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Effects of maternal immune activation in a mouse model of neurodevelopmental disorders

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Abstract

Autism Spectrum Disorder (ASD) is a complex neurodevelopmental condition with uncertain etiology, which is suspected to arise from the interaction during critical periods of brain development of genetic predisposition and environmental influences, that alone are not always enough to cause the development of the disorder. Among the environmental factors, Maternal Immune Activation (MIA) has emerged as a significant contributor to ASD risk, acting not as an isolated trigger but as part of a "multiple-hit" mechanism that amplifies susceptibility in genetically vulnerable individuals. Evidence from animal models demonstrates that exposure to MIA synergizes with mutations in several ASD-associated genes, producing behavioral and neurobiological changes greater than those induced by either factor alone. Collectively, these findings support a model where MIA interacts with genetic mutations to intensify neurodevelopmental alterations.

Keywords

ASD, MIA, multiple-hit model, ASD genetic animal model

Abbreviations

Autism spectrum disorder – ASD
Neurodevelopmental disorder – NDD
Maternal immune activation – MIA
Pre-pulse inhibition – PPI
Disrupted-in-schizophrenia 1 – DISC1
Gestational day – GD
Coronavirus disease 2019 – COVID-19
Dominant negative – DN
Nicotinic acetylcholine receptor alpha 7 subunit – α7nAChR
Nuclear receptor-related 1 protein – Nurr1
Tuberous sclerosis complex 2 – TSC2
Cholinergic receptor nicotinic alpha 4 subunit – ChRnα
Polyinosinic-polycytidylic acid – Poly:IC
Contactin-associated protein-like 2 – Cntnap2
Mammalian target of rapamycin – mTOR

Introduction

Autism Spectrum Disorder (ASD) is a neurological condition that affects 0.76% of individuals worldwide, characterized by deficits in communication, social interaction, and repetitive behaviours, with highly significant symptom variability and difficulty in diagnosis (1).

For years, the prevailing view was that environmental factors were the primary determinants for the development of this disorder. However, later on, research on ASD shifted its focus to genetics, indicating also a strong genetic contribution to its etiopathology. It is from this awareness that today we can affirm that ASD is a neurobiological disorder influenced by both genetic and environmental factors affecting the developing brain (2).

Risk genes for ASD are predominantly active during the mid-fetal period, between the 10th and 24th week post-conception, when they impact neuronal circuits during critical stages of brain development. These findings reinforce the hypothesis that ASD has an early genetic origin, highlighting the importance of investigating the molecular and cellular processes that occur during this period (1).

As mentioned, also environmental factors can act as important pathogenic regulators in the etiology of ASD. Children with ASD often show oxidative stress and methylation damage, which may be related to environmental pollution, exposure to chemical or toxic substances, and viral infection. In addition, when the pregnant mother is affected by certain antibodies and neurotoxins, her offspring can exhibit ASD-like symptoms (3).

This example is related to the recent concept of Maternal Immune Activation (MIA) during pregnancy as a risk factor in ASD development (4). In this scenario, the precise nature of the infection cause is not relevant (e.g., viral versus bacterial origins), since it is the actual response of the mother's immune system the true cause or the alterations in the neurodevelopment of the foetus (5).

Despite the relevant role of MIA in the development of Neurodevelopmental disorders (NDDs), data show that not all children of mothers who are infected during pregnancy develop ASD, suggesting that the co-presence of other determinants, such as genetic predisposition, might be responsible for the development of these NDDs. Indeed, it is currently thought that multiple "hits" (exposure to more than one risk factor) may be required for disease induction (6).

In this scenario, MIA may be one of two (or more) hits that together with others result in ASD (7). Based on this hypothesis, MIA appears to act as a "disease primer" able to make an individual more susceptible to the effects of genetic mutations and environmental exposures in triggering disease-related symptoms later in life (8). Examples of hits of genetic nature are mutations in ASD risk genes

(9) like *DISC1*, correlated to neurodevelopmental processes like neuronal migration and outgrowth (10), *TSC*2, which dysregulation is present in a subset of ASD cases (11), *Nurr*1, critical for the dopaminergic neuronal development (12), *α7nAChR,* that in the fetal brain suppresses inflammatory response, and *Cntnap2,* important in neuronal development and synaptic function (23). On the other hand, the environmental hits can be gestational diabetes mellitus (13), gut microbiota (14), or, as mentioned, MIA (7).

In this manner, the double or multiple-hit models represents a more global approach to understand disorders with multiple risk factors (8). Indeed, several genetic models have been made available to study ASD, and between them low dose Poly:IC, a synthetic double stranded RNA used to elicit MIA responses, has been proven to be the most representative in synergizing with mutations in ASD-associated genes, causing greater effects than either insult alone (8)

Methods

The compilation of articles cited in this review has been found through PubMed, using the following keywords: MIA, Autistic spectrum disorder, double hit model ASD, ASD genetic models, ASD mouse model, ASD double hit model.

Results and discussion

Various studies have investigated the interaction between a specific genetic mutation and the exposure to MIA induced by Poly:IC during gestation.

DISC1 mutation – PolyI:C interaction

Abazyan and colleagues (9) investigated how the interaction between DN-*DISC1* and the administration of PolyI:C to pregnant dams at gestational day 9 which resulted in social deficits, high levels of anxiety- and depressive-like behaviors in the offspring (17).

Another study (18), proved the synergistic interaction between PolyI:C MIA and *DISC1* point mutations exacerbate behavioural phenotypes such as sensory- and anxiety-like behaviours. This double-hit effect was dependent on the type of point mutation, showing how the *DISC1-L100P* $^{-/-}$ mutation produces schizophrenia-related behavioural, cellular, and biochemical phenotypes, while the *DISC1-Q3* $^{-/-}$ mutation results in a phenotype related to depression. In addition to that, they demonstrated that PolyI:C had a greater effect on maternal immune response and abnormal behaviour in *DISC1-L100P*$^{+/-}$ mutants compared with WT or *DISC1-Q31L*$^{+/-}$ mice (18).

TSC2 haploinsufficiency – Poly:IC interaction

Heterozygous mutations in *TSC*2 gene and the consequent disinhibition of the mTOR signalling, crucial for cell growth and metabolism, are emerging as well as common themes in the biology of ASD (11).

Through a model of heterozygous *TSC*2 mutations exposed to PolyI:C maternal immune activation, Ehninger and collegaues (11) were able to demonstrate significant interactive effects on gestational survival and postnatal behavioural traits in mice.

Moreover, grown-up mice exposed to both gestational PolyI:C and *TSC*2 haploinsufficiency showed deficient social exploratory behavior, while this phenotype was not observed following exposure to one of these factors alone (19).

