pacas, he did not include Perú in the known range of *A. taczanowskii*, having assumed that the name *thomasi* referred to a form of the Peruvian lowland paca. Later, THOMAS[10] reported 3 specimens from Perú as *Coelogenys paca*: 2 skulls of immature animals from Acobamba, 8000 ft., Depto. Pasco[11], and the skull of a male 'secured from natives' from Chihuangala, 4000 ft., Depto. Huánuco. The 2 from Acobamba are almost certainly *A. taczanowskii*; however, the specimen from Chihuangala is probably *A. paca*. Unfortunately, the Cordillera Carpish *A. taczanowskii*, whose chromosomes are reported herein, is represented by a skin only. Three skulls of this species were secured from a hunter from the same locality (LSUMZ 14437, 14438, and 14439). The only other reference to specimens from Perú is SANBORN's[8] report of purchasing a piece of skin of the mountain paca in Sandia, Depto. Puno[12].

Resumen. Se describe los cariotipos de *Anotomys leander* y *Agouti taczanowskii* del Perú. Las cromosomas de *A. leander* son 92, el número más alto conocido de un mamífero. Informes anteriores de *A. taczanowskii* en el Perú son discutido.

A. L. GARDNER

Museum of Zoology, Louisiana State University, Baton Rouge (Louisiana 70803, USA), 15 March 1971.

[10] O. THOMAS, Ann. Mag. nat. Hist., ser. 9, *20*, 594 (1927).

[11] Reported as Depto. Junín.

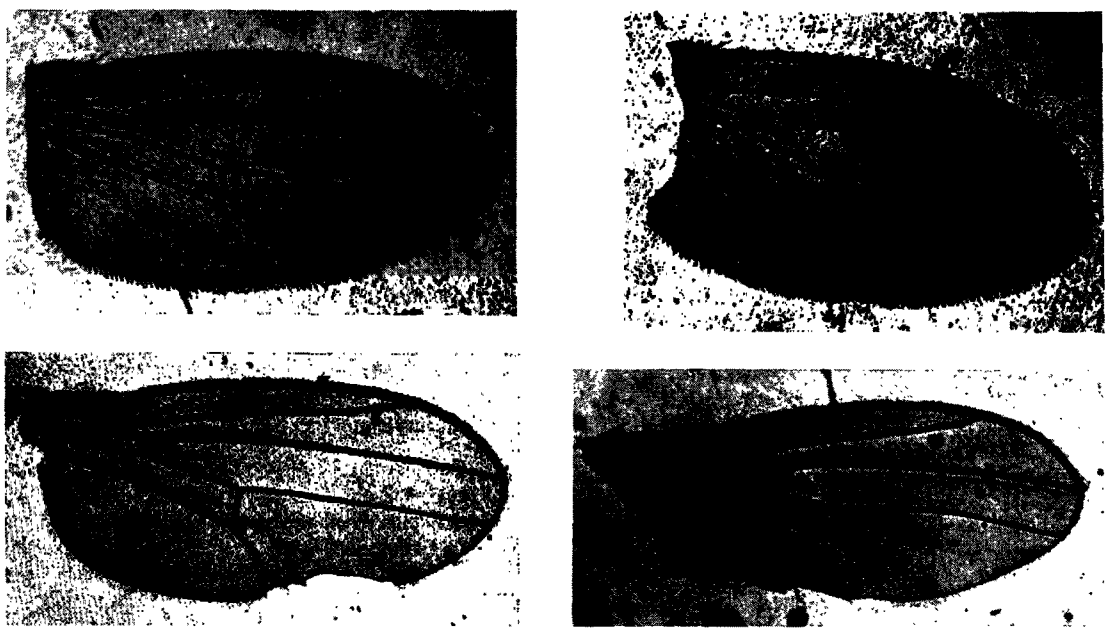
[12] The 1968 LSU Peruvian expedition was supported by JOHN S. MCILHENNY, Eugene du Pont III, and a Louisiana State University Graduate. Research Council grant to GEORGE H. LOWERY JR. I wish to thank JOHN P. O'NEILL of the Museum of Zoology, Louisiana State University, for assistance in the field, JAMES L. PATTON of the Museum of Vertebrate Zoology, University of California, Berkeley, for aid in preparing the karyotype of *Anotomys leander*, and GUY G. MUSSER of the Department of Mammalogy, American Museum of Natural History, for identifying the *Anotomys*. I also wish to thank MARY ANN GARDNER for preparation of the slides in the field.

