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# PROCESSING-IN-INTERCONNECT INSPIRED DENDRITIC COMPUTATION

**Keynote Speaker Chetan Singh Thakur**

Neuronal dendrites, akin to the neurobiological equivalent of interconnects, display a spectrum of linear and nonlinear mechanisms enabling them to perform fundamental computations. Neuroscience research emphasizes the vital role of dendrites in neural processing, challenging the conventional view of hardware interconnects, which primarily serve to transmit information passively.

Motivated by dendritic computation, our research introduces a novel SNN computing paradigm called TEMP (Time-to-Event Margin Propagation). This model harnesses the intrinsic computational capabilities of interconnects, including delay, sorting, and triggering operations, to facilitate both memory and computation. TEMP-based networks enable high representational capacity through combinatorial representations, and their basic primitives support energy-efficient and sparse processing. This approach contrasts traditional ML architectures, which heavily depend on local multiply-and-accumulate (MAC) operations.

# A BIOLOGICALLY GROUNDED SPIKING NEURAL NETWORK MODEL OF LEARNING IN THE VISUAL CORTEX

Presenter Marko Ruslim<sup>1#</sup>

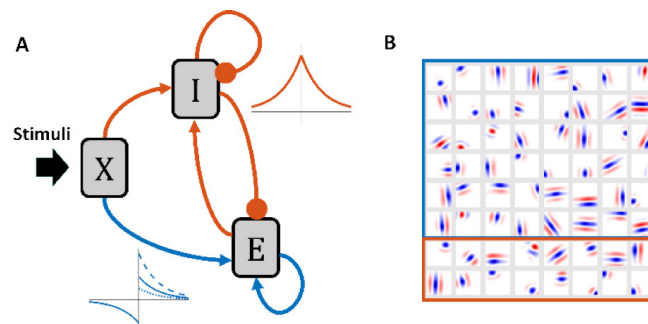
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Models that attempt to account for the physiological properties of the primary visual cortex (V1) often include features that are not biologically realistic. This puts into question how our brain can implement such models. The original sparse coding model, which posited that visual stimuli are represented by the activity of a small number of active neurons, was able to produce similar receptive fields to those observed electrophysiologically [1]. However, this model and its successive variants are not biologically plausible in several aspects, such as the possibility of negative firing rates, absence of spike-based plasticity and violation of Dale's law. We build upon previous models [2] and introduce a novel learning model that is biologically motivated by incorporating: spiking neurons with feedforward and recurrent synapses, separate excitatory and inhibitory populations, and Spike Timing-Dependent Plasticity (STDP).



**Figure 1:** Network structure and receptive fields. (A) LGN inputs ( $X$ ) receive visual stimuli and project to an excitatory population ( $E$ ) and an inhibitory population ( $I$ ), which also form recurrent connections. Insets: STDP learning rules for the blue and red connections. (B) Model receptive fields of 48 randomly chosen excitatory neurons (blue box) and 16 randomly chosen inhibitory neurons (red box).

Pre-processed natural scenes stimuli are presented to a population of Lateral Geniculate Nucleus (LGN) Poisson neurons consisting of 256 ON and 256 OFF cells, which project to 400 excitatory and 100 inhibitory Leaky Integrate-And-Fire (LIF) neurons. We use a minimal triplet STDP learning rule [3] for the excitatory-excitatory connections and a symmetrical STDP learning rule for all other connections (Figure 1A). Our model also includes a homeostatic rule that ensures a sparse code.

After training on natural images, our biologically-grounded learning model is able to learn a diverse set of receptive fields that has similar properties to experimentally observed V1 simple cells (Figure 1B). This includes small unoriented 'blob-like' features, as well as oriented Gabor-like filters.

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# BRAIN-WIDE CALCIUM IMAGING IN ZEBRAFISH AND GENERATIVE MODELLING REVEAL FUNCTIONAL NETWORK PROPERTIES OF SEIZURE DEVELOPMENT AND PROPAGATION

**Presenter Wei Qin<sup>1#</sup>**

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Epilepsy is a neurological disorder that causes recurrent seizures, but the exact mechanisms that trigger the transition from normal brain activity to seizure state are still unclear. Conventional methods, using data from humans, nonhuman primates, or rodents, have drawbacks for measuring the activity of single cells that may initiate or spread seizures<sup>[1]</sup>. Even at the network level, the changes in the network connectivity before and after state transitions are still not well understood. A method that can capture the dynamics of individual neurons and their interactions within the whole brain network would be useful for studying epilepsy. Zebrafish and calcium imaging provide such a method, as they enable simultaneous recording of neuronal activity in vivo with cellular specificity<sup>[2]</sup>.

Zebrafish share genetic and physiological similarities with humans and can exhibit seizure-like behaviours in response to various stimuli and drugs. One such drug is Pentylenetetrazol (PTZ), a pharmacological agent that blocks the inhibitory signalling of the neurotransmitter GABA, causing hyperexcitability and seizure-like activity. Additionally, mutations in the *scn1lab* gene, which encodes a sodium channel, can also cause spontaneous seizures in zebrafish<sup>[2]</sup>. In this study, we used in vivo light-sheet calcium imaging, brain-wide and at cellular resolution, on wildtype and *scn1lab* mutant zebrafish larvae under baseline and PTZ conditions.

In this study, we carried out calcium imaging on zebrafish with *scn1lab* mutations and wild-type controls at three levels of resolution: whole-brain, regional, and cellular. We compared the brain networks of the two genotypes and their changes after exposure to PTZ. We used network analysis and graph theory to quantify the differences in network structure and dynamics. We focused on the brain regions involved in ictogenesis and spread across different scales. Our results showed significant and consistent alterations in brain network functional connectivity and information flow in *scn1lab* mutants, suggesting that the mutations affect the functional organization of the brain. We also observed the evolution of the networks of both genotypes after PTZ treatment, where more nodes with high degrees emerged. Our findings demonstrate the unique advantages of zebrafish as an epilepsy research model and provide insights into the possible network mechanisms underlying seizure susceptibility and onset.

