This organism was found to display certain characteristics that are typical for *E. coli*, such as, the prompt fermentation of lactose and the production of a metallic sheen on EMB agar. On the other hand, the isolate was shown to have the following features consistent with *P. rettgeri*: citrate utilization, deamination of phenylalanine, hydrolysis of urea, absence of gas formation during glucose fermentation, and lack of fermentation of arabinose and maltose. This is why we tentatively chose to designate this organism a promptly lactose-fermenting strain of *P. rettgeri*. It is conceivable that this organism is a 'hybrid' that might have arisen as a result of an as yet unknown genetic interaction between a strain of *E. coli* and a strain of *P. rettgeri*. To the best of our knowledge, this is the first strain of *P. rettgeri* isolated that ferments lactose overnight[6-11].

This organism can be retrieved without difficulty from clinical material and all of the isolated strains consistently ferment lactose, constantly display identical antibiograms and produce the typical, unmistakable odor. A detailed summary of the outbreak due to this organism will be reported elsewhere (C. E. McCALL, in preparation)[12].

Zusammenfassung. Ein avirulenter, prompt Laktosefermentierender, Antibiotika-resistenter Stamm von *Proteus rettgeri* wurde aus klinischem Material isoliert.

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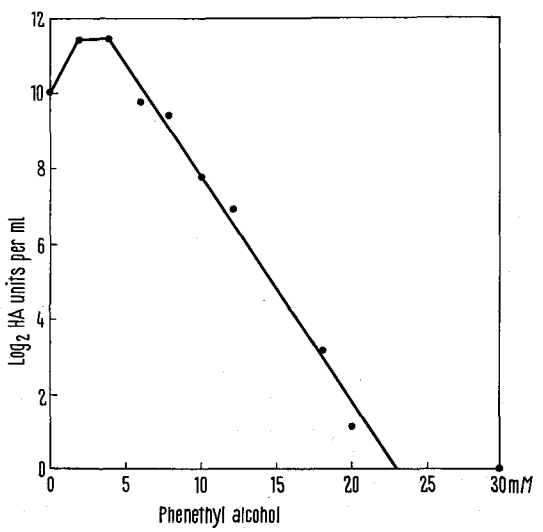
Effects of Phenethyl Alcohol on Influenza Virus Growth and Macromolecular Synthesis in Chick Embryo Cells

The bacteriostatic drug phenethyl alcohol has previously been shown to inhibit the replication of several DNA viruses[1-3], and the growth of small RNA viruses in bacteria[4] and animal cells in suspension culture[5]. In all cases the effect was reversible, and the drug had to be present during the time of viral replication in order to cause inhibition. The site of action of phenethyl alcohol is not known, although there are reports of alterations in cell membrane function in treated cells[6-8]. This results in inhibition of cell growth, and of the synthesis of RNA, DNA, and protein in both bacteria[9,10] and animal cells in suspension culture[11-13]. These effects on macromolecular synthesis and cell growth have been shown to be reversible following removal of the drug even after treatment for 12 days[11].

The replication of influenza viruses differs from that of small RNA viruses in a number of respects, and in particular displays a high degree of dependence on host cell metabolism[14]. It was therefore of interest to study the effect of phenethyl alcohol on influenza virus growth in monolayer cultures of chick embryo fibroblast cells. Since there are no previous reports of the action of phenethyl alcohol on cells in monolayer culture, as opposed to suspension cultures which are dividing rapidly[13], we also measured RNA and protein synthesis in treated cells.

Cell cultures were prepared and infected with influenza virus (fowl plague virus, Rostock strain) as detailed elsewhere[14]. Following virus adsorption, the cells were washed in phosphate-buffered saline, then incubated at 37 °C in growth medium supplemented with various concentrations of phenethyl alcohol[15]. After 24 h, the yield of virus released into the medium was assayed by haemagglutination titration (Figure). Each point represents the average haemagglutinin yield from 2 Petri dish cultures. It can be seen that very small concentrations of phenethyl alcohol, up to 5 m*M*, caused some increase in virus yield, but higher concentrations resulted in a marked inhibition which was complete at about 20 m*M*.

