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2021

Professor Marco Prinz

The Novo Nordisk Prize

Nomination of Marco Prinz

The Novo Nordisk Foundation is awarding the 2021 Novo Nordisk Prize to Professor Marco Prinz for his pioneering studies providing a massive collective step forward for understanding the function and role of microglia in the central nervous system (CNS), both in normal brain function and in the pathogenesis of inflammatory and neurodegenerative disorders. His groundbreaking results have important perspectives for novel concepts for treating people with CNS disorders.

Marco Prinz is 50 years old and graduated as a medical doctor from Humboldt-University (Charité), Berlin, Germany in 1996. He completed his dissertation in medicine at the same university in 1997, investigating the pathology of cortical interneurons in humans at the Institute of Neuroanatomy. During his postdoctoral studies at the Max Delbrück Centre for Molecular Medicine in Berlin-Buch, he studied the function of glial cells in the CNS, showing special interest in microglia. He then performed his residency in neuropathology at University Hospital Zürich in Switzerland and studied the role of the peripheral and CNS-restricted immune system for the pathogenesis of neurodegenerative disorders such as prion diseases under the supervision of Adriano Aguzzi. During that time, Marco Prinz obtained his first major scientific achievements, contributing to understanding a mechanism of prion transfer from the immune system to the nervous system through the positioning of specialized dendritic cells in the proximity of sympathetic nerves and identifying molecules (soluble dimeric prion proteins) to antagonize prion disease. These discoveries were published in the *Proceedings of the National Academy of Sciences of the United States of America* in 2002 and *Nature* in 2003 along with other prestigious publications.

After returning to Germany in 2003, Marco Prinz established his own research group at the Institute of Neuropathology of the University of Göttingen in Germany, where he began to focus on the developmental origin and the function of brain microglia,

the parenchymal macrophages of the CNS. In 2008, Marco Prinz was recruited to the University of Freiburg in Germany, and he was promoted to Professor and Director of the Institute of Neuropathology at this early career stage, being in charge of the entire neuropathological diagnostics at the Freiburg University Medical Center and the surrounding hospitals. He has consistently focused great skill and talent on the pathology of the CNS, and the major scientific contributions of Marco Prinz comprise his studies on the characterisation of microglia. Thanks to his pioneering discoveries, many published in the highest-ranking journals, it is now known that microglia share a core genetic signature and that they are extremely versatile and can adapt to changing environments. During the past almost two decades, Marco Prinz has been conducting highly original and clinically important research, generating novel and outstanding knowledge on microglia. Importantly, this has contributed to new and more comprehensive understanding of the pathophysiology of neuroinflammation and neurodegenerative disorders.

Microglia are tissue-resident macrophages in the CNS. They belong to a group of mononuclear phagocytes that comprises peripheral tissue macrophages, CNS-associated macrophages, dendritic cells and monocyte-derived cells. Microglia represent the main innate immune cells, and in contrast to other tissue macrophages, they have a unique solely yolk-sac origin. Microglia constitute the first immune CNS barrier against pathogens and environmental insults. As such, they have been shown to be

critical effectors and regulators of changes in CNS homeostasis during development and in health and disease. Marco Prinz's studies are leading in this field, and he has performed landmark studies changing our understanding of the role of microglia and other brain myeloid immune system cells in the normal brain and during CNS inflammatory and neurodegenerative disorders.

From the very beginning as an independent scientist, Marco Prinz focused his research on microglial cells, especially on the origin, function and fate of differentiated brain myeloid subtypes. It was assumed that bone marrow-derived monocytes, a subgroup of leukocytes, circulate in the blood and enter the tissues (where they differentiate into tissue-resident macrophages) in non-pathological conditions and during inflammation. At that time, the factors controlling microglia recruitment from the blood remained elusive, and the direct microglia precursor was unknown. However, this view markedly changed as a result of the breakthrough studies of Marco Prinz and colleagues. In 2007, they discovered new subtypes of mononuclear phagocytes with distinct roles in CNS disorders. Specifically, they discovered that monocytes expressing high levels of the CCR2 chemokine receptor are recruited into the brain during inflammatory conditions and give rise to microglia-like cells, making an important contribution to understanding microglia engraftment in the adult brain. This discovery was followed by several important contributions to understanding the role of CNS myeloid cells during brain autoimmunity. In particular, Marco Prinz identified a previously unknown protective role for the type I interferon receptor in experimental autoimmune encephalomyelitis, the animal model of multiple sclerosis. Subsequently, Marco Prinz identified the molecular mechanisms controlling myeloid cell recruitment into the brain during experimental autoimmune encephalomyelitis and extended his studies to Alzheimer's disease. These studies demonstrated that interfering with trafficking mechanisms of myeloid cells, especially chemokine receptors, may have therapeutic effects in animal models of Alzheimer's disease.

