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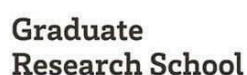
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Cortistatin, a neuropeptide with diverse effects and promising potentials

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Abstract

Cortistatin (CST) is a neuropeptide predominantly expressed in the cerebral cortex, sharing structural similarity with somatostatin but arising from a distinct gene. Studies demonstrate its neuroprotective role in models of seizures, ischemia and neurodegeneration. Additionally, CST acts as an immunomodulator in neuroinflammatory conditions and exhibits antinociceptive effects, reducing inflammatory and neuropathic pain. These diverse actions highlight CST as a promising therapeutic candidate for different neurological pathologies.

Keywords

Cortistatin, Neuroprotection, Neuroinflammation, Antinociception.

Abbreviations

CST – Cortistatin

PreproCST – Preprocortistatin

ProCST – Procortistatin

SSTR – Somatostatin receptor

CNS – Central nervous system

RA – Rheumatoid arthritis

BBB – Blood-brain barrier

IBD – Inflammatory bowel disease

MrgX2 – Mas-related G-protein coupled receptor X2

DRG – Dorsal root ganglia

GHSR – Growth hormone (GH)-secretagogue receptor

GPCR – G-protein-coupled receptor

KA – Kainic acid

ICV – Intracerebroventricular

MPTP – 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine

SNpc – Substantia nigra pars compacta

TH – Tyrosine hydroxylase

DAT – Dopamine transporter

BDNF – Brain-derived neurotrophic factor

Treg cells – Regulatory T cells

EAE – Experimental autoimmune encephalomyelitis

IP – Intraperitoneal

IL-12 – Interleukin-12

TNF- α – Tumor necrosis factor- α

IL-6 – Interleukin-6

IL-17 – Interleukin-17

IFN- γ – Interferon- γ

IL-1 β – Interleukin-1 β

SP – Substance P

CGRP – Calcitonin gene-related peptide

Introduction

Cortistatin (CST) is a cyclic peptide initially isolated from rat brain and named after its predominantly cortical expression, strong similarity with somatostatin and ability to depress neuronal activity. CST is cleaved from a prepropeptide known as preprocortistatin (PreProCST). PreproCST begins with a 27-residue apparent secretion signal sequence. In humans, cleavage of the preproCST to procortistatin (ProCST) would produce a protein that could be further processed at either of two tandem basic amino acid pairs to produce CST-29 and CST-14. CST-14 shares 11 of its 14 amino acids with somatostatin-14, but despite this high similarity, they are products of distinct genes. The distribution pattern of preproCST mRNA, determined by in situ hybridization on rat brain sections, indicates that preproCST mRNA is expressed in scattered neurons throughout the cerebral cortex and hippocampus, and is distinct from preprosomatostatin expression.

CST can bind to all somatostatin receptors (SSTR1-5) with affinities comparable to those of somatostatin (1). Previous studies have demonstrated that somatostatin receptors (SSTR) ligands exert neuromodulatory effects following ischemic injury (2), which has prompted investigation into the potential neuroprotective properties of CST in various models of central nervous system (CNS) injury. In addition, both SSTR ligands and CST have been shown to regulate immune response by balancing pro- and anti-inflammatory signals (3). This dual role has generated increasing interest in the potential

involvement of CST in neuroinflammatory processes, particularly in light of ongoing research exploring its therapeutic potential in inflammatory diseases such as rheumatoid arthritis (RA) and inflammatory bowel disease (IBD).

Furthermore, CST has been identified as a high potency ligand of Mas-related G-protein coupled receptor X2 (MrgX2), a human orphan G-coupled receptor with high expression in the dorsal root ganglia (DRG), particularly in the small-diameter neurons of the DRG associated with acute and neuropathic pain (4). Interestingly, overlapping distribution of CST and MrgX2 was seen in the DRG (5). This ligand-receptor pair has influenced researchers to study the role of CST in nociception. Another interesting coupling is with growth hormone (GH)-secretagogue receptor (GHSR), a G-protein-coupled receptor (GPCR), the endogenous ligand of which is acetylated ghrelin. CST is found to bind to GHSR with affinity comparable to that of ghrelin (3). This interaction is being investigated as a possible source of the analgesic effect of CST in models of neuropathic pain.

Following its discovery, many studies have investigated the potential effects of CST on the pituitary gland and systemic immune responses. These findings have led to proposals for the use of its analogues as therapeutic agents in the treatment of various autoimmune diseases. However, this review will visit the existing literature to highlight the potential roles of CST in neuroprotection, neuroinflammation, and pain modulation.

Methods

The PubMed database was searched using the following combination of keywords: (CST) AND (stroke OR "ischemic stroke" OR "cerebral ischemia" OR "cerebral infarction" OR neuroprotection OR neuroprotective OR neuroinflammation OR inflammation OR immunomodulation OR microglia OR astrocytes OR "blood-brain barrier" OR BBB OR cytokines OR apoptosis).

Initial search resulted in 103 articles. After title screening, duplicate records (n=1) and studies unrelated to the nervous system (n=44) were excluded. The remaining 58 papers underwent abstract screening to exclude studies outside the scope of this review (n=23).

Following that, full-text review was conducted to 35 articles. Of these, 18 articles (including both original research and review papers) were included in the final synthesis

Results and discussion

Figure 1 summarizes the overall roles that CST plays in the nervous system.

Neuroprotection

Since its discovery in 1996, many experimental studies have been done to establish the neuroprotective roles of CST. The earliest discovery showed that CST-14 reduces seizure severity and neuronal death following Kainic acid (KA) administration in rat models (6). In this study, CST was administered intracerebroventricularly (ICV), indicating a direct central effect, likely related to modulation of neuronal excitability and attenuation of excitotoxic damage (6).

Since then, many experiments focused on the roles that CST can play in cerebral ischemia. It has been found that CST expression is downregulated in non-somatostatinergic neurons following ischemic injury, suggesting a loss of endogenous protective signaling. Importantly, central (ICV) administration of CST significantly reduced ischemic damage