We also developed a generative model based on 6 wiring rules<sup>[3]</sup> to investigate how *scn1lab* mutations and PTZ affect network formation in the brain. We matched the generative models with the empirical data to evaluate the wiring rules and thus to infer the possible mechanisms of seizure development. The model considers the distance between ROIs and the wiring rules that control the formation of connections. Our model demonstrates that some wiring rules (e.g., homophily) can produce networks with similar properties to the functional connectivity derived from the empirical calcium imaging data. The findings suggest that WT and *scn1lab* mutants follow different principles during brain development. Our next step will be to compare the outputs of our models with high resolution anatomical data to pinpoint physical changes that may predispose *scn1lab* mutants to seizures. Moreover, we would like to try a combination of wiring rules that could provide predictive capability for seizure onset and offset. Such predictive models would set the stage for hypothesis-based tests of brain function using targeted ablations or optogenetic manipulations.

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# NEUROMORPHIC TECHNOLOGY FOR ENABLING NEURAL INTERACTION

**Keynote Speaker** Elisa Donati

Research Fellow with the Institute of Neuroinformatics, University of Zürich and ETH Zürich

The emergence of neuromorphic electronic circuits has the potential to revolutionize the field of bioelectronic medicine, enabling the development of highly accurate and targeted solutions for treating chronic diseases. By mimicking the structure and function of the nervous system, neuromorphic circuits can effectively interface with real neural processing systems, paving the way for real-time closed-loop interactions with biological tissues.

This talk will delve into the key characteristics of neuromorphic circuits that make them ideal for interfacing with the nervous system. It will also showcase the design and implementation of closed-loop hybrid artificial and biological neural processing systems, demonstrating their potential in various therapeutic applications.

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# NEUROMORPHIC IMAGING FLOW CYTOMETRY

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Imaging flow cytometry offers in-depth spatial information in delivering cell measurement, including cell morphology, texture and marker localization. This enables profound insights into single-cell studies. However, constructing such high-dimensional full-frame feedback can lead to a significant trade-off between throughput and spatial resolution. Moreover, fast-travelling fluorescent objects can be difficult to register by traditional photosensors owing to the limited amount of emission signal. Thus, it is imperative to explore the potential of next-generation imaging flow cytometry to address these challenges.

In this work, we adopted a neuromorphic photo-sensing technique to focus on the events of interest by asynchronous firing pixels to minimise data redundancy and latency. By incorporating its high dynamic range (> 120 dB), this application can be exceptionally sensitive against weak fluorescence signals. Herein, we provided a preliminary cytometric-like function in detecting and measuring 8, 15  $\mu\text{m}$  microparticles and human monocytic cell line (THP-1) under an event-focused vision, contributing highly consistent outputs with a commercialised flow cytometry.

Furthermore, the capacity of an event-based sensor in registering fluorescence signals was examined by capturing 6  $\mu\text{m}$  FITC-marked particles, achieving an outstanding performance compared to a conventional photosensor in different light scenarios.



# DIFFERENTIAL EFFECTS OF ANTI-SEIZURE MEDICATION ON LAYER 4 OF RAT V1

**Poster Presenter Artemio Soto-Breceda<sup>1,#</sup>**  
**P. Zarei Eskikand<sup>1</sup>, S. Sepehr Kazemi<sup>1</sup>,**  
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One of the main challenges in epilepsy treatment is the selection of anti-seizure medications (ASMs), which play an important role in seizure risk management and thus quality of life. With more than 30 types of ASMs and about 30% of people presenting drug resistant epilepsy, choosing an effective treatment can take years<sup>[1]</sup>.

Computational models of neural activity at the mesoscopic scale are frequently employed to describe epileptiform activity and infer changes in model parameters, with the goal of identifying the physiological mechanisms underlying seizures and epilepsy<sup>[2]</sup>. Despite their widespread use, these models are not typically validated for microscale neural dynamics such as conductance and synaptic strengths, which are critical for understanding drug effects.

This study presents a model of layer 4 of rat primary visual (V1) cortex following a multiscale framework and based on electrophysiological recordings of rat V1<sup>[3]</sup>. The model shows the distinct effects of GABAergic ASM on this specific region at the mesoscale level, which may have implications for their efficacy and side effects in epilepsy treatment. The micro-scale model, a biologically informed network of leaky integrate and fire (LIF) neurons was implemented with parameters taken from experimental results. Population size, connectivity and synaptic gains from the LIF were used as parameters in the mesoscale Neural Mass Model (NMM). Then, the firing rate function and post-synaptic potential of each synapse were fitted to a system of two differential equations per synapse describing the NMM dynamics. We validated the behaviour of the NMM against the LIF was across the parameter space using different metrics such as population firing rates, input current balance and frequency spectrum.

The effect of the different ASMs in the NMM was evaluated by measuring the recovery time from a current pulse injected into the circuit. Our results suggest distinct effects of diazepam and baclofen compared to that of muscimol in the models, which may help to determine approaches for epilepsy treatment depending on the source of the seizure. The multiscale model presented in this study offers a valuable instrument to investigate the intricate mechanisms of drug action on specific cortical networks and serves as a versatile platform for in silico testing of novel ASMs.

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# EVALUATING IMAGE PROCESSING ALGORITHMS FOR PROSTHETIC VISION USING GAIT ANALYSIS

**Poster Presenter Daniel Petrovski<sup>1</sup>**

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Retinitis Pigmentosa gradually destroys the photoreceptor cells in the retina, resulting in a steady deterioration of vision that eventually leads to complete blindness. Despite this, a significant number of retinal ganglion cells, responsible for transmitting visual information to the brain via the optic nerve, remain functional. The goal of retinal prostheses is to help patients affected by this condition regain their sight by using an implantable electrode array to stimulate the remaining retinal ganglion cells. The device captures visual information through a camera, processes it, and then activates the electrodes to stimulate the retinal neurons. However, the vision that is restored is rudimentary and very low resolution. To mitigate these issues, various image processing algorithms are employed, transforming high-resolution camera-captured images into signals suitable for low-resolution retinal prosthetic electrode arrays, with the aim to enhance patient outcomes.

