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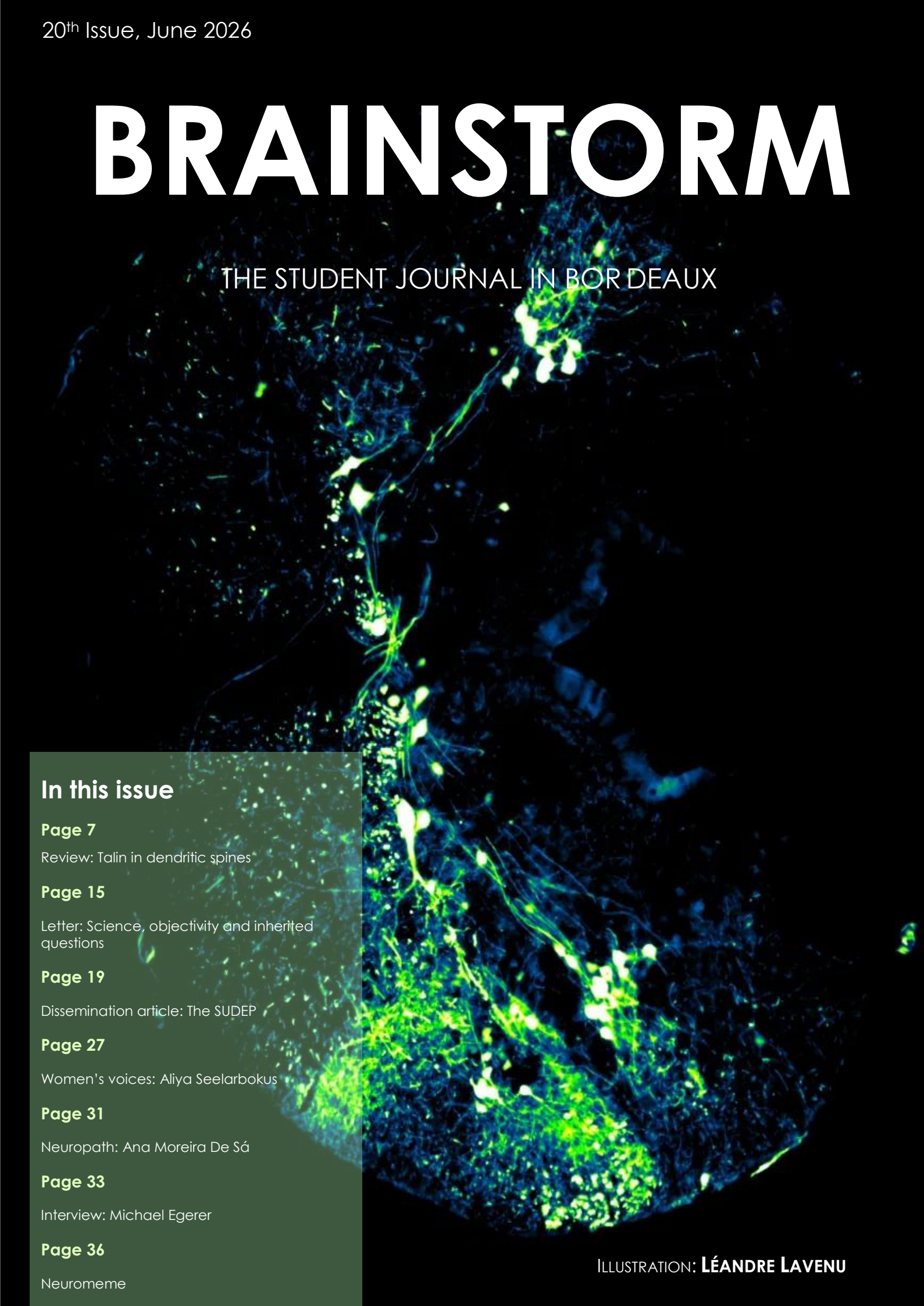

In this issue

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VALUES

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Talin in dendritic spines: an adaptable scaffold hiding in plain sight

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Abstract

Plasticity is a demanding task for a dendritic spine: this tiny compartment must remodel without losing synaptic contact, adjusting receptor content, reorganising the postsynaptic scaffold and reshaping actin. Such remodelling may also alter mechanical load at the membrane-cytoskeleton interface. This force-sensitive interface is still poorly understood in spines, but it has been studied extensively in adhesion biology. In that context, talin is a central integrin-actin linker that does more than bear tension: when stretched, it unfolds and changes its binding partners. This review explores whether these mechanosensitive properties could make talin a key organiser in dendritic spines.

Keywords

Talin; dendritic spine; integrin; mechanotransduction; MeshCODE; synaptic plasticity; adhesion;actin; vinculin

Abbreviations

AMPAR - α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid receptor

APP - amyloid precursor protein

CaMKII - calcium/calmodulin-dependent protein kinase II

CDK5 - cyclin-dependent kinase 5

DLC1 - deleted in liver cancer 1

F-actin - filamentous actin

FERM - band 4.1, ezrin, radixin and moesin

GTPase - guanosine triphosphatase

LTP - long-term potentiation

NMDAR - N-methyl-D-aspartate receptor

$PI(4,5)P_2$ - phosphatidylinositol 4,5-bisphosphate

PIPKIγ - phosphatidylinositol 4-phosphate 5-kinase type Iγ

PKA - protein kinase A

PKC - protein kinase C

PSD - postsynaptic density

Rap1 - Ras-related protein 1

RhoA - Ras homolog family member A

RIAM - Rap1-GTP-interacting adaptor molecule

ROCK - Rho-associated protein kinase

YENPTY - tyrosine-glutamate-asparagine-proline-threonine-tyrosine motif

Introduction

Adaptability is one of the nervous system's best tricks. Humans adapt to new cities, bad coffee and impossible deadlines; neurons adapt by changing how strongly they communicate. At excitatory synapses, this tuning is synaptic plasticity: the ability of synapses to strengthen or weaken through presynaptic and postsynaptic mechanisms. In the hippocampus, long-term potentiation (LTP) remains the classic example of memory-related synaptic change (1).

At many excitatory synapses, strengthening begins when neuronal activity triggers calcium entry through N-methyl-D-aspartate receptors (NMDARs) in the postsynaptic spine. This activates pathways that link activity to molecular reorganization, including calcium/calmodulin-dependent protein kinase II (CaMKII), protein kinase A (PKA) and protein kinase C (PKC). Together, these pathways promote insertion and stabilization of α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid receptors (AMPARs), converting a brief calcium signal into a longer-lasting increase in synaptic efficacy (2-4). Yet LTP is not only about adding receptors. For potentiation to persist, the postsynaptic compartment must be rebuilt. Spine heads enlarge, filamentous actin (F-actin) reorganizes, and the postsynaptic density (PSD) expands to stabilize a receptor-rich synaptic domain (5,6). This structural plasticity is not just the spine flexing its size: spine geometry, actin architecture and scaffold organization help determine whether inserted AMPARs remain trapped or diffuse away. In this sense, actin helps turning a transient biochemical event into a durable synaptic state.

