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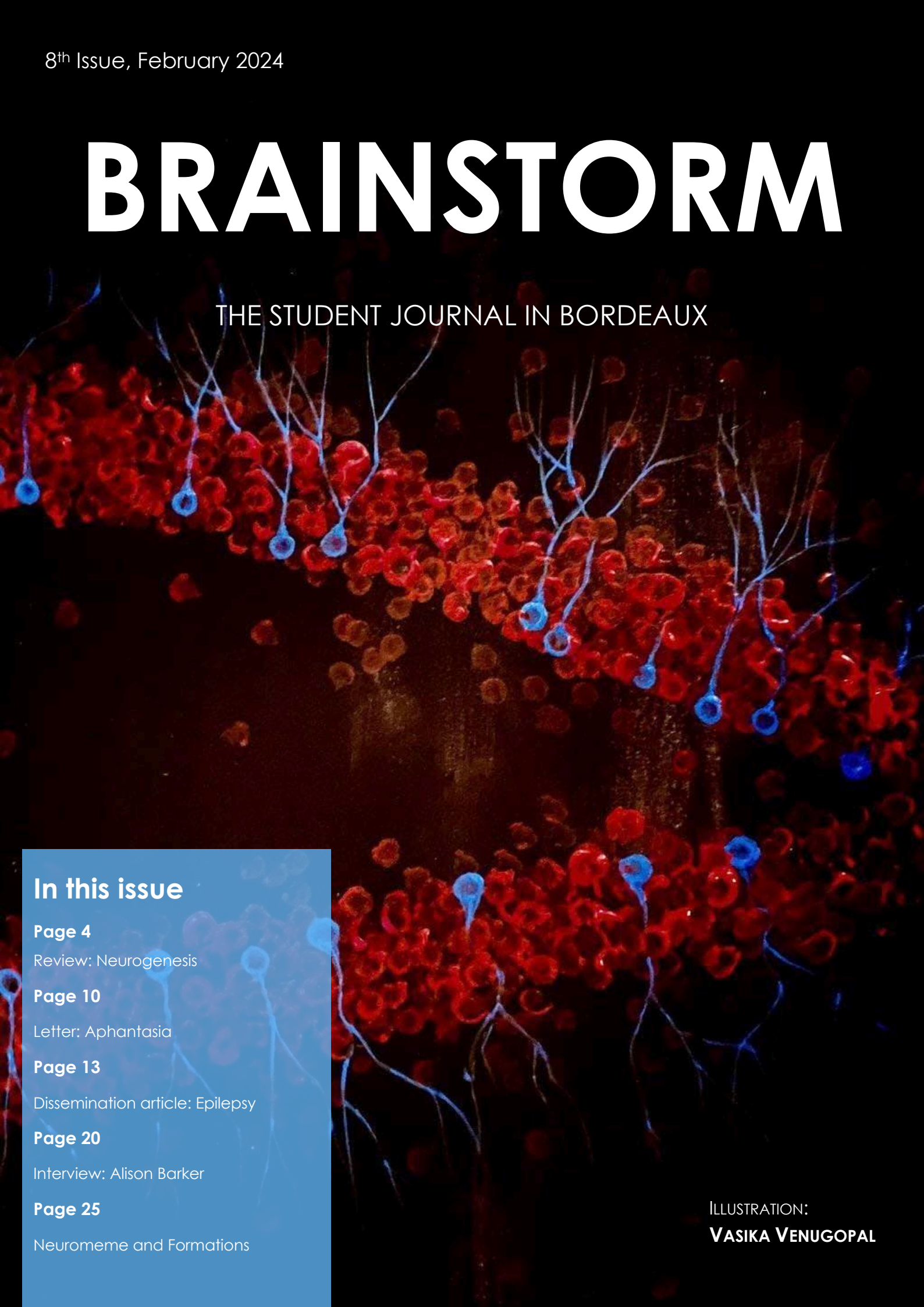

In this issue

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BRAINSTORM STUDENT JOURNAL

REVIEW

Adult neurogenesis in emotion: an overlooked mechanism for the study of mood disorders?

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Abstract

The discovery of adult neurogenesis unrevealed the brain's ability to self-renewal and adaptability to the ever-changing environment. Seemingly, the neurogenic processes occurring in the dentate gyrus, as part of the hippocampus, are of great importance due to its functional relevance in cognition and emotion. Within the scope of emotional processing, adult hippocampal neurogenesis (AHN) has been shown to be pivotal in anxiety, to mediate the action of anti-depressants and in the origin of stress-related behaviors. Therefore, AHN appears itself as a candidate target to investigate the mechanisms underlying neuropsychiatric disorders and as a potential therapeutical axis for more specific and effective treatments.

Keywords

Adult hippocampal neurogenesis, emotion, neuropsychiatric disorders, and therapeutical axis.

Abbreviations

abDGCs: adult-born dentate granule cells

AHN: adult hippocampal neurogenesis

ATD: anti-depressants

DG: dentate gyrus

EC: entorhinal cortex

5HTR: serotonin receptors

GR: glucocorticoid receptors

HPA: hypothalamus-pituitary-adrenal axis

MR: mineralocorticoid receptors

NMDAR: N-methtl-D-aspartate receptors

NMDAR: N-methtl-D-aspartate receptors

Introduction

Once perceived as an immutable structure, the brain was discovered to be plastic. The fascinating discovery of newly generated cells in the adult brain of rats by Joseph Altman challenged the common belief that after development new neurons and networks cease to appear (1). Indeed, neurogenesis is described as a neural developmental process but was confirmed to continue throughout life giving place to the continuous generation of progenitor cells (2). This process denominated adult neurogenesis relies on the proliferation, survival, and differentiation of progenitor cells into adult-newborn neurons that further integrate into established neuronal networks.

