We are IntechOpen, the world's leading publisher of Open Access books Built by scientists, for scientists

6,900
Open access books available

185,000
International authors and editors

200M
Downloads

Our authors are among the

154
Countries delivered to

TOP 1%
most cited scientists

12.2%
Contributors from top 500 universities



WEB OF SCIENCE™

Selection of our books indexed in the Book Citation Index in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?
Contact book.department@intechopen.com

Numbers displayed above are based on latest data collected.
For more information visit www.intechopen.com



Chapter 8

Countermeasure Development for Lumbopelvic Deconditioning in Space

Andrew Winnard, Dorothee Debuse and Nick Caplan

Additional information is available at the end of the chapter

http://dx.doi.org/10.5772/intechopen.72881

Abstract

Physical inactivity and lumbopelvic deconditioning have been linked to increased incidence of non-specific low back pain (LBP) and spinal injury in those who are exposed to microgravity (e.g. astronauts and individuals on long-duration bed rest) and in the general population. Astronauts have an increased risk of experiencing moderate to severe LBP during microgravity exposure and herniated intervertebral discs within 1 year following spaceflight. Atrophy and reduced motor control of the lumbar multifidus (LM) and transversus abdominis (TrA) muscles resulting from periods of deconditioning are linked to non-specific LBP and spinal injury risk in both post-flight astronauts and general populations. However, voluntary recruitment of these two key muscles is difficult and presents a rehabilitation challenge. This chapter reviews the concept of spinal stability as it relates to microgravity, discusses how existing exercise countermeasures used in space do not successfully maintain lumbopelvic muscle size, and introduces the functional readaptive exercise device (FRED) that shows potential to activate the LM and TrA muscles automatically and in a tonic fashion, which has relevance to rehabilitation of both astronaut and terrestrial populations.

Keywords: spinal, lumbopelvic, deconditioning, rehabilitation, astronaut

1. Introduction

There is a 53–68% risk of experiencing moderate to severe low back pain (LBP) during microgravity exposure [1] and fourfold increased risk of herniated intervertebral discs within 1 year following the spaceflight [2], which demonstrate a need to understand the underlying mechanisms of LBP and spinal changes that result from exposure to microgravity. Developing an effective rehabilitation programme to address and rehabilitate spaceflight-related spinal changes is also required. Atrophy and reduced motor control of the lumbar multifidus (LM)

IntechOpen

© 2018 The Author(s). Licensee IntechOpen. This chapter is distributed under the terms of the Creative Commons Attribution License (http://creativecommons.org/licenses/by/3.0), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited. CC BY

and transversus abdominis (TrA) muscles, resulting from periods of deconditioning, are linked to non-specific LBP and spinal injury risk in both post-flight astronauts and general populations [3–6]. However, voluntary recruitment of these two key muscles is difficult and presents a rehabilitation challenge [7].

This chapter discusses the key factors contributing to lumbopelvic deconditioning in spaceflight and shows how exercise countermeasures against deconditioning can be developed to aid both astronauts and people on Earth with LBP.

2. Link between segmental spinal stability and upright sagittal spinal motor control on Earth

Non-specific LBP is experienced in the lower region of the spine and is not attributable to a known cause or specific pathology such as infection, systemic disease, fracture or cauda equina syndrome [8]. Non-specific LBP is often multifactorial in its origin; this makes it complex to treat. Panjabi [9] was one of the first to recognise that abnormal spinal mechanics may be a common factor in people with back pain. He identified several potential triggers and causes of abnormal mechanics, including inflammation, biochemical and nutritional changes, immunological factors, structural changes in discs and endplates.

Atrophy [4, 10–13] and altered motor control [4] of the LM and TrA muscles are linked with the common symptom of altered mechanics. Both muscles have a substantial body of evidence linking their dysfunction and atrophy with LBP on Earth [4, 6, 14–18]. Similar patterns of muscle atrophy and LBP have also been observed in those who are exposed to microgravity [5, 6, 19, 20].

2.1. Deep and superficial lumbopelvic muscles in spinal stability

The paraspinal muscles can be divided into deep and superficial muscles based on a structural model of the spine presented by Bergmark [21] who provided the following definitions. **Deep muscles** have their origin and/or insertion at the vertebrae and have an action that includes controlling the curvature and/or structural stiffness of spine. Deep muscles include the LM and TrA muscles. The LM muscle controls and stabilises lumbar lordosis [22] during force transfer through the spine [23, 24] and provides segmental stiffness [25, 26]. The TrA muscle provides a transverse force, therefore increasing stiffness and extrinsic stability of the spine [4] by increasing intra-abdominal pressure [14, 27]. **Superficial muscles** control the large spinal movements and transfer loads between the thorax and pelvis, they do not directly increase stiffness or stability of the spine at a segmental level [21] but can increase global trunk stiffness [28]. Superficial muscles include erector spinae, the internal and external oblique muscles, rectus abdominis, quadratus lumborum and psoas.

Bergmark [21] also defined stability in engineering terms as the ability of a structure to maintain its equilibrium under loading. This definition was extended to define clinical spinal

http://dx.doi.org/10.5772/intechopen.72881

stability as the ability of the spine, under physiological loads, to limit structural displacement so as to prevent damage to spinal structures including the discs, ligaments and neural structures. The spine gains passive stability from bones, ligaments, tendons and fascia, while it was suggested that active stability is provided by deep muscles [21]. Studies using in vitro cadaveric specimens of human spinal segments found that the specimens became mechanically unstable at loads much less than those experienced by in vivo spines [25]. This finding highlighted the importance of the stabilising force provided by the LM and TrA muscles in allowing the spine to function under everyday loading.

2.2. Spinal stabilising system and motor control

To achieve spinal stability, the deep muscles must be controlled by precise coordination of activation and timing. The complete spinal stabilising system was, therefore, conceptualised by Panjabi [29] as having a neural control element, a passive spinal column (and ligaments) and an active system of deep muscles. The control system assesses and directs the deep muscles to provide varying levels of extrinsic stability while the passive elements of the spinal column provide intrinsic stability. To successfully provide control, actions are based on feedback from both the active and passive components. Mechanoreceptors in the passive structures indicate levels of force and stress, while feedback on muscle activation patterns and stretch are provided by the active system. In addition to the muscle feedback system, there is now strong evidence that LM and TrA are ideally activated in a feedforward mechanism, that is, they act in anticipation of changing loads. Importantly, the dysfunction of this feedforward control system has been linked with LBP [28].

