

# Mundulla Yellows in Eucalyptus

## An abiotic or biotic disorder?

A multi-disciplinary investigation of an unknown etiology



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## Executive Summary

A study of 40 *E. camaldulensis*, *E. leucoxyton* or *E. cladocalyx* trees from South Australia and Victoria was undertaken to investigate the role of biotic and abiotic causal agents in the plant die-back syndrome, Mundulla Yellows (MY). A further six *E. arenacea* trees were examined at Desert Camp in SA, in floristic and soil surveys.

Floristic surveys in SA, revealed that MY-like symptoms were present in plants with vastly different taxonomic positions. Conversely, closely related species were affected to markedly different degrees. Long-lived plant species were affected with MY symptoms, while annuals were apparently unaffected. Thirty-five new species, demonstrating MY-like symptoms, were identified in this study (additional to those listed in Mundulla Yellows Task Group, 2004. A Report on Mundulla Yellows in Australia. The Natural Resource Policies and Programs Committee of the Natural Resource Management Ministerial Council.).

In biotic investigations, no plant fungi, bacteria or phytoplasmas were identified as being associated with MY symptoms. Predominantly, ubiquitous saprophytes or secondary pathogens were isolated from symptomatic and asymptomatic sites, with slightly higher numbers recorded at asymptomatic sites. There was no association between nematodes and MY symptoms, nor was there an association between insect pests or vectors and MY, in an assessment of ten study sites.

In this study, MY symptoms were not transmissible by seed, mechanical inoculation (Victorian sites tested only) or grafting of selected material in repeated experiments. However, virus-like particles were detected at a single symptomatic SA study site, using Transmission Electron Microscopy (TEM). The characterisation (including mechanical inoculations) and the association of the virus-like particles with symptom expression are being further investigated. The absence of these particles in symptomatic trees from other sites suggests that they are not the primary cause of MY.

Viroid analysis detected multiple, faint RNA bands in both MY-affected and non-affected trees from independent study sites, indicating there was no clear association with MY.

MY symptoms were induced in *Eucalyptus camaldulensis* by planting healthy seed in sterilised and unsterilised soil, collected from underneath symptomatic trees. Soil collected from underneath non-symptomatic trees did not induce symptoms. This demonstrated that the soil factors play an important role in the development of MY symptoms but soil-borne biotic agents were not involved in symptom development.

All symptomatic sites, investigated in this study, contained alkaline subsoil and higher salt levels compared to asymptomatic sites. Soil from symptomatic sites had lower levels of available soil Fe and some sites also had lower levels of Mn when compared to asymptomatic site soils. Foliage analysis of some symptomatic sites revealed lower levels of Fe and Mn and higher levels of Na and Cl.

MY symptoms disappeared after applying a dilute chelated Fe solution to *E. camaldulensis* seedlings growing in soil from SA symptomatic site, in preliminary glasshouse experiments. In a pilot field trial, Fe and Mn were applied to symptomatic *E. leucoxylon* at a single Victorian study site, which also resulted in the reversal of MY symptoms. This experiment needs to be repeated in SA and at other locations.

This is the first report of MY symptom reversal. These results indicate that nutrients contribute to symptom development but clearly MY **is not** simply caused by a nutritional deficiency. Our data suggests that MY is caused by a complex interaction of soil properties (texture and parent material), nutrients, soil compaction, water availability, increased alkalinity and salinity, and the accumulation of bicarbonate in the soil solution.