Assessing the performance of retinal prostheses and the quality of the vision they restore typically involves measuring visual function, without considering functional outcomes for patients. Therefore, new assessment methods that prioritise functional vision are needed. One such functional outcome is the ability to navigate through different environments safely and confidently. When vision is compromised, individuals lose confidence in their ability to perceive their surroundings accurately, leading to an altered walking style that is more cautious. In this context, we propose using gait as an indicator of an individual's confidence to navigate in unfamiliar environments.

In this study, we conducted a gait analysis as participants with healthy gait and vision navigated through a simple low contrast obstacle course under simulated prosthetic vision with virtual reality goggles. Two image processing algorithms were subjected to comparative analysis within the context of simulated prosthetic vision. The first was a straightforward intensity map algorithm, which directly translated the grayscale image captured by the camera into prosthetic vision. The second employed a depth-sensing camera, resulting in a depth map image that was subsequently converted to prosthetic vision<sup>[1]</sup>.

Our results indicate a discernible difference in gait patterns between the two image processing algorithms, with variations observed in key gait parameters such as stride length, walking speed, swing phase, maximum toe clearance, and step width. Specifically, participants demonstrated a more confident walking style when navigating the obstacle course using the depth image processing algorithm, as compared to the intensity mapping algorithm. This observation underscores the potential of using gait as a reliable indicator of a person's confidence in their perception of the environment. Furthermore, it provides a novel approach to evaluating the performance of prosthetic vision, shifting the focus towards functional outcomes that directly impact the user's quality of life.

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# FPGA HARDWARE IMPLEMENTATION OF THE ODESA FRAMEWORK

**Poster Presenter** Ali Mehrabi  
Yeshwanth Bethi, André van Schaik, and Saeed Afshar

International Centre for Neuromorphic Systems, Western Sydney University, Australia

We demonstrate that the ODESA framework <sup>[1]</sup> and the online training of weights and thresholds can be implemented efficiently on a large scale in FPGA hardware. Our implementation consists of a multi-layer Spiking Neural Network (SNN) and individual training modules for each layer that enable online self-learning without using backpropagation.

By using simple local adaptive selection thresholds, a Winner-Takes-All (WTA) constraint on each layer, and a modified weight update rule that is more amenable to hardware, the trainer module allocates neuronal resources optimally at each layer without having to pass high-precision error measurements across layers.



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# OPTIMIZED DEEP EVENT-DRIVEN SPIKING NEURAL NETWORK ARCHITECTURE

**Poster Presenter Saeed Afshar**  
**Yeshwanth Bethi, and André van Schaik**

International Centre for Neuromorphic Systems, Western Sydney University, Australia

We present modular event-driven spiking neural architectures that use local synaptic and threshold adaptation rules to perform transformations between arbitrary spatio-temporal spike patterns. The architectures represent a highly abstracted model of existing Spiking Neural Network (SNN) architectures. We showcase Optimized Deep Event-driven Spiking neural network Architecture (ODESA) [1] that can simultaneously learn hierarchical spatio-temporal features at multiple arbitrary time scales.

ODESA performs online learning without the use of error back-propagation or the calculation of gradients. Using simple local adaptive selection thresholds at each node, the network rapidly learns to appropriately allocate its neuronal resources at each layer for any given problem without using an error measure.

The computations performed by the architectures are event-driven and the entire communication between layers is binary event-based. We provide a potential blueprint for future neuromorphic architectures that can be run asynchronously and enable low-power always-on learning systems.



# BALANCING PRIOR KNOWLEDGE AND SENSORY DATA: A PREDICTIVE CODING MODEL FOR COHERENT MOTION DETECTION AND IMPLICATIONS FOR SCHIZOPHRENIA

**Poster Presenter Fatemeh Nemat<sup>1</sup>**

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Motion detection is a continuous form of perception that heavily relies on past motion information. In our everyday lives, we are constantly processing motion information by predicting the movement of objects, such as cars while crossing a street. Apart from prior experience, the prediction of motion is also influenced by the motion of neighbouring objects within the receptive fields of neurons. This study addresses the question of how our brain extracts coherent motion from multiple moving objects in two-dimensional space. For example, in cases where a group of birds is in motion, our brain perceives both the shared coherent motion of this group of birds and their individual movement. Some studies have shown that patients with schizophrenia display deficiencies in specific motion detection tasks that require the estimation of coherent motion, such as random dot experiments<sup>[1]</sup>. We aim to address where deficiencies in motion detection tasks may arise in a predictive coding model of coherent motion detection.

The computational model is based on a single-layer classical predictive coding scheme<sup>[2]</sup>. A mechanism of surround suppression is also implemented, which suppresses neuronal activity when there is motion in the surrounding area of the receptive field. The effect of surround suppression depends on the prior history of neuronal activity within a time window. We tested the model with a random dot experiment where a set of dots moves in a coherent direction with noise added to the motion of each dot. In this experiment, we increased the level of noise step by step and evaluated the performance of the model. The results showed that as noise levels increase (from 0% to 60%), the time required for the model to converge also increases, progressing from 100 ms to 450 ms. We also evaluated the effect of prior experience on motion detection by changing the temporal window for using prior neuronal activity in motion detection. The results show that initially, when the window width is 0 and the model is not using prior information, the time to converge is 380 ms. With increasing window duration, the time to converge gradually drops to 280 ms when the window is 30 ms. With further increases in the time window beyond 30 ms, the time to converge gradually increases (reaching 400 ms with a time window of 80 ms) until the model does not converge at all.

