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50 & 100 YEARS AGO

50 YEARS AGO

There is something very depressing about contemporary biological journals. Paper after paper records observations or experiments, analyses them cautiously, and in a timid and tentative way compares them with previous observations and experiments on the same theme. That is about all: only rarely does the writer disclose how (in his view) his work is related to the broad panorama of biology. There are doubtless sufficient reasons for these omissions: many writers of papers undertake the research they describe for no other reason that that their supervisors 'put them on to it', and many editors of journals consider contemplation out of place in science and do not encourage authors to indulge in it...How refreshing it is, for example, to hear that the choice of a subject for research involves the "art of rejection", and to be told that this art can be compared with the art of the Chinese in designing the empty spaces in their pictures. It is refreshing, too, to be reminded...that the very observations one makes, and *a fortiori*, one's interpretation of them, are limited by the Zeitgeist and by unconscious philosophical assumptions derived from Spinoza.

From *Nature* 27 August 1955.

100 YEARS AGO

A somewhat lamentable aspect of modern science is the vast array of unorganized facts which are awaiting coordination; this is too often because they have been amassed without any definite idea of the purpose which they may serve; consequently it may happen that laborious observations belonging to one science may fail to attract the regard of a neighbouring science merely for want of the mutual acquaintance which would make them serviceable to each other; and in these days of exclusive specialisation the introduction which might lead to a happy union is, perhaps, not brought about for years.

From *Nature* 24 August 1905.

tails that propel them); plants; and chromoalveolates (including dinoflagellates and the apicomplexan parasites such as the malaria-causing *Plasmodium*). The complete sequences of these myosin proteins were then used to find protein domains present in the myosin tails and at the extreme N-termini of some of the molecules.

The analysis uncovered a rich variety of myosins throughout the eukaryotes, extending the catalogue to 37 distinct types of myosin — almost double the number known before. Previously unknown myosin tails containing unique combinations of protein domains were revealed, such as the type 3 myosin from the water mould *Phytophthora ramorum*. This has a series of ankyrin (ANK) protein–protein interaction domains followed by a FYVE domain that binds to the phosphoinositide PI(3)P. Moreover, the analysis confirms that no single myosin is common to all organisms, and that the complement of myosins in any given species ranges from two (in *Entamoeba*) to 13 (in *Phytophthora*). The diversity of myosins is likely to be reflected in the range of actin-based movements that a given cell type or organism can generate, and future functional studies of novel myosins may well reveal a wider range of roles for this group of motor proteins than previously suspected.

Is there a single ancestral myosin? The available data can only narrow down the possibilities to the presence of at least three ancestral myosin subfamilies in the eukaryotic cenancestor (Fig. 1). Previous studies had hinted that two of these, the myosin I and MSD myosins, were present in the earliest eukaryotes, and Richards and Cavalier-Smith's more extensive analysis provides a firm basis for this supposition. It also reveals that a third cenancestral myosin group consists of the MyTH4/FERM myosins, which are present not only in amoebae and multicellular animals (metazoans) but also in chromoalveolates.

So how did the different types of myosin evolve? As one might expect, it seems that following the appearance of the major cenancestral groups, the myosins diversified during eukaryotic evolution by gains and losses of protein domains. Notably, class II myosins, some of the best-studied myosins, are not ancient but arose during the evolution of the unikonts (organisms that have a single flagellum), which include the amoeboid, fungal and metazoan lineages. In addition, certain types of myosins were lost in some groups during evolution. For example, myosin I is missing from the plant lineage and, in an extreme example, no myosins could be found in *Trichomonas* and *Giardia* (both of which are primitive unicellular parasites) or red algae. Myosins in these lineages could have either diverged radically from the rest of the family or been lost altogether: given the existence of myosins in other rapidly evolving groups, it seems most likely that they were lost.

Richards and Cavalier-Smith[1] also address the larger question of the nature of the ancestral eukaryote. Their results are consistent with the emerging hypothesis that a fundamental separation between unikonts and bikonts (cells with two flagella), a group that includes plants, chromoalveolates and the excavates, is the earliest evolutionary divergence. They also infer some of the cellular structures that the common ancestor of these two groups must have possessed: it would have had a single cilium, a centriole and a mitochondrion, and would have had the ability to form a pseudopod. This ancestral cell would have had at least three different types of myosin, with the myosin I perhaps regulating formation of the pseudopod to aid cell movement, the MSD myosin contributing to both cell division and organelle movement, and the MyTH4/FERM myosin having a role in adhesion to substrates and perhaps even contributing to the formation of specialized actin-filled

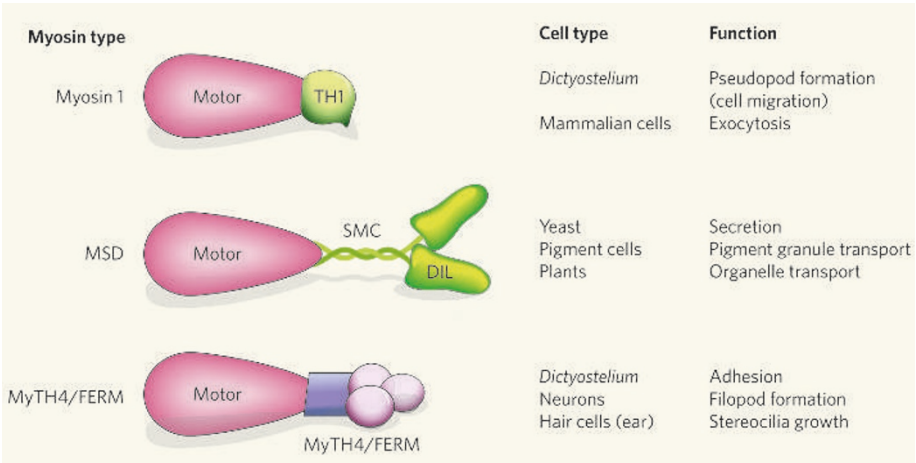

Figure 1 | Three likely ancestral myosins. Richards and Cavalier-Smith[1] propose that there were three ancestral myosins in the earliest eukaryotes, each with distinct tail domain structures. Listed are examples of organisms or cells expressing members of each myosin group and their known functions[2,3]. The TH1 domain would probably bind to charged lipids and target these myosins to membranes. The SMC domain would promote dimer formation and the DIL and MyTH4/FERM domains would target myosins to their cargo or subcellular location.

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