

Increased seawater temperatures cause temporal shifts in catabolic pathways of Antarctic krill *Euphausia superba*

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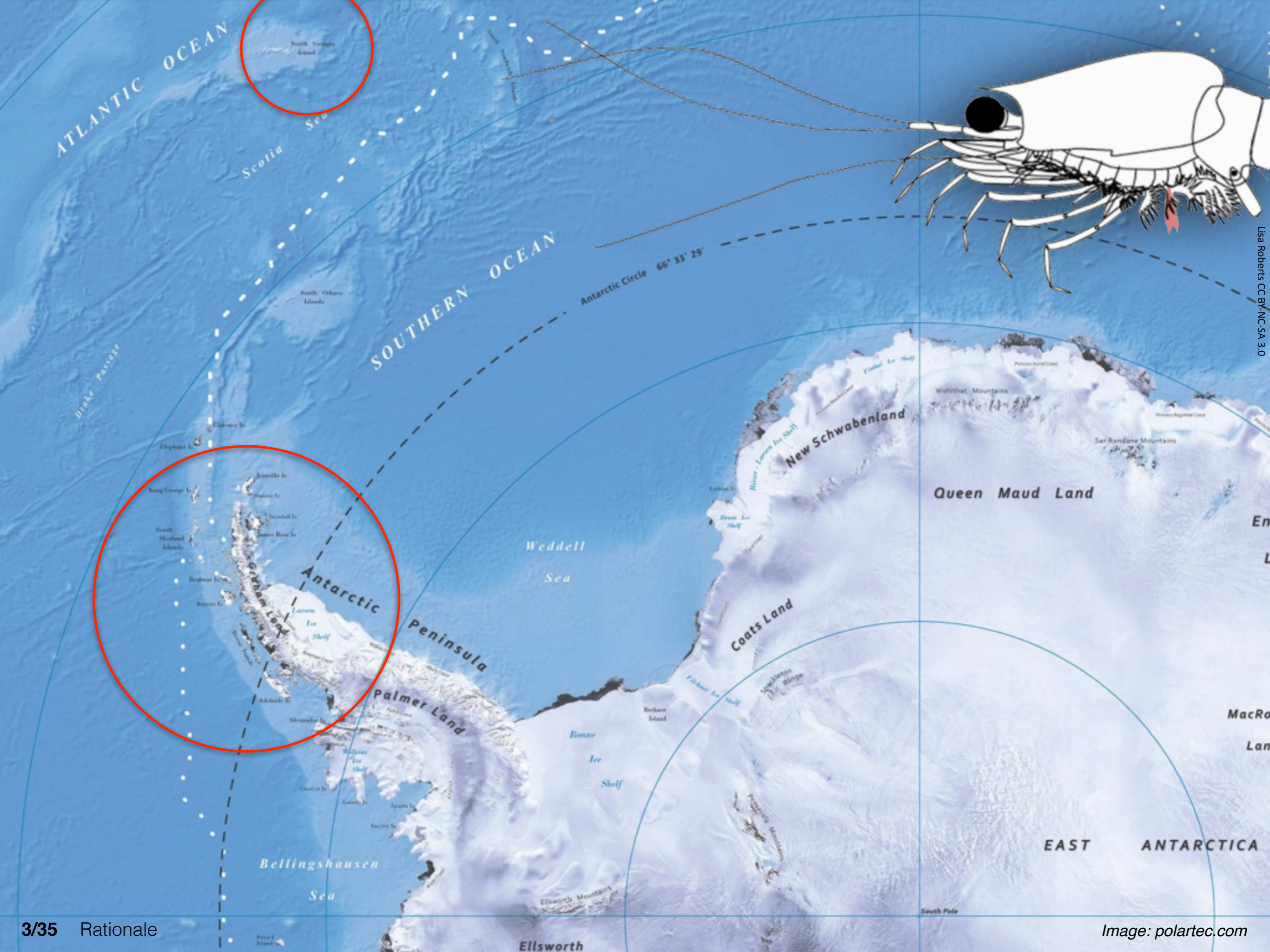
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Contents

- (Rationale - Why Krill?)
- Recap: Experiments at the AAD
- Results
 - Respiration
 - Enzyme Activities
 - Conceptual Energy Budget
- Conclusions & Outlook



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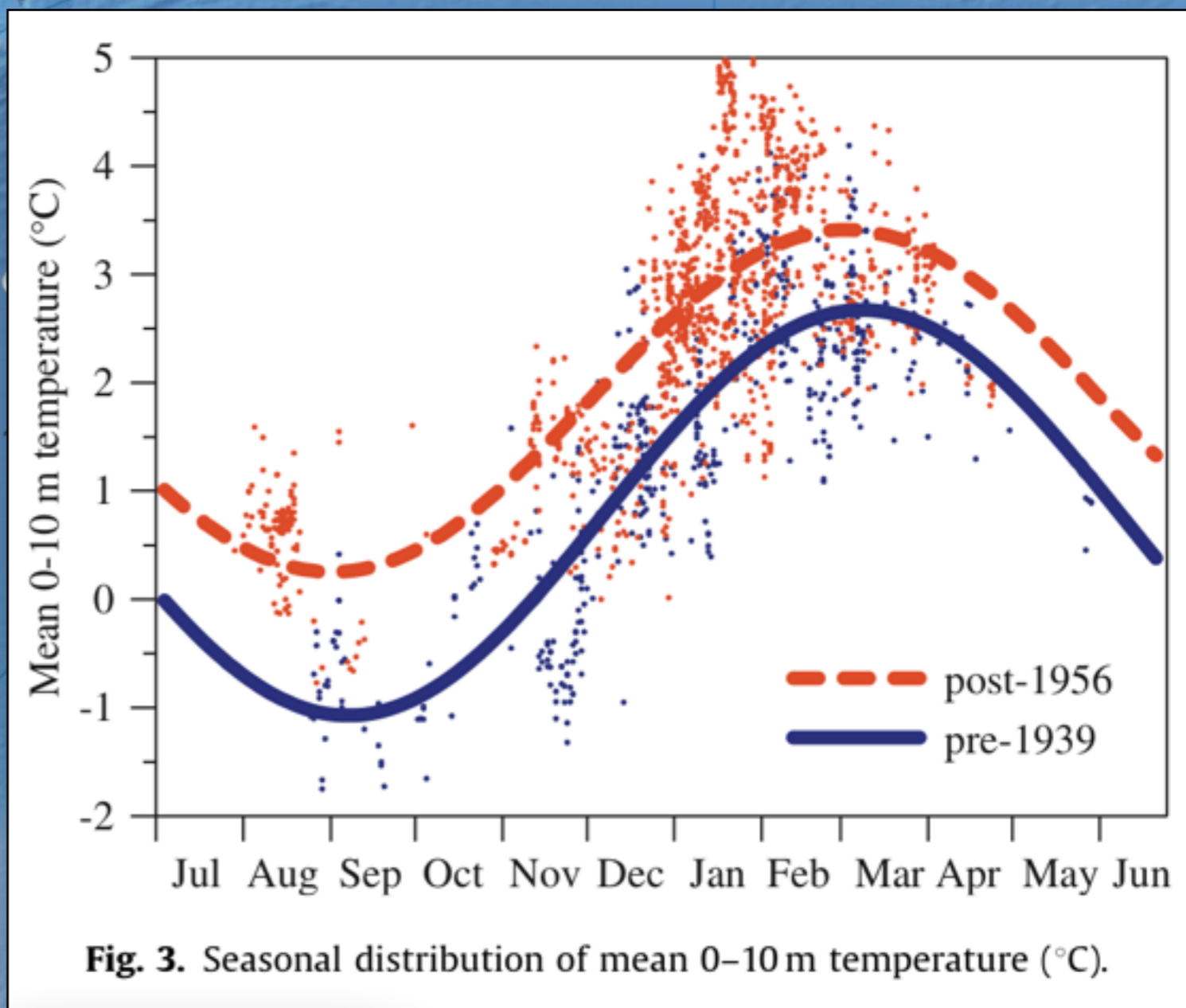
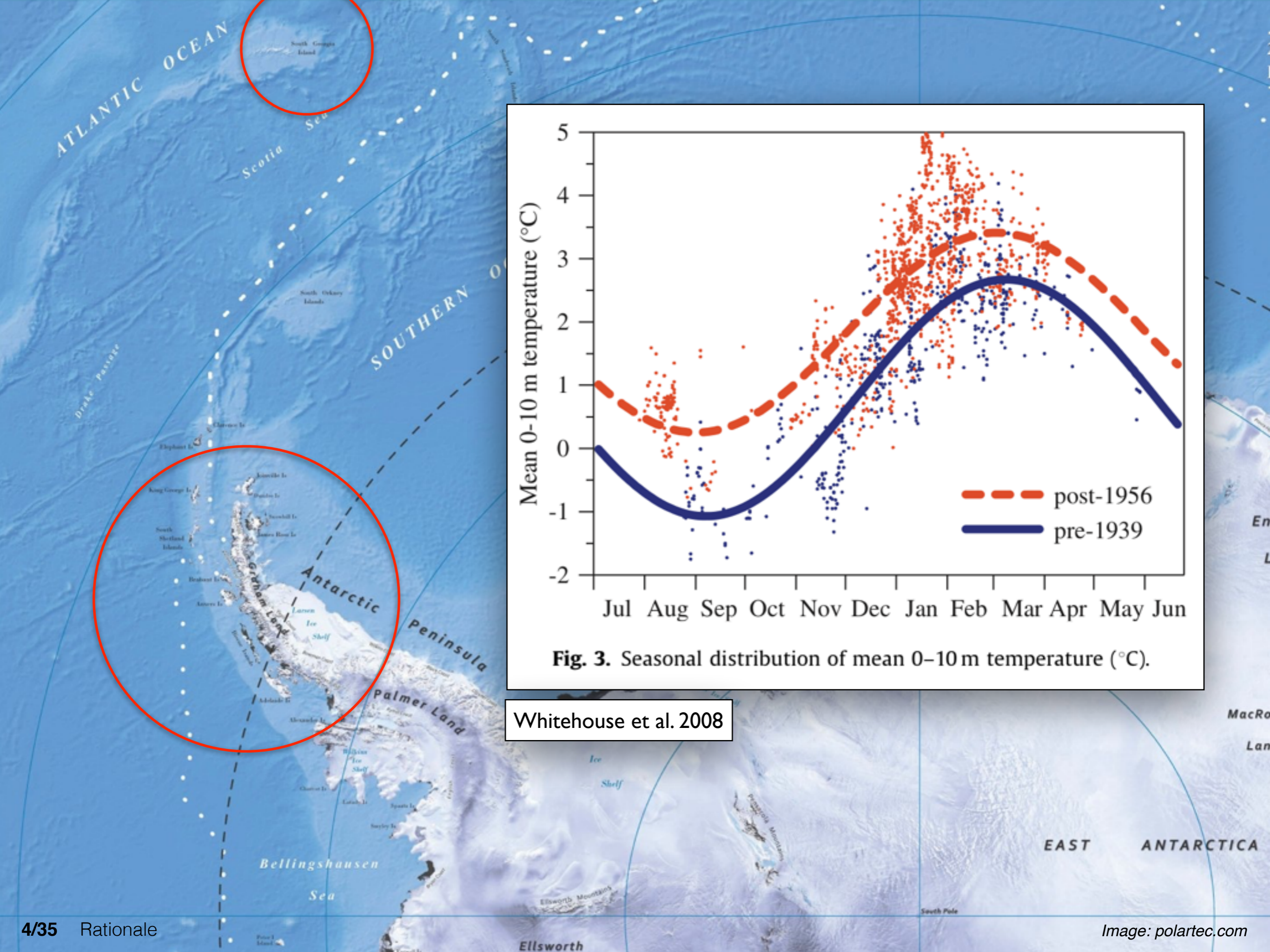


Fig. 3. Seasonal distribution of mean 0–10 m temperature (°C).

