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Hsp72 mRNA transcription, and Sweat Adaptations are greater post Heat Acclimation in Trained vs. Untrained individuals

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Heat Acclimation (HA)

- Intervention to improve ability to tolerate heat stress

- Four classic markers of HA [Sawka (2011) Comp Physiol. 1(4) 1883-928]

↓ Core temperature (-0.2 ± 0.1 °C)

↓ Heart rate (-5 ± 5 $b.min^{-1}$)

↑ Sweat rate (23 ± 38%)

↑ Performance/capacity (21 ± 28%)

[Tyler et al., (2016) Sports Medicine. 1-26]

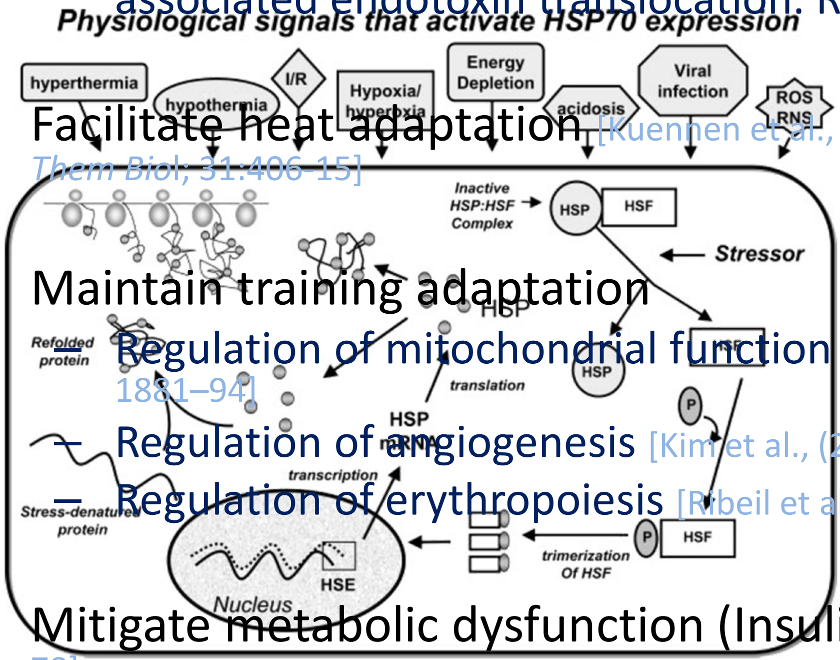
- Novel marker [Lee at al., (2015) *SJMSS*. 10.1111/sms.12621]

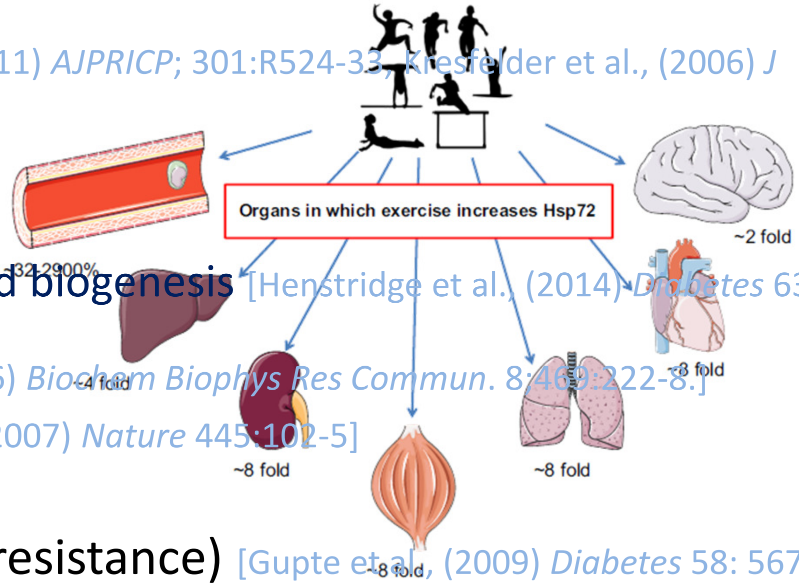
↑ cellular tolerance via Heat shock proteins

Heat Shock Proteins (HSP)

