Tissue Adaptation to Physical Stress: A Proposed "Physical Stress Theory" to Guide Physical Therapist Practice, Education, and Research
Michael J Mueller and Katrina S Maluf


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Tissue Adaptation to Physical Stress: A Proposed “Physical Stress Theory” to Guide Physical Therapist Practice, Education, and Research

The purpose of this perspective is to present a general theory—the Physical Stress Theory (PST). The basic premise of the PST is that changes in the relative level of physical stress cause a predictable adaptive response in all biological tissue. Specific thresholds define the upper and lower stress levels for each characteristic tissue response. Qualitatively, the 5 tissue responses to physical stress are decreased stress tolerance (eg, atrophy), maintenance, increased stress tolerance (eg, hypertrophy), injury, and death. Fundamental principles of tissue adaptation to physical stress are described that, in the authors’ opinion, can be used to help guide physical therapy practice, education, and research. The description of fundamental principles is followed by a review of selected literature describing adaptation to physical stress for each of the 4 main organ systems described in the Guide to Physical Therapist Practice (ie, cardiovascular/pulmonary, integumentary, musculoskeletal, neuromuscular). Limitations and implications of the PST for practice, research, and education are presented.


Key Words: Adaptation, Biomechanics, Force, Stress.

Michael J Mueller, Katrina S Maluf
Physical therapists have expertise in the application of interventions—such as exercise, postural instruction, orthotic devices, and modalities—that allow them to modify the physical stresses applied to tissues of the body.1-5 Physical stress is defined as the force applied to a given area of biological tissue.6 Exercise interventions that modify physical stress have been shown to decrease impairments, functional limitations, disability, and pain in a variety of patient populations.7-9 These same interventions can help people with and without disease increase muscle performance,10 bone mineral density,11 and fitness levels.9 An increasing amount of evidence indicates that exercise can have positive effects on disease processes such as diabetes,12 arthritis,7-9 and coronary artery disease.13

Many different theories and approaches currently used in physical therapy share fundamental principles that can be organized into a general theory to guide prevention and treatment of a broad range of patient problems. The general theory we present is based on fundamental principles that appear to govern the adaptive response of biological tissues to physical stress. Therefore, we refer to this theory as the Physical Stress Theory (PST). The PST integrates existing knowledge into a deliberately broad theory with potential application to physical therapist practice, education, and research.

Physical therapists have emphasized the evaluation and treatment of movement dysfunction.14-16 Movement can be defined by the basic physical components (mass × acceleration) of a segment or body. The mass × acceleration of a body segment is defined as force (force = mass × acceleration, Newton’s second law of motion). The application of force over a given area of tissue during movement results in stress to the tissue (stress = force/area, where force may be applied in any direction [tension, shear, compression]).9 Although movement is a major source of physical stress on tissues, other forces generated both inside the body (eg, isometric muscle contractions) and outside of the body (eg, gravity) also may contribute to tissue stress. The PST was developed to address how tissues, organs, and organ systems adapt to varying levels of physical stress (Tab. 1). In addition, this theory describes how other factors (Tab. 2) can modify both the level of physical stress and the response of biological tissues to a given stress level.

The PST focuses on the physical stresses that influence all biological tissues. Tissues are formed from groups of similarly specialized cells that cooperate to perform one or more functions within the body.17,18 The 4 fundamental types of tissue are: (1) epithelial tissue, which covers internal and external surfaces of the body and forms glands, (2) connective tissue, which provides structural and functional support to other tissues of the body, (3) muscular tissue, which has specialized contractile properties for producing movement, and (4) nervous tissue, which collects, transmits, and integrates stimuli to control the functions of the body.17,18 These 4 basic tissues combine to form organs. Groups of anatomically or functionally related organs are referred to as “organ systems.”17,18 Our discussion of the PST will be limited to tissues that form the organ systems identified as most relevant to physical therapists in the Guide to Physical Therapist Practice (ie, cardiovascular/pulmonary, integumentary, musculoskeletal, and neuromuscular systems).14 The PST does not address molecular or cellular mechanisms of adaptation. Rather, the PST identifies common principles from the literature that we suggest may be used to predict adaptive tissue changes that occur in response to physical stress.

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In its present form, we believe the PST can be applied most easily to the care of patients with primary problems involving the musculoskeletal and integumentary systems. However, we believe this theory also has potential applications for patients with primary problems involving the cardiovascular/pulmonary and neuromuscular systems. We contend that the PST relates to a continuum of care and, therefore, also has direct applications for issues related to wellness and the prevention of physical disabilities.

The purpose of this perspective is to present a general theory, based on principles of adaptation to physical stress, that we believe can be used to help guide physical therapy practice, education, and research. First, we will describe the fundamental principles of the PST, followed by a review of selected literature describing adaptation to physical stress for each of the four major organ systems described in the Guide to Physical Therapist Practice. The fundamental principles are based on an analysis of the literature and our clinical experiences. Because the principles are general and represent what we contend are the “common denominators” of a variety of research findings and clinical approaches, references are not provided in the description of the theory or in the listing of Fundamental Principles in Table 1. Supporting literature is provided in the subsequent section and is summarized in Table 3. The literature review also will address how we believe the PST relates to other physical therapy theories and approaches. Limitations and implications of the PST for practice, research, and education will then be discussed.

**Description of Physical Stress Theory**

The basic premise of the PST is that changes in the relative level of physical stress cause a predictable adaptive response in all biological tissue. Figure 1 illustrates primary adaptive responses and thresholds for tissue adaptation in response to physical stress. Factors that may influence either the level of physical stress on tissues or the adaptive response of tissues to a given stress level are outlined in Table 2. The fundamental principles of the PST are summarized in Table 1 and are described in further detail below.

**Fundamental Principle A**—Changes in the relative level of physical stress cause a predictable adaptive response in all biological tissue.
Table 3.
Effect of Physical Stress and Activity on Tissues of the 4 Major Organ Systems Listed in the Guide to Physical Therapist Practice

