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A Possible Explanation of the Gel-Sol Changes in Amoeboid Movement, Based on the Muscle Contraction Theories of Szent-Gyorgyi

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Introduction

The possibility that amoeboid movement and muscle contraction may be very similar and, perhaps, identical physico-chemical phenomena has occurred to numerous investigators during the past century. Ecker (1849) may have been the first to express this idea in the literature in terming muscle "differentiated contractile substance" and sarcode "undifferentiated contractile substance." Only recently, however, has evidence accumulated from the fields of biochemistry and physiology which bids fair to establish the reality of that possibility.

Szent-Gyorgyi (1949) has recently and vividly expressed in concrete language a theory of muscle contraction based on the results of his many biochemical studies of that tissue which may be employed to explain on biochemical grounds the course of events in amoeboid motion as set forth by Mast (1926, 1931, 1934) in the conversion of plasmasol to plasmagel, the contraction of the latter, and its reconversion to plasmasol.

Historical

Ecker (1849) has claimed that a pseudopodium was likely to be formed by protrusion of part of the body mass as a result of the contraction of part of the body posterior to the pseudopodium. M. Schultze (1861, 1863), Wallich (1863 a, b, c), and F. E. Schultze (1875), as well as Leidy (1879), were early investigators who remarked upon the contractility of the outer portion of the amoeboid protoplasm.

Heitzmann (1873) attempted to explain contractility in protoplasm by postulating a reticulum in three dimensions within the protoplast, the reticulum being of contractile, living material imbedded in a non-contractile, non-living fluid.

Theories of reticular structure and contractility of protoplasm held the foreground until the mid-1880's, despite many opponents, but gave way to and were superceded by granular theories of structure, and employment of surface tensions at interfaces to account for locomotion. Flemming (1882) believed protoplasm to be made

of filaments dispersed in a fluid; Altmann (1890), granules in fluid; and Butschli (1892), droplets. Studies by Quincke (1870, 1877) on surface tensions led Berthold (1886), Butschli (1892), Jensen (1901, 1902), McClendon (1911), K. Gruber (1912), L. Haberlandt (1919) and Furth (1922), to attempt to explain amoeboid movement according to changes in surface tension.

The opponents of the surface tension theories were many, and damage done them by Mast and Root (1916) and Beers (1924) in demonstrating that surface tensions could not account for the amount of force necessary to cut a ciliate in half in feeding, as amoebas are known to do, virtually ended the surface tension approach.

Hyman (1917) was one of the first to utilize the colloidal structure concept in explaining amoeboid movement, conceiving of changes in phase from sol to gel and the reverse, with tensions in the ectoplasmic gel furnishing the driving force. The contraction, she assumed, might be compared to the synereses of gels.

Pantin (1923) also employed gel-sol changes to explain his concept of amoeboid movement, describing the formation of ectoplasm (plasmagel) from endoplasm (plasmasol) at the advancing anterior end of the pseudopodium, and the reverse process taking place internally just in front of the posterior "tail piece". He, too, conceived of syneresis as the driving force.

Mast (1926, 1931, 1934) explained locomotion of amoebas in a manner which employed gel-sol changes, substituting elastic recoil of the gel for synerectic contraction as the driving force which results in the forward movement of the sol. Mast also devised an ingeneous explanation of layered construction, with convenient separation or adherence of the thin outer layer (plasmalemma) from or to the substrate and the layer immediately within (plasmagel), the outer layer being a clear, thin, elastic, protoplasmic sac. His employment, thus, of the plasmalemma satisfied objections lodged against gel-sol theories of movement, particularly objectives based on the "rolling" movement described by Jennings (1904), and the "walking" motion recounted by Dellinger (1906).

Gel and sol changes have not, however, satisfactorily explained the mechanism so as to exclude serious objections, particularly for protoplasmic structure, nor why there should be a gradient in the rigidity of the plasmagel as found by Marsland and Brown (1936), unless the contraction is synerectic, which Mast denies.

Newer concepts of the submicroscopic, micellar structure of protoplasm, indicating the presence of networks of polypeptide chains which are formed and dissociated repeatedly by establishment and

breakage of chemical linkages (Peters, 1937; Frey-Wissling; 1940; Seifritz, 1942; Szent-Gyorgyi, 1949; Goldacre and Lorch, 1950), point to the application of Szent-Gyorgyi's muscle-contraction theories to explain amoeboid gel-sol changes.

