

# Acclimation of the temperature set-points of the heat-shock response

Deepak Barua\*, Scott A. Heckathorn<sup>1</sup>

*Biology Department, Syracuse University, Syracuse NY 13244, USA*

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## Abstract

We surveyed studies that examined the heat-shock response (HSR) temperature set-points in response to changes in growth temperature to ask whether plasticity of the HSR set-points is common to all organisms, and whether it varies with thermal niche. Unexpectedly, sessile organisms exhibited lower acclimation in the HSR set-points compared to mobile organisms, and there was no difference in acclimation between eurytherms and stenotherms. The extent of acclimation differed among different heat-shock proteins, and the HSR temperature set-points (induction, maximal response, and shut-off). We show that HSR induction temperatures change in a step-wise fashion in response to growth temperature.

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## 1. Introduction

Temperature, a dominant factor in establishing growth, reproduction, and distribution of organisms, lacks spatial and temporal constancy in most environments. Consequently, organisms employ diverse adjustments at multiple levels of biological organization to deal with the fluctuating nature of the thermal environment (Precht, 1973; Cossins and Bowler, 1987; Hochachka and Somero, 2002). One such response, the heat-shock response (HSR), is the rapid expression of a distinct set of proteins, the heat-shock proteins (Hsps), with an acute increase in temperature (Lindquist, 1980). Hsps help protect cellular components from damage during heat stress, and facilitate their repair afterwards (Parsell and Lindquist, 1994). Extensive research on Hsps since their discovery has contributed to a better understanding of Hsps, their cellular targets, and mechanisms of action. In spite of this, several important

questions remain with respect to the ecological and evolutionary importance of the HSR (Feder and Hofmann, 1999; Hochachka and Somero, 2002). For example, while plasticity of Hsp gene expression in organisms is now well documented, the ecological and evolutionary significance of such plasticity for organisms in nature remains largely unexplored (Buckley and Hofmann, 2002; Tomanek and Somero, 2002).

The minimum heat-shock temperature required to induce the HSR ( $T_{on}$ ), the temperature of maximal response ( $T_{max}$ ), and the shut-off temperature ( $T_{off}$ ) appear to be species specific, and reflect the evolutionary history of organisms (Dietz and Somero, 1993; Tomanek and Somero, 1999). However, induction temperatures of the HSR also exhibit considerable plasticity as a function of developmental stage, growth temperature, and season (Dietz and Somero, 1992; Hofmann and Somero, 1995; Chapple et al., 1998; Roberts et al., 1997; Tomanek and Somero, 1999; Currie et al., 2000; Buckley and Hofmann, 2002). Organisms acclimated to warmer temperatures generally exhibit higher temperatures of HSR induction ( $T_{on}$ ), and such acclimatory changes probably help fine-tune the HSR to variable thermal

\*Corresponding author.

<sup>1</sup>Present address: Department of EEES, MS 604, University of Toledo, Toledo OH 43606, USA.

habitats (Hochachka and Somero, 2002). However, other studies have documented little or no change in the HSR temperature set-points with changes in growth temperature (Koban et al., 1987; Somers et al., 1989; Mason-Apps et al., 1990), and whether plasticity of the HSR is common to all organisms, and differs in organisms adapted to different thermal niches, is unclear (Tomanek and Somero, 1999). Also unknown are if and how changes in growth temperature affect the magnitude of expression of Hsps. Seemingly, increased growth temperature should increase magnitude of expression of Hsps in anticipation of higher temperatures and greater thermal damage. Alternatively, other growth-temperature-induced changes, e.g. greater membrane thermostability or increased cellular protectants (Hochachka and Somero, 2002), could decrease damage at higher temperatures and thereby reduce the requirement for more Hsps.

Eurythermal organisms tolerate broad ranges in environmental temperatures, unlike stenothermal organisms that are restricted to narrower ranges in more stable environments (Somero et al., 1996). Several authors have suggested that eurytherms should exhibit greater plasticity than stenotherms (Levins, 1969; Bradshaw, 1972; Somero et al., 1996; Bennett and Lenski, 1999; Huey et al., 2002), but whether acclimatory responses of the HSR set-point temperatures agree with this suggestion is unknown. In a similar manner, sessile organisms might be expected to show greater acclimation than mobile organisms. Plants and other sessile organisms are limited in their ability to escape from extreme thermal conditions, and are subsequently exposed to broader ranges of thermal environments (Bradshaw, 1972; Huey et al., 2002). Finally, in unicellular and simple multicellular organisms acclimation via biochemical mechanisms may be more important than in complex multicellular organisms, that have several alternative higher order mechanisms to cope with changes in temperature (Huey et al., 2002).