In mammals, new neurons were shown to be generated principally in two regions: the subventricular zone of the olfactory bulb, and the subgranular zone of the dentate gyrus (DG) in the hippocampus. Within these regions, a neurogenic niche composed of stem cells, neurotransmitters release and inputs received from other regions set the rate of proliferating and surviving cells as well as the maturation of newborn neurons. Added to intrinsic factors, environmental stimuli can change the tune of neurogenesis either by improving or disrupting its dynamics and networks (2).

Due to the relevance of DG in physiology and pathology of the human brain, a lot of attention has been given to adult hippocampal neurogenesis (AHN). Overall, this process has been described as a structural and functional plasticity mechanism important not only for the maintenance of physiological functions in memory processing (spatial learning, pattern separation, context discrimination…), but also in mood regulation (anxiety and anti-depressant action) (3). Additionally, the continuous generation of new neurons in the adult brain translates into functional adaptability since this process is regulated by internal and external factors. This process results in an adaptive response of the brain to the ever-changing environment.

In the scope of mood regulation, these adaptive responses can predispose vulnerability, since environmental factors detrimental for mental health, such as chronic stress exposure, are known to impair neurogenesis. Ultimately, this can affect hippocampal-dependent emotional functions (4). Concomitantly, neurogenic-potentiating factors can reverse these negative behavioral outcomes by increasing the number of surviving newborn neurons and their differentiation, thus restoring the loss of function. This is the case for anti-depressants, since they have been shown to potentiate neurogenesis, which is associated to their positive behavioral effects.

Therefore, AHN potential for self-renewal, functional relevance and adaptive responses highlights this mechanism as a utopic scenario for the mitigation of pathological conditions in neuropsychiatric disorders. In this review, the aim is to bring to light the functional relevance of AHN in emotion and the neurogenic therapeutical potential for a more specific and effective treatment of anxiety and depression.

Methods

The included studies were found using the Web of Science and PubMed search using different combinations of the following keywords: adult hippocampal neurogenesis, emotion circuits, anxiety, stress, anti-depressants, fluoxetine, memantine.

Results

AHN in emotional circuits

The dentate gyrus, an integral component of the hippocampal formation, serves as a crucial entry point for processing multi-sensory information originating from cortical regions. This involvement contributes significantly to both cognitive and emotional processing (5,6).

Traditionally, the circuitry of adult-born dentate granule cells (abDGCs) is characterized by unidirectional glutamatergic inputs originating from the entorhinal cortex (EC). The circuit connecting the EC to DG has been implicated in depression, as evidenced by optogenetic stimulation leading to antidepressive effects in animal models (7). Moreover, in addition to glutamatergic inputs, DG also receives monoaminergic projections from downstream structures, namely brainstem. Among these, the serotonergic projections of the raphe nuclei are known to be particularly relevant for mood regulation (5).

Lastly, the DG assumes a role in the stress response owing to its sensitivity to stress hormones. The presence of glucocorticoid and mineralocorticoid receptors (GR and MR) on the surface of mature newborn cells, specifically dentate granule cells (DGCs), underscores its involvement in regulating the negative feedback mechanism of the hypothalamus-pituitary-adrenal (HPA) axis (8). Overall, the sensitivity of DG to inputs and molecules crucial for mood regulation places it within the scope of the emotional circuit in both physiological and pathological contexts.

AHN in emotional functions: physiology and pathology

As a matter of fact, clinical studies suggest that patients suffering from neuropsychiatric disorders present impaired AHN observed by decreased hippocampal volume, impaired hippocampus-dependent functions, and decreased cell proliferation (9). In animal models, AHN has been shown to play a role in depressive- and anxiety-like behaviors, but also for contributing to the emergence of stress-related behaviors.

This was first revealed by the neurogenic-mediated action of mood stabilizers that concomitantly increased neurogenesis. The chronic administration of anti-depressants drugs (ATD) for 14 to 28 days successfully potentiated cell survival, proliferation and differentiation of newborn cells of the DG in adult rats, suggesting its neurogenic effect (10). Further on, the ATD-mediated neurogenesis was translated into the anti-depressant behavioral effects in mice. The administration of different types of ATD (namely fluoxetine, imipramine and desipramine) produced a neurogenic effect that was accompanied by a decrease in depressive-like behaviors (11). Importantly, after abolishing neurogenesis by X-ray irradiation restricted to the DG, these behavioral effects ceased to be reproduced, suggesting the AHN-mediated action of ATD. Moreover, its role in anxiety was shown by a correlation between depleted neurogenesis and anxiogenic phenotypes. Using a mouse model to specifically ablate newly generated neurons during a 6-week period followed by behavioral testing, anxiety-like behaviors were observed given by decreased time spent in anxiogenic regions of classical anxiety tests, namely the open field test and elevated plus maze (12).

Additionally, AHN as a plasticity mechanism highly sensitive to environmental stimuli has been shown to be responsive to susceptibility factors related to mental disorders, such as stress exposure. In this line of thought, several studies addressed the impact of chronic stress in neurogenesis by exposing animal models to stressors of different nature. Its exposure during pre- and post-natal period have consistently shown an impact in AHN by decreasing the level of cell proliferation, survival and differentiation. Prenatal stress imposed by restraint of pregnant female mice during the last week of gestation were shown to induce both anxiety- and depressive-like phenotypes (13,14) accompanied by decreased cell proliferation and number of mature DGCs, respectively.

Altogether, the abDGCs appear to be relevant in mediating functions that are known to be impaired in neuropsychiatric functions, and as they can be at the center of the etiology of some neuropsychiatric conditions, abDGCs deserve more attention in the field.

