I, James Parkinson, am here to reduce your stress levels[1]

Last week, Alois Alzheimer, whom I never met as he was born well after me, took the initiative and had the excellent idea of writing to you.
He told you about the current state of the disease that bears his name, and the most recent and extraordinary therapeutic prospects.
In these extreme times we live in, we can observe continuing progress in biology and medicine. I put pen to paper as I know many people are worried about this disease named after me, all the more so as there are so many of them and they are increasingly younger.

Who am I, James Parkinson?

I was born in the mid-18th century, in 1755. I spent my entire earthly existence in London. My father was a surgeon apothecary and I learned a lot from him. I practised surgery in Shoreditch, in the suburbs of London. I was impressed by the French Revolution, as were many others who were slow to gauge its excesses, which the French have as yet not fully accepted.
I joined ideological clubs: I wrote pamphlets for the London Corresponding Society against staunch conservatives, under the pseudonym "Old Hubert", for the purpose of defending the poor.
Some of your compatriots stupidly claim, to this day, "I don't like the rich". This is how you mathematically increase the number of poor people! I, James Parkinson, "like the poor who become rich". This is the future, and it is easy to understand, believe me!
I was elected by the suffrage of the time to sit in the House of Commons, one of the chambers of Parliament.
In 1799, I was already warning of the harmful effects of tobacco and alcohol in a short book of health tips I published.
In 1802 I developed a passion for geology. I published a voluminous treatise on "The organic remains of a former world" and contributed to the foundation of the Geological Society, alongside Charles Darwin.[2]
In 1817, when I was 62 – you see you should trust your elders – I published a short brochure, which enhanced my reputation: "An essay on the shaking palsy".
This is what I described: "Involuntary tremulous motion, in certain parts of the body, with reduced muscular strength; shaking not linked to movements and even when these parts are supported. Propensity to bend the trunk forwards, and to involuntarily pass from walking to running. The senses and intellects remaining unaffected".
I wrote this based on three cases: two cases I encountered in the street and the last one observed from afar... There was no need for statistics in my time!
I transferred my patients to my son: as with all sons sitting on their father's shoulders, they can see further. I could then devote my time to geology which fascinated me. My name was associated with a fossil bean, pandanacarpus parkinsonis. I finished my life in London, shortly before Christmas of 1824.
It was only half a century later, in 1872, that shaking palsy was to bear my name, on the initiative of professor Jean-Martin Charcot (1825-1893), the father of neurology and head of the Salpêtrière hospital.

The disease that bears my name primarily affects men

It is extremely rare before the age of 45. It concerns around 1% of the population over 65, men more so than women, with a peak of incidence at 70. Poor eating habits combined with pollution, years of

smoking (always very detrimental) and growing levels of stress, have increased the number of people who contract the disease: it affects 4 million people worldwide, 1% of the over 60 population and 3% of over 80s.
150,000 cases have been reported in France and an additional 8,000 new cases every year. With retirement age coming seventy years after the baby boom, a steady increase in the number of cases of these two diseases is to be expected. Bad eating habits, consistently on the increase thanks to processed food over the past few decades, added to the daily stress of modern life, do not bode well for the future.

My disease spares nobody

How, when we experience our first memory lapse, can we not fear this 21st century scourge that is Alzheimer's disease, or can we not live in fear of Parkinson's disease when the slightest tremor occurs? These neurodegenerative diseases haunt us, like a divine curse, an inevitability to which no one is immune in the most affluent countries. It is not unlike diabetes in the Middle Ages, this strange disease, unnamed at the time, which affected the fattest noblemen, clergymen and monastic communities. Exponentially so. "Divine punishment!" people thought. We have now stopped blaming God for all these diseases, including cancer.
An increasing number of celebrities are affected, as revealed regularly by the media, eager to show how the mighty have fallen, to impress us and appeal to our emotions. This feels like a countdown for those who are getting old. You are slowly approaching your demise...
Let's cast our mind back to the spring of 2005, when the slow death of Pope John Paul II, the world's most famous Parkinson's patient, was a televised event all over the world. It was a pathetic spectacle. This agony put an end to years of desperate struggle against his disease. Let's not forget about the decline of boxing champion Mohamed Ali, the fight of US actor Michael J. Fox (who starred in Back to the Future), that of French soprano Mady Mesplé (who wrote a book about it) or that of Robin William whom, as was revealed after he committed suicide, suffered from the onset of the disease. And what about the fate of writer and cartoonist François Cavanna or designer Sonia Rikyel? Even major dictators like Franco, Hitler or Mao had to deal with me and my famous disease, Parkinson's.

