TABLE I

*Influence of brain and corpora allata extracts on the release of lipids from the fat body* Achaea janata L.

| Individual glycerides | Microgram glycerides (as glycerol or palmitic acid equivalent) or fatty acids/ml Haemolymph | | |
|---|---|---|---|
| | Control | Brain extract | Corpora allata extract |
| Mono-glyceride[7] | 26·0±4·0 | 23·6±3·9 | 24·0±6·8 |
| 1,2-Diglyce-ride[7] | 32·0±2·3 | 59·0±6·0 (P 0·005) | 34·0±5·3 |
| 1,3-Diglyce-ride[7] | 29·6±3·7 | 52·0±2·9 (P 0·005) | 27·0±6·0 |
| Triglyceride[7] | 16·3±0·25 | 16·0±3·0 | 15·3±2·1 |
| Free fatty acids[7] | 36·7±7·0 | 43·0±6·8 | 39·0±6·0 |
| Total neutral lipid and free fatty acid content | 139·9 | 193·6 | 139·8 |

of aspects of lipid synthesis[3]. Isolated fat body of the *Cecropia* moth when assayed after the addition of corpora allata indicated decrease in the rate of lipid biosynthesis[5]. On the other hand it has been shown that an aqueous extract of corpora cardiaca produced a significant increase in DGL content of the haemolymph and augmented the lipid mobilization from the fat body[7]. In the desert locust neither extirpation nor implantation of corpora allata brought changes in the concentration of DGL content of the haemolymph[8]. It is believed now that the active agent in lipid metabolism is brain neurosecretary material which is stored and released by the corpora cardiaca[9]. In the present experiment it may be seen that both 1,2 and 1,3 DGL content of the haemolymph increased significantly with BE. The increase in FFA level with BE was insignificant. CAE has no appreciable influence on either glycerides or FFA levels of the haemolymph. The primary source of haemolymph DGL is the TGL store, contained within the fat body. The release of DGL from the fat body requires the hydrolysis of TGL and lipolytic activity has been reported in the fat body of several species of insects. It appears from these observations that the corpora allata in *A. janata* do not influence lipid mobilization from the fat body into the haemolymph. The significant increase in the 1,2 and 1,3 DGL level of the haemolymph containing BE supports the hypothesis that the neurosecretory material of the brain stimulates TGL (which constitutes 86% of the total neutral lipid) hydrolysis in the fat body, resulting in the release of DGL and FFA into the haemolymph.

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1. Vroman, H. E., Kaplains, J. N. and Robbins, W. E., *J. Insect Physiol.*, 1971, **11**, 899.
2. Walker, P. R. and Bailey, E., *Ibid.*, 1971, **17**, 1125.
3. Van Handel, E. and Lea, A. O., *Science, N.Y.*, 1965, **149**, 298.
4. Goldsworthy, G. J., *J. Endocr.*, 1971, **50**, 237.
5. Gilbert, L. I., In *Adv. in Insect Physiol.*, Beament, J. W. L., Treherne, J. E. and Wigglesworth, V. B., Academic Press, New York, 1967, **4**.
6. Kallapur, V. L. and Basalingappa, S., *Experientia*, 1977, **33**, 99.
7. —, *Indian J. Exptl. Biol.*, 1978, **16**, 608.
8. Beenakkers, A. M. Th., *Gen. Comp. Endocr.*, 1969, **13**, 3.
9. Keeley, L. L. and Van Waddill, H., *Life Sci. (II)*. 1971, **10**, 737.

## BLOOD 2-3 DIPHOSPHOGLYCERATE (DPG) AND POTASSIUM HOMEOSTASIS IN THYROID DISORDERS*

IT has been well documented that hyperfunction of thyroid gland leads to increased sodium content of red blood cells,[1–5] while conflicting observations have been reported on red cell potassium homeostasis in hyperthyroid state[6–7]. Since 2–3 Diphosphoglycerate (DPG) in blood regulates potassium homeostasis[8], it was proposed to examine the effect of thyroid hormone on blood DPG and potassium homeostasis in laboratory rats.

*Materials and Methods*

Adult male Sprague-Dawley rats were used in the investigation, Hypothyroidism was induced by single intraperitoneal injection of 1 mCi $^{131}$I in each rat. Hyperthyroidism was developed by daily i.p. injection of 90$\mu$g L-thyroxine to each rat for 14 days. Thyroid status was evaluated by serum concentration of protein bound iodine and growth rate of experimental rats. Potassium in plasma and red blood cell was estimated colorimetrically[9]. Influx of potassium in erythrocytes *in vitro*, using Rubidium-86 as the marker, was studied as per recommendations[10], using Nuclear Chicago Autogamma Spectrometer Model 4219. Blood DPG was estimated colorimetrically[11]. Reduced glutathione (GSH) content of blood was studied by colorimetric procedure[12].