This remodeling raises a mechanical question: If actin polymerization and contractility reshape the spine, they must redistribute load at the membrane-cytoskeleton interface. This load is unlikely to be uniform: dynamic F-actin near the spine tip drives expansion, stable actin pools maintain volume, and myosin II adds contractile tension (6-9). Whether this movement is lost or converted into useful force depends on how well actin grips the membrane. Adhesion

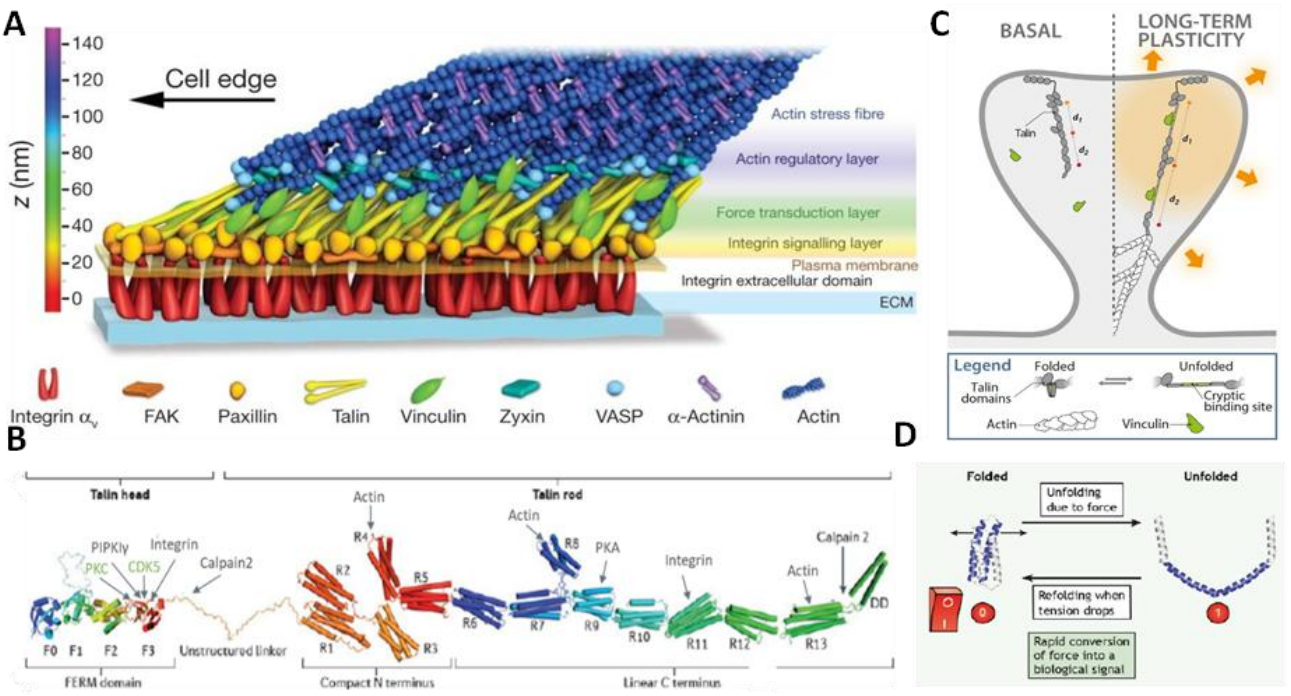

Figure 1. Talin architecture and proposed mechanotransduction in dendritic spines. **(A)** Schematic model of focal adhesion nanoscale architecture, adapted from Kanchanawong et al. (37), showing the stratified organization of integrin signaling, force-transduction and actin-regulatory layers. The model depicts experimentally determined protein positions, not protein stoichiometry. **(B)** Talin1 domain organization, showing the FERM head, rod domains and selected interaction partners relevant to adhesion, actin regulation and synaptic signaling. **(C)** Proposed talin-dependent mechanotransduction in an excitatory dendritic spine: during plasticity, actin-generated tension may stretch talin, expose cryptic vinculin-binding sites and strengthen actin-membrane coupling. **(D)** Talin rod domains can act as force-dependent binary switches, unfolding under tension and refolding when tension decreases, providing a possible conformational mechanism for mechanical signal encoding.

molecules can act like molecular clutches. N-cadherin-associated complexes engage actin during synapse maturation, spectrin and ankyrin provide an internal membrane scaffold, and integrins serve as anchoring and signaling hubs that support AMPAR stability and LTP maintenance (10-15). Perhaps the clearest sign that spine mechanics matter is that the spine can push back: during enlargement, postsynaptic force on the presynaptic bouton can enhance glutamate release through an actin-dependent pathway (16). Together, these findings show that plasticity has a physical side. What remains unclear is how a spine reads actin-generated force and converts it into lasting molecular organization.

This is where talin becomes interesting. Talin sits at the junction between membrane adhesion, F-actin and force-sensitive signaling (Fig. 1A). In adhesion biology, it is not merely a molecular cable: when stretched, talin rod domains unfold and expose binding sites for partners such as vinculin, allowing mechanical load to reshape the local molecular environment (Fig. 1B). Talin could offer what the synaptic picture is still missing: a way for force to become biochemical organization (Fig. 1C). Its postsynaptic role remains largely unknown, but detection in synaptic fractions and presynaptic links to actin assembly and endocytosis give it a credible foothold in the synapse (17,18). This review asks whether talin could provide a mechanochemical link between actin-driven spine remodeling and postsynaptic organization.

Methods

PubMed, Google Scholar and the supplied Zotero library were searched using combinations of: talin AND synapse, talin AND dendritic spine, talin AND mechanotransduction, MeshCODE, talin AND vinculin, integrin AND synaptic plasticity, actin AND LTP, synaptic proteome AND talin, and talin AND APP. Original studies and mechanistic reviews were prioritized. Non-neuronal adhesion papers were included only when they explained talin mechanisms directly relevant to the synaptic question, especially integrin activation, force-dependent unfolding, vinculin binding or state-dependent partner recruitment.

Results and discussion

Meet talin: adaptable by design

Before asking whether talin could contribute to synaptic mechanotransduction, we asked whether its structure fits a force-sensitive scaffold. Talin is built with a simple mechanical logic: one end faces the membrane, while the other reaches toward actin. Its membrane-facing head contains the band 4.1, ezrin, radixin and moesin (FERM) domain, which recruits talin to membrane-associated complexes. Extending from this head is a rod of 13 helical bundles, an interaction-rich platform for cytoskeletal and signaling partners (Fig. 1B) (19,20). Vertebrates express talin-1 and talin-2, with talin-2 brain-enriched (20). In its compact state, talin is autoinhibited: internal head-rod contacts hide key interaction sites until recruitment and activation (19).

This opening process is best understood from adhesion biology. Talin starts in a closed, inactive shape, like a folded clasp. Ras-related protein 1 (Rap1) recruits it to the membrane, either directly or through Rap1-GTP-interacting adaptor molecule (RIAM), while membrane lipids and phosphatidylinositol 4-phosphate 5-kinase type Iγ (PIPKIγ) help hold the clasp open (21–24). Once open, talin can grab β-integrins with one end and F-actin with the other, forming a bridge between extracellular adhesion and the cytoskeleton (Fig. 1A,B) (25). Whether spines use the same sequence is unknown, but it provides a useful model.

From force to signaling

Talin becomes interesting because this bridge is not rigid. When tension pulls on talin, rod bundles can unfold under piconewton-scale forces, exposing hidden vinculin-binding sites (Fig. 1D) (26,27). Vinculin can bind and reinforce the talin-actin connection, stabilizing the force-bearing complex (28). Thus, talin does not simply transmit force; it changes state in response to force. Each folded or unfolded domain can expose a different set of binding sites, like molecular switches. The MeshCODE hypothesis takes this logic one step further: if different switch combinations recruit different partners, talin-vinculin networks could, in principle, encode mechanical information and store a trace of past force (29). For dendritic spines, this remains hypothetical but attractive: potentiated spines must preserve receptor content, scaffold organization and actin architecture after the plasticity signal has faded (Fig. 1C).

