There is no evidence to suggest that E2ψ1 was present in the EO2 as a temperate phage; had it been temperate and become induced in vivo due to a component in the sheep's diet, then, assuming a uniform distribution of the inducer in the rumen, all the cells in the population would lyse within a short time. If, due to uneven mixing within the rumen, only a proportion of the EO2 were exposed to the inducer, there would only be a partial drop in the population density. Complete and gradual lysis could only be explained by postulating a regular intake of inducer which did not mix uniformly in the rumen; this is unlikely, however, since the same diet had been fed for more than 1 year during periods of high EO2 population densities and had been fed to other sheep containing EO2 in which bacteriophages did not subsequently appear. Bacteriophage E2ψ1 is therefore probably a virulent and not an induced temperate bacteriophage.

Recognition of the presence of both temperate and virulent bacteriophages in the rumen therefore brings another factor for consideration into the already complex field of ruminant nutrition and rumen ecology. Although it is as yet not known if other rumen bacteria have bacteriophage pathogens the number and variety of bacteriophage particles present in the rumen suggest that this is so. Temperate bacteriophages would not be expected to have a dramatic effect on the population densities of individual bacterial species unless that population was subjected to induction, perhaps by a plant constitutent in the diet. Virulent bacteriophages, on the other hand, as shown by the effect of E2ψ1 on EO2 would have a radical effect on specific populations. In terms of the well-being of the host animal, lysis of bacteria in the rumen with secondary fermentation of the products would involve losses of carbon compounds in the form of gases, but this would be partially offset by increased volatile fatty acid production. The effect of the loss of a single species, even of the important cellulolytic bacteria would probably be temporary because other species capable of occupying the same ecological niche would proliferate. Since, however, inducers of temperate phages are non-specific in their action, should such an inducer occur in the diet it is possible that several species could be lost simultaneously.

Resumen. Se han demostrado que dos cultivos de bacterias de rumen (un bastón gram negativo, designado W461, y una cepa de óvalos de EADIE) están infectados con bacteriófagos. El bacteriófago de W461 es templado; aquel de los óvalos es virulento y depleta rápidamente la población bacteriana en vivo.

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Pattern of Electrolyte Leakage in Downy Mildew Affected Sorghum

Alteration of permeability and ionic imbalance associated with plant disease development has been reported by many workers[1]. The present investigation reveals the pattern of electrolyte leakage in sorghum plants systemically affected by downy mildew, incited by *Sclerospora sorghi*.

Materials and methods. A highly susceptible variety (DMS 652) was used for the present investigation. Systemic infection was obtained following a previous method[2].

The loss of electrolyte was estimated by measuring the conductivity of the leachates. The electrolyte leakage was assessed from the roots, stem and leaves after 15, 30 and 45 days of sowing. The plant parts were initially washed thoroughly with tap water, subsequently with several changes of distilled water and finally rinsed with double distilled water. They were blotted dry and were suspended in sterile double distilled water in the ratio of 1 g of plant part to 10 ml of double distilled water. The conductivity of the bathing solution was measured after incubation at 25 °C for 24 h. All conductivity measurements were made with an ELICO model CM-82 conductivity bridge with platinum blackened electrodes. The temperature of the bathing solution was maintained at 25 °C during conductivity measurements. The results are expressed as specific conductivity (μmh/cm) of the leachates.

Results and discussion. The various parts of sorghum plant exhibited a pattern of loss of electrolytes and permeability change as a result of downy mildew attack (Table).

The greater leakage of electrolytes from diseased roots and leaves may be due to the higher absorption of electrolytes by diseased plants from the soil. Being an obligate

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Specific conductivity of bathing solutions of different parts of sorghum attacked by *Sclerospora sorghi*

Days after planting	Specific conductivity (μm h/cm) of bathing solutions of					
	Roots		Stems		Leaves	
	Healthy	Diseased	Healthy	Diseased	Healthy	Diseased
15	252.4	430.4	259.4	184.2	433.2	480.0
30	256.2	427.2	270.9	186.9	453.5	691.9
45	263.6	447.3	296.0	197.3	890.0	1530.8

parasite and a systemic one, such permeabilyty change brought about by *Sclerospora sorghi* is quite interesting. Though there is considerable leakage of electrolytes on the 45th day from diseased leaves, this may not be adversely affecting the fungus, which by this time enters the oospore stage in the leaves.