In 2012 and 2013, Marco Prinz and colleagues discovered a series of remarkable key characteristics of microglia and published the results in prestigious journals. First, they discovered a previously unknown role for cytosolic RIG-I-like helicases as negative modulators of CNS autoimmunity in animal models of multiple sclerosis. Next, they identified the microglia precursor in the yolk sac and, by a variety of fate-mapping studies, they contributed substantially to identifying the origin of microglia, showing that microglial cells represent a distinct tissue-specific macrophage population that is clearly genetically different from circulating myeloid cells. Further, they deciphered the pathways that are required for erythromyeloid precursors to develop into mature microglia during development. Then they discovered that transforming growth factor (TGF)- β -activated kinase 1 has a pivotal role in microglia-mediated inflammation during experimental autoimmune encephalomyelitis. These pioneering studies provided a massive collective step forward in knowledge about microglia function in the CNS.

In 2015, Marco Prinz made another fundamental discovery in neuroimmunology by showing that the gut microbiota is essential to regulating microglial maturation and activation during health and disease, suggesting that environmental factors can control microglia and may represent key elements to understanding and managing brain health. Importantly, the study indicated that microglia are particularly affected when a complex microbiota is absent, and their results showed that the absence of a complex host microbiota leads to defects in microglia maturation, differentiation and function, demonstrating a key role for the gut-brain axis in normal brain function. Further, the findings in this milestone contribution may assist in understanding and treating people with microglia-mediated inflammatory diseases, nervous system disorders, mental disorders and cancer of the CNS. Importantly, in a recent follow-up study, Marco Prinz and colleagues showed that host microbiota controlled microglia-mediated uptake of amyloid- β deposition in a mouse model of

Alzheimer's disease, in which RNA-sequencing analysis of FACS-purified hippocampal microglia uncovered distinct microbiota-dependent gene expression patterns, including genes attributed to phagocytosis or complement signalling and genes ascribed to Alzheimer's disease-linked activation of microglia, providing deeper knowledge about the high plasticity of the gut-microglia connection and treatment of people with microglia-mediated CNS disorders.

In his efforts to understand the role of myeloid cells in the brain, Marco Prinz made another landmark contribution to the field by clarifying the origin, fate and dynamics of macrophages at the CNS interfaces. By combining sophisticated techniques, such as large-scale single-cell RNA-sequencing with multiple approaches of fate-mapping, parabiosis and *in vivo* imaging, he challenged the view that non-parenchymal macrophages are bone marrow-derived and demonstrated that the perivascular and subdural meningeal macrophages are derived from embryonic precursors and are ontogenetically related to microglia. Further, they identified critical epigenetic regulators controlling microglia functions, demonstrating that histone deacetylases HDAC1 and HDAC2 are essential for microglial survival and expansion during development, whereas deleting these molecules reduces neuropathology and improves cognitive function in an Alzheimer's disease mouse model.

In 2019, Marco Prinz and colleagues provided another remarkable contribution to understanding how microglia orchestrate local immune responses. By combining two-dimensional technologies, single-cell RNA sequencing and time-of-flight mass cytometry, they identified microglia states in the human brain during homeostasis and disease. By this approach, they discovered a spectrum of transcriptional states in human microglia determined by their spatial distribution that change with ageing and brain tumour pathology. Using a highly translational approach by applying single-cell analysis of CNS tissue on mice, healthy human brains and the brains of people with multiple sclerosis,

Marco Prinz and colleagues discovered subtypes of microglia with distinct molecular hallmarks and diverse cellular kinetics that differed between neurogenerative conditions and toxic demyelination. By establishing transcriptional profiling of heterogeneous populations of microglia in healthy and diseased rodents and humans, Marco Prinz provided insights into the pathogenesis of various CNS disorders.

