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REPLICA MEAN FIELD LIMITS FOR NEURAL NETWORKS WITH EXCITATORY AND INHIBITORY ACTIVITY

BY

IOANNIS PAPAGEORGIOU (SÃO PAULO)

Abstract. We study Replica Mean Field limits for a neural system of infinitely many neurons with both inhibitory and excitatory interactions. As a result we obtain an analytical characterization of the invariant state. In particular, we focus on the Galves–Löcherbach model with interactions beyond the Property of Uniform Summability.

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1. INTRODUCTION

We study infinitely large neural networks. The activity of each neuron is described by the evolution of its membrane potential (see [60]), which is characterized by a brief depolarization called a spike or a jump (see [32, 37]). When a neuron spikes, it emits an action potential (see [40]). If a neuron i has membrane potential value x_i, then it spikes at a rate $\phi(x_i)$, where the functions $\phi : \mathbb{R}_+ \to \mathbb{R}_+$ are called intensity functions. When a neuron spikes its potential is reset to a non-negative resting value r_i, while neurons that interact with i receive an additional amount of either negative or positive potential which is added to their membrane potential.

In this paper, we study the Replica Mean Field limits of a system of infinitely many inhibitory and excitatory neurons.

One can study neural networks by focusing on the behavior of individual neurons, as in [27, 15, 33, 29, 31], where one concentrates on the examination of the jump times. In this case Hawkes processes are usually used which are memoryless since the point processes reset to a fixed value after a spike.

Alternatively, as in the current paper, the interactions among neurons in the network can be studied. In this case the entire evolution of the membrane potential of each neuron is studied. In fact, the evolution of the network between jumps is continuously analysed. Inhibition models for a finite number of neurons based

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on [37] are studied in [36, 61, 22], where a drift is not necessary since the neuron's membrane potential is reduced whenever a spike occurs in the system. The opposite of course happens in the case of excitatory neurons where a drift is crucial for the control of the system.

The case of networks with inhibitory and excitatory connections is studied in [62, 48, 54], while only inhibitory or excitatory connections are studied in [44]. In [32], a network with a deterministic drift has been considered.

The system of neurons that we consider in the current work contains infinitely many neurons that interact with each other through chemical synapses. Interactions between neurons occur when a neuron spikes. When a neuron i spikes, any neuron j that is connected to i receives an additional amount $w_{ij} \in \mathbb{R}$ of membrane potential called a synaptic weight. As for the membrane potential of the neuron i that spikes, it is reset to some non-negative value r_i. This description refers to the Galves–Löcherbach model (G-L) introduced in [29] to describe interactions of neural networks. In [29], the authors required the synaptic weights to satisfy the Property of Uniform Summability (PUS):

$$\sup_{i\in\mathbb{N}} \sum_{j\in\mathbb{N}} |w_{ji}| < \infty. \tag{1.1}$$

This is a Markovian process that belongs in the family of piecewise deterministic Markov processes introduced in [24, 25], which is frequently used in modeling chemical and biological phenomena (see for instance [7, 47, 23]). Piecewise deterministic Markov processes (PDMPs) have emerged as a powerful mathematical framework for modeling neuronal dynamics, particularly in integrate-and-fire models like Stein's neuronal model [58]. The hybrid nature of PDMPs, combining deterministic evolution with stochastic jumps, makes them particularly well-suited for capturing the behavior of neuronal membrane potentials, where continuous voltage dynamics are interrupted by discrete spike resets. PDMPs extend Stein's model by incorporating random jump processes that represent synaptic input events, stochastic ion channel behavior, or external perturbations. For example, in [46], PDMPs were used to model neurons with complex dendritic structures. The membrane potential at the trigger zone is modeled by a one-dimensional stochastic process with deterministic evolution between stochastic synaptic input events, which provides a synthesis of Stein's concept of the membrane potential behavior and the diffusion concept. Another application of PDMPs in neuroscience is presented in [51], where neuronal activity is formulated as an optimal control problem for infinite-dimensional PDMPs, incorporating both deterministic membrane potential evolution and stochastic transitions between different states of the ion channel.

Furthermore, hydrodynamic and mean-field limits of the model were studied in [19, 26, 16, 20], while properties of the invariant measure were studied in [38]. In [49, 34], some inequalities were also obtained. Phase transition and metastability were studied in [28, 5, 53, 4], and [6]. In [37, 39], no deterministic interactions have been considered.

In this paper, we study the Galves–Löcherbach model in the framework of [48] that allows interaction weights that go beyond the restrictive Property of Uniform Summability of the G-L model. In particular, we study the Replica Mean Field properties for the model beyond uniform summability. Replica Mean Field (RMF) limits were used by Baccelli and Taillefumer [9] to describe the stationary state of a finite neural network. Since [9] considered finitely many neurons, the system directly satisfied the uniform summability property. We will use the same approach here, but we will take advantage of the existence of inhibitory connections cancelling excitatory ones to obtain results about a system of infinitely many neurons that go beyond the Property of Uniform Summability.

The importance of studying neural networks that include both excitatory and inhibitory interactions follows from the fact that the lack of balance between the two types of interaction is associated with certain brain diseases (see [17, 12]). This takes place mainly when excitation dominates over inhibition (see [63, 13]). For this reason, the presence of inhibition is crucial in maintaining balance (see [14, 41, 66, 2]). Thus, when inhibitory neurons are included, the average local excitation can balance out with local inhibition [59, 2, 56, 57, 1, 64]. What is, however, important to notice is that this balance is not required only on a local level but globally within the network. This means that synapses between distant neurons should also be allowed (see [64, 3, 2]).