Whitehouse et al. 2008

Hypothesis


„Adult krill have a narrow temperature range of 0.5°C to 4°C for optimal growth and physiological functioning.“

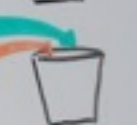


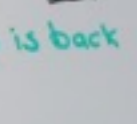
Weekends:
Flows off for 2 hours
As per weekdays, leave flows off for as long as possible

Tobi's Experiments
V2/V3 09/10 + V3 08/09 RTA

- Collect mounts + DIPs
- Preserve in 80% EtOH - record DIPs
- Check temp + flow
- Leave flows ON
- Prepare live algae / instant algae / Frippek for later feeding (random times)

8L Phaeo  500ml Frippek
20ml Thal
10ml Iso
10ml Pw

8L Pyram  500ml Frippek
20ml Thal
10ml Iso
10ml Pw

8 4L Gem  500ml Frippek
20ml Thal
10ml Iso
10ml Pw

↑ until Phaeo is back

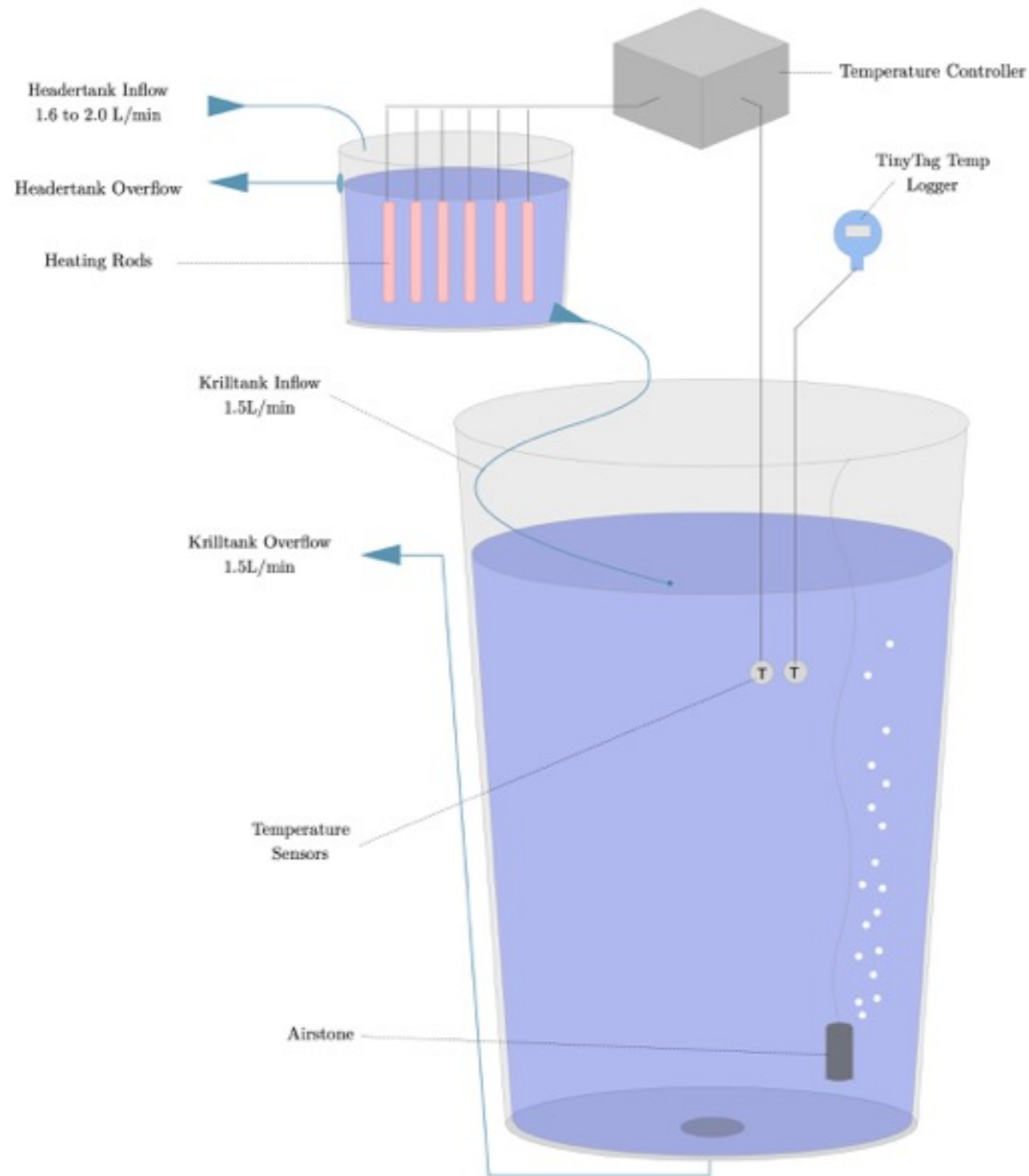
Tank B3
Refugees with Webcam
1L Phaeo
1L Pyram
1L Gem
200ml Frippek
2ml Thal
1ml Pw
1ml Iso

