Energy, Radiation and Temperature Regulation in Plants

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The temperature of any plant organ depends on the balance between incoming energy and energy loss. The energy balance comprises radiative transfer, sensible heat transfer, latent heat transfer and transfer to and from storage. Various biophysical mechanisms are available to plants, through manipulation of the energy balance terms, for temperature regulation.

Introduction

The temperature of any plant organ (trunk, leaf or root) depends on the balance between incoming energy and energy loss. Similarly, the temperature of the whole Earth–atmosphere system also depends on its energy balance and on the way the absorbed solar energy is distributed between different levels of the atmosphere and the ground. The surface air temperature varies over the Earth, depending on location and time of year, from as low as −69°C in Siberia in winter to as high as 58°C in deserts such as Death Valley, California, in summer. Although plant tissues can generally survive a wide range of temperatures (sometimes as low as that of liquid nitrogen or as high as found near geothermal active location), active growth is limited to a much narrower temperature range, with the optimum falling between about 15°C and 30°C for most plants. The environmental temperature and its seasonal variation are crucial factors determining the distribution of plants over the surface of the globe through the differing temperature sensitivities of survival, development, reproduction and production for different species.

When the rate of energy absorption exactly balances the rate of energy loss, the temperature of the absorbing tissue stays constant and is said to be in a ‘steady state’. However, when the rate at which energy is absorbed is greater than the rate of loss, the tissue in question will heat up, at a rate that depends on the difference between the incoming and outgoing fluxes and on its heat capacity. Therefore, large and massive tissues such as cactus tend to heat up more slowly than do thin tissues such as leaves, which track the changes in air temperature more closely.

An understanding of the energy balance of vegetation is also critical to the development of useful climate models for use in weather forecasting or in prediction of climate change. The global circulation models used for this work require detailed information on the soil–vegetation–atmosphere transfer (SVAT) processes to model accurately the significant feedback effects of vegetation on climate. Energy balance models are also useful for modelling of crop yield.

The different processes involved in energy exchange between plants and their environment will be discussed in some detail in subsequent sections of this article, with particular emphasis on radiative exchanges.

Energy Balance Equation

The main components of the energy exchange of any organ such as a leaf are illustrated in Figure 1 and comprise radiative transfer, sensible heat transfer, latent heat transfer and transfer to or from storage. The first law of thermodynamics (the principle of conservation of energy) states that energy cannot be created or destroyed, so we can write an equation showing the fact that the difference between all the energy fluxes (all in W m⁻²) into and out of the leaf equals the rate of energy storage (eqn [1]).

\[ \Phi_n - H - \lambda E = M + S \]  \[ 1 \]

where \( \Phi_n \) is the rate of net heat gain from radiation, \( H \) is the rate of net heat loss by what is called ‘sensible’ heat exchange by conduction and convection processes, \( \lambda E \) is the rate of ‘latent’ heat loss resulting from evaporation (or else a gain if condensation occurs), \( M \) is the net rate of heat storage in metabolic reactions (e.g. photosynthesis or respiration) and \( S \) is the net rate of physical heat storage (used to raise the temperature of the body).

Equation [1] applies equally to individual leaves and to whole canopies, though with some slight modifications. For example, for plant canopies it is common to separate the storage flux into heat transfer into the soil, \( G \), and the residual heat flux involved in changing the temperature of
Radiation exchange processes

Whether one is concerned with leaves or the whole Earth–atmosphere system, the main source of energy determining the temperature of objects is the sun. The incoming solar radiation has a peak in the visible and largely comprises wavelengths between about 0.2 m and 3 m. The solar spectrum is conveniently divided into the ultraviolet region (UV, 0.29–0.38 m) comprising up to 4% of the incoming short-wave (SW) energy; the photosynthetically active region (PAR, 0.38–0.71 m) comprising 21–46% of the SW energy; and the near infrared radiation (NIR, 0.71–4.0 m), which constitutes 50–70% of the incoming solar energy (Ross, 1981). The solar radiation is commonly called the short-wave to distinguish it from the long-wave radiation, otherwise known as ‘thermal infrared’ or ‘thermal’ radiation, that is given off by bodies as a function of their temperature. In view of the importance of radiation in the overall energy balance of vegetation, we will emphasize radiative exchanges and its controls in what follows. The net radiation absorbed (Φn), therefore, is the difference between the short-wave radiation from the sun that is absorbed and the net long-wave radiation emitted.

Short-wave radiation

Solar radiation reaches canopy surfaces both as the direct solar beam and as diffuse radiation. The diffuse component results primarily from atmospheric scattering by air molecules, clouds and aerosols (suspended particles) and from scattering from other vegetation and the soil; it therefore can come from any direction. The magnitudes of short-wave fluxes at the Earth’s surface change considerably during the course of the day and the year, both as a function of changes in the solar altitude and because of changes in transmission through the atmosphere (a strong function of atmospheric scattering). Optical properties of leaves and the canopy then determine the amount of energy actually absorbed (as outlined below).

The direct solar irradiance (Φs) on a horizontal surface is given by Lambert’s law as the radiant flux density normal to the solar beam multiplied by the sine of the solar elevation (θ = the cosine of the solar zenith angle). At the top of the atmosphere the irradiance is therefore equal to the flux density in the solar beam, the ‘solar constant’ (Φs0, equal to approximately 1370 W m⁻², varying by a few per cent from January to July as a result of the eccentricity of the Earth’s orbit round the sun), multiplied by the sine of the solar elevation. Because of attenuation in the atmosphere, the incoming short-wave irradiance at the ground therefore ranges from zero (at night) to around 1000 W m⁻² on a clear midday summer’s day with the sun more or less overhead.

Long-wave radiation

Long-wave radiation, confined to the spectral range between 3–4 μm and 100 μm, comes from the thermal emission from bodies at normal terrestrial temperatures. In practice, the long-wave radiation received by plant communities comes from the atmosphere and clouds as well as from other vegetation and the soil. The magnitude of this thermal radiation (ΦL, W m⁻²) emitted by any body is highly dependent on temperature of the emitter, changing with the fourth power of the temperature (in kelvin, K) according to the Stefan–Boltzmann law (eqn [2]).

$$\Phi_L = \varepsilon \sigma T^4$$  \[2\]

where the emissivity ε is a property of the surface emissivity and σ is the Stefan–Boltzmann constant (5.67 × 10⁻⁸ W m⁻² K⁻⁴). The emissivity of most vegetation is between about 0.95 and 0.995, while the emissivities of soils can be as low as 0.6 for sand and as high as 0.96 for dolomite.