- Augment in response to stress [Kregel (2002) *J Appl Physiol* 92: 2177–2186]
  - Oxidative, cytokine, muscular, thermal
- Present in multiple tissue sites [Henstridge et al., (2016) *J Appl Physiol* 120: 683–691]
- Most important – HSP70 family (HSPA1A; HSP72)

- Protection of vital organs [Amorim et al. (2015) *Temperature*; 2:499-505; Ely et al. *Temperature* 2:51-2]
  - Maintaining intestinal epithelial tight junction barriers. Increasing resistance to gut-associated endotoxin translocation. Reducing systemic inflammatory response

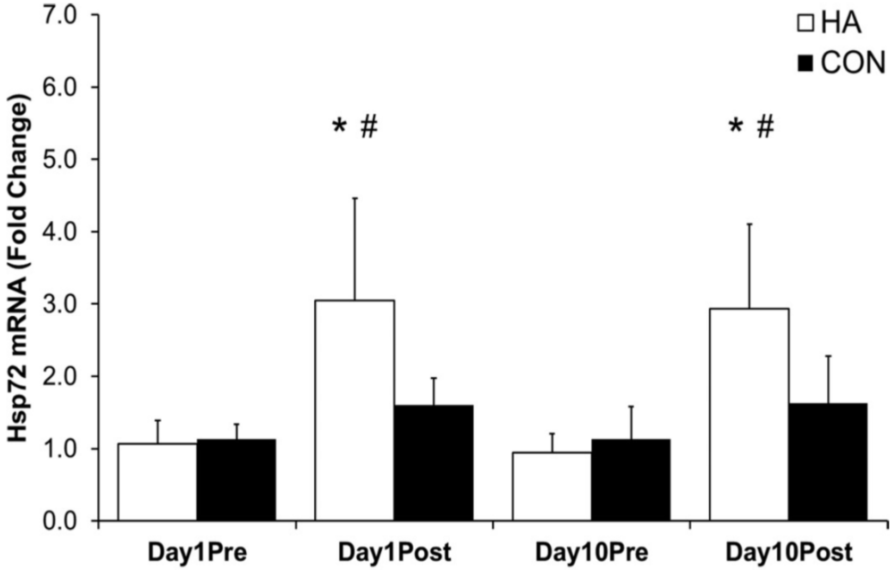




- Facilitate heat adaptation [Kuennen et al., (2011) *AJPRICP*; 301:R524-33; Kresfelder et al., (2006) *J Therm Biol*; 31:406-15]
- Maintain training adaptation
  - Regulation of mitochondrial function and biogenesis [Henstridge et al., (2014) *Diabetes* 63: 1881–94]
  - Regulation of angiogenesis [Kim et al., (2016) *Biochem Biophys Res Commun.* 8;469:222-8.]
  - Regulation of erythropoiesis [Ribeil et al., (2007) *Nature* 445:102-5]
- Mitigate metabolic dysfunction (Insulin resistance) [Gupte et al., (2009) *Diabetes* 58: 567–78]