Exp A Tank 2
Exp B Tank 2
Exp B Tank 1
Exp A Tank 3

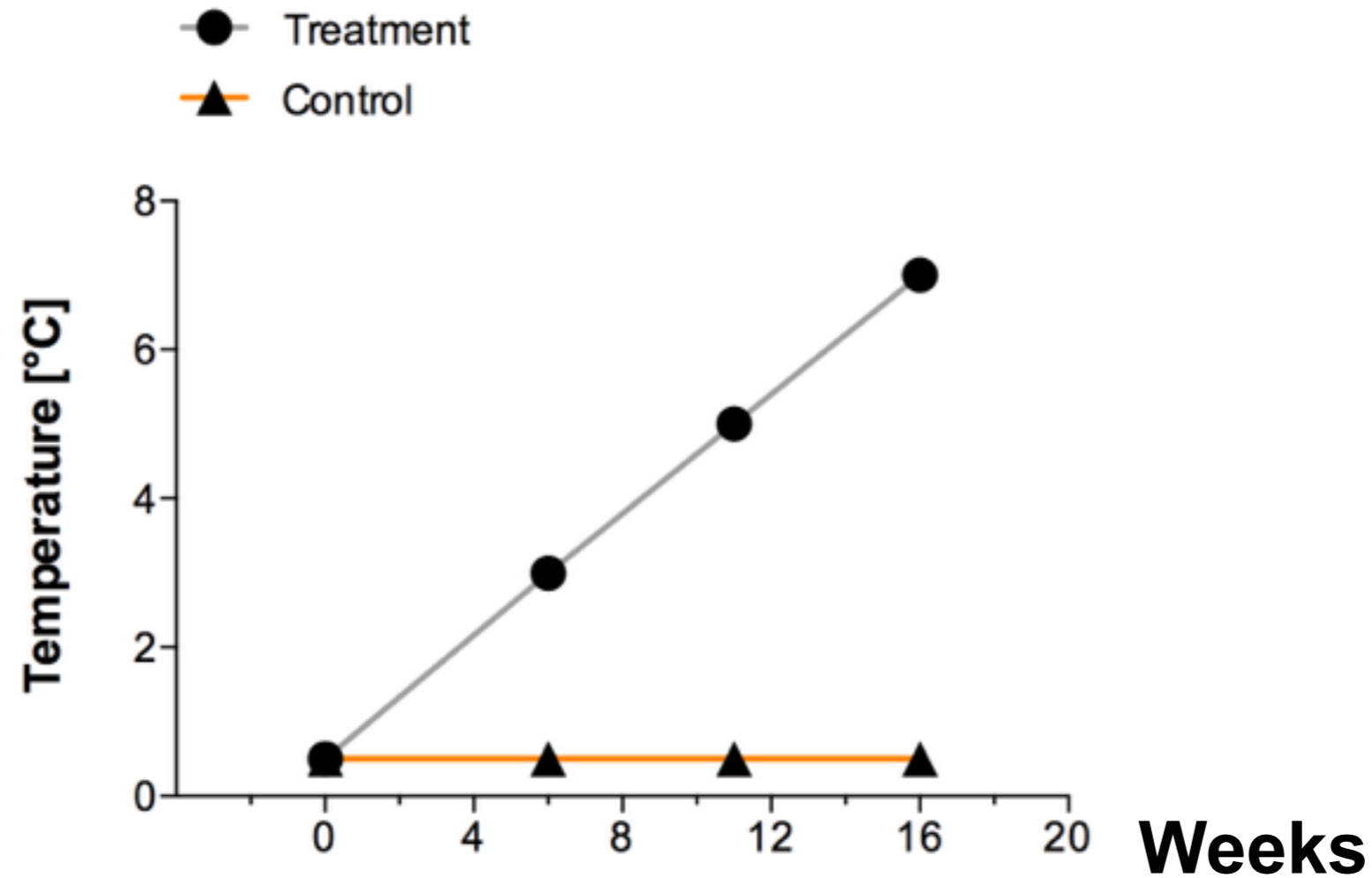




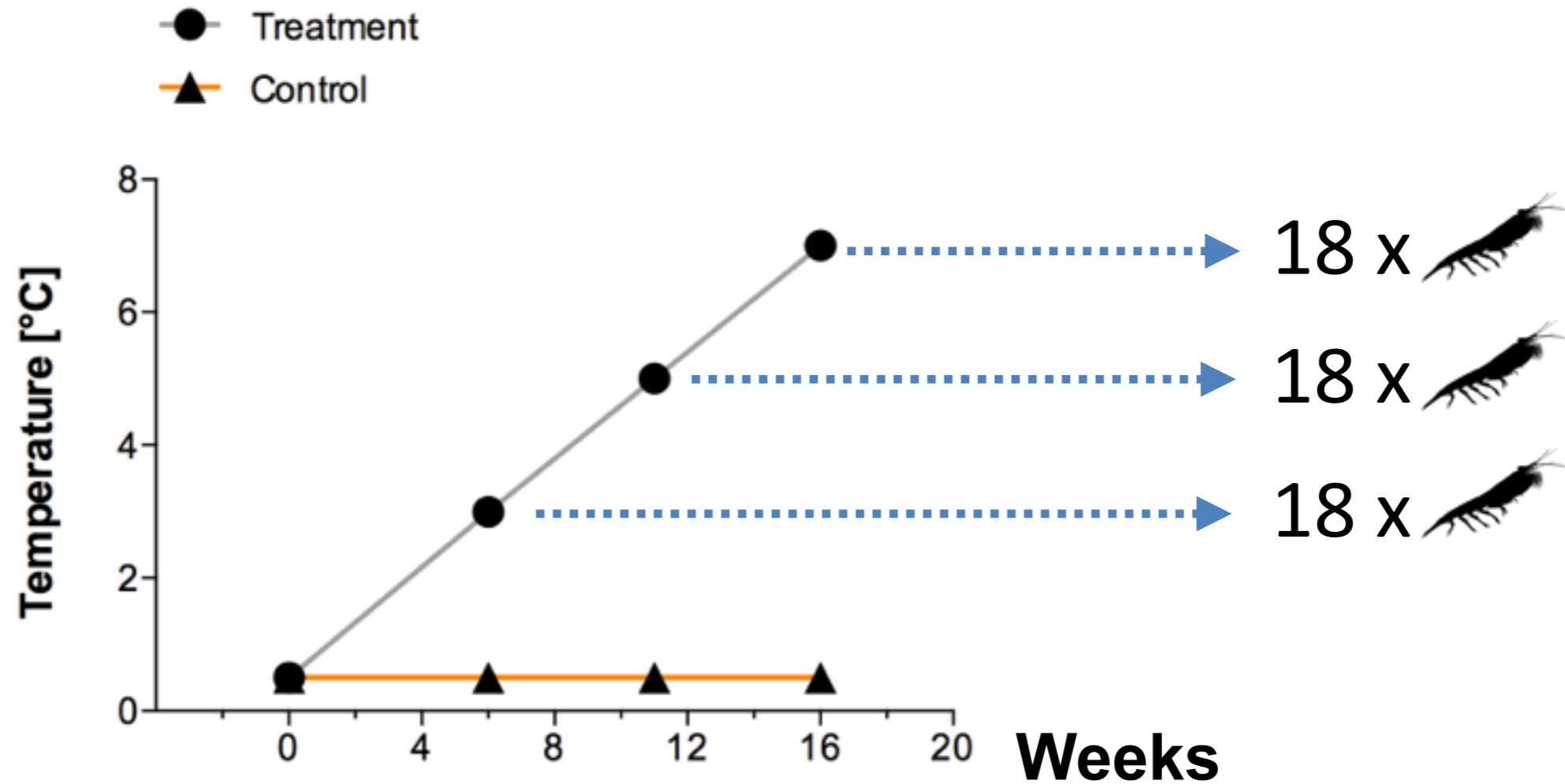
Experimental Setup



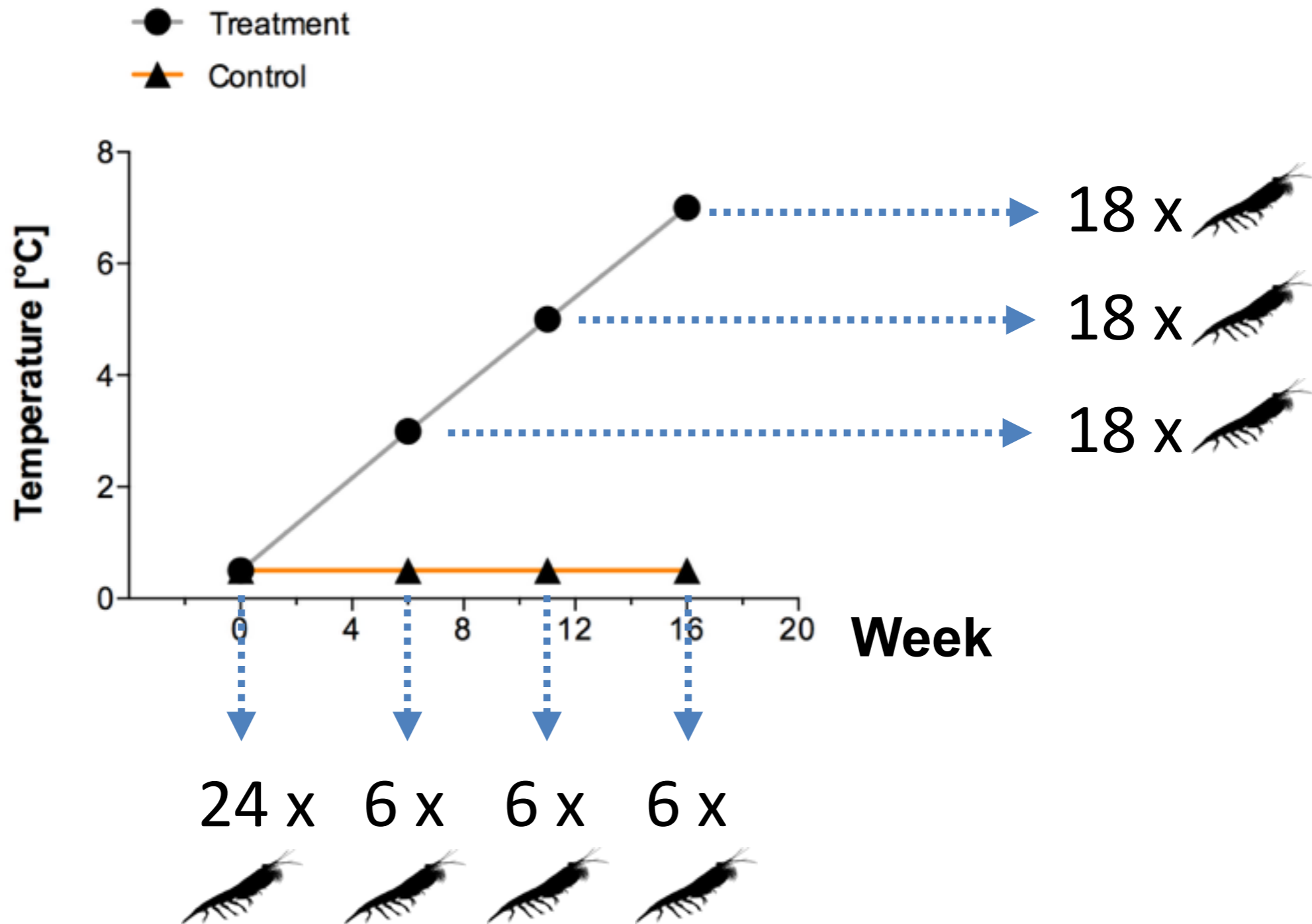
Sampling Scheme



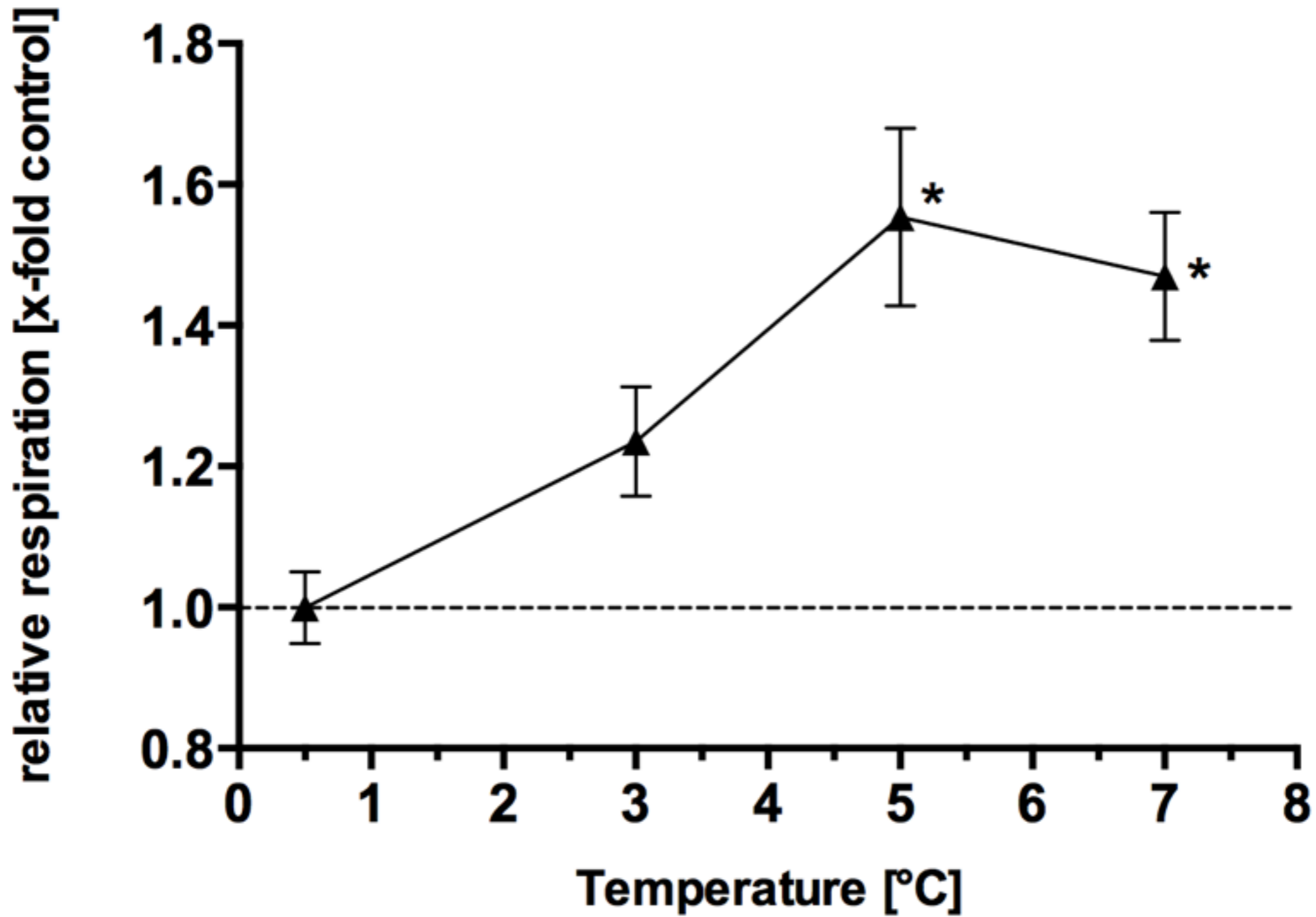
Sampling Scheme



Sampling Scheme



Respiration



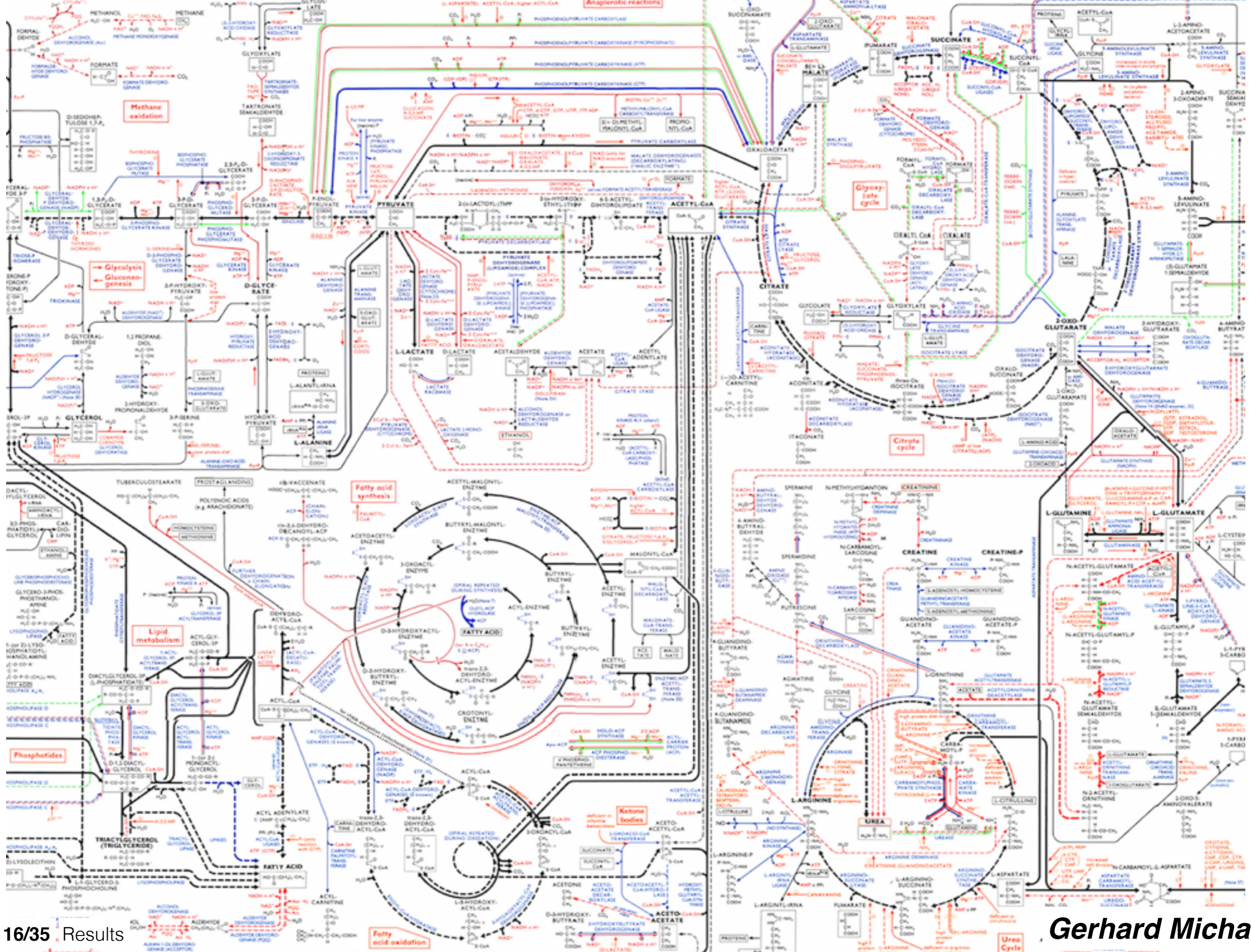
Respiration -> Energy Requirement

