“Approccio Ai Disturbi Psichici”

marco onofrj

Department of Neuroscience and Imaging, Institute of Neurology, University “G.dAnnunzio” Chieti-Pescara-Italy, Aging Research Center Ce.S.I., “ G. d’Annunzio” University Foundation, Chieti-Pescara, Italy















Licenza porto di fucile tiro a volo n° 471487-N, rilasciato dalla questura di Ascoli Piceno il 18.08.2011

Acquistato le seguenti armi:

Dall'armeria C.L.C.F. Sport Line s.r.l. di Ascoli Piceno, via Circonvallazione Nord 29

- Carabina semiautomatica cal. 30.06 marca Merkel matricola SR 11954 cat. 16136
- Fucile a pompa cal. 12 marca Smith&Wesson matricola 314B99
- N 100 a palla unica cal. 45 A.C.P.
- N 100 cartucce a palla unica per fucile da caccia 30.06

Dal sig. Rosetti Eugenio di San benedetto , via C. Colombo, n°34

- Pistola semiautomatica cal. 45 ACP marca STI matricola S003297 cat. 14477

Dal sig. Organtini, residente a Frazione Capodacqua, n°2

- Carabina cal. 7x57 marca Mauser matricola 79263
- N 80 cartucce a palla unica per fucile da caccia cal. 7x57

Inoltre dichiara di essere in possesso delle seguenti armi

- Fucile 2 canne sovrapposte cal. 12 marca Rizzini matr. 56880
- Carabina cal 375 HH Magnum marca Remmington matr. C6842109 cat. 599
- Carabina cal 22 L.R. BRNO matr. 244906
- Carabina cal. 30.06 marca F.N. matr. 11.67
- Pistola semiautomatica cal. 7,65 parabellum Beretta matr. 18074x
- Cat. 432 con kit di conversione per pistola semiautomatica cal. 22 L.R. mat. K05167U
- Carabina aria compressa cal. 4,5 marca Diana matr. 01230867
- Fucile 2 canne giustapposte cal. 12 marca Tanzi matr. 18634
- Fucile 2 canne giustapposte cal. 16 marca ?

. 10 Ottobre 2011

Prevalence of features:

Hypersexuality
77%Punding
61%Social isolation
45%Wandering
27%Gambling
22%Drug hoarding
22%Paraphilia
2%

Somatoform disorders in PD herald the onset of dementia and are frequently found indementia with lewy bodies

Crossing the Borders Between Neurology and Psychiatry in Functional Neurological Disorders

Victor Peralta, MD, PhD[1] and Anthony E. Lang, MD, FRCPC[2]*

[1]*Psychiatry Unit B, Complejo Hospitalario de Navarra. Pamplona, Spain*
[2]*Toronto Western Hospital, University of Toronto, Toronto, Ontario, Canada*

Movement Disorders, Vol. 26, No. 8, 2011

Background

Somatoform Disorder:Hysteria in PD : 1. Marsden CD Hysteria – a neurologist's view Psychol Med 1986, 16:277-288 2. Walters AS, Boudwin J, Wright D, Jones K. Three hysterical movement disorders. Psychol Rep. 1988 Jun;62(3):979-85.3. Lang AE, Koller WC, Fahn S. Psychogenic parkinsonism. Arch Neurol. 1995;52:802-10.

Psychiatric manifestations in PD/non-motor symptoms in PDDepression-Apathy-Anedonia

New Cathegories: Punding
compulsive disorders

J Neurol (2008) 255:31–36
DOI 10.1007/s00415-007-0655-z

ORIGINAL COMMUNICATION

Xinjun Li
Jan Sundquist
Helen Hwang
Kristina Sundquist

Impact of psychiatric disorders on Parkinson's disease

A nationwide follow-up study from Sweden

All people in Sweden hospitalized for psychiatric disorder and PD during the study period (1987 to 2001).Standardized incidence ratios (SIRs) with 95% confidence intervals(CI) for PD of 11.56

- Depression - Schizofrenia - Mood - Neurotic Personality Disorder

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- Depression
- Schizofrenia
- Mood
- Neurotic Personality Disorder

J Neurol
DOI 10.1007/s00415-010-5465-z

ORIGINAL COMMUNICATION

Frequency of dementia, depression, and other neuropsychiatric symptoms in 1,449 outpatients with Parkinson's disease

Oliver Riedel · Jens Klotsche · Annika Spottke · Günther Deuschl · Hans Förstl · Fritz Henn · Isabella Heuser · Wolfgang Oertel · Heinz Reichmann · Peter Riederer · Claudia Trenkwalder · Richard Dodel · Hans-Ulrich Wittchen

depression, 25% (13.2–47.9%) (MADRAS)deme
29% (12.2–59.4%) (MMSE, PANDA
validation)psychotic syndromes, 12.7% (3.1–4C
sleep disturbances (49%) anxiety (20%)

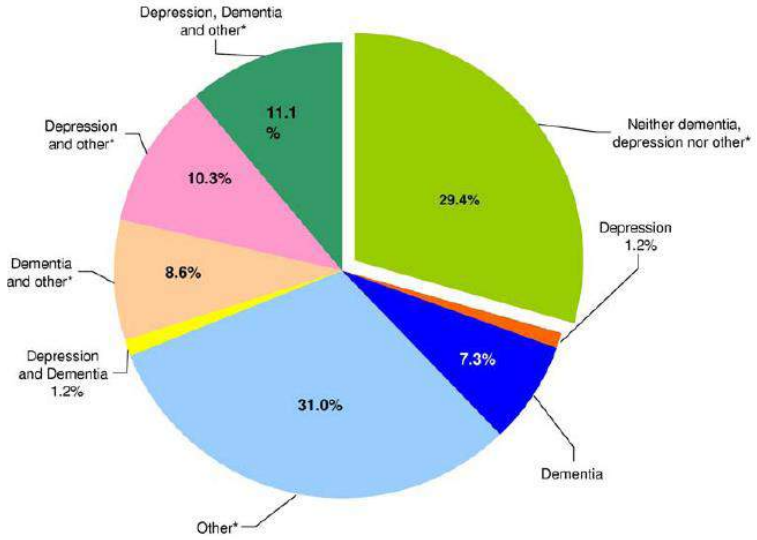

Fig. 2 Frequencies and combinations of dementia (DSM-IV), depression (MADRS $\geq$ 14), and other* neuropsychiatric symptoms in PD outpatients (n = 1,449) (*asterisk* visual misperceptions, hallucinations, paranoid symptoms, delusions, anxiety symptoms, compulsion symptoms)

Now somatiform disorder is quoted:

Neurol Sci (2010) 31:35–40
DOI 10.1007/s10072-009-0165-0

ORIGINAL ARTICLE

Psychiatric symptoms in Parkinson's disease assessed with the SCL-90R self-reported questionnaire

Chiara Siri · Roberto Cilia · Danilo De Gaspari · Federica Villa · Stefano Goldwurm · Catalano Marco · Gianni Pezzoli · Angelo Antonini

40%

Movement Disorders
Vol. 25, No. 13, 2010, pp. 2105–2113
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Anxious Personality Predicts an Increased Risk of Parkinson's Disease

James H. Bower, MD, MSc,[1*] Brandon R. Grossardt, MS,[2] Demetrius M. Maraganore, MD,[1] J. Eric Ahlskog, PhD, MD,[1] Robert C. Colligan, PhD,[4] Yonas E. Geda, MD, MSc,[3,4] Terry M. Therneau, PhD,[2] and Walter A. Rocca, MD, MPH[1,3]

quoted

Cohort study on somatoform disorders in Parkinson disease and dementia with Lewy bodies

Marco Onofrj, MD
Laura Bonanni, MD, PhD
Lamberto Manzoli, MD
Astrid Thomas, MD, PhD

Conclusions: The frequency of somatoform disorder (SFMD) (with catatonic signs) in Parkinson disease and dementia with Lewy bodies suggests that SFMD are part of the spectrum of Lewy body diseases. *Neurology*® 2010;74:1598–1606

Movement Disorders
Vol. 25, No. 15, 2010, pp. 2493–2500
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CME

Viewpoint

What Are the Most Important Nonmotor Symptoms in Patients with Parkinson's Disease and Are We Missing Them?

David A. Gallagher, MRCP,[1] Andrew J. Lees, MD, FRCP,[2] and Anette Schrag, MD, PhD, FRCP[1*]

Definition:

Suggestibility: Babinski

Hysteria has a purpose, hypocondria is an angst:Leonard

Doxomorfic: 300.11

Conversion symptoms typically do not conform to known anatomical pathways and physiological mechanisms, but instead follow the individual's conceptualization of a condition. A "paralysis" may involve inability to perform a particular movement

But the psychiatric Handbook most used by students suggest that Hysteria –Hypocondria underlie a malevolent personality !

