

of the scatter of individual members of a group and group means.

In the statistical analysis eight groups were defined with about thirty individuals/group (Table 2). Only one of 233 mandibles was misclassified in this analysis. Seventy-nine "unknown" mandibles were then tested: fifty-three were from other strains in Table 1, and twenty-six from strain LACG, one of the eight groups. The "unknowns" were recorded as belonging to a group if there was more than one chance in 100 of group membership. Two mandibles from strain HR were incorrectly classified as belonging to strain A2G, one mandible from C57BR was incorrectly classified as LACG, and one mandible from strain LACG was incorrectly classified as belonging to strain NZB. Thus, of the 312 mandibles studied only five were incorrectly classified. Using inbred, F1 hybrid, and non-inbred strains, 98% of the mandibles were correctly classified. Moreover, the technique could have been made considerably more precise if measurements on other bones had been included.

It may, therefore, be technically feasible to establish an international reference centre for strain identification, once samples of most of the known inbred strains have been measured. The technique also appears to be sufficiently precise to detect subline differences. Thus, further analysis showed that strains B10.A and B10.BR were genetically distinct, though closely related. The technique might therefore be used to study the development[†] of substrains, and could possibly be used to re-define the conditions under which a new substrain is recognized. At present, new sublines are established simply by transferring stock from one laboratory to another¹ even though genetically there should be no important differences developing from a number of generations. The method can also be applied to closed random-bred stock, and probably to other species.

MICHAEL FESTING

MRC Laboratory Animals Centre,
Woodmansterne Road, Carshalton, Surrey

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- ¹ Staats, J., *Standardized Nomenclature for Inbred Strains of Mice; Fourth Listing*, Cancer Research, 28, 391 (1968).
- ² *Biology of the Laboratory Mouse* (edit. by Green, E. L.) (McGraw-Hill, London, 1966).
- ³ Moutier, R., *Biochemical Differences as a Means of Genetic Control for Inbred Strains of Laboratory Animals*, Proc. IV ICLA Symposium, Washington, DC (1971).
- ⁴ Grüneberg, H., *The Genetics of the Mouse* (Martinus Nijhoff, The Hague, 1952).
- ⁵ Stein, K. F., *J. Genet.*, 55, 313 (1957).
- ⁶ Bailey, D. W., *J. Hered.*, 50, 26 (1959).
- ⁷ Blackith, R. E., and Reymont, R. A., *Multivariate Morphometrics* (Academic Press, London and New York, 1971).
- ⁸ Searle, A. G., *J. Genet.*, 52, 68 (1954).
- ⁹ Parrott, R. F., and Festing, M., *Standardised Laboratory Animals*, LAC Manual Series No. 2 (MRC Laboratory Animals Centre, Carshalton, Surrey, 1971).
- ¹⁰ Cooley, W. W., and Lohnes, P. R., *Multivariate Data Analysis* (John Wiley and Sons, Inc., New York and London, 1971).

Polar Auxin Transport in Leaves of Monocotyledons

ALMOST nothing is known about the establishment of cellular polarity underlying the polar auxin transport system of higher plants. Osborne¹ has suggested that the apical ends of cells derived from an apical meristem by sequential divisions are younger than the basal ends: their polarity and the basipetal transport of auxin are due to this age difference. Sachs² in his work on regenerating vascular strands has found that gradients of auxin may be responsible for establishing the cellular polarity and the subsequent transport of auxin in the direction of the initial gradient. Shoot tips and expanding dicot leaves contain relatively high levels of auxin. The basipetal polarity of auxin transport in petioles and stems is therefore associated with basipetal auxin gradients. In grass coleoptiles the greatest

amounts of auxin are found at the tip, where basipetal auxin transport is also associated with basipetal auxin gradients.

In monocot leaves which grow by a basal intercalary meristem, the pattern of cell division and of auxin distribution is more or less the reverse of that found in shoot tips. Sequential divisions of the basal meristem presumably make younger the basal ends of cells; and in growing monocot leaves the greatest amounts of auxin are found at the base^{3,4}. The polarity of auxin transport in monocot leaves is therefore of considerable interest.

Hertel and Leopold⁵ reported that in the primary leaf of *Zea mays*, auxin transport was basipetal. No other references to auxin transport in monocot leaves are available and I therefore tested the leaves of a number of species. In every case auxin transport was basipetal (Table 1).