Focal adhesions provide the clearest example of this mechanical logic (Fig. 1A) (37). These sites, commonly studied in fibroblasts and other adherent cells, anchor cells to their surroundings and link this attachment to actin through talin. They are not synapses, but they show that stretching talin can expose hidden binding sites and change nearby proteins. R8 gives an example. In its folded state, R8 can hold onto deleted in liver cancer 1 (DLC1), a Rho GTPase-activating protein that acts like a brake on Ras homolog family member A (RhoA), one of the Rho-family guanosine triphosphatases (GTPases) controlling actin. When force unfolds R8, this brake may be released, favoring RhoA/Rho-associated protein kinase (ROCK) signaling (30). This matter because Rho-family pathways shape actin during spine growth and stabilization. Talin, therefore, does not simply bear force; by changing shape, it can tune local actin-regulatory signaling.

The strongest synaptic link may be talin's connection to kinases that already control LTP. PKA regulates AMPAR insertion, late-phase LTP and hippocampus-dependent memory (3). Recent work shows that PKA can also bind talin directly: unfolding of talin R9 exposes a site for the PKA regulatory subunit RIIα, allowing force-dependent PKA association (31). If LTP generates tension inside spines, talin unfolding could position PKA where receptors, actin and adhesion complexes are reorganized.

PKC adds a second layer. During LTP, PKC helps sustain stronger synaptic transmission by phosphorylating proteins that regulate AMPAR function and postsynaptic stability (4). Talin itself can also be phosphorylated by PKC, suggesting that this kinase may tune talin during plasticity (32). Cyclin-dependent kinase 5 (CDK5), a neuronal kinase involved in NMDAR regulation and spine plasticity, has also been reported to phosphorylate talin (33,34). Talin's force-sensitive behavior may therefore depend not only on mechanical load, but also on plasticity-related signaling.

Why this matters for synapses

Although talin's postsynaptic functions remain largely unexplored, its partners already point toward neuronal processes. PIPKIγ generates phosphatidylinositol 4,5-bisphosphate [$PI(4,5)P_2$], a lipid involved in actin dynamics and clathrin-mediated endocytosis (35). At presynaptic sites, talin-2 binds PIPKIγ, promotes $PI(4,5)P_2$ accumulation, supports actin assembly and sustains synaptic vesicle endocytosis (18). Talin may also connect to Alzheimer 's-relevant amyloid precursor protein (APP) biology: its F3 subdomain binds the APP YENPTY motif, a short intracellular "address label" for trafficking adaptors, and talin loss alters APP processing (36). This suggests that talin's role may extend beyond adhesion mechanics to neurodegenerative vulnerability.

Overall, talin connects to processes that the spine uses during plasticity: actin remodeling, integrin signaling, kinase activity, membrane lipid signaling and protein cleavage. If talin is present and mechanically engaged at excitatory postsynapses, it could help convert actin-driven changes in spine shape into organized biochemical responses. In this view, talin would not simply mechanically support the synapse; it could help stabilize remodeling as a lasting molecular state.

Conclusions

Synaptic plasticity is usually discussed in terms of receptors, kinases, and changes in synaptic strength, but dendritic spines remind us that plasticity also has a structural side. During LTP, the postsynaptic compartment is not simply functionally updated; it is physically remodeled. Actin reorganizes, the PSD expands, receptors are stabilized, and the spine must preserve its synaptic contact while changing its internal architecture. How mechanical cues generated during this remodeling are sensed or translated into molecular organization remains an open question.

In this context talin represents an interesting player. The literature does not yet support calling talin a memory molecule, and the mechanical force landscape within spines remains far from resolved. However, talin has a rare combination of properties that fits the problem unusually well. It links integrins to F-actin, changes conformation under force, exposes new binding sites, recruits partners such as vinculin, and can be tuned by phosphorylation, phosphoinositide signaling and proteolysis. These are not isolated biochemical details; together, they describe a scaffold capable of coupling adhesion, actin, and molecular organization.

The synaptic evidence remains incomplete but suggestive. Integrins are already implicated in AMPAR stabilization and LTP maintenance; talin partners such as PIPKIγ operate at synapses; presynaptic talin has been linked to actin and endocytosis; and proteomic datasets place talin in synaptic and postsynaptic fractions. These observations do not prove that talin organizes dendritic spines. However, they suggest that talin is sufficiently linked to synaptic structure and plasticity that its relative absence from the synaptic plasticity literature is striking.

MeshCODE offers the most ambitious interpretation of talin biology: folded and unfolded talin domains could constitute a memory-related molecular code. Whether that idea applies to excitatory postsynapses is unknown. For now, the strongest conclusion is more cautious but still exciting: talin is an overlooked mechanochemical scaffold whose known biology makes it a serious candidate for shaping dendritic spine organization during plasticity. The synaptic talin story is not solved; it is just beginning.

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Science, objectivity, and inherited questions

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Can science still be objective when no one fully owns the question?

Very early in research, and even from the moment we enter a master's or PhD program, we learn to live with a paradox. The project we choose to commit ourselves to does not fully belong to us. It often begins before we arrive and continues after we leave. And yet, we are expected to make it our own.

This tension is part of how research operates. Each student develops a specific question within a broader scientific framework. A thesis becomes personal not because it leads to entirely different conclusions, but because it approaches a problem from a particular perspective.

In some laboratories, this continuity is seen as natural. Projects evolve through successive students, postdoctoral researchers, and collaborators. In others, the boundaries are less clear, making it more difficult to distinguish between inheritance and ownership.

My own work emerged from previous studies on the effects of metal nanoparticles on neural activity and respiration. Rather than starting from a completely new question, I entered an ongoing scientific conversation. My role was to refine certain mechanisms and connect observations across different experimental contexts.

This experience is not limited to the PhD. It extends across different stages of academic research and becomes even more pronounced in positions such as postdoctoral contracts, often characterized by shorter timeframes. Researchers often join projects that are already structured and designed to continue beyond their individual involvement. Scientific work is therefore rarely produced from a fully independent position. It is embedded within funding structures, evaluation systems, and collective trajectories.

This continuity is not simply an organizational aspect of research. It also shapes the conditions under which scientific knowledge is produced. The questions we investigate, the directions we pursue, and even the freedom we have to explore them are never entirely detached from the structures in which research takes place.

Seen from this perspective, objectivity becomes more than a methodological principle. It also becomes a question about the environment in which science is conducted, and about how knowledge is collectively constructed over time. Historians and sociologists of science have long shown that scientific knowledge is shaped not only by methods and results, but also by funding institutions, collaborations, publication system, and scientific evaluation processes.

In theory, scientific objectivity rests on the idea that knowledge should not depend on who produces it. Methods are designed to limit personal bias, and results are expected to be reproducible regardless of the researcher involved. This ideal remains central to how science defines its legitimacy.

In practice, however, research takes place within a set of constraints. Funding priorities, institutional agendas, and publication requirements all influence which questions are asked and which results are highlighted. Science also progresses slowly, requiring time for application, discussion, and methodological refinement. Yet researchers frequently work within short-term contracts and grants that demand rapid and measurable outcomes. Grant priorities are also rarely neutral: research connected to economic interests, public health concerns, or technological innovation is often more likely to receive support. As a result, some scientific directions become easier to pursue than others. This does not necessarily undermine the scientific process itself, but it does shape the conditions under which knowledge is produced. What emerges is not the disappearance of objectivity, but an objectivity that is continuously shaped by the structures within which science operates.

