Understanding the Lightning Process Approach to CFS/ME; a Review of the Disease Process and the Approach

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Abstract

Introduction: The Lightning Process (LP) is a neuro-physiological training programme based on self-coaching, concepts from Positive Psychology, Osteopathy and Neuro Linguistic Programming (NLP). It has a developing evidence base for its efficacy with a range of issues, including Chronic Fatigue Syndrome/ Myalgic Encephalomyelitis (CFS/ME), but little has been published about its hypothesis on the disease processes, and its approach to this disabling disease.

Objectives: This paper aims to resolve these gaps in the research and contextualise the approach within current theories and research into the disease.

Methods: A literature review was undertaken of the published evidence supporting the main current models of aetiology and disease process for CFS/ME. An evaluation of the LP’s conceptualisation of the aetiology of the disease and the hypothesis behind its approach was undertaken, through a review of the literature and semi-structured interviews with the programme’s original researcher (this paper’s lead author). These models were then compared to identify similarities and differences.

Results: The review identified that the LP adopts a multifactorial, multisystem disease process for the disease, which is well aligned with current research and established conceptual frameworks for CFS/ME pathology. It identified that the LP shared the established perspective that the illness is a physiological, and not a psychological, one. It found the LP applies a self-regulation approach to neuro-physiology processes to influence the physical disease process.

Conclusions: This paper resolves the identified gaps in the research and clarifies the hypotheses behind this approach, which has been identified by the evidence base as providing successful outcomes for some. It is hoped this clearer understanding of the approach will assist researchers, clinicians and those with this disabling disease to identify some additional options for potential recovery.

Keywords: neurology, physiology, intervention, hypothesis, fatigue

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Introduction and Objectives

The Lightning Process (LP) is a neuro-physiological training programme based on self-coaching, concepts from Positive Psychology, Osteopathy and Neuro Linguistic Programming (NLP). The intervention was developed in a similar way to other novel approaches, such as Motivational Interviewing (W. Miller & Rollnick, 1991), through an iterative process of practice-based evidence (Leeman & Sandelowski, 2012) and qualitative inquiries into clients’ experience.

It has been used by those seeking help with a range of issues, including Chronic Fatigue Syndrome/Myalgic Encephalomyelitis (CFS/ME), an illness defined as a chronic, fluctuating, neurological condition that causes symptoms affecting many body systems, particularly the nervous and immune systems. Early reports of complete recovery from many and an absence of results from others (ME association, 2010) resulted in a lack of clarity about its value in a field already prone to misinformation and strong debates. Further research has developed an evidence base, with an RCT finding the approach, when combined with specialist medical care, increases positive outcomes for some groups with CFS/ME, compared to specialist medical care alone (Crawley et al., 2017), a smaller study identifying positive outcomes for pain (Hagelsteen & Moen Reiten, 2015) and two studies reporting on patient experiences (Reme, Archer, & Chalder, 2013; Sandauet & Salamonsen, 2012). However, an outline of the theoretical basis of the LP intervention for CFS/ME has been missing from the evidence base, a gap which this article sets out to address.

Methods

The structure of this paper follows other authors’ suggestions (Adams, 2007; Miller, 1983) to identify peer-reviewed research to evaluate how a practice-based evidenced intervention fits with current models of aetiology and disease processes.

Firstly, a literature review of the published evidence was undertaken, supporting the main current models of aetiology and disease process for CFS/ME. Secondly, an evaluation of the LPs conceptualisation of the aetiology of the disease and the hypothesis behind its approach was undertaken. This was achieved through a review of the literature and semi-structured interviews with the programme’s original researcher and developer. Thirdly, a synthesised review of these models was then developed to compare, in order to identify similarities and differences.

Results

The results of the synthesised review are presented sequentially. It begins with models of aetiology and treatment followed by the evidence for, and a description of the LP’s approach.