The sensing of acute temperature changes and the subsequent initiation of the HSR involves a complex cascade of events (Morimoto, 1998). Mechanisms responsible for acclimation of the HSR to different growth temperatures are not fully understood (Hofmann et al., 2002), and investigations of the various steps of the HSR in response to acclimation might elucidate potential mechanisms of acclimation. Different classes of Hsps have different functions, cellular localization, and induction kinetics, and might also differ in susceptibility to acclimation. Hsp70 and Hsp90 are among the first Hsps induced in response to acute temperatures (Tomanek and Somero, 1999), while most small Hsps (sHsps) are induced at higher temperatures and later in the HSR (Howarth and Ougham, 1993). Hsp70 and Hsp90 play a pivotal role in the regulation of the HSR (Morimoto, 1998), and could potentially also

be involved in the process of acclimation of the HSR (Buckley et al., 2001; Tomanek and Somero, 2002), whereas other Hsps might not be involved. Additionally, while plasticity in the temperature of induction ( $T_{on}$ ) of the HSR has been commonly documented, it is unclear whether the temperatures of maximal synthesis ( $T_{max}$ ), and the shut-off temperature of the HSR ( $T_{off}$ ), are similarly plastic (Tomanek and Somero, 1999). And finally, it is not known whether there are any limits to acclimation (i.e. the range of temperatures over which acclimation is observed), or the change in temperatures that is necessary to elicit an acclimatory response in the HSR.

We surveyed the literature to ask whether acclimation of the HSR was common to most organisms, and if there were differences among organisms adapted to different thermal niches. Specifically, we compared the extent of acclimation of HSR induction temperatures ( $T_{on}$ ) based on taxa (vertebrates, invertebrates, plants, and protists, fungi and monera), mobility, and eurythermy. We examined the extent of acclimation in the different temperature set-points of the HSR ( $T_{on}$ ,  $T_{max}$ , and  $T_{off}$ ), and different Hsp classes (Hsp90, Hsp70 and small Hsps) and the heat-shock factor (HSF). We also examined the direction of change in the magnitude of expression of Hsps (i.e. increase or decrease in Hsp transcript or protein) with changes in growth temperature. Finally, we asked whether Hsp synthesis, mRNA transcription and HSF activation all exhibit acclimation, and if there were any general patterns in acclimation of the HSR.

## 2. Methods

From the literature, we surveyed over 400 previously published studies that had reported the temperature set-points of the heat-shock response. Of these, 33 studies for 33 different species had examined the set-points of the HSR for an organism at two or more growth temperatures. From these 32 studies, we extracted data on growth temperatures, the methods used to detect the HSR, the Hsps examined, the direction of change in magnitude of expression of Hsps, the induction temperature ( $T_{on}$ ), the temperature of maximal Hsp synthesis ( $T_{max}$ ), and the shut-off temperature ( $T_{off}$ ) of the HSR. For the purposes of this study, induction of the HSR was defined as the induction of Hsp protein synthesis, Hsp mRNA transcription, or the activation of HSF-DNA-binding activity in response to heat shock. From the data, we calculated the difference in growth temperatures examined, the corresponding difference in the temperature set-points of the HSR, and the change in the temperature set-points per unit change in growth temperature. When data for more than two growth temperatures were available, we also calculated the

