

# Human Resource & Neuroscience:

## The role of Neuroscience in Coping with Organisational Stress.

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**Abstract :** This paper aims to study the linkage between neuroscience and handling of Occupational Stress at Workplace . Human Resources is no more a mere support function but rather an important contributor to revenue earning capacity of the firm. Hence , the terminology Human Capital Management has almost replaced Human Resource Management . Man , is more an asset to the organisation and the economy investing upon whom , yields a greater ROI than any other asset. Hence , keeping a tap on their physiological configuration stands to be of paramount importance. This paper aims to study how the structure and function of Human Brain and the Nervous System tends to affect ones' coping mechanism at workplace & as well impacts the susceptibility of an incumbent to experience Work Stress. This study is of an exploratory nature , predominantly using literature review as its main mode of gathering data. Organisations should adopt engagement practices which doesn't not only look good in the powerpoints of the review meetings but rather be neurobiologically rewarding and only then , stress can be dealt with properly. As because one doesn't have control over the stressors but certainly does have control over their dealing strategies.

**IndexTerms** - Neuroscience , Human Resource Management , Stress , Organisational Stress , Occupational Stress , Coping .

### I. INTRODUCTION

Neuroscience examines the structure and function of the human brain and nervous system. Neuroscientists use cellular and molecular biology, anatomy and physiology, human behavior and cognition, and other disciplines, to map the brain at a mechanistic level.

The term 'Computational neuroscience' was coined by Eric L. Schwartz, at a conference to provide a review of a field, which until that point was referred to by a variety of names, such as Neural modeling, Brain theory, and Neural Networks. Later, Hubel & Wiesel discovered the working of neurons across the retina, in the primary visual cortex (the first cortical area).

### Theoretical neuroscience

Neuroscience encompasses approaches ranging from molecular and cellular studies to human psychophysics and psychology. The aim of computational neuroscience is to describe how electrical and chemical signals are used in the brain to interpret and process information. This intention is not new, but much has changed in the last decade. More is known now about the brain because of advances in neuroscience, more computing power is available for performing realistic simulations of neural systems, and new insights are being drawn from the study of simplified models of large networks of neurons.

Humans have an estimated hundred billion neurons, or brain cells, each with about a thousand connections to other cells. One of the great challenges of modern neuroscience is to map out all the networks of cell-to-cell communication—the brain circuits that process all thoughts, feelings, and behaviors. The resulting picture, emerging bit by bit, is known as "the connectome." The ability of the brain to elaborate new connections and neuronal circuits—neuroplasticity—underlies all learning.

Biology and psychology unite in the field of neuroscience, to tackle questions such as the brain's role in pain perception or the underlying cause of Parkinson's disease. Computer simulations, imaging, and other tools give

researchers and medical experts new insight into the physical anatomy of the brain, its five million kilometers of wiring, and its relationship to the rest of the mind and body.

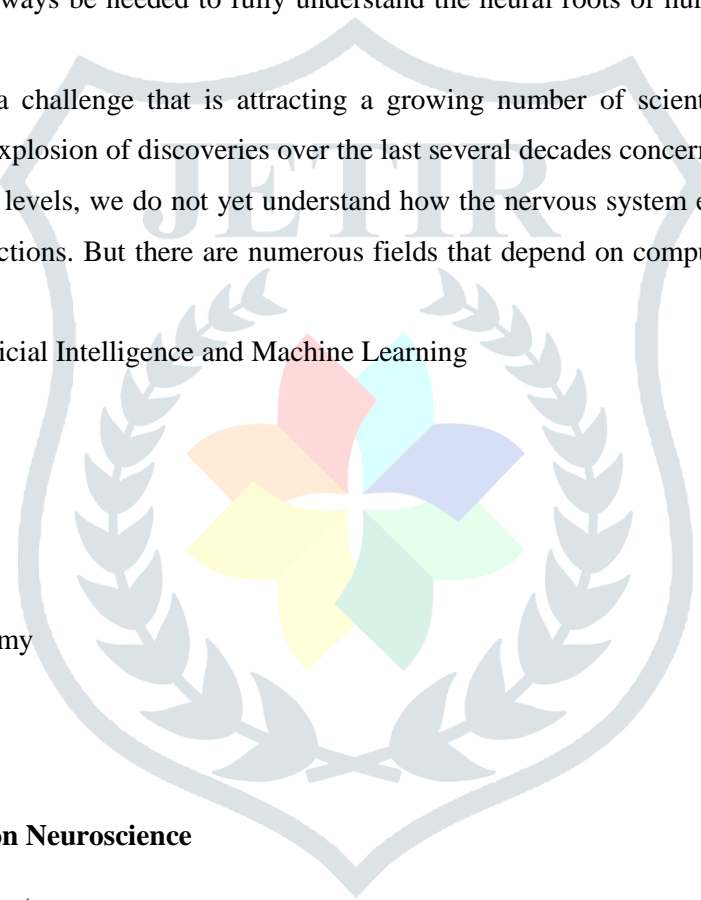
### How Neuroscience Helps Us Understand the Mind and Brain