DSM-IV-TR: 300.81 Somatization disorder:Classic presentation : female, low educational level

Somatoform disorder in Parkinson's Disease: code ?university education, male

Diagnostic criteria for 300.81 Somatization Disorder

A. A history of many physical complaints beginning before age 30 years that occur over a period of several years and result in treatment being sought or significant impairment in social, occupational, or other important areas of functioning.

B. Each of the following criteria must have been met, with individual symptoms occurring at any time during the course of the disturbance:

(1) *four pain symptoms:* a history of pain related to at least four different sites or functions (e.g., head, abdomen, back, joints, extremities, chest, rectum, during menstruation, during sexual intercourse, or during urination)

(2) *two gastrointestinal symptoms:* a history of at least two gastrointestinal symptoms other than pain (e.g., nausea, bloating, vomiting other than during pregnancy, diarrhea, or intolerance of several different foods)

(3) *one sexual symptom:* a history of at least one sexual or reproductive symptom other than pain (e.g., sexual indifference, erectile or ejaculatory dysfunction, irregular menses, excessive menstrual bleeding, vomiting throughout pregnancy)

(4) *one pseudoneurological symptom:* a history of at least one symptom or deficit suggesting a neurological condition not limited to pain (conversion symptoms such as impaired coordination or balance, paralysis or localized weakness, difficulty swallowing or lump in throat, aphonia, urinary retention, hallucinations, loss of touch or pain sensation, double vision, blindness, deafness, seizures; dissociative symptoms such as amnesia; or loss of consciousness other than fainting)

Suggestiblility

Nature Neuroscience **7**, 587 - 588 (2004) **Placebo-responsive Parkinson patients show decreased activity in single neurons of subthalamic nucleus**F Benedetti, L Colloca, E Torre, M Lanotte, A Melcarne, M Pesare , B Bergamasco, & Leonardo Lopiano

«Traité de psychiatrie» de Henri BARUK, Evolut. Psychiat, XXIV, 3, pp. 457-469.

Hysteria in von Economo encefalitisand Parkinsons's Disease

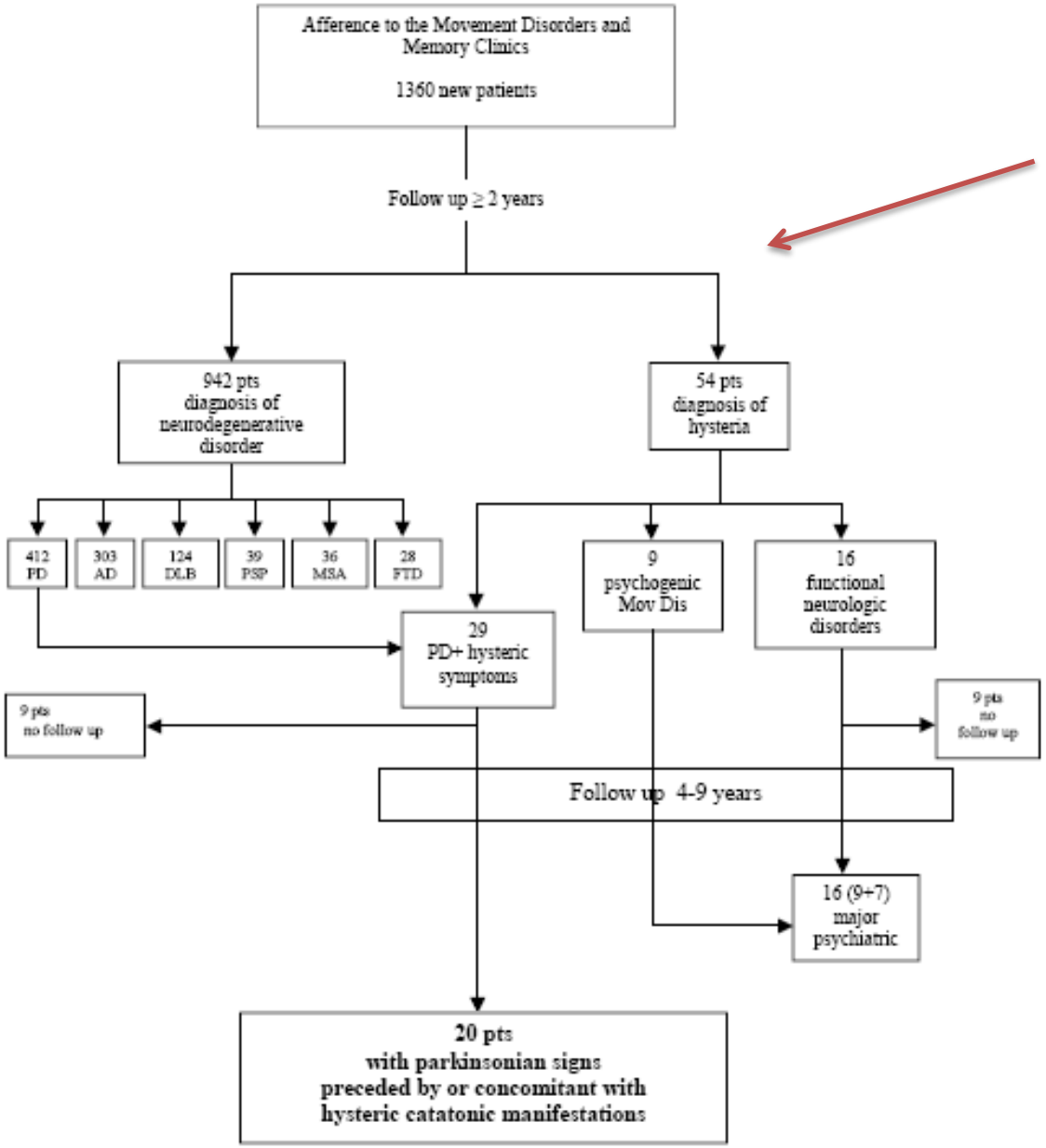
What are somatoform disorders?

1. Somatization disorder: Hysteria- Briquet syndrome 0,2-2%
2. undifferentiated somatoform disorder ?
3. Conversion disorder 3%
4. Hypocondria 3-13%

but also further differentiated somatoform disorders:

- complaint of pain in depressive episodes ?
- body dysmorfic disorder
- Somatic type delusion disorder

Study Design

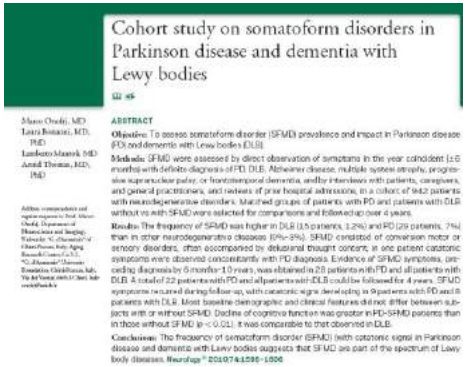


A Study Design

Selection Method:

1. Direct observation of SFMD

2. DSM IV R - semistructured interview
3. hospital charts
4. GP
5. SCL-90R
6. Unsuccessfull attempt : MMPI



Cohort study on somatoform disorders in Parkinson disease and dementia with Lewy bodies

ABSTRACT


DAD, WHAT'S A CONTROL FREAK?
THAT'S WHAT LAZY, SLIPSHOD, CARELESS, CUT-CORNER WORKERS CALL ANYONE WHO CARES ENOUGH TO DO SOMETHING RIGHT.
AM I IN THE PRESENCE OF THEIR KING? SHOULD I KNEEL?
IF ANYTHING WORKS IN THIS WORLD, IT'S BECAUSE ONE OF US TOOK CHARGE

Table 1 Comparison of the prevalence of somatoform disorders and catatonia in the PD-SFMD, and patients with DLB, AD, PD, PSP, MSA, FTD