	Temperature [°C]	Equation from linear regression	Individual energy requirement [Joule/d]
Treatment	0.5	$y = 0.3117x$	1,12
	3	$y = 0.4021x$	1,45
	5	$y = 0.5903x$	2,13
	7	$y = 0.6268x$	2,26
Control	0.5	$y = 0.3544x$	1,28
	3	$y = 0.3753x$	1,35
	5	$y = 0.3587x$	1,29
	7	$y = 0.4155x$	1,50

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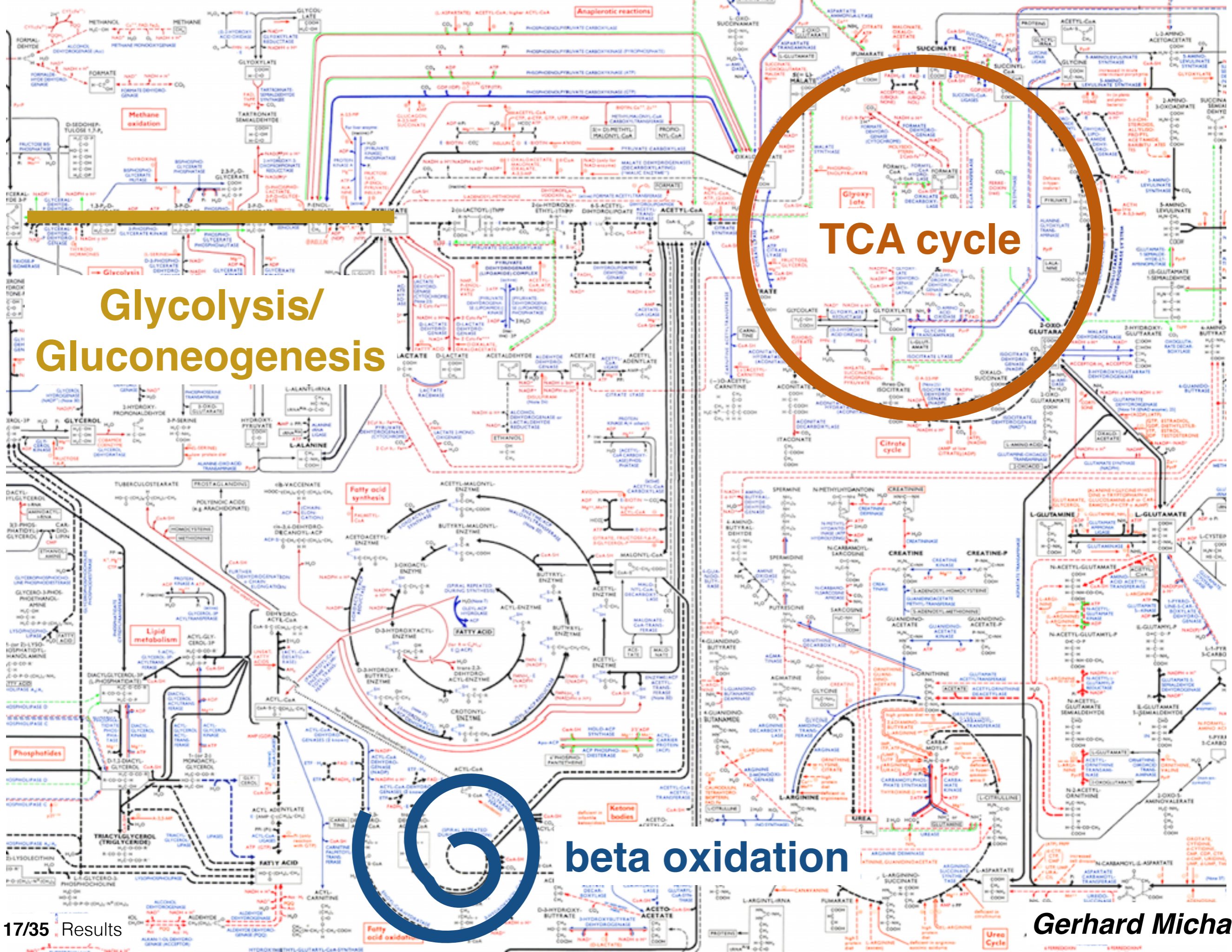
How are energy demands met?



