**CHARACTERISING NICOTINIC ACETYLCHOLINE RECEPTORS IN THE PLANT PARASITIC NEMATODE GLOBODERA PALLIDA**

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Plant parasitic nematodes (PPN) cause $125b crop damage annually and with stricter regulation of pesticides they are posing an increasing threat to food security.

*G. pallida* is a cyst nematode; infective juvenile worms locate and invade host potato roots within a short time period while the environmental conditions are optimal. Therefore, locomotion is an essential component of its invasion strategy. It belongs to the phylum Nematoda, which also includes the free-living model genetic nematode *C. elegans*. We explored the idea that the neurobiological basis of *C. elegans* locomotion is likely to be conserved between nematodes and provide a route to new molecular targets for pest control.

Acetylcholine (ACh) is the excitatory neurotransmitter at *C. elegans* body wall neuromuscular junction, acting on nicotinic ACh receptors (nAChRs) to control muscle contraction and locomotion. The pesticide aldicarb, which blocks cholinesterase, causes spastic paralysis in *C. elegans* through chronic elevation of synaptic ACh. This compound also immobilises *G. pallida* consistent with a role for ACh in regulating its motility. Similarly, nicotine is effective against both *C. elegans* and *G. pallida*. However, levamisole, a nAChR agonist which is highly effective at paralysing *C. elegans*, is ineffective against *G. pallida*. In *C. elegans* the levamisole sensitive receptor is comprised of *unc-38*, *unc-63*, *lev-8*, *lev-1* and *unc-29* subunits*.* Thus *C. elegans* mutants for *unc-38* have uncoordinated locomotion and are highly resistant to levamisole. The newly available genome sequence for *G. pallida* identified homologues to *unc-38*, *unc-63* and *unc-29* subunits. Here we have focused on providing insight into the functional role of *G. pallida* *unc-38* by using transgenic *C. elegans* as a platform for expression. Thus we expressed either *C. elegans* or *G. pallida unc-38* in body wall muscle of *unc-38(x20)* mutant *C. elegans*. Both constructs rescued the motility of the mutant suggesting *G. pallida* UNC-38 is functionally equivalent to *C. elegans* UNC-38. However, the *G. pallida unc-38* expressing worms were not sensitive to levamisole. Currently we are investigating the structure-activity relationship of UNC-38 with respect to levamisole sensitivity.

Acknowledgements: Funded by the BBSRC Grant no. BB/J006890/1.