Symptoms easy to describe, intracerebral lesions identified

There are 3 major tell-tale signs: slowness of movement, a certain muscle rigidity and tremors in the extremities or throughout the limbs, particularly, when they are at rest. It can also be accompanied by difficulties for the patients to express themselves and great anxiety, which causes depression.
All these symptoms are responsible for instability when standing or walking, which gets worse: small steps, walking bent forward, broadening of the support polygon, i.e. the ground bearing surface of the feet.
The lesions which cause these disorders are located in the centre of the brain, affecting the basal ganglia which normally ensure the harmony and control of your movements, and your postural stability.

Consequences on the brain: very sick neurons[3] in the center of your brain

You have made tremendous progress in two and a half centuries, especially recently, because you know what happens in these basal ganglia. In addition, you have now identified the location of the keys that regulate your static states at night and dynamic states during the day. This is referred to as sleep and wake regulation.

As you already know, the neurons are interconnected by "synapses" or "synaptic boutons", the purpose of which is to produce neurotransmitters.
Your chemists and biologists have discovered, among the causes of the disease named after me, the deficiency in a neurotransmitter produced by the neurons of these ganglia in the centre of the brain.
One of these basal ganglia is called Locus Niger while the other is the Striatum or striated ganglia[4].
They normally contain neurons capable of secreting a very specific neurotransmitter[5]: Dopamine[6].

Your experts call them dopaminergic neurons and they account for one tenth of all the neurons in your body, i.e. a maximum of 10 million. The transmissions occur in the synapses or synaptic boutons. The number of synaptic boutons is estimated at nearly 100,000 for every dopaminergic neuron of the striatum and locus Niger.
It is within these boutons that dopamine is synthesised, to be subsequently stored in vesicles or granules. A single storage vesicle features approximately 5,000 dopamine molecules, and 5 receptors, D1 to D5, are specific to dopamine.
To enhance its synthesis, you can essentially use L-Dopa (di-hydroxy-phenylalanine)[7] which is its immediate precursor.
An enzyme found in dopaminergic neurons, DOPA decarboxylase[8], transforms DOPA[9] into Dopamine.
So you understand why L-Dopa is an important medication for Parkinson's disease. It helps offset the major dopamine deficiency in the striatum which is the root of all troubles.

Dopamine is produced in the brain thanks to two essential amino acids contained in food.
In fact, according to our colleague, Pr Jean Costentin, "it starts with an initial amino acid, L-Phenylalanine, that the body can transform into L-Tyrosine, although this stage is not necessary as L-Tyrosine is contained abundantly in food. It is necessary for the thyroid function. It easily accesses the brain, where it is captured by all neurons, in particular dopaminergic neurons".
He points out that "it is useless to increase L-Tyrosine as it will not provide any more L-Dopa than what it spontaneously produces. The only solution is to add L-Dopa which will be transformed into dopamine thanks to the L-Dopa decarboxylase enzyme, which features strongly in the liver, intestine and blood"[10].

This information is not consistent with that of our colleague, doctor Lemoine[11], psychiatrist, doctor in neuroscience, who recommends Tyrosine in the treatment of Parkinson's disease, claiming that it stimulates the formation of dopamine. However he advises against the simultaneous use of vitamin B6 which destroys dopamine (hence the danger he sees in Magnesium supplements, often enriched in B6), brewer's yeast, neuroleptic or antipsychotic drugs which give the same result.