For early-career researchers, this tension becomes especially visible because these constraints directly shape both scientific work and professional opportunities. They are trained to think independently while working within projects that are rarely entirely self-defined. They are encouraged to develop originality, yet evaluated on their ability to produce within existing frameworks. In this sense, they are neither fully interchangeable nor fully autonomous. They contribute to a process that depends on them while also extending far beyond them. As a result, the question of objectivity can no longer be separated from the conditions under which scientific work is produced.

This directly shapes the conditions of scientific objectivity. It is not that individuals deliberately introduce bias, but rather that the structure of research itself influences what can be asked, explored, and ultimately recognized. When directions are already partly defined, when time is limited, and when results are expected, the space for deviation and for genuinely unexpected questions can narrow.

Despite this, science remains fundamentally a collective attempt to approach truth. Its strength lies in its capacity for self-correction, in the plurality of perspectives, and in the fact that no single contribution defines the whole. Objectivity, in this sense, is not the absence of subjectivity, but what emerges from its confrontation across people, methods, and time.

Perhaps, then, the question is not whether science is still objective, but how individuals come to find their place within it. How does one contribute to knowledge that is meant to outlast them? How does one develop a scientific voice within a system that values both continuity and originality?

To do science is therefore not simply to produce knowledge in isolation. It also means navigating a system shaped by collaboration, institutional constraints, inherited questions, and individual curiosity. Objectivity, in this sense, is not something fixed or entirely detached from society, but something continuously negotiated and collectively constructed.

Further reading:

Thomas Kuhn, *The Structure of Scientific Revolutions*

Robert K. Merton, *The Sociology of Science*

Bruno Latour and Steve Woolgar, *Laboratory Life*

Like lightning in a cloudless sky: the sudden unexpected death in epilepsy

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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.

In memory of Aymeric Ramain (1997-2019).

One day, while you are watching TV, playing video games or cards, studying for an exam, having lunch, or doing sport, you see your son or daughter, your brother or sister, or a friend falling to the ground, convulsing and unconscious. At the time, you may not have understood what was happening, but a few weeks or months later, the neurologist will put a name to what happened: it was an epileptic seizure. The neurologist will, of course, explain to the family what epilepsy is: “Epilepsy is a chronic brain disorder characterized by the occurrence of seizures. Seizures can vary widely in severity, ranging from those that are impossible to notice to those that cause spasms and loss of consciousness. They are caused by excessive electrical activity in the brain, generally, this activity is limited to a small area (focal seizures), but sometimes it spreads to the entire brain (generalized seizures). Both types of epileptic seizures can result in motor or non-motor symptoms (absences). Only tonic-clonic seizures (or “grand mal”) are the most impressive and dangerous for the patient, in fact, they can cause dramatic accidents. There are medications available to reduce the frequency and severity of seizures, or even eliminate them entirely. In the event of a severe seizure, try to move the patient away from any dangerous situations, place them on the side and check their breathing after the seizure.”

After years and years of recurring seizures, you know how to react. Fear never completely disappears, but routines emerge around unpredictability. The problem is that no medication has been able to reduce frequency of seizures or even stabilize the progression of the disease. The school is now requesting that he be placed in a medical institution, as the seizures have become too frequent and too severe. This institution is located several hundred kilometers away, due to the scarcity of such institutions. Beyond the frequent trips back and forth, his absence weighs heavily on you. When he was a child, he had thousands of different jobs in mind, now, none. The institution advises him to work in a factory, in an office, or in facility security. Having seizures in a professional environment has become a source of extreme fear for him. The idea of working has become a nightmare. Fatigue and anxiety have set in. He went back home to rest. One morning, you walk into his room and find him in his bed… gone.

Definition and conceptual analysis

SUDEP, or Sudden Unexpected Death in EPilepsy, is the leading cause of death among patients with epilepsy, however, it remains one of the least known among the general public and patients' families. This trend is also seen at the scientific literature level. In 2025, 10,500 published articles containing the word "Epilepsy" are found on PubMed (an online scientific database for life sciences and biomedical articles) and only 191 containing the word "SUDEP" (Figure 1). That is why it is essential to raise public awareness of SUDEP in order to better understand this phenomenon, both within the scientific community and among the patient's family and loved ones. With this in mind, this article aims to raise public awareness of SUDEP, potential ways to prevent it, and the burden placed on loved ones following such a loss.

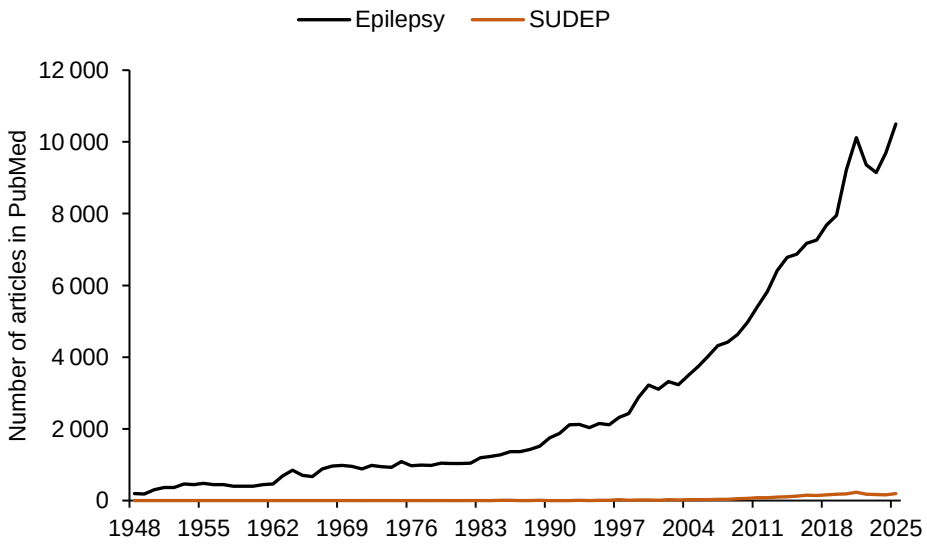

Figure 1. Number of articles found in PubMed containing either the word "Epilepsy" (black) or "SUDEP" (red) from 1948 to 2025.

The canonical definition of SUDEP has been established in its unified version in 2012 as follows: "Sudden, unexpected, witnessed or unwitnessed, nontraumatic and non-drowning death, occurring in benign circumstances, in an individual with epilepsy, with or without evidence for a seizure and excluding documented status epilepticus (seizure duration ≥30 min or seizures without recovery in between), in which postmortem examination does not reveal a cause of death." (1)

It is very important to note that SUDEP (and all forms of "sudden death") is a quite unique concept in medicine. First, it is a diagnosis of exclusion: there is no anatomical or toxicological explanation, and no status epilepticus, trauma/injury, or drowning. Second, it is an essentially probabilistic medical concept: based on all available information at a specific point in time (i.e. current medical knowledge, current medical classifications, current investigation techniques, and the quality and practices of the autopsy), no medical cause can be found to explain the death. Third, it is a post-mortem concept.