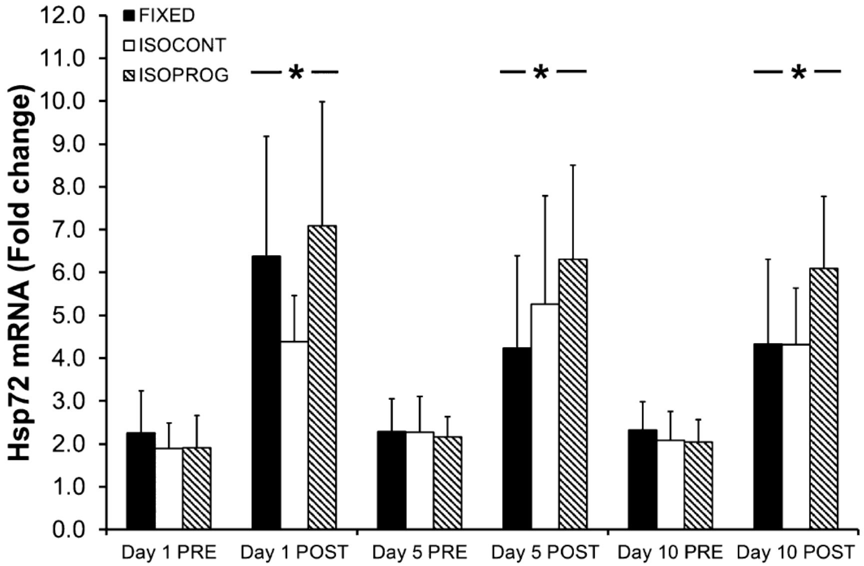
Hsp72 mRNA & Heat Acclimation

- Greater ↑ in Hsp72 mRNA HA vs CON



Gibson et al., (2015) *J Appl Phys*. 19, 889-899

- No difference in Hsp72 mRNA with different HA protocols



Gibson et al., (2015) *SJMSS*. 25, 259-268

Effect of “fitness” on Heat Acclimation

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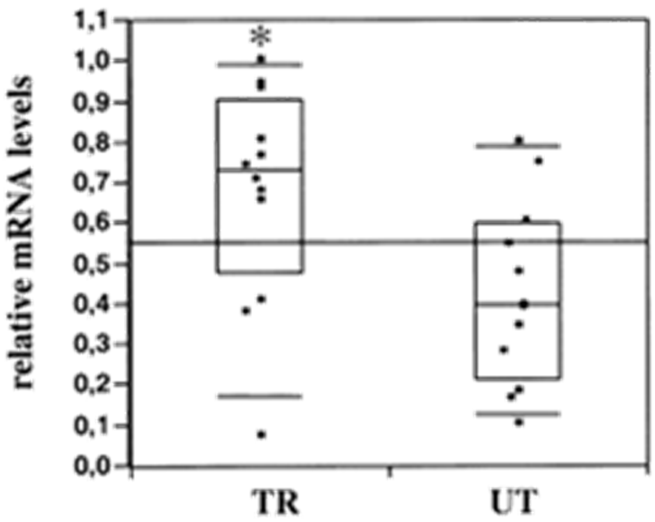
- 10 sessions
- 60 min HA
- 4.8 km.h^{-1}
- 3-7%
- 40°C,
- 30% R.H.

	Moderately Fit 46 mL.kg^{-1}.min^{-1}	Highly Fit 60 mL.kg^{-1}.min^{-1}
Δ Resting T_{rec}	- 0.17°C	- 0.11°C
Δ Exercising T_{rec}	- 0.17°C	- 0.21°C * $P < 0.05$
Δ End HR	- 7 b.min^{-1}	- 10 b.min^{-1}
Δ Sweat Rate	+0.21 %BM	+0.35 %BM
???		

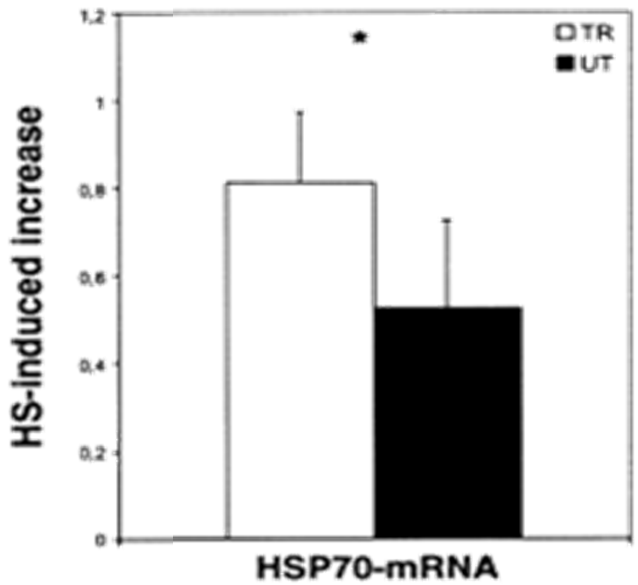
Cheung and McLellan (1998) *J Appl Phys* 84:1731-1739,