2.3. Segmental stability and the neutral zone

During dynamic loads into spinal flexion and extension, there is displacement of each vertebra, which allows flexibility. At low loads, the spine was observed to be highly flexible and then stiffening as loads increased. A neutral zone was defined as the range of segmental displacement within which there is a minimal resistance to the displacement [29]. This is represented graphically in **Figure 1** with the neutral zone being represented by a ball in a bowl. The motion of the ball represents the displacement motion of the vertebral segment, while the steepness of the sides represents varying stability with steeper sides demonstrating increased resistance to displacement. As segmental spinal stability increases, the neutral zone becomes smaller, demonstrated by placing the ball in a wine glass. As segmental spinal stability decreases, the neutral zone gets larger, demonstrated by placing the ball in a flat bowl (**Figure 1**).

It was hypothesised that decreased stability may be caused either by damage to the passive stability system and/or abnormal activity or control of the active system that leads to a larger neutral zone [29]. An increase in the neutral zone is likely to be associated with increased stress on spinal structures so, it results in pain. Therefore, interventions were suggested for unstable painful spines which aimed at reducing the neutral zone through retraining control of the active stability system or through the use of spinal fusion [29].

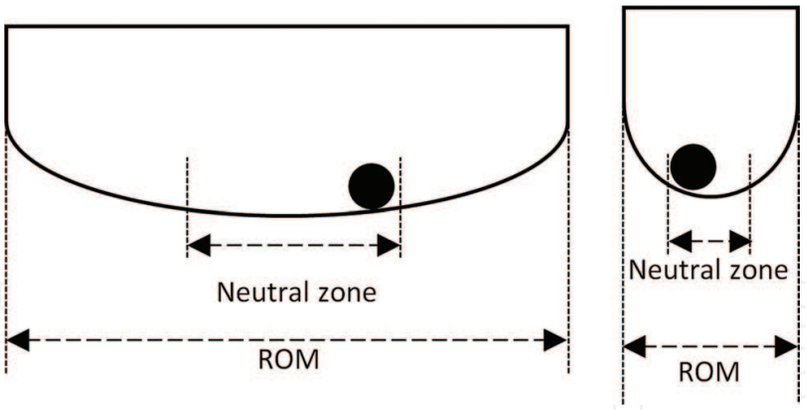


Figure 1. A visual representation of the neutral zone as a flat dish with less stability (left) and a wine glass with more stability (right) (Adapted from Panjabi [29]).

2.4. Theory linking low back injury with altered motor control and low back pain

The theory of how low back injuries can cause altered motor control and lead to low back pain was summarised by Panjabi [9] as follows:

1. Initial trauma occurs in spinal structures such as ligaments. This can be either a long-term build-up of microtrauma or an acute injury.
2. During dynamic loading of the injured spine, mechanoreceptor signals sent to the neural control system, produced by the injured tissue are now corrupted due to injury.
3. The motor control area of the brain finds a mismatch between expected signals and those actually being received. This causes control unit output to the active stability system in response to dynamic loading to also become corrupted.
4. Corrupted output from the control unit leads to the changes in the activation of the deep muscles in response to the dynamic load. These changes lead to abnormal activation and timing of the active stabilising deep muscles—LM and TrA. This then causes altered spinal mechanics.
5. Abnormal activation patterns of the deep muscles causes their returning feedback to also become corrupted, causing further mismatch in signals being received by the control unit.
6. Increased corruption of control unit output occurs in response to continued dynamic loading. This has great potential to lead to segmental instability, increased segmental neutral zone and higher stresses on spinal structures.
7. Inflammation of stressed spinal tissues around unstable segments is then likely to occur and nociceptive pain signals produced.
8. If left unchecked, chronic non-specific LBP may develop.

Evidence supporting these hypotheses exists from several experimental studies. Danneels et al. [11] conducted a comparison study of chronic LBP and matched non-symptomatic participants,

which found reduced cross-sectional area of LM in the lower lumbar spine. In the study, 32 clinical participants were compared with 23 matched no-LBP volunteers, and the LM cross-sectional area was measured using CT scans. A study in pigs by Hodges et al. [12] found that induced L4 spinal disc lesions resulted in LM cross-sectional area decreases at the same level of the injury within 3 days, compared to no change in no-LBP controls. Injury to the L3 nerve root resulted in LM cross-sectional area reduction at the affected level and down to L4, L5 and S1 levels in 15 induced injury pigs compared to six controls. The controls were, however, still subjected to a sham surgical procedure that involved all the same steps as the injured pigs apart from the inducing of the injury. A comparison study by Hides et al. [3] of 26 first episode acute unilateral low back pain patients with 51 healthy controls, found that LM asymmetry in the people with back pain, isolated to the symptomatic level compared to symmetrical LM muscles in the no-LBP controls. A comparison study by Hodges and Richardson [4] of 15 LBP patients with 15 no-LBP matched controls used electromyography to assess the activation and timing of TrA in response to upper limb movements. It was observed that TrA activation was consistently delayed in the people with back pain. A comparison study by Ferreira and Hodges [13], in which 10 low back pain patients compared with 10 healthy matched controls, found consistently reduced changes in TrA thickness in their group with back pain during lower limb exercises, which was measured using ultrasound imaging.

While Panjabi's theories were seminal to improve our understanding of lumbopelvic pain, they missed one important factor, and that is the fact that, unlike the feedback control of many superficial muscles, there is a feedforward control of the deep spinal muscles LM [24, 30] and TrA [31, 32]. In other words, the LM and TrA muscles work in anticipation of loads and movements, not in response to them, thus, providing spinal stability. Importantly, there has also been evidence that the anticipatory activation of the deep spinal muscles is impaired in people with LBP compared to non-symptomatic controls [4, 31, 33] and that this is reversible with certain exercise approaches [34]. Based on a considerable body of literature, key authors in the field [16, 35] also suggest that secondary compensatory postural mechanisms are likely to contribute further to LBP, which would go some way towards explaining chronic LBP in the face of minimal tissue abnormality. It should be noted that the studies used to have small sample sizes may not be strongly statistically powered.

