TEMPERATURE EFFECT ON THE *IN VIVO* PRODUCTION OF FUSARIC ACID

USING a thermostatically controlled miniature glass-house,[1] the *in vivo* production of fusaric acid, a toxin/antibiotic produced by *Fusarium vasinfectum* Atk., has been studied at varying temperatures and reported hereunder. Cotton plants, Karunganni 2 (*Gossypium arboreum*) grown in sterilised and *F. vasinfectum* infested and uninfested soils were treated to varying temperatures, *viz.*, 32·5° C., 35·0° C. and 37·5° C. (± 0·1° C.). At the end of fourteen days, fusaric acid content of these plants was determined by the modified agar-cup technique developed in this laboratory.[2] Chromatographic studies of the α-amino constituents of plants growing at the three different temperatures indicated the consistent presence of cystine at 37·5° C. and its absence at the two other lower temperatures. The disease incidence and the *in vivo* fusaric acid content of cotton plants grown under the three different temperatures are presented in Fig. 1.

While it is obvious that there is a progressive decrease in wilt with increasing temperatures, a gradual accumulation of detectable fusaric acid in plants was favoured by increasing temperatures. This behaviour can be explained in the light of our present knowledge on the role of heavy metals, particularly Fe''' which is known to potentiate lycomarasmin activity in excised shoots of tomato.[3] It is, therefore, quite possible to expect fusaric acid to behave in a manner analogous to lycomarasmine in its ability to chelate with Fe''' or any other metallic ions of the host. Since cystine has a strong chelating ability with free Fe of the host tissue,[4] it seems logical to expect a decreased toxicity of fusaric acid at 37·5° C., presumably due to the preferential

chelation of cystine liberated at 37·5° C., with free iron or any other metallic ions of the host rendering them unavailable for effective phytotoxicity of fusaric acid.

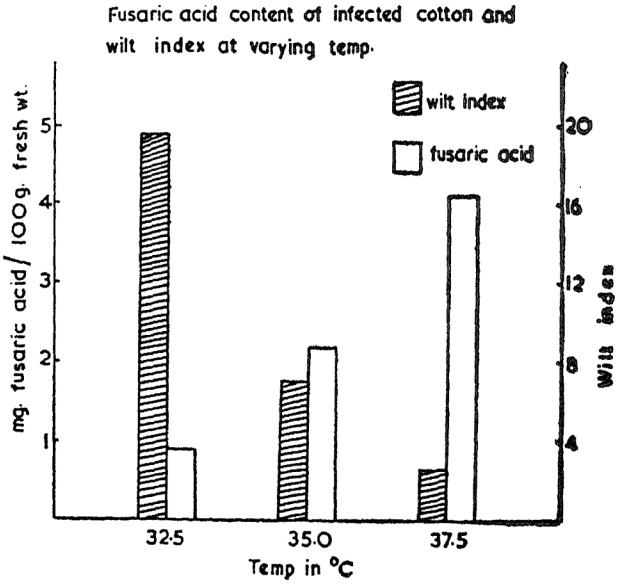

FIG. 1

Our knowledge of the changes of fusaric acid in tomato cuttings indicates that a major portion of this toxin is immediately metabolised in plants into various fractions, some of which are known to be more injurious to the host tissue than the unmetabolised fusaric acid.[5] Therefore, it is also quite possible that higher temperatures inhibit the formation of certain enzyme systems of the host responsible for the decarboxylation and N-methylation of fusaric acid with the result that a greater quantum of unmetabolised and less injurious free fusaric acid accumulates in the plants incubated at higher temperatures. An understanding of the quality of bound and free metallic ions available in the host at varying temperatures of incubation and the enzyme status of the host as well, is bound to throw considerable light on this problem and work on these lines is being pursued.

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