Obviously, the food we eat, which provides us with these valuable amino acids, influences our behaviour, particularly the way our brain functions. Our brain needs effective blood flow to provide essential oxygen and nutrients. Neurons also need to receive good cholesterol[12] to produce myelin which enables the transmission of nerve impulses.

In the disease that bears my name, Parkinson's, as well as in the disease named "Lewy body dementia", a protein known as alpha-synuclein accumulates inside the dopaminergic neurons in the form of filaments. This accumulation interrupts the transmission of nerve impulses to the musculotendon system and disrupts the stability and functional harmony of the limbs as soon as the body awakens.
Movement disorders, particularly tremors, occur when 70% of the neurons of the substantia nigra are destroyed. This is why you will benefit from understanding the tremendous discovery that I, James Parkinson, am about to present to you.

You cannot imagine the internal suffering - and external too because of how others look at you - of patients as soon as this diagnosis is mentioned! In addition, modern medicine is not very effective, except at the beginning and as long as the patient remains responsive to treatments.

Searching for potential and uncertain causes of the neurodegenerative diseases that bear our names, myself James Parkinson and my colleagues Alois Alzheimer, Arnold Pick and Friedrich Lewy.

Hippocrates, our master and the father of Medicine, said 500 years BC: "Search for the cause and treat it, search for the cause of the cause and treat it".

The disease named after me has become part of neurodegenerative diseases like the one described by Alzheimer, as well as diseases affecting the brain referred to as "frontotemporal dementia"[13], Picq's disease[14] or Lewy body dementia.

In these diseases, incoherence dominates the clinical picture: incontinence, loss of speech, inability to write or read. Additional examinations (scan, Petscan) show that the brain is badly or under-irrigated in fronto-temporal regions. Positron Emission Tomography (PET) helps visualise the brain's activity, but this examination is less accessible as it is very costly. More importantly, it does not affect the inevitable evolution of the disease in any way. It is unfortunately difficult to ask these patients about their eating and social habits in the broad sense.

However they do follow modern consumption habits dominated by an excess of cow's milk products in particular, industrially processed food, full of preservatives, flavour enhancers, artificial flavourings, etc. One cannot help but link these poor nutritional habits in the broad sense with the onset of a variety of metabolic disorders. These bad habits are the source of almost all auto-immune diseases, when the body starts producing antibodies against itself. More specifically, depending on genetic susceptibility, attacking the thyroid, pancreas, muscles, digestive tract, joints, central or peripheral nervous system, the skin, etc.

We have all noticed how eating too many proteins and consuming too much alcohol at night overloads the digestive tract and disrupts sleep. When repeated, these excesses can initially
disrupt the upper digestive tract (the esophagogastric region with a reflux of acidic stomach fluids which burn the oesophagus), and the lower digestive tract (the end of the small intestine and the colorectal region).

– In the upper digestive tract: is Helicobacter pylori implicated?
These are increasingly common in modern society, associated with stress in all its forms. They can lead to the appearance of various digestive disorders.
We should highlight the link between the disease that bears my name and poor eating habits, resulting in the presence of Helicobacter pylori[15] in the stomach. These bacteria only live in the human stomach. They are responsible for the superficial inflammation of the stomach and eventually ulcers, which can develop into stomach cancer, for which the prognosis is not good. Helicobacter pylori can live in the stomach for a long time, protected against acidity by secreting an enzyme, urease, which neutralises gastric acid. These bacteria can therefore survive and proliferate in the mucus of the stomach lining.
It appears that man is the exclusive reservoir of H. pylori. Smoking when infected with these bacteria increases the risk of development into stomach cancer, which could be multiplied sixteen-fold.
The risk of duodenal ulcer and perforated duodenal ulcer also increases. H. pylori seems to be essentially transmitted human-to-human, with the possibility of indirect transmission due to the limited

yet possible survival of these bacteria in the environment. In addition, smoking halves the effectiveness of the treatment designed to eradicate the bacteria.
The links between H. pylori and the disease named after me are uncertain. Nevertheless they are possible, which appears to be proven indirectly when treatments suppress H. pylori[16].
Epidemiological studies are required to confirm these links. This involves detailed interviews of all patients suspected of having contracted the disease, in terms of their dietary habits and any disorders relating to the presence of H. pylori in the stomach.

