Infinite–Dimensional Cerebellar Controller for Realistic Human Biodynamics

Vladimir G. Ivancevic∗ Tijana T. Ivancevic†

Abstract

In this paper we propose an $\infty$–dimensional cerebellar model of neural controller for realistic human biodynamics. The model is developed using Feynman’s action–amplitude (partition function) formalism. The cerebellum controller is acting as a supervisor for an autogenetic servo control of human musculo–skeletal dynamics, which is presented in (dissipative, driven) Hamiltonian form. The $\infty$–dimensional cerebellar controller is closely related to entropic motor control.

Keywords: realistic human biodynamics, cerebellum motion control, $\infty$–dimensional neural network

Contents

1 Introduction 2

2 Sub-Cerebellar Biodynamics and Its Spinal Reflex Servo–Control 4

3 Cerebellum: The Adaptive Path–Integral Comparator 9

∗Human Systems Integration, Land Operations Division, Defence Science & Technology Organisation, P.O. Box 1500, Edinburgh SA 5111, Australia (Vladimir.Ivancevic@dsto.defence.gov.au)

†School of Electrical and Information Engineering, University of South Australia, Mawson Lakes, S.A. 5095, Australia (Tijana.Ivancevic@unisa.edu.au)
1 Introduction

Realistic human biodynamics (RHB) is a science of human (and humanoid robot) motion in its full complexity. It is governed by both Newtonian dynamics and biological control laws (see [Iva04, IB05, II06a, II06b, II06c]).

There are over 200 bones in the human skeleton driven by about 640 muscular actuators (see, e.g., [Mar98]). While the muscles generate driving torques in the moving joints, subcortical neural system performs both local and global (loco)motion control: first reflexly controlling contractions of individual muscles, and then orchestrating all the muscles into synergetic actions in order to produce efficient movements. While the local reflex control of individual muscles is performed on the spinal control level, the global integration of all the muscles into coordinated movements is performed within the cerebellum [II06a, II06b].

All hierarchical subcortical neuro–muscular physiology, from the bottom level of a single muscle fiber, to the top level of cerebellar muscular synergy, acts as a temporal \( < | out > | in > \) reaction, in such a way that the higher level acts as a command/control space for the lower level, itself representing an abstract image of the lower one:

1. At the muscular level, we have excitation–contraction dynamics [Hat77a, Hat78, Hat77b], in which \( < | out > | in > \) is given by the following sequence of nonlinear diffusion processes [II06a, II06b]:

   \[
   \text{neural action potential} \sim \text{synaptic potential} \sim \text{muscular action potential} \\
   \sim \text{excitation contraction coupling} \sim \text{muscle tension generating}. 
   \]

---

1 Here we need to emphasize that human joints are significantly more flexible than humanoid robot joints. Namely, each humanoid joint consists of a pair of coupled segments with only Eulerian rotational degrees of freedom. On the other hand, in each human synovial joint, besides gross Eulerian rotational movements (roll, pitch and yaw), we also have some hidden and restricted translations along \((X, Y, Z)\)-axes. For example, in the knee joint, patella (knee cap) moves for about 7–10 cm from maximal extension to maximal flexion). It is well-known that even greater are translational amplitudes in the shoulder joint. In other words, within the realm of rigid body mechanics, a segment of a human arm or leg is not properly represented as a rigid body fixed at a certain point, but rather as a rigid body hanging on rope–like ligaments. More generally, the whole skeleton mechanically represents a system of flexibly coupled rigid bodies. This implies the more complex kinematics, dynamics and control then in the case of humanoid robots.
Its purpose is the generation of muscular forces, to be transferred into driving torques within the joint anatomical geometry.

2. At the spinal level, \( \langle \text{out}|\text{in} \rangle \) is given by autogenetic–reflex stimulus–response control \[\text{Hou79}\]. Here we have a neural image of all individual muscles. The main purpose of the spinal control level is to give both positive and negative feedbacks to stabilize generated muscular forces within the ‘homeostatic’ (or, more appropriately, ‘homeokinetic’) limits. The individual muscular actions are combined into flexor–extensor (or agonist–antagonist) pairs, mutually controlling each other. This is the mechanism of reciprocal innervation of agonists and inhibition of antagonists. It has a purely mechanical purpose to form the so-called equivalent muscular actuators (EMAs), which would generate driving torques \( T_i(t) \) for all movable joints.

3. At the cerebellar level, \( \langle \text{out}|\text{in} \rangle \) is given by sensory–motor integration \[\text{HBB96}\]. Here we have an abstracted image of all autogenetic reflexes. The main purpose of the cerebellar control level is integration and fine tuning of the action of all active EMAs into a synchronized movement, by supervising the individual autogenetic reflex circuits. At the same time, to be able to perform in new and unknown conditions, the cerebellum is continuously adapting its own neural circuitry by unsupervised (self–organizing) learning. Its action is subconscious and automatic, both in humans and in animals.

Naturally, we can ask the question: Can we assign a single \( \langle \text{out}|\text{in} \rangle \) measure to all these neuro–muscular stimulus–response reactions? We think that we can do it; so in this Letter, we propose the concept of adaptive sensory–motor transition amplitude as a unique measure for this temporal \( \langle \text{out}|\text{in} \rangle \) relation. Conceptually, this \( \langle \text{out}|\text{in} \rangle \)–amplitude can be formulated as the ‘neural path integral’:

\[
\langle \text{out}|\text{in} \rangle \equiv \langle \text{motor}|\text{sensory} \rangle = \int_{\text{amplitude}} D[w, x] e^{iS[x]}.
\]  

(1)

Here, the integral is taken over all activated (or, ‘fired’) neural pathways \( x^i = x^i(t) \) of the cerebellum, connecting its input sensory–state with its output motor–state, symbolically described by adaptive neural measure \( D[w, x] \), defined by the weighted product (of discrete time steps)

\[
D[w, x] = \lim_{n \to \infty} \prod_{t=1}^{n} w_i(t) dx^i(t),
\]  

(2)

in which the synaptic weights \( w_i = w_i(t) \), included in all active neural pathways \( x^i = x^i(t) \), are updated by the standard learning rule

\[
\text{new value}(t + 1) = \text{old value}(t) + \text{innovation}(t).
\]
More precisely, the weights $w_i$ in (2) are updated according to one of the two standard neural learning schemes, in which the micro–time level is traversed in discrete steps, i.e., if $t = t_0, t_1, ..., t_n$ then $t + 1 = t_1, t_2, ..., t_{n+1}$.

1. A self–organized, unsupervised (e.g., Hebbian–like [Heb49]) learning rule:

$$w_i(t + 1) = w_i(t) + \frac{\sigma}{\eta}(w^d_i(t) - w^a_i(t)),$$

where $\sigma = \sigma(t)$, $\eta = \eta(t)$ denote signal and noise, respectively, while superscripts $d$ and $a$ denote desired and achieved micro–states, respectively; or

2. A certain form of a supervised gradient descent learning:

$$w_i(t + 1) = w_i(t) - \eta \nabla J(t),$$

where $\eta$ is a small constant, called the step size, or the learning rate, and $\nabla J(n)$ denotes the gradient of the ‘performance hyper–surface’ at the $t$–th iteration.

Theoretically, equations (1–4) define an $\infty$–dimensional neural network (see [IA07, IAY08, II08c]). Practically, in a computer simulation we can use $10^7 \leq n \leq 10^8$, roughly corresponding to the number of neurons in the cerebellum [II07a, II07b].

The exponent term $S[x]$ in equation (1) represents the autogenetic–reflex action, describing reflexly–induced motion of all active EMAs, from their initial stimulus–state to their final response–state, along the family of extremal (i.e., Euler–Lagrangian) paths $x_{\min}^i(t)$. ($S[x]$ is properly derived in [89] below.)