Glycolysis/ Gluconeogenesis

TCA cycle

beta oxidation

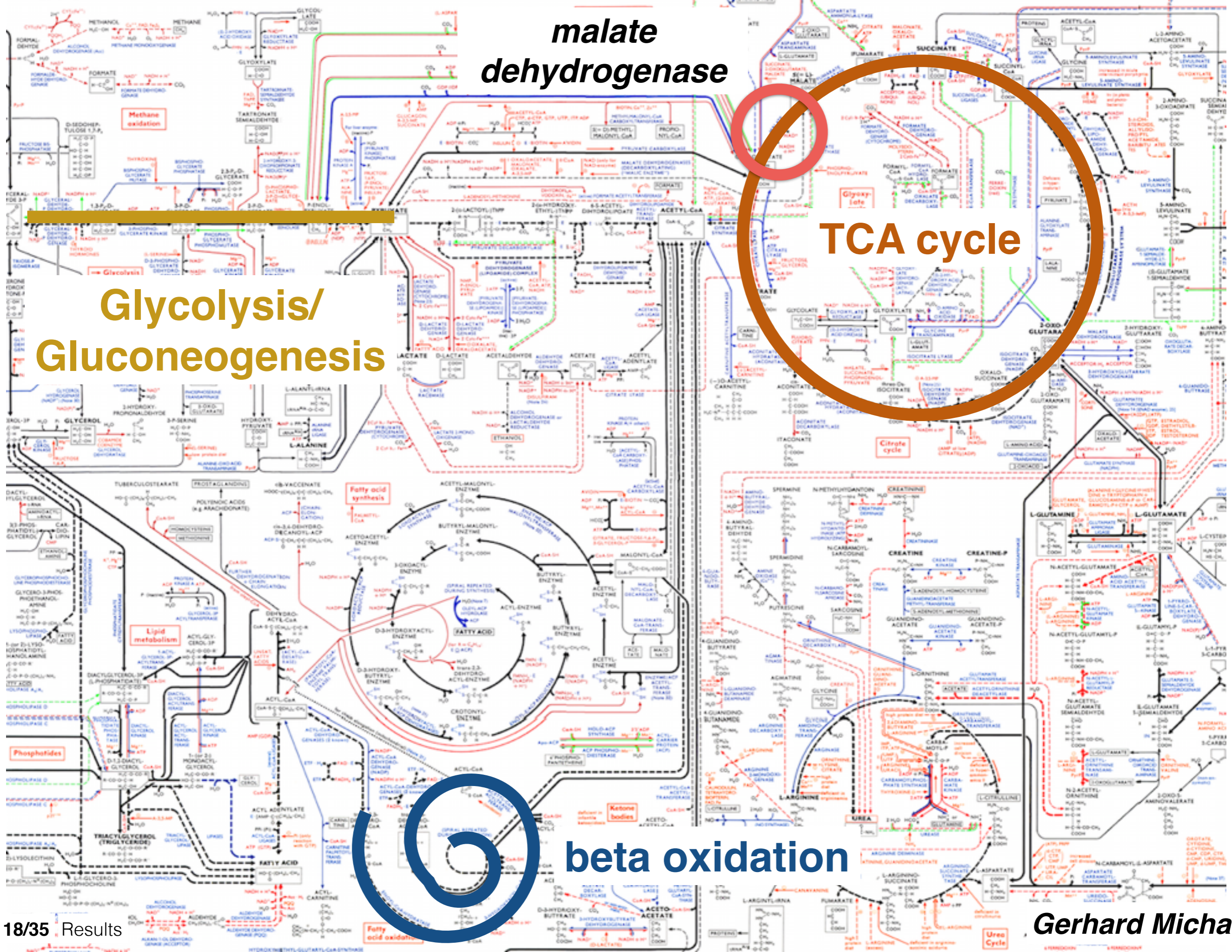


**malate
dehydrogenase**

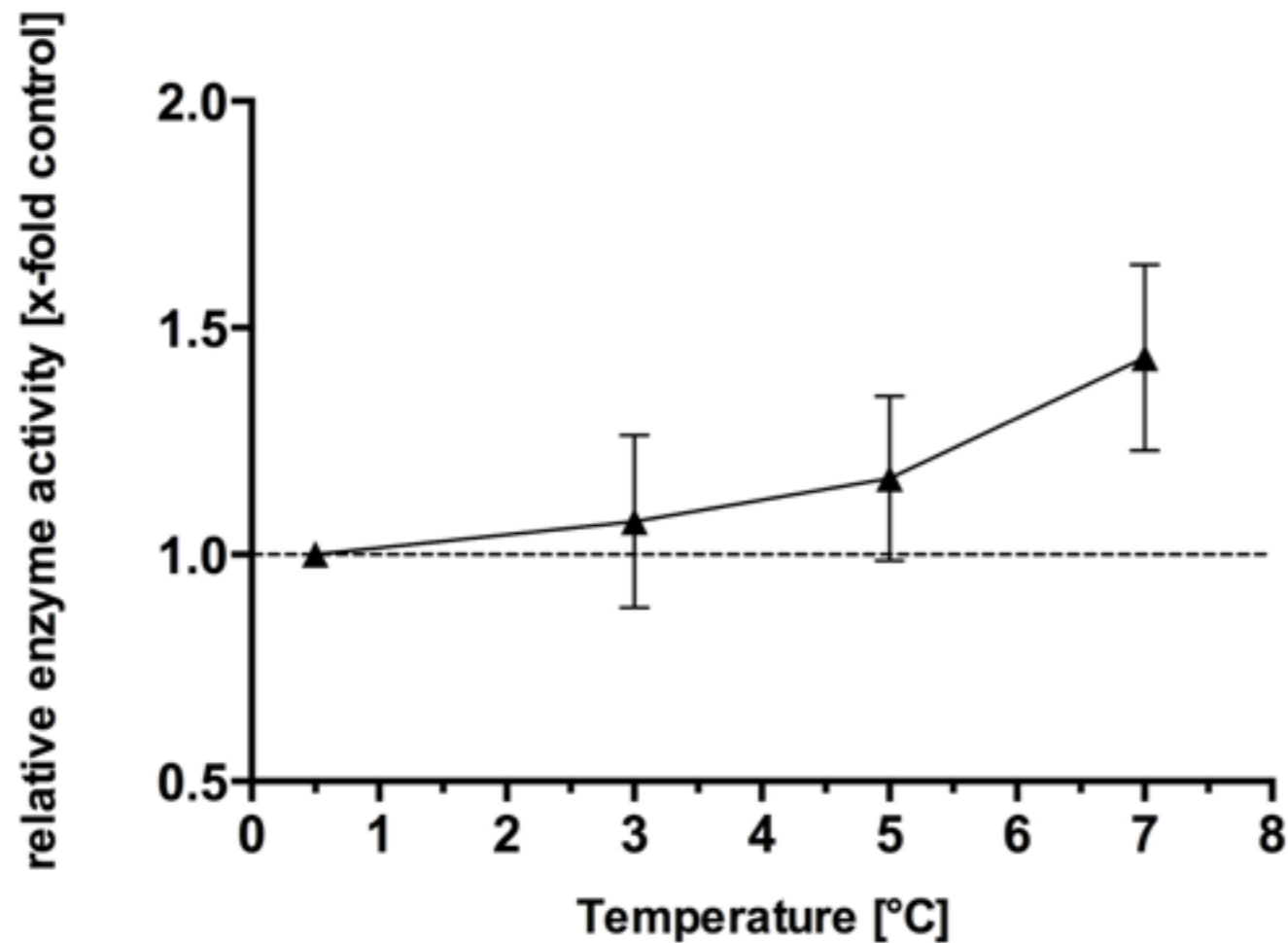
**Glycolysis/
Gluconeogenesis**

TCA cycle

beta oxidation



Malate Dehydrogenase MDH



- Key enzyme in TCA cycle, catalyzes oxidation of malate to oxaloacetate
- also involved in other pathways (shuttling of TCA intermediates to cytosol)
- mirrors respiration to some extent

**malate
dehydrogenase**

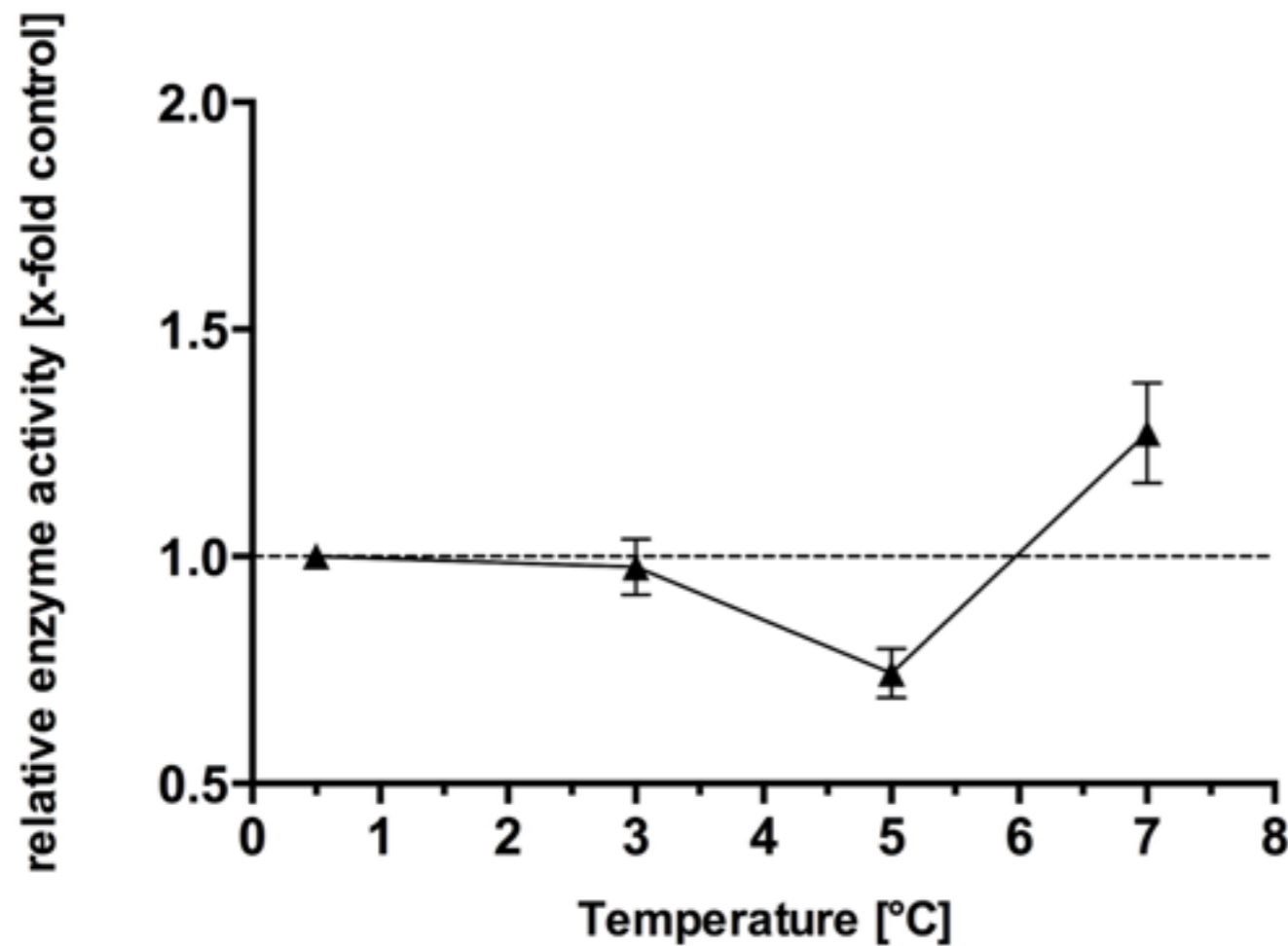
**Glycolysis/
Gluconeogenesis**

**citrate
synthase**

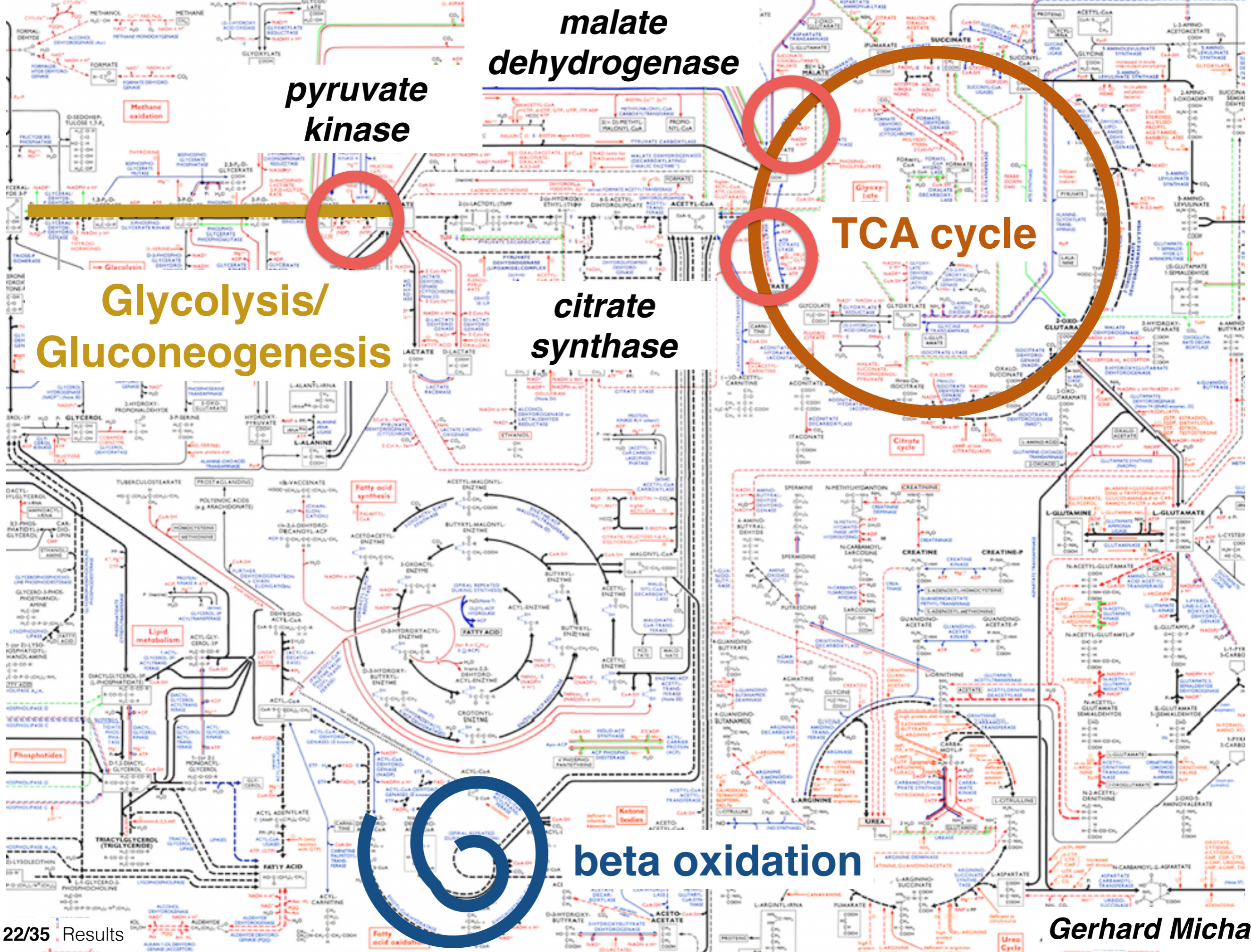
TCA cycle

beta oxidation

Citrate Synthase CS



- catalyzes first reaction in the cycle: condensation of the acetate residue from Acetyl CoA and one molecule oxaloacetate
- acts as central crossing point for various pathways
- balances oxidative and biosynthetic pathways
- entry point for fat synthesis (Acetyl-CoA shuttle to cytosol)



