therefore not be competition *in utero* (except in the case of twins) between embryos as in the mouse. Hence, embryos showing developmental abnormalities may have a greater chance of being born. Thus the increase in the frequency of developmental abnormality with maternal age in man may represent a true increase, while in the mouse the apparent observed decrease may be spurious.

In *Drosophila*, where the eggs are laid and there is not embryo formation *in utero*, competition of the type postulated will be non-existent, so that with age the incidence of developmental abnormalities might be expected to increase with maternal age as a result of poorer developmental stability of the offspring of old females. In *D. melanogaster* for two characters, egg length variance and sterno-pleural chaeta number asymmetry, suggestive evidence has been presented in agreement with this expectation¹⁵. Similarly, in the case of lethal homozygous Curly genotypes, where the embryos or first instar larvae die, an increasing proportion of genotypes die as embryos as maternal age increases¹⁸. An analogous effect occurs for a larval-pupal lethal factor where larvae from young females mainly develop as far as the pre-pupal stage, but from older females developmental standstill occurs almost exclusively prior to puparium formation¹⁹.

In man, it would therefore be of some interest to study the incidence of developmental abnormality in twins and single births in great detail. A more suitable organism for this work may be the sheep where the incidence of twinning is relatively high in some stocks.

It would also be of interest to compare the effect of teratogenic agents in mouse and man as affected by maternal age in view of the recent evidence for congenital abnormality in man due to thalidomide. The thalidomide data in man may be adequate for the detection of a maternal age effect if it exists. This may give information on a recent suggestion²⁰ that thalidomide operates by preventing a previously malformed foetus from being eliminated naturally.

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¹ Penrose, L. S., *Proc. Roy. Soc., B*, **115**, 431 (1934).

² Penrose, L. S., *J. Ment. Sci.*, **85**, 1141 (1939).

³ Penrose, L. S., *J. Obstet. Gynaec.*, **46**, 645 (1939).

⁴ Murphy, D. P., *Ann. N.Y. Acad. Sci.*, **57**, 503 (1954).

⁵ Edwards, J. H., *Brit. J. Prev. and Soc. Med.*, **12**, 115 (1958).

⁶ Rank, B. K., and Thomson, J. A., *Med. J. Austral.*, **2**, 681 (1960).

⁷ Holt, S. B., *Ann. Eugen.*, **12**, 220 (1945).

⁸ Holt, S. B., *Ann. Eugen.*, **14**, 144 (1948).

⁹ Wright, S., *Amer. Nat.*, **60**, 552 (1926).

¹⁰ Searle, A. G., *Ann. N.Y. Acad. Sci.*, **57**, 558 (1954).

¹¹ Green, E. L., *Genetics*, **26**, 192 (1941).

¹² Hauschka, T. S., and Brown, E., *Ann. N.Y. Acad. Sci.*, **57**, 465 (1954).

¹³ Reed, S. C., *Genetics*, **21**, 339 (1936).

¹⁴ Russell, E. S., *Ann. N.Y. Acad. Sci.*, **57**, 597 (1954).

¹⁵ Parsons, P. A., *J. Exp. Biol.*, **39**, 251 (1962).

¹⁶ Bodmer, W. F., *Nature*, **190**, 1134 (1961).

¹⁷ Bodmer, W. F., *Heredity*, **16**, 485 (1961).

¹⁸ Hanna, B. L., Sawin, P. B., and Sheppard, L. B., *Genetics*, **47**, 519 (1962).

¹⁹ Hadorn, E., *Developmental Genetics and Lethal Factors* (Methuen, 1962).

²⁰ Arnall, P., *New Statesman*, **64**, 286 (1962).

MISCELLANEOUS

An Improved Technique for the Preparation and Scanning of Thin-layer Chromatograms

THIN-LAYER chromatography offers many advantages over paper chromatography for the separation of biological compounds. While thin-layer chromatogram procedures are rapid and efficient, separations on glass plates do not allow scanning of the chromatograms on present equipment. Further, recommended methods for the removal of the thin layers from glass plates require additional work and are not satisfactory as spots may be distorted, back-

Fig. 1. Separation and scanning of serine, asparagine, valine and leucine from thin-layer chromatograms developed on plastic plates

grounds discoloured, and parts of the silica-gel layer lost due to adherence to the plate.

Thin-layer chromatography is used in our laboratories for investigations of the effect of diet on Newcastle disease virus infection^{1,2}. The following technique was devised and found to be satisfactory for the thin-layer chromatogram separation and scanning of the free amino-acids of avian livers (Fig. 1).

Thin-layer plates are prepared by applying the silica gel by standard procedures on the matted side of otherwise clear plastic. Product No. VCA 3310-C1 of the Union Carbide Plastics Co., Clifton, New Jersey, was found to be resistant to the various solvents and satisfactory for use in our separations. (Note, standard glass plates are still used when separations require caustic solvents for the identification of compounds.) The size of the plastic squares is limited only by the length of the developing tank. The plastic plate is spotted with the desired number of samples and placed in the solvent. Following a run, the plate is air-dried and stained. After the colour is developed and the plate completely dry Tuffilm Spray No. 543 (M. Grumbacher, Inc., New York) is applied. This product has been found to penetrate the silica gel without disturbing the spots and, when dry, leaves the plate with the appearance of an acetate strip. After the preceding treatment the plate can be readily cut into strips and scanned in standard equipment.

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¹ Squibb, R. L., *Poultry Sci.*, **40**, 425 (1961).

² Sanslone, W. R., and Squibb, R. L., *J. Nutr.*, **76**, 86 (1962).