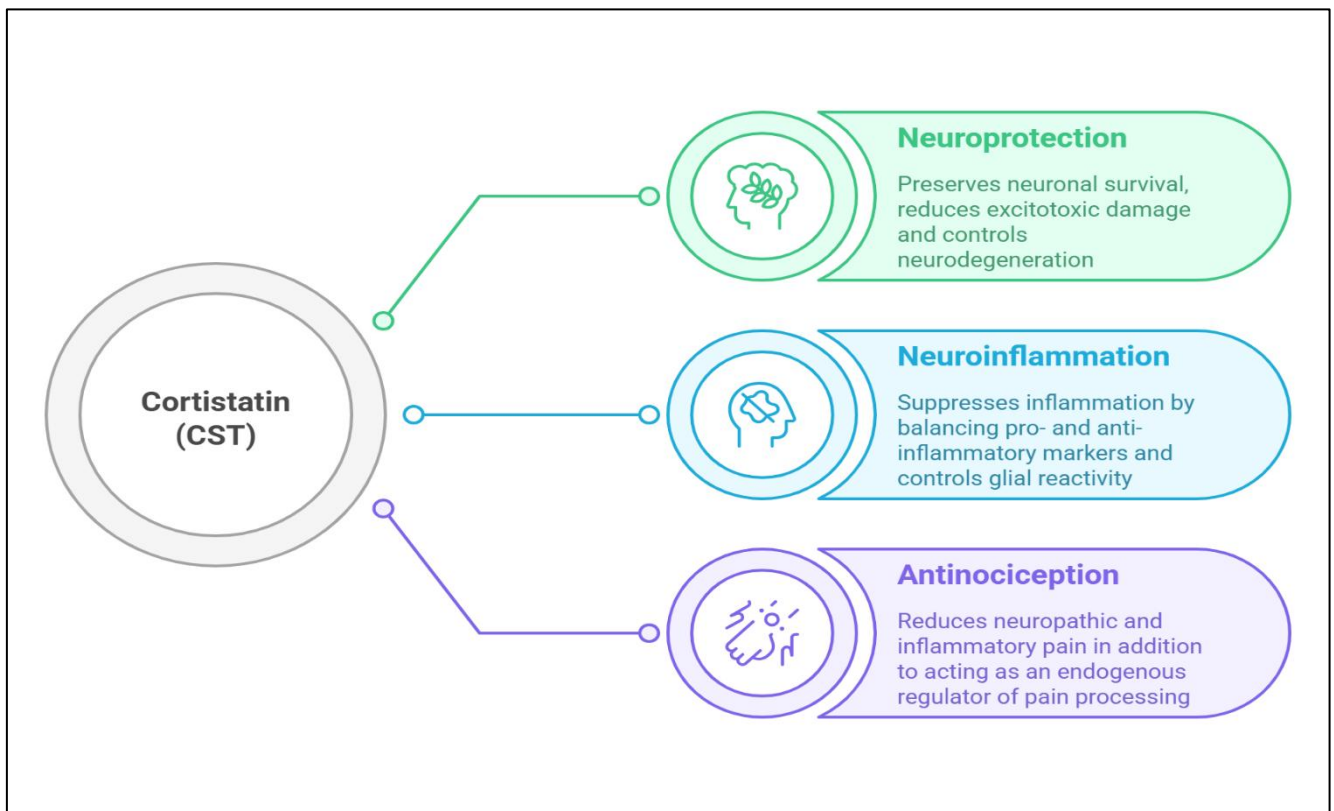


Figure 1: The multifaceted roles of CST in the nervous system

compared to saline-treated controls by (52%) (7). These protective effects are thought to involve modulation of neuronal injury processes, although the precise intracellular mechanisms were not fully characterized.

In addition, in chemically induced ischemia in rat retina, intravitreal administration of CST and attenuated damage caused by the ischemia in a concentration dependent manner (8). More recent evidence indicates that CST retains therapeutic efficacy when administered intraperitoneally up to 24 hours after stroke, significantly improving neuronal survival. This delayed neuroprotection reflects the pleiotropic actions of CST, encompassing not only direct effects on neuronal viability but also modulation of post-ischemic inflammatory responses, including reduced production of pro-inflammatory mediators. In addition, CST contributes to the preservation of BBB integrity, mitigating vascular dysfunction and limiting secondary injury processes such as edema and immune cell infiltration. Together, these findings highlight CST as a multimodal therapeutic agent targeting the neuronal, immune, and vascular components of ischemic stroke pathology. (9).

From a pathophysiological perspective, CST was found to have anti-atherosclerotic effects in a mouse model of hyperlipidemia. Following intraperitoneal administration, CST reduced the development of arterial plaques and inhibited foam cell formation, suggesting a role in regulating inflammatory and metabolic processes that may indirectly contribute to cerebrovascular protection (10).

Further support for CST's neuroprotective potential comes from recent experimental evidence in neurodegenerative models. In a mouse model of Parkinson's disease induced by 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP), intraperitoneal administration of CST significantly preserved dopaminergic neurons in the substantia nigra pars compacta (SNpc) and maintained striatal dopaminergic innervation. This was accompanied by restoration of dopamine levels and normalization of key dopaminergic markers such as

tyrosine hydroxylase (TH) and dopamine transporter (DAT). Notably, CST also restored brain-derived neurotrophic factor (BDNF) expression, suggesting that its neuroprotective effects include the restoration of neuronal survival pathways (11).

These findings indicate that CST has the potential to be a therapeutic agent used in adjuvant to reperfusion therapy following stroke; and as a neuroprotective agent in neurodegenerative diseases. However, much about the exact role of CST and its interaction with other inflammatory modulators in ischemia and neurodegeneration are yet to be discovered.

Neuroinflammation

In the periphery, CST acts as a pleiotropic modulator of the immune system—meaning it exerts multiple, context-dependent effects on different immune cell populations—by balancing pro- and anti-inflammatory responses and promoting the differentiation of regulatory T cells (Treg cells), a subset of T lymphocytes that suppress autoreactive immune responses and maintain immune tolerance (12).

Experiments support the therapeutic role of CST in autoimmune neuroinflammation. In a mouse model of experimental autoimmune encephalomyelitis (EAE) that mimics multiple sclerosis, intraperitoneal (IP) CST treatment markedly reduced disease incidence and severity, with many treated animals showing only mild symptoms or complete recovery 20–30 days after disease onset. CST therapy reduced inflammatory infiltration into the central nervous system and decreased the expression of pro-inflammatory mediators including interleukin-12 (IL-12), tissue necrosis factor- α (TNF- α), interleukin-6 (IL-6), interleukin-17 (IL-17), interferon- γ (IFN- γ), while preserving anti-inflammatory cytokine levels (13).

CST also demonstrates protective effects in infection-associated neuroinflammation. In a rat model of *Klebsiella pneumoniae* meningoencephalitis, CST was administered ICV, resulting in reduced leukocyte recruitment, decreased pro-inflammatory cytokines in brain

tissue and cerebrospinal fluid, minimized neuronal apoptosis, and preserved neuronal survival in the cortex and hippocampus (14). Similarly, in sepsis-associated encephalopathy, CST administered intraperitoneally reduced neuroinflammatory responses, by inhibiting overactivation of microglia and preserving the BBB integrity (15). Recent evidence also shows how crucial CST is for cerebrovascular homeostasis since its deficiency leads to endothelial dysfunction, increased immune activation, leukocyte infiltration and disruption of BBB integrity (16).

Importantly, recent findings from neurodegenerative disease models further reinforce the immunomodulatory role of CST. In the previously mentioned MPTP model of Parkinson's disease, IP administration of CST significantly reduced both central and systemic inflammation. In the brain, CST decreased the expression of key pro-inflammatory cytokines such as TNF- α and interleukin-1 β (IL-1 β) and attenuated astrocyte and microglial activation. Moreover, CST induced a shift in microglial phenotype toward a more ramified, homeostatic state, rather than a reactive, pro-inflammatory profile. Similarly, CST reduced circulating levels of inflammatory mediators, including IL-6, TNF- α , demonstrating that its effects extend beyond the brain to the peripheral immune system (11).

Altogether, growing evidence highlights the strong immunomodulatory effects of CST, suggesting that its therapeutic application in neuroinflammatory conditions and infections may become feasible in the near future.