2. REPLICA MEAN FIELD LIMITS

Replica Mean Field (RMF) limits were used by Baccelli and Taillefumer [9] in order to describe the stationary state of a system of a finite number of neurons (see also [10, 8, 11, 55, 65, 18]). In RMF, if we have a system of K neurons, we consider a number of replicas of this system. If the RMF consists of M replicas, every one of them represents a copy of the initial neural system. As a result, there exists a copy of any of the $i \in K$ neurons in every replica. When a neuron spikes, it will interact with neurons from other replicas. Assume that a neuron i interacts with another neuron j in the initial system. In the RMF, when the neuron $i \in K$ from replica n spikes, then it will choose uniformly one of the $M-1$ copies of j from any of the other $M-1$ replicas different from n to interact. The RMF framework provides a biologically plausible way to analyze large, intensity-based neural networks while preserving key finite-size effects. Unlike classical mean-field approximations, which assume infinitely large homogeneous networks, the replica approach introduces independent copies (or replicas) of a finite network with randomized connectivity. This method is particularly relevant for biological neural networks, where sparse connectivity, stochastic activity, and finite-size effects play a crucial role. By incorporating randomness into neural interactions, the RMF model better reflects real-world neural architectures, such as cortical microcircuits and hippocampal networks. In addition, it provides a robust framework for

studying synaptic plasticity, learning, and information processing under realistic neural conditions.

The linear G-L model considered in [9], given that it consists of finitely many neurons, directly satisfies the PUS condition (1.1). We will use the same approach here, but with some modifications that will allow us to obtain results about a system of infinitely many neurons.

The idea behind the RMF structure is to express the moments of the invariant measure in terms of structural characteristics of the network, such as the drift a_i, the reset value r_i and the synaptic weights w_{ij}. In particular our aim is to obtain expressions of the moments of spiking rates $\beta_i^k = \mathbb{E}[N_i((0,1])^k]$ in terms of the aforementioned features. To do so, we will derive an ODE for β_i^k through the RMF limit.

The results presented in the current article as well as the methods to obtain them follow very closely the work in [9]. For a complete and comprehensive analysis of the RMF approach, one should consult [9]. Here, we will mention only the absolute necessary aspects of the method that will be used. In general, Replica Mean Field models consist of a finite number of copies of the initial model which replaces the initial interactions with higher level inter-replica interactions. Then, assuming the Poisson Hypothesis, when letting the number of replicas go to infinity we consider independence between the replicas. In the actual schema considered in [9], every replica consists of a finite number of neurons. In our case, however, where a network of infinitely many neurons is studied, we will consider replicas of infinitely many neurons.

The finite-replica model of infinitely many neurons is defined analogously to the finite-neurons case, as follows:

For a finite model consisting of M replicas, let $m \in \{1, \dots, M\}$ and $i \in \mathbb{N}$, $N_{m,i}$ denote the spiking activity of the neuron (m, i), that is, the neuron i belonging to the replica m. Accordingly, $\phi(x_i^m)$ for $m \in \{1, \dots, M\}$ and $i \in \mathbb{N}$ represents the spiking activity of the neuron (m, i); unlike the typical model where one neuron i interacts with any neuron j of its network through a synaptic weight w_{ij}, in the M-replica model, every neuron (m, i) of a replica m interacts only with the neurons (n, j) belonging to other replicas, i.e. $n \neq m$, through the same intensity weight w_{ij}, in such a way that the replica n is chosen in a uniform way among the $M - 1$ replicas $\{1, \dots, m-1, m+1, \dots M\}$ different from m.

Although we consider a network of infinitely many neurons, we will assume that for any neuron $i \in \mathbb{N}$, there is only a finite set K_i of neurons that receive a synaptic weight different from zero from i,

$$K_i := \{j \in \mathbb{N} : w_{ij} \neq 0\},$$

with $|K_i| < \infty$. Equally we assume that for a neuron $i \in \mathbb{N}$, there is only a finite set K^i of neurons from which i receives a synaptic weight different from zero,

$$K^i := \{j \in \mathbb{N} : w_{ji} \neq 0\},$$

with $|K_i| < \infty$. Furthermore, we will assume that for every $i \in \mathbb{N}$,

$$K^i \subset \{1, \dots, i-1, i+1\}.$$

In essence this is a feedforward neural network, where the succeeding neuron $i+1$ has also been added, in order to avoid trivial cases under Hypothesis (H1) presented below. Feedforward neural networks, where each neuron receives input from all preceding neurons, are fundamental to various biological systems and play crucial roles in sensory processing, motor control, and neural development. These structures facilitate sequential information transmission, enabling precise timing and efficient computation. In particular, synfire chains, which are characterized by precisely timed neural sequences, have been observed in motor and sensory circuits, contributing to processes such as speech and movement coordination [1].

One striking example of a generation of a sequential neural network occurs in songbirds, where synfire chains in the High Vocal Center drive sequential firing during song production [30]. Similarly, repetitions of spontaneous synaptic input pattern in neocortical neurons in vivo and in vitro were studied in [35].

Since the set of neurons K^i that influences a neuron i is always finite, our system is infinite-dimensional in the weak sense that K^i can increase to infinity as $i \to \infty$. As we will see later, when we study ergodicity this is important in order to talk about a single neuron's stationarity. Furthermore, it should be noted that we do not assume that the size of the sets K_i or K^i is uniformly bounded in i.

3. LINEAR INTENSITY AND LINEAR INTERACTION

In this section, we study the RMF limit for the linear G-L model, where the intensity function is linear $\phi(x_i) = x_i$. In order to stay closer to the stochastic framework of [9], we introduce a variation in the model of the previous section, that is, when a neuron i spikes, the membrane potential does not settle necessarily to 0 as in [29], but to some value $r_i \geqslant 0$. We will consequently study both the cases where $r_i = 0 \ \forall i \in \mathbb{N}$ and $r_i > 0 \ \forall i \in \mathbb{N}$.