In addition, and in the context of a large international effort, Marco Prinz and colleagues characterised microglia morphology and transcriptional programmes across 10 species spanning more than 450 million years of evolution, showing that human microglia express a conserved core programme but also significant heterogeneity compared with other mammalian cells of the same type. Along with these milestone studies, Marco Prinz and colleagues also identified the hexosaminidase subunit beta as a stably expressed microglia core gene, providing a valuable new genetic tool to specifically study microglia functions in the CNS. Finally, by integrating gene expression profiling, genetics and comprehensive phenotyping, Marco Prinz and colleagues discovered that pharmaceutical stimulation of sterol synthesis in myelin-phagocytosing microglia and macrophages boosted the repair of acutely demyelinated lesions, suggesting a novel therapeutic strategy for myelin repair in experimental multiple sclerosis.

Marco Prinz's scientific activities over the past 25 years have been outstanding. He has delivered numerous technically and conceptually innovative contributions to neuroimmunology. Marco Prinz has applied new and constantly evolving strong tools integrating genetic, genomic, metabolomic, molecular biological and computational modelling to dissect pathophysiological mechanisms essential for developing CNS disorders.

During the past two decades, Marco Prinz has contributed a steady stream of highly significant discoveries revealing pivotal roles of microglia for the CNS during health and disease. Marco Prinz and colleagues have shown that microglia both respond to and induce

CNS disorders, including multiple sclerosis, Alzheimer's disease and other neurodegenerative disorders, such as amyotrophic lateral sclerosis and Parkinson's disease. He has been the driving force in developing and implementing highly translational international clinical research in CNS disorders. Thus, Marco Prinz's contributions to understanding the significance of microglia are extensive, original and groundbreaking. This impressive scientific accomplishment is highly remarkable and outstanding.

Marco Prinz is an exceptional scientist, and his career is studded with high-quality and high-impact publications and remarkable achievements and recognitions in neuroimmunology, which is truly excellent. Marco Prinz has already been honoured with several national and international prizes and awards, including the Gottfried Wilhelm Leibniz Prize. He has attracted considerable external funding, including extensive European Union research funding. Further, Marco Prinz has shown exceptionally great leadership and mentored many clinical scientists and numerous young researchers and PhD students who are now continuing their own research. Marco Prinz has an impressive scientific output of more than 300 original studies, many published in high-impact journals such as *Science*, *Nature*, *Nature Medicine*, *Nature Neuroscience*, *Nature Immunology*, *Nature Genetics*, *Cell*, *EMBO Journal*, *Journal of Clinical Investigation*, *Journal of Experimental Medicine* and *Proceedings of the National Academy of Sciences of the United States of America*. He has also authored several review articles in his field, and many of his reviews and original articles are frequently cited. Marco Prinz's research has contributed to the international scene in a leading role and with important impact. Together with his colleagues, he has made seminal discoveries regarding CNS macrophage biology. Thus, Marco Prinz stands out as a pre-eminent clinical scientist, and his scientific translational work in neuroimmunology has contributed to important discoveries documented from his many highly original publications. His landmark studies on the endogenous immune system of the CNS during development,

homeostasis and disease revealed the role of microglia in normal brain function and the pathogenesis of inflammatory and neurodegenerative disorders, suggesting novel therapeutic strategies for treating people with CNS disorders.

In summary, the Committee on the Novo Nordisk Prize finds that Marco Prinz is a worthy recipient of the 2021 Novo Nordisk Prize based on his systematic, comprehensive, clinically important and highly original international research on microglia and for its impact on medical science.





**Suddenly everyone
wants to play with
the brain's most
boring cells**

Microglia are the brain's garbage collectors. Studying them for a lifetime probably sounds reasonably boring to most people. However, for Marco Prinz, the world's most boring brain cells were his path into the incredible world of brain research. For almost 25 years, his research has revealed that microglia are much more than garbage collectors. They are the key to the brain's immune system and thus to understanding autoimmune brain disorders such as Alzheimer's and multiple sclerosis. Microglia may be the key to treating people who have these diseases. Additionally, microglia also control many other key functions of the brain. For his pioneering studies, Marco Prinz is receiving the 2021 Novo Nordisk Prize.