On the Teratogenic Effect of Thymidine and its Suppression by Deoxycytidine in *Drosophila melanogaster*

In some of the earlier experiments[1,2] it was found that thymidine, when added to the food, has teratogenic and mutagenic effect in *D. melanogaster*. The most conspicuous of the teratogenic changes observed were the cuts on the wing margins (the number, size and potition of these varying, affecting one or both of the wings) and the abnormalities of the wing-veins (Figure). The other abnormalities were a) an increase in the number of scutellar bristles, b) irregularity of the abdominal bands and c) the malformation of the legs. The abnormalities were found to appear independently or in conjunction with others.

It has been reported by several authors that thymidine, at high concentrations inhibits the cell-growth in tissue-culture experiments and that this effect can be compensated by adding cytidine to the tissue medium[3–5]. It was thought that there might be a similar effect in *D. melanogaster* and the thymidine-action might be compensated in the same way as in tissue cultures.

The experiments described here were performed under exactly similar conditions as reported earlier[1]. In all of these experiments, grade A thymidine and deoxycytidine obtained from Calbiochem (Switzerland) were employed. The concentration of thymidine and deoxycytidine respectively were kept at 2%. To study the compensatory effect of deoxycydidine, equal amounts of thymidine and deoxycytidine were weighed separately, mixed together



Some of the typical abnormalities of the wings and the veins in *D. melanogaster*

and added to the medium to give a concentration of 2% thymidine + 2% deoxycytidine. Ore-R cultures of *D. melanogaster* were started on different media and the flies which had developed on these media checked for any visible abnormalities. F_1 males were tested for the presence of sex-linked recessive lethals by employing the M-5 technique. Control cultures were always started simultaneously.

Whereas in all repeats on thymidine medium, a very high (but varying) percentage of hatching adults showed the abnormalities described above, the addition of an extra 2% of deoxycytidine to the thamidine-containing medium completely suppressed the abnormalities of the wings and the veins. The other abnormalities, such as extra bristles and irregular abdominal sementation, were not significantly affected. Deoxycytidine at 2% concentration had no effect. It was neither toxic nor teratogenic.

For the study of the mutagnic effect the M-5 technique was employed. The results obtained are shown in the Table.

It is evident that deoxycytidine at 2% concentration is not mutagenic ($t = 1.27$, $p > 0.20$) and that 2% of deoxycytidine added to the thymidine (2%) medium does not counterbalance the mutagenic effect ($t = 0.59$, p 0.50) of thymidine. Thymidine at 2% concentration is, as reported earlier[1], mutagenic ($t = 3.78$, $p < 0.001$).

Induction of sex-linked recessive lethals

	Control	2% Thymidine	2% Deoxycytidine	2% Thymidine + 2% Deoxycytidine
No. of chromosomes tested	2465	1030	2691	1038
No. of lethals	8	21	15	17
% – lethals	0.32	2.04	0.55	1.64

These results show that deoxycytidine does not compensate the mutagenic effect of thymidine; however, in agreement with the tissue-culture experiments, it does counter-balance the teratogenic effect concerning the wings and the veins. In the case of tissue-culture experiments, the compensatory effect of deoxycytidine is explained by assuming that the presence of excessive exogenous thymidine leads to the formation of a high level of TTP (thymidine tri-phosphate) which in turn produces a deficiency in deCTP (deoxycytidinephosphate, a component necessary for the DNA synthesis) in the cells, and hence the cell-growth is inhibited[4,6]. The cell-growth can be maintained if deoxycytidine is added to the tissue medium. The suppression of thymidine-teratogenesis in *D. melanogaster* by deoxycytidine may also be explained on the same basis. This is an interesting observation. It would mean that the nutritional imbalance can be a cause of congenital abnormalities. This is so in the case of *D. melanogaster*. Whether it is also the case with other organisms remains to be seen.