Antinociception

CST has been shown to exert antinociceptive effects similar to those of somatostatin supporting its role as an endogenous regulator of pain processing (3). Experimental studies further demonstrate that CST deficiency results in increased sensitivity to thermal stimuli, enhanced visceral pain responses, and exacerbated hyperalgesia and allodynia in inflammatory and post-operative pain models. Conversely, exogenous CST administration reduces

pain-related behaviors through multiple routes, including intrathecal (spinal), local, and intraperitoneal (systemic) administration, suggesting both central and peripheral mechanisms of action. These effects are associated with reduced production of pro-inflammatory mediators and attenuation of inflammatory responses in both peripheral tissues and the spinal cord (17).

More recent evidence shows that CST can minimize neuropathic pain in models of peripheral nerve injuries. CST reduced hyperalgesia and allodynia, two hallmark features of chronic neuropathic pain. Furthermore, CST deficiency exacerbated neuropathic pain responses, suggesting an endogenous effect that CST has in alleviating pain signals. Interestingly, the analgesic effects of CST were blocked by sstr2 and GHSR1 antagonists indicating a complex mechanism of action for CST as an antinociceptive agent. Additionally, CST was found to decrease the expression of pronociceptive peptides substance P (SP) and calcitonin gene-related peptide (CGRP) in DRG neurons (18).

Overall, these findings indicate that CST reduces pain through modulation of inflammatory and neuroimmune processes, although the precise receptor subtypes and intracellular signaling pathways involved remain to be fully elucidated.

Conclusions

Although CST was discovered recently, research into its biological roles has expanded rapidly. Increasing evidence highlights its diverse physiological functions, particularly its immunomodulatory and neuroprotective properties, which make it a promising therapeutic agent in autoimmune and inflammatory diseases. CST's ability to bind to multiple receptor families and its presence in different regions of the central nervous system add further complexity to its biological actions. These characteristics have drawn interest from neuroscientists seeking to better understand how CST interact with neurons and glial cells.

Existing literature suggests that CST participates in diverse processes. However, many aspects of its mechanisms of action remain hidden, particularly regarding its signaling pathways.

This literature review aims to highlight current knowledge surrounding CST and its potential roles in neuroprotection, neuroinflammation, and nociception. It also illustrates how the study of a single neuropeptide with different biological functions can be helpful in understanding disease pathophysiology and may ultimately contribute to the development of novel therapeutic strategies.

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Who do we brain researchers think we are?

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Christopher Stevens is a postdoc at INCIA. He originates from Dublin, Ireland, but did his university studies in philosophy in Paris (Nanterre, Paris X and La Sorbonne, Paris IV) before coming to Bordeaux to study neuroscience. He has a PhD in neuroscience from the University of Bordeaux (2022) and a PhD in philosophy from Université Bordeaux-Montaigne (2026). Currently, he is a postdoc in Serge Ahmed's Choice, Addiction and Neurodysfunctions team, working on the question of how to model addiction-like phenotypes in non-human animals.

As neuroscientists, we are accustomed to seeing and using phrases like “the neurobiological underpinnings of [such and such a phenotype of interest].” We also know that the phenotypes whose neural bases we investigate are rarely easy to isolate and define, representing a challenge that often demands years of back and forth between research at the internal neurobiological and research at the externally observable (behavioral or symptomatic) levels. Indeed, where the object of investigation is not satisfactorily defined, it can be the neurobiological underpinnings of something quite distinct from our stated target that we end up fluorescing, recording, or activating/inhibiting, constituting research digressions which often delay progress in the fitting direction. Illustrative of the challenge is the winding path of research that led to behavioral and neuronal differentiation of the “passive” immobility of learned helplessness and the “active” mobility-inhibiting freezing of adaptive fear response (1).

At more abstract levels of brain function, this definitional challenge only becomes trickier. Little surprise, then, it is here that certain questions essential to progress in neuroscience become indistinguishable from questions philosophy has been reflecting on for millennia: What is belief? What is thought? What is wanting or desire? Do our behaviors result from free will or are they determined by physical causes?

One of the seeds of the modern scientific enterprise was itself the fruit of philosophical reflection on how the interaction of desire, belief, and reasoning can unconsciously orient behavior in unwilling directions. Francis Bacon, albeit in the language of his own time, situated our inquiring minds in a continuous process of cognitive confrontation: our beliefs (hypothetical conceptions about how nature works) versus our perceptions (observations of how nature compares to these hypotheses). He also described how we tend to resolve dissonances in these confrontations by altering or diminishing facts that may otherwise contradict our prior beliefs, rather than by revising our beliefs in order to make them accommodate the new information as objectively as possible :

“Once an individual's understanding has settled on something (either because it is an accepted belief or because it pleases them), it draws everything else also to support and agree with it. And if it encounters a larger number of more powerful contradicting examples, it either fails to

notice them, or disregards them, or makes fine distinctions to dismiss and reject them” (*Novum Organum*, Book I, Aphorism XLVI) (2).

In short, Bacon was laying the foundations for the study of cognitive biases, phenomena he labelled *idola mentis* (“idols of the mind”) because of how they captured human minds yet had no real power behind them. Most pointedly, however, Bacon observed that his highly educated peers were just as susceptible to these self-deceptive “idols” as unschooled minds. Demonstrating impressive psychological perspicacity, he asserted that, precisely *because* of their heightened intellectual capacities, his fellow scholars possessed remarkable skill in taking otherwise contradicting information and molding it into elegant arguments in favor of their preferred preexisting theories.

In modern psychological terms, this type of phenomenon is called *motivated reasoning* (3). And when reasoning is motivated towards confirming ‘my side’ of a given situation (e.g. *my* political position, *my* spiritual conviction, *my* scientific hypothesis), it is aptly named *myside bias* (4). Bacon’s proposed remedy to this kind of biased thinking, his *novum organum* (“new instrument”), was the empirical method; a methodological tool for making observable reality, rather than human reason, the measure of all true knowledge of – and thus power over – nature. Hence his famous maxim “knowledge is power.”

It is probable that Bacon, a dualist with respect to mind and body, never considered the possibility that one day belief and desire would themselves become objects of empirical biological research. But, as neuroscientists, this is precisely the unique-among-researchers situation we operate in: subject to the effects of such biases while in the very activity of subjecting their biological substrate, the central nervous system, to empirical method.

For my own doctoral research into myside bias, one of the most influential discoveries in neurobiology was the identification – in a series of classic papers from McDonald and White (5–7) – of multiple memory systems. Each of these was described as being centered on (but not limited to) particular brain regions: habit-like stimulus-response memory centered on the striatum; affective memory centered on the amygdala; and relational episodic memory (i.e. relating *wheres*, *whats*, and *whens*) centered on the hippocampus. This memory system dissociation suggested to me a hypothesis that the kind of myside bias first described by Bacon could be explained by cortico-hippocampal deliberation over episodic and semantic cognitive content (i.e. reasoning over experiences and facts) weighted by inputs from more deeply embodied habit-like striatal and/or affective amygdalar memories.

