Parkinson 2017

3rd International Conference on

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Posters

Weller A et al., J Alzheimers Dis Parkinsonism 2017, 7:5 (Suppl)
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Olfactory hallucination in a super smeller

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Introduction: True hypersomnia in a patient with phantosmia has not heretofore been described.

Case study: A 19-year-old right –handed female presented with a 4-month history of sudden onset of hallucinated smell and taste after swimming, when water infused her nostrils. The phantosmia was an unpleasant, fruity, rotten aroma which was always concurrent with the taste of rotten fruit. The phantom taste was 7/10 in intensity. The smell was always the same aroma but of variable intensity. It would occur every day up to three times a day, and usually 6-7/10 in intensity, and it would last from 1 hour to many hours. Since the onset of this, she found that some odorants when phantosmia is present have enhanced intensity, more than 150 % of normal, including kitchen aromas, bleach, and soap.

Results: Abnormalities in Chemosensory Testing: In the absence of phantosmia: Olfaction: Alcohol Sniff Test: 30 (normosmia). Suprathreshold Amyl Acetate Odor Intensity Testing: hyperosmia. Retronasal Olfactory Testing: Retronasal Smell Index: 3 (reduced). Gustatory Testing: Propylthiouracil Disc Taste Test: 8 (normogeusia). When phantosmia is present: Alcohol Sniff Test: 13 (hyposmia).

Discussion: Possibly the phantosmia changed her focus of attention to ambient aroma, enhancing her intensity perception and thus reducing her olfactory threshold; such attention reduced olfactory stimuli threshold has been seen in industrial workers exposed to solvents (Schwartz, 1989). Possibly the primary abnormality is hyperosmia: Her olfactory sensitivity threshold may be so low that she detects odors in the environment that others don't, which are interpreted as phantosmia and phantogeusia (due to reverse retronasal olfaction). This case highlights the need to test those who complain of phantogeusia and phantosmia for olfactory sensitivity, it also suggests treatment approaches for resistant phantosmia and phantogeusia including physical or pharmacological measures to reduce the underlying olfactory ability. Further studies in this arena are warranted.

Biography

Weller A is a fourth year Medical Student with extensive research in Neurology and Neurodegenerative disorders. He has Associate Degree in Psychology, Bachelor Degree in Business, currently pursuing Masters in Public Health. He is interested in all types of neurology research as I consider it as an enormous and expanding field of medicine.

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Notes:

Azza A Ali et al., J Alzheimers Dis Parkinsonism 2017, 7:5 (Suppl)
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Modulatory effects of bisphenol A, caffeine, epigallocatechin-3-gallate and their combinations against parkinsonism in rats

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Background: Parkinson's disease (PD) is the most common neurodegenerative movement disorder. It is associated with selective loss of dopamine (DA) neurons and levels in the brain leading to the appearance of motor as well as non-motor symptoms. Excessive exposure to Manganese (Mn) has been associated with increased risk of developing classic PD and manganism. Bisphenol A (BPA) is a synthetic estrogen-like substance; its exposure is almost universal and has showed neuroprotection against neuronal degeneration. Caffeine (Caf) is the most consumed psychostimulant in the world and demonstrated as a promising neuroprotective and in symptomatic treatment of PD. Epigallocatechin-3-gallate (EGCG) has known interactions with caffeine and considered as a powerful antioxidant, anti-inflammatory and anti-apoptotic with dopaminergic neuroprotective effect.

Objective: The objective of this study is to evaluate and compare the behavioral effects of BPA, Caf, EGCG and their combinations against PD induced by Mn in rats.

Methods: Rats were divided to 7 groups. One group was normal and 6 groups received daily for 5 weeks MnCl2 (10 mg/kg) either alone or in combination with each of the following: BPA (50 mg/kg), caffeine (10 mg/kg), EGCG (5 mg/kg), caffeine and EGCG and combination of all. Five behavioral tests were used (grid, bar, swimming, open-field and Y-maze tests). In addition, biochemical changes in monoamines, AChE, GSK-3B as well as excitotoxicity, apoptotic, neuroinflammatory and oxidative markers were also evaluated besides histopathological examinations.

Results: Behavioral data showed that Mn induced increase in catalepsy, delay in decision making, disruption in neuromuscular co-ordination and vigilance as well as decrease in locomotor, emotionality and exploratory activities together with impairment of spatial memory. All used treatments improved most behavioral impairments however Caf and EGCG co-administration showed more pronounced improvements than each one alone even in the presence of BPA. Biochemical and histopathological examinations in the striatum and frontal cortex confirmed behavioral one. EGCG showed marked protection from neuronal degeneration in all brain regions than Caf which still showed some nuclear pyknosis in cerebral cortex and hippocampus.

