brains of animals receiving the radiochemical on day 22 of gestation, the number of labelled glia cells was reduced, indicating a decline in gliogenesis. From an overall point of view there were no differences in the white and the grey matter in gliogenesis. It seemed as if to sweep in broad strokes from caudal to rostral regions of the brain. An analysis of coronal sections did not reveal any mediolateral pattern of gliogenesis.

In the cerebellum intensely labelled glial elements were found in the deep cerebellar nuclei and the medullary layer surrounding them during the same period as described above, but not in the cerebellar cortex, which had only lightly labelled glial cells. This indicated that, in all likelihood, gliogenesis, in the sense of origin of glia cells out of last mitosis of their precursors and followed by their differentiation, was not present in the embryonic cerebellar cortex.

Another important fact observed was the presence of intensely labelled ependymal cells along the ventricular lumen in the brains of animals receiving thymidine-H^3 during embryogenesis. They, being highly specialized and different from their precursor neuroepithelial cells, must have arisen out of neuroepithelial cells during embryonic development. This represented ependymogenesis, and it was seen to run parallel to gliogenesis (figure 2).

Discussion and conclusions. The findings in this study have shown that gliogenesis during embryonic period is not confined to some selected structures, but is found to take place in the entire central nervous system except cerebellar cortex. Fujita[11] has demonstrated that gliogenesis in the cerebellar cortex is primarily a postnatal developmental event. The exact percentage of total glia cells formed during embryogenesis cannot be established from this study, but the results suggest this to be significant. In continuation with this, gliogenesis may be found during postnatal development and, at a low-rate, during adulthood[12-13].

Ependymogenesis was seen to follow a course parallel to gliogenesis, suggesting that these 2 histogenetic events may take place at the same time and, possibly, independently. This implies that as the last of the neuroepithelial cells undergo final mitoses to give rise to daughter cells determined to differentiate into the ependymal cells, the precursors of neuroglial elements, i.e. glioblasts, located in the embryonic white and grey matter undergo mitoses to give rise to the glial cells of embryonic origin. Some glioblasts or their undetermined daughter cells may continue to proliferate for long duration to contribute to gliogenesis during postnatal development.

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Comparison of native and microwave irradiated DNA[1]

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Summary. Chemical changes induced in the testicular DNA of irradiated Swiss male mice are described. Parameters quantified were hyperchromocity and melting temperature. The data supports the possibility that microwave causes strand separation.

A portion of the population is being exposed to increasing number of devices which emit potentially hazardous microwaves. Most of these devices have gained their access to the consumer through medical, industrial and household applications.

Material and methods. The genetic damage caused by microwave exposure is not well documented. The purpose of this study was to determine structural changes in the testicular DNA induced by exposing to microwaves. Swiss mice, 56 days old (Charles River Breeding Laboratories, Wilmington, MA), were irradiated in a walk-in anechoic chamber at Walter Reed Army Medical Center, Washington, DC. The equipment and calibration procedures have been described elsewhere[2,3]. The irradiation frequency was 1.7 GHz, and the power density was 50 mW/cm^2 with an exposure time of 30 min. The results were compared with sham irradiated controls. The parameters quantified were a) hyperchromicity, and b) melting temperatures. A Gilford 2400 spectrophotometer was used for obtaining thermal profiles.

Each animal was laid supine on a platform in front of the waveguide in the near field. To avoid movement in the field during irradiation the mice were anesthetized with 1% phenobarbital. Care was taken so that only the testes were irradiated, the rest of the body being shielded with loaded urethane foam (Eccosorb-An). Sham irradiated controls were placed outside the waveguide during exposure of the irradiated group. Animals were observed during irradiation via a closed circuit television monitor. At the termination of the exposures, the males were sacrificed, the testes pooled and DNA isolated according to the procedure outlined by Britten et al.[4,5]. Because of the problems in introducing thermocouples in the field, the body temperature during irradiation was not monitored. However, rectal temperatures were taken before and after irradiation. Normal temperatures of Swiss mice varied between 35 and 37 °C. Rectal temperatures taken after exposure indicated an increase of 1–2 °C. Minimal temperature changes occurred in the sham irradiated controls.

