OPINION

Central nervous system illnesses and salidroside's pharmacological effects

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ABSTRACT

Salidroside (SAL) is a high-altitude-extracted phenylpropanoid glycoside monomer from Rhodiola. It has been demonstrated to be neuroprotective in illnesses of the Central Nervous System (CNS) and to have protective effects against myocardial infarction, liver cancer, renal fibrosis, and other organ diseases. In particular, SAL can alleviate neurological dysfunction and a number of pathogenic reactions in CNS illnesses. The pharmacological effects of SAL on inflammation, oxidative stress, apoptosis, autophagy, and neural regeneration were clarified by this review. Additionally, it is evaluated how SAL influences different signaling pathways to control pathogenic processes in CNS disorders. Uncertainty persists regarding the connections between distinct routes and the processes at various phases of the disease process.

INTRODUCTION

The Himalayas, northwest Asia, and North America are where you may mostly find rhodiola, a perennial flowering herb of the genus *Rhodiola rosea*. It is a versatile plant with culinary and medicinal uses that thrives in chilly regions above 2000 meters above sea level. Phenylpropane glycoside chemical salidroside is obtained from Rhodiola plants. 2-[4-hydroxyphenyl] ethyl-D-glucopyranoside is its chemical name. Its molecular mass is 300.31 and its formula is C14H20O7. SAL has the appearance of a colorless, clear needle-like crystal and a melting point of roughly 160 °C. It disintegrates readily in methanol and water, but not in ether. Physical techniques such thermal reflux, ultrasonic, microwave, and ultra-high pressure can be used to directly extract SAL from natural plants; however, supercritical CO2 extraction best integrates the benefits of many techniques so that the extraction rate of SAL reaches 99.6%. Saccharomyces cerevisiae and glutamic acid strains can both be used to extract SAL. In addition, tyrosine can catalyze uridine diphosphate-dependent glycosyltransferase to produce tyrosol, which can subsequently be used to biosynthesize SAL. Currently, intravenous injections and gavage are two ways to administer SAL. After being administered to animals, tyrosol, a particular aglycone, is created by deglycosylating SAL and may be discovered in their tissues. According to other research, SAL can be broken down by acids or enzymes into aglycone and glucose. As a result, SAL can exert biological effects by being broken down into tyrosol in the stomach by gastric acid and digestive enzymes. The SAL liposomes bioavailability increases the area under the blood concentration-time curve and extends SAL's half-life and peak time. SAL has a 20 to 2 hour half-life that varies depending on the medication concentration and the type of animal. SAL is mostly dispersed throughout the body's organs with higher blood flow after it has been absorbed, and it is then eliminated by the liver and kidney. SAL has been used for a very long time throughout Europe, America, and Asia. It is widely used to decrease the body's responses to changes in altitude and to enhance cardiopulmonary function. Research has shown that SAL's protective impact is concentration-dependent. Additionally, different concentrations of SAL don't seem to have any harmful effects on healthy tissues or cells. As a result, it is a medication that is both reliable and efficient and is frequently employed in research. SAL has been shown in studies on numerous disorders to have anti-inflammatory, antioxidant, anti-aging, and anti-tumor

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properties. Cells respond defensively to stimulation, infections, and injury by inducing inflammation. Reactive Oxygen Species (ROS), nitric oxide, and other oxidants can all be produced by the recruited immune cells in addition to a huge range of cytokines and lysosomal enzymes. These elements promote localized cell growth and prevent localized cell apoptosis. Fibrosis and granuloma may develop if inflammatory responses are not gradually curbed or restricted. Important mediators in inflammatory pathways that contribute to the promotion of inflammation and fibrosis include Toll-Like Receptor 4 (TLR4), Nuclear Factor-B (NF-B), and Mitogen-Activated Protein Kinases (MAPK, including JNK, ERK, and p38). SAL inhibited monocyte adhesion to CMECs in TNF-treated Cardiac Microvascular Endothelial Cells (CMECs), attenuated the release of inflammatory mediators like IL-1, IL-6, and Monocyte Chemoattractant Protein-1 (MCP-1), and downregulated the NF-B and MAPK pathways, suggesting that SAL exerts a potential anti-inflammation therapeutic effect on atherosclerosis. The TLR4/NF-B and MAPK signaling pathways, as well as the production of inflammatory factors (IL-6, IL-1, and TNF-), as well as the deposition of collagen I and III and epithelial-interstitial transformation, as well as renal tubular injury and renal interstitial fibrosis, were all found to be inhibited by SAL, according to research. SAL was also discovered to suppress microglia adhesion and phagocytosis in a study using a depression model by deactivating the ERK1/2, p38, and NF-B signaling pathways and decreasing iNOS expression, hence lowering mental and behavioral dysfunctions. In addition, SAL inhibited IL-1-induced chondrocyte inflammation by decreasing inflammatory mediators like NO, prostaglandin-E2, iNOS, and COX-2, as well as the Matrix Metalloproteinases (MMP)-1, -3, and -13, which are linked to the breakdown of bone tissue protein. Signal Transducer and Activator of Transcription 3 (STAT3) is phosphorylated by Janus Kinase (JAK) to create a dimer that is transported into the nucleus and controls the activation of inflammatory genes. After hepatic hypoxia, SAL has been shown to reduce the generation of proinflammatory substances throughcontrolling the JAK/STAT3 pathway. By increasing pro-IL-1 and NLRP3 expression, the important inflammatory activator NF-B prepares the NLRP3 inflammasome for activation. Studies have shown that Sirt1, TXNIP, and AMPK are among the signal transducers that SAL uses to regulate the NF-B/NLRP3 signaling pathway. In order to control inflammation, SAL can also downregulate GSK-3 signaling. SAL therefore controls the TLR4/NF-B, MAPK/NF-B, and JAK/STAT3 pathways to modify inflammatory factors. ROS are produced by cell organelles and include a variety of oxidants (mitochondria, chloroplasts, peroxisomes, etc.). Superoxide Dismutase (SOD), Catalase (CAT), and glutathione peroxidase are examples of antioxidants that can become out of balance as a result of an excessive buildup of ROS (GSH-Px). This condition, known as "oxidative stress," aids in cell destruction and death. One typical consequence of diabetes is endothelial dysfunction brought on by oxidants. An earlier investigation found that SAL upregulated the accumulation of oxygen products (ROS and Malondialdehyde [MDA]) in vivo, downregulated the accumulation of antioxidant enzymes (SOD, CAT, and GSH-Px), and inhibited oxidative stress in diabetes-related vascular endothelial cells. By raising the levels of antioxidants (HO-1, SOD, GSH-Px, and NQO1) through the AMPK/Nrf2 pathway, SAL may also reduce oxidative stress brought on by Low-Density Lipoprotein (LDL) in the body. NADPH Oxidase 2 (NOX2) was downregulated andmitochondrial membrane potential was restored as a result of AMPK activation by SAL. When a cell is hypoxic, the Hypoxia-Inducible Factor-1 (HIF-1) can encourage glycolysis, which sustains the energy supply by generating ATP. Two crucial enzymes that support mitochondrial respiration are ISCU1/2 and COX10. According to studies, pretreatment with SAL boosted the activity of HIF-1, ISCU1/2, COX10, MiR-210, and oxidases, promoted the production of ATP in mitochondria, and prevented apoptosis in PC12 cells and microglia that had been exposed to COCl2-induced hypoxia.