Zusammenfassung. Thymidin-Zusatz im Nährboden erweist sich bei *Drosophila melanogaster* als mutagen und teratogen. Bei Zusatz von Deoxycytidin wird der teratogene Effekt zumindest teilweise aufgehoben. Auf die mutagene Wirkung des Thymidins hat Deoxycytidin jedoch keinen Einfluss.

O. PARKASH[7]

Institut für allgemeine Biologie der Med. Fakultät, A-1090 Wien IX (Austria), 22 March 1971.

[1] O. PARKASH, Experientia *23*, 859 (1967).
[2] O. PARKASH, Experientia *24*, 726 (1968).
[3] H. J. BARR, J. Cell. comp. Physiol. *61*, 119 (1963).
[4] J. E. CLEAVER, *Thymdine Metabolism and Cell Kinetics* (North Holland Publishing Co., Amsterdam 1967).
[5] J. E. CLEAVER and R. N. HOLFORD, Biochim. biophys. Acta *103*, 654 (1965).
[6] R. B. PAINTER, R. M. DREW and R. E. RASMUSSEN, Radiat. Res. *21*, 355 (1964).
[7] The author is thankful to D. SPERLICH for guidance and friendly discussions.

Das Vorkommen von «Myrosinase» als Hinweis auf die systematische Stellung der Batidaceae

Die systematische Stellung der bisher nur in 2 Arten bekanntgewordenen Familie der Batidaceae – Halophyten tropischer und subtropischer Küsten der Neuen Welt und einiger Pazifikinseln – ist bis heute umstritten. Schon seit langem wurde die Möglichkeit verwandtschaftlicher Beziehungen zu den Centrospermae, insbesondere zu den Chenopodiaceae, erwogen. Die für diesen Verwandtschaftskreis typischen Betacyane, deren Vorkommen bei den Batidaceae als eindeutiger Hinweis hätte gelten können (HEGNAUER[1]), konnten bei dieser Familie jedoch nicht nachgewiesen werden[2]. Auf Grund gewisser Ähnlichkeiten im Blütenbau, der endospermlosen Samen und des Vorkommens von Rudimentärstipeln wurde in neuerer Zeit auch die Möglichkeit einer Verwandtschaft mit den Capparidales erörtert[3,4]. Nach unserer Auffassung könnten dafür auch Übereinstimmungen im Aufbau der Infloreszenzen und andere, vor allem auch anatomische Details sprechen, die bei einer erneuten morphologisch-systematischen Bearbeitung der Familie gefunden wurden (B. SCHMIDT, Manuskript abgeschlossen). Im Zusammenhang mit diesen Untersuchungen lag es für uns nahe, auch nach phytochemischen Kriterien für die verwandtschaftliche Zuordnung der Batidaceae zu suchen.

Die Capparidales-Familien (Capparidaceae, Cruciferae, Resedaceae, Tovariaceae; Moringaceae (?)) sowie die Tropaeolaceae und Caricace sind durch das Vorkommen von Glucosinolaten sowie des glucosinolatspaltenden Enzyms «Myrosinase» ausgezeichnet. Es erschien uns daher wünschenswert, entsprechende Untersuchungen an Batidaceae vorzunehmen, wenngleich hier typische Myrosinzellen nicht nachzuweisen waren. Eine chromatogra-

[1] R. HEGNAUER, Chemotaxonomie III (Birkhäuser-Verlag, Basel/Stuttgart 1964), p. 236.
[2] T. J. MABRY und B. L. TURNER, Taxon *13*, 197 (1964).
[3] A. A. PULLE, *Compendium van de Terminologie*, Nomenclatur en Systematiek der Zaadplanten (N. V. A. Osthock, Utrecht 1952), p. 318.
[4] TH. ECKARDT, Ber. dt. bot. Ges. *72*, 411 (1959).