examined are shown here with the normal disomic parent. Minor changes in gel pattern were observed in some of the other ditelocentric lines but in this communication we are only concerned with those lines illustrated. It can readily be seen that removing the arm of the 1D chromosome of 'Chinese Spring' results in the disappearance of 2 slow-moving bands. Apparent quantitative differences in these bands are observable in the proteins extracted from other ditelocentric lines but only the 1D chromosome appears to be associated with their complete removal. It is of interest to note that WELSH and HEHN[3] in studies with monosomics, found that the 1D chromosome had the greatest influence on flour dough quality as measured on the 'Farinograph' and fermentation time.

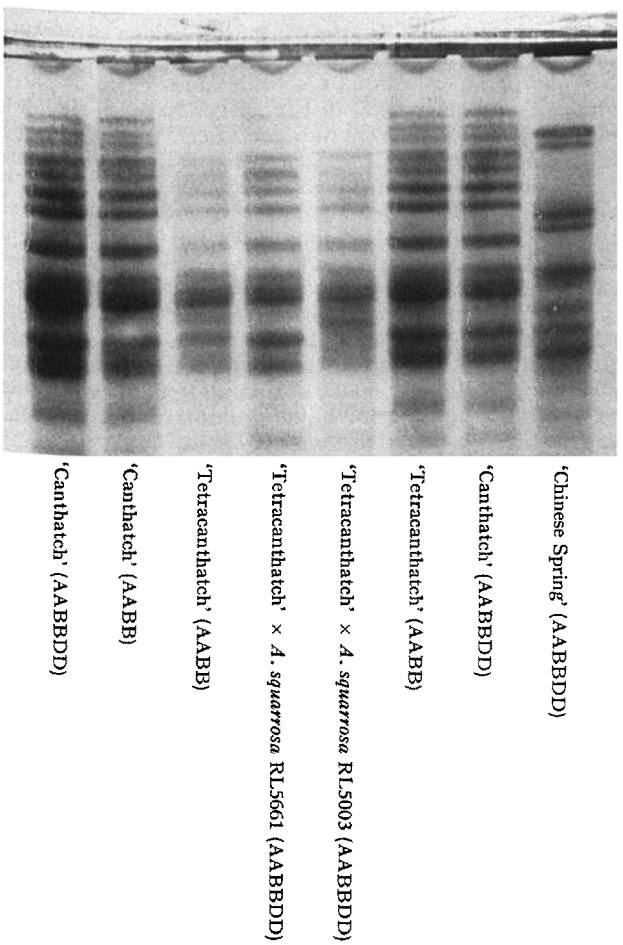

Fig. 2. Starch gel electrophoretic patterns of gluten proteins from related hexaploid and tetraploid wheats.

Figure 2 shows the starch gel patterns obtained with the hexaploid wheat 'Canthatch' together with its tetraploid derivative 'Tetracanthatch'. Protein patterns from hexaploids reconstituted from 'Tetracanthatch' and 3 varieties of *A. squarrosa* L. are shown together with that of 'Chinese Spring' for comparison. It is quite obvious that removal of the complete D genome from 'Canthatch' has had no observable effect on starch gel pattern. The proteins from reconstituted hexaploids show some differences in pattern but unavoidable differences in the amount of protein loaded into the gel make comparison of these samples difficult.

There is no doubt that individual protein components in gluten represent heritable characters. In a polyploid like wheat it appears that the genetic information controlling the synthesis of many of these proteins may be duplicated on different genomes. The only clear-cut example of single chromosome control shown in these studies is for the 1D chromosome of 'Chinese Spring'. There are 2 obvious interpretations of the results with 'Canthatch' and its tetraploid derivative where the removal of the D genome had no effect on protein pattern. The first explanation is simply that the chromosomes on the D genome are not controlling the synthesis of any of the proteins examined. Perhaps a more likely explanation is that the genes in the D genome controlling protein synthesis are duplicated in the A and B genomes. The fact that A, B and D genomes are all believed to have arisen from a single progenitor lends more credence to the latter explanation. The apparent contradiction of the results with 'Chinese Spring' where removal of part of a chromosome on the D genome caused a definite change in the protein 'profile' and the observation that removal of the whole D genome from 'Canthatch' caused no change, emphasises the complexity of the inheritance of these characters.

