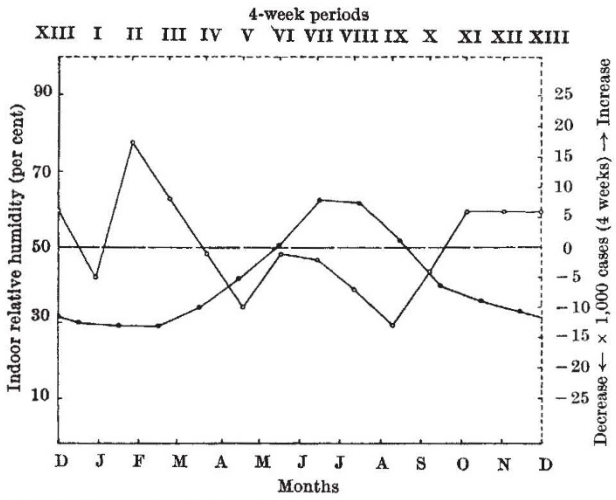


Fig. 3. Seasonal variation of relative humidity (●) and increment of measles morbidity (○) in England and Wales. The mean monthly relative humidity values (period 1921–50) have been plotted at mid-month. The increase in the number of measles cases (means over the period 1946–62) during each four weeks period has been plotted at the beginning of the period and not at mid-period, as the date of infection is probably two weeks before registration

increment of the morbidity curve for measles. The increment is generally positive at low relative humidity values and negative for relative humidity values above 40 per cent. In other words, measles morbidity increases during the period of low indoor relative humidity and decreases in the period of high humidity. Analogous results were obtained using data for other countries.

The marked influence of relative humidity on virus survival together with the negative correlation between the seasonal variation of relative humidity and of measles morbidity suggest that relative humidity indoors might be an important factor in the seasonal variation of measles.

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## Canine Distemper Virus cultured in a Diploid Monkey Cell Strain

THERE have been reports of the growth of virulent strains of canine distemper virus in primary tissue cultures derived from chick embryos[1], dogs[2], ferrets[3], rabbits[4], marmoset and human beings[5], and in continuous cell lines derived from dogs[5] and human beings[4].

This preliminary communication is to report the growth of a virulent strain of canine distemper virus in the cell strain *BS–C–1* which was derived from primary grivet monkey kidney tissue[6] (kindly supplied by Mrs. Hope E. Hopps). The *BS–C–1* cells were used between passages 36 and 48. They were sub-diploid or diploid, having a chromosome number ranging from 58 to 60 (ref. 7). Canine distemper virus has been serially passaged 22 times in *BS–C–1* cells, with production at each passage, after 7–9 days, of a cytopathic effect characterized by multi-nucleated giant cells containing intranuclear and intracytoplasmic inclusion bodies. Uninoculated *BS–C–1* cells were free of cytopathic changes. After 9 and after 21 serial passages, the titre of canine distemper virus in *BS–C–1* cell cultures, 7 days post-infection, was $10^4$ $TCID_{50}$/ml. This was determined by the development in *BS–C–1* cells of intracytoplasmic inclusion bodies.

The line of *BS–C–1* cells could be grown serially, and could be maintained free of the extraneous viruses which might occur in primary cell cultures and which could exhibit variable interference effects. *BS–C–1* cells have provided a useful culture system for growth of canine distemper virus because of their uniformity.

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# PSYCHOLOGY

## Further Observations on Differences in Eosinopenia between Inbred Strains of Mice

Thiessen and Nealey[1] recently reported differences among five inbred strains of mice in behavioural reactivity and in adrenocortical activity, in response to taking blood counts and handling. On several measures, the *C57BL/Crgl* strain displayed greater sensitivity to the testing conditions than did other strains. Resting blood eosinophil-level was higher, test-retest reliability was higher and the correlation between resting-level and extent to which the second count differed from the initial count was greater. These findings corroborate the conclusion of Wragg and Speirs[2] that the *C57* genotype is maximally sensitive to measures of eosinopenic responses and, in addition, point out the value of the *C57BL/Crgl* strain for experimental purposes.

The findings of Thiessen and Nealey[1] differ from those of Wragg and Speirs[2] in one important way. Wragg and Speirs found that for the *C57BR/cd* mouse, maximum eosinopenia occurred at 3·5 h after the initial count, but Thiessen and Nealey found that for the *C57BL/Crgl* mouse, significant eosinopenia was not apparent at 3·5 h after the initial count. The reported discrepancy was assumed to be due to slight differences in the time interval between counts, or to variations in handling procedure. An additional possibility, impossible to evaluate in our original report, is that the *C57* genotype of our investigation did respond with eosinopenia to the taking of blood samples, but had fully recovered by the time the second count was taken 3·5 h later. If such is the case, it would suggest that genetic separation of the *C57* animals has resulted in striking differences between sublines in the rate of eosinophil cyclicity and thus, presumably, in differences of adrenocortical activity.

To test the possibility that *C57BL/Crgl* animals respond with rapid eosinopenia and rapid recovery to experimental manipulations, 5 groups of 10 animals each were investigated. The groups differed in the time permitted to elapse between the initial eosinophil count and a second count. The time intervals for the different groups were 15 min, 1, 2, 3 and 4 h. To assess the possibility that other strains used in our previous investigation may also demonstrate significant eosinopenia during the earlier periods following baseline measures, 50 *C3H/Crgl/2* mice, judged to be insensitive to tail cutting and handling[1], were also tested at these same intervals. Animals were housed individually at 33 days of age and tested 7 days later, following the standardized procedure previously used with these same strains[1]. Blood samples were obtained by cutting off the tip of the tail. The number