Multiplicative Synaptic Normalisation and a Non-Linear Hebb Rule Underlie a Neurotrophic Model of Competitive Synaptic Plasticity

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Abstract

Synaptic normalisation is used to enforce competitive dynamics in many models of developmental synaptic plasticity. In linear and semi-linear Hebbian models, multiplicative synaptic normalisation fails to segregate afferents whose activity patterns are positively correlated. To achieve this, the biologically problematic device of subtractive synaptic normalisation must be used instead. Our own model of competition for neurotrophic support, which can segregate positively correlated afferents, was developed in part in an attempt to overcome these problems by removing the need for synaptic normalisation altogether. However, we now show that the dynamics of our model decompose into two decoupled subspaces, with competitive dynamics being implemented in one of them through a non-linear Hebb rule and multiplicative synaptic normalisation. This normalisation is “emergent” rather than imposed. We argue that these observations permit biologically plausible forms of synaptic normalisation to be viewed as abstract and general descriptions of the underlying biology in certain, scaleless models of synaptic plasticity.
1 Introduction

Activity-dependent competition between afferent neurons for control of target neurons is a ubiquitous feature of mammalian neuronal development (Purves, 1994). These competitive interactions are thought to lead, for example, to the development of ocular dominance columns (ODCs) — inter-digitated domains of control by the left and right eyes — in the primary visual cortex of higher mammals such as Old World monkeys and cats (Hubel & Wiesel, 1962; LeVay et al., 1978, 1980). Understanding the mechanisms underlying competition in the nervous system is of central importance to developmental neuroscience, both experimentally and theoretically.

Theoretically, several approaches to competition have been developed (for reviews, see Swindale, 1996; van Ooyen, 2001), including the Bienenstock-Cooper-Munro model (Bienenstock et al., 1982), neurotrophic models (e.g., Harris et al., 1997; Elliott & Shadbolt, 1998a,b), covariance models (Sejnowski, 1977; Linsker, 1986a,b,c), and various other approaches (e.g., Swindale, 1980; Fraser & Perkel, 1989; Montague et al., 1991; Tanaka, 1991). But perhaps the most popular models are based on linear or semi-linear Hebbian rules coupled with various forms of synaptic normalisation as a means of enforcing competitive dynamics (von der Malsburg, 1973; Miller et al., 1989; Goodhill, 1993). Although multiplicative synaptic normalisation (von der Malsburg, 1973) is not biologically implausible, it does not lead to afferent segregation in the presence of positive correlations in the activity patterns between afferent cells. To segregate afferents in the presence of positive correlations, subtractive synaptic normalisation must be used instead (Goodhill & Barrow, 1994; Miller & MacKay, 1994).

Whether or not models actually need to be able to segregate positively-
correlated afferents in order to be biologically relevant is currently a moot ques-
tion. ODCs in Old World monkeys develop prior to birth and are adult-like at
birth (Horton & Hocking, 1996). Recent data has also questioned whether, as
previously assumed, ODCs develop in the ferret after eye-opening (Crowley &
Katz, 1999, 2000). In the cat, however, despite data revealing non-Hebbian de-
velopmental processes (Crair et al., 1998), it is still tenable to assume that ODCs
develop after eye-opening, and therefore in the presence of positively correlated
inter-ocular images. Thus, models of at least cat ODC development must be able
to segregate positively correlated afferents in a plausible manner.

In previous work, we have criticised the use of synaptic normalisation on two
grounds. First, we argued (Elliott & Shadbolt, 1998a,b, 1999; Elliott et al.,
2001) that synaptic normalisation simply describes rather than seeks to explain
the nature of competition in the nervous system. Second, we have argued (El-
liott & Shadbolt, 1998a,b, 1999; Elliott et al., 2001) that subtractive synaptic
normalisation is biologically implausible, for reasons explained later. Inspired
by experimental results implicating neurotrophic factors in activity-dependent
synaptic competition (reviewed in McAllister et al., 1999), and in an attempt
to overcome the difficulties of synaptic normalisation, we have built a mathe-
matical model based on activity-dependent competition for neurotrophic factors
involving anatomical plasticity, and later extended the model to include simul-
taneous physiological plasticity (Elliott & Shadbolt, 1998a,b, 1999; Elliott et al.,
2001). Critically, our neurotrophic model segregates positively correlated affer-
ents, as required for application to the development of ODCs (Elliott & Shadbolt,
1998b).