The downward long-wave irradiance (ΦL,d) from the sky depends both on air temperature (and hence time of year) and particularly strongly on cloudiness: under cold clear skies in winter, ΦL,d may be around 200–250 W m⁻²; on cloudy days in summer, ΦL,d may reach around 380 W m⁻², reflecting the higher effective temperature of the emitter (Monteith and Unsworth, 1990). In contrast, the emitted upward long-wave radiation calculated according to eqn [2] can vary from 230 W m⁻² at −20 °C to
580 W m\(^{-2}\) at 45\(^\circ\)C, so the net long-wave flux in practice varies between around zero to a loss of 100 W m\(^{-2}\). The net long-wave radiation exchange is a major, yet often neglected, component of the overall energy budget of canopies, often being of similar magnitude to short-wave fluxes.

**Radiation regime within vegetation canopies**

The propagation of radiation, both short-wave and long-wave, through plant canopies is crucial both for photosynthesis and for the energy balance of different leaves. When radiation entering a canopy encounters a leaf or another canopy element such as a stem, it may be absorbed, reflected or transmitted. The corresponding optical properties of the vegetation elements, namely the absorption coefficient \((\alpha)\), reflection coefficient \((\rho)\), transmission coefficient \((\tau)\) and in the long-wave range the emissivity coefficient \((\varepsilon)\) are all functions of radiation wavelength \((\lambda)\). Figure 2 presents the optical properties of ‘typical’ leaves in the short-wave part of the spectrum.

The probability that a given ray of light interacts with a leaf or canopy element depends strongly on the canopy architecture, which includes factors such as the density distribution of leaves and branches in space and their orientation relative to the incident beam. For a detailed discussion of canopy geometry effects, see Monteith and Unsworth (1990). Because of the complexity of canopy architectures, it is common to make rather gross simplifications relating to leaf arrangement (often assumed to be random), orientation (often assumed horizontal) and shape (often assumed to be flat). The leaf area density of the whole canopy or its layers is described using the leaf area index \((L, \text{m}^2 \text{m}^{-2})\), which is the projected area of one side of the leaves per unit area of ground.

The penetration of radiation in plant canopies has been treated in many advanced studies (e.g. Ross, 1981). A convenient simplification was introduced by Monsi and Saeki in the 1950s, who pointed out that if one assumes that the canopy behaves as a turbid medium, one would expect the intensity of radiation to decrease exponentially with depth. This Beer’s law approximation has proved to be a reasonable assumption for most plant canopies. It describes well the general features of radiation penetration in horizontally homogeneous canopies so that the short-wave irradiance, \(\Phi_S\) (W m\(^{-2}\)), on a horizontal surface at any level can be described by eqn [3].

\[
\Phi_S = \Phi_{S,0} \exp(-kL)
\]  

[3]

where for downward radiation, \(\Phi_{S,0}\) is the irradiance at the top of the canopy, \(L\) is the cumulative leaf area index from the canopy top to a given height inside and \(k\) is an extinction coefficient. The value of \(k\) depends on the angular distribution of leaves in the canopy, the elevation angle of the incident radiation and the optical properties of the leaves (including reflectance, transmission and absorption). For a canopy where all the leaves are horizontal, \(k\) is equal to 1 for all angles of incident radiation, but for other leaf angle distributions other values of \(k\) are required. A more extensive discussion of the relationship of \(k\) to leaf angle can be found in Monteith and Unsworth (1990) and in Jones (1992).

**Sensible heat exchange processes**

The term sensible heat exchange is used to cover the transfer between the plant and its surroundings by the processes of conduction and convection. Conduction is the transfer of heat from an area of higher temperature to one of lower temperature by the transfer of energy between the adjacent molecules without mass transfer of the molecules, as for example in a metal bar. Convection, on the other hand, is the transfer of heat where the movement of the molecules themselves and their associated kinetic energy transfers the heat. The transfer of sensible heat from a leaf to the surrounding atmosphere therefore involves both conduction through the solid leaf material and conduction from the leaf surface to the adjacent air molecules, and the critical and largely rate-determining convection through the adjacent air layer – the boundary layer. In the study of the heat balance of plant leaves and canopies, the convective process are particularly important.

‘Forced’ or ‘free’ convection

When a body is exposed in a moving air stream (wind), the heat transfer is said to be by forced convection, but when the air movement is caused by temperature gradients in the

![Figure 2](https://via.placeholder.com/150)

Figure 2  The spectral properties of typical plant leaves over the visible and near infrared wavelengths. Leaf absorptance \((\alpha)\) is high in the visible or photosynthetically active (=PAR) wavelengths between 400 and 700 nm, while reflectance \((\rho)\) and transmittance \((\tau)\) are higher in the near infrared. After Jones (1992).
air itself (e.g. the rise of warm air over a radiator), the
process is termed free convection. Forced convection
dominalesmostsituationsinthefield,withfree
convection only becoming significant under very low wind
speeds and when leaf or canopy temperatures exceed the air
temperature by around 10°C or more. The efficiency of
heat transfer in the boundary layer depends on the
structure (eddies or turbulence) of the boundary layer
and on its thickness. The greater the degree of turbulence in
the air stream, the more rapid is heat or mass transfer, and
hence the lower the resistance to heat transfer. With
smoother surfaces and lower wind speeds, the air stream
tends towards a more laminar flow structure and hence
there is increased resistance to heat transfer.

Resistance analogues
The transfer of heat, whether by conduction or by
convection or even as latent heat in evaporation, is one
example of the general transport equation. In its integrated
form this can be written as eqn [4].

\[ \text{Flux density} = \text{proportionality constant} \times \text{driving force} \]

[4]

where the ‘driving force’ may be the temperature or
concentration gradient and the ‘proportionality constant’
or ‘transfer coefficient’ is a measure of the conductance of
the flow pathway to heat or mass transfer. Such a system
and its control are conveniently described by means of
‘resistance analogues’, using the analogy with Ohm’s law
for electrical current. Using resistance analogues one can
write the general equation [5] for the rate of sensible heat
loss (H) for an element having temperature \( T_{\text{leaf}} \) and an air
temperature (\( T_a \)).

\[ H = \rho_a C_P \frac{T_{\text{leaf}} - T_a}{r_{\text{ah}}} \]

[5]

where \( r_{\text{ah}} \) (s m\(^{-1}\)) is the resistance to sensible heat transfer
in the boundary layer (= the boundary layer resistance), \( \rho_a \)
the density of air and \( C_P \) is the specific heat capacity of
air. The factor \( \rho_a C_P \) is used to ensure that similar units are
used for the resistance for heat flux as for the resistance for
mass transfer (see below).