The potential of newborn neurons as a treatment of mood disorders

Due to the correlation between stress-meditated effects of neurogenesis and mental illness, AHN has become a target mechanism for the treatment of mood disorders. As mentioned above, the neurogenic-mediated action of antidepressants was revealed to be effective for increasing neurogenesis but also for allowing to overcome negative behavioral outcomes of depression. These

ATD are described as Selective Serotonin Reuptake Inhibitors (SSRIs) that seemingly increase levels of serotonin that act upon serotonin receptors (5HTR) expressed by mature DGCs. Specifically, the type 1A (5HT1AR) seem to mediate the neurogenic effect of ATD and its anti-depressive like behavioral effects. In a study by Samuels and colleagues (15), specific depletion of 5HT1AR on mature DGCs abolished the neurogenic-mediated effect of ATD. This study also proposed that SSRIs boost neurogenesis by stimulating the secretion of neuroprotective growth factors, thus enhancing survival and differentiation of adult-newborn neurons.

Other drugs with neurogenic and neuroprotective potential have been proposed for treatment of mood disorders. Memantine, an N-methtl-D-aspartate receptor (NMDAR) antagonist commonly used for the treatment of Alzheimer's Disease was proven to restore impaired AHN in models of neurodegeneration (16), while recently it has been suggested as a therapeutic approach for anxiety disorders (17).

However, the mechanism behind the neurogenic potential of these drugs remains an open question. Further insight could contribute to a better understanding of the underlying molecular and cellular processes on the origin of the emotional impairments associated to neuropsychiatric disorders. Ultimately, mood stabilizer drugs with a neurogenic potential could contribute for a more specific targeted therapeutical approach.

Discussion

AHN is a fascinating mechanism not only for its utopic scenario of cell self-renewal and plasticity but for mediating pivotal function in cognition and emotional behaviors regulated by a tight interaction with biological and environmental factors. Specifically, its role in mood regulation should be given more attention due to its functional relevance and to the potential of newborn neurons in regenerating loss of function.

In physiology, AHN is inserted into the emotional circuitry, being connected to upstream and downstream structures implicated in mood regulation, notably the EC and the raphe nuclei But also, for participating in anxiety behaviors and regulating the negative feedback of stress response. In pathology, AHN dysfunction seems to be at the heart of the etiology of stress-related disorders, as shown by the correlation between stress-mediated impairment of neurogenesis and the observed behavioral deficits. All and all, AHN has been implicated in pathological conditions of neuropsychiatric disorders, notably in stress-related disorders, such as depression and anxiety. This evidence highlights neurogenic treatments as promising therapeutical strategies for mood disorders and as useful to identify the underlying molecular mechanisms, as these treatments suggest the implication of serotonergic system and the glucocorticoid receptors mediated by adult-newborn neurons. At the same time, the impact of stress at different life stages could contribute to a better understanding of the etiology of mental disorders. By exposing animal models to stress at different developmental stages (early postnatal, adolescence or adulthood) and therefore targeting different periods of neurogenesis, would allow to pinpoint the critical time windows for emergence of mental disorders.

Conclusion

Understanding brain's structural plasticity and ability to adapt to the presented challenges can be useful for restoring loss of function as a therapeutical approach in human pathologies, showing that adult neurogenesis deserves more attention.

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LETTER

Imagination Without Visual Imagery: Journey into the World of Aphantasia

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How I found out I have Aphantasia

For most of my life, whenever someone asked me to close my eyes and visualize something, I interpreted it to mean as remembering it, sensing it, or experiencing it. It surprised me that visualization was a literal term and that many people are capable of seeing images when they close their eyes.

The first time I learned about aphantasia was through an internet video I watched during my twenties and it blew my mind! I felt like everyone else except me had a superpower, when I realized others could use their imaginations to create visual images. Immediately, I felt compelled to learn more about this condition.

What is Aphantasia:

Visual mental imagery implies the ability to produce a quasi-perceptual visual representation with the mind's eye. When people are asked to imagine an object, such as an apple, most individuals can mentally recreate a vivid picture. However, the definition and intensity of this mental imagery may differ among subjects. Some people are incapable to create any visual imagery at all and they are defined as aphantastics. Aphantasia is a primarily congenital, lifelong neurodevelopmental condition where visual mental imagination is either entirely absent or weak and unclear (1,2). Studies have recognized several comorbidities and tracts associated with aphantasia (1,2,3,4,5).

It's worth to note that aphantasia represents a spectrum of conditions, with subjects experiencing different levels of ability in creating mental images. Moreover, some people experiencing aphantasia can still have sensory-based imagination related to sounds, tastes, or smells.

Hypothesis have been raised suggesting that aphantasics might not experience visual imagery deficits, but rather an impaired ability to report their visual imaginations. This would imply a lack of metacognition, the capacity of exactly introspect about our thoughts (6).

From this point of view, the concept of aphantasia would represent only a failure in reporting and comparing one's own imagery to others.

Nevertheless, this hypothesis does not take into account that aphantasia can also be an acquired condition, with subject that are still remembering how their previous experiences of visual imagination was. For instance, this is the case of the individual MX, which experienced the onset of aphantasia in a late phase of life, as consequence of a surgical intervention of coronary angioplasty (7).

In the past, different attempts to evaluate the prevalence of aphantasia have given variable results, mostly due to methodological limitations.

In the recent times, to solve this point the Vividness of Visual Imagery Questionnaire (VVIQ) has been established as common and reliable methodology.