Models of aetiology and treatment

Aetiology, symptoms and treatment of CFS/ME - Diagnosis of the illness is recognised to be challenging and is complicated by the variance in aetiology and presentations. Onsets can vary widely and include post infection (bacterial or viral), trauma, anaesthetic, drug reaction, emotional stress and unknown aetiology (Panelli, 2017). There is some familial clustering and although genetic factors have been suggested, they remain unconfirmed (Edwards, McGrath, Baldwin, Livingstone, & Kewley, 2016). Symptoms also vary in intensity from case to case and over time, but include post exertional malaise and fatigue that is unresponsive to rest, and often include pain, cognitive impairment, general malaise, autonomic dysregulation, unrefreshing sleep, digestive issues and hypersensitivity to a range of stimuli such as noise, light and scents.

This multifactorial and multisystem illness presents a challenge to a simple single cause and effect model of health and, despite searches for a novel infectious agent, and a single pathognomic test, nothing consistent has yet been identified, resulting in the unsatisfying situation of diagnosis by exclusion. This has resulted in a strong treatment focus on symptomatic relief, with CBT, to help cope with the illness, and pacing, to increase exercise tolerance and reduce inflammation, remaining the main NICE approved approaches (NICE, 2007). In the absence of identifying the aetiology, authors suggest there is a need to explain the physiological disturbance behind the symptoms (Edwards et al., 2016).

The physiological disturbance and the LP hypothesis of the disease process. There is general agreement (Edwards et al., 2016) concerning the importance in the disease process of the activation of the Sympathetic Nervous System (SNS), sensitisation of the Central Nervous System (CNS), dysregulation of immune and Hypothalamus Pituitary Axis (HPA) systems, and addressing issues within these systems are central to the LP model. The hypothesis of the LP is that although the symptoms of the disease are precipitated by the original agent or incident, they are maintained by the aberrant ongoing response to that original event, which disrupts the usual process of recovery. As a result the
LP’s primary focus is on considering how to restart the disrupted recovery process. Although there are a number of proposed theories accounting for this disrupted recovery process, such as the severity of infection (Hickie et al., 2006) and genetic predisposition (Falkenberg, Whistler, Murray, Unger, & Rajeevan, 2013; Nater et al., 2008), the aetiology of why the recovery process is disrupted is currently uncertain.

Disrupted Recovery Process. Physical, chemical and even emotional threats to the body, independent of their cause, trigger a stress response (Selye, 1936) which, the LP theory suggests, has a significant impact on the disease process. In the LP this stress response is termed the Physical Emergency Response (PER) to clarify its physical nature and distinguish it from the more common interpretation of stress as a purely cognitive-emotional response. This additional terminology was created to clarify the LP’s position that, although it is considering the physiological effects of the humoral stress response, it is not suggesting the illness is psychological in origin.

The short-term activation of the PER is a valuable adaptive physiological shift to threat, however there are physiological consequences to its activation. These include: temporary arousal of the Sympathetic Nervous System, changes in blood flow to the limbs and away from most organs, alteration in blood sugar management, a switch from reflective to more reactive cognition, a decrease in digestive function, increased vigilance and an interruption of sleep and a suppression of immune function.

The LP hypothesis suggests two stages to the development of CFS/ME and these elements are supported by a number of authors (see figure 1):

1) That in CFS/ME the PER begins to become chronically activated, causing 1) dysautonomia and neurological sensitisation, 2) altered immune responses, 3) impaired digestion, 4) disrupted sleep and 5) poor cognition. These disturbances correlate to key symptoms of the illness, 1) symptoms in multiple systems including neural, muscular and circulatory systems (Wyller, Eriksen, & Malterud, 2009) 2) non-recovery of original and subsequent infections, poor lymphatic drainage of tissues (Perrin, 2005), inflammation in CNS (Nakatomi et al., 2014) 3) food intolerances and Gastro Intestinal symptoms (Lakhan & Kirchgeissner, 2010) 4) poor, unrefreshing sleep 5) brain fog, difficulty concentrating.