To test this hypothesis, we adapted Aline Marighetto’s mouse model of hippocampus-dependent everyday-like episodic memory (EdM) (8) by placing it in competition against a previously acquired habit-like stimulus-response tactile discrimination rule (of the kind shown by McDonald and White to be striatus-dependent). Among our observations, two were particularly striking: 1) In mice who had previously acquired a habit-like tactile discrimination rule, responses became significantly more biased towards it as the episodic memory challenge became more difficult to resolve, i.e. more cognitively ambiguous; 2) this biased responding wasn’t faster and more heuristic, but tended to be significantly more deliberative than decision-making in control mice (9).

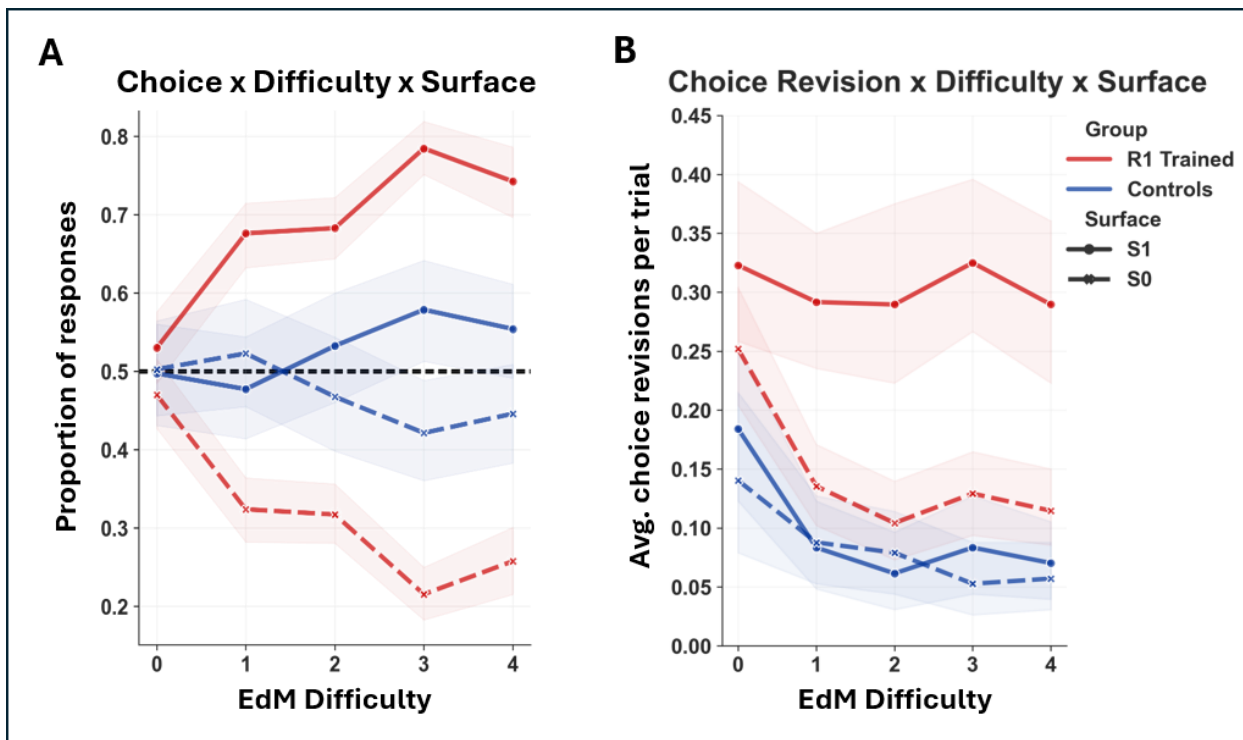


Figure 1 - Bias towards a habit-like behavioral rule increases as task difficulty increases but manifests deliberately, not heuristically. **A)** On the easiest trials of the episodic EdM task (level 0), mice previously trained to habitually respond to a tactile stimulus-response rule ('R1 Trained' group, $n=40$, red lines) displayed almost no response preference for the surface previously learned to be "always rewarded" (S1). However, the proportion of responses biased to S1 increased significantly as a function of increasing EdM difficulty, i.e. as the EdM trials became putatively more difficult to cognitively represent and resolve. In control mice ($n=19$, blue lines), no significant difference in surface choice was observed. **B)** Replicating prior EdM research (13), deliberative choice revision in control mice decreased as a function of increasing EdM difficulty (putatively due to EdM challenge-mediated representational weakening: for details see 13). In R1 Trained mice, however, choice revision biased towards S1 (solid red line), but not choice revision towards S0 (dashed red line), remained significantly higher at all difficulty levels. This result suggests that, in a process reflective of motivated reasoning, the previously learned habit-like tactile rule not only amplifies neurocognitive deliberation but does so as a means of biasing behavioral output towards itself.

These results were compelling because they reflect two of the most characteristic features of myside bias as described in psychological studies in humans: 1) That it is preferentially expressed, not in a heuristic 'think fast' "system-1" manner (10), but rather through a deliberative process of motivated reasoning, and; 2) that its prevalence increases as a function of a subject's cognitive uncertainty with respect to a given challenge (11,12).

Our experimental behavioral results thus indicate neurocognitive processes by which a willed or intentional behavior (e.g. to objectively process and adaptively act on novel environmental information) can be overridden by a deeper, unwilled, and sometimes opposing behavior (e.g. to defend established beliefs against challenge). That this typically occurs in a manner which nevertheless leaves us believing that we are, in fact, still behaving in the way we intended is what makes myside bias such a difficult challenge to overcome. It also raises profound philosophical questions about the nature of the self and free will: when the executive intention to be rational is unconsciously overridden under the weight of a "deeper" neural system, *who* carries out the consequent action?

Nor is this kind of 'self-splitting' phenomenon unique to myside bias: in forthcoming work, we identify a form of it in everyday-like memory function (13), and in my current research project – with Serge

Ahmed – I have begun exploring a similar question in the domain of addiction, where the conflict between an addict’s will to recover and their persistent craving for the object of addiction can often lead to unwilling relapse. Nevertheless, as neuroscientists defined by our (brain) research habits, it is myside bias and its potential negative effects which impact us most: in how we define the concepts and objects whose neural bases we seek to identify, in how we formulate and test our neuroscientific questions, in how we interpret the data we collect, and in how we communicate all of this to our peers.

The pragmatist philosopher John Dewey – who saw himself as continuing the empirical Baconian tradition (14) and is often identified as the godfather of the “critical thinking” movement (15) – also conceived of the self and the will in a multiple systems-like manner, as an “interpenetration of habits” (16). Moreover, he used this habit-based conception to promote the practical importance of working towards developing better “habits of thought.” Intentionally *practicing* the behaviors one wants to become habits was, for Dewey, therefore the most concrete way to manifest and exercise one’s will.

Using what we are discovering about how habit-like (putatively striatum-centered) behaviors can override our executive (cortico-hippocampal) intentions, we can now add a neurocognitive explanatory layer to enrich this “moral” conception of habit development. In other words, if our true intention as brain researchers is, for example, to exercise critical thinking, then we should understand better than all other researchers why it is not sufficient to attempt to do so only now and then. Rather, it should be practiced regularly, to the point of becoming a neuronally instantiated habit of thought, lest some other *less* critical prior habit – such as merely endeavoring to confirm our own hypothesis – override our good intentions without us even (consciously) realizing it.