According to these authors, the *TSC2*$^{+/-}$ mutation could interact with MIA and induce neurological damage in different ways. MIA is thought to perturb fetal brain development, at least in part due to effects of cytokines in the developing nervous system. Importantly, *TSC*/mTOR signaling is downstream of multiple factors implicated in gestational immune activation, including various cytokines and growth factors. Accordingly, it is possible that disinhibited *TSC*/mTOR signaling downstream of mediators of gestational immune activation effects (i.e., cytokines, growth factors)

amplifies their impact on the *TSC2*$^{+/-}$ fetal brain (20).

Nurr1 mutation – PolyI:C interaction

In another study, *Nurr1* heterozygous knockout mice were injected with PolyI:C at GD 17, and their behavioral phenotypes were examined in adulthood. PolyI:C-treated *Nurr1* heterozygous knockout mice exhibited hyperactivity in the novel environment, sensorimotor gating pre-pulse inhibition, attention deficits, and impaired of working memory (21).

However, according to other authors, these double-hit effects were only present if Poly I:C injections occurred during early (GD9.5), but not late (GD17.5) gestation (5,21).

α7nAChR mutation - Poly:IC interaction

Another study by Wu et al. (22) investigated the role of both the maternal and fetal mutations in α7nAChR in the development of MIA-induced behavioral abnormalities.

Since α7nAChR in the fetal brain suppresses inflammatory response by inhibiting IL-6 production, consequentially, the loss of α7nAChR in the offspring increases their vulnerability to MIA-induced autistic and schizophrenia-like symptoms.

Maternal choline supplementation triggered an anti-inflammatory response in the fetal brain and thus decreased the MIA-induced IL-6 elevation during embryonic stage and the autistic- and schizophrenia-like behaviors at adulthood.

These evidences raise the possibility that the abnormal behaviors in MIA offspring produced by elevated IL-6 are modulated by activation of α7nAChRs in the fetal brain (22).

Cntnap2 deficiency - Poly:IC interaction

The two hit model combining Poly:IC and genetic mutations was also studied in relation to another gene involved in ASD: *Cntnap2*. The use of PolyI:C MIA in *Cntnap2*$^{+/-}$ and *Cntnap2*$^{-/-}$ animal models, in this case rats, demonstrated that the genetic deficiency increased the susceptibility to the environmental insult. Rats carrying the Cntnap2 mutation were indeed more sensitive to the detrimental effects of MIA on sensorimotor gating, i.e., increased startle and reduced PPI (23).

Conclusions

ASD is a neurodevelopmental disorder that affects 0.76% of individuals worldwide, even though this percentage only accounts for approximately 16% of the global child population, indicating a discrepancy likely due to differences in stigmatization of the pathology and access to healthcare and diagnostic services (24). On top of this, the difficulty in the diagnosis and treatment of this disorder is also related to the fact that ASD is a complex multifactorial disease, with both genetic and nongenetic factors playing a role.

Understanding how these non-genetic factors combine with or modulate genetic susceptibility and affect brain development represents an imminent research area that might potentially transform how ASD pathologies are diagnosed and prevented (25).

Between the non-genetic factors, recent data have been demonstrating the role of MIA exposure and its synergism with genetic risk factors, making the offspring more vulnerable to various types of impairments in adolescence and adulthood. This suggests a relationship between MIA and neurodevelopmental disorders such as ASD.

The risk of MIA and its influence in the development of NDDs has gained a higher interest in the scientific community also due to the increase incidence of new pandemic infections, like the recent COVID-19, that can impair pregnancies and the healthy development of the fetus. As a consequence, this scenario causes the impellent need to study and understand better the influence of MIA in effecting the fetus development in order to find a way to counteract or reduce the detrimental effects of these infections over the new generations. In this prospective, the potential value of genetic models for exploring gene–gene as well as gene–environment and environment–environment

interactions relating to ASD should be seriously considered.

This is why, in recent years, there has been the development of double-hit animal models characterized by specific genetic mutations that could interact with MIA exposure, which effects are later analyzed through behavioral tests.

Despite that, there is still much to learn about MIA's pathophysiological mechanism, like the precise contribution of the maternal, placental and fetal compartments to the production of, and response to, cytokines which are thought to be the main mediators of MIA's effects on the fetal brain.

Nevertheless, MIA and double-hit animal model have proven to be neurodevelopmental models with substantial validity and incredible potential. Once rigor, reproducibility, and transparency are improved, they can become fundamental tools in the study of ASD.

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Opinion: A critical point of view on whether and where to do a PhD

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The academic world is reshaping. The access to higher education is being democratized, and competition for PhD's funding is more demanding than ever before. Now, the requirements to find a professorship are relentless: standing out is compulsory (1). Not only that, but a recent survey from *Nature* has reported that PhD students around the world are in many cases overworking, stressed, unsatisfied with their supervision, or even feeling harassed in some cases (2). These changes raise a burning question: **is it worth pursuing a PhD nowadays?**

After spending several years in university, most of us tend to be indecisive and hesitant about what to do next. As a biologist with a growing interest in cancer research, I wanted to pursue a scientific career in academic research, for which a PhD is a *sine qua non* condition (at least in Spain). Below, I summarize the lessons I have learned along the way, not only from my personal experience, but also from my PhD colleagues in Spain and other countries. This letter attempts to present the considerations we wish to have taken before making some decisions, and to fill a gap on the lack of a "critical view" of what a PhD could give you. Hopefully, this letter will serve others to choose more consciously about whether and where to do a PhD.

First: what should a PhD give you?

A PhD is a journey where you need to take responsibility for your own research project. You should acquire the skills to understand the problem, its background, limitations and open questions, and (at later stages) actively propose and make decisions about how to answer them. Ideally, you should find evidence that could answer a question that was previously unknown. By the end of the journey, you should be an expert in your field of research and be able to work autonomously at a very high level. Not only that, but you should acquire many soft skills (e.g. critical thinking, problem solving, teamwork, oral

and written communication, time and project management) that would apply to research, but also to other professional situations.

Second: do you (really) want to do a PhD?

This may sound like a simple question, but there are layers to it. A PhD is a long and (at times) tedious process. Depending on the country, a PhD can last between three years, up to a decade in very extreme cases. That is a fair amount of procrastination before going out to the "real world", the one without clear milestones set by an academic institution. These are the four lessons I learned along the way:

1. A PhD is very different from any other university degree you may do. There are no exams *per se*, and a research project can go very different ways depending on the project needs, supervision and collaborations, technical resources, skills, and (importantly, but often dismissed) luck. You need to be prepared for the unexpected. In a bachelor's or a master's degree, you study, do your exams, and get a grade based on your performance. "Easy". In a PhD, you may spend months (if not, years) trying to get a single experiment to work, which may not have anything to do with how intelligent or skilled you are. If you want to do a PhD, expect periods of frustration; modifications in your initial project, or even switching to a new one after a couple of years; and unexpected issues that will escape your control. In return, you will learn to be resilient, to foresee unexpected problems, and to become flexible when you encounter future issues (just to name a few skills).