HSP72 and training status

- Trained = ↑ basal Hsp72 mRNA



- Trained = ↑ transcription of Hsp72 mRNA with *in vitro* heat stress



Fehrenbach et al., (2000) *J Appl Phys* 89 704 - 710

Aim

Determine differences in…

- Physiological markers (**resting core temperature, resting heart rate, sweat rate**) between **trained** and **untrained** individuals in response to 10 days of isothermic HA
  - **Hypothesis:** Equality of physiological adaptations between trained and untrained

- Transcription of **Hsp72 mRNA** between **trained** and **untrained** individuals in response to 10 days of isothermic HA
  - **Hypothesis:** Greater increase in Hsp72 mRNA during *in vivo* HA intervention in trained

Participants and Method

- Upper and lower quartile of participants (ranked by VO_{2peak}) from two previous experiments
  - Gibson et al., (2015) *SJMSS*. 25(1), 259-268
  - Gibson et al., (2015) *J Appl Phys*. 19 (8), 889-899

- Preliminary testing (VO_{2peak} and anthropometry)

- Ten days of isothermic HA T_{rec} ≥38.5°C (40°C/40%)

  - Pre session

Resting T_{rec}, HR, NBM, Hsp72 mRNA

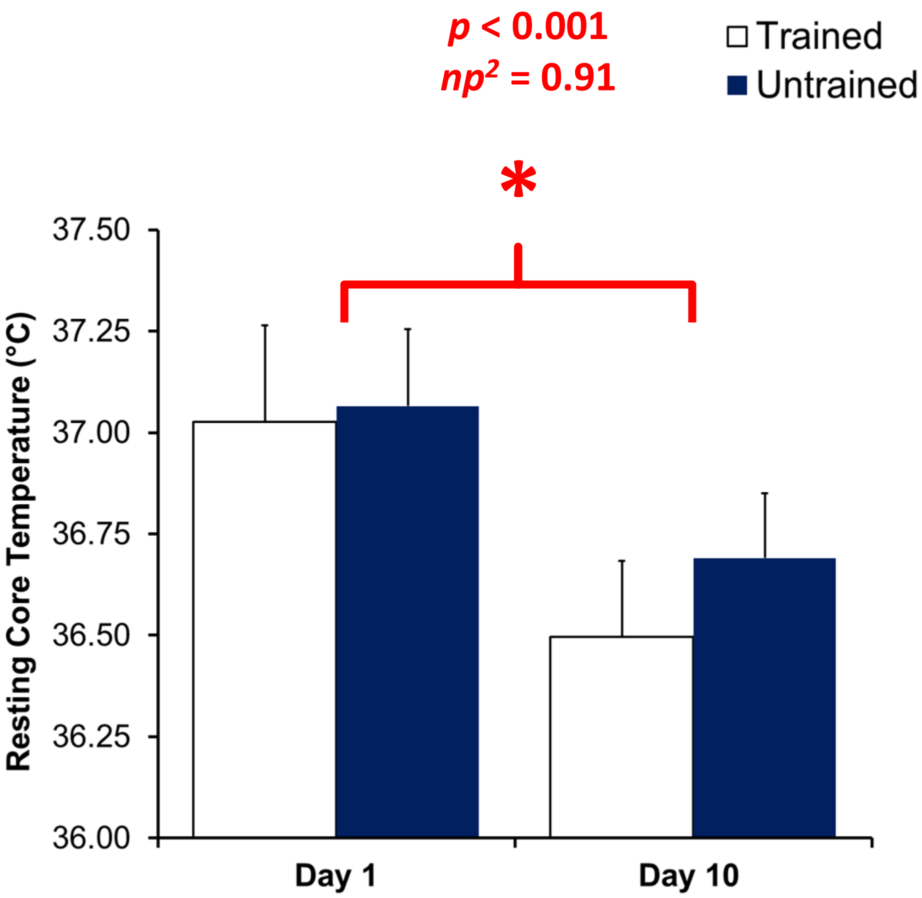
  - Post session

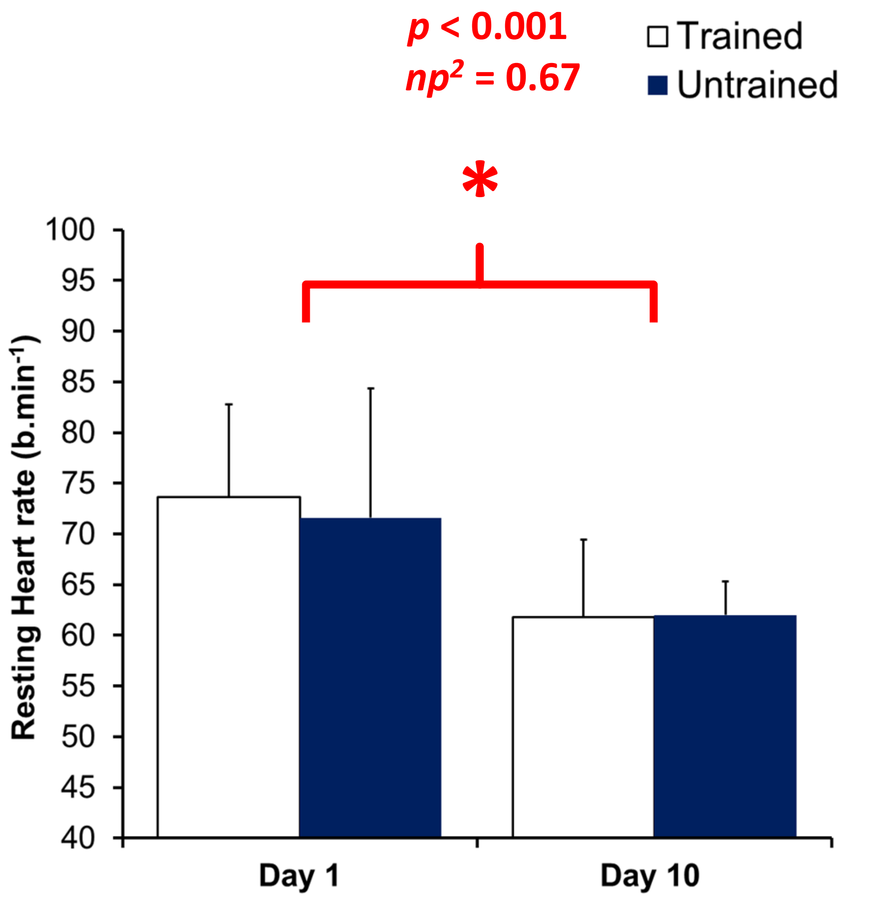
NBM, Hsp72 mRNA

Trained (T; n = 6)

Untrained (UT; n = 6)

Results - Core temperature and Heart rate





Discussion – Core temperature and Heart rate

- Equality of adaptation between T and UT in agreement with previous work [Cheung and McLellan (1998) *JAP* 84:1731-1739]

