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Fear of reinjury matters after ACL injury

Yong Woo An*
Department of Kinesiology and Dance, New Mexico State University, USA

Abstract

Purpose: Fear of re-injury has been linked to functional joint instability after an anterior cruciate ligament injury. However, it is still unknown how fear and function are linked sequentially in patients with a knee sprain. Therefore, the purpose of this narrative review article was to provide a comprehensive summary of the interrelationships between neuromuscular control, cognition, and emotion, the neural mechanisms underlying cognitive and emotional regulation processes, and potential emotional regulatory training after knee ligamentous injury.

Methods: Previous research articles and more relevant research articles through a cited reference emphasizing fear of re-injury, cognition, emotion, neuromuscular control, and anterior cruciate ligament injury searched from PubMed databases were reviewed.

Results: Higher fear of re-injury in patients after an anterior cruciate ligament (ACL) injury may be associated with diminished neuromuscular control. As negative emotion requires better cognitive neural processing in the several brain's regions that are also essential for maintaining dynamic joint stability, increased neural demands as a result of higher fear of re-injury can disrupt neuromuscular control and long-term pathological sequalae. Therefore, enhanced neural efficiency in cognitive control networks through executive function training may help ACL patients to regulate high fear of re-injury and dynamic restraint systems to maintain functional joint stability during intense physical activity.

Conclusions: As negative feelings can interfere with the cognitive processing for neuromuscular control, executive function training may improve affective control and muscle coordination. Understanding the interrelationships that exist between neuromuscular control, cognition and emotion help to not only develop better rehabilitation program, but also improve patient outcomes after an anterior cruciate ligament injury.

Key words: Anterior cruciate ligament rupture, Neuroplasticity, Fear of re-injury and movement, Emotional regulation, Functional joint instability

Introduction

It has been suggested that some anterior cruciate ligament (ACL) patients who suffer long-term disabilities have altered muscular contraction patterns (Swanik, Lephart, Swanik, Stone, & Fu, 2004), and significantly greater fear of participating in intense physical activity (Gignac et al., 2015; Lee, Karim, & Chang, 2008). Moreover, ACL patients with relatively higher fear demonstrate lower knee function

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Correspondence : anyong@nmsu.edu

during activities of daily living when compared to other ACL patients who have less fear of re-injury/movement (Ross, 2010). Furthermore, higher fear of re-injury has been suggested as one of reasons leading to long-term pathological sequalae such as a recurrent rupture (Ardern, Taylor, Feller, Whitehead, & Webster, 2013). According to Morrey et al. (Morrey, Stuart, Smith, & Wiese-Bjornstal, 1999), ACL patients show progressively improved emotional responses throughout the rehabilitation process. These patients express heightened arousal levels both immediately following the ACL injury and at clearance to physical activity participation, even after completion of an extensive rehabilitation program (Morrey et al., 1999). Given this fact, the direct correlation between reduced subjective knee function scores and augmented fear of re-injury/movement in ACL patients may suggest that negative feelings can alter dynamic muscle contraction mechanisms (Lentz et al., 2015). However, it is still unknown how fear and function are linked sequentially.

An ACL injury may change existing neural networks in the central nervous system (CNS), and therefore ACL patients have varying cortical adaptations in regions of the brain responsible for awareness of sensory inputs from multimodal sources. As a result, the CNS reorganization, which is referred to as neuroplasticity, may differ with respect to proprioceptive feedback and neuromuscular control among ACL patients (Kapreli & Athanasopoulos, 2006). This is critical as several regions of the brain that depend on this information for neuromuscular control and maintenance of dynamic joint stability are also associated with cognitive motor planning strategies (LeDoux & Damasio, 2013). Moreover, it has been suggested that several frontal cortical regions related to cognitive processing for voluntary movements are also highly associated with the regulation of emotion (LeDoux & Damasio, 2013). An unpleasant emotional state, such as fear, increases cortical activity in these frontal areas of the brain in order to regulate emotional responses, by projecting significant information to other brain regions responsible for sensorimotor control (Horn & Swanson, 2013; Morris & Dolan, 2004). It has been shown that unconscious "fight-or-flight" behaviors, in response to unanticipated events, will alter muscle contraction patterns necessary for joint stability (Okada, Hirakawa, Takada, & Kinoshita, 2001). This may indicate that emotional dysregulation resulting from negative emotional stimuli can interrupt a normal cascade of neurocognitive processes associated with the muscle coordination to protect joints (dynamic restraint) and maximize patients' functional outcomes (Okada et al., 2001). However, the lack of understanding surrounding the CNS's role has created a barrier to determining how negative feelings after an ACL injury is associated with diminished knee function in ACL patients. Therefore, the purpose of this article is to assess the interrelationships that exist between neuromuscular control, cognition and emotion, to identify the neural mechanisms underlying cognitive and emotional regulation processes, and to discuss potential emotional regulatory training after an ACL injury to lessen barriers to a patient's full function.

