Table 2. Antimutagenic Action of Spermine on Mutations to Streptomycin-Resistance induced in *E. coli UC* 879 by Caffeine and Ultraviolet Light

(See Table 1 for details)

Treatment	$N_{initial}$ $\times 10^{10}$	$N_{surviving}$ $\times 10^{8}$	$m \times 10^{-10}$	$\frac{m \text{ control}}{m \text{ treated}}$	P
Caffeine	0·8	—	1·5	11·5	⩽0·001
Caffeine + spermine	1·7	—	0·13		
Control	1·5	—	0·48		
Ultra-violet, 25 sec	1·4	0·53	220	8·5	0·024
Spermine + ultra-violet	3·2	0·66	26		
Control	1·4	—	0·46		

ment was repeated once with essentially identical results. The ultra-violet irradiation was accomplished with a Westinghouse 36*GT*5 germicidal lamp at a dose of approximately 150 ergs/mm² to effect a killing rate of approximately 99 per cent[11]. Individual aliquots of a culture which had been grown for 9 h on a shaker (with or without added spermine during growth) were irradiated in a darkened room. All further handling was carried out in the dark to prevent photoreactivation. In all cases cells grown in the presence of spermine had lower mutation rates than the control cells ($P = 0{\cdot}02$).

The mutational event in all these experiments occurred in the absence of the indicator by which the event was scored. Furthermore, there was no effect on the growth rate of streptomycin-sensitive or -resistant organisms when spermine was included in the culture medium. Thus there is no chance that any form of selection could account for the observed changes in the incidence of mutations[12]. Preliminary results suggest that other polyamines may also suppress the random mutation rate much as they were reported by Sevag and co-workers[1–3] to delay the outgrowth of resistant populations in the presence of antibiotics. It is concluded from these results that the antimutagenic action of polyamines is a general property of these compounds, that it is independent of the mutational end-point being measured, and that it is even more pronounced when mutations are induced than when randomly occurring mutations are studied.

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H. G. Johnson
M. K. Bach

Department of Biochemistry,
The Upjohn Co.,
Kalamazoo, Michigan.

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VIROLOGY

Derivatives of 2-Aminoethyl-guanidine and of a Eugenol as Virus Inhibitors

It is a well-known fact that certain structurally different chemicals inhibit the synthesis of viral RNA[1–3]. In this communication a few substances which inhibit virus multiplication are described. These are the following: 2-(β-aminoethyl) pyridine (AEP), its substituted derivatives; the 2-pyridyl-ethyl-guanidine sulphate (Pyg)[4], the 2 - (5 - allyl - 3 - methoxy - 2 - hydroxy - benzyl) - β - (α - pyridyl) ethyl amine (*H*-99)[5] and 'Ismelin' 2-octahydro-1-azocynil ethyl guanidine sulphate[4]. Guanidine hydrochloride was used as control.

Experiments were carried out on primary monkey kidney cell monolayer cultured in Petri dishes by the plaque method[6]. The virus inhibitory effect of chemicals was tested against 10–100 plaque-forming units of Sabin's type 1 (*LSc* 2*ab*) virus strain. The plaque assay has already been published in detail[7]. Usually 1 h after virus infection, plates were overlaid with nutrient agar containing 20, 50 or 100 μg/ml. of substances respectively. Sometimes substances were added to the cell sheets, applying a second overlayer 4, 24, 32 h after virus infection respectively. The results are presented in Table 1.

Table 1. Effect of Chemicals on the Plaque-forming Capacity of Type 1 (*LSc* 2*ab*) Virus Strain (Sabin)

Exp. No.	Treatment after virus infection (h)	Concentration of substances (μg/ml.)	Controlled untreated plates	Denomination of substances: 'Ismelin'	Pyg	*H*-99	AEP	Guanidine
				Average number of plaques				
1	1	100	130	Zero	Zero	Zero	N.D.	N.D.
	1			Zero	Zero	Zero	70	
2	4	100	98	Zero	Zero	N.D.*	N.D.	N.D.
	24			39	Zero	N.D.	N.D.	
	1	100		Zero	Zero			
3	1	20		50	4			
	24	100	137	100 (Very small)	Zero	N.D.	N.D.	N.D.
	32	100		N.D.	17			
	1	100		Zero	Zero	Zero		Zero
4	1	50	49	Zero	N.D.	N.D.	N.D.	N.D.
	1	20		Zero	Zero	40		3

*N.D., not done.

As Table 1 indicates, the Pyg was the most effective but its component the AEP had only a slight inhibitory effect. Substance *H*-99 showed a strong inhibition in a concentration of 100 μg/ml., but at 20 μg/ml. its effect was negligible. The inhibitory effect of 'Ismelin' was expressed at any given concentration (100, 50, 20 μg/ml.). When 'Ismelin' was applied 4 h after infection it could inhibit the formation of visible plaques completely, and applying 24 h after infection the plaque number was strongly but not completely inhibited. In the case of Pyg, 4 and 24 h after virus infection the inhibitory effect was complete; moreover, 32 h after infection it was still at about 80 per cent. Consequently these support the hypothesis that 'Ismelin' and Pyg can inhibit the late phase of virus synthesis.

It is remarkable that the highly active chemicals are known as anti-hypertensive substances[8], and one of them, 'Ismelin', is already being used as a therapeutic drug.

Sándor Pácsa

Institute of Microbiology,
Pécs.

Olga H. Hankovszky
Kálmán Hideg

Institute of Pharmacology,
Pécs, Hungary.

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GENETICS

Puffing of Salivary Gland Chromosomes after Treatment with Carbon Dioxide

After larvae of the Oregon *R* strain of *Drosophila melanogaster* had been placed in an atmosphere of CO_2 for 1 h late in the third instar, the salivary-gland chromosomes were examined and compared with those of a similar group of the same age. The chromosomal regions