To conclude, a timely note of caution regarding the development of science-conducive habits of thought seems fitting. It has now been widely demonstrated that generative AI, in its current publicly available forms, can produce a range of negative cognitive effects in users, effects which the foregoing neurocognitive considerations on biases and habits can only underline. The tendency for genAI interfaces to sycophantically reinforce users’ preexisting theories can make executive inhibition of priors more difficult, progressively weakening the neurocognitive capacity to develop a habit of critical thought, and leading in extreme but numerous cases to myside bias hardening into ‘AI psychosis’ (17–19). Similarly, more and more publications are already describing behavioral dependencies whereby frequent genAI users are developing addiction syndromes (20–22), using AI to offload cognitive effort to such an extent that, like those substance abusers who use drugs to offload emotional burden, they can no longer function without it and their own investigative will is reduced to posing questions to an algorithm (23). Indeed, given all that we have empirically discovered about neurocognition in the century since Dewey penned his thoughts, these negative effects on brain function, and the reasons we ought therefore to actively avoid them, should be more salient to the neuroscience community than to any other: *caveat promptor*.

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When music makes us move

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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.

Imagine a great song. With the melodies of the guitar and vocals and the pulsing rhythm of the bass and drums, something seems to itch inside you to move. It makes you dance; it might motivate you to start cleaning, or the music might accompany you on a run. If something so simple yet so powerful can make anyone active, could it also help people who have trouble initiating movement?

Parkinson's disease (PD) is a progressive neurodegenerative disorder that affects both movement and cognitive functions, thereby strongly reducing a person's quality of life (1). One main symptom associated with the disease is bradykinesia, which essentially means slowness in movement. This is associated with difficulties initiating movements, unexpected pauses in movement and can result in problems with balance, leading to a fear of falling, which in turn, can further limit a patient's mobility (2,3). The disease is characterized by the degeneration of dopamine-producing neurons in the substantia nigra, which project among others, to the basal ganglia, a brain region involved in controlling and initiating movement (4). Current treatments range from medication to deep brain stimulation and rehabilitation (1). However, research also shows that adding different forms of therapy, such as music therapy, to the treatment plan, can improve quality of life and alleviate symptoms associated with the disease (5,6).

Music therapy has been shown to have behavioral, cognitive, psychosocial and motor benefits in patients with neurodegenerative disorders such as PD (7). One of its main focuses in Parkinson's disease is gait rehabilitation, which aims to help restore a more regular walking rhythm (8). This therapy method uses a treadmill together with external rhythmic cues, known as rhythmic auditory stimulation (RAS).

These cues can range from a simple metronome click, to music with a clear and regular beat. The idea is that an external rhythm may help compensate for difficulties in generating an internal rhythm, which normally relies heavily on the main brain circuits affected by Parkinson's disease. Instead, the rhythm provided by sound can engage auditory and motor brain areas through a different pathway, helping the person coordinate their movements with the beat (8,9).

But what if this therapy could be taken home?

Having access to a treadmill at home, especially one with an integrated RAS system, might not be possible for everyone, and limiting training to scheduled therapy sessions can make regular practice difficult. New approaches using smartphones could provide a more convenient and accessible alternative (10). Users can choose music based on their preferences from different playlists, while the beats per minute are adjusted to match their ideal, personalized, walking tempo. This approach thereby not only increases the motivation to walk as you get to choose the music yourself, but it also allows the user to incorporate music cued walking into real life routines, such as outdoor walks. This could make rhythmic training not only more accessible but also have a higher impact on a patient's quality of life.

Music therapy will not cure Parkinson's disease, but it offers a complementary approach that can potentially improve important aspects of everyday life. And Parkinson's disease is only one example of how music can interact with the brain. Music-based therapies have also been used to improve speech in people who have suffered a stroke (11), and to reduce anxiety in people with dementia (12).

Now, despite these promising effects, we still do not fully understand exactly how music produces these changes in the brain. Which pathways are being activated? How can an external rhythm help us move when our internal timing system is impaired? And how can singing sometimes help someone produce speech after a stroke? Researchers are starting to tackle these questions by discussing the need of studies looking at brain activity during music therapy approaches for neurodegenerative diseases. Answering these questions could help us understand the complexity of brain connectivity and plasticity, and how the brain can find alternative ways to produce movement or speech through something as simple and powerful as music.

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Women's Voices: inspiring the neuroscientist community

Silvia Sposini

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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!

Hello, welcome to Brainstorm Women's Voices! To kick things off, could you give us a short introduction of yourself and your career path so far?



Silvia Sposini: Hello everyone, my name is Silvia Sposini and I'm a newly appointed Assistant Professor at the University of Bordeaux.

I'm Italian and I completed both my Bachelor's and Master's in Italy. As soon as I finished school, I knew I loved studying and I found biology fascinating. While I wanted to be a marine biologist and follow orcas, my family had higher expectations for me. We eventually reached a compromise. I decided to go for biology, but through a more realistic path, so I chose cellular and molecular biology.

Towards the end of my Master's, I started an internship in Italy in a biochemistry department laboratory, which I loved. I knew that I loved being in the lab, but I also wanted to explore something outside Italy. I managed to find a short internship abroad, which ended up being at Imperial College London which I later extended. Then I applied for PhD positions everywhere in Europe and ended up getting a position at Imperial College, so I decided to stay.

Towards the end of my PhD, I thought it was time for a change—not only science-wise, but also in my personal life. I thought it was the right time to experience life in another country. Again, I started looking for postdoc positions everywhere in the world. I kind of knew what I wanted to do: use microscopy and continue to study what I had been doing during my PhD. I've always worked on G protein-coupled receptors, or GPCRs. I wanted to stay in that field but also explore a new physiological setting. My PhD

was on reproductive biology, and for my postdoc I decided to move to neuroscience. It was something I had always wanted to do but had never had the chance to study or work on.

I thought Bordeaux and the IINS were the perfect combination of these two things. Specifically, I contacted David Perrais because he had the expertise and research topics that were interesting to me at that time, which is how I arrived here in Bordeaux. I also came up with an idea that I had developed between my PhD and my postdoc. I proposed to him a project looking at G protein-coupled receptors, membrane trafficking, and how this has an impact on the signaling of these proteins, which was something new to the Institute. I applied for several postdoctoral funding schemes, and I was lucky enough to get incredible funding from the UK Wellcome Trust, which funded my research for six years.

Over these years, the project evolved. I wondered many times, “What will I do next when I grow up?” There have been many times when I thought I might quit academia because we all know there are hard times. But I think my dream and aspiration have always been to stick to public research. Eventually, I managed to get there, and I’m incredibly happy and honored to be in this situation.

That was an incredible journey. To focus more on what you do now, what is the topic of interest in your research that you’ll be following for the next few years



Silvia Sposini: The theme of research is essentially this: there are many molecules, hormones, peptides and so on that are sensed and detected by neurons and dictate how neurons respond. The molecules that translate an extracellular signal into an intracellular message for the cell to decode and then translate into a cellular function are actually receptors, and most of them are G protein-coupled receptors.

They are hugely studied in model systems such as immortalized cell lines, but very poorly studied in neurons. However, we know that neuromodulators have a huge impact on the brain in general and modulate essentially all possible physiological functions—sleep, mood, appetite, memory and so on. So, my aim is really to investigate, at a molecular level, how these G protein-coupled receptors do their job and how they influence neuronal functions.