In this paper, we present the results of further analysis of our neurotrophic
model. We have found that the model’s dynamics decouple into two essentially
independent subspaces, with the competitive dynamics residing exclusively in one
subspace. In this latter subspace, we show that a non-linear Hebb (or Hebb-like)
rule governs synaptic growth. To our surprise, and contradicting widely-held
beliefs about the capacity of multiplicative synaptic normalisation to segregate
positively correlated afferents, we find that competition is implemented through
multiplicative synaptic normalisation. This normalisation, however, is not im-
posed, but, in a sense that we shall explain, is “emergent”. We then argue that
the key feature of our model that allows this Hebb-like, synaptic normalisation
description is that the dynamics in the competitive subspace are independent
of an overall synaptic scale (i.e., the absolute number of synapses). We suggest
that many such models may satisfy this property, thus perhaps sanctioning the
use of synaptic normalisation as an acceptable, abstract characterisation of the
underlying competitive process.

2 Reformulations of the Model

In this section we first write down our basic model of anatomical, competitive
developmental synaptic plasticity based on competition for neurotrophic support,
and then state a number of key results that, in the non-linear Hebb formula-
tion, will be seen to be rather more transparent. We then introduce a change of
variables, from which an energy function reformulation of the model will reveal
two basically decoupled sets of dynamics: one competitive set leading to afferent
segregation; and one non-competitive set determining an overall synaptic scale.
Finally, we derive the non-linear Hebb rule formulation, using the decoupled dy-
namics of the energy function formulation to extract an “emergent” normalisation
process.
2.1 The Basic Model

Let afferent cells be labelled by letters such as \( i \) and \( j \), and target cells be labelled by letters such as \( x \) and \( y \). Let the number of synapses between afferent cell \( i \) and target cell \( x \) be \( s_{xi} \), and let the activity of afferent cell \( i \) be \( a_i \in [0,1] \). Then the basic equation governing the time-evolution of \( s_{xi} \) is given by

\[
\frac{ds_{xi}}{dt} = \epsilon s_{xi} \left[ \frac{(a + a_i)}{\sum_j s_{xj}(a + a_j)} \sum_y \Delta_{xy} \left( T_0 + T_1 \frac{\sum_j s_{yj}a_j}{\sum_j s_{yj}} \right) - 1 \right];
\]

see Elliott & Shadbolt (1998a) for a detailed derivation and justification. The quantities \( T_0 \) and \( T_1 \) represent, respectively, an activity-independent and maximum activity-dependent release of NTFs by target cells; \( a \) represents a resting uptake term of NTFs by afferents; and \( \epsilon \) is an overall “learning rate”. The function \( \Delta_{xy} \) embodies lateral interactions between target cells \( x \) and \( y \). In previous work, we have considered \( \Delta_{xy} \) to arise only through the diffusion of NTFs between target cells, so that \( \Delta_{xy} \geq 0 \forall x, y \) (Elliott & Shadbolt, 1998a,b, 1999). However, we can also consider \( \Delta_{xy} \) to arise from both excitatory \((\Delta_{xy} > 0)\) and inhibitory \((\Delta_{xy} < 0)\) lateral synaptic interactions between target cells, either enhancing or reducing the release of NTFs by target cells. In this case, we can ignore NTF receptor dynamics, which in previous work we have also considered (Elliott & Shadbolt, 1998a). For convenience, and without much loss of generality, we will assume that \( \sum_y \Delta_{xy} = 1 \forall x \).

The quantity \( c = T_0/(aT_1) \) is a critical parameter in our model. Previous work has shown that, for \( \Delta_{xy} = \delta_{xy} \) (the Kronecker delta), when \( c < 1 \) afferent segregation occurs (that is, all but one of \( s_{xi} \) go to zero at each target cell \( x \)), while for \( c > 1 \), afferent segregation breaks down (Elliott & Shadbolt, 1998a). For \( c < 1 \), segregation occurs for all but perfectly correlated afferent activity patterns (Elliott & Shadbolt, 1998a). In the presence of a general \( \Delta_{xy} \), the
critical value of $c$ is reduced below unity and also becomes a function of afferent correlations, so that too-extensive (positive) lateral interactions or too strong (but not perfect) afferent activity correlations can lead to a breakdown of afferent segregation (unpublished observations). The parameter $a$ also plays a direct role in segregation. As $a \to \infty$, the rate of segregation tends to zero. In the limit, the ratio of the number of synapses supported by any pair of afferents on a given target cell remains fixed (Elliott & Shadbolt, 1998a).