In most classical neuronal models, it is assumed that, following an action potential, the membrane potential is reset to either a hyperpolarized value or to its resting potential – represented as zero in our model – that is, $r_i = 0$. This ensures that the neuron remains inactive for a period, commonly referred to as the refractory period. However, evidence suggests that the membrane potential does not always return exactly to its resting value after a spike. In [21], the authors investigated the afterpotential characteristics and firing patterns of developing rat hippocampal CA1 neurons. These neurons undergo substantial electrophysiological changes during postnatal development, particularly in their resting membrane potential (RMP). Their results showed that younger neurons exhibit a less negative RMP compared to mature neurons. A similar observation was reported in [43], which examined postnatal cerebral cortical pyramidal neurons in rats; however, in

that study, the authors attributed the observed depolarization to a measurement artefact. More recently, in [42], the authors analysed a large database of leech heart interneuron models and found that many neurons exhibit multistability. This means that a single neuron model can display multiple stable behaviors, such as quiescence and bursting, including different quiescent resting states. These findings highlight how neurons may leverage multistability for functional flexibility, but also how such dynamics could contribute to dysfunction.

The generator of the process X is given for any test function $f : \mathbb{R}_+^{\mathbb{N}} \to \mathbb{R}$ and $x \in \mathbb{R}_+^{\mathbb{N}}$ by

$$\mathcal{L}f(x) = -\sum_{i\in\mathbb{N}} a_i g(x_i)\frac{d}{dx_i}f + \sum_{i\in\mathbb{N}} \phi(x_i)[f(\Delta_i(x)) - f(x)] \tag{3.1}$$

for $g : \mathbb{R}_+ \to \mathbb{R}_+$ and $a_i \geqslant 0$ for all $i \in \mathbb{N}$, where

$$(\Delta_i(x))_j = \begin{cases} \max\{x_j + w_{ij}, 0\}, & j \neq i, \\ r_i, & j = i. \end{cases} \tag{3.2}$$

In order to form the infinitesimal generator of the M-replica model for the (3.1)–(3.2) model we need to introduce the set $V_{m,i}$ of neurons that are targeted by the neuron (m, i). When a neuron j in replica n, denoted (n, j), spikes, it interacts by sending a weight w_{jz} to every neuron $z \in K_j$. Since in the replica model there are $M - 1$ different neurons z it chooses one of the $M - 1$ replicas uniformly, say s, and interacts with this (s, z) neuron. Since every neuron (m, i) can be targeted by K_i different neurons, $V_{m,i}$ has cardinality $|V_{m,i}| = (M - 1)^{K_i}$, where

$$V_{m,i} = \big\{v \in \{1, \dots, M\}^{K_i} : v_i = m \text{ and } v_j \neq m,\ j \neq i\big\}.$$

The infinitesimal generator for the M-replica Markovian dynamics is

$$\begin{aligned}\mathcal{L}^M[f_u](x) = &-\sum_{i\in\mathbb{N}}\sum_{m=1}^{M} a_i g(x_i^m)\frac{d}{dx_i^m}f_u(x) \\ &+\sum_{i\in\mathbb{N}}\sum_{m=1}^{M}\frac{1}{|V_{m,i}|}\sum_{v\in V_{m,i}}\phi(x_i^m)[f(x + w_{m,i,v}(x)) - f(x)],\end{aligned}$$

where the weights distributed after neuron (m, i) spikes are

$$[w_{m,i,v}(x)]_{j,v_j} = \begin{cases} w_{ij} & \text{if } j \neq i,\ v_j \neq m, \\ r_i - x_i^m & \text{if } j = i,\ v_j = m, \\ 0 & \text{otherwise.} \end{cases}$$

We consider two main hypotheses about the drift and the intensity functions ϕ, g:

HYPOTHESIS (H1). The linear G-L model as in [9]:

$$\phi(x_i) = x_i, \quad g(x_i) = x_i \quad \text{and} \quad \min_{i\in\mathbb{N}} r_i > 0.$$

HYPOTHESIS (H2). For $z > 0$,

$$\phi(x_i) = x_i + z, \quad g(x_i) = x_i.$$

The computer simulations presented by C. Pouzat [50] for the G-L model designed to emulate the stochastic dynamics of neural networks present a firing behavior of neurons that is driven by a rate function, which is conceptually similar to the functions $\phi(x_i)$ in (H1) and (H2). In Hypothesis (H1), the firing rate is directly proportional to the membrane potential, suggesting a linear relationship between the neuron's activity and its current state. This linear assumption is often used in theoretical models of neural networks, including the G-L model, to simplify the analysis of network dynamics. On the other hand, Hypothesis (H2) introduces a shift in the firing rate.

For any set $S \subset \{1,\dots,M\}$ and $K \subset \mathbb{N}$, define $V_u^{K,S}(x) = e^{u\sum_{i\in K}\sum_{m\in S} x_i^m}$. Then

$$\begin{aligned}\mathcal{L}^M[V_u^{K,S}](x) = &-\sum_{i\in\mathbb{N}}\sum_{m\in S} a_i g(x_i^m)\frac{d}{dx_i^m}V_u^{K,S}(x)\\ &+\sum_{i\in\mathbb{N}}\sum_{m=1}^{M}\frac{1}{|V_{m,i}|}\sum_{v\in V_{m,i}}\phi(x_i^m)[V_u^{K,S}(x+w_{m,i,v}(x)) - V_u^{K,S}(x)].\end{aligned}$$

Since a neuron (j,m) with $j \notin \bigcup_{i\in K} K^i$ does not contribute synaptic weights to the sum $\sum_{i\in K}\sum_{m\in S} x_i^m$, we can write

$$\begin{aligned}\mathcal{L}^M[V_u^{K,S}](x) = &-\sum_{i\in K}\sum_{m\in S} a_i g(x_i^m) u V_u^{K,S}(x)\\ &+\sum_{i\in K}\sum_{m\in S}\frac{1}{|V_{m,i}|}\sum_{v\in V_{m,i}}\phi(x_i^m)\big[e^{u(r_i-x_i^m)+\sum_{j\in K\cap K_i,\, u_j\neq m} uw_{ij}} - 1\big]V_u^{K,S}(x)\\ &+\sum_{i\in K^c\cap\bigcup_{j\in K}K^j}\sum_{m=1}^{M}\frac{1}{|V_{m,i}|}\sum_{v\in V_{m,i}}\phi(x_i^m)\big[e^{\sum_{j\in K\cap K_i,\, u_j\neq m} uw_{ij}} - 1\big]V_u^{K,S}(x)\\ &+\sum_{i\in K}\sum_{m\in S^c}\frac{1}{|V_{m,i}|}\sum_{v\in V_{m,i}}\phi(x_i^m)\big[e^{\sum_{j\in K\cap K_i,\, u_j\neq m} uw_{ij}} - 1\big]V_u^{K,S}(x).\end{aligned}$$

Next we consider ϕ of polynomial order, that is, $\phi(x_i) = (x_i)^r + z$ for some positive $r \in \mathbb{N}$ and $z > 0$.