– In the lower digestive tract: is intestinal permeability implicated?
Intestinal permeability, more or less diffuse inflammation, and the alpha-synuclein pathology are involved from the onset of Parkinson's disease. It is thought that they actually contribute to creating the disease. Eradicating H. Pylori would improve the absorption of Levopoda and therefore the motor symptoms.
The characterisation of intestinal permeability is improving, linked to gluten intolerance (modern seeds) and excess calcium derived from animals, which cross the intestinal barrier and even the blood-brain barrier. This permeability initially results in alterations of the intestinal transit, which is sometimes too rapid or sometimes too slow, with bloating and diffuse intestinal discomfort.

The real causes have finally been identified: Sleep-Wake regulation disorders

It all happens at the top of hormonal glands, in the pineal gland or epiphysis[17].

The identification of the Sleep-Wake system is what led to the discovery of the causes of Parkinson's disease and Parkinsonian syndromes, as well as the explanation of its symptoms.

The discovery by my 21st century colleague Pr Jean Bernard Fourtillan, in Poitiers, France, is extremely important as it helps comprehend the entire negative dynamic which disrupts the limbic system[18] starting from the pineal gland.

This limbic system, like an intracerebral ring, links the basal ganglia. The disruptions cause the symptoms of the disease that bears my name.

This discovery of the century is presented in the letter from his colleague and friend, Pr Henri Joyeux, in which he humorously gets the pineal gland itself to talk.

Read it and re-read it to fully comprehend this formidable discovery. You will notice that it is intended for the researchers and the erudite of your century as well as the general public. True democracy! Why should this discovery be reserved for insiders?

Let me, James Parkinson, summarise this wonderful discovery in a few sentences:

- The pineal gland simultaneously secretes 3 hormones which naturally pass into the bloodstream: Melatonin, 6-Methoxyharmalan and Valentonin. Three successive acetylation stages involving the N-acetyltransferase (NAT) enzyme enable this synthesis.

- As they are biosynthesised in the pineal gland, the 3 hormones are released into the blood. They spread across the body and reach their sites of action: their specific receptors located on the neurons regulate the mental (sleep, wake, cognition, memory, voluntary muscle contractions, etc.) and vegetative life, which automatically functions beyond our control: contraction of smooth muscles, cardiac

movements, respiratory rate by contractions of diaphragmatic muscles, regular blinking of the eyelids, etc.
Melatonin, MLT, is the neuroprotective hormone protecting the neurons from the destructive action of oxygenated free radicals.
6-Methoxyharmalan, 6-MH, has extremely powerful psycho-stimulating properties, similar to those of LSD. It is referred to as the wake or cognition hormone and, more generally, the day hormone.
Valentonin, aka VLT, is the actual sleep hormone. It is referred to as the night hormone.

– This secretion is controlled by the suprachiasmatic nucleus, above the junction, at the base of the brain, of the optic nerves.

– At bedtime, this nucleus which receives less and less light (including in visually impaired people) triggers the secretion of these 3 hormones via the pineal gland, for 8 hours.

– This is how, for a person who usually goes to bed at 10pm, the suprachiasmatic nucleus triggers the secretion of the 3 hormones through a nerve route, from 10pm and for 8 hours. The pineal secretion stops at 6am.

