Pharmacology Biochemistry & Behavior, Vol. 19, pp. 43–47, 1983.

Changes in Activities of Fucokinase and Fucosyltransferase in Rat Hippocampus after Acquisition of a Brightness Discrimination Reaction

N. POPOV, S. SCHMIDT, S. SCHULZECK, R. JORK, B. LÖSSNER AND H. MATTHIES

Institute of Pharmacology and Toxicology
Medical Academy, Leipzigerstrasse 44, 3090 Magdeburg, G.D.R.

Received 3 January 1983

POPOV, N., S. SCHMIDT, S. SCHULZECK, R. JORK, B. LÖSSNER AND H. MATTHIES. *Changes in activities of fucokinase and fucosyltransferase in rat hippocampus after acquisition of a brightness discrimination reaction.* PHARMACOL BIOCHEM BEHAV **19**(1) 43–47, 1983.—Activities of enzymes involved in utilization of the glycoprotein precursor L-fucose (fucokinase and fucosyltransferase) were studied in rat hippocampal tissue after acquisition of a brightness discrimination reaction. Fucokinase activity was increased immediately after training, while fucosyltransferase revealed decreased values. However, 7 hr after training fucokinase activity showed normal values, while fucosyltransferase activity rose in trained animals over active and passive controls. The results are discussed in the light of a regulatory role that fucokinase and fucosyltransferase may play in fucose utilization under altered functional conditions.

Rat Hippocampus Glycoprotein synthesis L-Fucose Fucokinase Fucosyltransferase Brightness discrimination

PREVIOUS gel electrophoretic [27], lectin-binding [25,28] and histoautoradiographic [24] studies, in which rats had learned a brightness discrimination task, of hippocampal glycoproteins (with intraventricularly injected L-[1-^{3}H] fucose as precursor) revealed a biphasic increase in incorporation rate and glycoprotein content. The first increase occurred immediately upon completion of training, followed by a silent period at control levels, while the second increase was observed 7 to 9 hr after training.

Further results obtained after intraventricular and intraperitoneal application of L-fucose confirmed the assumption that glycoproteins with fucosyl endgroups in their glycan chains seem to play an essential role in the consolidation of a long-term memory trace. The metabolic system of L-fucose utilization operating during the consolidation phase seems to be an important step, as the retention of the acquired behavior tested was significantly improved in studies with two different tasks, i.e., a brightness discrimination and a conditioned avoidance of a shuttle-box paradigm, by application of L-fucose. The effect was specific to L-fucose, the physiological substrate participating in protein fucosylation, while D-fucose was ineffective [31].

L-Fucose has to be activated by enzymic steps, beginning with the phosphorylation by fucokinase [6, 10, 29], followed by formation of the activated sugar, GDP-L-fucose [9], and finally leading to the coupling of GDP-L-fucose to an acceptor glycoprotein by fucosyltransferase [4, 17, 30, 32]. Therefore, we studied the activities of fucokinase and fucosyltransferase in hippocampal tissue after acquisition of a brightness discrimination reaction in rats.

METHOD

Male rats of Wistar origin weighing approximately 180 g were used throughout. The animals were provided with food and water ad lib.

The radioactive substances, L-[1-^{3}H]fucose (specific activity 5.3 Ci=196 GBq/mmole), L-[1-^{14}C]fucose (specific activity 57 mCi=2.1 GBq/mmole) and GDP-L-[U-^{14}C] fucose (specific activity 181 mCi=6.7 GBq/mmole), were purchased from the Radiochemical Centre, Amersham, Great Britain.

Fetuin and ovomucoid were obtained by SERVA, Heidelberg, F.R.G.

Behavioral Model

The rats were assigned to one of three experimental groups: trained animals, active controls (pseudotrained animals) and passive controls.

The animals were trained in a Y-shaped chamber to attain a shock-motivated brightness discrimination reaction [22]. The learning task was to escape from a mild electric shock applied to the grid floor of the starting compartment, into the illuminated alley of the Y-chamber. This is contrary to the rat's innate reaction to escape predominantly into the dark.

Whenever the rat entered the non-illuminated alley of the chamber it was punished by foot-shock. The side of illumination was changed after every three trials (runs) so as to avoid position training. The runs were considered as positive responses only if the animals entered the illuminated alley of the chamber. The rats had to perform 40 runs during a training session of approximately 45 minute duration. The training criterion was reached when the rat had performed not less than 28 positive runs and at least 7 positive changes, i.e., positive responses after the side of illumination was changed. Only animals that had reached the criterion after a single session of 40 runs were included in enzymic studies. The active control was subjected to an equal number of foot-shocks, performing the same number of runs as the corresponding trained animal, but illumination and foot-shock release were paired randomly so that the animal could not learn (pseudotraining). The passive controls were allowed to stand in the home cage.

