

Cell Motility

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The retention of cell motility, and its associated molecular machinery, in mature human cells is best explained through the lens of its evolutionary history. In eukaryotic cells the molecular system of motility, although extremely adaptive in its phenotypic expression, consist of a limited number of constituents encoded by a highly conserved set of genes. (Cappuccinelli P, 1980). These key constituents, namely dyenin ATPase motor proteins, and microtubules, originated from and, in fact, still integrate closely with, another important cellular system, the cytoskeleton. Just as the components of the cellular motility form a natural extension of the cytoplasm, so does its functionality. While the cytoskeleton acts to maintain homeostasis by regulating transport in the internal cellular environment (Fletcher and Mullins, 2010), The motility system maintains homeostasis by regulating the cells external environment, i.e by moving to different external environments.

The evolutionary advantage provided by the loose form of environmental regulation afforded by cellular motility is, perhaps, most apparent in the context of a single cellular organism. Protists rely heavily on their motility and associated chemo-taxis pathways to attain their food, either in actively seeking out high nutrient environments, (Fenchel and Blackburn, 1999), or in the predation of prokaryotes prey species. (Pernthaler, 2005). Additionally, motility is also central in evading damage from toxins within the environment, (Ermilova *et al*, 2007), and predation by other protists (Jakobsen, 2001). Overall, motility in protists is clearly an evolutionary positive trait, with no obvious disadvantages.

The advantages motility conferred to single cellular eukaryotes serve well to explain the original presence of cellular motility in early multicellular eukaryotes, and such it could be argued that the retained motility structures present in adult human cells are merely the remnants of an historically advantageous cellular system. However, in isolation, this explanation is woefully insufficient, as it omits the many distinct evolution disadvantages, as well as advantages which motility can confer to multicellular organisms. For example, cellular motility is absolutely essentially in effective tissue repair, (Rosen and Goldberg, 1989), but is also active in the development and progression (metastasis) of cancers (Condeelis, 2005). In consideration of these mixed consequences the widespread retention of motility factors in higher eukaryotes becomes unclear. In fact, it might seem more evolutionary positive for most mature adult cells to loose their capacity for motility during specialization, restricting the

motility required for tissue repair and embryologists to a few specialized cell lines, and in so doing minimizing the associated risks.

However, despite the perceived disadvantages of widespread motility/motility factor retention, there is still a sound evolutionary explanation for its occurrence. The route of this explanation lies in the nature of multicellularity itself. In certain contexts, multicellular organism are best understood, not as singular entities but rather as a symbiotic communities consisting of many different colonies of specialized cells. The general consensus is that multicellularity arose from increased specialization in colonial associations, due to the increased chances of survival and or reproductive success afford to each cell within the colony. (Michod *et al*, 2006). Although in many contemporary multicellular eukaryotes this specialization has lead to an almost complete co-dependence between tissue types, it must be remembered that evolution still operates at a cellular as well as individual/community level. In some instances theses evolutionary forces may align, such as in the effective digestion of pathogens by macrophages which provides a food source for the macrophages and immunity against disease for the cellular community/organism. Conversely, in some cases these evolutionary forces may conflict as in the formation of metastatic tumors: Tumor cells best ensure their own survival and reproduction by rapid division and migration to evade the immune system, even though this activity might compromise the overall survival of the cellular community/organism, analogous to an algal bloom which leads to the formation of a marine dead zone. The retention of cellular motility factors in general, can be similarly explained. That is, despite the risks and inefficiencies implied for the cellular community, for an individual cell the capacity for motility, or the capacity to regain motility through mutation remains an highly evolutionarily positive trait.

In conclusion the retention of cell motility factors in adult human tissues is a direct result of the evolutionary positivity of motility. The cellular motility which arose in single cellular eukaryotes, was retained, (or at least its molecular basis was maintained) in multicellular eukaryotes because for multicellular organisms evolutionary pressure is felt at a cellular as well as at an organismal level. Therefore tissue cells maintain the potential for motility as it aid in their individual survival and reproduction, even while it risks the survival and reproduction of the organism as a whole.

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