milk[15], pigeon milk has a higher percent of unsaturated fatty acids, particularly the linoleic and linolenic acids which are known to be the essential fatty acids for tissue metabolism[16].

Though the biological[2] and chemical assays[17] of pigeon milk for vitamins A, B1, B2 and C have revealed a low concentration, it was reported, based on the growth attained by the squabs, that their amount is adequate[2]. Moreover, it may be mentioned in this context that chicks when orally fed with small amounts of pigeon milk (0.25 g/day/chick in addition to standard ration) showed a significant increase in growth compared to controls, the increased growth being continued for many weeks even after the feeding of pigeon milk was stopped[17]. In view of these observations, it is suggested that an unidentified growth-promoting factor(s) coupled with the dietary balance of essential fatty acids and amino acids[7] might contribute to the phenomenal rate of growth in pigeon squabs[18].

Zusammenfassung. Taubenmilch ist lipidhaltig und enthält zwei wesentliche Fettsäuren: Linol- und Linolensäure. Ein nicht identifizierter Faktor zusammen mit einer fett- und aminosäurehaltigen Diät ist imstande, auf Jungvögel wachstumsfördernd zu wirken.

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Table II. The fatty acid composition of pigeon milk

Fatty acid	Percentage
1. Unsaturated	
Palmitoleic acid	8.2
Oleic acid	39.4
Linoleic acid	12.6
Linolenic acid	5.0
2. Saturated	
Palmitic acid	17.6
Myristic acid	0.7
Stearic acid	14.8
Arachidic acid	1.7

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Induction of Brain Tumours in High Yield by Administration of N-Ethyl-N-Nitrosourea to Newborn Rats

Though it has long been possible to induce experimental brain tumours in animals using topical application[1] or direct implantation[2,3] of carcinogenic polycyclic hydrocarbons, tumours of the central and peripheral nervous systems can now be induced much more easily by systemic administration of carcinogens such as N-methyl-N-nitrosourea[4].

Transplacental induction of such tumours has been studied with the very effective and less toxic N-ethyl-N-nitrosourea (ENU) in single i.v. doses of 5–80 mg/kg administered to rats in late pregnancy[5,6]. Tumours of the central and peripheral nervous systems occurred in 193 of 222 offspring of the treated mothers, and it was estimated that the nervous system of the foetal rat is about 50 times more sensitive to ENU carcinogenesis than that of the adult animal.

ENU is also an effective carcinogen for the nervous system of the rat when administered as a single dose to the newborn animal[7,8] and we used this more convenient method in our pathological studies which will be reported in detail elsewhere[9]. We were particularly interested to observe that our experiments yielded an unexpectedly high proportion of brain tumours, indicating that this procedure will be of interest to workers requiring a source of such tumours for therapeutic trials or tumour transplantation experiments. Results with our first series of 34 rats are therefore described briefly here and compared with those of other workers.

Three litters of random-bred Wistar-derived albino rats and 2 litters of random-bred Lister hooded rats were injected s.c. 24 h after birth with ENU freshly dissolved in a little ethanol and made to volume with sterile physiological saline to give a dose of 10 mg/kg body weight. Numbers of animals surviving to weaning were 14/17 albino and 20/21 hooded. They were housed on sawdust in wire cages and maintained on Thompson cube diet 42 and water ad libitum.

When functional neurological disturbances were seen the animals were anaesthetized with ether and killed by per-aortic perfusion of formalin-acetic acid-methanol mixture (FAM) (1:1:8). The brain, spinal cord and main nerve trunks were dissected in toto and immersion fixed in FAM. The blocks were embedded in paraffin wax, sectioned, and stained for routine neurohistological examination[9].

