The Role of Salivary Glands in the Removal of Excessive Blood Plasma Norepinephrine

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Standard high-performance liquid chromatography was used to study the contents of epinephrine and norepinephrine in rat submaxillary salivary glands, oral cavity mucosa, and in saliva during background and evoked secretion as well as in plasma after parenteral injection of norepinephrine solution. The pilocarpine-induced stimulation of secretory function of the salivary glands increases epinephrine content in the oral cavity mucosa, while in the salivary gland its concentration decreases, although the norepinephrine content remains at the control level. The concentrations of these amines do not vary in saliva, but their release with saliva increases. Presumably, the salivary glands accumulate and release catecholamines during the secretion cycle.

Key Words: *salivary glands; catecholamines; oral cavity mucosa*

The salivary glands release catecholamines with saliva during background and evoked secretion [4,7,9]. During stress, this function of salivary glands seems to be very important, because stress increases plasma contents of epinephrine (EN), norepinephrine (NEN), and other biogenic amines. It is still unclear whether the release of individual catecholamines by salivary glands is selective under conditions when their plasma level is excessive. We attempted to find out how much the release of exogenous NEN with saliva is changed when its plasma level is elevated.

MATERIALS AND METHODS

Experiments were carried out on 57 random-bred male albino rats weighing 250±25 g. The rats were divided into the following groups: intact animals with background saliva secretion; rats with saliva secretion evoked by pilocarpine (1 mg/kg) after parenteral injection of NEN (40 mg/kg); rats with background saliva secretion after parenteral injection of NEN (40 mg/kg). To synchronize secretory glands of gastrointestinal tract, all animals were deprived of food 24 h prior to experiment. Access to water was not limited. Saliva samples for determination of catecholamine levels were taken during a 40-min period under Nembutal anesthesia (40 mg/kg). Ninety minutes postinjection, blood samples (0.5 ml), pieces of the left submaxillary salivary gland (10 mg), and samples of oral cavity mucosa were taken from narcotized rats. NEN and EN were determined using high-performance liquid chromatography with electrochemical detection [2,3,5,6,8].

The results were statistically analyzed using Student's test [1].

RESULTS

The effect of exogenous NEN on the catecholamine level in salivary gland, oral cavity mucosa, and saliva was studied. Table 1 shows that 1.5 h postinjection of NEN, its content did not differ from the control in all examined tissues, although plasma level of EN increased. It should be noted that an increase in plasma NEN did not affect its content in salivary gland and in oral cavity mucosa. Other data were obtained during stimulation of the salivary gland

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TABLE 1. Norepinephrine (NEN) and Epinephrine Levels in Salivary Gland, Oral Cavity Mucosa, and Blood of Rats Injected with NEN without Pilocarpine *(M±m)*

Tissue	Control	After injection of NEN
Norepinephrine		
Salivary gland, ng/g	925±118 (9)	1051±83 (8)
Oral cavity mucosa, ng/g	296±34.4 (8)	226±32 (9)
Blood, ng/ml	3.2±0.78 (8)	3.8±0.76 (10)
Epinephrine		
Salivary gland, ng/g	69±15 (8)	72.2±15.2 (9)
Oral cavity mucosa, ng/g	67.7±15.7 (8)	57.4±12.6 (9)
Blood, ng/ml	0.33±0.05 (8)	1.1±0.26 (9)*

Note. *$p<0.01$ compared with control. Here and in Table 2: brackets show the number of cases.

TABLE 2. Norepinephrine (NEN) and Epinephrine Levels in Salivary Gland, Blood, Oral Cavity Mucosa, and in Salivary Gland in Rats during Stimulation of Salivary Glands with Pilocarpine after Injection of NEN *(M±m)*

Tissue	Control	After injection of NEN
Norepinephrine		
Oral cavity mucosa, ng/g	272±51.6 (9)	747.6±139.9 (12)**
Salivary gland, ng/g	1455±349 (9)	1213.9±235.2 (10)
Salivary concentration, ng/ml	4.67±0.54 (9)	4.43±0.98 (10)
Salivary release, ng/40 min	0.75±0.08 (11)	1±0.14 (11)***
Blood, ng/ml	3.7±0.42 (9)	3.31±0.59 (13)
Epinephrine		
Oral cavity mucosa, ng/g	40.2±7.5 (7)	64.6±11 (11)*
Salivary gland, ng/g	100±21.6 (7)	34.7±7.3 (9)**
Salivary concentration, ng/ml	1.18±0.25 (10)	0.82±0.11 (12)
Salivary release, ng/40 min	0.03±0.05 (12)	0.15±0.2 (12)***
Blood, ng/ml	0.59±0.08 (8)	0.4±0.07 (10)

Note. *$p<0.05$, **$p<0.01$, ***$p<0.001$ compared with control.

secretion by pilocarpine (Table 2). Although there were no changes in plasma NEN and EN, in the oral cavity mucosa EN concentration was markedly increased, while in the salivary gland it decreased, NEN content remaining at the control level. The salivary concentration of NE and NEN did not vary, but their release with saliva increased.

Our data show that salivary gland acquires the ability to accumulate and release catecholamines mainly during the secretory cycle. At this period, the neurotransmitter concentration in the salivary gland remains at the control level (NEN) or decreases (EN). Enhancement of NEN release with saliva seems to contribute to an increase in the concentration of the neurotransmitter in the oral cavity mucosa, which is characterized by increased permeability to these substances.

Our findings indicate that the decrease in EN plasma concentration during secretory cycle was partially evoked by an increase of its release with saliva. It shows that the salivary glands can remove excessive catecholamines from blood plasma.

REFERENCES

1. I. A. Oivin, *Patol. Fiziol.*, No. 4, 76-79 (1960).
2. A. D. Yudaev and V. N. Titov, *Lab. Delo*, No. 3, 34-38 (1990).
3. H. Aliger and J. McManus, *Clin. Lab. Med.*, **8**, No. 2, 110-112 (1985).
4. M. Cattel, H. C. Wolff, and D. Clark, *Am. J. Physiol.*, **109**, 375-377 (1934).
5. D. Clinton, *J. Neurosci. Methods*, No. 11, 275-273 (1984).
6. P. Hjemdahl, *Methods Enzymol.*, **42**, 521-534 (1987).
7. K. A. Houpt, K. M. Kendrich, R. F. Parrot, and C. F. De La Riva, *Horm. Metab. Res.*, **20**, No. 3, 189-190 (1988).
8. B. Kagedal and D. S. Goldstein, *J. Chromatogr.*, **429**, 177-233 (1988).
9. P. S. Larsen, *J. Pharmacol. Exp. Ther.*, **54**, 341-345 (1935).