The blood-brain barrier is established prenatally to prevent influx of immune cells at a later time. Thus, the brain hosts its own immune system with a key actor called microglia. In addition, the microglia play a central role in both monitoring and defending the brain and maintaining its normal functions. At the beginning of his career, Marco Prinz became fascinated by these cells, which received almost no attention in the mid-1990s and were primarily thought to be the brain's garbage collectors.

"Most people found them boring, because they were thought to simply remove waste during illness. Our research has shown that their function extends far beyond this role and that the development of microglia dates back to a remarkably short period during fetal development. Microglia are key in maintaining the normal function of the brain, and their failure can therefore also lead to several very serious brain disorders," explains Professor Dr. Marco Prinz, Director at the Institute of Neuropathology, Medical Center, University of Freiburg, Germany.

Exotic research

Microglial cells were not at the top of Marco Prinz's wish list when he completed his medical studies at Humboldt-University (Charité) in Berlin in 1996. The brain had always fascinated him, but with the still rather rudimentary conditions in eastern Germany at that time, the opportunities for research

were very limited. However, he got the chance to investigate brain cell staining to study neuronal growth among children with Down's syndrome.

"We used very old-fashioned silver staining techniques, but my studies with neuroanatomist Eveline Schulz made me want to explore the brain more, and this led me to choose research rather than the medical path."

This was just after the Berlin Wall fell, and in an attempt to integrate eastern and western Germany, a decision was made to build new research institutes in eastern Germany. One was the Max Delbrück Center for Molecular Medicine (MDC) in Berlin.

"I asked around a bit: what is your research field? One researcher worked with stroke and another worked with encephalitis. And this guy who works on encephalitis then said he was collaborating with someone else, so I thought, 'Okay, I'll try my luck there and see what he does in the lab'."

Most brain researchers worked with neurons and the signals between them, but biochemist Uwe-Karsten Hanisch did not. He believed that inflammation of the brain is essentially mediated by a special type of cells called microglia.

“At the time, they were thought to be a kind of garbage collector that simply cleaned up after brain infections. Very few people were working with these very special immune cells in the brain. This was a really exotic research field, and yes, by that time, microglia were probably the most boring cells in the brain.”

Trigger release

Despite Marco Prinz's medical background and sparse education in traditional research, Uwe-Karsten Hanisch took Marco Prinz under his wing as a postdoctoral fellow. This turned out to be an excellent choice for both of them, and soon they produced a steady stream of articles on microglia and interleukins, the immune system's tiny signalling molecules. However, brain researchers showed little interest because their focus was elsewhere.

In 1997, Stanley Prusiner from the United States received the Nobel Prize for his explanation of the cause of bovine spongiform encephalopathy (mad cow disease) and the human counterpart Creutzfeldt-Jakob disease. He coined the term prion from the words proteinaceous and infectious to explain how the misfolding of proteins caused the infection in diseases. Adriano Aguzzi from the University Hospital of Zurich, who took Prinz in as his postdoctoral fellow was also one of the world-leading experts on prion diseases during those days.

“The people I met here were the outstanding scientists and teachers: Adriano Aguzzi, a natural and driven neuropathologist; Charles Weissmann, who pioneered molecular genetics, cloned type I interferons and was also one of the founders of Biogen, worked in a nearby laboratory; and then there was Rolf Zinkernagel, who had actually won the Nobel Prize in Physiology or Medicine just a few years before. They were really demanding and brilliant scientists from whom I learned my basic understanding of molecular medicine.”

During this time, Marco Prinz achieved his first major scientific advances, which contributed to understanding a mechanism for transferring prions from the immune system to the nervous system. The results were published in major journals such as *Proceedings of the National Academy of Sciences of the United States of America* in 2002 and *Nature* in 2003 and other leading journals.

“In hindsight, I clearly had to be there to move on to bigger things. It was a really intense time, and learning how to conduct experiments and think about science was important. This was something that I really learned together with these incredibly serious individuals.”