<table>
<thead>
<tr>
<th>Organ System</th>
<th>Tissue/Organ</th>
<th>Stress/Activity Level</th>
<th>Low</th>
<th>Normal</th>
<th>High</th>
<th>Excessive</th>
</tr>
</thead>
<tbody>
<tr>
<td>Musculoskeletal</td>
<td>Bone</td>
<td>↓ BMD</td>
<td>No change</td>
<td>↑ BMD</td>
<td></td>
<td>Fracture</td>
</tr>
<tr>
<td></td>
<td></td>
<td>↓ strength</td>
<td></td>
<td>↑ strength</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Muscle</td>
<td></td>
<td>↓ contractile protein</td>
<td>No change</td>
<td>↑ contractile protein</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>↓ fiber diameter</td>
<td></td>
<td>↑ fiber diameter</td>
<td></td>
<td>Strain</td>
</tr>
<tr>
<td></td>
<td></td>
<td>↓ peak tension</td>
<td></td>
<td>↑ peak tension</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>↓ peak power</td>
<td></td>
<td>↑ peak power</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tendon</td>
<td></td>
<td>↓ CSA</td>
<td>No change</td>
<td>↑ CSA</td>
<td></td>
<td>Strain</td>
</tr>
<tr>
<td></td>
<td></td>
<td>↓ stiffness</td>
<td></td>
<td>↑ stiffness</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>↓ strength</td>
<td></td>
<td>↑ strength</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ligament</td>
<td></td>
<td>↓ CSA</td>
<td>No change</td>
<td>↑ CSA</td>
<td></td>
<td>Sprain</td>
</tr>
<tr>
<td></td>
<td></td>
<td>↓ stiffness</td>
<td></td>
<td>↑ stiffness</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>↓ strength</td>
<td></td>
<td>↑ strength</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cartilage</td>
<td></td>
<td>↓ proteoglycan content</td>
<td>No change</td>
<td>↑ proteoglycan content</td>
<td></td>
<td>Degeneration or tear</td>
</tr>
<tr>
<td></td>
<td></td>
<td>↓ cartilage thickness</td>
<td></td>
<td>↑ cartilage thickness</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>↓ stiffness</td>
<td></td>
<td>↑ stiffness</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Integumentary</td>
<td>Skin</td>
<td>↓ collagen content</td>
<td>No change</td>
<td>↑ collagen content</td>
<td></td>
<td>Abrasion or wound</td>
</tr>
<tr>
<td></td>
<td></td>
<td>↓ collagen fiber diameter</td>
<td></td>
<td>↑ collagen fiber diameter</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>↓ skin thickness</td>
<td></td>
<td>↑ skin thickness</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>↓ strength</td>
<td></td>
<td>↑ strength</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cardiopulmonary/vascular</td>
<td>Heart</td>
<td>↓ cardiac muscle mass</td>
<td>No change</td>
<td>↑ cardiac muscle mass</td>
<td></td>
<td>Fibrosis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>↓ capillary density</td>
<td></td>
<td>↑ capillary density</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>↓ stroke volume</td>
<td></td>
<td>↑ stroke volume</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Blood vessels</td>
<td></td>
<td>↓ vascular diameter</td>
<td>No change</td>
<td>↑ vascular diameter</td>
<td></td>
<td>Fibrosis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>↓ arterial compliance</td>
<td></td>
<td>↑ arterial compliance</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neuromuscular</td>
<td>Neurons</td>
<td>↓ maximum discharge rate</td>
<td>No change</td>
<td>↑ maximum discharge rate</td>
<td></td>
<td>Axonal demyelination and degeneration</td>
</tr>
<tr>
<td></td>
<td></td>
<td>↑ recruitment threshold</td>
<td></td>
<td>↑ recruitment threshold</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>↑ activation during MVC neuron loss</td>
<td></td>
<td>↑ activation during MVC neurogenesis (hippocampus)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Strength is defined as maximum stress, strain, and/or energy density prior to failure. ↑ = increase, ↓ = decrease, BMD = bone mineral density, CSA = cross-sectional area, MVC = maximum voluntary contraction.
their structure and composition to best meet the mechanical demands of routine loading. Deviations from routine or steady-state loading provide a stimulus for tissue adaptation that allows tissues to meet the mechanical demands of a novel environment.

Fundamental Principle B—Biological tissues exhibit 5 characteristic responses to physical stress. Each response is predicted to occur within a defined range along a continuum of stress levels. Specific thresholds define the upper and lower stress levels for each characteristic tissue response. The relative relationship between these thresholds is fairly consistent between people, whereas the absolute values for thresholds vary greatly.

Fundamental Principle C—Physical stress levels that are lower than the maintenance range result in decreased tolerance of tissues to subsequent stresses. Atrophy is one common mechanism by which tissues become less tolerant of subsequent physical stresses (Fig. 2). Atrophy occurs when tissue degeneration exceeds tissue production, and can be defined as a general increase in bulk of a tissue. In general, biological tissues adapt to increased levels of stress by increasing cross-sectional area, density, or volume (Tab. 3). Other examples of adaptations that may increase tissue stress tolerance include hormonal changes, altered cell membrane excitability, and changes in the material properties of tissues (Tab. 3).

Fundamental Principle D—Physical stress levels that are in the maintenance range result in no apparent tissue change. Tissue homeostasis occurs when tissue degeneration is equal to tissue production, resulting in tissue turnover without net gain or loss. The range of stress levels that promote tissue homeostasis is defined as the maintenance stress range and may be different for different people. This steady-state or equilibrium response occurs when tissues are exposed to the same levels of stress to which they have become accustomed.

Fundamental Principle E—Physical stress levels that exceed the maintenance range (ie, overload) result in increased tolerance of tissues to subsequent stresses. Hypertrophy is one common mechanism by which tissues become more tolerant of subsequent physical stresses (Fig. 3). Hypertrophy occurs when tissue production exceeds tissue degeneration, and can be defined as a general increase in bulk of a tissue. In general, biological tissues adapt to increased levels of stress by increasing cross-sectional area, density, or volume (Tab. 3). Other examples of adaptations that may increase tissue stress tolerance include hormonal changes, altered cell membrane excitability, and changes in the material properties of tissues (Tab. 3). Although stress overload can promote tissue hypertrophy and improve stress tolerance, adequate recovery between bouts of increased stress is needed for this adaptive response to occur (see below).

Fundamental Principle F—Excessively high levels of physical stress result in tissue injury. For the purposes of this theory, injury is defined as tissue damage caused by excessive stress resulting in pain or discomfort, impaired function of the tissue, or both. Damages that are not felt
or are not causing noticeable dysfunction are not considered clinically significant injuries. The maximum stress threshold is defined as the amount of stress the tissue can bear just before it fails, if the tissue is fully rested and recovered from previous stresses. Stress levels that exceed the maximum stress threshold are considered excessive and result in tissue injury.

Fundamental Principle G—Extreme deviations from the maintenance stress range that exceed the adaptive capacity of tissue result in tissue death. The PST represents the potential range of responses of viable tissues to a given level of physical stress. To the extent that the stress level can be altered, viable tissues exhibit a variable stress response. For example, healthy tissues that are injured as a result of exposure to high stress levels may return to the maintenance range if stress levels are appropriately reduced. In cases where stress levels deviate substantially from maintenance conditions, tissues may no longer be viable and tissue death can result. Thus, tissue death can occur when tissues are exposed to either extremely high or extremely low stress levels and are unable to adapt or recover.

Fundamental Principle H—The level of exposure to physical stress is a composite value, defined by the magnitude, time, and direction of stress application (Fig. 4). Stress magnitude refers to the amount of stress (force per unit area) on a tissue at any given moment in time. Time factors include the duration, the number of repetitions, and the rate at which stress is applied to tissues of the body. Each of these factors has a direct relationship with the level of stress exposure. For example, a longer duration of stress application results in a greater level of stress on the tissue. The direction of stress application also influences tissue adaptation. Stress will have a different effect depending on whether it is applied in tension, compression, shear, or torsion. Use of a composite measure to quantify the level of stress exposure implies that stress levels may be altered by changing one or more of the stress-related variables that comprise this measure.

Fundamental Principle I—Individual stresses combine in complex ways to contribute to the overall level of stress exposure. Tissues are affected by the history of recent stresses. In the PST, the effect of any given stress will depend on the previous stress experience of the tissue. For example, 1 repetition of a biceps muscle curl against a resistance of 13.6 kg (30 lb) may have little effect on subsequent muscle performance. However, 3 sets of 10 repetitions, 3 times a week, for 2 weeks can lead to muscle hypertrophy and increase the muscle’s ability to generate force. One consequence of the cumulative effect of repeated stresses is that tissues require periods of rest in which the level of stress exposure is substantially reduced so that they can adapt and recover from previous stresses.

Fundamental Principle J—Excessive physical stress that causes injury can occur through 1 or more of the following 3 mechanisms: (1) a high-magnitude stress applied for a brief duration, (2) a low-magnitude stress applied for a long duration, and (3) a moderate-magnitude stress applied to the tissue many times.
principle is closely related to Fundamental Principle H (Tab. 1), which identifies stress magnitude and time factors (eg, duration and number of repetitions of stress application) as important variables used to determine the composite level of stress to which tissues are exposed.

Fundamental Principle K—Regardless of the mechanism of injury, inflammation occurs immediately following tissue injury and renders the injured tissue less tolerant of stress than it was prior to injury. In the context of the PST, the threshold for subsequent tissue injury is lowered as a result of previous injury and the presence of inflammation. Stress levels that did not cause pain before the tissue was injured would then have the potential to cause pain and further tissue damage. Injured and inflamed tissues must be protected from subsequent excessive stress until acute inflammation subsides. Continued stress, even at a level considered normal for uninjured tissue, can prevent tissue regeneration following injury.

