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Movement Systems as Dynamical Systems

The Functional Role of Variability and its Implications for Sports Medicine

Keith Davids,[1] *Paul Glazier*,[2] *Duarte Araújo*[3] and *Roger Bartlett*[4]

1 School of Physical Education, University of Otago, Dunedin, New Zealand
2 School of Sport, P.E. and Recreation, University of Wales Institute, Cardiff, Wales
3 Faculty of Human Kinetics, Technical University of Lisbon, Lisbon, Portugal
4 Centre for Sport and Exercise Science, Sheffield Hallam University, Sheffield, England

Abstract

In recent years, concepts and tools from dynamical systems theory have been successfully applied to the study of movement systems, contradicting traditional views of variability as noise or error. From this perspective, it is apparent that variability in movement systems is omnipresent and unavoidable due to the distinct constraints that shape each individual's behaviour. In this position paper, it is argued that trial-to-trial movement variations within individuals and performance differences observed between individuals may be best interpreted as attempts to exploit the variability that is inherent within and between biological systems. That is, variability in movement systems helps individuals adapt to the unique constraints (personal, task and environmental) impinging on them across different timescales. We examine the implications of these ideas for sports medicine, by: (i) focusing on intra-individual variability in postural control to exemplify within-individual real-time adaptations to changing informational constraints in the performance environment; and (ii) interpreting recent evidence on the role of the angiotensin-converting enzyme gene as a genetic (developmental) constraint on individual differences in physical performance.

The implementation of a dynamical systems theoretical interpretation of variability in movement systems signals a need to re-evaluate the ubiquitous influence of the traditional 'medical model' in interpreting motor behaviour and performance constrained by disease or injury to the movement system. Accordingly, there is a need to develop new tools for providing individualised plots of motor behaviour and performance as a function of key constraints. Coordination profiling is proposed as one such alternative approach for interpreting the variability and stability demonstrated by individuals as they attempt to construct functional, goal-directed patterns of motor behaviour during each unique performance. Finally, the relative contribution of genes and training to between-individual performance variation is highlighted, with the conclusion that dynamical systems theory provides an appropriate multidisciplinary theoretical framework to explain their interaction in supporting physical performance.

In the last 20 years, ideas from scientific paradigms such as chaos theory and the sciences of complexity have been integrated with concepts and tools from dynamical systems theory to re-shape our understanding of movement behaviour.[1,2] Dynamical systems theory has been successfully applied to the study of coordination in nervous systems and movement control,[3-5] movement development[6-9] and skill acquisition.[2,10] In particular, the dynamical systems framework has influenced the way that movement scientists view inter- and intra-individual variability in motor performance, as a function of learning and development across a lifespan.

Traditional approaches to the study of motor behaviour have operationalised variability with measures of variance in motor output (e.g. standard deviation around the distribution mean of a dependent variable measured over repeated trials). In the cognitive sciences, for example, the search for motor invariance led to a narrow interpretation of variability in motor behaviour as evidence of noise or random fluctuations at different levels of the movement system (e.g. anatomical, mechanical, physiological). From a dynamical systems theoretical perspective, variability in movement systems is considered to be a central theoretical issue worthy of study in its own right, and has been related to the sensorimotor equivalence that arises from the abundance of motor system degrees of freedom characterising the human body.[11]

From this new perspective, variability of performance has been viewed as more functional, since a consistent outcome can be achieved by different patterns of joint relations owing to the dynamics of the joint biomechanical degrees of freedom (DOF). For example, a defining feature of a chaotic system is that deterministic processes can drive fluctuations in system output that apparently seem random. In principle, a range of deterministic and stochastic processes could contribute to the observed fluctuations in movement and its outcome. With such a view, noise may have a positive role in preventing a system from becoming too stable in complex environments so that functional movement solutions may be found during exploratory behaviour of athletes during practice or rehabilitation.[12] Furthermore, there is growing evidence that the nature of movement variability is driven by the interaction of the various sources of constraints on action, and this leads to the uniqueness of system dynamics for a particular performer under a specific set of task constraints. This task-specific view may provide a better framework for understanding the role of inter- and intra-individual variability in the provision of diagnoses and treatment interventions in human movement by sports medicine specialists.

This paper provides a brief overview of the application of dynamical systems theory to the study of motor behaviour before making inferences about the need to revise understanding of inter- and intra-individual levels of variability in sport and exercise, and health behaviours for sports medicine. We draw parallels between the biological determinism implicit in traditional conceptions of variability as noise superimposed on the production of idealised motor representations, and the biological determinism prevailing in some medical science interpretations of perceptual-motor disorders and the role of genes in constraining health and physical performance.

It will become clear that a broader understanding of individual variability, including performance variability, requires careful interpretation of data by sports clinicians, since it can be functional in facilitating adaptations of individual biological systems to the changing constraints of dynamic environments. To contextualise discussion, we selectively focus on two important areas for sports medicine: (i) postural control; and (ii), the relative role of genes and the environment in constraining inter-individual variations in health and performance. We evaluate the implications of this new view of variability for the ubiquitous ‘medical model’ in clinical assessments of health and movement behaviours.

1. Dynamical Systems Theory and the Study of Movement Behaviour

Dynamical systems theory is a multidisciplinary, systems-led approach, encompassing mathematics, physics, biology, psychology and chemistry, to des-

cribe systems that are constantly changing and evolving over different timescales.[13] A central tenet of dynamical systems theory is that natural phenomena can be explained, at multiple scales of analysis, with the same underlying abstract principles regardless of system structure and composition.