In this study, a biologically plausible model based on the predictive coding technique is presented that is capable of successfully detecting the coherent motion of stimuli. Our model emphasizes the crucial role of the balance between relying on prior knowledge and sensory data, as this balance significantly impacts the overall performance of the model. Our model is motivated by the idea that abnormalities in motion detection tasks in schizophrenia might result from either overly relying on prior knowledge or ignoring prior knowledge and responding strongly to noise.

Acknowledgment - This research is funded by Early Career Research Grant from the University of Melbourne.

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# REVVING UP MOTOR UNITS AFTER NON-INVASIVE SPINAL CORD STIMULATION IN PATIENTS WITH TETRAPLEGIA

Poster Presenter Jessica Catherine Martin<sup>1,3</sup>  
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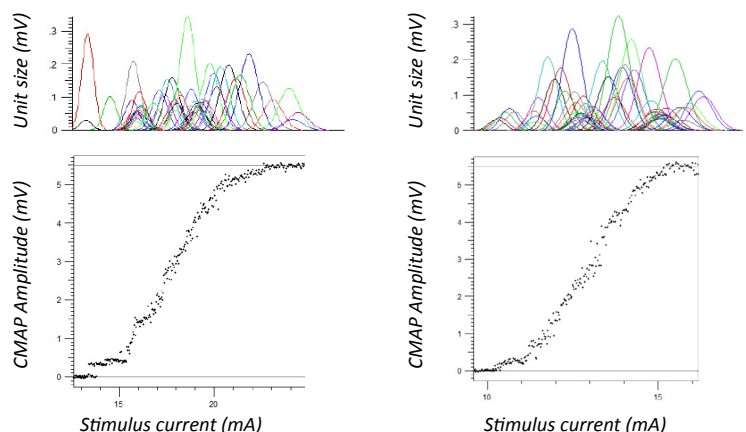
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**Introduction:** Motor Unit Number Estimation (MUNE) may be an important technique to evaluate longitudinal change in motor function, particularly in the context of spinal cord injuries, which are profoundly debilitating. Such injuries disrupt supraspinal control of sensorimotor pathways and severely disrupt afferent input from the periphery at the level of injury. In Australia, tetraplegia, characterized by varying degrees of impairment in the arms, hands, legs, and trunk, affects 42% of individuals living with spinal cord injuries, incurring significant societal costs<sup>[1]</sup>.

While therapeutic interventions to recover lost function are limited<sup>[2]</sup>, transcutaneous spinal cord stimulation (TSS) shows promise in enhancing the excitability of spinal circuitry below the injury site. Extensive research has explored its capacity to improve lower limb function, trunk stability, bladder function, and spasticity<sup>[3]</sup>. TSS also holds potential for restoring upper limb function, although much of the supporting evidence is based on case reports and case series. The Transcutaneous Electrical Spinal Cord Neuromodulation (TESCoN) trial aims to evaluate the combined approach of TSS with intensive physical therapy, recognizing the key role of cutaneous and proprioceptive input in TSS potentiation.

**Methods:** Each TESCoN therapy session lasts a maximum of two hours per day, 4 - 5 days per week for four weeks. 80 participants are expected to be recruited using an early phase adaptive basket trial design. Motor axons of the median nerve are electrically stimulated at the wrist, and compound muscle action potential (CMAP) responses for the abductor pollicis brevis muscle are recorded using QTracS software and TROND recording protocol<sup>[4]</sup>. These responses are used to estimate the number of contributing motor units (MUNE) using the MScan-Fit-2 program<sup>[5]</sup>.

**Results:** Preliminary analysis was conducted for 6 participants for whom baseline and post-intervention MUNE data have been collected. Baseline MUNE averaged  $58 \pm 20$ , which is notably lower than the values reported for healthy people ( $135 \pm 37$ )<sup>[6]</sup>. Following the intervention, post-intervention MUNE increased to  $69 \pm 24$ . Large scale variations in MUNE between the hands were observed in patients with hemiplegia. As further participants are recruited, this technique holds the potential to differentiate individuals who are functionally stable from those who with clinically significant motor function improvements.



**Figure 1:** Left panel: baseline MUNE (49 units) and right panel: post-intervention MUNE (59 units) for the right abductor pollicis brevis muscle in a patient with incomplete tetraplegia (AIS D)

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# SPIKE TIMING DEPENDENT GRADIENT FOR DIRECT TRAINING OF FAST AND EFFICIENT BINARIZED SPIKING NEURAL NETWORKS\*

**Poster Presenter Mostafa Rahimi Azghadi<sup>3#</sup>**  
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Spiking neural networks (SNNs) are well-suited for neuromorphic hardware due to their biological plausibility and energy efficiency. These networks utilize sparse, asynchronous spikes for communication and can be binarized. However, the training of such networks presents several challenges due to their non-differentiable activation function and binarized inter-layer data movement.

The well-established backpropagation through time (BPTT) algorithm used to train SNNs encounters notable difficulties because of its substantial memory consumption and extensive computational demands. These limitations restrict its practical utility in real-world scenarios. Therefore, effective techniques are required to train such networks efficiently while preserving accuracy. In this paper, we propose Binarized Spike Timing Dependent Gradient (BSTDG), a novel method that utilizes presynaptic and postsynaptic timings to bypass the non-differentiable gradient and the need of BPTT. Additionally, we employ binarized weights with a threshold training strategy to enhance energy savings and performance.

Moreover, we exploit latency/temporal-based coding and the Integrate-and-Fire (IF) model to achieve significant computational advantages. We evaluate the proposed method on Caltech101 Face/Motorcycle, MNIST, Fashion-MNIST, and Spiking Heidelberg Digits. The results demonstrate that the accuracy attained surpasses that of existing BSNNs and single-spike networks under the same structure. Furthermore, the proposed model achieves up to 30× speedup in inference and effectively reduces the number of spikes emitted in the hidden layer by 50% compared to previous works.



