The molecular basis of metabolic syndrome: studies in zebrafish

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Propositions accompanying the dissertation

The molecular basis of metabolic syndrome

Studies in zebrafish

1. The SHP1/2 inhibitor NSC-87877 is able to reverse a diabetic phenotype in zebrafish larvae. *(This thesis, Chapters 2 and 4)*

2. Disruption of the *lepb* gene influences mortality and bacterial burden in infected zebrafish. *(This thesis, Chapter 3)*

3. The PTPN6 protein modulates the insulin and leptin signaling pathways. *(This thesis, Chapter 4)*

4. Treatment with glucocorticoids prevents the metabolic alterations that occur in wasting syndrome during mycobacterial infection. *(This thesis, Chapter 5)*

5. Leptin orthologues have a very similar structure in all vertebrates and thus display functional similarities in different species. *(Adapted from Londraville et al., 2014)*

6. The PTPN6 protein is a mediator between hyperinsulinemia-derived insulin resistance and immune suppression. *(Adapted from Marín-Juez et al., 2014)*

7. It is surprising that although leptin is effective for reversing leptin deficiency-induced obesity and lipodystrophy, it is not yet used to treat typical obesity *(Adapted from Farr et al., 2015)*

8. The mechanism of action of adjunctive glucocorticoid therapy, which induces faster clearance of bacteria and reduces morbidity and mortality in patients with different forms of TB, is still ill-defined *(Adapted from Tobin et al., 2012).*

9. Although fish are an excellent experimental model for diabetes research, it is not known whether they develop diabetes under natural conditions.

10. The next pandemic will be caused by antibiotic-resistant bacteria.

11. Doing a PhD research project means that you know that you actually do not know.

12. In research, the biggest limitation is our own imagination.

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