Introduction

Low back pain (LBP) is a common pathology often labelled as non-specific and categorised according to the duration for which it has persisted i.e. acute (<6 weeks), sub-acute (6-12 weeks) or chronic(>12 weeks) [1,2]. Many believe the 'natural history' of LBP results in most of low back injuries and acute LBP recovering before chronicity develops, and that the majority of costs associated with the condition are due those who do develop chronic LBP [1]. Thus chronic LBP is often considered an entirely different entity from low back injury and acute LBP. However, while a variety of further co-morbidities are associated with chronic LBP, such as psycho-social factors, it should be noted that logically all *"...chronic back pain always starts as acute back pain"* (pp 967) [3]. Indeed, in contrast to the common notion of LBPs natural history involving recovery of most acute LBP, evidence suggests that a considerable proportion (69% to 75%) of low back injury and subsequent acute LBP develops in chronic LBP [4,5], often with increasing frequency and severity [6]. This questions whether acute LBP in many instances was ever really recovered.

LBP is prevalent across modern populations [1,7-9] though prevalence does vary at a given point in time, as well as duration (12-33% for point prevalence, 22-65% for 1 year prevalence, 11-84% lifetime prevalence [10]). More recently it has been suggested that around one third of the UK population are affected each year [1] and Waddell and Burton [8] suggest that 60 – 80 % of adults will suffer from LBP at some point in their life. Numerous studies further reveal high rates of prevalence of LBP, including chronic LBP and those with LBP of a recurrent nature [11]. Complicit with this prevalence are considerable economic costs ranging into the billions [12-14] contributed to by direct costs involved with treatments and care, but more so by indirect costs including working losses due to employee absence, production loss etc [15-17]. That LBP represents a significant burden to individuals as well as presenting significant economic costs provides strong evidence for need of further research elucidating its potential causes, as well as treatments that appropriately address such causes to confer improvement in its multifactorial symptoms.

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LBP is a multifactorial condition associated with a wide range of physical symptoms [18,19]. A wide range of 'potential' mechanisms for pain in the lumbar spine exist [1,20,21]. Abnormality or injury in several structures in the lumbar spine, including the facet joints [22,23], intervertebral discs [24,25], and ligamentous tissues [26] are prevalent in LBP and can contribute to pain. In fact any of the anatomical structures of the spine may contribute to pain [22,26-30] though it is not always possible to directly attribute pain to a specific peripheral structure through diagnostic means in individual cases; hence 'non-specific' is offered as a label in up to 85% of cases [31]. In light of this Lee and Vleeming [32] have introduced an integrated multifactorial model of function in relation to the pelvic girdle which has been adapted here to focus on the lumbar spine. Lumbar spine function in the context of LBP therefore comprises several factors and the array of symptoms in LBP could be considered instead as interrelated deficiencies in function under the areas of; form closure (structure), force closure (force produced by myofascial action), motor control (neural patterning and control), and emotions (psychological and psycho-social factors). This model posits that LBP and the function of the lumbar spine are linked, that the various commonly associated symptoms are potentially interrelated dysfunctions that together result in, or from, the condition of LBP, and that improvement in function should be considered in a variety of areas. Further, it should be considered that dysfunction in one area may therefore have the potential to instigate dysfunction in other areas of function and thus might be considered a primary factor in LBP. One factor that that has been researched extensively for its role in LBP is deconditioning of the lumbar extensor musculature.

Lumbar Extensor Disuse/Deconditioning in LBP

It is apparent that despite difficulty in accurately identifying true prevalence due to differences in methods, most studies highlight LBP as a wide ranging issue across a wide range of modern populations. The high prevalence of LBP suggests that a common factor from the multifactorial model may be involved in LBP, and thus potentially its associated multifactorial symptoms. Many authors have historically suggested weakness or deconditioning of the muscles that extend the lumbar spine as this predominantly influential factor [33-36]. In fact this is found to be a commonly associated factor in LBP in both cross sectional and prospective study. The concept has in fact recently been reviewed by

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the author's research group and it has been concluded that evidence from a triumvirate of methodological approaches supports it as a possible causative mechanism in LBP [37]. Specifically it appeared that deconditioning of the lumbar extensor musculature but not the hip extensor musculature was associated with LBP in cross sectional studies and also prospectively associated with low back injury and development of acute LBP. This review also suggested that there is further evidence supporting causality by the criteria put forth by Austin Bradford Hill [38]; criteria such as biological plausibility and experimental reversibility [39,40]. These factors combine to offer why lumbar extensor deconditioning offers a quite convincing explanation of why LBP is such a wide ranging condition.