2. Ask yourself what you would like to do in the future. Some career paths may benefit from having (or directly, require you to have) a PhD degree. For example, if you would like to lead your own research team in a research institution then, sure, you will be required to have a PhD. However, if you just want to do research, there are positions like scientist (in the private sector), or research engineer and laboratory technician (in the public sector) that do not require a PhD degree. Some positions do not exist in some countries (e.g. I found out about research engineers when I came to France as a postdoc). If you are open-minded and willing to move to a different country, possibilities expand. Bear this in mind and explore your interests and options. You may not need a PhD title for whatever you want to do.

3. Note that you can acquire many of the soft and hard skills that a PhD will give you somewhere else. Some professors and researchers idealize the benefits of doing a PhD, but the truth is that they will depend on your particular case: your work environment, your personality, the techniques that your project will require, and whether they are established in your team or not, etc. You may find a job in a private company where you could face similar problems and learn to work with the same technology as in a PhD project. Also, consider that PhD positions are worse paid than similar jobs in private companies.

4. If you do not have a contract, do NOT do it for free. Many people refer to PhD fellows as "students" and gaslight them into thinking that doing a PhD is not a job. It is, and it is very demanding in every possible way.

In conclusion, deciding to do a PhD is a long-term commitment that has several advantages and disadvantages that really depend on what is your envisioned career path. Avoid idealizing the journey and be aware of the problems that will be coming along with it, and everything should be fine.

Ok, I do want to do a PhD. Where should I do it?

Let's now imagine that you decide you want to go on and pursue a PhD. There are two components here:

(A) One is simpler: you need to choose a university program to enroll in. This may sound trivial, but let me tell you, some programs (and/or countries) do have very strict requirements for graduation. Importantly, some universities pay little to no attention to how these programs work, and you may have to manage a nontrivial amount of paperwork close to the end of your PhD if you make the wrong choice. This means that you would stress due to (1) your PhD manuscript being finally submitted and (2) simultaneously doing paperwork for the university that may not be needed in a different program. Easy recommendation: ask current and former PhD fellows about their experiences with PhD programs before enrolling in the first program you see.

(B) The second one is much more important: how do you decide **where** to do your PhD. Mind that you can do a PhD in a company or in the private sector (the so called "industrial" PhDs), but I will focus on what I have experienced personally or (indirectly), through my friends and peers. Here are my ranked personal recommendations when choosing a team to do a PhD:

A. Find a place where you will enjoy what you do. You need to like your (1) project, (2) your advisor/supervisor, and (3) the place where you will be for the next 4 years. Importantly, **do NOT rush your decision**. A PhD takes a long time, and you need to feel comfortable about what you are doing and where you are doing it as much as you can.

B. Find a thesis advisor that matches your personality. Some advisors like to be on top of the projects, while others let you be unless you actively seek help. Ask yourself what you prefer, because this is often linked to points C and D.

C. Be aware of the stage the research group is at. Younger groups have a stronger need for publications to keep funding rolling. This means that you will probably have more active supervision, but also higher pressure for obtaining positive results and publishing. You will probably learn better how to deal with early-stage situations because techniques and projects may be in a more naïve state, which may "delay" the obtention of "applied" results. Also, you may have less funding, technical resources, and knowledgeable colleagues, so your way in the PhD may be harsher in that way. You will definitely need to become autonomous if you do a PhD in a small team.

   On the contrary, very mature teams may have better funding and resources and more established projects. Thus, you may not have to worry that much about setting up techniques/equipment or fine-tuning protocols. However, mature teams often have several projects pending publication, and they can allow themselves to delay publishing some results. In other words, competition for publication may high in a different way. In some extreme cases you may not even publish, which can be very demotivating and hinder your career development. On the other hand, you will likely have the opportunity to learn many more different techniques in a larger team, and have the opportunity to interact daily with (more) people that have completely different areas of technical expertise.

D. Following up on that thought, mind the size, work environment, structure and organization of the team. **In general**, small teams are often (but not necessarily) early-stage ones, and they have a higher need (and ease) for keeping a nice structure, organization and work environment. Big teams can go many different ways. They can be very organized and divided into collaborative groups of people that work on different projects, or (in extreme cases) they can be chaotic and

competitive on publishing exactly the same project. My advice is to screen for red flags. Try to avoid this type of places as much as possible:

i. Places where people do not get along. This is a go or no go. Nobody likes an unpleasant work environment.

ii. Teams where there are many more PhD fellows than technicians and postdocs. Sadly, some teams use PhDs as cheap technical labor and do not encourage the acquisition of hard and soft skills that a PhD is meant for. This is not always the case, but can be a red flag. Ideally, look for a team with a balanced structure of postdocs and PhDs.

iii. Related to (ii), teams with micromanaging by the principal investigator (PI). In some places, you may be treated as a technician that only executes orders. There is nothing bad about doing only technical work, but that is not the intention of a PhD. If that is the case, you will likely get burnt out early in your journey and drop off the PhD program.

iv. In the same line, teams where it is assumed that you will work overtime or during the weekends by default. A PhD may punctually demand working outside a regular 9-5 schedule, but this should not be assumed as the norm.

v. Teams where PhD fellows do not get encouraged to supervise master or early PhD fellows (at any point). This would help you acquire team management and leadership skills, which will help you flourish in your professional future.

vi. Teams where PIs are detached from the teams' projects. There, you may never get any feedback on your experimental set up, even if you ask for it. You need guidance before you get to be independent. Make sure that you can have regular meetings throughout the PhD. They will be key for your success.

vii. If you are a foreigner, pay attention to whether there are other foreigners in the teams. You may be (and feel) excluded, and communication may not be fluent with your team at first. This can be anecdotic if your work environment is welcoming, since it will help you integrate faster in the country's culture. Just be mindful of it.

E. Be aware that competitive/famous centers attract people mainly interested in their reputation. Doing a PhD in a very competitive research center has the advantage of counting on advanced research infrastructure that will help boost the progression of your project. However, many people are attracted to these places because the experience will look nice in their CVs afterwards. My advice is: whatever the research center you go to, focus on finding a team that is moved by passion about science, not influence or publications in high-impact journals.

Ok, but how can I know all of that?

You may be thinking that you have no idea about how to know all these things. The answer is simpler than you may think: you need to ask. When you do an interview for a PhD project, remember: you are also evaluating if you do want to do a PhD with that team and/or advisor. Check their publications and team composition beforehand, and interview not only with the PI, but also with a PhD (and/or a couple of members from the team) that have been there for a while. Ask them many questions: What is the average time to complete a PhD in the team? How many publications do people usually have when they leave for their next position? Do people get along with each other, or are there subgroups of people? Who is going to supervise me? How often do you meet with you PhD fellows? …

Judge their answers carefully and interview with (minimum) a couple different teams if possible. Importantly, be straightforward about what interests you, and what does not. If everything matches, you will see it clearly. And if not, you will see it too. This is not an easy task but, hopefully, after all these advices, it will get a little bit easier.