PROPOSITION 3.1. *Assume that ϕ is an increasing function of polynomial order. For every finite set of neurons $K = \{1,\dots,s\}$ for some $s \in \mathbb{N}$, such that for*

$K^i \subset \{1,\dots,i-1,i+1\}$ for any $i \in K$ and $u>0$, there exist $c>0$ and $d>0$ such that

$$\mathcal{L}^M[V_u^{K,M}](x) \leqslant -cV_u^{K,M}(x) + d\mathbf{I}_B(x).$$

Proof. Since $K^i \subset \{1,\dots,i-1,i+1\}$ we have $\{\bigcup_{j\in K} K^j\} \subset \{1,\dots s+1\}$, and so

$$\mathcal{L}^M[V_u^{K,M}](x) = -\sum_{i\in K}\sum_{m=1}^{M} a_i g(x_i^m) u V_u^{K,M}(x)$$
$$+\sum_{i\in K}\sum_{m=1}^{M}\frac{1}{|V_{m,i}|}\sum_{v\in V_{m,i}}\phi(x_i^m)\big[e^{u(r_i-x_i^m)+\sum_{j\in K\cap K_i, u_j\neq m} uw_{ij}}-1\big]V_u^{K,M}(x).$$

Since ϕ is of polynomial order, for every $u>0$ and $x \geqslant 0$ there exists an $\tilde{x} \in [0,\infty)$ such that $\phi(\tilde{x})e^{-u\tilde{x}} \geqslant \phi(x_i)e^{-ux_i}$ for all $x_i \geqslant b$ for some $b>0$. We can compute

$$\mathcal{L}^M[V_u^{K,M}](x)$$
$$\leqslant \sum_{i\in K}\sum_{m=1}^{M}\frac{1}{|V_{m,i}|}\sum_{v\in V_{m,i}}\big[\phi(\tilde{x})e^{+ur_i-u\tilde{x}+u\sum_{j\in K\cap K_i,\,u_j\neq m} w_{ij}}-\phi(x_i^m)\big]V_u^{K,M}(x).$$

If for some $c>0$, we consider the compact set

$$R_c^{KM} := \Big\{x\in\mathbb{R}_+^{KM}, x_i>b : \sum_{i\in K}\sum_{m=1}^{M}\phi(x_i^m)$$
$$\leqslant \sum_{i\in K}\sum_{m=1}^{M}\frac{1}{|V_{m,i}|}\sum_{v\in V_{m,i}}\phi(\tilde{x})e^{+ur_i-u\tilde{x}+u\sum_{j\in K\cap K_i,\,u_j\neq m} w_{ij}}+c\Big\},$$

then outside R_c^{KM} we have $\mathcal{L}^{K,M}V_u^{K,M}(x) \leqslant -cV_u^{K,M}(x)$, while for $x\in R_c^{KM}$ we get $\mathcal{L}^{K,M}V_{u,1}^{K,M}(x) \leqslant -cV_{u,1}^{K,M}+d$ for some positive constants c and d. Hence

$$\mathcal{L}^M V_u^{K,M} \leqslant -cV_u^{K,M} + d\mathbf{1}_{R_c^{KM}} \quad \blacksquare.$$

REMARK 3.1. In [9], the Foster–Lyapunov inequality was used to prove ergodicity for a Replica model which consists of a system of finite neurons, so that the MGF of all the neurons in the system with respect to the stationary measure can be calculated.

However, in our case, we will limit ourselves to one neuron, with the goal of studying an ODE for $\Lambda^{m,i}(u) = \mathbb{E}^{\{1,\dots,i+1\}}[e^{u(x_i)}]$, where $\mathbb{E}^{\{1,\dots,i+1\}}$ is the stationary measure referring to the partial network containing only the first $i+1$ particles $\{1,\dots,i+1\}$. Since, by the construction of the model, every $i\in\mathbb{N}$ can receive synaptic weights only from finitely many neurons

$$K^i \subset \{1,\dots,i-1,i+1\},$$

the behavior of any neuron i depends exclusively on a finite system of neurons $j \leqslant i+1$. In that way, $\Lambda^{m,i}(u) = \mathbb{E}^{\{1,\dots,i+1\}}[e^{u(x_i)}]$ can be studied as in the case of neuron i belonging to a finite system of neurons $\{1,\dots,i+1\}$. It is important at this point to clarify that ergodicity in our context does not refer to ergodicity for the whole infinite-dimensional system but to the individual neuron. For this reason, the stationarity of only individual neurons i is studied through the RMF limit $\Lambda^i = \lim_{m\to\infty} \Lambda^{m,i}$. The regeneration argument used below to establish ergodicity ultimately relies on the fact that each neuron is influenced by a finite set of neurons, each of which is in turn influenced by an even smaller set. In that way, the regeneration time for every neuron is finite. This allows us to obtain uniform results with respect to i, and thus conclude the desired property for all $i \in \mathbb{N}$. In this case, $\mathbb{E}^{\{1,\dots,i+1\}}$ is a measure that is obtained in a probability space of $i+1$ random variables $\{1,\dots,i+1\}$, with boundary conditions the values of the remaining r.v. $\{j : j \geqslant i+2\}$, which however, by the construction of the network, do not affect the first $i+1$ particles.

We can now prove Harris ergodicity.

