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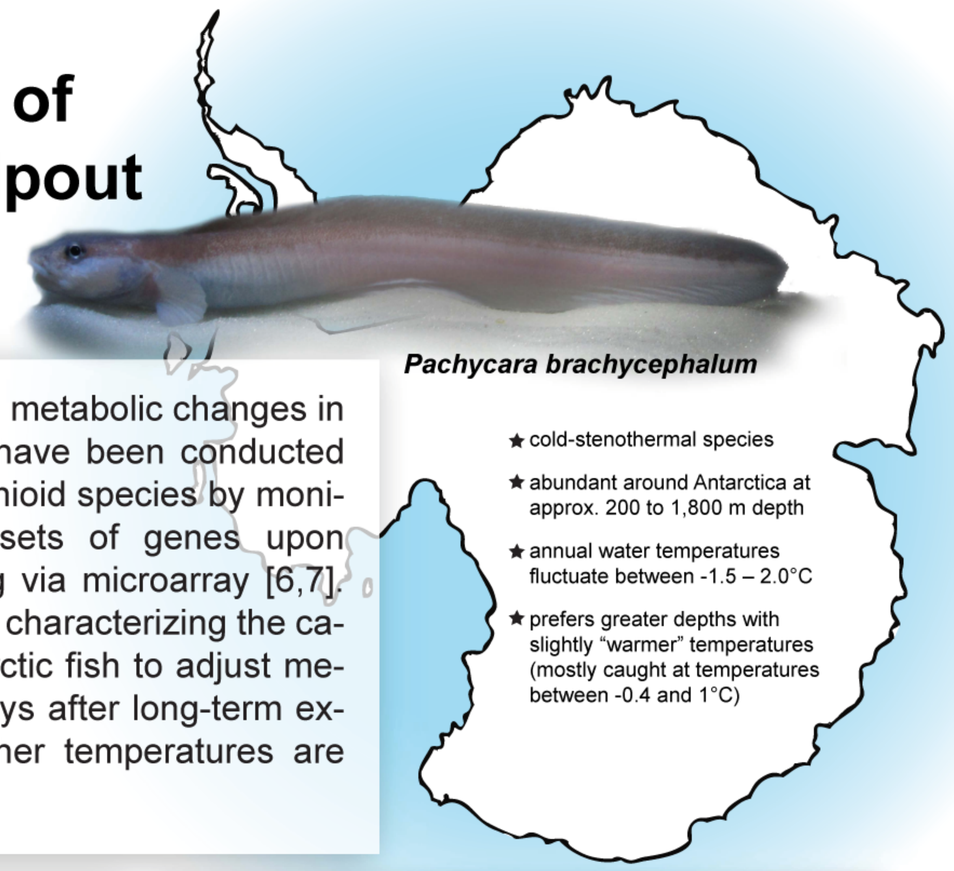
Linking transcriptomics to physiology - Effects of chronic thermal acclimation in the Antarctic eelpout (*Pachycara brachycephalum*)

