Alterations in the Pituitary- Thyroid Axis in Camel *Camelus dromedarius* Infected by Larvae of Nasal Bot Fly *Cephalopina titillator*

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**Abstract:** The present study used the Chemiluminescent Microparticle Immunoassay (CMIA) to examine the alterations in pituitary-thyroid function, in camels infected with third instar larvae of *Cephalopina titillator*, by measuring the levels of triiodothyronine (T3) and thyroxine (T4) of the thyroid gland and level of thyroid stimulating hormone (TSH) of the pituitary gland. The results indicated that the infection of camels by larvae of the nasal bot fly caused a case of hypothyroidism. This was evidenced from the declines recorded in blood levels of T3 and T4. Also the data obtained point to the occurrence of a parallel decrease in the level of blood TSH. It appears that the depressed release of TSH during infection with *C. titillator* together with the subsequent decline in T3 and T4 levels from thyroid gland, might reflect the direct effect of infection on pituitary gland, and suggest decreased synthesis of T3 and T4 in response to larval infection of the nasal bot fly.

**Key words:** *Cephalopina titillator*, *Camelus dromedarius*, T3, T4, TSH

**Introduction**

The camel nasal bot fly, *Cephalopina titillator* (subfamily: Oestridae) is the only species in its genus (Sharma, 1992). It develops in the nasal cavities of camels and shows a high degree of host specificity. High level of infestation causes congestion of nasal cavity with mucous, severe inflammatory and degenerative changes, leading to extensive damage of nasopharyngeal tissues and the formation of lymphoid nodules at the site of larval attachment in the pharynx. The affected camels may snort, show abnormal behaviour and nervous symptoms including restlessness, cramp, excitation, convulsions, difficulties in breathing and may lead to death following complications due to secondary infections (Hussein et al., 1982 and 1983; Higgins, 1985; Derhalli et al., 1989 and Musa et al., 1989). In the previous studies, no results have linked *C. titillator*-infected camels with serum thyroid hormone concentrations (T3 and T4) or serum thyroid stimulating hormone (TSH) level. On the other hand, it was reported that *Trypanosoma congoense* infection rapidly impaired the function of the thyroid gland in goats as defined by considerable plasma thyroxine (T4) decrease (Mutayoba et al., 1988) and hypothyroidism in cattle (Abbe and Eley, 1992). Experimental infection of rabbits with *Trypanosoma congoense* induced a rapid decline in both T3 and T4 (Lomo et al., 1996). Similarly, studies on human sleeping sickness showed a significant decrease in 3, 5, 3'-triiodothyronine (T3) and (T4) without TSH variation (Boersma et al., 1989). Al-Qarawi et al. (2001) indicated that *Trypanosoma evansi* caused a significant case of hypothyroidism evidenced as a decrease in the thyroid triiodothyronine (T3) and thyroxine (T4) blood levels associated with a parallel decrease in the level of blood pitutary thyroidic hormone (TSH). The objective of the present investigation was to measure the levels of T3, T4 and TSH in the camel *Camelus dromedarius* during the infection with larvae of *Cephalopina titillator*.

**Materials and Methods**

**Experimental Animals:** For this study, adult male and female camels, *Camelus dromedarius* were used. Animals were obtained from abattoir of El-Basatiein, Cairo, Egypt

**Groups of Animals Under Investigation:** Camels were divided into two main groups. Each group consists of 7 animals

**Group 1:** This group is the control animals. They were free from any larval instars of the studied insect, *Cephalopina titillator*

**Group 2:** The animals of this group were infected by the larvae of *C. titillator* with third instar larval burdens of 10 – 15 (mean 13) per each camel

**Blood and Tissue Sampling:** Animals were sacrificed by cervical dislocation. Blood was collected in separated clean centrifuge tubes, allowed to coagulate and serum was separated by centrifugation at 3000 r.p.m. for 20 min. Serum specimens were quickly kept frozen at – 20°C until needed for analysis of thyroid hormones (triiodothyronine, T3 and thyroxine, T4) and thyroid stimulating hormone (TSH). The levels of T3, T4 and TSH in blood serum were determined using the Chemiluminescent Microparticle Immunoassay (CMIA). CMIA kits were provided by Abbott Laboratories Diagnostics Division. Abbott Park, IL 60064, USA.