Even though Marco Prinz had to put aside the microglia while he was in Zurich, they continued to turn up. Indeed, growing evidence showed that microglia play a key role not only as a primitive immune cell that cleans up after an infection but also both in connection with brain damage in general and with several other disorders of the central nervous system.

“The accumulation of these microglia actually turned out to be a very common feature of most of the scrapie diseases such as mad cow disease and the equivalent in sheep and goats. So the microglia were closely related to the prions, and it was even suggested that microglia might be the trigger releasing these neurodegenerative prion diseases.”

Much more central role

The microglia may even not be as boring as people thought. After 4 years in Zurich, Marco Prinz returned to Germany and became group leader at the Institute of Neuropathology, University of Göttingen. The future seemed bright, but convincing potential donors and research councils that studying inflammation of the brain is important was difficult.



“

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“They said: he has done some good research, but he has no evidence for what he wants to do now. I got positive feedback, but rejection after rejection. You really have to be in the right place at the right time in research.”

2005 turned out to be the pivotal time. Neuroscience was experiencing a renaissance. With new laser scanning imaging techniques, confocal microscopy and new techniques for studying the brain in mice using green fluorescent proteins, for which Roger Tsien received the Nobel Prize in 2008, opportunities to study brain cells in great detail exploded.

“Now we could suddenly follow the actual development of the cells and thus also see what factors trigger the development of microglia in the embryo, and we increasingly realised that microglia are not simply the first important immune barrier against pathogens and environmental changes. They seemed to play a much more central role in the brain.”

Link to specific diseases

In 2007, Marco Prinz and his colleagues in Göttingen discovered that immune cells, which express high levels of chemokine receptor CCR2, develop when inflammation occurs in the brain's microglia-like cells, such as in the autoimmune disease encephalomyelitis – the animal equivalent of multiple sclerosis.

Almost overnight, microglia suddenly went from being the dullest to the most exciting topic, and I had been working with microglia for more than 10 years, which gave me a huge advantage over many other researchers.

In 2008, Marco Prinz was offered a professorship at the University of Freiburg, where he became director of the Institute of Neuropathology at this very early stage in his career. He was also made responsible for neuropathological

diagnostics at the University's medical centre and the surrounding hospitals.

“This enabled us to better identify the links to specific diseases, including Parkinson's, Alzheimer's, Huntington's and multiple sclerosis, and it soon became clear quite quickly that microglia have many functions in the brain – both during development and maturation and in regenerating brain cells.”

200 genetic links

Convinced of the central role of microglia, Marco Prinz now decided to focus his research on their origin and how they evolved. At the time, it was thought that monocytes (immune cells derived from bone marrow) – the immune system's garbage collectors that clean up and destroy foreign organisms and dead cells – circulate in the bloodstream and penetrate the body's various tissues and that these differ from the tissue's own garbage collectors.

“If true, this fascinating concept could revolutionise treatment: simply manipulating the bone marrow would better combat diseases in various tissues.”

However, Marco Prinz and his colleagues discounted this theory and showed in a groundbreaking study that both the brain tissues' own garbage collectors – the microglia and the cells in perivascular tissues, the sites around blood vessels – originated from similar stem cells in the fetal yolk sac.

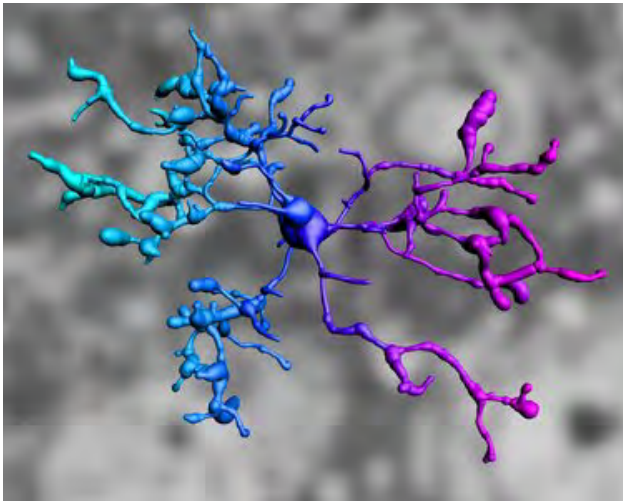
10% of brain cells are plastic immune cells

The brain's immune system is therefore off limits. The microglia and the brain's other immune cells arise in the fetal state, and when a baby is born, the influx of immune cells from the periphery to the brain is shut off, just like to other central organs. “The brain is somehow protected by immune privilege. The development of the immune system differs from that of the skin and the kidneys, for example. You can live without a kidney,

but some organs have to have immune privilege, so evolution has found a way to protect these key organs, such as men's testicles, women's ovaries and then the brain."