This writer has been unable to find in the literature as yet any works which establish by microchemical analyses the presence in amoebas of myosin, actin, adenosine-triphosphate, and direct evidence of the inter-action of these substances. However, Goldacre and Lorch (1950), by means of injections of adenosine-triphosphate into amoeboid protoplasm in movement, using a micropipette, achieved greater contraction in areas of gel already in contraction, and delay of formation of gel at the anterior end where the sol-gel transformation normally occurs. They postulated, on the strength of these experiments, a pattern of association, contraction and dissociation of polypeptide chains which very closely resembles the muscle contraction theories of Szent-Gyorgyi, except that the proteins involved are not named.

Increasingly accumulating evidence demonstrates the presence of enzymes in amoebas (Holter and Kopac, 1937; Holter and Doyle, 1938; Andersen and Holter, 1949) and, by implication, the involvement of them in biochemical processes occurring within and conducted by amoeboid protoplasm.

According to Szent-Gyorgyi (1949), the two principle proteins of muscle, actin and myosin, bind and dissociate depending on concentrations of potassium and calcium ions within very narrow ranges. The relaxed muscle is in a precariously balanced equilibrium which can be upset by changes in the concentration of potassium of as little as 0.01 M if magnesium ions are also present. In the relaxed muscle, myosin exists in straight chains, hydrated and binding to it magnesium ions; actin is then present in a globular form.

Hydrated, extended myosin has a great affinity for adenosine-triphosphate, easily forming a chemical attachment to it, but does not react with it so long as actin remains globular and unattached to myosin. Actin forms molecular fibers and micelles by changing from its globular polymer if potassium or sodium ions are added which change the concentration of either more than 0.01 M. These fibers attach to one another at their ends and along their broad sides, and attach by their ends to the ends of myosin fibers.

The actino-myosin-adenosine-triphosphate complex, once formed, is extremely unstable. The protein fibers tend to shorten, either folding or twisting, and dehydrate, becoming energy poor. If the system is not allowed to shorten, tension and isometric state of con-

traction are developed. Adenosine-triphosphate promotes the attachment of actin fibers to myosin, and so long as it is present they remain fastened together. Myosin, however, has ATP-ase activity, breaks the adenosine-triphosphate, releasing energy which promotes myosin contraction. As adenosine-triphosphate is destroyed, the actin fibers dissociate from the contracted myosin fibers and returns to the globular form. The myosin fibers hydrate spontaneously at normal ionic concentrations of muscle, and again bind magnesium ions and adenosine-triphosphate. The actin molecules retain their globular form until sufficient potassium and/or sodium ions are present to cause them to again assume the fibrous state. In muscle these needed ions are provided by the action of the nerve impulse in changing the permeability of the sarcolemma to these ions as the impulse passes the myoneural junction.

Discussion

Application of these events postulated by Szent-Gyorgyi for muscle to the gel-sol changes of amoeboid movement entails little or no change. Present evidence does not yet permit the proteins to be positively identified as actin and myosin, but Goldacre and Lorch (1950) have demonstrated the efficacy of adenosine-triphosphate in promoting contraction in amoebas, so that some complex employing ATP is certainly present to utilize its stored energy.

If one were to assume that the proteins are myosin and actin, (and that assumption appears reasonable), the sol could be identified as equivalent to muscle in the relaxed, precariously equilibrated state; and the gel equivalent to muscle which has been stimulated to become fibrous, being either isometrically or isotonically contractile, depending upon the tensions, locally, upon the complex which prevent or permit shortening.

Local weakening of the peripheral gel in a resting amoeba might occur at any point where ionic changes of potassium or sodium would be sufficient to dissociate the actin-myosin linkage. Sol would be pushed through the weakened area against the plasmalemma, stretching it circumferentially and linearly so that pseudopodal formation would begin. Stimuli created by conditions in the surrounding liquid through which the plasmalemma were pushed might serve to change its permeability (as might also the stretching of it) so that potassium ions or sodium ions might enter or escape, changing the concentrations of those ions enough to cause linkage of actin and myosin within the sol just behind the plasmalemma. These formed fibers, pushed peripherally and adding more linkages by their broad sides to other similarly formed complexes, would make

a firm gel tube, the protein fibers of which would enter into isometric contraction. Since the gel tube cannot compress the liquid sol within it, the tube would keep its shape and isometrically contracted condition, until the volume of the sol flowing through it were lessened. This volume would be lessened in the sol near the posterior end in the same amount and at the same rate as the sol were pushed forward into the lengthening pseudopod. The protein complex in the posterior end of the body, particularly nearest the posterior end, would undergo isotonic contraction and shortening.