2.2 Energy function formulation

Introducing the new variables $s^+_x = \sum_i s_{xi}$ and $v_{xi} = s_{xi}/s^+_x$, so that $\sum_i v_{xi} = 1$, Eq. (1) can be rewritten as

$$s^+_x \frac{dv_{xi}}{dt} = \epsilon T_1 v_{xi} \left[ \frac{\sum_j (a_i - a_j) v_{xj}}{\sum_j (a + a_j) v_{xj}} \right] \sum_{yj} \Delta_{xy} (ac + a_j) v_{yj}$$

(2)

and

$$\frac{ds^+_x}{dt} + \epsilon s^+_x = \epsilon T_1 \sum_{yj} \Delta_{xy} (ac + a_j) v_{yj}.$$  

(3)

We now average over the ensemble of afferent activity patterns. To achieve this we assume, for tractability, that for $n$ distinct afferents, $i = 1, \ldots, n$, there are just $n$ distinct activity patterns, with pattern number $i$ being defined by $a_i = 1$ and $a_j = p \forall j \neq i$, with $p \in [0, 1]$. Defining $\mu$ as the average activity of an afferent, so that $n\mu = 1 + (n - 1)p$, and introducing the parameter $r = (1 - p)/(a + p)$, we obtain after some algebra

$$ns^+_x \frac{dv_{xi}}{dt} = \epsilon T_1 v_{xi} \left[ a(c - 1) \left( \sum_j \frac{1 + r\delta_{ij}}{1 + rv_{xj}} - n \right) + (1 - p) \sum_j \frac{1 + r\delta_{ij}}{1 + rv_{xj}} \sum_y \Delta_{xy} (v_{yj} - v_{xj}) \right],$$

(4)

and

$$\frac{ds^+_x}{dt} + \epsilon s^+_x = \epsilon T_1 (ac + \mu),$$

(5)
where, in these two equations, we have dropped for notational convenience the \( \langle \rangle \)
brackets on the variables \( v_{xi} \) and \( s_x^+ \), these brackets denoting ensemble-averaging.
Restricting to \( n = 2 \) afferents for increased tractability and writing \( v_x = 2v_{xi} - 1 \)
for any one of the two afferents \( i \), we then obtain

\[
\frac{dv_x}{dt} = \epsilon T_1 r^2 \frac{1 - v_x^2}{(2 + r)^2 - r^2 v_x^2} \sum_y \Delta_{xy} v_y, \tag{6}
\]

\[
\frac{ds_x^+}{dt} = \epsilon \left[ T_1 (ac + \mu) - s_x^+ \right], \tag{7}
\]

where

\[
\Delta_{xy} = (a + \mu) \Delta_{xy} - (ac + \mu) \delta_{xy}. \tag{8}
\]

From Eqs. (6) and (7) we easily obtain a Lyapunov or energy function \( E \), such
that \( dE/dt \leq 0 \) always, where

\[
E = E_S + E_C, \tag{9}
\]

with

\[
E_S = \frac{1}{2} \sum_x \left[ T_1 (ac + \mu) - s_x^+ \right]^2 \tag{10}
\]

and

\[
E_C = -\frac{1}{2} \sum_{xy} v_x \Delta_{xy} v_y. \tag{11}
\]

In fact, this form for \( E \) generalises rigorously to \( n > 2 \) afferents, as we shall
demonstrate elsewhere.

The stable solutions of Eqs. (6) and (7), and thus the solutions of the unaveraged Eq. (1) when \( \epsilon \) is sufficiently small that the \( s_{xi} \) change slowly compared to
the afferent activities \( a_i \), correspond to the minima of \( E \). \( E \) cleanly decomposes
into two pieces that may be minimised independently. \( E_S \) merely sets the overall
scale for \( s_x^+ \) and is minimised directly by setting \( s_x^+ = T_1 (ac + \mu) \forall x \). The dynamics embodied in this minimisation [and therefore the solutions of Eqs. (5) or (7)]

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are therefore trivial and uninteresting. Minimisation of $E_C$, on the other hand, with $v_x \in [-1, 1]$ corresponding to a target cell “spin” variable denoting control by one afferent (negative $v_x$) or the other (positive $v_x$), encapsulates the competitive dynamics of the model. The eigenvalues of the matrix $\hat{\Delta}$ entirely determine the character of these dynamics, and $E_C$ is, of course, exactly the Hamiltonian of a spin glass (with $v_x \in \{-1, +1\}$).