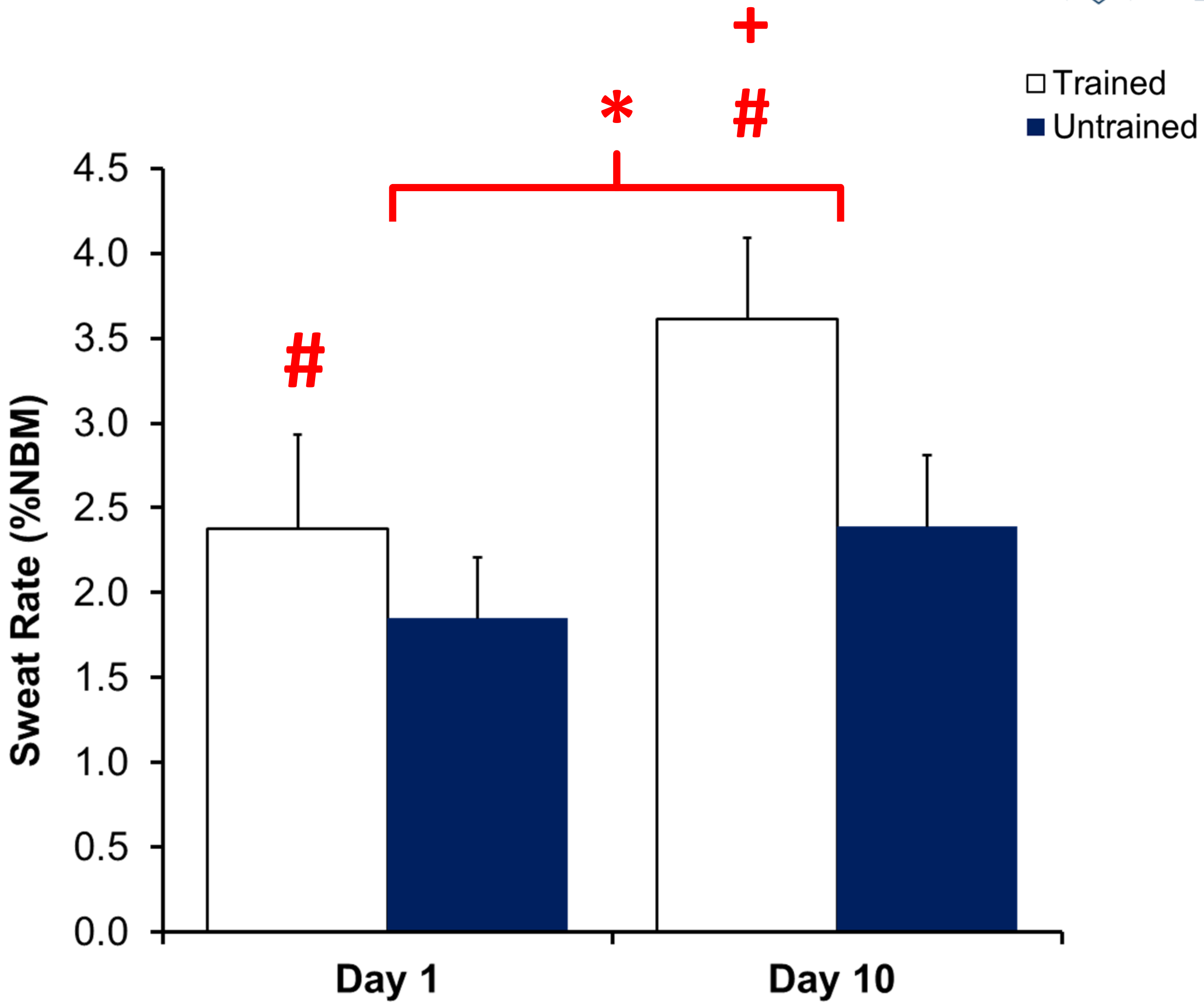
Rest T_{rec} [T = -0.5 ±0.2°C; UT = -0.4 ±0.2°C]

- – Regulation of heat balance at POAH via afferent feedback improved irrespective of training status

Rest HR [T = -12 ±4 $b.min^{-1}$; UT = -11 ±12 $b.min^{-1}$]

- – Equality of PV expansion possible in T vs UT?
- – ↓ HR may be result of improved myocardial compliance, rather than maintained/increased SV via ↑PV/↓SkBf [Periard et al., (2016) Autonomic Neuroscience 196 52-62]

