Phytophotodermatitis caused by contact with a fig tree 

*(Ficus carica)*

José G B Derraik, Marius Rademaker

**Abstract**

Two arborists presented acutely with blistering eruptions affecting their forearms, hands, and fingers. The previous day, both men had pruned branches from a large fig tree, *Ficus carica*, which had sustained damaged during a storm. The following morning, both complained of a burning discomfort which rapidly evolved into erythema and bullae on skin that had been in direct contact with the tree branches. These symptoms gradually resolved over 4 to 6 weeks. Although phytophotodermatitis from *Ficus carica* has been reported, it is often poorly recognised and there is a need to raise awareness amongst arborists, orchardists, forestry workers, gardeners, and health professionals.

Plant dermatitis (phytodermatitis) is caused by a reaction in the skin following contact with certain plants or plant parts. They can be irritant such as cactus spine injuries, urticarial (e.g. from stinging nettles), allergic from plants such as *Primula obconica* or *Toxicodendron succedaneum*, or they can be phytophototoxic in nature. Phytophotodermatitis is generally a toxic reaction due to direct skin exposure to certain plants or plant parts, followed by exposure to ultraviolet (UV) light. The most common plants to cause phytophotodermatitis belong to the Apiaceae (Umbelliferae) family. Other plant families that can cause phytophotodermatitis include Rutaceae, Moraceae, and Fabaceae.

**Case report**

Two male arborists were cleaning up storm damaged limbs from a large fig tree, *Ficus carica*, which was heavily laden with fruit (e.g. Figures 1 and 2). The work took place in Auckland between 10:30 am and noon, on a dry, relatively clear summer day (80% relative humidity, temperature 24°C, total UV exposure in 1.5 hours 3.58 mJ).

Both workers were dressed in short-sleeved shirts or singlets. During the removal of the storm damage, they wrapped their arms (predominantly their right arms) around the fig tree branches when dragging them to the wood chipper.

Some 9 hours later, the first arborist noted a burning sensation on his right arm, which he attributed to sunburn. However, over the next 12 hours, the skin on this arm became swollen, erythematous, and was sore to touch. Within 24 hours of contact with fig tree parts, bullae appeared on the forearm, wrist, and back of the hand.
These symptoms on the first arborist persisted for over 2 weeks despite the use of alternative remedies, including a mixture of lavender oil and Aloe vera gel. As the acute erythema settled, post-inflammatory pigmentation developed, which slowly resolved over a month.

The second worker also experienced a burning sensation on his right forearm some 9 hours after working with the fig tree. Blistering of skin was noticed approximately 31 hours after contact with the fig tree (Figure 3), at which point the arborist covered the blisters and bullae with manuka honey. His condition progressively worsened with swelling and formation of large bullae on the affected arm (Figure 3). Circa 51 hours after contact with the plant, he attended the accident and emergency (A&E)
department at the local hospital. Initially the pain and blistering were restricted to the arm which had been wrapped around the fig branches—but subsequently he developed pain and swelling on the left arm, chest, and legs which had also been in contact with the fig tree. The discomfort progressed such that he was unable to work for approximately 10 days.

He responded slowly to topical corticosteroids and oral non-steroidal anti-inflammatories. The symptoms gradually resolved over 4 to 6 weeks (Figure 3).

**Discussion**

The history and clinical appearances was pathognomonic of a phytophotodermatitis which, in these two cases, was secondary to contact with the fig tree. Phytophotodermatitis is the interaction of plant compounds, most often psoralens, with sunlight on human skin; this results in an acute dermatitis.¹ It is usually a phototoxic reaction, as opposed to a photoallergic reaction. As a result, no prior sensitisation is necessary and anybody can be affected.² Other types of phytodermatitis include urticarial dermatitis, irritant contact dermatitis, and allergic contact dermatitis.

The eruption of phytophotodermatitis usually begins 24 hours after exposure and peaks at 48–72 hours. Phytophototoxicity may be amplified by both humidity and perspiration. It typically manifests as a burning erythema that may subsequently blister, and post-inflamatory hyperpigmentation lasting weeks to months may ensue. In some patients, the preceding inflammatory reaction may be mild and go unrecognised by the patient.

Phytophotodermatitis occurs most commonly in the spring and summer when furocoumarins are at their highest concentration in plants, and when UV levels are also at their peak. The incidence of phytophotodermatitis is unknown, but will vary according to the risk of exposure to psoralens. Because furocoumarins are found in a wide range of wild and domestic plants (Table 1), a variety of patient groups may become exposed.