2.3 Non-linear Hebb rule formulation

We now return to $n$ afferents in the unaveraged system defined by Eqs. (2) and (3), and restrict to a consideration of one target cell only, so that we examine competition on a pointwise basis. We then may set $\Delta_{xy} = \delta_{xy}$ and drop the $x$ subscript on the $s_x^+$ and $v_{xi}$ (and $s_{xi}$) variables. Our results, however, easily generalise to multiple target cells with a general $\Delta_{xy}$.

The energy function analysis above reveals that the $n$ independent degrees of freedom in the $n$ $s_i$ variables decompose into $n - 1$ degrees of freedom in the $n$ scaleless $v_i$ variables ($\sum_i v_i = 1$, by construction) that entirely capture the competitive dynamics of the model, and one degree of freedom in the $s^+$ variable that entirely captures the scaling dynamics of the model. As argued above, these latter scaling dynamics are quite trivial and uninteresting and we may discard them without any important loss of generality, restricting attention to the $(n - 1)$-dimensional competitive subspace only. Hence, our basic equation is just

$$\frac{dv_i}{dt} = v_i \left( \frac{a c + \sum_j v_j a_j}{a + \sum_j v_j a_j} \right) \sum_j (a_i - a_j)v_j$$  \hspace{1cm} (12)

with $\sum_i v_i = 1$, where we have absorbed a factor of $\epsilon T_1$ into a redefinition of time. In this subspace, we have a synaptic growth rule, Eq. (12), which automatically normalises the $v_i$ such that $\sum_i v_i = 1$ always. We stress, however, that this
normalisation is the result of the definition of the \( v_i \) variables \( (v_i = s_i/s^+) \) and the fact that these variables are key in capturing the competitive dynamics, with the scaling dynamics decoupling.

Nevertheless, from a purely mathematical point of view, we may ask what is the form of the growth rule underlying Eq. (12), and how is the “emergent” normalisation \( \sum_i v_i = 1 \) maintained? Defining

\[
\Pi = \frac{ac + \sum_j v_j a_j}{a + \sum_j v_j a_j},
\]

which is a purely post-synaptic, although non-linear term, and

\[
\pi_i = a_i v_i,
\]

which is a pre-synaptic term, we may rewrite Eq. (12) as

\[
\frac{dv_i}{dt} = \pi_i \Pi - v_i (\sum_j \pi_j) \Pi.
\]

The first term on the right-hand-side (RHS) is a non-linear Hebb growth rule, where, for the moment, we define a Hebb growth rule as any synaptic growth rule that is expressible as the product of a pre-synaptic term and a purely post-synaptic term. Were this the only term on the RHS of Eq. (15), it would induce the unconstrained synaptic growth characteristic of Hebb rules. How, then, is this unconstrained growth forced to remain in the \((n - 1)\)-dimensional subspace in which \( \sum_i v_i = 1 \)? Were we to impose this through multiplicative synaptic normalisation, we would modify the Hebb rule by subtracting from the unconstrained growth term a term proportional to \( v_i \) and such that this additional term forces \( \sum_i dv_i/dt = 0 \). In our case, this term would be \( v_i (\sum_j \pi_j) \Pi \), which is exactly the second term on the RHS of Eq. (15).

Eq. (15), therefore, represents a non-linear Hebb rule, with a non-linear post-synaptic term \( \Pi \), together with multiplicative synaptic normalisation. Yet, this
model segregates afferents for all but perfectly correlated afferent activity patterns (when $\Delta_{xy} = \delta_{xy}$), explicitly contradicting the widely-held view that, in order to segregate positively correlated afferents, multiplicative normalisation fails and subtractive normalisation must be used (Goodhill & Barrow, 1994; Miller & MacKay, 1994). Furthermore, it contradicts, or at least dramatically reduces the significance of claims that non-linear Hebbian dynamics are reducible to linear Hebbian dynamics (Miller, 1990), for linear dynamics will not segregate positively correlated afferents under multiplicative normalisation (Goodhill & Barrow, 1994; Miller & MacKay, 1994). We discuss these issues more fully later.