Complete shortening of the myosin-like part of the gel would be achieved at the internal boundry of gel against sol at the posterior end of the amoeba. As adenosine-triphosphate were destroyed, the actin-like protein would become globular, releasing myosin, forming sol from gel. with the resultant dissociation of the proteins of the complex and the dispersion of them in the liquid.

The sol thus formed would be pushed forward by peripheral and posterior gel contractions. During its flow towards the anterior end of the organism, myosin fibers would straighten and reform the linkage with magnesium ions, and with adenosine-triphosphate as it would be reconstituted by transfer of high energy phosphate bonds to the adenylic acid and adenosine di-phosphate residues split during contraction from the original adenosine tri-phosphate. On reaching the anterior end the sol would again encounter an area where a change in the postassium ion concentration would once more shift the globular actin to its chain-form polymer, instigating another cycle of gel formation and contraction.

Not only the continuous advance of a naked amoeba may be explained by the events in muscle contraction, but also the various other contractile activities performed by other amoeboid protoplasm.

Since, according to Szent-Gyorgyi (1949), actin and myosin do not form a fibrous complex until the potassium ion concentration is right to change the actin from its globular to its fibrous polymer, that cation must be brought in at the concentration sufficient to promote the change. Before the complex can contract, magnesium ions must be present, as well as adenosine-triphosphate. Depending upon the locales and concentration of the various chemical materials, and the order and amounts in which they appear at the locales concerned, almost any contractile pattern might be accounted for.

Summary

1. The similarity of contraction in amoeboid protoplasm and muscle was noted as early as 1849 by Ecker.

2. Early theories of contractility in protoplasm did not satisfactorily explain amoeboid movement.

3. Theories based on surface tension changes, and on synereses of gels have not been adequate in explaining protoplasmic contractions.

4. Theories based on the colloidal structure of protoplasm and gel-sol changes have not been satisfactory, unless it can be explained how and why the local as well as the general gel-sol changes and the reverse occur.

5. The muscle contraction theories of Szent-Gyorgyi, employing the concept of ionic changes in potassium and magnesium in triggering the polymeric changes and the association of actin and myosin-ATP in a contractile complex, are adequate to explain changes in gel to sol and from sol to gel in amoeboid movement in its many patterns and sequences.

Literature Cited

Altmann, R., 1890. Die Elementarorganismen und ihre Beziehungen zu den Zellen. Veit and Co., Leipzig.

Andersen, N. and H. Holter, 1949. The Genera of Amoebae. Science, 110: 114-115.

Beers, C. Dale, 1924. Observations on Amoeba feeding on ciliate Frontonia. Brit. J. Exp. Biol., 1: 335-341.

Berthold, G., 1886. Studien über Protoplasmamechanik. A Felix, Leipzig.

Bütschli, O., 1892. Untersuchungen über mikroscopische Schäume and das Protoplasma. W. Engelmann, Leipzig.

Dellinger, O. P., 1906. Locomotion of Amoebae and allied forms. J. Exp. Zool., 3: 337-358.

Ecker, A., 1849. Zur Lehre vom Bau und Leben der contractilen Substanz der niedersten Thiere. Zeit. Wiss. Zool., 1: 218-245.

Frey-Wissling, A., 1948. Submicroscopic Morphology of Protoplasm and its Derivatives. Elsevier, New York.

Fürth, O., 1922. Zur Theorie von der Struktur der Zellen. Jena Zeit, Naturw., 14: 458-465.

Goldacre, R. J. and I. J. Lorch, 1950. Folding and unfolding of protein molecules in relation to cytoplasmic streaming, amoeboid movement and osmotic work. Nature; 166: 497-500.

Gruber, K., 1912. Biologische and experimentalle üntersuchungen an Amoeba proteus. Arch. Protistenk., 25: 316-376.

Haberlandt, L., 1919. Uber amöboide Bewegung., Zeit. Biol. 69: 409-436.