Moreover, the term SUDEP contains the word "unexpected" in its name and definition, a term that usually never appears in medicine. It reflects both the suddenness of the event and the current limits of medical prediction. Medical practice is fundamentally organized around anticipation: identifying pathological processes before irreversible outcomes occur. SUDEP challenges this framework because it remains identifiable only retrospectively.

SUDEP occupies an unusual position in medicine: it is simultaneously a clinical concern, a forensic diagnosis, and a residual explanatory category. Defined largely by exclusion and recognized only retrospectively after death, it challenges conventional assumptions that diseases or medical conditions correspond to identifiable mechanisms

observable during lifetime. For all these reasons SUDEP represents not only a neurological and medical challenge but also an epistemological one.

Does SUDEP merely reflect the current limitations of medical knowledge and techniques, or does it reveal the intrinsic impossibility of predicting its occurrence?

This question is not purely theoretical. If SUDEP is viewed as a temporary limitation of biomedical knowledge, then new advances could improve prediction. Conversely, if certain forms of SUDEP are inherently resistant to accurate prediction due to their multifactorial, probabilistic, and heterogeneous nature, then anticipation may remain out of reach.

It is important to note that these possibilities are not mutually exclusive. The current inability to predict SUDEP does not imply that any progress is impossible, just as partial unpredictability does not eliminate the value of continuing research. On the contrary, identifying risk factors, understanding physiological mechanisms, improving seizure control, and developing preventive strategies remain essential clinical and scientific goals. At present, there is no sufficient reason to conclude that SUDEP will always remain beyond the reach of a better medical understanding.

Epidemiology and risk factors

The incidence of SUDEP in the general epilepsy population is estimated at approximately 1-1.6 cases per 1,000 patient-years (2,3). However, this risk is not uniformly distributed among patients. Over the past decades, several major risk factors have been consistently identified. One of the most comprehensive studies on SUDEP risk factors was conducted using Swedish national data on all individuals diagnosed with epilepsy between 1998 and 2005 (60,952 individuals), followed until 2011, and identified cases of SUDEP by reviewing the medical records of patients with epilepsy who had died (2).

The most important risk factor found is the occurrence and frequency of tonic-clonic seizures (TCS). Patients who experienced one or more TCS during the year preceding death show a markedly increased incidence of SUDEP, estimated at approximately 287-296 per 100,000 person-years. Nocturnal TCS constitute another major risk factor and are associated with an incidence of approximately 206 per 100,000 person-years. Similarly, nonadherence to antiseizure medications (ASMs) is associated with a comparable incidence of approximately 206 per 100,000 person-years.

Social and behavioral factors also appear to play a significant role. Living alone or lacking nighttime supervision is associated with an incidence of approximately 182 per 100,000 person-years, suggesting that immediate intervention after seizures may influence survival. In addition, alcohol dependence and substance abuse are associated with substantially elevated incidences of SUDEP, estimated respectively at approximately 292 and 236 per 100,000 person-years.

Importantly, SUDEP risk appears to increase considerably when several risk factors coexist. Patients with a history of nocturnal TCS who also live alone present an incidence approaching 516 per 100,000 person-years. The highest reported incidence is observed in patients combining multiple major risk factors: including recent TCS, nocturnal TCS, nonadherence to ASMs, and absence of nighttime supervision, reaching approximately 1,808 per 100,000 person-years.

These observations suggest that SUDEP is the result of cumulative physiological, behavioral, social and environmental factors.

Potential mechanisms

SUDEP remains inherently probabilistic: although several risk factors significantly increase its likelihood, a residual risk persists in all individuals with epilepsy, including in the absence of recognized risk factors (3). This raises a fundamental question: why can epilepsy lead to sudden death?

To address this question, it is important to understand the physiological nature of epileptic seizures. 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" (4). This abnormal neuronal activity may remain focal (i.e. constrained to a limited brain area) or become generalized, involving bilateral neural networks simultaneously. In some cases, focal seizures can secondarily generalize and spread throughout the brain (5). Both focal and generalized seizures may evolve into generalized tonic-clonic seizures, the seizure type most strongly associated with SUDEP. They typically consist of two successive phases:

- The tonic phase, characterized by generalized muscular stiffening,
- Followed by the clonic phase, characterized by repetitive muscular jerking.

Importantly, generalized seizures do not affect only motor or cognitive functions such as movement, attention, or consciousness. They may also disrupt central autonomic networks involved in the regulation of respiration, cardiovascular activity, arousal, and homeostatic reflexes. Consequently, widespread epileptic activity (particularly during generalized TCS) may transiently affect vital physiological systems necessary for survival.

One of the major advances in understanding the pathophysiology of SUDEP came from the MORTEMUS study (6), an international retrospective collaboration analyzing cases of sudden death occurring during long-term video electroencephalography (VEEG) and electrocardiographic (ECG) monitoring in epilepsy monitoring units across Europe, Israel, Australia, and New Zealand. For the first time, the study provided direct observations of the chronological sequence of physiological events preceding some cases of SUDEP.

The sequence is the following:

1. A generalized tonic-clonic seizure
2. A short period of normal or increased heart and respiratory rates
3. The occurrence of central apnea associated with severe bradycardia (i.e. decreased heart rate) and transient asystole (i.e. cardiac arrest)
4. Postictal generalized EEG suppression

In approximately one-third of cases, death occurred rapidly after this sequence. In the remaining cases the sequence continues:

5. Transient restoration of cardiac function associated with abnormal and possibly ineffective respiration (probably aggravated by the prone position)
6. Respiration then progressively deteriorates until terminal apnea
7. Followed ~5 minutes later by terminal asystole

From the MORTEMUS study it can be suggested that the main mechanism underlying SUDEP may involve an early centrally mediated breakdown of autonomic regulation following generalized TCS. More research is needed to find such mechanism(s). Severe disruption of respiratory and cardiac functions appears to occur during the postictal period, potentially leading either to immediate death or to delayed terminal cardiorespiratory arrest after several minutes of progressive physiological deterioration, likely aggravated by hypoxia (6).

These findings considerably shifted the understanding of SUDEP. Rather than resulting from an isolated cardiac arrest alone after seizures, SUDEP increasingly appears to involve a complex interaction between respiratory dysfunction, autonomic deregulation, impaired arousal, and cardiac failure following severe seizures.

Prevention and communication

Another important finding of the MORTEMUS study is that the sequence of events leading to SUDEP may include a potential window for life-saving intervention. Notably, all patients who received cardiopulmonary resuscitation within the first three minutes following cardiorespiratory arrest were successfully resuscitated (6). Although the number of observed cases remains limited, these findings suggest that early intervention during the postictal period may influence survival outcomes.

This observation has important implications for prevention strategies. Several studies suggest that nocturnal supervision and seizure monitoring systems (including baby monitors, pulse oximeters, or seizure detection devices) may help reduce the occurrence or severity of critical postictal events (7). More specifically, immediate postictal interventions such as repositioning the patient from a prone to a lateral or supine position, providing tactile or verbal stimulation, and initiating cardiopulmonary resuscitation, when necessary, may help restore arousal and improve respiratory function after seizures (3,7).

Despite these potential preventive measures, awareness of SUDEP remains limited among many patients, caregivers, and even healthcare professionals. Relatively few neurologists (around 7%) discussed SUDEP with patients and families (8), and there is a substantial scarcity in clinicians' knowledge regarding SUDEP (9,10). This reluctance may partly reflect the ethical difficulty of communicating a potentially fatal but incompletely predictable risk.

