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PROCEEDINGS OF SONA 2019 (14TH MEETING OF SOCIETY OF NEUROSCIENTISTS OF AFRICA/17TH MEETING OF NEUROSCIENCE SOCIETY OF NIGERIA)

Plenary Speakers Abstracts

IBRO KAVLI LECTURE

S1

Space and time in the brain

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The ability to map space is critical to survival. Without it, animals would not find food or partners and they would be eaten by predators. Neural circuits for finding oneś way thus exist in all animals. In mammals, space is mapped by complex neural networks in the hippocampus and entorhinal cortex. These brain areas contain specialized position-coding cell types, including grid cells, which we discovered in the medial entorhinal cortex in 2005. Grid cells are active only when animals are at certain locations, locations that tile environments in a periodic hexagonal pattern. More recently, we have found that the medial entorhinal cortex contains a further variety of specialized cell types, including not only grid cells but also cells that monitor direction, speed and local borders. I will present these cell types and their properties in the first part of the talk. I will further show that when spatial behavior is tested in environments with salient objects or landmarks, a hitherto undescribed subset of medial entorhinal cells fires in a vector-like manner at distinct distances and directions from objects in the recording enclosure, irrespective of where in the enclosure the object is located, and irrespective of the identity of the object. The number of vector-coding cells is comparable to that of grid cells in the same region. In the second part of the lecture, the focus will be on the representation of time, which is less well understood than space. I will show how episodic temporal information is encoded across scales from seconds to hours within the overall population state of the lateral part of entorhinal cortex. I will also demonstrate that the representation of time in this region depends on the structure of experience and so may diverge from clock time. In the hippocampus, the task-dependent representation of time in lateral entorhinal cortex may be integrated with spatial inputs from medial entorhinal cortex, allowing the hippocampus to store a unified representation of experience. Deficiencies in the function of the hippocampal-entorhinal system may be at the core of neurological diseases where spatial orientation is affected, such as Alzheimerś disease.

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James kimani lecture

S2

The redox-sensitive APE1 is a master cellular regulator for inflammatory pain condition

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Inflammatory pain is a chronic pathology characterized by maladaptive plasticity that relies on functional reorganization of dorsal horn networks. In the central nervous system, the inappropriate response after neuronal damage depends on alterations in synaptic transmission and neuronal connectivity of local microcircuits. Chronic inflammatory pain is associated with global changes in the gene expression of damaged neurons. To understand molecular mechanisms underlying inflammatory pain, it is essential to elucidate how gene expression is altered and how these changes contribute to the development and maintenance of chronic pain. Interestingly, oxidative stress has been identified as a hallmark of neurological disorders, including chronic pain and it contributes to cell death after nerve injury. The oxidative stress sensor, apurinic/apyrimidinic endonuclease/redox factor (APE1/Ref–1) is a ubiquitous multifunctional protein involved in the DNA base exci-

sion repair (BER) pathway. APE1 is strongly upregulated in various cell types following oxidative stress.

Until now there has been no data on APE1 expression and function in nervous tissues after induction of chronic pain. Therefore we studied APE1 expression and subcellular distribution in an inflammatory pain model produced by Complete Freund's Adjuvant (CFA) injection. We demonstrated that despite a global decrease in APE1 expression under inflammatory conditions, APE1 redox function accounts for pain sensitization by blocking selectively APE1-redox activity under inflammatory conditions using €-3-[2-(5,6–dimethoxy–3–methyl–1,4–benzoquinonyl)]–2–nonylpropenoic acid (E3330). Indeed, E3330 treatment causes impairment of APE1 redox activity and subcellular localization, thus enhancing nuclear localization. In the rat CFA model, we demonstrated that E3330 exerts potent anti-inflammatory effects through the inhibition of APE1 redox activity and subsequent suppression of down-stream transcription factor activation (e.g. NF-Kb) and inflammatory molecule production. E3330 injection results in pain alleviation in inflammatory rats. Further studies are still in progress to decipher the possible mechanism(s) underlying this APE1 regulatory effect.