2.5. Lumbopelvic adaptations to microgravity

Astronauts returning from long-duration space missions (~6-months duration) [36] and participants following long-duration bed rest studies, which are commonly used to simulate microgravity exposure [37], have a range of muscular and postural problems. Human spaceflight results in exposure to an altered gravity state, mostly eliminating weight bearing and axial loads, resulting in physiological changes and potentially increased injury risk [36, 38, 39]. Buckey [36] grouped these changes into broad themes allowing them to be listed briefly as follows: bone loss, psychosocial, radiation biological, muscle loss, balance and postural control, cardiovascular and nutritional. These changes include decreased balance and proprioception, decreased muscle mass, force and power with increased loss of technique (specifically affecting lower limb antigravity muscles and lumbopelvic segmental control muscles) [36], decreased ability to control posture—specifically, the ability to achieve a balanced pelvic tilt

and normal spinal curves in the sagittal plane, increased risk of spinal injury from poor spinal positioning during everyday activities—especially involving trunk flexion, increased chance of poor global movement patterns, and risk of injury from musculoskeletal weakness and atrophy [5, 19, 40, 41]. Those in microgravity also experience lengthening of the spine due to swelling and hyper-hydration of the intervertebral discs which, in turn, become deconditioned resulting in increased risk of disc injury [19].

Gernand [38] reported the implications of these physiological changes on subsequent safe functioning when returning to a gravity-loaded environment, highlighting the need for both countermeasure interventions during spaceflight and rapid and effective rehabilitation following spaceflight. For spaceflight of around 6 months, Gernand [38] noted significant bone and muscle loss, as well as altered postural control, leaving the body susceptible to fractures, muscle injury and the potential to develop osteoporosis. Muscle atrophy and altered motor control have been specifically observed in the lumbopelvic region [42]. A European Space Agency (ESA) report by Snijders et al. [43] reported LBP in 12 out of 20 astronauts during spaceflight. The report highlighted the importance of maintaining spinal movements, as end range flexion and extension exercises were anecdotally noted as being employed to ease pain during spaceflight. A relationship was also highlighted between LBP and atrophy of deep spinal muscles, particularly LM, during bed rest studies [44]. Wing et al. [1] reported that 53–68% of astronauts experienced moderate to severe back pain when in space. On landing after a shuttle mission, a US astronaut reported severe LBP which was later linked with a herniated nucleus pulposus at the L4-5 intervertebral (IV) disc and required surgical intervention [2]. Johnston et al. [2] also reported that astronauts had a more than fourfold increased risk of herniated disc pulposus within the first year following spaceflight, compared with controls. Sayson and Hargens [42] suggested that this back pain and disc injury could be caused by a range of factors linked to spinal lengthening and reduced loading. A review by Belavy et al. [19] supported this, suggesting the increased lumbar IV disc herniation risk in the astronaut population was most likely caused by long-term disc tissue deconditioning resulting from swelling of the discs due to unloading during spaceflight. However, the review only considered IV discs in isolation and did not refer to any potential predisposing factors such as spinal motor control. It should be noted that data from actual astronaut studies are usually from small samples, and therefore, statistical power is often low. Earth-based simulation studies, such as bed rest studies, are therefore, useful to increase the overall sample size on which to base conclusions.

Lumbopelvic adaptations to microgravity include adoption of a flexed posture (**Figure 2**) [36], spinal lengthening, increased intervertebral disc height and disc deconditioning, altered spinal curvatures [42] and atrophy of the lumbopelvic musculature. Selective atrophy of spinal extensors without corresponding atrophy of the psoas muscle was also seen in terrestrial individuals with LBP compared to no-LBP controls by Danneels et al. [11]. Atrophy and motor control changes in the LM muscle have been linked with LBP [3, 10] and development of poor intersegmental control of the lumbar spine [15, 21, 27, 45], which can potentially cause increased stress on spinal structures, resulting in pain [25, 29, 46].

Humans exposed to sustained microgravity develop a risk of significant spinal injury as a result of microgravity-induced poor intersegmental control of the lumbar spine combined with loaded activities, such as extra-vehicular activity, physically demanding medical procedures, landing

http://dx.doi.org/10.5772/intechopen.72881

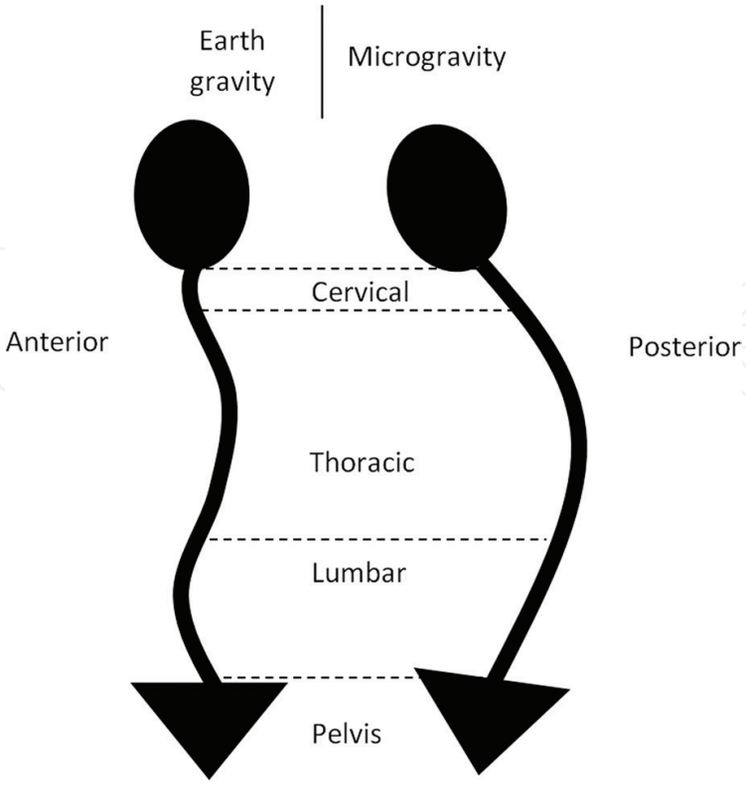


Figure 2. Postural adaptation to microgravity, showing loss of normal spinal curvature and increased flexion of the spinal column.

and return to a g-loaded environment, which have the potential to be at least as demanding as those undertaken in normal Earth gravity [38]. It is necessary, therefore, to know what physiological changes occur, which could lead to increased injury risk, and which interventions, both preventative and rehabilitative, can be used to minimise and effectively rehabilitate physiological compromise. The current evidence also suggests that interventions to address lumbopelvic physiological adaptations are likely to be a required element of any rehabilitation programme following exposure to microgravity. While Evetts et al. [47] indicated that European post-flight rehabilitation includes specific training for lumbopelvic posture and spinal muscles involved in intersegmental control of the lumbar spine, they highlighted a need to compare the effectiveness of interventions to advance the treatments given to astronauts. Such improvements are also likely to aid terrestrial healthcare with more effective interventions for people with LBP and post-bed rest rehabilitation [47].

