A lectin in *Nemopanthus mucronatus* to papain treated porcine erythrocytes

C. S. Tumosa

Philadelphia Police Laboratory, Franklin Square, Philadelphia (Pennsylvania 19106, USA), 12 April 1977

Summary. Extracts of the mountain holly, *Nemopanthus mucronatus*, contained an agglutinin to papain treated porcine erythrocytes but not untreated or neuraminidase treated cells. Human erythrocytes similarly treated did not react.

Plant agglutinins or lectins have been used to determine human and animal blood group factors[1,2], to differentiate various species of animals[3] and to examine the structure of the red cell[1,4]. Several new lectins reacting with receptor sites uncovered by the action of enzymes have expanded the tools available to probe the sub-surface geometry of cells[5–8].

A lectin was found in the dried fruit of the mountain holly, *Nemopanthus mucronatus*, which agglutinated papain treated porcine erythrocytes to a titer of $^1/_{16}$ but which did not agglutinate untreated or neuraminidase treated cells. Human erythrocytes of various ABO(H), MN and RH–Hr phenotypes did not react if similarly treated.

The lectin from the mountain holly appears to react with a sub-surface antigen on the porcine erythrocyte which is not unblocked by the release of sialic acid through neuraminidase treatment but which is unblocked by the proteolytic effect of papain treatment.

This behavior indicates that the receptor site for the lectin is not at the periphery of the cell membrane since the reduction of the zeta potential by neuraminidase would have a greater effect on sites closer to the periphery of the cell than those buried deeper. The lectin becomes effective only after the removal of sterically hindering proteins on the cell membrane by a proteolytic enzyme. A similar reactivity has been noted between the *Amaranthus caudatus* lectin and bovine erythrocytes[6].

The receptor site for the lectin is also different from the T antigen found in many animal species[6] and inhibition experiments with simple sugars indicate a glucose-like specificity.

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Effects of various hormones and adrenalectomy on the levels of amylase in rat pancreas and parotid gland[1]

T. Takeuchi, M. Ogawa and T. Sugimura

Biochemistry Division, National Cancer Center Research Institute, Chuo-ku, Tokyo 104 (Japan), 1 February 1977

Summary. Dexamethasone, adrenocorticotropic hormone and thyroxine increased the amylase activities in both the pancreas and the parotid gland of infant rats. After adrenalectomy, the amylase activities of the pancreas and parotid gland were about half the control levels, suggesting that both glucocorticoid and thyroxine are involved in maintaining the amylase activities in these organs.

Administration of glucocorticoid to immature rats is known to include increase in the levels of pancreatic amylase or chymotrypsin from the immature to the mature level[2–4]. However, the abilities of hormones other than glucocorticoid to cause premature induction of pancreatic or parotid amylase in rats, and the effects of the absence of glucocorticoids on this enzyme are unknown.

Materials and methods. Donryu-strain rats were used. Hormones were injected s.c. on days 6, 7 and 8 after birth. Amylase activities in the pancreas and parotid gland were assayed on days 8, 10, 13 and/or 16 after birth. The doses of hormones injected per g b.wt per day were as follows: dexamethasone, 0.1 μg; testosterone propionate, 10 μg; adrenocorticotropic hormone (ACTH), 80 mU; growth hormone, 10 μg; L-thyroxine, 10 μg; insulin, 2 mU; and glucagon, 10 μg. Adrenalectomy was performed by the posterior approach in immature rats (day 14 after birth) (experiment 1) and young adult rats (day 40 after birth) (experiment 2)[5]. The amylase activities in the pancreas and parotid gland and the serum corticosterone level were assayed 4 and 12 days after adrenalectomy. Amylase activity was assayed as described by Ceska et al.[6] and expressed as μmoles of maltose hydrolized per min at 37 °C. Serum corticosterone was assayed by competitive binding radioassay as described by Murphy[7].

Results and discussion. After injection of ACTH or dexamethasone rats showed less increase in b.wt than control rats and, after injection of growth hormone, rats

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Table 1. Effect of various hormones on pancreatic amylase activity (mean ± SD)

	Day after birth 6	8	10	13	16	Adult
Control	30 ± 8	38 ± 13	50 ± 26	82 ± 53	105 ± 47	298 ± 32
Dexamethasone		170 ± 81*	290 ± 80**	98 ± 85	108 ± 33	
ACTH		108 ± 30*	205 ± 50**	86 ± 37		
Thyroxine		83 ± 27*	201 ± 98	227 ± 25	123 ± 13	
Dexamethasone and Thyroxine		177 ± 21***	203 ± 81*	211 ± 45**	111 ± 12	

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.