Disease groups N of patients	DLB 124	AD 303	PD 412	PSP 40	MSA 36	FTD 28
Age	70.2 (8.3)	72.0 (10.6)	63.9 (12.5)	65.1 (2.1)	64.8 (3.2)	66.0 (3.0)
MMSE	22.8 (2.5)	23.5 (3.0)	27.2 (1.6)	25.2 (1.3)	26.6 (1.4)	22.0 (1.5)
FAB	14.6 (2.5)	15.5 (2.8)	17.0 (3.0)	11.0 (1.3)	16.8 (1.2)	11.7 (1.8)
NPI	20.5 (8.2)	11.8 (5.6)	5.0 (3.8)	6.1 (4.4)	5.5 (2.7)	14.4 (5.4)
Presence of SFMD n (%)	15 (12.0)	3 (1.0)	29 (7.0)	1 (2.5)	0	0
SFMD interview positive n (%)	28 (22)	7 (2.3)	40 (9.7)	1 (2.5)	0	0
Number of hospitalisations	4.0 (2.6)	3.8 (2.1)	1.8 (1.6)	4.5 (2.1)	4.2 (3.0)	3.2 (2.2)
(Number of hospital discharges with definite SFMD diagnosis /number of patients)	32/16	8/4	36/29	6/1	0/0	0/0

Table 3 Demographic, clinical, neuropsychological, and neuroimaging data at admission to the study. Comparisons of SFMD patients with age and disease duration matched PD and DLB patients

Variables	PD-SFMD (n = 29)	PD (n = 87)	DLB-SFMD (n = 15)	DLB (n = 45)
Age, years	70.6 (5.5)	70.1 (5.3)	72.0 (3.4)	73.5 (4.7)
Male gender	48%	51%	60%	52%
Educational level, years	12.9 (6.8)	12.3 (3.4)	12.1 (3.0)	12.2 (4.0)
University education % of patients	68%	29%	32%	28%
Mini Mental State Examination (MMSE)	27.1 (1.3)	27.2 (1.7)	23.3 (1.3)	23.1 (1.2)
Neuropsychiatry Inventory (NPI)	9.3 (1.9)	3.7 (2.7)	11.1 (2.2)	11.2 (3.5)
Frontal Assessment Battery (FAB)	16.8 (1.2)	17.3 (1.0)	14.5 (1.2)	14.5 (1.8)
Clinician Assessment of Fluctuation (CAF)	0.0 (0.0)	0.0 (0.0)	4.7 (1.9)	4.6 (2.3)
REM sleep Behavior Disorders (RBD)	38%	24%	60%	67%
Visual Hallucinations (VH)	0%	0%	13%	20%
Somatization subscale of the Symptom Checklist 90R (SCL-90R-SS)	32.2 (8.8)	22.3 (8.0)	22.0 (9.6)	22.5 (8.0)
Beck Depression Inventory (BDI)	29.5 (8.7)	29.6 (7.6)	28.9 (5.5)	28.7 (7.0)
Unified Parkinson's Disease Rating Scale-subscale III (UPDRS-III)				
B.t.	10.2 (2.9)	10.7 (2.9)	8.4 (2.6)	8.6 (2.2)
A.t.	6.0 (2.5)	7.1 (3.4)	6.0 (2.1)	5.8 (2.4)
Hoehn/Yahr Staging (H/Y)	1.2 (0.3)	1.1 (0.2)	1.8 (0.4)	1.8 (0.3)
Total Equivalent L-Dopa dose	424 (44)	419 (50)	106.7 (40.6)	111.5 (22.0)
SPECT DAT Scan				
M	45%	67%	40%	29%
B	55%	33%	60%	71%

Table 2 SFMD phenomenology in PD[a]

	No. of patients		Coincident with diagnosis		In follow-up		Prior to diagnosis	
SFMD symptoms	PD	DLB	PD	DLB	PD	DLB	PD	DLB
Total	29	15	29	15	22	15	29	15
Hypochondriasis, %	90	87	90	87	90	87	90	87
Motor conversion, %								
Paresis	76	60	28	33	48	7	31	40
Abnormal postures	38	47	24	20	21	27	0	7
Globus pharyngis	52	47	0	7	52	47	7	7
Psychogenic parkinsonism	3	0	0	0	0	0	3	0
Catatonic signs	31	53	3	7	31	53	0	13
Sensory conversion, %								
Anesthesia	17	53	10	7	7	27	7	27
Body deformation delusions	45	20	17	7	10	7	42	7
Multilocalized pain with gastrointestinal symptoms	100	73	17	27	69	27	93	53

Abbreviations: DLB = dementia with Lewy bodies; PD = Parkinson disease; SFMD = somatoform disorder.

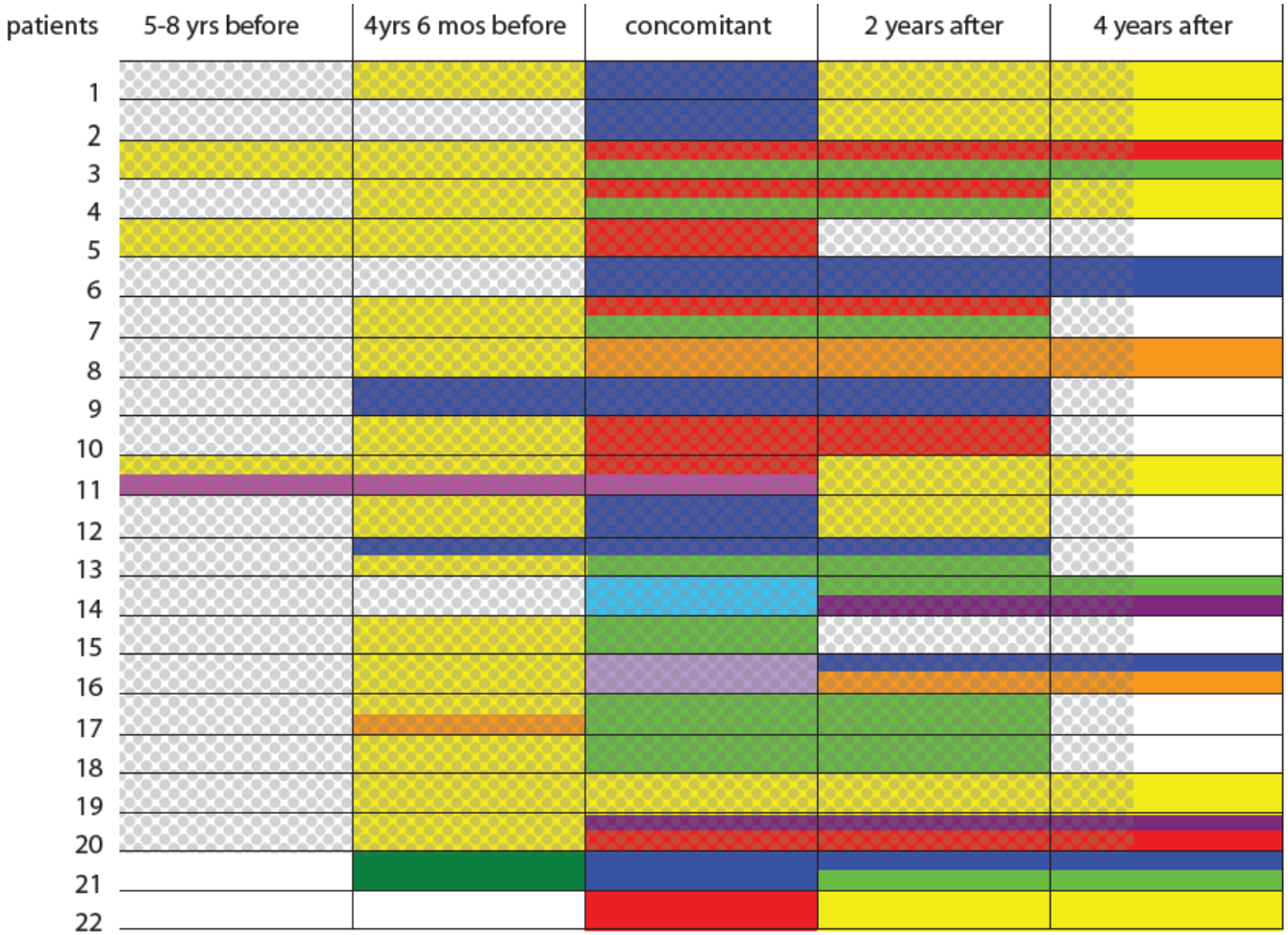
[a] Further details of conversion symptoms are reported in Results. Prevalences of SFMD symptoms in PD are calculated based on the number of patients with PD at onset of the comparative studies. Note that catatonic signs were reported in 2 patients before DLB diagnosis.

What are somatoform disorders?

