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Adaptation for Fitness

Intense CrossFit workouts improve your fitness—but how? Dr. Lon Kilgore explains how doing Grace can cause adaptive changes at the cellular level and result in improved performance.

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Any study of exercise physiology must begin with an understanding of what it is we wish to know. Exercise physiology is an applied science, meaning it is intended to solve a problem. The problem needing solving is that we—you, me and our trainees—are not as physically fit as we could be.

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The solution that needs to be provided by our study should be a defined means of improving fitness levels. The discipline of exercise physiology should provide us with an understanding of how the body adapts to exercise to make us more fit. We can begin that quest with a look at the work of one individual, Hans Selye, MD.

Who Is Hans Selye and Why Do I Care?

Adaptation is not a new concept. Friedrich Nietzsche's quote, "That which does not kill us makes us stronger," is a famous adage used in reference to the many challenges we face in life. The fact that it's from the 1800s means we have known for hundreds of years that the human body, when presented with a sub-lethal physical, psychological or chemical stress, can adapt to the source of stress, allowing the body to tolerate incrementally larger similar stresses.

Numerous and earlier historical writings in science and medicine provide observations that mirror Nietzsche's. But none of these writings provided us with anything other than anecdotes—nice observations of the end results of adaptation. It was not until the 1936 synthesis of the general adaptation syndrome by Selye that we had our first understanding of how the adaptation occurred. Selye, an endocrinologist and professor at McGill University in Montreal, Que., spent a lifetime pursuing a goal of understanding how humans responded and adapted to all types of stress. His work in this area forms the essential foundation of exercise physiology. The entirety of the discipline exists as extensions of Selye's theory of biological adaptation.

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Courtesy of the University of Montreal

Dr. Hans Selye started researching "stress" in the 1920s, and his general adaptation syndrome still forms the basis of most exercise science.

Through Selye's own works and his analysis of other scientists' discoveries, he was able to develop a generalized pattern of organismic responses and adaptations to a variety of stressors. The general adaptation syndrome outlines a series of stages through which the body passes as it successfully adapts, or the stages that lead to a failure to adapt.

Selye's 1936 paper was titled *A Syndrome Produced by Diverse Nocuous Agents* and examined structural and functional changes in organisms, single-cell to human, after exposure to "nocuous" (harmful) stresses such as injury, cold exposure, intoxication, drugs and—most important for our purposes—exercise.

Selye proposed that all organisms mount an acute response, then a chronic adaptation after surviving exposure to stress. The final adaptation enables the organism to tolerate a subsequent and more intensely stressful exposure to the same type of stress—a physiological expression of Nietzsche's popular quote.

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Stress produces adaptations, and by precisely measuring those adaptations we can determine the best form and amount of stress needed to produce optimal changes in physiology.

The general adaptation syndrome has three basic stages: alarm, resistance and exhaustion. The former two are quite useful and represent a positive adaptation leading to survival. The latter stage represents a failure to adapt to an overwhelming stress and might result in death of the organism. Let's look at each stage individually and examine what occurs and why.

It should be understood that if exercise is to drive adaptation (fitness gain), the work done in training must continually progress in load. No increased load leads to no improvement in fitness.

Setting off the Alarm

In the alarm stage, the body experiences a novel stress or novel level of magnitude or frequency of a previously experienced stress. That the magnitude or frequency of stress application exceeds the levels previously experienced is very important. It takes such a level of stress to disrupt the internal equilibrium of the cell, tissue or organism and induce the alarm stage.

Once the cascade of events is triggered, we see the physiologic intent of this stage is survival at all costs. The stressed cell or tissue diverts all available resources—energy, metabolic resources and architectural substrates—from carrying out normal functions to the maintenance of cell-structure integrity. Making new and replacing normal cellular chemicals and structures slow to a crawl while creation of cell-stabilizing stress proteins, acute-phase proteins and beneficial inflammatory mediators increases. This selective increase in production acts like basic life support, keeping the cell from being damaged further in the presence of the stress.

Once the stress is removed, there is a fairly rapid return of homeostasis, within six to 48 hours (Selye's proposition). It must be understood that the alarm stage is the stimulus for adaptation, and for us it is the stimulus for improved fitness. If this is understood, it should also be understood that if exercise is to drive adaptation (fitness gain), the work done in training must continually progress in load. No increased load leads to no improvement in fitness.

The idea of progression, just like the concept of adaptation, is not new. Its origin is often credited to the Greek Olympian Milo of Croton (circa 400 B.C.), who became the strongest of all of the original Olympians by reputedly lifting a bull each day of its life, from calf to full-grown bull. So the idea of progression as a training reality has been accepted for a couple of millennia. Selye simply provided us with a lucid physiological explanation for how and why it occurred.

Resistance Is Not Futile

The second phase of the syndrome is the resistance stage. During this stage, the organism starts producing more metabolic and structural elements that are required to enhance its ability to withstand another exposure to the damaging stress. That makes sense: resistance to stress is developed. While the alarm stage is absolutely crucial to initiating fitness gains, the resistance stage is where fitness gains actually occur. I would propose that a better name for this stage would be the "adaptation phase."