Fundamental Principle L—The stress thresholds required to achieve a given tissue response may vary among individuals depending on the presence or absence of several modulating variables. In developing the PST, we identified 4 categories of variables (Tab. 2) that can modulate either: (1) the level of stress on tissues, or (2) the response of tissues to a given level of physical stress (ie, threshold values for tissue adaptation and injury). Because of the complex interaction among variables, it is not yet possible to determine the absolute value of the thresholds for tissue adaptation proposed within this theory. However, we propose that the PST can be used to construct models that predict the relative influence of both stress-related and nonmechanical variables on tissue adaptation and injury. With additional research, it should be possible to estimate the relative threshold value, based on a percentage of maximum stress tolerance, that is required to promote a desired tissue response given the individual circumstances of each patient. Rehabilitation programs then could be tailored to enhance factors contributing to tissue repair and to minimize factors contributing to tissue failure.

Factors that can change the level of stress on tissues or the threshold values for tissue adaptation and injury are summarized in Table 2 and include movement and alignment, extrinsic, psychosocial, and physiological factors. Each of these factors is described below, and those factors that can be modified with physical therapy interventions are emphasized.

**Movement and Alignment Factors**

According to the PST, movement is the most important factor that physical therapists can use to influence tissue adaptation. As noted, movement occurs because of forces. Forces are applied to tissues over a given area that result in stresses, which contribute to tissue adaptation as outlined in this theory. Physical therapists often focus on the treatment of movement dysfunction,\(^{15,16,21,22}\) and one theory has designated movement as its core principle.\(^{15}\) By comparison, the core principle of the PST is tissue adaptation in response to physical stress. As summarized in Table 3, movement can have beneficial effects (eg, exercise-related tissue hypertrophy) as well as detrimental effects (eg, overuse injury) on tissues of the body. According to the PST, movement and alignment factors are considered primarily for their ability to place stress on biological tissue. The components of movement and alignment considered by this theory are muscle performance, motor control, posture and alignment, and physical activity.

**Muscle Performance**

Muscle performance (force generation, muscle length) is a critical aspect of movement that can influence tissue stress. Muscles are highly adaptable. They generate movement and, hence, forces that place stress on tissues. Muscle also is an important “shock absorber,” and muscle contraction is well recognized for its ability to protect bones, cartilage, and ligaments from excessive stress.\(^{28}\) As a general theory, the PST can be used in combination with other theories and approaches that provide a more detailed analysis of the mechanisms by which muscle performance contributes to stress on tissues of the body.\(^{24–30}\)

**Motor Control**

Motor control has been defined as the study of the nature and cause of movement,\(^{31}\) and it, therefore, represents a major component of physical therapists’ expertise.\(^{21}\) Evaluation of the ways in which people control their movements to accomplish tasks provides physical therapists with insight into how stresses are applied to tissues of the body during movement. For example, Maluf et al\(^{32}\) have proposed that the daily repetition of similar movements and postures may result in excessive stress on tissues of the low back. These authors suggest that physical therapists can identify and modify motor recruitment patterns which potentially contribute to patients’ low back pain during the performance of daily activities. Maluf et al\(^{32}\) contend that an important role of physical therapists is to identify patterns of movement that contribute to excessive tissue stress and to teach patient-appropriate movement strategies to prevent tissue injury and pain.

**Posture and Alignment**

Kendall et al\(^{25}\) have emphasized the relationship between posture, impairments, and pain. The Kendalls’ basic premise, based on their clinical observations, is that there is a standard or “ideal” posture and that
deviations from this ideal posture lead to characteristic patterns of musculoskeletal impairments and pain.\textsuperscript{25(p125)} For example, the Kendalls predict that a person with excessive lumbar lordosis would have weak abdominal and hamstring muscles, with short, strong low-back and hip flexor muscles.\textsuperscript{25(p126)}

Some studies\textsuperscript{33–37} have questioned, and even refuted, a large relationship between these variables in people with and without back pain. Rather than emphasizing an ideal standard of posture and hypothesizing that there is a large relationship among specific postures, impairments, and pain patterns, the PST proposes that pain is caused by excessive tissue stress and that postural deviations are one of many potential variables that contribute to the excessive stress levels that result in pain. We commonly observe people with “poor” posture who are pain-free and other people with “good” posture who have pain. The types of activities performed by people varies widely, resulting in different stress demands on tissues of the body.

The PST predicts that no one ideal posture exists for all people because tissues will adapt to meet the unique stress demands of each person. Injury occurs when tissues are unable to adapt to meet the demands of a given posture or task. Therefore, rather than comparing a person’s posture to an ideal standard, the therapist’s examination should focus on the postures or movements that cause pain.\textsuperscript{26,32,38,39} Within this context, postural deviations become one of many potential factors that may place stress on injured tissues. In some people, the postural deviation may be the primary factor contributing to excessive tissue stress (see “Implications for Physical Therapist Practice” and “Implications for Research” sections). In our view, the PST expands upon the Kendalls’ theory by proposing that postural deviations are one important component of musculoskeletal pain; however, pain patterns should be evaluated in a broader context that considers other potential sources of tissue stress.

**Physical Activity**

Physical activity is another component of movement that results in tissue stress. The US Department of Health and Human Services and the American College of Sports Medicine have adopted the definition of physical activity as “bodily movement that is produced by the contraction of skeletal muscle and that substantially increases energy expenditure.”\textsuperscript{40(p4)} Physical activity may be divided into the specific subcategories of occupational, leisure, and self-care activities. The PST predicts that physical activity improves health because it increases stress on a broad range of tissues, making the tissues more tolerant of subsequent physical activity. Because the tissues are more tolerant of physical stress, they are less likely to be injured. This reduction in the likelihood of injury occurs regardless of whether the tissue is part of the cardiovascular/pulmonary, integumentary, musculoskeletal, or neuromuscular system. Increased physical activity has been linked to many positive health benefits, including lower risk for non-insulin-dependent diabetes mellitus,\textsuperscript{41} stroke,\textsuperscript{42} and obesity.\textsuperscript{43} The federal government, in Healthy People 2010, has set a number of goals to increase physical activity (Objective 22; Physical Activity and Fitness) in people who are otherwise healthy. We believe that physical therapists should use their expertise to provide instruction on how people can increase overall physical activity without injuring specific structures (ie, back or knee).

**Extrinsic Factors**

Extrinsic factors are factors outside of the body that can influence either the level of stress on tissues or the thresholds for tissue adaptation and injury. For the purpose of this perspective, those extrinsic factors that can be modified or utilized by physical therapists are emphasized.

Orthotic devices can be used to modify physical stress on biological tissues and can be used as an adjunct to other interventions in several phases of tissue adaptation. An orthotic device can be used to relieve stress from injured tissue (eg, a resting hand splint for patients with carpal tunnel syndrome, a lumbosacral corset for a person with low back pain). An orthotic device also can be used to apply stress to tissue to cause a change in the tissue (eg, orthotic devices that apply low loads for prolonged periods to increase muscle length and joint range of motion at the elbow).\textsuperscript{44} According to the PST, orthotic devices are an appropriate adjunct to treatment when other means of movement can not adequately control stress on the tissue to meet desired guidelines. We contend that components of the orthotic device should be chosen for their ability to achieve a desired stress level on the tissue. Likewise, taping\textsuperscript{45,46} and assistive devices (eg, crutches, walkers, canes) may be effective adjuncts to help modify stress on injured tissues.

