The genome of Mantamonad sp. str. SRT306 illuminates the evolutionary dynamics of membrane-trafficking machinery in eukaryotes.

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In studying the evolutionary dynamics of membrane-trafficking proteins, three patterns have emerged. Two such patterns are easily interpreted. Widely distributed and conserved proteins have allowed the reconstruction of a sophisticated endomembrane system in the Last Eukaryotic Common Ancestor, while lineage-specific proteins have shown the extensive innovation that has happened in descendant lineages. The third pattern, however, is less easily explained. There exist membrane-trafficking proteins that are widely distributed across eukaryotic taxa but are also frequently not found present in eukaryotic genomes, i.e. a “Patchy” distribution. Two immediate explanations arise for such a pattern. One is Lateral Gene Transfer, a well-documented phenomenon that occurs in microbial eukaryotes. The other explanation, and the one best supported by the weight of evidence in this case, is that these genes were present in a LECA whose complexity allowed for cellular redundancy. Since the LECA is inferred to be a free-living heterotroph, the genes have been lost repeatedly in some specialized and streamlined descendant lineages. A prediction of the latter hypothesis is that, as more genomes of free-living heterotrophs are sequenced, these patchy proteins will be found in greater abundance and together in the same genomes. We have sequenced and analyzed the genome of Mantamonad sp. str. SRT306 looking specifically at membrane-trafficking proteins. The HTAC vesicle coat adaptors are here described and provide several examples of patchy proteins. Further examination of other membrane-trafficking complexes systems similarly demonstrate the presence patchy proteins, which we argue is most consistent with the evolutionary dynamics of patchy proteins in the membrane trafficking system being explained by a sophisticated LECA at the edge of cellular complexity.