The VVIQ represent a 16-item measure that require subjects to create several visual images and then rate them with a 5-point scale, as follow *(5) perfectly clear and as vivid as normal vision, (4) clear and reasonably vivid, (3) moderately clear and vivid, (2) vague and dim, or (1) no image at all, you only 'know' that you are thinking of the object.*

Figure 1. VVIQ scoring. Source: Aphantasia – "Mind-blindness" – The inability to visualize mental images – Cascading Insights

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The VVIQ represent a robust way to measure visual imagery validated by resting state fMRI data. Subject characterized by aphantasia present a lower connectivity between frontal and visual areas of the brain; It is consistent with parallel measures, such as questionnaire and behavioural (pattern glare) tasks for sensory sensitivity (3).

An overall VVIQ score of 16–32 constitute the pure aphantasia condition, in which visual imagination is almost totally absent (1). However, aphantasics often report vague and scattering 'flashes' of visual imaginations. While the exact range of phenomenologies is yet to be charted, a test-score of 16–32 captures the absent or highly impoverished imagery of aphantasics, so we adopt this here. Although a small number of studies have used other thresholds (e.g.,23 or 25,2,4), it has been suggested that a score of 16–32 would represent a robust and conservative standard for future studies.

We can then conclude that Aphantasia is defined as a condition where visual mental imagination is either entirely absent or weak and unclear, although other forms of sensory imagination can still be present.

Most of the people experiencing aphantasia (including myself) realize it only late in life if not at all…perhaps our readers will be now thrilled by the idea of taking the VVIQ test!

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DISSEMINATION ARTICLE

Epilepsy – what do we know so far?

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This section has been created in collaboration with the Maison du Cerveau, an association that brings together all those involved with diseases from the nervous system. Our goal is to increase visibility and to provide information about these pathologies, treatments, and research advancements for the general public.

The history of epilepsy is a long and ancient one, with the disease itself having been misjudged and misdiagnosed for the majority of it. One of the oldest descriptive accounts of epilepsy were retrieved from a Babylonian clay tablet[i] which dates to around 6th century B.C, and opens with the text (1):

'If epilepsy falls once upon a person or falls many times, it is the result of possession by a demon or departed spirit.'

While the historical artifact, discussed by Wilson and Reynolds in their paper in 1990, offers insight on how epilepsy was perceived in that era, what is remarkable is that some of these ancient descriptions of seizures do not seem very different from those we find presently. For example, one such extract goes to detail what is now recognized as a focal seizure (2):

'If at the time of his possession, while he is sitting down, his (left) eye moves to the side, a lip puckers, saliva flows from his mouth, and his hand, leg and trunk on the left side jerk (or, twitch) like a (newly)-slaughtered sheep, it is miqtu[ii]'

Still, needless to say that present understanding of seizures and epilepsy has greatly evolved since then – significantly after the introduction of EEG in the 19th century and the 20th century advances in cellular and molecular techniques.

Global and national burden of epilepsy

According to a WHO report of 2023, 50 million people globally suffer from epilepsy, making it the fourth most common neurological disorder. In France, about 650,000 people are estimated to be affected by this disease, which roughly corresponds to 1% of the population (3). Despite its prevalence and lengthy history, full understanding of it remains elusive, and in some parts of the world it is still grossly misunderstood to be a condition of a spiritual or demonic disposition.

i. Tablet belonging to The British Museum's "Babylonian Collection" which is one (#26) of the forty tablets belonging to the ancient medical texts known as 'Saikikku' or 'All Diseases' (1).

ii. Miqtu, meaning 'falling sickness', is the Babylonian term for 'epilepsy'(2).

What is epilepsy and how does it arise?

The definition of 'epilepsy' was last updated in 2014, and is characterized as a disease in which the person 'demonstrates a pathological and enduring tendency to have recurrent seizures (4). An 'epileptic seizure' is defined as a 'transient occurrence of signs and/or symptoms due to abnormal excessive or synchronous neuronal activity in the brain' (5). In other words, a seizure is a clinical presentation of epilepsy, caused by an abnormal activation of neuronal clusters in the brain. This abnormal firing of neurons is generally recognized to be due to an imbalance of the excitatory (glutamatergic)/inhibitory (GABAergic) transmission in the brain (6).

Most studies on epileptogenesis shows that the two key phenomena at play for most types of epilepsy seem to be (1) the hyper-excitability of neurons and (2) their hyper-synchronization. The hyper-excitability of a neuron is its increased tendency to be activated by a particular stimulus, such as sound, light or touch (depending on the affected brain region). When this hyper-excitability leads to a point where the excitation outweighs inhibition, it can result in these neurons firing rapidly and repetitively in a cycle that is sustained by positive-feedback (7). For this hyper-excitability to trigger a seizure, a significant population of neurons needs to be firing simultaneously at a similar rate (in synchrony) – meaning a hyper-synchronous neuronal hyper-excitation. Various studies describe alterations in neuronal morphology and complex cellular interactions (8) to be behind transient atypical, synchronous neuronal firing patterns (such as abnormal burst firing) that bring about a seizure.

Because numerous molecular and cellular processes dictate neuronal function, the factors that could disrupt the equilibrium are just as many. According to a 2011 classification, epilepsy can fall under four main groups based on its etiology (9):

1. Idiopathic epilepsy: This is a subclassification of generalized epilepsy and concerns epilepsies that do not present structural abnormalities or any indications of the disease through diagnostic imaging. Idiopathic epilepsy is considered to be (or likely to be) of mainly genetic presentation. Some types of epilepsy that fall under this category include: childhood absence epilepsy (CAE), juvenile absence epilepsy (JAE) and juvenile myoclonic epilepsy (JME).