1.1 Movement Systems as Dynamical Systems

The study of movement systems as dynamical systems contains two strands of modelling work. The first approach investigates information-force transactions between a performer and the environment using the analytical tools of nonequilibrium thermodynamics.[14,15] Kinematic patterns in movement systems are viewed as the product of force (kinetic) fields, which lawfully give rise to flow (informational) fields. This programme of work examines coordination as an emergent process in dynamic environments, based on the intentions of the performer.[16,17] A second, related research line is founded on 'pattern dynamics' which initially sought applications of synergetics and nonlinear dynamics to human movement.[18] The goal of this programme is to construct dynamical equations of motion of relevant coordination phenomena that capture the stability and loss of stability associated with persistence and change in movement systems, for example occurring during performance transitions as well as longer-term changes due to learning, development or rehabilitation in individuals.

In dynamical systems, spontaneous pattern formation between component parts has been found to emerge through processes of self-organisation. Such systems are typically 'open' thermodynamic systems engaged in constant energy transactions with the environment.[17] Self-organisation is manifested as transitions between different organisational states emerging due to internal and external constraints pressurising system components into change.

The concept of constraints from dynamical systems theory perhaps has the greatest implications for a new view of individual variability for sports medicine. Constraints have been defined as boundaries or features, which interact to limit the form of biological systems searching for optimal states of organisation.[19,20] Constraints reduce the number of configurations available to a dynamical system by structuring the state space of all possible configurations available to it. There are many classes of constraints that can shape the behaviour of a dynamical system and Newell[21] categorised them as organismic (pertaining to the individual), the task and the environment (see figure 1; note that this paper advocates an adaptation of the model to the study of human behaviour including movement coordination).

2. Bernstein's (1967) Problem

Key ideas from dynamical systems theory have been allied to the theoretical insights of the pioneering Russian physiologist and biomechanist, Nikolai Bernstein who formulated the fundamental problem for movement systems as "the process of mastering the redundant degrees of freedom" or more succinctly "the organisation of the control of the motor apparatus".[22] Bernstein used the term redundant DOF to refer to the (bio)mechanical DOF that exceed the minimum number required to successfully accomplish any given motor task.[23] Newell and Vaillancourt[24] proposed that DOF are the "number of independent coordinates required to uniquely describe the configuration of a system". For instance, there are seven DOF in the human arm, three at the shoulder, one at the elbow and three at the wrist, far more than needed for most typical interceptive arm movements, providing flexibility and security in adapting to changing environmental conditions.[25,26]

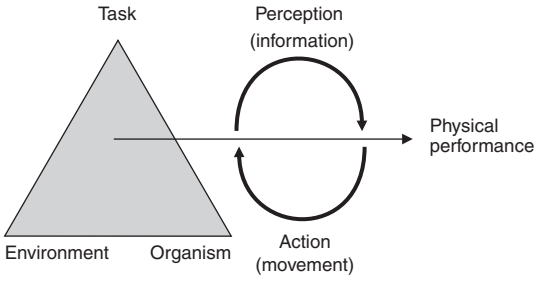

Fig. 1. Newell's model of interacting constraints adapted to illustrate the resulting effects on variability of physical performance.

At different scales of analysis, the human movement system is replete with redundancy. For example, molecular biologists have found that part of the DNA code specifies which proteins (long strings of amino acids) are made during development. Twenty different kinds of amino acids have been found and it appears that DNA code is read in groups of consecutive nucleotides, arranged into triplets. For each triplet there is one amino acid. With 64 possible triplets and 20 amino acids, there is redundancy even at this molecular level of the human body (i.e. more than one nucleotide triplet matches the same amino acid).[27]

At the behavioural level, Bernstein's initial solution to the redundant DOF problem was that humans eliminated a portion of the DOF by "rigidly and spastically fixing"[22] joint articulations to restrict segmental rotation and translation. Movement systems cope with the redundant DOF by introducing temporarily strong, rigid couplings between multiple DOF resulting in more controllable single 'virtual' DOF complexes termed 'coordinative structures'.[28] Coordinative structures constrain the interconnections made by parts of the movement system during functional movements. They have been defined by Kay[29] as "…an assemblage of many microcomponents …assembled temporarily and flexibly, so that a single microcomponent may participate in many different coordinative structures on different occasions". Coordinative structures reduce the dimensionality (i.e. complexity) of the dynamical movement system by allowing humans to exploit the inherent interconnectedness of the anatomical system. An essential feature of a coordinative structure is that if one of the component parts introduces an error into the common output, the other components automatically vary their contribution to movement organisation and minimise the original error.[30] Flexibility in adapting to local conditions is enhanced by the capacity of parameters of coordinative structures to be tuned by information so that movement goals can be achieved.[28]

Despite recent claims that the original problem of motor redundancy was itself inadequately formulated,[30-32] Bernstein's preliminary insights have provided the foundations for substantial theorising and experimentation in motor control. Kelso and Schöner[33] argued that, contrary to traditional theoretical emphases, a greater understanding of the behaviour of human movement systems *qua* complex biological systems would be revealed in organisational principles rather than hard-wired mechanisms or psychological constructs such as 'representations' encoded within the central nervous system. Variability of movement has a functional role in selecting for the coordinative structures that emerge under constraint as less functional states of organisation in the motor system are explored and abandoned.[2,17,19,28,34] This retreat from biological determinism recognises that biological systems are required to generate both stable (persistent) and flexible (variable) behavioural output in response to changing intentions and dynamic environmental conditions, and signals a new way for understanding variability observed in movement systems.