# NEUROMORPHIC OLFACTION: FROM ODOURS TO SPIKE-REPRESENTATIONS

**Poster Presenter Nik Dennler<sup>1,2</sup>  
André van Schaik<sup>2</sup> and Michael Schmuker<sup>1</sup>**

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In the realm of sensory technology and artificial intelligence, the emergence of neuromorphic olfaction has recently become a promising candidate to bridge the gap between biological olfaction and artificial systems<sup>[1, 2, 3]</sup>. The self-similar nature of odour plumes and the intermittent odour encounters when navigating an environment<sup>[4]</sup> suggest the use of fast and asynchronous sampling techniques<sup>[5]</sup>.

We present a particularly promising technology that may be used to address this, which is an electronic nose utilising Metal Oxide (MOx) gas sensors<sup>[6]</sup>. The employed sensors offer versatility and sensitivity in odor detection, where the gas dependent resistance can be sampled at arbitrary fast sampling rates. Further - yet exploratory - work is being done on optical electronic noses<sup>[7]</sup>, which provide a real-time and scalable sensor modality, as well as on Planar Laser-Induced Fluorescence (PLIF)<sup>[8]</sup>, which is often used to visualize odour plumes and to gain insights into their dynamic behavior.

Taking a step forward, we propose novel pathways for neuromorphic olfaction. Asynchronous sampling of the different sensor modalities, as well as processing techniques, are suggested. In summary, this study addresses the pressing needs of neuromorphic olfaction and investigates promising technologies and innovative approaches to bring us closer to bridging the gap between odor plumes and spike-representations, enabling a new era of artificial olfaction with applications in environmental monitoring, robotics, and various other domains.

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# TRANSITIONS IN A MODEL OF MARMOSET PFC

**Poster Presenter Bernard A Pailthorpe#**

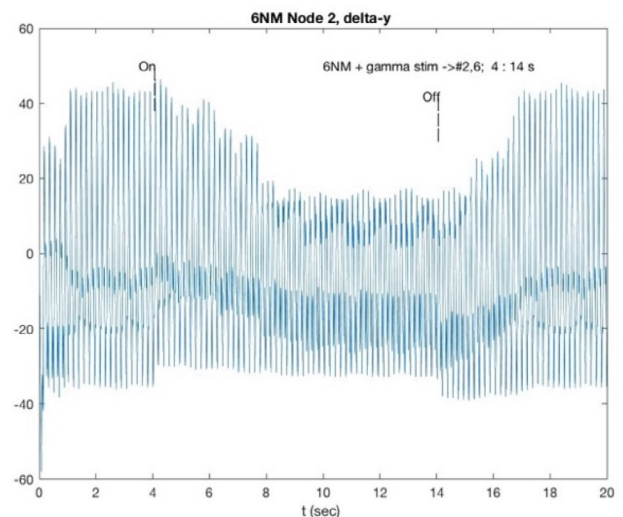
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Network analysis of Marmoset cortex connectivity data reveals an interesting cluster in the Marmoset pre Frontal Cortex (pFC). Link analysis identified a tightly coupled, 3D star shaped cluster of 6 anatomical areas around the Dorso lateral pFC, containing one out- (A10) and two in-hubs (A32V, A11), as measured by participation coefficient. Other nodes are A32, A9 and A46D. Each node was modelled using Jansen-Ritt (JR) neural masses, comprising 3 neural assemblies, following Wilson-Cowan (WC) and JR. This system provides an opportunity to probe how simple oscillator models can elucidate the workings of pFC.

The nodes are heterogeneous in two ways: due to the local link weights, and their characteristic frequency band (following size variation). The sigmoidal voltage-to-firing rate conversion,  $S(v)$ , was modelled using a distribution of synaptic weights (erfc form), rather than firing thresholds (erf), as initially discussed by WC. Simulations of leaky integrate and fire (LIF) neurons (80% excitable and 20% inhibitory) <sup>[following 1]</sup> indicate that resonant frequencies of nodes varied with their size, i.e. number of neurons. Each node had dominant spectral power in one of the theta, alpha, beta or gamma bands. Other model parameters followed with the original WC and JR work. Nodes were connected with directed, weighted links derived from the Marmoset data. Signals were assumed to travel at a velocity of 1 m/s. Model outputs are the time dependent local field potential (LFP) for each node, and the average for the cluster, enriched by interference of the outputs of the linked oscillators. The cluster exhibits a range of oscillations and phase plane behaviour similar to earlier studies of neural masses, with added complexity due to the link weights, signal delays and mixed frequencies. Generally the system is stable in response to constant or simple stimuli. Previous studies showed transitions in response to an ensemble of visual stimuli <sup>[2]</sup>.

The model was stimulated by pulse trains of: constant amplitude; and also amplitude modulated by oscillatory envelopes in the theta to gamma bands. Those represent travelling waves incoming from distant brain regions, as associated with cognitive tasks <sup>[3]</sup>. Targets for the stimuli were: all nodes; the hubs singly, in pairs, and as a triple. The LFP for A32V showed a transition that varied with the nature of the input[s]. This occurred when the two in-hubs were both stimulated, but not individually: effectively functioning as an AND logic gate. A constant stimulus induces a fast transition, over 0.3 s. Modulated stimuli induce slower transitions over 4 - 6 s, lasting longest for the beta band. A low amplitude intermediate steady state lasted 4 - 7.6s, longest for beta. Short stimuli (off at 8.4s) eliminate the intermediate steady state. Off resonance stimuli induced fast changes and short steady state (both < 1s). Turning off the modulated stimulus caused the original LFP waveform to resume, over ~3s (cf. Figure).

The results show that a simple oscillator model can exhibit local state pFC transitions in response to biologically relevant stimuli. Suppressed oscillatory power is consistent with observations during working memory tasks <sup>[4]</sup>.