Cognition, Fear of re-injury/movement and Neuromuscular Control

The cognitive management strategy for motor planning is a critical factor in providing early preparatory and continuous reactive muscle contractions needed for maintaining functional joint stability (Swanik, Covassin, Stearne, & Schatz, 2007; Swanik et al., 2004). Therefore, any brief failure in coordination or judgment can temporarily interrupt muscle stiffness regulatory strategies intended to protect the joint (Kirkendall & Garrett, 2000). This would leave static restraints such as the ACL vulnerable to excessive loads, regardless of laxity, because the entire dynamic restraint mechanism would be compromised (Swanik, 2015).

Sudden unanticipated events often result in a universal unconscious startle response in the extremities (Maslovat, Kennedy, Forgaard, Chua, & Franks, 2012). This brief, and involuntary startle response, may be a result of increased errors in motor planning processes because the brain's cognitive network is not sufficient to simultaneously prepare for the overabundant environmental cues (DeAngelis et al., 2014). Altered joint stiffness regulation strategies have been displayed when an unexpected acoustic stimulus is delivered prior to a knee perturbation (DeAngelis et al., 2014).

Particularly, previous findings have revealed higher short-range stiffness (0-4°), indicating spinal reflexive responses through the CNS, with early quadriceps contraction prior to joint loading, and both attenuated long-range stiffness (0-40°) and muscle activation during and after the knee loading (DeAngelis et al., 2014). It has been suggested that early activation of the quadriceps, before the unanticipated knee perturbation, results in knee extensor moments and increased anterior tibial translation, which would exacerbate ACL loading (DeMorat, Weinhold, Blackburn, Chudik, & Garrett, 2004). Moreover, decreased muscle activation during and after the knee movement implies insufficient energy absorption by muscles surrounding the knee, which ultimately impairs dynamic restraint capabilities (Swanik et al., 2004). This may indicate that the cognitive processing needed for anticipation of joint sensation is interrupted during an unexpected, startling event, thus resulting in altered preparatory (feed-forward) and reactive (feedback) muscle contractions that are incompatible with optimal joint stiffness and maintenance functional stability (Bryant, Newton, & Steele, 2009; Swanik et al., 2004).

Previous studies have consistently demonstrated that frightening stimuli can significantly influence functional performance (Noteboom, Fleshner, & Enoka, 2001). For instance, in response to a sudden life-threatening event, a person is likely to exhibit a defensive behavior to either escape from or resist the situation. This "fight or flight" response has been observed in patients with numerous musculoskeletal injuries, such as a chronic low back pain (CLBP) and ACL injury (Hartigan, Lynch, Logerstedt, Chmielewski, & Snyder-Mackler, 2013). According to these studies, CLBP and ACL patients tend to avoid intense physical activities that are associated with the previously experienced pain or injurious situations because of the expectancy of having a relapse of pain or re-injury. Moreover, these patients reported significant development of subjective fear and diminished functional abilities over the long term. It has been suggested that advanced neural activity related to cognitive processes in the brain can suppress fear responses (Schweizer, Grahn, Hampshire, Mobbs, & Dalgleish, 2013). As executive-function skills can control these negative feelings, ACL patients with greater fear of re-injury/movement may need enhanced cognitive management skills of muscle stiffness regulatory strategies in order to prevent unpleasant experiences of knee "giving way," when they confront intensive knee functional tasks (LeDoux & Damasio, 2013; Swanik et al., 2007). However, it is unclear how negative emotions, particularly a fear of re-injury/movement, alters the neuromuscular control system. Moreover, determining how negative emotions can be regulated in ACL patients and its effect on functional joint instability has not yet been investigated.