3. Motor System Variability

As we noted in the introductory section, an important theme in dynamical systems accounts of human movement behaviour is that careful inferences need to be drawn about the variability observed in movement behaviour by clinicians and sport scientists.[12,24,35] In some parts of the system, higher levels of variability can actually reflect subconscious compensatory measures by individuals. In sport, compensatory variability can be observed in performers who are skilful at exploiting the high dimensionality offered by the many motor system DOF. Evidence shows that skilled performers can freeze or unfreeze the DOF in the chain of movement as the prevailing task constraints demand.[24] Less skilled performers, in comparison, tend to rigidly fix DOF and may show as much or more variability that is not functional, owing to weak adaptation to task constraints. Furthermore, through ageing and illness, human movement systems tend to show a loss of complexity, defined as inadequate adaptation to environmental changes and hence reduced functionality.[24]

An example of functional variability was reported in data on the shooting performance of skilled and unskilled marksmen by Arutyunyan and colleagues.[36] They identified different levels of variability in each joint of the upper arm in skilled performers. This finding has since been replicated and interpreted as contributing to success in the task, since the gun and performer's motor apparatus were viewed as forming a complex system.[37] Higher levels of variability in the shoulder and elbow joints complemented each other to allow the wrist (and therefore gun) to maintain a stable position. The interface of the gun with the hand provided the lowest magnitude of intra-trial variability, fitting the need to be very stable at this location. The same high levels of variability were not seen in the shoulder and elbow joints of unskilled shooters, with the consequence that the pistol position remained unstable and more variable during shooting.

The findings of the studies on pistol shooting[36,37] provide a powerful rationale for clinicians and coaches to consider functional variability of motor behaviour as a key criterion of successful performance, rather than the ability to replicate an ideal movement template or common optimal motor pattern.[38] That is, motor patterns emerge under different task constraints to achieve stable task outcomes, and do not appear as pre-determined, invariant, anatomical assemblages. Kugler and Turvey[17] argued that descriptions of goal-directed movement behaviour should focus on the functions of an action, not on the specific anatomical units involved because "… an act is functionally specific, its variability is in reference to preserving the function it fulfils rather than preserving any particular aggregation of body parts that it happens to involve".

4. Coordination Profiling

The new view of variability signals that the motor system's inherent noisiness results in variability being omnipresent, unavoidable, and yet functional in helping to produce stable movement outcomes. Despite the search for invariance in cognitive science studies of motor control over the past decades, studies have revealed that even elite athletes are unable to reproduce invariant movement patterns despite years of practice.

Schöellhorn and Bauer[39,40] developed a pattern recognition algorithm for identifying individual movement patterns for various types of sport and everyday life tasks based on the production of non-linear, categorising, self-organised neural network maps. This method was used to qualitatively analyse intra- and inter-individual levels of performance variability in athletes during complex sport actions. The algorithm effectively measures the 'space' between separate performances within and between each individual. A smaller amount of space between performances could be interpreted as signifying lower levels of inter- or intra-individual variability, depending on whether the analysis was between different athletes or over repeated trials for the same performer. In this way, clusters of performances could be picked up, providing a qualitative measure of variability in movement patterns. Performance clusters of higher density signified lower levels of variability, with lower density of clusters implying greater levels of variability.

In one study,[40] two discus throwers were filmed with high-speed cameras during several training and competition periods. The result was 45 trials for analysis by a decathlete and eight trials for analysis by a specialist discus thrower. Data were obtained on positions and angular velocities of all body and limb joints from the point of the last step of the athlete to the moment the discus left the hand. The data revealed clustering across different training and competitive sessions for each individual. The movements were more similar during each individual session and varied between different sessions. These findings belie the traditional view of expert performance being characterised by invariant features. Clearly, even elite athletes come to training and competition sessions unable to reproduce identical movement patterns despite years of practice. In another study by Bauer and Schöellhorn[40] higher levels of inter-individual variation were found within the clusters of the international athletes, compared with the national athletes, rejecting the idea of

the existence of common optimal movement patterns in highly skilled performers.

Further evidence of both inherent and functional variability has emerged from studies of interceptive actions in sport such as long jumping, triple jumping, and table tennis reviewed by Davids and colleagues.[41] In long jumping, for example, the literature suggests that both expert and novice performers exhibit a two-phase approach strategy that includes: (i) an initial acceleration phase during which athletes attempt to maintain a stereotypical stride pattern while progressively increasing their stride length as they accelerate down the track; followed by (ii), a zeroing-in phase during which athletes attempt to modify stride length parameters over the final strides. Analyses of inter-trial footfall variability in relation to the take-off board have revealed an ascending-descending trend that corresponds with the two phases of the run-up. During the acceleration phase, small inconsistencies in stride length, representative of inherent variability effects in motor systems, are accumulated until approximately 4 strides before the take-off board. After optimal horizontal velocity has been reached, visual control takes over during the zeroing-in phase and stride length is regulated to remove the initial variability created during the acceleration phase, indicating that the variability observed in the last four strides is functional. This approach has also been observed in other sports such as golf. In a study examining the variability of ground reaction forces during the backswing, Koenig and coworkers[42] reported that all golfers (n = 14) regardless of handicap (range from 1–18) showed increasing variability of force production from the initiation of the back swing to the mid-point of the downswing, followed by a decrease during the impact phase, and a further increase in the follow-through.