**Glycolysis/
Gluconeogenesis**

**pyruvate
kinase**

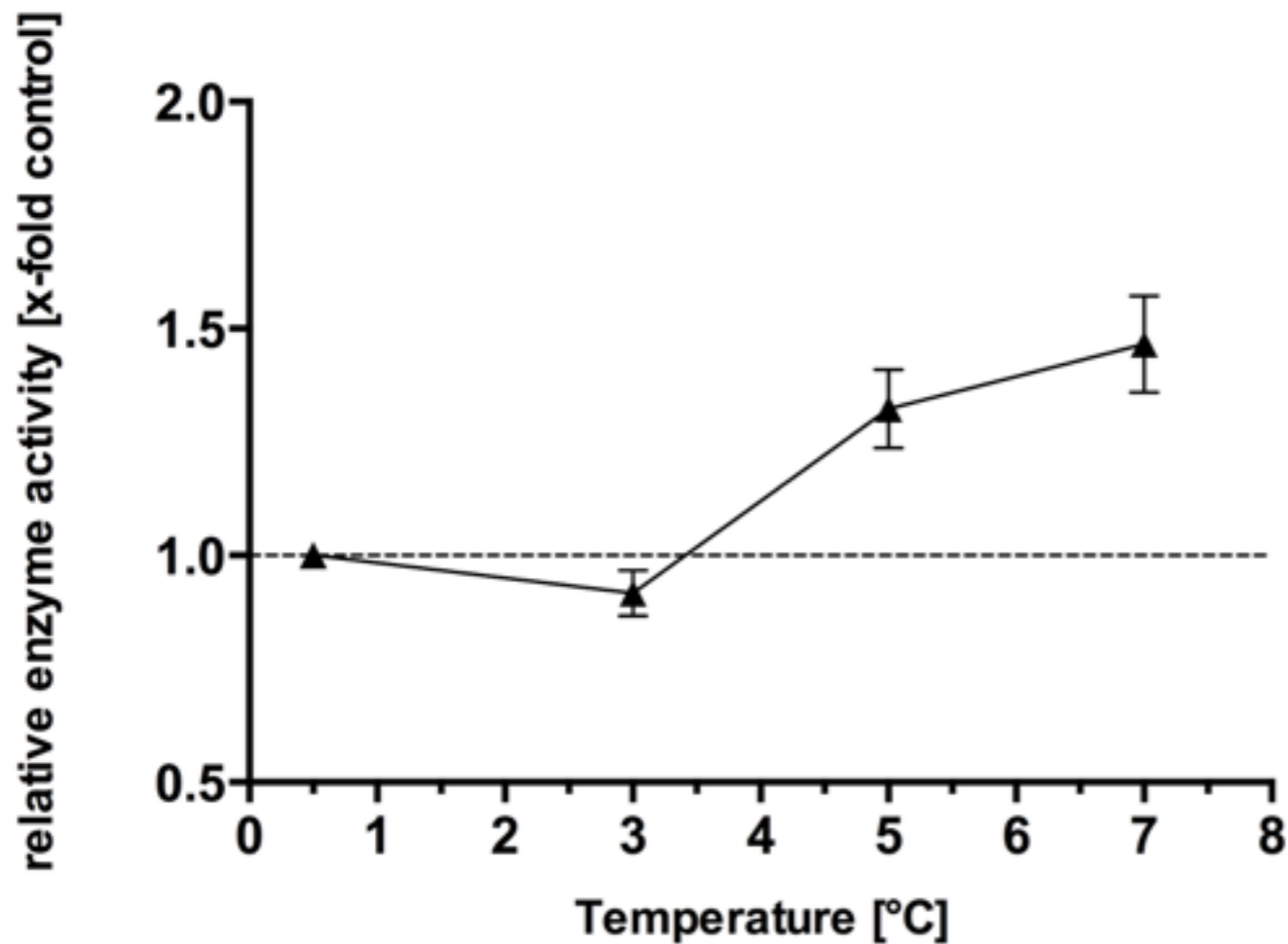
**malate
dehydrogenase**

TCA cycle

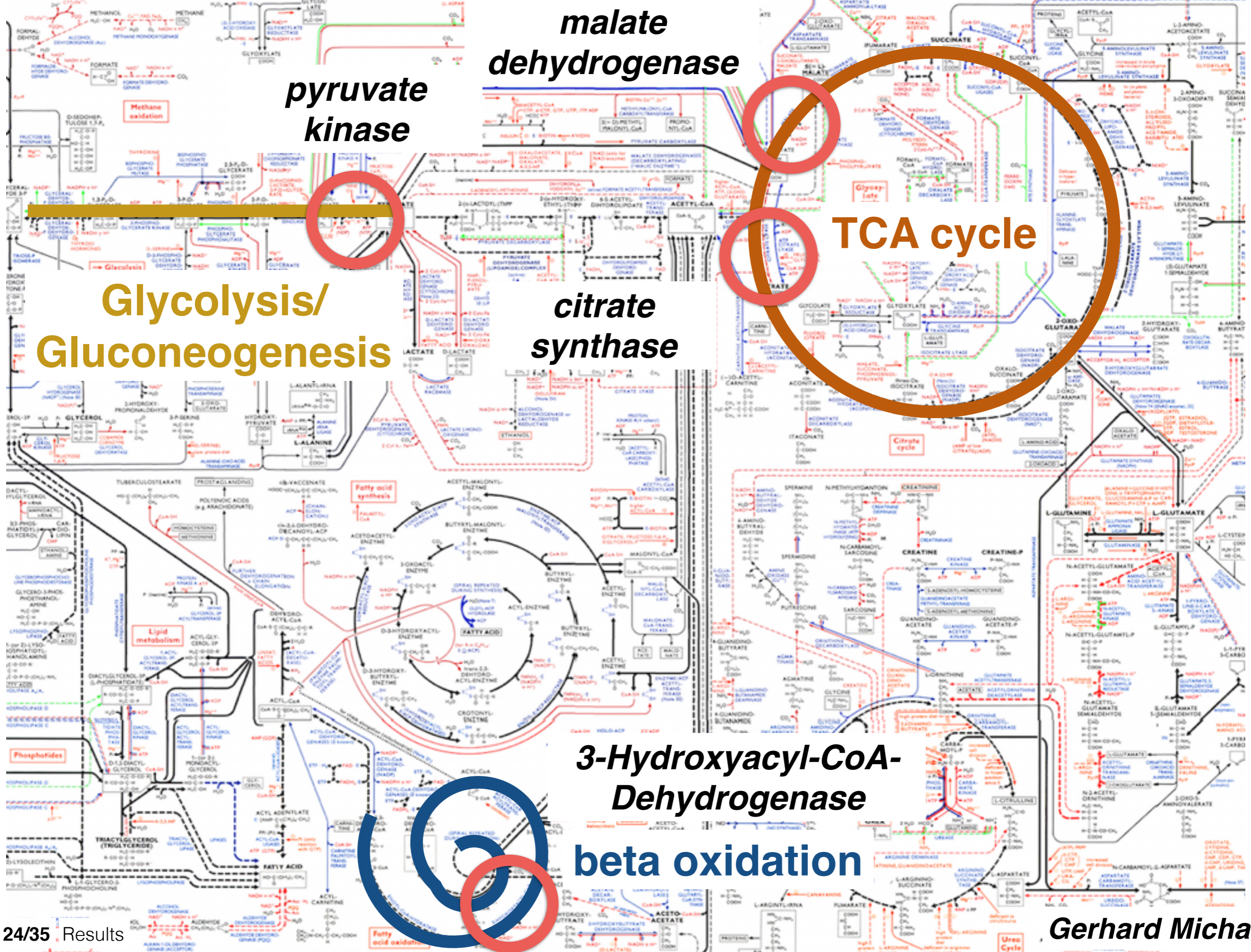
**citrate
synthase**

beta oxidation

Pyruvate Kinase PK



- Key enzyme in glycolytic pathway, catalyzes transphosphorylation from PEP and ADP to pyruvate and ATP
- constitutes primary metabolic intersection (*Munoz 2003*)
- suggested to play an important role in the transition to anaerobic metabolism (*Vial et al. 1992*)