The first animal was killed 197 days after treatment. The last, killed at 680 days, was the only one in which no tumour was found. A total of 71 neural tumours was found, of which 53 (74%) were situated in the brain (Table). The preferred site was in the cerebral hemisphe-

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Tumours of the nervous system induced in rats by single neonatal administration of N-ethyl-N-nitrosourea

Authors	Dose (mg/kg)	Rats injected	Rats with tumours	Nervous-system tumours				Other tumours	Days to first tumour	Median induction time (days)
				Brain	Cord	Cranial nerves	PNS			
Druckrey, Schagen and Ivankovic[8]	5	28	9 (32%)	8	1	0	0	0	238	500
Druckrey, Schagen and Ivankovic[8]	10	16	11 (69%)	2	2	10	7	0	161	310
Druckrey, Schagen and Ivankovic[8]	20	16	16 (100%)	3	6	13	12	1	187	240
Jones, Searle and Smith[9]	10	34[a]	33 (97%)	53	9	7	2	8	197	392

[a] Excluding 4 dying very early.

res, where all but 6 of the brain tumours occurred. Histologically, the most common tumours were subependymal-plate gliomas (17) and oligodendrocytomas (10). There were 9 tumours in the spinal cord. Of the 9 tumours (13%) in the peripheral nervous system, 7 were situated in the cranial nerves and their ganglia.

The only direct comparison possible with our results is afforded by the experiments of Druckrey, Schagen and Ivankovic[8], who employed neonatal administration of ENU over the dose range 5–80 mg/kg and also investigated the effects of administration at 10 and 30 days of age.

As is seen from the Table, using the same dose and age at administration as Druckrey et al., our proportion of treated animals developing nervous-system tumours was markedly greater (97%), though the median induction time was longer. Our animals developed relatively fewer tumours of the cranial and peripheral nerves but more spinal and very many more brain tumours. Though Druckrey et al. obtained a relatively higher proportion of brain tumours at 5 mg/kg only 32% of these rats developed tumours. Similarly, when a high proportion of brain tumours occurred following repeated i.v. administration of MNU[4], only 66% of animals developed nervous-system tumours.

A possible reason for the higher yield of brain tumours in our experiment is our use of non-pure-line albino and hooded rats of the types which are widely distributed and used, whereas Druckrey et al. used pure line BD-IX rats which have been inbred for very many years. Though the influence of dietary differences cannot of course be excluded, our results suggest that albino or hooded rats, treated once neonatally with ENU at about 10 mg/kg, may be considerably more useful as a source of brain tumours for therapeutic and other studies than is apparent from the existing literature.

Several points should be stressed. The usefulness of chemically induced tumours for various studies depends on the development by the animals of well-defined signs, such as paralysis or marked loss of weight due to the tumours, but sudden haemorrhage into a tumour occasionally causes death of an animal prior to the appearance of such signs. Moreover, 9 of the 53 brain tumours in our series were microtumours only found on histological examination.

Our preliminary results at higher dosages (25 and 50 mg/kg) accord with published work in that, although the latent period is shortened, a higher proportion of peripheral nerve tumours then develop. The effect of dividing these larger doses over several days on the proportions of tumours induced might well be of interest.

Zusammenfassung. Durch einmalige Verabreichung von 10 mg/kg N-Äthyl-N-Nitrosoharnstoff am 1. Tag nach der Geburt wurden bei 33/34 Ratten 53 Hirntumoren, 9 Tumoren des Rückenmarkes, aber nur 9 im peripheren Nervensystem erzeugt, was eine bedeutend gesteigerte Rate von Hirntumoren bedeutet.

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Action of Tobacco on Lipolysis and its Modification by Insulin

The ability of nicotine to release catecholamines and therefore to stimulate lypolysis is well known. The purpose of this communication was to investigate the effect of smoking on plasmatic free fatty acid (FFA), and its modification by previous insulin treatment.

Methods. 15 smoking men (10–15 cigarettes a day) and 15 non-smoking men were tested. None of them were ill or under treatment that could influence the basal levels and behavior of the FFA. All of them fasted and lay in bed for 12 h before the test. Basal levels of FFA in both groups were determined initially and after smoking 2 black tobacco cigarettes with and without the inhalation of the smoke. Finally the experiment was repeated after the administration of 10 IU of insulin. The concentration of FFA in plasma has been determined by the method previously described by Dole and Meinertz[1].

Results. The results are shown in the Table; a significant elevation of FFA in smokers can be observed in