In summary, it seems that increasing expertise does not lead to movement invariance and the construction of a single, pre-determined motor pattern, as argued in cognitive science theories of motor control. This misconception is an example of the biological determinism that influences much theory and experimentation in human behaviour.[27] Data on sport performance suggest that practitioners should engage in 'coordination profiling', and resist interpreting performance data in terms of proximity of individuals to a perceived common optimal motor pattern.[38] Coordination profiling refers to the use of individualised, in-depth analyses to examine how each individual performer uniquely satisfies specific task constraints during goal-directed behaviour.[43] It recognises that individuals approach performance, training, practice, and rehabilitation with distinct intrinsic movement system dynamics shaped by many important constraints. With the increasing capacity for in-depth analysis of movement coordination brought about by technological advances, the existence of subtle individualities or 'signature' patterns is becoming apparent, even in highly constrained tasks.[3] The powerful constraints on individual performance variation underlines why the statistical technique of pooling individual data in the study of motor coordination (i.e. reporting group means and standard deviations) may have limited value since, according to Kelso,[3] "one might as well average apples and oranges".

5. Coordination Profiling and the Medical Model

Parallels can be drawn between the sports science and medical literature. The misconception of 'common optimal movement patterns' also exists in the study of perceptual-motor disorders and has implications for sports medicine. For example, in the area of disability studies, the implicit 'medical model' or 'disability as tragedy model', used by many clinicians provides a unitary, biologically determined perspective of health and movement behaviour in which variability, viewed as deviation from an 'accepted' norm, is seen as dysfunctional and an index of abnormality.[44] In this model, health and performance behaviours are identified as problems for the individual since they may deviate from what is perceived as population norms. Rather, the alternative view prompted by dynamical systems theory is that variability in behaviour may be viewed as an adaptation to unique organismic constraints such as genes, motor system structure or personality. Vari-

ability has a functional role in helping individuals adapt to ever-changing constraints imposed on them by environmental, anatomical and physiological changes due to disease, illness, injury and ageing. An implication of this view for clinicians is that behaviours exist on a spectrum characterising the boundaries of naturally occurring variability.

The terms 'impaired' and 'elite' performance in sport and exercise need to be understood in relation to the constraints on each individual.[45] This is because the precise location that a movement system inhabits on the performance spectrum emerges from the multitude of constraints pressurising it at specific points during the lifespan. Since the constraints on each individual are many and unique, it follows that coordination solutions will differ within and between individuals in order to maintain functionality. As Latash and Anson[45] note, the "phenomena of variability of voluntary movements by themselves indicate that 'correct' peripheral motor patterns may form a rather wide spectrum". This fundamental principle applies to healthy individuals across the lifespan as well as those with injuries, diseases and perceptual-motor disorders. Latash and Anson[45] proposed that adaptations to constraints should not necessarily be perceived as pathological since motor patterns may be optimal for the conditions affecting the individual's motor system at any given point in time. Typically, although motor patterns in individuals with cognitive, perceptual and motor deficits may be defined by some clinicians as 'abnormal' compared with 'common optimal motor patterns' idealised in the 'medical model', they may be better viewed as functional and emergent under the confluence of constraints that each individual needs to satisfy.

Therefore, treatment interventions in sports medicine should not be directed towards the achievement and maintenance of an 'ideal' motor pattern. This is not the point of therapy or rehabilitation programmes. The overarching aim of an intervention should be to help individuals satisfy the unique conflagration of constraints that impinge upon them in order to improve their functional capacity in the performance contexts they face.

In the concluding sections of this paper, we examine the implications of these ideas for sports medicine by examining the study of postural control and for assessing the genetic basis of physical performance.

6. Interacting Constraints on Postural Control

During the past decade, an increasing number of investigations into postural control have been conducted within a dynamical systems framework,[46-48] with an important line of enquiry attempting to determine how different sensory systems (i.e. visual, acoustic, haptic, proprioceptive) interact and contribute to postural behaviour.[49,50] In many studies, movement scientists and clinicians have typically used a 'constraints-led' approach whereby different informational and task constraints have been experimentally manipulated to examine their effects on postural control in individuals with and without movement disorders.[51] Controlling posture and maintaining balance whilst performing rudimentary motor tasks such as standing or walking is a major challenge for many elderly people and for patients with movement disorders. The potential for severe injury or even fatality caused by accidental falling due to a loss of postural stability has prompted extensive analysis of the task and individual constraints on the regulation of posture.[52-54]

One example of a 'constraints-led' approach was proposed in a study by Davids et al.[47] which examined the interaction of task and informational constraints on postural behaviour in healthy individuals and patients with anterior cruciate ligament (ACL) deficiency. The ACLs represent an important part of the knee joint complex as they provide mechanical stability to prevent anterior displacement of the tibia on the femur and proprioceptive information about postural sway.[55-58] Postural control was assessed using a static postural sway meter and a dynamic stabilometer under six different conditions: standing on both legs with eyes open and closed; standing on the injured leg with eyes open and closed; and standing on the non-injured leg with eyes open and closed.[47] Under static task conditions, results show-

ed that proprioceptive information was not a major factor in postural control (figure 2 and figure 3). Under more dynamic task constraints, significant differences in postural control between the ACL-deficient group and the control group were observed only when vision was unavailable suggesting that vision is the most important source of exproprioceptive information. Clearly, these findings have substantial practical implications for the design of clinical tests to assess functional postural behaviours.

