meiosis by a direct method, and at the same time to correlate this with the sensitivity pattern. Mavor[5,6] showed that irradiation of females caused primary nondisjunction. The same ought to be true also in males if meiotic stages are irradiated. The appearance of individuals in which the paternal sex-chromosomes both have entered the sperm cell will indicate that the cell had been treated before anaphase 1. In the present experiment the occurrence of *XO* males has been used as an indication of genetical damage. At the same time the induction of nondisjunction between the *X* and *Y* chromosomes in the males has served as a criterion of early meiosis (prior to anaphase I).

The following stocks were used: the females were taken from a *y w sn* ♀♀ × *y w sn*; sc^8Y ♂♂ stock and the males from a y^{16} ♀♀ × y^{16}; sc^8Y ♂♂ stock. The normal offspring would be *y* females and *w sn* males. Individuals which had lost the *X* and/or sc^8Y, and consequently were *XO*, would phenotypically be *y w sn*; and eggs fertilized by an *X*; sc^8Y carrying sperm (nondisjunction) would give wild-type females due to the presence of the y^+ locus in the *Y* chromosome (*y w sn*; y^{16}; sc^8Y). As these females also may arise through induced crossing-over between the *X* and the *Y* chromosome, each wild-type female was tested individually. The majority of these were due to nondisjunction. The males were picked out when 0–1 day old. They were either irradiated when they had woken up after treatment with ether or kept without females for 3 days prior to irradiation. The males were irradiated with a dose of 1,100 r. (2 min.). Immediately after irradiation the males were transferred to a mating box. The first mating period lasted 4 hr., but from the 4th day onwards each period consisted of 24 hr. Between the first mating period and the 4th day the males were kept with an excess of females.

Table 1. 0–1-day and 3–4-day-old y^{16}; sc^8Y males were given 1,100 r. in air and were then mated to *y w sn* females. Total number of offspring as well as rate of *XO* males and wild-type females are given

Mating period after irradiation	0–1-day-old males			3–4-day-old males		
	Percentage *XO* ♂♂	Percentage wild type ♀♀	Total offspring	Percentage *XO* ♂♂	Percentage wild type ♀♀	Total offspring
0–4 hr.	0·46	0·01	20,538	0·32	0·03	44,635
Fourth day	0·33	0·03	62,692	0·20	0·04	43,016
Fifth day	0·75	0·03	50,552	0·43	0·02	38,766
Sixth day	1·33	0·01	24,735	0·61	0·02	17,571
Seventh day	2·21	0·22	18,100	1·24	0·12	14,155
Eighth day	3·47	0·51	13,350	1·55	0·21	11,706
Ninth day	2·39	0·19	7,772	0·96	0·13	8,724
Tenth day	0·54	0·18	27,946	0·49	0·14	8,312
Eleventh day	0·46	0·14	30,044	0·19	0·05	7,807

From Table 1, which shows the results after irradiation of 0–1-day- and 3–4-day-old males, it can be seen that, independent of the age of the males at time of irradiation, the number of induced *XO* males rises steeply from the 4th day, reaching a peak on the 8th day after treatment and then decreasing towards the 11th day. As regards the frequency of induced wild-type females, the rate is practically unchanged from the first mating period until the 6th day after irradiation. On the 7th day after irradiation there is a sharp increase in the frequency of wild-type females. The frequency reaches a peak during the 8th day after irradiation, followed by a drop towards the 11th day.

It is of special interest to compare the frequency of induced *XO* males and the rate of induced nondisjunction females during the 4th–10th day after irradiation. It can be seen that the peak of *XO* males coincides with the peak of nondisjunction females. Since the occurrence of nondisjunction females is used as the criterion of early meiosis (prior to anaphase I) there is an agreement between the highest X-ray sensitivity and cells treated at early meiotic stages.

About the same sensitivity pattern is obtained after irradiation of 0–1 and 3–4 day old males respectively. There exists, however, a discrepancy in the amplitude of the sensitivity. The frequency of induced *XO* males, as well as the rate of induced nondisjunction females, is much lower in the experiments with 3–4 day old males. This difference is statistically significant except for the first mating period. There is a fairly good agreement between these findings and Lüning's[1] conclusions that 6·7-day-old males vary much less than 0–1-day-old males between different stages of spermiogenesis.

These experiments show that the treated meiotic cells become available for insemination during the 7th day after irradiation and onwards. From the present material it can be concluded that the peak of sensitivity presumably corresponds to cells treated during metaphase I or before anaphase I is completed. This conclusion is supported by sensitivity studies performed on different material[7–10].

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VIROLOGY

Virus Survival as a Seasonal Factor in Influenza and Poliomyelitis

The pronounced seasonal fluctuations in the morbidity of influenza and poliomyelitis are largely unexplained. Both diseases can probably be transmitted by airborne infection and by many other ways which involve the survival of the causative virus in the dry state. For many organisms survival in air can be taken as indicator for survival in the dry state in general. Consequently it seemed worth while to study the influence of temperature and humidity on the survival of the viruses of influenza and poliomyelitis in air.

Earlier experiments with aerosols[1] of many organisms showed that—at least in the range of indoor temperature—the influence of temperature is small (Q_{10} = 2–3) compared with the influence of humidity. Furthermore it became evident that the relative humidity and not the absolute humidity is the determining factor.

