yolk bodies was difficult to trace, presumably due to loss of the coat on the cytoplasmic surface.

However, since in our material numerous dense bodies acceptable as phagolysosomes (Figure 5) where fibrin seemed to be in the process of breakdown were present, we think that the partially coated vacuole is a type of phagosome by which the synovial cells of this patient were impounding fibrin from the joint space.

Zusammenfassung. Bei einem Individuum mit Multipler Sklerose, Psoriasis und Arthritis konnte man bei elektronenmikroskopischer Kontrolle von Synovialgewebe des Knies feine Filopodien nachweisen, die wahrscheinlich an der Aufnahme von Zellfragmenten, Fibrin und Erythrozyten im Gelenkspalt beteiligt sind.

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Biogenic Amines in a Retransplantable Neurogenic Teratocarcinoma

Monophyletic neurogenic teratocarcinomas are tumors composed of embryonal carcinoma cells, which are the stem cells of the tumor, and neuroectodermal tissues derived from the stem cells[1]. Despite the fact that this neuroectodermal tissue stems from rapidly proliferating malignant cells, it resembles in many respects the normal neural tissue derived from normal embryonic cells[1,2]. In this study we have analysed the neurogenic teratocarcinomas for their content of biogenic amines, known to have a specific role in neurotransmission, in order to get some insight into the functional status of this peculiar tumor.

Materials and methods. A retransplantable neurogenic teratocarcinoma, described in detail elsewhere[1], was obtained upon retransplantation of murine embryo-derived experimental teratocarcinomas. Tumor was histologically composed of embryonal carcinoma cells and neural tissue of varying degrees of maturity as determined histochemically and ultrastructurally[1]. Maturity of various elements of the tumor was, however, predominantly on the cytological level and the overall histological organization of the tumor was rather low. Neural tissue in the tumor did not mimic any organized neutral structure except embryonic neural tubes. Synapses and gliovascular junctions were noticed as the signs of the highest structural organization.

Tumor-bearing mice were decapitated, their tumors and brains removed, washed in ice cold saline, dried with filter paper and stored at 15 °C until the next day, when biochemical analyses were performed. 5-Hydroxytryptamine (5-HT) and 5-hydroxyindolacetic acid (5-HIAA) were extracted and spectrofluorometrically determined according to the method of CURZON and GREEN[3]. Noradrenalin (NA) and dopamine (DA) were also determined spectrofluorometrically using a slight modification of the method described by LAVERTY and TAYLOR[4]. Identification of each substance investigated was performed by recording their excitation and fluorescence spectra. In addition to untreated animals, an experimental group of 4 tumor-bearing animals received 25 mg/kg body weight of trancylpromine (Parnate, SK & F–385) i.p. 1.5 h prior to decapitation. Another group of 4 animals bearing neurogenic teratocarcinomas was given probenecid in a dose of 200 mg per kg body weight by single i.p. injection 1.5 h prior to decapitation.

Results. As shown in the Table, the tumors examined contained 5-HT and 5-HIAA in amounts comparable or even higher than those measured in normal mouse brains. There was, however, much more variation in the content of these substances in tumors than in the brains. It is notable that the tumors did not contain NA and DA in measurable amounts.

Treatment of tumor bearing animals with trancylpromine caused an increase of 5-HT in tumors for 29% ($P < 0.05$) and a decrease of 5-HIAA for 60% ($P < 0.01$) in relation to control values. Injection of probenecid to tumor-bearing animals caused no detectable increase in the content of 5-HIAA in the tumors.

Discussion. Our data show that neurogenic teratocarcinomas contain 5-HT and 5-HIAA, despite their structural immaturity on the histological level. Remarkable is, however, the absence of NA and DA in the tumors.