You mentioned that with your permanent position you can continue your research, but you’ll also be teaching. What do you think will change now that you will do both? And what does it mean to you to educate and train the next generation of researchers?



Silvia Sposini: I must admit that when I was applying for positions, I applied for some positions that involved teaching and some others that didn’t. I was scared by the teaching—not only by the workload, but also by the responsibility. However, during the process and when I knew I got this position, a lot of the uncertainty disappeared. This was also thanks to the people I met throughout the years and the feedback I received from students and colleagues in general. I thought, “Maybe if people see me this way, it means I can do it.” That gave me a bit more energy to take on this role. As I said, it’s a big responsibility, but it’s nice. I see mainly the positive aspects of having an impact on the new generation of scientists.

Throughout your career, do you feel like you have faced any specific difficulties because you are a woman in science and are working in very competitive environments?



Silvia Sposini: Up until the end of my PhD, I would have said no, not at all, just because I was in an environment where women were the majority and I could easily picture myself as one of them. Then I moved here and realized that neuroscience is quite a male-dominated world. This, combined with the fact that I was new to neuroscience, made it tricky to see myself in the position of a teacher or researcher in this field.

However, again, it was thanks to the people surrounding me who pushed me towards positions in neuroscience. I thought, “Okay, if they think they can see me there, it means I can do it.” So, I decided to go for it. To be fair, I think these barriers can be overcome. If you want to do something, you can do it. I don’t want to say that these barriers don’t exist, because it’s true that not seeing women among your peers is a big factor, but they can be overcome. At the end, what counts in research and science in general is the content of your character and your mind, and not really whether you’re a man or a woman. I do recognize that it’s really important to provide examples to the new generation and to have a much more balanced environment in terms of men and women. I think this will make everyone’s life easier, especially for the new generation.

As you also have children, do you feel that affects you specifically in a working environment like academia? Does this affect the possibilities or access to opportunities that you have?



Silvia Sposini: The examples I’ve had in life were fundamental to seeing myself as a scientist and a mother. All my supervisors and mentors have a family and showed me that the two things are possible. So when I felt it was my moment to build a family, I went for it without too much thinking. I thought, “If those people managed, I’m sure it is feasible.”

And again, it is feasible. It poses some challenges. But for me, what I’ve seen is that having a family helps my productivity in a way. I have my working hours. I do my 200% in that time. Then I come back home and I’m not a scientist anymore. I’m just a mother to my children. Because building a family was something I really wanted and makes me incredibly happy and fulfilled, maybe I can also do better at work. I think it’s a mutual relationship. I’m also really happy to give an example to my daughters: a woman, when she has children, doesn’t have to stop working. But you need a network of people around you—people who understand you and who help you when needed.

What I can say is that, in these years, I received all the help I could have needed. Just to make it more personal, I had some key experiments that I really wanted to finish before the birth of my second daughter. However, we can’t plan everything in life, and I was hospitalized to give birth to my daughter the day before these huge experiments. So my network came to help. David Perrais and Julie Angibaud in my team took over and ran the experiment successfully. They gave me all the peace of mind to focus on more important things at that moment.

One of the best compliments I’ve ever had, actually, was this last year, which has been difficult because I was trying to manage everything—family, work, applying for a position, with the pressure of not getting one. A PhD student came to me after she knew I got the permanent position and said: “Thank you so much, because you’ve been an example for women, showing us that it is possible to get a permanent position and it is possible to do it while being a mother.”

However, I think you still need to have something clear in mind, which has been hard for me to accept—and is still hard to accept. You need to give up on something. You need to accept that you can’t do everything. The example of not being the one performing my dream experiment has been hard, but I had to let go. Life teaches you a little bit.

I think that’s always the case. Even with this interview, if we were interviewing a man, we almost never would ask him if he can manage with his kids.



Silvia Sposini: Absolutely. I think we should ask men those questions too. And guess what? I think they will be surprised! If they say that parenthood has not affected them at all, maybe they should go home and think about it!

I once heard a researcher say: “I achieved a lot in my life, but I also had three children, and I was not the primary caregiver. When the school called because a child was sick, it was usually my wife who went to pick them up.” I appreciated the honesty of that reflection, and I think recognizing such contributions is important.

As you mentioned the importance of a network and mentorships, are there certain people that have shaped or influenced you as a person and your career?



Silvia Sposini: Absolutely. At each step of your life and career, I think you can identify someone who has a role as a mentor for you. For me it was every supervisor I had. They are still people with limitations, as everyone has—no one is perfect. But from everyone, I learned something.

Now that I’ve built, little by little, and want to continue building, this network of people around me, I know who I can go to for advice on science experiments, or about life, or when I have an opportunity and wonder, “Should I take it? Is it not a good moment?” It can be your supervisor, but it can also be a colleague. Sometimes, even students have had this role for me, teaching me a lot.

Regardless of who this person is, it’s really important to have a network. You can’t achieve something on your own. I really stress this, especially for young people: reach out to people you want to get advice from. Ask them for help. Ask them to review your cover letters, or just get advice. You can’t expect busy people to come to you and give you advice, but if you go and ask, 90% of people are really happy to help. So don’t be shy. Just go and ask.

Regarding what you said about going to someone and asking for advice, is there any piece of advice that you would give, especially to other women pursuing a career in academia?



Silvia Sposini: I think many of the points I’ve mentioned so far could be considered helpful advice. Really build your network and don’t be scared to face changes in life. That can be the birth of a child or moving country. If you’re motivated enough, you should follow what you really feel.

Many years ago, when I was between my PhD and postdoc, a lot of people were going to the US because you only have a Nature paper if you go to the US. I felt this pressure, and I was about to compromise a lot on my personal life to go there. At one point, I decided to stop and tell the PI I had contacted: “Sorry, I know this was going well, but I just realized I don’t want to do it.” At the end, they completely understood.

So, I think good advice is also not to feel the pressure of what has been done before, or what we think should be done. Just follow what you’re comfortable with. Going a little bit out of your comfort zone is okay, but don’t do things that others are doing just because you think that’s how things should be done.

NeuroPaths: Exploring careers beyond academia

Franck Aby, CEO of Apateya

Aude Verboven¹

¹PhD student at the IMN, Bordeaux Neurocampus

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 NeuroPaths, we speak with **Franck Aby**, who is the CEO of the start-up Apateya, focusing on developing a new treatment against neuropathic pain. He moved from Ivory Coast to France for his studies where he pursued a bachelors in biochemistry. He then took a break from sciences for a few years before starting a Master online : the Euromediterranean Master in Neuroscience (EMN). He then completed a PhD at the IINS in Bordeaux, obtained in 2019 and focused on identifying the causes of neuropathic pain. Following his PhD, he continued with a post-doctoral position at the IMN with a maturation project aiming to transform his thesis project into a concrete proof of concept with the aim to develop a new treatment for neuropathic pain. That is how he ended up creating the startup Apateya in 2024.

Are you interested in knowing more about the CEO of a scientific start-up job as a career path? Then this section is for you!

Chairman and CEO of a scientific start-up

Franck Aby

*I aim to demonstrate the value of
fundamental research in meaningful
applications*



What is your role about?

