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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1. Sansaki, S., *Jpn. J. Genet.*, 1938, **13**, 285.
2. Jucci, C., *Bull. Zool.*, 1932, **3**, 23.

## 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 $\rho_0$ and $\rho_i$ 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 $\theta$). Rapid shape patterns continue throughout the cycle[2]. The variation of $\omega_n$ with $\rho_0/\rho_i$ 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 $\mu$m and the critical diameter at the time of division is estimated to be 12.6–18.9 $\mu$m (assuming that the volume doubles just before

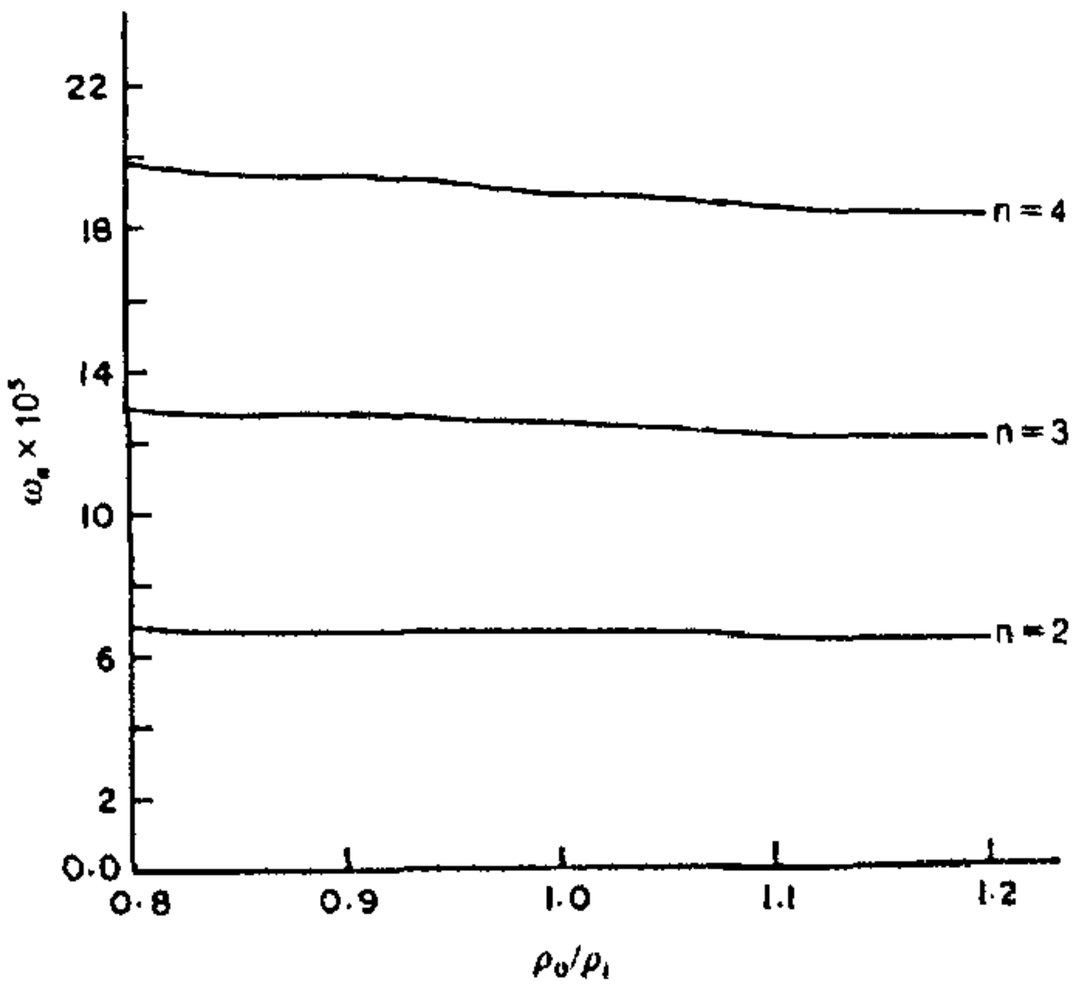


**Figure 1.** Frequency of vibration of a cell as a function of density ratio ($T/\rho_i b^3 = 9 \times 10^{10}$ s$^{-2}$).

mitosis). Abnormal vibration characteristics for different modes of oscillation of a cancer cell are shown in figure 3 (with time $t$). The period of the cell cycle has been divided into time intervals $t_1$, $t_2$, etc. The different phases of the cycle are also indicated in figure 3. The cells divide repeatedly (at a faster rate than do the corresponding normal ones[12]) without spending the normal time in interphase. The amount of time required for most cancer cells to pass through the S, $G_2$ and division phases of the cell cycle may be approximately the same as for normal cells but the $G_1$ period appears to be considerably shorter.