Both viruses were aerosolized for 2 min. with an indirect spray (mean droplet diameter 6μ) into a

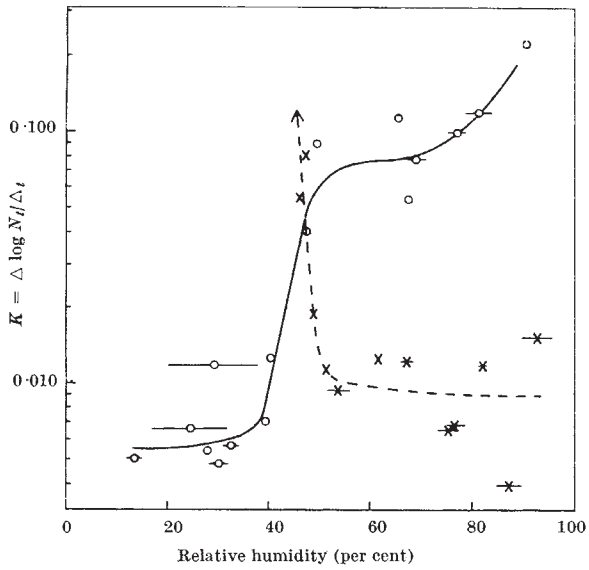

Fig. 1. Death-rates for influenza virus (○) and poliomyelitis virus (×) in air plotted against relative humidity

static system (4 M^1). Influenza virus (PR_8) was suspended in one part allantoic fluid and one part 2 per cent Difco peptone. Poliomyelitis virus (type 1 *CSl*) was suspended in one part Hanks salt solution with lactalbumin-hydrolysate and 5 per cent horse serum and one part peptone. At various times samples were taken for 2 min. with modified capillary impingers containing 1 per cent peptone or 1 per cent peptone with Dulbecco buffer.

Virus was titrated by amnion inoculation for influenza and by the plaque method on human amnion cells (strain *U*) for polio-virus.

From the logarithmic phase of the inactivation curves at various relative humidities, death-rate constants were calculated, using $K_2 = \Delta \log N_t/\Delta_t$ (t denoting time in minutes). K is plotted against relative humidity in Fig. 1. For influenza virus the death-rate is high (0·091 ± 0·024) at 50–90 per cent relative humidity and low (0·0073 ± 0·0031) in the range 15–40 per cent relative humidity. The transition is rather sharp. These results are mainly in accordance with the limited experiments published previously by other authors[2–6]. For poliomyelitis the inverse is true. Inactivation is slow (K = 0·0089 ± 0·0036) at high relative humidity and very fast at low relative humidity. In fact, below 45 per cent no virus could be detected 30 sec. after spraying, which means that less than 0·03 per cent of the virus was left. The transition is again sharp.

In temperate climates influenza generally is a winter disease, occurring during the period when the relative humidity indoors is low as a result of heating. In Fig. 2 the seasonal variation of indoor humidity is plotted. The period of increasing influenza morbidity coincides with the period during which the survival of the virus in air is optimal.

Increase of poliomyelitis morbidity occurs during summer, that is, during the period when the indoor relative humidity is optimal for the survival of the virus in air (and presumably in the dry state in general).

Consequently the relative humidity, indoors, is considered as an important environmental factor contributing to the seasonal fluctuations of the morbidity of these two virus diseases.

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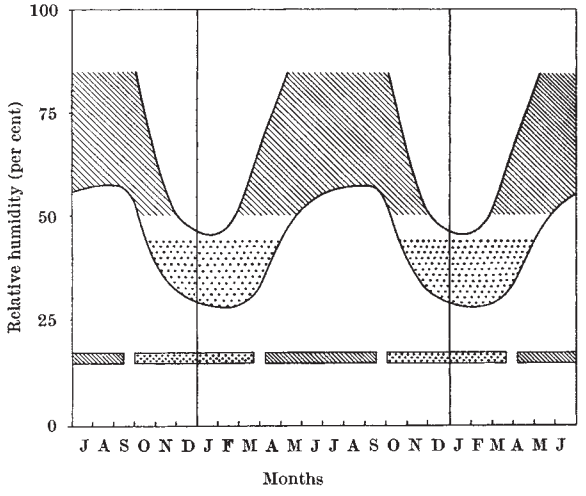

Fig. 2. Seasonal variation of relative humidity indoors in the Netherlands. The upper and lower curves represent mean maximal and minimal relative humidity as calculated from temperature and absolute humidity. The range for optimal virus-survival is stippled for influenza virus and hatched for polio virus. The period of increasing morbidity of influenza (data for England and Wales) and for poliomyelitis (Dutch data) are given at the bottom of the figure as stippled and hatched bars

Association of *Olpidium brassicae* and Tobacco Necrosis Virus

Tobacco necrosis virus infects the roots of many plants, usually without symptoms. The virus is soil-borne[1–3], but the conditions necessary for natural infection have not been determined. Attempts to obtain artificial infection of roots by adding virus suspensions to roots of plants grown in sterilized soil commonly fail.

The relationship of *Olpidium brassicae* to tobacco necrosis virus infection was investigated because of claims[4–7] of an association between tobacco necrosis virus and lettuce big vein disease, with which *O. brassicae* is invariably associated[8,9].

Further circumstantial evidence for the association of tobacco necrosis virus and *O. brassicae* was obtained in the present work by microscopic detection of *Olpidium* in the roots of *Cleome spinosa*, *Fragaria vesca*, *Chenopodium amaranticolor*, tobacco, and French bean infected with tobacco necrosis virus.

When suspensions of *Olpidium* zoospores from lettuce plants shown by root assay to be free of tobacco necrosis virus, and suspensions from macerated virus-infected cowpea leaves were simultaneously watered on to potted lettuce seedlings, much greater