Somatization disorder: Hysteria- Briquet syndrome 0,2-2%undifferentiated somatoform disorder ?Conversion disorder 3%Hypocondria 3-13%

but also further differentiated somatoform disorders:

- complaint of pain in depressive episodes ?
- body dysmorfic disorder
- Somatic type delusion disorder



KoroXyphoidEvacuation ritualEkbohm syndrom

Hypochondria

Pseudo Motor Pattern

- Unilateral or bilateral bent knee and tiptoeing posture
- Overt catatonia
- Hemiparesi-Paraparesis
- Psychogenic bizarre parkinsonism preceding the onset of PD

Non Motor Pattern

- Delirium of penis shrinkage
- Delusion of ingrowths of the xyphoid process
- Delusion of bowl deformation
- Delusion of migrating cutaneous allergy (Ekbom)

Body Dysmorphogenesis
Sensory delusions

- Multilocalized recurrent pain responding to unorthodox manupilations
- Hemianesthesia or partial anaesthesia

Motor conversion disorders

HYSTERIC GAIT YOPD

Bent Knees/tip toeing PDD

Burper DLB

Dancing Gait

Pseudo paralisis

Silly walk

Catatonia DLB

Non Motor Somatic Pattern

KoroXyphoidEvacuation ritualEkbohm syndrom

-Viscosity-Mannerism-verbal aggressiveness- paranoid delusion-Psychogenic tremorRestlessnessVerbosity treatment refusal psychological Romberg knee buckling Globus pharyngis

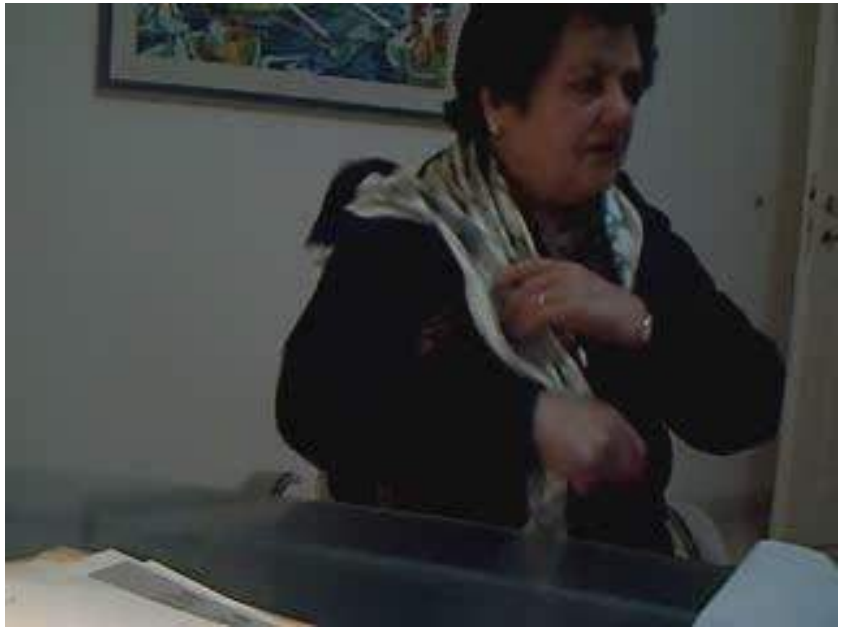


Table 4 Demographic, clinical, neuropsychological data at admission to the study and after 2 and 4 years of follow-up in SFMD patients and patients with PD or DLB matched for education and MMSE score at baseline

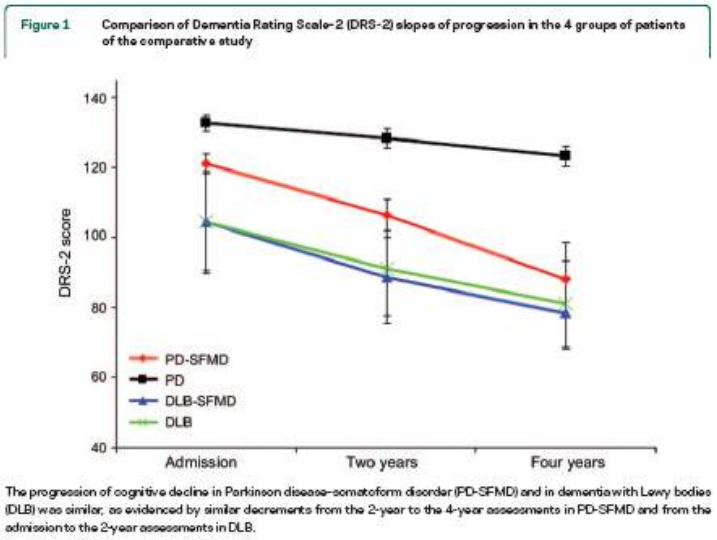
Variables	Phase	PD-SFMD (n=22)	PD (n=66)	DLB-SFMD (n=15)	DLB (n=30)
Age, years	Admission	70.7 (6.1)	70.0 (5.2)	72.0 (3.4)	73.2 (4.6)
Male gender	—	50%	50%	63%	43%
Mini Mental State Examination (MMSE)	Admission	26.8 (1.2)	27.2 (1.6)	23.3 (1.3)	23.0 (1.2)
	2 years	23.9 (1.5)	26.8 (1.5)	17.5 (1.4)	17.3 (2.2)
	4 years	17.9 (1.9)	26.4 (1.4)	12.1 (1.5)	12.0 (2.4)
Dementia Rating Scale-2 (DRS-2)	Admission	121.2 (2.8)	132.7 (2.8)	104.4 (13.9)	104.3 (14.7)
	2 years	106.4 (4.5)	128.3 (3.0)	88.7 (11.1)	88.6 (15.3)
	4 years	88.1 (10.5)	123.2 (3.2)	78.3 (10.2)	78.4 (12.4)
Neuropsychiatry Inventory (NPI)	Admission	9.2 (2.0)	3.6 (2.7)	11.1 (2.2)	11.3 (3.4)
	2 years	13.6 (1.8)	4.0 (1.9)	19.7 (1.7)	18.9 (4.9)
	4 years	19.8 (3.5)	4.7 (1.9)	27.2 (3.0)	26.7 (3.6)
Frontal Assessment Battery (FAB)	Admission	16.5 (0.4)	17.4 (0.9)	14.5 (1.2)	14.5 (1.9)
	2 years	14.4 (0.6)	16.6 (1.4)	11.5 (1.1)	11.6 (2.1)
	4 years	11.4 (0.6)	14.9 (2.1)	9.9 (1.0)	9.8 (1.8)
Clinician Assessment of Fluctuation (CAF)	Admission	0.0 (0.0)	0.0 (0.0)	4.7 (1.9)	4.6 (2.2)
	2 years	0.2 (0.4)	0.1 (0.2)	5.9 (1.2)	6.2 (2.2)
	4 years	1.0 (1.2)	0.2 (0.6)	7.1 (1.6)	7.2 (2.4)
REM sleep Behavior Disorders (RBD)	Admission	41%	23%	60%	67%
	2 years	68%	42%	80%	87%
	4 years	86%	62%	87%	90%
Visual Hallucinations (VH)	Admission	0%	0%	13%	20%
	2 years	9%	2%	40%	43%
	4 years	59%	3%	93%	93%
Unified Parkinson's Disease Rating Scale-subscale III (UPDRS-III)	Admission	10.4 (3.1)	10.8 (2.9)	8.4 (2.6)	8.6 (2.1)
	2 years	22.8 (3.2)	23.6 (3.1)	10.9 (2.5)	11.1 (2.4)
	4 years	35.4 (3.6)	34.7 (3.8)	13.9 (2.6)	13.7 (2.9)
Hoehn/Yahr Staging (H/Y)	Admission	1.2 (0.3)	1.1 (0.2)	1.8 (0.4)	1.8 (0.4)
	2 years	1.8 (0.3)	1.8 (0.3)	1.9 (0.3)	2.1 (0.2)
	4 years	2.2 (0.4)	2.1 (0.3)	2.0 (0.1)	2.4 (0.3)
Equivalent L-Dopa dose (mg/day)	Admission	390 (80)	407 (160)	106 (42)	113 (37)
	2 years	578 (50)	604 (62)	253 (62)	286 (58)
	4 years	780 (140)	857 (104)	310 (58)	336 (50)