In all cases, the secretion of the 3 hormones by the pineal gland lasts 8 hours, regardless of the time of year or duration of the night.
I should point out the opposite effects of Valentonin and 6-Methoxyharmalan on alertness, blood pressure and muscle tone.
Valentonin, the sleep hormone, reduces blood pressure, muscle tension and body temperature while we sleep. We rest our neurons just as much as our muscles.
Conversely, 6-Methoxyharmalan increases blood pressure to improve the vascularisation of all the organs necessary for an active life, notably the entire muscular system for the activity of a day consisting of 16 waking hours.

Here are the key elements of Pr JB Fourtillan's discovery which apply to the disease named after me

In patients suffering from Parkinson's disease, pineal secretion is greatly reduced, in correlation with the symptoms.

– The secretion of Melatonin is not enough to eliminate the oxygenated free radicals which damage the neurons. They are poorly protected at the beginning of every night. Therefore the simultaneous production of 6-Methoxyharmalan and Valentonin is insufficient.

– This results in the progressive destruction of the dopaminergic neurons of the substantia nigra, illustrated by the increasing prominence in the neurons of the substantia nigra, the alpha- synuclein protein.
- This protein abnormally builds up inside the cell bodies of the dopaminergic neurons, in the form of insoluble filament aggregates. These deposits interrupt nerve impulses, which prevents the synthesis of dopamine at pre-synaptic level, at the junction of all neurons which activate the muscular system in particular.

All this is wonderfully described and illustrated in the video: « The true causes and the treatment of Parkinson's disease », codirected by Pr J B Fourtillan and Pr H Joyeux.

What I have to say next essentially concerns the experts

The two key hormones, Valentonin and 6-Methoxyharmalan, normally travel to their sites of action through the blood, and regulate in turn the mental and vegetative life of the body, during both sleep (8 hours) and wake periods (16 hours).
This regulation is carried out, as with all hormones, by acting on the specific hormone receptors, by modulating:

– in a selective manner, the responses of a few receptors of the 7 major neurotransmitters, including: the 5-HT_{2C} receptors of serotonin (serotonergic receptors 5-HT_{2C}; the α_2 receptors of noradrenaline (noradrenergic receptors α_2); the D_1 and D_2 receptors of dopamine (dopaminergic receptors D_1 and D_2).

– the secretions of the body's 7 endocrine glands.The pineal gland therefore controls the functioning of all endocrine glands, notably the hypophysis and, through the hypophysis, the thyroid-adrenal-sexual glands.

The disease named after me, James Parkinson, and the so-called Parkinsonian syndromes such as Lewy body dementia and Pick's disease, are caused by a significant drop in the pineal secretions of the 3 hormones which make up the Sleep-Wake system.
As all of you who are interested in this disease will have understood, the regulation of alertness, blood pressure and muscle tone result from the competing actions of Valentonin and 6-Methoxyharmalan on the aforementioned receptors.

From 10pm to 6am, Valentonin reduces alertness through the allosteric activation of serotonergic receptors 5HT2C, which as a result maintains the state of sleep.

Like Yin and Yang, Valentonin and 6-Methoxyharmalan have opposite effects.

Standard treatments are effective at the onset of the disease but become ineffective fairly quickly: there are 16 types of medication. It should be noted that their action is limited to muscle relaxation; insufficient muscle contraction is not treated. Antiparkinsonian dopaminergic agents therefore only treat part of the disease.

While there is currently no treatment against the disease of my colleague Alois Alzheimer (as attested by the delisting of the 4 medications offered to patients), a lot of molecules are proposed to patients suffering from the disease that bears my name.
This is only designed for the physicians and patients concerned.
Here are these treatments, along with their trade and generic names, available to all those concerned. They must understand their action mechanisms, their adverse effects or the effects they are attempting to neutralise.

The 3 Anticholinergic antiparkinsonian drugs

the purpose of which is to reduce the problems induced by antipsychotic drugs. They are used to reduce the Parkinsonian side effects (extrapyramidal disorders) observed in psychotic patients treated with neuroleptic drugs.
-Biperiden (Akineton®, Akinophy® and generic drugs): Biperiden hydrochloride. -Trihexyphenidyl (Artane®, Parkinane and generic drugs): Anticholinergic drug with antimuscarinic action.
-Tropatepine (Lepticur® and generic drugs).