Just as computers are hard-wired with electrical connections, the brain is hard-wired with neural connections. These connections link together its various lobes and also link sensory input and motor output with the brain's message centers, allowing information to come in and be sent back out.

One major aim of current neuroscience research, then, is to study how this wiring works and what happens when it's damaged. New developments in brain scanning allow researchers to see more detailed images and determine not only where there may be damage but also how that damage affects, for instance, motor skills and cognitive behavior in conditions like multiple sclerosis and dementia.

A rapidly expanding discipline, neuroscience findings have grown by leaps and bounds over the past half-century. More work, however, will always be needed to fully understand the neural roots of human behavior, consciousness, and memory.

Understanding the brain is a challenge that is attracting a growing number of scientists, from many disciplines. Although there has been an explosion of discoveries over the last several decades concerning the structure of the brain at the cellular and molecular levels, we do not yet understand how the nervous system enables us to see, hear, learn, remember and plan certain actions. But there are numerous fields that depend on computational neuroscience, a few are listed below,

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- a) Deep Learning, Artificial Intelligence and Machine Learning
  - b) Human psychology
  - c) Medical sciences
  - d) Mental models
  - e) Computational anatomy
  - f) Information theory

## Literature Review

### A. Literature Review on Neuroscience

#### Hubel and Wiesel Experiment

This experiment seems to be a harbinger for the neuronal insights that have been discovered. It has laid the foundation for exploring computational neuroscience in depth. Let's see what's inside this.

**Professors David Hubel and Torsten Wiesel have experimented in the 1950s wherein they recorded the neuronal activities of the cat across the retina, as they moved a bright light.** They've filed a few exciting observations while the experiment was going on, they are

- Neurons fired only in some instances, but not always.
- The activity of neurons changed depending on the orientation and location of the line of light.

(Don't worry about the neuronal jargon, we would be exploring all the terms in the following topics.) The electrical and chemical signals recorded in the cells connecting the retina to the brain were converted to sound signals. These sound signals were then played, which resulted in 'Snap! Pop!' crackling sounds. These weren't continuous, instead

played only when the neuron fired. Henceforth, it has established a fundamental understanding of how neurons extract the information cast by the retina, and then clearly explained how the visual cortical neurons (present in the primary visual cortex, V1, in the brain) can formulate an image.

### **Neural cells, Anatomy and Electrical Personality of neurons**

So, to get a clear understanding of how the brain works and how we're able to perceive the world around us, let's look at the primary part of the brain, namely the **neurons**. These are the computational units of human brain.

The brain can be broken down into individual discrete parts called neurons. There're many neuronal shapes possible, say, in the visual cortex, the neuron is pyramidal, and in the cerebellum, they are called the Purkinje cells.

### **Structure of neurons**

A neuron consists of three main parts namely Soma, Dendrites, and Axon. **Soma is the cell body. Dendrites are the input ends of the neurons whereas the axon is the output end.** So, the input is received by the dendrites from the axons of the adjacent neuron. These inputs give rise to an **Excitatory Post-Synaptic Potential (EPSP)**, and when taken as a combination from several other neurons, it provides an **Action Potential or a Spike**. This spiking happens only when the input reaches a certain threshold.