Conclusion: Neuronal degeneration induced by Mn was partially improved with BPA. Co-administration of Caf and EGCG showed more pronounced protection than each one alone with superiority of EGCG.

Biography

Azza A Ali has completed her PhD specialized in Pharmacology and Toxicology from Faculty of Pharmacy, Cairo University, Egypt. Her Postdoctoral studies included different scientific aspects especially on neurodegenerative disorders. She has also developed research line of behavioral pharmacology in Egypt. She is Member of many scientific societies such as (AAPS) and Alzheimer's Association (ISTAART). She is also Editorial Board Member of many international Journals such as *Brain Disorder & Therapy*, *Acta Psychopathologica*, *EC Pharmacology* and *Toxicology* as well as Organizing Committee Member at the 7th International Conference on Dementia & Care Practice. She has published more than 50 papers in reputed journals, supervised and discussed more than 80 PhD and MSc thesis and actively participated by oral and posters presentations at many international conferences especially on Alzheimer's disease and Dementia as Dementia Conferences 2015, 2016 and Alzheimer's Association International Conference (AAIC 2016). She has many appreciation certificates and certificate of best presentation award at 19th International Conference on Environmental Pollution and Pollution Control (ICEPPC 2017). Now she is the Head of Pharmacology and Toxicology Department at Al-Azhar University, Egypt.

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Comparative study on the potential role of cocoa, epigallocatechin-3- gallate, coenzyme Q10 and their combination against manganese-induced parkinsonian like syndrome in rats

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Background: The worldwide prevalence of Parkinson's disease (PD) is increasing day by day. It is the most common movement disorder which occurs due to complex interactions between environmental and genetic factors. There are strong associations between excessive exposure to Manganese (Mn) and neurodegenerative diseases characterized by extrapyramidal motor disorder similar to PD. Cocoa is a potent antioxidant and can protect cells against oxidative stress. Epigallocatechin-3-gallate (EGCG) is responsible for most of green tea's role in promoting good health with powerful antioxidant, anti-inflammatory and anti-apoptotic as well as dopaminergic neuroprotective effects. Coenzyme Q10 (CoQ10) is anti-aging and radical scavenger with effectiveness in improving cognitive disorders.

Objective: To evaluate and compare the potential effects of Cocoa, EGCG, CoQ10 and their combination against PD induced by Mn in rats.

Methods: Rats were divided to 6 groups. One group was normal and 5 groups received daily for 4 weeks MnCl2 (10 mg/kg IP) either alone or in combination with each of the following: Cocoa (24 mg/kg PO), EGCG (5 mg/kg IP), CoQ10 (200 mg/kg PO) and their combination. Five behavioral tests were used (Grid, Bar, Swimming, Open-field and Y-maze tests). In addition, biochemical changes in monoamines, AChE, BDNF, GSK-3, Glutamate, GABA, INOS, Cox 2 as well as apoptotic, neuroinflammatory and oxidative markers were also evaluated besides histopathological examinations.

Results: Behavioral data showed that Mn induced increase in catalepsy, delay in decision making, disruption in neuromuscular co-ordination and vigilance as well as decrease in locomotor, emotionality and exploratory activities together with impairment of spatial memory. All used treatments improved most behavioral impairments however co-administration of Cocoa, EGCG and CoQ10 showed more pronounced improvements than each one alone. Biochemical and histopathological examinations in the striatum confirmed the behavioral ones. Cocoa and EGCG showed marked protection from neuronal degeneration in all brain regions than CoQ10 which still showed some nuclear pyknosis in cerebral cortex and hippocampus.

Conclusion: Neuronal degeneration as well as behavioral changes induced by Mn was partially improved either by Cocoa, EGCG or CoQ10 with superiority of EGCG and Cocoa but their combination showed more pronounced protection than each of them alone.