The form of $\Pi$ allows us to see more readily the model’s behaviour in certain limits. When $c = 1$, we have that $\Pi \equiv 1$. In this case, Eq. (15) is purely pre-synaptic and so we would expect, as in fact observed (Elliott & Shadbolt, 1998a), chaotic oscillations in the $v_i$ as they grow or decay independently of each other (except through $\sum_i v_i = 1$). In the limit $a \to \infty$ with $ac$ held fixed (because $ac = T_0/T_1$, $T_0$ and $T_1$ being parameters independent of $a$), $\Pi \to 0$. Hence, as $a$ grows the $v_i$ evolve more slowly, so that segregation slows down, and, in the limit, the $v_i$ are constant in time.

When $c < 1$, $\Pi < 1$ and is a monotone increasing function of $\sum_j v_j a_j$ on $[0, \infty)$ (although the actual range of this sum is just $[0, 1]$), while for $c > 1$, $\Pi > 1$ and is a monotone decreasing function of $\sum_j v_j a_j$. $\Pi$ should therefore not in general be regarded as a post-synaptic firing rate, but rather as a post-synaptic “plasticity rate”, which, of course, depends on the summed post-synaptic response, $\sum_j v_j a_j$. While we have defined a Hebb rule as any synaptic growth rule that is expressible as the product of a pre-synaptic and a post-synaptic term, many Hebb rules are often stated in more restricted forms involving only correlations between pre-synaptic and post-synaptic activity. For $c < 1$, because
Π is a monotone increasing function of $\sum_j v_j a_j$, our Hebb rule definition for our model is equivalent to the more restricted definition. Hence, for $c < 1$, Eq. (15) is Hebbian in both senses, and because our model segregates afferents only for $c < 1$, this justifies our use of our more general definition of a Hebbian model. However, for $c > 1$, Π is monotone decreasing. Therefore, the growth rule in Eq. (15), although Hebbian by our definition, is not Hebbian according to the more restricted definition. Indeed, for $c > 1$, Eq. (15) would often be regarded as an anti-Hebbian rule, rewarding anti-correlations between pre-synaptic and post-synaptic activity. We thus see the dynamical importance of the point $c = 1$: it corresponds to the transition in the model between a (classical) Hebbian rule and a (classical) anti-Hebbian rule.

3 Discussion

We have extended our earlier analysis of our neurotrophic model of anatomical, competitive synaptic plasticity, which can segregate afferents in the presence of positively correlated afferent activity patterns, and shown that a change of variables reveals two essentially decoupled sets of dynamics. One set determines an overall synaptic scale, and can be discarded without any important loss of generality. The other set completely determines the competitive dynamics underlying the model, independent of the synaptic scale. These competitive dynamics can be viewed in two contrasting ways, either as the dynamics of a spin glass, which we have not pursued at any length here, or they can be formulated precisely as a non-linear Hebbian model with synaptic normalisation implemented multiplicatively (cf. Wiskott & Sejnowski, 1998). In contrast to many other models of synaptic competition, this normalisation is not imposed, but rather is “emergent”, in the sense that the interesting, competitive interactions of the model restrict them-
selves dynamically to a lower dimensional subspace in which a set of variables can be found that fully capture these dynamics and that satisfy a normalisation constraint. Critically, even though synaptic normalisation is implemented multiplicatively in this lower dimensional subspace, our model can segregate positively correlated afferents. Indeed, in the absence of lateral interactions between target cells (so that \( \Delta_{xy} = \delta_{xy} \)), the model can segregate all but perfectly correlated afferents.

The fact that a non-linear Hebb rule and multiplicative synaptic normalisation can segregate even strongly positively correlated afferents is intriguing. It is well-known that multiplicative normalisation together with a linear Hebb rule, or a linear Hebb rule coupled with non-linearities such as a winner-take-all mechanism, cannot segregate positively correlated afferents (von der Malsburg, 1973; Goodhill & Barrow, 1994; Miller & MacKay, 1994) and that subtractive rather than multiplicative normalisation must be used (Miller et al., 1989; Goodhill, 1993). The fact that certain non-linear Hebbian models are reducible to linear Hebbian models (Miller, 1990) has led to the widespread belief that, in general, no Hebbian model, whether linear or non-linear, can segregate positively correlated afferents under multiplicative normalisation. Our results constitute an explicit and constructive counter-example to these beliefs.