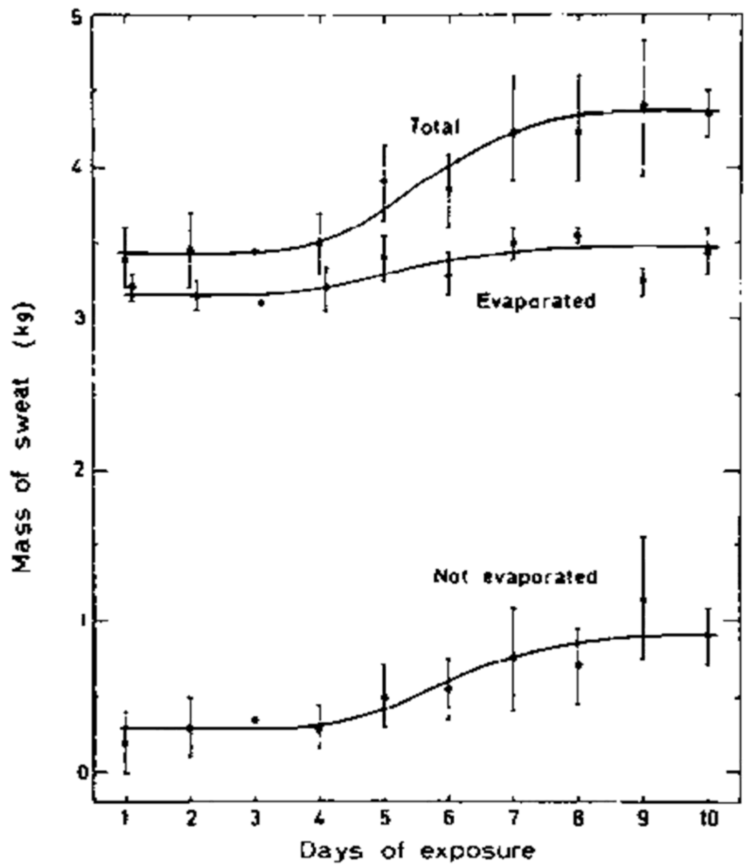
Results - Sweat rate adaptations



= $p = 0.003$; $np^2 = 0.59$. * = $p < 0.001$; $np^2 = 0.82$. + = $p = 0.029$; $np^2 = 0.39$.

Discussion – Sweat rate adaptations

- Greater sweat adaptation in T (+1.2 ±0.6 %NBM; +52%) vs UT (+0.5 ±0.4 %NBM; +29%) due to
  - ↑ sweat sensitivity ↑ gland output ↑ cutaneous vasodilation
  - Determine role of training status on sweat sensitivity, cholinergic, α- & β- adrenergic, plasma ATP
- Trained individuals may become hypohydrated more quickly (E_{max} dependent).