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The hidden battle: mental health and resilience in neurodegenerative diseases

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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.

A few weeks ago, I was sitting comfortably with my mother, enjoying one of those classic mother-daughter movie moments that I missed so much. Our choice fell on “The Father”, a wonderful 2020 movie starring Anthony Hopkins — and one I strongly recommend. Without giving too much away, the film portrays the reality of a man living with dementia.

What struck me most was the way the protagonist’s emotions were depicted — the confusion, the fear, the fleeting moments of clarity. It made me reflect: how does the awareness of our own condition shape our emotional state? Do the resulting anxiety and depression themselves worsen the symptoms? How do people living with neurodegenerative diseases face their everyday lives?

During my master’s studies, I had the chance to attend a meeting between doctors and Parkinson’s disease patients. What stayed with me was the incredible strength with which these individuals confronted their illness — and the pain in their voices as they described their daily routines, always trying to hold their heads high despite everything.

With this article, I would like to explore an often-overlooked aspect of neurodegenerative diseases: the interplay between the symptoms and mental health, and the ways patients cope with it.

1. The overlooked burden of mental health in neurodegenerative diseases

People living with neurological disorders such as dementia or Parkinson’s disease frequently experience psychiatric comorbidities, most notably depression and anxiety.

Research estimates that around 25 to 50% of individuals with dementia suffer from depression [1, 3], and anxiety may affect up to 75% of patients [1]. In Parkinson’s disease, about 17% of patients

experience major depression, and anxiety occurs in 4–40% of cases — a wide range that largely reflects differences in how anxiety is measured [1]. Studies vary considerably in the tools they use, from standardized questionnaires to clinical interviews based on diagnostic criteria, and each method captures different aspects of anxiety. Prevalence rates also shift depending on the examined patient population, such as whether individuals are in early or advanced stages of the disease, or whether they are recruited from community samples or specialized clinics. Furthermore, some studies focus on specific types of anxiety, including generalized anxiety, panic symptoms, or Parkinson's-specific anxiety linked to motor fluctuations. These methodological variations explain why reported anxiety rates span such a broad interval.

Despite their high prevalence, depression and anxiety are still frequently underdiagnosed and undertreated — with only about 20% of Parkinson's patients with depression receiving adequate therapy [1]. Several factors may explain this striking gap. First, the core motor symptoms of Parkinson's disease often dominate both clinical attention and patient conversations, leaving psychiatric symptoms in the background. Many patients also interpret feelings of sadness, anxiety, or apathy as an inevitable consequence of living with a chronic illness, and therefore do not report them. On the clinical side, the overlap between depressive symptoms and Parkinsonian features — such as fatigue, slowed thinking, reduced facial expression, or sleep disturbances — makes diagnosis challenging and contributes to frequent misattribution. These factors combine to create a situation in which emotional symptoms remain invisible, even though they deeply affect patients' daily functioning and overall prognosis [1].

2. Depression and disease progression: a bidirectional relationship

In Parkinson's disease, psychiatric symptoms such as depression, apathy, and psychosis are not merely emotional side effects — they often correlate with disease progression and poorer prognosis [2]. Whether these emotional disturbances directly worsen neurological decline or are manifestations of shared neurobiological mechanisms remains unclear, but they consistently predict faster cognitive and functional deterioration.

Depression and neurodegeneration appear deeply interconnected and researchers have proposed several hypotheses to explain this link:

1. Depression as a risk factor: Chronic depression induces biological stress responses, such as inflammation and altered neurotransmitter function, which may increase vulnerability to neurodegeneration.

2. Depression as an early symptom (prodrome): Late-life depression might represent an early sign of an emerging neurodegenerative process rather than an independent condition.

3. Neurodegeneration as a cause of depression: Damage to brain regions involved in emotional regulation may directly lead to depressive symptoms.

4. Shared underlying mechanisms: Common pathways — vascular changes, neuroinflammation, or genetic predispositions — may explain the frequent co-occurrence of both disorders.

This bidirectional relationship complicates diagnosis and treatment but also provides opportunities for early intervention and prevention [3].

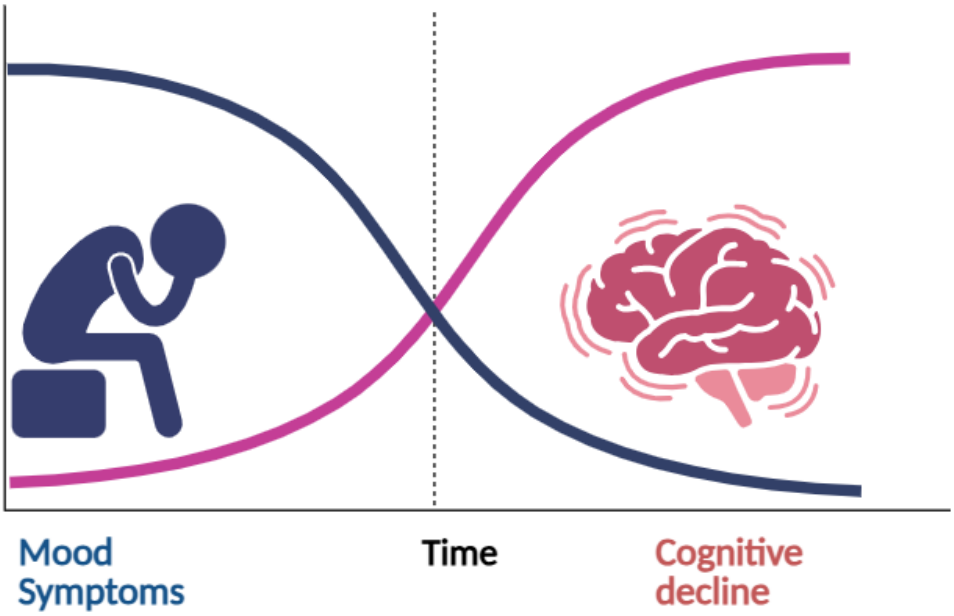

Figure 1. The overlap of the mood symptoms and cognitive decline. (Created in https://BioRender.com)

3. The clinical face of depression in Alzheimer's, Parkinson's, and Huntington's disease

In Alzheimer's disease, depressive symptoms are reported in up to 80% of patients during the disease course [4]. This figure includes both diagnosed and previously unrecognized symptoms identified through research assessments. Despite such high prevalence, depression in Alzheimer's often remains an "invisible burden" because it is overshadowed by cognitive decline. Emotional distress is frequently attributed to memory loss or behavioral changes, leading caregivers and clinicians to overlook it. As a result, even when depression is identified, it may remain insufficiently treated, contributing to faster cognitive decline and greater caregiver strain.