2. Symptomatic epilepsy: It is defined as epilepsy that is a result of an injury to the brain, and is associated with notable neuroanatomical abnormalities or pathological anomalies in the brain. Examples of epilepsy in this subclass include those caused by an infection of the central nervous system (CNS), brain tumor, head injury, or other neurodevelopmental and congenital conditions. Examples in this class include early myoclonic encephalopathy, epilepsy with myoclonic absence, West syndrome, and Lennox-Gastaut Syndrome.

3. Provoked epilepsy: This concerns epilepsy which has its presentation rooted in a systemic dysfunction or an external factor, with no remarkable structural or pathological changes of the CNS. This subcategory includes reflex epilepsies (having a mostly genetic character) and are known to be triggered by environmental factors, internal mental condition or a combination of both.

4. Cryptogenic epilepsy: This category deals with those epilepsies that are assumed to have a symptomatic presentation, but with no identifiable cause – or in other words, unknown epilepsy.

Epilepsy – Understanding the seizure types

Epilepsy is a heterogenous disorder with a multifactorial origin. This makes classification of epilepsy quite challenging. Without fully understanding the characteristics of seizures (since an epilepsy type can have more than one kind), it can be tricky to navigate around the treatment options in order to manage seizures, while minimizing adverse effects. Among other reasons, this led to a more recent classification (see Figure 1) established by the International League Against Epilepsy (ILAE) based on (10):

1. Onset of seizure, whether it is localized, generalized or unknown

2. Features, such as awareness and specific motor or non-motor behaviors that manifest

3. Other symptoms associated with the seizure such as degree of body movement

Focal onset seizures

Focal onset seizures, otherwise known as partial seizures, are abnormal neuronal activity occurring in a localized area of brain, in one of the brain hemispheres. These types of seizures are further classified depending on whether the onset is motor or non-motor and level of awareness during the seizure. Non-motor features can be hallucinations (cognitive seizure), hot or cold flashes, goosebumps, panic (emotional seizure) accelerated heartbeat, tingling of the skin (sensory seizure) and unresponsiveness (behavior arrest seizure). Depending on the type of motor behavior, a focal onset seizure could be classified as (5,10,11):

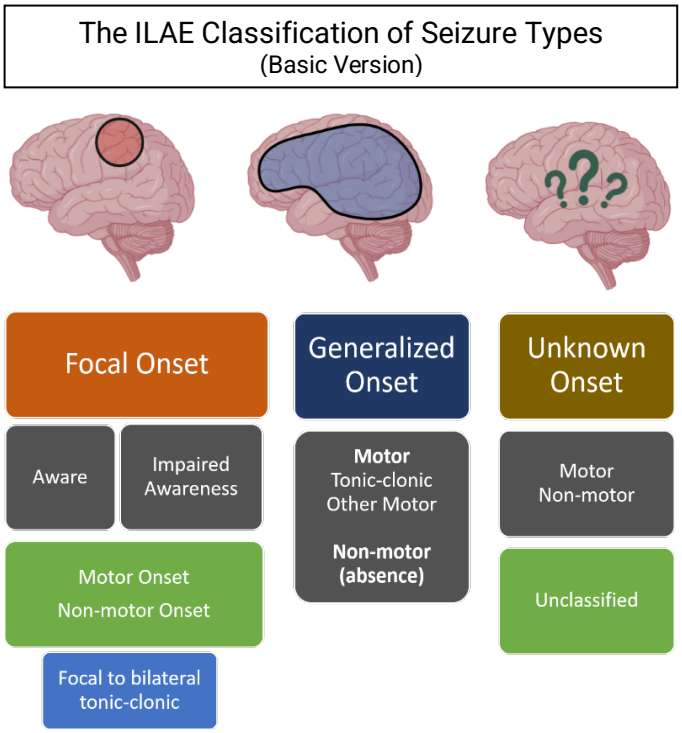

Figure 1 ILAE 2017 revised classification of seizure types. According to the revised guidelines, seizures are classified under on their onset into three types: Focal, Generalized and Unknown.

- Tonic seizures: abnormal and persistent contractions of the muscles which can present itself as stiffness in the limps and posture

- Clonic seizures: sustained twitching or rhythmic jerking of the limbs due to brief, recurring contractions of the muscles

- Atonic seizures: loss of muscle tone in a limb which can result in a person 'falling limp'

- Myoclonic movements: un-sustained irregular, non-rhythmic twitching of the limbs as a result of muscles rapidly contracting and relaxing in one side of the body

- Automatisms: coordinated and repeated occurrences of specific motor actions such as swallowing, rubbing, clapping or tapping

- Hyperkinetic seizures: commonly found to be a consequence of abnormal activity in the frontal lobe, these seizures are characterized by agitated movements such as thrashing and kicking

- Epileptic spasms: abnormal movements seen in children that are defined by abrupt flexions at the waist and limbs

Generalized onset seizures

Generalized onset seizures are abnormal neuronal activities that occur simultaneously in both hemispheres of the brain and are categorized as: motor or non-motor/absence based on seizure types. Generalized seizure also differ from focal ones in that there is an impairment of awareness associated with it (12).

Generalized onset non-motor seizures (or 'petit mal' seizures), typically feature a brief period in which a person is idle (or seems to have spaced out). These seizures can include a myoclonic type where a 'blank stare' is accompanied by sudden twitching or jerking of certain muscles.

Generalized onset motor seizures, previously known as 'grand mal' seizures, can be epileptic spasms, tonic, clonic, myoclonic, atonic, tonic-clonic, clonic-tonic, myoclonic-tonic-clonic or myoclonic-atonic. The last four mentioned are classified based on which type of phase precedes the next. For example, the myoclonic-tonic-clonic seizures are characterized by twitching arms, then a tonic rigidness, followed lastly by a rhythmic clonic jerking; whereas, the myoclonic-atonic seizure is described as transient jerking of the limbs or chest, and a subsequent limpness (10,13,140).