- Future direction: Control for H_{prod} (W) and exercise duration to determine whole-body sweat rate.
  - ↑ H_{prod} ($W.kg^{-1}$) = ↑ rise in T_{rec} = ↑ duration of ~ max sweating. [Jay and Cramer (2015) *Temperature* 2 (1) 42-43]
  - Greater duration exercising (D10 vs D1; T +16 min (47%) UT +5 min (8%))



Mitchell et al,. (1976). *J Appl Physiol* 40, 768–778

Results - Hsp72 mRNA

* = $p < 0.001$; $np^2 = 0.91$
\# = $p = 0.004$; $np^2 = 0.57$

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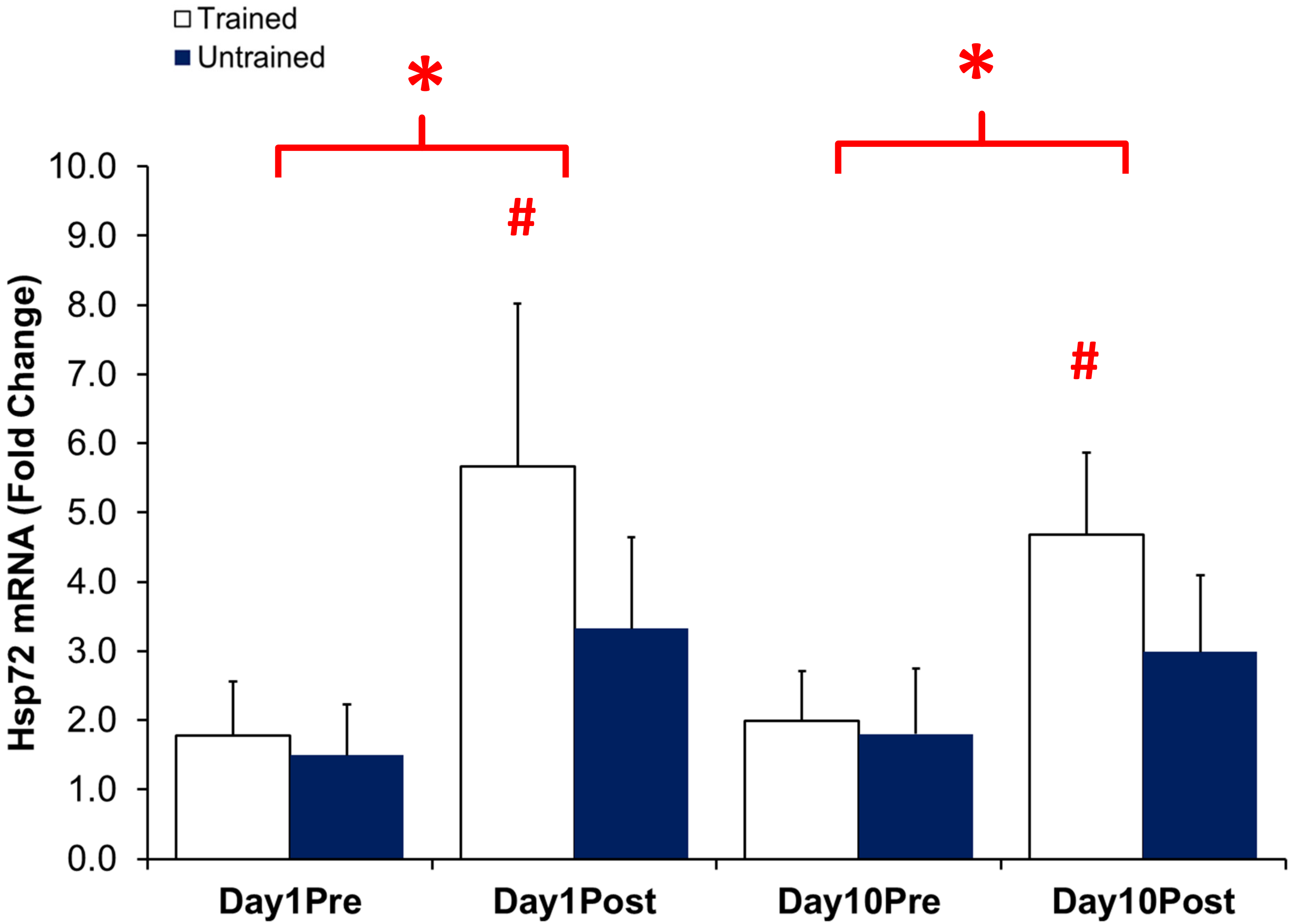


