Nitric oxide synthase activation is a unique mechanism of garlic action.

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Garlic (Allium sativum L.) is thought to decrease blood pressure, inhibit platelet aggregation, reduce blood fat and cholesterol as well as possessing antibacterial and antifungal properties [1]. The main active agents in garlic extracts are allicin and ajoene, but several other chemicals have been isolated and studied [2,3]. Nitric oxide (NO) is linked to hypertension [4] and the relevant properties of garlic prompted us to investigate whether the above mentioned effects of garlic extracts are due to the activation of nitric oxide synthase (NOS), the enzyme that produces NO.

Garlic extracts were prepared by 1% alcoholic extraction of fresh cloves of garlic or from Kwai garlic tablets (gm/mL). This was diluted and the effects upon calcium-dependent NOS activities determined in 2 different in vitro systems. Human platelets were isolated and prepared for assaying NOS activity in the presence and absence of garlic extracts at different dilutions. Garlic extracts were also added to human placental villous tissue and incubated at 37°C for 45 min. Tissues were then separated by centrifugation and supernatants removed for determination of nitrite concentrations by chemiluminescence [5]. The incubated tissues were rapidly frozen and homogenates prepared for determination of NOS activities. Assays were performed in platelets and homogenates by following the conversion of radiolabelled L-arginine to L-citrulline in the presence and absence of the specific inhibitor, N'-monomethyl-L-arginine [6].

NOS activity increased in a dose dependent manner in platelet preparations after incubation with garlic extract (Fig. 1). The incubation of placental tissues with garlic extracts also showed an increase in NOS activity and the metabolite, nitrite, in a dose dependent manner (Fig. 2). NOS activities are suppressed at higher concentrations of garlic, although NO production must have increased, as reflected by increased nitrite concentrations. This is probably due to NO acting as a feedback modulator of its own synthesis [7].

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