Leishmania are major human pathogens that, like all trypanosomatid parasites, use their single flagellum for vital roles including motility, surface attachment and environmental sensation. Normal motility is necessary for *Leishmania* to establish infection of the sandfly vector however comparatively little is known about how they control their motility to navigate the sandfly gut. We previously showed that differences between the proximal and distal flagellum, specifically in the outer dynein arms and dependent on a paralogous pair of docking complex heterodimers, are conserved between *Trypanosoma brucei* and *Leishmania* and are important for controlling flagellum beat type. We asked how common proximal-distal asymmetry was, if it always involves the outer dynein arms and if it is always docking complex-dependent. To address this, we used TrypTag to identify 25 proximal and 26 distal flagellum-specific proteins, and subjected the ~30 with a *Leishmania* ortholog to analysis. Each was endogenously tagged with a fluorescent protein, to ask if their asymmetry was similar to *T. brucei*, identifying 9 with a conserved proximal localisation and 9 with a conserved distal localisation. To test if their asymmetric position was dependent on the proximal and distal docking complex, we used a combinatorial protein tagging and gene deletion strategy. This showed that the localisation of 3/9 proximal and 4/9 distal are docking complex-dependant, indicating that there are at least two mechanisms generating asymmetry in the flagellum. In deletion mutants of each proximal/distal-specific protein, electron microscopy showed only very small changes in electron density. Therefore, to address whether asymmetries involved the outer dynein arms we tested if proximal/distal-specific proteins were dependent on the outer dynein arm motor proteins. This showed that 4 proteins are outer dynein arm-associated in some way. Our work shows that there is complex proximal/distal adaptation of axoneme molecular composition through at least two mechanisms, and particularly involving the outer dynein arms. In the future, we will test our panel of mutants for defects in swimming and flagellum beating to fully map, genome-wide, the contribution of proximal/distal-specific flagellar proteins to flagellum beat control.