TABLE I

Liquid holding recovery after UV treatment at different time periods

Time in hours of liquid holding (in dark) at 30°C	Percent survival — UV dose	
	4000 ergs/ mm²/sec.	6000 ergs mm²/sec.
0 hr.	0·334	0·00155
1 hr.	0·336	0·00153
2 hrs.	0·333	0·00159
4 hrs.	0·312	0·00146
6 hrs.	0·286	0·00139

TABLE II

Effect of temperature on liquid holding recovery

Time in hrs. of liquid holding (in dark)	Temperature of liquid holding	Per cent survival — UV dose	
		4000 ergs/ mm²/sec.	600 ergs/ mm²/sec.
0 hr.	..	0·334	0·00155
2 hrs.	10° C	0·330	0·00153
2 hrs.	20° C	0·333	0·00155
2 hrs.	30° C	0·333	0·00156
2 hrs.	38° C	0·336	0·00156
2 hrs.	40° C	0·450	0·00170
2 hrs.	42° C	0·665	0·00308
2 hrs.	45° C	9·000	0·03875
2 hrs.	50° C	Nearly all cells die	

observed that the total number of bacteria in the untreated suspension had been reduced to a considerable extent, while in the ultraviolet treated suspension the number was not reduced to a similar extent. This obviously caused a relatively higher percentage of survival. Further, it was interesting to note that, at 45° C the survival percentage was consistently increased twenty-five fold of the initial survival rate, although at 42° C only two fold increase was noticed. At still higher temperature, *i.e.*, by liquid holding at 50° C for 2 hours time, it was found that none of the cells could survive and so the question of heat reactivation of UV damaged cells in this case could not arise (Table II). This phenomenon is in sharp contrast to the normal liquid holding recovery found in most other organisms, where LHR occurs maximally at room temperature.

The present finding may be best explained as a kind of "thermal reactivation" operative in dark and without an energy source. Here the reactivation may be due to (1) increased decay rate of labile photoproducts at elevated temperatures or (2) due to the presence of some other mode of repair operative at 40° C or above. The exact mechanism of this recovery is however not yet clear. Experiments to elucidate the mechanism concerned are now in progress.

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SPOROSTATIC ACTIVITY OF VOLATILE CULTURE METABOLITE(S) OF *ASPERGILLUS FLAVUS*

VOLATILE sporostatic activity of culture metabolite(s) of *Aspergillus flavus* was demonstrated against the macroconidia of *Fusarium solani f. coeruleum* a rot causing pathogen. Delaying as well as inhibition of spore germination was found, due to the volatile metabolite(s).

Introduction

The failure of spores to germinate in contact with parent cultures or of other fungi is well known in a wide range of fungi. Biologically active fungal volatile metabolites (Dick and Hutchinson[1]; Hora and Baker[2])

are mostly aldehydes and other miscellaneous compounds (Park and Robinson[3]; Rabinson and Park[4]; Robinson, Park and Garrett[5]) which in low concentration delay the spore germination. Inhibition of spore germination is due to the continuous production of volatile substances from the mycelium.

Materials and Method

The test fungus as well as assay fungus were isolated from the soil, B.H.U. campus. The *F. solani f. coeruleum* was tested for its pathogenic nature.

The test fungus was cultured in sterilized czapek's-dox broth, after 10 days of incubation at room temperature (27 ± 2° C). When the thick mycelial mat formed in static culture, was separated and placed in sterilized petriplates.

The sporostatic activity was tested by placing two glass rods over the mycelium over which microscope slide with boiled, sterilized cellophane, streaked with spores of the assay fungus (4×10^5 macroconidia/ml) was inverted. The lids of the petriplates were replaced by the dish of a similar size (as lower) and sealed with tape to check the drying off of the cellophane and mycelium. The plates were set in duplicate. For control similar slides were inverted in sterilized moist chamber over the glass rods. The slides were observed periodically for the spore germination.

