from untreated seed plants were used as control. All the petri dishes were inoculated with 7 mm mycelial discs of a 3-day-old culture of *S. rolfsii* isolated from infected groundnut plants (cv TMV-2). Radial growth of the fungus was measured after 72 h of incubation.

Persistence of tolclofos methyl in sandy loam, red loam and black loam soils was studied over 12 weeks. Characteristics of the soils are given in table 1. Samples of each soil type were passed through a 4.68 mm sieve after pounding. The soils were sterilized in an autoclave at 20 psi for 30 min twice on alternate days. One kg of sterile soil of each soil type was mixed with fungicide (1 and 2 g/kg of soil). The treated soils were kept in plastic beakers and sterile water was added regularly except on the day prior to soil sampling. Control soils were maintained without the addition of the fungicide.

Persistence of the chemical to a depth of up to 5 cm in the soils was studied by bioassay of samples collected using a cork borer. Fungicide present in the soil was extracted by taking 5 g of air-dried soil with 5 ml of acetone in glass vials (100 × 25 mm). The vials were then thoroughly shaken and left for 10 min to allow the soil to settle. The supernatant was transferred to sterile glass vials (100 × 25 mm) and the acetone was evaporated. The residue was taken up in sterile distilled water as before, and the bioassay carried out. Each treatment, including the control, was replicated thrice.

The data (table 2) show that tolclofos methyl was taken up by groundnut following dry seed treatment and was redistributed among different parts of the plant. All the extracts, prepared from plants on days 1, 3 and 7 and every week thereafter after germination, inhibited growth of *S. rolfsii*. The whole plant extracts from plants raised from seeds treated with 0.1% and 0.2% fungicide limited fungal growth to a greater extent than the corresponding root and shoot extracts. Extracts from older plants age (raised from treated seeds) were less inhibitory to the fungus than extracts from younger plants. Extracts prepared from fungicide-treated soils completely inhibited growth of *S. rolfsii*.

The inhibition of *S. rolfsii* by shoot and leaf extracts shows that the fungicide moved upwards in the plant system. The higher inhibition in the case of treatment with 0.2% tolclofos methyl may be due to higher initial uptake, and translocation and persistence of the fungicide in plant system. The lower inhibition by extracts from older plants (raised from treated seeds) may be due to dilution of the fungicide or degradation of the fungicide to non-fungitoxic products.

The better persistence of the fungicide in the treated soils may be due to slow rate of degradation by abiotic factors and absence of biological degradation.

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A NOTE ON THE SPONTANEOUS OILY LARVAL MUTANT IN SILKWORM, *BOMBYX MORI* L

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DURING the course of our breeding studies in June 1988 some spontaneous oily mutant larvae were observed in a polyvoltine silkworm breed of *Bombyx mori* L. The larvae were highly oily and translucent when compared to normal creamy-white larvae (figure 1). The mutant larvae were separated and their performance was assessed and compared with that of the normal breed. After three generations the characters of the mutant strain were consistent; hence it is breeding true.

More than 25 different genes, located at different loci, are known to cause translucency of the skin. The integument of the silkworm larva is usually opaque, containing whitish crystals of urate[1]. But several mutants have become known which have