Some interesting findings on centre of pressure (COP) dynamics from the postural sway meter were also reported.[47] In postural control research, COP profiles have been routinely used to indicate postural stability as they are related to postural sway.[59] Davids et al.[47] found that the mean and maximum COP velocity of the control group was greater than that of the ACL-deficient group (see figure 4 and figure 5). According to previous research, these

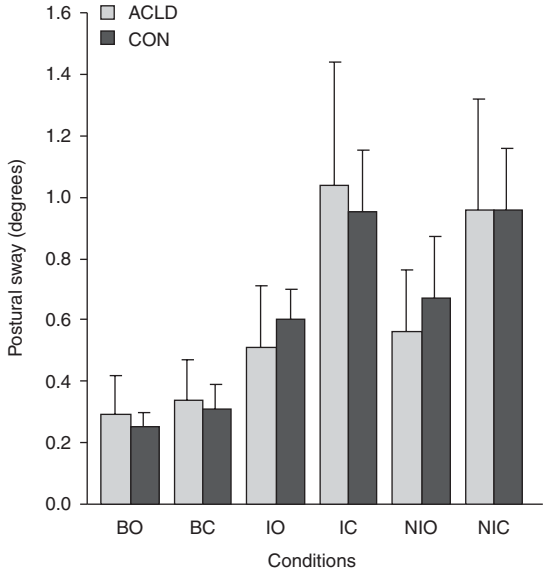

Fig. 2. Whole body sway data under the static task constraints of the postural sway meter as a function of type of stance and informational constraints (reproduced from Davids et al.,[47] with permission). **ACLD** = anterior cruciate ligament deficient group; **BC** = standing on both feet with eyes closed; **BO** = standing on both feet with eyes open; **CON** = control group; **IC** = standing on the injured leg with eyes closed; **IO** = standing on the injured leg with eyes open; **NIC** = standing on the non-injured leg with eyes closed; **NIO** = standing on the non-injured leg with eyes open.

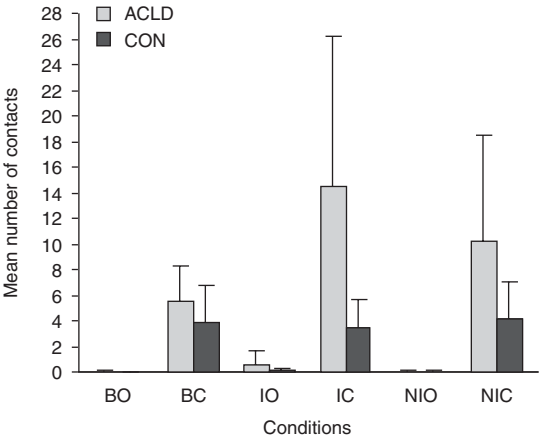

Fig. 3. Data on the number of contacts with the ground on the stabilometer under the dynamic task constraints as a function of type of stance and informational constraints (reproduced from Davids et al.,[47] with permission). **ACLD** = anterior cruciate ligament deficient group; **BC** = standing on both feet with eyes closed; **BO** = standing on both feet with eyes open; **CON** = control group; **IC** = standing on the injured leg with eyes closed; **IO** = standing on the injured leg with eyes open; **NIC** = standing on the non-injured leg with eyes closed; **NIO** = standing on the non-injured leg with eyes open.

contradictory findings provide an indication of greater postural stability in the ACL-deficient group than in the controls since COP velocity is considered to have an inverse relationship with the ability to control posture.[60,61] Alternative explanations of participants reducing postural sway in order to reduce pain or injury were discounted because there were no reports of discomfort or self-concern during the performance of this static balancing task. Davids et al.[47] suggested that the higher COP velocity in the control group should not be interpreted as greater instability but rather as indicative of normal exploratory behaviour (i.e. generation of postural sway) in discovering stable solutions to the postural control problem.[49,50,62,63]

This line of reasoning has also been followed in a number of other studies investigating the relationship between COP variability and stability in individuals with movement disorders such as Parkinson's disease.[51] A feature of individuals with Parkinson's disease is the inherent lack of postural stability and the proneness to accidental falling. An implicit assumption of much previous research is

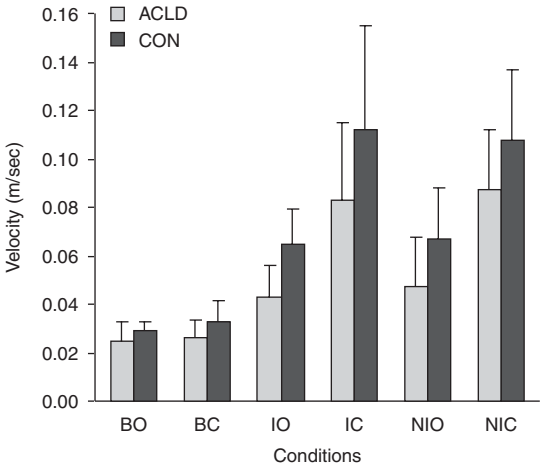

Fig. 4. Mean centre of pressure velocity data under the static task constraints of the postural sway meter as a function of type of stance and informational constraints (reproduced from Davids et al.,[47] with permission). **ACLD** = anterior cruciate ligament deficient group; **BC** = standing on both feet with eyes closed; **BO** = standing on both feet with eyes open; **CON** = control group; **IC** = standing on the injured leg with eyes closed; **IO** = standing on the injured leg with eyes open; **NIC** = standing on the non-injured leg with eyes closed; **NIO** = standing on the non-injured leg with eyes open.

