Acacia gum (gum arabic)

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Acacia gum (AG) is the dried gum of the stem and branches of acacia trees (family leguminosae) and various other acacia trees throughout the world and it is often referred to commercially as gum arabic. AG is a complex polysaccharide consisting mainly of calcium salts of polyarabic acid, but also contains magnesium and potassium ions. It is a high molecular weight polysaccharide molecule containing D-galactopyranose, D-glucuronic acid, L-rhamnopyranose and L-arabofuranose. On hydrolysis, acacia yields hexoses, arabinose, galactose, rhamnose and glucuronic acid. AG is generally recognized as safe by the US FDA. It is widely used in the production of foods such as puddings, frostings, candy, beverages and chewing gum. It has demulcent properties and is often added to medicines for that purpose [1–4]. AG is a water soluble fermentable polysaccharide resistant to gut enzymes and thus can be described as a dietary fiber. The principle fermenter bacteria capable of using acacia as the only carbohydrate source are bacteriods and bifidobacterium. The proportion of these flora rise after acacia ingestion and return to initial levels after cessation of ingestion. AG is completely degraded in the colon [5].

The energy value of AG is 14.7 ± 0.5 kJ/g, lower than the energy value of starch 17.4 ± 0.4 kJ/g [6]. AG administered to men for 3 weeks has no effect on glucose tolerance, but decreases serum cholesterol [7].

The introduction of a therapeutic agent into clinical use has always been associated with fear of toxic effects. However, it is interesting to note that in experimental studies on animals, AG has been shown to have a protective effect against the toxicity of a number of drugs, such as gentamicin, acetaminophen and doxorubicin. In addition, AG has no teratogenic or carcinogenic properties [8–11]. In rats, AG has no histopathological or hematological toxicity when administered for 13 weeks at doses as high as 5 g/kJ/day. It has been suggested that the renoprotective effect of AG is possibly through inhibition of the production of oxygen-free radicals that cause perodxidation. However, this has yet to be proven. It has, however, been shown that AG has no effect on the concentration of some free-radical scavengers (reduced glutathion, ascorbic acid, lipid perodxidation and superoxide dismutase) on the kidneys and liver of healthy rats [12–16].

There are no limitation to the use of AG as a food additive as the experimental evidence of safety demanded by the international food safety has already been met [17].

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