**Table 1. Examples of plants known to cause phytophotodermatitis, and the main sensitising compounds associated with them**

<table>
<thead>
<tr>
<th>Family</th>
<th>Species</th>
<th>Common Names</th>
<th>Main Compounds</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apiaceae</td>
<td><em>Ammi majus</em></td>
<td>Bishop's weed, large bullwort</td>
<td>5-MOP, 8-MOP, imperatorin</td>
</tr>
<tr>
<td></td>
<td><em>Apium graveolens</em></td>
<td>Celery</td>
<td>Psoralens, 5-MOP, 8-MOP</td>
</tr>
<tr>
<td></td>
<td><em>Heracleum spathylium</em></td>
<td>Cow parsnip, common hogweed</td>
<td>5-MOP, 8-MOP, imperatorin, phellopterin</td>
</tr>
<tr>
<td></td>
<td><em>Heracleum mantegazzianum</em></td>
<td>Giant hogweed</td>
<td>5-MOP, 8-MOP, imperatorin</td>
</tr>
<tr>
<td></td>
<td><em>Pastinaca sativa</em></td>
<td>Parsnip</td>
<td>5-MOP, 8-MOP, imperatorin, isopimpinellin</td>
</tr>
<tr>
<td>Fabaceae</td>
<td><em>Psoralea corylifolia</em></td>
<td>Babchi, scurf pea</td>
<td>Psoralens</td>
</tr>
<tr>
<td>Moracea</td>
<td><em>Ficus carica</em></td>
<td>Fig</td>
<td>Psoralens, 5-MOP</td>
</tr>
<tr>
<td>Rutaceae</td>
<td><em>Citrus bergamia</em></td>
<td>Bergamot</td>
<td></td>
</tr>
<tr>
<td></td>
<td><em>Citrus maxima</em></td>
<td>Pomelo, pummelo, shaddock</td>
<td>5-MOP</td>
</tr>
<tr>
<td></td>
<td><em>Dictamus albus</em></td>
<td>Gas plant</td>
<td>5-MOP</td>
</tr>
</tbody>
</table>

5-MOP = 5-methoxypsoralen, 8-MOP = 8-methoxypsoralen.

The two cases presented were at very high risk of developing phytophotodermatitis because of their prolonged and significant contact, high summer levels of...
furocoumarins in the plant, peak summer UV levels, exposed skin, warm temperature, and perspiration.

Whilst photoallergic reactions are a cell-mediated immune response in which the antigen is the light-activated photosensitising agent, phototoxic reactions result from direct damage to tissue caused by light activation of the photosensitiser.

The main photosensitisers in plants are furocoumarins and consist of psoralens (5-methoxypsoralens, 8-methoxypsoralens), angelicin, bergaptol, and xanthotal (Table 1).1–3

The photochemical excitation of these furocoumarins is induced by UV radiation, usually within the UVA wavelengths of 320–400 nm (peak activity is around 335 nm).13

Two types of toxic reactions occur: one oxygen-independent, where the UV-activated furocoumarins bind to RNA and nuclear DNA; and the other: an oxygen-dependent reaction where the induced compounds cause cell membrane damage and oedema.1,3–6 These reactions consequently lead to cell death (sunburnt cells and apoptotic keratinocytes).

Ficus carica, which is believed to have originated in western Asia, was brought to the Mediterranean as early as 5000 BC. In New Zealand, it is commonly cultivated as a fruit tree in home gardens and appears to be widespread in the North Island (particularly in northern regions); it can also be found in some areas of the South Island, particularly in those areas that experience long, hot summers (Melanie Newfield, personal communication, 2007).

Ficus carica belongs to the Mulberry family (Moraceae). The leaves and unripened fruit of figs contain the furocoumarins, psoralen, and bergapten, as well as the coumarins, umbelliferone, 4’,5’-dihydropsoralen, and marmesin. The furocoumarins are lipid-soluble and can penetrate the epidermis with ease.7–10

There are a number of other Ficus species which may cross react with F. carica including Weeping fig (F. benjamina), Cluster fig (F. racemosa), and Sycamore Fig (F. sycomorus).

Eating figs does not cause photosensitisation, unless the juice is smeared onto the face. However, anaphylaxis has been reported after eating figs; in some of these cases, this may represent a cross-reaction with natural rubber latex.11–12

Although phytophotodermatitis from Ficus carica has been previously reported, it is often poorly recognised. As the cases reported here illustrate, contact with fig and other plant sources of furocoumarins can cause severe local reactions.

It is important that awareness is raised amongst the general public—especially those people whose occupations lead to a greater likelihood of exposure: arborists, orchardists, forestry workers, and gardeners.

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References:


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