*Schistosoma mansoni* is a blood fluke species responsible for schistosomiasis. Human infection is currently treated by a single drug, praziquantel. While praziquantel's mechanism of action has recently been revealed to operate through a transient receptor potential melastatin ion channel, drug resistant fears remain and fuel the search for alternative chemotherapies. As basic investigations of *S. mansoni* biology and biological processes could reveal vulnerabilities suitable for drug discovery, we present a functional genomics characterisation of two *S. mansoni* gene products putatively involved in energy metabolism. One gene product (SmFHII) is homologous to human class II fumarase while the second (SmFHI) is more closely related to the distinct class I fumarase commonly associated with prokaryotes. These enzymes are active in the mitochondria (TCA cycle), cytosol (urea cycle) and nucleus (DNA-damage repair). BLASTp analyses of SmFHI and SmFHII homologues revealed that only the lophotrochozoans possess a class I fumarase, while class II fumarases can be found in all other representative metazoan groups examined. The predicted localisation of SmFHI and SmFHII were determined using TargetP and revealed that SmFHI is predicted to possess a mitochondrial targeting peptide and likely responsible for mitochondrial activity. Relative expression of these genes across the human infective lifecycle stages was determined by qRT-PCR and showed that both genes are highly expressed within this crucial part of the lifecycle. Finally, RNA interference (RNAi) studies revealed that knockdown of both genes (*SmFHI* AND *SmFHII*) led to a defect in adult worm motility; single knockdown of *SmFHI* OR *SmFHII* did not significantly reduce this phenotype. These results suggest that *S. mansoni* fumarases can perform compensatory functions/activities and that these (e.g. respiration, DNA-damage repair or urea cycle) are involved in adult worm motility. Total oxygen consumption in RNAi treated worm cultures is currently being measured to quantify how loss of fumarase function affects respiration. RNAi of *SmfhI* and *SmfhII* will be performed on schistosomula to determine how these genes affect *in vitro* development, phenotype and motility. Additionally, the use of known fumarase class II inhibitor 2,3-Dicarboxyaziridine in tandem with predicted class I inhibitors are to be tested on adult worms and schistosomula. Together, these results will reveal the importance of SmFHII and SmFHI to schistosome viability, respiration and development.