### **Peeking Inside**

Interestingly, neurons can be defined as a "leaky bag of charged liquid." So, all of a sudden, how have chemicals cropped up? It's a crucial thing which many of us aren't aware of. Neurons deal entirely with chemicals, and chemical reactions drive all the spikes and synapses. We indeed have  $\text{Na}^+$ ,  $\text{Cl}^-$ ,  $\text{K}^+$ , et al. in our brains.

Contents of a neuron are enclosed within a lipid bilayer, and the lipid is "fat" in simple terms. This bilayer is impermeable to charged ions, such as  $\text{Na}^+$ ,  $\text{K}^+$ ,  $\text{Cl}^-$  et al. So, how do these chemicals move among the neurons? To answer this, let's deep dive into Ionic channels.

### **Ionic Channels**

The "Ionic Channels" allow the transmission of these ions, i.e., to pass in and out of the neurons. This results in a Potential Difference which exists between the insides and the outer part of the neuron, the inside potential is  $-70\text{mv}$  relative to the outside.

We have  $\text{Na}^+$ ,  $\text{Cl}^-$  on the outside, whereas  $\text{K}^+$ , Organic Anion- are present in the inside of a neuron. Vice-versa is possible too, but the ionic concentrations are lower in this case.

So, how's the potential always  $-70\text{mv}$ ? This is maintained by pumping the ions in and out of the neurons, i.e., by expelling  $\text{Na}^+$  out and allowing  $\text{K}^+$  in. Ionic channels permit only specific neurons to pass and can be classified into three gated channels,

Voltage-Gated — Probability of opening the channel depends on membrane voltage.

Chemically Gated — Binding to a chemical causes the channel to open.

Mechanically Gated — Pressure or stretch influences the channel to open/close.

Ionic passage across the neuron membrane

### **Neuronal signaling**

Neuronal signaling is the interaction that happens among the neurons by the transmission of the signals. The gated channels discussed above allow for neuronal signaling, let's see how,

Firstly, the inputs from other neurons activate the chemically gated channels, i.e open the channels, which lead to changes in the local membrane potential.

Next, this leads to the opening/closing of voltage-gated channels resulting in Depolarization(a positive change in voltage) and Hyperpolarization(a negative change in voltage). Repolarization is where the cell is brought back to the actual potential.

A strong enough depolarisation will lead to the spike or the action potential.

This indeed opens the Na<sup>+</sup> channels(voltage-gated), followed by rapid Na<sup>+</sup> influx(out to in) which drives more channels to open until they inactivate.

When slowly the Na<sup>+</sup> channels start to inactivate, K<sup>+</sup> outflux(in to out) restores membrane potential or the K<sup>+</sup> channels open, reducing the spike. This is Repolarization.

Thereafter, the cell is made more negative as the K<sup>+</sup> channels stay open and continue to let the positive ions exit the neuron. This is termed as Hyperpolarization.

As the potassium channels close, the sodium-potassium pump works to reestablish the resting state again.

After the spike is generated, it is propagated along the axon.

Along the axon, Na<sup>+</sup> channels open first, causing the rise of the Action Potential, followed by the closing of the Na<sup>+</sup> channels and the opening of the K<sup>+</sup> channels, which lead to the fall of Action Potential.

## **B.Literature Review on Human Resources And Neuroscience**

### **Organisational Stress and Neuroscience**

Stress has an impact upon the immune, circulatory, and nervous systems [Esch et al. 2002a; Esch et al. 2002b; Esch et al. 2002d]. However, the underlying physiology reveals high conformance, since the stress phenomenon and its impact are associated with common stress response pathways [Stefano et al. 2005a; Charmandari et al. 2005]. In fact, stress affects immunological [Esch et al. 2002a], cardiovascular [Esch et al. 2002b], and neurodegenerative or mental diseases/disorders [Esch et al. 2002d], and this may include both positive and negative aspects [Stefano et al. 2005a; Esch et al. 2002a; Esch et al. 2002b; Esch et al. 2002d; Charmandari et al. 2005].

