



Fig. 4. An ovary of guinea-pig, 30 days after birth. An interstitial cell. A, nucleus; J, lipide drop; P, figure myelinic. $\times 30,000$.

It is known (ECKSTEIN and PASTER⁵, STANFIELD et al.⁶) that gonadotropic hormones increase the activity of some enzymes connected with the metabolism of carbohydrates and proteins (succinic dehydrogenase, isocitric dehydrogenase).

We think the high histoenzymologic activity of the interstitium is due to the gonadotropic hormones, especially to FSH. This is proved by the rapid growth of the follicles and the presence of the described iso-folliculia. The enzyme inactivity of the interstitium and its morphological transformation probably shows that, in a short period of time, the incretion of gonadotropic hormones is held up. It may be this phenomenon has a more complicated hormonal correlative mechanism, but it is interesting for it may be a general rule for the prepuberty of mammalia.

Résumé. Ont été examinés 427 ovaires de cobayes et de jeunes filles. L'ovaire, chez le cobaye de 30 jours de la vie postnatale et chez la jeune fille âgée de 10 ans, est caractérisé par des follicules en croissance de grandeur presque égale (isofolliculie) et par la disparition presque complète de l'activité enzymatique de l'interstitium.

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⁵ B. ECKSTEIN and Z. PASTER, *Acta Endocr.* 18, 1274 (1958).

⁶ D. A. STANFIELD and Y. W. ROBINSON, *Endocrinology* 76, 390 (1965).

Effect of Temperature on Survival of γ -Irradiated Seeds of *Arabidopsis thaliana*

In the study of the biological effects of radiation, considerable effort has been devoted to modification of the response of seeds by varying the pre- or post-radiation environment. This report deals with the question of how irradiation of dry seeds and post-treatment growing temperatures may interact to influence the response of plants of *Arabidopsis thaliana* (L.) Heynh. The investigation grew out of observations made in the course of radiobiological studies with this organism in which there was a marked difference in the number of surviving individuals, depending upon post-radiation conditions¹. Evidence is presented for temperature sensitivity of radiation injury in seeds which is unrelated to induced free radicals.

Materials and methods. Seeds of the race 'Estland' of *A. thaliana* from a stock supplied by Dr. J. LANGRIDGE of Canberra were equilibrated with dry calcium chloride at room temperature prior to exposure. Radiation exposures of 150 kR were made using a cobalt-60 γ source.

Seeds were immediately hydrated in distilled water for a minimum of 1 h and then transferred aseptically to nutrient agar². All cultures were placed at 5°C for the first 24 h to overcome possible dormancy before being transferred to growth chambers. Post-irradiation treatment of 1 h in distilled water followed by 24 h on agar at 5°C should eliminate any free radicals formed due to irradiation. Thus any differential effects observed following exposure to different temperature regimes would not be attributable to interactions of radicals and temperature.

Results. The survival to flowering is less in irradiated populations than in the non-irradiated ones, as would be expected at this dosage (Table I). With respect to

¹ O. SHIFRIS, personal communication.

² J. LANGRIDGE, *Aust. J. biol. Sci.* 10, 243 (1957).

temperature, the number of survivors dropped at the higher temperature, 27°C. A decrease was seen in the non-irradiated population as well as the irradiated one, but to a greater degree in the latter. This drop in survival of non-irradiated seed, as well as irradiated, at 27°C indicates that the lower temperature (20°C) is closer to the optimum for growth in this material.

An effort was made to determine whether a critical period existed during which irradiated seeds were more sensitive to temperature differences by giving a brief exposure to near-optimum temperature (Table II). In both treatments, seeds were initially placed at 5°C for 24 h. In the first group, which was cultured continuously thereafter at 27°C, the survival was 46.4%. However, if a near-optimum temperature treatment was given during germination, followed by the higher temperature for the remaining period, survival was increased significantly to 77.4%.

In this case, an interval of only 4 days at 20°C was sufficient to increase the percentage survival from 46 to 77. It is possible that if this period were further shortened, there would still be a significant effect on survival. The magnitude of the difference in response between these 2 treatments indicates that the initial stages of growth are the more sensitive ones to temperature variation. It will be noted that lethality at 27°C in this experiment is less than was observed in the previous experiment for both irradiated and non-irradiated groups (Table I). Although the measured air temperatures were the same, the difference may reflect a discrepancy in temperature within the culture tubes, that in the earlier experiment being higher due to closer proximity to the light source.

To determine if alterations in post-radiation survival affect the composition of subsequent generations, the progenies from both these groups were cultured and examined. Table III shows the mean-square expectation for the within-plot variances of irradiated and non-irradiated populations. The estimate of the component, σ_G^2 , represents the transmissible variation induced by radiation, which is assumed to be genetic; however, it could also include physiological damage if, for example, some damaged cytoplasmic component were transmitted through the female parent. On the basis of a highly significant F value, it was concluded that the population with the greater survival in the first generation also showed greater genetic variability among its progeny. From these same individuals, estimates of fertility, based on percentage pollen stainability, were obtained (Table IV), which indicate significantly more sterility in the progeny of the near-optimum temperature population. There is also evidence for non-homogeneity of variance from the difference in their respective errors, 5.7 vs. 3.2.