Table 2. Effect of various hormones on parotid amylase activity (Mean ± SD)

	Day after birth 6	8	10	13	16	Adult
Control	3.0 ± 0.8	2.6 ± 0.8	5.1 ± 1.9	12.6 ± 5.2	25.9 ± 9.8	603 ± 43
Dexamethasone		12.4 ± 3.1***	19.1 ± 5.4**	18.0 ± 3.1	32.1 ± 14.5	
ACTH		8.2 ± 2.1**	15.4 ± 7.8*	13.0 ± 5.6		
Thyroxine		5.9 ± 1.7*	14.8 ± 4.2**	54.1 ± 17.2**	23.2 ± 14.1	
Dexamethasone and Thyroxine		10.8 ± 1.9***	29.8 ± 4.1***	48.9 ± 13.7**	43.1 ± 2.8**	

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.

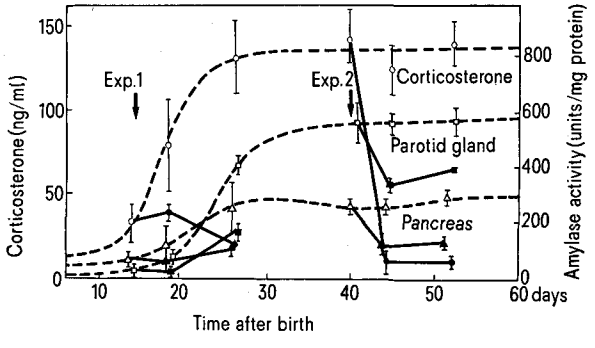
gained slightly more weight than control rats. Other hormones did not affect increase in b.wt.

Dexamethasone increased the amylase activity in the pancreas and parotid gland of immature rats[4]. Morphologically, pancreatic acinar cells of treated immature rats showed more increase in number of adult type electron-dense zymogen granules than those of controls. ACTH induced increases in the amylase activities in the 2 organs, which may be due to the stimulated corticosterone (tables 1 and 2).

Thyroxine induced amylase activity in both the pancreas and parotid gland. Yeh and Moog[8] found that thyroxine did not elevate the plasma corticosterone level in hypophysectomized rats, and claimed that this effect was independent of adrenocortical function. The effects of simultaneous administration of thyroxine and dexamethasone on the pancreas and parotid gland show the characters of each hormone; the rapid increase of amylase activity may be due to dexamethasone and the prolonged effect to thyroxine (tables 1 and 2).

Testosterone, growth hormone, insulin and glucagon had no effect on the amylase activity in either the pancreas or parotid gland. Adrenalectomy decreased the serum corticosterone level more in young adult rats than in infant rats; after adrenalectomy, the serum corticosterone in infant rats (experiment 1) was about one-fifth of the control level, whereas that in young adult rats (experiment 2) it was about one-tenth. However, adrenalectomy resulted in decreases in the amylase activities in the 2 organs to about half to three-quarters of the control levels in rats of both ages (figure). Thus, although factors secreted from the adrenal gland maintain the amylase activities in both organs at normal levels, other factors such as thyroxine also seem to be involved in maintaining the levels.

The serum corticosterone and thyroid hormone increase to adult level in the periweanling period[9–11], when amylase in the pancreas and parotid gland begin to increase[11]. The developmental change in the serum glucocorticoid and thyroid hormone seems to influence amylase activity in the 2 organs. This work revealed the ability of the 2 hormones to cause premature induction of amylase in the pancreas and parotid gland.



Serum corticosterone and amylase activities after adrenalectomy. ●—● Serum corticosterone level; ▲—▲ amylase activity of the pancreas; and ■—■ amylase activity of the parotid gland in adrenalectomized rats. ○—○ Serum corticosterone level; △—△ amylase activity of the pancreas and □—□ amylase activity of parotid gland in control rats. Values are expressed as mean ± SD in 3–4 experiments. More detailed data on the developmental change of serum corticosterone in controls were reported previously[11].

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