**Glycolysis/
Gluconeogenesis**

**pyruvate
kinase**

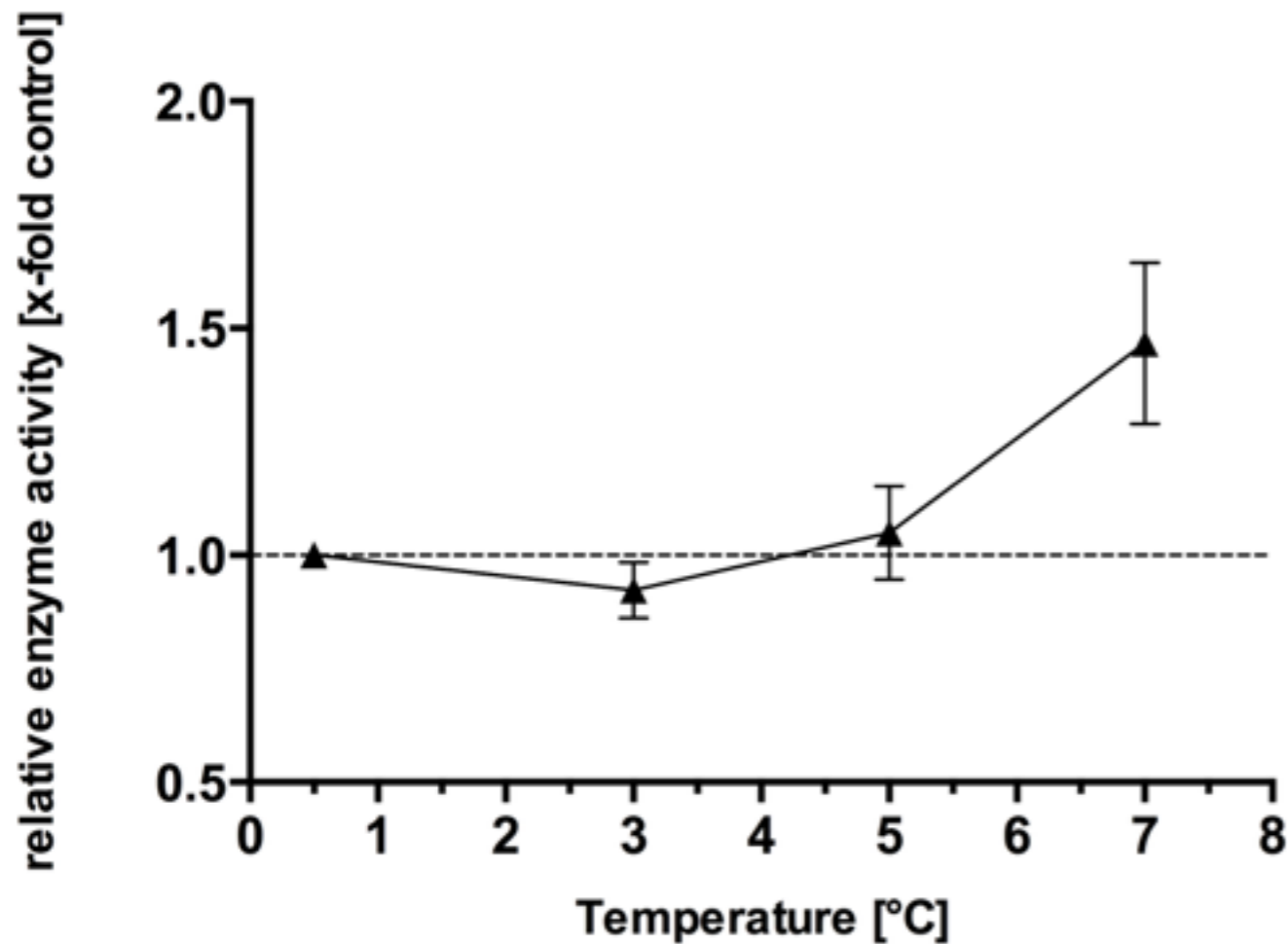
**malate
dehydrogenase**

TCA cycle

**citrate
synthase**

**3-Hydroxyacyl-CoA-
Dehydrogenase
beta oxidation**

3-Hydroxyacyl-CoA-DH HOAD



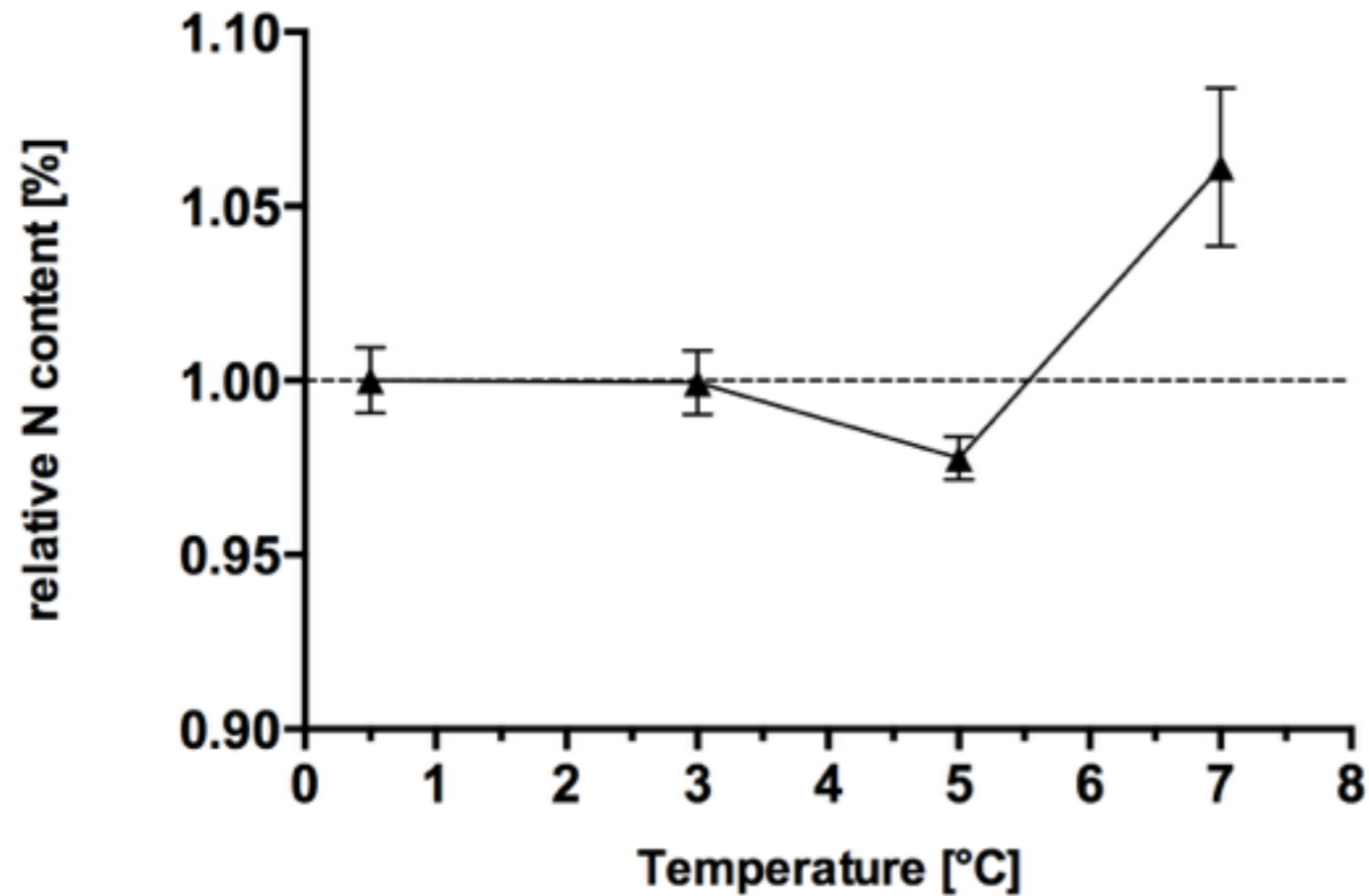
- 3rd step in beta oxidation
- marker enzyme for utilization of lipids

Glucose Catabolism

- ATP is allosteric inhibitor of PK -> upregulation of PK when ATP required
- upregulation -> less gluconeogenesis, no demand for synthesis of glucose

Protein Catabolism

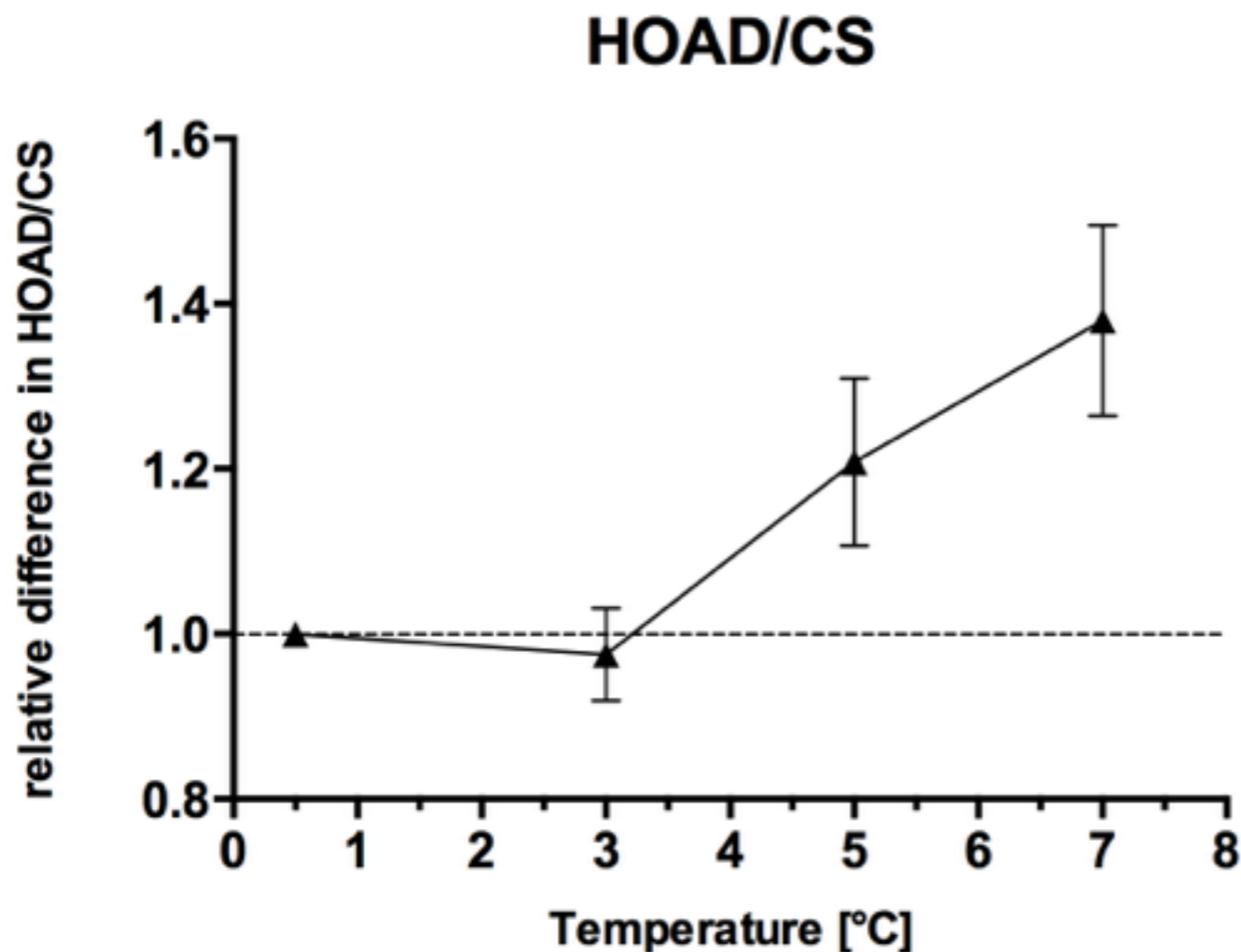
Nitrogen content



MDH going up \leftrightarrow not mirrored by CS:

- points to a role of MDH other than that in the cycle series: downstream shuttling of intermediates of protein catabolism into TCA?
- other studies show higher capacity for protein breakdown with increasing temperature (Schwerin et al. 2009)

Lipid Catabolism



Normalization to CS as central crossing point in metabolism (Windisch et al. 2011):

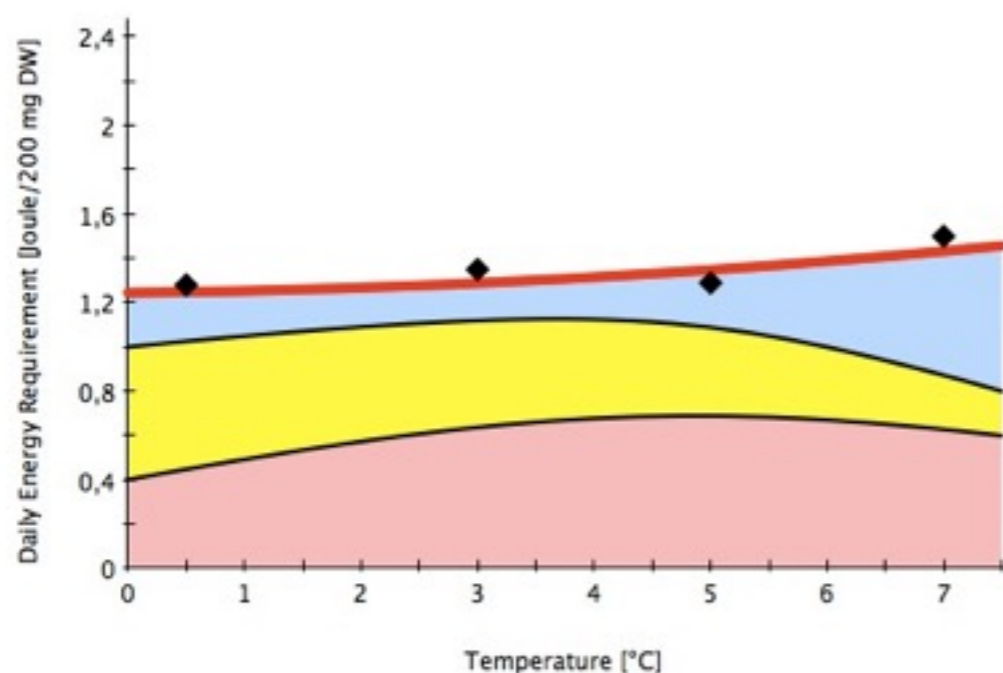
- increase in ratio hints at tendency towards lipid oxidation, NOT lipid synthesis

