Removal of ^{14}C-BUdR incorporated in DNA of newborn and young adult rat liver

	Time (d)						
Newborn	0*	0.33	1	2	6	13	21
^{3}H/^{14}C-ratio	3.86 ± 0.43	–	2.96 ± 0.34	–	4.52 ± 0.86	5.17 ± 0.82	12.21 ± 0.77***
^{3}H-TdR-DNA labelling (dpm/mg)	7928 ± 687	–	3848 ± 355***	–	2368 ± 330***	2322 ± 56***	2333 ± 84***
^{14}C-BUdR-DNA labelling (dmp/mg)	2054 ± 170	–	1300 ± 112***	–	524 ± 63***	449 ± 21***	191 ± 10***
Young adult							
^{3}H/^{14}C-ratio	13.19 ± 0.95	13.39 ± 0.58	14.45 ± 1.02	17.35 ± 0.93**	17.19 ± 1.37**	21.49 ± 1.60***	–
^{3}H-TdR-DNA labelling (dpm/mg)	2507 ± 172	2384 ± 259	2066 ± 164	1856 ± 299	1874 ± 433	1676 ± 168***	–
^{14}C-BUdR-DNA labelling (dpm/mg)	190 ± 20	178 ± 24	143 ± 11**	107 ± 16***	109 ± 29**	78 ± 24***	–

* 0-time-values were obtained from a group of rats killed 1 h after i. p. injection of 7.0 μCi ^{3}H-TdR together with 1.0 μCi ^{14}C-BUdR/100 g b. wt (^{3}H/^{14}C-ratio = 7) to female Wistar rats (100–120 g) in groups of 5. The same compounds were s.c. injected to newborns in quadruplicate groups of 14 rats in the case of 0 and 1 times and of 9 in the case of 6, 13, 21 times. Animals were killed at various times and radioactivity was counted on triplicated 2.5 mg DNA samples. Data are reported as mean ± SEM. ** $0.05 > p > 0.01$. *** $p < 0.01$.

radioactivity measured, in triplicate, on 2.5 samples in a previously calibrated Intertechnique SL 32 spectrometer provided with external ^{226}Ra standardization.

Results. The time-course of the removal of ^{14}C-BUdR incorporated in DNA of newborn and young adult rats liver is shown in the table: results are expressed both as the mean of the ratios ^{3}H/^{14}C and as the mean of DNA specific activity for each isotope ± SEM. The ^{3}H/^{14}C-ratios in DNA of newborns and young adults 1 h after injection are quite different (3.86 and 13.19, respectively). They also differ from the ratio of the injected mixture (7.0) and from the ratio we found in regenerating rat liver (7.04 as mean of 2 different experiments)[2] under identical experimental conditions. These differences in nucleoside mixture uptake could be due either to a dilution of the labelled compounds in the different cell nucleotide pools or to a lower discriminating capacity of DNA polymerases of newborn as regards BUdR incorporation (a kind of enzymatic 'immaturity'). We have no data supporting the one or the other hypothesis: however Packard et al.[5,6] have found no difference in the levels of incorporation of TdR or different halogenated nucleosides (BUdR, IUdR) into mice embryo DNA, even using teratogenic doses[3].

A significant BUdR removal from liver DNA began 2 d after treatment in young adults, but only after 21 d in newborns where the increase in ^{3}H/^{14}C-ratio found at 13th d did not appear to be significant. The noticeable decrease in ^{3}H-TdR labelling we found in newborns is closely related to liver growth, as shown in the figure.

Discussion. The in vivo removal here reported can be attributed to DNA repair: in fact, it is neither explainable on a purely physico-chemical basis nor is it due to cellular death followed by preferential TdR uptake by viable cells, since the amount and the radioactivity of the injected ^{3}H-TdR and ^{14}C-BUdR were many times lower than teratogenic or mutagenic or toxic dose[3,6–9]. BUdR removal is an earlier phenomenon in normal young adults than in hepatectomyzed animals (it begins at day 2 and 4, respectively) but is longer lasting in the former group (no evidence is given that BUdR removal ends at day 13 in young adults while, in hepatectomyzed rats, it ends at day 8)[2]: in newborns, the same process is evident only at 21st day, i.e. at the end of weaning. The amount of BUdR removal is 3fold in newborns while less than 2fold in adults, when considering the ratio between the data from the last and the first estimation: this could be due to incorporation of excised BUdR in growing liver. The impairment of repair activity found in newborns could likely be related to their greater sensitivity to carcinogens.