The 13 dopaminergic drugs to offset the Dopamine deficiency.

1. Dopaminergic agonists

– Apomorphine (Apokinon® and generic drugs): acts as a dopamine competitive agonist.
– Pergolide (Célance® and generic drugs): acts by directly stimulating the post-synaptic dopaminergic receptors of the nigrostriatal system. It inhibits the secretion of prolactin, increases the serum concentrations of growth hormone and reduces those of Lutein, a Hormone acting on Ovaries and Testicles.
– Piribedil (Trivasta® and generic drugs).
– Pramipexole (Sifrol®): non-ergot derived dopamine agonist prescribed for the treatment of early-stage Parkinson's disease and the restless legs syndrome.
– Ropinirole (Adartrel®, Requip®, Ropinirole Mylan): Antiparkinsonian drug from the dopaminergic family. Offsets the dopamine deficiency by stimulating dopamine receptors. Has notable side effects.
-Rotigotine (Neupro®): dopaminergic agonist.

2. Amantadine

– Amantadine (Mantadix®): weak antagonist of glutamate receptors which increases the release of dopamine and blocks the re-absorption of dopamine.

3. Selective MAO-B inhibitors

– Rasagiline (Azilect®): irreversible monoamine oxydase inhibitor, used as monotherapy at the onset of Parkinson's disease or as an adjuvant treatment in more advanced cases. It belongs to the selective MAO-B inhibitor family. It increases dopamine levels by blocking its
degradation in the brain, thereby improving certain symptoms observed during the disease, such as muscular stiffness and slowness of movement.

– Selegiline (Depreny®, Otrasel®, Selegiline Mylan): molecule from the amphetamine class, more specifically derived from methamphetamine. It is a monoamine oxydase inhibitor (MAOI) which inhibits the degradation of catecholamines (adrenaline, noradrenaline and dopamine) and serotonin.
It is used in the treatment of Parkinson's disease, alone or in combination with medications containing levodopa (Modopar®, Sinemet®) to reinforce their action.

4. COMT inhibitors

– Entacapone (Comtan®): Inhibits the breakdown of catecholamines. Catecholamines are destroyed by the MAO (monoamine oxydases) and COMT enzymes (catechol-O- methyltransferases). It prevents this destruction by inhibiting these enzymes: it is referred to as a COMT inhibitor (COMTI).
Alone or combined with carbidopa[19] and levodopa, it is used to treat the disease named after me.

5. Levodopa + dopadecarboxylase inhibitors

Levodopa absorbed through the digestive system, then through blood, passes into the brain tissue and transforms into dopamine to offset this deficiency. The substitution treatment is effective on akinesia and rigidity. Tremors are less pronounced and occur at a later stage.
L DOPA is superior to dopamine agonists in controlling the symptoms, with however more motor complications. It slows down the progression of the disease and slightly improves the patient's autonomy (compared with the dopamine agonists).

L DOPA remains the preferential treatment for the elderly, especially in case of early-stage cognitive impairments, which can contraindicate the use of other Antiparkinsonian medications.

– Modopar®: Levodopa + Benserazide[20] (dopadecarboxylase inhibitor).
This medication contains levodopa (or L-dopa), which transforms into dopamine in the body, and benserazide which stabilises the effect of levodopa. Its purpose is to offset the dopamine deficiency which characterises Parkinson's disease. It essentially acts on muscular rigidity and the reduction in resting tremors, specific to this condition.

– Sinemet®: Levodopa + Carbidopa (dopadecarboxylase inhibitor).
This medication contains levodopa (or L-dopa), which transforms into dopamine in the body, and Carbidopa which stabilises the effect of levodopa. Its purpose is to offset the dopamine deficiency which characterises Parkinson's disease. It essentially acts on muscular rigidity and the reduction in resting tremors, specific to this condition.