Example output LFP ( $dy = y_e - y_i$ ) of neural mass modelling A32V in the pFC cluster during the induced transition. Gamma modulated 100Hz pulse stream arrives to A32V and A11 at 4.0 s and off at 14.0 s.

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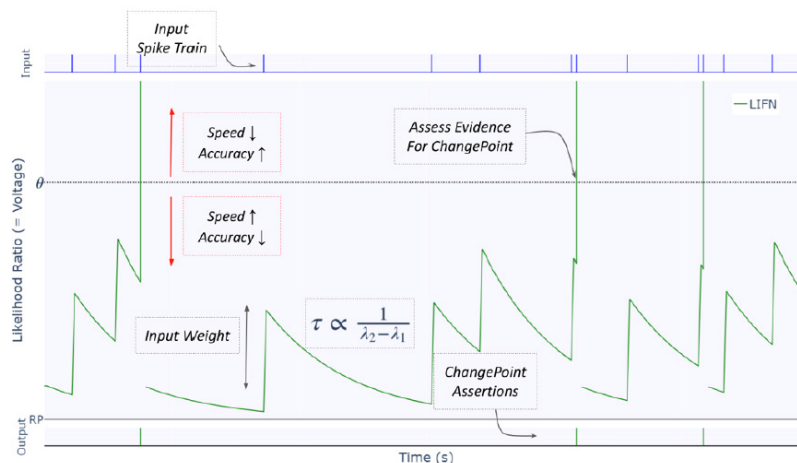
# SPIKING NEURONES AS CHANGEPOINT DETECTORS

Poster Presenter Shivaram Mani<sup>1</sup>

Travis Monk<sup>1#</sup> and André van Schaik<sup>1</sup>

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This figure shows that spiking neurones strongly resemble online changepoint detectors. In online changepoint detection (CPD) problems, we observe samples of data as they are drawn from some distribution (input raster plot above). At some unknown time, those samples are abruptly drawn from a different distribution.

Our goal is to detect that changepoint as we observe the samples<sup>[1]</sup>. For example, we might want to detect the instant that the rate of input spikes changes from one value to another. Online CPD algorithms solve this problem by computing a test statistic of the samples under the hypotheses that they were drawn from one distribution or the other, e.g. a likelihood ratio (green trace). They compare that test statistic with a threshold (horizontal dashed black line), signal when they cross, reset, and repeat.

Similarly, spiking neurones compare their membrane potential with a threshold, generate an action potential when they cross, reset, and repeat. We will show that the membrane potential of the leaky integrate-and-fire neurone model is the likelihood ratio of a family of compound Poisson processes<sup>[2]</sup>. Therefore the leaky integrate-and-fire neurone implements online CPD when its inputs are Poisson-distributed. This result is novel. We suspect that our derivation is just one example of a principle that can generalise beyond simple neurone models.

If real spiking neurones implement CPD on their inputs, then we can offer rigorous and intuitive statistical interpretations of neural membrane potentials, action potentials, resting potentials, synaptic weights, and thresholds. We can also make a novel and testable prediction: the electrophysiology of a spiking neurone is related to the spiking statistics of its inputs. We will derive one example of this relationship and demonstrate it in auditory neurones of the barn owl<sup>[3]</sup>. CPD offers explanations for the basic design and function of spiking neurones and nervous systems.

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# ESTIMATING USER-SPECIFIC CURRENT SPREAD AND NEURAL HEALTH FOR COCHLEAR IMPLANT USERS

Poster Presenter Xiaowei Xia<sup>1#</sup>

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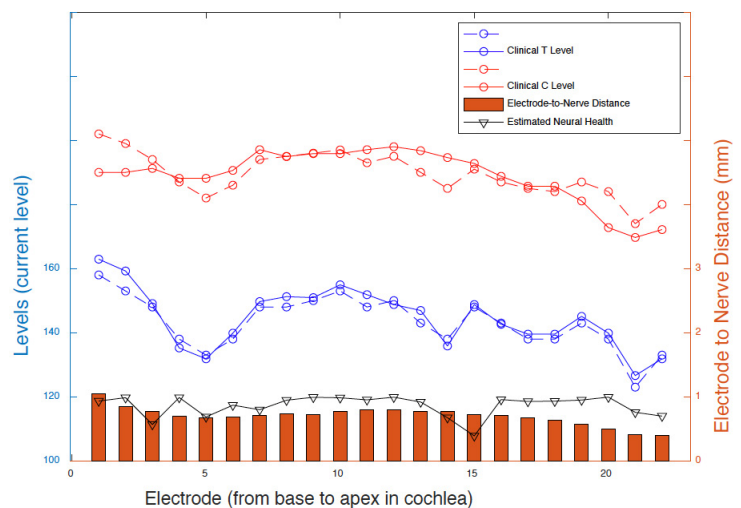
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Individual variability of hearing performance of cochlear implant (CI) users is still very large. The hearing performance of CI users can be potentially improved by adjusting the coding strategies according to the patterns of current spread and neural health in the cochlea. These two parameters are important for the development of better sound coding strategies, but they are user-specific and it can be very time consuming to measure them in the clinical setting. In this research, we developed a method to estimate current spread and neural health in the implanted cochlea using a user-specific computational model and basic clinical psychophysical measurements. We used an existing stochastic model to predict electrically simulated auditory nerve responses [1,2]. User-specific parameters were added to the model to incorporate the neural behaviour for individual subjects, specifically the current spread from each electrode and the distribution of surviving neurons across the auditory nerve. Previously collected measurements from six users [3] were used that comprised threshold levels of current to perceive each electrode (T levels), maximum comfortable current levels (C levels), and electrode to auditory nerve distances.

The model was considered to be delivering T level current on an electrode when 0.04% of nerve fibres were activated, and C level current when 0.1% of nerve fibres were activated. Using a genetic algorithm, we varied current spread and neural health so that the model output matched T and C levels of each user as closely as possible. Across six users, the model provided accurate simulated results with a very low error, giving an average root-mean-square error of 2% between the simulated and clinical measurements.