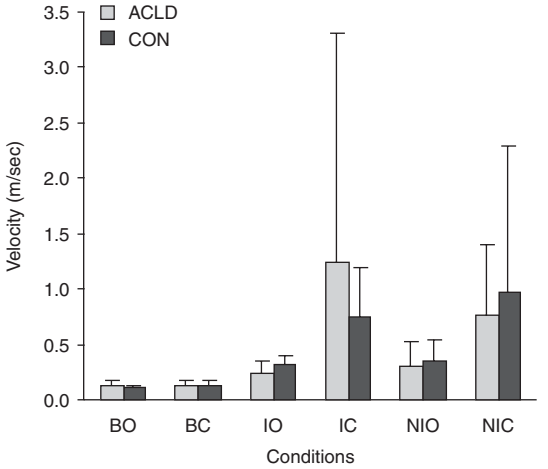

Fig. 5. Maximum centre of pressure velocity data under the static task constraints of the postural sway meter as a function of type of stance and informational constraints (reproduced from Davids et al.,[47] with permission). **ACLD** = anterior cruciate ligament deficient group; **BC** = standing on both feet with eyes closed; **BO** = standing on both feet with eyes open; **CON** = control group; **IC** = standing on the injured leg with eyes closed; **IO** = standing on the injured leg with eyes open; **NIC** = standing on the non-injured leg with eyes closed; **NIO** = standing on the non-injured leg with eyes open.

that postural instability is associated with large and highly variable COP oscillations.[64] However, investigations conducted within a dynamical systems framework suggest that under certain task constraints healthy individuals exhibit greater COP oscillations than individuals with Parkinson's disease.[65,66] These findings suggest that movement variability plays a functional role in the detection and exploration of stability boundaries during bipedal stance and locomotion.[67,68] Additionally, higher levels of postural sway variability, found in participants with eyes closed (compared with eyes open), indicated greater use of perceptual information in order to control posture and reduce effects of random fluctuations in the musculature of the postural control system.[67,68] Riley and Turvey's[12] comments on these findings succinctly capture the implication for sports medicine: "More variable does not mean more random, and more controllable does not always mean more deterministic".

7. Genes and the Environment as Constraints on Human Behaviour

In science, perhaps the most controversial analysis about variability in human behaviour concerns the relative influence of genetic and environmental influences. How these constraints shape variations in human performance is a question of increasing interest in sports medicine. This problem is a manifestation of the longstanding nature-nurture debate that has dogged biological science, education, sociology, philosophy and psychology for many years, meaning the identification of the precise proportion of performance variation in a population accounted for by genetic characteristics or environmental influences. Much has been written about this particular dualism in science, and resolution of the debate has proved difficult. For excellent analyses see Lewontin,[27] and Johnston and Edwards.[69]

From a dynamical systems perspective, it is clear that both nature and nurture can act as constraints on behaviour, although there have been few attempts to determine how organismic constraints of a genetic

basis interact with environmental or task constraints, as predicted by Newell's (1986) model. Nevertheless, it is possible to interpret existing data of relevance to sports medicine, based on the interaction of genetic and environmental constraints. For example, there is growing consensus in the study of human obesity that the contribution of genetic factors is exacerbated in environments that differ in cultural constraints including caloric availability.[70] Genetic propensity towards adiposity has less of a constraining influence on individuals in environments where caloric availability was lower, whereas these same individuals would be at greater risk in other environments. Such environments can be categorised as high or low risk, depending on the prevalence of other significant cultural constraints including work patterns imposed on traditional meal times, and popularity of non-physically active pastimes such as computer games and TV watching. Thus, the effects of the interaction of genes and environment on phenotypic expression of behaviour can be best understood at the level of individual risk, rather than as defective behaviour.

The past few years have also seen increasing work on the role of genes in defining the level of athletic performance attainable by individuals. In the main, research has focused on genetic and environmental contributions to physical, typically endurance, performance[71] although there have also been some attempts to evaluate relative contributions to acquisition of motor skill.[72] The significant amount of inter-individual variation observed in response to training of the cardiovascular system has led to many investigators questioning the extent to which genetic diversity may be responsible for the data.[73]

The search for the genetic basis of many human capacities, such as physical performance, has engendered strong rhetoric in some quarters, with some molecular biologists calling it the 'biological counterpart to the holy grail',[74] and some sport scientists asserting that genes are responsible for up to half of the variation in physical performance between individuals within a population.[75] Currently, the role of the angiotensin-converting enzyme (ACE) gene is receiving considerable attention in the sports medicine literature and in section 8 we evaluate the evidence for its role as a genetic constraint on variation in physical endurance performance.

