motor performance (thioridazine), while others inhibit both functions equally (chlorpromazine, prochlorperazine) and yet others (perphenazine) predominantly reduce the conditioned avoidance response and the motor activity. It seems therefore that the sedative and anti-emotive effects of these drugs are independent of each other.

Further experiments with the above-mentioned drugs (to be published) revealed a striking parallelism between their inhibitory potency on the conditioned escape response and their cataleptic activity in rats. Previous studies[10] and recent findings[6,11] have also stressed the parallelism between the cataleptic effect of these compounds in animals and the incidence of extrapyramidal side-effects in man. The depressant effect of these drugs on conditioned and motor performance of experimental animals is apparently not directly related to their therapeutic tranquillizing effect, but rather to the manifest depression, apathy or extrapyramidal symptoms. The inhibitory effect on emotional behaviour, on the other hand, seems rather to be related to their therapeutic activity.

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PATHOLOGY

An Attempt to Produce Malignant Change with Deoxyribonucleic Acid from Rat Sarcoma and Hepatoma

Benoit, Leroy, Vendrely and Vendrely[1,2,3,4] have described changes in the pigmentation of the Pekin duckling after injection of deoxyribonucleic acid from the Khaki-Campbell drake, which they interpreted as a somatic mutation. Perry and Walker[5] and Bearn and Kirby[6] have repeated similar work in the rat and failed to produce any change. Hewer and Meek[7] injected young mice with deoxyribonucleic acid from herring sperm, and within 23 days produced death from malignant disease of the intestine. Leuchtenberger, Leuchtenberger and Uyeki[8] produced cytological changes in the livers of mice by intraperitoneal injection of deoxyribonucleic acid prepared from breast cancers of C_3H mice. This work has now been repeated in the rat using deoxyribonucleic acid prepared from rat sarcoma and rat hepatoma.

The deoxyribonucleic acid used in these experiments was prepared from rat hepatoma and rat sarcoma by the method described by Kirby[9,10,11]. The final product was precipitated and dried and then made into a highly viscous suspension by adding 0·9 per cent saline. 56·8 mgm. of rat sarcoma deoxyribonucleic acid were injected in equal amounts into 18 newly born Wistar rats subcutaneously and intraperitoneally within 3 hr. of birth. Abdominal distension was caused with each injection, but no mortality resulted. 57·3 mgm. of hepatoma deoxyribonucleic acid were injected into 8 newly born rats in the same way. Each rat received either 3 mgm. of sarcoma or 7 mgm. of hepatoma deoxyribonucleic acid.

All animals survived and were weaned at 3 weeks. They grew normally from then on, and at nine months are all well. No tumours are present.

These results show, at present, a failure to produce malignant change using deoxyribonucleic acid from the rat sarcoma and hepatoma.

Deoxyribonucleic acid is now of very considerable interest in view of the transformations produced in viruses by Avery, Macleod and McCarthy[12] and the somatic mutations produced in ducks by Benoit, Leroy, Vendrely and Vendrely[1,2,3,4]. As a working hypothesis it is widely accepted that deoxyribonucleic acid is the primary genetic material[13]. In these transformation experiments the molecules of deoxyribonucleic acid become incorporated into the host and so produce a change in the virus or cell type from then on. The cancer cell may be considered as a mutant cell which proceeds to grow as a result of this mutation in an abnormal manner. On this theory, it should be possible to produce malignant changes in normal cells using deoxyribonucleic acid from cancer cells. One obstacle is to effect the incorporation of the deoxyribonucleic acid from malignant cells into the normal cell. It is believed that many workers are proceeding along these lines of research at the present time and, therefore, it is of importance to report methods that have failed to produce positive results.

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Differentiation Between a Growth-Promoting Factor and a Tumour-Susceptibility Factor in Eggs

Szepsenwol[1] reported that feeding a diet composed mainly of cooked eggs to mice resulted in a significant increase in the number of animals spontaneously developing tumours. Denton[2] found that feeding egg yolk increased the growth of chicks. Recently Hradec[3] presented evidence, based on fractionation studies, which indicated the identity of the tumour susceptibility-enhancing and the growth-promoting factors.