Résumé. L'hérédité des composants protéiques du gluten a été étudié au moyen de l'électrophorèse en gel d'amidon. Une branche du chromosome 1D de la variété du blé dit «Chinese Spring» est en corrélation avec la présence de 2 composants protéiques. Le composant du gluten n'a aucun effet lorsqu'on prélève le génome D entier à la variété «Canthatch».

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[3] J. R. WELSH and E. R. HEHN, Crop Science *4*, 320 (1964).

Hydrolysis of Human Leucocyte Phospholipids by Snake Venoms

The A-phospholipases of *Vipera palestinae* (VP) and *Naja naja* (NN) venoms do not split the phospholipids of intact erythrocytes, but are able to do so in the presence of a lytic factor (LF) in NN venom, a basic protein[1]. These venom phospholipases differ in their ability to attack the phospholipids in platelets[2], osmotic erythrocyte ghosts[1] and mitochondria[3,4], hydrolysis occurring with NN phospholipase but not with VP phospholipase. In the present study, the action of these venom components on leucocyte phospholipids was investigated.

Leucocytes were separated from normal blood according to the procedure described by BERGMEYER[5]. Venom phospholipases and LF were prepared as described pre-

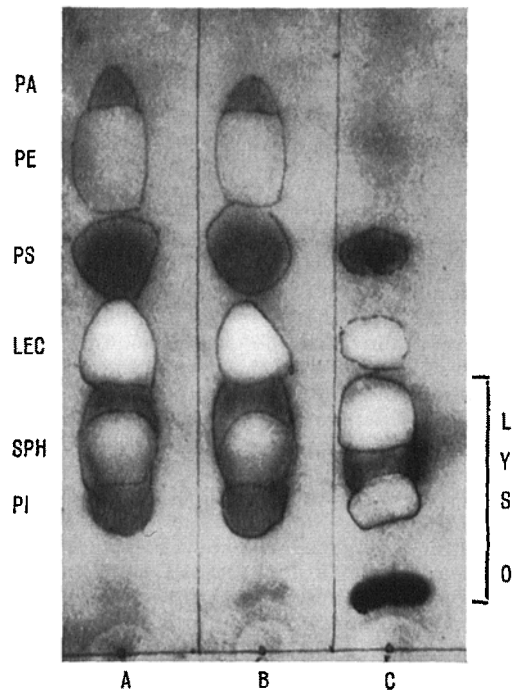

Fig. 1. Action of NN and VP on leucocytes. Incubation system: $3 \cdot 10^7$ cells in 1.7 ml buffered saline containing 1 μmol Ca^{2+} were incubated at 37 °C for 1 h with 1000 μg VP or 450 μg NN venom protein[6]. These amounts have equal phospholipase activity on egg yolk[1]. Silicic acid paper chromatography of leucocyte phospholipids: A, non-treated; B, VP venom-treated; C, NN venom-treated; PA, phosphatidic acid; PE, phosphatidylethanolamine; PS, phosphatidylserine; LEC, lecithin; SPH, sphingomyelin; PI, phosphatidylinositol; Lyso, lyso products.

