Table 1. Estimated tension values at the mesophyll evaporating surface, and mean rates of photosynthesis during the measuring period.

Plant No.	Tension (bars)	Photosynthesis (mg CO_2 min^{-1} g^{-1} leaf dry wt.)
1	320	3.77 ± 0.07*
2	180	15.50 ± .28
3	240	5.26 ± .17

* ± Standard error.

spheric relative humidity were calculated. The humidity was expressed as negative potential ($-d$) in meters of water (*8*), which has the advantage of being in absolute units as well as being corrected for different air temperatures. Leaf temperatures were either equal to or lower than air temperatures. Where leaf temperature was lower, actual transpiration values were corrected to isothermal conditions as follows. The relationship between T and vapor pressure gradient is linear—that is, T/vapor-pressure-gradient = a constant, K. A mean value for this constant for each plant was calculated from data obtained on occasions when leaf and air temperatures were equal. Where leaf and air temperatures differed, the change in saturated vapor pressure caused by this temperature difference was calculated, neglecting, for this purpose only, the inaccuracy introduced by assuming the evaporating surface to be saturated. The difference in gradient was multiplied by the mean constant, K, and added to the measured transpiration value. This correction greatly improved the correlations.

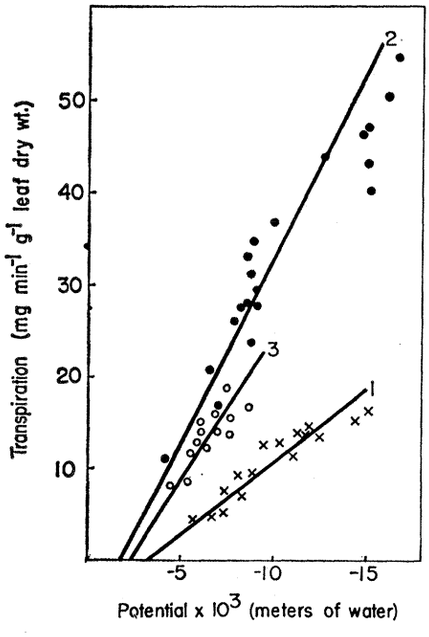

Fig. 1. Estimation of mesophyll saturation deficit in *Reaumuria hirtella* by regression analysis of transpiration rate and atmospheric water potential. The intercept on the base line measures the deficit. The correlation coefficients (r) are −0.924 for plant 1; −0.772 for plant 2; and −0.645 for plant 3.

A major source of error in these estimations arose from inaccuracies in measurements of leaf temperature; such inaccuracies could lead to relatively large errors in the calculated vapor pressures. Since inaccuracies in measurements of mean leaf temperature amounted to ±0.3°C, the resulting variability of the estimated mean tension values for plants 1, 2, and 3, respectively (Table 1), was ±40, ±25, and ±30 bars.

Highest transpiration rates were associated with the lowest mesophyll saturation deficit (plant 2, Fig. 1). The slope of the regression lines is a measure of stomatal resistance. Again, this was lowest in plant 2, which also had a much higher rate of photosynthesis (Table 1).

The measured tension values are high (Table 1). Shimshi (*4*) has published values up to 90 bars for nonwilted maize. However, *Reaumuria* is a halophyte growing under desert conditions, with an average of 30 percent of soluble salts in the cell sap (*9*). The effect of this high salt concentration may not be confined to mere molar reduction of the saturated vapor pressure. As shown by Boon-Long (*2*), solutes may concentrate at the evaporating surface, causing a reduction in vapor pressure greater than that calculated from the concentration of the vacuolar sap. High salt concentrations may also reduce cell permeability (*2*). The physical effect of the retreat of water columns into the micro-capillaries of the cell wall (*10*) would contribute to a further reduction of vapor pressure. Though these effects combine to reduce actual transpiration, they nevertheless do not affect the estimated saturation deficits, because they are functions of the transpiration flux and would disappear at zero transpiration. The actual and theoretical lines should therefore converge to intercept the abscissa at the same point.

P. C. WHITEMAN
D. KOLLER

Department of Botany,
The Hebrew University of Jerusalem,
Israel

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Genetic Disparity and Cancer Induction by Normal Tissue Implants in Amphibia

Abstract. *Fifty percent of the implants of normal adult* Triturus cristatus *kidney made into the forelimbs of immature but postmetamorphic* Xenopus laevis *hosts initiated the formation of lymphosarcoma at the site of implantation. Donor-host genetic disparity as it relates to the intensity of the reaction, when homografts, heterografts, and xenografts are compared, appears to be one of several factors which play a role in the postembryonic induction of both lymphosarcomas in* Xenopus laevis *and accessory limb structures in* Triturus viridescens.