According to Marco Prinz, curing many of the numerous brain disorders requires learning more about the brain's own immune system and especially learning to understand the origin and function of microglia and how to manipulate them.

"Most of the billions of cells in the brain are neurons and are not very plastic. However, the 10% that are microglia are very plastic, and in recent years we have found that about half of all genes associated with a higher risk of brain disorders are present in microglia. So perhaps we can cure diseases by manipulating the microglia. However, this is a huge puzzle we are assembling, and although each piece adds something new, there is still a long way to go."



IMARIS-based three-dimensional reconstruction of a microglia cell (from Goldmann, Wieghofer et al. 2013).

Gut linked to the brain

In 2015, Marco Prinz and colleagues found a surprising piece of the puzzle when they revealed that the microorganisms in the human gut play a key role in regulating the maturation and activation of microglia.

This suggests that environmental factors may actually control the microglia and may prove to be key elements in understanding and managing brain health. In particular, microglia appear to be especially affected, since insufficient diversity and complexity of the microbiome can lead to defects in the maturation, differentiation and function of microglia.

The study indicated a key role for the communication between gut and brain in health and in disease. In a later study, in which they linked the composition of the microbiome to activation of special genes on microglia, the researchers showed that dysregulation can ultimately result in disease.

"These genes can be linked directly to the development of Alzheimer's disease. Since then, we have carried out similar studies on Parkinson's disease and multiple sclerosis with similar results, so we first try to understand how the sick person's microbiome has changed and then we identify the factors linking the gut and microglia that make people sick and, ultimately, how to change the microbiome to affect and cure the disorder."

Brain confetti party

In his efforts to understand the diverse role of microglia in the brain, Marco Prinz and his group recently became more and more aware that microglia, in addition to being the brain's local garbage collectors, also help to create plasticity and structure in the brain. By combining single-cell RNA sequencing and mass spectrometry, they created a breakthrough in understanding the normal and diseased brain.

This enabled us to identify and characterise the gene expression in the cells and compare this with, for example, the development of tumours. The gene expression and plasticity of the microglia play a special role in this development. Whether the altered genetic expression is the cause and whether this changes because of the development of the tumour is still too early to say.

Researchers have also turned their attention to the microglia in connection with the scientific black box of nervous system and mental disorders. Today, we know that both autism and schizophrenia alter neuronal networks, and these previously boring cells are suspected once again.

“We have developed a confetti system to colour the microglia in mice red, yellow, green and blue – depending on whether they are expanding by creating daughter cells, contracting or remaining the same. This enables us to monitor the development of the cells in the normal state and during the development of various diseases.”

A real goldmine

Twenty-five years after Marco Prinz launched himself into boring brain cells, microglia are today considered key regulators of the central nervous system – both in health and disease.

“They seem to be involved in every form of brain pathology: in neurodegeneration such as Alzheimer’s disease, amyotrophic lateral sclerosis and Parkinson’s disease; in neuroinflammatory diseases such as meningitis and multiple sclerosis; in nervous system and mental disorders such as autism and schizophrenia; and even in pathological conditions.”

The microglia are thus not simply immune cells that clean up after infections that can damage the brain, including in autoimmune diseases such as Parkinson’s disease in which the

immune system attacks itself. The cells also build and shape the environment in the neuronal networks. As Marco Prinz collects more and more information about microglia, they are increasingly proving to be a real gold mine.

“Many companies are trying to target these cells therapeutically. Today we know that, although Alzheimer’s, Huntington’s and Parkinson’s and multiple sclerosis are multifactorial and complicated diseases with more than 200 genetic mutations and risk factors for each disease, the microglia provide most of the genetic risk factors.”

Although Marco Prinz and colleagues dream of using their knowledge of the cells to understand and possibly cure the diseases, this remains complicated.