< MMSE

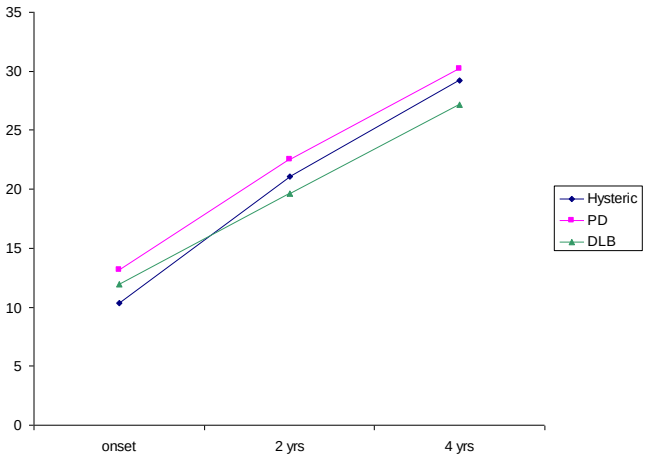
< DRS-2

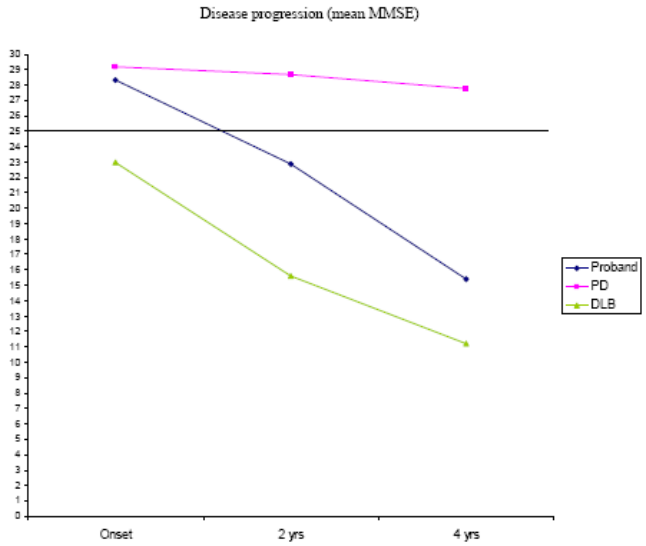
> RBD

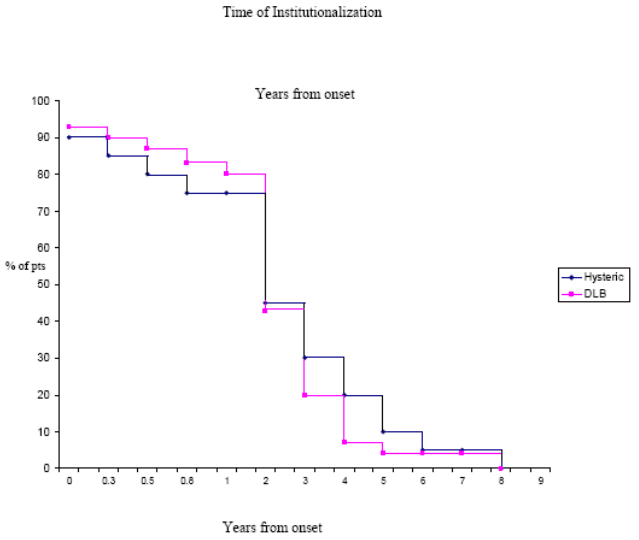
> VH



PD motor signs progression (mean UPDRS)







Conclusion:

Somatoform disorders are observed in a high percentage of PD patients: 4-7% Somatoform disorders are rare in all other diseases but LBD where prevalence is 12%the quality of symptoms is outstanding look for clinical patterns and the combination with catatonia

Somatoform disorders including somatic delusions and catatonic manifestations should be listed among psychotic disorders of LBD

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journal homepage: www.elsevier.com/locate/jns

Updates on Somatoform Disorders (SFMD) in Parkinson's Disease and Dementia with Lewy Bodies and discussion of phenomenology

Marco Onofrj [a,*], Astrid Thomas [a], Pietro Tiraboschi [b], Gregor Wenning [c], Francesco Gambi [d], Gianna Sepede [d], Massimo Di Giannantonio [d], Caterina Di Carmine [a], Daniela Monaco [a], Valerio Maruotti [a], Fausta Ciccocioppo [a], Maria Chiara D'Amico [a], Laura Bonanni [a]

Ebmeier KP, O'Brien J, Taylor J-P (eds): Psychiatry of Parkinson's Disease.
Adv Biol Psychiatry. Basel, Karger, 2012, vol 27, pp 125–132

Somatoform Disorders in Parkinson's Disease and Dementia with Lewy Bodies Evidence Underlying Psychotic Traits

Marco Onofrj[a] · Astrid Thomas[a] · Laura Bonanni[a] · Massimo di Giannantonio[b] · Francesco Gambi[b] · Gianna Sepede[b]

[a]Neurology Clinics and [b]Psychiatry Clinics, Department of Neuroscience and Imaging and Aging Research Center, Ce.S.I., 'Gabriele d'Annunzio' University Foundation, University G. d'Annunzio of Chieti-Pescara, Chieti, Italy

VIEWPOINT

Functional (Psychogenic) Symptoms in Parkinson's Disease

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Psychogenic Parkinsonism

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Headache
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Expert Opinion

Functional/Psychogenic Neurological Symptoms and Headache

Jon Stone, MB, ChB, FRCP, PhD; Randolph W. Evans, MD

Key words: functional/psychogenic neurological symptom, headache attributed to somatization disorder, conversion disorder

(*Headache* 2011;51:781-788)

Neurol (2012) 259:124–131
OI 10.1007/s00415-011-6140-8

ORIGINAL COMMUNICATION

'sychiatric symptoms screening in the early stages of Parkinson's lisease

aulo Bugalho · Joaquim Alves da Silva ·
ıês Cargaleiro · Madalena Serra · Bernardo Neto

SPECIAL ARTICLE

ONLINE FIRST

The Evolution of Academic Neurology

New Information Will Bring New Meaning

William Mobley, MD, PhD; Roger N. Rosenberg, MD

LETTERS: PUBLISHED ARTICLE

Functional or Somatoform Disorders in Parkinson's Disease?

I read with interest the paper by Pareés et al. entitled "Functional (psychogenic) symptoms in Parkinson's disease."[1] "Functional" is an old term that is now revisited.[2] We have previously labeled the same disorders as Somatoform Disorders following the recommendation of editors in earlier publications.[3-5] Whether this term will be accepted for use again is open to question. I would like to call to attention the authors' comments regarding our publications suggesting that "some of the symptoms .... were non-motor symptoms of PD." This was not the case. The symptoms we described were not part of the non-motor manifestations of Parkinson's disease (PD), as clearly illustrated by the single case description published previously[6] in website attachments to the paper. Nor do I agree with their comments[1] suggesting that our studies "did not compare" PD patients with "other neurologic disorders." This is not accurate as the patient population described included those with dementia and Lewy bodies, Progressive Supranuclear Palsy, Multiple System Atrophy, Fronto-Temporal Dementia and Alzheimer's disease in addition to patients with PD.[7] On the other hand, I do concur that the suggested explanation for these disorders based on the Bayesian model of hierarchical organization of perceptions — motor outputs[8-9] is promising and that future functional MRIstudies in such patients might enlighten our understanding and approach to these fascinating conditions.

Marco Onofrj

Neurology, Dept. Neuroscience and Imaging; Aging Research Center, CeSI, University Foundation, University of Chieti-Pescara, Chieti, Italy

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1. Pareés I, Saifee TA, Kojovic M, et al. Functional (psychogenic) symptoms in Parkinson's disease. Mov Disord 2011;26:2322–2327.
2. Onofrj M, Bonanni L, Manzoli L, Thomas A. Cohort Study on somatoform disorders in Parkinson disease and dementia with Lewy bodies. Neurology 2010;74:1598–1606.
3. Onofrj M, Thomas A, Tiacchella F, et al. Updates on somatoform disorders (SMD) in Parkinson Disease and Dementia with Lewy bodies and discussion on phenomenology. J Neurol Sci 2011;310:166–171.
4. Edwards MJ, Adams RA, Brown H, et al. A Bayesian account of "Hysteria". Brain 2012;135:3495–3516.

Received: 2 July 2012; Revised: 16 August 2012; Accepted: 8 September 2012
Published online 15 October 2012 in Wiley Online Library (wileyonlinelibrary.com). DOI: 10.1002/mds.25714

Reply:

We thank Professor Onofrj for his supportive comments[1] on our manuscript.[2] We are pleased to hear that the Bayesian model of the brain to explain functional symptoms is found promising. We appreciate that his studies compared Parkinson's disease with other movement disorders (atypical parkinsonism) and indeed with other neurological disorders such as Alzheimer's disease.[3-4] Following the recommendation of our reviewer, we emphasised the difficulties in confidently classifying sensory symptoms as functional and specifically differentiating them from nonmotor symptoms in Parkinson's disease. However, after a closer review of the supporting data from their article[3] we agree that sensory symptoms displayed by some patients reported fit better with functional symptoms rather than with sensory symptoms that can be found within the spectrum of nonmotor symptoms of Parkinson's disease. Nevertheless, although functional overlap in Parkinson's disease can commonly be seen in clinical practice, further research is still required to confirm this potential vulnerability and understand the underlying pathophysiological mechanism.