Some approximate formulae are available that enable
one to predict the heat transfer resistances between leaves
of different shapes and sizes as a function of air velocity.
For example, the resistance to heat transfer (s m\(^{-1}\)) under
laminar forced convection conditions for flat leaves is
approximated by eqn [6].

\[ r_{\text{ah}} = 151 \, (d/u)^{0.5} \]

[6]

where \( d \) is approximately the mean diameter of the leaf (m)
and \( u \) is the wind velocity (m s\(^{-1}\)). The values for other
shapes may be found in appropriate texts (Monteith and

Latent heat exchange
Central to the control of leaf and plant temperature is the
role of transpiration, with the latent heat required to
evaporate the water at the leaf’s surface (the latent heat of
evaporation of water, \( \lambda = 2.5 \, \text{MJ} \) for every kilogram of
water evaporated) being carried away with the evaporating
water. Mechanisms that control transpiration therefore
play an important role in regulating leaf temperature.

The rate of transpiration depends on the resistances to
both water vapour transfer through the stomata and water
vapour and heat transfer through the boundary layer
surrounding the leaf. The basic equation describing latent
heat flux (\( \lambda E \), W m\(^{-2}\)) from a leaf is eqn [7].

\[ \lambda E = \frac{M_w \lambda}{R (273 + T_a)} - \frac{e_s (T_{\text{leaf}}) - e_s (T_a)}{(r_W)} \]

[7]

where \( r_W \) (s m\(^{-1}\)) is the total resistance to water loss
(actually the sum of \( r_{\text{LW}} \), the ‘leaf’ resistance (largely due to
the stomata), and \( r_{\text{aW}} \), the boundary layer resistance for
water vapour), \( e_s (T_{\text{leaf}}) \) (Pa) is the saturation vapour
pressure at leaf temperature, \( e_s (T_{\text{aW}}) \) (Pa) is the vapour
pressure of the air surrounding the leaf, \( M_w \) (\( \approx \) 29) is the
effective molecular mass of air, and \( R \) is the gas constant
(\( = 8.3144 \, \text{J} \, \text{K}^{-1} \, \text{mol}^{-1}\)). The first term on the right-hand
side of this equation simply generates the correct units for
\( \lambda E \).

This form of equation is appropriate if one knows all the
relevant values, including the leaf temperature. More
frequently, however, one knows only the environmental
conditions, so a form of equation now known as the
‘Penman–Monteith equation’ that was first put forward by
Penman in 1948 and subsequently modified by Monteith in
1965 is used (eqn [8]).

\[ \lambda E = \frac{[s (\phi_s - G) + \rho_a C_p g_n D]}{s + (\gamma g_H/g_w)} \]

[8]

where \( G \) is the heat storage in the soil, \( D \) is the vapour
pressure deficit of the air (\( = e_s (T_a) - e_s \)), \( s \) is the slope of
the curve relating saturation vapour pressure to temperatu-
ture, \( \gamma \) is the psychrometric constant (\( = 66.1 \, \text{Pa} \, \text{K}^{-1}\) at
20°C) and \( g_H = 1/r_{\text{ah}} \) and \( g_w = 1/(r_{\text{aw}} + r_{\text{LW}}) \) are the
conductances to heat and water vapour. This is a very
powerful equation that allows one to predict \( \lambda E \) as a
function of physiological and environmental variables,
including transfer resistances, humidity and incident
radiation. For further discussion of the derivation and
operation of this equation see Monteith and Unsworth
(1990) and Jones (1992).

Heat and mass fluxes from canopies and from individual
leaves are determined by the same processes, though the
relevant boundary layers determining exchanges with
leaves may be only a few millimetres deep, while in
canopies the boundary layer of air that is influenced by the
vegetation may be tens or hundreds of metres in extent.
Energy storage and dynamics of leaf temperature change

Transfer of energy to and from storage in plant canopies and in the soil is an important factor regulating the dynamics of temperature in plant canopies. The energy flux going into physical storage (G for soil or S for leaf or canopy) is defined by the product of the rate of temperature change (ΔT/t) and the heat capacity of the system (C_p, where m is the mass in kg), according to eqn [9].

\[ S = (\Delta T/t) \cdot C_p m \]  

[9]

The specific heat capacity, \( C_p \) (J kg\(^{-1}\) K\(^{-1}\)) varies from 1010 for air, to 3500–4000 for leaves and 800–1550 for soils. In practice, when studying the energy balance of vegetation canopies, we frequently ignore the heat capacity of the leaves, because energy storage in such systems is dominated by the large mass of the soil.

When the rate of energy absorption exactly balances the rate of energy loss, the temperature stays constant and is said to be in a ‘steady state’. In a natural environment, however, environmental conditions are never constant, so temperatures vary continually. The rate of change of leaf temperature (dT\(_{\text{leaf}}\)/dt) depends both on the heat capacity of the tissue and the rate at which heat is gained or lost (as a function of the temperature difference between the leaf and the environment and the conductance to heat transfer). A convenient measure of the rate of change is the ‘rate constant’ for the change (τ), which is the time taken for 63% of the final change after a change of environmental conditions. The value of τ is given by eqn [10].

\[ \tau = \text{Heat capacity}/(\rho C_p \{1/r_a + s/\gamma r_w\}) \]  

[10]

where \( s/\gamma \) is the rate of increase of latent heat content to the increase of sensible heat content of saturated air. Examination of this equation shows that the time constant increases with heat capacity and with both the boundary layer and stomatal resistances because increasing resistances slow the rate of heat exchange.

Magnitudes of energy balance components

In general the energy use in metabolism and fluxes into or out of storage tend to be at least an order of magnitude less than the main energy fluxes (H, λE, \( \Phi_S \), \( \Phi_L \)) (see Table 1). For example the efficiency of conversion of incoming radiation into photosynthesis is normally in the range of only 1.5–3%. The instantaneous flux into soil, \( G \), may be up to 10% of \( \Phi_n \), i.e. up to 100 W m\(^{-2}\), though over a daily cycle night-time cooling approximately balances day-time heating.

The radiative fluxes dominate the overall energy balance of leaves or canopies. The energy absorbed from the incident solar radiation may be up to 1000 W m\(^{-2}\) and is largely dissipated through the three other mechanisms (\( \Phi_L \), λE and H). Under typical weather conditions the net long-wave radiation for a leaf or upper canopy layer may vary from \( 20 \) to \( -140 \) (W m\(^{-2}\)), where negative values indicate losses, so that canopies are mostly net long-wave emitters. When the vegetation is not subject to water deficits and the stomata are open, the leaf temperature is usually below air temperature so the term H is positive (the leaf gaining heat from the air by up to 100 W m\(^{-2}\)). In such cases all the absorbed short-wave radiation is dissipated through latent heat and long-wave radiation losses. When the water supply is poor and plant water deficits increase, the stomata close and the proportion of latent heat loss decreases and the sensible heat loss increases.

