indicated by the observation that the width at half-height of the radiochemical peak is more than twice that of the corresponding chemical peak (*7*).

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References and Notes

1. Cyclohexane-d_{12} was obtained from Merck & Co., Ltd., Montreal, Quebec. Its isotopic purity (>99 moles percent) was confirmed by mass spectrometric analysis.
2. C. Phillips, *Gas Chromatography* (Butterworths, London, 1956), p. 15.
3. A. I. M. Keulemans, *Gas Chromatography* (Reinhold, New York, 1957), p. 16.
4. R. T. Davies, Jr., and R. W. Schiessler, *J. Phys. Chem.* 57, 966 (1953).
5. K. E. Wilzbach, *J. Am. Chem Soc.* 79, 1013 (1957); P. Riesz and K. E. Wilzbach, in preparation.
6. O. Redlich *et al.*, *J. Am. Chem. Soc.* 72, 4161 (1950).
7. This research was carried out at Argonne National Laboratory under the auspices of the U.S. Atomic Energy Commission.

19 July 1957

Sensitivity of Hamster to Colchicine

In 1952, Orsini and Pansky (*1*) reported that hamsters seem to possess a natural resistance to colchicine. They found that the hamster would survive when it was injected with dosages ranging from 0.12 to 10 mg per 100 g of body weight. Upon noting this work, we undertook an investigation to discover the lethal dosage for the hamster and to note any gross effects that might occur (*2*).

Young mature males 10 to 12 weeks of age were used for the entire series. The animals were deprived of food for 24 hours before injection but were allowed water at liberty. The weights, dosage, and subsequent history of the animals were recorded, and all animals were injected intraperitoneally in the morning.

Several animals were injected with dosages up to 10 mg/100 g of body weight, and no outward effects of the drug were noted. At the 15-mg level, slight paralysis and loss of weight were observed. The dosage was increased to 30 mg and increased by 10-mg steps thereafter. With the 30- and 40-mg dosages, all animals displayed slight paralysis in the rear quarters, drowsiness, inability to maintain equilibrium, and a marked loss of weight. The severity of these symptoms increased with increased dosages. When given 50 to 70 mg/100 g of body weight, the majority of the animals went into a coma preceded by paralysis and surges of transient tetany, from which they did not recover. One of the animals that received 50 mg and two of those that received 60 mg displayed severe nasal hemorrhages before death. No diarrhea or bloody stools were present, as has been reported for the rat (*2*). The results are recorded in Table 1.

On autopsy, pinpoint hemorrhages were present on the small and large intestines. Histological sections were made of the small intestines to observe any mitotic variation. In all cases there was a marked increase in the number of metaphase figures and an absence of spindle fibers in many cells.

Eleven males which survived the previous treatment were kept to observe any latent effects that might develop. The animals were checked, weighed, and placed with females in heat many times during the following 6 months. The males that received the 50- and 60-mg dosages never regained the tremendous weight lost, and two of the animals that received 60-mg dosages died within 3 months. Animals of these groups were hypersensitive and unsure of balance, as if their nervous or muscular system, or both, had been affected. Animals of the groups that received 30- and 40-mg dosages appeared normal and regained most or all of the weight lost.

None of the 11 males which received from 30 to 60 mg/100 g of body weight mounted a female, but all would go through the preliminary actions of breeding. However, males that received a dosage of 15 mg/100 g of body weight were fertile. Six months after the beginning of the experiment, the animals were sacrificed. In the two that had received 60-mg dosages, the following conditions were noted: the liver adhered to the diaphragm, the intestines were adhered to themselves and to the body wall, and the testes were approximately one-half of normal size. In the other groups, adhesions were not as evident and occurred only among the intestinal loops.

Histologically, the testes of these animals showed a conspicuous absence of secondary spermatocytes, spermatids, and spermatozoa. In many instances all cell types were sloughed in clumps into the lumen of the tubules. The secondary spermatocytes, spermatids, and sperm were completely absent in about one-third of the tubules of the groups that received 60 mg dosages but ranged to near normal in the group that received 30 mg dosages.

From the data given it is evident that the lethal dosage of colchicine for the hamster is approximately 70 mg/100 g of body weight. The presence of paralysis and the loss of consciousness indicate that the effects of colchicine on the nervous system are the main factor causing death. Colchicine will cause an arrest of cell mitosis in the metaphase stage in the hamster. In other work (*3*) the following is shown: (i) the effect of colchicine on the mitotic index of the crypts of Liberkühn at the 1 mg/100 g dosage level; (ii) the optimal dosage for maximal arrested metaphases; (iii) dosages that inhibit reproduction in the female; (iv) dosages that cause resorption of the fetuses in late pregnancy (*4*).

Table 1. Survival of hamsters following administration of various dosages of colchicine.

Dosage (mg/100 g of body wt.)	No. in group	Deaths		Survivals (No.)
		No.	Time after injection (hr)	
30	4			4
40	4	1	27	3
50	5	1	30	3
		1	45	
60	8	1	2	5
		1	3	
		1	108	
70	8	3	0.5	0
		2	2	
		2	3	
		1	45	
75	5	2	0.5	0
		2	2	
		1	3	

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References and Notes

1. M. W. Orsini and B. Pansky, *Science* 115, 88 (1952).
2. Colchicine was obtained from the Nutritional Biochemicals Corp., Cleveland, Ohio.
3. A description of this work is in preparation.
4. This investigation was supported in part by research grant RG4473 from the National Institutes of Health, U.S. Public Health Service.

15 July 1957

Alpha-Rhythm Responsiveness in Normal, Schizophrenic, and Brain-Damaged Persons

Routine examination of electroencephalographic records does not show that the electroencephalograms of schizophrenics differ in any consistent manner from those of normal patients (*1*). However, more active electroencephalographic techniques which introduce experimental variables in order to test for electroencephalographic changes hold more promise. Berger (*2*) long ago noted that sensory stimulation produced alpha blocking among normal persons. Later, Liberson (3) reported less reduction in alpha activity in response to light (a flash every 2 seconds) among catatonics than among psychoneurotics. Mundy-Castle (*4*) has emphasized the usefulness of more rapid photic stimulation, capable of producing alpha driving (increased amplitude or change in frequency) for