Although less characterized by guilt or suicidal ideation than major depressive disorder, PD-related depression still severely impacts quality of life and disease trajectory [4].

In Huntington's disease (HD), depression compounds severe motor and cognitive deterioration, contributing to extremely high suicide rates — over four times those of the general population [4].

Across all these disorders, early identification and treatment of depressive symptoms are essential, not only to improve emotional well-being but also to mitigate cognitive and functional decline.

4. Diagnostic and therapeutic challenges

In neurological disorders, many depressive symptoms — such as apathy, fatigue, reduced initiative, or changes in sleep and appetite — closely resemble the cognitive and physical changes caused by these conditions. As a result, these emotional symptoms are often mistaken for a natural consequence of the disease rather than a co-occurring disorder that requires specific treatment [3,4].

In clinical settings, the primary focus often falls on managing motor or cognitive decline, leaving psychiatric symptoms in the background. Yet these symptoms are not secondary: people with

neurological diseases face an increased risk of suicidal thoughts and behaviours, particularly in conditions such as Huntington's disease, Parkinson's disease, multiple sclerosis, Alzheimer's disease, and ALS. [4]

Addressing this hidden burden requires earlier recognition and routine monitoring of mood symptoms, ideally through closer collaboration between neurologists, psychiatrists, and caregivers. Integrating mental-health assessment into standard neurological care remains one of the most effective ways to ensure that depression is promptly identified and appropriately treated.

5. Conclusions

A shared genetic basis between Alzheimer's and depression has also been reported, reinforcing the idea of a bidirectional interaction.

Psychosocial factors further exacerbate these vulnerabilities. The progression of neurological illness often leads to social isolation, communication difficulties, and loss of autonomy, all of which heighten depressive symptoms [4].

The COVID-19 pandemic further amplified these challenges: isolation and reduced access to care worsened depression and anxiety among patients with chronic neurological diseases [4].

Depression and anxiety are not peripheral symptoms but central components of neurodegenerative diseases. They influence disease progression, quality of life, and even mortality. Early detection, better-adapted diagnostic tools, and integrated care approaches that address both neurological and psychiatric needs are essential to improve patients' well-being and support them throughout the course of their illness.

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Women's Voices: inspiring the neuroscientist community

Maria-Carmen Medrano

Sara Carracedo[1]
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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 **Maria-Carmen Medrano**, a neuroscientist from Cabreton (La Rioja, Spain) whose career spans several countries and research fields. After completing her Bachelor in Pharmacy and a Master's in Pharmacology, she pursued a PhD on noradrenergic modulation of opioid tolerance and withdrawal. Her postdoctoral journey has taken her through leading laboratories in Spain, France, the UK and Canada, where she has explored neurophysiology, pain and addiction using multidisciplinary approaches. Now based at the Bordeaux Neurocampus, she continues her work on sensorimotor integration and noradrenergic modulation, and has recently been appointed Lecturer in animal and human physiology at the University of Bordeaux. In this interview, she reflects on her scientific path and shares insights into the demands of parenting alongside a scientific career.

Sara Carracedo: Could you tell us about your path in neuroscience?

Maria Carmen: I have a PhD in Pharmacology and I've built an international postdoctoral career in neuroscience, working in top academic labs across Spain, France, the UK, and Canada. My research looks at how addiction and chronic pain affect the brain, especially in relation to attention and cognition. I use a mix of techniques (electrophysiology, pharmacology, and behavioral analysis) to explore the brain circuits involved, from the spinal cord all the way up to regions like the cingulate cortex, the locus coeruleus, and the ventral tegmental area.

Along the way, I've also been teaching and mentoring students at both the undergraduate and Masters level, which has been a big part of my journey. Since November 2023, I've been working with the "Motor control and cognition" (MOCOCO) team at INCIA at the Bordeaux Neurocampus. My current

project examines how noradrenaline modulates the corticospinal circuit, with a focus on sensory-motor integration and its clinical applications.

And most recently, something I'm very excited about, I've been appointed Maîtresse de Conférences (Lecturer) in animal and human physiology, starting this September (2025). It's a big milestone for me and something I've been working toward over the last ten years of research and teaching.

Sara Carracedo: What is your current research about at INCIA?

Maria Carmen: At INCIA, I'm developing a translational and integrative research project focused on the neurophysiology of sensorimotor integration, in both physiological and pathological conditions, with a particular emphasis on the role of noradrenergic modulation. By investigating how pain and movement interact, from the spinal cord to the cortex and including autonomic regulation, I aim to better understand the complex relationships between nociception, motor control, and higher brain functions. Acute pain plays a crucial protective role, guiding motor adjustments to avoid injury. But when pain becomes chronic, it can disrupt motor function, leading to loss of strength, coordination, and mobility. My project seeks to explore these reciprocal interactions in depth and to identify how they're modulated by noradrenergic circuits, which could help guide more integrated therapeutic approaches. To study these mechanisms, I combine electrophysiological recordings in rodents, transcranial magnetic stimulation (TMS) in humans, and pupillometry in both species as a non-invasive readout of noradrenergic neuromodulation.

Sara Carracedo: As a mother, how have you dealt with the demands of parenting alongside a scientific career? Do you feel that family responsibilities have affected your visibility or opportunities in research?

Maria Carmen: I became a mother at the end of my third postdoc project. My contract finished right when my son was born, and at the same time, my partner was doing his PhD. We're both foreigners, and without family nearby to help, we had to face the first months of parenthood on our own. I felt very supported by my team and supervisor, and I made the conscious choice to take a break from research, simply because I wanted to. It was the right decision for me and, as it happened, COVID arrived at the same time, which in some ways reinforced that choice.

Coming back wasn't easy. I've managed to find new postdoc opportunities, to continue publishing, and to keep growing as a researcher. But managing work and parenting, with no external help, while both of us were in precarious academic positions, was incredibly demanding. The uncertainty of the job market becomes especially heavy when you have someone else depending on you. Still, I've been lucky to have a very supportive partner, going through similar struggles as me, and that we deeply understand one another's pressures and needs.

Opportunities sometimes come later, and others are simply gone. But I've learned to stay focused and confident, and to keep going while also embracing family life and everything it brings. It's not easy, but it has taught me resilience, clarity, and renewed motivation.

Sara Carracedo: What structural support (or lack thereof) have you encountered regarding childcare or parental leave in academia? How do you think institutions could evolve to better support early-career scientists with families?

Maria Carmen: Childcare support for early-career researchers is unfortunately very scarce and expensive, especially during the first years when the child's needs are greatest. Considering the modest salaries and precarious conditions of PhD students and postdocs, affordable and flexible childcare options are simply out of reach for many. On top of that, practical challenges like school holidays or days off (for example, Wednesdays without school in France) make it impossible to work a regular full week without interruptions or extra childcare solutions.