Biography

Azza A Ali has completed her PhD specialized in Pharmacology and Toxicology from Faculty of Pharmacy, Cairo University, Egypt. Her Postdoctoral studies included different scientific aspects especially on neurodegenerative disorders. She has also developed research line of behavioral pharmacology in Egypt. She is Member of many scientific societies such as (AAPS) and Alzheimer's Association (ISTAART). She is also Editorial Board Member of many international Journals such as *Brain Disorder & Therapy*, *Acta Psychopathologica*, *EC Pharmacology* and *Toxicology* as well as Organizing Committee Member at the 7th International Conference on Dementia & Care Practice. She has published more than 50 papers in reputed journals, supervised and discussed more than 80 PhD and MSc thesis and actively participated by oral and posters presentations at many international conferences especially on Alzheimer's disease and Dementia as Dementia Conferences 2015, 2016 and Alzheimer's Association International Conference (AAIC 2016). She has many appreciation certificates and certificate of best presentation award at 19th International Conference on Environmental Pollution and Pollution Control (ICEPPC 2017). Now she is the Head of Pharmacology and Toxicology Department at Al-Azhar University, Egypt.

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G Shilau et al., J Alzheimers Dis Parkinsonism 2017, 7:5 (Suppl)
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9-tert-butyl-apomorphine (DTBA) as an effective soluble compound with antioxidant activity for treatment of experimental Parkinsonism

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Objective: To evaluate antiparkinsonic effect of the investigated compound were used two different model of experimental Parkinsonism in white rats: 1st – classical model with overdosege of neuroleptic drugs and 2nd – new one, with injection the initiator free-radical oxidation (FRO), FRO - Fe-ascorbate mixture, into *substantia nigra* of the brain.

Background: One of the causes of Parkinsonism might be activation of lipids peroxidation (LPO). But there might take place and other FRO initiator reactions, apart from LPO inhibition, that should also be considered – of radical fragmentation processes. The bifunctional organic compounds like dopamine in particularly can undergo FRO, that is reactions of radical fragmentation. In former investigations we were found, that the products of free-radical oxidation (FRO) of dopamine on adding Fe-ascorbate *in vitro* resulted in predominant formation 4-(2-aminoethyl)-benzoquinone-l,2 along with other products.

Results: Treatment of dopamine with Fe-ascorbate mixture resulted in predominant formation 4-(2-aminoethyl)-benzoquinone-l, 2 along with other products. The new fenol's derivative DTBA inhibit FRO of dopamine *in vitro*: Antioxidant activity of DTBA in 10 times exceed the same one of dibunolum. This compound (DTBA) has not negative reaction (vomiting, nausea) that caused by Apomorphine. Animals treated with the new phenol derivative DTBA demonstrated considerably faster recovery from catalepsy.

Conclusions: In the mechanism of Parkinsonism development an important role may belongs not only LPO but FRO (that is reactions of radical fragmentation) of dopamine initiated by non-heme Fe and phenol derivatives with AO-activity might be used as affective means for the treatment of Parkinsonism.

Biography

G Shilau has completed his PhD at age of 29 years old from Byelorussian State Medical University. He worked as Senior Scientific Worker in the Laboratory of The Biochemistry of Neurohormones over Mention University and then as leading Scientific Worker, Central Scientific-Investigating Laboratory of Byelorussian Medical Postgraduate Academia. Currently, he works as deputy Director of the Center of Medical Information EOCEN and continues his scientific work in close cooperation with Laboratory of free-radical process chemistry of the Research Institute of Physical Chemical Problems of the Belarusian State University. He has published more than 40 papers in reputed journals.

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Genetic analysis of 20 facioscapulohumeral muscular dystrophy (FSHD) probands by southurn blot analysis and investigation of genotype/phenotype correlation

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Facioscapulo humeral muscular dystrophy (FSHD) is a dominantly inherited disease that is characterized with involvement and weakness of the facial and scapular muscles. It's one of the most common forms of myopathies with prevalence 1:7500-1:15000. FSHD is caused by genetic and epigenetic factors and can be classified into two subgroups: FSHD1 and FSHD2. FSHD1-accounts for around 95% of the cases- is associated with contraction of D4Z4 macrosatellites at the subtelomeric region of chromosome 4. Normal individuals carry 11-100 repeats whereas FSHD1 patients have 1-10 D4Z4 repeats. 20 Iranian FSHD probands were recruited. All cases consented to participate after being informed about the project. Their clinical presentations were recorded precisely. DNAs were extracted using salting out method and digested by EcoRI and EcoRI/BlnI enzymes. The southern blot was optimized for DIG-labelled probe P13E11. The average age at onset of patients was 15-/+6.5 (range 1-49 years). 12 out of 20 patients were familial and remaining cases were sporadic. Respectively, four (20%), six (30%) and ten (50%) of patents presented mild, severe and moderate type of the disease. Results of Southern blot showed 19 of 20 (95%) patients shared less than nine D4Z4 repeats. One patient presented more than 11 D4Z4 repeats. Our findings showed a correlation between genotype and phenotype. More severe type of the disease war related to less number of the D4Z4 repeats.