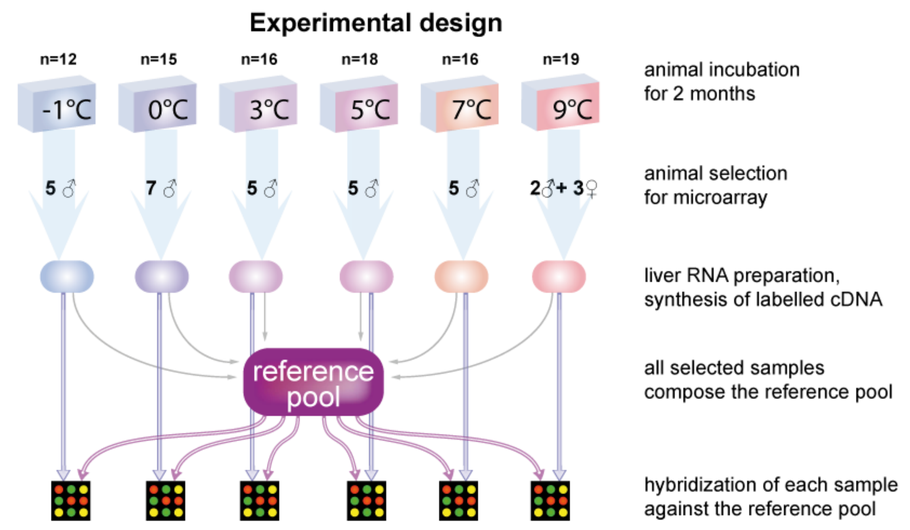


Introduction

Thermal gradients account for the biogeographic distribution of species along latitudinal clines, as well we across the costal and tidal zones [1]. Thus, species-specific thermal tolerances are limiting and correlate with natural habitat temperature variations [2].
Due to the local influence of global climate change, highly adapted stenothermal organisms currently experience rising water temperatures around the Antarctic Peninsula [3,4]. Predictive models indicate that the warming trend is especially strong in polar regions [5]. The capacity for acclimation may therefore become exceedingly critical for species survival in those regions.
To our knowledge, surveys investigating detailed metabolic changes in Antarctic fish have been conducted in two notothenioid species by monitoring broad sets of genes upon acute warming via microarray [6,7]. However, data characterizing the capacity of Antarctic fish to adjust metabolic pathways after long-term exposure to higher temperatures are still missing.

Methods

A species-specific microarray comprized of 15.854 contigs of a normalized cDNA library of *P. brachycephalum* [8] was designed. cDNA samples prepared from liver RNA of animals kept for 2 months at different temperatures were analyzed by single hybridizations (n = min 5 per treatment) against a reference pool consisting of pooled cDNA from all treatments.