maximum change in the temperature set-points per unit change in growth temperature. To reduce pseudoreplication, we analyzed a single sample per species. When multiple data from different investigations for a species were available, we used sequential criteria for selecting one of the studies. We selected studies that reported the greatest change in HSR set-point temperatures per unit change in growth temperature. To avoid inconsistencies resulting from difference in methodologies, we did not mix data from different studies within a species. When data for multiple proteins for a species within an investigation were available we selected one of the proteins in the following order: Hsp70, Hsp90, Hsp60, small Hsps (sHsps), and then others (except for the comparison of different classes of Hsps, for which we used data for all proteins in a species). We selected data with the highest resolution (i.e. smallest temperature intervals between measurements). When data from multiple methods for the same species were available, we first chose protein synthesis data, followed by immunologically detected Hsp accumulation, RNA synthesis, and lastly, HSF-DNA-binding. The author's original determination of  $T_{on}$ ,  $T_{max}$  and  $T_{off}$  was used when provided; when not specified, these were determined from data provided. Organisms were classified into taxonomic groups; as vertebrates (V), invertebrates (I), plants (P), and protists, fungi and monera (PFM). Organisms were classified as mobile or sessile, and eurytherms or stenotherms, based on adult life stages. Endotherms were included with stenotherms. Analysis of variance was used for comparison of means.

### 3. Results

Most studies that have investigated the set-points of the HSR of organisms acclimated to different growth temperatures demonstrate significant plasticity (Table 1). However, some show that the HSR, or aspects of it, do not respond to changes in acclimation temperature, and appear to be more genetically fixed. Studies that demonstrate lack of acclimation in the HSR include those of vertebrates, invertebrates, plants, and protists. These cases, which represent unrelated taxa, and both eurytherms and stenotherms, appear to be exceptions rather than a norm. Changes in the HSR set-point temperatures, where they occur, are generally positive, i.e. increases in growth temperature resulted in an increase in the HSR temperature set-points. However, an opposite trend occurs in rainbow trout (Currie et al., 2000) and sea lamprey (Wood et al., 1999). Acclimation has been demonstrated for different components of the HSR, including activation of the heat-shock-factor's DNA-binding ability, transcription of Hsp genes, and translation of Hsp proteins.

The direction of change in magnitude of expression of Hsps (amount of Hsp transcript or protein) was not consistent with changes in growth temperature (Table 1). Of the 20 studies that examined magnitude of expression in Hsps, nine showed no change, seven showed an increase, while four showed a decrease in magnitude with increasing growth temperature. We could not identify any trends in the direction of change in magnitude of expression of Hsps with respect to phylogeny, mobility, or eurythermy of organisms.

To investigate patterns in the extent of acclimation, we examined the change in induction temperatures ( $T_{on}$ ) per unit change in growth temperature. Phylogeny did not affect the extent of acclimation in induction temperatures (Fig. 1A) ( $P = 0.26$ ). Interestingly, plants (P), and protists, fungi and monera (PFM), all sessile organisms, tended to have lower acclimation of  $T_{on}$ . Substantiating this, sessile organisms (including sessile animals) had lower acclimation compared to mobile organisms (Fig. 1B) ( $P < 0.05$ ). Acclimation of  $T_{on}$  was marginally higher in stenotherms than in eurytherms (Fig. 1C), but not significantly different ( $P = 0.74$ ). Where sufficient data were available, similar results were observed in comparisons within taxa; i.e. lower acclimation in sessile organisms within invertebrates, and greater acclimation in stenotherms within vertebrates (data not shown).

The extent of acclimation of  $T_{on}$  for HSF, Hsp90, Hsp70, and small Hsps were significantly different ( $P < 0.05$ ). Small Hsps show limited acclimation compared to HSF, Hsp90 and Hsp70 (Fig. 2). In support of this, for the few cases where sufficient data were available, we observed lower acclimation of small Hsps as compared to Hsp70 in paired tests within species. In comparing the different set-points of the heat-shock response (Fig. 3), the temperature of induction ( $T_{on}$ ) appears to be the most responsive to changes in growth temperature, followed by the maximal temperature of the response ( $T_{max}$ ), and lastly the shut-off temperature ( $T_{off}$ ) of the HSR ( $P < 0.05$ ). This was true for both the average change in the set-point temperature per unit change in growth temperature, and the maximum change in the set-point temperatures per unit change in growth temperature (data not shown). The shut-off temperature ( $T_{off}$ ) of the HSR rarely showed any acclimation in response to changes in growth temperature.

In the studies examined, complete acclimation was rare; i.e. the extent of acclimation (change in induction temperature per unit change in growth temperature) was almost always less than one. Generally, induction temperatures did not change in a continuous fashion with growth temperature, but in steps as illustrated by Fig. 4 for six representative species.

