

Broccoli dry extract



Broccoli extract has antioxidant and **anticancer** properties due to its components, among which sulforaphane stands out. Since the first isolation of broccoli, **sulforaphane** was shown to have cancer chemoprotective properties in rats. In the early 1990s, more than 3000 publications have described its efficacy in rodent disease models, underlying mechanisms of action or, to date, more than 50 clinical trials examining pharmacokinetics, pharmacodynamics, and disease mitigation (1).

Helicobacter pylori infection causes gastric ulcers due to an increase in pH in the stomach caused by the production of ammonia. Sulforaphane has demonstrated its function to inhibit this production, so it could also be used to treat H. Pylori infection (1,3).

Antioxidant effect

Its antioxidant activity may be due, among other mechanisms, to the increase in Glutathione (GSH), one of the most important regulators of oxidative stress. A decrease in its activity seems to be involved in many diseases. Sulforaphane has been shown to be involved in an increase in GSH, and therefore reduces the damage caused by oxidative stress.

High bioavailability

Furthermore, it has been shown to have an absolute bioavailability (figure 1) of around 80%, while other antioxidant molecules exhibit a bioavailability of around 1-8%.

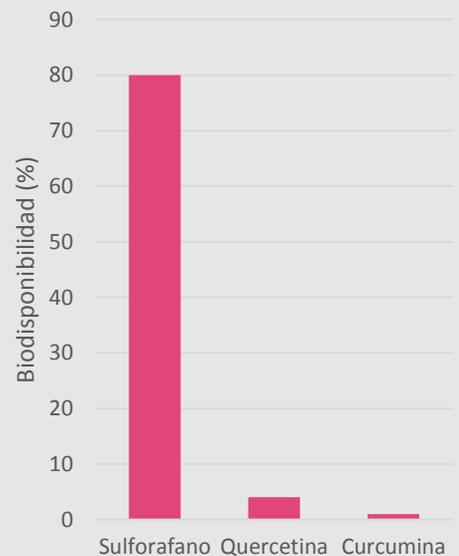


Figura 1. Biodisponibilidad de sulforafano, quercetina y curcumina.

Antiinflammatory Effect

There are also many studies on the anti-inflammatory effect of Sulforaphane. Figure 2 shows a study in which 30 grams of broccoli were administered daily to a group of healthy, overweight people. In a period of 70 days, it was found that markers of inflammation such as interleukin-6 and C-reactive protein decreased considerably.

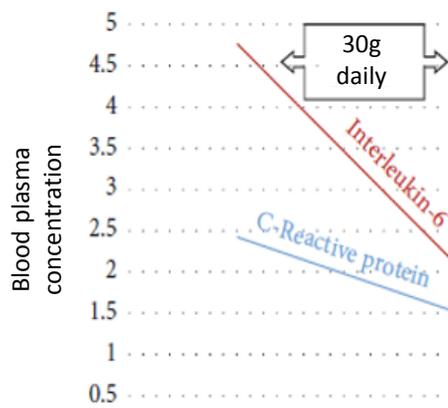


Figure 2. Markers of inflammation in blood plasma after ingesting 30 grams of broccoli daily. Study conducted in healthy overweight patients.

Diabetes type 2

Sulforaphane has been shown to reduce hepatic glucose production and improve glucose control in patients with type 2 diabetes (Figure 3). This action is due to the activation of transcription factors and intracellular mechanisms that modulate glucose production in hepatocytes.

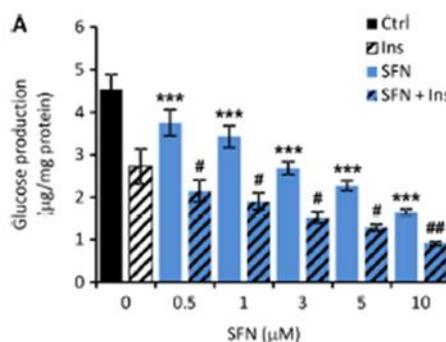


Figure 3. Glucose production in hepatocytes after the administration of insulin (Ins), sulforaphane (SFN) and SFN + Ins.

References

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