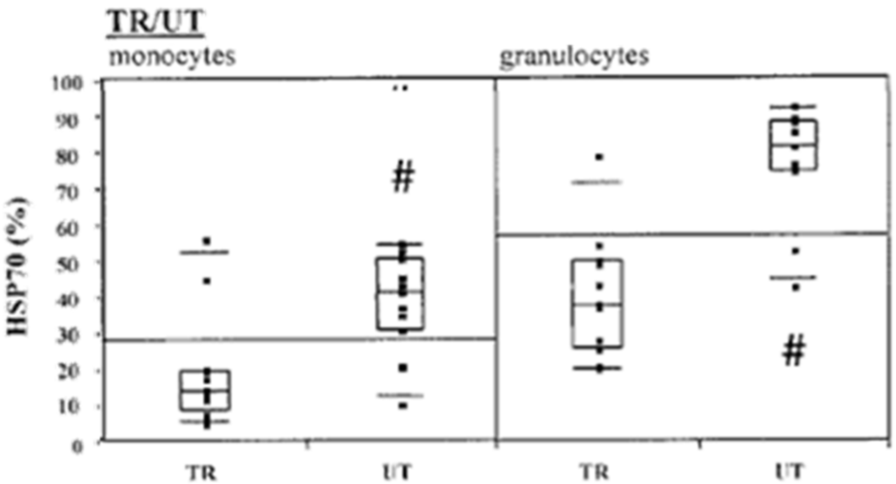
Discussion – Hsp72 mRNA

- Greater Hsp72 mRNA increase in T (+160%) vs UT (+82%)
  - Improved transcriptional activation now shown *in vivo*

  In vitro; Fehrenbach et al., (2000) *J Appl Phys* 89 704 – 710

- Future direction: Unknown if protein translation similar under equal endogenous criteria

  Fehrenbach et al., (2000) *Med Sci Sp Ex* 32 (3) 592-600



Conclusions

1. Isothermic HA able to induce physiological adaptations in both Trained and Untrained individuals.

   T_{rec} and HR demonstrate equality (effect of v.highly trained unknown)

   Sweat adaptations may demonstrate accelerated response in Trained individuals

2. Greater Hsp72 mRNA increase in Trained individuals

   Improved *In Vivo* transcriptional activation

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Dr Lee Taylor

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