We have frequently criticised synaptic normalisation for being a mathematical device that simply imposes rather than seeks to illuminate synaptic competition (Elliott & Shadbolt, 1998a,b, 1999; Elliott et al., 2001). Even if we accept that synaptic normalisation underlies competition in the nervous system, and accepting that normalisation could arise from decay or homeostatic mechanisms controlling synaptic efficacy (Turrigiano et al., 1998), it appears to us implausible to assume, as required by subtractive normalisation, that these mechanisms should
regulate synaptic efficacy in a fashion that is independent of the concentrations of any of the pre-synaptic or post-synaptic components that, together, determine synaptic efficacy. If a model of synaptic plasticity requires an arguably implausible form of synaptic normalisation to segregate positively correlated afferents, then perhaps that model should be regarded as implausible.

Prior to our results above, such a conclusion would have been unpalatable, as it would have left a vacuum in the space of conventional competitive, synaptic normalisation-based Hebbian models that can segregate positively correlated afferents. A few other models will segregate positively correlated afferents (e.g., Bienenstock et al., 1982; Harris et al., 1997), but these do not enforce hard constraints via synaptic normalisation on summed synaptic efficacy. Our present model, in its non-linear Hebb reformulation, fills this vacuum by being an explicit, constructive example of a simple Hebbian model that uses multiplicative normalisation to achieve competitive dynamics. This model is almost certainly not unique: presumably an infinite number of non-linear Hebbian models exist that are capable of segregating positively correlated afferents using various forms of synaptic normalisation that do not require biologically problematic assumptions. Our model should therefore be construed, from a mathematical point of view, as merely an existence proof of one such model.

How do we reconcile our previous criticisms of synaptic normalisation (of any form) as a mathematical device, and our presentation of our neurotrophic model as a competing alternative, with the results above, showing an underlying multiplicative synaptic normalisation in the competitive dynamics of our model? There are two related aspects to our reply.

First, the synaptic normalisation that we have found was in no way imposed from the outset. After changing variables so as to eliminate an overall synap-
tic scale, we found that the competitive dynamics reside entirely in the scaleless variables (the $v_{xi}$), which by definition satisfy a normalisation equation, and, moreover, the dynamics of the scaleless variables are basically independent of the dynamics of the scaling variables (the $s_x^+$); at least, the coupling is completely trivial and uninteresting and can be ignored without any important loss of generality. In this sense, then, we have referred to this normalisation as “emergent”.

Second, mathematically, it will almost always be possible to perform a change of variables and thereby introduce a set of scaleless variables that satisfy a normalisation equation. The only case in which this may not be possible is when the transformation would introduce possibly singular variables, but we shall ignore this possibility. Whether or not the scaling and scaleless dynamics decouple, it will also always be possible to ask whether the scaleless dynamics can be mathematically separated into a general growth term (a Hebb or Hebb-like term, for example) and a term that maintains the normalisation equation (the normalisation term that sets the derivative of the summed scaleless variables to zero). If the competitive dynamics reside entirely in the scaleless variables, and if the scaleless and scaling variables do not interact in any important fashion, then any such model will possess an underlying synaptic growth rule with competition implemented by some form of synaptic normalisation. Given that neurons exhibit such properties as homeostasis and gain control, which can be thought of as mechanisms to eliminate dependence on, or to adjust to, synaptic scale, it is possible that many models of synaptic plasticity can so be reformulated.

On this view, in those classes of model of synaptic plasticity in which the underlying competitive dynamics are scale-independent, “emergent” synaptic normalisation is seen to be inevitable, fully capturing, mathematically-speaking, the competitive dynamics. Thus, when rooted in a biologically-plausible model of
synaptic plasticity, synaptic normalisation, provided that its emergent form is not implausible, would appear to be an acceptable, abstract and general description of the underlying biology. Can we reverse this statement and argue, therefore, that we may impose competitive dynamics on any given synaptic growth rule by enforcing a hard normalisation constraint? The answer is probably in the affirmative, provided that such a model is regarded as tentative, awaiting a derivation from an underlying model whose scaleless dynamics correspond to the given synaptic growth rule. Of course, if it is found that the synaptic growth rule requires an implausible form of synaptic normalisation to achieve afferent segregation in the presence of positive correlations, as we believe that linear and semi-linear Hebb rules do, then that particular synaptic growth rule should be discarded.

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