Unknown onset seizures

As the name suggests, these include all the seizures for which the origin is hard to establish. This can be the case for seizures that occur in the sleep or when the individual is alone without anyone to witness describe the clinical presentation of it (10). These can be motor, non-motor or unclassified due to lack of information. It is important to note this category does not aspire to characterize seizures, rather is a way to classify seizures that could not be placed in the other two categories.

How is a diagnosis established?

Establishing a correct and precise diagnosis for epilepsy is essential in order to treat and manage the disorder. This is another reason why the classification of epilepsy is constantly revised and updated as new information is uncovered. In order to make an accurate diagnosis, healthcare professionals will need maximum information pertaining to the seizure (what happened before, during and after the event). Here, descriptions of the symptoms by a witness can be essential in identifying the seizure type.

The diagnostic process also includes a thorough review of an individual's medical history, and several tests, such as: a complete neurological examination, brain scans, blood tests, and sometimes a genetic test. Usually, an electroencephalogram (EEG) is also performed, which is a non-invasive test that allows the recording of the brain's electrical activity. Since epilepsy is marked by an altered neural activity, an EEG can be useful to detect these changes and record any possible seizure. These can allow professionals to determine the epileptogenic zones (area of the brain heavily implicated in seizure generation) and recognize the type of change in brain activity (such as in neural firing patterns) for them to be able to decide the best treatment procedure to adopt.

Common therapeutic approaches

While epilepsy does not have a solid cure, there exist various strategies for affected individuals to manage seizures. Certainly, the approaches will vary from person to person, but the first step is generally anticonvulsants or anti-epileptic drugs (AEDs). These differ based on the mechanism of action and can be calcium channel blockers, sodium channel blockers, glutamate antagonist, GABA potentiators or have combination of these mechanisms15. Depending on the type of seizures, AEDs would be prescribed as a monotherapy or as combination therapy.

AEDs can be an effective means of controlling seizures in approximately 70% patients with a recent onset of epilepsy, but 30% of the affected population continue to suffer from drug-resistant and uncontrolled seizures. This can have severe consequences on their quality of life, with loss of independence, depression and risk of injury (16,17). An epileptic surgery is usually considered when at least two AEDs are ineffective in obtaining seizure control in patients when the epileptogenic zone is identified (such as can be done for lesional epilepsy). The goal is to remove the brain area where the seizure originates to prevent their occurrence or to reduce their severity. Importantly, medication is often prescribed after surgery to manage seizures.

Conclusion

Epilepsy has been a condition that has been widely misunderstood in the past. Unfortunately, in some parts of the world, it still continues to be met with social judgements and fear. To deal with this, it is imperative to dedicate resources towards educating the public and removing the misconceptions that are associated with it. Epileptic patients suffer from a negative impact on their quality of life as well as a risk to it. While there is no cure for it, there exist therapeutic strategies and neurophysiological care for patients for them to ameliorate their physical health and permit them to have normal personal and professional lives.

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INTERVIEW

The necessity of a big picture science

Juan García-Ruiz[1]
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This interview was extracted from the website www.neuronhub.org founded by one of the members of the Editorial Board of BrainStorm (Juan Garcia-Ruiz).

What's neuronhub? It is an outreach website hosting interviews with researchers from all corners of the planet about their work in the field of neuroscience. The idea is that you get something from people who have a long career in science, that you learn something new and cool, and above all that you don't lose track of the latest discoveries in neuroscience that are being made in other parts of the world.

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First person plural

If I ask you about the origin of civilization, you may first think of different regions such as the Mesopotamian (the modern-day Iraq), Ancient Egypt, the Indus Valley (current northwest India and Pakistan) or China, which were the birthplace of the earliest human organization as a society. These are the regions in which 4000 or 5000 years ago, humans started developing complex social, political, and economic systems. Besides, it is the origin of human division of labor.

You guessed something like that, didn't you? Not a bad attempt to guess the first civilization ever, except for the fact that you are completely wrong! What if I told you that there was a civilization that originated 30 million years ago in Eastern Africa (present-day Ethiopia, Kenya, Djibouti and Somalia)? That's right! And you know what? This civilization was not human. Let me introduce you to naked mole rats!

Naked mole rat. Photo: Mehgan Murphy.

Naked mole rats are these hairless, not too graceful rodents that have been inhabiting the East African underground for tens of millions of years and that you've probably only seen in pictures. They say beauty lies within. Certainly not within the earth! But researchers still love them because of their impressive physiology. First, they are the only existing cold-blooded mammals. Plus, they have a very low respiratory and metabolic rate, which makes them adapt to extremely low-oxygen conditions like the ones found underground. In addition, they are super-powerful immortal beings known for their insensitivity to certain types of painful stimuli and their great longevity. But the most interesting thing about these animals is that they live in a complex society in the same way as humans.

The phenomenon of living in a high level of organization is known as eusociality. What does it mean a high level of organization? Basically, that they cooperate between individuals in the offspring care, that they divide the labor, and that their colonies are constituted by overlapping generations of individuals (in the same way we live in families with grand-children, parents and grand-parents). Another way of seeing these colonies of eusocial animals is as a superorganism. We can imagine them not just as a group of individuals working together, but is instead a single organism in which the individual members are analogous to the cells in a body. They have specialized roles that they satisfy for the sake of the group, much like the cells in a multicellular body have specialized functions that contribute to the overall health and survival of the organism.

A crucial feature of eusocial species or superorganisms is to have good communication between its constituents. For this reason, to better understand how these organized societies form and succeed, it is very important to understand how their individuals interact. The work of some researchers such as Alison Barker focuses on that. Stay with us a little longer and we will tell you what language naked mole rats speak.