Isabel Pareés, MD; Tabish A. Saifee, MRCP, and Mark J. Edwards, MD, PhD

Sobell Department of Motor Neuroscience and Movement Disorders, UCL Institute of Neurology, Queen Square, London, UK

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1. Onofrj M. Functional motor or somatoform disorders in PD? Mov Disord 2011;26:1997.
2. Pareés I, Saifee TA, Kojovic M, et al. Functional (psychogenic) symptoms in Parkinson's disease. Mov Disord 2011;26:2322–2327.
3. Onofrj M, Bonanni L, Manzoli L, Thomas A. Cohort study on somatoform disorders in Parkinson disease and dementia with Lewy bodies. Neurology 2010;74:1598–1606.
4. Onofrj M, Thomas A, Tiacchella F, et al. Updates on Somatoform Disorders (SMD) in Parkinson's Disease and Dementia with Lewy Bodies and discussion of phenomenology. J Neurol Sci 2011;310:166–171.

Received: 23 August 2012; Accepted: 8 September 2012
Published online 15 October 2012 in Wiley Online Library (wileyonlinelibrary.com). DOI: 10.1002/mds.25712

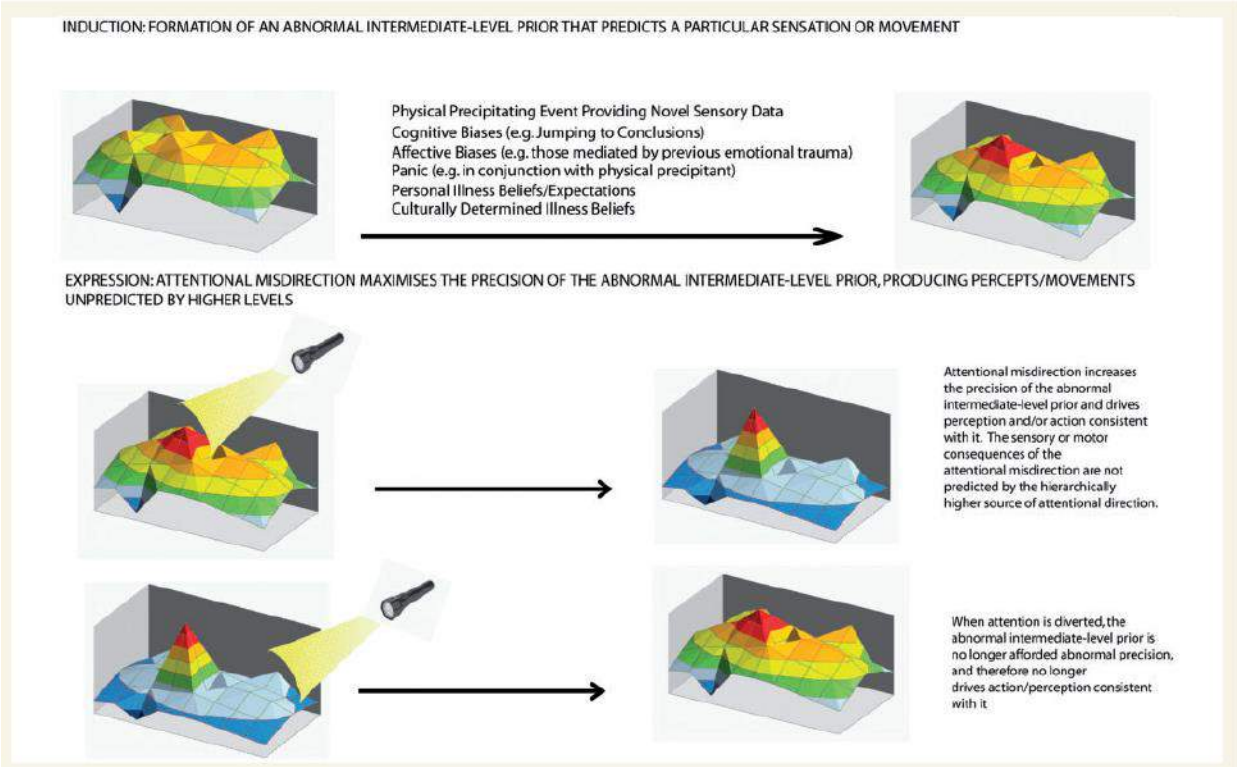
doi:10.1093/brain/aws129 Brain 2012: 135; 3495–3512 | 3495

BRAIN
A JOURNAL OF NEUROLOGY

OCCASIONAL PAPER

A Bayesian account of 'hysteria'

Mark J. Edwards,[1,*] Rick A. Adams,[2,*] Harriet Brown,[2] Isabel Pareés[1] and Karl J. Friston[2]



Why catatonia is in a continuum with hysteria ?

Thanatosis

Sham- Death reflex or playing possum

Merskey H. Conversion, dissociation, or doxomorphic disorder. In: Halligan W, Bass C, Marshall J. Contemporary Approaches to the Study of Hysteria: Clinical and Theoretical Perspectives. Oxford, UK: Oxford University Press; 2001:171–183.

Shorter E. Hysteria and catatonia as motor disorders in historical context. Hist Psychiatry 2006;17:461–468.

An injured Virginia Opossum lies incapacitated in front a dog.

Continuum

HysteriaCatatoniaGanser SyndromeCotard Syndrome

Allucinazioni visive

marco onofrj

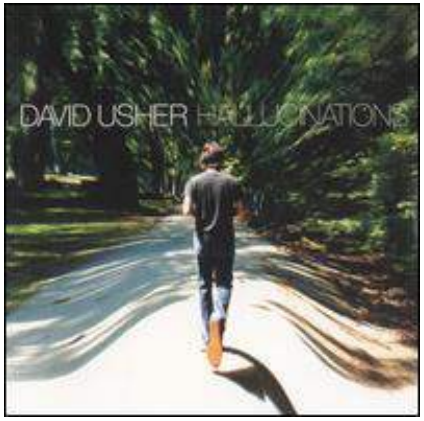
Department of Oncology and Neuroscience, Neurophysiopathology, University "G.D'Annunzio" Chieti-Pescara-Italy, Aging Research Center Ce.S.I., " G. D'Annunzio" University Foundation, Chieti-Pescara, Italy

Table 1
Definitions and glossary of VH and other visual phenomena

Visual phenomenon	Definition
Illusions	Misinterpretation of images.
Pareidolias	Complex visual illusions involving ambiguous forms that are perceived as meaningful object as faces-people.
Visual hallucinations	Perceptions of images not present in the visual field i.e. images in the absence of external sensory stimulus. Thus VH are considered a "unitary pathological symptom distinct from illusion".
Phosphenes	Unstructured lights such as flashes, sparkles, colored dots, zig-zag lines or rainbows, black and white or colored, static or moving.
Photopsias	Geometric elementary structured images, often recurring in a repetitive form.
Visual distortions	Illusions, as the perceived image consists of the distortion of an image which is present or was present in the visual field of the subject.
Visual allesthesias	Condition in which visual images are transposed from one half of the visual field to the other, either vertically or horizontally.
Micropsia	Reduced size of the object.
Macropsia	Enlarged object.
Pelopsia	The object appears closer than actual.
Teleopsia or Telopsia	The object appears farther than actual.
Metamorphopsia	The distortion of objects or figures, like enlargement of particulars, e.g. elongated necks, fanglike dentures.
Kinetopia	The illusion of movement of a static object.
Palinopsia	The visual perseveration or recurrent appearance of a visual image after the stimulus has disappeared.
Poliopia	The multiplication of the visual image in the visual field.
Tassellopsies (or Teicopsies)	Hallucinations consisting of the perception of brick-like textures in the visual field and include fortification spectra and heat waves appearing in migraine.
Dendropsies	Hallucinations of tree branches (dendron).
Hypnagogic hallucinations	Appearing when falling asleep.
Hypnopompic hallucinations	Appearing when waking from sleep.
Simple hallucinations	Phenomena like photopsia or perception of static images.
Complex hallucinations	Kinetic/kinematic with preserved or disturbed insight.
Extracampine hallucinations	Sensation of presence of somebody/something (e.g. guardian angel) at the border or external to the visual field.
Serotine misinterpretations	These terms refer to occurrence of illusions (i.e. moving leaves interpreted as people) in the late afternoon / early evening (serotine). This disorder is mostly described in late AD and is an essential part of the "sundowning" phenomenon and is also defined as Pareidolia.
Movement hallucinations	Sensation of passage (brief vision of persons or animals passing on the sides of the visual field).
Minor forms of VH	Include: presence of extracampine or presence hallucinations; passage hallucinations; or as sometimes described (although phenomenological incorrect) illusions.
Formed hallucinations	Formed hallucinations with various contents (persons, animals, objects, interacting with each other and with the patient in complex scenes).
Blurred or formed	Blurred hallucinations are described as indefinite or not fully formed images, like presence and passage VH. Formed images can be inanimate or animate figures, and can be static or moving in complex interactions.
Moving images/Kinetic and kinematic (movie like)	Can be simple or minor, blurred, complex or formed.
Simple or complex	The classification in simple/complex VH can be confounding because the terms are sometimes used to describe VH characterized by preserved vs. disturbed insight.
Benign or malignant	VH can be described as benign or malignant. The terms have been applied in the characterization of VH in 1) the context of retained insight or disrupted insight 2) the proneness of the disturbance to remain stable or to progress with PD 3) labelling the nature of the disturbance as mild (not bothersome for the patient) or severe (affecting patient quality of life). However, this classification criterion has been challenged because VH severity and frequency tend to progress, and thus "the term benign hallucinations of PD should be considered generally unsound and dropped from operative vocabulary" [45].
Early or late	VH in PD have also been tentatively classified as early, appearing within 5 years from the onset of PD or late. This classification too has been challenged.