Control of Energy Exchanges in Canopies

Plant–atmosphere coupling

An important concept in our understanding of adaptations of plants to high and low temperature environments is that of ‘environmental coupling’. A canopy, a plant or a leaf are said to be ‘well coupled to the environment’ if changes in the environmental conditions are readily reflected in variables such as leaf temperature. A weakly coupled leaf responds only slightly to changes in, for example, air

Table 1  Approximate guide to typical magnitudes of instantaneous fluxes of energy into a leaf (W m\(^{-2}\)) where positive values represent energy gains by a grass lawn. Over periods of minutes or more, fluxes into storage in the leaf can be ignored

<table>
<thead>
<tr>
<th>Component</th>
<th>Night-time</th>
<th>Mid-day (W)(^{a})</th>
<th>Mid-day (S)(^{a})</th>
</tr>
</thead>
<tbody>
<tr>
<td>Short-wave (( \Phi_S ))</td>
<td>0</td>
<td>300 to 900</td>
<td>300 to 900</td>
</tr>
<tr>
<td>Long-wave (( \Phi_L ))</td>
<td>-50</td>
<td>-90 to -10</td>
<td>-100 to 0</td>
</tr>
<tr>
<td>Sensible heat (H)</td>
<td>-50 to 50</td>
<td>-400 to 200</td>
<td>-700 to -100</td>
</tr>
<tr>
<td>Latent heat (λE)</td>
<td>-60 to 50</td>
<td>-650 to -200</td>
<td>-100 to 0</td>
</tr>
<tr>
<td>Metabolism (M)</td>
<td>1 to 4</td>
<td>-25 to -5</td>
<td>-2 to -1</td>
</tr>
</tbody>
</table>

\(^{a}\)W represents values for a well-watered crop and S a water-stressed crop.
temperature. A leaf is relatively poorly coupled to or is isolated from the environment where there is a large boundary layer resistance to heat exchange, for example as occurs with large leaves or when there is a low wind speed.

Ecological Examples of Adaptations to Extreme Temperatures

In most environments plants are subject to extreme temperatures at some time of the year, so they have evolved a range of mechanisms enabling them to overcome the constraints imposed by these extremes. Perhaps the most general mechanism for tolerating extreme temperatures is through periodicity in growth, with the plants growing only during the time(s) of year when favourable temperatures occur, becoming dormant over the summer or winter periods when temperatures may fall outside the ideal range. Nevertheless, a wide range of mechanisms are available that extend the range of growth temperatures. In this article we concentrate only on the biophysical mechanisms that enable plants to maintain tissue temperatures within the normal physiological range, rather than considering those biochemical mechanisms that enable plants to tolerate extreme tissue temperatures, whether high or low. Further information on biochemical temperature tolerance mechanisms may be found in texts such as Long and Woodward (1988), Crawford (1989), and Larcher (1995).

Avoidance of high-temperature stress

Understanding of the leaf energy balance and also of gradients of environmental temperature is crucial to understanding the mechanisms available to plants for tolerance of temperature extremes. In high-temperature environments such as deserts, reduction of tissue temperatures is critical for continued productivity and even for survival. The range of adaptations that enable plants to tolerate high temperature environments include both metabolic adaptations that allow them to maintain function at higher than usual temperatures and mechanisms that favour a reduction of tissue temperature. Here we consider only the latter mechanism.

Cooling of leaves can come about either as a result of a reduction of energy input (e.g. reduced absorption of incident short-wave radiation) or as a consequence of increased heat loss (e.g. through enhanced evaporation, or sensible heat loss). The effects of some factors, however, such as those affecting the boundary layer resistance to heat transfer (e.g. leaf size or hairiness), can be very complex. For example, decreasing leaf size, and hence decreasing the boundary layer resistance, may increase evaporative cooling, but at the same time it increases sensible heat transfer, which, if the leaf is already below air temperature, will tend to cause the temperature to rise.

Reducing radiation absorption

The main ways in which leaves minimize the absorption of solar radiation and thus limit heating include the following:

- Leaves may have a high reflectivity, often achieved by having a reflective waxy cuticle or by the presence of a downy layer of epidermal hairs.
- A coating of hairs or spines can have another effect, that of shading the more sensitive tissues in the mesophyll of the leaf from high irradiances, so that heat is dissipated from the less thermally sensitive (sometimes even dead) tissues in the hairs or spines. For example, the apical spines and hairs in the cactus *Carnegia gigantea* can reduce the daily maximum meristem temperature by as much as 10 K (see Nobel, 1988).
- Adjustment of leaf angle in relation to the solar beam can also be important. Vertically orientation of leaves or phyllodes, as in *Eucalyptus* species, for example minimizes radiation interception at high solar angles around mid-day, when heating is of greatest concern. Many species, especially legumes, also have a marked capacity to orient their leaves in relation to the solar beam (so called ‘heliotropic’ movements). In conditions when such leaves are water stressed and the capacity for evaporative cooling is therefore limited, plants such as the cowpea (*Vigna unguiculata*) orient their leaves parallel to the solar beam, thus greatly reducing the amount of solar energy absorbed (e.g. Shackel and Hall, 1979).

Enhanced heat loss

A contrasting adaptation to tolerance of high temperatures is maximizing heat loss. Perhaps the most effective aspect of this is maximizing transpiration rate and its associated latent heat cooling. This involves having high stomatal conductance, either as a result of wide stomata or by having large numbers of stomata. Unfortunately, it is not a strategy that is universally applicable, as it relies on an ample supply of water, which is clearly not always available. Indeed, it is particularly under desert conditions when water supply is limited that high temperatures become of greatest concern and this mechanism loses its utility.

It is also possible to enhance sensible heat loss by reduction in the boundary layer resistance to heat transfer, for example by the development of narrow or dissected leaves. Of course, this is of no benefit if the air temperature is particularly high, as a reduction in the boundary layer resistance causes leaf temperature to track environmental temperature more closely. Nevertheless, possession of thin
leaves is a widespread adaptation of plants to hot environments.

**Environmental damping**

A number of plant species, including the cacti and other desert succulents, avoid the diurnal extremes of temperature (both hot and cold) as a result of the thermal damping conferred by their high thermal mass. The long time constant conferred by a combination of high thermal mass and the frequently associated high boundary layer resistance to sensible heat transfer can result in bulk tissue temperatures fluctuating only a small amount over a typical day. For example, calculations have indicated that where the mid-height surface temperature of *Carnegia gigantea* has a diurnal range of 33 K, the core temperature may fluctuate by only 6 K. Field observations have confirmed this calculation, with the maximal core temperature being as much as 13 K below the maximum surface temperature.