However, one issue that many authors have with this explanation of LBP is very clearly surmised by Crossman et al. [41]. They note that, of the studies suggesting the presence of lumbar extensor deconditioning in LBP, *"in none of these studies were any mechanisms offered up to explain how "normal" paraspinal muscle could "dysfunction" to predispose to LBP."* Yet it may be the case that the lumbar extensors as an isolated muscle group exist in a potential state of specific chronic 'disuse,' and thus become 'deconditioned' in the first instance independent of physical activity levels due to their anatomy [36,42]; in essence relatively weak lumbar extensors comparable to strong hip extensors. This seems further apparent as a recent review has shown that most forms of activity and exercise appear to provide little to no conditioning effect [43]. Although 'disuse' is often considered as a general reduction in physical activity [44-46], it seems here that 'disuse' could instead be specifically considered as applicable to the lumbar extensors due to the difficulty in conditioning them, thus leading to their 'deconditioning'. In a sense, specific 'disuse' may lead to specific 'deconditioning' of the lumbar extensors, which may further lead to injury and LBP. But this is not simply a reduction in general activity levels; it is due to the inability to effectively maintain their condition as a consequence of their anatomy as the hip extensors appear to 'take-over' much of the load bearing [47-49]. Yet, in consideration of this, a pertinent question may be 'why' the anatomical arrangement of our lumbar spine and pelvis should be as such to result in such outcomes? Fortunately questions such as these can be informed by considering the evolutionary heritage of our species.

An Evolutionary Hypothesis to Explain LBP

"Nothing in biology makes sense except in the light of evolution"

Dobzhansky [50]

Dobzhansky's [50] now famous phrase lays at the forefront of all scientists' minds when examining biological phenomena. If we are to consider the deconditioning hypothesis as appropriate to explain the high prevalence of LBP then it should fit with evidence regarding the evolution of our species. Every organism is a product of its particular evolutionary history. In his original paper on the 'Disuse Syndrome', Bortz II [44] considers LBP as a 'disease of civilisation' and posed that disuse (defined as general physical inactivity) over the course of human history has been the primary cause of a many of these diseases of civilisation including LBP. However, whilst positing evolutionary reasoning for the Disuse Syndrome, Bortz II [44] fails to consider what morphological changes might have occurred over the course of the evolutionary history of *Homo* which may have important implications in the prevalence of LBP over and above simple definitions of inactivity. Again a fault in the consideration of Disuse Syndrome in chronic LBP from this evolutionary perspective is the definition of disuse as being lack of general physical activity. However, this definition fails to consider specific activity of the part of the body in question, in this case the lumbar spine and the lumbar extensor musculature. Indeed, and perhaps more importantly, Gracovetsky [51] has highlighted, the spine specifically has played an integral role in the evolution of locomotion, preceding the involvement of the peripheral anatomy, and this evolution has permitted the eventual habitual bipedal gait seen in *Homo sapiens*. In consideration of the earlier multifactorial model it should perhaps not be surprising that the important role of the lumbar spine in gait is linked to its anatomical evolution and that this may provide some explanation for the link between for example, abhorrent gait and LBP (expanded upon below), perhaps amongst other associations that might be further explained by consideration of this line of reasoning.

If instead, however, the ideas of Bortz II [44] are correct in concerning lifestyles due to modern civilisation, as predominant factors in chronic LBP then we would expect to see far lower rates of LBP in those populations that are free from this influence and still follow physically active *traditional*

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lifestyles. Indeed, rates of other so called '*diseases of civilisation'* such as obesity, cancer, heart disease, type II diabetes etc., (which have risen dramatically in prevalence over the previous half century in western populations) are almost non-existent in human populations adhering to their traditional diet and lifestyles [52-54].

LBP in Traditional Indigenous Populations

The World Health Organisation (WHO) reports rates of back disorders as being highly ranked *worldwide* as a cause of morbidity [55]. There is some evidence that higher rates of LBP are positively associated with income and urbanisation [56] in addition to being slightly more common in more 'developed' countries [57]. Anderson [58] has argued from an evolutionary perspective using Polanyi's [59] concept of dual level control (figure 4) to discuss how an evolutionary 'mismatch' hypothesis might explain any differences between 'modern' and 'traditional' cultures in terms of LBP rates.

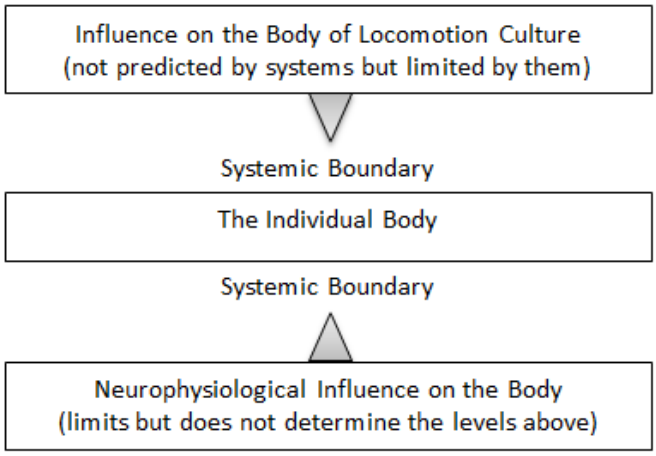

Figure 4. Dual-level control in consideration of 'locomotion culture'from Anderson [58]

