronic kidney disease (CKD). These mineral-enriched plaques induce arterial stiffening, putting patients at risk for fibrosis, inflammation, and oxidative stress on a cellular level. It is well known that VC is a highly controlled process mainly regulated by the vascular smooth muscle cells (VSMCs). In the physiological conditions, VSMCs have a contractile phenotype and maintain the structural and functional homeostasis of the vessel wall. However, under pathological conditions, VSMCs transform into osteoblast-like cells and secrete collagenous extracellular matrix.		
3	Possible Protective Effects of Sulforaphane and Ginkgo Biloba Combination on Induced Alzheimer's Disease in Rats		Alzheimer's disease (AD) is one of the most prevalent neurodegenerative illnesses, and yet, no workable treatments have been discovered to prevent or reverse AD. Extracellular amyloid beta (A β) plaques and intracellular neurofibrillary tangles comprised hyper-phosphorylated Tau protein (pTau) represent the major hallmarks of AD. In addition, the downstream cognitive symptoms can be caused by non-A β factors including oxidative		In progress

			stress, inflammation, mitochondrial dysfunction and lipid perturbation. Despite the increasing incidence and prevalence of the disease, clinical studies on disease-modifying medication have largely failed, making AD therapy one of the most difficult fields of modern medicine. Moreover, all the scientific advances in recent decades that have increased our understanding of the cellular and molecular bases of AD, still, viable medicines to cure or slow the disease progression are very poor.Thus, these circumstances have enticed the researchers in this study to investigate natural replacement therapy, which have fewer side effects and are highly effective in managing AD and memory loss. Sulforaphane (SFN) is a phytocompound with antioxidant, anti-inflammatory, and antiapoptotic effects. Since oxidative stress, inflammation, and mitochondrial dysfunction are involved in neurodegenerative diseases, the interest of the scientific community for it has grown. Therefore, based on its properties, SFN was investigated		

			in main neurodegenerative diseases including Alzheimer's disease (AD), Parkinson's disease (PD), and multiple sclerosis (MS).		