Biography

Afagh Alavi has completed her MSc, PhD and Postdoctoral studies from University of Tehran. She has been working as an Assistant Profesor in the University of Social Welfare and Rehabilitation Sciences. She has published more than 13 papers in reputed journals.

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Tappakhov Alexey et al., J Alzheimers Dis Parkinsonism 2017, 7:5 (Suppl)
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Two neurodegenerative disorders (Parkinson's disease and essential tremor) in extrem Siberia (the Yakut population)

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The Sakha (Yakutia) Republic is the largest region of the Russian Federation and is located in the north-eastern part of the Eurasian mainland. It has a population of 959 689 (2016), consisting mainly of ethnic Yakuts (49.9%) and Russians (37.8%). About 40% of Yakutia lies above the Arctic circle and all of it is covered by permafrost which greatly influences the region's ecology and limits forests in the southern region. We first studied the two most common neurodegenerative disorders (Parkinson's disease (PD) and essential tremor (ET)) in the Yakut population. The prevalence of PD in Yakutia was 67 per 100,000 of the adult population. The prevalence of the disease in Yakutsk was 76.5 per 100,000 population, and in the regions it varied widely from 9.8 to 185.6 per 100,000 population. We did not reveal statistical differences in prevalence of PD among the Yakut (75.4 per 100,000) and Russian (73.4 per 100,000) population. As for the essential tremor, we diagnosed this disease in 32 patients, including Yakuts – 37.5% (12 people), Russians – 34.4% (11 people). The mean age of patients with ET was 67.3 ± 1.83 years (range 43 to 85 years). The greatest number of patients accounted for the age group of 70-79 years (12 people, 37.5%). In 14 (43.8%) patients the disease was hereditary. 43.8% (14 patients) had a later onset of the disease (over 60 years), and 28.1% (9 patients) had an early onset (20-30 years).

Biography

Tappakhov Alexey is a neurologist and postgraduate student in the Department of neurology and psychiatry at the North-Eastern Federal University in Yakutsk. Scientific interest is devoted to the study of neurodegenerative diseases and movement disorders. Alexey Tappakhov study of epidemiologic, genetic and clinical features of Parkinson's disease in the Sakha (Yakutia) Republic. He is looking for reliable neuroimaging, neurophysiological biomarkers of Parkinson's disease. Alexey Tappakhov has published more than 10 papers about Parkinson's disease and other neurodegenerative disorders.

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Tourette syndrome in Argentina

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I will briefly explain my own story about how I created the Association. When my daughter was 8 years old – at present she is 20 - she was diagnosed with TS. It was a long 2-year journey until a diagnose was reached. When my daughter was 6 year old I began to notice several tics. During a routine control with her pediatrician, who knows her since she was born, I told him about certain sounds she made and I insisted on these tics such as winks and short head movements. Of course I received the same answer, "there is absolutely nothing wrong with your daughter". As the year went by and the situation worsened for new problems appeared such as anger and some phobia, the environment at home was one of constant fighting, screaming and swearing. Then came the string of doctors and medical studies, until a final diagnose was reached. At that time my daughter was 8 years old. Then came hard years, years of research, years of acceptance, years of trial and error, good and bad doctors, struggling with family and denial, years of explaining to all her teachers and to the parents of her classmates. Years spent explaining that she was able to do the same things like any other girl her age first, then as a teenager, years sending medication to camp trips, to a graduate trip, with the look of apprehension for the responsibility and especially the fear of people who did not understand that it was neither dangerous nor contagious, that they just had to make sure she took her medication and played, enjoyed, danced, sang, etc. We thought it would be a good idea to transmit all the experience and research to other families with children with TS. On September 19th, 2012, the Argentine Association for Tourette Syndrome was created, primarily to inform and educate teachers that our children would not have a bad experience throughout their school years, the most important experience of their growth. AATS is a non-profit association where all is done with the heart and with one's own efforts, besides the collaboration received from professionals attending the conferences. The workshops were declared of Educational Interest by THE LEGISLATURE OF THE AUTONOMOUS CITY OF BUENOS AIRES also BY THE NATIONAL MINISTRY OF EDUCATION - Commission of Public Health and of Education of the LEGISLATURE of the PROVINCE OF BUENOS AIRES – and by The National Institute against Discrimination. Our Facebook page includes over 18000 people and there are many countries throughout Latin America. Our purpose is to spread, inform and educate because we believe that children must have a happy education, where they can enjoy going to school and not coming back crying because of lack of information on behalf of teachers, principals, classmates and families.