Footwear is another extrinsic factor that can influence stresses applied to the foot. Narrow toe boxes are thought to apply damaging stresses to the forefoot that lead to the formation of bunions and hammer-toe deformities.\textsuperscript{47} However, footwear also can be used for therapeutic purposes such as helping to decrease stress on the foot and ankle at heel strike (ie, shock absorption) or helping to control excessive movement of the rear foot.\textsuperscript{48}

The ergonomic environment is another extrinsic factor that can be modified to influence tissue stress. Changes in the ergonomic environment can be achieved by...
redesigning the job site or work tools to reduce the demands of jobs that require high force, high repetition, and awkward postures. Primary principles of ergonomic design include reduction of extreme joint motion, excessive force magnitudes, and highly repetitive movements. Appropriate size and placement of a workstation can reduce extreme joint motion. Tools or machines can be designed to reduce excessive forces or repetitive motions (eg, using an electric drill rather than a manual one).

Modalities (eg, heat, cold, electrical stimulation) are extrinsic factors that can be used to modify the level of tissue stress or the response of biological tissues to stress application. As described in the PST, modalities have a secondary role in treatment but may be indicated to augment the body’s own adaptive capabilities. For instance, electrical stimulation can be used to augment short- and long-term muscle force production, especially in the presence of pathology that limits the normal force generating capacity of muscle (eg, after ligament injury or spinal cord injury). Heat is an example of a modality that can be used to modify the response of tissues to physical stress. For example, elevation of muscle temperature is thought to help prevent strain injuries (ie, raise the threshold for muscle injury) by allowing muscles to stretch more and tolerate higher loads before failure.

Gravity is an extrinsic factor that physical therapists can use to modify the external load on the body. By modifying the orientation of the body or limbs with respect to the ground, physical therapists can increase or decrease forces exerted on the body during movement and, consequently, alter the forces that must be generated internally to produce movement. Modalities or devices, such as aquatic therapy or weight-supported walking devices, also may be used to modify tissue stress by altering the effects of gravity.

**Psychosocial Factors**

Psychosocial factors represent those factors that are unique to the lifestyle of each person and that potentially can be modified. Psychosocial factors can have a profound influence on tissue adaptation, especially as related to tissue injury. We believe psychosocial factors primarily influence individual threshold values for tissue adaptation and injury. For example, researchers investigated the role of mechanical and psychosocial factors in the onset of forearm pain in a 2-year-long prospective study (N=1,953). Besides linking injury to repetitive movements of the arm or wrist (relative risk of 4.1 and 3.4, respectively), the investigators reported that people who were “only occasionally” or “never” satisfied with support from supervisors and colleagues had a relative risk of 4.7 (95% confidence interval=2.2, 10) for forearm injury. These data indicate that, in some circumstances, psychosocial factors can be as important as mechanical factors for tissue injury. Although we recognize that psychosocial factors can play an important role in a person’s response to physical stress, this role is not developed extensively in this perspective. We encourage others to develop the psychosocial component of the PST.

**Physiological Factors**

Physiological factors influence the ability of tissue to respond to physical stress, but these factors often are difficult, if not impossible, to modify. Physiological factors assume a less important role in physical therapy treatment compared with the factors described previously because physical therapists usually cannot modify or treat physiological factors directly. Physical therapists should be aware of the influence of physiological factors, however, because these factors will affect the prognosis of tissue adaptation and recovery from injury.

Medications can influence tissue physiology and, consequently, the ability of tissues to adapt to physical stress. For example, corticosteroid use has a complex effect on tissue. Corticosteroids can simultaneously cause a decrease in inflammation in one tissue (positive effect on stress tolerance) and cause atrophy in other tissues such as skin, bone, and muscle (negative effect on stress tolerance). Physical therapists do not prescribe medications. However, the impact of medications on the adaptive capacity of tissue is important, and physical therapists should be aware of these effects on their patients. In addition, exercise may help to offset muscle atrophy from medications such as glucocorticoids.

Age is another important physiological factor. Although it is beyond the scope of this perspective to discuss the effects of age on tissue adaptation, it is important to discuss several general points. Aging has a negative effect on tissue adaptation. The magnitude of this negative effect in humans, however, is not clear. Kohrt believes that a considerable portion of the negative effects that have been attributed to aging may be due to age-associated decreases in activity. Two predictions regarding the effects of age can be derived from the PST. The first prediction is that aging lowers the ability of tissues to tolerate stress and has a general tendency to lower the threshold for injury, similar to the effect illustrated in Figure 2. The level of stress required to promote tissue hypertrophy and increase stress tolerance generally is greater for young subjects who are healthy compared with older subjects. Young people, therefore, can exercise and stress their tissues more aggressively with less fear of injury than older people. The second prediction is that the negative effects of aging can be modified through increased stress on the tissue. Increased stress, applied through progressive increases in activity or exer-
exercise, theoretically can help prevent or reverse some of the negative effects of aging (Fig. 3). Increasing evidence suggests that tissues remain responsive to physical stress well into old age and that positive adaptations to increased activity and exercise can result in decreases in functional limitations and disability.7,8,10

Systemic pathology includes the many diseases that can affect the ability of tissues to adapt to physical stress. Examples of systemic pathology that physical therapists often encounter are diabetes mellitus and rheumatoid arthritis. Similar to the effects of aging, many forms of systemic pathology lower the ability of tissue to tolerate stress, and they have a general tendency to lower the threshold for injury. One example of this principle is the effect of peripheral neuropathy on cortical bone mass in the feet and hands of patients with diabetes. Patients with diabetes and peripheral neuropathy have been shown to have lower cortical bone mass, with a higher incidence of metatarsal fractures, compared with a matched group of patients with diabetes alone.60 As interpreted by the PST, pathology associated with peripheral neuropathy reduces the injury threshold for bone to a level experienced during normal walking.

Although the PST recognizes the negative impact of systemic pathology on tissue adaptation, it also proposes that carefully applied physical stresses can have positive effects on people with chronic disease. Growing evidence supports this prediction.9,15,61–65 Traditionally, people with rheumatoid arthritis have been excluded from vigorous activities that might exacerbate joint inflammation.9 However, studies have shown that progressive resistive exercises and aerobic exercise programs can increase muscle performance, fitness levels, and bone mineral density with no exacerbation of disease activity in patients with rheumatoid arthritis.9,61,62 The positive effects of aerobic exercise for patients with cardiovascular disease have been well documented.13 Likewise, evidence indicates that increased activity, even in small increments, can have positive effects on reducing the burden of hyperinsulinemia and diabetes.63 William H Herman, an associate editor for Clinical Diabetes, summarized the effect of exercise on diabetes:

An ideal treatment for type 2 diabetes would lower blood glucose concentrations acutely, improve long-term glycemic control, and enhance insulin sensitivity. It would improve mild to moderate hypertension, reduce low-density lipoprotein cholesterol and triglycerides, increase high-density lipoprotein cholesterol, and serve as an adjunct to caloric restriction for weight reduction. Finally, it would increase patients’ sense of well-being and improve their quality of life. As a drug, such an agent would be a blockbuster, with greater than $1 billion per year sales potential. Yet such a treatment exists and has been recognized for centuries. It is regular physical activity—that is exercise.12

Although systemic pathology may lower the threshold for tissue injury, many people with systemic pathology respond positively to exercise, both in terms of increased stress tolerance and reduction of disease complications.