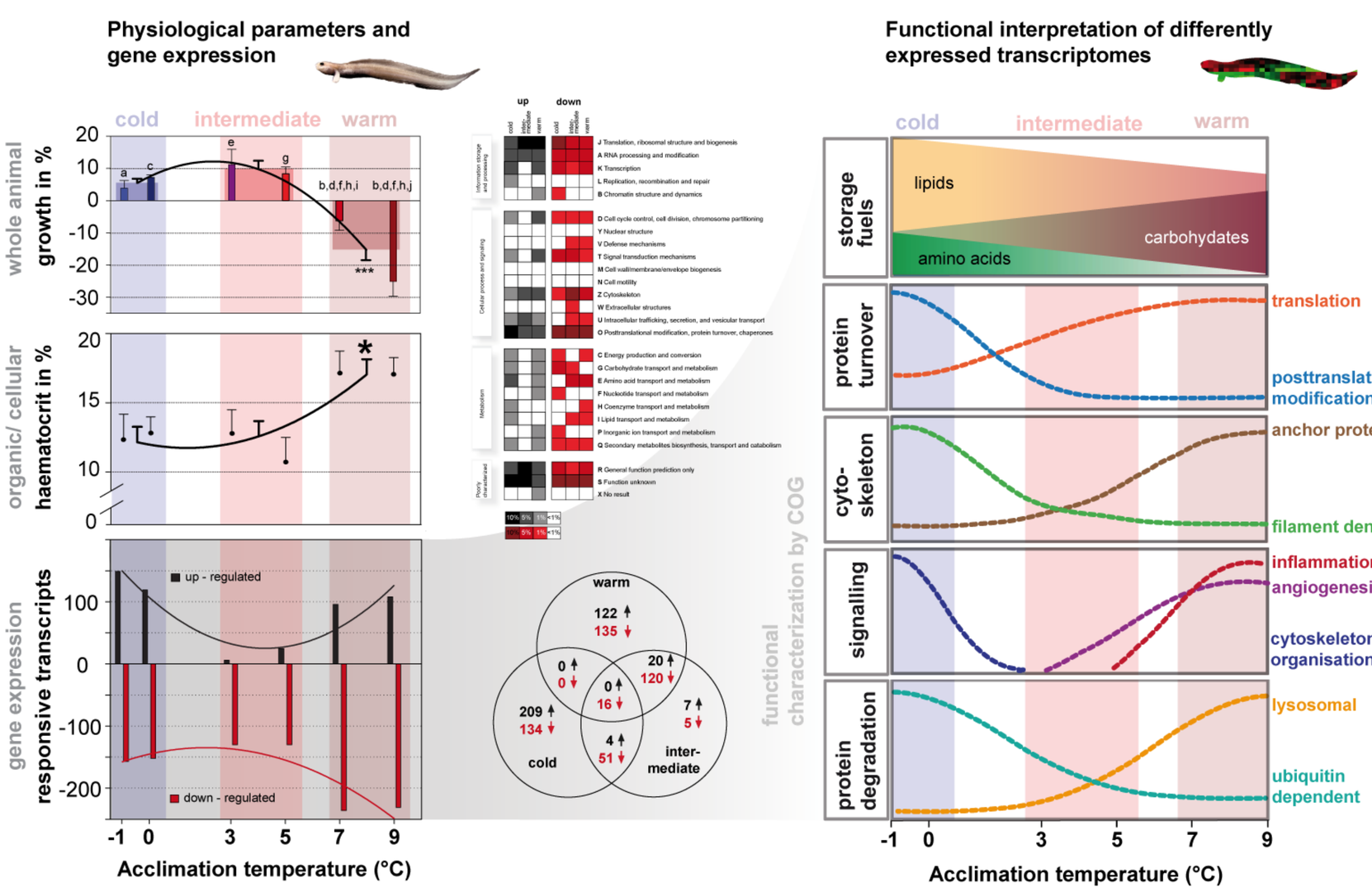
Objectives

1. Characterization of the thermal plasticity of *P. brachycephalum* on a broader molecular scale after chronic thermal exposure to differnt temperatures within its thermal window and beyond.

2. Linking the effects of thermal acclimation from the systemic and cellular level to the gene expression level to identify effective mechanisms defining climate sensitivity.

Results

Animal growth performance (GP) indicate a thermal optimum at 3°C; at 6°C the GP gets lost.
Above that temperature higher haematocrit levels indicate an onset of a temerature-dependent oxygen and capacity limitation of the thermal tolerance.
The magnitude of long-term altered transcripts correlates to the growth optimum by a minimum of altered transcript diversity. Affected metabolic pathways are involved in energy consumption and conversion. Different protein modification and degradation pathways become effective at different temperatures. The subcellular organization is characteristic at certain temperatures. Signalling pathways are largely involved in cellular maintenance and the balance between stress and homeostasis responses.



Conclusion

Chronic thermal exposure causes transcriptomic adjustments below and above the optimum temperature reflecting imbalances in homeostatic mechanisms, at the same time being paralleled by unfavourable shifts in energy budget. Some of the transcriptomic changes may be responses to such unfavourable shifts (e.g. the preferred usage of carbohydrate–based fuels with increasing temperatures to prepare for hypoxemic events), or be causative in stimulating baseline energy demands, e.g. by an enhanced protein turnover via ubiquitin-related enzymes in the cold. The expression profiles of housekeeping proteins suggest that large protein complexes become functionally impaired in the warmth, indicated by a seemingly futile expression of e.g. components of the transcription or the protein synthesis machinery. A maintenance at warm temperatures is only possible by using up body´s own energy reserves as well as through a restructuring of tissue (angiogenesis) for an efficient oxygen supply. Subsequently, thermal limitation largely depends on the degree of protein adaptation and the available functional repertoire of genes.

References

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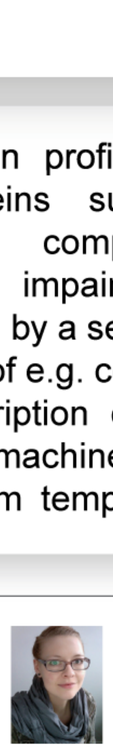

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