Table 1

Change in the magnitude of expression of Hsps (MAG) and temperature set-points of the heat-shock response ( $T_{on}$ —induction,  $T_{max}$ —maximal response,  $T_{off}$ —shut-off) per unit change in growth temperature

Genus	Species	Taxon	Mob	E/S	Reference	PAR	MAG	$T_{on}$	$T_{max}$	$T_{off}$
<i>Aurelia</i>	<i>aurita</i>	I	M		Black and Bloom, 1984	70	–	0.00	0.22	
						83		0.00		0.11
<i>Bombyx</i>	<i>mori</i>	I	M		Hsieh et al., 1995	G		1.33		
<i>Crassostrea</i>	<i>gigas</i>	I	S	E	Friedman et al., 1999	G		0.40		
<i>Drosophila</i>	<i>melanogaster</i>	I	M		Lindquist, 1980	70	+	0.60	0.20	0.00
<i>Drosophila</i>	<i>subobscura</i>	I	M		Pascual and deFrutos, 1988	70	–	0.50	0.00	0.00
						90		0.50	0.00	0.00
						L	–	0.00		0.00
<i>Drosophila</i>	<i>triantarctica</i>	I	M	E	Goto and Kimura, 1998	70	+	0.25	0.00	0.00
<i>Gillichthys</i>	<i>mirabilis</i>	V	M	E	Dietz, 1994	70	0	0.70		
						90	0	0.50		
					Buckley and Hofmann, 2002	70	+	0.86	0.86	0.71
						90		0.86		0.29
						HSF	+	0.47	0.60	
<i>Gillichthys</i>	<i>seta</i>	V	M	E	Dietz and Somero, 1992	90	0	0.50		
<i>Homo</i>	<i>sapiens</i>	V	M	S	Abravaya et al., 1991	HSF	–	0.50	0.50	
<i>Ictalurus</i>	<i>punctatus</i>	V	M	E	Koban et al., 1987	70	0	0.00	0.14	
						90		0.00	0.14	
<i>Iguana</i>	<i>iguana</i>	V	M	S	Bols et al., 1990	70		0.00	0.00	0.00
						39		0.00	0.00	0.14
<i>Leucosporidium</i>	<i>fellii</i>	F	S	E	Deegenars and Watson, 1998	90,70				0.00
<i>Leucosporidium</i>	<i>scottii</i>	F	S	E	Deegenars and Watson, 1998	90,70		0.50		0.00
<i>Lolium</i>	<i>temulentum</i>	P	S	E	Ougham, 1987	70		0.00	0.00	0.00
						L		0.00	0.00	0.00
<i>Montastrea</i>	<i>faveolata</i>	I	S	S	Black et al., 1995	70		0.17		
<i>Mus</i>	<i>musculus</i>	V	M	S	Kaneko et al., 1997	70	0	0.60		
						110		1.00		
<i>Mytilus</i>	<i>californianus</i>	I	M	E	Roberts et al., 1997	70	0	0.20	0.40	
<i>Oncorhynchus</i>	<i>mykiss</i>	V	M		Currie et al., 2000	L	+	0.00	0.00	
						70	+	–0.42	0.00	
					Legoff and Michel, 1999	HSF		0.33	0.00	
<i>Opuntia</i>	<i>ficus indica</i>	P	S	E	Somers et al., 1991	70,L		0.00	0.00	0.30
<i>Oryzias</i>	<i>latipes</i>	V	M	E	Oda et al., 1991	70		0.22		
<i>Perkinsus</i>	<i>marinus</i>	PR	S	E	Tirard et al., 1995	H,L		0.00		
<i>Petromyzon</i>	<i>marinus</i>	V	M		Wood et al., 1999	90,70	0	1.25	0.00	
<i>Physarum</i>	<i>polycephalum</i>	PR	S		Wright and Tollon, 1982	70		0.00		
<i>Saccharomyces</i>	<i>cerevisiae</i>	F	S		Hjorth-Sorensen et al., 2001	HSF	+	0.50	0.00	
					Chatterjee et al., 1997	G	+	0.17	0.25	0.17
<i>Saxifraga</i>	<i>cernua</i>	P	S	E	Mason-Apps et al., 1990	70,L	0	0.00	0.00	0.00
<i>Sorghum</i>	<i>bicolor</i>	P	S	E	Howarth, 1989	70		0.50		
						L		0.00		
<i>Spinacea</i>	<i>oleracea</i>	P	S		Somers et al., 1989	70		0.00	0.00	0.00
						L		0.00		
<i>Sporobolomyces</i>	<i>salmonicolor</i>	F	S		Berg et al., 1987	88			0.09	
<i>Synechocystis</i>	<i>PCC6803</i>	M	S		Horvath et al., 1998	70	0	0.14	0.29	
						60	0	0.43		
						L	0	0.29		
<i>Tegula</i>	<i>brunnea</i>	I	M		Tomanek and Somero, 1999	70	–	0.30	0.30	0.00
<i>Tegula</i>	<i>funeralis</i>	I	M	E		70	+	0.00	0.30	0.00
<i>Tegula</i>	<i>montereyi</i>	I	M			70	+/-	0.30	0.30	0.00
<i>Vigna</i>	<i>unquiculata</i>	P	S		Heuss-LaRosa et al., 1987	70		0.50		
						60		0.00		
						90		0.33		
						L		0.33		

