Papanicolaou's procedure[10]. 200 consecutive cells were randomly counted and grouped as mesothelial cells, polymorphonuclear (PMN) leukocytes, lymphocytes, histiocytes, monocytes, mast cells, bare nuclei (light to dark staining nuclei without cytoplasm) and daisy cells. Randomly counting a fixed number of cells rather than counting the number of cells in an absolute volume eliminates the extreme variation resulting from counting cells per unit volume; often only a few drops were aspirated. By dividing the average cell count by 2, the percent distribution of each individual mean cell count was obtained. The SE for each mean cell count was calculated, and the percent coefficient of SE variation, a rough index of experimental error, was obtained by dividing the SE by the mean and multiplying by 100.

The normal peritoneal fluid cellular content of adult female dogs as compared to women

	Adult female dogs	Menstrual women
No. of subjects	23	34
Body weight (kg)	9.9 ± 0.2	–
Cell type	Percent distribution of cells ± SE	
Mesothelial cells	25.0 ± 1.6 (6.0)	51.8 ± 3.2
PMN leukocytes	51.4 ± 1.9 (3.0)	11.9 ± 2.2
Lymphocytes	12.9 ± 0.8 (6.0)	23.0 ± 1.9
Histiocytes	6.7 ± 1.1 (16.0)	8.8 ± 1.1
Monocytes	3.6 ± 0.5 (13.0)	–
Mast cells	–	–
Bare nuclei	–	–
Daisy cells	–	–

Percent coefficient of standard error variation in parenthesis.

The percent distribution of peritoneal fluid cellular content of adult femal dogs is dissimilar to that of women or any other species studied. For example, mesothelial cell, PMN leukocyte and lymphocyte proportions were found to be 25.0%, 51.4% and 12.9% respectively, as compared with 55.4%, 16.7% and 7.3% in female rabbits and 51.8%, 11.9% and 23.0% in women. The dog PMN leukocyte distribution, in particular, was significantly greater than that recorded in other species (table). Peritoneal fluid specimens in the dog were cloudy and viscous, while specimens in most other species studied were found to be clear, colorless and of low viscosity. This data suggests that cellular exfoliation from the abdominal cavity in dogs is quite different from that found in other species. However, we do not feel this would prevent the dog from being used as an animal model in future studies examining the effects of inflammation, cancer, hormones, pregnancy and other factors of peritoneal fluid cytology. In any case, the present study does extend our knowledge of species differences and aids in our understanding the cellular response of abdominal serous fluid in health and disease.

Removal of 5-bromo-2-deoxyuridine incorporated in liver DNA of newborn and young adult rats[1]

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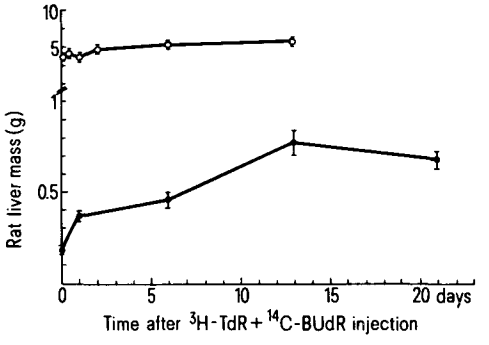
Summary. 5-bromo-2-deoxyuridine (BUdR) removal from liver DNA in newborn and young adult rats has been demonstrated: it forms the basis of a reliable method to measure DNA repair in tissues provided with detectable DNA synthetic activity.

Recently, removal of 5-bromo-2-deoxyuridine (BUdR) incorporated in DNA of regenerating rat liver has been demonstrated and attributed to repair activity[2]. Other evidence of excision repair of BUdR from DNA of mice embryo comes from Skalko's and Packard's work: BUdR, but not 5-iodo-2-deoxyuridine (JUUR), is removed in a 2-day-period and this could explain the higher toxicity of equivalent dose of the latter halogenated compound[3]. Therefore we have searched for a similar BUdR removal in newborns and young adults.



Time-course of liver mass (g) in 1-day-old (●—●) and young adult (100–120 g) (○—○) rats which were injected with a mixture of ^{3}H-TdR and ^{14}C-BUdR (7.0 μCi + 1.0 μCi/100 g b.wt). In young adults, data are the mean of 5 values ± SEM, while in newborns, the mean of 56 values for 1 h and 1 d groups, and of 36 values for 2, 6 and 21 d groups ± SEM is reported.

Material and methods. 1-day-old Wistar rats were s.c. injected, in quadruplicate groups, with a mixture of ^{3}H-thymidine (TdR) and ^{14}C-BUdR (18.5 Ci/mmole and 60 mCi/mmole, respectively. The Radiochemical Centre, Amersham, England) in the ratio ^{3}H/^{14}C = 7 (7.0 μCi TdR plus 1.0 μCi BUdR/100 g b. wt). The same tracers were i.p. injected, in the same dose, in female rats (in groups of 5) weighing 100–120 g. In young adults, livers were removed at hours 1 and 8 and at days 1, 2, 6 and 13, and processed separately. In newborns, 14-liver pools in the case of 1 h and 1 d groups and 9-liver pools in the case of 6, 13 and 21 d groups were carried out. DNA was extracted using a phenol method[4], as we previously reported[2], and

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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)

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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