Mass transfer changes owing to changes in membrane fluidity (rigidity), growth patterns alter, and the cells divide at inappropriate times. Each type of cancer cell has a recognizably different abnormality in frequency spectrum which is different from that of the homologous normal resting cell[9] and is a characteristic of the severity of the malignancy. Cells become cancerous when normal $G_1$ periods become defective. The rate at which a cancer cell can divide depends partly on the severity of the defects in the controls[14].

One can conclude that alterations in interfacial tension due to changes in surface properties and

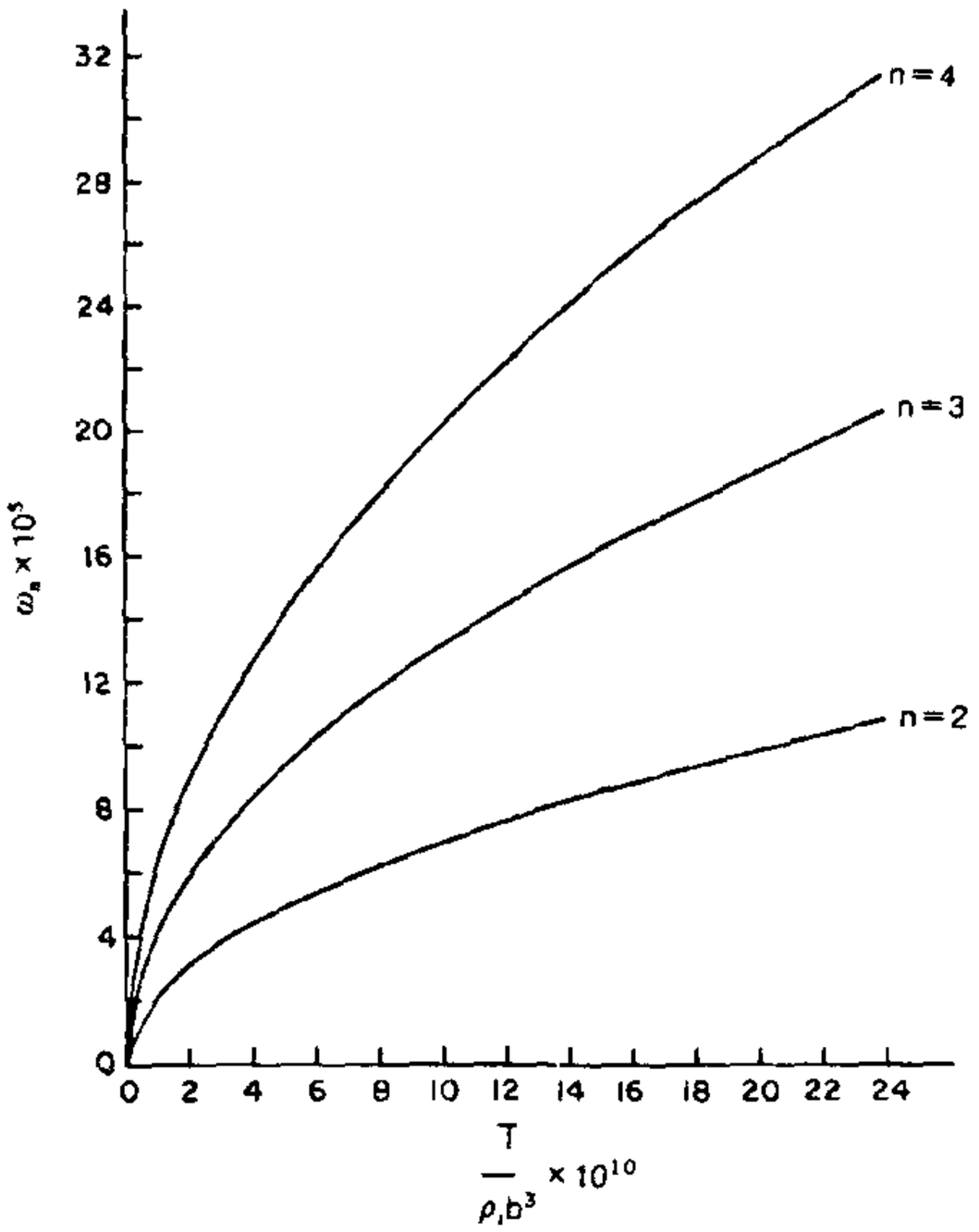


**Figure 2.** Frequency of vibration of a cell as a function of tension parameter $T/\rho_1 b^3$.

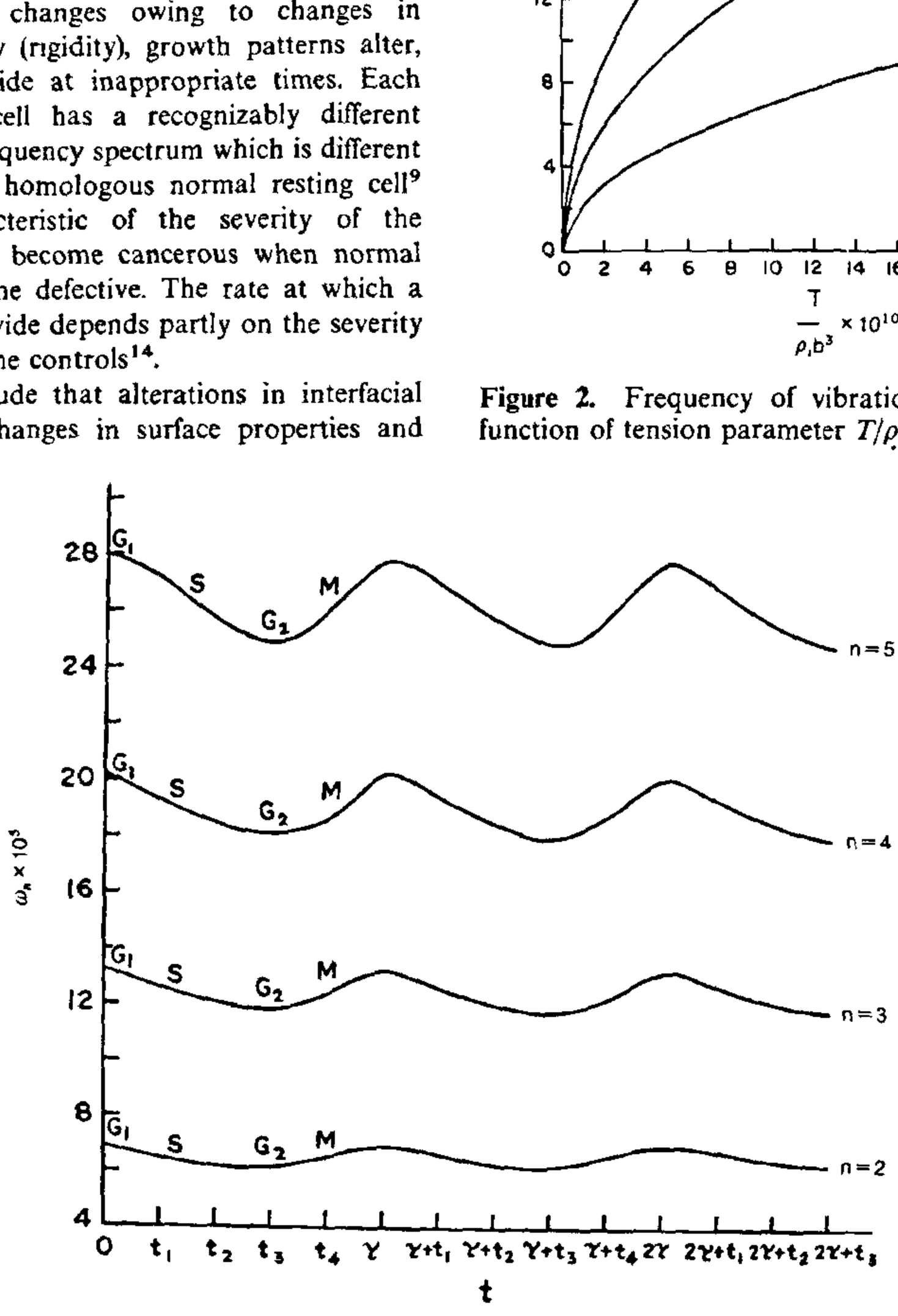


**Figure 3.** Plot of $\omega_n$ vs $t$ for different modes of oscillation of a cancer cell ($\rho_0/\rho_1 = 0.95$).

abnormal vibration characteristics are important factors in the establishment of the malignant state.