For the study of fucokinase activity in the hippocampus, the animals were killed by neck blow and decapitated in three series (10 min, 4 hr and 7 hr after training), while four series (10 min, 4 hr, 7 hr and 24 hr after training) were adopted for fucosyltransferase activity investigations. Animals within a series were each killed and prepared for enzyme assay at the same time of day.

Assay of Fucokinase Activity

The enzyme fucokinase (ATP: 6-deoxy-L-galactose-1-phosphotransferase, E.C. 2.7.1.52) catalyzes the phosphorylation of L-fucose to form β-L-fucose 1-phosphate.

The elaborated assay for determination of fucokinase activity in brain tissue is a modification of the liver tissue procedure described by Ishihara *et al.* [11]. In our assay, L-1-^{14}C]fucose was also employed as substrate, but the radioactivity of the formed L-[1-^{14}C]fucose 1-phosphate was determined by double-label counting after addition of L-[1-^{3}H]fucose 1-phosphate as internal standard, during the last minute of incubation. Briefly, in each case the hippocampal tissue of one rat was homogenized in a mixture the volume of which was nine times that of the tissue and consisted of 130 mM potassium fluoride, 60 mM Tris-HCl-buffer, pH 8.5 (measured at 23°C) and 3 mM dithiothreitol [16]. The potassium fluoride concentration indicated was found to be the optimal initial amount which, in the incubation medium, drops to 84 mM and inhibits the activity of GDP-L-fucose pyrophosphorylase, i.e., it blocks further conversion of formed β-L-fucose 1-phosphate to GDP-L-fucose. All procedures were carried out at 0–4°C, unless otherwise indicated. The homogenate was centrifuged at 20,000 g for 30 minutes. The resulting supernatant was then centrifuged at 20,000 g for another 15 minutes. This supernatant served as the crude enzyme source, of which 100 μl were thoroughly mixed with 10 μl ATP (50 mM), 10 μl Tris-HCl-buffer (500 mM), pH 8.5 (measured at 23°C), and 20 μl bidistilled water. The reaction was started in a water bath at 37°C by addition of 10 μl of a solution containing 50 mM $MgCl_2$ and 0.5 mM L-[l-^{14}C]fucose (96,000 dpm=1.6 kBq). The mixture was incubated at 37°C for 45 minutes while being thoroughly mixed at 5 minute intervals. During the least minute of incubation, 96,000 dpm (1.6 kBq) L-[1-^{3}H]fucose 1-phosphate (prepared in our laboratory) were added and the reaction was terminated by quickly placing the incubated mixture on an anion-exchange microcolumn (0.35 ml Dowex 1×8, 200–400 mesh, in the formiate form) at room temperature. The untransformed L-fucose was eliminated by two washing procedures—with 18 ml and 8 ml of 0.01 M and 0.02 M ammonium formiate buffer, pH 7.5, respectively. The elution of formed L-fucose 1-phosphate was started by addition of 2.5 ml of 1.5 M ammonium formiate buffer. Since the main quantity of the reaction product appeared after addition of 1.5 ml of 2.0 M ammonium formiate buffer, the latter eluate was mixed with 0.5 ml methanol and 13 ml scintillation cocktail (6 g PPO, 0.2 g POPOP, 8 ml acetic acid, 100 g naphthalene in 1 l 1,4-dioxane) for radioactivity measurement. The samples were counted on a Multimat scintillation spectrometer (Intertechnique, Plaisir, France) to estimate double-labeling (^{3}H and ^{14}C). The measured ^{14}C-radioactivity of formed L-fucose 1-phosphate was corrected by ^{3}H-radioactivity counts of the internal standard added at the end of the incubation period. Under control conditions, i.e., in material obtained from passive controls, approximately 20,000 dpm of L-[1-^{14}C]fucose 1-phosphate formed per 500 μg protein after 45 minute incubation were measured in each sample. Fucokinase activity is expressed as pmoles L-fucose 1-phosphate formed, μg^{-1} protein, hr^{-1} (Table 1).

Assay of Fucosyltransferase Activity

The enzyme fucosyltransferase (GDP-fucose: glycoprotein fucosyltransferase, E.C. 2.4.1.68) catalyzes the transfer of L-fucose from GDP-L-fucose to nascent endogenous glycoconjugates (predominantly of growing glycoprotein molecules) or to exogenous acceptors such as desialylated fetuin, ovomucoid etc.