We also consider obesity a physiological factor within the context of the PST that can be modified, but not easily. Obesity is defined as an increase in body weight beyond the limitation of skeletal and physical requirements, as the result of an excessive accumulation of fat.64 Body weight provides an indication of the stresses that must be borne by tissues of the body during physical activity. The PST predicts that obesity is a risk factor for certain types of injury and a negative factor for recovery from injury. In addition, obesity has been linked with low activity levels.45 People who are obese, therefore, may be at greater risk of injury than people who are not obese because high-stress, low-repetition activities are thought to be more damaging than low-stress, high-repetition activities.65–68 Evidence exists to support this prediction. For example, a population-based (n = 350, 95 men and 255 women, 55 years of age or older) longitudinal study (mean follow-up duration = 5.1 years) reported that the risk of incident radiographic knee osteoarthritis was significantly increased among subjects with higher baseline body mass index (odds ratio = 18.3, 95% confidence interval = 5.1–65.1).69

**Literature Review**

The PST focuses on how biological tissues respond to physical stress. Existing evidence and approaches that share common principles with the PST will be described next for each of the 4 major systems described in the Guide to Physical Therapist Practice.14 Because physical therapists often work with patients with injuries, each section will begin by discussing how physical stress can contribute to injury. Discussion of the effects of physical stress on injury will then be followed by a discussion of the effects of physical stress on tissue atrophy, maintenance, and hypertrophy. These effects are summarized in Table 3.

**Musculoskeletal System**

Some authors29,70 have described mechanical principles involved in the management of patients with low back pain. McGill70 proposed that injury, or “failure of a tissue,” occurs when the applied load exceeds the failure tolerance of the tissue. In addition, McGill70 contended that the structures of the back are influenced by the history of recent physical stresses, so that the accumulation of individual stresses can cause injury. He argued that the characteristics of the load (load rate, mode of
load compression, bending, torsion, shear) and the properties of the tissue determine the type and extent of tissue damage.

McGill\(^{70}\) described the same 3 mechanisms of injury to low back tissues that are proposed within the PST (Principles H, I, and J, Tab. 1). One mechanism of injury to low back tissues is a single application of a relatively high load or stress. McGill provided the example of a person riding a snowmobile who is thrown from the machine and lands on a flexed spine and experiences a sudden posterior disk herniation. A second mechanism of injury to low back tissues is multiple moderate loads or stresses applied to the structures of the low back. In this case, injury is the result of accumulated trauma. McGill provided the example of a worker lifting boxes in which tissues of the low back are loaded repeatedly, causing a slow degradation of the tissue tolerance. A third mechanism of injury is low loads sustained by the low back over a long period of time. An example of this mechanism of injury is construction workers who install rods in the floors of a new building and remain in a flexed posture for prolonged periods of time.

Injury to other types of tissue can result from the same mechanisms as those described above for the low back. Bones can fracture from one bout of high-magnitude force (eg, sudden impact fracture of the femur bone from a motor vehicle accident) or from bouts of moderate-magnitude force that occur many thousands of times (eg, stress fracture of the tibia or metatarsal bones from running,\(^{71}\) prolonged marching\(^{72}\)). Similar to bones, ligaments can tear with one bout of a high-magnitude force (eg, injury of the medial collateral ligament from a tackle to the lateral knee). Increasing evidence, however, suggests that ligaments also can fail from repeated bouts of moderate-magnitude stress.\(^{73}\) For example, women have a much higher incidence of anterior cruciate ligament injury from noncontact sporting events compared with men.\(^{74}\) Huston et al\(^{75}\) speculated that this finding may be related to anatomical differences, such as a wider pelvis and greater Q angle in women, that result in greater cumulative stress on the anterior cruciate ligament during typical activities such as walking or running. Radin and colleagues\(^{65–68}\) have described similar mechanisms of injury (Principle J, Tab. 1) involved in degeneration of articular cartilage and joints.

McPoil and Hunt\(^{27}\) also have recognized the importance of mechanical stress in the management of foot and ankle injuries. These authors have described a treatment approach based on a “Tissue Stress Model.” This approach proposes that clinicians focus on reducing excessive mechanical stress from injured tissues in the foot and ankle rather than attempting to place the foot in an ideal posture, or subtalar joint neutral position, as advocated by the Root Theory.\(^{74}\) The authors use a case study to illustrate application of their model.\(^{27}\)

Stress levels that are higher than the maintenance stress range, yet are lower than the threshold value for injury, can have positive effects on tissue adaptation. Based on Principle E of the PST, musculoskeletal tissues subjected to levels of stress that are higher than normal become more tolerant to subsequent physical stresses and are more resistant to injury (ie, tissues become stronger). Principle I suggests that this type of adaptation occurs only when tissues are able to recover and adapt to previous bouts of physical stress. Controlled increases in physical stress through progressive resistive exercise cause muscle fibers to hypertrophy and become capable of generating greater force. Likewise, higher-than-normal levels of physical stress can promote remodeling in bone.\(^{75}\) Wolff’s Law provides an excellent example of how one specific biological tissue, bone, responds to physical stress by remodeling. Wolff’s Law states that the thickness, number, and orientation of trabeculae will correspond to the distribution of mechanical stresses on bone.\(^{75}\) A consequence of stress-induced bone remodeling is that the strength of bone is greatest in the direction in which loads are most commonly imposed.\(^{76}\) For example, the maximum stress tolerated by bone just prior to failure has been found to be higher for compressive loads than for tensile or shear loads, reflecting the predominantly compressive loads experienced by bone during weight bearing.\(^{77}\) Runners with a documented increase in bone mineral density (BMD) in the leg, but not arm, compared with nonathletes provide one example of mechanically induced adaptations in bone.\(^{54}\) Similarly, contralateral differences in arm BMD have been observed in volleyball, basketball, and tennis players, but not in swimmers.\(^{78,79}\)

Similar to bone, models of tendon and ligament adaptation predict that deviations from a typical range of strain (change in length relative to original length) stimulus values promote adaptive responses in these tissues. According to these models, strain values of 1.5% to 3.0% are required to maintain tissue homeostasis.\(^{80}\) Strain values above this range will lead to increases in the cross-sectional area and stiffness of tendon and ligament, whereas strain values below this range predict decreases in these parameters. The models further predict that the more the strain stimulus deviates from steady-state values, the faster adaptive responses occur in tissue. Model-based simulations of tissue adaptation are consistent with histology data for tendon and ligament in animals and humans.\(^{80}\) Other research has demonstrated that both tendon and ligament respond to exercise-induced stress with increases in cross-sectional area, stiffness, and tensile strength.\(^{81–84}\)
Evidence also supports the idea that tissues within the musculoskeletal system atrophy and become less tolerant of physical stress if stress on the tissue diminishes below a baseline level.\(^{59,85–88}\) Unfortunately, tissues typically atrophy at a faster rate than they hypertrophy.\(^{59,85}\) Muscle can lose 6% to 40% of its ability to generate force over a 4- to 6-week period of bed rest or immobilization.\(^{86}\) Bone mineral density also is lost in response to diminished physical stress. Leblanc et al\(^ {87}\) found that BMD is reduced 5% to 4% at the femoral neck and lumbar spine after 17 weeks of bed rest in young men who are healthy. Holick\(^ {88}\) summarized the current literature by indicating that unloading of the skeleton, either due to strict bed rest or in zero gravity, leads, on average, to a 1% to 2% reduction in BMD at selected skeletal sites each month.

Likewise, ligaments respond to reduced mechanical stress. Woo and colleagues\(^ {85,89}\) have documented a decline in the mechanical properties of the rabbit medial collateral ligament in response to 9 weeks of immobilization. These researchers reported that the stiffness, the ultimate load before failure, and the energy-absorbing capacity of the immobilized medial collateral ligament-bone complex were approximately one third of those variables in the contralateral, nonimmobilized control limb.\(^ {89}\)

**Integumentary System**

Tissues in the integumentary system also demonstrate patterns of response to physical stress similar to those described for the musculoskeletal system. Mechanisms of excessive stress resulting in injury to the skin have been described by Brand\(^ {24}\) regarding his work with patients with Hansen disease and foot ulcers. Brand\(^ {24}\) proposed the same 3 basic mechanisms of injury to skin that we describe for musculoskeletal tissues and that are outlined in this theory (Principle J, Tab. 1). According to Brand,\(^ {24}\) direct mechanical damage to the skin can occur with high levels of pressure (100 kg/cm\(^2\) or 1,300 psi) applied for a brief duration, such as when a person steps on a tack. Ischemic skin lesions can occur when a relatively low magnitude of pressure (1–5 psi) is applied to the skin for a long duration, such as when people wear tight shoes or are confined to bed for prolonged periods of time. "Inflammatory autolysis," similar in concept to cumulative trauma injury, can occur when pressures of a moderate magnitude (20 psi) are applied to the skin hundreds or thousands of times each day, such as stresses applied to planar tissues during normal walking.\(^ {24}\) Integument failure due to repetitive stress was shown when repetitive stress (10,000 repetitions per day) was applied to denervated rat footpads in an attempt to simulate stress on the foot during walking. Footpad ulcers occurred within 7 to 10 days of commencing the simulated walking procedures.\(^ {90}\) Brand\(^ {24}\) and others\(^ {91,92}\) subsequently documented similar relationships among time, pressure, and skin breakdown in patients with diabetes and peripheral neuropathy.