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1. D'Amato, F., *Nuclear Cytology in Relation to Development*, Cambridge Univ. Press, London, New York, 1977, pp. 540.
2. Braun, A. C., In: *The Cancer Problem*, Columbia Univ. Press, New York, London, 1969, pp. 29, 71.
3. Swann, M. M., *Cancer Res.*, 1958, **18**, 1118.
4. Wood, H. N. and Braun, A. C., *Proc. Natl. Acad. Sci.* USA, 1965, **54**, 1532.
5. Rosen, R., *Progress in Theoretical Biology*, vol. 6, Academic Press, New York, London, 1981, p. 4.
6. Nason, A. and Goldstein, P., *Biology*, Addison–Wesley, California, London, Ontario, 1969, pp. 199.
7. De, S., *Natl. Acad. Sci. Lett.*, 1986, **9**, 385.
8. De, S., *Proc. Natl. Acad. Sci. India*, 1988 (in press).
9. De, S., *J. Theor. Biol.*, 1987, **125**, 117.
10. Ackerman, E., *Biophysical Science*, Prentice-Hall, Englewood Cliffs, N. J., 1962, pp. 238.
11. Enterline, H. T. and Coman, D. R., *Cancer*, 1950, **3**, 1033.
12. Rédel, G. P., *Genetics*, Macmillan, New York, Collier Macmillan, London, 1982, pp. 497.
13. Pardee, A. B., In: *Cell Growth*, (ed.) C. Nicolini, Plenum Press, New York, London, 1982, pp. 673.
14. Weisz, P. B. and Keogh, R. N., *Elements of Biology*, 4th edn, McGraw-Hill, New York, London, Tokyo, 1977, pp. 326.

## FECUNDITY OF MAYFLIES OF WESTERN GHATS OF PENINSULAR INDIA

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FECUNDITY is the total number of eggs produced by the female during her life span regardless of the fate of the eggs. For most insect groups, it is difficult to measure fecundity accurately in their natural environment because the eggs are continually maturing and are oviposited throughout the life span. Mayflies are excellent material for the study of fecundity under natural conditions. Adult mayflies usually live for only a day or two and all the eggs are produced prior to the subimago stage and the total potential fecundity can be determined accurately by examining subimagos, imagos that have not oviposited, and even last instar nymphs[1].

Mayfly fecundity has been correlated positively with body length[1–5]. A more meaningful calculation for making comparisons between species would be the relationship between egg production and unit body length. In the present study, six families of Ephemeroptera were chosen and the relationship between their egg production and unit body length was analysed. Eggs from the abdominal and thoracic body cavities of the last instar (with darkened wing pad) nymphs of mayflies were removed to a Sedgwick rafter and counted. The relationship between fecundity and body length was statistically analysed.

The correlation between the number of eggs produced by six families of Ephemeroptera and their body length is presented in figures 1–6. The average number of eggs per mm of body length in the six families ranged from 201 to 1843. The data of Clifford and Boerger[1] for Bigory River mayflies of Canada, of Hunt[2] and Britt[3] for Ephemeridae, and of Minshall[6] for Heptageniidae were 137–222 eggs/mm, 300–350 eggs/mm and 100–200 eggs/mm respectively.

In the present study the $r$ value which ranged from 0.90 to 0.99 (highly significant) confirms the general view that fecundity increases with increasing body length of the nymphs. However, Minshall[6] found that beyond a certain size (10.5 mm) the number of eggs decreased with increasing size of the individual. This apparent decline in egg number with increasing body size (up to 31 mm in Ephemeridae) has not been observed in the present study.

Though the nymphs of Ephemeridae are the longest among the members of the six families under investigation, the rate of egg production is high (1843 eggs/mm) only in Heptageniidae. The ephemerids are burrowing and sandy forms whereas heptageniids are rheophilic and are restricted to torrential areas of rock-bottomed streams. This difference in ecology should account for production, in heptageniids, of more eggs as a compensatory measure for the loss of eggs washed away by torrents.