8. The Angiotensin-Converting Enzyme (ACE) Gene

The ACE gene is one of a number of candidate genes associated by research with inter-individual variability in physical endurance performance.[76-85] In muscle, the angiotensin I-converting enzyme has the role of degrading vasodilators, bradykinin and tachykinin, and stimulating production of the vasoconstrictor angiotensin II during physical performance.[86] Sequence variation in DNA has led to the existence of two alleles of the ACE gene, I and D (I for insertion and D for deletion), which combine to produce polymorphisms in genotype (II, ID, DD).

The D variant of the gene has been linked with relatively higher ACE activity. For example, early work with army recruits found that the II polymorphism of the gene has been associated with lower activity levels of ACE in muscle than the DD allele, and an increased response to physical training.[87] Recruits with ACE genotype II differed by as much as 1100% in response to repetitive upper-arm exercises compared with DD genotype peers. Individuals with a heterogeneous genotype (DI) were associated with levels of performance between both homozygous genotypes. In sport, a higher prevalence of the II genotype has been found in elite endurance athletes including mountaineers able to climb to 7000m without the aid of oxygen, Olympic-standard endurance runners and elite rowers.[78,87,88]

8.1 The ACE Gene: Some Critical Issues

Although some work on endurance performance in elite athletes has failed to support the more functional role of the I allele of the ACE gene,[80] the cohort of athletes in that study consisted of 120 performers chosen from a variety of sports with different task constraints which possibly masked any observed effects (including 26 hockey players, 25 cyclists, 21 skiers, 15 track and field athletes, 13 swimmers, 7 rowers and 5 gymnasts). Such a mixed

group of athletes may not have had the requisite levels of phenotypic homogeneity to lead to valid estimates of the genetic basis of performance. Moreover, it has become clear that carriers of the D allele appear to have an advantage in training and performance when task constraints emphasise power over a shorter duration.[78,79] In fact, the D allele has been related to increased quadriceps muscle strength gains following 9 weeks of isometric training.[89] It is possible that the D allele may confer some performance and training benefit in task constraints requiring power (perhaps through its effect on greater angiotensin II genotype and muscle hypertrophy), and similarly the I allele an effect under endurance task constraints. The implication is that variability at the level of individual genes provides functionality and adaptability in movement systems needing to perform a variety of activities in a complex environment. This suggestion also emphasises that, in experiments on the ACE gene variants and sport performance, a clear understanding of differences in task constraints is needed to ensure homogenous cohorts of athletes are carefully examined to avoid the loss of genetic association.

Finally, research on the ACE gene is progressing rapidly and there are some indications that its role in constraining physical performance may be somewhat different than originally perceived. For example, there has been some doubt cast on the relationship between the presence of the I allele of the ACE gene and the responsiveness to endurance training originally proposed in some studies.[87,88,90] The locus of the ACE gene has been identified as chromosome 17q23 and genomic scanning for presence of candidate genes for baseline maximum oxygen uptake ($\dot{V}O_{2max}$) performance or responsiveness to training failed to confirm evidence of linkage.[91] These findings on a sedentary population were supported by a frequency analysis, which failed to find a relationship between the accumulation of alleles I and II and endurance performance in 192 elite athletes (skiers, runners and cyclists) and 189 controls.[71] Interestingly, the highest frequencies reported for both the elite athlete group and controls were for the ID genotype (0.46 and 0.47, respectively). Nevertheless, future research needs to ascertain whether: (i) the effect of the I allele of the ACE gene on endurance performance is mediated via peripheral muscle effects and changes in efficiency; and (ii) the effect of the D allele on performance under power task constraints is mediated via increased angiotensin II acting as a local hypertrophic factor in muscle.

In interpreting the data from studies of the genetic constraints on physical performance and motor skill acquisition, there is enormous potential for confusion amidst the rhetoric. On the face of it, the data in favour of a strong genetic constraint on physical performance seem compelling. Whilst most researchers working in the area of genetic variations in human performance agree with Hopkins'[75] opinion that athletes are born and made, a clear interpretation of the data on the ACE gene is needed to understand how athletic performance emerges under interacting constraints.

An example of the need for care in drawing appropriate conclusions from data, in the face of the rhetoric that human physical performance is *strongly* influenced by genetic factors,[78] was provided in a study by Bouchard and colleagues.[91] They attempted to estimate the proportion of influence attributable to genetic and environmental constraints on familial resemblance for $\dot{V}O_{2max}$ during exercise on a cycle ergometer in sedentary individuals.[91] For this purpose, exercise performance of fathers, mothers, sons and daughters was measured in 86 nuclear families. Maximum heritability including genetic and non-genetic causes for physical performance accounted for 51% of the total adjusted phenotype variance. Several models of interacting constraints were tested and results showed that there was 2.6–2.9 times more variance between families than within families.

Unfortunately, the approach taken in this study by Bouchard et al.[91] meant that genetic and familial environmental influences could not be fully quantified separately although "inferences about their respective contributions to the phenotype variance could be made by inspection of the pattern of familial correlations". It is important to note here that

correlations do not imply causation, and that the focus on the constraints imposed by the shared familial environment only, precludes the influence of *wider* environmental constraints, such as socio-cultural changes in society, including impact of media images, government education programmes, and peer group pressure, being factored into the equation.

