the staphylococci-binding site is located on Aα and Bβ chains involving C-terminal portion but not N-terminal disulfide knot. γ chain has no binding site for staphylococci. Abnormal fibrinogens with defects in the N-terminal portion interacted normally with staphylococci. The degree of binding paralleled the extent of clumping reaction between staphylococci and ^{125}I-fibrinogen or its plasminderived fragments. However, monomeric S-carboxymethyl Aα and Bβ chains showed a high degree of binding without concomitant clumping suggesting requirement for dimeric form of polypeptide chains for clumping. Conclusion: Aα and Bβ chains but not γ chain have homologous regions in terms of staphylococci-binding site on C-terminal portion responsible for clumping reaction. Thus, staphylococci can be used to measure abnormal fibrinogens with defects in the N-terminal region.

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B. Kudryk and B. Blomback (The New York Blood Center, 310 East 67 Street, New York, New York 10021, U.S.A.): **Immunochemical Studies on the NH_2-Terminal "Disulfide Knot" and Related Structures. (29)**

A radioimmunoassay for the NH_2-terminal portion of human fibrinogen (N-DSK) was recently used to measure cross-reactivity between several related fragments (Eur. J. Biochem. *46:* 141–147, 1974). The results obtained showed that "cold" N-DSK and the NH_2-terminal portion of plasmic Fragment E ("E-knot") were strong competitors while Fragment E itself was a poor competitor in the system ^{125}I-labeled N-DSK/anti-N-DSK. In this paper we report results from further experiments designed to study the basis for the observed immunological difference between Fragment E and "E-knot". Using the N-DSK radioimmunoassay system we have now shown that formic acid denaturation alone cannot account for this difference. Additionally, intact mixtures of CNBr-treated Fragment E competed to the same degree as "E-knot". The latter finding would suggest that there appears to be no interaction of the split COOH-terminal peptides (from Fragment E) with "E-knot" resulting in the "masking" of epitopes. However, at this point we cannot be certain that such "masking" of epitopes by these peptides does in fact occur in intact Fragment E.

In a related study we have also tested the cross-reactivity of a disulfide core fragment obtained from N-DSK by trypsin digestion. This fragment (DS-4) has an approximate molecular weight of 8,000 and has 4 disulfide bridges. The latter does not react on immunodiffusion using antisera to N-DSK. However, in the N-DSK radioimmunoassay system DS-4 showed very strong competition. Approximately 73% inhibition was observed at the 10 nmol level. The significance of these results with respect to the location of highly antigenic regions of N-DSK will be discussed.

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D. A. Lane, M. Brasher, V. V. Kakkar and P. J. Gaffney (Department of Surgery, King's College Hospital Medical School, and National Institute of Biological Standards and Control, London, England): **Characterisation of a Soluble High Molecular Weight e Fragment Released by Plasmin from Cross-linked Fibrin. (30)**

The heterogeneity of fragments released by plasmin degradation of cross-linked human fibrin has been examined. Six components (labelled 1–6) have been detected by PA gel electrophoresis and the two components (3 and 4) that make up the D dimer complex have been examined in relation to fragments D and E. Using two dimension immunoelectrophoresis (with PA gel in the first dimension) and immunodiffusion, together with SDS PA gel electrophoresis of the fragments and their subunits, it has been shown that the more anodic component (band 4) of the complex is D dimer. Band 3, which is the recently reported high MW component (Gaffney et al., Clin. Sci. Mol. Med., in press) that reacts with antisera to fragments D and E, is shown to be a strong non-covalent association of D dimer and E. This observed heterogeneity of the components of fibrin digestion is significantly more complex than has been previously reported.