In essence the concept of dual level control in this context posits that an organism is limited by its genotype and phenotype with regards to the limits set upon the bodies locomotion possibilities, but that the environment of 'locomotion culture' influences the locomotion patterns utilised within those limits [58]. Anderson [58] argues that the cultural environment influence on locomotion ultimately impacts upon LBP risk as there is evidence that traditional cultures often adopt what would be considered a posture involving a 'stabilised' lumbar spine during locomotion, dancing and working [60-64], and thus

likely experience less in the way of tissue stress and are better able to deal with unexpected perturbations. Thus an argument from this perspective offers that there is an evolutionary 'mismatch' between the organism and its environment; in this case the modern human anatomy and its interaction with the environment of 'locomotion culture' it finds itself in. The traditional locomotion cultures presented by traditional populations are suggested to be the ideal environment for our anatomy and as corollary the modern locomotion culture presents a mismatch. However, evidence available examining a wide range of traditional indigenous population's show that rates of LBP are as high, and in some cases higher, than in 'modern' populations (table 1). This would tend to suggest that any locomotion culture differences may be of little importance in determining whether a population is at greater risk of LBP. Further, as noted, most exercises and activities offer little in the way of stimulus to condition the lumbar extensors [47-49] and so it would be unlikely that such movements associated with locomotion culture would impact upon LBP, at least through this mechanism. It could be argued that socioeconomic differences might impact upon LBP rates independently of physical activity in traditional and indigenous populations. However, as noted it might be expected based on the positive association between income and urbanisation [56] and the slightly greater rates in more 'developed' countries [57] that this should in fact result in lower rates of LBP in traditional populations.

Table 1. Studies of LBP prevalence in traditional and indigenous populations.

Population	***Prevalence***	***Supporting studies***
Australian Aborigines	4.3% point prevalence 39% point prevalence 64% 7 week prevalence ~19% lifetime prevalence (sig higher than non-Indigenous ~17%)	[65-67]
Sami Reindeer Herders	~35% Men, ~60% Women 1 week prevalence 60% 12 month prevalence (no assc. with work)	[68,69]
Maasai (compared to matched Norwegians)	Subjective Health Complaints sig greater in Maasai (p = 0.002)	[70]
New Guinea Highlanders	75% point prevalence	[71]
Rural Chinese	64% 12 month prevalence (Ass with physical exposures i.e. farming)	[72]
Rural Thai	~8% lifetime prevalence ~1% 30 day prevalence	[73,74]
Thai Rice Farmers	49% point prevalence 56% 12 month prevalence 77% lifetime prevalence Trunk extension endurance sig less compared to asymptomatic	[75,76]

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Rural Indonesians	Rural – 15.25% Urban – 22.5%	[77]
Rural Malaysians	11.6%	[78]
Rural Philippines	14.4%	[79]

A handful of studies do suggest that some traditional cultures report extremely low rates of LBP including some studies of Australian Aborigines and rural Thai. However, in combination with the totality of evidence suggesting that LBP is common to a range of traditional populations similar to these it is reasonable to consider these findings as outliers. Explanation for these outliers concerns mainly the reduced inclination of indigenous peoples to report pain to 'outsiders' due to cultural barriers [80]. In particular, in one study up to 50% of an Australian Aborigine population suffered with LBP but preferred not to make this public [81]. Recent research also suggests that, though LBP is present in Aboriginal populations, where there is resultant disability it may be partly due to the influence of modern healthcare professionals reinforcing negative beliefs contrary to their cultural ones [82]. In addition most rural Thai, Malaysians and Philippines opt not to utilise westernised health care provided to them and instead self-medicate using traditional healing methods [78,83,84]. Some traditional healing methods such as bonesetting used by traditional Finns are reported subjectively as improving LBP more than drugs or physical therapy [85]. However, in native American Indians and Alaskan natives though subjective reports associated with their traditional healing methods were positive, those using such methods had significantly worse health outcomes and were more likely to report LBP [86]. Whether traditional healing methods are in anyway efficacious has yet to be determined, however, the point here is that LBP is common to almost all populations examined and that outliers are easily explained by their reluctance to report pain and instead to rely on traditional healing methods. Though it should be noted that data does not exist for many 'underdeveloped' or traditional populations around the world and as such it may be that there are still populations with relatively low prevalence of LBP.

We have focused on extant populations thus far, both 'modern' and 'traditional.' However, there is evidence of lumbar spine damage and degeneration potentially pursuant to LBP in many examples of extinct species of *Homo* and other early human populations. These include *Homo erectus* [87], *Homo*

heidelbergensis [88], ancient Egyptians and Nubians [89], ancient Chinese populations [90] and Ötzi the Iceman [91]. This included disc herniation, spondylolisthesis, scoliosis, osteophytosis, rheumatoid arthritis, osteoarthritis, lumbar spur formation, and ankylosing spondylitis all of which have the potential to cause LBP [1,20-30]. So in consideration of Anderson's [58] invocation of dual level control in determining LBP in humans it would seem that the cultural environment, despite varying considerably between populations, has little impact upon LBP prevalence. Thus instead we might follow this line of reasoning to examine the flip side of the model; the biological make-up of the organism itself. As noted, the anatomical arrangement of modern *Homo's* lumbar spine and pelvis has followed an evolutionary history closely entwined with the emergence of different locomotor patterns. Therefore, the important role that the lumbar spine, and particularly its musculature, plays in human gait, and how gait is affected when this musculature is in dysfunction, how our present primary locomotor pattern may have emerged, and what anatomical changes accompanied this will be considered.