While universities and research organizations offer some childcare facilities, they are mostly designed for permanent staff. There is very little specifically tailored to postdocs or PhD students, who arguably need this support the most, given their unstable contracts and uncertain futures. This gap creates a real barrier for early-career scientists trying to balance family and research.

Institutions could evolve by developing more inclusive and flexible childcare programs, accessible regardless of contract status. Subsidies or partnerships with local childcare providers to make services affordable would be a huge help. Specific support for foreign researchers, who often lack family nearby, is also essential. More flexible working hours or remote work options, especially during school holidays, could also ease the pressure.

Ultimately, creating an environment where early-career scientists with families feel supported would not only improve well-being but also promote equality and retention of talented researchers in academia.

Sara Carracedo: What advice would you give to early-career researchers who are considering starting a family?

Maria Carmen: My advice would be: if that's what you want, go for it! Having a family and pursuing a scientific career can definitely be challenging, but it's also deeply rewarding and absolutely possible. The key is to plan ahead as much as you can. From the very beginning of your pregnancy, start thinking about childcare options: look into what's available, talk to other researchers or friends who have gone through it, and don't hesitate to ask for advice and support.

Building a good support network is crucial, whether it's your partner, colleagues, or friends, because balancing family and research is easier when you're not alone. Also, be kind to yourself: there will be times when things feel overwhelming or delayed, and that's normal. It's important to stay flexible and patient and remember that your career path might look different from others, and that's okay.

Finally, keep communicating openly with your supervisors or teams about your needs and challenges. Many people are more understanding than you might expect, and having that dialogue can help create a better environment for you and others who follow.

NeuroPath: Exploring careers beyond academia

Ioannis Bakogiannis, Senior Editor at *Nature Mental Health*

Juan Garcia-Ruiz[1]
[1]Neurocentre Magendie, INSERM

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 **Ioannis Bakogiannis**, Senior Editor at Nature Mental Health, part of the Nature Portfolio. Ioannis brings more than three years of experience in scientific publishing and almost four years working across Nature Portfolio journals. With a background of over a decade in Neuroscience, Mental Health, and the Life Sciences, he describes himself as a goal-driven scientist who thrives in dynamic, collaborative environments. He earned his PhD in Bordeaux, where he investigated how diet-induced obesity affects memory and hippocampal connectivity under the supervision of Guillaume Ferreira at the NutriNeuro Lab (INRAE). Since January 2024, Ioannis has also been co-chair of the SN Pride Network at Springer Nature Group.

Are you interested in knowing more about the clinical research associate job as a career path? Then this section is for you!

Senior Editor at Nature Mental Health

Ioannis Bakogiannis

Advancing the foundations of mental health research

What is your role about?

I am a scientific in-house editor, responsible for the assessment and peer review process within the journal *Nature Mental Health*. My daily tasks include reading papers across a wide range of fields under the general umbrella of mental health, evaluating submissions, identifying reviewers when sending papers out for peer review, and making editorial decisions, which can often be challenging. I also attend conferences and symposia to stay informed about current research and participate in content commissioning for opinion pieces (Comments, News & Views, Perspectives, etc.). Additionally, editors write Q&As in the form of interviews and Research Highlights, which aim to showcase research published elsewhere. Some of these tasks may vary depending on the journal.

What made you choose this professional path?

After completing my PhD, I felt experimentally saturated. I wanted to remain in research while broadening my knowledge of other fields, exploring new techniques and projects, and engaging more in conceptual tasks and scientific writing. During my PhD, I attended a talk by a Nature Neuroscience editor at FENS, where she spoke specifically about her editorial career. She inspired me to follow this path and, in the end, we became colleagues. Therefore, thank you, Leonie!

What's a matching profile?

For an editorial role, a PhD in a relevant field to the journal is required, as well as a passion for science, critical thinking, an eye for detail, the ability to read and evaluate the strengths and weaknesses of papers and great communication skills. In most positions, a post-doctoral position is not required but is welcomed.

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

Read papers (especially in bioRxiv or medRxiv), try to evaluate them from an editorial perspective (would you publish this paper and why?) and keep an eye out for opportunities on Springer Nature's job portal and follow journals on LinkedIn or other social media.

Main responsibilities

- Handling original research papers and working closely with editorial teams on all aspects of the editorial process, including manuscript selection and overseeing peer review.
- Making well-reasoned editorial decisions on submitted manuscripts in the light of expert advice.
- Commissioning and editing content such as Reviews and opinion pieces (varies by journal).
- Be eager to travel and represent the journal in Conferences and Symposia.
- Meet tight deadlines for different content types.
- Work together with other editors and departments, such as Art Editors, Editorial Assistants, and Production.

Requirements

A PhD in a relevant field and the characteristics I mentioned above.

Working conditions

- Work environment: Working with other editors and being tightly linked to researchers and current trends in science.
- Pressure level: High-paced because of meeting strict editorial deadlines.
- Work-life balance: Good work-life balance with the possibility of hybrid working conditions.
- Salary (interval): Varies per location (there is no location in France, but there are offices in Europe, including Berlin, London, Madrid and Milan).

Do you have further questions?

Contact Ioannis at ioannis.bakogiannis@nature.com

Like father, like worm

Interview with Oded Rechavi

Juan Garcia-Ruiz[1]
[1]Neurocentre Magendie, University of Bordeaux

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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Darwin is often considered the father of evolution. In school, we're usually taught that Darwin got it right with his theory of evolution. We're also told that Lamarck was basically wrong, and we associate him with the "crazy" idea of the inheritance of acquired traits. You know, the story about giraffes stretching their necks to reach food on tall trees and then passing their longer necks on to their offspring. So according to what we learn, Darwin is the genius, and Lamarck is the loser. But… are they really?

It turns out both were brilliant. And, actually, Lamarck wasn't the only one proposing the inheritance of acquired traits: it was a shared assumption at the time. He mentioned it in his work, but it was not a distinctive insight of his theory, and he didn't believe in it more strongly than Darwin did. So it makes

no sense to make such a distinction between Darwin and Lamarck because they both accepted the idea. The real difference is that Darwin made crucial contributions with his ideas of natural selection, variation, and adaptation to environmental pressures. But Lamarck also had important insights, like the gradual transformation of species and the influence of the environment on an organism's form.

So why is Lamarck still associated with the inheritance of acquired traits? The reason is historical. Later Darwin's followers, the neo-Darwinists of the 19th and 20th centuries, wanted to separate their new, selection-based theories from the older "soft inheritance" ideas. They needed someone to represent that outdated way of thinking, and Lamarck became the perfect symbol. That's how the label "Lamarckian" came to mean anything related to the inheritance of acquired traits.