THEOREM 3.1. *For every $i \in \mathbb{N}$ and $m \in \{1,\dots,M\}$ the system of $i+1$ neurons $\{x_{m,1}(t), x_{m,2}(t), \dots, x_{m,i+1}(t)\}_{t\in\mathbb{R}}$ is ergodic.*

Proof. First we notice that according to [45], the Foster–Lyapunov drift condition of the last proposition implies that the process is non-explosive and that the set R_c^{KM} is positive recurrent.

Following [9], Hypothesis (H1) implies that for large c, the set R_c^{MK} is also a regeneration set, since $\min_{i\in K} \inf_t \phi(x_i(t)) > \min_{i\in K} r_i > 0$. We can conclude the same for Hypothesis (H2), since according to Robert and Touboul [52], regeneration is guaranteed when neurons spike consecutively and spontaneously, which is the case when the spike intensity is always strictly positive, as in the current case where $\min_{i\in K} \inf_t \phi(x_i(t)) > \min_{i\in K} z > 0$.

The non-explosiveness of the Markov dynamics, together with the fact that the set R_c^{MK} is positive recurrent and a regeneration set, implies the Harris ergodicity of the Markov chain $\{x_{m,1}(t), x_{m,2}(t), \dots, x_{m,i+1}(t)\}_{t\in\mathbb{R}}$. ∎

Having established the ergodicity of the process, we can now obtain the ODE that describes the stationary measure. For some $z \geqslant 0$, define

$$\Lambda^{m,i}(u) := \mathbb{E}^{\{1,\dots,i+1\}}[e^{u(x_i^m+z)}],$$

where $\mathbb{E}^{\{1,\dots,i+1\}}$ is the expectation with respect to the stationary measure. We will derive an ODE for the RMF limit of $\Lambda^{m,i}$, that is,

$$\Lambda^i := \lim_{m\to\infty} \Lambda^{m,i}.$$

It should be noted that [9] dealt with the more general moment generating function $\mathbb{E}[e^{u\sum_{i\in K} x_i^m}]$, from which after using the Poisson Hypothesis an ODE for Λ^i

was obtained and consequently an analytic expression for $\beta_i^1 = \mathbb{E}[N_i(0,1]]$. In our case, starting directly from $\Lambda^{m,i}$ will allow us to reproduce the result from [9] following the same proof, but slightly simplified, because due to only one random variable involved we will avoid the complicated calculation of the quantities vanishing in the limit. Furthermore, we have perturbed the density function by a positive constant $z > 0$, so that we can consider Hypothesis (H2) where we obtain regeneration even when $r_i = 0$.

PROPOSITION 3.2. *Assume $g(x_i) = x_i$ and $\phi(x_i) = x_i + z$ for some $z \geqslant 0$. For every $m \in \{1, \dots, M\}$ and $i \in \mathbb{N}$, Λ^i satisfies the following ODE:*

$$0 = -(1 + a_i u)\frac{d}{du}\Lambda^i(u)(x) + \sum_{j \neq i}(e^{uw_{ji}} - 1)\beta_j \Lambda^i(u) + \beta_i e^{ur_i},$$

where $\beta_i = \mathbb{E}^{\{1,\dots,i+1\}}[x_i] + z$.

Proof. For $m \in \{1, \dots, M\}$ and $i \in \mathbb{N}$, for the function

$$V_u^{\{i\},\{m\}}(x) = \exp^{u(x_i^m + z)}$$

we have

$$\begin{aligned}\mathcal{L}^M[V_u^{\{i\},\{m\}}](x) = {} & -a_i g(x_i^m) u V_u^{\{i\},\{m\}}(x) + \phi(x_i^m)[e^{ur_i - ux_i^m} - 1]V_u^{\{i\},\{m\}}(x) \\ & + \sum_{j \neq i}\sum_{n \neq m}\frac{1}{|V_{n,j}|}\phi(x_j^n)[e^{uw_{ji}} - 1]V_u^{\{i\},\{m\}}(x).\end{aligned}$$

Since $\mathbb{E}^{\{1,\dots,i+1\}}[\mathcal{L}^M[V_u^{\{i\},\{m\}}]] = 0$, we obtain

$$\begin{aligned}0 = {} & -(1 + a_i u)\frac{d}{du}\Lambda^{m,i}(u)(x) + \frac{d}{du}\Lambda^{m,i}(u)(x)\mathbf{I}_{u=0}e^{ur_i} \\ & + \sum_{j \neq i}\sum_{n \neq m}\frac{1}{|V_{n,j}|}[e^{uw_{ji}} - 1]\mathbb{E}^{\{1,\dots,i+1\}}[(x_j^n + z)V_u^{\{i\},\{m\}}(x)].\end{aligned}$$

Since $|V_{n,j}|$ depends only on j, the last term becomes

$$\begin{aligned}& \sum_{j \neq i}\sum_{n \neq m}\frac{1}{|V_{n,j}|}[e^{uw_{ji}} - 1]\mathbb{E}^{\{1,\dots,i+1\}}[(x_j^n + z)V_u^{\{i\},\{m\}}(x)] \\ & \qquad = \sum_{j \neq i}(e^{uw_{ji}} - 1)\frac{1}{|V_{n,j}|}\sum_{n \neq m}\mathbb{E}^{\{1,\dots,i+1\}}[(x_j^n + z)V_u^{\{i\},\{m\}}(x)].\end{aligned}$$

We finally get

$$\begin{aligned}0 = {} & -(1 + a_i u)\frac{d}{du}\Lambda^{m,i}(u)(x) + \frac{d}{du}\Lambda^{m,i}(u)(x)\mathbf{I}_{u=0}e^{ur_i} \\ & + \sum_{j \neq i}(e^{uw_{ij}} - 1)\frac{1}{|V_{n,j}|}\sum_{n \neq m}\mathbb{E}^{\{1,\dots,i+1\}}[(x_j^n + z)V_u^{\{i\},\{m\}}(x)],\end{aligned}$$

or equivalently

$$0 = -(1+a_i u)\frac{d}{du}\Lambda^{m,i}(u)(x) + \mathbb{E}^{\{1,\dots,i+1\}}[x_i^m + z]e^{ur_i} + \sum_{j\neq i}(e^{uw_{ji}}-1)\frac{1}{|V_{n,j}|}\sum_{n\neq m}\mathbb{E}^{\{1,\dots,i+1\}}[(x_j^n+z)V_u^{\{i\},\{m\}}(x)].$$