Another strategy found in some other desert species is for temperature-sensitive meristems to be located either below ground level (thus taking advantage of the thermal damping provided by the soil), or else well above the ground surface. The advantage of this latter approach is that the sensitive meristems are located well above where the highest temperatures occur on a diurnal cycle.

**Avoidance of low-temperature stress**

Avoidance of low-temperature stresses has two aspects, both the avoidance of temperatures that lead to tissue damage through frost and tissue freezing or chilling injury, and the mechanisms that raise tissue temperatures closer to the physiological optimum for growth than would otherwise occur.

**Biophysical temperature elevation**

In arctic and other cold environments, the growing season is very short. There is therefore a premium on mechanisms that can raise tissue temperatures above the rather low air temperatures that occur in the spring. Such mechanisms are widespread in arctic species and are based on a combination of mechanisms to maximize solar heat gain and mechanisms to minimize loss of the heat gained.

A common feature of many arctic plants is the way in which flowers can act as solar ‘traps’ or reflectors, often focusing the sun’s rays on the sensitive reproductive tissues. Indeed it has been shown that this mechanism can raise tissue temperatures in the centres of flowers of plants such as *Dryas octopetala* by as much as 5–10 K above air temperature in the immediate vicinity (Crawford, 1989; Larcher, 1995). More important, however, is the reduction of heat loss that is achieved as a result of the low stature of most arctic plants, so that they remain within the relatively undisturbed boundary layer where wind speeds, and thus rates of heat loss, are low. Tissue temperatures in this boundary layer within a few centimetres of the ground may be 10 or more degrees above air temperatures, as a result of this reduced heat loss.

Another possible biophysical mechanism involved in freezing avoidance could involve the so-called freezing exotherm, which refers to the heat released as some of the tissue or external water freezes, thus releasing the latent heat of fusion. This mechanism can delay cooling of critically sensitive tissues when exposed to low temperatures, but in general is unlikely to contribute much to species differences in frost tolerance.

**Thermogenesis**

There has been much speculation in recent years that plant tissues in low-temperature environments can actually raise their temperatures significantly by means of thermogenic reactions. In addition to the widespread cytochrome oxidase respiratory pathway (COP), plants possess a respiratory pathway that is not found in animals called the alternative oxidase pathway (AOP). This latter pathway is less efficient in the conversion of carbohydrate into ATP, as a result potentially releasing more of the bound energy as heat.

It is now well known that the spadices (flower spikes) of a number of species in the Araceae including the skunk cabbage (*Symlocarpus foetidus*) and the arum lily (*Philodendron selloum*) can heat up well above air temperature (by as much as 35 K) as a result of operation of the AOP during their flowering period in the early spring (see Seymour, 1997). This thermogenesis reaction is now believed to result not directly from the fact that these tissues respire largely through the AOP, but rather because of the very high absolute respiration rates that occur at this time in these tissues.

**The tropical alpine form**

The characteristic life form of high mountains in the tropics is the pachycaul form of herbaceous plants such as *Lobelia* and *Senecio*, which form dense rosettes of leaves with a large erect tree-like flowering stalk. In these plants the dense rosette of leaves closes at night and, together with the dense covering of hairs on the leaves, probably acts as insulation, protecting the sensitive tissues from frost. In the higher regions of the tropics, frosts are likely at any time of year, so indigenous plants are unable to depend on seasonal frost-hardening to survive.

**References**

Further Reading


Heat Shock Proteins (HSPs): Structure, Function and Genetics

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Heat shock proteins are evolutionarily well-conserved, ATP-binding proteins that function as molecular chaperones in facilitating protein folding and degradation. They are expressed constitutively in various cell types, but the expression of certain family members is strongly induced in response to stress stimuli.

Introduction

The stress response was discovered in 1962 by Ferruccio Ritossa, who detected a new puffing pattern upon heat shock in the polytene chromosomes of the fruitfly Drosophila melanogaster. Today it is known that all organisms share a common molecular stress response, which includes a dramatic change in the pattern of gene expression and an elevated synthesis of a family of stress-induced proteins (heat shock proteins, Hsps). Expression of Hsps usually results in repair of damaged and misfolded proteins and survival of the cell, mainly through their chaperone function. The diverse inducers of Hsp synthesis include (1) environmental stress, such as exposure to heat shock, oxidants and heavy metals; (2) nonstress conditions, including certain stages during normal cell growth, development and differentiation; and (3) various disease states, such as ischemia and inflammation. The common signal generated by various stimuli is likely to be protein damage, but the exact stress-sensing mechanism in the cell is currently not known.

Heat Shock Protein Families

The group of stress-induced proteins of mammalian cells and their Escherichia coli homologs include major protein families of 100, 90, 70, 60 and 40 kilodaltons (kDa), as well as the small Hsps of 15–30 kDa (Table 1: Lindquist and Craig, 1988). The majority of studies of the heat shock response have focused on the evolutionarily conserved eukaryotic Hsps of 70 kDa. Human cells contain several Hsp70 family members with common structural and functional properties but different stress inducibilities. The Hsp70 family includes the highly stress-inducible Hsp70, the constitutively expressed heat shock cognate protein Hsc70, the mitochondrial glucose-regulated protein Grp75, and Grp78/BiP, the expression of which is increased upon various stimuli that interfere with the endoplasmic reticulum (ER) function, such as calcium depletion and glucose starvation.

Structure of Hsps

Most Hsps consist of a highly conserved amino (N)-terminal adenosine triphosphatase (ATPase) domain and a carboxy (C)-terminal substrate-binding domain (Figure 1). In Hsp70, for example, binding of ATP drives conformational changes in the peptide-binding domain, altering its affinity for substrates. In its ATP-binding state, Hsp70 and the unfolded polypeptide form a weak complex, which is stabilized upon ATP hydrolysis. The substrate is released upon entering a new ATP cycle. Repeated cycles of binding and release of an unfolded polypeptide from Hsp70 are modulated by various cochaperones, such as members of the Hsp40 family.