But setting Darwin and Lamarck aside for a moment, what if the idea of inherited acquired traits wasn't completely wrong after all? Today, modern biologists, especially those studying epigenetics, are finding that the environment can influence heredity. This is called transgenerational inheritance. I spoke with one of the leading researchers in this field, Professor Oded Rechavi from Tel Aviv University. For the past ten years, his lab in the Department of Neurobiology has been challenging one of biology's core dogmas, showing that acquired traits can indeed be inherited through mechanisms involving small RNA molecules. He received several distinctions, including the Schmidt Science Polymath Award, the Blavatnik Award, the Kadar Award, the Krill Wolf Award, the Alon and F.I.R.S.T (Bikura) Prizes, and the Gross Lipper Fellowship.

Juan García Ruiz: What is your research about?

Oded Rechavi: We study many different things, but our main focus is on transgenerational epigenetic inheritance, which is the kind of inheritance that happens independently of changes in the DNA sequence. Most people know DNA as the material that carries hereditary information, but in some organisms, inheritance can also involve other molecules like RNA. This kind of inheritance follows different rules than DNA-based inheritance and allows for some fascinating phenomena, the most interesting being the inheritance of acquired traits. In other words, changes that happen during an individual's lifetime can somehow be passed on to the next generations.

JGR: Is this related somehow to epigenetics?

OR: People use the word epigenetics in different ways. Personally, I define it as any inheritance that occurs across cell divisions or across generations without changes to the DNA sequence itself. There are several mechanisms that make this possible: chemical modifications to the DNA, changes in chromatin structure (like histone methylation), or inheritance through RNA molecules. Some people even include prions when they talk about epigenetics.

JGR: How does this happen mechanistically in case of RNA epigenetics?

OR: First, an important disclaimer: everything I describe applies to our studies with these simple worms called C.elegans. Time will tell whether this is also true in other organisms. We expect that it might be, but we are not certain yet.

In these worms, people identified a mechanism called RNA interference (RNAi) by which small RNA molecules regulate gene expression by silencing specific genes. This turned out to be conserved in other

organisms like humans. So when I talk about RNA inheritance, I am talking about RNA molecules that can regulate gene expression also in next generations through this same process.

For molecules to be inheritable, they need to have a way to amplify themselves. DNA can replicate because each strand serves as a template to make the complementary one, and that's how genetic information is passed on. In the case of RNA, there are enzymes called RNA-dependent RNA polymerases that use RNA itself as a template to make more RNA. Without this amplification, the RNA signal would fade over time, becoming weaker in each generation. But what we see is that these RNA responses can actually persist across multiple generations.

JGR: Does this mean that the production of these RNA molecules in the ancestors need to occur in the sex cells to be transmitted to the next generations?

OR: The interesting thing, and one of the reasons small RNAs are so fascinating, is that in C. elegans (and some other organisms), these molecules can actually move between cells (Chen & Rechavi, 2021). They can travel from somatic cells to the germ cells that produce the next generation. So small RNAs can be made in any tissue, and they will eventually reach the germline and be passed on. These organisms even have dedicated mechanisms that ensure this transfer happens.

JGR: For how many generations can RNA-based information be transmitted?

OR: When worms are exposed to different stressors, you can observe epigenetically transmitted responses that persist for 3 to 5 generations at the population level. However, at the individual level, some worms can carry this information across hundreds of generations. We have identified genes that regulate how long these inherited responses last; we call them motek genes, an acronym for MOdified Transgenerational Epigenetic Kinetics. In mutants that are defective in motek genes, we observe that at the population level they all inherit RNAs for hundreds of generations. They are genes that encode for proteins that act like a clock, limiting how long the inheritance persists.

The basic idea behind the motek genes is that worms seem to "expect" that their offspring will live in a similar environment, but not indefinitely. Generally, if you keep worms in the same environment for multiple generations, the inherited response will last longer.

JGR: At the individual level, how can worms "decide" what will become an adaptive transgenerational response? How is it ensured that RNA is produced in a way that makes it stable enough to be passed on to the offspring?

OR: There are a few possibilities for when a transgenerational response can actually be adaptive. If the environment experienced by the parents matches that of their offspring, then the inherited response is obviously beneficial. But there are also cases where the parental response turns out to be disadvantageous for the next generation because the environments don't match. So, really, all possibilities are on the table.

To better understand this, we performed some evolution experiments in the lab. We found that when worms are grown at high temperatures, this changes the pool of small RNAs (sRNAs) in their sperm and harms sperm function in their offspring. Because these worms are hermaphrodites (they produce both sperm and eggs), having defective sperm means they can't fertilize themselves. In response, they secrete a hormone that attracts males, leading them to mate with others instead (Toker et al., 2022).

Mating increases genetic variability since it mixes genomes. Once that happens, the resulting genetic change becomes encoded in DNA — a more stable and long-lasting modification. So this is one way that short-term transgenerational RNA responses can lead to more permanent evolutionary changes, even within just a few generations.

JGR: You usually focus on challenging triggers like starvation or antiviral responses. Has this mechanism also been observed in non-challenging traits, which aren't protective per se, but still provide some kind of advantage?

OR: There's a really interesting paper showing that you can actually teach a worm to prefer a particular odor, and that this preference can then be passed on to the next generation. That study came out before small RNA inheritance became a big topic. You could imagine that a similar mechanism might be at play there, but it still needs to be studied in detail.

JGR: What have been your main findings in the field of transgenerational transmission?

OR: In my postdoc I showed that challenging worms with viruses produce transgenerational responses. It was the first time that inherited small RNAs were sequenced, and also the first time that we showed that these RNA-dependent RNA polymerases, the enzymes that amplify small RNAs, play a role in RNA inheritance. It was already known that these enzymes amplified RNA, but not their role in inheritance. Later on, we also showed for the first time that exposing worms to challenging environments could lead to transgenerational changes in their offspring's physiology.

In addition, we discovered genes that control how long these inherited effects last: the motek genes (Houri-Ze'evi et al., 2016). We found that there are specific rules that determine how these responses are passed on across generations and among different worm lineages, explaining why some worms inherit a lot of small RNAs, while others don't.

More recently, we've shown that producing small RNAs just in the brain of a worm is enough to trigger transgenerational changes (Posner et al., 2019). This opens up the possibility that brain activity could influence the next generation, something that hasn't been demonstrated before. When we alter the levels of small RNAs specifically in the brain, it affects not only RNA inheritance but also the offspring's behavior. For example, modifying small RNA production in the parental brain changes how efficiently their descendants can find food.

Finally, I'd mention the evolutionary experiment I talked about earlier: when worms are grown at hitgh temperatures, it alters their behavior in the next generations about whether to mate or not. This has direct evolutionary consequences because it increases genome diversity.

JGR: I have a provocative question. Why is studying worm physiology important for society?

OR: I'm very interested in worms, but not because I'm a zoologist. The thing is, about four out of every five animals on this planet are worms, numerically speaking. So if we understand something fundamental about worms, we're learning something about life in general. So even if all our findings turned out to apply only to worms, I'd be happy. But of course I'd even happier if it also applied to other organisms, including humans. This is something we still don't know.