Swanik et al. (Swanik et al., 2007) attempted to link a noncontact ACL injury and neurocognitive characteristics in intercollegiate athletes by using a computerized neurocognitive test battery (ImPACT: Immediate Post-Concussion Assessment and Cognitive Testing). The executive functioning baseline measurements of non-injured athletes compared to ACL-injured athletes revealed slower reaction time and processing speed, as well as diminished visual and verbal memory scores. These components are thought to represent cerebral performance associated with working memory and goal-directed decision-making processing necessary for neuromuscular control (Consiglio, Driscoll, Witte, & Berg, 2003; Lamm, Windischberger, Moser, & Bauer, 2007; Maroon et al., 2000; Moser, Schatz, & Jordan, 2005). However, very limited studies have attempted to examine the effects of neurocognitive intervention in ACL patients. Cappellino et al. (Cappellino et al., 2012) utilized neurocognitive exercises as an alternative rehabilitation approach following an ACL rupture. These exercises required using an ACL patient's recognition of joint positions, various patterns of body movements and joint angles, and transition of joint load in addition to traditional proprioceptive and perceptive neuromuscular control programs. It was reported that ACL patients who performed the neurocognitive exercises showed improved muscle coordination and decreased pain and edema at six months after reconstruction compared to others who underwent a common physical therapy program. These findings suggest that use of an individual's attention during proprioceptive and perceptive rehabilitation may facilitate and attune existing affective control networks (Bonnard,

de Graaf, & Pailhous, 2004). As a result, precise cognitive awareness and enhanced neuromechanical coupling can offer better muscle stiffness regulation strategies to protect the knee in response to an unanticipated event (Swanik et al., 2007). However, a negative feeling and its subsequent neural responses can instantly interfere with cognition and motor planning needed for coordination and the avoidance of unintentional injuries, as several cortical and subcortical areas are responsible for both the regulation of emotion and cognition (Amaral & Strick, 2013; Ohman, 2005). Although it has been suggested that executive-function skills are associated with ACL injury proneness and knee function, the direct relationships between cognition, fear, and joint stiffness regulation strategies in ACL patients with long-term disability, as well as higher fear of re-injury/movement, remain unknown (Cappellino et al., 2012; Gignac et al., 2015; Lentz et al., 2015; Swanik et al., 2007). Therefore, understanding neural mechanisms underlying fear and its neurophysiological reactions will offer better insight into the role of executive-function skills for fear of re-injury/movement, its effects on functional joint instability, and best practices to improve each patient's functional outcome following an ACL injury.

Negative Emotion: fear and its neurophysiological reactions

Human emotional response is a natural physiological homeostatic process regulated by the CNS (LeDoux & Damasio, 2013). Fear is an unconscious emotional awareness responding to an unanticipated frightening stimulus, whereas the feeling of fear is a conscious behavioral and cognitive response. In order to examine fear responses, many researchers have reproduced fearful situations by using visuospatial stimuli, such as emotionally provocative pictures or films (Bradley & Lang, 2006; Chen, Katdare, & Lucas, 2006). In several psychological studies of emotion, researchers have observed different neurophysiological reactions, such as cardiac outputs, corresponding to either unconscious or conscious fear regulation processes in subjects responding to fear-related pictures (Ax, 1953; Bradley & Lang, 2006; Horn & Swanson, 2013; Lang & Bradley, 2007). The CNS influences these changes that are mediated by the peripheral nervous system, particularly, by either independent activation of the parasympathetic or sympathetic nervous systems, or through reciprocal regulation between them (Bradley & Lang, 2006; Lang & Bradley, 2007). It is well known that the parasympathetic nervous system is responsible for quick activation in target organs, by releasing rapidly dissipating acetylcholine neurotransmitters along a short length of post-ganglionic fibers (Bradley & Lang, 2006; Lang & Bradley, 2007). Conversely, the sympathetic nervous system is responsible for slower, but longer lasting activation in target organs, by releasing slowly dissipating noradrenaline neurotransmitters through relatively lengthy post-ganglionic fibers (Bradley & Lang, 2006; Lang & Bradley, 2007). Activation of the parasympathetic branches decreases heart rate and blood pressure, whereas the sympathetic branches often increases heart rate and blood pressure (Bradley & Lang, 2006; Lang & Bradley, 2007).