Figure 1 shows the optimised and clinical T and C levels and estimated neural health for one user. Our method may provide an accurate estimation of current spread and neural health in the cochlea of CI users, which may contribute to the development of individualised CI sound coding strategies for better hearing.



**Figure 1.** Clinical and optimised simulated measurements for one CI user. T and C levels are shown on the left y-axis. Electrode to auditory nerve distances are shown on the right y-axis. Neural health is shown as a proportion, 0-1.





# SEIZURE WARNING SYSTEM USING CRITICAL SLOWING DOWN

Poster Presenter Yueyang Liu<sup>1</sup>

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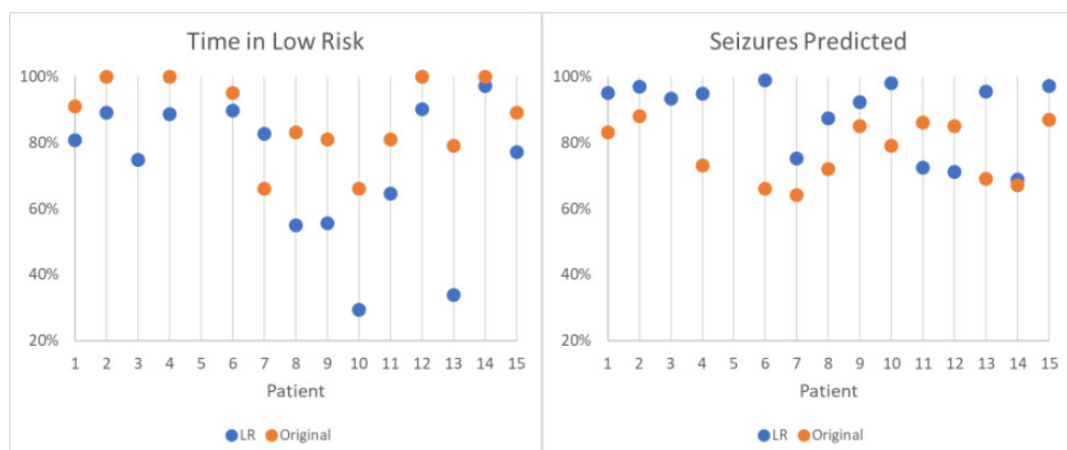
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Considering the human brain as a dynamic system that has the ability to rapidly change state, we can interpret entering epilepsy seizures as a change of state. Brain recordings may show transition signals, called critical slowing down, which exist before transitions of state in dynamic systems. This critical transition might not be detected in short recordings, but can be more accurately detected with the help of long-term data. Following previous research of critical slowing down as a biomarker of impending seizures<sup>(1)</sup>, we extracted these biomarkers as features by doing Hilbert Transformation with autocorrelation and variance for every two minutes from 14 patients with long-term iEEG recordings<sup>(2)</sup> and used logistic regression incorporate with all iEEG channels to train a seizure warning system. Five windows (10 minutes) before the seizure were labeled as pre-ictal stage, and would quantify as success if one of these were successfully predicted. On average across all patients, the warning system predicted 88% of seizures and remained 72% of the time in low risk. The proposed method is more sensitive compared to<sup>(1)</sup>, and can potentially be tuned for patient-specific requirements.



**Figure 1** The logistic regression model is able to predict more seizures than the original results (1), but having a relatively lower time spent in high risk, meaning that the logistic regression model is more sensitive.



# ASYNCHRONOUS DATA PROCESSING FOR EVENT CAMERAS

Poster Presenter Ziwei Wang<sup>1</sup>

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Event cameras, inspired by biological vision systems, capture per-pixel asynchronous intensity change for each pixel without a certain output data rate. Such cameras are ideal for robotics applications since they have high temporal resolution, high dynamic range and low latency. Traditional computer vision methods often accumulate events into image-like pseudo-frames that sacrifices the real-time performance of events. To overcome this, we would like to introduce the following four asynchronous data processing algorithms for event cameras, where each event is processed independently as it arrives. Without accumulating or windowing events, these approaches preserve the rich temporal information and are well-suitable for implementation on low-level hardware, including Field Programmable Gate Arrays (FPGAs) and Application-Specific Integrated Circuits (ASICs).

**Event-Frame Fusion for High-Speed HDR Video Reconstruction<sup>[1]</sup>**: an asynchronous linear filter architecture, fusing event and frame camera data, for HDR video reconstruction and spatial convolution that exploits the advantages of both sensor modalities.

**Event Data Preprocessing<sup>[2]</sup>**: a comb filter to preprocess event data to remove unwanted flicker events from an event stream asynchronously. It achieves over 4.6 times relative improvement in the signal-to-noise ratio compared to the raw event stream due to the effective removal of flicker from fluorescent lighting.

**Optical Communication<sup>[3]</sup>**: a smart visual beacon architecture with both LED modulation and asynchronous event camera demodulation algorithms. It achieves up to 4 kbps in an indoor environment and lossless transmission over a distance of 100 meters at a transmission rate of 500 bps.

**High-Speed Visual Tracking<sup>[4]</sup>**: an asynchronous event blob tracker that achieves highly accurate tracking and event blob shape estimation even under challenging lighting conditions and high-speed motions. The microsecond time resolution achieved means that the output can be used to derive secondary information such as time-tocontactor range estimation, enabling applications to real-world problems such as collision avoidance in autonomous driving.