**Statistical Analysis:** The results are reported as the mean ± S.E. Differences between means were determined by using one way analysis of variance (ANOVA) and F-test followed by Tukey's multiple range test using the Statistical Package for the Social Sciences (SPSS) version 10. Non-significant level was taken at P> 0.05.
Table 1: Changes in levels of triiodothyronine (T3), thyroxine (T4) and thyroid stimulating hormone (TSH) in serum of camels infected with third instar larvae of Cephalopina titillator.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Groups</th>
<th>Mean ± S.E</th>
<th>% of Change</th>
</tr>
</thead>
<tbody>
<tr>
<td>Triiodothyronine (T3)</td>
<td>Non-infected</td>
<td>120.0 ± 20.80</td>
<td>-78.52 ***</td>
</tr>
<tr>
<td></td>
<td>Infected</td>
<td>25.78 ± 7.08</td>
<td></td>
</tr>
<tr>
<td>Thyroxine (T4)</td>
<td>Non-infected</td>
<td>11.62 ± 1.33</td>
<td>-23.58 *</td>
</tr>
<tr>
<td></td>
<td>Infected</td>
<td>8.88 ± 1.89</td>
<td></td>
</tr>
<tr>
<td>Thyroid stimulating hormone (TSH)</td>
<td>Non-infected</td>
<td>0.014 ± 0.003</td>
<td>-14.29</td>
</tr>
<tr>
<td></td>
<td>Infected</td>
<td>0.012 ± 0.002</td>
<td></td>
</tr>
</tbody>
</table>

Numbers of camels in each group = 7
Non-significant at P>0.05.
*** Very highly significant at P<0.0001.
* Significant at P<0.01
Each bar represents a percentage of change value.
*** Very highly significant at P<0.0001

Results and Discussions

Results of thyroid hormones (T3 and T4) and thyroid stimulating hormone (TSH) in blood serum of non-infected (control) and infected camels are shown in Table 1 and graphically illustrated in Fig. 1.

Triiodothyronine (T3): The response of thyroid hormones for the action of third larval instar of C. titillator infection shows a very highly significant decrease (F=146.35, P<0.0001) in the level of T3 in serum. The value (expressed as percentage of change) was -78.52%.

Thyroxine (T4): The analysis of T4 level in serum of infected camels showed a value of -23.58%. (expressed as percentage of change). This value indicates the occurrence of a significant decrease (F=6.07, P<0.01).

Fig. 1: Effect of infection with 3rd instar larvae of Cephalopina titillator on triiodothyronine (T3), thyroxine (T4) and thyroid stimulating hormone (TSH) in serum of camels

Thyroid stimulating hormone (TSH): Studies were undertaken to examine the TSH level of serum in response to third larval instar of C. titillator infection. The results revealed the occurrence of a non-significant decrease (F=0.40, P>0.05) of -14.92%.

Although the literature contains many references to the effect of different parasites on the pituitary-thyroid axis in mammals (Ikede and Losos, 1975; Mutayoba et al., 1988; Boersma et al., 1989; Abebe and Eley, 1992; Abebe et al., 1993; Lomo et al., 1996; Haroun et al., 2000 and Al-Qarawi et al., 2001), no direct estimates of the effect of nasal bot fly C. titillator on serum T3, T4 and TSH in camels have been made before.

The present results indicated that larvae of C. titillator caused a significant case of hypothyroidism evidenced as decrease in the thyroid triiodothyronine (T3) and thyroxine (T4) blood levels associated with a parallel decrease in the level of blood pituitary thyrotropic hormone (TSH). These data suggest that the depressed release of TSH during infection together with the subsequent decline in T3 and T4 levels from thyroid gland, might reflect the direct effect of infection on pituitary and thyroid gland. In support of these observations, Al-Qarawi et al. (2001) indicated that decreases in the levels of T3, T4 and TSH of blood in camels infected with Trypanosoma evansi were recorded.

