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A SHORT REVIEW ON THE CHEMICAL COMPOSITION AND THE APPLICATIONS OF CHEST NUT

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Abstract:

Chestnuts, unlike other nuts and seeds, are relatively low in calories, carry less fat, but are rich sources of minerals, vitamins and phyto-nutrients that immensely benefit health.Another unique feature of chestnuts is that they chiefly made of starch in contrast to other seeds and nuts, which are high in calorie, protein, and fat. Chestnuts nutrition composition is, therfore, comparable to that of other staple starch foods such as sweet potato, sweet corn, potatoes, plantain, etc., Nevertheless; they are still good sources of minerals, vitamins and some good-quality protein than cereals and tubers.

Key words: *Chestnuts, vitamins and phyto-nutrients.*

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INTRODUCTION:

The fruit is a capsule with a thick, leathery husk that contains the dark nuts. As the husk dries, the nuts are released. The pink and white flowers of the plant grow in clusters. The horse chestnut is native to the Balkan region of southeastern Europe and western Asia, but now is cultivated worldwide[1-3]. The dried ripe seeds of the plant are of most medicinal interest.chestnuts have been used in traditional medicine and for a variety of other commercial applications for centuries. Extracts of the bark have been used as a yellow dye, and the wood has been used for furniture and packing cases. In the western United States, the crushed unripe seeds of the California buckeye were scattered into streams to stupefy fish, and leaves were steeped as a tea to remedy congestion. The horse chestnut has been used as a traditional remedy for arthritis and rheumatism, as well as for gynecological bleeding and as a tonic. Even though the seeds are toxic, several traditional methods were employed to rid them of their toxicity. Seeds were buried in swampy, cold ground during the winter to free them of toxic, bitter components, then eaten in the spring after boiling[4-7]. American Indians roasted, peeled, and mashed the poisonous nuts, then leached the meal in lime water for several days, creating a meal used to make bread.

Chestnuts belong to the family Fagaceae, which also includes oaks and beeches. The four main species are commonly known as European, Chinese, Japanese, and American chestnuts, some species called **chinkapin** or **chinquapin**

- **European species** sweet chestnut (Castanea sativa) (also called "Spanish chestnut" in the US) is the only European species of chestnut, though it was successfully introduced to the Himalayas and other temperate parts of Asia. Unrelated but externally similar species of horse chestnut are abundant around Europe.
- **Asiatic species** Castanea crenata (Japanese chestnut), Castanea mollissima (Chinese chestnut), Castanea davidii (China), Castanea henryi (Chinese chinkapin, also called Henry's chestnut – China) and Castanea seguinii (also called Seguin's chestnut – China)
- **American species** These include Castanea dentata (American chestnut – Eastern states), Castanea pumila (American- or Allegheny chinkapin, also known as "dwarf chestnut" – Eastern states), Castanea alnifolia (Southern states), Castanea ashei (Southern states), Castanea floridana (Southern states) and Castanea paupispina (Southern states)[8].

Chestnuts should not be confused with horse chestnuts (genus Aesculus), which are not related to Castanea and are named for producing nuts of similar appearance, but which are mildly poisonous to humans, nor should they be confused with water chestnut (family Cyperaceae), which are also unrelated to Castanea and are tubers of similar taste from an aquatic herbaceous plant [7,8]. Other trees commonly mistaken for chestnut trees are the chestnut oak (Quercus prinus) and the American beech (Fagus grandifolia), [9-10] both of which are also in Fagaceae.

Medical uses

Chronic venous insufficiency (CVI) is a disorder affecting approximately 25% of the European community, women in particular (Piechal et al., 2005). CVI is caused by inborn or acquired anomalies in the functioning of the venous system, resulting from primary defects in a vein wall and valve structure as well as insufficiency thereof, and by factors influencing the weakened tension and structure thereof, such as hormonal changes, pregnancy, obesity, limited activity, working in a sitting or standing position, and oral contraceptives (Sudoł-Szopińska et al., 2006). As a result of the weakened vascular tension and structure, blood congestion, vascular bed overflow, and hypoxia occur; in consequence, mitochondrial oxidative phosphorylation is inhibited, and the content of adenosine triphosphate (ATP) is lowered. A decrease in ATP content in endothelium cells induces a series of cellular modifications, such as an increase in cytosolic calcium concentration, the release of inflammatory mediators, such as prostaglandins (Michiels et al., 1993), and the platelet-activating factor (PAF) (Arnould et al., 1993) which result in the recruitment, activation and adhesion of polymorphonuclear neutrophils (Arnould et al., 1996). Leukocytes adhering to the vascular wall release phospholipase A_2responsible for production of inflammation precursors, toxic oxidative metabolites, and lysosomal enzymes (elastase, collagenase). They also lead to the increased activity of hyaluronidase that degrades hyaluronic acid, the major constituent of capillary endothelium. The increased activity of other enzymes being components of vascular walls, *i.e.* β-*N*-acetylglucosaminidase, β-glucuronidase and arylosulphatase, responsible for degradation of proteoglycans, has also been observed in chronic venous insufficiency. Degradation of hyaluronic acid and proteoglicans results in violation of the integrity of blood vessel walls, increased capillary permeability, and fragility (ESCOP, 2003). In the course of an inflammatory reaction, histamine and