Moreover, conclusions on the contribution of the genetic component to performance were somewhat speculative. Despite the fact that maximal heritabilities reported in this study were inflated by familial non-genetic contributions, the effects of maternal transmission of mitochondrial DNA to the fertilised zygote were proposed as being optimally allied to the father's environmental contribution. Bouchard et al.[91] argued that the data 'revealed' that "maternal influence, perhaps by mitochondrial inheritance, accounts for as much as 30% of the familial transmission". The authors' conclusion that, "Based on the present results, we estimate that 'mitochondrial' heritability is in the range of 30–35%", seems a speculative interpretation of the data based on correlational statistics, a limited range of environmental constraints impacting on model construction and no evidence from DNA analysis.

8.2 Problems of Interpreting Data from Genetic Studies

In order to avoid confusion over the relative influences of genetic and environmental constraints on behaviour it is worth reiterating what is already known in this area of work. It is important to note that heritability of a trait is constrained by genetic and environmental factors to some extent and that research in behavioural genetics is concerned with explanations of hereditary influences at the level of populations, not individuals. As most geneticists working on physical performance understand, genes work in combination to influence biological function, refuting the idea of successful athletes being differentiated on the presence of a single gene (for a similar argument in developmental theory see Johnston and Edwards[69]). It has also become clear that genes are not biologically determinate, since even the most ardent geneticists agree that transmission of genetic information between generations is less than perfect.[92] DNA is simply a copy of information transmitted between generations, which is read by cellular machinery in the production of proteins that create the individual, part by part. This is important to note because the view of DNA as information bearer has been replaced with the fallacy of DNA as blueprint, plan, or master molecule.[27]

8.3 Genes and Variability in Movement Systems

The presence of genetic material should not be viewed as a blueprint for success in sport. As Johnston and Edwards[69] have pointed out, it is "a very long step from polypeptide sequences to behaviour – a step …that covers much incompletely understood territory". Attempts to see genes as building plans or coded blueprints are one of the great artificialisms of human conceptualisations of nature.[93] It has become a 'central dogma' of how people think about the process of evolution.[94] In relation to this argument, it is interesting to note that a major aspect of Darwin's legacy was to change the emphasis in evolutionary theory from a transformational model (focus on group modal properties) to a variational model (focus on variability amongst individual members of a species). Moreover, a major argument against the blueprint conceptualisation of the role of genes is found in evidence that identical twins are not identical! For example, evidence from the study of phenotypically identical twins showed that their fingerprints differ and the shape of their brains can differ by as much as 40%.[95] This change in emphasis was geared to the idea that the internal constraints on variability (e.g. genes) are causally dependent on the environmental constraints that select for them.[27]

Parallels can be drawn with the revision of understanding of the concept of movement variability in dynamical systems accounts of behaviour. Lewontin's criticisms of the stance of biological determinism[27] is reflected in the medical model's rejection of polymorphism and the implicit notion of variability as deviation from a 'perfect ideal'. Genetic diver-

sity is the norm and biological systems are not DNA-determined. There is no single, standard, normal DNA sequence that we all share and estimates are that we differ in DNA sequence by 0.1% (~3 million nucleotides) including inherited sequences from parents. It takes more than DNA to produce a living organism, which cannot be computed from DNA sequences. According to Lewontin,[27] "A living organism at any moment of its life is the unique consequence of a developmental history that results from the interaction of and determination by internal and external forces".

In summary, it seems that the evidence strongly supports the view that genetic and environmental factors do interact to constrain performance variability observed between sport performers. Genetic diversity may be responsible for a small part of training or performance response in individuals. It appears that the influence of genetic factors is often very small, and only when there is a favourable interaction with important environmental constraints are performance benefits observed. The implication is that elite endurance athletes of a less favourable genotypic disposition can succeed with the appropriate training environment, but it can be concluded that performers with a more favourable genotype, who interact with their training environment in an appropriate manner, are more likely to receive a greater training response.

9. Conclusions

In this article we have highlighted how dynamical systems theory has paved the way for a new interpretation of movement variability, avoiding the biological determinism implicit in traditional theories of motor control. It is becoming clear that the human motor system is intrinsically noisy, and the new perspective on movement variability is seen as functional in allowing individuals to adapt to the multitude of unique constraints on performance. We have argued that sports clinicians need to understand the nature of the interacting constraints, which shape emergent movement behaviour, warranting reconsideration of some practices and approaches in sports medicine. The new, more positive, interpretation of the role of variability implies that it should be studied as an inherent part of motor system output, and a concomitant focus of sports medicine should be on developing techniques, such as coordination profiling, in order to study the relationship between variability of performance and the nature of the interacting constraints impinging on each individual. To support our arguments, we examined some of the current literature on postural control and the genetic basis of endurance performance as vehicles for understanding the pervasiveness of biological determinism in traditional theories of human behaviour, exemplified by the search for motor invariance or 'common optimal motor patterns' in sport and exercise science and the pervasiveness of the 'medical model' in sports medicine.

The main implications for sports medicine include: (i) an integrated explanatory framework may be more beneficial in understanding physical performance in sport, rather than unitary scales of analysis, (e.g. social, psychological, physiological, molecular); (ii) variability in movement behaviour may be functional due to the interacting constraints on behaviour; and (iii), a better comprehension of human functioning (and malfunctioning) may be obtained by describing the dynamics of different sub-systems of the body and by understanding macro- (performer-environment) and micro- (at the level of muscles and joint complexes) level interactions.

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Correspondence and offprints: Professor *Keith Davids*, School of Physical Education, University of Otago, PO Box 56, Dunedin, New Zealand.
E-mail: kdavids@pooka.otago.ac.nz