In the present work as well as in previous investigations [15], fucosyltransferase activity in Triton X-100-solubilized microsomes from rat hippocampus was determined essentially as described by Zatz and Barondes [32] by measuring the incorporation of L-[U-^{14}C] fucose from GDP-L-[U-^{14}C]fucose into desialylated fetuin and ovomucoid. Fetuin was desialylated chemically as described by Suckling and Hunter [30]. All critical procedures were carried out at 0–4°C. Under the conditions applied the fucosyltransferase reaction was shown to be linear over at least 60 minutes and over a range from 10 to 60 μg of solubilized microsomal protein [15]. Under control conditions, i.e., in material obtained from passive controls, approximately 1900 dpm per 30 μg protein after 30 minute incubation were measured in each sample. Fucosyltransferase activity is expressed as pmoles fucose transferred to acceptor, μg^{-1} protein, hr^{-1} (Table 2).

Protein Determination

Protein estimations were performed by the method of Lowry *et al.* [18] using recrystallized bovine serum albumin as the reference substance.

Statistics

Student's *t*-test in pairs and groups was used to compute the statistical significance of differences between means obtained from trained animals and corresponding controls.

RESULTS

Fucokinase activity was studied in hippocampal tissue of 30 triplets (30 trained animals, 30 active controls and 30 passive controls). The trained animals performed 30.00±0.46 (S.E.M.) positive runs (responses) out of a total of 40 runs exhibiting 7.86±0.22 positive runs after changing the alley-illumination side during the training session. Fucosyl-

TABLE 1

FUCOKINIASE ACTIVITY IN RAT HIPPOCAMPUS AT DIFFERENT TIMES AFTER ACQUISITION OF A BRIGHTNESS DISCRIMINTION TASK

Series	Time after training	Number of triplets	Passive controls	%	Active controls	%	Trained animals	%
1	10 min	9	2.83 ± 0.26	100	3.14 ± 0.32	111	3.71 ± 0.36*	131
2	4 hr	11	2.43 ± 0.10	100	2.35 ± 0.14	97	2.75 ± 0.23	113
3	7 hr	10	2.87 ± 0.13	100	2.96 ± 0.27	103	2.91 ± 0.22	101

Fucokinase activity is expressed as pmoles L-fucose 1-phosphate formed; μg^{-1} protein; hr^{-1}; means ± S.E.M. *Indicates $p<0.05$ (Student's t-test in pairs) for means of trained animals vs. active and passive controls.

TABLE 2

FUCOSYLTRANSFERASE ACTIVITY IN RAT HIPPOCAMPUS AT DIFFERENT TIMES AFTER ACQUISITION OF A BRIGHTNESS DISCRIMINATION TASK

Series	Time after training	Number of triplets	Passive controls	%	Active controls	%	Trained animals	%
			Desialofetuin as exogenous acceptor					
1	10 min	19	0.97 ± 0.05	100	0.86 ± 0.08	89	0.72 ± 0.05*†	74
2	4 hr	5	0.87 ± 0.02	100	0.89 ± 0.04	102	0.91 ± 0.06	105
3	7 hr	12	1.00 ± 0.10	100	0.99 ± 0.08	99	1.41 ± 0.14*†	141
4	24 hr	5	1.00 ± 0.03	100	1.04 ± 0.03	104	1.07 ± 0.06	107
			Ovomucoid as exogenous acceptor					
1	10 min	9	0.25 ± 0.02	100	0.24 ± 0.02	96	0.19 ± 0.02*†	76
3	7 hr	12	0.19 ± 0.02	100	0.19 ± 0.02	100	0.24 ± 0.02*	126
4	24 hr	5	0.19 ± 0.02	100	0.21 ± 0.02	111	0.21 ± 0.02	111

Fucosyltransferase activity is expressed as pmoles fucose transferred to acceptor; μg^{-1} protein; hr^{-1}; means ± S.E.M.; * and † indicate stastistically significant differences ($p<0.05$) between means of trained animals vs. active and passive controls, computed by using Student's t-test in pairs and groups, respectively.

transferase activity was studied in 41 triplets (123 rats). The trained animals performed 32.34±0.36 positive runs showing 8.96±0.23 positive reactions after changing the side of illumination. Thus, all trained animals involved in this study reached the learning criterion.