Table 1 Auxin Transport by Sections of Young Leaves

Plant	C.p.m. in receiver blocks	
	Acropetal transport	Basipetal transport
Amaryllidaceae		
<i>Galanthus nivalis</i> L (8)	1	2,098
<i>Narcissus</i> sp (6)	0	4,616
Cyperaceae		
<i>Carex pendula</i> L (4)	6	553
Gramineae		
<i>Hordeum vulgare</i> L (6)	2	117
Iridaceae		
<i>Crocus purpureus</i> Weston (6)	0	142
<i>Iris</i> sp (4)	2	999
Liliaceae		
<i>Chlorophytum comosum</i> (Thunb.) Jacques (8)	2	51
<i>Cordylone australis</i> (Forst.) Hook. f. (4)	0	106
Orchidaceae		
<i>Cymbidium lowianum</i> Rchb. f. (4)	0	784
Palmae		
<i>Erythea armata</i> (Wats.) Wats. (6, leaflets)	1	52

Leaves were collected from plants growing outdoors and from tropical species in glasshouses. Sections (7 mm) were placed horizontally, supported by a strip of filter paper coated with petroleum jelly, on glass slides, and were supplied with agar (1% w/v) donor and receiver blocks. Donor blocks contained [¹⁴C] indol-3-yl acetic acid (52 mCi/mM, Amersham) at a concentration of 3.0 μM. During the transport period (3.5 h) the sections were kept in the dark in Petri dishes lined with moistened filter paper. Receiver blocks were then placed in scintillation vials with liquid scintillator⁶ (4 ml.) and counted for at least 10 min on a Packard TriCarb scintillation counter. Background counts (25–30 c.p.m.) were subtracted from the results. The numbers of leaf sections used are shown in parentheses.

Table 2 Auxin Transport in Primary Leaves

Region of leaf	C.p.m. above background			
	11-day-old plants		19-day-old plants	
	Acropetal transport	Basipetal transport	Acropetal transport	Basipetal transport
Tip	1	28	1	1
Middle	1	670	0	49
Base	0	525	0	106
Leaf-leaf sheath junction	1	495	1	107
Leaf sheath	2	346	0	117

7 mm sections were taken from different regions of primary leaves of *Avena sativa* plants grown in daylight at 22° C. Procedure as in Table 1. 10 sections were used for each sample. Transport time 3.5 h.

In leaves of young plants of *Avena sativa*, basipetal auxin transport took place across the meristematic region at the base of the leaf and also in the leaf sheath (Table 2), which grows by a basal meristem⁶. Plants germinated and grown in darkness yielded similar results. Less auxin transport was found near the leaf tip than in the younger, more basal parts of the leaf and younger leaves had a greater ability to transport auxin than older leaves (Table 2). A decline in the ability of cells to

transport auxin as they grow older has been observed in a number of other species and tissues⁷⁻¹⁰.

The hypothesis that polarity is determined by the difference in age between the apical and basal ends of cells becomes more complicated if it is to explain how both apical and basal meristems give rise to cells with a basipetal polarity. Its greatest weakness is in explaining the basipetal polarity of cambial derivatives¹¹ which arise not by transverse but by longitudinal divisions. The possibility that gradients of auxin determine the polarity of cells also seems at first sight unlikely to provide an explanation for the development of basipetal auxin transport in monocot leaves; but too little information is available about the anatomy of the basal meristems and the detailed pattern of auxin distribution in and around the meristematic region for any firm conclusions to be made.

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A. R. SHELDRAKE

Department of Biochemistry,
University of Cambridge

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- ¹ Osborne, D. J., in *Transport of Plant Hormones* (edit. by Vardar, Y.), 97 (North Holland, Amsterdam, 1968).
- ² Sachs, T., *Ann. Bot.*, **33**, 263 (1969).
- ³ van Overbeek, J., *Bot. Gaz.*, **100**, 133 (1938).
- ⁴ van Overbeek, J., *Amer. J. Bot.*, **33**, 263 (1947).
- ⁵ Hertel, R., and Leopold, A. C., *Planta (Berl.)*, **59**, 535 (1963).
- ⁶ Bray, G. A., *Analyt. Biochem.*, **1**, 279 (1960).
- ⁷ Esau, K., *Plant Anatomy* (Wiley, New York, 1957).
- ⁸ Jacobs, W. P., *Amer. J. Bot.*, **37**, 248 (1950).
- ⁹ Leopold, A. C., and Guernsey, F. S., *Bot. Gaz.*, **115**, 147 (1953).
- ¹⁰ Leopold, A. C., and Lam, S. L., *Physiol. Pl.*, **15**, 631 (1962).
- ¹¹ Sheldrake, A. R., *J. Exp. Bot.* (in the press).