Even though there was increased leakage of electrolytes from diseased roots and leaves, there was a suppression of escape of electrolytes from the stem. This could very well be due to accumulation of electrolytes, which may provide nutrition to the fungus which is still in the mycelial stage in the stem. This may also compensate for the loss through the roots.

Loss in permeability is encountered in hosts attacked by facultative parasites where a toxin or enzyme is involved in the pathogenesis[1]. The ionic imbalance and permeability change seen in downy mildew affected sorghum suggests that the elaboration of a principle for the pathogenesis by an obligate parasite like *Sclerospora sorghi* cannot be ruled out.

Résumé. On étudie la perte d'électrolytes dans les plantes de sorghum infectées par le mildiou lanugineux. C'est dans les racines et les feuilles malades qu'elle fut le plus marquée mais elle ne se produisit pas dans la tige malade. Elle eut son maximum dans les feuilles malades et augmenta au fur et à mesure des progrès de la maladie.

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Intrarenally Induced Infection in Rats: Kidney Infecting Dose and Sex Dependence

Experimental research on pyelonephritis requires a suitable animal. However, handy animals are not known to suffer naturally from this disease. It is necessary, therefore, to overwhelm the high local or general resistance of healthy animals against renal infection. Various methods have been developed which in the past 10 to 15 years proved the 2 main pathogenetical mechanisms of this disease in humans. These 2 infection routes are 1. the ascending or retrograde, 2. the haematogenous way. Experimentally there are a few disadvantages in these methods: 1. necessary pre-lesion within the kidney in order to make it sensitive to the infection, 2. the unknown quantity of bacteria reaching the kidney and being responsible for success in infection, 3. an unfixed rate of lethality in the animals caused by the high infection dose necessary.

For special purposes we have found the intrarenal infection model suitable because of its fastness, simplicity, and the possibility of inoculating exactly measured doses of bacteria. This method is an unphysiological one, but the result is apparently the same as in both the other modes of infection, although the special pathomechanisms might be quite different. The method was introduced in rabbits[1], modified for rats[2], and performed by only a few research groups recently[3–5]. Prat has taken it for chemotherapeutical investigations (personal communication).

We have used rats at the age of 5 to 7 months from a Wistar inbred strain held in our institute for about 15 years. For injection procedure the rats were etherized and infected through the ethanol-disinfected skin into the left kidney fixed by 2 of the operator's fingers. Inoculation was done with suspensions of a haemolyzing *Escherichia coli* 06 strain derived from urine of a female patient with urinary tract infection. Overnight at 37 °C bacteria grown on nutrient agar slants in tubes of 16×160 mm size were washed off with 10 ml saline which resulted constantly in a content of 1.5×10^9 bacteria per ml of suspension. This original suspension was diluted stepwise to suitable concentrations which were injected by a fine needle on microlitre syringes. At the same time bacterial counts were performed by plating serial dilutions on blood- and ENDO-agar, resulting next day in the exact amount of bacteria injected. Lastly we have injected planned bacterial concentrations because deviations from the desired amounts controlled by reading the plates on the next day were very small, if any.

In order to find the 'critical' infection dose to give reproducible results for possible biological standardization of this model, an equal volume (0.02 ml) of arithmetically diluted suspensions was injected intrarenally.

Table I. Incidence of infection dependent on the amount of injected bacteria (strain *E. coli* 06 2791/71)

No. of injected bact. per kidney (in 0.02 ml)	No. bacteriol. pos. / No. infected kidneys
$\leqq 1 \times 10^4$	0/30
2×10^4	4/8
4×10^4	8/15
8×10^4	5/10
1.6×10^5	13/15
$\geqq 3.2 \times 10^5$	16/16

Table II. Bacteriological findings from the infected (2×10^7 *E. coli*) left kidneys

Autopsy at days after infection	No. positive / No. infected	Sex of animals	Statistical significance
31–32	24/24	♀	—
	16/20	♂	
83	3/5	♀	—
	0/5	♂	
157–165	8/18	♀	$+(p<0.05)$
	1/13	♂	

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