6. Levodopa + Entacapone (COMT inhibitor) + Carbidopa (dopadecarboxylase inhibitor).

– Stalevo®: Levodopa + Entacapone + Carbidopa.
Antiparkinsonian drug from the dopaminergic family. Its purpose is to offset the dopamine deficiency in certain brain regions, responsible for the disorders characteristic of Parkinson's disease.
It contains levodopa (or L-dopa), which transforms into dopamine in the brain, and carbidopa and entacapone which stabilise the effect of levodopa by preventing its degradation.

It is used in the treatment of Parkinson's disease in patients who cannot be stabilised with levodopa combined with carbidopa or benserazide alone, particularly in those suffering from end-of-dose fluctuations (on-off effect).

None of these treatments are very effective, as you know, you the experts in the disease named after me and you the patients. Fortunately things are about to change.

I am sorry for cramming so much information in one letter.

Now I will let you catch your breath. Obviously you are awaiting the new treatment.
Over the next few weeks, and by mutual agreement, Alois Alzheimer and I, James Parkinson, will interview Pr Jean-Bernard Fourtillan, who discovered the common causes of the diseases that bear our names. He will present to you the imminent future.

Best regards.

Pr James Parkinson

Post-scriptum

On website fonds-josefa.org :

- Book and Brochure:

“The pineal gland and the Sleep-Wake system. Therapeutic applications” by Pr J.B. Fourtillan, for sale on Amazon. To order from web site fonds-josefa.org

- The Videos You Tube:

« Pr J B Fourtillan answers questions from Pr H Joyeux »,
Pr J.B. Fourtillan & Pr H. Joyeux (You Tube : duration: 1 h)
« The pineal gland and the sleep-wake system. Therapeutic applications »,
Pr J.B. Fourtillan (YouTube : duration: 37 min)
« The Sleep-Wake system in Creation »,
Pr J.B. Fourtillan (You-Tube, duration : 14min 35 s + 2 min 13)
« The causes and the treatment of Alzheimer's disease have finally been discovered ! », Pr J.B. Fourtillan & Pr H. Joyeux (You Tube : duration 13 min 30)
« The true causes and the treatment of Parkinson's disease »,
Pr J.B. Fourtillan & Pr H. Joyeux (You Tube : dur. 14 min 25)

- Donations will help us independently finance the development of the pharmaceutical forms of the patches, see Make a donation on website fonds-josefa.org

Sources:

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[1] This information is taken from two of our books, published by the Editions du Rocher:
1/ In 2015 – Tout savoir pour éviter Alzheimer et Parkinson (Everything you need to know to avoid Alzheimer's and Parkinson's disease) with Dominique Vialard).
2/ In 2016 – La lutte contre le stress, un remède anti-cancer (Combating stress to prevent cancer) with André and Luc Joyeux.

[2] In 1825, Charles Robert Darwin (1809-1882) followed in the footsteps of his father, a physician who treated the poor in Shropshire. He left for Edinburgh, Scotland, to study medicine, but did not understand the surgery of his time and abandoned his medical studies. At age 22, he embarked on a five-year journey (1831-1836) on board HMS Beagle, a Royal Navy corvette, initially as a geologist. This journey was to make him famous.

[3] See letters 111, "I, your brain" and 112, "Take good care of your neurons" on the www.professeur-joyeux.com website, "All letters" section

[4] Sweden's Arvid Carlsson (Nobel Prize for Medicine in 2000) demonstrated that the striatum featured a high level of dopamine.

[5] See the list, names and roles of all neurotransmitters in the publication Tout savoir pour éviter Alzheimer et Parkinson, Rocher publishers.

[6] The acronym of DihydrOPhenyl-ethylAmine, Dopamine enhances the appeal for novelty, pleasure; it is useful for initiating the action, reducing withdrawal, lack of motivation, indecisiveness. When deficient, it is involved in the movement disorders of Parkinson's disease.