TABLE I

*Thyroid function: Blood DPG, GSH and potassium content of plasma and red cells*

(Data represent mean ± SEM. Figures in parentheses denote number of rats)

| Group | Plasma potassium meq/litre plasma | Red cell potassium meq/litre cells | Influx of $^{86}Rb$ in red cells cpm/0·2 ml cell | DPG $\mu$ mole/ gm Hb | GSH mg/100 ml blood |
|---|---|---|---|---|---|
| Control | 7·1 ± 0·4 (6) | 42·7 ± 1·2 (6) | 33110 ± 3353 (6) | 21·1 ± 1·0 (6) | 24·7 ± 1·0 (6) |
| Experimental (Hypothyroid) | 6·2 ± 0·1 (6) | 41·9 ± 1·0 (6) | 37362 ± 3827 (7) | 22·2 ± 1·0 (6) | 16·7 ± 0·6 (7)* |
| (Hyperthyroid) | 9·7 ± 0·2 (4)* | 102·7 ± 0·7 (4)* | 50652±2148(4)** | 39·4 ± 1·9 (4)* | 22·1 ± 0·1 (4) |

* $p < 0·001$; ** $p < 0·01$.

*Results and Discussions*

Table 1 reveals significant increase in the popassium-content of plasma and red cell with simultaneous increase in influx of $^{86}Rb$ in erythrocytes (*in vitro*) of hyperthyroid rats. It has also been observed that excess of thyroid hormone in circulation significantly enhances blood DPG in experimental rats, while potassium homeostasis and blood DPG in hypothyroid rats remain same as control. In contrast, blood GSH content in hypothyroid rat was diminished with its unchanged value in hyperthyroid state.

The phenomenon of increased plasma and red cell content of potassium with simultaneous increase in blood DPG observed in the present study was similar to that which occurs in hypoxic condition, reported from this laboratory earlier[13–14]. But the major difference between the two observations was that blood GSH in hypoxic state increases[14] along with DPG. While in the present investigation, blood DPG was increased along with simultaneous increase in plasma and red cell potassium in hyperthyroid state, without any alteration of blood GSH content. This could be due to effect of thyroid hormone on metabolic handling of sulphydryl group in the system.

Increase in blood DPG in hyperthyroid state observed in the present investigation is in agreement with the published reports[15–16] but it fails to support the findings of unaltered blood DPG in rats with hyperthyroid state, induced by the injection of triiodothyronine[17]. Results of potassium homeostasis reported here are not in agreement with the reported studies[6–7] on human patients. Reason for this observed difference could be due to difference in species employed in the study.

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1. Bockelman, W. A., *Nature*, 1958, **181**, 1136.
2. Goolden, A. W., Bateman, G. D. and Torr, *S. Br. Med. Jour.*, 1971, **2**, 552.
3. Smith, E. K. and Samuel, P. S., *Clin. Sci.*, 1970, **38**, 49.
4. Cole, C. H. and Wadelel, R. W., *J. Clin Endo. and Metab.*, 1976, **42**, 1056.
5. Pimparkar, B. D., Godas, P. V., Amin, B. M. and Chaubel, K. A., *Ind. J. Med. Res.*, 1977, **65**, 112.
6. Awaad, H. K. and Goolden, A. W. H., *Clin Sci.* 1960, **20**, 113.
7. Ismail Beigi, F. and Edelman, I. S., *Am. J. Physiol.*, 1973, **225**, 1172.
8. Gardos, G., *Acta Biochem. Biophys. Acad. Sci. Hung.*, 1966, **1**, 139.
9. Weichselbaum, T. E., *Am. J. Clin. Path.*, 1946, **7**, 40.
10. Dunn, A. and Arditti, J., *Experimental Animal Physiology*, Hold Rinehart and Winston, New York, 1969, p. 49.
11. *Sigma Technical Bulletin Number* 665, 1974.
12. Bewtler, E., Duron, O. and Kelly, B. M., *J. Lab. Clin. Med.*, 1963, **61**, 882.
13. Sarkar, S. R., Banerji, R., Singh, L. R. and Chaudhuri, B. N., *Curr. Sci.*, 1977, **45**, 522.
14. Singh, L. R., Banerji, R. and Chaudhuri, B. N. (Communicated).
15. Snyder, L. M. and Reddy, W. J., *J. Clin. Inv.*, 1970, **49**, 1993.
16. Miller, W. W. J., *Am. Med. Assoc.*, 1970, **211**, 1824.
17. Duhm, J., Deuticke, B. and Gerlach, E., *Naturwiss.*, 1969c, 56, 329.

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