The inhibitory effect of phenethyl alcohol on influenza virus replication was not due to irreversible cell damage. Cells were incubated with 10 m*M* phenethyl alcohol for 2 h, then washed free of the drug before infection. Virus yields 24 h later did not differ significantly from those obtained from untreated cells. In further experiments phenethyl alcohol (10 m*M*) was added at intervals after infection and the haemagglutinin yield determined 24 h post-infection. Inhibition was observed when the drug was added at any time up to 3 h post-infection: thereafter the effect diminished and if added later than 10 h post-infection the virus yield at 24 h was normal. Since virus



Effect of various concentrations of phenethyl alcohol on the yield of influenza virus from chick embryo fibroblast cell monolayers.

release in this system commences at 3 h and is almost complete by 10 h[14], these results would suggest that the drug acts as a general inhibitor of virus maturation rather than specifically blocking a single early event.

In order to study the effect of phenethyl alcohol on RNA and protein synthesis in chick fibroblast cells, 48 h monolayer cultures were washed in phosphate-buffered saline, then incubated in fresh growth medium, with or without addition of drug (10 m*M*), and supplemented with uridine-5-^{3}H (10 μc/ml, specific activity 5 c/mmole) and DL-valine-4-^{14}C (0.5 μc/ml, specific activity 36.6 c/mmole). At intervals, duplicate cultures were analysed for incorporation of radioactivity into RNA, protein, and the trichloroacetic acid (TCA) soluble nucleotide pool as described in detail elsewhere[14,16]. The incorporation of uridine into RNA was inhibited by about 50% at all periods studied, but this inhibition was largely accounted for by a decrease in the radioactivity of the TCA-soluble nucleotide pool (Table). This suggests that a primary effect of the drug is on the incorporation of uridine into the cell, and supports the notion that phenethyl alcohol exerts a primary effect on the cell membrane[13,17]. Protein synthesis was inhibited only slightly (Table); much greater inhibition of protein synthesis has been found in studies using actively growing suspended cell cultures and bacteria[10,11,13]. In the latter cases inhibition of protein synthesis was accompanied by decreased cell proliferation, whereas the monolayer cultures which we used were already in the stationary phase of growth. Thus our results are not inconsistent with the conclusion that phenethyl alcohol acts by affecting a cell membrane-associated process concerned with the control of cellular growth[13]. Further studies using phenethyl alcohol are being directed towards the interrelationship of influenza virus multiplication with cellular metabolism.

Effect of phenethyl alcohol (10 m*M*) on macromolecular synthesis in chick embryo fibroblast cells

Time (h)	Incorporation of ^{3}H-uridine				Incorporation of ^{14}C-valine	
	TCA-soluble nucleotides (cpm × 10^{-4})		RNA (cpm/μg × 10^{-1})		Protein (cpm/mg × 10^{-2})	
	Control	Treated	Control	Treated	Control	Treated
0.5	3.3	1.9	4.9	2.9	9.5	9.0
1.5	12.2	5.2	39.7	21.2	30.9	27.8
2.5	15.5	11.5	64.6	36.6	58.8	44.6
3.5	16.7	11.7	101.9	52.0	87.1	66.8

All values are means obtained from duplicate cultures.

Zusammenfassung. Phenethylalkohol wirkt virusstatisch und es wird vermutet, dass die mit der Zellmembran in Beziehung stehenden Vorgänge der Zellvermehrung beeinflusst werden.

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The Host-Parasite Interface of Strigeoid Trematodes

VIII. Surface Specialization of the Adhesive Organ of Cardiocephaloides physalis (Lutz, 1926)

Strigeoid trematodes (Platyhelminthes) possess a posterior hind-body and an anterior fore-body which bears, on its ventral surface, a ventral sucker and posterior to this, the adhesive organ, which is used to achieve very intimate contact with the host tissues. The fore-body is cup-shaped in some species[1] and in these the adhesive organ consists of 2 lobes and the parasite lies with the mouth of the fore-body cup pressed against the host gut mucosa so that the gut villus becomes drawn into the cup to lie between the 2 lobes. Previous electron microscope studies[2-6] have indicated that in certain strigeoid species the tegument of the adhesive organ lobes in contact with the host tissues is specialized to form a chambered, placenta-like surface[2].

Cardiocephaloides physalis is a poorly known form and it seemed appropriate to determine the presence of

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