2 Sub-Cerebellar Biodynamics and Its Spinal Reflex Servo–Control

Subcerebellar biodynamics includes the following three components: (i) local muscle–joint mechanics, (ii) whole–body musculo–skeletal dynamics, and (iii) autogenetic reflex servo–control.

2.1 Local Muscle–Joint Mechanics

Local muscle–joint mechanics comprises of [Iva06, II06a, II06b]:

\footnote{Note that we could also use a reward–based, reinforcement learning rule [SB98], in which system learns its optimal policy:

$$\text{innovation}(t) = |\text{reward}(t) - \text{penalty}(t)|.$$}
1. Synovial joint dynamics, giving the first stabilizing effect to the conservative skeleton dynamics, is described by the \((x, \dot{x})\)-form of the Rayleigh–Van der Pol’s dissipation function

\[
R = \frac{1}{2} \sum_{i=1}^{n} (\dot{x}^i)^2 [\alpha_i + \beta_i(x^i)^2],
\]

where \(\alpha_i\) and \(\beta_i\) denote dissipation parameters. Its partial derivatives give rise to the viscous–damping torques and forces in the joints

\[
\mathcal{F}_{\text{joint}}^i = \partial R / \partial \dot{x}^i,
\]

which are linear in \(\dot{x}^i\) and quadratic in \(x^i\).

2. Muscular dynamics, giving the driving torques and forces \(\mathcal{F}_{\text{muscle}}^i = \mathcal{F}_{\text{muscle}}^i(t, x, \dot{x})\) with \((i = 1, \ldots, n)\) for RHB, describes the internal excitation and contraction dynamics of equivalent muscular actuators [Hat78].

(a) Excitation dynamics can be described by an impulse force–time relation

\[
\begin{align*}
F_{\text{imp}}^i &= F^0_i(1 - e^{-t/\tau_i}) \quad \text{if stimulation} > 0 \\
F_{\text{imp}}^i &= F^0_i e^{-t/\tau_i} \quad \text{if stimulation} = 0,
\end{align*}
\]

where \(F^0_i\) denote the maximal isometric muscular torques and forces, while \(\tau_i\) denote the associated time characteristics of particular muscular actuators. This relation represents a solution of the Wilkie’s muscular active–state element equation [Wil56]

\[
\dot{\mu} + \gamma \mu = \gamma S A, \quad \mu(0) = 0, \quad 0 < S < 1,
\]

where \(\mu = \mu(t)\) represents the active state of the muscle, \(\gamma\) denotes the element gain, \(A\) corresponds to the maximum tension the element can develop, and \(S = S(r)\) is the ‘desired’ active state as a function of the motor unit stimulus rate \(r\). This is the basis for the RHB force controller.

(b) Contraction dynamics has classically been described by the Hill’s hyperbolic force–velocity relation [Hil38]

\[
F^\text{Hill}_i = \frac{(F^0_i b_i - \delta_{ij} a_i \dot{x}^j)}{(\delta_{ij} \dot{x}^j + b_i)},
\]

where \(a_i\) and \(b_i\) denote the Hill’s parameters, corresponding to the energy dissipated during the contraction and the phosphagenic energy conversion rate, respectively, while \(\delta_{ij}\) is the Kronecker’s \(\delta\)–tensor.

In this way, RHB describes the excitation/contraction dynamics for the \(i\)th equivalent muscle–joint actuator, using the simple impulse–hyperbolic product relation

\[
\mathcal{F}_{\text{muscle}}^i(t, x, \dot{x}) = F_{\text{imp}}^i \times F_{\text{Hill}}^i.
\]
Now, for the purpose of biomedical engineering and rehabilitation, RHB has developed the so-called hybrid rotational actuator. It includes, along with muscular and viscous forces, the D.C. motor drives, as used in robotics \[VBS90, Iv06, II06a\]

\[
F^\text{robo}_k = i_k(t) - J_k \ddot{x}_k(t) - B_k \dot{x}_k(t),
\]

with

\[
l_k i_k(t) + R_k i_k(t) + C_k \dot{x}_k(t) = u_k(t),
\]

where \(k = 1, \ldots, n\), \(i_k(t)\) and \(u_k(t)\) denote currents and voltages in the rotors of the drives, \(R_k\), \(l_k\) and \(C_k\) are resistances, inductances and capacitances in the rotors, respectively, while \(J_k\) and \(B_k\) correspond to inertia moments and viscous dampings of the drives, respectively.

Finally, to make the model more realistic, we need to add some stochastic torques and forces \[IS01\, II07a\]

\[
F^\text{stoch}_t = B_{ij}[x^i(t), t] dW^j(t)
\]

where \(B_{ij}[x(t), t]\) represents continuous stochastic diffusion fluctuations, and \(W^j(t)\) is an \(N\)-variable Wiener process (i.e. generalized Brownian motion), with \(dW^j(t) = W^j(t + dt) - W^j(t)\) for \(j = 1, \ldots, N\).

### 2.2 Hamiltonian Biodynamics and Its Reflex Servo–Control

General form of Hamiltonian biodynamics on the configuration manifold of human motion is formulated in \[IS01\, Iva02\, Iva04\, IB05\, II06a\, II06c\] using the concept of Euclidean group \(SE(3)\) (see Figure 1).

Briefly, based on affine Hamiltonian function of human motion, formally \(H_a: T^*Q \rightarrow \mathbb{R}\), in local canonical coordinates on the symplectic phase space (which is the cotangent

| Subgroup | Definition |
|----------|------------|
| \(SO(3)\), group of rotations in 3D (a spherical joint) | Set of all proper orthogonal \(3 \times 3\) - rotational matrices |
| \(SE(2)\), special Euclidean group in 2D (all planar motions) | Set of all \(3 \times 3\) - matrices: |
| \(SO(2)\), group of rotations in 2D subgroup of \(SE(2)\)-group (a revolute joint) | Set of all proper orthogonal \(2 \times 2\) - rotational matrices included in \(SE(2)\) - group |
| \(\mathbb{R}^3\), group of translations in 3D (all spatial displacements) | Euclidean 3D vector space |
Figure 1: The configuration manifold \( Q \) of the human musculoskeletal dynamics is defined as an anthropomorphic product of constrained Euclidean SE(3)–groups acting in all major (synovial) human joints.

bundle of the human configuration manifold \( Q \) \( T^*Q \) given as

\[
H_a(x, p, u) = H_0(x, p) - H^j(x, p) u_j,
\]

where \( H_0(x, p) = E_k(p) + E_p(x) \) is the physical Hamiltonian (kinetic + potential energy) dependent on joint coordinates \( x^i \) and their canonical momenta \( p_i \), \( H^j = H^j(x, p) \), \( j = 1, \ldots, m \leq n \) are the coupling Hamiltonians corresponding to the system’s active joints and \( u_i = u_i(t, x, p) \) are (reflex) feedback–controls. Using (5) we come to the affine Hamiltonian control RHB–system, in deterministic form

\[
\begin{align*}
\dot{x}^i &= \partial_{p_i} H_0 - \partial_{p_i} H^j u_j + \partial_{p_i} R, \\
\dot{p}_i &= F_i - \partial_{x^i} H_0 + \partial_{x^i} H^j u_j + \partial_{x^i} R, \\
o^i &= -\partial_{u_i} H_a = H^j, \\
x^i(0) &= x^i_0, \\
p_i(0) &= p_i^0, \\
(i = 1, \ldots, n; \quad j = 1, \ldots, Q \leq n),
\end{align*}
\]