Alison Barker is an American neuroscientist. She currently has her own group at the Max Planck Institute for Brain Research in Frankfurt. She studied biochemistry in the US at Brown University. She became interested in the chemistry in the brain, which led her to start a PhD in neuroscience at University of California, San Francisco. In the middle of her PhD her supervisor moved to Munich and she joined him. At that stage she was interested in studying visual processing behaviors in zebrafish: how animals can perceive something about their world, how that gets integrated in the brain and how it ultimately leads to a behavioral output. Then from her postdoc she shifted gears completely and started to study auditory information processing in social contexts taking the naked mole-rats as a model.

Juan García Ruiz: When we think of social animals, the first thing that comes to mind is humans, ants and bees. Is this feature limited to those species or instead all the species have some extent of organization?

Alison Barker: When people think of social hierarchies in the animal kingdom the most common examples are bees or other insects that have a queen, workers and other individuals doing specific tasks. This was mainly thought to exist in invertebrates and there were some hypotheses about why that might be in terms of genetic fitness and the ability to support individuals that are highly related. But there was a fantastic discovery in the early 80s by Jennifer Jarvis: she found the first eusocial mammal. When I say eusocial I mean fitting three criteria that was first described in insects: reproductive division of labor, cooperative care of the young and existence of multiple overlap of generations. All three criteria are present in the naked mole rats. They show a very strict reproductive division of labor, which means that only a subset of individuals breed in the colony. The naked mole rats actually have a structure that is very similar to bees. They have a queen that is responsible for reproduction, the only breeding female, and there are generally one to three breeding males, depending on the size of the colony. The rest of the colony divides their work into very specialized tasks, like soldiers which defend the colonies and workers, which will forage for food.

A subset of the workers also care for the young. And the older individuals stay in the colony, so different generations overlap within the same colony. To answer to your question, there are a lot of species that are incredibly social and cooperative, but this sort of high level cooperation and this very strict hierarchy is rare.

JGR: What are the main forms in which social animals can communicate?

AB: Any social interaction is a complicated process that depend on multiple channels that can be decoded and integrated. For the naked mole rats, we know that they rely a lot on olfaction, audition and touch. Like many subterranean rodents they are mostly blind, so we don't include the vision in our studies.

JGR: How crucial is the high-level societal organization for a species that has evolved this way? Otherwise said, could a naked mole rat survive on its own?

AB: One of the reasons why I love this animal is that it is such a nice example of the power of evolution. They are very well adapted to their ecological niche. Their natural habitat is located in Ethiopia, Kenya and Somalia, in very dry lands along the equator, so very little grows there and the periods of the year in which they can forage and look for food are limited. One of the reasons why the naked mole rats do so well in these environments is because they share resources. Plus, naked mole rats are cold-blooded, so they cannot regulate their own body temperature. This makes a lot of sense if you think of where they live. The temperature doesn't really change that much along the equator. So in one sense they have given up some features and have developed others to survive, like this highly cooperative organization. I guess the naked mole rats could survive on their own for a certain period of time, but I think without their colony they wouldn't do very well.

JGR: What are the big unknowns in the field of eusociality?

AB: Some of the biggest questions are how eusociality evolved, and how it has been maintained. One of the most striking things about this is that whenever you belong to some social group you give up some of your own agency. For instance in a society we give up some personal freedoms for the benefits of being in a society. In the extreme case of the naked mole rats they give up their ability to reproduce. So one of the big unknowns is the understanding of this weighting, what are the brain circuits that allow us to cooperate and override the basic urge for self-preservation or reproduction.

JGR: What makes naked mole rats an interesting model to study social communication?

AB: I decided to study the naked mole rats because in their case the eusociality is very extreme. In general, in science you always look for the greatest signal-to-noise ratio so that you can really pick the things that stand out. Biology is messy and there are many factors that cannot be controlled, so if the behavior is very robust, that is a very nice place to start. The other reason why I chose the naked mole rats is because they are very easy to have in the lab, so then you can start adapting technology used in other rodents to better study neural circuits or molecular mechanisms.

JGR: What are the main questions your team is trying to elucidate?

AB: We focus on a specific type of communication. We are very interested in how vocalizations help social groups organize and how they enforce social bonds. We want to understand how these social signals are encoded and decoded. When an animal produces a social sound, another animal in the group has to detect it, process it and determine its meaning to generate its own behavior. There are many steps, and we want

to better understand how it takes place in the brain. Naked mole rats are the perfect model to study this because besides being very cooperative, they are also highly vocal. They make at least 25 different vocalizations, and they can use these diverse sounds in specific social contexts. We know for example that there is a specific type of vocalization which is like a greeting call and is called the soft chirp. When two individuals encounter one another they emit this sound reciprocally. This provides multiple levels of information, such as the individual identity, or which social group they belong to. You can think of it in the way humans use dialects or accents: with a single vocalization you can get a lot of information about the other person. We are trying to focus on this and then move into the brain. We are also interested in how the social dynamics of the colony can change their vocal dialects and how it develops: how do young animals learn when they are born which dialect to use in each social context.

JGR: Can naked mole rats use these 25 vocalizations in a flexible way, for instance by combining them to create more complex meanings? Or do they use them in a more stimulus - response way?

AB: We are trying to figure that out. Vocalizations are very stereotyped responses in a lot of animals. Of course in humans it's something extremely flexible. I think naked mole rats are in the middle. There is some kind of plasticity in their calls. But we are just scratching the surface. We have looked at this one vocalization type, and now we are trying to figure out what is going on with the other 24.

JGR: What are the main discoveries that your team made?