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S3

Why study brains and behaviour of different mammals?

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The majority of neuroscience research is 1 ocused on 3 species, rat, mouse and human. The general aim is to understand the basis of human neural function and dysfunction, and to develop therapeutics or interventions; however, over 80% of potential therapeutics developed in rodent models do not translate to the human condition, i.e. these rodent models are inefficient. Comparative neuroscience research does not directly aim to discover cures to human neural dysfunctions, rather, comparative studies: (1) may guide the translation of data generated in rodent models to humans; (2) may reveal more appropriate model animals for the study of specific human neural dysfunctions; (3) provide data relevant to understanding the animals under investigation that may lead to improved conservation and management strategies; and (4) may inform us of the processes that have led to the evolution of the human brain, enhancing our understanding of ourselves and our existence. The African continent houses 25% of the world's Eutherian mammal species, making the mammalian neural biodiversity of the continent a great natural resource for understanding processes of brain evolution, with the potential to reveal model animals more suitable for use in the investigation of human neural dysfunction than laboratory rodents. Here I will present two examples of ongoing from research in my laboratory demonstrating how comparative research can: (1) greatly benefit human health on a global scale; and (2) revolutionize conservation efforts on a continental scale. Novel observations regarding sleep in large diurnal mammals have led to a new understanding of the timing of sleep, how this relates to the environment, why we sleep too much in the industrialized world, and how we may improve sleep and alleviate symptoms of sleep disorders. The brain of the plains zebra indicates that zebras are the key species of the mass migrations of large mammals on the African savannahs. Understanding the plains zebra brain, how this relates to migratory behaviours, and expanding our knowledge of plains zebra migrations in Africa can lead to major changes in the way conservation measures are implemented, to the benefit of all flora and fauna dependent upon these migrations for survival.

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S4

Stroke: from neuroprotection to neurorehabilitation

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Background: Stroke is a devastating disorder that affects approximately 15 million people worldwide. One in Six people worldwide will have a stroke in their lifetime. Over the last four decades, the stroke incidence in low and middle-income countries has more than doubled. Despite the high prevalence of ischemic stroke, only limited therapeutic options exist. Restoring blood flow using clot-dissolving drugs remains limited to a narrow window. More, administrating those compounds can cause serious side-effects like bleeding. An emerging view favors combinational therapy to stimulate protective and regenerative mechanisms.

Summary: We demonstrated that alpha-linolenic acid (ALA is an essential omega–3 polyunsaturated fatty acid required as part of our daily diet), has neuroprotective effects against stroke damage. Its administration increases neurogenesis, synaptogenesis and neurotrophin expression. Mice fed with an ALA-enriched diet displayed reduced mortality rate, infarct size and increased spontaneous reperfusion after ischemia. Our work provides new insights into the potential of omega–3 polyunsaturated fatty acids as nutraceuticals in stroke prevention and protection. Upon stroke, the nervous tissue degenerates rapidly and is often difficult to rescue. Therefore, it is important to improve recovery and rehabilitative interventions. To be transferable to the patient, these interventions must be well-designed. Understanding how the injury evolves in a particular animal model will enable us to administer a given intervention within the appropriate time window, thereby improving efficacy and transferability. We have studied the time course of neuronal death and brain injury in the endothelin-1 stroke model in rats. We evaluated the potential of enriched rehabilitation to enhance recovery of post-stroke motor and cognitive deficits. We show that enriched rehabilitation, which combines enriched environment with tailored reaching therapy, enhances stroke recovery by stimulating neuroplasticity processes. Additionally, alternate and moderate wheel running, which promotes hippocampal neurogenesis, can be implemented in the rehabilitative battery as part of a combinational therapy post-stroke.

Key message: The fine-grained understanding of the pathophysiology of stroke and the implementation of combined neuroprotective and neurorestorative approaches are opening new avenues for stroke treatment.

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S5

Is nodding syndrome an *onchocerca volvulus* associated epilepsy?

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Human onchocerciasis, caused by infection by the filarial nematode *Onchocerca volvulus,* is a major neglected public health