However, studies consistently indicate that most patients and caregivers wish to be informed about SUDEP, preferably at the time of diagnosis or shortly thereafter through direct face-to-face discussions (11-13). Importantly, communication about SUDEP may itself contribute to prevention by improving treatment adherence, encouraging proactive safety behaviors, increasing nighttime supervision, and promoting knowledge of first aid measures and cardiopulmonary resuscitation techniques.

In order to help healthcare professionals in communicating surrounding SUDEP an adaptation of the SPIKES protocol has been created (14):

- Setting: set the stage for a collaborative discussion by preparing a comfortable, private space and ensuring that all parties whom the patient wishes to involve are present.
- Perception: assess the caregiver and/or patient's baseline knowledge about SUDEP using open-ended inquiry.
- Invitation: ask caregivers and/or patients directly about how much and what kind of information will be helpful to them. If a child is present, ask permission before proceeding with the conversation.
- Knowledge: share your knowledge about SUDEP in a clear, direct, and comprehensive manner.
- Empathy: acknowledge and respond to caregiver and patient emotions.
- Summarize: summarize the information that has been presented using lay language and present a plan for next steps, including referrals to further resources.

Communication about SUDEP is not just informational, it constitutes an integral component of prevention and patient care.

Burden on caregivers and loved ones

Beyond its neurological and physiological aspects, SUDEP also places a heavy psychological and social burden on families and caregivers. The sudden and unexpected nature of the death has a profound impact on their grieving process. In many cases, it is the family members who discover the body, often during the night or early in the morning, under circumstances that appear harmless. This brutal confrontation with death, combined with the lack of any obvious explanation, can lead to intense traumatic reactions, persistent feelings of guilt, anger, a sense of helplessness, and long-term psychological distress.

Several recent studies (15,16) have examined the experiences of parents and loved ones following a death due to SUDEP. One of the main findings highlights the central role that prior knowledge of SUDEP plays in the grieving process and the resulting coping mechanisms. Parents who had not been informed of the existence of SUDEP prior to the death often reported extreme shock, deep guilt, anger toward healthcare professionals, and maladaptive and persistent grief reactions. Some also reported persistent counterfactual thinking, wondering if the death could have been prevented had they been informed earlier. In contrast, parents who had prior knowledge of SUDEP described a less traumatic experience, characterized by lower levels of guilt and anger, greater acceptance, and a stronger sense of agency in understanding the event.

Longitudinal studies also suggest that grief following a SUDEP evolves over time. Although sadness often persists for years, feelings of anger, guilt, and relationship difficulties tend to gradually subside, while acceptance gradually sets in. The first few months following the death are particularly difficult, with significant repercussions on mental health, interpersonal relationships, and daily functioning. It is important to note that supportive social interactions (particularly exchanges with understanding peers, family members, healthcare professionals, and support groups) have consistently been identified as major factors facilitating adaptation and the psychological healing process.

These studies have also highlighted the challenges families may face immediately following a death from SUDEP. Parents often reported difficult interactions with emergency responders, law enforcement, and healthcare professionals, who were not sufficiently informed about SUDEP or ill-prepared to handle the aftermath of such an event. Such experiences can further exacerbate psychological distress, confusion, and feelings of abandonment.

These observations suggest that the impact of SUDEP extends far beyond the death itself. Because of the sudden and unexpected nature of SUDEP, loved ones are often confronted simultaneously with grief, a sense of bewilderment, and questions that arise in hindsight. Communication about SUDEP therefore serves not only a preventive function but also has a major psychological and ethical dimension. Informing patients and their families about SUDEP can help reduce the traumatic impact of the loss, facilitate the implementation of proactive coping strategies, and alleviate the profound sense of incomprehension often associated with sudden and unexpected death in epilepsy.

Conclusion

In conclusion, SUDEP occupies an unusual and challenging position in medicine. Although defined retrospectively and essentially by exclusion, it nevertheless constitutes a major clinical reality and one of the leading causes of premature death in people with epilepsy. Over recent decades, important progress has been made in identifying risk factors, understanding the physiological mechanisms involved, and developing preventive approaches centered on seizure control, nocturnal supervision, early intervention, and education of patients and their caregivers.

Current evidence increasingly suggests that SUDEP does not result from a single isolated mechanism, but rather from a complex interaction between severe seizures, respiratory dysfunction, loss of autonomic homeostasis, impaired arousal, and cardiac failure. At the same time, the persistence of residual unpredictability of SUDEP highlights the current limits of medical knowledge and prediction rather than the impossibility of future progress. Therefore, biomedical research remains essential.

Beyond its biological mechanisms, SUDEP also raises major ethical, psychological, and social issues. The burden for caregivers and families extends far beyond the death itself, often combining traumatic grief, incomprehension, guilt, and retrospective questioning. The literature consistently shows that communication about SUDEP plays a central role not only in prevention, but also in psychological preparation and coping. Informing patients and families about SUDEP should therefore not be considered optional, but rather an integral component of comprehensive epilepsy care. As the words of parents who have lost a child with epilepsy are the most accurate, beautiful, and meaningful: “No parent should ever lose their child. Secondly, no parent should ever lose their child to something they had no earthly idea about.” (15)

SUDEP informs us that medicine is sometimes faced with uncertainty itself. Understanding, predicting, preventing, and communicating about SUDEP remain inseparable dimensions of the same clinical and human challenge.

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Women's Voices: inspiring the neuroscientist community

Aliya Seelarbokus

Sara Carracedo[1], Chiara Galizia[2]
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Women's Voices is an interview section created in partnership with the Neurocampus Parity and Inclusion Committee (NeuroPIC), a local group committed to promoting equality and organizing actions to close the gap between women and men in academia. The goal of this section is to increase the visibility of early career female researchers at the Bordeaux Neurocampus of the University of Bordeaux. We interview researchers about their scientific contributions, insights and opinions about equity, diversity, and gender bias in academia. Through these interviews, we aim not only to highlight their achievements but also to serve as inspiration for our scientific community and other female scientists.

Together, we will bridge the gap!

This month in Women's Voices, we interview **Aliya Seelarbokus**, a second year PhD student from Mauritius, working on addictive-like eating in patients with stroke and obesity at INCIA. After completing her Bachelor of Science in Food and Human Nutrition and a Master's degree in Clinical and Public Health Nutrition in the UK, she moved to France for a PhD. Early on in her studies she became interested in the relationship between the Mediterranean diet and dementia, opening up the path to combining nutrition with neuroscience. In this interview she discusses her perspective on gender in the scientific work field across different countries, and the opportunities and challenges offered to international students in Europe.

Sara Carracedo: Could you tell us a bit about your academic journey and what led you to work in neuroscience?

Aliya Seelarbokus: My academic journey began in Mauritius, my home country. After completing high school in 2017, I moved to the United Kingdom (UK) to begin my Bachelor of Science (BSc) in Food and Human Nutrition, with no idea at all of one day integrating into the neuroscience field! During my second year at Newcastle University, I took a special liking to the role of the Mediterranean diet in the onset and progression of dementia and was particularly interested in healthy ageing. Owing to my genuine interest and curiosity for emerging research studies around this topic, I secured a 1-year contract as a Research Assistant at the Human Nutrition and Exercise Research Centre at Newcastle University. This first position allowed me to build on the knowledge I had accrued over my first two years of study and increased my eagerness to follow an academic career pathway in the field of nutrition and neuroscience. In 2022, I pursued my Master's in Clinical and Public Health

Nutrition at University College London (UCL) and I had the opportunity to publish my first systematic review examining the role of the Mediterranean diet among patients with Parkinson's disease. As I knew I had to pursue a PhD to meet my career goals, I explored PhD opportunities in France due to family commitments and was very fortunate to have been selected by the Bordeaux Neurocampus International PhD Program! Currently, I am working on food addiction and addictive-like eating behaviors among patients with stroke and obesity.