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The involvement of caspase family in Parkinson's disease: A study of neurotoxicity in human dopaminergic neuron differentiated from stem cells

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Parkinson's (PD) disease is characterized by the death of dopamine-containing neurons (DCN), which clinically translates into impaired motor functions and neuropsychiatric problems. Since the majority of studies have been using animal models of PD or post-mortem samples from PD patients, the progression of the disease in human cells still remains to be fully characterized. Therefore, there is an imperative need to go back to basics and understand the mechanism of the events happening prior to the dopaminergic cell death. To this aim, a human dopaminergic cell model was developed from a human neural progenitor ReNcells, and to which a neurotoxin 6OHDA was applied to mimic the different stages of PD. The objective of this study was to investigate the progression of the disease particularly in relation to the involvement of the caspase, a family of cysteine proteases which acts as pro-apoptotic proteins that promote cell death via activation of the caspase cascade. Our results showed that 6OHDA-induced toxicity triggered caspases-2 and -8 activation, which in turn activated caspase-3 leading to death of DCN. Additionally, activation of caspases-2, -3, and -8 was reduced by z-VAD-FMK in 6OHD-treated cells. Our results suggest that caspase-2 cause's cell death might be via an indirect NF kappaB route. This study has established a PD model which can provide better insight to PD pathogenesis on a biochemical and molecular level, leading to a better understanding of PD and potential for new treatments.

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An overview of sleep disturbances in Parkinson's disease

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This review provides an overview of the most common sleep disturbances in PD, including: Insomnia, REM behavior disorder, restless legs, circadian disturbances, sleep-disordered breathing, excessive daytime sleepiness and other Parkinson's sleep related disturbances. Furthermore, we explore the pathogenesis and management of each sleep disorder in this population. The early diagnosis and treatment of sleep problems may be a valuable instrument in the prevention and/or prognosis of neurodegenerative disorders including PD. The use of questionnaires for screening of different sleep disturbances can be useful, but polysomnographic studies continue being the gold standard and are needed to confirm the diagnosis in many cases.

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Multiple GPCR heteroreceptor complexes: New targets for treatment of Parkinson´s diseases

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The introduction of allosteric receptor–receptor interactions in GPCR heteroreceptor complexes of the CNS gave a new dimension to brain integration and neuropsychopharmacology. The molecular basis of learning and memory was proposed to be based on the reorganization of the homo- and heteroreceptor complexes in the post junctional membrane of synapses. Long-term memory may be created by the transformation of parts of the heteroreceptor complexes into unique transcription factors which can lead to the formation of specific adapter proteins. The observation of the GPCR heterodimer network (GPCR-HetNet) indicated that the allosteric receptor-receptor interactions dramatically increase GPCR diversity and biased recognition and signaling leading to enhanced specificity in signaling. Dysfunction of the GPCR heteroreceptor complexes can lead to brain disease. The findings of dopamine (D2R) and adenosine (A2AR) hetero and isoreceptor complexes in the brain over the last decade gave new targets for drug development in Parkinson´s diseases. We studied the possible reorganization of the A2A-A2A homoreceptor complex and the A2AR-D2R, A2AR-mGluR5 heteroreceptor complexes in the dorsal and ventral striatum in the hemiparkinson rat using the proximity ligation assay. The results were obtained in the dorsolateral striatum comparing the 6-OHDA lesioned side with the unlesioned side, 4 weeks after the lesion (6-OHDA microinjections into the medial forebrain bundle). The A2AR-D2R heteroreceptor complex was found to be significantly increased on the lesioned side ($p< 0.05$, Student's paired t-test , N= 4 rats), which was true also for the A2AR-mGluR5 heteroreceptor complex ($p< 0.05$, Student's paired t-test , N= 4 rats) . The A2AR-A2AR homoreceptor complex was not significantly altered on the lesioned side vs the unlesioned side. Thus, the loss of DA terminals and DA transmission in the dorsal striatum on the lesioned side leads to an altered balance of the hetero and homoreceptor complexes with significant increases of the A2AR-D2R and A2AR-mGluR5 heteroreceptor complexes on the lesioned side. In contrast, the A2AR-A2AR homoreceptor complexes were not altered on the lesiond side vs the unlesioned side. These results may be interpreted as indicating that in the untreated hemiparkinsonian rat the A2AR-D2R and A2AR-mGluR5 heteroreceptor complexes become more dominant favoring excitation of the dorsal striato-pallidal GABA neurons mediating motor inhibition. Hypokinesia becomes increased. The hypothesis is given that changes in the function of the dopamine and adenosine heteroreceptor complexes may especially help us understand the molecular mechanisms underlying the motor complications of long-term therapy in Parkinson's disease (PD) with levodopa and DA receptor agonists. In the indirect pathway the potential role of the A2AR-D2R, A2AR-D2R-mGluR5 and D2R-NMDAR heteroreceptor complexes in PD are covered.