In hypothyroid animals the synthesis of long chain fatty acids from glucose and other carbohydrate precursors, as well as their oxidation, were reduced (Bray and Goodman, 1968). Since both of these processes require movements of metabolites across the mitochondrial membrane, it has been postulated that the metabolic defect in hypothyroidism may reside in the mitochondrial membrane (Bray and Goodman, 1968). Lipogenesis as well as lipolysis were reported to be largely affected by thyroid hormones (Fisher and Bull, 1967).

Roncarli and Murthy, 1975): T3 and T4 have pronounced physiological effects in the control of respiration and energy metabolism as well as in the biogenesis of the mitochondria. Their production is important in conditions such as disease that leads to alter metabolic
status. The thyroid gland is one of the endocrine glands, which is affected during parasite infection.

In our study, the infection was indicated by a significant reduction in the level of T3 and T4 blood concentrations. This impairment in thyroid function could be explained on the basis of a direct or/and an indirect (through the pituitary gland) effect of the larvae on the thyroid gland. The direct effect may be mediated by biologically active factors of parasite origin such as phospholipase A (Tizard et al., 1978), protease (Lonsdale-Eccles and Grab, 1986) and peptidase (Knowles et al., 1989) thereby causing thyroid dysfunction. On the other hand, indirect pathological changes which occur during parasites infection can lead to pituitary dysfunction (Ikedo and Loscos, 1975 and Matyoba, et al., 1988). The presence of larvae of C. titillator within the nasopharyngeal cavity and their causes for abnormal movement of the head, bleeding from the nostrils and meningitis in camels (Hussein et al., 1982; Al-Ani et al., 1991 and Bekele, 2001), might alter the pattern of pituitary TSH production and thereby prevent normal thyroid function. Protozoans of parasitic origin have been implicated in the gonadotrophic dysfunction in rats infected with T. brucei brucei (Humblart et al., 1990). Thus, an indirect antithyroid effect could have been mediated through; activation of epithelial cells, monocytes and macrophages in the immune system during infection releasing cytokines which activate and down regulate the endocrine system. The results of De Souza et al. (1989) and Honegger et al. (1991) suggested that IL-1 may be involved in the paracrine and/or autocrine regulation of pituitary function. The two molecular forms of IL-1: IL-1β and IL-1β affect the CNS and the endocrine system (Schmidt and Toci, 1990). In addition, IL-1 and II-6 (Sepolsky et al., 1987; Batman et al., 1989 and Tsagarakis et al., 1989) are known to be stimulants of the hypothalamo-pituitary axis while TNF (cachectin) is known to inhibit the secretion of pituitary hormones such as growth hormone (Walton and Cronin, 1989) and TSH (Pang et al., 1989). Pang et al. (1989) implicated TNF in causing impairment of hypothalamic-pituitary-thyroid function in rats and suggested that TNF is one of the mediators responsible for alterations in the thyroid function. On the other hand, thyroid-stimulating hormone concentrations decline following subcutaneous injection of recombinant human IL-1β in rats (Dubois et al., 1988).

The weakness that has been pointed to in the study implemented by Abebe and Eley (1992) and Al-Qarawi et al. (2001) in their failure to study the pituitary TSH output has been our target to strengthen the present study and achieve our conclusion which indicated a dysfunction of the secretomotoric function in the pituitary-thyroid axis during the state of larvae of C. titillator infection in camel.

The alteration in the T3 and T4 dynamics in the study of Al-Qarawi et al. (2001), in the camels infected with Trypanosoma evansi was not consistent with the results of Lomo et al. (1996) in the rabbits infected with Trypanosoma congoense. The depression in the T3/T4 ratio in the previous study of Al-Qarawi et al. (2001), may be due to the release of T3 by the thyroid gland under the influence of small amounts of pituitary hormones (Nejad et al., 1973).

According to the above developments, it is evident that infection with larvae of C. titillator would tend through their multiple functions to cause decrease in thyroid hormones synthesis which might explain the decrease in T3 and T4 during the experiment. Also the concomitant decrease in blood TSH level might as well as stimulate the decline of the T3 and T4 in blood.

References


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