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DISSERTATION SUMMARY

Isolation of new planar polarity mutants in *Drosophila melanogaster*

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Polarity is a fundamental atribute of living organisms and cells. One of the most common form is the apical-basal polarity. Many cells are, however also polarized within the plane of the tissue, and thus this type of polarity is called planar cell polarity (PCP) or tissue polarity. In *Drosophila* all adult cuticular structures are polarized within the plane whereas in vertebrates a similar polarity is seen in the arrangement of fish scales, bird feathers, hair of mammals, the orientation of cilia in the oviduct or the stereociliar bundles in the neurosenory epithelium of the inner ear (Fanto McNeill 2004).

Despite the grate progress that has been made in vertebrates during the past few years, Drosophila system remained the best studied example of PCP. PCP in flies is most evident in the wing, which is covered by uniformly polarized, distally pointing hairs, in the epidermis, where sensory bristles point to the posterior, and in the eye, where PCP results in a mirror symmetry arrangement of ommatidia. Polarization in these tissues is controlled by the PCP gene products, mutants of which gene impair planar organization. Some of the PCP gene which have been placed into the core group, appear to affect polarity in all of the tissues. The key members of the core group are frizzled (fz), strabismus (stbm)-Van Gogh (Vang), dishevelled (dsh), flamingo (fmi), also known as starry night (stan), diego (dgo) and prickle-spiny legs (pk-sple).

In addition to the core genes, several other genes, like fuzzy, inturned, fritz, multiple wing hair, roulette, nemo, RhoA, mishapen and jun have been classified as secondary polarity genes becouse their function is required in only a subset of tissues (Adler and Lee 2001). These genes are thought to function as effectors of the core genes.

Taken together, a very simple model of *Drosophila* PCP establishment can be drawn: the products of the core genes, in response to an as yet not precisely identified polarity signal, build up a signalling center that controls polarity through tissue specific effectors. While a lot has been learned about the core genes, many important questions remain. How do they interact at the molecular level? How is this pathway connected to others? What are the effector genes and how can they control coordinated physical changes in cell behaviour?

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In order to contribute to the answering of above mentioned questions by isolating new tissue polarity genes, we initiated a large scale mutagenesis screen for the left (2L) and right arm (2R) of the second chromosome and the right (3R) arm of the third chromosome of *Drosophila melanogaster*. We used the FRT/Flp mosaic system (Xu and Rubin 1993) which is employed for the first time to identify polarity mutants. To encrease the sensitivity of the screen, we performed an F_2 screen which offers the advantage of recognizing phenotypes with low penetrance and eliminates the sterility problems of F_1 screens. As a mutagene we used EMS in 30 mM concentration and ENU in 1,6 mM concentration.

After the mutagenesis of the 2R, 2L and 3R and screening of about 22,800 crosses, we found 58 strong and many weeker PCP mutants. These mutants showed a random orientation of bristles on notum and hairs on wings, multiple wing hairs, misrotated and symmertrical ommatidia and chirality flips in eyes.

In order to map our mutants and to test whether they are carrying new genes we initiated complementation analysis. The complementation analysis has shown that on the 2L we isolated 18 fmi, 1 stbm, 1 pk and 3 new alleles, on the 2L 1 ds, 4 frtz and 3 new alleles, and on the 3R 27 new alleles. The crosscomplementation of these alleles revealed that the new alleles on the 2L and 2R are unique, while on the 3R we could distinguish one complementation group with 5, one with 2 members and the others were unique. We roughly mapped our mutants based on lethality with deficiencies and the most interesting mutants were also mapped by recombination to determine whether the mutation and lethality are correlated. In order to determine if our mutants can be members of the PCP pathways, we examined their genetical interaction in eye with the two well characterized PCP genes dsh and fz. We have found that most of our mutant enhances or suppresses these genes, so they can be fitted in PCP pathway at different levels.

References

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