3. Management of low back pain on Earth using the motor control approach to improve spinal stability and control

Management of segmental instability using specific motor control exercises aimed at normalising the recruitment patterns of the deep muscles was first summarised by O'Sullivan [48]. The first stage of training is learning to isolate and correctly voluntarily contract the deep muscle system. The voluntary contractions are intended to be low level and at 30–40%

maximal voluntary contraction. Contractions are taught in postures such as supine, prone and four-point-kneeling while patients are asked to perform abdominal drawing in using TrA while maintaining a neutral lumbar lordosis. In addition to this, patients are taught: differentiation of lumbar, pelvic and hip movements and diaphragmatic breathing and maintenance of neutral lordosis in different postural sets such as sitting and standing.

Live biofeedback with use of palpation, ultrasound imaging or possibly electromyography can be included to help isolate TrA and LM activation [49]. Treatment is then progressed to the second stage where the deep muscle recruitment learned in stage one is incorporated into functional movement, and compensatory muscle strategies are discouraged. Patients are taught movements such as sit to stand, walking, bending and twisting while maintaining activation of deep muscles. The third and final stage of training is for patients to carry the newly learned and stable functional movements into their activities of daily life.

The 'bare bones' have evolved since 2000 into an evidence-based and integrated approach which is summarised by Hodges et al. [28] and too complex to discuss here. However, it is based on a considerable amount of good evidence, one of which will be summarised in the following paragraph.

Hides et al. [49] assessed LM size in athletes with LBP and determined the effectiveness of a motor control intervention. Ten participants with back pain underwent a 6-week intervention programme of learning to correctly activate TrA and LM. Live biofeedback using ultrasound imaging was used during muscle activation teaching. Abdominal drawing in exercises were used to teach recruitment of TrA while maintaining a normal, relaxed, breathing pattern, followed by participants attempting to swell the LM muscle while holding a breath out and keeping the spine still with a neutral lumbar lordosis. Initially, activation was taught in lying and then progressed to upright sitting and standing, all the while maintaining a neutral lumbar lordosis. Further progression to functional movements was then performed. By the end of the programme, pain scores had dropped from an average of 4.3–2.3 ($p < 0.05$). Before treatment, asymmetry had been observed in LM cross-sectional area, which also significantly decreased, while overall muscle size increased. This is just one study that provides evidence that motor control exercises including recruitment of deep muscles can improve clinical outcomes.

4. In-flight countermeasures for lumbopelvic deconditioning in space

On the International Space Station (ISS), astronauts take part in up to 2.5 h of exercise each day including running, cycling and strength training. These exercise countermeasure programmes are known to be relatively successful at preventing bone loss and loss of muscle mass in some regions of the body. However, as mentioned earlier, they are not specifically targeted at preventing lumbopelvic muscle loss.

A number of bed rest studies have investigated potential inflight countermeasures, reporting on their effects on lumbopelvic musculoskeletal parameters. However, no studies have tested

http://dx.doi.org/10.5772/intechopen.72881

the operational countermeasures currently in use on the ISS [50]. Tested countermeasures included lower body negative pressure treadmill running, resistance exercises with external vibration, resistance exercise alone, rowing like exercises using a flywheel device and self-performed exercises designed to mobilise the spine. In a recent systematic review of these bed rest studies, Winnard et al. [50] identified that no single potential countermeasure can successfully prevent all lumbopelvic musculoskeletal adaptations to simulated microgravity. For example, resistive vibration exercise was the only countermeasure, which is able to protect against lumbopelvic muscle adaptations, but it did not prevent spinal morphology changes such as loss of lumbar lordosis. No other countermeasure tested was able to do more than partially prevent (at best) the lumbopelvic muscle adaptations. This demonstrates a need for further research into new interventions to better protect the spine during microgravity exposure. It might be possible to translate ground-based interventions into new countermeasures or develop new ones based on the current lumbopelvic deconditioning rehabilitation theory. Any new interventions will need testing in ground-based microgravity simulations before incurring costs associated with actual spaceflight testing. Lower body negative pressure treadmill was the most effective currently researched countermeasure against lumbopelvic deconditioning. As this countermeasure is not yet used in operational spaceflight and no countermeasure is fully effective, there remains a need for rehabilitation.

5. Rehabilitation following actual and simulated spaceflight

Due to the lack of effective in-flight countermeasures targeted at preventing lumbopelvic musculoskeletal adaptations, astronauts require significant rehabilitation to reduce injury risk on their return to Earth. During spaceflight, a general pattern of selective extensor muscle atrophy has been seen throughout the body [51]. Decrease in spinal extensor volume has been reported as being greater than hip flexor (psoas muscle) decline in astronauts [52]. Anecdotal accounts also appear to show selective atrophy of trunk extensor muscles concomitant with improved flexor muscle performance immediately post mission [53]. This muscle imbalance results in temporary loss of lumbopelvic posture, flexion of thoracic spine and hyperextension of cervical spine, the centre of gravity is moved anteriorly and increases the risk of musculoskeletal injury [47]. Hides et al. [40] suggested that deep spinal muscle changes such as atrophy of LM and TrA muscles, along with selective hypertrophy of spinal flexors over extensors [5], may impact on the ability of the spine to distribute loads appropriately shortly after spaceflight simulation via bed rest. The European approach to post-space mission rehabilitation addresses the muscle imbalance and uses motor control training in a way very similar to that described for people with LBP on Earth and based mostly on the existing terrestrial evidence transferred to a post-spaceflight setting. Initially, postural control, muscle control and muscle balance are restored, followed by the use of strategies to normalise muscle recruitment. Astronauts are then supported to redevelop postural alignment in line with the centre of gravity and to develop adequate motor control before they start to exercise with elements of loading and strength training. The latter is only started after the astronauts have regained correct postural alignment and control [54, 55].