Skin also exhibits positive adaptations to controlled increases in physical stress. Clinicians have observed that skin adapts to progressive loading with improved stress tolerance. A common practice for treating patients with a new orthotic or prosthetic device is to slowly progress the magnitude and duration of weight-bearing to allow skin and underlying tissues to adapt to new weight-bearing forces.\(^ {93}\) In addition, the incidence of blisters in the feet of runners has been found to be highest in the early stages of training, and it is much lower in people who consistently run relatively long distances (eg, 48 km [30 miles] per week) as compared with people who are just beginning to run.\(^ {94}\)

Sanders et al\(^ {105}\) have provided a review of the growing body of literature that suggests that skin exhibits an adaptive response to stress similar to the response of other biological tissues. Skin under tension shows changes similar to ligaments and tendons under tension. Increases in collagen fibril diameter, collagen cross-linking, and sulfated proteoglycan content render skin more resistant to tensile forces. Skin adapts to increases in shear stress with an increase in the size and density of cells at the basement membrane and an increase in the thickness of the stratum corneum (the outermost cell layer on the surface of the skin). Increased thickness of the stratum corneum results in a greater volume of skin through which shear loads can be distributed. Distribution of shear loads causes a reduction in the shear gradient and, consequently, decreases the potential for skin breakdown.

**Cardiovascular/Pulmonary System**

Similar to what can occur to tissues in the musculoskeletal and integumentary systems, excessive physical stress can injure tissues in the cardiovascular/pulmonary system. Mechanical stretch induced by high blood pressure is thought to be an initial event that leads to cardiac hypertrophy and eventual cardiac failure.\(^ {96}\) High blood pressure is an established risk factor for conditions such as stroke,\(^ {97}\) myocardial infarction,\(^ {98}\) and atherosclerotic disease.\(^ {99}\) In addition, vigorous and unaccustomed physical activity has been associated with a higher incidence of cardiovascular events.\(^ {100}\)

The positive effects of physical stress in the form of exercise also are well documented for tissues in the cardiovascular/pulmonary system. McArdle et al\(^ {113}\) outlined fundamental principles of exercise training for the cardiopulmonary system. The "overload principle" states that exercise overload specific to an activity must be applied to enhance physiologic improvement and bring
about a training effect. As described for other systems, achieving overload without tissue injury requires an appropriate balance among training intensity (or magnitude), frequency, and duration (time factors). When developing training programs, physical therapists also must consider whether stress levels designed to improve cardiovascular performance have the potential to cause injury to tissues in the musculoskeletal system.

Training can enhance cardiovascular function in athletes, people who are sedentary, people with disabilities, and people with previous cardiac impairments. Training causes adaptations in tissues within the cardiovascular and pulmonary systems, including an increase in the weight and volume of the heart, an increase in the size of the left ventricular cavity and the thickening of its walls, an increase in plasma volume, an increase in maximum cardiac output, and an increase in lung volumes. Intensive training can increase aerobic capacity 15% to 30% during the first 3 months, with as much as a 50% increase over a 2-year period (Tab. 3).

Similar to other systems described, the positive effects of physical stress (ie, aerobic exercise training) are quickly reversible for tissues in the cardiopulmonary system. Reductions in metabolic and exercise capacity can occur after only 1 to 2 weeks of detraining, and many other improvements are lost within several months of detraining. McArdle et al, for instance, reported a 25% reduction in maximum oxygen consumption in 5 subjects who remained bedridden for 20 consecutive days. Maximal stroke volume and cardiac output also decreased, resulting in a loss of aerobic capacity by an average of 1% per day.

**Neuromuscular System**

In our opinion, adaptation of the nervous system in response to physical stress has not been studied as extensively as the adaptive response of tissues in the musculoskeletal, integumentary, and cardiovascular/pulmonary systems. Increasing evidence, however, suggests that physical stress and activity affect the peripheral and central nervous systems in a manner similar to that of other tissues in the body. Excessive stress can cause injury to tissues within the nervous system via the 3 mechanisms discussed for other systems. Examples of nerve injuries caused by high-magnitude stress applied over a brief duration include spinal cord injuries resulting from gunshot wounds or motor vehicle accidents. Nerve injury also may be caused by lower-magnitude stresses applied repetitively or over a long duration. Examples of these mechanisms of nerve injury include carpal tunnel syndrome, tarsal tunnel syndrome, thoracic outlet syndrome, ulnar nerve palsy, spinal stenosis, and lumbar root lesions.

Novak and Mackinnon described excessive physical stress from various postural alignments and repetitive motions that can contribute to nerve injury and pain. The incidence of cumulative trauma disorders has risen dramatically over the last 15 years and has been linked to jobs that require high-force and high-repetition activities, especially when performed in awkward postures. The odds ratio for developing carpal tunnel syndrome has been reported to be greater than 15 for high-force, high-repetition jobs compared with jobs requiring low-force, low-repetition activities. Pressures within the carpal tunnel have been shown to be higher when the wrist is in extreme flexion or extension compared with a neutral position. Furthermore, jobs requiring excessive wrist deviations are considered a risk factor for injury to the median nerve.

Lumbar spinal stenosis is another condition that can place excessive physical stress on neural tissues. Movements and postures of lumbar extension can further reduce the diameter of the spinal canal and vertebral foramen in people with spinal stenosis, causing compression of lumbar nerves and resulting in lower-extremity pain and paresthesias. People with lumbar stenosis generally have reduced lower-extremity symptoms when their spine is flexed because this posture allows the vertebral foramen to widen, thereby reducing compressive and shear forces on neural tissues.

Mechanical injury of nerve tissue can occur with low-magnitude stresses applied for a long duration (compression injury) or from repetitive bouts of moderate-magnitude stresses (cumulative trauma disorders). Although some nerve injuries may require surgical correction, we believe one role of the physical therapist is to determine how excessive stress on the nerve can be relieved in a conservative fashion to facilitate healing of the nervous tissue.

We have limited our discussion of the neuromuscular system to nerve injuries caused by mechanical stress on the nerve and surrounding structures. Although beyond the scope of this perspective, a growing body of literature suggests that neurons adapt to high and low levels of physical activity by altering their electrical activity (rate of discharge, threshold of recruitment). The observation that physical activity can stimulate neural adaptation has important implications for the rehabilitation of patients with primary neurological disorders (eg, stroke, traumatic brain injury). We predict that the fundamental principles outlined in the PST can be used to describe neural adaptations following central or peripheral nervous system injury, and we encourage others to investigate this prediction.
Limitations of the Physical Stress Theory

The PST represents an effort to integrate common principles from the approaches and evidence described above. We also expand existing theories to place the role of tissue injury within a continuum of tissue viability so that the theory encompasses pathological conditions and principles of wellness and prevention. Like any theory, the PST has limitations. These limitations will be discussed next, followed by a discussion of the implications of the theory for physical therapist practice, education, and research.

