REPORT ON THE 38th ANNUAL DROSOPHILA RESEARCH CONFERENCE, March1998

Sarah Goulding

Postgraduate student with Dr. A. Jarman

Funding to attented this meeting was kindly provided by the James Rennie Bequest and The Small Project Grants Trust

Over one thousand *Drosophila* researchers came to Washington to attend this years meeting. The Edinburgh fly community was represented by three labs, those of Prof. Mary Bownes, Dr. Ilan Davis and that of my supervisor Dr. Andrew Jarman.

The four day conference schedule was overwhelming: presentations were given from 9 am until 10.30 p.m. every day. The meeting was opened with a lecture by Mel Green that focused on the historical perspectives of the field. Two plenary sessions were held, one at the beginning and one which closed the meeting; during these invited speakers talked about their labs' research. Talks by Ross Cagan on eye development and Iswar Hariharan on cyclin-dependent kinase inhibitors had particular relevance to our own work in the lab.

The number of participants and diversity of topics meant that talks had to be grouped under main areas of research and these sessions were run concurrently. Each ten minute presentation was rigorously timed by the chair to allow people to move to different sessions. I quickly learned that coffee breaks were an essential part of the day.

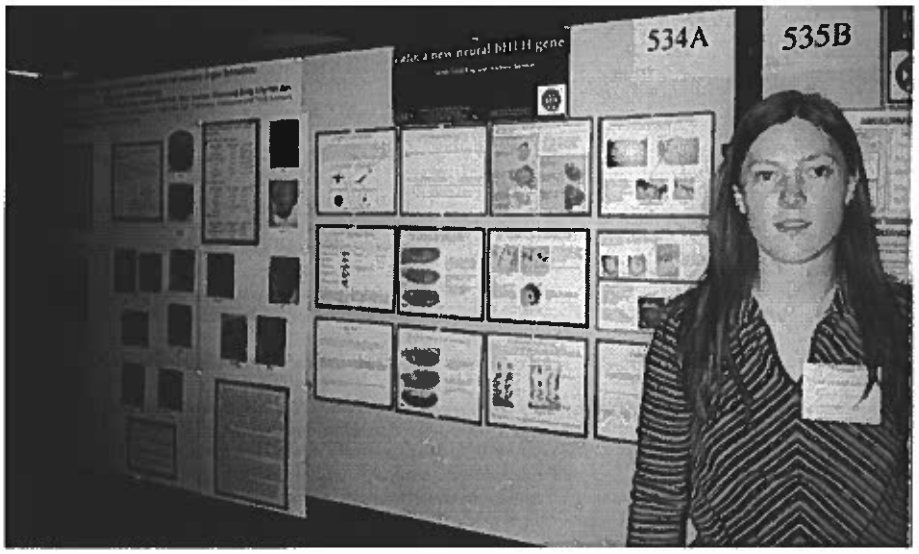
During coffee breaks, in between sessions and after meals in the evenings people convened in the poster exhibition hall. Authors were given specific times to be beside their own work. I presented a poster (one of over six hundred) entitled **'*cato*: a new neural bHLH gene'** which described my postgraduate work (see attached abstract and photograph). This was definitely the most rewarding part of the meeting. My poster was well received and those that spoke to me about it gave me encouraging feedback and ideas for future experiments.

While the meeting was too large to really 'network' with the more famous researchers I was able to meet up with old friends who had gone into similar areas of research and meet new people through them. I also discused our lab's work with colleagues of my supervisor.

In summary the four days presented a challenge in trying to absorb as much as possible from the masses of research that was presented and I enjoyed giving a poster about my own studies. I benefited greatly from

attending this meeting, not only in terms of the experience of presenting my own work, but also learning from others and making contacts for work and the future. I would like to thank the James Rennie Bequest and the Small Project Grants Trust for giving me their financial support.

POSTER ABSTRACT :*cato* a new neural bHLH gene



atonal and members of the achaete-scute complex (AS-C) are proneural genes of *Drosophila melanogaster*. They encode nuclear proteins with a basic helix-loop-helix (bHLH) motif, indicative of their function as transcriptional regulators.

I have identified new genes by degenerate PCR which contain bHLH domains with closest similarity to atonal. RNA in situ experiments have shown that these genes are expressed at times consistent with their having a function in both larval and adult peripheral nervous system (PNS) development.

One of these genes, *cato* (for cousin of atonal) shows a non-proneural pattern of RNA expression. Instead, this pattern is more similar to that of asense, a pan-neural gene: the RNA is seen in sense organ precursor cells (SOPs) and their daughters. Early expression in the embryo is seen in cells that give rise to chordotonal (ch) organs. A later more complex pattern emerges that also includes external sense (es) organ precursors. This two-phase expression pattern is also reflected during adult PNS development. Some phenotype analysis has been carried out using the antibody MAb22C10 which stains the PNS neurons and their dendrites in stage 16 embryos.

Specific cells appear to be affected in embryos of deficiency stocks for regions that uncover *cato*: the v'ch1 neuron is often missing, or twinned. The lch5 cluster and vch neurons are misaligned in some segments.

Preliminary misexpression data indicate that *cato*has a role during sense organ formation at a later stage of neurogenesis than atonal. Our present model of *cato* function is that it is required for sense organ cell fate and/or differentiation.