2) That the CNS role in dampening down this PER activation and restoring homeostasis is also interrupted by the ongoing PER via two mechanisms. Firstly, the flood of unusual, alarming signals from the wide range of affected systems have an overwhelming effect on the CNS. This produces an overwhelming array of information for the CNS process, which requires detailed and appropriate responses. Normally this would be manageable, however, the second consequence of the PER is the affect it has on the CNS’ ability to process and respond to these signals in an effective way. This is due to the direct impact of altered hormonal and neurotransmitter levels, caused by the PER, on the neurological tissues (Joëls & Baram, 2009; Popoli, Yan, McEwen, & Sanacora, 2012).

These altered levels result in changed synaptic thresholds and altered signal processing (Landgrebe et al., 2008). Simply put, this means signals that should be amplified, such as increasing blood flow or lymphatics to the muscles, can be supressed, and others that should be quietened down, such as pain from tissues, can be amplified. This inability of the CNS to respond appropriately to the incoming signals or manage the outgoing instructions to the tissues prevents effective re-regulation of the various systems affected by the disease process. As a result, the unresolved physiological changes perpetuate and worsen, creating an additional threat to the body and re-triggering a further cascade of PER changes (Craddock et al., 2014).

As this altered body-wide state of dysregulation and sensitisation continues, a further factor, neuroplasticity, begins to have an effect. Neuroplasticity is the ability of the nervous system to change as a result of usage and is vital in the process of learning and responding to change. This ongoing adaptive process causes pathways that are most used to become faster, easier to activate and have a bigger effect on brain function as a whole. Unfortunately, in this case, the repeated activation of these disruptive pathways results in them becoming more influential and efficient, and this enhances the stability of the altered neurological and physiological responses (Edwards et al., 2016). The effect of this widespread dysfunction can affect all body systems and produce a wide range of fluctuating symptoms, which are therefore too extensive to list here. However, with fatigue and muscular symptoms being a core symptom of the illness, the muscular system is particularly interesting to consider further. The reduction in activity levels caused by the altered physiology, as described above, has a direct effect on the condition of muscles and also on the venous and lymphatic fluid circulation. The fluid circulation in these systems partly relies on movement to encourage the return of fluid from the extremities; as a result, a lack of
activity prevents good function of these vital systems. As they have an essential role in removing the by-products of metabolism from the tissues, and are an essential part of the circulatory and immune system, poor function in these systems creates a worsening of symptoms across all systems, compromising immune function and creating an extra threat. This threat triggers a further PER. Finally the psycho-social effects of experiencing the severity of the illness, the lack of sustained recovery and sometimes the lack of awareness of this ‘invisible’ illness by others, creates a further threat response, adding another turn to this now self-perpetuating cycle (Falkenberg et al., 2013).

**LP approach: Evidence and description**

**New intervention possibilities.** Considering the disease as a disruption of the normal recovery process raises interest as to how to restore normal homeostasis in the systems affected. Approaches that could directly influence these homeostatic systems might have an impact on the disease process. These homeostatic mechanisms are usually managed by an involuntary process of minutely adjusting and checking to ensure levels are within normal limits. As a result of this moment by moment variability, pharmaceuticals, that are difficult to deliver with the precision and variability required, are not the first choice for management of such systems. It was also considered that these involuntary systems were beyond conscious control. However research has suggested that, with training, it is possible to have influence on such systems, including blood pressure and heart rate (Campbell, Labelle, Bacon, Faris, & Carlson, 2012; Carlson, Speca, Faris, & Patel, 2007; Chen, Yang, Wang, & Zhang, 2013), blood sugar levels (Hartmann et al., 2012; Rosenzweig, Reibel, Greeson, & Edman, 2007; Youngwanichsetha, Phumdoung, & Ingkathawornwong, 2014), SNS activation, temperature regulation and immune system function (Carlson, 2012; Davidson, 2003; Ditto, Eclache, & Goldman, 2006; Kox et al., 2014), improved muscle function (Brick, McElhinney, & Metcalfe, 2018) and hormone production (Speer, Bhanji, & Delgado, 2014; Speer & Delgado, 2017).