By the Poisson Hypothesis, for $m \neq n$,

$$\lim_{M\to\infty}\mathbb{E}^{\{1,\dots,i+1\}}[(x_j^n+z)e^{u(x_i^m+z)}] = \lim_{M\to\infty}\mathbb{E}^{\{1,\dots,i+1\}}[x_j^n+z]\mathbb{E}^{\{1,\dots,i+1\}}[e^{u(x_i^m+z)}] = \beta_j\Lambda^i.$$

We finally obtain

$$0 = -(1+a_i u)\frac{d}{du}\Lambda^i(u)(x) + \sum_{j\neq i}(e^{uw_{ji}}-1)\beta_j\Lambda^i + \beta_i e^{ur_i}. \quad \blacksquare$$

The main result of this section about the linear G-L model follows. It should be noted that the main condition (3.3) does not allow purely inhibitory models, that is, with all weights $w_{ji} \leqslant 0$. But the inequality still allows some weights to be negative as long as the terms of the sum with positive weights compensate.

THEOREM 3.2. *Assume the linear G-L model satisfies* (H1) *or* (H2). *If the RMF limit* $\beta_i = \mathbb{E}^{\{1,\dots,i+1\}}[\phi(x_i)]$ *satisfies*

$$\sum_{j\in K^i}(1-e^{-w_{ji}/a_i})\beta_j > 0 \tag{3.3}$$

for any $i \in \mathbb{N}$, *then* β_i *solves the following system of equations:*

$$\frac{1}{\beta_i} = \int_{-\infty}^{0}\exp\Big(l_i(u) - \sum_{j<i}\beta_j h_{ij}(u)\Big)\,du, \tag{3.4}$$

where the functions $l_i(u)$ *and* $h_{ij}(u)$ *are*

$$l_i(u) = \frac{r_i}{a_i}(e^{a_i u}-1), \tag{3.5}$$

$$h_{ij}(u) = e^{-w_{ji}/a_i}\left(\mathrm{Ei}\left(\frac{w_{ji}}{a_i}e^{ua_i}\right) - \mathrm{Ei}\left(\frac{w_{ji}}{a_i}\right)\right) - u, \tag{3.6}$$

witj Ei *denoting the exponential integral function.*

A useful corollary follows.

COROLLARY 3.1. *Assume the linear G-L model satisfies* (H1) *or* (H2) *and* $\sup_{i,j\in\mathbb{N}}|w_{ji}|<\infty$. *If*

$$\sum_{j\in K^i}\beta_j<\infty,$$

then (3.4) *holds with* l_i, h_{ij} *as in* (3.5) *and* (3.6).

REMARK 3.2. If we cancel the drift, i.e. take the limit $a_i\to 0$, then (see again [9]) we get the following explicit expressions of l_i and h_{ij}:

$$l_i(u)=r_iu \quad\text{and}\quad h_{ij}(u)=\frac{e^{w_{ji}u}-1}{w_{ji}}-u.$$

Before we present the proof of Theorem 3.2, we present some examples of linear G-L networks that go beyond the PUS condition and satisfy the hypotheses of Theorem 3.2.

EXAMPLE 3.1. (A) Assume (H1). We will consider the case where there is no drift, i.e. $a_i=0$ for all $i\in\mathbb{N}$. Furthermore, for simplicity, assume that every i receives weights only from $j<i$. Then, as pointed out in Remark 3.2, the functions l_i and h_{ij} have an explicit expression which leads to the following simple form of (3.4):

$$\frac{1}{\beta_i}=\exp\biggl(\frac{\sum_{j<i}\beta_j}{w_{ji}}\biggr)\int_{-\infty}^{0}\exp\Bigl(\Bigl(r_i+\sum_{j<i}\beta_j\Bigr)u\Bigr)\exp\biggl(-\sum_{j<i}\frac{\beta_je^{w_{ji}u}}{w_{ji}}\biggr)\,du. \tag{3.7}$$

We assume that $w_{ij}\geqslant 0$ for all $i,j\in\mathbb{N}$.

Since, because of Corollary 3.1, it is sufficient to assume $\sum_{i\in\mathbb{N}}\beta_i<\infty$, we need β_i's to decrease in i sufficiently fast. But from the construction of the model, we know that every neuron i receives synaptic weights only from the neurons $j<i$. In that way, we guarantee that a neuron i affects only the neurons j that follow it, $j>i$, and is not affected by the neurons after it, $j>i$. In that way, β_i depends only on the behavior of the neurons $j<i$, while the values of $\{\beta_j, j<i\}$ depend neither on the value of r_i, nor on the values of the synaptic weights w_{ji}.

That implies that for fixed $\{\beta_j, j<i\}$, we can choose inductively the values w_{ji} and r_i in such a way that $\beta_i\leqslant 1/2^i$. This can happen in the following way. As in the previous example, from Remark 3.2 we obtain the expression (3.7). The right hand side of (3.7) increases as $w_{ij}\to 0$. In fact, when $w_{ij}\to 0$ we have

$$\frac{\sum_{j<i}\beta_j(1-e^{w_{ji}u})}{w_{ji}}\to -u\sum_{j<i}\beta_j,$$

and so

$$\frac{1}{\beta_i}\uparrow\int_{-\infty}^{0}\exp(r_iu)\,du=\frac{1}{r_i},$$

which implies that the value of β_i can be chosen as small as we desire as long as r_i and w_{ij} are sufficiently small. Since the weights r_i, w_{ij} are chosen small, we can consider the case where, for any $i \in \mathbb{N}$, $w_{ij} > a > 0$ for all $j > i$, for some small a uniformly in i and j. Then $\sum_j w_{ji} = (i-1)a \to \infty$ as $i \to \infty$, and the PUS condition does not hold.