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# A CENTURY OF THE ALPHA RHYTHM AND ITS RELATIVES: A UNIFIED THEORY AT LAST

**Presenter Peter. A. Robinson<sup>1#</sup>**

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Berger first recorded human EEG on 6 July 1924, noting the  $\sim 10$  Hz alpha rhythm to be the most prominent brain activity <sup>[1]</sup>. Alpha is concentrated over visual cortex at the back of the head, sometimes displays a double peak, and is suppressed by visual inputs <sup>[2]</sup>; the beta rhythm occurs at its harmonic. Later, the  $\sim 10$  Hz mu rhythm was discovered, concentrated over sensorimotor cortex near the crown of the head, suppressed by motor activity, and sometimes associated with  $\sim 20$  Hz activity <sup>[2]</sup>. The  $\sim 10$  tau rhythm is concentrated over auditory cortex near the ears and is suppressed by sound. Early theories argued that separate groups of neurons fire at  $\sim 10$  Hz at the relevant locations, but these were ad hoc and lacked explanatory power <sup>[3]</sup>. More recently, the alpha rhythm was argued to be a natural mode of activity in the cortex <sup>[3]</sup> or of the corticothalamic (CT) system <sup>[4,5]</sup>, and was analyzed using neural field theory (NFT).

Here, we show that just 4 corticothalamic eigenmodes of activity can explain the key features of alpha, mu, and tau rhythms, including frequency structure and topography <sup>[5]</sup>. Splitting is due to eigenmodes having different frequencies, while CT loops account for the basic 10 Hz frequency and correlations between alpha and beta, and between mu and its harmonic. We predict that split-mu, split-tau, and second-harmonic tau rhythms can occur, including split second-harmonic mu and tau; split-beta has already been observed. Spatial peaks are due to constructive interference of modes in the relevant sensory region, supported by enhanced CT gains, and suppressed when those gains are reduced by attention <sup>[5]</sup>.

Several predictions remain to be confirmed experimentally and fits of theory to data will enable brain states to be probed in real time, as is the case for spectra including the basic alpha rhythm <sup>[7]</sup>. Links to evoked responses and other phenomena can also be made via NFT.

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# A RECONFIGURABLE NEUROMORPHIC COMPUTE SYSTEM FOR BRAIN-SCALE SIMULATION

**Presenter André van Schaik**  
**Mark Wang**

International Centre for Neuromorphic Systems, Western Sydney University, Australia

Reverse engineering the brain was chosen in 2008 as one of 14 “Grand Challenges” for the 21st Century by the (US) National Academy of Engineering <sup>[1]</sup>. They consider improving our understanding of how the brain works as one of the most important engineering contributions to science in this century, with applications for building smarter computers and prosthetics and understanding neurological disorders.

Over the past decade, we have made tremendous progress in machine learning and AI, but arguably we are not much closer to understanding how brains achieve their remarkable data processing capabilities. Current machine learning relies on a brute-force statistical approach requiring vast amounts of labelled examples to train the machine and bears little resemblance to how brains learn. Both the training and performance of the task consume large amounts of power and this inefficiency has become an issue <sup>[2]</sup>. Understanding how biological brains can learn these tasks from far fewer examples and do so consuming only 25 Watts will not only allow us to understand our own brains better but also to build better artificial brains.

Progress in understanding the brain is hampered by our inability to simulate neural networks in software on a scale comparable to the human brain, which has 10<sup>11</sup> neurons and 10<sup>14</sup> synapses. The problem is similar to the reason that Artificial Neural Networks failed to deliver their promised gains in the 1990s: the hardware of the time was simply not up to simulating the sizes of networks needed. It was only with the development of Graphics Processing Units (GPUs), multicore CPUs, and cheap, fast memory chips, that researchers overcame this bottleneck; we can now simulate very large Convolutional Neural Networks (CNNs). However, CNNs are very different from the neural networks found in biology. While CPUs and GPUs can simulate bio-inspired neural networks, their performance is far below what is needed <sup>[3]-[6]</sup>. Therefore, a new type of neuromorphic compute system is needed. Several billion-dollar programs in the past decade have aimed to address this need (e.g., US DARPA Synapse, EU Human Brain Project) but none has yet succeeded. This is largely because, when these programs started, the only way to build an electronic brain was to design custom Integrated Circuits (chips) to implement the computations. Custom chips take a large effort and a long time to design and manufacture for every iteration and cost tens of millions of dollars each. This significantly restricts the pace of innovation.

Undoubtedly, custom chips will someday set the gold standard in terms of performance and power efficiency for creating an electronic brain, but this is still a long way off. Instead, we are building a world-first reconfigurable neuromorphic compute system capable of brain-scale simulation using Field Programmable Gate Arrays (FPGAs). The system will support simulating spiking neural networks at 200 trillion synaptic operations per second, which is on par with the estimated number of synaptic operations in a human brain <sup>[7], [8]</sup>. The machine will consist of three racks containing 92 Bittware 520N-MX boards, each with an Intel Stratix 10MX FPGA, 16GB of high-bandwidth memory and 256GB of Intel Optane memory, in Dell host servers.

To enhance the utility of our system, we will make the system remotely accessible and develop a software front-end that allows description of the neural models and design of the neural networks in the popular programming language Python. This front-end will translate these descriptions into a format that can then be used to configure the hardware system directly. The development of this front-end will enable researchers to use the platform without needing detailed knowledge of the hardware configuration.

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The advantages of our approach are manifold: (a) we can use existing commercial hardware in tried and proven settings in modern data centres; (b) the approach is scalable, from using a few devices to simulate a small brain with millions of neurons, to using three full datacentre racks to simulate a human sized brain with 86 billion neurons, and beyond; (c) once proven, the system would be easy to replicate at data centres around the world; (d) future improvements to the hardware architecture, as well as novel neuron models and synaptic learning rules, can be incorporated in the existing hardware simply by reconfiguring it (whereas these are fixed on the custom Integrated Circuits); and (e) neuroscientists and neuromorphic engineers from around the world will be able to perform brain-scale neural network simulation from 2023 onwards already.

The system will be built at and hosted by Western Sydney University, and the research is performed in partnership with the Universities of Sydney and Melbourne, Forschungszentrum Juelich, and with support from Intel, Molex, Dell, and Xenon. It has been funded in part by ARC LIEF grant LE230100034.

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