Heitzman, C., 1873. Untersuchungen über das Protoplasma. I. Bau des Protoplasmas. Sitzber. Kaiserl. Akad. Wiss. Math.-Naturwiss. Cl., 67 (III. Abth.): 100-115.

Holter, H. and W. L. Doyle, 1938. Studies on Enzymatic Histochemistry. XXVIII. Enzymatic Studies on Protozoa". J. Cell. and Comp. Physiol., 12: 295-308.

Holter, H. and M. J. Kopac, 1937. Studies on Enzymatic Histochemistry. XXIV. Location of Peptidase in Amoeba". J. Cell. and Comp. Physiol., 10: 423-437.

Hyman, Libbie, 1917. Metabolic gradients in Amoeba and their relationships to the mechanism of amoeboid movement". J. Exp. Zool., 24: 55-99.

Jennings, H. S., 1904. Movement and Reactions of Amoeba. Publ. Carnegie Instn., No. 16: 129-134.

Jensen, P., 1901. Untersuchungen über Protoplasmamechanik. Pfluger's Arch. ges. Physiol., 87: 361-417.

Jensen, P., 1902. Die Protoplasmabewegung. Ergebn. Physiol. (Part 2): 1-42.

Leidy, J., 1879. Fresh-Water Rhizopods of North America. Report U. S. Geol. Survey, 12: Washington.

MeClendon, J. F., 1911. Ein Versuch, amöboide Bewegung als Folgerscheinung des wechselden elektrischen Polarisationzustandes der Plasmahaut zu erklaren. Pfluger's Arch. ges. Physiol., 140: 271-280.

Marsland, D. A., and D. E. S. Brown, 1936. Amoeboid movement at high hydrostatic pressure. J. Cell. and Comp. Physiol., 8: 167-178.

Mast, S. O., 1926. Structure, movement, stimulation and locomotion in Amoeba. J. Morph., 41: 347-425.

Mast, S. O., 1931. Locomotion in *Amoeba proteus* (Leidy). Protoplasma, 14: 321-330.

Mast, S. O., 1934. Amoeboid movement in *Pelomyxa palustris* (Greeff). Physiol. Zool., 7: 470-478.

Mast, S. O., and F. M. Root, 1916. Observations on ameba feeding on rotifers, nematodes and ciliates, and their bearing on the surface tension theory. J. Exp. Zool., 21: 33-49.

Pantin, C. F. A., 1923. On the physiology of amoeboid movement. J. Marine Biol. Assoc., 13: 26-49.

Peters, R. A., 1937. Proteins and cell organization. In Perspectives in Biochemistry, J. Needham and D. E. Green, Eds. Cambridge U. Press, Cambridge.

Quicke, G., 1870. Ueber Capillaritäts-Erscheinungen an der gemeinschaftlichen Oberfläche zweier Flüssigkeiten. Ann. Phys. Chem., 139: 1-89.

Quincke, G., 1877. Ueber den Randwinkel und die Ausbreitung von Flüssigkeiten auf Fest Körpern. Ann. Phys. Chem., 2: 145-194.

Schultze, M., 1861. Ueber Muskelkörperchen und das, was man eine Zelle zu nennen habe. Arch. Anat., 1-27.

Schultze, M., 1863. Das Protplasma der Rhizopoden und der Pflanzenzellen. W. Engelmann, Berlin.

Schulze, F. E., 1875. Rhizopodenstudien IV. Arch. Mikr. Anat., 11: 329-353.

Seifritz, W., 1942. Some physical properties of protoplasm and their bearing on structure. In Structure of Protoplasm, W. Seifritz, ed., Iowa State College Press, Ames.

Szent-Gyorgyi, A., 1949. Attacks on Muscle. Science, 110: 411-413.

Wallich, G. C., 1863a. Further observations on an undescribed indigenous Amoeba, with notices on remarkable forms of Actinophrys and Difflugia. Ann. Mag. Nat. Hist., 12 (3rd series): 365-371.

Wallich, G. C., 1863b. On the value of distinctive characters in amoeba. Ann. Mag. Nat. Hist., 12 (3rd series): 111-151.

Wallich, G. C., 1863c. Further observations on distinctive characters and reproductive phenomena of the Amoeban Rhizopods. Ann. Mag. Nat. Hist., 12 (3rd series): 329-337.

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