Organisms classified as vertebrates (V), invertebrates (I), plants (P), protists (PR), fungi (F) or monera (M). The different HSR induction parameters (PAR) studied: G—general protein synthesis; H—high-molecular-weight Hsps; L—low-molecular-weight Hsps; HSF—heat-shock-factor activation; numbers indicate corresponding Hsps. Organisms classified into eurytherms (E) or stenotherms (S) and by mobility (MOB) as mobile (M) or sessile (S).

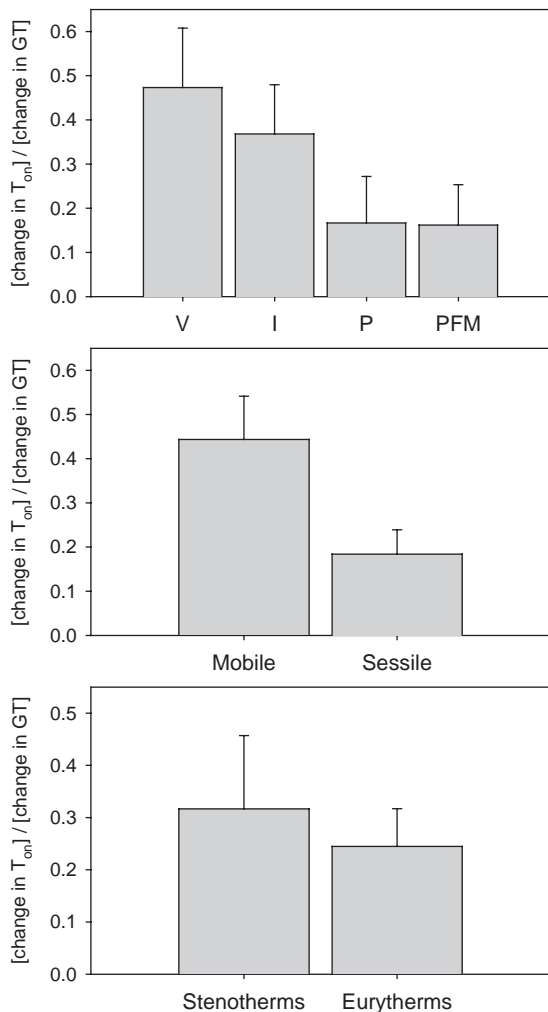


Fig. 1. Change in HSR induction temperature ( $T_{on}$ ) per unit change in growth temperature grouped by (A) taxa: V—vertebrates ( $n=9$ ), I—invertebrates ( $n=11$ ), P—plants ( $n=6$ ), PFM—protists, fungi and monera ( $n=5$ ); (B) mobility: mobile organisms ( $n=17$ ), sessile organisms ( $n=14$ ); and (C) eurythermal ( $n=14$ ) and stenothermal ( $n=4$ ) organisms. Error bars represent  $\pm 1$  standard error.