If this kind of inheritance also happens in humans, the implications would be huge. This would mean that when we talk about heredity, we shouldn't just focus on DNA sequences, but also consider RNA.

Beyond that, our work is driven mostly by curiosity, and we try to challenge different dogmas, things people say "can't happen". For example, it was believed that it was impossible to inherit acquired traits, but our research shows that this is not entirely true. So, as an exercise in creativity and in rethinking the limits of biology, I think this kind of work really matters.

JGR: What are the frontiers of the field of transgenerational inheritance? What are the black boxes?

OR: The biggest questions are: does this kind of inheritance affect evolution? And if so, how? What are the barriers to small RNA inheritance? Does it happen in other organisms, and if it does, are the same mechanisms at play?

JGR: What do you like most about doing research? What did you write in your cover letters to explain how you fell in love with science?

OR: Being a scientist is kind of a loophole. We often joke about the downsides of academia, but honestly, there are so many advantages. We get to follow our curiosity and have direct control over what we do. We can be creative, even if that creativity has to operate within certain constraints. Our environment is constantly changing, and we get to work with smart, curious people all the time. We set our own schedules and enjoy an unparalleled level of freedom in our work. I don't think I'd trade it for any other job. Well, maybe for being an NBA player.

JGR: Do you remember any advice from another scientist that had a big impact on you?

OR: I actually remember bad advice (laughs). A lot of young investigators tend to be very cautious. They don't want to hire too many people or take big risks early on. I think that's a mistake. But the good advice I got was to "just go for it" from the start. Don't wait until you are super established to take chances. You never know what's going to work. Even projects that seem safe might fail. So it's better to give everything from the beginning. That energy and commitment will give you the motivation to keep going.

JGR: Do you think we'll ever change the way we publish scientific research?

OR: Yes, I do. I think the current situation is only temporary. A short phase in the long history of publishing. In fact, I am building a platform for changing the situation which is called q.e.d (qedscience.com) to do something about it. I really believe that this model where you wait for years for peer review, only to get rejected and start all over again, won't last forever. q.e.d is an AI that gives you the feedback you need, and we built it to speed publication and to change the entire landscape of academic publishing.

I think we'll move toward publishing preprints, and journals and editors will act more like curators, selecting the most interesting papers and highlighting them. q.e.d will offer readers another dimension helping them understand and judge what they read critically. Each journal might even develop its own approach. In the future, I imagine scientists will simply upload their papers online in whatever form they like, and the community, owing in part to critical AI services, will decide what deserves attention.

JGR: Do you have a book recommendation?

OR: Yes, a few actually. It's not exactly a science book, and it's a bit controversial, but it's related to transgenerational inheritance. It's an excellent book called The Case of the Midwife Toad by Arthur Koestler, about a rather infamous scientist named Paul Kammerer. Another great one is The Common Thread by John Sulston. It's about the race to sequence the human genome, and it's really fantastic. And of course, there's the classic What Is Life? by Erwin Schrödinger.

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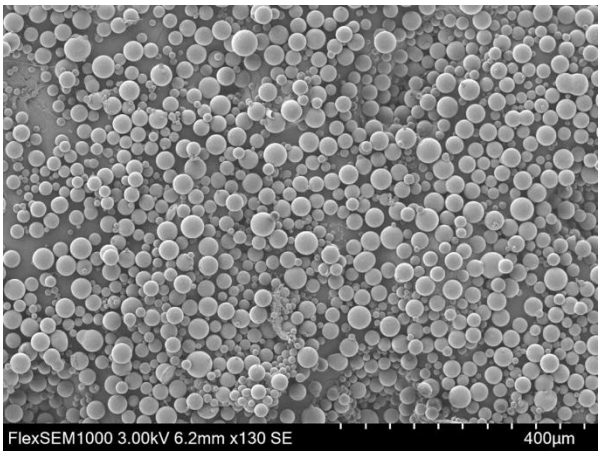
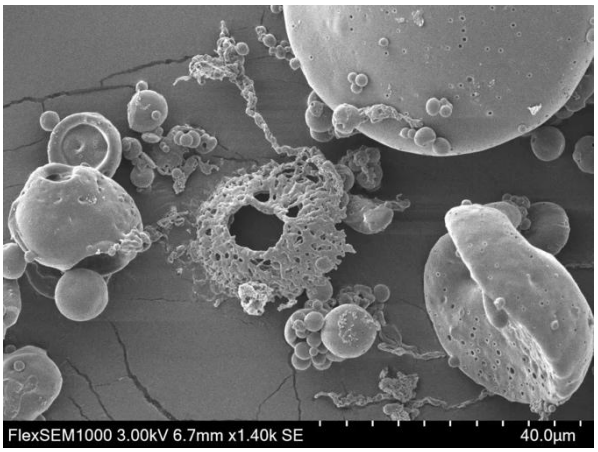
Neurofailure

Expectation vs reality By Aurelia Poerio

This Scanning Electron Microscopy image captures one of the first attempts at producing poly(lactic-co-glycolic) acid microspheres for neurotrophic factor delivery. As the name suggests, they were supposed to be perfectly round… instead, a little monster came out!





Formations

Surgery training

The Graduate Program proposes an English session for the surgery training on rodents (EU directive 2010/63). It will take place from 11/02/2026 to 13/02/2026.

PhD students will have priority in our selection. Due to the high demand we cannot guarantee that all M2 students will be accepted.

For registration, write to neurocampus.graduateprogram@u-bordeaux.fr with the following information before 9th January 2026:

- Research team
- Thesis director / Internship supervisor
- Techniques to be used following this training

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 is pursuing a PhD focused on the role of lactate in basal synaptic transmission. Although he speaks near-perfect French, Juan comes from Huelva, Spain. He is also the co-founder of neuronhub (www.neuronhub.org).

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!

Sara Carracedo

Born in Spain, Sara is a Postdoctoral student at the IMN. She holds a Veterinary Medicine Bachelor's degree from the University of Santiago de Compostela, the NeuroBIM Master's degree, and a PhD in neurosciences from the University of Bordeaux. Her Postdoc at the IMN focused on understanding the neuroimmune role of P2X4 receptor in Amyotrophic lateral sclerosis.

Daniele Stajano

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.

Ludovica Congiu

Hailing from Sardinia (Italy), Ludovica obtained a master's degree in Neuropsychobiology at the University of Cagliari and pursued a Ph.D. in neuroscience at the Universitätsklinikum Hamburg-Eppendorf (UKE) in Hamburg. She is a Postdoctoral researcher at INCIA.

Simon Lecomte

Simon is originally from Lyon, France. He did his Bachelor's of Psychology from Strasbourg, after which he did the NeroBIM master's degree from the University of Bordeaux. He is a PhD student in the IIINS where he is studying how the Fragile X Syndrome impacts the presynaptic mechanisms at the DG-CA3 synapses.

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.

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