Table 1 Major heatshock protein families

<table>
<thead>
<tr>
<th>Family</th>
<th>E. coli hom.</th>
<th>Function in eukaryotes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hsp100</td>
<td>Clp</td>
<td>ATP-dependent protein disaggregation and degradation, thermotolerance</td>
</tr>
<tr>
<td>Hsp90</td>
<td>HtpG</td>
<td>Conformational regulation of signal transduction molecules</td>
</tr>
<tr>
<td>Hsp70</td>
<td>DnaK</td>
<td>ATP-dependent stabilization of hydrophobic regions in extended polypeptides, protein transport, thermotolerance</td>
</tr>
<tr>
<td>Hsp60/</td>
<td>GroEL/</td>
<td>Chaperonins: ATP-dependent facilitation of folding to the native state</td>
</tr>
<tr>
<td>Hsp10</td>
<td>GroES</td>
<td>Binding to unfolded polypeptides, cochaperone for Hsp70</td>
</tr>
<tr>
<td>Hsp40</td>
<td>DnaJ</td>
<td>ATP-independent prevention of protein aggregation upon various stress stimuli, thermotolerance</td>
</tr>
<tr>
<td>Small Hsps</td>
<td>Ibp</td>
<td></td>
</tr>
</tbody>
</table>

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Heat Shock Proteins (HSPs): Structure, Function and Genetics

Functions of Heat Shock Proteins

The correct folding of many proteins depends on a preexisting protein machinery composed of molecular chaperones, which also function as a means of defense against protein denaturation and aggregation caused by stress. Molecular chaperones mediate folding of newly translated polypeptides in the cytosol and within organelles, and also stabilize proteins to prevent aggregation. The Hsp70 proteins in mitochondria and in the ER have principal roles in protein translocation. Hsp90 and its cochaperones are also involved in signal transduction.

Chaperone function

The key characteristic of chaperones is their ability to recognize nonnative conformations of polypeptides, protecting them from aggregation by binding to hydrophobic residues that have been exposed during translation or translocation or following stress-induced damage. The newly synthesized polypeptides can be maintained in a folding-competent state, and efficient folding is achieved by cycles of controlled binding and release of the polypeptide from the chaperone. Hsp70 proteins function as ATP-dependent molecular chaperones by assisting folding of newly translated polypeptides, guiding protein translocation across organelar membranes, disassembling oligomeric protein structures, facilitating proteolytic degradation of unstable and abnormal proteins, and controlling the biological activity of regulatory proteins (Bukau and Horwich, 1998). In addition, the constitutively expressed Hsc70 functions as an ATPase in the disassembly of clathrin-coated vesicles during transport of membrane components through the cell. (See Protein Folding and Chaperones.)

A functionally distinct chaperone family, the chaperonins, has also been implicated in protein folding in the cytosol. Chaperonins are oligomers composed of back-to-back rings of identical or closely related, rotationally symmetric subunits of approximately 60 kDa. The chaperonins assist a variety of newly synthesized, partially folded and newly translocated proteins to reach their native forms by enclosing the whole nonnative polypeptides within the chaperonin central cavity and facilitating folding through multiple ATP hydrolysis-dependent cycles of binding and release (Netzer and Hartl, 1998). The chaperonins include the widely studied bacterial GroEL and its cochaperonin GroES, the closely related eukaryotic mitochondrial Hsp60/Hsp10 complexes, and the cytosolic TCP1-ring complex (TRiC). The chaperonins and Hsp70 act sequentially at least in mitochondria, where unfolded proteins imported from the cytosol first interact with Grp75 and thereafter are transferred to Hsp60.

α-Crystallins were originally recognized as eye lens proteins, but owing to their heat-inducibility they have also been referred to as small Hsps. The small Hsps have a role in a wide range of cellular activities, including functioning as ATP-independent molecular chaperones in polymerization of actin filaments (Narberhaus, 2002). The small Hsps associate to form large oligomeric structures, which can form stable complexes with folding intermediates in order to protect them from irreversible aggregation. Release and refolding of intermediates into the native state requires close cooperation with other cellular chaperones.

Hsps and thermotolerance

Preconditioning (i.e. an exposure to nonlethal temperatures) protects cells from subsequent exposure to higher, otherwise lethal, temperatures or other stresses, thereby inducing thermotolerance. The first indication that Hsps could be involved in this process was based on the correlation between their inducible synthesis and development of thermotolerance. Subsequently, Hsp70, Hsp27 and Hsp110 have been shown to participate in conferring cellular resistance against stress.

The acquisition of thermotolerance has potent clinical relevance (Morimoto and Santoro, 1998). Overexpression of Hsp70 protects myocardium from ischemia and reperfusion injuries, most probably by preventing protein aggregation during ischemic stress. Increased Hsp70 levels can be obtained, for example, by predisposing the heart to short-term stress, thereby improving the ability of the heart to withstand subsequent, more severe stress. Upon cerebral ischemia in rat, the hippocampal neurons expressing increased levels of Hsp110 and Hsp70, owing to transient short ischemia, acquire tolerance against subsequent severe ischemia. Interestingly, anti-inflammatory drugs, such as sodium salicylate and indomethacin, promote acquisition of thermotolerance by potentiating Hsp70 expression already at febrile-range temperatures. Increased expression of ubiquitin, which is a bona fide stress protein, has been detected during
ischemia, and degradation of proteins via the ubiquitin system appears to be part of the neuronal recovery mechanism following cerebral ischemia.

**Hsps and protein degradation**

Upon extreme stress, the damaged proteins cannot be successfully refolded but must be destroyed. This is accomplished mainly through the ubiquitin–proteasome-mediated proteolysis, an essential pathway of protein degradation. The ubiquitin–proteasome pathway can be blocked by specific inhibitors, resulting in accumulation of abnormal and damaged proteins, which in turn activates the heat shock response with Hsp70 and other molecular chaperones. (See Protein Degradation and Turnover.)

Protein deposition and aggregate formation are implicated in the disease pathogenesis of a number of depository neurodegenerative diseases, such as cystic fibrosis, Alzheimer and Parkinson diseases, prion disorders and polyglutamine diseases (e.g. Huntington disease). In a mouse model for a polyglutamine disease, high levels of Hsp70 afford protection against neurodegeneration, suggesting that the protein-folding machinery is key to the control and regulation of neuronal protein aggregation. (See Protein Misfolding and Degradation in Genetic Disease.)

Among known chaperones, Hsp104, a member of the Hsp100 family in the budding yeast *Saccharomyces cerevisiae*, is unique in a sense that it does not prevent aggregation but is able to dissolve aggregates of high molecular weight (Glover and Lindquist, 1998). It is also indispensable for acquisition of thermotolerance in yeast. Refolding from the aggregated state requires not only Hsp104, but also specific interactions with additional chaperones, such as Hsp70 and Hsp40. Whether a functional mammalian homolog of Hsp104 exists remains to be shown.

**Hsp90 chaperone function extends to signal transduction**

The 90-kDa Hsps are a class of abundantly and constitutively expressed chaperones that undergo an open to close conformational change after addition of ATP, analogous in many respects to the Hsp70 proteins. Hsp90 is highly effective in converting the denatured substrate in the absence of ATP to a folding competent state, which can subsequently be refolded upon addition of Hsp70, a cochaperone, and ATP. The Hsp90 homologs in the ER and mitochondria of higher eukaryotes are termed Grp94 and TRAP-1 respectively.