The passive controls were considered for assessment of diurnal fluctuations which were negligible. To interpret the findings for trained animals and active controls (pseudotraining), reference was made in each case to the passive controls within a series. The results are presented in Tables 1 and 2.

Figure 1 summarizes the training-induced changes in enzyme activities. As shown in this figure, 10 minutes upon completion of training fucokinase activity was increased vs. active and passive controls, while fucosyltransferase activity (studied by means of two different exogenous acceptors) was decreased below the level of control animals, and, vice versa, 7 hr after training fucokinase activity decreased to control levels, while fucosyltransferase activity was significantly increased in hippocampal tissue. Twenty-four hours after acquisition of the brightness discrimination task fucosyltransferase activity was found to have reached the control level.

DISCUSSION

Glycoproteins which are major constituents of synaptic and other neuronal membranes, are thought to play an essential role in determining the efficiency of interneuronal connections [1, 2, 3, 7, 8, 19].

The present findings suggest that fucokinase and fucosyltransferase, two enzymes which are involved in fucose activation and utilization, respectively, may play a regulatory role in glycoprotein metabolism under altered functional conditions. Thus, an increase in fucokinase activity accompanied by a lowered fucosyltransferase activity was observed 10 minutes after acquisition, while 7 hours upon completion of training the fucokinase activity attained control values, whereas the fucosyltransferase activity revealed increased values in trained animals over active and passive controls.

The early change in fucokinase activity is thought to be the result of conformational alterations of the existing enzyme due to short-term operating regulatory processes involving, for instance, cyclic nucleotides, thus activating fucokinase immediately after training. Preliminary studies on

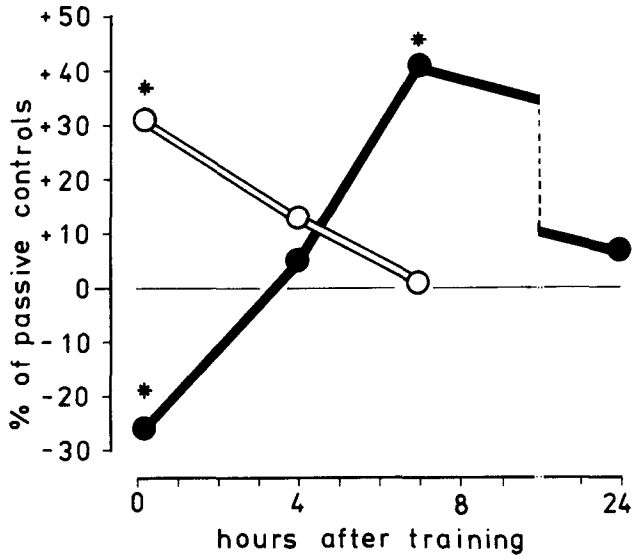

FIG. 1. Changes in activities of fucokinase (○) and fucosyltransferase (●) in rat hippocampus of trained animals expressed as percentage deviations as referred to the activities of the corresponding passive controls (zero line) at different times after acquisition of a brightness discrimination task. The asterisks indicate statistically significant differences between means obtained for trained animals vs. those obtained for active and passive controls. Further explanations in text.

hippocampal slices in vitro (Jork *et al.*, in preparation) showed an increased formation of fucose 1-phosphate, the product of fucokinase reaction, as a result of the interaction of dopaminergic agonists with corresponding receptor sites in hippocampal structures. The percentage increase in fucose 1-phosphate was similar in both experiments, (a) immediately after training and (b) upon treatment of slices with dopaminergic drugs. Moreover, in both experiments fucose incorporation into hippocampal tissue was also increased. Since dopaminergic inputs may mediate emotional influences or reward, weighting in this way the significance of specific information, it can be postulated that during acquisition of an acquired behavior distinct systems operating in the hippocampus can influence metabolic processes by controlling enzymic reactions through second messenger system. It is a reasonable assumption that, with enhancing, learning-induced activation of the dopaminergic influence during acquisition of an acquired behavior, glycoproteins are increasingly synthesized, transported through the dendritic tree and incorporated into the formerly facilitated synaptic structures in order to maintain their activated states as a permanent trace.

The reduced activity of fucosyltransferase 10 minutes after training is consistent with lowered values obtained from experiments on hippocampal slices treated with dopamine or apomorphine [15], and cannot be explained to date. In the latter finding obtained in vitro, fucosyltransferase was not believed to be essential as a regulatory system in an increased utilization (incorporation) of fucose after treatment with dopaminergic drugs. This has appeared to be true, at least, of the phase immediately after interaction with dopaminergic agonists. On the other hand, 7 hours after training the activity of fucosyltransferase was enhanced, suggesting that this enzyme may play an essential role for changes in fucosylation processes during the late consolidation phase of memory formation.