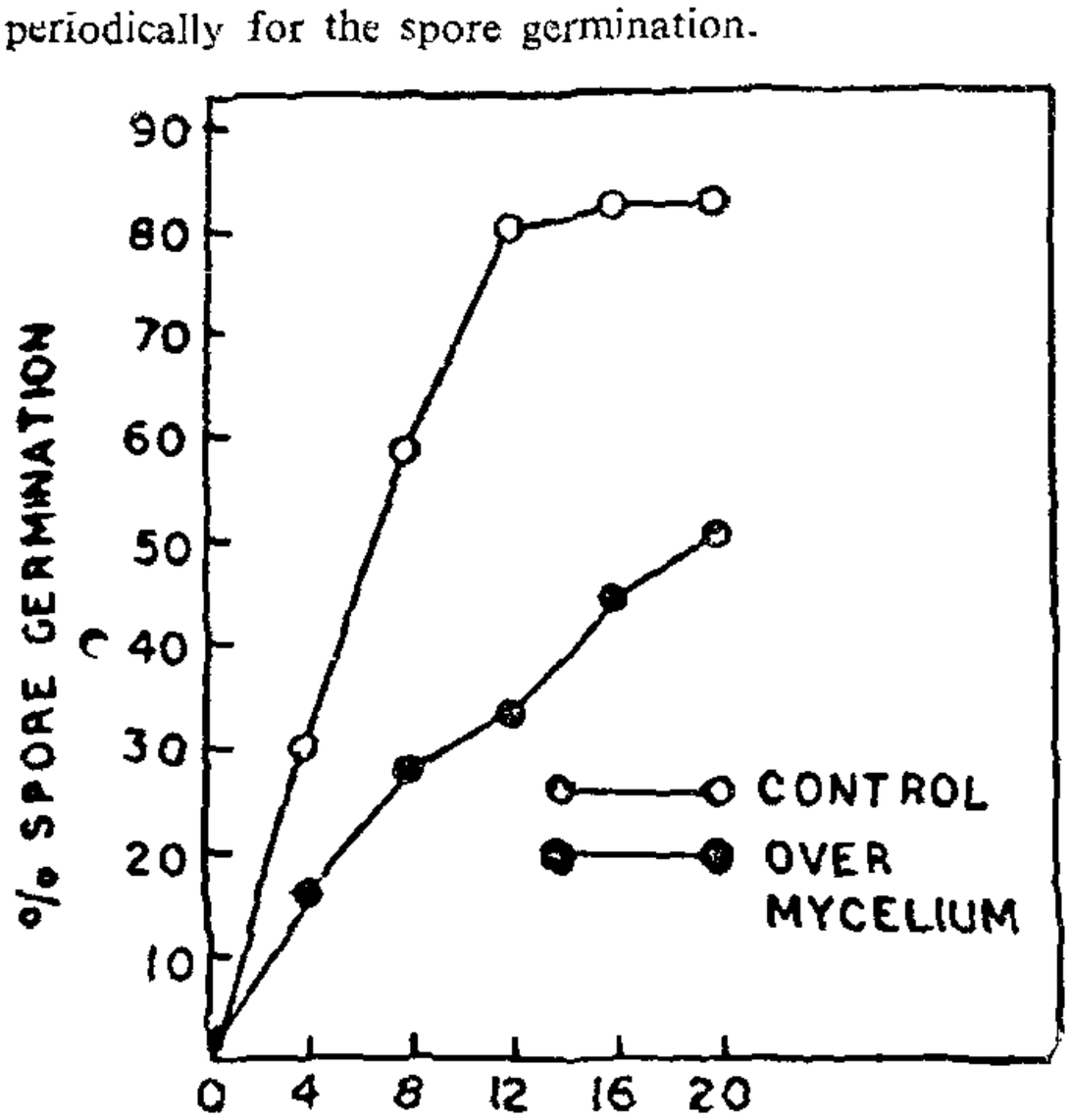

FIG. 1

Viability of the spores was tested by transferring the lides showing the inhibition of spore germination, to controlled condition and observing the germination after certain period of incubation when maximum germination was found.

Results and Discussion

The experimental result indicates that the mycelial mat of *A. flavus* produces volatile inhibitory substances to the spore germination which is confirmed by testing the viability of the spores of the assay fungus.

The *A. flavus* is known to produce non-volatile vacuolation factors like aspergillic acid, aflatoxin and other miscellaneous toxins. These volatile and non-volatile metabolites in soil might be responsible for the mycostasis of several other pathogenic forms of soil inhibiting fungi.

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CHANGES IN THE AMOUNT OF ASCORBIC ACID IN NORMAL AND REGENERATING BARBELS OF FISH *HETEROPNEUSTES FOSSILIS* (BLOCH)

VITAMIN C is very necessary for the repair of wounds in mammals, including man[1]. One of the obvious debilitations of scurvy is the strikingly poor repair of wounds. Vitamin C deficiency interferes with collagen fibre synthesis[2] and thus in the repair of wounds.

Though distribution of ascorbic acid has been studied by numerous investigators[3,4], its distribution in the normal and regenerated barbels of fish has not been studied. In the present investigation, distribution of ascorbic acid has been studied in normal and regenerated barbels and regeneration blastema.

15 live *H. fossilis* (15–20 cm) were acclimatized in the laboratory aquaria for 9–10 days. Normal barbels from 5 fishes were cut and fixed. The barbels of remaining 10 fishes were amputated leaving half the length of the barbels. Amputated barbels of 5 fishes were removed (after 4 days) in which blastema formation has taken place. In remaining 5 fishes the amputated barbels were allowed to regenerate, and the regenerated barbels (fully formed after 30 days of amputation) were removed and fixed. Silver nitrate