Lumbar Spine Kinematics in Asymptomatic and Symptomatic Human Gait

Gait describes the pattern of movement during locomotion. In humans the normal walking gait pattern consists of the stance phase and swing phase, which can be further broken down to determine initial contact and loading response (together encompassing weight acceptance), midstance and terminal stance (together encompassing single limb support) during the stance phases, and pre swing, initial swing, mid swing and terminal swing (together encompassing advancing limb swing) during the swing phase.

Thurston and Harris [92] observed that the movements of the lumbar spine related to identifiable events as described within the gait cycle and reported normative data for its kinematics including displacement or range of motion (ROM) and wave pattern. Other early studies began by examining whole trunk kinematics, usually identifying the trunk segment using a sternum and sacral marker, measured both relative to the pelvis and room coordinates, and identifying a small range of flexion/extension displacement (~2-12^{o}) [93-95]. Saunders et al. [96] originally presented a deterministic model of

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locomotion as the translation of the body's centre of gravity through space with the least expenditure in energy. They identified three of six major determinants of gait, each corresponding to movement in the three axes, and related to motion about the pelvis. Control of these determinants are fundamental for reducing displacement of the centre of mass (COM) during walking and maintaining its sinusoidal pattern [97]. This energy conservation mechanism during gait explains experimental data demonstrating small ROM of the trunk during gait. Advancements in the accuracy of measurement techniques [98] and more specific segmentation of the spine [99,100] as opposed to the trunk as a whole have resulted in even smaller ranges of excursion being identified, similar to those found in Thurston & Harris' [92] work, providing further evidence to suggest the important role that control of the lumbar spine plays in gait. Indeed Gracovetsky's [51] spinal engine model highlights the important fact that from evolutionary perspective locomotion was initially achieved through motion of the vertebrate spine, evolving from predominantly a lateral bend, to flexion-extension driven propulsion until finally a combination of spinal movements result in the pelvis being driven and controlled by the spine as seen in human bipedal gait.

The kinematic waveform pattern that the lumbar spine follows during gait has been consistently reproduced in studies with healthy participants [92,99-101]. It is acknowledged that patterns of lumbar movement in the saggital plane are variable between subjects but consistent in repeated trials [99,100] and that movements in the frontal plane are most reproducible [99,100,102]. Modelled as a rigid body the spine follows a smooth oscillating pattern with small inter-segmental movements. Syczewska and colleagues [103] found that the small segments ranging from T_{10} to S_1, including the entire lumbar spine, always bend laterally toward the support leg.

Such fine control of the lumbar spine during gait is undoubtedly aided by the musculature. Most studies examining muscular contributions to gait have focused upon the lower extremities [104,105]; however others have identified the active role that the spinal musculature plays [93,101,106]. Thorstensson and colleagues [93] showed that the EMG pattern of the multifidus and longissimus during gait involved two bursts of activity per cycle each corresponding to foot strike. They concluded that this activity in

relation to the pattern of trunk movement suggested that the lumbar erector spinae muscles main function during gait is to control and restrict excessive trunk movement about the frontal plane. Callaghan and colleagues [101] demonstrated similar bimodal activity corresponding to greater peak in the musculature ipsilateral to the contacting foot.

The amplitude in EMG activity of lower extremity musculature can be highly variable between subjects which presents difficulties in identifying 'normal' EMG signals, yet within subject signals can be identified and are highly repeatable [104,107]. Nonetheless the activity of the lumbar musculature does appear to follow a particular pattern seemingly to stabilise superior segments against inertial and gravitational forces during both single foot contacts [93,101,106]. Interpreted in concurrence with the pattern of lumbar frontal movement ipsilateral to the support leg [103], the important role in motor control of the ispilateral lumbar musculature during gait is beyond doubt. However, as noted, deconditioning of this musculature is commonly associated with LBP. Indeed it has been suggested that deficiencies in motor control during gait may produce excessive stresses to the lumbar spine, which may contribute to development of LBP [110]. However, a recent review has suggested there is evidence against walking itself being causally associated with LBP [107]; thus the gait observed in LBP participants might be justifiably considered a symptom of the condition and perhaps of lumbar extensor deconditioning.

Healthy participants demonstrate relatively low stride-to-stride variability in lumbar kinematic patterns during both level and incline gait [109]. However, greater stride-to-stride variability at the lumbar spine in all movement planes [110], greater frontal plane coordination variability of the pelvis and trunk [111,112] and more rigid transverse plane coordination variability of the pelvis and trunk [112-114] is reported in LBP participants compared with healthy controls. It also appears that pain *per se* may not be responsible for these gait differences. Lumbar spine kinematics during gait appear to be complex and developed over time, as patterns are evident before pain is experienced [115] and both induced pain and fear of pain produce little change in muscle activity in LBP patients [116]. Indeed recently studies have shown that even those with a previous history of LBP who are currently asymptomatic demonstrate

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abnormal gait patterns [117,118]. Thus the pain *per se* may not be the factor responsible. There is contrasting evidence reporting no residual effect upon gait from an episode of low back pain in nurses returning to work with very low pain levels [102]. Yet, a lack of directly comparable control group makes this conclusion questionable [102].