Discussion and conclusions. If one correlates these results from progeny testing with the original survival of irradiated seed, greater radiation damage, as reflected by variation in flowering time and fertility, is found to be associated with greater survival. The fact that less damage is associated with lowered survival indicates that the missing fraction of the population had received greater radiation damage than the survivors. This pattern could arise if the more damaged individuals had been preferentially eliminated from the population during the early stages of growth.

The role of temperature in the expression of radiation damage should be viewed through its influence on cellular metabolism. It has been emphasized previously that cellular activity is the factor which reveals the lesion caused by irradiation³. The radiation response may not be

Table I. Survival to flowering in M_1 generation

Treatment (°C)	Irradiated			Control		
	Survivors	Non-survivors	Survival (%)	Survivors	Non-survivors	Survival (%)
5 + 20	76	24	76.0	86	14	86.0
5 + 27	11	89	11.0	63	37	63.0

$$\chi^2 = 21.1; P < 0.01.$$

Table II. Survival to flowering in M_1 generation

Temperature treatment (°C)	Irradiated			Control		
	Survivors	Non-survivors	Survival (%)	Survivors	Non-survivors	Survival (%)
5 + 27	130	150	46.4	187	13	93.4
5 + 20 + 27	127	37	77.4	144	6	96.0

Analysis of variance (angular transformation)

Source of variation	df	Mean square	F
Radiation	1	112,592.8	137.1*
Temperature	1	22,749.4	27.7*
Interaction	1	11,016.0	13.4*
Error	∞	821	

* $P < 0.01$.

Table III. Estimates of variance components for flowering time in M_2 generation

Temperature treatment (°C)	Within-plot mean squares		Difference σ_G^2	F
	Irradiated $\bar{\sigma}_E^2 + \sigma_G^2$	Control $\bar{\sigma}_E^2$		
5 + 27	7.44	3.56	3.88	2.17*
5 + 20 + 27	13.37	4.94	8.43	

* $P < 0.02$.

Table IV. Pollen sterility (%) in M_2 generation

Temperature treatment (°C)	Irradiated	Control
5 + 27	12.6 $\pm$ 3.2	<1
5 + 20 + 27	31.0 $\pm$ 5.7	<1

³ L. H. GRAY, Prog. Biophys. biophys. Chem. 2, 240 (1951).

equivalent if temperatures which are sub-optimal, but sufficient to allow germination and growth, are compared⁴⁻⁸. KONZAK et al.⁸ suggested that temperature influences radiation damage through alteration of the reaction rate for induced free-radical species. However, this mechanism should not apply in the present case since post-irradiation treatment would be expected to eliminate free radicals.

The joint effects of ionizing radiation and temperature in determining biological damage can be viewed as an example of the ecological concept of limiting factors. It is a well-known principle that when conditions are not optimum for a species with respect to one ecological factor, the limits of tolerance may be reduced with respect to other ecological factors. There is evidence that some conditions in the natural environment may operate as stress factors in combination with radiation^{10,11}. In addition, experimentation with *A. thaliana* over a range of temperatures has shown that temperature tolerance of a number of races is limited because of genes whose products become inactive at high temperatures^{12,13}.

Zusammenfassung. Nach früher akuter Berührung mit γ -Bestrahlung kann im allgemeinen der Erholungsprozess bei *Arabidopsis thaliana* (L.) Heynh. Samen durch relativ kleine und kurze Umgebungsveränderungen (4 °C während 4 Tagen) stark modifiziert werden. Diese werden nach ver-

meintlicher Deaktivierung radiochemischer Produkte angewendet und beeinflussen nicht nur die behandelte, sondern auch die folgende Generation.

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⁴ O. E. V. GELIN, *Agri Hort. Genet.* 11, 66 (1953).

⁵ L. EHRENBERG, *Hereditas* 41, 123 (1955).

⁶ L. EHRENBERG, *Bot. Notiser* 108, 184 (1955).

⁷ L. EHRENBERG and U. LUNDQVIST, *Hereditas* 43, 390 (1957).

⁸ C. F. KONZAK, H. J. CURTIS, N. DELIHAS and R. A. NILAN, *Can. J. Genet. Cytol.* 2, 129 (1960).

⁹ A. T. NATARAJAN and K. R. NARAYANAN, in *Repair from Genetic Radiation Damage and Differential Radiosensitivity in Germ Cells* (Ed. F. H. SOBELS; Pergamon, New York 1963), p. 413.

¹⁰ W. S. OSBURN JR., *Science* 134, 342 (1961).

¹¹ W. S. OSBURN JR., in *Radioecology* (Eds. V. SCHULTZ and A. W. KLEMENT JR.; Reinhold, New York 1963), p. 319.

¹² J. LANGRIDGE and B. GRIFFING, *Aust. J. biol. Sci.* 12, 177 (1959).

