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Some Aspects of Theories of Spontaneous Coagulation

A. I. McMullen *

(Rubber Research Institute of Malaya)

Apart from the important technical questions concerned in the problem of the spontaneous breakdown of the Hevea latex colloidal suspension, the academic speculations arising in discussion of this interesting phenomenon have far-reaching implications in the study of plant physiology and have not yet been satisfactorily answered despite (or because of!) fifty years of spasmodic researches in the problem.

A brief description of the Colloid Chemist's picture of the organisation of molecules at the hydrocarbon-serum interface in Hevea latex is given with the corresponding theoretically possible methods of disorganisation of same, thus leading to flocculation and coagulation.

The causative agents of this disorganisation are regarded in the literature as being bacteria or enzymes and much controversy has existed as to which of these is essentially responsible. Much of the contradictory evidence is due to lack of uniformity in methods and material and partly to lack of differentiation between native enzymes and those originating in bacteria from external sources.

It is adduced from the reliable evidence available in the literature and from data supplied from research projects being carried out at the Rubber Research Institute of Malaya that spontaneous coagulation, as it is normally encountered, is due to the activity of contaminating micro-organisms, but that destabilisation of sterile latex or pre-coagulation of fresh latex is caused by an enzyme system set in motion by activators liberated from the lutoids present in the so-called "yellow fraction." The theory is advanced that the activating agent, most likely (on available evidence) Ca ion or Ca proteinate, is present in high concentration inside the lutoid "cell", or round the lutoid membrane. Such a concentration difference may be maintained in the latex vessels by normal biological dynamic equilibrium energy or by physio-chemical mechanisms as described by Danielli.

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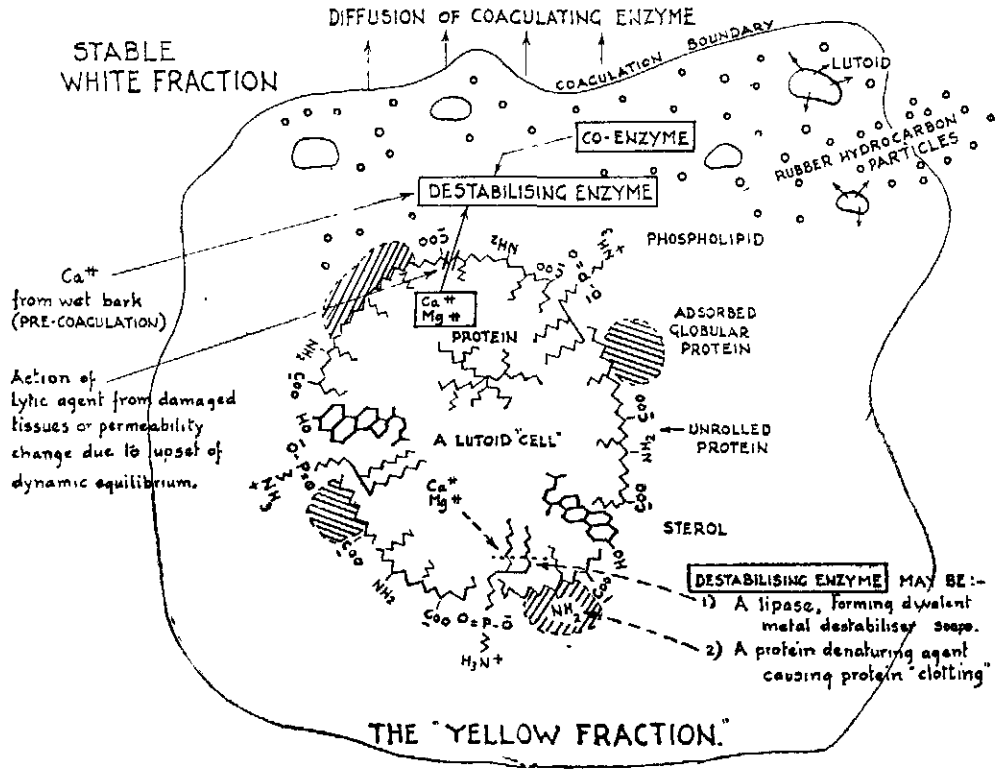
When the latex is removed from this environment the activating ion is released into the serum in high concentration by change in membrane permeability due to breakdown of the equilibrium mentioned, or by the action of a lytic agent originating from damaged tissues or protoplasmic lining. The activator then unites with co-enzyme already present in the serum, giving rise to the active destabilising enzyme, in the neighbourhood of the lutoids. This enzyme then attacks the protein component of the protective layer surrounding the hydrocarbon particles by a denaturing action and flocculation occurs. This mechanism is illustrated in the schematic picture shown. (In this the structure of the lutoid is much simplified since surface techniques have shown that membranes behaving as osmotic barriers must be multi-molecular in thickness).

If the lutoids are allowed to settle under gravity then local or pre-coagulation takes place and complete destabilisation proceeds slowly as active enzyme or activator diffuses out from the coagulating centre. If the lutoids are maintained in a dispersed condition by shaking, total (but no fractional) coagulation occurs.

Discussion

It was suggested that possibly the explanation of such an apparently complex process as the spontaneous coagulation of sterile latex on the theory of a single mechanism may not represent the complete picture and that it might be necessary to consider that two influences are at work during disruption of the "mosaic" interfacial layers, namely a protein denaturing agent and a lipase producing long chain fatty acid from lipoid molecules.


DIFFUSION OF COAGULATING ENZYME
STABLE WHITE FRACTION
COAGULATION BOUNDARY
LUTOID
RUBBER HYDROCARBON PARTICLES
CO-ENZYME
DESTABILISING ENZYME
PHOSPHOLIPID
Ca++ from wet bark (PRE-COAGULATION)
Ca++ Mg++
PROTEIN
ADSORBED GLOBULAR PROTEIN
Action of lytic agent from damaged tissues or permeability change due to upset of dynamic equilibrium.
A LUTOID "CELL"
UNROLLED PROTEIN
STEROL
DESTABILISING ENZYME MAY BE:-
1) A lipase, forming dyvalent metal destabiliser soaps.
2) A protein denaturing agent causing protein "clotting"
THE "YELLOW FRACTION."