serotonin – the enzymes affecting the increase in capillary permeability – are released, too.

Chemical constituents

Horse chestnut seeds are rich in saponins (3–5%), over thirty of which have been isolated and identified. The main compound is aescin – a mixture of acylated triterpene glycosides. Three fractions of aescin, denoted as crypto-, α-, and β-aescin have been described in the literature. Cryptoaescin contains C-28-*O*-acetyl saponins, and β-aescin contains C-22-*O*-acetyl saponins, whereas α-aescin is a mixture of crypto- and β-aescin. β-Aescin (mainly made up of aescin Ia (**1**) and aescin Ib (**2**)) is the major active component of extracts from horse chestnut seeds, whereas α-aescin (made up mainly of isoaescin Ia (**5**) and isoaescin Ib (**6**)) is less bioactive (ESCOP, 2003). Horse chestnut seeds also contain flavonoids: quercetin and kaempferol derivatives, proanthocyanidins, sterols, and significant amounts of starch (ESCOP, 2003).

The effectiveness of HCSE and aescin as the treatment for chronic venous insufficiency results from the anti-oedematous and anti-inflammatory activity as well as their influence on the tension of blood vessel walls. The biological activity of both horse chestnut seed extract (HCSE) standardised for the content of aescin and isolated aescin has been confirmed by numerous *in vitro*, *in vivo*, and the clinical studies (Pittler and Ernst, 2012)

Anti-inflammatory and anti-oedematous activity

It has been confirmed that aescin is mainly responsible for the anti-inflammatory and anti oedematous activity of the horse chestnut seed extract (Table 1). The research results have shown that the complete extract has been around 100 times more effective as the treatment for inflammation and lymphoedema in rats than the extract from which aescin was removed (Guillaume and Padioleau, 1994).

The mechanism of the anti-inflammatory and anti-oedematous activity of HCSE and aescin is multi-directional and has been proposed on the basis of results of *in vitro* and *in vivo* studies . It was demonstrated that aescin counteracted ATP reduction and an increase in the activity of phospholipase A_2 responsible for the release of precursors of inflammatory mediators (Arnould et al., 1996 and Sirtori, 2001). Moreover, aescin reduced neutrophil adhesion and aggregation, which was shown in *ex vivo* studies on an isolated human umbilical vein. Incubation thereof in hypoxic conditions in an aescin solution or without it proved that aescin inhibited adhesion of neutrophils to the vascular endothelium, activated them, and produced leukotriene B4 and the superoxide anion (Bougelet et al., 1998). In an experimental model of rat pleurisy, HCSE reduced plasma extravasation and leukocyte migration to the pleural cavity as well as the release of inflammatory mediators, which resulted in inhibition of inflammation and oedema (Guillaume and Padioleau, 1994). In *in vitro* studies, the aescin restrained hyaluronidase activity by 93%, which resulted in inhibition of permeability and the loss of plasma from the cells of vascular endothelium and, as a consequence, in a decrease in the occurrence of oedema (Facino et al., 1995). In addition, aescin decreased the activity of lysosomal enzymes, which shifted the proteoglican synthesis-breakdown balance towards synthesis thereof. *In vivo* studies showed that aescin administered intraperitoneally to rats for three weeks significantly reduced degradation of mucopolysaccharides in the connective tissue (the xiphoid process) (Panigati, 1992). Aescin and HCSE are also characterised by the antihistaminic and antiserotonin activity. It was demonstrated that HCSE administered orally to rats diminished or inhibited excessive permeability of skin capillaries caused by previous administration of histamine and serotonin (Guillaume and Padioleau, 1994). The later research proved that aescin Ib (**2**), IIa (**3**), and IIb (**4**) had the antihistaminic and anti-serotonin activity while aescin Ia (**1**) mainly inhibited histamine (Matsuda et al., 1997). The anti-exudative activity of aescin is also connected with selective sensitisation of vascular smooth muscles to Ca^{2+} ions, which leads to the increased tension and sealing thereof and, as a result, to a decrease in the inflammation caused by vascular endothelium hypoxia. Results of *in vitro* studies confirmed the above. Aescin caused concentration-dependent contraction rings of inferior vena cava from male rats incubated in normal Krebs. In Ca^{2+-} free Krebs there was essentially no contraction to aescin, but in aescin-treated veins incubated in Ca^{2+}free Krebs, stepwise addition of extracellular $CaCl_2$ caused corresponding increases in contraction (Raffetto and Khalil, 2011).