Allucinazioni visive

Illusioni



Allucinazioni semplici

- Extracampine
- Animali-oggettti
- Passaggio
- Movimento

formate o sfumate (blurred)

with visual disturbances = Charles-Bonnet Syndrome

G. 1. Stone, coloured photograph.

FIG. 2. Tree bark, coloured photograph.

An Artist's View of Drug-Induced Hallucinosis

Georg Ebersbach, MD*
Movement Disorders, Vol. 18, No. 7, 2003

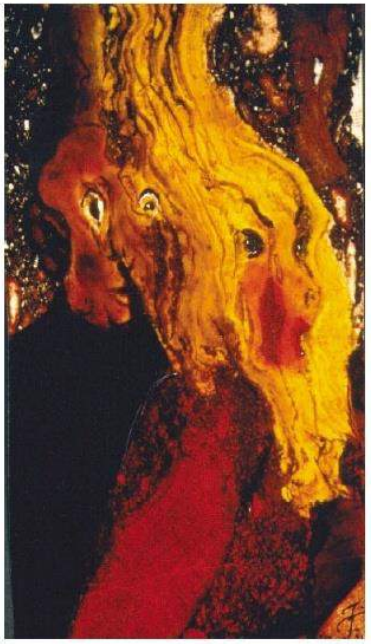
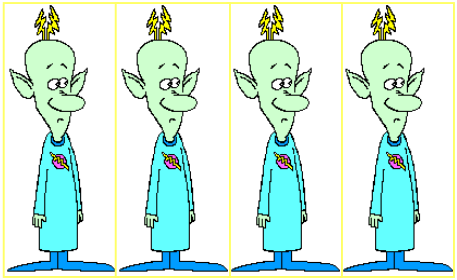


FIG. 3. Marble, coloured photograph.

without visual disturbances = peduncular hallucinosis



Hallucinations

with

low drug levels

Similarity

with

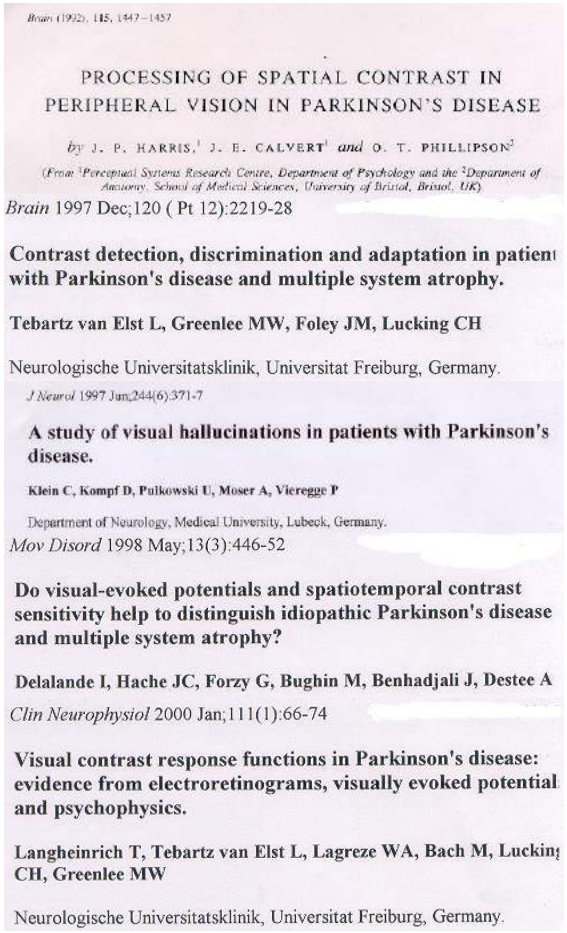
visual dysfunction

in PD

Onofrj M., Bodis-Wollner I. Dopaminergic deficency causes delayed visual evoked potentials in rats. Ann. Neurol., 11: 484-490, 1982.

Bodis-Wollner I, Marx MS, Mitra S, Bobak P, Mylin L, Yahr M. Visual dysfunction in Parkinson's disease. Loss in spatiotemporal contrast sensitivity. Brain. 1987 Dec;110 (Pt 6):1675-98.

Bodis-Wollner I, Onofrj M. Visual system in Parkinson's Disease. In: Advances in Neurology. Eds. Yahr MD, Bergmann KJ. 1986;45:323-327



Brain (1992), 115, 1347–1457

PROCESSING OF SPATIAL CONTRAST IN PERIPHERAL VISION IN PARKINSON'S DISEASE

by J. P. HARRIS,[1] J. E. CALVERT[1] and O. T. PHILLIPSON[2]

(From [1]Perceptual Systems Research Centre, Department of Psychology and the [2]Department of Anatomy, School of Medical Sciences, University of Bristol, Bristol, UK)

Brain 1997 Dec;120 (Pt 12):2219-28

Contrast detection, discrimination and adaptation in patient with Parkinson's disease and multiple system atrophy.

Tebartz van Elst L, Greenlee MW, Foley JM, Lucking CH

Neurologische Universitatsklinik, Universitat Freiburg, Germany.

J Neurol 1997 Jun;244(6):371-7

A study of visual hallucinations in patients with Parkinson's disease.

Klein C, Kompf D, Pulkowski U, Moser A, Vieregge P

Department of Neurology, Medical University, Lubeck, Germany.

Mov Disord 1998 May;13(3):446-52

Do visual-evoked potentials and spatiotemporal contrast sensitivity help to distinguish idiopathic Parkinson's disease and multiple system atrophy?

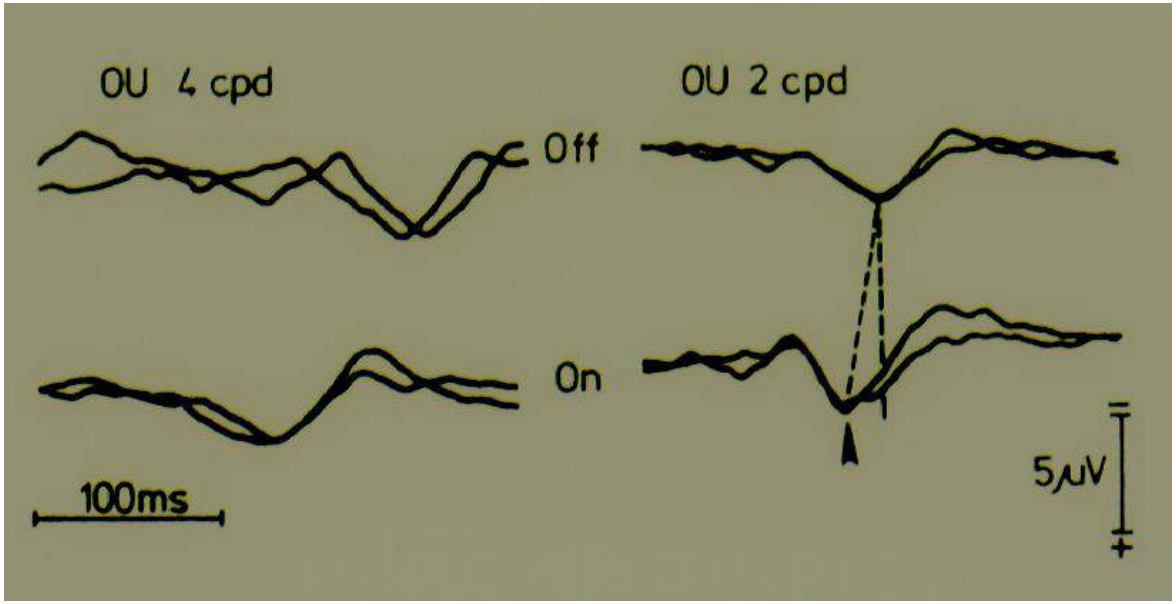
Delalande I, Hache JC, Forzy G, Bughin M, Benhadjali J, Destee A

Clin Neurophysiol 2000 Jan;111(1):66-74

Visual contrast response functions in Parkinson's disease: evidence from electroretinograms, visually evoked potential and psychophysics.