The African clawed toad, *Xenopus laevis*, is subject to the spontaneous development of highly lethal metastatic lymphosarcomas (*1*). The cancer forms primarily in the liver, spleen, and kidney, but will metastasize elsewhere into visceral and perivisceral regions. The organs affected are progressively destroyed. The cells of the cancer are largely lymphoblastic in appearance and frequently one finds lymphoid cells which have peripherally arranged chromatin and prominent nucleoli (*2*). The same type of cancer can be induced to form in other *Xenopus* species and subspecies (*3*), in the European newt, *Triturus cristatus* (*4*), and in other Anuran species, for example *Rana pipiens, Rana esculenta,* and *Bufo bufo bufo* (*5*), after the initial

breakdown of the transplanted cancer material within the host. The new cancers arise through host cell transformation. This has been determined with assurance for inter-order transfers where extreme differences in cell size allow distinctions to be made between donor or host origin (*6*). Recently, it was demonstrated that this cancer can be transferred to *Xenopus laevis* by means of filtrates of lymphosarcoma. Filtrates of normal tissues gave negative results. These filtrate experiments coupled with the positive results obtained by using frozen stored lymphosarcoma suggest a viral etiology for this cancer. Frozen normal tissue implants failed to induce cancer (*7*).

We demonstrated previously that homografts of normal adult kidney of the clawed toad, *Xenopus laevis* (*8*), and heterografts of normal and neoplastic kidney from the frog, *Rana pipiens* (*9*), made into *Xenopus* hosts are capable of initiating the formation of lymphosarcoma at the site of implantation and in the visceral organs of the host, while the implant is being destroyed by the graft rejection mechanism. These implants were placed into either the dorsal lymph sac or the forelimb of postmetamorphic but immature stage 66 (*10*) *Xenopus laevis* hosts.

In this report we describe experiments with xenografts whereby portions of normal adult kidney taken from the European newt *Triturus cristatus* were implanted into *Xenopus* hosts. The term xenograft is being used to characterize heterografts of greater genetic disparity than genera. Previous implantation work with amphibia (*11*) has indicated more dramatic differences between xenografts and heterografts than between homografts (within the same species) and heterografts (between species and genera but within the same order) with regard to their ability to induce the development of accessory structures in salamander limbs.

Ten *Xenopus* hosts received an implant of approximately 2 mm^3 in each forelimb in accordance with a technique described in detail previously (*11*). The implanted limbs were observed macroscopically over a 62-day period, after which time the hosts were killed and their limbs and visceral organs were removed and prepared for histological analysis. The hosts were anesthetized in 2.5 percent urethan when the implants were made, and were then maintained at 23°C and fed with *Tubifex tubifex* worms.

A strong response against the xenografts was apparent 7 days after implantation. In over one-half of the hosts, the skin became ulcerated over the foreign tissue, and the kidney material became reduced in mass. The implant continued to decrease in size throughout the 62-day period. By 62 days, only six small mounds could be observed macroscopically in the 20 limb sites.

Histological examination of all the implanted limbs revealed that only one limb bore any extraordinary tissue mass without accompanying lymphosarcoma development. This one limb had a mass of tissue which had become encapsulated, and the foreign kidney structure had been replaced by host-formed fibrous scar. The difference in cell size between the donor tissue (large cells) and host tissue (small cells) made it possible for the origins of the tissues to be determined with confidence.

In three of the host limbs there was no evidence of either kidney tissue or lymphosarcoma, but each of the remaining 16 host limbs contained lymphosarcoma with at least a small amount of fibrous scar. In some of these limbs there was evidence of epidermal activity related to implant removal. In amphibia the epidermis is capable of engulfing necrotic implant material and ejecting it from the limb site (*4, 11*). In two limbs the lymphosarcoma was invasive into adjacent musculature and skin, while in the others the cancer was localized as lymphoid masses in the lymph sac and dermis of the skin in the implant region. None of the ten hosts bore lymphosarcomas of the visceral organs, which indicates that the limb tumors were related to the implantation of *Triturus* kidney and were not metastases of spontaneous visceral lymphosarcomas. The development of lymphosarcoma in response to a cytolysing foreign implant can be distinguished readily from the normal rejection response on the basis of two criteria: (i) the prevalence of lymphoblastic cell foci, and (ii) the invasiveness of the lymphoid cell population.

In comparing the results obtained with homografts, heterografts, and xenografts of normal kidney placed into the limbs of *Xenopus*, one finds an increase in the proportion of definitive tumors which form as the genetic disparity between the donor and host increases. Definitive lymphosarcoma developed in only 5 out of 30 host limbs when kidney homografts were used, whereas 9 limbs out of 29 developed lymphosarcoma when kidney heterografts were used. In the experiment reported here, tumors developed in 16 out of 20 sites of xenograft implantation. These xenograft-induced cancers were also more extensive than those induced by homo- or heterografts. We suggest that this correlation may be of some interest if one recalls the increase in inductive power of amphibian kidney implants with increasing genetic disparity in previously reported experiments in which the formation of accessory structures was induced in urodele limbs (*11, 12*). This analogy may become more meaningful when the data are considered in conjunction with results obtained from (i) experiments in which kidney homografts in *Xenopus* forelimbs induced both lymphosarcoma and the formation of accessory cartilage nodules, and (ii) experiments in which the carcinogen methylcholanthrene, implanted as mounds of crystals in *Xenopus* forelimbs (*2*), also induced both lymphosarcoma and the formation of accessory limb structures. We suggest that implants of both foreign tissue not containing lymphosarcoma and carcinogens initiate cancer development in *Xenopus* by activating latent virus in host lymphoid cells, but will discuss this hypothesis in detail elsewhere (*9*).

LAURENS N. RUBEN
MICHAEL BALLS
Station de Zoologie Expérimentale,
L'Université de Genève,
Chêne-Bougeries,
Geneva, Switzerland

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