Side effects

The effectiveness of HCSE and aescin as the treatment for chronic venous insufficiency is also connected with the influence on the tension of veins. As a result of an increase in the tension of vein walls, the blood flow through the vessels is accelerated and venous outflow improves. The consequence is that venous blood congestion decreases, which, in turn, enhances microcirculation as erythrocyte dispersion increases and tissue oxygenation improves, the flow through *vasa vasorum* accelerates, the time of leukocyte migration through the blood vessels and,

therefore, the chance for activation thereof initiating the cascade of an inflammatory reaction is lowered.
Weak genotoxic activity of a commercial dry extract as well as fluid extracts of horse chestnut seeds was demonstrated in the Ames test on strains of *Salmonella typhimurium* TA 98 (EMEA, 2012 and ESCOP, 2003).
HCSE studies on rats and rabbits (100 and 300 mg/kg bw) showed no teratogenic activity. A fall in the average body weight of the foetus was only observed after application of large doses of HCSE.
HCSE single dose toxicity studies (Venostatin retard preparation, standardised for the content of 50 mg of aescin in 240–290 mg extract) conducted on animals (mice, rats, guinea pigs, and rabbits) demonstrated greater toxicity of the extract when administered intravenously and intraperitoneally (LD_{50} 6.8–465 mg/kg bw) than after oral administration (LD_{50} 910–2600 mg/kg bw). The LD_{50} values ranged from 3 to 17 mg/kg bw after intravenous and intraperitoneal administration of aescin to laboratory animals (EMEA, 2012).
The studies of chronic oral toxicity of HCSE were carried out on dogs (20, 40, 80 mg/kg bw; 5 days/week/3 month) and rats (100, 200, 400 mg/kg bw; 5 days/week/3 month). The obtained results proved a lack of toxicity of the administered extract.
In the study of sub-acute intravenous toxicity, HCSE was administered to rats at doses of 9, 30, 90 mg/kg/day for 60 days. The dose of 90 mg/kg caused death of 8 out of 30 animals as early as in the first days of the study; the doses of 30 and 9 mg/kg did not lead to any significant disorders and were safe for the animals (Liehn et al., 1972).
Aescin, at a dose of 2 × 50 mg/kg bw, given to young, 32-day old rats, did not affect fertility, and nephrotoxicity was not detected either (EMEA, 2012).
A lack of the nephrotoxic properties of aescin was also confirmed in a study on the influence of free and albumin-bound aescin on renal tubular transport performed on an isolated, artificially perfused frog kidney[11]. It was proven that the low harmfulness resulted from the ability of aescin to bind with plasma proteins (50%), and the concentration of free aescin filtered through the kidney was considered too low to be able to have a toxic effect (Barnes et al., 2007).

CONCLUSION:

The nuts are an excellent source of minerals such as iron, calcium, magnesium, manganese, phosphorus and zinc, besides providing a very good amount of potassium (518 mg / 100 g). Potassium helps counter hypertensive action of sodium, lowers heart rate and blood pressure. Iron helps prevent microcytic-anemia. Magnesium and phosphorus are important components of bone metabolism. Further, they are also rich in many important B-complex groups of vitamins. 100 g of nuts provide 11% of niacin, 29% of pyridoxine (vitamin B-6), 100% of thiamin, and 12% of riboflavin.Chestnuts, like hazelnuts and almonds, etc., are free from gluten. And for the same reason, they are one of the popular ingredients in the preparation of gluten-free food formulas intended for use in gluten-sensitive, wheat allergy, and celiac disease patients.

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