No studies have investigated lumbopelvic rehabilitation approaches following actual spaceflight, and only one study has investigated rehabilitation approaches following simulated spaceflight (bed rest) [40]. The study assessed specific motor control (SMC) exercises compared to a control group performing trunk and general strength exercise (TFS) programme in a supine position. Results favoured SMC for restoring spinal length and posterior disc height, suggesting it may reduce the risk of IV disc injury during rehabilitation. However, TFS was favoured for training LM muscle and restoring lordosis angle and overall disc volume. Overall, it was suggested that SMC is favourable over TFS because SMC is expected to place less force on the discs and is associated with the lower rate of change in disc volume and anterior disc height [40]. Lower forces on the discs during rehabilitation—at a time when the discs may be deconditioned and vulnerable to injury—is expected to help restore posture and motor control with reduced risk of damage to the discs in the process. Therefore, in line with current ESA rehabilitation practice, a training programme starting with SMC when disc injury risk is high, then progressing to general trunk strengthening once lumbar postural control is restored would seem to be indicated. Other rehabilitation methods that train the LM muscle and maintain lordosis angle, without high axial loading, would also be worth investigating. Additionally, as noted previously, due to the low sample sizes from the terrestrial evidence on which these methods are based, further studies to improve statistical power would be useful to ensure a robust evidence-based approach. While motor control training has been shown to be useful in LBP rehabilitation, and is already used in the rehabilitation of European astronauts [47, 54, 55], many people have difficulty in recruiting LM, in particular, voluntarily [7]. This presents a challenge to physiotherapists involved in evidence-based practice for LBP. Many of the exercises used early in motor control training also lack functional relevance to activities of daily living, and there is a drive to make rehabilitation more functional [45, 56, 57]. As such, new interventions must be developed to address these challenges.

6. Developing a new countermeasure for lumbopelvic deconditioning in space

6.1. Early development of the functional readaptive exercise device (FRED)

In an attempt to address the challenges discussed earlier relating to motor control training, Debuse et al. [53] investigated the effects of a new exercise device, the functional readaptive exercise device (FRED) (**Figure 3**), that aims to recruit the LM and TrA muscles.

FRED exercise constitutes a combination of weight-bearing, an unstable base of support (at the feet), an upright 'standing' posture with a relatively stable lumbopelvic area, and functional lower limb movement, combined with real-time visual feedback of performance. As the FRED offers no resistance to lower limb movement, it requires good balance and coordination in order to achieve a smooth, controlled cyclical motion. Exercise on the FRED has been shown to recruit LM and TrA automatically (i.e. with no conscious effort by participants) and

http://dx.doi.org/10.5772/intechopen.72881



Figure 3. Current prototype of the functional readaptive exercise device.

to recruit them differentially [53]. More recently, FRED exercise has been shown to promote tonic activity of LM, assessed through measurement of superficial muscle activity using surface electromyography [58], as well as the deep lumbopelvic muscles using intramuscular electromyography [59], which is considered the most rigorous way of investigating muscle activity [60]. FRED exercise was shown to result in more selective activation of the LM and TrA muscles than over-ground walking [59], and it was found to reduce lumbopelvic movement when compared to over-ground walking, especially axial rotation of the spine [61]. To date, these studies are all based on normal terrestrial gravity, and the next step will be a clinical trial of the FRED following bed rest as a simulation of space flight. A musculoskeletal modelling study that examined the potential role of the FRED in the recruitment of lumbopelvic muscles in both +1 and 0 Gz environments, Lindenroth et al. [62] predicted that FRED exercise is able to facilitate lumbopelvic muscle recruitment in microgravity similar to how it recruits the same muscles in Earth gravity.

Based on the early research findings relating to the early and current prototypes of FRED, it can be hypothesised that the device uses several mechanisms in combination, to produce rehabilitation effects on several of the problems found in spinal instability simultaneously within one intervention [58], as presented in **Table 1**.

These potential mechanisms show how the FRED has already demonstrated the ability to automatically activate both LM and TrA in an asymptomatic population without need for conscious muscle recruitment. This might have potential to solve the LM and TrA conscious recruitment difficulties found in traditional spinal motor control rehabilitation [7]. The exercise is dynamic, functional, weight-bearing, in an upright posture and relevant to common daily activities such as walking. These are all elements of motor control exercises covered in **Section 4**. It appears, therefore, that the device might be a useful intervention to train the LM and TrA muscles and segmental spinal stability.

Problem	FRED mechanism
Poor lumbopelvic motor control of deep spinal muscles	Exercising using a pattern of moving the feet in a quasi-elliptical path in antiphase with minimal resistance from the device or support from the upper limbs Exercising while maintaining a stable pelvis and upright trunk while having to maintain an even speed within one revolution. The abovementioned points create a need for greater control of the lower limbs and pelvis during an unstable dynamic movement. Greater control is particularly needed in resisting a fast descent of the foot in the forward-most position of the cycle. The movement is functional and similar to over-ground walking. Therefore, muscle activation training is learned in a functional movement, hoped to produce carry over into other functional daily activities. Clinical observations seem to indicate that relatively greater rear foot loading in standing results in greater recruitment of LM, whereas relatively greater front foot loading in standing has a deactivating effect on LM. It is hypothesised that correct exercise on FRED results in reduced front foot loading. FRED provides visual feedback that encourages users to exercise at a constant, controlled speed and frequency ratio, which is hypothetically the most energy efficient movement [63]. Additional feedback encourages users to maintain even movements throughout the exercise, training control of the lumbopelvic area and lower limbs during dynamic functional movements. It is thought that efficient and smooth controlled movement on FRED may improve LM and TrA neuromotor control. The exercise has already been shown to activate LM and TRA without the need to consciously trigger the activation in non-symptomatic populations [53]. In addition to this, LM was shown to have constant tonic activity throughout exercise cycle on the device in an electromyography study. The muscle was active for more time than during over-ground walking [58].
Reduced ability to control spinal posture and balance	Previous kinematic research has shown FRED exercise promotes an increased degree of anterior pelvic tilt during upright posture [61]. Increased anterior pelvic tilt, within a range where the thoracolumbar junction remains the inflexion point between lumbar lordosis and thoracic kyphosis, has been shown to create a well-balanced sagittal spinal posture [64]. Electromyography data have also shown that this type of posture produces the highest LM and TRA recruitment [22], though this study investigated sitting postures. Additionally, users of the device are required to exercise in an upright posture. It is hoped that these elements together mean FRED exercise promotes a balanced upright sagittal posture, with recruitment of LM and TrA. Having improved control of balanced posture is also hoped to improve overall balance.
Atrophy of spinal extensors	EMG data from FRED exercise show that it promotes increased activation of spinal extensors over flexors [58]. This may be relevant to the rehabilitation of astronauts who show increased flexion postures when in space [36]
Weakness of lower limb anti-gravity muscles	Previous kinematic research shows FRED exercise involves constant hip and knee flexion in a dynamic and gravity-loaded exercise, therefore, constantly loading lower limb extensor muscles [61]. This loading is expected to improve strength in the lower limb extensors, which is a common aim of traditional interventions for reducing falls risk in older people [65]