**The LP approach.** Supported by this research, the LP’s approach is to systematically develop individuals conscious influence on their CNS, and through that on homeostatic mechanisms. In advance of attending a seminar, the training programme begins with a pre-course audio programme that highlights the concepts of conscious control of the CNS and the consequential role of the patient as an active participant in the change process. This is followed up with a conversation with a practitioner to answer any questions and help the individual decide if they wish to proceed with this approach. On attending the three consecutive half day’s seminar, the individuals are coached through a three-phase strategy: Awareness; Interruption; and Redirection, outlined below. The three consecutive day

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**Figure 1: The self-perpetuating spiral. Adapted from Parker (2012)**

[Diagram showing the self-perpetuating cycle of Neuroplasticity, PER/Stress Response, Neurological sensitisation, Dysregulation of Immune System and HPA, Multiple systems affected, Overload of input and neurological sensitisation affects CNS ability to dampen this cycle, Increase in symptoms, system wide dysfunction, effects on blood and lymphatic flow, reduced activity levels, etc., Neuroplasticity enhances stability of cycle through usage, Original causative agent.]

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structure provides opportunities for deeper familiarity with the tools, practice, focused coaching and feedback on progress. Once the seminar is completed, a minimum of three hours, and more if required, follow up sessions are provided to support the adoption of the new skillset.

**The three-phase strategy.** For the purpose of this paper there follows a brief outline of the process, however the details of the complete 3 day process is also available for those interested in a fuller understanding of its mechanics (Parker, 2013).

**Awareness:** A key element of the training is for the individual to develop an awareness of which neurological pathways they are activating. They learn to identify if those pathways contribute to the homeostatic imbalance, or encourage the restoration of function and health. Participants are introduced to the research into conscious influence on physiological processes, and the association between the use of language and changes in neurological activity (Eck, Richter, Straube, Miltner, & Weiss, 2011; Richter et al., 2014; Richter, Eck, Straube, Miltner, & Weiss, 2010). The trainer also assists the participants to develop a new awareness of the type of language being used, which helps them to notice which pathways are being activated.

**Interruption:** Once the activation of unhelpful pathways has been identified a number of cognitive, linguistic, embodied cognition and gentle movement techniques are used to interrupt those pathways. This interruption process is deigned to alter the involuntary use of these pathways (Adamczyk & Bailey, 2004). Interrupting these dysregulating pathways has been found to encourage improved neuro-endocrine function and resilience (Barber, Bagsby, & Munz, 2010; Burgdorf, M. Colechio, Stanton, & Pankspepp, 2017; Carney, Cuddy, & Yap, 2010; Cohen & Pressman, 2006; Faymonville, Boly, & Laureys, 2006; Posner, Russell, & Peterson, 2005; Quoidbach, Berry, Hansenne, & Mikolajczak, 2010).

**Redirection:** The final phase is to adopt a compassionate self-coaching role. This provides a mechanism to gain access to effective coaching by applying the skills of coaching to oneself. A structured self-coaching strategy is then employed to firstly access a sense of self-compassion and support (Neff, Kirkpatrick, & Rude, 2007). Secondly, to help the individual to identify what their desired affective or physiological state is (Bandler & Grinder, 1979; Duckworth, Kirby, Gollwitzer, & Oettingen, 2013). And thirdly, to encourage the activation of that desired state, by structured and detailed re-vivification of appropriate reference desired states/memories whenever required (Faymonville et al., 2006; Grinder & Bandler, 1981; Langer, 2009; Quoidbach et al., 2010; Speer et al., 2014; Speer & Delgado, 2017). This gives the individual an opportunity to activate new pathways that encourage improved physiology and restoration of homeostasis.

It is proposed that through using this sequence, and via instrumental learning and neuroplastic processes, provided by repetition, the old ‘anti-homeostatic’ pathway can be ‘hijacked’ and rerouted, increasingly by default, to trigger new more helpful pathways (Briones et al., 2005; Hunter & Stewart, 1993; Murphy & Corbett, 2009; Vrensen & Nunes Cardozo, 1981).