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The proper application of stress will result in increased performance until a person's genetic ceiling is reached. Few athletes ever approach this limit.

The duration of this phase is greatly variable from days to months, depending on a number of issues including the magnitude of homeostatic disruption. Was the workload a little or a lot more than normal? Was it a single or cumulative overload? Is the trainee fit or unfit? The end result is always the same: an enhanced physiological ability to tolerate a specific stress.

Here is a good place to introduce the basic premise of specificity. While the stages of the general adaptation syndrome follow the same pattern regardless of stress type, a specific stress such as running 10 kilometers for the first time will produce a set of physiologic adaptations intended to make the trainee able to run the distance again with a lower degree of perceived stress as well as less, if any, homeostatic disruption. Running 10 kilometers generally will not improve sprint speed or squat strength to any appreciable extent because long, slow distance running cannot induce the specific set of adaptations required to do so effectively.

Exhaustion on a Cellular Level

The third stage is known as the exhaustion stage. Selye envisioned it as a stage where the organism's adaptive capacity was overwhelmed, or exhausted. Homeostasis has been disrupted, and the magnitude of disruption is so profound that recovery is impossible. The repercussions of this stage can be quite dire, with death being among the possibilities.

We all know exercise can kill you—Selye considered it a noxious stress—but deaths among healthy exercising individuals are rare. This means the third stage, relative to exercise, is usually manifested as something less dire. We will call it "overtraining."

We will look at Stage 3, or overtraining, as being induced by excessive volume (duration, frequency or both) or intensity (level of exertion relative to maximal ability) of exercise—or a combination of both. The physiological results of overtraining are quite diverse, individual and destructive, but in general they are marked by an inability to compete or train at expected levels. Fitness has decayed.

The Flow of Adaptive Information

When we disrupt homeostasis, a series of events affects our physiology at the most molecular of levels. We affect the operation of our genes, little segments of DNA (deoxy-ribonucleic acid) sequestered in the nuclei of our cells. Our genes control pretty much everything about our anatomy and our physiology through a handy-dandy little informational flow: DNA makes RNA (ribonucleic acid) makes protein makes function.

Initially a novel exercise stress shuts down, represses or down-regulates the activity of many normally active genes in favor of activating, promoting or up-regulating the activity of other survival genes. This is Selye's first stage. The up-regulation of survival genes and down-regulation of normally active genes leads to a different profile of proteins produced by the cell and changes the nature of the functions of the cell, tissue or organism. In this instance, normal cell metabolism and function are repressed in favor of producing transient architectural proteins and other emergency proteins that aid in cellular survival.

After the exercise stress has been removed, the survival status of the cell is not immediately altered. Many of the emergency proteins and their related functions remain present and function for some time. But over the days following a single exercise stress, during the resistance

stage, the normally active genes become un-repressed and begin amping up their production and function again. But this time either more copies of them will be activated or they will experience an increased efficiency in function. We can also see previously inactive genes become active in order to augment function.

As survival-gene activity and their products' activities abate, the new and enhanced set of genes now active will produce new architectural proteins (things like actin and myosin) and metabolic proteins (such as enzymes controlling energy production) that set up improved performance. The magnitude of change following a single exercise bout is not truly large and may in fact be immeasurable in practice, but the cumulative result of a series of homeostatically disruptive training sessions will be measurable in terms of strength or endurance depending on the type of training done.

Realize here that the body will arm itself for survival by activating specific genes that contribute to its ability to survive (physical fitness in our example), a further demonstration of the relevance of the concept of specificity.

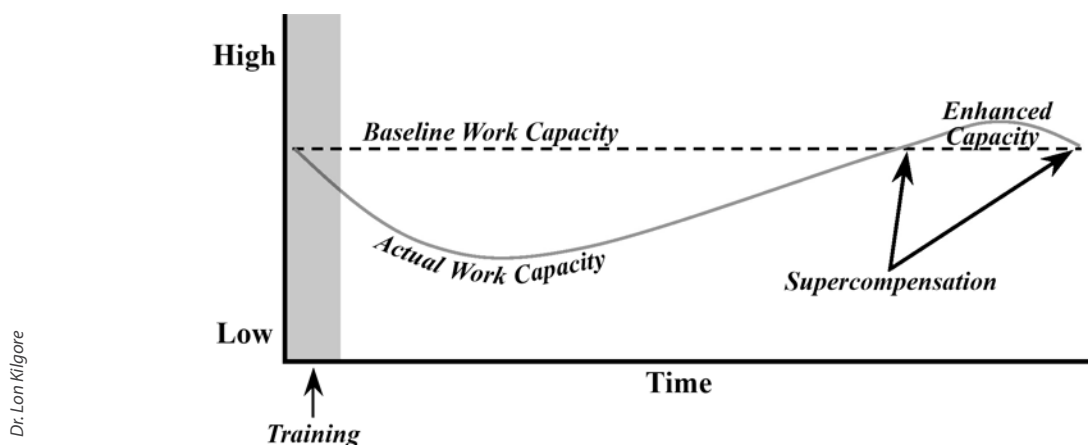
Making Fitness Gain out of Performance Loss

During Stage 1 of the general adaptation syndrome, we commonly see a depression in physical capacity: our performances in training or competition are less than our best. We feel tired, a little sore, or maybe sluggish. This is normal, expected and even desired as these sensory phenoma tell us that we have indeed disrupted homeostasis, our training goal at this point.