Sara Carracedo: You've worked in several countries, including the UK and France. What have you learned from navigating different academic systems across Europe?

Aliya Seelarbokus: When leaving the UK last year, I had mixed feelings as I was so well-settled in Britain's education system, but I was also very excited to discover a new country, a different academic system and culture! The transition from the UK to France has been fairly smooth as I realized that we are all connected through research, especially during a PhD where we are always looking out for collaborations with international universities. All of my PhD work is conducted in English and therefore there were no issues with the language, although it helped that I already knew French. I think that the key differences between both education systems are primarily during the Bachelor's and Master's years as the duration of the studies in the UK and France are slightly different. For example, in the UK, my BSc lasted for 4 years (with a professional year included) while my MSc was for a single year. In France, I understood that a BSc (or equivalent) is usually 3 years and typical Master's may be up to 2 years. However, both education systems converge to the same end point – where the PhD would be fairly similar across Europe as we collaborate internationally through mobility, conferences, and publication teams.

Sara Carracedo: How has mobility shaped your perspective on gender and inclusion in science?

Aliya Seelarbokus: If we consider the latest 2025 report of the French higher education and research ministry, we still note that more than half of the doctoral graduates are men and their work conditions are said to be better than for women. However, on a personal note, I have not faced any difficulties regarding my inclusion in science up to now, neither in the UK, nor in France. In my lab, I strongly believe that women are given opportunities, and we are all equally supported, irrespective of our gender. Therefore, I hope that there would not be any issues which would crop up as I pursue my academic journey.

Sara Carracedo: Have you faced any administrative or institutional barriers due to your international status? What could European institutions do better to support researchers from abroad?

Aliya Seelarbokus: No, there were no major barriers faced due to my international status. In fact, when I was at UCL, more than half of my classmates were international students and we were highly supported. However, once you've completed your studies in the UK, we have 2 years to stay under a work visa. After these 2 years, it is relatively hard to find a job and a sponsorship in the UK as an international applicant.

In Europe, depending on the country you are applying to, there are some PhDs which primarily target home students, and overseas students need to pay for the outstanding fees, which are very expensive.

However, I have been greatly supported by the International Neurocampus Graduate Program at the University of Bordeaux, as this program specifically targets and privileges applicants from abroad. Having specific programs both for home and international applicants does help to maximize opportunities for all.

Sara Carracedo: What would you say to other female scientists considering mobility as part of their career?

Aliya Seelarbokus: Be ready to take the challenge with unwavering commitment in the pursuit of your goals! Mobility should be seen as an exciting opportunity, and everyone should be given the chances as it helps you grow both professionally and personally!

NeuroPath: Exploring careers beyond academia

Ana Moreira De Sá, CRO scientist in preclinical research

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The world of science offers many exciting paths, and academia is just one of them. Each year, both the public and private sectors actively seek PhD graduates to fill diverse roles. However, many of them may seem unfamiliar to most of us. At Brainstorm, we want to help you explore career options that align with your interests, and aspirations.

That's why we created NeuroPath: a section dedicated to highlight scientific related careers outside academia. We reached out to professionals, who like us, have earned a PhD in neurosciences, most of them from the Neurocampus, but chose to apply their expertise in different fields. Through their stories, they share insights into their career journeys, their current positions as well other practical questions.

Science is a lifelong pursuit, but the path you take is yours to choose.

Follow the one that excited you the most!

This month in NeuroPath, we speak with **Ana Moreira De Sá**, currently working as a preclinical scientist in a contract research organization, MOTAC Neuroscience. She completed her academic training at the University of Bordeaux, where she obtained a Bachelor's degree in Molecular, Cell Biology and Physiology in 2014, followed by a Master's degree in Cellular Biology, Physiology and Physiopathology in 2016. She then pursued a PhD in Neuroscience from 2016 to 2019, focusing on neurological diseases, particularly ALS. Following her PhD, she continued with a postdoctoral position in Neuroscience from 2019 to 2020, before transitioning into industry.

Are you interested in knowing more about working in a contract research organization as a career path? Then this section is for you!

CRO scientist in preclinical research

Ana Moreira de Sá

I help with the development of future medicines for neurological disease

What is your role about?

As a preclinical neuropharmacology scientist for a CRO, my role involves the partnering with pharmaceutical companies worldwide to accelerate CNS drug discovery - from target validation through to pharmacokinetics studies. I run and oversee a small team performing experiments in rodent models (both mice and rats), with validated platforms for neuropsychiatric disorders. Due to confidentiality, most of the times I do not know the identity of the compounds tested; however, I am aware of the disease targets to apply the appropriate experimental models. Our company may be involved in a specific part of a project or follow it from start to finish. Since the company also has a "Translational Medicine Department", when successful, there is also some follow up on the clinical steps.

What made you choose this professional path?

After completing my PhD in Neuroscience, I wanted to move into a space where my work would have a more direct and tangible impact – contributing to the development of new drug targets and therapeutic strategies in close collaboration with pharmaceutical patterns and clinical teams. I was also attracted to industry because it offers clearer career progression, better work-life balance, and a more applied, translational approach compared to fundamental academic research.

What's a matching profile ?

An ideal candidate usually needs to have hands-on experience with rodent models and *in vivo* techniques - behavioral testing, surgical procedures, and electrophysiology. Data analysis and presentation skills are also very much appreciated. For "senior" roles like this, a PhD, along with experience in training and mentoring students is equally expected.

Do you have some advice for people interested in following this path?

The industry job market is highly competitive at the moment, so your CV needs to make a strong first impression – translate your PhD skills into industry-relevant skills/language. Keep your LinkedIn profile up to date and don't hesitate to reach out to the hiring manager when applying. Strong professional references who can speak to both your technical and interpersonal skills will further solidify your candidacy.

Main responsibilities

- Overseeing and conducting experiments (*in vivo* work)
- Experimental design and study regulatory documentation
- Data acquisition, analysis, visualization, and report preparation
- Contribution to study reports for clients, publications, and presentations
- Training and mentoring of junior staff

Requirements

This role calls for strong organizational, communication and teamwork skills, alongside a proactive and detail-oriented mindset. The ability to juggle multiple projects and people is equally important.

Working conditions

- **Work environment:** Given the company's relatively small size (~20 people), there is close collaboration and open communication across all levels. The team is super international and young, and company meetings are held frequently - roughly every two months.
- **Pressure level:** As a CRO, experiments are client-funded, so the delivery of reliable results on the previously established time is critical - failure has direct financial consequences for the business. That said, timelines are planned collaboratively with each client, which keeps deadlines realistic.
- **Work-life balance:** The work-life balance is very good. Schedule is fixed from 9:30 to 17:30 and any overtime work is balanced out with compensatory time off later.
- **Salary:** Again, since it is a relatively small company, the initial salary is similar to an experienced post-doc in academia. But the good thing is that salary revisions and progress evaluations are made often (every 6 months).

Do you have further questions?

