

# Exploring the mango – poison ivy connection: the riddle of discriminative plant dermatitis

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A relationship between sensitivity to poison oak or poison ivy and mango dermatitis has been suggested by previous publications. The observation that acute allergic contact dermatitis can arise on first exposure to mango in patients who have been sensitized beforehand by contact with other urushiol-containing plants has been documented previously. We report 17 American patients employed in mango picking at a summer camp in Israel, who developed a rash of varying severity. All patients were either in contact with poison ivy/oak in the past or lived in areas where these plants are endemic. None recalled previous contact with mango. In contrast, none of their Israeli companions who had never been exposed to poison ivy/oak developed mango dermatitis. These observations suggest that individuals with known history of poison ivy/oak allergy, or those residing in area where these plants are common, may develop allergic contact dermatitis from mango on first exposure. We hypothesize that previous oral exposure to urushiol in the local Israeli population might establish immune tolerance to these plants.

**Key words:** allergic contact dermatitis; Anacardiaceae; *Mangifera*; mango dermatitis; poison ivy; poison oak; *Toxicodendron*; urushiol. © Blackwell Munksgaard, 2005.

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The mango tree (*Mangifera indica*) is cultivated worldwide. Allergic reactions to mango have been described both after ingestion of the intact unpeeled fruit and after contact with the plant, in particular with its stem (1–3). However, it is not commonly recognized that a skin reaction to mango may develop on first exposure to the plant. Such a reaction may be caused by presensitization to long-chain phenols present in both poison ivy and poison oak and various parts of the mango plant. We wish to describe our experience with a group of 17 American youths visiting Israel, all of whom came from a part of the US where poison ivy sensitivity is endemic. All developed a rash after having been employed in mango picking.

## Case Reports

17 individuals from a group of 32 tourists visiting Israel presented at our dermatology clinic with a rash. The permanent residence of these 17 patients was the Oakland Bay area, Northern California. The other individuals of the group lived in other states of the US where poison oak/poison ivy sensitivity is not known. Their mean age was  $16.7 \pm 2.5$ , the group comprising

11 males and 6 females. 14 patients had a widespread eczematous urticarial rash. The distribution of the rash was linear in 14 patients and photosensitive in 4. The patients with the photosensitive distribution had mild eczema on their arms and forearms. 1 week prior to the presentation, the entire group has been employed in mango picking for a few hours every day at a kibbutz in Northern Israel in the vicinity of the sea of Galilee. Although a group of about 30 Israeli youths had been involved simultaneously in the same activity of mango picking, none of the Israeli participants had developed any dermatological manifestations. Detailed interview showed that among the American tourist group with a rash, there was no exposure to cosmetics, soaps or special food common to all subjects with skin involvement. Likewise, no difference was found in the dietary or working conditions of the American tourists and their Israeli companions. The common feature of all subjects who had a severe rash was a history of previous exposure to poison oak and/or poison ivy. 3 of the 17 patients, who had a mild rash only, had no history of prior reaction to poison ivy or oak but had also lived in an area where the plant is endemic.

1 patient presented with severe eczema with secondary infection. None of the patients had fever, angioedema or respiratory complaints. 2 patients within the group required hospital admission and were treated by intravenous corticosteroids, with rapid resolution of symptoms. The rest of the group was managed by topical corticosteroids, with subsequent relief of symptoms.

### Discussion

Mango dermatitis has been described as urticarial and eczematous in character with a linear distribution (1). The features of dermatitis observed in the present group of patients are in agreement with these characteristics. The diagnostic enigma in the present outbreak of mango dermatitis was the sharp distinction between the presence of dermatitis in all the American youths involved in the mango-picking activities and the complete absence of such symptoms among the young Israelis participating in the same activities.

Prior exposure to poison oak or poison ivy has been documented as the most common sensitizing factor in the development of mango dermatitis. In the USA, over 50% of the general population may be allergic to poison ivy, and it can be assumed that in some areas, the incidence of sensitization may be even higher (4). The cross reactivity between poison oak, poison ivy, mango and other plants of the Anacardiaceae family, genus *Toxicodendron*, is due to the presence of alk(en)yl-catechols (i.e. urushiol) in these plants (1). The non-volatile composition of mango latex was investigated and 5-resorcinol was identified as mango dermatitis allergen. Besides the allergenic properties, the catechols, resorcinol and phenols act as preservatives against microbial infections (5).