Apatheya's goal is to bring a new treatment to the market for neuropathic pain, without opioids and with fewer side effects. We are currently at the preclinical stage, working toward clinical development. As the CEO of a startup company, I am responsible for developing the company's overall strategy. This includes establishing the roadmap, defining the key milestones and timeline for the entire project, and identifying the right partners. These partners may include financial partners, as well as advisors and various public or private institutions that can help us move the project forward. They may provide us with facilities and equipment, for example, or bring specific expertise in areas that are critical to the project. My role also involves managing the team, both from a professional standpoint and from a more human perspective, ensuring commitment, motivation, and cohesion within the group. Most of all, my role is to believe in what we are doing.

What made you choose this professional path?

After completing my PhD, I wanted to turn my research into something that could have a real impact on people's lives. And I wanted to do it myself, to show that it was possible. For me, this is also a way to demonstrate the value of fundamental research, with the ultimate goal of helping people.

What's a matching profile?

An ideal candidate would technically be someone with experience who has done this before. But for a more junior profile, I would look for someone who is well supported by experienced people. This person needs to be open-minded, eager to learn, organized, and with a great deal of humility.

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

The main advice I would give people would be to believe in themselves, be a good listener, not be afraid to learn, and surround themselves with good people. And above all, take the leap. It's a big step to take because of the uncertainty, but uncertainty will always be there. It shouldn't stop you from moving forward.

Main responsibilities

- Overall strategy
- Identifying and dealing with the partners
- Establish the roadmap
- Managing all employees
- Believe in the project

Requirements

This role calls for strong discipline and organization skills alongside an ability to know what we want and not being afraid of adversity. At times, a bit of luck can also be useful.

Working conditions

- **Work environment:** Really challenging (especially in France), but extremely interesting in terms of learning and encounters.
- **Pressure level:** Very high
- **Work-life balance:** Complicated, there is always a lot of work, but it's easier when we really like what we do. We can still have time off and completely disconnect from time to time.
- **Salary:** I pay myself a salary that can be very variable from one month to another. At first, it is very low, because we give all we can to the company. With time it increases, and depending on how it goes, it can get pretty high.

Do you have further questions?

Contact Franck at franck.abby@Apatheya.com

More, more, more

Interview with Katia Befort

Juan Garcia-Ruiz¹

¹Glia-neuron interactions team, Neurocentre Magendie, INSERM

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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Perhaps the only difference between me and other people is that I've always demanded more from the sunset.

Nymphomaniac, volumen I, Lars Von Trier

Addiction is a persistent dependence on a substance or activity that comes with serious consequences for health. Nothing you didn't already know. But how does the picture look in Europe?

Some numbers

Addiction represents a significant burden on our society today. According to the latest report from the *European Monitoring Centre for Drugs and Drug Addiction* (2020): amphetamines had a total of 2 million users aged 15 to 64 last year; 2,7 million for MDMA; 4,3 million people used cocaine; and 25,2 million used cannabis. 34% of treatment requests were related to heroin and other opioids. 82% of fatal overdoses were due to opioid use. What's more, according to the WHO, the European Union is the region with the highest alcohol consumption in the world: one in five citizens aged 15 or older admits to drinking five or more drinks (60 grams of alcohol) in a single sitting at least once a week.

As for tobacco, things aren't much better. According to the European Commission (2019), tobacco use is the most common cause of premature death in the European Union, claiming 700 000 lives a year. The data shows that 26% of the general population and 29% of young people aged 15-24 smoke. But I won't bore you with more numbers. Katia Befort, a doctor in molecular biology and pharmacology, has much more to tell us about addiction.

Juan García Ruiz: Could you introduce yourself briefly?

Katia Befort: My name is Katia Befort. I studied molecular biology at the University of Strasbourg. I then did a thesis in molecular biology and pharmacology, and when I finished, I did a postdoc in Boston. When I came back to France, I went through the CNRS recruiting process and got a research position. In 2001, I started my research in Strasbourg at the Institute of Genetics and Molecular and Cellular Biology, where I worked for thirteen years as part of a large team. In 2014, I moved to the Laboratory of Cognitive and Adaptive Neurosciences. On top of that, I spent a year in Canada specializing in neurotoxicology, in a small city called Kingston.

JGR: What is addiction from a molecular point of view?

KB: What we currently understand best is the brain's reward circuit, which gets activated by drug use and is involved in addiction. With repeated drug use, neurons and neural networks end up adapting, and this eventually generates addiction.

From a molecular standpoint, addictive behaviors are characterized by the expression of different genes in the neurons of the reward circuit. How does this happen? Repeated drug use causes certain epigenetic modifications that change the structure of chromatin, making it easier for certain transcription factors to access it, which ultimately results in the expression of some genes. One of the things we want to understand is whether these transcription factors are the same regardless of which drug is being used. For the past several years, I've been studying excessive food consumption, focusing on sugar and fat. Our starting hypothesis is that overeating triggers the same epigenetic and gene-expression changes as drugs do.

JGR: How do you study addiction in the lab?

KB: In our lab, we study addiction using animal models like rats and mice. For example, we look at addiction to cocaine or to painkillers like morphine and its derivatives, as well as cannabinoids. Currently, we don't use genetically modified animals, we simply give the drug to the animal and study the changes at a molecular level. In the past, though, I did work with mice that lacked certain morphine receptors.

JGR: Does addiction exist in animals outside the lab?

KB: Yes. Some studies have shown that monkeys living in certain tropical areas seek out fermented fruit and drink for its alcoholic juice. Similar behaviors have also been observed in elephants.

JGR: How can a behavior that's a priori beneficial, like eating, turn into something harmful, like a sugar addiction?

KB: How do we become addicted? What we're chasing with drugs is that initial rush of euphoria. Humans are drawn to that sensation and tend to repeat the behaviors that produce it, like using certain substances. Repeated use contributes to the onset of addiction. This is accompanied by a shift in the person's emotional state, so that taking the drug generates less and less pleasure over time, to the point of eventually causing discomfort instead. The person then falls into a loop where they consume more and more just to escape the discomfort caused by the absence, or reduction, of that initial euphoria. This can happen with behaviors that are, in principle, beneficial. All it takes is for the reward circuit and the person's emotional state to be altered by repeated exposure.

JGR: Opioids seem to have interested you quite a bit: enough that you wrote a thesis on the subject. What have you learned from that work?

KB: When I started, relatively little was known about this topic. Some things were known about opioids like morphine, such as the existence of their receptors, but they hadn't been well characterized. We were one of the first labs to manage to clone these receptors in order to understand them. This allowed us to identify the delta receptor, which was a major breakthrough, since at the molecular level we had no idea how it worked, and it opened up several new lines of research. For instance, 20 years before these discoveries, pharmaceutical companies were already trying to understand how morphine worked. The fact that we characterized these opioids and their receptors was a major contribution to understanding their mechanisms of action.

JGR: From what I've seen in your publications, opioid receptors seem to play a role in sociability, in the consumption of palatable food, and in emotional states. How are these three behaviors related?