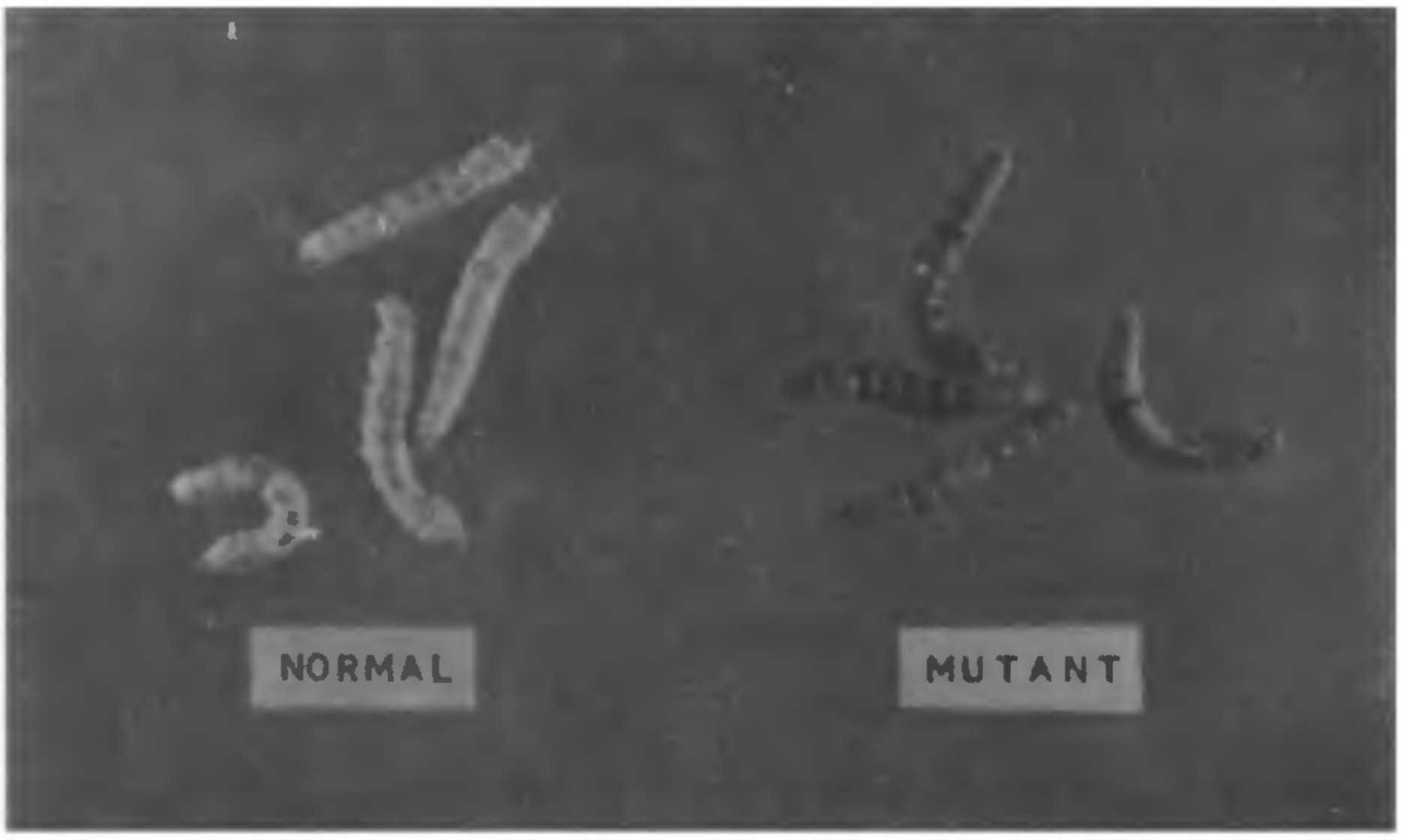

Figure 1. Normal and oily mutant larvae of silkworm.

translucent skin. The translucency varies according to the mutant strain, mainly depending on the quantity of urate in the epidermal cells[1,2].