One of the most predominant neurophysiological responses associated with a fear-related stimulus is a cardiovascular reaction. Originally, it was proposed that a negative emotional stimulus triggers the sympathetic system to accelerate defensive behaviors by increasing heart rate (Ax, 1953). However, many recent studies employing negative emotional stimulus showed an initially decelerated heart rate followed by an accelerated heart rate (Adenauer, Catani, Keil, Aichinger, & Neuner, 2010; Bradley, Hamby, Löw, & Lang, 2007). Furthermore, a more arousing negative stimulus induced greater cardiac deceleration and delayed and longer activation of the subsequent cardiac acceleration. This initial cardiac deceleration elicited by the parasympathetic nervous system indicates increased sensory intakes by the brain, which reflects initial unconscious awareness of the fearful stimulus. On the contrary, the heart rate acceleration that follows is a result of the sympathetic dominance, indicative of the internal cognitive processing for recognition and preparation for an appropriate "fight or flight" behavior (Bradley & Lang, 2006).

Unlike normal fear-related cardiac responses, people who are emotionally vulnerable have shown somewhat

different heart rate reactions (Adenauer et al., 2010; Globisch, Hamm, Esteves, & Ohman, 1999; Ohman, 2005). Particularly, patients with emotional disorders, such as spider or snake phobias, show a relatively large or early onset of heart rate acceleration in response to high-fear-related stimuli compared to neutral stimuli and control subjects who display normal cardiac responses (Globisch et al., 1999; Wendt, Lotze, Weike, Hosten, & Hamm, 2008). Negative stimuli in patients with posttraumatic stress disorder (PTSD), who typically report difficulty in controlling emotions, also reveal similar cardiac responses to animal phobic individuals compared to both neutral stimuli and other non-PTSD groups (Adenauer et al., 2010). These findings may suggest that minimal fear-related sensory information in these patients can very quickly activate the conscious affective control processing, which means that they fail to recognize important environmental cues for successful anticipation of movement during a sudden high velocity physical activity (Bryant et al., 2009). The prolonged recognition and appraisal processes may also interrupt cognitive neural networks related to muscle coordination because these neurophysiological reactions are a product of the CNS, particularly the simultaneous modulation of neural interconnections between subcortical and cortical regions of the brain (Horn & Swanson, 2013). These findings may infer that the fear network in the brain has a substantial role in regulation of negative feeling, as well as maintenance of functional joint stability.

The amygdala, which is one of the limbic system structures, is interconnected with other cortical areas associated with fear-related perception, cognition and motor planning through multiple afferent and efferent pathways. Therefore, it is thought to be the center of the fear response and subsequent motor behaviors (Amaral & Strick, 2013). Fear-related neural processes begin with activation of the amygdala, which simultaneously projects the fear-related sensory inputs to the hypothalamus and brainstem, as well as to the cerebral cortex (LeDoux & Damasio, 2013; Ohman, 2005). This early activation of the amygdala to the hypothalamus and brainstem is an indication of unconscious, automatic detection of fear-related stimulus, whereas the continuous cortical feedback between the amygdala and the cerebral cortex is associated with increased cognitive processing in the fear network for conscious regulation of negative emotional responses (Liddell, Williams, Rathjen, Shevrin, & Gordon, 2004).

A number of neuroimaging techniques have enabled the examination of neural interconnections between these sub-cortical and cortical areas during fear responses. Functional MRI (fMRI) studies have shown increased amygdala activation and its neural functional connectivity with the dorsal anterior cingulate cortex (dACC) and the orbitofrontal cortex (OFC) during presentation of fear-related facial pictures (Morris & Dolan, 2004; Williams et al., 2006). These cortical areas are thought to heighten cognitive awareness of the body in order to prepare voluntary movements (Amaral, 2013; Clark, Mahato, Nakazawa, Law, & Thomas, 2014; Ward et al., 2015). It is possible that feelings of fear can increase neural recruitment demands in these areas, and subsequently alter cognitive motor planning during unanticipated events (Olson & Colby, 2013; Swanik et al., 2007). While these findings demonstrate that prefrontal areas are important for regulating both emotion and neuromuscular control through using of fMRI techniques, electroencephalograph (EEG) technique also offer concurrent neural interactions in real time that may exist between those areas (Crosson et al., 2010).