AB: An important finding that we made when I was a postdoc working at the Max Delbrück Center for Molecular Medicine in Berlin is related to the soft chirp, a type of vocalization I mentioned before. We found out that this type of vocalization provides information about the individual naked mole rats identity and about which colony they are from. We could test this and we showed that naked mole rats hearing a soft chirp from their own colony would preferentially respond to that rather than to a soft chirp from another colony or an artificial sound, which suggests that they can recognize the general features of the colony in these vocalizations.

Another interesting finding is that these dialogues can be learnt early in life. So if you move a young naked mole rat to another colony, they will learn the dialect of the adoptive colony. If this was completely genetic, we would predict that regardless of where the animals were raised, they would speak the birth dialect. But that was not the case. Something quite interesting we discovered about those dialects is that they seem to be dependent on the presence of the queen of the colony. We had a colony in which the queen was lost in several overthrow situations. So there were periods in which there was no queen in the colony and we could see that the dialects got really messy and fell apart, and when there was a new queen the vocalizations became clear again. This means that the social dynamics of these animals are really important.

JGR: Do you share the opinion that research is today a challenging career choice?

AB: Research has always been complicated I would say. It is difficult to quantify that appropriately. But I think that one thing that is really powerful is the open-data initiatives we have today. Diversity in science is very important, and this first requires access. Having access to papers or to code is very important and I think that's really moving science forward.

JGR: Do you have a book recommendation?

AB: One book that I think all scientists should read is Mary Shelley's Frankenstein. This is not strictly scientific but it speaks to scientific ethics. Briefly, Frankenstein was a doctor that created a creature. He was so concerned about reanimating dead matter that he didn't think about the consequences: what is the meaning? What are my responsibilities? And after he did it, he just let this creature run free and took no responsibility. Obviously that ended poorly for him. I read that when I was sixteen years old and it made a deep impression on me. As scientists we can do things that nobody did before, but we should always think of the consequences of our actions.

JGR: What can humans learn from other social species?

AB: Humans sometimes live with blinders on and have this very narrow view of things. Certainly from naked mole rats we can learn about cooperation and about resources sharing. One of the things that is most interesting when studying a rodent like the naked mole rats is to think about how we think of communication and how it can take on many different forms. It has been like an eye-opener. It made me rethink about the interactions we have as humans, what does it mean to communicate, or how do we structure language. Plus, studying naked mole rats is also powerful because it gets you to see something done in a different way, so it puts things in perspective.

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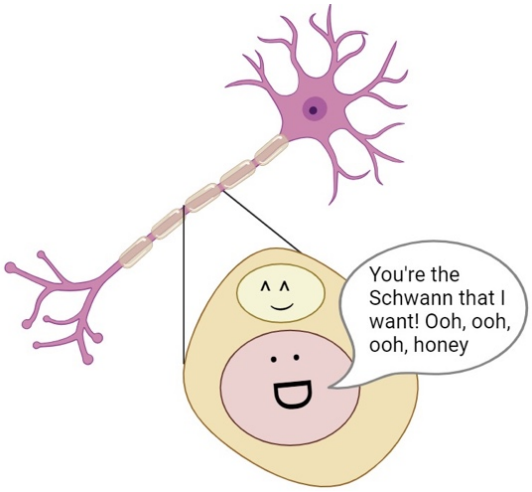

Carmen Guerrero, 1st year PhD student at the IMN

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Editorial board

Sara Carracedo

Sara is a PhD student at the Neurodegénératives Diseases Institute (IMN). She comes from Pontevedra, Spain and holds a Veterinary Bachelor's degree from the University of Santiago de Compostela and the NeuroBIM Master's in Neurosciences from the University of Bordeaux. Her PhD is focused on understanding the microglial and neuronal role of P2X4 receptor in ALS pathogenesis in which she is interested in neuroimmune interactions, microglial functions, and P2X4 trafficking.

Juan García-Ruiz

With two Bachelor's degree, in Psychology and Biochemistry, and the NeuroBIM Master's degree from the University of Bordeaux, Juan is now pursuing a PhD where he is focuses on the role of lactate in basal synaptic transmission, which allows him to combine his research interests in biochemistry, electrophysiology and neurometabolics. Although he speaks near-perfect French, Juan comes from Huelva, Spain. He is also the co-founder of neuronhub (www.neuronhub.org).

Simon Lecomte

Simon is originally from Lyon, France. He did his Bachelor's of Psychology from Strasbourg, after which he did the NeroBIM master's degree from the University of Bordeaux. He is a PhD student in the IINS where he is studying how the Fragile X Syndrome impacts the presynaptic mechanisms at the DG-CA3 synapses from which one can guess that his interests lie in memory, synaptic communication and the hippocampus.

Ludovica Congiu

Ludovica is an Italian researcher hailing from the beautiful island of Sardinia. After obtaining a master's degree in Neuropsychobiology at the University of Cagliari, she pursued a Ph.D. in neuroscience at the Universitätsklinikum Hamburg-Eppendorf (UKE) in Hamburg. The project focused on characterizing the role of the cell adhesion molecule L1 in affecting mitochondrial activity and metabolism. Currently, she works as an Assistant Ingénieur at the IMN, where she is investigating the role of P2X4 receptors in ALS and anxiety disorders.

Khadija Inam

Khadija is a Clinical Research Associate in the General and University Psychiatry (PGU) Unit of Centre Hospitalier Charles Perrens. With a Bachelor's degree in Applied Biosciences from the National University of Sciences and Technology, Pakistan, and a Master's degree in Neurosciences from the University of Bordeaux, France, she has seamlessly transitioned from the vibrant academic corridors to the cutting-edge realm of clinical trials. Her expertise is mainly focused on the complex spectrum of neuropsychiatric disorders—Schizophrenia, Depression, Anxiety, and Bipolar Disorder.

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