#### 4. Discussion

In this study, we took a broad synthetic approach to examine patterns in acclimation of the HSR. Given the limited nature of the data, we were unable to comprehensively compare patterns within taxa, or control for non-independence in the data, and this might confound some of the patterns observed. Nevertheless, these results provide novel insight into acclimation of the HSR in diverse organisms adapted to different thermal niches. Experimental studies of closely related species will permit testing of the hypotheses generated from this comparative analysis.

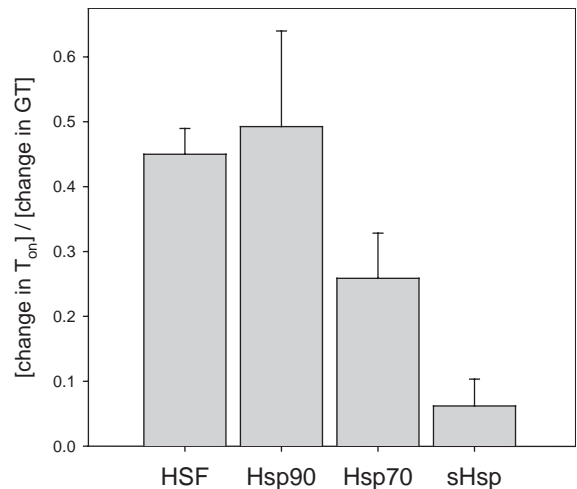


Fig. 2. Change in HSR induction temperature ( $T_{on}$ ) per unit change in growth temperature grouped for: heat-shock-factor—HSF ( $n=4$ ), Hsp90 ( $n=8$ ), Hsp70 ( $n=25$ ) and small Hsps (sHsps) ( $n=10$ ). Error bars represent  $\pm 1$  standard error.

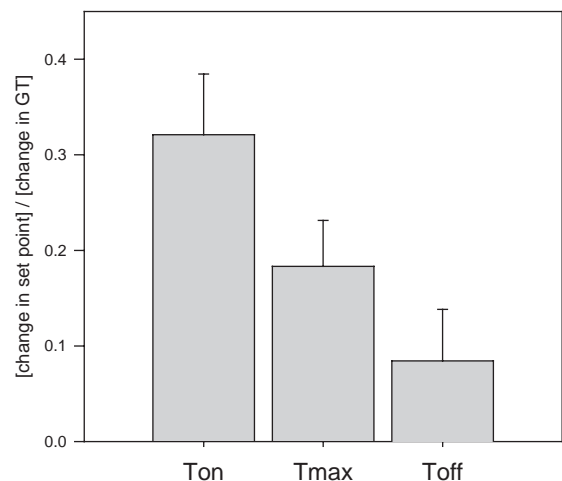


Fig. 3. Change in the set-point for HSR induction temperature— $T_{on}$  ( $n=31$ ), temperature of maximal response— $T_{max}$  ( $n=21$ ), and shut-off temperature— $T_{off}$  ( $n=14$ ) of the HSR. Error bars represent  $\pm 1$  standard error.

The temperature set-points of the HSR exhibit considerable plasticity in response to changing growth temperatures. While this is generally acknowledged (Buckley and Hofmann, 2002), it is unclear why other organisms do not exhibit such plasticity (Koban et al., 1987; Somers et al., 1989; Mason-Apps et al., 1990). We could not identify any common pattern in species lacking plasticity of the HSR. In general, however, a high proportion of plants displayed limited acclimation of the HSR—four of the six plant species examined