(where \( \partial_u \equiv \partial/\partial u \), \( F_i = F_i(t, x, p) \), \( H_0 = H_0(x, p) \), \( H^j = H^j(x, p) \), \( H_a = H_a(x, p, u) \),
\[ R = R(x, p), \] as well as in the fuzzy–stochastic form \([\text{IS01, II07a}]\)

\[
dq^i = \left( \partial_{p_i} H_0(\sigma_\mu) - \partial_x H^j(\sigma_\mu) u_j + \partial_x R \right) dt, \\
dp_i = B_{ij}[x^i(t), t] dW^j(t) + \\
\left( \bar{F}_i - \partial_x H_0(\sigma_\mu) + \partial_x H^j(\sigma_\mu) u_j + \partial_x R \right) dt, \\
d\bar{o}^i = -\partial_u H_a(\sigma_\mu) dt = H^j(\sigma_\mu) dt, \\
x^i(0) = \bar{x}_0^i, \quad p_i(0) = \bar{p}_0^i \\
\]

In (6)–(7), \( R = R(x, p) \) denotes the joint (nonlinear) dissipation function, \( \sigma^j \) are affine system outputs (which can be different from joint coordinates); \( \{\sigma\}_\mu \) (with \( \mu \geq 1 \)) denote fuzzy sets of conservative parameters (segment lengths, masses and moments of inertia), dissipative joint dampings and actuator parameters (amplitudes and frequencies), while the bar (\( \bar{\cdot} \)) over a variable denotes the corresponding fuzzified variable; \( B_{ij}[q^i(t), t] \) denote diffusion fluctuations and \( W^j(t) \) are discontinuous jumps as the \( n \)–dimensional Wiener process.

In this way, the force RHB servo–controller is formulated as affine control Hamiltonian–systems (6–7), which resemble the autogenetic motor servo (see Appendix), acting on the spinal–reflex level of the human locomotion control. A voluntary contraction force \( F \) of human skeletal muscle is reflexly excited (positive feedback \( +F^{-1} \)) by the responses of its spindle receptors to stretch and is reflexly inhibited (negative feedback \( -F^{-1} \)) by the responses of its Golgi tendon organs to contraction. Stretch and unloading reflexes are mediated by combined actions of several autogenetic neural pathways, forming the so–called ‘motor servo.’ The term ‘autogenetic’ means that the stimulus excites receptors located in the same muscle that is the target of the reflex response. The most important of these muscle receptors are the primary and secondary endings in the muscle–spindles, which are sensitive to length change – positive length feedback \( +F^{-1} \), and the Golgi tendon organs, which are sensitive to contractile force – negative force feedback \( -F^{-1} \).

The gain \( G \) of the length feedback \( +F^{-1} \) can be expressed as the positional stiffness (the ratio \( G \approx S = dF/dx \) of the force–\( F \) change to the length–\( x \) change) of the muscle system. The greater the stiffness \( S \), the less the muscle will be disturbed by a change in load. The autogenetic circuits \( +F^{-1} \) and \( -F^{-1} \) appear to function as servoregulatory loops that convey continuously graded amounts of excitation and inhibition to the large (alpha) skeletomotor neurons. Small (gamma) fusimotor neurons innervate the contractile poles of muscle spindles and function to modulate spindle–receptor discharge.
3 Cerebellum: The Adaptive Path–Integral Comparator

3.1 Cerebellum as a Neural Controller

Having, thus, defined the spinal reflex control level, we proceed to model the top subcortical commander/controller, the cerebellum (see Appendix). The cerebellum is responsible for coordinating precisely timed (out|in) activity by integrating motor output with ongoing sensory feedback (see Figure 2). It receives extensive projections from sensory–motor areas of the cortex and the periphery and directs it back to premotor and motor cortex [Ghe90, Ghe91]. This suggests a role in sensory–motor integration and the timing and execution of human movements. The cerebellum stores patterns of motor control for frequently performed movements, and therefore, its circuits are changed by experience and training. It was termed the adjustable pattern generator in the work of J. Houk and collaborators [HBB96]. Also, it has become the inspiring ‘brain–model’ in robotic research [SA98, Sch98, Sch99].

Figure 2: Schematic (out|in) organization of the primary cerebellar circuit. In essence, excitatory inputs, conveyed by collateral axons of Mossy and Climbing fibers activate directly neurones in the Deep cerebellar nuclei. The activity of these latter is also modulated by the inhibitory action of the cerebellar cortex, mediated by the Purkinje cells.

The cerebellum is known to be involved in the production and learning of smooth coordinated movements [TGK92, FSB97]. Two classes of inputs carry information into the
cerebellum: the mossy fibers (MFs) and the climbing fibers (CFs). The MFs provide both plant state and contextual information [BC81]. The CFs, on the other hand, are thought to provide information that reflect errors in recently generated movements [Ito84, Ito90]. This information is used to adjust the programs encoded by the cerebellum. The MFs carry plant state, motor efference, and other contextual signals into the cerebellum. These fibers impinge on granule cells, whose axons give rise to parallel fibers (PFs). Through the combination of inputs from multiple classes of MFs and local inhibitory interneurons, the granule cells are thought to provide a sparse expansive encoding of the incoming state information [Alb71]. The large number of PFs converge on a much smaller set of Purkinje cells (PCs), while the PCs, in turn, provide inhibitory signals to a single cerebellar nuclear cell [FSB97]. Using this principle, the Cerebellar Model Arithmetic Computer, or CMAC–neural network has been built [Alb71, MCK92] and implemented in robotics [Sma98], using trial-and-error learning to produce bursts of muscular activity for controlling robot arms.

So, this ‘cerebellar control’ works for simple robotic problems, like non-redundant manipulation. However, comparing the number of its neurons ($10^7 - 10^8$), to the size of conventional neural networks (including CMAC), suggests that artificial neural nets cannot satisfactorily model the function of this sophisticated ‘super–bio–computer’, as its dimensionality is virtually infinite. Despite a lot of research dedicated to its structure and function (see [HBB96] and references there cited), the real nature of the cerebellum still remains a ‘mystery’.

![Figure 3: The cerebellum as a motor controller.](image)

The main function of the cerebellum as a motor controller is depicted in Figure 3. A coordinated movement is easy to recognize, but we know little about how it is achieved. In search of the neural basis of coordination, a model of spinocerebellar interactions was
recently presented in [AG05], in which the structural and functional organizing principle is a division of the cerebellum into discrete micro–complexes. Each micro–complex is the recipient of a specific motor error signal, that is, a signal that conveys information about an inappropriate movement. These signals are encoded by spinal reflex circuits and conveyed to the cerebellar cortex through climbing fibre afferents. This organization reveals salient features of cerebellar information processing, but also highlights the importance of systems level analysis for a fuller understanding of the neural mechanisms that underlie behavior.

3.2 Hamiltonian Action and Neural Path Integral

Here, we propose a quantum–like adaptive control approach to modeling the ‘cerebellar mystery’. Corresponding to the affine Hamiltonian control function (5) we define the affine Hamiltonian control action,

\[ S_{aff}[q, p] = \int_{t_{in}}^{t_{out}} d\tau \left[ p_i \dot{q}_i - H_{aff}(q, p) \right]. \]  

(8)

From the affine Hamiltonian action (8) we further derive the associated expression for the neural phase–space path integral (in normal units), representing the cerebellar sensory–motor amplitude \( \langle \text{out} | \text{in} \rangle \),

\[ \langle q_{out}^i, p_{out}^i | q_{in}^i, p_{in}^i \rangle = \int \mathcal{D}[w, q, p] e^{i S_{aff}[q, p]} \]

\[ = \int \mathcal{D}[w, q, p] \exp \left\{ i \int_{t_{in}}^{t_{out}} d\tau \left[ p_i \dot{q}_i - H_{aff}(q, p) \right] \right\}, \]

with

\[ \int \mathcal{D}[w, q, p] = \int \prod_{\tau=1}^{\eta} \frac{w_i(\tau) dp_i(\tau) dq_i(\tau)}{2\pi}, \]

where \( w_i = w_i(t) \) denote the cerebellar synaptic weights positioned along its neural pathways, being continuously updated using the Hebbian–like self–organizing learning rule (3). Given the transition amplitude \( \text{out} | \text{in} \) (9), the cerebellar sensory–motor transition probability is defined as its absolute square, \( |\langle \text{out} | \text{in} \rangle|^2 \).