Table 1. FRED mechanisms.

6.2. Developing FRED for rehabilitation from lumbopelvic deconditioning in space

Following the early research on the early prototype of the FRED, a range of mechanistic studies were completed on the prototype shown in **Figure 3** in order to develop it for use as a rehabilitation intervention in groups with lumbopelvic muscle deconditioning (e.g. low back pain, astronauts). Winnard et al. [63] compared the thickness of LM and TrA at a range of movement amplitudes in an asymptomatic sample. A large body of evidence has linked dysfunction and/or atrophy of TrA and LM to lumbopelvic deconditioning and LBP [3, 4, 9, 11, 12]. Importantly, these muscles are difficult to recruit voluntarily, this presents a challenge

in terms of their rehabilitation [7]. Therefore, the automatic recruitment of TrA and LM during FRED exercise would appear to offer an advantage over current practice. Increasing crank amplitude was observed to increase movement variability, the range of TrA and LM thickness peaks, as well as mean TrA muscle thickness [63]. These outcomes are all measures of motor control of either global movement or muscle recruitment.

In a large sample of both symptomatic (LBP) and asymptomatic participants, FRED exercise promoted increased lumbar extension and anterior pelvic tilt compared to over-ground walking [64]. Attaining a lordosis throughout the lumbar spine below the thoracolumbar junction is a common goal of current interventions [48]; it is the sagittal spinal position where LM tends to be most effectively recruited [22, 24, 65]. Although this finding alone does not indicate that the correct lordosis is promoted by FRED exercise, when combined with the finding that FRED exercise appears to recruit key lumbopelvic muscles automatically, there is increased likelihood that the spinal position promoted during FRED exercise is more conducive to LM recruitment than walking.

In the same sample, the FRED caused increased anteroposterior and mediolateral centre of mass variation compared to walking [64]. This suggests an increased challenge to balance and, therefore, motor control during FRED exercise. This may form part of the motor control mechanism of FRED exercise and adds to the overall evidence that FRED exercise is in line with current motor control interventions and adds weight to the justification for a clinical trial.

7. Conclusion

Astronauts undergo significant lumbopelvic musculoskeletal deconditioning following their exposure to microgravity. Many experience low back pain, and there is a fourfold increase in the incidence of intervertebral disc injury on their return to Earth as compared to their non-astronaut peers. It is known that the spine lengthens, normal posture is lost, intervertebral discs change their morphology, LM and TrA muscles atrophy and a flexor-extensor lumbopelvic muscle imbalance occur during spaceflight. Current in-flight countermeasures aim to generally prevent physiological adaptations to microgravity. However, they are not specifically targeted enough to do so for the lumbopelvic spaceflight adaptations. Astronauts, therefore, require rehabilitation upon return to Earth's gravity to reduce injury risk. The European rehabilitation interventions follow current evidence-based practice for treating people with LBP in whom dysfunction of TrA and LM is a key contributing factor to their symptoms such as motor control training.

FRED exercise shares many of the characteristics of motor control exercise. The findings of a range of mechanistic studies show that it results in automatic and tonic activation of LM and TrA, promotes normal lumbopelvic positioning against gravity, works trunk and lower limb extensors more than flexors, challenges balance and motor control, and is a functional progressive exercise. On this basis, as well as the results of a very recent clinical study on people with chronic LBP, we are confident that FRED exercise would complement and enhance

current astronaut rehabilitation practice. Future research should now investigate the effectiveness of the FRED in larger terrestrial populations with low back pain as well as following simulated and actual spaceflight.

Author details

Andrew Winnard[1], Dorothee Debuse[2] and Nick Caplan[1]*

*Address all correspondence to: nick.caplan@northumbria.ac.uk

1 Northumbria University, Newcastle upon Tyne, UK

2 Lunex University, Differdange, Luxembourg

References

[1] Wing P, Tsang I, Susak L, Gagnon R, Potts J. Back pain and spinal changes in microgravity. Orthopedic Clinics of North America. 1991;**22**(2):255-262

[2] Johnston S, Campbell M, Scheuring R, Feiveson A. Risk of hernated nucleus pulposus among US astronauts. Aviation, Space and Environments Medicine. 2010;**81**(6):566-574

[3] Hides JA, Stokes MJ, Jull GA, Cooper DH. Evidence of lumbar multifidus muscle wasting ipsilateral to symptoms in patients with acute/subacute low back pain. Spine. 1994; **19**(2):165-172

[4] Hodges P, Richardson C. Inefficient muscular stabilization of the lumbar spine associated with low back pain: A motor control evaluation of transversus abdominis. Spine. 1996;**21**(22):2640-2650

[5] Hides JA, Belavy DL, Stanton W, Wilson SJ, Rittweger J, Felsenberg D, et al. Magnetic resonance imaging assessment of trunk muscles during prolonged bed rest. Spine. 2007; **32**(15):1687-1692

[6] Hides J, Lambrecht G, Stanton W, Damann V. Changes in multifidus and abdominal muscle size in response to microgravity: Possible implications for low back pain research. European Spine Journal. 2016;**25**(Suppl 1):175-182

[7] Van K, Hides J, Richardson C. The use of real-time ultrasound imaging for biofeedback of lumbar multifidus muscle contraction in healthy subjects. The Journal of Orthopaedic and Sports Physical Therpay. 2006;**36**(12):920-925