In the present case, the oily character is very conspicuous and the mutant larvae were easily separated from the original batch. Of the 21 oily mutant larvae 11 were males and ten females. The mutant is identified as the *og* mutant, i.e. the Giallo Ascoli translucent of Sansaki[1], which is situated on chromosome 9 at locus 7.4. The translucency is of an extremely high degree. Translucent larvae are thought to have less ability than the normal type to retain uric acid in various organs and tissues of ectodermal origin. Translucency genes are more or less lethal. In the present case, the mortality was very high: of the 11 males only four larvae spun cocoons and of the 10 females only two spun cocoons. Sexual markings are very clear and it can be differentiated with the naked eye easily even after the third moult. All the four males and two females

Table 1 *Performance of oily mutant and normal polyvoltine silkworm*

Generation		Larval duration (h)	Cocoon wt (g)	Shell wt (g)	Cocoon shell ratio (%)
Mutant	June–July '88	504	1.322	0.205	15.5
	Sept.–Oct. '88	510	1.302	0.200	15.4
	Nov.–Dec. '88	553	1.320	0.210	15.9
	Mean	522.33333	1.31467	0.20500	15.6
	SD	26.72838	0.01103	0.00500	0.26459
Normal	June–July '88	576	1.006	0.139	13.8
	Sept.–Oct. '88	594	1.087	0.150	13.8
	Nov.–Dec. '88	636	1.107	0.152	13.7
	Mean	602.000	1.06667	0.14700	13.76666
	SD	30.78961	0.05349	0.00700	0.05820
	t	3.3843*	7.8657**	11.6773**	11.7211**

emerged from the cocoons. Only one pair was recovered, as the other female was unable to pair with a male. All the eggs laid by the mated female moth were fertilized. In the subsequent generation the survival of the larvae and moths was observed to be satisfactory. Three generations (June–July, August–September and November–December 1988) of mutant and normal were compared and the data are given in table 1.

It is evident from table 1 that the cocoon characteristics of the mutant are superior to those of the normal. Another interesting feature is reduction in larval duration by 3 days even after three generations in the mutant strain. The improvement noticed in the mutant strain is 23% in cocoon weight, 39% in shell weight and 13% in cocoon shell ratio.

As the cocoon characters of the mutant are better than those of the normal and are breeding true, the mutant can be utilized in future breeding programmes to improve the economic characters of polyvoltine breeds.

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VIBRATION CHARACTERISTICS OF CANCER CELLS

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A cancer is a malignant mass of cells that divide uncontrollably to the extent that normal cells are deprived of nutrients[1]. In cancerous transformation cells lose, partially or completely, their normal surface properties[2]. It involves a radical reorientation in biosynthetic metabolism for cell growth and division[3]. Progressive increase in the permeability of the cell membrane[4], lack of contact inhibition of growth[2], and changes in membrane fluidity[5] (i.e. viscosity and rigidity of the cell membrane) are some of the well-known characteristic features of tumour cells. Cancerous growth reflects a disturbance in the cellular system. Various types of cell disorders are known to occur in man and other organisms[6], each of which has a characteristic frequency spectrum and amplitude of oscillations. The equations for the characteristic frequencies of oscillation of normal cells have been used to explain neoplasia in terms of altered characteristic frequencies.

The vibration characteristics of normal cells have recently been studied[7–9]. If one ignores the viscosity and relaxation parameter, the frequency equation of a normal resting cell can be written as[8,10]

$$\omega_n^2 = \frac{T}{\rho_i b^3} \times \frac{n(n-1)(n+2)}{\left[1+\left(\frac{n}{n+1}\right)\frac{\rho_0}{\rho_i}\right]}, \quad (n \neq 1), \qquad (1)$$

where n is a positive integer, T the interfacial tension, b the radius of the cell, and ρ_0 and ρ_i the density of the extracellular and intracellular fluid respectively. The surface and interface displacements are proportional to the Legendre polynomials[7] P_n^0 (cos θ). Rapid shape patterns continue throughout the cycle[2]. The variation of ω_n with ρ_0/ρ_i for different modes of oscillation of the cell is shown in figure 1. As the tension parameter $T/\rho_i b^3$ of the membrane changes (increased membrane fluidity is a characteristic of cancer and more rigid membranes are characteristic of aging), frequency of vibration changes (figure 2).