Stress can either exert ameliorating or deleterious effects, depending on a multitude of factors (e.g., individual, endogenous, or exogenous elements) [Esch, 2002f; Esch, 2003d; Esch et al. 2002a; Esch et al. 2002b; Esch et al. 2002c, Esch et al. 2002d; Esch et al. 2003b; Jones et al. 2001]. However, clinically, negative influences of stress upon health and disease processes seem to predominate [Esch, 2002f; Stefano et al. 2005a; Esch et al. 2002a; Esch et al. 2002b; Esch et al. 2002d], which may especially be true in modern societies, where stress-related health issues and complaints almost have an epidemic character [Esch, 1999; Esch, 2002f; Esch, 2003d; Jones et al. 2001; Salavecz et al. 2009; Siegrist & Wahrendorf, 2009; Stefano et al. 2005a]. SM strategies, therefore, are of growing importance and acceptance since they address a “basic physiological process” in these societies and countries [Ernst et al. 2009; Esch, 2002f; Esch, 2003d; Le Tourneau, 2003; Richardson & Rothstein, 2008; Salavecz et al. 2009; Stefano et al. 2005a].

The brain is the central organ of stress and adaptation above normal tissue adaptive responses [McEwen, 2009]. When the brain perceives/senses an experience/ stimulus as stressful, physiological and behavioral responses (stress responses) are initiated, leading to allostasis and adaptation, i.e., adaptive or allostatic stress responses [Esch 1999; Esch et al. 2003b; McEwen, 1998; Sterling & Eyer, 1988]. Thereby, the goal is to keep balance, self-organize and maintain autonomy under challenge and ultimately to survive [Esch, 1999; Esch, 2003d].

When an organism chooses the right or successful strategy to fight a stressor and meet the challenge, a boost of rewarding (and stress-reducing) signalling molecules is released into the blood through the brain's reward and motivation centres, in the course of the evolving event or afterwards, to make the individual feel good, become positively motivated and reinforce (and memorize!) the beneficial behavior [Esch & Stefano, 2004c; Esch & Stefano,

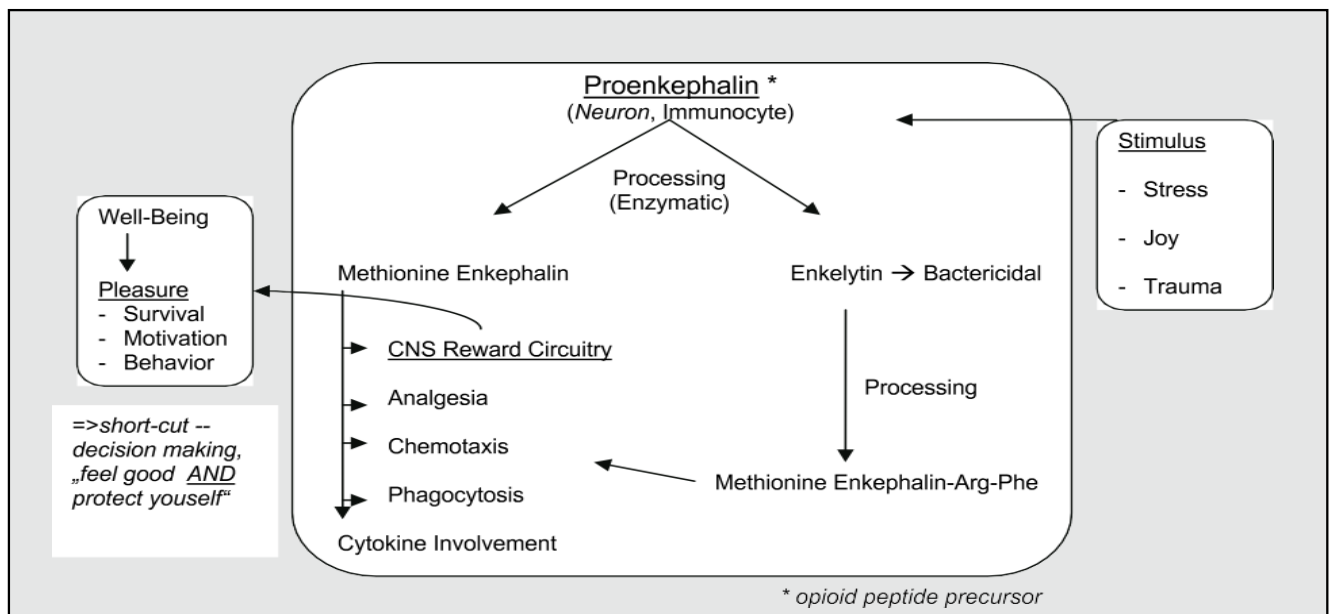
2005a; Esch & Stefano, 2005b; Esch & Stefano 2007b; Mantione et al. 2008; Stefano & Esch, 2005c; Stefano et al. 2001a; Stefano et al. 2007a; Stefano et al. 2008c]. Successful adaptation is thus endogenously rewarded (see below). As a result of these ongoing processes of adaptation, over time, allostatic load can accumulate, and the overexposure to neural, endocrine, and immune stress mediators can have adverse effects on various organ systems, leading to the possible onset or progression of diseases [Esch et al. 2002a; Esch et al. 2002b; Esch et al. 2002d; McEwen, 1998]. Hence, profound physiological and molecular connections between stress and various disease processes exist [Charmandari et al. 2005; Esch, 2003d; Stefano et al. 2005a].