EBV Antibody in Sera of Non-human Primates

SEVERAL investigators have assayed sera of non-human primates for antibody to the Epstein-Barr virus (EBV). Gerber and Birch¹ reported the widespread distribution of complement-fixing antibody derived from the chimpanzee, baboon, rhesus and African green monkey. In a subsequent study, Gerber and Rosenblum² reported that many rhesus monkeys bled within 4 days of capture were also EBV seropositive, but Henle and Henle³ had previously failed to demonstrate EBV antibody using the indirect immunofluorescence method in sera from the chimpanzee, baboon and rhesus monkey. Two of 4 baboons, however, inoculated in our laboratory with 10⁸ EB3 cells were found by the Henles³ to have low levels of antibody 4-8 weeks after exposure. Landon and Malan⁴ have reported the presence of EBV antibody in rhesus and cynomolgus monkeys at birth. This antibody disappeared after 8-10 months, although sera from the mothers of these animals remained positive. Levy *et al.*⁵ were able to demonstrate antibody in 2 of 3 chimpanzees bled immediately after capture in the jungle. The number of animals used, species and geographic distribution, and finally the lack of history on the animals involved in all of these studies, plus the need for a suitable experimental host system, suggested that additional studies were worth pursuing.

Our primary interest in EBV antibody was two-fold: first, the presence of antibody in relation to phylogeny, and second, the presence of antibody in newly-captured animals. To answer these questions, sera were obtained from representative Old and New World monkeys and apes after varying periods of time in captivity, and from chimpanzees and baboons immediately after capture (Fig. 1). All sera were tested by the indirect immunofluorescent method described by Henle and Henle⁶, using goat anti-human IgG and P3HR-1 cells. Anti-

monkey (squirrel) gamma globulin conjugate (Dr V. Dunkel (NIH)) was also used, and gave essentially the same results. This material was used to exclude possible lack of cross reactivity when using anti-human preparations.

As shown in Fig. 1, only sera from the Old World monkeys and apes yielded positive reactions. All the sera from both gorillas and chimpanzees (with one chimpanzee exception) had antibody with titres ranging from 1:20 to greater than 1:160. Also, 6 chimpanzees bled at the time of capture were all seropositive (titres 1:10 to greater than 1:160), and antibody was also present in baboon and rhesus sera. It will be noted, however, that only half (approximately) of these sera were positive and the titres were somewhat lower especially among the baboons. Again, two sera from baboons obtained on capture were also positive. Both of the New World species tested, that is, marmosets and howlers, were completely devoid of EBV antibody at the 1:10 level.

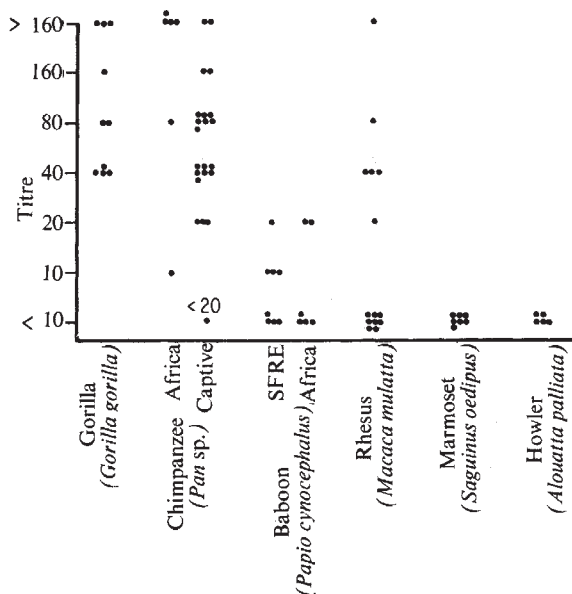


Fig. 1

Dunkel (personal communication) arrived at essentially the same results following testing of sera derived from the chimpanzee (*Pan sp.*), baboon (*Papio sp.*), rhesus (*Macaca mulatta*), cynomolgus (*M. fascicularis*), bonnet (*M. radiata*), African green (*Cercopithecus aethiops*), talapoin (*C. talapoin*), squirrel (*Saimiri sciureus*), and owl (*Aotus trivirgatus*) monkeys, marmoset (*Callithrix*) and the prosimian bushbaby (*Galago crassicaudatus*). The last four species were found to be negative.

The positive results obtained here are of interest, as they demonstrate a possible phylogenetic relationship to man as regards infection with EBV. Furthermore, the results also indicate a descending relationship as one passes from the apes down through the monkeys. Newly captured animals also have antibody, a finding that would suggest infection in nature with this or an antigenically similar virus, such as that causing infectious mononucleosis: this suggestion also has been put forward by Leby *et al.*⁵. While we are in agreement with this concept, our experiences in the wild⁷ make us cautious in stating that this is the only possibility. Both chimpanzees and baboons live close enough to man's environment to make it impossible to rule out human contact of some sort. These data would suggest, however, that a non-human primate "reservoir" for EBV may exist. Additionally the chimpanzee or baboon may serve as a model for the study of this virus.

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