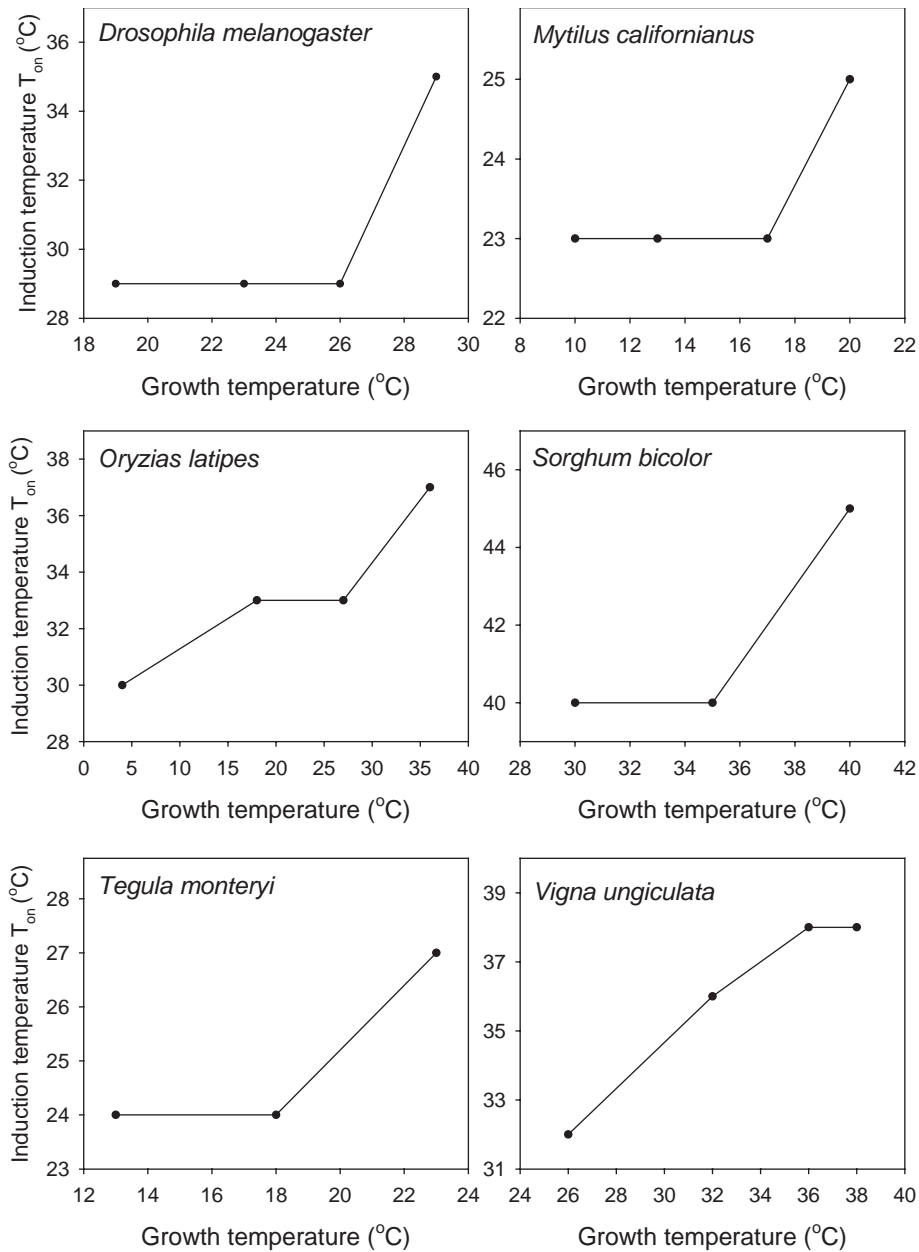


Fig. 4. Change in HSR induction temperatures in response to change in growth temperature for: fruit fly—*Drosophila melanogaster*, japanese killfish—*Oryzias latipes*, california mussel—*Mytilus californianus*, sorghum—*Sorghum bicolor*, monterey turban snail—*Tegula monteryi* and cowpea—*Vigna unguiculata*.

showed limited acclimation. Similarly, no consistent patterns were observed in the direction of change in the magnitude of expression of Hsps with growth temperature.

Contrary to our expectations, acclimation of induction temperature ( $T_{on}$ ) was lower in sessile organisms than in mobile organisms, and marginally (though non-significantly) greater in stenotherms than in eurytherms.

We suggest two possible, non-mutually exclusive, explanations for the observed results. First, equal changes in growth temperature might not be equally consequential for organisms adapted to different thermal niches. For example, a degree change in growth temperature may affect homeothermic organisms adapted to stable and constant temperatures very differently from eurythermic organisms that experience

wide diurnal and seasonal temperature fluctuations. Second, the lower acclimation in sessile organisms could be a consequence of broader tolerance ranges. In contrast to mobile organisms, plants and other sessile organisms are exposed to a broader range of temperatures, and consequently, are predicted to have wider tolerance ranges (Bradshaw, 1972; Huey et al., 2002). Increased temperature tolerance may be associated with a decrease in the acclimatory capacity (Ushakov et al., 1977; Stillman, 2003), and sessile organisms may rely on broader tolerance ranges rather than on plasticity.