Common pathophysiological pathways in stress-related diseases have been described [Esch & Stefano 2002e; Esch & Stefano, 2007b; Esch et al. 2002c; Esch et al. 2003a; Stefano et al. 2005a], and they critically involve stress hormone (e.g., cortisol, norepinephrin (NE)) and, in particular, NO activity (see below). Moreover, as noted earlier, stress may trigger the activation of a damaged or insufficient biochemical cascade designed to address such “normal” perturbations. In this scenario an individual may have trouble terminating this response, which, under these circumstances, will allow for a long and chronic response since the terminating processes are not functioning or fully functional [Fricchione et al. 1997].

In addition, stress effects and (patho-) physiological consequences are potentially ‘transferred’ not only within the individual (e.g., systemic interactions between mind, brain and body [Esch, 2008a; Komaroff, 2001; Sapolsky, 2004; Stefano et al. 2001a; Stefano et al. 2005a], but also towards other ‘neighbouring’ cells and organisms, even those initially not under stress, e.g., by the means of verbal/non-verbal communication or the exchange of molecular and physical information [Esch, 1999; Esch, 2003d; van Wijk et al. 2008a; van Wijk et al. 2008b; Wiegant et al. 1996].

Furthermore, stress mediation and specific impact of stress hormone activity may be carried over biologically and conserved beyond generation borders, since parents and their offspring show stress response commonalities and physiological/neurobiological as well as stress behavioral coupling [Bakoula et al. 2009; Charmandari et al. 2005; Chin et al. 2009; Moles et al. 2004]. This transfer of stress consequences via neurobiological, physical, or chemical coupling can even include genetic alterations, and these effects may be relatively stable [Chin et al. 2009; Esch, 1999; Meyer et al. 2001; Wiegant et al. 1996].

A brief physical or mental ‘assault’ may allow an organism to deal with both an appraised or perceived stress through various detailed allostatic compensatory mechanisms [Stefano et al. 2005d]. If the situation were to continue chronically, the organism might become vulnerable, susceptible to the negative aspects of the stress response, such as in the case of prolonged immune down-regulation [Esch et al. 2002a, Stefano & Scharrer, 1994; Stefano et al. 1995c; Stefano et al. 1996a; Stefano et al. 1996b; Stefano et al. 2000d].