Despite the decreased fucosyltransferase activity observed immediately after training, previous studies in our laboratory on hippocampal glycoproteins did reveal an increased incorporation rate of labeled fucose during the early post-training phase [12, 13, 14, 24, 27]. Similarly, studies on hippocampal slices [13,14] as well as other experiments in vivo [5] revealed an increased incorporation into brain tissue under the conditions of unchanged [5] or even decreased [15] fucosyltransferase activity. So, the reduced or unchanged fucosyltransferase activity in behavioral experiments as well as after treatment of tissue slices by dopaminergic drugs had no influence on the increased fucose incorporation into glycoproteins in vivo and in vitro. Consequently, fucosyltransferase is felt not to be the limiting step of the enzyme chain of fucose utilization.

To summarize, the present findings permit the assumption that the fucokinase reaction is most likely influenced by short-acting regulatory mechanisms operating during or immediately after training, while the increase in fucosyltransferase activity 7 hours after training is hardly explained by qualitatively similar mechanisms. Since protein synthesis as well was increased 7–9 hours after training [20, 21, 23, 26] using the same behavioral model, it can be assumed that the increased fucosyltransferase activity reflects an increased formation of proteins in the late consolidation phase rather than a late conformational change of the enzyme.

It should be the aim of future investigations to elucidate the limiting step within the regulatory chain controlling fucose utilization under functional conditions.

ACKNOWLEDGEMENTS

The skillful technical assistance of Mrs. Renate Sülldorf, Mrs. Helga Pakendorf, Mrs. Uta Riechert and Mrs. Dora Nüss is gratefully acknowledged.

REFERENCES

1. Barondes, S. H. Brain glycomacromolecules and interneuronal recognition. In: *The Neurosciences, Second Study Program*, edited by F. O. Schmitt. New York: Rockefeller University Press, 1970, pp. 747–760.
2. Bogoch, S. *The Biochemistry of Memory: With an Inquiry into the Function of the Brain Mucoids*. New York: Oxford University Press, 1968.
3. Bogoch, S. Glycoproteins of the brain of the training pigeon. In: *Protein Metabolism of the Nervous System*, edited by A. Lajtha. New York: Plenum Press, 1970, pp. 555–569.
4. Broquet, P., M. N. Perez-Gonzalez and P. Louisot. Characterization of a rat brain fucosyl-transferase. *J Neurochem* **32**: 599–606, 1979.
5. Burgoyne, R. D. and S. P. R. Rose. Changes in glycoprotein metabolism in the cerebral cortex following first exposure of dark-reared rats to light. *J Neurochem* **34**: 510–517, 1980.
6. Coffey, J. W., M. Miller and O. Z. Sellinger. The metabolism of L-fucose in the rat. *J Biol Chem* **239**: 4011–4017, 1964.

