Fehlende Reizwirksamkeit der Fructose ist bisher von keinem Insekt bekannt. Im Gegenteil, dieser Zucker zählt – ausser bei der hinsichtlich ihrer Ernährung stark spezialisierten Seidenraupe[10] – bei allen untersuchten Insekten zu den Monosacchariden mit der grössten Reizwirksamkeit[3]. Das dürfte darauf zurückzuführen sein, dass der Fructose sowohl der 1C- als auch der Furanose-Bindungsort zur Verfügung stehen[1,11]. (Zucker, die an zwei verschiedenartige Bindungsorte angelagert werden können, sind wirksamer als solche, bei denen diese Möglichkeit nicht besteht[2]).

Da bei der Baumwollwanze Fructose keine Reizwirksamkeit besitzt, ist zu folgern, dass die Zuckerrezeptorzellen dieses Insekts weder einen 1C- noch einen Furanose-Bindungsort aufweisen[12,13].

Summary. The bug *Dysdercus intermedius* has gustatory receptors on the terminal segments of its antennae. Thirsty bugs discriminate between dry and wet cellulose swabs: only if they are stimulated with moist swabs, do they extend the proboscis. The stimulating effectiveness of different mono-, oligosaccharides and glycosides was studied by touching the antennae of hungry bugs and observing the extension of the proboscis. The result, that fructose is completely ineffective, entitles one to the assumption that the sugar receptor cells of *Dysdercus* possess neither a 1C site nor a furanose site.

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Interaction of *Bordetella pertussis* Vaccine Treatment and Lymphocytic Choriomeningitis Virus Infection in Mice

Transitory spleen hypertrophy and lymphocytosis developed in mice following the intravenous treatment with *Bordetella pertussis* vaccine[1]. The immunological reactivity of mice alters parallel with these consequences. The humoral immune response to heterologous antigen increases[2,3], while the cellular immune response shows decrease[4–7].

Fatal choriomeningitis develops in mice after intracerebral administration of lymphocytic choriomeningitis virus (LCM) as a result of the cellular respons of the organism[8]. The question whether the simultaneously administered B. pertussis vaccine and LCM virus influence each other's effect, was studied in our experiments.

Materials and methods. 6- to 8- week-old mice of both sexes, from the Swiss breed, were used in our experiments, in the following arrangement:

Group	P	P-LCM	LCM	C
No. of mice	28	28	28	28
	28	—	—	28
i.v. treatment	*B. pertussis* vaccine	*B. pertussis* vaccine	Phys. saline	Phys. saline
i.cer. treatment	Phys. saline	LCM virus 100 LD/50	LCM virus 100 LD/50	Phys. saline

Animals were treated i.v. with a single dose of 0.3 ml vaccine (9×10^9 killed bacteria). LCM virus infection was performed on the day of treatment with *B. pertussis* vaccine. Neurological symptoms, characteristic of LCM virus infection, were observed, and the mortality rate of animals was recorded. The relative spleen weight of the individual groups was determined. The brains of mice which died in the LCM group were subjected to histological examination. Brains of the animals of P-LCM group-succumbing after the 9th day or sacrificed on the 18th day, were excised and cut in half for virus reisolation and histological examination.

Results. The mortality rate observed in the individual mice-groups is demonstrated in Figure 1. No death was observed in the groups P and C during the experimental period. Animals belonging to the LCM group succumbed without exception 7 to 9 days following the infection, displaying neurological symptoms. Histological examination revealed lymphocytic choriomeningitis in their brains. The period of succumbing was rather prolonged in the P-LCM group as compared with the LCM group, the death rate being in the former only 43% until the 9th day. Virus was isolated from the brain of each animal succumbing or being sacrificed after the 9th day, despite the fact that 25% of these animals failed to show symptoms of meningitis. Correspondingly, no lymphocytic infiltration could be observed in the choriomenings either.