[8] Balague F, Mannion AF, Pellise F, Cedraschi C. Non-specific low back pain. Lancet. 2012; **379**:482-491

[9] Panjabi MM. A hypothesis of chronic back pain: Ligament subfailure injuries lead to muscle control dysfunction. European Spine Journal. 2006;**15**:668-676

[10] Hides J, Gilmore C, Stanton W, Bohlscheid E. Multifidus size and symmetry among chronic LBP and healthy asymptomatic subjects. Manual Therapy. 2008;**13**:43-49

[11] Danneels LA, Vanderstraeten GG, Cambier DC, Witrouw EE, De Cuyper HJ. CT imaging of trunk muscles in chronic low back pain patients and health control subjects. European Spine Journal. 2000;**9**:266-272

[12] Hodges P, Holm AK, Hannson T, Sten H. Rapid atophy of the lumbar multifidus follows experimental disc or nerve root injury. Spine. 2006;**25**:2926-2988

[13] Ferreira PH, Ferreira ML, Hodges PW. Changes in recuitment of the abdominal muscles in people with low back pain. Spine. 2004;**29**(22):2560-2566

[14] Hides J, Stanton W, Mendis M, Sexton M. The relationship of transversus abdominis and lumbar multifidus clinical muscle tests in patients with chronic low back pain. Manual Therapy. 2011;**16**(6):573-577

[15] Hodges P, Moseley G. Pain and motor control of the lumbopelvic region: Effect and possible mechanisms. Journal of Electromyography and Kinesiology. 2003;**13**:361-370

[16] Macdonald D, Moseley L, Hodges P. Why do some patients keep hurting their back? Evidence of ongoing back muscle dysfunction during remission from recurrent back pain. Pain. 2009;**142**(3):183-188

[17] Saunders S, Coppieters M, Hodges P, editors. Reduced tonic activity of the deep trunk muscle during locomotion in people with low back pain. In: World Congress of Low Back Pain and Pelvic Pain; 2004

[18] Wallwork T, Stanton W, Freke M, Hides J. The effect of chronic low back pain on size and contraction of the lumbar multifidus muscle. Manual Therapy. 2009;**14**(5):496-500

[19] Belavy DL, Adams M, Brisby H, Cagnie B, Danneels L, Fairbank J, et al. Disc herniations in astronauts: What causes them, and what does it tell us about herniation on earth? European Spine Journal. 2015;**24**:251

[20] Evetts S. Anecdotal Accounts of Selective Atrophy of Trunk Extensors over Flexors in an Astronaut Population. Personal Communication 2015

[21] Bergmark A. Stability of the lumbar spine – A study in mechanical engineering. Copenhagen: Department of Solid Mechanics, Lund Institute of Technology; 1989

[22] Claus AP, Hides JA, Moseley LG, Hodges PW. Different ways to balance the spine. Spine. 2009;**34**:208-214

[23] Macintosh J, Valencia F, Bogduk N, Munro R. The morphology of the human lumbar multifdus. Clinical Biomechanics. 1986;**1**(4):196-204

[24] Moseley L, Hodges P, Gandevia S. Deep and superficial fibers of the lumbar multifidus muscle are differentially active during voluntary arm movements. Spine. 2002;**27**(2): 29-136

[25] Panjabi MM. The stabilizing system of the spine. Part 1. Function, dysfunction, adaptation, and enhancment. Journal of Spinal Disorders & Techniques. 1992;**5**(4):383-289

[26] Kiefer A, Shirazi-Adl A, Parnianpur M. Synergy of the human spine in neutral postures. European Spine Journal. 1998;**7**(6):471-479

[27] Hodges P. Lumbopelvic stability: A functional model of the biomechanics and motor control. In: Richardson C, Hodges P, Hides J, editors. Therapuetic Exercise for Lumbopelvic Stabilization. Edinburgh: Churchull Livingston; 2004. pp. 13-28

[28] Hodges P, Cholewicki J, Van Dieen J. Spinal Control: The Rehabilitation of Back Pain State of the Art and Science. Edinburgh: Churchill Livingstone Elsevier; 2013

[29] Panjabi MM. Clinical spinal instabiilty and low back pain. Journal of Electromyography and Kinesiology. 2003;**13**:371-379

[30] Hodges PW, Moseley GL, Gabrielsson A, Gandevia SC. Experimental muscle pain changes feedforward postural responses of the trunk muscles. Experimental Brain Research. 2003;**151**(2):262-271

[31] Hodges PW, Richardson CA. Altered trunk muscle recruitment in people with low back pain with upper limb movement at different speeds. Archives of Physical Medicine and Rehabilitation. 1999;**80**(9):1005-1012

[32] Hodges P, Cresswell A, Thorstensson A. Reparatory trunk motion precedes upper limb movement. Experimental Brain Research. 1999;**124**:69-79

[33] Ferreira PH, Ferreira ML, Maher CG, Refshauge K, Herbert RD, Hodges PW. Changes in recruitment0020of transversus abdominis correlate with disability in people with chronic low back pain. British Journal of Sports Medicine. 2010;**44**(16):116-172

[34] Hides JA, Stanton WR, McMahon S, Sims K, Richardson CA. Effect of stabilization training on multifidus muscle cross-sectional area among young elite cricketers with low back pain. The Journal of Orthopaedic and Sports Physical Therapy. 2008;**38**:101-108

[35] Van Dieen J, Kingsma I. Spine function and low back pain: interactions of active and passive structures. In: Hodges P, Cholewicki J, Van Dieen J, editors. Spinal Control: The Rehabilitation of Back Pain. Edinburgh: Churchill Livingstong; 2014. pp. 41-59

[36] Buckey JC. Space Physiology. New York: Oxford University Press; 2006

[37] Pavy-Le Traon A, Heer M, Narici MV, Rittweger J, Vernikos J. From space to Earth: Advances in human physiology from 20 years of bed rest studies (1986-2006). European Journal of Applied Physiology. 2007;**191**:143-194

[38] Gernand JM, editor. Risk Assessment and Control Through Countermeasure System Implementation for Long-Term Crew Exposure to Microgravity; 2004. Anaheim: IMECE

[39] di Prampero PE, Narici MV. Muscles in microgravity: From fibres to human motion. Journal of Biomechanics. 2003;**36**:403-412