Evidence instead suggests the lumbar extensor musculature play a role in gait variability in LBP [111,116,117,120-125]. Variability in gait patterns is combined with poorer erector spinae activity adaptability to unexpected perturbations [116], or walking velocity changes [121]. In fact, the findings of numerous studies are suggestive of muscular dysfunction of the lumbar extensors during gait in those with LBP compared with asymptomatic controls [111,116,119-121]. Hanada et al. [125] also report that where asymptomatic controls significantly activated their rectus abdominus and internal obliques more, symptomatic participants had significantly greater activation of the lumbar extensors. More recent work shows evidence of greater lumbar extensor activity in LBP participants compared with controls [122], at a range walking velocities [123], and that neither disability nor fear of movement is associated with this greater activity [122]. However, different coping strategies may be associated with either greater activity (catastrophizing) or greater relaxation during double support (distraction) suggesting some influence of cognitive control [124].

Human gait is normally quite robust in the face of muscular weakness of the lower limbs (except the plantar flexors, hip abductors and flexors [126]). The lumbar spine's primary role in driving human bipedal gait however seems to significantly affect this robustness [51]. It is possible that the greater activation of the lumbar extensors, and altered lumbar spine kinematics during gait in chronic LBP participants, is a manifestation of the lumbar extensor deconditioning commonly associated with chronic LBP. Indeed it has been recently shown that weak lumbar extensor musculature is associated with poorer control of the lumbar spine during gait [127] and thus greater activation in the face of fatigue due to deconditioning could be a compensatory attempt to maintain control of the lumbar spine during gait. Hart et al. [128] demonstrate that inducing fatigue in the lumbar extensors impacts lumbar kinematics during running gait of healthy participants and chronic LBP participants. Arjunan et al. [129]

also show significantly greater lumbar extensor activity during running gait in chronic LBP participants. Indeed, as noted prospective evidence supports lumbar extensor deconditioning as being a risk factor for low back injury and pain. Thus it may be responsible for the development of the gait variability associated with chronic LBP also. Further, a recent study has shown that control of the lumbar spine during gait is improved through specific strengthening of the lumbar extensor musculature [130].

Thus far the importance of the lumbar spine and it's musculature in particular in controlling human bipedal gait has been explored. The importance of spinal movement in the emergence of different locomotor repertoires has also been alluded to. However, an illustration of the emergence of bipedalism and how anatomical evolution of the lumbar spine has been intertwined with it has not yet been presented, nor any evidence yet to support the question of *'why'* the lumbar musculature could '*decondition'* from specific '*disuse*' to result in LBP and its accompanying symptoms such as gait abnormality. The final section here shall attempt to provide this illustration though it should be noted that the specific route for the emergence of bipedalism is not entirely agreed upon within the paleo-anthropological literature and so here discussion will remain general in nature as it pertains to deconditioning's relationship with LBP.

The Emergence of Bipedalism

A general view of the gradual acquisition of bipedalism (Figure 5) in the infra-order Anthropoidea suggests adaptations in predominant habitual locomotion styles from 1) arboreal quadrupedalism (both generalised prongrade and more forelimb dominated orthogrady including some brachiation), to 2) semi-terrestrial quadruped (with greater utilisation of forelimb suspension behaviour including brachiation and bridging), to 3) biped over the past ~20 million years [131]. Though it should be noted that the specific placements of different species are often the cause of much debate and this presentation is not universally agreed upon [131-133]. Evidence for very early specialisation for arboreal quadrupedalism has been inferred from observation of the locomotor repertoires of both non-primate arboreal quadrupeds such as Wooly Opossums [134] and in early fossil evidence from primate ancestors such as *Plesiadapis* and *Necrolemur* [135]. Arboreal quadrupedalism in extant species with similar

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anatomical arrangement generally includes pronograde postures and considerable utilisation of leaping movements involving utilisation of the 'spinal-engine' in flexion and subsequent extension for propulsion, in addition to some climbing and some evidence of brachiation [136]. Varied extant Old World Monkeys (OWMs), generally considered anatomically similar to early Miocene forms, exhibit these behaviours [137-139]. Indeed the anatomy of early Miocene forms such as *Proconsul* and *Morotopithecus*[1] suggest that their locomotor patterns involved similar arboreal quadruped positional behaviours [140-144]. The transition to a more semi-terrestrial quadrupedal locomotor pattern included more frequent orthograde postures including climbing, more frequent brachiation, bridging and perhaps knuckle walking [132,136]. The extant great ape species all exhibit these positional behaviours though to varying degree's dependent upon individual species specialisation [132,136,145]. Later Miocene forms also exhibit anatomy to suggest that their locomotor patterns may have been similar to this semi-terrestrial quadrupedalism including different dryopithecines and morotopithecines [135,140,143,146]. Increased bipedalism has been shown in extant semi-terrestrial quadruped great apes in response to certain environmental stimuli such as introduction of items requiring carrying [147]. Early species such as *Oreopithecus* and the subfamily Hominini, such as *Ardipithecus Australopithecus,* and *Homo* exhibit adaptations suggesting the gradual change from species exhibiting semi-terrestrial quadrupedalism, including specialism for orthograde climbing, to species exhibiting more habitual bipedalism [148,158-160,168].