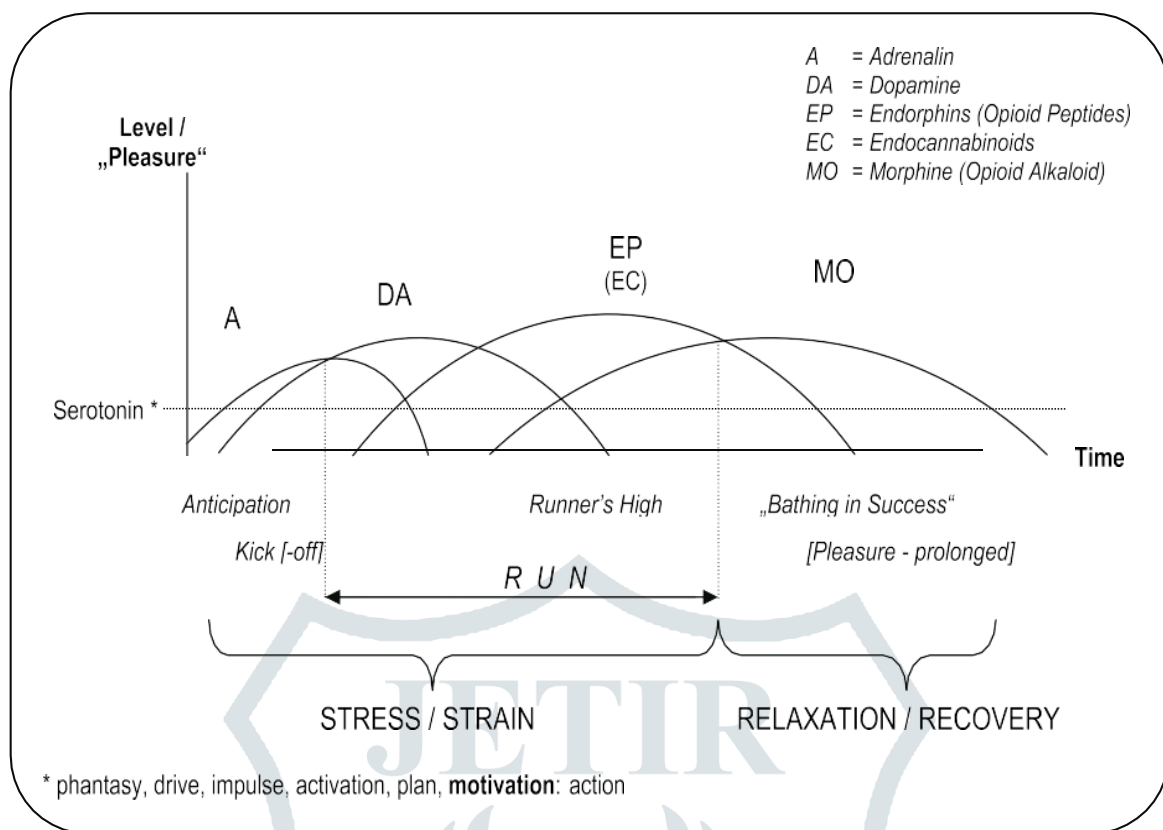
**Opioid peptides as stress hormones.** Stress triggers a release of proenkephalin that gets processed into enkelytin and Met-enkephalin. This step characterizes opioid peptides as stress hormones at the border to stress autoregulation, i.e., endogenous stress reduction, since they have the functions of a ‘double-edged sword’: in part, the opioid peptides/endorphins enable the stressed organism to stay active beyond the normal or physiological duration of a stress cycle (e.g., ca. 90 minutes max. in humans), by signalling the individual to keep up with the stress activity (because it might be biologically necessary), while reducing pain and other physiological companions of a prolonged stress response. In this case, the activated immune response and defence is upheld, typical signs of stress response activity prevail. However, the opioid peptides already prepare for the relaxing and recovering part of stress, i.e., stress management, since motivation and behavioral adjustments are positively influenced; references: [Fricchione & Stefano, 1994; Stefano & Scharrer, 1994; Stefano & Kream, 2008a; Stefano et al. 1996b; Stefano et al. 1998a; Stefano et al. 2001a; Stefano et al. 2005a; Stefano et al. 2005d].