In the phase–space path integral (8), \( q_{in}^i = q_{in}^i(t), q_{out}^i = q_{out}^i(t); p_{in}^i = p_{in}^i(t), p_{out}^i = p_{out}^i(t); t_{in} \leq t \leq t_{out}, \) for all discrete time steps, \( t = 1, ..., n \to \infty, \) and we are allowing for the affine Hamiltonian \( H_{aff}(q, p) \) to depend upon all the \( (M \leq N) \) EMA–angles and angular momenta collectively. Here, we actually systematically took a discretized differential time limit of the form \( t_{\sigma} - t_{\sigma-1} \equiv d\tau \) (both \( \sigma \) and \( \tau \) denote discrete time steps) and wrote \( \frac{(q_{\sigma} - q_{\sigma-1})(t_{\sigma} - t_{\sigma-1})}{(t_{\sigma} - t_{\sigma-1})} \equiv \dot{q}_i. \) For technical details regarding the path integral calculations on Riemannian and symplectic manifolds (including the standard regularization procedures), see [Kla97, Kla00].
Now, motor learning occurring in the cerebellum can be observed using functional MR imaging, showing changes in the cerebellar action potential, related to the motor tasks (see, e.g., [MA02]). To account for these electro–physiological currents, we need to add the source term \( J_i(t)q^i(t) \) to the affine Hamiltonian action (8), (the current \( J_i = J_i(t) \) acts as a source \( J_i A^i \) of the cerebellar electrical potential \( A^i = A^i(t) \)),

\[
S_{aff}[q,p,J] = \int_{t_{in}}^{t_{out}} d\tau \left[ p_i \dot{q}^i - H_{aff}(q,p) + J_i q^i \right],
\]

which, subsequently gives the cerebellar path integral with the action potential source, coming either from the motor cortex or from other subcortical areas.

Note that the standard Wick rotation: \( t \mapsto -t \) (see [Kla97, Kla00]), makes our path integral real, i.e.,

\[
\int \mathcal{D}[w,q,p] e^{iS_{aff}[q,p]} \overset{\text{Wick}}{\longrightarrow} \int \mathcal{D}[w,q,p] e^{-S_{aff}[q,p]},
\]

while their subsequent discretization gives the standard thermodynamic partition function (see Appendix),

\[
Z = \sum_j e^{-w_j E^j/T}, \tag{10}
\]

where \( E^j \) is the energy eigenvalue corresponding to the affine Hamiltonian \( H_{aff}(q,p) \), \( T \) is the temperature–like environmental control parameter, and the sum runs over all energy eigenstates (labelled by the index \( j \)). From (10), we can further calculate all statistical and thermodynamic system properties (see [Fey72]), as for example, transition entropy \( S = k_B \ln Z \), etc.

### 3.3 Entropy and Motor Control

Our cerebellar path integral controller is closely related to entropic motor control [HN08a, HN08b], which deals with neuro-physiological feedback information and environmental uncertainty. The probabilistic nature of human motor action can be characterized by entropies at the level of the organism, task, and environment. Systematic changes in motor adaptation are characterized as task–organism and environment–organism tradeoffs in entropy. Such compensatory adaptations lead to a view of goal–directed motor control as the product of an underlying conservation of entropy across the task–organism–environment system. In particular, an experiment conducted in [HN08b] examined the changes in entropy of the coordination of isometric force output under different levels of task demands and feedback from the environment. The goal of the study was to examine the hypothesis that human motor adaptation can be characterized as a process of entropy conservation that is reflected in the compensation of entropy between the task, organism motor output,
and environment. Information entropy of the coordination dynamics relative phase of the motor output was made conditional on the idealized situation of human movement, for which the goal was always achieved. Conditional entropy of the motor output decreased as the error tolerance and feedback frequency were decreased. Thus, as the likelihood of meeting the task demands was decreased increased task entropy and/or the amount of information from the environment is reduced increased environmental entropy, the subjects of this experiment employed fewer coordination patterns in the force output to achieve the goal. The conservation of entropy supports the view that context dependent adaptations in human goal-directed action are guided fundamentally by natural law and provides a novel means of examining human motor behavior. This is fundamentally related to the Heisenberg uncertainty principle [II08b] and further supports the argument for the primacy of a probabilistic approach toward the study of biodynamic cognition systems.

The action–amplitude formalism represents a kind of a generalization of the Haken-Kelso-Bunz (HKB) model of self-organization in the individual’s motor system [HKB85, Kel95], including: multi-stability, phase transitions and hysteresis effects, presenting a contrary view to the purely feedback driven systems. HKB uses the concepts of synergetics (order parameters, control parameters, instability, etc) and the mathematical tools of nonlinearly coupled (nonlinear) dynamical systems to account for self-organized behavior both at the cooperative, coordinative level and at the level of the individual coordinating elements. The HKB model stands as a building block upon which numerous extensions and elaborations have been constructed. In particular, it has been possible to derive it from a realistic model of the cortical sheet in which neural areas undergo a reorganization that is mediated by intra- and inter-cortical connections. Also, the HKB model describes phase transitions ('switches') in coordinated human movement as follows: (i) when the agent begins in the anti-phase mode and speed of movement is increased, a spontaneous switch to symmetrical, in-phase movement occurs; (ii) this transition happens swiftly at a certain critical frequency; (iii) after the switch has occurred and the movement rate is now decreased the subject remains in the symmetrical mode, i.e. she does not switch back; and (iv) no such transitions occur if the subject begins with symmetrical, in-phase movements. The HKB dynamics of the order parameter relative phase as is given by a nonlinear first-order ODE:

\[ \dot{\phi} = (\alpha + 2\beta r^2) \sin \phi - \beta r^2 \sin 2\phi, \]

where \( \phi \) is the phase relation (that characterizes the observed patterns of behavior, changes abruptly at the transition and is only weakly dependent on parameters outside the phase transition), \( r \) is the oscillator amplitude, while \( \alpha, \beta \) are coupling parameters (from which the critical frequency where the phase transition occurs can be calculated).
4 Appendix

4.1 Houk’s Autogenetic Motor Servo

About three decades ago, James Houk pointed out in [Hou67, HSG70, Hou78, Hou79] that stretch and unloading reflexes were mediated by combined actions of several autogenetic neural pathways. In this context, “autogenetic” (or, autogenic) means that the stimulus excites receptors located in the same muscle that is the target of the reflex response. The most important of these muscle receptors are the primary and secondary endings in muscle spindles, sensitive to length change, and the Golgi tendon organs, sensitive to contractile force. The autogenetic circuits appear to function as servo-regulatory loops that convey continuously graded amounts of excitation and inhibition to the large (alpha) skeletomotor neurons. Small (gamma) fusimotor neurons innervate the contractile poles of muscle spindles and function to modulate spindle–receptor discharge. Houk’s term “motor servo” [Hou78] has been used to refer to this entire control system, summarized by the block diagram in Figure 4.

![Figure 4: Houk’s autogenetic motor servo.](image)

Prior to a study by Matthews [Mat69], it was widely assumed that secondary endings
belong to the mixed population of “flexor reflex afferents,” so called because their activation provokes the flexor reflex pattern – excitation of flexor motoneurons and inhibition of extensor motoneurons. Matthews’ results indicated that some category of muscle stretch receptor other than the primary ending provides important excitation to extensor muscles, and he argued forcefully that it must be the secondary ending.