Conclusions

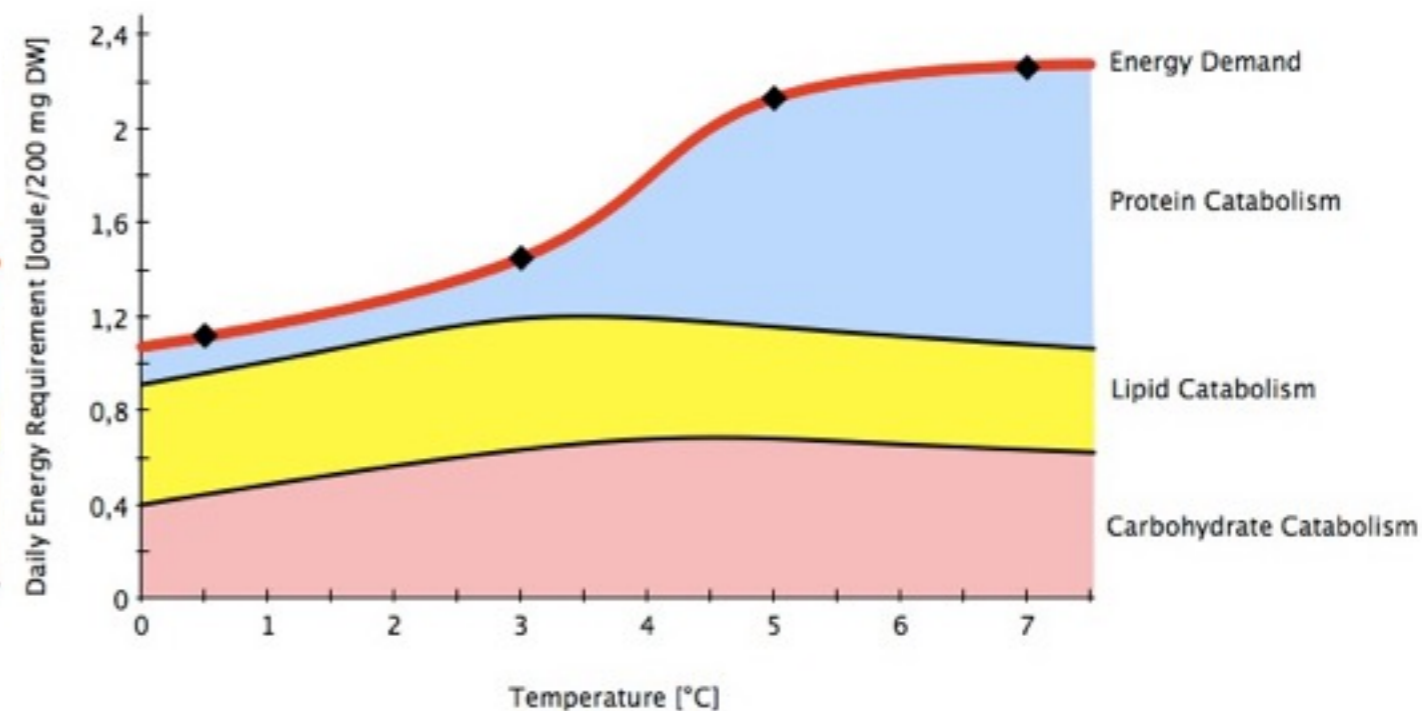
- Increased seawater temperature possibly leads to:
 - *earlier onset and heavier reliance on protein catabolism*
 - *prolongation of lipid oxidation*

Conclusions

Control



Treatment



Implications:

- Krill relies on productive summer months to accumulate lipid reserves for winter - **prolonged lipid oxidation may impede the buildup of these crucial reserves** - overwinter-ability affected
- **Energy** channeled towards higher maintenance **will lack elsewhere**, for example maturation

Outlook

Differential Gene Expression

- validate enzyme activities on genetic level
- fill gaps in the puzzle

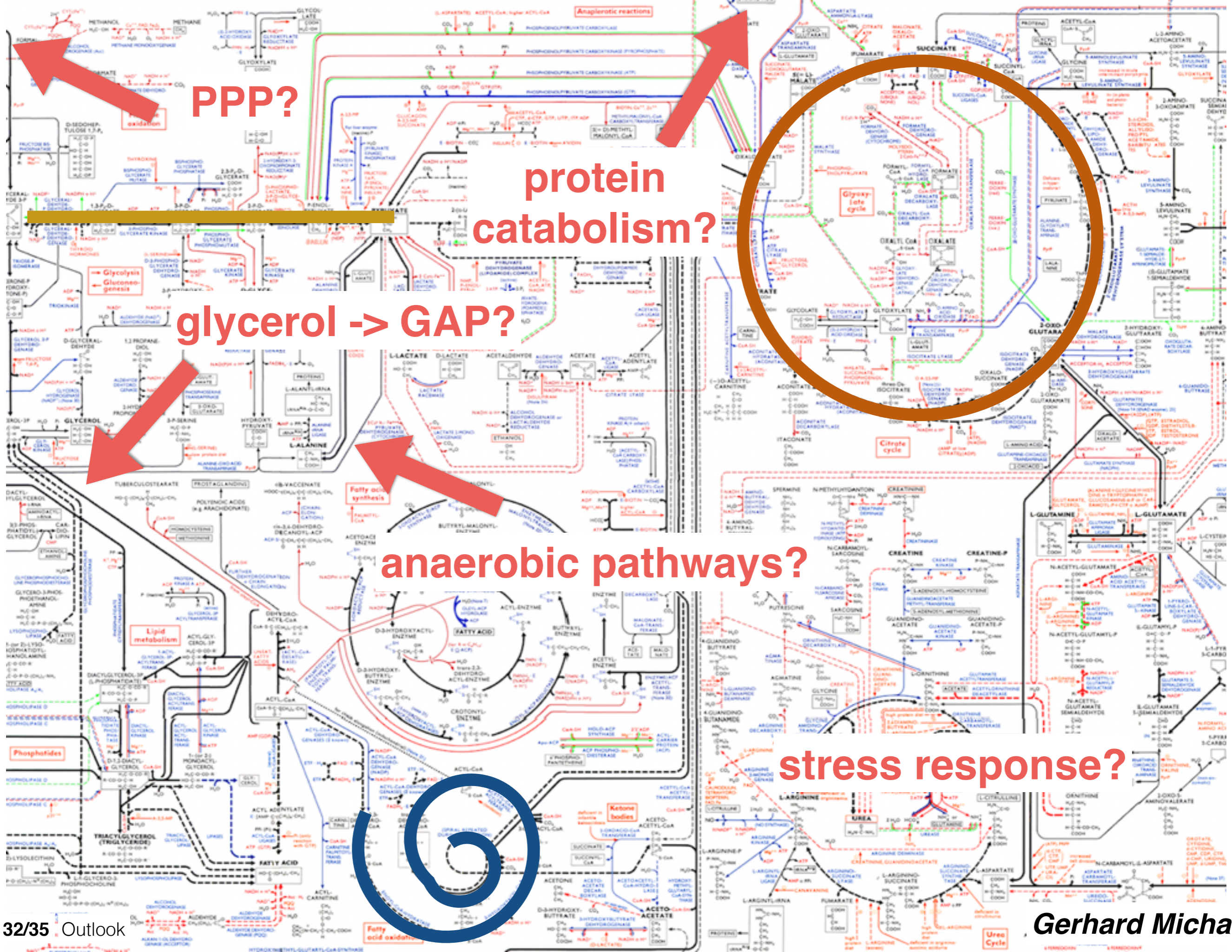
PPP?

protein catabolism?

glycerol -> GAP?

anaerobic pathways?

stress response?



Outlook

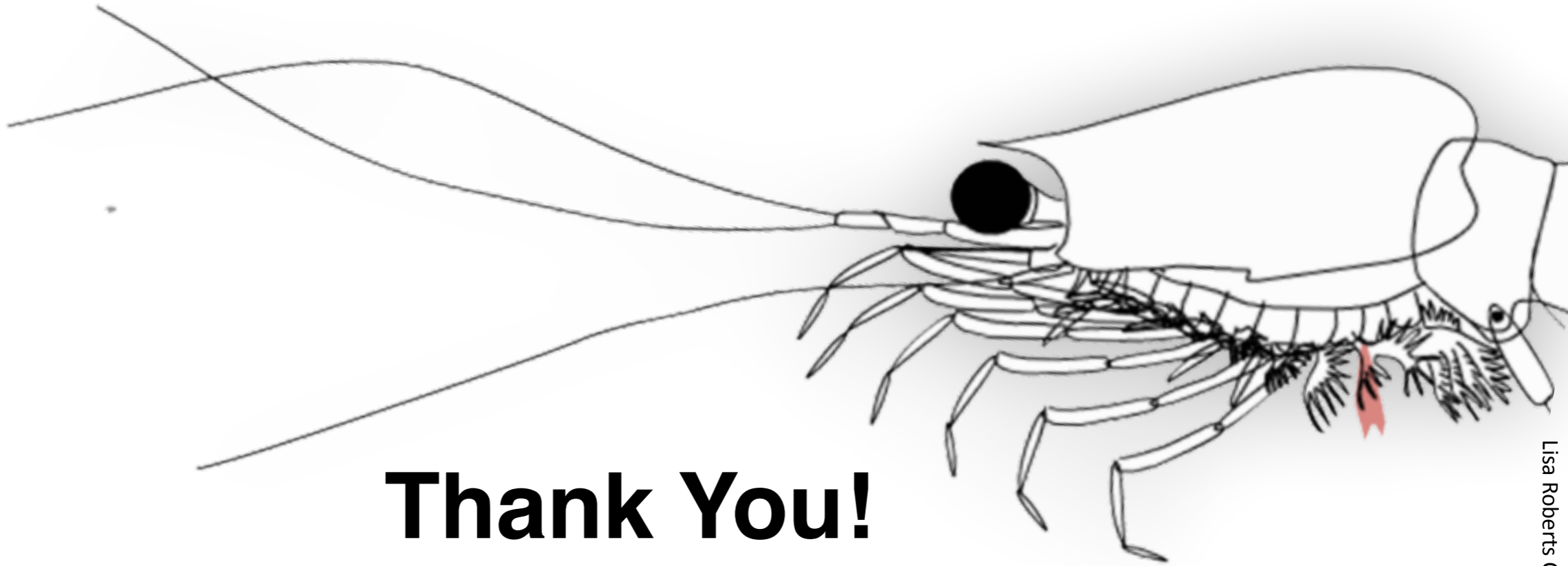
Temperature Compensation of Clock Genes

- Dissociation of environmental events (blooms, sea-ice retreat) and endogenously controlled physiology (regression, maturation, spawning)



Acknowledgments

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Thank You!

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