(B) Assume (H1). If we consider the linear G-L model with a drift $a_i > 0$, then the same reasoning as in the previous example can be applied to (3.4). In that case, the values of β_j for $j < i$ do not depend on w_{ji} or r_i as in (A), nor on a_i. Then the smaller the values of w_{ij}, r_i and the greater the value of a_i, the smaller the value of β_i. Thus, the PUS condition will not hold, as in the previous example, as long as we choose, for any $i \in \mathbb{N}$, the weights $w_{ij} > a > 0$ for all $j > i$.

Proof of Theorem 3.2. Theorem 3.2 follows directly from [9, Theorem 3.6]. The proof of that result is based on the following proposition:

PROPOSITION 3.3 ([9, Proposition 5.2]). *If $\tau > 0$ and $f(-\tau) > 0$, then the ODE*

$$\left(1 + \frac{u}{\tau}\right)\Lambda'(u) + f(u)\Lambda(u) - g(u) = 0 \tag{3.8}$$

has a unique continuous solution

$$\Lambda(u) = \int_{-\tau}^{u} e^{-\int_v^u \frac{f(\omega)}{1+\omega/\tau}\, d\omega} \frac{g(v)}{1 + v/\tau}\, dv.$$

One observes that for the functions

$$g(u) = -\beta_i e^{ur_i} \quad \text{and} \quad f(u) = -\sum_{j \neq i} (e^{uw_{ji}} - 1)\beta_j,$$

and $\tau = 1/a_i$, the ODE (3.8) has already been proven in Proposition 3.2. It remains to impose conditions on the linear G-L model so that the condition $f(-1/a_i) > 0$ is satisfied, that is,

$$\sum_{j \neq i} \big(e^{-\frac{1}{a_i} w_{ji}} - 1\big)\beta_j < 0. \quad \blacksquare$$

4. NON-LINEAR INTENSITY AND NON-LINEAR INTERACTION

In this section, we will consider cases that go beyond the case of linear ϕ and g. To do so, we will introduce a new type of slightly modified interactions as defined below in (4.2). The generator of the process, for any test function $f : \mathbb{R}_+^N \to \mathbb{R}$ and $x \in \mathbb{R}_+^N$, is as follows:

$$\mathcal{L}f(x) = -\sum_i a_i g(x_i) \frac{d}{dx_i} f + \sum_i \phi(x_i)[f(\Delta_i(x)) - f(x)] \tag{4.1}$$

for $g(x_i) \geqslant 0$ for all $x_i \geqslant 0$, and $a_i \geqslant 0$ for all i, where

$$(\Delta_i(x))_j = \begin{cases} ((x_j)^r + (w_{ij})^r)^{1/r}, & j \neq i, \\ r_i, & j = i. \end{cases} \tag{4.2}$$

As in the case of the linear G-L model, we consider two different hypotheses, one with an intensity function that is always strictly positive and one where it is not, in which case a non-zero reset value is required for the neuron that spikes.

HYPOTHESIS (H3). For some $r > 0$ and $r_i > 0$,

$$\phi(x_i) = (x_i)^r, \quad g(x_i) = x_i \quad \text{and} \quad \min_i r_i > 0.$$

HYPOTHESIS (H4). For some $r > 0$ and $z \geqslant 0$,

$$\phi(x_i) = (x_i)^r + z, \quad g(x_i) = x_i.$$

The main result of this section about the non-linear model with non-linear interactions follows:

THEOREM 4.1. *Assume the non-linear model* (4.1)–(4.2) *with intensity function and drift as in* (H3) *or* (H4)*. If the RMF limit*

$$\beta_i = \mathbb{E}^{\{1,\dots,i+1\}}[\phi(x_i)] = \mathbb{E}^{\{1,\dots,i+1\}}[(x_i)^r] + z,$$

for some $r > 0$ and $z \geqslant 0$, satisfies

$$\sum_{j \in K^i} (1 - e^{-W_{ji}^r/a_i})\beta_j > 0$$

for any $i \in \mathbb{N}$, then β_i solves the following system of equations:

$$\frac{1}{\beta_i} = \int_{-\infty}^{0} \exp\Big(l_i(u) - \sum_{j<i} \beta_j h_{ij}(u)\Big),$$

where the functions $l_i(u)$ and $h_{ij}(u)$ are

$$l_i(u) = \frac{r_i^r}{a_i}(e^{a_i u} - 1),$$
$$h_{ij}(u) = e^{-w_{ji}/a_i}\left(\mathrm{Ei}\left(\frac{W_{ji}^r}{a_i} e^{u a_i}\right) - \mathrm{Ei}\left(\frac{W_{ji}^r}{a_i}\right)\right) - u,$$

with Ei *denoting the exponential integral function.*

The proof is identical to the proof of Theorem 3.2. We only need to check the analogue of Proposition 3.2 for the non-linear interactions and intensity function. In analogy to the previous subsection, we now define, for some $z \geqslant 0$ and $r > 0$,

$$\Lambda^{m,i}(u) = \mathbb{E}^{\{1,\dots,i+1\}}[e^{u((x_i^m)^r+z)}],$$

where $\mathbb{E}^{\{1,\dots,i\}}$ is the expectation with respect to the stationary measure. Similarly, the RMF limit of $\Lambda^{m,i}$ is

$$\Lambda^i := \lim_{m\to\infty} \Lambda^{m,i}.$$

PROPOSITION 4.1. *Assume $\phi(x) = x^r + z$ and $g(x) = x$. For every $m \in \{1,\dots,M\}$ and $i \in \{1,\dots,K\}$, Λ^i satisfies the following ODE:*

$$0 = -(1+a_i u)\frac{d}{du}\Lambda^i(u)(x) + \sum_{j\neq i}(e^{uw_{ji}} - 1)\beta_j\Lambda^i + \beta_i,$$

where $\beta_i = \mathbb{E}^{\{1,\dots,i+1\}}[(x_i)^r] + z$,

Proof. For $m \in \{1,\dots,M\}$ and $i \in \{1,\dots,K\}$, consider the function

$$V_{u,r}^{\{i\},\{m\}}(x) = e^{u((x_i^m)^r+z)}.$$

We have

$$\begin{aligned}
&\mathcal{L}^M[V_{u,r}^{\{i\},\{m\}}](x) \\
&\quad = -a_i g(x_i^m) u V_{u,r}^{\{i\},\{m\}}(x) + \phi(x_i^m)[e^{-u(x_i^m)^r} - 1]V_{u,r}^{\{i\},\{m\}}(x) \\
&\qquad + \sum_{j\neq i}\sum_{n\neq m}\frac{1}{|V_{n,j}|}\phi(x_j^n)[V_{u,r}^{\{i\},\{m\}}(\Delta_i(x)) - V_{u,r}^{\{i\},\{m\}}(x)].
\end{aligned}$$