In the PST, we assume that tissue exposure to physical stress can be quantified, and we assume that knowledge of the level of stress exposure and factors that influence the adaptive capacity of tissues (eg, age, disease) can be used to predict tissue adaptation and injury. In its current form, however, the PST does not define absolute threshold values for tissue adaptation and injury. For example, we do not know whether a tissue is about to be injured until it begins to show signs of inflammation (ie, pain, heat, swelling, or redness). Although attempts are being made to identify biological markers that may identify thresholds of adaptation or injury (ie, integrins in the muscle119 and serum keratan sulfate in intervertebral disks120), much more research is needed to define these thresholds in multiple tissue types.

We believe the PST provides an appropriate framework for investigating the influence of multiple factors on the level of physical stress required for tissue adaptation and injury avoidance. Despite the absence of specific values for thresholds of change, the PST currently provides an overall framework that outlines relative relationships along a continuum of tissue responses to physical stress. Elaboration and refinement of this preliminary framework should eventually allow for a quantitative understanding of the thresholds that indicate when atrophy, maintenance, hypertrophy, injury, and death of various tissues begin.

Another limitation of this theory is that it describes changes at the tissue level and does not indicate how tissue change is related to functional limitations or disability. Therefore, we believe the PST must be used in combination with other theories or models of disablement121 to address the entire spectrum of physical disability. We believe that physical disablement models are critical for understanding the overall relationship between disease or pathophysiology and resulting impairments, functional limitations, and disability.121 We further believe that the PST can be used to complement the physical disablement models by helping us to understand the mechanisms of injuries and repair at a tissue level.

Certain areas within physical therapy are not well developed in the PST in its current form. For example, the PST does not address issues of adaptation in the central nervous system. In addition, the PST does not identify the specific psychosocial factors that most influence tissue adaptation and injury. We believe the current theory, however, does provide a useful framework a wide range of physical therapy issues and has implications for practice, research, and education.

Implications for Physical Therapist Practice

We believe the PST provides a useful framework to approach patient care. It emphasizes the role of physical stress by addressing factors known to influence the level of physical stress and the response of tissues to stress application. According to this theory, the primary role of the physical therapist is to modify physical stresses to achieve a desired goal. Two broad goals will be discussed. The first goal is to reduce pain and subsequent impairments, functional limitations, and disabilities that result from injury. The second goal is to increase activity tolerance. Each of these goals will be discussed further in subsequent sections.

Implications for Treating Patients With Tissue Injury

The PST provides a general model for evaluating and treating people with injury. Chronic tissue injury, defined as injury that results in pain lasting greater than 8 weeks, often is caused by stresses of moderate magnitude that are repeated hundreds or even thousands of times a day.24,49,63 Examples of injuries resulting from this mechanism include many forms of back and cervical pain,70 patellofemoral pain,122 tendinitis injuries (eg, Achilles tendon, posterior tibial tendon), impingement syndrome of the shoulder, stress fractures, neuropathic plantar ulcers,24 and carpal tunnel syndrome.49 The primary questions asked in the evaluation and treatment of these and other forms of tissue injury are: (1) What factors appear to be contributing to excessive stress on the injured tissue? and (2) How can these contributing factors be modified to reduce stress on the tissue and allow the tissue to heal?

We believe proper identification of the injured tissue usually can help determine the factors contributing to excessive stress on the tissue. For example, suspected tendinitis might lead the clinician to investigate sources of stress caused by movements that are controlled by the muscle and tendon. In many cases, however, physical therapists are not able to confirm injury of a specific tissue using the tools currently available to them. The PST describes general principles of tissue adaptation and injury, applicable to all types of biological tissue, that can be used to guide treatment regardless of whether the injury can be localized to a specific structure. In our view, appropriate treatment, therefore, does not always
require accurate identification of the injured structure. For instance, if low back pain can be eliminated by teaching the patient to move at the hips rather than moving at the lumbar spine when movements of the lumbar spine cause pain, identifying the specific injured tissue is of little benefit.

We believe general principles that govern the response of all biological tissues to stress can be used to guide physical therapy intervention, even when pathology of a specific structure cannot be determined. In this context, we contend that the most important question to guide treatment becomes: What factors appear to be contributing to excessive stress on the injured tissue? This question relates to Fundamental Principles F, I, J, and K in Table 1. In our opinion, the factors in Table 2 should be systematically evaluated for their potential to contribute to excessive tissue stress or for their potential to modify the response of tissues to stress application. In our experience, various types of injury tend to be associated with a similar set of contributing factors. For example, we have observed that factors contributing to overuse injuries of the foot and ankle often include movement and alignment factors (ie, excessive pronation or supination, changes in activity) and extrinsic factors (ie, footwear). Factors that contribute to excessive tissue stress or modify the tissue response can be added to or subtracted from the model depending on individual characteristics of the patient and the therapist’s current working set of hypotheses.

We suggest that a physical therapist develop a hypothesis regarding the factors that are contributing to excessive tissue stress and causing injury. The next logical question is: How can I modify these contributing factors to reduce stress on the tissues and allow the tissues to heal? Successful outcomes, in our opinion, help to confirm the initial hypothesis, whereas poor outcomes direct the clinician to investigate other potential sources of stress not identified by the initial hypothesis. This approach is consistent with the hypothesis-oriented algorithm for clinicians.

The basic principles discussed can be illustrated with several examples. Maluf et al described evaluation and treatment of a patient with chronic low back pain. The authors speculated that repetition of rotation and extension movements of the lumbar spine contributed to excessive stress and injury to structures within the low back region. Low back symptoms improved following instructions designed to restrict lumbar rotation and extension movements during the performance of daily activities. McPoil and Hunt described a similar approach in the treatment of a patient with heel pain. The authors speculated that a recent change in activity level, poor footwear, and excessive pronation at the subtalar joint contributed to excessive stress on the plantar fascia and resultant heel pain. The treatment plan, therefore, was designed to modify these factors to decrease stress on the plantar fascia (eg, modifying the patient’s work schedule to limit weight-bearing time, purchasing shoes with cushioned midsoles, obtaining temporary orthotic devices to control excessive pronation).

Similar principles can be applied to the evaluation and treatment of many types of wounds. Mueller and Diamond described an approach emphasizing reduction of stress on plantar tissues in a patient with diabetes, peripheral neuropathy, and a chronic plantar ulcer (21 months). They speculated that a fixed equinus and rear-foot varus deformity contributed to excessive pressure on tissues in the forefoot during walking and prevented the chronic ulcer from healing. The wound healed after a total contact cast, followed by protective footwear, was applied to reduce excessive stress at the site of the wound. An approach based on principles of stress reduction also has been described for the treatment of peripheral nerve injuries such as carpal tunnel syndrome and brachial plexus injuries. Novak and Mackinnon have described how postures, movements, impairments, and physical activities can contribute to stress on peripheral nerve tissue. Furthermore, these authors have described how factors contributing to excessive stress on peripheral nerves can be modified to allow the nerve tissue to heal and, consequently, reduce the symptoms associated with nerve injury.

Even after sources of excessive stress have been identified and removed, we contend that injured tissues will be less tolerant of stress than they were prior to injury due to pain, inflammation, and disuse associated with the injury (Principles C and K, Tab. 1). Therefore, after pain and inflammation have subsided, previously injured tissues, in our opinion, should be exposed to progressively higher levels of physical stress to gradually restore the tissues’ ability to tolerate greater levels of stress (Principle E, Tab. 1). Stress, we contend, should be applied progressively, with attention paid to the body’s need for rest and recovery.