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Results and discussion. Recording of the melting curves were repeated at least 3 times to ensure reproducibility of results. Thermodynamic theories[6,7] predict that if intramolecular heterogenity is pronounced, the melting curves will become unusually broad and in some cases become polyphasic. Consequently recorded melting curves were normalized to the total hyperchromicity and finally transferred to a derivative form by numerical differentiation[8]. Figure 1 shows a plot of percent hyperchromicity on the Y-axis and temperature on the X-axis. Figure 2 shows the first derivative plot which is essentially a chromicity rate plot against temperature, and shows a distinct difference between the 2 inflection points. Also the plots show that structural changes in the treated DNA occurred at a lower temperature than the similar changes in the native DNA.

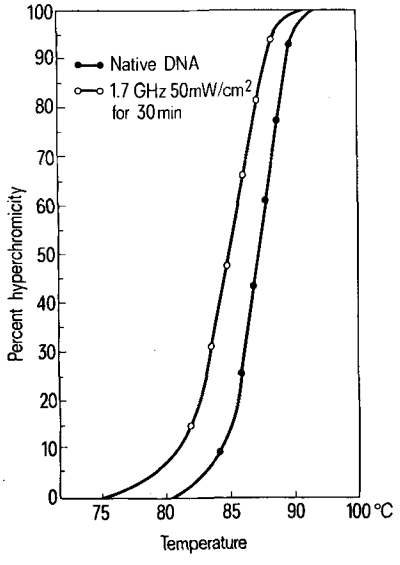

Fig. 1. Thermal profiles of native and irradiated DNAs.

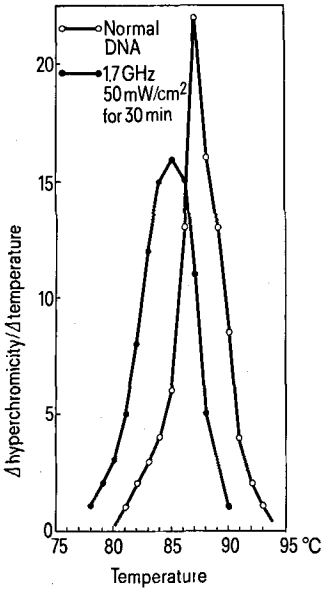

Fig. 2. Thermal profiles in differential plot of normal and irradiated DNA from the testes of Swiss mice.

Nucleotide composition of normal and irradiated DNA

Normal DNA		Irradiated DNA	
G–C	= 43%	G–C	= 38.3%
A–T	= 57%	A–T	= 61.7%
G	= 21.5%	G	= 19.5%
C	= 21.5%	C	= 19.5%
A	= 28.5%	A	= 30.85%
T	= 28.5%	T	= 30.85%
Pu/Py	= 1.0	Pu/Py	= 1.0
A/T	= 1.0	A/T	= 1.0
G/C	= 1.0	G–C	= 1.0
A + T/G + C	= 1.32	A + T/G + C	= 1.6
T_m	= 87 °C	T_m	= 85 °C
Hyperchromicity	= 30%	Hyperchromicity	= 24%

Mice were exposed to 1.7 GHz 50 mW/cm² for 30 min.

$$G + C = \frac{T_m - 69.1}{0.41}{}^{9}.$$

Pu/Py = purines/pyrimidines.

$$\% \text{ Hyperchromicity} = \frac{\text{final optical density} - \text{initial optical density}}{\text{final optical density}} \times 100.$$

The table shows the base composition for guanine and cytosine was 21.5% in the control group and 19.5% in the irradiated group; adenine and thymine 28.5% in the control group and 30.85% in the irradiated group. There was no difference in the base ratios, however, the asymmetry ratio for the control group was 1.32 while in the irradiated group was 1.6. A hyperchromic shift from 30% in the control to 24% in the irradiated sample was observed, and the melting temperature (T_m) changed from 87 °C in the control to 85 °C in the irradiated group. These observations support the possibility that irradiation causes strand separation since changes in the optical density reflects a decrease in hydrogen bonding. Consequently it is suggested that a point mutation could occur if there were imprecise base pair matching.

The proper significance of the departure of the hyperchromicity and melting temperature from the norm needs further investigation however, this preliminary work indicates that well defined and judiciously controlled experiments on DNA shall establish the degree and extent of the damage.

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