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Dilation of nervi corporis allati 2 (NCA 2) – a neurohaemal structure in *Chrysocoris stolli* Wolf. (Heteroptera: Pentatomidae)

R. K. Singh, S. B. Singh, T. R. Khan and T. K. Singh

Post-Graduate Department of Zoology, Magadh University, Bodh-Gaya, Bihar (India), 4 April 1977

Summary. The NCA 2 is dilated in about the middle of its length. Neurosecretory material is stored in this dilation. It serves as a secondary neurohaemal organ for lateral neurosecretion.

The general pattern of the neuroendocrine system in *Chrysocoris stolli* is similar to that of other heteropteran insects[1–13] but with one major exception, that is the discovery of dilations in the nervi corporis allati 2(NCA 2) which serve as neurohaemal structures not previously described in any insect belonging to this group. While the neurosecretory material produced by the medial neurosecretory cells (MNSC) of the protocerebrum passes

directly to the aorta (which serves as a neurohaemal organ for medial neurosecretion) via the nervi corporis cardiaci 1 (NCC 1), the product of the lateral neurosecretory cells (LNSC) is conveyed to the corpora cardiaca (CC) through the NCC 2. Some axons of the NCC 2 traverse the CC to form short, thick nervi corporis allati 1 (NCA 1) which enter into the corpus allatum (CA). The CA is a single globular structure having a notch in the middle which gives it a bilobed appearance (figure 1). The lateral neurosecretion is brought to the CA via the CC and the NCA 1. The axons of the NCA 1 constitute long NCA 2. The NCA 2 come out from the CA and go to the suboesophageal ganlion. While the NCA 1 is uniform in diameter, the NCA 2 is dilated at about the middle of its length, forming a kind of saccular structure (figure 2). In sections stained with paraldehyde fuchsin, this dilated region of the NCA 2 was observed to contain abundant purple stained neurosecretory material, which resembled in its staining reaction the neurosecretory material produced by the LNSC and present in the NCC 2, CC, NCA 1 and CA. However, no stainable material was observed in NCA 2 beyond the dilated region. This strongly suggests that the dilated region of the NCA 2 serves as a secondary neurohaemal site for lateral neurosecretion, the primary neurohaemal organ being the CC. Hence all the neurosecretory material produced by LNSC does not end in the CC but some passes to the NCA 2. This lateral neurosecretory material is stored in the dilated region of the NCA 2 and is released from here into the blood. Though a neurohaemal area in the NCA 2 has been reported in 2 orthopteran insects, viz. *Acheta domestica*[14, 15], and in *Gryllodes sigillatus*[16], it was not reported before in any heteropteran insect. Moreover, while in *Gryllodes* the dilatation of the NCA 2 serves as a secondary neurohaemal organ for medial neurosecretion, in *Chrysocoris* it is the secondary neurohaemal structure for lateral neurosecretion.

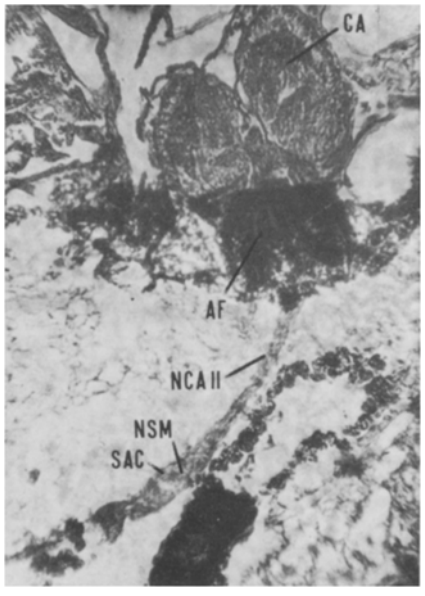

Fig. 1. Frontal section of corpus allatum (CA) of *Chrysocoris stolli* showing NCA II and sacculation (SAC) containing neurosecretory material (NSM). The black structure at the proximal end of NCA II is an artifat (AF).

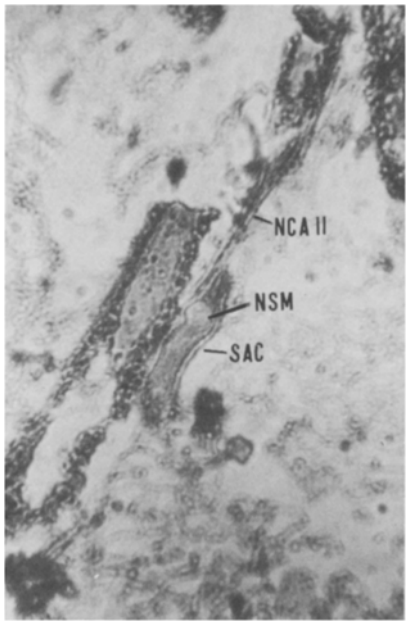

Fig. 2. Photomicrograph of a section (taken at a higher magnification) showing the sacculation (SAC) in NCA II in about middle of its length containing accumulated PF positive neurosecretory material (NSM).

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