7. Irwin, L. N. Glycolipids and glycoproteins in brain function. In: *Reviews of Neuroscience*, vol 1, edited by S. Ehrenpreis and I. J. Kopin. New York: Raven Press, 1974, pp. 137–179.
8. Irwin, L. N., R. A. Barraco and D. M. Terrian. Protein and glycoprotein metabolism in brains of operantly conditioned pigeons. *Neuroscience* **3:** 457–463, 1978.
9. Ishihara, H. and E. C. Heath. The metabolism of L-fucose. IV. The biosynthesis of guanosine diphosphate L-fucose in porcine liver. *J Biol Chem* **243:** 1110–1115, 1968.
10. Ishihara, H., D. J. Massaro and E. C. Heath. The metabolism of L-fucose. III. The enzymatic synthesis of β-L-fucose 1-phosphate. *J Biol Chem* **243:** 1103–1109, 1968.
11. Ishihara, H., H. Schachter and E. C. Heath. L-Fucose kinase from pig liver. *Methods Enzymol* **28:** 399–402, 1972.
12. Jork, R., G. Grecksch, M. Jirka, B. Lössner and H. Matthies. Apomorphine and glycoprotein synthesis in rat hippocampus. *Pharmacol Biochem Behav* **12:** 317–318, 1980.
13. Jork, R., B. Lössner and H. Matthies. Dopamine and macromolecule synthesis in rat hippocampus. *Pharmacol Biochem Behav* **11:** 247–249, 1979.
14. Jork, R., B. Lössner and H. Matthies. The influence of dopamine on the incorporation of different sugars into total proteins of hippocampal slices. *Pharmacol Biochem Behav* **13:** 303–304, 1980.
15. Jork, R., S. Schmidt, S. Schulzeck, B. Lössner, N. Popov and H. Matthies. Mechanisms of dopamine induced changes in hippocampal glycoprotein metabolism. *Pharmacol Biochem Behav* **17:** 203–207, 1982.
16. Kilker, R. D., D. K. Shuey and G. S. Serif. Isolation and properties of porcine thyroid fucokinase. *Biochim Biophys Acta* **570:** 271–283, 1979.
17. Louisot, P. and P. Broquet. Subcellular localization of glycosyl-transferases in synaptosomes and mitochondria of brain. In: *Central Nervous System. Studies on Metabolic Regulation and Functions*, edited by E. Genezzani and H. Herken. New York: Springer Verlag, 1974, pp. 164–166.
18. Lowry, O. H., H. J. Rosenbrough, A. L. Farr and R. J. Randall. Protein measurement with the Folin phenol reagent. *J Biol Chem* **193:** 265–275, 1951.
19. Margolis, R. K. and R. U. Margolis. Structure and distribution of glycoproteins and glycosaminoglycans. In: *Complex Carbohydrates of Nervous Tissue*, edited by R. U. Margolis and R. K. Margolis. New York: Plenum Press, 1979, pp. 45–73.
20. Matthies, H. Learning and memory. In: *Advances in Pharmacology and Therapeutics*, vol 5, *Neuropsychopharmacology*, edited by C. Dumont. New York: Pergamon Press, 1978, pp. 117–135.
21. Matthies, H., W. Pohle, N. Popov, B. Lössner, H.-L. Rüthrich, R. Jork and T. Ott. Biochemical mechanisms correlated to learning and memory formation. Facts and hypotheses. *Acta Physiol Hung* **48:** 335–355, 1976.
22. Ott, T. and H. Matthies. Some effects of RNA precursors on development and maintenance of long-term memory: Hippocampal and cortical pre- and post-training application of RNA precursors. *Psychopharmacologia* **28:** 195–204, 1973.
23. Pohle, W. and H. Matthies. Incorporation of ^{3}H-leucine into brain cells after learning. *Pharmacol Biochem Behav* **2:** 573–577, 1974.
24. Pohle, W., H.-L. Rüthrich, N. Popov and H. Matthies. Fucose incorporation into rat hippocampus structures after acquisition of a brightness discrimination. A histoautoradiographic analysis. *Acta Biol Med Ger* **38:** 53–63, 1979.
25. Popov, N. and H. Matthies. Changes in hippocampal glycoproteins during learning and memory processing. In: *Neurobiology of the Hippocampus*, edited by W. Seifert. London: Academic Press, 1983, pp. 473–487.
26. Popov, N., S. Schulzeck and H. Matthies. Changes in labeling of soluble and solubilized hippocampus proteins after a learning experiment in rats. *Acta Biol Med Ger* **35:** 213–219, 1976.
27. Popov, N., S. Schulzeck, W. Pohle and H. Matthies. Changes in the incorporation of [^{3}H]fucose into rat hippocampus after acquisition of a brightness discrimination reaction. An electrophoretic study. *Neuroscience* **5:** 161–167, 1980.
28. Popov, N., S. Schulzeck, W. Pohle and H. Matthies. Alterations in rat hippocampal glycoproteins after acquisition of a brightness discrimination reaction. In: *Neuronal Plasticity and Memory Formation. IBRO Monograph Series, Nr. 9*, edited by C. Ajmone Marsan and H. Matthies. New York: Raven Press, 1982, pp. 193–196.
29. Richards, W. L. and G. S. Serif. Canine thyroid fucokinase. *Biochim Biophys Acta* **484:** 353–367, 1977.
30. Suckling, A. J. and G. D. Hunter. Glycosyl transferase activity in normal and scrapie-affected mouse brain. *J Neurochem* **22:** 1005–1012, 1974.
31. Wetzel, W., N. Popov, B. Lössner, S. Schulzeck, R. Honza and H. Matthies. Effect of L-fucose on brain protein metabolism and retention of a learned behavior in rats.. *Pharmacol Biochem Behav* **13:** 765–771, 1980.
32. Zatz, M. and S. H. Barondes. Particulate and solubilized fucosyl transferase from mouse brain. *J Neurochem* **18:** 1625–1637, 1971.