Changes in vibrational frequency from that of the normal resting cell[9] may be the cause of several metabolic diseases. The diameter of carcinoma cells[11] is 10–15 μm and the critical diameter at the time of division is estimated to be 12.6–18.9 μm (assuming that the volume doubles just before

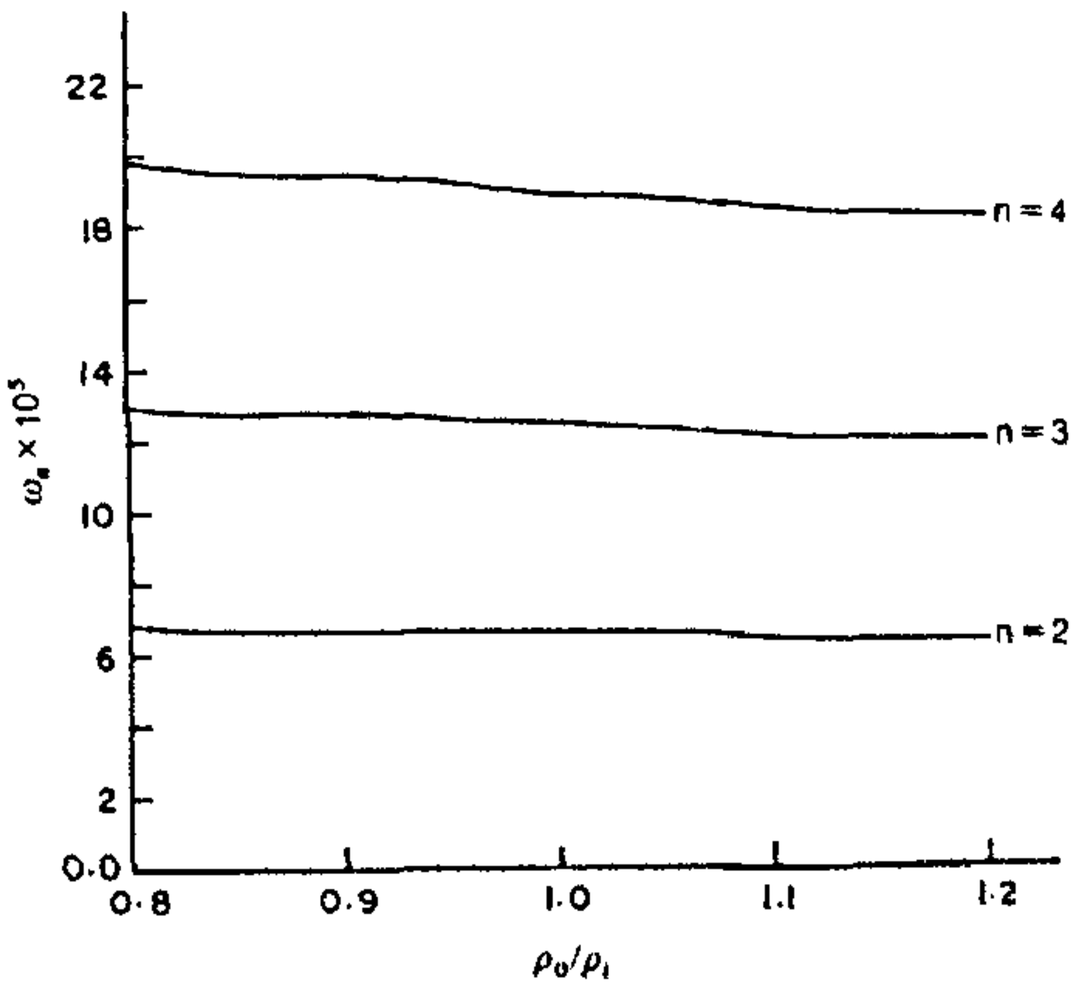

Figure 1. Frequency of vibration of a cell as a function of density ratio ($T/\rho_i b^3 = 9 \times 10^{10}$ s^{-2}).