Implications for Improving Activity Tolerance

Our examples were designed to illustrate how to promote healing and progressive strengthening of injured tissues. The goal of helping patients to improve activity tolerance will now be considered. Examples of this goal include increasing muscle peak force production, achieving independence in transfers, and improving walking tolerance. Activity can be considered a form of physical stress. Therefore, a systematic approach for evaluating and treating individuals with the goal of improving activity tolerance can be derived from the
PST. We propose that the following questions are relevant to the goal of increasing activity tolerance: (1) What is the activity goal? (2) What are the current modifiable factors limiting the activity goal? and (3) How should these factors be modified to meet the activity goal? For example, the activity goal of an elderly person may be to stand independently from a sitting position. A physical therapist may hypothesize that the primary modifiable factors limiting this goal are: (1) lower-extremity muscle atrophy resulting in poor force production, (2) decreased dorsiflexion range of motion, (3) poor motor control (movement and alignment factors), and (4) a low seat surface (extrinsic factor). The person’s age is an important physiological factor that may limit the potential for tissue adaptation and lower the threshold for tissue injury. A treatment plan designed to modify these factors based on the PST might include: (1) a progressive resistive exercise program for lower-extremity extensor muscles with at least 70% of maximum effort, 2 to 3 times a week, for several weeks to increase muscle force production, (2) stretching exercises to increase dorsiflexion range of motion, (3) practice standing from a seated position using appropriate movement strategies, and (4) advising the patient to obtain a higher chair so that lower muscle force production is needed to achieve the activity.

The overload principle (Principle E, Tab. 1), in which maintenance stress levels are raised to the range of stress that promotes tissue hypertrophy, has tremendous implications for physical therapy and forms the basis for progressive resistance exercises to increase muscle performance and activity tolerance. Physical stress must be of sufficient magnitude and repetition to cause the desired change in performance. The role of the physical therapist, in our view, is to instruct people in the appropriate magnitude and repetition of exercise or activity to provide an adequate stimulus for hypertrophy of intended tissues without injuring other tissues. Although clinicians are accustomed to using the overload principle to achieve hypertrophy of muscle, these same principles can be applied to other tissues such as ligament, tendon, and skin (Tab. 3).

Implications for Physical Therapist Education
In a time when scientific and medical knowledge is expanding at an exponential rate, we believe the PST can help educators focus on appropriate content for a substantial portion of the physical therapy curriculum. According to the PST, key elements of the curriculum should include a clear understanding of how the body adapts to physical stress and how physical therapists can apply or modify stress on the body to achieve desired outcomes (Tabs. 1 and 3).

In the PST, we propose that several key content areas should be emphasized. Kinesiology, we argue, should be taught with an emphasis on tissue mechanics and how different types of movement contribute to stress on specific tissues. Classes related to exercise physiology, in our view, should present guidelines for exercise and activity prescription that are designed to fall within the range of stress that promotes tissue hypertrophy while avoiding injury. We contend that instruction about orthotic devices or modalities should focus on their use in modifying either the level of stress applied to the body or the body’s response to stress. The content of classes related to medicine and pathology should focus on providing an understanding of how particular diseases or pathologies modify the body’s response to various forms of physical stress including exercise and activity. Classes related to clinical diagnosis and management could be organized using the approach described in the section titled “Implications for Physical Therapist Practice.” We contend that the list of factors in Table 2 could form the focus of factors to consider for evaluation and intervention. Although there are other content areas that must be addressed in a curriculum (eg, administration), we contend that the factors listed above are the most important and could serve as the focus of content unique to a physical therapy curriculum. Students should be trained to develop a clinical hypothesis that identifies the most important factors contributing to a patient’s problem, to address each modifiable factor, and to revise the initial hypothesis based on evaluation of treatment outcomes.

Implications for Research
The PST provides a framework for generating research hypotheses that focus attention on what we believe to be the most important and basic treatment technique used by physical therapists: modifying physical stresses to facilitate tissue adaptation and prevent injury. Although emphasizing the role of physical stress (Tab. 1), the PST also recognizes the multifactorial nature of tissue adaptation and injury (Tab. 2).

We believe this theory helps to clarify and expand on documented theoretical approaches currently used in physical therapy. Disablement models have provided an excellent theoretical framework for studying the relationship among impairments, functional limitations, and disability. We agree that a limitation of these models, however, is that they often do not address the underlying cause of the patient’s symptoms. For example, disablement models postulate that back pain leads to certain functional limitations, such as difficulty performing household chores. However, we believe these models do not encourage identification of the specific mechanisms that cause the pathology leading to low back pain. The PST helps to fill this theoretical void by proposing that
excessive physical stress causes the pathology, resulting in pain and subsequent disability.

We believe the PST also can be used to complement the American Physical Therapy Association’s current Clinical Research Agenda for Physical Therapy.128 The agenda was designed to “support, explain, and enhance physical therapy clinical practice by facilitating research that is useful primarily to clinicians.”128(p499) The Clinical Research Agenda consists of 72 questions, organized according to the elements of the physical therapist patient/client management model. In addition to generating new research questions, we believe the PST provides a theoretical framework to help direct research for questions in the research agenda. For example, question 1.4.9. of the agenda asks, “Do measures of postural alignment in people with spinal disorders influence clinical decision making, and, if so, how?”128(p505) The PST directs the investigator to consider posture and alignment factors in the context of how these factors might contribute to excessive physical stress on the spine. Based on the PST, an investigator might hypothesize that, in the general population, posture and alignment contribute a small but meaningful effect size to spinal dysfunction. The PST suggests that other factors such as movement patterns, age, ergonomic environment, and psychosocial factors also must be considered in clinical decision making when examining and managing patients with spinal dysfunction. Furthermore, we believe the PST provides an organized framework for identifying and studying the relative contribution of each of these factors to the development, persistence, and recurrence of spinal disorders. This is one example of the many multifactorial problems encountered by physical therapists. In the PST, we suggest that contributing factors should not be investigated in isolation when studying the etiology of any given disorder.

Another example of how the theory could be used to generate and test hypotheses put forth in the agenda is in the management of skin breakdown on the plantar surface of the foot in patients with diabetes and peripheral neuropathy. Question 2.2.1. asks, “What are the modifiable risk factors for cumulative trauma syndrome?”128(p505) Neuropathic plantar ulcers are one of many conditions that can be considered cumulative trauma syndrome.24 Investigators91,129 have determined that the location of plantar ulcers corresponds with areas of high physical stress, identified by peak plantar pressure. Identifying the magnitude of pressure that predicts injury of plantar tissues, however, has been elusive.130 The PST predicts that there is no single threshold of peak pressure that predicts injury for all patients. Rather, the theory suggests that a combination of stress-related variables (eg, magnitude, duration, repetition, and direction of pressures) must be considered when assessing a patient’s risk for skin breakdown (Fundamental Principles H and J, Tab. 1).24 Furthermore, the threshold for injury is influenced by individual movement and alignment factors (eg, activity level, foot deformities), extrinsic factors (eg, type of shoes), psychosocial factors (eg, adherence), and physiological factors (eg, severity of peripheral vascular disease and peripheral neuropathy) (Tab. 1). Researchers who are attempting to investigate the complex issue of skin breakdown would need to account for these many factors. The PST provides a framework for connecting the factors that can be used to guide research questions for a broad range of problems encountered by physical therapists.

Conclusion
The PST emphasizes the application and modification of physical stress on tissues of the human body to elicit positive adaptations and avoid injury. Many of the thoughts presented in this article are not new. The summary and integration of these thoughts into a defined theory, however, does offer a comprehensive approach that we believe can help to direct physical therapist practice and research for a wide range of people with and without impairments. We assert that there is considerable evidence to support current physical therapist interventions directed at modifying physical stress. We challenge readers to test, modify, and develop this theory through practice and published research. Our hope is that readers will see common elements described in this perspective in their own areas of practice and that physical therapy can continue to move forward in a more systematic and evidence-based manner.

References


Tissue Adaptation to Physical Stress: A Proposed "Physical Stress Theory" to Guide Physical Therapist Practice, Education, and Research
Michael J Mueller and Katrina S Maluf

References
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