Direct observation of electrophysiological signal changes in the brain areas corresponding to a specific event or stimulus, which is referred to as event-related potential (ERP), has provided a cascade of sequential neural responses in the cerebral cortex to external stimuli (Sur & Sinha, 2009). The brain shows different ERP components such as latency and amplitude corresponding with the type of neural events. In general, early latency and peak ERPs are related to perceptive processing, while late latency and peak ERPs are considered as neurocognitive processing. Several emotion studies utilizing EEG also showed different ERP components responses to emotional stimuli (Krolak-Salmon, Hénaff, Vighetto, Bertrand, & Mauguière, 2004). Affective stimuli showed an early deflecting peak potential at about 200ms (N2) and positive peak potential around 300ms (P3) after the onset of stimuli, which have implied unconscious

automatic detection of emotional stimuli. Additionally, a late negative peak potential at about 430ms (N4) and positive peak potential within a range of 300 to 1000ms (P3b) are also observed. These late ERP components are known to represent cognitive integration during conscious emotional responses (Bradley & Lang, 2006; Bradley et al., 2007; Liddell et al., 2004). Interestingly, the brain has showed different ERP component responses based on a variety of arousal levels. A fearful stimulus, which provokes greater arousal than a neutral stimulus, induces a greater and early onset of N2 and P3a over the frontal and centroparietal sites and, in turn, elicits larger and later onset of P3b at the centro-parietal and posteroparietal areas. The time course of the cerebral performances indicates that fearful stimuli can delay the beginning of cognitive processes in the fear network and require longer time for regulation of the frightening situation. These altered cerebral functions are more predominant in individuals who are emotionally susceptible to a negative stimulus (Ohman, 2005).

Neuroimaging studies have shown that individuals with an animal phobia produce increased activity in subcortical and cortical areas, responding to fear-related stimuli (Carlsson et al., 2004; Wendt et al., 2008). This population also demonstrated enhanced P3a and P3b relative to non-phobic controls (Leutgeb, Schäfer, & Schienle, 2009; Schienle, Schäfer, Stark, & Vaitl, 2009). Moreover, patients with military and civilian related post-traumatic stress disorder (PTSD) have also shown dissimilar ERPs than non-PTSD individuals such as larger increased N2, altered P3a, and extended period of P3b when exposed to trauma-related stimuli (Attias, Bleich, & Gilat, 1996). A large, early positive ERP implies rapid detection of the dangerous stimulus as a result of the enhanced afferent subcortical conveyance by the amygdala. This is thought to be the location of memories from previous fear-related experiences, so the positive ERP suggests the memories are being transmitted to the prefrontal cortex (Liddell et al., 2004; Morris & Dolan, 2004; Williams et al., 2006). The early induction of, and extent of late positive ERP may indicate increased cortical activation demands related to cognitive regulation processing in fear network.

It is suggested that emotion-related motor behavior is a result of affective control of emotional responses through a cortical pathway between the frontal and parietal cortices (Olson & Colby, 2013). This fear network begins from the OFC, to the premotor cortex through the dorsolateral prefrontal cortex (DLPFC), and from there to the primary motor cortex (M1). Although it is unidentified how negative emotions alter neuromuscular control in ACL patients, it is known that the frontoparietal neural network is highly associated with cognitive control of working memory and regulation of emotion (Schweizer et al., 2013). Irregular cortical activation in the prefrontal and somatosensory cortices in response to a fearful stimulus may imply abnormal neuroplasticity (Mahan & Ressler, 2012). Therefore, it is possible that high fear of re-injury/movement may interfere not only with the affective regulatory neural network, but also with the cognitive motor planning network needed for neuromuscular control in ACL patients. For this reason, it is suggested that improving executive-function skills can aid to mediate negative emotion, as well as quickly suppress emotion-related behaviors due to high neural connectivity between fear and cognition networks (Gyurak, Goodkind, Kramer, Miller, & Levenson, 2013; Gyurak et al., 2009; Gyurak, Gross, & Etkin, 2011).

Emotional Regulation: Executive Functioning Training

Emotional regulation is an integrated cognitive behavioral process related to perceiving, evaluating, analyzing, and modulating the emotional state (Gyurak et al., 2009). Therefore, it is important that several brain regions temporarily work together for optimal neurocognitive processing. Neuroimaging studies have shown that neurocognitive function skills can contribute to improving fear network responsible for cognitive emotion regulatory processing. Desbordes et al. (Desbordes et al., 2012) demonstrated in a fMRI study that healthy controls had inhibited cortical activity in the amygdala in response to emotion-related images after 8-week mindful-attention training (MAT), which aims to down-regulate emotional response by cultivating internal and external awareness such as one's breathing, mental events,