Once the tools have been mastered, physical and mental repetition processes are taught to enhance the familiarity with the new neurology and to prepare for specific situations which have been identified as previously symptom producing; these include implementation intention (Gollwitzer, 1999) , pseudo orientation in time (Erickson, 1954), future pacing (Grinder & Bandler, 1981) and brain rehearsal (Parker, 2013) approaches.

**Increasing physical endurance.** Changing the physiological response to exercise is a priority for recovery from CFS/ME and so is discussed in more depth here.

The standard models employed are pacing, CBT and GET. These models primarily advocate gradual change through small step, incremental usage and the physiological effects of exercise (Cox, Ludlam, Mason, Wagner, & Sharpe, 2004). The CBT elements often add coping strategies for managing the illness and an identification of where cognitive appraisals of lack of ability are at odds with actual ability. Instead, the LP employs a pacing approach to recovery, combined with a neurological model for influencing physiological change and increasing exercise tolerance. This appears to contribute to the rapid change in ability experienced by many (Reme et al., 2013). However, due to the dominance of the other models, and their experience of slow and variable change, it has given rise to caution about the LP approach. This in turn has fuelled inaccurate opinions, based on those models, that the LP must encourage individuals to ignore their illness (Reme et al., 2013). From the outline above, it can be seen that this opinion does not reflect the actual approach.

**The LP approach to increasing physical endurance.** Through self-coaching, and initially with support to ensure goals are achievable, appropriate desired exercise goals are set (this could be walking 10 steps or running a mile depending on the current level of severity of the illness). The LP tools are then used to improve...
physiological ability prior to increasing any physical exertion, based on the brief description of the 3 phases outlined above. This is not simply pretending, hoping or ‘faking it’, and it requires some practice to actually make a physiological shift, through use of in-depth re-vivification of previous successful exercise experiences.

Once the individual feels confident they have assisted their physiology to change to the required level for the exercise goal, the exercise is commenced. If during the exercise they feel they might be over doing it or notice symptoms or signs of activating physiologically unhelpful pathways, then the LP approach is to stop the activity. Next they take a supportive self-coaching role towards themselves, and then use the LP tools once again to shift their physiology until it is at an appropriate level to continue on with their goal. However, if they are unable to achieve the required change in physiology to continue, or feel their current physical limit of endurance has been almost reached, then the activity is stopped. Whatever the outcome they are asked to remain supportive and kind to themselves throughout this process.

After the physical exercise has finished, the process is then used to assist positive changes in the physiology. In this case it is directed to in-depth re-vivification of previous successful post-exercise experiences. This encourages physiological processes that support effective recovery in muscles and joints that maybe unused to exercise and to avoid post exertional fatigue associated with the condition (Brick et al., 2018; Speer & Delgado, 2017).

**Adaptation.** As many of those seeking help for this condition are unable to concentrate for extended periods, tolerate noise or light, or travel, the process is often adapted to meet their needs, and can be taught in manageable chunks of 10 minutes or less, at low sound levels, in the dark and at home, to meet participants needs. As a result the range of those receiving the approach spans from those able to attend sessions in a clinic to the extremely debilitated.

**Discussion and Conclusion**

This paper set out to review the LP’s perspective and approach to this debilitating disease and to contextualise the approach within current theories and research. And as such it is the first peer reviewed article to explore these themes. It found that the LP is aligned with current models of the disease process and it adopts a multifactorial, multisystem disease process model for the disease. It identified that the LP shared the established perspective that the illness is a physiological, and not a psychological, one. It found the LP applies a self-regulation approach to neuro-physiology processes to influence the physical disease process. The review identifies that, although supported by the literature, these hypotheses have yet to be evidenced experimentally. It is hoped that this paper encourages further quantitative research that tests these hypotheses through fMRI and biochemical analyses. It is also hoped that a review of the existing evidence supporting the efficacy of the approach is undertaken to further develop the evidence base.

In conclusion this paper resolves the identified gaps in the research and clarifies the hypotheses behind this approach. It is hoped this clearer understanding of the approach will assist researchers, clinicians and those with this disabling disease to identify some additional options for potential recovery.

**Bibliography**