The primary and secondary muscle spindle afferent fibers both arise from a specialized structure within the muscle, the muscle spindle, a fusiform structure 4–7 mm long and 80–200 \( \mu \) in diameter. The spindles are located deep within the muscle mass, scattered widely through the muscle body, and attached to the tendon, the endomysium or the perimysium, so as to be in parallel with the extrafusal or regular muscle fibers. Although spindles are scattered widely in muscles, they are not found throughout. Muscle spindle (see Figure ??) contains two types of intrafusal muscle fibers (intrafusal means inside the fusiform spindle): the nuclear bag fibers and the nuclear chain fibers. The nuclear bag fibers are thicker and longer than the nuclear chain fibers, and they receive their name from the accumulation of their nuclei in the expanded bag-like equatorial region—the nuclear bag. The nuclear chain fibers have no equatorial bulge; rather their nuclei are lined up in the equatorial region—the nuclear chain. A typical spindle contains two nuclear bag fibers and 4-5 nuclear chain fibers.

The pathways from primary and secondary endings are treated commonly by Houk in Figure [4], since both receptors are sensitive to muscle length and both provoke reflex excitation. However, primary endings show an additional sensitivity to the dynamic phase of length change, called dynamic responsiveness, and they also show a much–enhanced sensitivity to small changes in muscle length [Mat72].

The motor servo comprises three closed circuits (Figure 4), two neural feedback pathways, and one circuit representing the mechanical interaction between a muscle and its load. One of the feedback pathways, that from spindle receptors, conveys information concerning muscle length, and it follows that this loop will act to keep muscle length constant. The other feedback pathway, that from tendon organs, conveys information concerning muscle force, and it acts to keep force constant.

In general, it is physically impossible to maintain both muscle length and force constant when external loads vary; in this situation the action of the two feedback loops will oppose each other. For example, an increased load force will lengthen the muscle and cause muscular force to increase as the muscle is stretched out on its length-tension curve. The increased length will lead to excitation of motoneurons, whereas the increased force will lead to inhibition. It follows that the net regulatory action conveyed by skeletomotor output will depend on some relationship between force change and length change and on the strength of the feedback from muscle spindles and tendon organs. A simple mathematical derivation [NH76] demonstrates that the change in skeletomotor output, the error signal of the motor servo, Should be proportional to the difference between a regulated stiffness and the actual stiffness provided by the mechanical properties of the muscle, where stiff-
ness has the units of force change divided by length change. The regulated stiffness is determined by the ratio of the gain of length to force feedback.

It follows that the combination of spindle receptor and tendon organ feedback will tend to maintain the stiffness of the neuromuscular apparatus at some regulated level. If this level is high, due to a high gain of length feedback and a low gain of force feedback, one could simply forget about force feedback and treat muscle length as the regulated variable of the system. However, if the regulated level of stiffness is intermediate in value, i.e. not appreciably different from the average stiffness arising from muscle mechanical properties in the absence of reflex actions, one would conclude that stiffness, or its inverse, compliance, is the regulated property of the motor servo.

In this way, the autogenetic reflex motor servo provides the local, reflex feedback loops for individual muscular contractions. A voluntary contraction force $F$ of human skeletal muscle is reflexly excited (positive feedback $+F^{-1}$) by the responses of its spindle receptors to stretch and is reflexly inhibited (negative feedback $-F^{-1}$) by the responses of its Golgi tendon organs to contraction. Stretch and unloading reflexes are mediated by combined actions of several autogenetic neural pathways, forming the motor servo (see [II06a, II06b, II06c]).

In other words, branches of the afferent fibers also synapse with with interneurons that inhibit motor neurons controlling the antagonistic muscles – reciprocal inhibition. Consequently, the stretch stimulus causes the antagonists to relax so that they cannot resist the shortening of the stretched muscle caused by the main reflex arc. Similarly, firing of the Golgi tendon receptors causes inhibition of the muscle contracting too strong and simultaneous reciprocal activation of its antagonist.

4.2 Cerebellum and Muscular Synergy

The cerebellum is a brain region anatomically located at the bottom rear of the head (the hindbrain), directly above the brainstem, which is important for a number of subconscious and automatic motor functions, including motor learning. It processes information received from the motor cortex, as well as from proprioceptors and visual and equilibrium pathways, and gives ‘instructions’ to the motor cortex and other subcortical motor centers (like the basal nuclei), which result in proper balance and posture, as well as smooth, coordinated skeletal movements, like walking, running, jumping, driving, typing, playing the piano, etc. Patients with cerebellar dysfunction have problems with precise movements, such as walking and balance, and hand and arm movements. The cerebellum looks similar in all animals, from fish to mice to humans. This has been taken as evidence that it performs a common function, such as regulating motor learning and the timing of movements, in all animals. Studies of simple forms of motor learning in the vestibulo–ocular reflex and eye–blink conditioning are demonstrating that timing and amplitude of learned movements are encoded by the cerebellum.
When someone compares learning a new skill to learning how to ride a bike they imply that once mastered, the task seems imbedded in our brain forever. Well, imbedded in the cerebellum to be exact. This brain structure is the commander of coordinated movement and possibly even some forms of cognitive learning. Damage to this area leads to motor or movement difficulties.

A part of a human brain that is devoted to the sensory-motor control of human movement, that is motor coordination and learning, as well as equilibrium and posture, is the cerebellum (which in Latin means “little brain”). It performs integration of sensory perception and motor output. Many neural pathways link the cerebellum with the motor cortex, which sends information to the muscles causing them to move, and the spino-cerebellar tract, which provides proprioception, or feedback on the position of the body in space. The cerebellum integrates these pathways, using the constant feedback on body position to fine–tune motor movements [Ito84].

The human cerebellum has 7–14 million Purkinje cells. Each receives about 200,000 synapses, most onto dendritic spines. Granule cell axons form the parallel fibers. They make excitatory synapses onto Purkinje cell dendrites. Each parallel fibre synapses on about 200 Purkinje cells. They create a strip of excitation along the cerebellar folia.

Mossy fibers are one of two main sources of input to the cerebellar cortex (see Figure 5). A mossy fibre is an axon terminal that ends in a large, bulbous swelling. These mossy fibers enter the granule cell layer and synapse on the dendrites of granule cells; in fact the granule cells reach out with little ‘claws’ to grasp the terminals. The granule cells then send their axons up to the molecular layer, where they end in a T and run parallel to the surface. For this reason these axons are called parallel fibers. The parallel fibers synapse on the huge dendritic arrays of the Purkinje cells. However, the individual parallel fibers are not a strong drive to the Purkinje cells. The Purkinje cell dendrites fan out within a plane, like the splayed fingers of one hand. If we were to turn a Purkinje cell to the side, it would have almost no width at all. The parallel fibers run perpendicular to the Purkinje cells, so that they only make contact once as they pass through the dendrites.

Unless firing in bursts, parallel fibre EPSPs do not fire Purkinje cells. Parallel fibers provide excitation to all of the Purkinje cells they encounter. Thus, granule cell activity results in a strip of activated Purkinje cells.

Mossy fibers arise from the spinal cord and brainstem. They synapse onto granule cells and deep cerebellar nuclei. The Purkinje cell makes an inhibitory synapse (GABA) to the deep nuclei. Mossy fibre input goes to both cerebellar cortex and deep nuclei. When the Purkinje cell fires, it inhibits output from the deep nuclei.

The climbing fibre arises from the inferior olive. It makes about 300 excitatory synapses onto one Purkinje cell. This powerful input can fire the Purkinje cell.

The parallel fibre synapses are plastic—that is, they can be modified by experience. When parallel fibre activity and climbing fibre activity converge on the same Purkinje cell, the parallel fibre synapses become weaker (EPSPs are smaller). This is called long-term
Figure 5: Stereotypical ways throughout the cerebellum.

depression. Weakened parallel fibre synapses result in less Purkinje cell activity and less inhibition to the deep nuclei, resulting in facilitated deep nuclei output. Consequently, the mossy fibre collaterals control the deep nuclei.