The clinical features, course and pathogenesis of mango dermatitis are essentially the same as those of poison ivy dermatitis. Keil et al. (3) documented the relationship between mango dermatitis and hypersensitivity to poison ivy by showing that most of their patients who had a positive response to a patch test with 3-n-pentadecyl catechol (which is a standard agent for poison ivy allergy) also had a positive reaction to mango-stem sap. These observations might explain the high incidence of antecedent poison ivy dermatitis among those contracting mango dermatitis reported in the literature.

Several findings in the present report support a connection between poison ivy sensitivity and mango dermatitis: (1) all subjects with mango dermatitis were residents of Northern California

where exposure to poison ivy and poison oak is very common, and several patients had actually developed allergic skin reactions after exposure to these plants in the past, (2) most, if not all, of them stated that they had never been in contact with mango previously. None of the Israeli-born members of the same group developed mango dermatitis as compared with a prevalence of 100% among all the 17 American subjects with a typical rash of varying degree of severity. This discriminative nature of mango dermatitis in relation to the 2 groups indicates that it is not exposure to mango per se, but previous sensitization, most probably to poison ivy and oak, that is the critical factor determining the presence or absence of mango dermatitis. An interesting question is why Israelis do not develop allergic contact dermatitis from urushiol? Geroso and Elpern (6) described the low incidence of mango dermatitis in people born in the Hawaiian islands as opposed to newcomers or visitors. They speculated that if the primary exposure to urushiol is through the skin from contact with poison oak/poison ivy, these individuals will develop delayed-type hypersensitivity to urushiol. Conversely, if the first exposure to urushiol occurs through ingestion, it is presented to the gut-associated lymphoid tissue allowing the induction of specific immunological tolerance.

Several studies have examined the relationship between prior enteral or cutaneous exposure to the offending agent (such as poison ivy) and prevention of cutaneous sensitivity. These studies were performed on animal models and showed contradictory results. Chase (7) prevented cutaneous sensitization to 2:4 dinitrochlorobenzene in guinea pigs by prior feeding of the chemical. In contrast, Wechsler (8) showed that prior feeding or injection of substances related to poison ivy did not protect guinea pigs against contact-skin sensitization to these agents. More recently, Ikeda et al. (9) published a study in which they had examined the relationship between epicutaneous and oral hyposensitization to urushiol in an experimental model of contact dermatitis in guinea pigs. Their findings showed that the hyposensitizing potency of repeated oral dosing was greater than that of repeated epicutaneous dosing. The potency of combined dosing was superior to that of either oral or epicutaneous dosing alone. Furthermore, the phenomenon of cutaneous hyposensitization following oral desensitization has been documented for other allergens such as nickel, Kerosuo et al. (10) showing that adolescents wearing nickel-releasing orthodontic braces before ear piercing had a lower incidence of nickel allergy than subjects wearing no braces

at all or with braces only after ear piercing. The mechanism of the induction of immune tolerance is not fully understood. It has been found that oral nickel chloride administration to naïve mice-induced tolerance due to persistent suppressive T-cell activity (11).

*Toxicodendron* (Rhus) and related plants are rare in Israel as well as mango which is a tropical plant cultivated only in a few rural areas. In most Israelis, the first exposure to urushiol is through ingestion. Along with the hypothesis offered by Geroso and Elpern (6) and evidence available from previous studies, one can hypothesize that Israelis acquire immunological tolerance to urushiol and, therefore, do not develop any rash upon mango picking.

Finally, we suggest that the likelihood of mango dermatitis in patients presensitized to poison ivy or to poison oak mandates special care and preventive measures, such as protection from direct contact with mango leaves, stems and skin of the mango fruit, in subjects with a known history of poison ivy allergy or in those who live in areas where such a problem is endemic.

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