[40] Hides JA, Lambrecht G, Richardson CA, Stanton WR, Armbrecht G, Pruett C, et al. The effects of rehabilitation on the muscles of the trunk following prolonged bed rest. European Spine Journal. 2011;**20**:808-818

[41] Belavy DL, Armbrecht G, Richardson CA, Felsenberg D, Hides JA. Muscle atrophy and changes in spinal morphology, is the lumbar spine vulnerable after prolonged bed-rest? Spine. 2011;**36**(2):132-145

[42] Sayson J, Hargens A. Pathophysiology of low back pain during exposure to microgravity. Aviation, Space and Environmental Medicine. 2008;**79**:365-373

[43] Snijders CJ, Richardson CA, Hides JA, Pool-Doudzwaard AL, Belavy DL. Low back pain during flight – 1 year post-flight science report. European Space Agency Report. 2011. ESA Proposal number 2003-DSM-MUS-100

[44] Pool-Goudzwaard AL, Belavý DL, Hides JA, Richardson CA, Snijders CJ. Low back pain in microgravity and bed rest studies. Aerospace Medicine and Human Performance. 2015;**86**(6):541-547

[45] Hodges P, Cholewicki J. Functional control of the spine. In: Vleeming A, Mooney V, Stoeckart R, editors. Movement, Stability and Lumbopelvic Pain: Integration of Research and Therapy. 2nd ed. Edinburgh: Churchill Livingstone; 2007. pp. 489-512

[46] Panjabi MM. The stabilizing system of the spine. Part 2. Neutral zone and instability hypothesis. Journal of Spinal Disorders. 1992;**5**(4):390-397

[47] Evetts S, Caplan N, Debuse D, Lambrecht G, Damann V, Petersen N, et al. Post space mission lumbo-pelvic neuromuscular reconditioning: A European perspective. Aviation, Space and Environmental Medicine. 2014;**85**(7):764-765

[48] O'Sullivan PB. Lumbar segmental "instability": Clinical presentation and specific stabilizing exercise management. Manual Therapy. 2000;**5**(1):2-12

[49] Hides J, Stanton W, McMahon S, Sims K, Richardson C. Effect of stabilisation training on multifidus muscle cross-sectional area among young elite cricketers with low back pain. Journal of Orthopaedic & Sports Physical Therapy. 2008;**38**(3):101-108

[50] Winnard A, Nasser M, Debuse D, Stokes M, Evetts S, Wilkinson M, Caplan N, et al. Systematic review of countermeasures to minimise physiological changes and risk of injury to the lumbopelvic area following long-term microgravity. Musculoskeletal Science and Practice. 2017;**27**:S5-S14

[51] Edgerton VR, McCall GE, Hodgeson JA, Gotto J, Goulet C, Fleischmann K, et al. Sensorimotor adaptations to microgravity in humans. The Journal of Experimental Biology. 2001;**204**:3217-3224

[52] LeBlanc A, Rowe R, Schneider V, Evans H, Hedrick T. Regional muscle loss after short duration spaceflight. Aviation, Space, and Environmental Medicine. 1995;**66**(12):1151

[53] Debuse D, Birch O, Gibson ASC, Caplan N. Low impact weight-bearing exercise in an upright posture increases the activation of two key local muscles of the lumbo-pelvic region. Physiotherapy Theory and Practice. 2013;**29**(1):51-60

[54] Lambrecht G, Petersen N, Weerts G, Pruett C, Evetts S, Stokes M, et al. The role of physiotherapy in the European Space Agency strategy for preparation and reconditioning of astronauts before and after long duration space flight. Musculoskeletal Science and Practice. 2017;**27**:S15-S22

[55] Petersen N, Lambrecht G, Scott J, Hirsch N, Stokes M, Mester J. Postflight reconditioning for European astronauts – A case report of recovery after six months in space. Musculoskeletal Science and Practice. 2017;**27**:S23-S31

[56] Richardson C, Hides J. Open chain segmentral control and progression into function. In: Richardson C, Hodges P, Hides J, editors. Therapeutic Exercise for Lumbopelvic Stabilization. 2nd ed. Edinburgh: Churchill Livingstone; 2004. pp. 233-246

[57] Richardson C, Hides J. Closed chain segmental control. In: Richardson C, Hodges P, Hides J, editors. Therapeutic Exercise for Lumbopelvic Stabilization. 2nd ed. Edinburgh: Churchill Livingstone; 2004. pp. 221-232

[58] Caplan N, Gibbon KC, Hibbs A, Debuse D. Phasic-to-tonic shift in tunk muscle activity during low-impact weight bearing exercise. Acta Astronautica. 2014;**104**:388-395

[59] Weber T, Debuse D, Salomoni S, Elqueta Cancino E, De Martino E, Caplan N, et al. Trunk muscle activation during movement with a new exercise device for lumbo-pelvic reconditioning. Physiological Reports. 2017;**5**(6):e13188

[60] Richardson CA, Jull GA. Muscle control – Pain control. What exercises would you prescribe. Manual Therapy. 1995;**1**:2-10

[61] Gibbon KC, Debuse D, Caplan N. Low impact weight-bearing exercise in an upright posture achieves greater lumbopelvic stability than overground walking. Journal of Bodywork & Movement Therapies. 2013;**17**:462-468

[62] Lindenroth L, Caplan N, Debuse D, Salomoni S, Evetts S, Weber TA. Novel approach to activate deep spinal muscles in space – Results of a biomechanical model. Acta Astronautica. 2015;**116**:202-210

[63] Winnard A, Debuse D, Wilkinson M, Samson L, Weber T, Movement NC. Amplitude on the functional re-adaptive exercise device: Deep spinal muscle activity and movement control. European Journal of Applied Physiology. 2017;**117**:1597-1606

[64] Winnard A, Debuse D, Wilkinson M, Tahmosybayat R, Caplan N. The immediate effects of exercise using the functional re-adaptive exercise device on lumbopelvic kinematics in people with and without low back pain. Musculoskeletal Science and Practice. 2017;**27**:S47-S53

[65] O'Sullivan PB, Dankaerts W, Burnett AF, Farrell GT, Jefford E, Naylor CS, et al. Effect of different upright sitting postures on spinal pelvic curvature and trunk muscle activation in a pain-free population. Spine. 2006;**31**(19):707-712