The basket cell is activated by parallel fibers afferents. It makes inhibitory synapses onto Purkinje cells. It provides lateral inhibition to Purkinje cells. Basket cells inhibit Purkinje cells lateral to the active beam.

Golgi cells receive input from parallel fibers, mossy fibers, and climbing fibers. They inhibit granule cells. Golgi cells provide feedback inhibition to granule cells as well as feedforward inhibition to granule cells. Golgi cells create a brief burst of granule cell activity.

Although each parallel fibre touches each Purkinje cell only once, the thousands of parallel fibers working together can drive the Purkinje cells to fire like mad.

The second main type of input to the folium is the climbing fibre. The climbing fibers go straight to the Purkinje cell layer and snake up the Purkinje dendrites, like ivy climbing a trellis. Each climbing fibre associates with only one Purkinje cell, but when the climbing fibre fires, it provokes a large response in the Purkinje cell.

The Purkinje cell compares and processes the varying inputs it gets, and finally sends its own axons out through the white matter and down to the deep nuclei. Although the inhibitory Purkinje cells are the main output of the cerebellar cortex, the output from the
cerebellum as a whole comes from the deep nuclei. The three deep nuclei are responsible for sending excitatory output back to the thalamus, as well as to postural and vestibular centers.

There are a few other cell types in cerebellar cortex, which can all be lumped into the category of inhibitory interneuron. The Golgi cell is found among the granule cells. The stellate and basket cells live in the molecular layer. The basket cell (right) drops axon branches down into the Purkinje cell layer where the branches wrap around the cell bodies like baskets.

The cerebellum operates in 3’s: there are 3 highways leading in and out of the cerebellum, there are 3 main inputs, and there are 3 main outputs from 3 deep nuclei. They are:

The 3 highways are the peduncles. There are 3 pairs (see [Mol97] [Har97] [Mar98]):

1. The inferior cerebellar peduncle (restiform body) contains the dorsal spinocerebellar tract (DSCT) fibers. These fibers arise from cells in the ipsilateral Clarke’s column in the spinal cord (C8–L3). This peduncle contains the cuneo–cerebellar tract (CCT) fibers. These fibers arise from the ipsilateral accessory cuneate nucleus. The largest component of the inferior cerebellar peduncle consists of the olivo–cerebellar tract (OCT) fibers. These fibers arise from the contralateral inferior olive. Finally, vestibulo–cerebellar tract (VCT) fibers arise from cells in both the vestibular ganglion and the vestibular nuclei and pass in the inferior cerebellar peduncle to reach the cerebellum.

2. The middle cerebellar peduncle (brachium pontis) contains the pontocerebellar tract (PCT) fibers. These fibers arise from the contralateral pontine grey.

3. The superior cerebellar peduncle (brachium conjunctivum) is the primary efferent (out of the cerebellum) peduncle of the cerebellum. It contains fibers that arise from several deep cerebellar nuclei. These fibers pass ipsilaterally for a while and then cross at the level of the inferior colliculus to form the decussation of the superior cerebellar peduncle. These fibers then continue ipsilaterally to terminate in the red nucleus (’ruber–duber’) and the motor nuclei of the thalamus (VA, VL).

The 3 inputs are: mossy fibers from the spinocerebellar pathways, climbing fibers from the inferior olive, and more mossy fibers from the pons, which are carrying information from cerebral cortex (see Figure 6). The mossy fibers from the spinal cord have come up ipsilaterally, so they do not need to cross. The fibers coming down from cerebral cortex, however, do need to cross (as the cerebrum is concerned with the opposite side of the body, unlike the cerebellum). These fibers synapse in the pons (hence the huge block of fibers in the cerebral peduncles labelled ‘cortico–pontine’), cross, and enter the cerebellum as mossy fibers.
The 3 deep nuclei are the **fastigial**, **interposed**, and **dentate nuclei**. The fastigial nucleus is primarily concerned with balance, and sends information mainly to vestibular and reticular nuclei. The dentate and interposed nuclei are concerned more with voluntary movement, and send axons mainly to thalamus and the red nucleus.

The main function of the cerebellum as a motor controller is depicted in Figure 3. A coordinated movement is easy to recognize, but we know little about how it is achieved. In search of the neural basis of coordination, a model of spinocerebellar interactions was recently presented in [AG05], in which the structure-functional organizing principle is a division of the cerebellum into discrete micro–complexes. Each micro–complex is the recipient of a specific motor error signal, that is, a signal that conveys information about an inappropriate movement. These signals are encoded by spinal reflex circuits and conveyed to the cerebellar cortex through climbing fibre afferents. This organization reveals salient features of cerebellar information processing, but also highlights the importance of systems level analysis for a fuller understanding of the neural mechanisms that underlie behavior.

The authors of [AG05] reviewed anatomical and physiological foundations of cerebellar information processing. The cerebellum is crucial for the coordination of movement. The authors presented a model of the cerebellar paravermis, a region concerned with the control of voluntary limb movements through its interconnections with the spinal cord. They particularly focused on the olivo-cerebellar climbing fibre system.

Figure 6: Inputs and outputs of the cerebellum.
Climbing fibres are proposed to convey motor error signals (signals that convey information about inappropriate movements) related to elementary limb movements that result from the contraction of single muscles. The actual encoding of motor error signals is suggested to depend on sensorimotor transformations carried out by spinal modules that mediate nociceptive withdrawal reflexes.

The termination of the climbing fibre system in the cerebellar cortex subdivides the paravermis into distinct microzones. Functionally similar but spatially separate microzones converge onto a common group of cerebellar nuclear neurons. The processing units formed as a consequence are termed ‘multizonal micro-complexes’ (MZMCs), and are each related to a specific spinal reflex module.

The distributed nature of microzones that belong to a given MZMC is proposed to enable similar climbing fibre inputs to integrate with mossy fibre inputs that arise from different sources. Anatomical results consistent with this notion have been obtained.

Within an individual MZMC, the skin receptive fields of climbing fibres, mossy fibres and cerebellar cortical inhibitory interneurons appear to be similar. This indicates that the inhibitory receptive fields of Purkinje cells within a particular MZMC result from the activation of inhibitory interneurons by local granule cells.

On the other hand, the parallel fibre–mediated excitatory receptive fields of the Purkinje cells in the same MZMC differ from all of the other receptive fields, but are similar to those of mossy fibres in another MZMC. This indicates that the excitatory input to Purkinje cells in a given MZMC originates in non–local granule cells and is mediated over some distance by parallel fibres.

The output from individual MZMCs often involves two or three segments of the ipsilateral limb, indicative of control of multi–joint muscle synergies. The distal–most muscle in this synergy seems to have a roughly antagonistic action to the muscle associated with the climbing fibre input to the MZMC.

The model proposed in [AG05] indicates that the cerebellar paravermis system could provide the control of both single– and multi–joint movements. Agonist-antagonist activity associated with single–joint movements might be controlled within a particular MZMC, whereas coordination across multiple joints might be governed by interactions between MZMCs, mediated by parallel fibres.

Two main theories address the function of the cerebellum, both dealing with motor coordination. One claims that the cerebellum functions as a regulator of the “timing of movements.” This has emerged from studies of patients whose timed movements are disrupted [IKD88].

The second, “Tensor Network Theory” provides a mathematical model of transformation of sensory (covariant) space-time coordinates into motor (contravariant) coordinates by cerebellar neuronal networks [PL80] [PL82] [PL85].