[1] Moroto specimens have been reconstructed by several authors in comparison with other species with a range of interpretations. These have included suggestion of a long lumbar spine (6-7 veterbrae), yet also adaptations indicating stiffness of the column, and also adaptations suggesting arboreal quadrupedalism and/or orthograde, fore-limb dominated positional behaviours. Hence here it is cited as an example spanning several categories of locomotor and lumbo-pelvic changes.

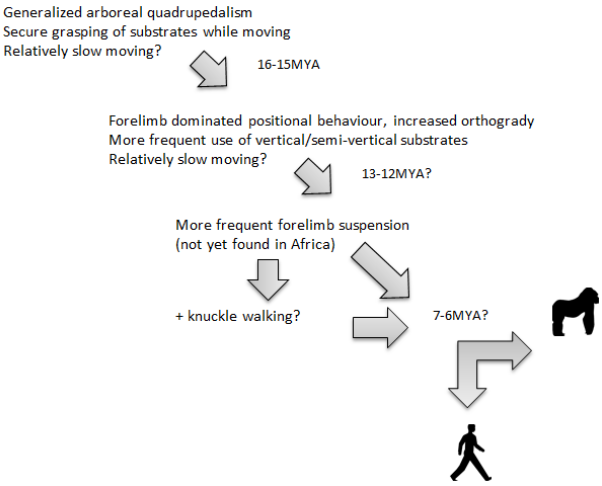

Figure 5. General representation of locomotor evolution in primates from Nakatsukasa [131].

This general pattern of change in locomotor repertoire was necessarily accompanied by considerable restructuring of the lumbo-pelvic anatomy further adding weight to Gracovetsky's [51] assertion of the important role of the spine. Again a general view of these changes occurring in tandem with the general view presented of locomotor patterns includes change from 1) a long mobile lumbar vertebral column, laterally facing pelvis and large lumbar extensors to 2) a short lumbar vertebral column, posterior location of the transverse process, lengthening of the ilia, reduction of extensor musculature and increase in passive rigidity through entrapment and invagination and 3) to re-lengthening of the vertebral column, reduction in length and broadening of the ilia and sacrum. Again it should be noted that this scenario is not entirely agreed upon in the literature and indeed it is argued that this is noted in the next section.

Small bodied arboreal quadrupeds including OWMs, and early Miocene forms including *Proconsul, Nachalopithecus* and some morotopithecines generally have lumbar vertebral numbers ranging from 6 to 7 [141,142,149,150]. Larger bodied semi-terrestrial species including extant great apes and dryopithecines instead have a shorter lumbar spine of 3 to 4 lumbar vertebrae [144,150-152]. The transition to bipedalism was accompanied by a lumbar spine consisting of 5 to 6 vertebrae, though there is some disagreement as to whether this constituted a lengthening from shorter backed species or a shortening from longer backed arboreal quadrupeds [133,150,152-164]. Nevertheless these anatomical

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arrangements of vertebral number were also accompanied by structural changes to the vertebral bodies themselves. Extant and extinct longer backed small bodied arboreal quadrupeds have craniocaudally elongate lumbar vertebrae with ventrally positioned transverse processes [141,151,165] whereas larger bodied semi-terrestrial species contrastingly have craniocaudally shorter vertebrae with posteriorly positioned transverse processes [150,166,167]. Adaptations permitting bipedality included a loss of the styloid process from the vertebral body, this particular change being first present in *Morotopithecus* indicating early changes that might permit eventual bipedal locomotion, and is present in *Oreopithecus, Ardipithecus, Australopithecus* and *Homo* [166,167]. The structure and orientation of the pelvis is narrow with laterally facing iliac blades in extant OWMs, *Proconsul* and gibbons [141]. A lengthening of the ilia is clearly present in extant great apes and also in *Morotopithecus* and *Dryopithecus* [143,146,150,168]. Forms either capable of, or exhibiting habitual bipedalism, including *Oreopithecus, Ardipithecus, Australopithecus* and *Homo* exhibit a shorter ilia with broadening and anterior angling in addition to a broadening of the sacrum [158-160,168].

These anatomical changes occurred along with invagination of the spinal column in the family Hominoidea. Earlier species of small bodied arboreal quadrupeds had long mobile lumbar vertebral columns. Larger semi-terrestrial quadrupeds had a shorter lumbar vertebral column, increased passive rigidity through entrapment due to the lengthened ilia, and spinal invagination resulting in increased column rigidity. Bipeds instead have a relatively longer more mobile lumbar vertebral column. The changes in musculature of the lumbo-pelvic complex included both the lumbar and hip extensors permitting changing gait patterns. It is suggested a general reduction in lumbar extensor musculature occured with changes in the hip extensors primarily reflecting pelvic rearrangement of their attachments and their changing roles around ~3 millions years ago (e.g. shortened ilia reducing the glutei's moment and along with the hamstrings being now being primarily involved with controlling trunk and head during support phase, and control and deceleration of the limb during the final stages of the swing phase [169,170]). Pronograde arboreal quadrupeds have relatively large lumbar extensor musculature cross sectional area (CSA) likely due to its involvement in the flexion/extension movement of leaping [141,142,171]. Orthograde semi-terrestrial quadrupeds contrastingly have significantly reduced lumbar