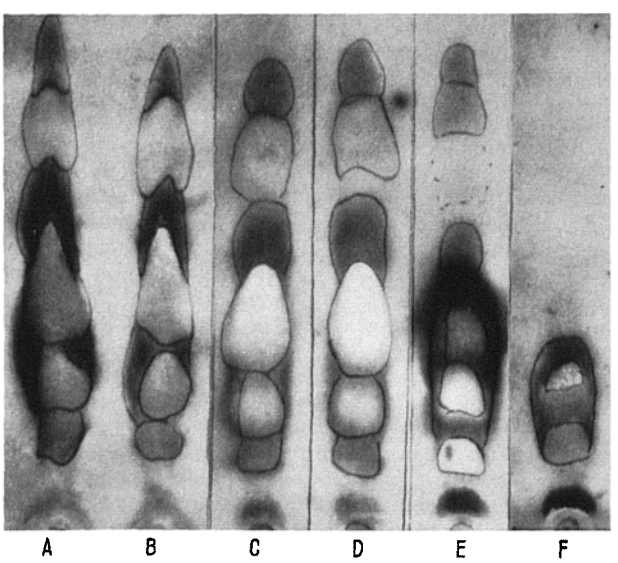

Fig. 2. Action of venom phospholipases and LF on leucocytes. Incubation system as in Figure 1 using 250 μg VP phospholipase and 125 μg NN phospholipase, having the same activity when examined on egg yolk. LF was added in the amount of 310 μg according to the proportion of the protein content of the LF and phospholipase fractions in the whole NN venom. Silicic acid paper chromatography of leucocyte phospholipids: A, non-treated; B, LF-treated, C, VP phospholipase-treated; D, NN phospholipase-treated; E, VP phospholipase + LF; F, NN phospholipase + LF. For identification of phospholipid spots see Figure 1.

viously[1,3]. Following incubation of leucocytes with venoms or venom fractions, the phospholipids were extracted from the leucocytes and determined by silicic acid paper chromatography[3].

Incubation of washed leucocytes with VP venom did not cause leucocyte phospholipid splitting (Figure 1). The VP venom-treated cells did not absorb trypan blue and retained the ability to phagocytize latex particles[7]. In contradistinction, NN venom caused disruption of leucocytes with almost complete splitting of the leucocyte phosphatidic acid, phosphatidylethanolamine, phosphatidylserine and lecithin.

Isolated VP and NN phospholipases had no phospholipid splitting action on leucocytes (Figure 2), nor did they render them permeable to trypan blue or impair their phagocytic capacity. Storage of the leucocytes at + 4 °C overnight prior to phospholipase treatment did not modify these results. On the other hand, when subjecting the stored cells to recentrifugation or hypertonic solution, NN phospholipase caused a considerable degree of phospholipid splitting with disruption of many cells. VP phospholipase, however, did not affect the cells, even after such treatment. Homogenization of leucocytes made their phospholipids susceptible to the action of both VP and NN phospholipases. Isolated NN-LF did not cause splitting of leucocyte phospholipids, but rendered them susceptible to the action of both venom phospholipases (Figure 2).

It appears that the leucocyte, in respect to the susceptibility of its phospholipids to the snake venom phospholipases, behaves in a manner analogous to the erythrocyte, different snake venom phospholipases acting on the cell according to the degree of damage. Whereas the phospholipases of both venoms are not able to attack the phospholipids of intact leucocytes, moderate damage renders them susceptible to NN phospholipase only, and homogenization to both venom phospholipases.

It is noteworthy that the phagocytic ability of the leucocytes was affected only when the cells were destroyed by either whole venom (NN) or phospholipases (VP or NN) combined with LF. In no instance was phagocytic ability affected in the absence of phospholipid splitting.

Résumé. Les phospholipases A des venins du serpent *Vipera palestinae* et du *Naja naja* ne clivent pas des phospholipides de leucocytes intacts, sauf en présence d'un facteur lytique se trouvant dans le venin de *Naja naja*. L'activité des phospholipases des deux venins sur les leucocytes endommagés n'est pas la même.

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[1] E. CONDREA, A. DE VRIES and J. MAGER, Biochim. biophys. Acta *84*, 60 (1964).
[2] C. KIRSCHMAN, E. CONDREA, N. MOAV, S. ALOOF and A. DE VRIES, Archs int. Pharmacodyn. Thér. *150*, 372 (1964).
[3] C. KLIBANSKY, J. SHILOAH and A. DE VRIES, Biochem. Pharmac. *13*, 1107 (1965).
[4] E. CONDREA, Y. AVI-DOR and J. MAGER, Biochim. biophys. Acta *110*. 337 (1965).
[5] H. U. BERGMEYER, *Methoden der enzymatischen Analyse* (Verlag Chemie, Weinhein 1962), p. 748.
[6] O. H. LOWRY, N. J. ROSENBOROUGH, A. L. FARR and R. J. RANDALL, J. biol. Chem. *193*, 265 (1951).
[7] Y. RABINOWITZ, Blood *23*, 811 (1964).