KB: The brain regions involved in the behaviors you mention belong to the reward circuit, so it makes sense that they're related to one another. As for opioids, we've identified three receptors: mu, delta, and kappa. Each receptor is involved in a different aspect. For example, mu receptors are linked to the euphoric and analgesic effects of a drug, while delta receptors are more closely tied to the emotional component of drug use. Whether a given brain region is more associated with one behavior or another depends on the expression level of each receptor there.

JGR: Part of your research has focused on compulsive eating in industrialized countries. For instance, you've studied sweeteners. Have you drawn any conclusions comparing sugar and sweeteners?

KB: The mechanisms of action of sweeteners aren't fully understood yet. We studied a sugar-consumption model, and originally, we used saccharin as a control. We wanted something with a sweet taste but no caloric input. What we found, though, was that animals consuming saccharin showed very different behaviors from those consuming sugar. That opened up a whole series of questions about sweeteners. This is especially interesting given that humans tend to favor sweeteners to avoid gaining weight, which makes understanding the underlying molecular mechanisms all the more necessary.

JGR: Fat has a better energy-to-quantity ratio than sugar. Given that, why do we become addicted to sugar more easily than to fat?

KB: I don't think I'd considered that before. As far as I know, the most addictive combination is sugar and fat together. It's true that fat addiction on its own isn't as much of a problem. I don't think it's really a biological issue so much as a societal one. In the world we live in, sugar carries a lot of weight. You see that reflected in advertising, not to mention how common it is in the food industry to add sugar to all kinds of products. That makes it much easier to develop a sugar addiction than a fat addiction.

JGR: Do you think addiction is a recent phenomenon? Today we talk about addiction to video games, food, tobacco, alcohol and others. Did other forms of addiction exist in the past, or is this something tied to our current lifestyle?

KB: I think that in the distant past, humans spent their time simply surviving. We've now entered an era where everything is available to us, and this shift in our society makes us more inclined to try everything in front of us. Of course, this kind of access varies a lot depending on a country's level of development. But even the poorest, least industrialized countries have easy access to junk food, for example. So in the end, sources of addiction are within everyone's reach. I think addiction is tied to a society that exposes us so readily to things. It's a matter of access, of possibility. It's the same with drugs: the more accessible ones, like alcohol and tobacco, have a much larger share of the population addicted to them.

JGR: Tobacco use has been declining since the early 2000s. How do you see the future of our society when it comes to addiction? Are you optimistic? Are we learning?

KB: I don't think addiction will ever disappear completely. Humans will always have a strong curiosity and a tendency toward risk-taking. That said, I do think the object of addiction will keep shifting. Research will allow us to better understand the mechanisms of addiction, and with that, we'll be able to improve prevention and help people with addiction more effectively. Right now, for instance, there are no real treatments to prevent relapses. Research is essential in the fight against addiction, but I don't think it will ever be eradicated entirely.

JGR: Any message you'd like to share with readers?

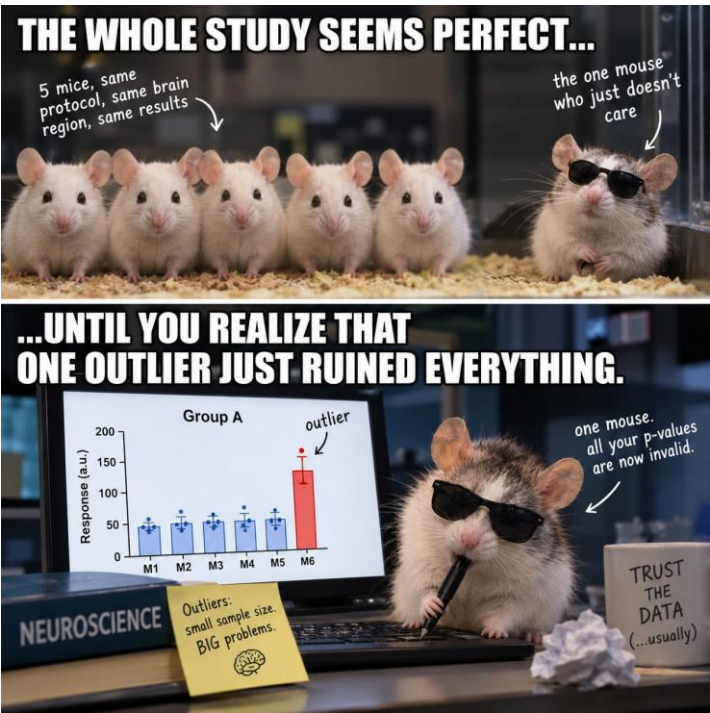
KB: Keep asking questions, stay curious. Be open to new ways of thinking, work abroad if you can, embrace culture shock, change your surroundings. It's not always easy to motivate young people when they're unsure whether they want to go into research, because even we researchers sometimes have doubts and go through difficult moments. But I think that's true in every field. So if research interests you even a little, give it a shot. You have to dare to try!

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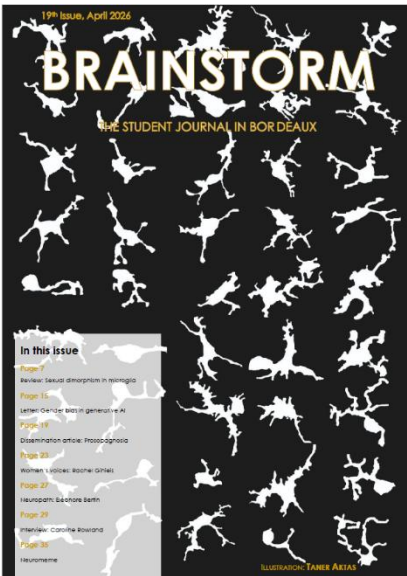
Neuromeme



By Ludovica Congiu in collaboration with ChatGPT

Cover contest winner

The winner of the cover contest is **Taner Aktas**, congratulations to him! (19th issue)



Editorial board



Juan Garcia-Ruiz

With two Bachelor's degree, in Psychology and Biochemistry, and the NeuroBIM Master's degree from the University of Bordeaux, Juan finished his PhD focused on the role of glucose and lactate in sustaining neuronal communication. 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 was a PhD student at the IINS where he was studying how the Fragile X Syndrome impacts the presynaptic mechanisms at the DG-CA3 synapses.



Toshiko Sekijima

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!

Aude Verboven

Aude, directly coming from Bordeaux, is a PhD student at the IMN. She previously graduated from the MultiPublic track of Bordeaux Neurosciences Master. She is currently studying the dopaminergic afferences to pain modulating nuclei in the context of Parkinson's disease.





Chiara Galizia

Chiara is a PhD student at the Interdisciplinary Institute for Neuroscience. She has a Bachelor's degree in Psychology from the Radboud University and a Master's in Neuroscience and Cognition from Utrecht University. Currently, she is studying AMPA receptor dynamics in synaptic plasticity.

Poorvi Ravindra

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.



Ludovica Congiu

Ludovica, originally from Sardinia, Italy, trained in Neuropsychobiology at the University of Cagliari and obtained her Ph.D. in Neuroscience at the Universitätsklinikum Hamburg-Eppendorf (UKE) in Hamburg. She is currently a Postdoctoral Researcher at INCIA, where her research investigates Congenital Central Hypoventilation Syndrome (CCHS), with a particular focus on defining the role of microglia in disease pathophysiology.

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