

Emerging clinical and therapeutic applications of *Nigella sativa* in gastroenterology

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Abstract

Nigella sativa (*N. sativa*) decreases DNA damage and thereby prevents initiation of carcinogenesis in colonic tissue secondary to exposure to toxic agents such as azoxymethane. *N. sativa* is of immense therapeutic benefit in diabetic individuals and those with glucose intolerance as it accentuates glucose-induced secretion of insulin besides having a negative impact on glucose absorption from the intestinal mucosa. *N. sativa* administration protects hepatic tissue from deleterious effects of toxic metals such as lead, and attenuates hepatic lipid peroxidation following exposure to chemicals such as carbon tetrachloride.

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Key words: *Nigella sativa*; Thymoquinone; Colon cancer; Glutathione-S transferase; Schistosomiasis

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TO THE EDITOR

Yildiz *et al*^[1] in their recent article, published in the September issue of the “*World Journal of Gastroenterology*”, have clearly highlighted the efficacy of *Nigella sativa* (*N. sativa*) in relieving the deleterious effects of ischemia reperfusion

injury in the liver. Their findings bring into highlight the increasing clinical and therapeutic applications of *N. sativa* and its derivatives in the field of gastroenterology.

N. sativa decreases DNA damage and thereby prevents initiation of carcinogenesis in colonic tissue secondary to exposure to toxic agents such as azoxymethane^[2]. In fact, sustained delivery of thymoquinone (derived from *N. sativa*) is almost as effective in causing apoptosis of colon cancer cells as sustained delivery of 5-fluorouracil^[3]. Similarly, hepatic metastasis from tumors such as mastocytomas is markedly decreased following administration of *N. sativa*^[4]. *N. sativa*, when used in combination with *Hemidesmus indicus* and *Smilax glabra*, also seems to decrease hepatic carcinogenesis secondary to exposure to agents such as diethylnitrosamine^[5]. These anti-carcinogenic effects are mediated in part by thymoquinone secondary to its inhibitory influence on the NF-κB activation pathway^[6].

N. sativa is of great therapeutic benefit in diabetic individuals and those with glucose intolerance, as it accentuates glucose-induced secretion of insulin, besides having a negative impact on glucose absorption from the intestinal mucosa^[7,8]. In fact, *N. sativa* attenuates the damage to β-cells of the pancreas following exposure to toxic elements such as cadmium^[9]. Similarly, *N. sativa* administration attenuates the ulcerative effects of ethanol on gastric mucosa by decreasing the glutathione-S transferase levels in gastric mucosa^[10].

Besides these effects, *N. sativa* also demonstrates anti-parasitic effects. For instance, its administration decreases the number of eggs as well as worms in schistosomiasis, which tends to affect hepatic and intestinal tissues^[11]. In addition, *N. sativa* attenuates the side effects associated with some common medications used by gastroenterologists. For instance, cyclosporine, used by gastroenterologists for disorders such as recalcitrant Crohn's disease, is often associated with nephrotoxic side effects, which can be limited by *N. sativa* due to its anti-oxidant properties^[12]. Similarly, *N. sativa* administration protects hepatic tissue from deleterious effects of toxic metals such as lead and attenuates hepatic lipid peroxidation following exposure to chemicals such as carbon tetrachloride^[13,14].

Thymoquinone, derived from *N. sativa*, has also been demonstrated to induce apoptosis of human colon cancer cells^[15]. The above examples clearly illustrate the massive clinical and therapeutic potential of *N. sativa*.

Personally, I believe that the anti-carcinogenic effects of *N. sativa* hold the maximum therapeutic potential. Given the significant benefits associated with its administration, broad-spectrum studies are clearly and urgently needed to further assess and elaborate its therapeutic benefits in gastroenterology.

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