It is known that embryonal neural tissues contain less 5-HT and 5-HIAA than the adult ones[5]. The data obtained on teratocarcinomas indicate that histologically immature neural tissues do not necessarily have lower amounts of 5-HT and 5-HIAA than normal adult brain if the cells forming those tissues have attained full cytologic maturity. It is also evident that the neural tissues derived from malignant stem cells do not lack

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Content of 5-HT, 5-HIAA, NA and DA in the brain and the neurogenic teratocarcinoma of mice

	5-HT	5-HIAA	NA	DA
Brain	637 ± 37 (6)	302 ± 26 (6)	264 ± 13 (4)	458 ± 42 (4)
Tumor	1123 ± 304 (6)	276 ± 71 (6)	0 (4)	0 (4)

Values are given in nanograms per gram of tissue and represent the means ± S.E. Number of animals is given in brackets.

the capacity to form and store 5-HT and 5-HIAA. It is highly suggestive that 5-HT and 5-HIAA measured in this study were formed in the tumor. This was supported by an increase in the content of 5-HT and a decrease in the content of 5-HIAA in tumors after application of trancylpromine. Trancylpromine is an inhibitor of monoaminoxidase[6] and the changes observed after administration of this drug indicate that the enzyme exists in the neurogenic teratocarcinomas. Injection of probenecid, an inhibitor of active transport of 5-HIAA from neural tissues into the blood stream[7], did not affect the content of this substance in the tumors. We were thus unable to demonstrate the existence of an active transport for 5-HIAA out of the tumor, although this does not mean that 5-HIAA does not get out of the tumors. The absence of active transport could somehow be related to the histological immaturity of the neural tissue in the tumors.

Zusammenfassung. Neurogene Teratocarcinome weisen 5-HT und 5-HIAA-Konzentrationen auf, die ähnlich gross oder auch höher sind als im Gehirn der erwachsenen Maus. Die Tumoren sind reich an Monoaminoxydase Ein aktiver Transport von 5-HIAA aus den Tumoren konnte nicht nachgewiesen werden.

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Effect of Hemiovariectomy and Strain of Rat on Serum Gonadotropin Levels

The compensatory increase in both size and function of the remaining ovary in the rat following hemiovariectomy may be due to a reduction in gonadal steroid inhibition of the hypothalamus and pituitary gland resulting in a rise in serum gonadotropin levels. Although Edgren et al.[1] could not detect changes in gonadotropin levels following hemiovariectomy, Benson et al.[2] observed elevated plasma FSH levels 4 days after removal of 1 ovary. Using radioimmunoassays, Howland and Skinner[3] were able to detect elevations in serum levels of both LH and FSH 1 day after removal of an ovary on the day of estrus. Hemiovariectomy as late as 8 PM on day 3 of the cycle (estrus = day 1) in rats with 4-day-cycles or 2 AM on day 4 in rats with 5-day-cycles leads to doubling of the number of ova shed by the remaining ovary at the next estrus[4]. Therefore if a rise in serum levels of gonadotropins is necessary for the increase in the ova shed by the remaining ovary at the subsequent estrus, hemiovariectomy at metestrus or diestrus might lead to a rapid elevation in serum gonadotropin levels. The following study was conducted to test this hypothesis.

Materials and methods. Sprague-Dawley or Long-Evans strain rats from our own colony that were 8–10 weeks old were used in this study. The rats were assigneg to 1 of the 3 treatment groups (Table) on the mornind that a predominantly leucocytic vaginal smear was obtained. Early in the afternoon of the same day the surgical procedures were carried out. The rats were lightly anesthetized with ether and were hemiovariectomized or sham-hemiovariectomized. The control animals received no treatment. On the following morning (approximately 21 h after surgery) the animals were removed from their cages and decapitated. This was done quickly with care being taken to avoid exciting the animals in an attempt to minimize any possible effects of acute stress. Trunk blood was collected and allowed to clot. Serum samples were frozen until assayed for FSH and LH.

The concentration of LH in individual serum samples was determined by radioimmunoassay[5]. FSH concentrations were determined by a similar procedure using materials distributed by the National Institute of Arthritis

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Serum levels of LH and FSH (mean ± S.E.M.) in control, sham-hemiovariectomized and hemiovariectomized rats of 2 strains

Strain	Treatment	No. of rats	Serum LH [a] (ng/ml)	Serum FSH [b] (ng/ml)
Long-Evans	Control	11	23.6 ± 2.6	191 ± 20
	Sham	13	20.0 ± 3.8	204 ± 24
	Hemi	15	20.7 ± 2.8	289 ± 22
Sprague-Dawley	Control	16	17.0 ± 1.8	213 ± 21
	Sham	15	11.8 ± 1.2	174 ± 7
	Hemi	15	14.7 ± 1.3	350 ± 26

[a] Strain $P < 0.01$. [b] Treatment $P < 0.01$.