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Über die Bildung rudimentärer Zilien bei Melanozyten

Nach der zur Zeit vorherrschenden Lehrmeinung stammen alle Pigmentzellen von der Neuralleiste ab und damit auch die melaninbildenden Melanophoren bzw. die Melanozyten¹. Wie Untersuchungen an Embryonen und Feten des Menschen gezeigt haben, lassen sich bereits von der 10. Woche an im subkutanen Bindegewebe (Subkutis) histochemisch Melanoblasten als Vorstufen der Melanozyten nachweisen, und am Ende des 4. Monats sind schon alle Bereiche der Epidermis mit Melanozyten besiedelt. Hier liegen sie an der Grenze der Epidermis zum Corium hin innerhalb der Basalzellschicht, wo sie als «Dendritenzellen» mit langen Zytoplasmafortsätzen in die oberen Epidermisschichten reichen. Histologisch erscheinen sie nach HE-Färbung als sogenannte «Klarzellen» bzw. «cellules claires»² mit wenig tingiertem Zytoplasma.

Gutartige Wucherungen der Melanozyten führen zur Bildung von Zellnestern oder Zellsträngen, die dann je nach ihrer Lage als Junktionsnaevus, Compound-Naevus, corialer Naevus usw. bezeichnet werden³. Gelegentlich können die Melanozyten bzw. die Naevuszellnaevi entarten, so dass es dann zur Bildung von malignen Melanomen kommt.

Von manchen Autoren werden die Naevuszellen nicht von Melanozyten bzw. von Melanoblasten abgeleitet, sondern von den ebenfalls aus der Neuralleiste stammenden Neuroblasten⁴. Nach einer weiteren Hypothese soll das Muttergewebe der Naevi weder epithelialer noch SCHWANNscher (REMAKScher) Herkunft sein, sondern von einem besonderen (mesodermalen) Hüllgewebe der peripheren Nerven, den periendoneuralen Häutchenzellen, abstammen. Schliesslich ist auf Grund von embryologischen⁵⁻⁷, histochemischen und elektronenmikroskopischen Untersuchungen eine gemeinsame Herkunft von Melanozyten und Mastzellen vermutet worden⁸.

In letzter Zeit ist die Ultrastruktur der Melanozyten bzw. der malignen Melanomzellen eingehend untersucht

worden. Von den übrigen Zellen der Epidermis unterscheiden sie sich in erster Linie durch das Fehlen von Desmosomen und Tonofilamenten. Wie in den meisten anderen Somazellen, so finden sich bei den Melanozyten Zentriolen, und zwar gewöhnlich in Form eines Diplomsoms (Figur 1). Diese Zellstrukturen sind elektronenoptisch erstmals von BERNHARD und HARVEN⁹ sowie von HARVEN und BERNHARD¹⁰ dargestellt worden. Sie bilden ein Röhrenchensystem mit einer Länge von etwa 0,3–0,5 μ m und einem Durchmesser von etwa 0,15 μ m. Die einzelnen Röhrrchen bestehen aus 2 oder 3 Untereinheiten (Subfilamenten). Bei ziliotragenden Zellen ist dieses Zellorganell für die Bildung der Zilien verantwortlich, z. B. beim Trachealepithel^{11,12}. Gewöhnlich erfolgt dies nur bei epithelialen Zellen.

Bei Untersuchungen an benignen und malignen Pigmentgeschwülsten des Menschen konnten wir bei einzelnen Zellen von zwei Naevuszellnaevi die Bildung von rudimentären Zilien beobachten (Figur 2). Dabei erreichen

¹ D. STARCK, in *Handbuch der Haut- und Geschlechtskrankheiten* (Ed. J. JADASSOHN; Springer Berlin, Göttingen, Heidelberg, New York 1964), Erg.-Werk Bd. 1/2, p. 139.

² P. MASSON, *Anals Anat. path. med. chir.* 3, 417 und 657 (1926).

³ A. SCHAUER und A. VOGEL, *Medische Welt, Stuttg.* 3, 149 (1967).

⁴ H. STORCK, *Hautarzt* 21, 187 (1970).

⁵ F. FEYRTER, *Virchows Arch. path. Anat. Physiol.* 301, 417 (1938).

⁶ F. FEYRTER, *Virchows Arch. path. Anat. Physiol.* 346, 117 (1969).

⁷ F. FEYRTER und J. Böck, *Albrecht v. Graefes Arch. Ophthal.* 178, 67 (1969).

⁸ M. OKUN, *J. invest. Derm.* 44, 285 (1965).

⁹ W. BERNHARD und E. DE HARVEN, *C. r. Acad. Sci., Paris* 242, 288 (1956).

¹⁰ E. DE HARVEN und W. BERNHARD, *Z. Zellforsch.* 45, 378 (1956).

¹¹ S. SOROKIN, *J. Cell Biol.* 15, 363 (1962).

¹² V. KALNINS und K. PORTER, *Z. Zellforsch.* 1, 100 (1969).