or even training. Moreover, an intentional cognitive re-thinking, referring to reappraisal, has known to be associated with working memory processes, and it showed increased cortical activity in the prefrontal areas but decreased activity in the limbic system, such as the amygdala and insula, in response to negative emotional stimuli (Goldin, McRae, Ramel, & Gross, 2008). These findings may suggest that the enhanced prefrontal functioning is a cognitive neural compensation in the fear network utilized in an attempt to quickly inhibit fear-related reactivity in the limbic system (Riemann & Lephart, 2002). Although many types of neurocognitive components mentioned above have been engaged in emotion regulation, executive functions may play a key role in the augmentation of affective control implicated in neuromuscular control (Gyurak et al., 2013; Gyurak et al., 2011; Schweizer et al., 2013).

Increased neural demands in the cortical areas during emotional regulation can disrupt neuromuscular control because the preparatory motor planning needed for feed-forward muscle contraction strategies also relies on high cognitive processing in the cerebral cortex (Noteboom et al., 2001; Okada et al., 2001; Riemann & Lephart, 2002). Executive-function skills are associated with goal-directed motor behaviors because the cognitive processes include recognition, preparation, implementation and evaluation of an external stimulus (Zelazo & Cunningham, 2007). Attention, working memory, reaction time and decisional accuracy may be particularly critical components in the control of emotion regulation and muscle coordination as these cognitive characteristics are highly associated with neural activation in the frontoparietal areas as well as unintentional musculo-skeletal injuries (Goldin et al., 2008; Schweizer et al., 2013; Swanik et al., 2007). Schweizer et al. (Schweizer et al., 2013) utilized an emotional dual n-back task, matching a word via verbal cue with an emotional face paired with the word of n trials back, in order to improve working memory capacity. The results revealed improved executive functions and emotional regulation. In comparison to pre-training of working memory, individuals had decreased cortical activation in the frontoparietal networks during executive functioning task. These findings may suggest that decreased cortical activation provides evidence for improved neural productivity (Kelly & Garavan, 2005). Therefore, enhanced neural efficiency in cognitive control networks through executive function training may help ACL patients to regulate high fear of re-injury/movement and dynamic restraint systems to maintain functional joint stability during intense physical activity.

Conclusion

ACL-injured individuals who have failed to resume pre-injury levels of physical activity report higher fear of re-injury/movement and suffer repetitive functional joint instability, when compared to others who are able to cope with physical activity without functional limitations (Ardern et al., 2013; Lee et al., 2008). Evidence of neuroplasticity in emotionally vulnerable individuals, such as animal phobic and PTSD patients, in response to particular animals or traumatic stimuli respectively, suggest that increased cortical activation in the fear network can interfere with goal-directed cognitive motor planning processes. Findings from neuro-imaging studies on ACL injuries corroborate re-organized cortical activation in ACL patients. As the PNS and CNS both are critical in the voluntary movements and emotion regulation, it is not surprising that emotional regulation also involves greater cortical activation to compensate for the increased fear-related sensory inputs to the CNS (Krolak-Salmon et al., 2004). However, researchers have observed a greater reduction in accuracy and reaction time in the general population in response to fear-related stimuli compared with neutral or happy facial expressions (Calvo & Lundqvist, 2008). This finding may suggest that increased cognitive neural processing demands in the prefrontal cortex as a result of frightening stimuli during an unanticipated physical activity may not be indicative of better planning of movement or anticipation for a joint perturbation, but may merely alter the cognitive processing for the neuromuscular control system necessary for maintaining dynamic joint stability (Carlsson et al., 2004). Therefore, it is possible that ACL patients with ACL injury-related visual stimuli may unconsciously activate and extremely increase neural activation in motor planning

network in response to an unexpected event such as an acoustic startle stimulus, due to increased sensory resources for both the feed-forward and feedback muscle contraction mechanisms in addition to fear regulation. Moreover, executive function is thought to provide cognitive regulation of negative emotions so that augmentation of ACL patient's cognitive capacity may also improve affective control and muscle coordination, thus maintaining functional knee stability (Chen et al., 2006; Goldin et al., 2008; Gyurak et al., 2011; Ohman, 2005). Therefore, neurocognitive function interventions that are incorporated with neuromuscular control exercises, for instance a dual task emphasizing cognitive processing during a drop-jump performance, should be considered after an ACL injury to optimize patient outcomes.

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