cies given above (19 per cent positive and 81 per cent negative) suggest that the gene responsible for the 'new' antigen has a frequency in American Negroes of about 0·10.

Dr. Ruth Sanger very kindly confirmed some of our findings. She pointed out that unless the antigen is found in a White family, where blood-group segregation is usually more informative, it will probably take a long time to exclude the 'new' antigen from the remaining known blood-group systems. For this reason, we propose the tentative name, Js, for the antigen, omitting a superscript a or b until such time as its proper status is delineated.

It is of interest that this patient made another antibody, anti-V, to an antigen common in Negroes but rare in Whites; the anti-V could be removed by appropriate absorption leaving the anti-Js intact.

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Cytoplasmic Variation in *Nectria stenospora*

DURING an investigation of the suitability of *Nectria stenospora* Berk. and Br. for the study of fungal variation, a case of cytoplasmic variation occurred which, at first appearance, closely simulated a gene-controlled effect.

The eight ascospores from a single ascus of unknown parentage were isolated on potato dextrose agar and, on the characters of colony colour and texture, were grouped into four pairs. The colour of the colony was controlled by a single pair of alleles, with 'yellow' dominant to 'pale', at least in heterokaryotic colonies. The texture of plate cultures was 'uniform', 'ragged' (uniform except at edge of plate where growth was ragged) or 'tasselled' (an intricate pattern of tassel- or fan-shaped areas of denser, sporulating mycelium separated by areas with reduced hyphal density and sporulation). It was thought that texture was controlled by genes at two loci. Colony characteristics are shown in Table 1.

Table 1. CHARACTERISTICS OF COLONIES DERIVED FROM ASCOSPORES OF ONE ASCUS

Ascospore colony	Characteristics	Postulated genotype
1, 2	Pale; uniform	y; a^1b^1
3, 4	Yellow; tasselled	Y; a^2b^2
5, 6	Pale; ragged	y; a^1b^2
7, 8	Yellow; ragged	Y; a^2b^1

Although these characteristics were always maintained if the strains were transferred by mycelium, analyses of colonies derived from single, uninucleate conidia have shown that only about 32 per cent of colonies derived from a 'tasselled' parent are 'tasselled', whereas about 10 per cent of colonies from 'ragged' parents are 'tasselled'. Only strains 1 and 2 ('uniform') breed true on subculturing with single conidia. The supposition that the tasselling effect must therefore be controlled by cytoplasmic particles has been confirmed by Jinks's heterokaryon test[1]. Using colour of the colony as a nuclear marker, a heterokaryotic colony, phenotypically 'tasselled', was made between strains 1 ('pale', 'uniform') and 3 ('yellow', 'tasselled'). Among colonies grown from single conidia were some combining the characters 'pale' and 'tasselled'. Thus 'tasselled' had become dissociated from the 'yellow' nucleus and associated with the 'pale'.

The 'tasselled' character must therefore be cytoplasmically controlled, and the resemblance to a two-gene effect in the original ascus is due to a fortuitous irregularity in distribution of the cytoplasmic particles among the ascospores, only strains 3 and 4 having sufficient for its expression and 1 and 2 having few or none. The presence of the particles in strains 5–8 is revealed in mono-conidial subcultures when a conidium, by chance, incorporates a greater-than-average number of particles[2].

The investigation has also revealed great variation between strains in regard to sexual reproduction, some apparently being sterile, some heterothallic and others homothallic, although preferentially outbreeding. Details of the work will be presented in full elsewhere.

This work was undertaken while one of us (A. G.) held a Commonwealth Scientific and Industrial Research Organization Australian Studentship for the Honours Year.

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A Change of Pathogenic Race in *Fusarium oxysporum* f. *pisi* induced by Root Exudate from a Resistant Host

THE soil-borne fungus *Fusarium oxysporum* causes wilt in many economically important host plants and, like many plant pathogenic fungi, it consists of several specialized forms, each including a number of pathogenic races. A striking feature of *Fusarium* wilts is their sudden occurrence in crops in which they were previously unknown[1,2]. Such new disease outbreaks clearly suggest that either saprophytic forms of the fungus have become parasitic or that existing parasitic races have extended their host-range. Among genetic mechanisms already proposed to explain such changes of pathogenicity in *Fusarium* and other imperfect fungi are mutation, heterokaryosis, which allows haploid nuclei to associate in various ways, and a parasexual mechanism[3], which allows permanent genetic recombination without a sexual stage[4]. On the other hand, there is some evidence that pathogenicity can be influenced by the host. For example, Reddick and Mills[5] increased the varietal host range of *Phytophthora infestans* by successively transferring haploid zoospore cultures on to a series of potato varieties of increasing resistance to this fungus. Bawden[6], in discussing the role of plant hosts in microbial ecology, pointed out that this approach can be extended by first correlating the mechanism of resistance with the presence in the host of some toxin specific to the fungus pathogen or with the lack of some metabolite essential for its growth.

Pea wilt seemed to be a suitable subject with which to study this problem, for exudates from the roots of pea varieties that act as differential hosts for the different races of *F. oxysporum* f. *pisi* affect the spores of the three pathogenic races differentially[7]. For example, spore germination of race 1 is inhibited in root exudate from the race 1-resistant pea variety, Wilt-Resistant Alaska, whereas the growth of race 2,