The average relative spleen-weight is somewhat lower in the LCM group than in the control group. This corresponds to the effect of LCM virus to cause lymphoid atrophy[9]. The average spleen-weight of mice belonging to group P was found to be high between the 9th and 12th day[1], which corresponds to the hypertrophy of the

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spleen occurring after pertussis vaccination. The average relative spleen-weight found in the P-LCM group shows no significant deviations as compared to the control group, thus the effect of *B. pertussis* vaccine to cause spleen hypertrophy and that of the LCM virus to induce atrophy in this organ fail to become manifested in this case (Figure 2). According to our results, the simultaneously introduced *B. pertussis* vaccine and LCM virus influence each other's effect.

Accordingly, the course of intracerebral LCM virus infection shows the same changes in mice treated with *B. pertussis* vaccine as in immune depressive states provoked by other methods[8,10–21]. Our results support the data which suggest that pertussis vaccine treatment reduces the cellular immune response to heterologous antigen being simultaneously present in the organism.

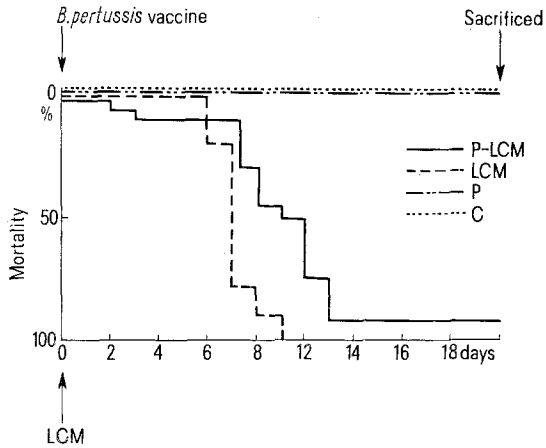

Fig. 1. Mortality rate in the individual mice-groups.

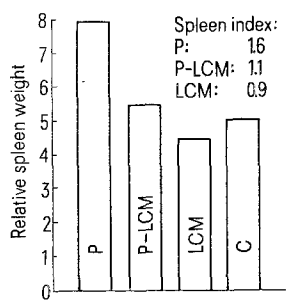

Fig. 2. The average relative spleen-weight on the 7 to 11th day of the experiment. Spleen index: P, 1.62; P-LCM, 1.12; LCM, 0.9.

Zusammenfassung. Gleichzeitig verabreichte *Bordetella pertussis* Vakzine und lymphozytäre Choriomeningitis Virus bewirkt keine Splenomegalie und auch keine lymphozytäre Choriomeningitis.

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Inhibition of Induction of Group A Bacteriocins of *Serratia marcescens* by Rifampin

The rifamycin antibiotics (rifampin) are known to specifically inhibit the bacterial enzyme deoxyribonucleic acid (DNA)-dependent ribonucleic acid (RNA) polymerase[1,2]; thus, rifampin has been employed to study various aspects of bacterial RNA and protein synthesis. Because the rifamycins are such specific inhibitors of bacterial DNA-dependent RNA polymerase, they have been used to determine the role of the drug-sensitive host RNA polymerase during the intracellular replication of a number of bacterial viruses; DNA phages (e.g., T4 and λ) were found to be sensitive to rifampin throughout replication[3,4]. The growth of the RNA phages f2 and $Q\beta$ was not inhibited when rifampin was added 4 to 5 min after infection; however, when the drug was added before or immediately after phage infection, viral replication was markedly suppressed[5,6]. Very recently it has been shown that the major pathway whereby phage inhibit host syntheses requires protein synthesis; on the other hand, the inhibition of host syntheses by phage ghosts was not affected by inhibitors of protein synthesis (puromycin, rifampin, and chloramphenicol)[7].

We recently developed a technique for typing clinical isolates of *Serratia marcescens*[8], based on sensitivity to 10 selected group A bacteriocins[9]. Meanwhile we have determined that these 10 bacteriocins are phage tails that consist of cores only (subgroup I) or of tails that are made up of cores and contractile sheaths (subgroup II)[10]. During the course of our investigations concerning the properties of these bacteriocins, rifampin was employed in attempts to answer the following three questions: 1. Does rifampin inhibit the induction of these bacteriocins by mitomycin C, i.e., does induction require functioning host cell DNA-dependent RNA polymerase? 2. Does rifampin inhibit the killing activity of these bacteriocins, i.e., is the