**Neurobiology of stress management** In the above section, we looked at underlying principles of the stress response and depicted some of its neurobiological key players, and finally brought those in line with the idea of an endogenous autoregulation, i.e., physiological self-care and SM. In the following section of this report, we will focus upon the neurobiological commonalities that can be found between the columns of a professional SM regimen, particularly with regard to the molecular effectors.

Indeed, the catecholamine pathway may have arisen from endogenous MO biosynthesis, coupling these signalling processes in an even more intimate relationship as also made evident by common enzymes in the synthesis of these chemical messengers [Mantione et al. 2008; Neri et al. 2008; Stefano & Kream, 2007b; Stefano & Kream, 2009c].

## Behavior

There exists a high congruency between the different techniques and approaches towards behavioral stress reduction when it comes to underlying neurobiological pathways (see below).



**Discussion :** Stress is natural and can be helpful. Stress at appropriate levels, for example, can improve problem solving and cognitive function [Huether et al. 1999; Stefano et al. 2005a; van Praag et al. 2008]. It may act as a trigger for adaptive modifications, e.g., of the structure and the function of the brain, and thus serve to adjust, in a self-optimizing and autoregulative manner, the behavior of an individual to the ever-changing requirements of its external world and environments [Esch, 2003d; Huether et al. 1999; Huether, 1996; McEwen, 2009]. In this regard, stress offers organisms a positive coping strategy, enabling the organism, so endowed, a greater chance of survival. In part, this explains why many of the stress components found at the cellular, tissue and organismic levels in protists, invertebrates and vertebrates have been pre-served during the long course of evolution.

Stress can, however, have deleterious effects in all organisms, and these are related to the dose, form and duration of stress and its underlying (patho-) physiology [Esch et al. 2002a; Esch et al. 2002b; Esch et al. 2002d; Huether et al. 1999; Sachsse et al. 2002; Stefano et al. 2005a]. Accordingly, stress reduction/termination is an innate protection potential to ameliorate stress and counteract its dangerous downside. Hence, SM is natural too, but it has to be physiologically ‘permitted’.

The underlying autoregulation involved in stress and SM manifestations shows a neurobiological overlap (while not denying the specific parts and shares of SM approaches) pointing towards a more general neurobiological/life-sustaining principle, i.e., unspecific or common effects [Esch et al. 2004b; McEwen, 2001; McEwen, 2009]. In this, endogenous SM response pathways consist of the same ‘hardware’ and chemical messengers as, for example, the placebo response, namely the CNS motivation, motor and reward pathways, located predominantly in the limbic brain [Esch & Stefano, 2004c; McEwen, 2001; McEwen, 2009; Stefano et al. 2001a].

The basic truth appears to be that natural or biological and positive activities, i.e., comforting or ‘wellness’ interventions in cognitive and higher noncognitive organisms, serve the goal of survival, appetitive motivation and

health for the individual and the species. These so endowed organisms are rewarded by an overlapping pleasure physiology (**Fig. 7**).

The active use of such self-regulatory potentials may be principally possible, learnable and trainable, e.g., individually by the use of mind/body or cognitive behavioral SM techniques [Esch, 2008a; Esch & Stefano, 2007b; Komaroff, 2001], yet these activities and ‘complementary medical’ interventions (e.g., [Ernst et al. 2008; Ernst et al. 2009; Esch et al. 2004b; Esch et al. 2007a; Stefano & Esch, 2005b]) via their physiological effects are unconsciously and automatically self-activated during stress to reduce it.

**Conclusion :** As stress is natural so is SM (almost like day and night). However, we must take care to keep this healing potential in mind and reserve time and space in our daily routines and stressful lives to let autoregulation happen and, therefore, function. Otherwise we delay the ever accumulating ‘stress’ to deal with the mental and bodily consequences of a lowered stress-induced resistance to diseases. Importantly, stress reduction can be learned, it is neurobiologically rewarding and pleasurable and one must simply learn to take the first step.

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