The Hsp90 system is one of the best-known chaperone machines in eukaryotes. The Hsp90 proteins are specifically involved in the folding and conformational regulation of a number of signal transduction molecules, such as protein kinases (e.g. Src and Raf) and transcription factors, of which steroid hormone receptors (SHR) are well-characterized examples of cytosolic proteins that rely on molecular chaperones for folding (Figure 2; Richter and Buchner, 2001). A functional basis for SHR–chaperone interactions is the establishment and maintenance of the receptor in an inactive state. Association of client proteins with Hsp90 complexes in general is important for the stability and functional repression of the signal transducer; that is, inhibition of their abilities to bind DNA, to oligomerize and to interact with the transcriptional coregulatory proteins in the absence of ligand binding or other stimulatory signal. The role of Hsp90 in signal transduction is further supported by the various signal transduction phenotypes in *D. melanogaster* that are heterozygous for Hsp90.

**Quality control in ER**

The rough ER is an organelle that is specialized for folding and assembly of polypeptides, a property reflected by high concentrations of molecular chaperones, such as Grp78/BiP and the membrane-bound chaperone calnexin, in ER. The ER-resident proteins contain at their C-terminus a KDEL-motif of four amino acids, which is responsible for the retention in ER. Protein misfolding and aggregation and aggregation of unfolded proteins in the ER induce a signal that selectively activates transcription of genes encoding ER-resident chaperones. Consequently, Grp78/BiP and the other chaperones bind to the abnormal proteins, facilitating their solubilization. This cascade has been termed the unfolded protein response (UPR).

The assembly and modification of proteins of the secretory pathway occur in the ER and are a prerequisite for export to the Golgi apparatus. Grp78 has a key role in monitoring protein transport through the cell: if Grp78/BiP is not itself secreted but remains in the ER, proteins bound to it will be also retained. The quality control (i.e. preferential recognition of unfolded and incorrectly assembled proteins by molecular chaperones) provides a means of delaying secretion until the proteins are accurately folded and modified, and thereby also ensures that the abnormal proteins never leave the cell or reach its surface (Ellgaard and Helenius, 2001). The aberrant proteins are retained in the ER in complexes with molecular chaperones until they are degraded by the ubiquitin–proteasome pathway.

**Antiapoptotic proteins**

Stimuli that induce the stress response may also induce apoptosis, depending on the duration and intensity of the stress signal. Apoptosis (or programed cell death)
is an active form of cellular self-destruction that is essential to maintaining homeostasis in an organism. Central to apoptotic signaling is the apoptotic effector machinery, including a cascade of proteases called caspsases, the activation of which ultimately dissembles the cells. Apoptosis is critical during embryonic development and in removing damaged cells, and its malfunction can even lead to development of cancer. Small Hsps, especially Hsp27, function as antiapoptotic proteins upon oxidative stress by inhibiting cytochrome c release. The inducible Hsp70 prevents apoptosis by interfering with the signaling cascades and by impairing the activation of effector caspsases (Jäätteli, 1999).

The ability of Hsps to prevent apoptosis induced by several anticancer drugs as well as other stimuli suggests that they could enhance tumorigenesis and limit the effectiveness of cancer therapy. Indeed, Hsp70 and Hsp27 are abundantly expressed in many malignant human tumors, and their expression in certain cancer types, such as breast cancer, correlates with poor prognosis and resistance to therapy.

Genetics

The Hsp family members are encoded by multiple genes residing in various chromosomal loci in the human genome. The hsp70 and hsp90 multigene families will be introduced more closely below.

Hsp70 genes

The Hsp70 loci within the major histocompatibility complex (MHC) class III region on chromosome 6 contains three intronless genes (Table 2). The hsp70-1 and hsp70-2 genes (HSPA1A and HSPA1B, respectively) are 12 kb apart and encode an identical heat-inducible protein, but have divergent 5' and 3' untranslated region (UTR) sequences. The third gene, HSPA1L (also known as hsp70-HOM), is located 4 kb telomeric to HSPA1A. HSPA1L encodes a constitutively expressed protein that shares 90% identity with hsp70-1, the sequences differing most in the C-terminal 100 amino acids. Comparison of the human hsp70-1,
hsp70-2 and hsp70-HOM genes with their rat and mouse counterparts indicates that the interspecies similarity is higher than the intraspecies similarity. The same applies to the 3' UTR sequences of these genes, each of the three genes being more closely related to the respective orthologous gene than to the other two genes. The intronless hsp70B0 gene (HSPA6) encodes a more basic, heat-inducible protein of 70 kDa sharing a 77% similarity with hsp70-1. hsp70B, which is approximately 95% identical to hsp70B0, is located on the same chromosome (1q23.1). Hsp70B messenger ribonucleic acid (mRNA) expression has been detected upon heat shock in fibroblasts. Despite transcription, the hsp70B gene (HSPA7) has been suggested to be nonfunctional.

HSPA2 (encoding Hsp70T), located on chromosome 14, is an intronless human homolog of the murine hsp70-2 gene abundantly expressed in testis and skeletal muscle. Male Hsp70-2–/– mice lack postmeiotic spermatids and mature sperm and are infertile, whereas female mice undergo normal meiosis and are fertile.

**Hsp90 genes**

The Hsp90 proteins are mainly encoded by two genes, HSPCA (hsp90α) and HSPCB (hsp90β), on chromosomes 1 and 6, respectively. A gene family for human hsp90α has also been identified (Table 3). This family contains four HSPCA-like genes (HSPCAL1–4), of which at least one (HSPCAL4) encodes a functional protein. HSPCB, in contrast, has two pseudogenes, one on chromosome 4 and the other on chromosome 15. Hsp90β-deficient mice show placental abnormalities, suggesting that the role of Hsp90β in placental development cannot be compensated by Hsp90α alone.

**Regulation of Heat Shock Gene Expression**

Heat shock gene expression is regulated at multiple levels. Regulation at the transcriptional level through activation of specific transcription factors, HSFs, is most widely studied and will be described in more detail in this article. Human Hsp70-1 is expressed both constitutively and inducibly, and the expression pattern is reflected by the Hsp70-1 promoter structure (Figure 3).