Studies of motor learning in the vestibulo–ocular reflex and eye-blink conditioning demonstrate that the timing and amplitude of learned movements are encoded by the
cerebellum [BK04]. Many synaptic plasticity mechanisms have been found throughout the cerebellum. The Marr–Albus model mostly attributes motor learning to a single plasticity mechanism: the long-term depression of parallel fiber synapses. The Tensor Network Theory of sensory–motor transformations by the cerebellum has also been experimentally supported [GZ86].

### 4.3 Feynman’s Partition Function

Recall that in statistical mechanics, the so–called partition function $Z$ is a quantity that encodes the statistical properties of a system in thermodynamic equilibrium. It is a function of temperature and other parameters, such as the volume enclosing a gas. Other thermodynamic variables of the system, such as the total energy, free energy, entropy, and pressure, can be expressed in terms of the partition function or its derivatives.\footnote{There are actually several different types of partition functions, each corresponding to different types of statistical ensemble (or, equivalently, different types of free energy.) The canonical partition function applies to a canonical ensemble, in which the system is allowed to exchange heat with the environment at fixed temperature, volume, and number of particles. The grand canonical partition function applies to a grand canonical ensemble, in which the system can exchange both heat and particles with the environment, at fixed temperature, volume, and chemical potential. Other types of partition functions can be defined for different circumstances.}\footnote{A canonical ensemble is a statistical ensemble representing a probability distribution of microscopic states of the system. Its probability distribution is characterized by the proportion $p_i$ of members of the ensemble which exhibit a measurable macroscopic state $i$, where the proportion of microscopic states for each macroscopic state $i$ is given by the Boltzmann distribution,

$$p_i = \frac{1}{Z} e^{-E_i/(kT)} = e^{-(E_i - A)/(kT)},$$

where $E_i$ is the energy of state $i$. It can be shown that this is the distribution which is most likely, if each system in the ensemble can exchange energy with a heat bath, or alternatively with a large number of similar systems. In other words, it is the distribution which has maximum entropy for a given average energy $<E_i>$.}

The partition function of a canonical ensemble\footnote{The partition function of a canonical ensemble is defined as a sum

$$Z(\beta) = \sum_j e^{-\beta E_j},$$

where $\beta = 1/(k_B T)$ is the ‘inverse temperature’, where $T$ is an ordinary temperature and $k_B$ is the Boltzmann’s constant. However, as the position $x_i$ and momentum $p_i$ variables of an $i$th particle in a system can vary continuously, the set of microstates is actually uncountable. In this case, some form of coarse–graining procedure must be carried out, which essentially amounts to treating two mechanical states as the same microstate if the differences in their position and momentum variables are ‘small enough’. The partition function then takes the form of an integral. For instance, the partition function of a gas consisting of $N$ molecules is proportional to the $6N$–dimensional phase–space integral,

$$Z(\beta) \sim \int_{\mathbb{R}^{6N}} d^3 p_i d^3 x_i \exp[\beta H(p_i, x_i)],$$

where $H = H(p_i, x_i), \ (i = 1,...,N)$ is the classical Hamiltonian (total energy) function.} is defined as a sum

$$Z(\beta) = \sum_j e^{-\beta E_j},$$

where $\beta = 1/(k_B T)$ is the ‘inverse temperature’, where $T$ is an ordinary temperature and $k_B$ is the Boltzmann’s constant. However, as the position $x_i$ and momentum $p_i$ variables of an $i$th particle in a system can vary continuously, the set of microstates is actually uncountable. In this case, some form of coarse–graining procedure must be carried out, which essentially amounts to treating two mechanical states as the same microstate if the differences in their position and momentum variables are ‘small enough’. The partition function then takes the form of an integral. For instance, the partition function of a gas consisting of $N$ molecules is proportional to the $6N$–dimensional phase–space integral,
More generally, the so-called configuration integral, as used in probability theory, information science and dynamical systems, is an abstraction of the above definition of a partition function in statistical mechanics. It is a special case of a normalizing constant in probability theory, for the Boltzmann distribution. The partition function occurs in many problems of probability theory because, in situations where there is a natural symmetry, its associated probability measure, the Gibbs measure (see below), which generalizes the notion of the canonical ensemble, has the Markov property.

Given a set of random variables \( X_i \) taking on values \( x_i \), and purely potential Hamiltonian function \( H(x_i) \), \( (i = 1, ..., N) \), the partition function is defined as

\[
Z(\beta) = \sum_{x_i} \exp \left[ -\beta H(x_i) \right].
\]

The function \( H \) is understood to be a real-valued function on the space of states \( \{X_1, X_2, \ldots \} \) while \( \beta \) is a real-valued free parameter (conventionally, the inverse temperature). The sum over the \( x_i \) is understood to be a sum over all possible values that the random variable \( X_i \) may take. Thus, the sum is to be replaced by an integral when the \( X_i \) are continuous, rather than discrete. Thus, one writes

\[
Z(\beta) = \int dx_i \exp \left[ -\beta H(x_i) \right],
\]

for the case of continuously-varying random variables \( X_i \).

The Gibbs measure of a random variable \( X_i \) having the value \( x_i \) is defined as the probability density function

\[
P(X_i = x_i) = \frac{1}{Z(\beta)} \exp \left[ -\beta E(x_i) \right] = \frac{\exp \left[ -\beta H(x_i) \right]}{\sum_{x_i} \exp \left[ -\beta H(x_i) \right]},
\]

where \( E(x_i) = H(x_i) \) is the energy of the configuration \( x_i \). This probability, which is now properly normalized so that \( 0 \leq P(x_i) \leq 1 \), can be interpreted as a likelihood that a specific configuration of values \( x_i, (i = 1, 2, ..., N) \) occurs in the system.

As such, the partition function \( Z(\beta) \) can be understood to provide the Gibbs measure on the space of states, which is the unique statistical distribution that maximizes the entropy for a fixed expectation value of the energy,

\[
\langle H \rangle = -\frac{\partial \log(Z(\beta))}{\partial \beta}.
\]

The associated entropy is given by

\[
S = -\sum_{x_i} P(x_i) \ln P(x_i) = \beta \langle H \rangle + \log Z(\beta).
\]
The principle of maximum entropy related to the expectation value of the energy $\langle H \rangle$, is a postulate about a universal feature of any probability assignment on a given set of propositions (events, hypotheses, indices, etc.). Let some testable information about a probability distribution function be given. Consider the set of all trial probability distributions which encode this information. Then the probability distribution which maximizes the information entropy is the true probability distribution, with respect to the testable information prescribed.

Now, the number of variables $X_i$ need not be countable, in which case the set of coordinates $\{x_i\}$ becomes a field $\phi = \phi(x)$, so the sum is to be replaced by the *Euclidean path integral* (that is a Wick–rotated Feynman transition amplitude in imaginary time), as

$$Z(\phi) = \int \mathcal{D}[\phi] \exp \left[ -H(\phi) \right].$$

More generally, in quantum field theory, instead of the field Hamiltonian $H(\phi)$ we have the action $S(\phi)$ of the theory. Both Euclidean path integral,

$$Z(\phi) = \int \mathcal{D}[\phi] \exp \left[ -S(\phi) \right], \quad \text{real path integral in imaginary time} \quad (11)$$

and Lorentzian one,

$$Z(\phi) = \int \mathcal{D}[\phi] \exp \left[ iS(\phi) \right], \quad \text{complex path integral in real time,} \quad (12)$$

are usually called ‘partition functions’. While the Lorentzian path integral $\text{(12)}$ represents a quantum-field theory-generalization of the Schrödinger equation, the Euclidean path integral $\text{(11)}$ represents a statistical-field-theory generalization of the Fokker–Planck equation.