But how do we turn a homeostatic disruption and reduction in physical ability into a positive, fitness-enhancing result? It's fairly simple but also extremely complex. The idea of super-compensation is fairly well known and elementary. When we train, we become fatigued. Fatigue exists not just as the feeling of tiredness we get after training hard but also as a set of biochemical and architectural phenomena occurring in cells, tissues and systems. We can consider it the opposite of fitness, the ability to do work. In fact, fatigue is defined as a reduction in the ability to do work. The balance and timing of the physiological processes of both fitness and fatigue can produce fitness gains through super-compensation. Think of it as the disruption of homeostasis and the occurrence of Stage 1 of the general adaptation syndrome events that induce fatigue, metabolically and structurally.

Also occurring during Stage 1 is the reduction of normal function. Fitness, or the ability to do work, will have been compromised as a protective device to prevent further damage. Over time, the results of Stage 1 will diminish, or fatigue will diminish and the emergency proteins and processes will return to their low baseline: "normal" or absent levels of production or activity.

Right after the withdrawal of stress (the end of the training session or series of cumulative sessions), anabolic processes kick in to restore function (fitness) to the system as rapidly as possible. The magnitude of activity is very large in the time shortly after the end of training but decays in the hours and days following cessation of training. The decay will at some point return back to the original level



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Figure 1: Graphical representation of super-compensation. Homeostatic disruption induced by training reduces work capacity. During recovery, work capacity increases to baseline, and if appropriate time is allowed, a transient increase in work capacity is realized. It is imperative to understand that the training undertaken must be difficult enough to disrupt muscular homeostasis, and it must be followed by adequate rest. If those two conditions are not met, super-compensation cannot occur.

of function existing prior to the training stimulus. The return of fatigue or the return of fitness back to baseline is not remarkable and does not lead to fitness gain. It is the timing of decay in both that leads to fitness gain.

The general adaptation syndrome and the concept of super-compensation are important to the practitioner because he or she must, through the delivery of training, elicit these physiological phenomena in a controllable, reliable and repeatable manner.

If the rate of fatigue reduction (recovery) is slower than the rate of fitness restoration, then the net effect is either no fitness gain or possibly even a retention of Stage 1 levels of reduced performance ability. If however, the rate of fatigue reduction is faster than the decay of fitness restoration processes, we will experience super-compensation and enhanced fitness, an increased ability to do work (Figure 1). Super-compensation represents the successful entry and completion of Stage 2 of the general adaptation syndrome. At this point, recovery methodologies become relevant. Adequate nutritional support and sleep provide the body with the elements necessary for maximizing the magnitude of fitness-restoration processes while also facilitating more rapid fatigue reduction. Not only can poor nutritional habits and inadequate sleep duration reduce the degree of super-compensation produced by a training session or program, but such neglect can also move a trainee toward our application of Selye's third stage: overtraining.

The general adaptation syndrome and the concept of super-compensation are important to researchers and practitioners alike. They are important to the former as these two entities provide a conceptual basis for the study of the human during exercise, with the intent of improving physical fitness. They are important to the latter because the practitioner must, through the delivery of training, elicit these physiological phenomena in a controllable, reliable and repeatable manner.

Unfortunately, the practitioner is largely left to his own devices in this task as relevant experimental scientific literature regarding such programming is sparse and often questionable in content. It is advisable for aspiring coaches and trainers to not only become knowledgeable about anatomy and physiology of the human body and how its structure and function dictate exercise programming, but also to apprentice under successful professionals in order to learn how to apply that knowledge toward effective professional exercise practice.



About the Author

Lon Kilgore is a professor at Midwestern State University, where he teaches applied physiology and anatomy. He has also held faculty appointments at Kansas State University and Warnborough University (IE). He graduated from Lincoln University with a bachelor of science in biology and earned a PhD in anatomy and physiology from Kansas State University. He has competed in weightlifting to the national level since 1972 and coached his first athletes to national championship event medals in 1974. He has worked in the trenches, as a coach or scientific consultant, with athletes from rank novices to professionals and the Olympic elite, and as a collegiate strength coach. He has been a certifying instructor for U.S.A. Weightlifting for more than a decade and a frequent lecturer at events at the U.S. Olympic Training Center. His illustration and authorship efforts include books, magazine columns and research journal publications.