But under the new interaction regime introduced in (4.2) we now have

$$V_{u,r}^{\{i\},\{m\}}(\Delta_i(x)) = e^{u((x_i^m)^r+z+(w_{ji})^r)}$$

and so

$$\begin{aligned}
&\mathcal{L}^M[V_{u,r}^{\{i\},\{m\}}](x) \\
&\quad = -a_i g(x_i^m) u V_{u,r}^{\{i\},\{m\}}(x) + \phi(x_i^m)[e^{-u(x_i^m)^r} - 1]V_{u,r}^{\{i\},\{m\}}(x) \\
&\qquad + \sum_{j\neq i}\sum_{n\neq m}\frac{1}{|V_{n,j}|}\phi(x_j^n)[e^{uw_{ji}^r} - 1]V_{u,r}^{\{i\},\{m\}}(x).
\end{aligned}$$

Since $\mathbb{E}^{\{1,\dots,i+1\}}[\mathcal{L}^M[V_{u,r}^{\{i\},\{m\}}]] = 0$ we obtain

$$\begin{aligned}
0 = &-(1+a_i u)\frac{d}{du}\mathbb{E}^{\{1,\dots,i+1\}}[V_{u,r}^{\{i\},\{m\}}(x)] \\
&+ \frac{d}{du}\mathbb{E}^{\{1,\dots,i+1\}}[V_{u,r}^{\{i\},\{m\}}(x)]\mathbf{I}_{u=0}e^{ur_i^r} \\
&+ \sum_{j\neq i}\sum_{n\neq m}\frac{1}{|V_{n,j}|}[e^{uw_{ji}^r} - 1]\mathbb{E}^{\{1,\dots,i+1\}}[((x_j^n)^r + z)V_{u,r}^{\{i\},\{m\}}(x)].
\end{aligned}$$

Since $|V_{n,j}|$ depends only on j, the last term becomes

$$\sum_{j\neq i}\sum_{n\neq m}\frac{1}{|V_{n,j}|}[e^{uw_{ji}^r}-1]\mathbb{E}^{\{1,\dots,i+1\}}[((x_j^n)^r+z)V_{u,r}^{\{i\},\{m\}}(x)]$$
$$=\sum_{j\neq i}(e^{uw_{ji}^r}-1)\frac{1}{|V_{n,j}|}\sum_{n\neq m}\mathbb{E}^{\{1,\dots,i+1\}}[((x_j^n)^r+z)V_{u,r}^{\{i\},\{m\}}(x)].$$

We finally get

$$0=-(1+a_iu)\frac{d}{du}\mathbb{E}^{\{1,\dots,i+1\}}[V_{u,r}^{\{i\},\{m\}}(x)]$$
$$+\frac{d}{dx_i^m}\mathbb{E}^{\{1,\dots,i+1\}}[V_{u,r}^{\{i\},\{m\}}(x)]\mathbf{I}_{u=0}e^{ur_i^r}$$
$$+\sum_{j\neq i}(e^{uw_{ij}}-1)\frac{1}{|V_{n,j}|}\sum_{n\neq m}\mathbb{E}^{\{1,\dots,i+1\}}[((x_j^n)^r+z)V_u^{\{i\},\{m\}}(x)],$$

or equivalently

$$0=-(1+a_iu)\frac{d}{du}\Lambda^{m,i}(u)(x)+\mathbb{E}^{\{1,\dots,i+1\}}[x_i^m]e^{ur_i^r}$$
$$+\sum_{j\neq i}(e^{uw_{ji}}-1)\frac{1}{|V_{n,j}|}\sum_{n\neq m}\mathbb{E}^{\{1,\dots,i+1\}}[((x_j^n)^r+z)V_u^{\{i\},\{m\}}(x)].$$

By the Poisson Hypothesis, when M goes to infinity and $m\neq n$,

$$\lim_{M\to\infty}\mathbb{E}^{\{1,\dots,i+1\}}[(x_j^n)^re^{u(x_i^m)^r}]=\lim_{M\to\infty}\mathbb{E}^{\{1,\dots,i+1\}}[(x_j^n)^r]\mathbb{E}^{\{1,\dots,i+1\}}[e^{u(x_i^m)^r}]$$
$$=\mathbb{E}^{\{1,\dots,i+1\}}[(x_j)^r]\mathbb{E}^{\{1,\dots,i+1\}}[e^{u(x_i)^r}].$$

This leads to

$$0=-(1+a_iu)\frac{d}{du}\mathbb{E}^{\{1,\dots,i+1\}}[e^{u(x_i)^r}]$$
$$+\sum_{j\neq i}(e^{uw_{ji}^r}-1)\mathbb{E}^{\{1,\dots,i+1\}}[(x_j)^r+z]\mathbb{E}^{\{1,\dots,i+1\}}[e^{u(x_i)^r}]$$
$$+\mathbb{E}^{\{1,\dots,i+1\}}[(x_i)^r+z]e^{ur_i^r}.\ \blacksquare$$

As in the previous section where the linear G-L model was studied, similar examples can be constructed.

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Ioannis Papageorgiou
Universidade Federal do ABC (UFABC) - CMCC
Santo André, São Paulo, Brazil
E-mail: i.papageorgiou@ufabc.edu.br, papyannis@yahoo.com

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