extensor CSA with the iliocostalis situated more laterally. This general reduction appears to be due to the increased passive rigidity offered by the short lumbar spine, entrapment and invagination. A stable base such as this was necessitated perhaps by increased body size and involvement in more specialised locomotion such as bridging [132,136,140,143,145,146] and the lateral relocation of the iliocostalis is thought to have been important for brachiation through an increased moment for lateral flexion [166]. Comparative anatomy between arboreal quadruped species such as OWMs and habitual bipeds such as modern humans indicate a considerable reduction in lumbar extensor CSA for habitual bipeds suggesting lower force producing capacity of this musculature in relative terms [166,169,172]. This has combined with changes in the musculature of the hip to result in the present arrangement in modern humans. The glutei and hamstrings are capable of greater hip extension torque production in quadrupeds, both pronograde and orthograde, due to hip extension being heavily involved in propulsion during locomotion [169-171]. The shift to habitual bipedality involved reorganisation and enlargement of the glutei and a retainment of the hamstrings, though their specific roles in locomotion changed. In habitual bipeds the glutei are involved in control of the trunk during the support phase in walking and running and the hamstrings controlling the swing phase of the gait cycle [170,173,174]. It is difficult to draw direct comparison with regards to the relative abilities of the musculature in different species to produce force and subsequently torque across their articulations. However, based on the changes that appear to have occurred it could be inferred that anatomically modern humans in essence may bear the evolutionary compromise of relatively strong hip/trunk extensors retained from earlier quadruped ancestral forms, relatively weak lumbar extensors occurring in part due to the increase in passive rigidity in the lumbar spinal column in earlier species, in combination with a long flexible lumbar spine. Understanding the origins of this anatomical arrangement it is possible to conceptualise an evolutionary hypothesis in an attempt to explain some of the common observations regarding LBP and deconditioning in the literature.

Conceptualising the Hypothesis

Returning to the concept of dual-level control presented earlier it was discussed that two possible general influences on the individual organism might be invoked to perhaps explain the rates of LBP

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seen in humans; the environment of 'locomotion culture' and the biological constraints set by the organisms make-up. We have seen that cultural influences seem to exert relatively little influence upon rate of LBP by the fact that numerous traditional populations have high rates of LBP similar to more 'modern' populations (table 1). This would tend to argue against the hypothesis of an evolutionarily determined 'mismatch' between organism and environment as seems to be the case for other conditions. Contrastingly it would seem that the biological constraints set by human anatomy bear the major influence. As a result of their evolutionary history tying anatomical changes in the lumbo-pelvic complex with changes in predominant locomotor patterns, modern human's bear the compromise of a long flexible lumbar column with relatively little passive stability and relatively weak lumbar extensor musculature. Before proceeding to fully invoke this hypothesis as an explanation for a number of observations regarding LBP, deconditioning and the role and form of muscular conditioning using exercise for the lumbar extensors it is important to highlight potential areas of debate against it.

A common argument against the assertion that LBP is a result of the anatomy corresponding to our ability to habitual perform bipedal locomotion is that if it were indeed the case *'Natural Selection would have taken its toll'* and it would be unlikely that we would have survived as a species. However this argument makes two assumptions; 1) that the presence of LBP would have had to negatively impact upon reproductive success and, 2) this would have had to have been greater than the positive impact of being habitually capable of bipedalism. Some have argued that LBP is a condition that presents itself most commonly past reproductive age and thus it would be likely to persist as a trait [174]. However it should be noted that amongst the high prevalence of LBP seen in adult populations there is considerable evidence of similarly high rates of LBP in children, adolescents and young adults from as young as 10 years of age with rates being either unaffected by physical activity, exercise or sport and in some cases increased [175-181]. From this it could be deduced that LBP must have had little impact upon reproductive success in order for it to still remain as a highly prevalent condition. In fact many evolutionarily derived traits have both positive and negative effects upon reproductive success and are termed as 'costly' [182]. What is important is the net effect upon reproductive success. As long as the positive impact outweighs the negative the net effect will be tipped in favour of persistence in the gene

pool. In the case of bipedality, assuming the hypothesis presented here that LBP is a consequence of our evolutionarily derived anatomy required for bipedalism then the positive effects associated with habitual bipedalism must have outweighed the negative effects associated with that trait.

In addition, some adaptations favouring a more stabilised spinal morphology appear to have occurred with the adoption of habitual bipedalism. For example, comparison of the vertebral body CSA between *Homo erectus* and *Homo sapiens* suggests modern humans have proportionately larger CSAs [88]. This suggests that because the stress the intervertebral discs and vertebral bodies experience is proportional to the force over the loaded area, the greater CSA in modern humans may represent a degree of adaptation to what appears to be the inherently unstable lumbar morphology required for habitual bipedality in *Homo*. Indeed, recent work also shows that modern humans whose lumbar vertebral body shape and size is more similar to that of chimpanzee's (referred to as a more ancestral shape) typically present with greater prevalence of Schmorl's nodes [183]. Additionally there appears to have been an increased posterior translation of the posterior superior iliac spine (PSIS) [166,167]. This relocation increased the moment arm over which the lumbar extensors exert force effectively increasing the torque they are capable of producing. This may represent some adaptation to enhance their relative strength despite a general reduction in the musculatures CSA.