Although the heat shock response is a ubiquitous phenomenon, certain cells show a reduced induction of heat shock gene expression upon stress stimuli. For example, in some human leukemia and retinoblastoma cell lines, induction of Hsp70-1 transcription upon heat shock is diminished, leading to impaired thermotolerance. This appears to be due to hampered binding of HSF1 and other transcription factors to the hsp70-1 promoter, possibly because of a high methylation state. The suppression seems to be specific to the hsp70-1 gene (HSPA1A), as HSPA1B and HSPA6 mRNA expression is induced normally. Unresponsiveness to heat shock in several mouse cell lines is consistently associated with a complete loss of Hsp70 promoter accessibility and extensive methylation. These findings indicate that distinct hsp70-encoding genes might contribute to the heat shock response in a cell-type-dependent manner.

Table 2 Human hsp70 multigene family

<table>
<thead>
<tr>
<th>Gene</th>
<th>Symbol</th>
<th>Chromosome</th>
<th>Expression</th>
<th>Protein size (aa)</th>
<th>OMIM</th>
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</thead>
<tbody>
<tr>
<td>hsp70</td>
<td>HSPA1A</td>
<td>6p21.3</td>
<td>Inducible</td>
<td>641</td>
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<td>6p21.3</td>
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<td>641</td>
<td>603012</td>
</tr>
<tr>
<td>hsp70-HOM</td>
<td>HSPA1L</td>
<td>6p21.3</td>
<td>Constitutive</td>
<td>641</td>
<td>140559</td>
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<tr>
<td>hsp70B'</td>
<td>HSPA6</td>
<td>1q23.1</td>
<td>Inducible</td>
<td>643</td>
<td>140555</td>
</tr>
<tr>
<td>hsp70B</td>
<td>HSPA7</td>
<td>1q23.1</td>
<td>Inducible</td>
<td>n.a.</td>
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</tr>
<tr>
<td>hsp70T</td>
<td>HSPA2</td>
<td>1q24.1</td>
<td>Constitutive</td>
<td>639</td>
<td>140560</td>
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<tr>
<td>n.a.</td>
<td>HSPA3</td>
<td>21</td>
<td>n.a.</td>
<td>n.a.</td>
<td>140570</td>
</tr>
<tr>
<td>hsc70, hsp73</td>
<td>HSPA8</td>
<td>1q23.3–q25</td>
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<td>646</td>
<td>600816</td>
</tr>
<tr>
<td>grp78, BiP</td>
<td>HSPA5</td>
<td>9q34</td>
<td>Inducible</td>
<td>654</td>
<td>138120</td>
</tr>
<tr>
<td>grp75, mortalin</td>
<td>HSPA9B</td>
<td>5q31.1</td>
<td>Inducible</td>
<td>679</td>
<td>600548</td>
</tr>
</tbody>
</table>

OMIM: Online Mendelian Inheritance in Man Database in NCBI’s Entrez System; n.a.: not available; aa: amino acids.

Table 3 Hsp90 family

<table>
<thead>
<tr>
<th>Gene</th>
<th>Symbol</th>
<th>Chromosome</th>
<th>OMIM</th>
</tr>
</thead>
<tbody>
<tr>
<td>hsp90α</td>
<td>HSPCA</td>
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</tr>
<tr>
<td>hsp90α-like 1</td>
<td>HSPCAL1</td>
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<tr>
<td>hsp90α-like 2</td>
<td>HSPCAL2</td>
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<td>hsp90α-like 3</td>
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<tr>
<td>hsp90α-like 4</td>
<td>HSPCAL4</td>
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<tr>
<td>hsp90β</td>
<td>HSPCB</td>
<td>6p12</td>
<td>140572</td>
</tr>
</tbody>
</table>

Heat Shock Proteins (HSPs): Structure, Function and Genetics
CCAAT box in its hsp70-HOM gene, in contrast, contains neither an HSE nor a boxes, which contribute to the basal expression of Hsp70-1. The genes (thereby mediating transcription of the heat shock HSFs acquire DNA-binding activity to the HSE, heavy metals and bacterial and viral infections, most inducers, such as elevated temperatures, oxidants, form under normal conditions. In response to various are synthesized constitutively and stored in a latent promoter of the strictly heat-inducible Hsp70B lacks TATA and CCAAT boxes, which contribute to the basal expression of Hsp70-1. The hsp70-HOM gene, in contrast, contains neither an HSE nor a CCAAT box in its 5’ flanking region.

Heat shock transcription factors

Like many inducible transcriptional regulators, HSFs are synthesized constitutively and stored in a latent form under normal conditions. In response to various inducers, such as elevated temperatures, oxidants, heavy metals and bacterial and viral infections, most HSFs acquire DNA-binding activity to the HSE, thereby mediating transcription of the heat shock genes (Figure 4).

Since the isolation of a single HSF gene from S. cerevisiae and D. melanogaster, several members of the HSF family have been found in vertebrates and plants, such as HSF1, HSF2 and HSF4 from mouse and human (Pirkkala et al., 2001). In vertebrates, HSF1 is the general HSF that mediates stress-induced heat shock gene expression in response to environmental stressors. HSF2 is refractory to classical stress stimuli but is activated during nonstressful conditions. HSF4 is known to induce heat shock gene expression upon heat stress, but otherwise its function is largely uncharacterized. Avian cells express a unique HSF3 in addition to the mammalian HSF1 and HSF2 homologs. Disruption of the HSF3 gene results in impaired heat shock response and loss of thermostolerance in avian cells expressing normal levels of HSF1. This is due to the inability of HSF1 to trimerize even upon heat shock in the absence of HSF3, indicating that HSF3 directly influences HSF1 activity and has a dominant role in the regulation of avian heat shock response.

The yeast homolog of vertebrate HSF1 is essential for cell growth and viability, suggesting a role in regulation of basal heat shock gene expression. HSF1 knockout mice have severe defects in the chorioallan-


**Further Reading**


**Web Links**

Heatshock 70 kDa protein 1A (HSPA1A); LocusID: 3303. LocusLink: http://www.ncbi.nlm.nih.gov/LocusLink/LocRpt.cgi?l=3303

Heatshock 70 kDa protein 1B (HSPA1B); LocusID: 3304. LocusLink: http://www.ncbi.nlm.nih.gov/LocusLink/LocRpt.cgi?l=3304

Heatshock 70 kDa protein 1-like (HSPA1L); LocusID: 3305. LocusLink: http://www.ncbi.nlm.nih.gov/LocusLink/LocRpt.cgi?l=3305

Heatshock 90 kDa protein 1, α-like 3 (HSPCAL3); LocusID: 3324. LocusLink: http://www.ncbi.nlm.nih.gov/LocusLink/LocRpt.cgi?l=3324

Heatshock 90 kDa protein 1, β (HSPCB); LocusID: 3326. LocusLink: http://www.ncbi.nlm.nih.gov/LocusLink/LocRpt.cgi?l=3326