Contact Ana at ana.moreira.sa@gmail.com

When the brain falls short

Interview with Michael Egerer

Juan Garcia-Ruiz[1]
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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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In the study of human behavior, researchers sometimes rely on *in vitro* experiments involving cells, tissues and organs. In other cases, computational models are necessary. Neuroscience itself covers many different dimensions of behavior. Yet one discipline is often overlooked in these discussions: sociology.

Our knowledge is built through concepts we create in society. Take gambling addiction as an example. Is the relationship between humans and gambling the same everywhere in the world? Clearly not. The way societies perceive gambling, and the way institutions define problematic behavior, strongly influence how we understand it. Therefore, how we study gambling addiction (or addiction more broadly) always depend on social and cultural context.

Looking at an issue from multiple perspectives is often the best way to understand it. In the case of addiction, understanding the biochemistry of the nervous system is not enough on its own. Focusing exclusively on what happens in the brain can be limiting. This is why neuroscience can benefit from being complemented by the sociology of addiction.

To offer a different perspective, encourage critical thinking, and avoid the reductionism that can come from explaining everything through biology alone, we spoke with Michael Egerer. Trained as a sociologist and currently a researcher at University of Helsinki, he studies the regulation of the gambling industry and the concept of addiction.

Juan García Ruiz: How do you study addiction?

Michael Egerer: Currently I am involved in a study looking at what is called the brain disease model of addiction (from a social point of view). The aim is to understand the implications of considering addiction as a matter of the brain, and how this can affect treatments and prevention, or yet how it is used by public discourse.

JGR: Have you already come to any conclusions?

ME: I just finished a manuscript on focus group interviews with persons in treatments for addiction. This brain disease model of addiction is promoted heavily by the National Institute of Drug Abuse in the USA. They are strategically pushing the idea that addiction is a chronic relapsing brain disease. This idea implies that what you do with the brain and medication can be good for treating addiction, and I am not discussing this part because this is not my topic. But they also argue that this model would decrease stigma saying that addiction is a disease. But you have other groups of researchers that think that if you say that these people have no autonomy anymore of what they are doing, then it actually increases stigma. People that have no agency in post-modern society, they cannot lead their own life.

From the interviews, my conclusion is that this debate is incomplete. It is not only a question of autonomy versus lack of autonomy. Another dimension that is often missing is biography. What I found is that the brain disease model can also help people make sense of their own life story. It can provide a more coherent narrative of what has happened to them over time.So in that sense, both positions in the stigma debate capture only part of the story. They focus on whether the model increases or decreases stigma, but they tend to overlook how these explanations also shape biography and identity.

JGR: How do we become addicted? Is there such a thing as a predisposition to addiction?

ME: We start to consider gambling an issue as soon as it causes problems. So, the etiology converges with the definition of the phenomenon. Of course there is a precise catalogue of criteria for identifying and diagnosing what is nowadays called Gambling disorder in the diagnostic manuals. Jim Orford simplified addiction basically towards the excess (for example, a loss of discrimination, meaning people can no longer use something in an appropriate way) and the emergence of problems. But then, such a definition risks including (and pathologizing) unlimited amount of unusual behaviours.

There are obviously factors predisposing persons to addiction, but I really would say that instead of continuing the contest between nature and nurture, I would like to put more emphasis on regulation. One becomes more easily addicted, others less, that is true. But both benefit from efficient regulation and prevention. Prevention should of course not be limited to education only, but offer also regulatory incentives. Normalisation is a key-matter here, though overdoing de-normalisation has its downsides as well.

JGR: How long has gambling addiction existed?

ME: Gambling addiction has probably always existed as long as there has been gambling. Although I want to point out that gambling itself is not human nature, there are cultures without gambling. But if you look at how addiction emerged you have to consider two factors. The first one is the nature of the problem which has changed over the last 200 years, and especially in the last 50 years. You have a globalization of gambling, industrialization and digitalisation of gambling. All these changes increase the offer of gambling and makes it more available. The second one is the perspective of social control. In comparison to previous times, people are expected nowadays to control themselves. Obvious external control, like sanctions, has diminished in the last 200 years. That puts the individual in the center of the control of gambling, and the same goes for the control of substance abuse.

JGR: Gambling policy is complicated because the industry generates significant revenue. From your perspective, what can realistically be done?

ME: This is of course a political question. Independently from what I think, there has to be a compromise between the different stakeholders that are involved. There are no magic bullets. Regulation has advantages and disadvantages. It needs to be balanced somehow. What is important is to keep in mind the weakest groups and keep them in the public and political discussion. It is much easier for the industry to lobby their interests (or even for the state itself). In the gambling field, looking at the research that has been done, it has been beneficial to disentangle the different interests institutionally. It's good to create independent organizations that supervise the industry. Clearly gambling regulation shouldn't be managed only by the same ministry, which has specific interests in gambling revenue. It's politically easier to get the money from gambling than to raise the income taxes for instance.

JGR: Is gambling regulation only focusing on setting limits?

ME: Education also matters, and there are lots of prevention and education programs on gambling. However, the problem is not necessarily that gambling regulation focus on setting limits, but the wrong limits are set. Gambling regulation focuses so far on individualizing the responsibility. Maybe you heard about the so-called responsible gambling. That concerns mostly the responsible gamblers. So gamblers need to make the right choices, with the help of gambling tools for instance. But the thing is that whenever they get into trouble, this is also their fault. The industry get often away with only mild limits concerning gambling addiction. So setting limits should also take into account regulatory framework on the industry.

JGR: Is there one message you would like people to remember?

ME: The dichotomy is not state paternalism versus individual freedom, as it is often claimed. At least not if we are talking about individual citizen freedom. The real dichotomy is between state paternalism and industry freedom, I would say. So again, the issue is to find the right spot between the two ends of the continuum. If you have only freedom from state paternalism, then you are more vulnerable to industry manipulation. If you let the industry to run free, then the individual citizen becomes manipulated in many ways. One should not be fooled that a minimal state control increases citizen freedom. It is important to be aware that state regulation also secures individual freedoms.

JGR: Would you recommend a book to our readers?

ME: I find very interesting Ian Hacking's *Rewriting the soul* (1995). He is a Canadian philosopher. This book does not specifically deal with addiction, but it deals with mental health. I think it demonstrates very well how crucial are concepts like mental health or addiction in defining what we consider reality, or defining the framework that allow us to understand who we are and who other people with these problems are. As a plus, I would say it's also fairly easy and quick to read for a book by a philosopher.

Neurometa

"In the loving memory of all the souls we lost to the SP8" Vasika Venugopal

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Toshiko, originally from New Zealand, is currently PhD student at the Nutrition et Neurobiologie Intégrative (Nutrineuro). She holds a bachelor's in Biology from the University of Hawaii and a master's in agro-biomedical Science from the University of Tsukuba, Japan. She is also passionate by scientific illustration!

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Poorvi, originally from India obtained her Bachelor of Engineering in BMS College of Engineering, Bengaluru. She then moved to the land of bicycles and tulips to pursue a Master's degree in Molecular and Cellular Life Sciences. She is currently doing a PhD at the IINS, studying NMDAR clustering in early synaptogenesis.

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Daniele Stajano was born in Naples (Italy). He has a Bachelor's degree in Biology and a Master's degree in Neurobiology. After his Ph.D. in neurosciences at the ZMNH of Hamburg (Germany), he joined as postdoctoral student the IINS. He is currently interested in molecular mechanisms orchestrating brain maturation in neurodevelopmental disorders such as the autistic spectrum disorder.

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