[7] Phenylalanine is an essential amino acid which we must consume in our food via all cereals, leguminous vegetables (2.2g/100 gr), roots and tubers, nuts and seeds, fruit and vegetables, red and white meat (1.3g/100gr), eggs (0.75mg/100gr), fish and seafood. In a nutshell, a varied diet is the assurance of never lacking in phenylalanine. The daily need is approximately 25mg/kg of body weight.

[8] In order to work, decarboxylase needs magnesium, zinc and vitamins B2 and B6 (pyridoxine) which can be easily found in a balanced diet.

[9] There are two chemical forms of Dopa, both optical isomers: L-Dopa and D-Dopa. L-Dopa (levorotatory stereoisomeric form) is metabolised by the body and can cross the blood-brain barrier. Its decarboxylation by DOPA-decarboxylase produces dopamine. It is prescribed for the disease that bears my name. It increases the level of dopamine but induces depression due to the drop in the level of serotonin. It slows down the progression of the disease, slightly improving the patient's autonomy.

[10] Pr Jean Costentin, La Dopamine dans tous ses états, Docis, 2015.

[11] Dr P. Lemoine, Soigner sa tête sans médicament, ou presque, Robert Laffont, 2014.

[12] Good cholesterol can be found in liquid egg yolk which you can eat for breakfast every day in the form of soft-boiled eggs.
[13] Proteins build up in the neurons and block their functioning. Progression is generally faster and mental deterioration more pronounced, with behavioural disorders, delusions, visual hallucinations, loss of reasoning ability, inability to count. It primarily affects elderly subjects. It is combined with tremors, falls, depressive syndromes, airway obstructions, which can lead to respiratory resuscitation, therefore becoming end-of-life signs. This disease is characterised by the frequent appearance of a compulsive taste for sugary foods, certainly linked to stress.
[14] Arnold Pick (1851-1924), Czechoslovakian neurologist and psychiatrist. Friedrich Heinrich Lewy (1885- 1950), a German physician, neuroanatomist and psychiatrist, was the first to discover abnormal deposits of proteins (Lewy bodies) in the early 1900s.
These relatively rare diseases are characterised by the progressive and fairly rapid attack of the frontal and temporal brain regions. In France, an estimated 5,000 people are already affected between the ages of 50 and 60. The neurons in these regions degenerate and die for unknown reasons. No epidemiological study has been carried out on these subjects' eating and social habits.
[15] John Robin Warren and Barry Marshall who discovered it in 1982 were awarded the Nobel Prize for Medicine in 2005.
[16] Bacteria 'linked' to Parkinson's disease – Testerman Traci, BBC Online, 23 May 2011 and Eradication of Helicobacter pylori infection improves levodopa action, clinical symptoms and quality of life in patients with Parkinson's disease –
Hashim H, Azmin S, Razlan H, Yahya NW, Tan HJ, Manaf MR, Ibrahim NM – PLoS One. 2014 Nov 20;9 (11):e112330. doi: 10.1371/journal.pone.0112330. Nov 2014.
[17] See letter no. 137 - I, your pineal gland, aka epiphysis
[18] From the Latin "limbus" meaning "edge" as the limbic system is situated on the edge of the cortex, towards the inside.
[19] Carbidopa is a catecholamine used in Parkinson's disease to inhibit aromatic L-amino acid decarboxylase (or DOPA decarboxylase) and therefore the peripheral metabolism of L- DOPA (or levodopa), which helps increase the proportion of levodopa likely to cross the blood- brain barrier to reach the central nervous system. (https://www.vidal.fr/substances/796/carbidopa/)
[20] Benserazide is used in Parkinson's disease to inhibit aromatic L-amino acid decarboxylase (or DOPA decarboxylase) and therefore the peripheral metabolism of L-DOPA (or levodopa), which helps increase the proportion of levodopa likely to cross the blood-brain barrier to reach the central nervous system.