As noted, there is also debate regarding the general presentation of the evolutionary history of our species. For example there is some disagreement regarding the stages of locomotion presented [131-133] with this debate centring on whether the last common ancestor (LCA) of modern humans and the great apes walked with a bent-hip-bent-knee gait when bipedal and whether their lumbar spine was long and flexible (like OWMs) or short and stable (like in the great apes) [133,150,152-164]. It is beyond the scope of this article to examine this area of debate but it should be noted that the final circumstance, a long flexible yet unstable lumbar spine in humans being related to LBP, can be supported irrespective of whether an intermediary involving a short back LCA stage in locomotor evolution and its corresponding anatomical evolution occurred [147,150]. A general reduction in the stability of the

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lumbar spine through reduction in the musculature and a retainment of the bulk of the powerful hip extensor musculature appears to have occurred irrespectively.

So what observations regarding LBP can be explained by this hypothesis? The widespread nature of LBP in a range of culturally and environmentally diverse human populations is more convincingly explained by some common trait regarding their physical make-up. That it appears evident that as a species we have relatively unstable and weak lumbar spines this might put them at greater risk of injury, degeneration, and thus potentially pain. As noted in the beginning of this article, specific deconditioning of the lumbar extensors is consistently associated with LBP both cross-sectionally and prospectively. This may be due to this species wide trait, though evidently the relative strength of the lumbar extensor musculature and stability of the lumbar spine will vary within the population affecting risk relative to other individuals. On the whole however such high rates of prevalence conceivably correspond with this. The relative size and strength of the hip extensor musculature in combination with the relatively weak lumbar extensor musculature perhaps further serves to explain the persistence of specific lumbar extensor weakness. In addition it explains the notorious difficulty in conditioning the lumbar extensors when they are exercised in a compound fashion whether through general physical activity or through trunk extension based exercises compared to isolated lumbar extension exercise [43]. In essence the hip extensors to a degree relinquish the lumbar extensors of their role in load bearing thus permitting their continued relative disuse and subsequently deconditioning, or at least not permitting their condition to improve. This offers an explanation for Crossman and colleagues [41] concern regarding '*how'* the normal lumbar extensors might be or become deconditioned to predispose to LBP in the first place. The 'normal' lumbar extensors are indeed weak and the lumbar spine relatively unstable in humans while the relative strength of the hip extensors confounds attempts to condition or recondition them and may potentially allow them to exist in their own specific state of relative 'disuse.' This apparently stems from the evolutionary history of our species and might be the predominant factor within the multifactorial model of LBP that imparts the presence of other symptoms particularly gait abnormalities.

From this hypothesis then what other predictions might be offered and how might they be tested? It might be expected that, despite differences in positional behaviour, locomotion and skeletal morphology potentially confounding comparisons, evidence of spinal disorders that might confer LBP might broadly be relatively less prevalent in extant non-hominin primates due to the greater relative stability their lumbar spines offer, whether through muscular action or passive rigidity. In fact there is already some evidence that this is the case. Both ape and monkey species are almost entirely free of spondylolysis [174]. In addition studies of chimpanzee's, bonobo's and gorilla's show significantly lower rates of spinal degeneration of all kinds [184-186] with most injuries instead occurring from aggression or fall related trauma [185,187,188]. Though there is some evidence of spinal degeneration in extinct bipedal hominin species and early humans [87-91] systematic study of spinal degeneration in other extinct non-hominin primate species does not seem to have been conducted. The present hypothesis might predict that prevalence of lumbar spine degeneration might also be low in such species. Future research therefore should look to conduct cross-sectional comparative analysis and differential diagnosis across the populations noted (extinct non-hominin primate species, extinct hominin species, extant primate species, and extant humans) systematically, and with a preferably large sample size such as to reduce the confounding impact of differences in positional behaviour and locomotion, where it might be hypothesised that prevalence of potentially pain causing lumbar injury and degeneration will be higher in hominin species compared with non-hominin species.

Conclusions

To conclude this article and summarise the discussion, the evolutionary hypothesis presented here that the adaptations permitting habitual bipedalism have left us with a lumbo-pelvic anatomy comprising weak lumbar extensors, strong hip extensors and a lack of stability, may serve to explain the high prevalence of LBP in humans. It also may serve to explain why deconditioning of the lumbar extensors specifically is a common factor in LBP; why atypical gait patterns are present in LBP and related to this; why conditioning these muscles is notoriously difficult with most exercise movements; and it may also explain why exercises allowing optimal isolation of these muscles for conditioning are quite effective in rehabilitation of LBP. Of course in the beginning of this manuscript it was noted that LBP

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is a multifactorial condition with a number of factors associated with it. Many of these may in fact stem from the initiation of low back injury and LBP that is a result of the evolutionarily determined anatomy of modern humans. However, it is not the intention to argue vehemently for a singular cause of LBP independently. Certainly, though the hypothesis presented here may be compelling there is scope for the relationships of factors associated with LBP to be bi-directional and for other factors within the multifactorial condition to exert causative pressures also. For example, many of the anatomical influences discussed may influence the high rates of LBP in humans in some cases, but specific deconditioning of the lumbar extensors may be the result of injury and LBP and not necessarily causative.

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