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Parkinson's disease – Lessons from the grave

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Since the 1990s medical research has been focused on large scale drug trials. Individual case histories have, largely, been ignored. It is always difficult to have a precise individual prognosis for people with Parkinson's (PWPs) as it is such a heterogenous disorder. However, a retrospective provides a blow by blow history of exactly how each case played out. It isn't perfect because we don't have the holy grail setting the successful protocol out for everyone to follow. This Poster is a preliminary attempt to gather such information with a magnificent seven cases who came together on their Parkinson journey via the ask the doctor forum when it was in the capable hands of Dr. Abraham Lieberman, of the Muhammed Ali Parkinson Center in Phoenix, Arizona. Giving the limited space available in a poster, the focus will be on the most important lesson from each case, especially if repeated in more than one of this small sample. Such Case Histories could be gathered from many surviving caregivers, although the process of grief and other recovery processes can be excruciatingly painful. Caregivers have only recently been given credit for their role and may still be overlooked for the valuable repository of real life information they represent.

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Astrocyte iron metabolism is involved in the nigral iron accumulation in Parkinson's disease

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Nigral iron accumulation plays a key role in Parkinson's disease (PD). It is worth attention that unlike neurons and other glia cells, astrocytes have few iron deposit. Our previous results show that 6-hydroxydopamine (6-OHDA) up-regulates iron import and down-regulates iron export in nigral dopaminergic neurons, thus leading to cellular iron accumulation. While 6-OHDA promotes iron traffic in astrocytes by up-regulating both iron import and export, thus avoiding iron deposit. Therefore, astrocytes may be "iron pump". Then, if the enhanced iron traffic in astrocytes is the source of iron accumulated in dopaminergic neurons? What is the mechanism of the enhanced iron traffic in astrocytes? In this study, we found that 6-OHDA that may be produced by auto-oxidation in dopaminergic neurons, promoted iron traffic in astrocytes by the activation of hypoxia-inducible factors (HIFs) in astrocytes. In addition, it was HIF-2α, but not HIF-1α that regulated the iron transport in astrocytes. The study is to explore the mechanisms of iron metabolism in astrocytes and its role in the nigral iron accumulation in PD, thus providing insight into the control of astrocytes function to protect neurons as therapeutic strategies of PD.

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Managing Life with Parkinson's disease before and after deep brain stimulation surgery

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Parkinson's disease is a highly destabilising disease and comes down as a curse not only to the victim but to his or her family members and relatives. It is incurable and tends to destroy the quality of life .Therefore, the affected family and the victim should not surrender , instead should accept this as an opportunity to invent and practise new systems in their daily life that will overshadow the ill effects of the disease. Activities like regular get together of family members, friends, well wishers , celebrating birthdays and anniversaries should be held regularly to reduce the risks of PD. Visits to nearby touirist spots , picnic etc will help to overcome the drudgery and infuse freshness in the mind of the victim. My wife was first diagonised with PD in 2004 and she was put to prescribed medication which improved her condition. But I studied a lot on the disease and knew that it will progress with time and a stage will come when she will be near immobilised. So, I started planning for other forms of changes in daily life. Being an Engineering professional, I was invited at various international conferences to present papers. Some organisers readily agreed to bear the cost of my wife's visit along with me which helped us to network with many participants and later there was regular skype talk with many of them after the event. Thus, on return, we felt fresh and invigorated. During this 12 years till 2016, I had the opportunity to participate at about 12 events and she also got the opportunity to come out of her mental block due to the disease. In he year 2012 she has underwent a deep brain stimulation surgery , a major and highly expensive treatment . Post this surgery, her tremor has completely disappeared and she is feeling that her life is improving . Though she needs help- in walking and movements but her spirit is very high . At this event we request the organiser to let her participate and read out her own story of how she is facing the ordeal with courage and firtitude.

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