In this issue

How do wasps choose among syconia?

Fig trees and their pollinating wasps exhibit one of the best known instances of insect–plant co-evolution, where the partners exhibit an obligate dependence on each other for their reproduction. Every species of *Ficus* is pollinated by a specific species of the agaonid wasp. These wasps (called foundress wasps), besides serving as pollen vectors, lay eggs in to a proportion of the flowers inside the figs (called as syconium; *pl* syconia). The fig trees gain in

terms of reproduction benefits from having their flowers pollinated and dispersal of pollen grains. But the only benefit for the pollinating wasps is the availability of seeds for rearing their off springs. As more number of pollinating wasps entering the syconium could increase the efficiency of pollination, they can also increase their oviposition rates. However, question arises why the pollinator wasps have not evolved to increase their fecundity in terms of increased number of eggs or entering in large numbers into the syconium to ruin the fig trees to collapse the association. Many of the theories explaining the conflict were given by studying the characters of *Ficus* spp. Ramya *et al.* (**page 520**) study the behaviour of pollinator wasps entering the syconium. The pollinators restrict themselves before overcrowding inside the syconium. In fact the pollinators are capable of assessing the load of wasps inside fig before entering the syconium. Assessment of syconium is done through various clues and one of them could be the wings on the ostiole which are left behind by the preoccupied wasps. Pollinators are capable of judging the syconium in terms of receptivity and pre-wasp load before entering. The study shows that pollinators avoid competition and interference for oviposition. These studies on entry behaviour of pollinators were first attempted in laboratory conditions to determine the role of tiny pollinators in maintaining mutualism by balancing the seeds and wasps production.

Breeding system of *Emex australis*

Emex australis a native of Cape region of South Africa was deliberately introduced into Western Australia by settlers who thought it had the potential of a vegetable. Hence it was given the name 'Cape Spinach'. However, soon it became an aggressive weed and spread throughout the agricultural areas of Western Australia. Having spread to many countries including India it causes immense damage to wheat. In India it has so

far been recorded only from Jammu district of Jammu and Kashmir state. *E. australis* is a winter annual found growing in dense patches between December and May. The plant body initially a rosette of leaves, later on differentiates shoots at the base. Flowers are unisexual, colourless, odourless and nectarless suited to self- and/or wind-pollination. Sex ratio is male-biased as is typical of wind-pollinated taxa. Analysis of results obtained from different pollination treatments suggests the plants to be facultatively xenogamous. Breeding system of this type coupled with prolonged seed dormancy contributes to its aggressiveness and rapid spread. Proper de weeding at the seedling stage proves effective in its control because most of the chemical and biological methods have failed to contain it. See **page 554**.

Role of autophagy in neurodegenerative diseases

Autophagy, a cellular protein degradation process, has been implicated in various human physiological and pathological conditions, such as development, immunity, cancer, neurodegeneration and longevity. This has led to a rapid growth in its field of research over the last decade and has generated considerable interests amongst a wide range of biologists. Autophagy functions predominantly as a cell survival process and is critical for tissue homeostasis. In this issue, Sovan Sarkar (**page 514**) provides a brief overview of the role of autophagy in neurodegenerative diseases. While several mutant aggregate-prone proteins associated with neurodegenerative diseases have been shown to be autophagy substrates, recent reports suggest that autophagy dysfunction occurring in such disease contexts could contribute to neurodegeneration by accumulation of the mutant proteins. Enhancing autophagy by small molecules has been demonstrated to be protective in several models of neurodegenerative diseases by facilitating the clearance of these mutant proteins. The chemical inducers of autophagy offer great potential for future therapeutic studies not only in the context of neurodegeneration, but also for diverse human diseases where autophagy acts as a protective pathway.