

## Pharma Europe 2016 : The anti-inflammatory activity of Ginsenoside Rg3-enriched red Ginseng extracts in RAW264.7 cells and protects LPS-induced septic shock in mice - Man Hee Rhee- Kyungpook National University

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Ginseng, the root of *Panax ginseng* Meyer, had been used in Easter Asia, including Korea, China and Japan for a thousand of years. The potential benefits of ginseng range from energizing the body to providing the longevity. Ginsenoside Rg3 (Rg3), one of the most effective ginseng saponin, has anti-inflammatory and anti-cancer effects. Numerous previous studies have unraveled the Rg3 Pharmacological effects. One recent study has also revealed its protective cardiovascular effects in hyper sensitive rats. Here we investigated the anti-inflammatory activity of Rg3-enriched red ginseng extract in vitro on murine macrophages RAW 264.7 cells. We found that Rg3-enriched red ginseng extract in dose dependent manner attenuate the expression of proinflammatory cytokines after LPS induction. The MTT assay for dose dependent concentrations of Rg3- enriched red ginseng extract show no cytotoxicity and nitric oxide concentration show a decreasing trend with increase in dose. The mRNA expression of proinflammatory cytokines like IL-1 $\alpha$ , IL-6, iNOS, COX-2 and TNF- $\alpha$  show the same trend like that of nitric oxide production. Furthermore, it was found that a remarkable attenuation of inflammation by oral treatment of Rg3-RGE extract in the mice survival study with LPS-induced septic shock. Rg3 enriched ginseng fraction can be hailed as a potent anti-inflammatory therapeutic agent in future. Nonsteroidal mitigating drugs (NSAIDs) reduce torment by checking the cyclooxygenase (COX) enzyme. On its own, COX protein orchestrates prostaglandins, making aggravation. In entire, the NSAIDs keep the prostaglandins from consistently being incorporated, decreasing or taking out the agony. Some basic instances of NSAIDs are headache medicine, ibuprofen, and naproxen. The fresher explicit COX-inhibitors are not ordered along with the conventional NSAIDs despite the fact that they probably share a similar method of activity. Then again, there are analgesics that are usually connected with calming drugs yet that have no mitigating impacts. A model is paracetamol (known as acetaminophen or Tylenol in the U.S). Rather than NSAIDs, which decrease agony and aggravation by restraining COX proteins, paracetamol has - as ahead of schedule as 2006 - been appeared to obstruct the reuptake of endocannabinoids, which just lessens torment, likely clarifying why it has negligible impact on irritation; paracetamol is now and again joined with a NSAID (instead of a narcotic) in clinical practice to improve the help with discomfort of the NSAID while as yet getting the injury/illness tweaking impact of NSAID-initiated aggravation decrease (which isn't gotten from narcotic/paracetamol blends). Nonsteroidal calming drugs (NSAIDs) are individuals from a medication class that diminishes torment, diminishes fever, forestalls blood clots, and in higher dosages, diminishes irritation. Reactions rely upon the particular medication yet to a great extent incorporate an expanded danger of gastrointestinal

ulcers and drains, coronary episode, and kidney disease. The term nonsteroidal recognizes these medications from steroids, which while having a comparable eicosanoid-discouraging, mitigating activity, have a wide scope of different impacts. First utilized in 1960, the term served to separate these prescriptions from steroids, which were especially derided at the time because of the implications with anabolic steroid abuse. NSAIDs work by repressing the action of cyclooxygenase catalysts (COX-1 or COX-2). In cells, these proteins are associated with the union of key natural middle people, to be specific prostaglandins, which are engaged with irritation, and thromboxanes, which are associated with blood thickening. There are two sorts of NSAIDs accessible: non-specific and COX-2 selective. Most NSAIDs are non-particular and repress the movement of both COX-1 and COX-2. These NSAIDs, while lessening irritation, likewise repress platelet accumulation (particularly anti-inflammatory medicine) and increment the danger of gastrointestinal ulcers/bleeds. COX-2 specific inhibitors have less gastrointestinal reactions however advance apoplexy and considerably increment the danger of cardiovascular failure. Thus, COX-2 specific inhibitors are commonly contraindicated because of the high danger of undiscovered vascular disease. These differential impacts are because of the various jobs and tissue localisations of each COX isoenzyme. By repressing physiological COX movement, all NSAIDs increment the danger of kidney disease and through a related instrument, heart attack. moreover, NSAIDs can dull the creation of erythropoietin bringing about sickliness, since hemoglobin needs this hormone to be delivered. Delayed use is hazardous and contextual analyses have indicated the wellbeing hazard with celecoxib. The most unmistakable NSAIDs are anti-inflammatory medicine, ibuprofen, and naproxen, all accessible over the counter (OTC) in most countries. [8] Paracetamol (acetaminophen) is commonly not considered a NSAID since it has just minor mitigating action. It treats torment basically by blocking COX-2 and hindering endocannabinoid reuptake solely inside the cerebrum, however very little in the remainder of the body. Headache medicine, the main NSAID ready to irreversibly hinder COX-1, is likewise demonstrated for antithrombosis through hindrance of platelet accumulation. This is helpful for the administration of blood vessel apoplexy and counteraction of antagonistic cardiovascular occasions like coronary failures. Anti-inflammatory medicine represses platelet conglomeration by restraining the activity of thromboxane A<sub>2</sub>. In an increasingly explicit application, the decrease in prostaglandins is utilized to close a patent ductus arteriosus in neonates on the off chance that it has not done so physiologically after 24 hours. NSAIDs are valuable in the administration of post-employable dental torment following intrusive dental methods, for example, dental extraction. When not contra-demonstrated they are preferred over the utilization

of paracetamol alone because of the mitigating impact they provide. When utilized in blend with paracetamol the pain relieving impact has been demonstrated to be improved. There is powerless proof recommending that taking pre-usable absense of pain can lessen the length of post usable agony related with putting orthodontic spacers under neighborhood anaesthetic. Combination of NSAIDs with pregabalin as preemptive absense of pain has indicated promising outcomes for diminishing post employable torment intensity. The viability of NSAIDs for rewarding non-malignant growth ceaseless torment and disease related torment in kids and young people isn't clear. There have not been adequate quantities of top notch randomized controlled preliminaries directed

#### Biography

Man Hee Rhee graduated in Veterinary Medicine in 1989 and completed his MS degree in 1991 and then PhD degree in 2000 in Neurobiology Department, Weizmann Institute of Science, Israel. He then spent 3 years in the Department of Cell Biology & Physiology, Washington University School of Medicine, USA as a Research Associate. He has joined in Kyungpook National University as Associate Professor. He has published more than 150 papers in reputed journals and has been serving as an Editorial Board Member of repute.

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