**References**

[AG05] Apps, R., Garwicz, M., Anatomical and physiological foundations of cerebellar information processing. Nature Rev. Neurosci. 6, 297–311, (2005)

[Alb71] Albus, J.S., A theory of cerebellar function. Mathematical Biosciences, 10, 25-61, (1971)

[BC81] Bloedel, J.R., Courville, J., Cerebellar afferent systems. In J. Brookhart, V. Mountcastle, V. Brooks, and S. Geiger, editors, Handbook of Physiology, Sect. 1. The Nervous System. Motor Control. American Physiological Society, Bethesda, MD, (1981)
[Hat78] Hatze, H., A general myocybernetic control model of skeletal muscle. Biol. Cyber., 28, 143–157, (1978)

[Heb49] Hebb, D.O., The Organization of Behavior, Wiley, New York, (1949)

[Hil38] Hill, A.V., The heat of shortening and the dynamic constants of muscle. Proc. Roy. Soc. B76, 136–195, (1938)

[Hou67] Houk, J.C., Feedback control of skeletal muscles. Brain Res., 5, 433–451, (1967)

[Hou78] Houk, J.C., Participation of reflex mechanisms and reaction-time processes in the compensatory adjustments to mechanical disturbances. Progr. Clin. Neurophysiol. 4, 193–215, (1978)

[Hou79] Houk, J.C., Regulation of stiffness by skeletomotor reflexes, Ann. Rev. Physiol. 41, 99–114, (1979)

[Hou79] Houk, J.C., Regulation of stiffness by skeletomotor reflexes. Ann. Rev. Physiol., 41, 99–114, (1979)

[IA07] Ivancevic V., Aidman E., Life-space foam: A medium for motivational and cognitive dynamics. Physica A 382, 616–630, (2007)

[IAY08] Ivancevic V., Aidman E., Yen L., Extending Feynman’s Formalisms for Modelling Human Joint Action Coordination. Int. J. Biomath. (to appear)

[IB05] Ivancevic, V., Beagley, N., Brain–like functor control machine for general humanoid biodynamics. Int. J. Math. Math. Sci. 11, 1759–1779, (2005)

[II06a] Ivancevic, V., Ivancevic, T., Human–Like Biomechanics. Springer, (2006)

[II06b] Ivancevic, V., Ivancevic, T., Natural Biodynamics. World Scientific, (2006)

[II06c] Ivancevic, V., Ivancevic, T., Geometrical Dynamics of Complex Systems. Springer, (2006)

[II07a] Ivancevic, V., Ivancevic, T., Neuro–Fuzzy Associative Machinery for Comprehensive Brain and Cognition Modelling. Springer, Berlin, (2007)

[II07b] Ivancevic, V., Ivancevic, T., Computational Mind: A Complex Dynamics Perspective. Springer, Berlin, (2007)

[II07c] Ivancevic, V., Ivancevic, T., Applied Differential Geometry: A Modern Introduction. World Scientific, Singapore, (2007)
[II08a] Ivancevic, V., Ivancevic, T., Complex Nonlinearity: Chaos, Phase Transitions, Topology Change and Path Integrals, Springer, (2008)

[II08b] Ivancevic, V., Ivancevic, T., Quantum Leap: From Dirac and Feynman Across the Universe to Human Body and Mind. World Scientific, Singapore, (2008)

[II08c] Ivancevic, V., Ivancevic, T., Nonlinear Quantum Psychodynamics with Topological Phase Transitions. NQJ (to appear)

[IKD88] Ivry, R.B., Keele, S.W., Diener, H.C., Dissociation of the lateral and medial cerebellum in movement timing and movement execution, Exp. Brain. Res. 73(1), 167–80, (1988)

[IS01] Ivancevic, V., Snoswell, M., Fuzzy–stochastic functor machine for general humanoid–robot dynamics. IEEE Trans. SMCB, 31(3), 319–330, (2001)

[Ito84] Ito, M., Cerebellum and Neural Control. Raven Press, New York, (1984)

[Ito90] Ito, M., A new physiological concept on cerebellum. Rev. Neurol, (1990)

[Iva02] Ivancevic, V., Generalized Hamiltonian biodynamics and topology invariants of humanoid robots. IJMMS, 31(9), 555–565, (2002)

[Iva04] Ivancevic, V., Symplectic Rotational Geometry in Human Biomechanics. SIAM Rev. 46(3), 455–474, (2004)

[Iva06] Ivancevic, V., Lie–Lagrangian model for realistic human bio-dynamics. IJHR 3(2), 205–218, (2006)

[Kel95] Kelso, J.A.S., Dynamic Patterns: The Self Organization of Brain and Behavior. MIT Press, Cambridge, MA, (1995)

[Kla00] Klauder, J.R., Beyond Conventional Quantization, Cambridge Univ. Press, Cambridge, (2000)

[Kla97] Klauder, J.R., Understanding Quantization. Found. Phys. 27, 1467–1483, (1997)

[MA02] Mascalchi, M. et al.: Proton MR Spectroscopy of the Cerebellum and Pons in Patients with Degenerative Ataxia, Radiology, 223, 371, (2002)

[MGK92] Miller, W.T., Glanz, F.H., Kraft, L.G., CMAC: An associative neural network alternative to backpropagation. In C. Lau, editor, Neural Networka. Theoretical Foundations and Analysis, pages 233-240. IEEE Press, New York, (1992)

[Mar98] Marieb, E.N.: Human Anatomy and Physiology. (4th ed.), Benjamin/Cummings, Menlo Park, CA, (1998)
Matthews, P.B.C., Evidence that the secondary as well as the primary endings of the muscle spindles may be responsible for the tonic stretch-reflex of the decerebrate cat. J. Physiol. London 204, 365–93, (1969)

Matthews, P.B.C., Mammalian Muscle Receptors and Their Central Action, Williams & Wilkins, Baltimore, (1972)

Molavi, D.W., Neuroscience Tutorial, School of Medicine, Washington Univ. (1997)

Nichols, T.R., Houk, J.C., The improvement in linearity and the regulation of stiffness that results from the actions of the stretch–reflex. J. Neurophysiol. 39, 119–142, (1976)

Nijmeijer, H., van der Schaft, A.J., Nonlinear Dynamical Control Systems. Springer, New York, (1990)

Park, J., Chung, W.-K., Geometric Integration on Euclidean Group With Application to Articulated Multibody Systems. IEEE Trans. Rob. 21(5), 850–863, (2005)

Pellionisz, A., Llinas, R., Tensorial approach to the geometry of brain function: cerebellar coordination via a metric tensor. Neurosci. 5, 1125–1136, (1980)

Pellionisz, A., Llinas, R., Space-time representation in the brain. The cerebellum as a predictive space-time metric tensor. Neurosci. 7(12), 2949–2970, (1982)

Pellionisz, A., Llinas, R., Tensor network theory of the meta-organization of functional geometries in the central nervous system. Neurosci. 16(2), 245–273, (1985)

Schaal, S., Atkeson, C.G., Constructive incremental learning from only local information. Neural Comput., 10, 2047–2084, (1998)

Sutton, R.S., Barto, A.G., Reinforcement Learning: An Introduction. MIT Press, Cambridge, MA, (1998)

Schaal, S., Robot learning. In M. Arbib (ed.) Handbook of Brain Theory and Neural Networks (2nd ed.), MIT Press, Cambridge, (1998)

Schaal, S., Is imitation learning the route to humanoid robots?. Trends Cogn. Sci., 3, 233–242, (1999)

P. van der Smagt, Cerebellar control of robot arms. Conn. Sci. 10, 301–320, (1998)
[TGK92] Thach, W.T., Goodkin, H., Keating, J., The cerebellum and the adaptive coordination of movement. Ann. Rev. Neurosci. 403–442, (1992)

[VBS90] Vukobratovic, M., Borovac, B., Surla, D., Stokic, D., Biped Locomotion: Dynamics, Stability, Control, and Applications. Springer, Berlin, (1990)

[Wil56] Wilkie, D.R., The mechanical properties of muscle. Brit. Med. Bull. 12, 177–182, (1956)