“The brain is probably the most complex thing in the universe. Developing new types of therapy can therefore easily turn out to be much more complicated than we expect, so our initial vision is to use the new knowledge to develop better diagnostics, whereas new microglia-based therapies are probably a little further in the future.”

A diagnostic alarm clock

According to Marco Prinz, a diagnostic method based on microglia may become a reality in the not-so-distant future. Currently, many brain disorders are diagnosed using small biopsies.

“When a biopsy is taken, people already have the disease, often already at an advanced stage. Microglia are ultimately the first cell to respond during almost any brain disease. Identification of some molecules, genes or markers that are really typical of microglia-specific disease will enable us to identify a disease much earlier and perhaps intervene with a treatment before they become too advanced.”



Microglia exhibit a cell-specific molecular signature, and thus, Marco Prinz can imagine basing a diagnostic method on deviations from this signature. Out-of-balance microglia may indicate early disease since many brain diseases take years or even decades to develop.

“Once the symptoms appear, it is often too late. One could imagine identifying some of the microglia-specific molecules

in the future using magnetic resonance imaging or positron emission tomography among living patients at higher risk. This could act like an alarm clock in the brain that tells us, for a specific patient, that the microglia in a particular region are activated, and this can then inform our diagnosis and perhaps even our treatment in the long term,” concludes Marco Prinz.

The Novo Nordisk Prize

- Advances in medical sciences

The Novo Nordisk Prize recognises an active scientist for her/his excellent research, inspirational leadership and mentoring, leading to a major discovery or breakthrough in biomedical science. The prize is intended to reward and further support biomedical research in Europe.

The Prize of DKK 5 million (EUR 672,000) consists of a research grant of DKK 4.5 million (EUR 605,000) and a personal award of DKK 0.5 million (EUR 67,000). An additional DKK 0.5 million will be awarded for hosting an international symposium within the Prize recipient's field(s) of research. In addition, in celebration of the award, the recipient gives a lecture lasting about 1 hour at his or her workplace, sponsored by the Foundation.

Nomination call

Nominations are invited from the scientific community worldwide defined as academics and scientists working in companies. The public call is published on the Foundation's website in the spring. The nomination and review processes are confidential.

Selection process

At the meetings the Committee considers the nominees' research contributions and medical impact of their discoveries based on the submitted nominations. The short-listed candidates are then selected for further evaluation including a comprehensive bibliometric analysis and international peer review.

The peer reviews, bibliometric report and the nominees' scientific leadership impact serve as basis for the Committee deliberations and decision of awarding the Prize. The award event usually takes place in the spring at the Novo Nordisk Foundation Prize Celebration.

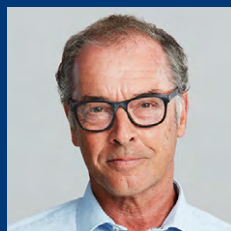
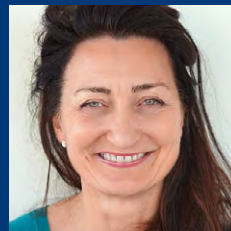
History of the Prize

The prize, originally DKK 50,000, was first awarded on 16 February 1963. The prize was called the Novo Prize from 1963 until 1989, when it was renamed the Novo Nordisk Prize. Until 2020 the Prize was given for a Danish contribution. From 2021 the prize is awarded for a European contribution.

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Committee on the Novo Nordisk Prize



The 2021 Committee comprised the following nine members, appointed by the Novo Nordisk Foundation's Board of Directors:

- » Jørgen Frøkiær, chair - *Aarhus University Hospital, Aarhus University*
- » Harriet Wallberg, - *University of Helsinki*
- » Jaakko Kaprio - *Karolinska Institutet*
- » Rolf Reed - *University of Bergen*
- » May-Britt Moser - *Norwegian University of Science and Technology*
- » Lars Fugger - *John Radcliffe Hospital, University of Oxford*
- » Liselotte Højgaard - *Rigshospitalet, University of Copenhagen*
- » Niels Henrik von Holstein-Rathlou, *SVP - Novo Nordisk Foundation*
- » Mads Krogsgaard Thomsen, *CEO - Novo Nordisk Foundation*

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