Of the set-points of the HSR, the temperature of induction ( $T_{on}$ ) is the most plastic, while the shut-off temperature ( $T_{off}$ ) appears to be relatively fixed. This implies that acclimation to different growth temperatures may not influence responses to extreme temperatures close to the critical upper limits of organisms, like the shut-off temperatures of the HSR. Small Hsps showed limited acclimation compared to HSF, Hsp90, and Hsp70. This probably reflects the different roles of the various classes of Hsps. HSF, Hsp90, and Hsp70 play an integral part in the signal transduction pathway that results in the induction of the HSR, unlike small Hsps.

Generally, the relation of induction temperature and growth temperature was a step function, for which we propose the following explanation (Fig. 5). Behavioral regulation and physiological adjustments help organisms maintain regular homeostasis for temperatures around their optimal growth temperature, and acclimation of the HSR induction temperatures would be unnecessary within this range (OGT–A in Fig. 5). Increase in growth temperatures beyond this range, likely modify the morphology, physiology, or biochemistry (e.g. changes in leaf morphology in plants, changes

in cell membrane composition, etc.). Such changes would alter the sensitivity to high temperatures and result in a change of the HSR induction temperature. This acclimation is probably limited, and beyond some upper limits (B–C in Fig. 5), we will see no additional changes in the HSR induction temperatures.

Acclimation has been demonstrated in transcriptional activation of HSF, transcription of Hsp mRNA, and protein synthesis. However, factors upstream of HSF can also alter induction of the HSR. For example, growth temperatures alter membrane lipid composition and stability (Hazel, 1995; Logue et al., 2000), and membrane composition can alter HSR induction temperature independently of growth temperature (Carratu et al., 1996; Chatterjee et al., 1997, 2000; Horvath et al., 1998; Török et al., 2001). Factors upstream of the HSR, per se, may play an important role in acclimation of the HSR. A number of recent studies have examined mechanisms of HSR acclimation, but neglect possible mechanisms upstream of the HSR (Buckley et al., 2001; Buckley and Hofmann, 2002; Hofmann et al., 2002; Tomanek and Somero, 2002).

Acclimation of the HSR is now well documented, and additional studies may only be warranted if demonstrating novel aspects of plasticity in the HSR, or addressing unresolved questions. For example, the proximate mechanisms that underlie acclimation of the HSR are currently unknown. Experimental tests of the proposed cellular thermometer model of HSR induction have been equivocal in trying to explain acclimation of HSR induction temperatures (Buckley et al., 2001; Buckley and Hofmann, 2002; Tomanek and Somero, 2002). The minimum temperature necessary to elicit acclimation, the range of temperatures over which acclimation of the HSR is observed, and how these relate to environmental temperatures naturally experienced by organisms are also unknown. Is acclimation of the HSR apparent in stenothermal organisms in response to smaller changes in growth temperature, and limited to smaller ranges of temperatures than in eurythermal organisms? Finally, the ecological and evolutionary significance of such acclimation remains to be determined (Buckley and Hofmann, 2002; Tomanek and Somero, 2002). Understanding the causes and consequences of phenotypic plasticity of the HSR, and other acclimatory mechanisms (Stillman, 2003), will be especially important in predicting the impact of global climate change on natural populations.

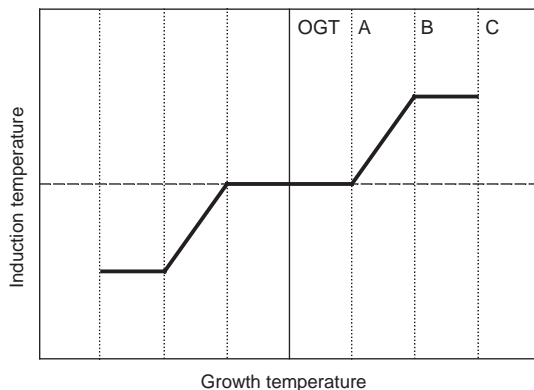


Fig. 5. Model for change in HSR induction temperature in response to change in growth temperature. OGT—optimal growth temperature, A—upper limit for buffering changes in growth temperature, B—upper limit of acclimation for induction temperatures, and C—critical upper limit.

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