Langheinrich T, Tebartz van Elst L, Lagreze WA, Bach M, Luckin CH, Greenlee MW

Neurologische Universitatsklinik, Universitat Freiburg, Germany.



PD Patients in Virtual reality

1-3h

Drug wash-out
Night-early morning dose

Study 3 hours

Hallucinations/ Illusions	**1st h**	**5%**
	2nd h	**15%**
	3rd h	**40%**

End of dose deterioration

L-Dopa
+
DA dose

60 min from dose	10%
150 min from dose	35%

Movement Disorders
Vol. 20, No. 2, 2005, pp. 130–140
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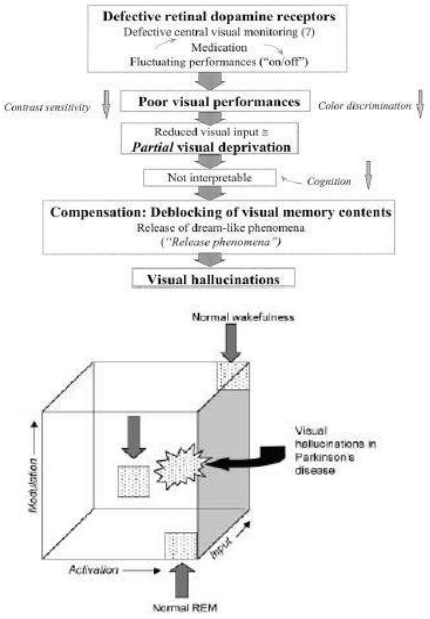
Review

Repeated Visual Hallucinations in Parkinson's Disease as Disturbed External/Internal Perceptions: Focused Review and a New Integrative Model

Nico J. Diederich, MD,[1,2]* Christopher G. Goetz, MD,[2] and Glenn T. Stebbins, PhD[2]



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ELSEVIER

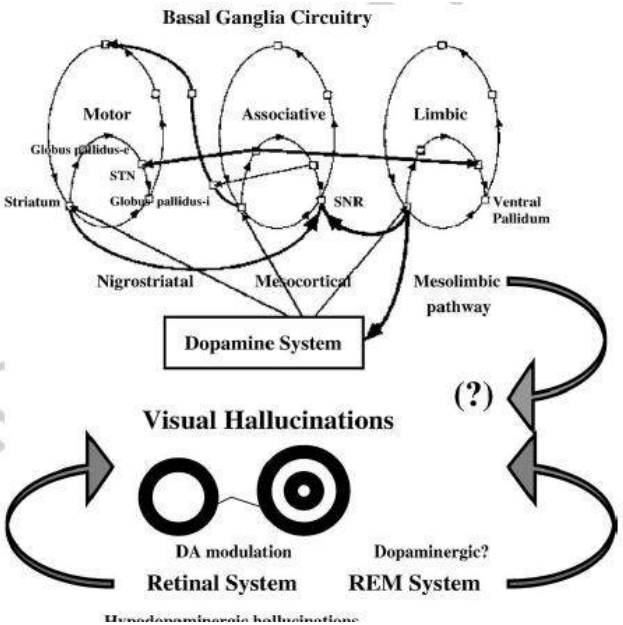
Journal of the Neurological Sciences xx (2006) xxx – xxx

Journal of the Neurological Sciences

www.elsevier.com/locate/jns

Visual hallucinations in Parkinson's disease: Clues to separate origins

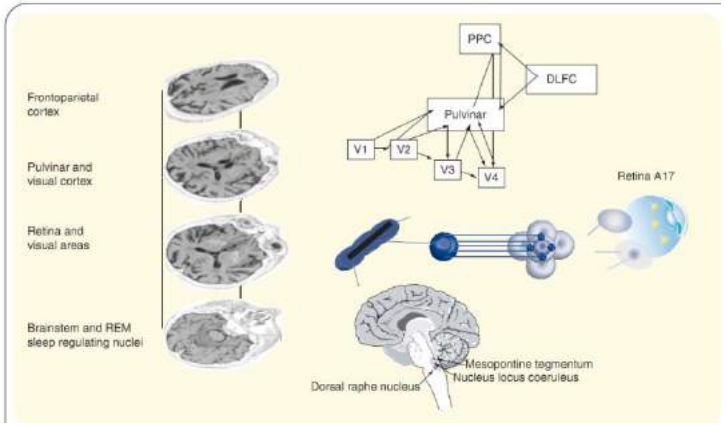
M. Onofrj [a,b,*], L. Bonanni [a,b], G. Albani [c], A. Mauro [c], D. Bulla [d], A. Thomas [a,b]



New approaches to understanding hallucinations in Parkinson's disease: phenomenology and possible origins

Marco Onofrj†*, Astrid Thomas and Laura Bonanni*

Expert Rev. Neurotherapeutics 7(12), 1731–1750 (2007)



Default-mode network

In 2001, the default mode network (DMN) was first proposed as an interconnected set of brain regions that is active when the brain is in a resting state and typically deactivated during memory encoding and other cognitively demanding tasks focused on processing of external stimuli. The DMN includes the dorsal and ventral medial prefrontal cortices, medial and lateral parietal cortex, and parts of the medial and lateral temporal cortices.

Functional network disruption in the degenerative dementias

Michela Pievani, Willem de Haan, Tao Wu, William W Seeley, Giovanni B Frisoni

Lancet Neurol 2011; 10: 829–43

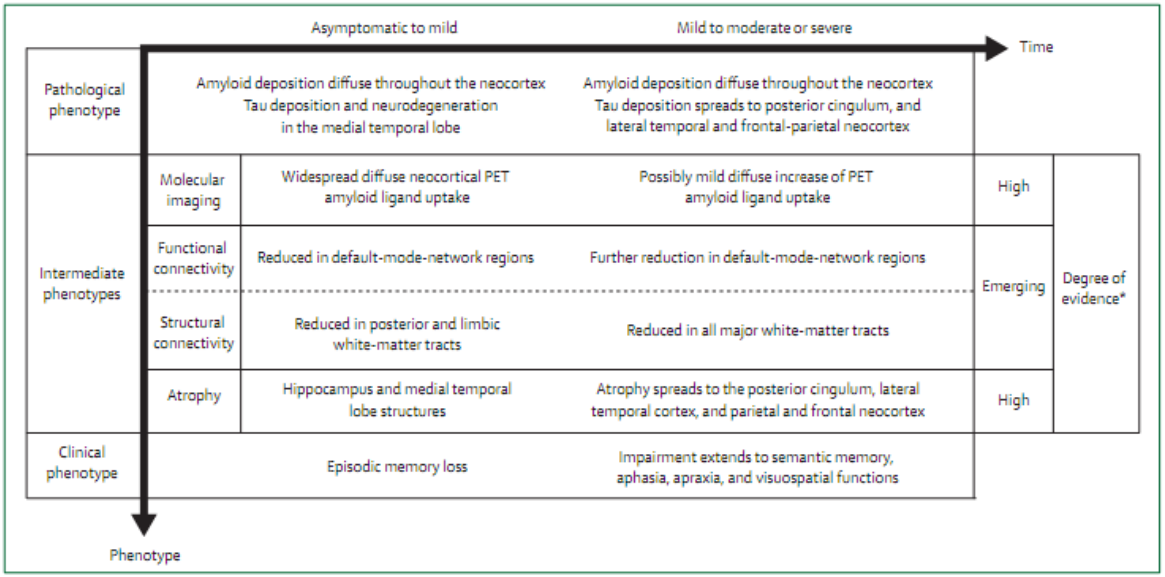


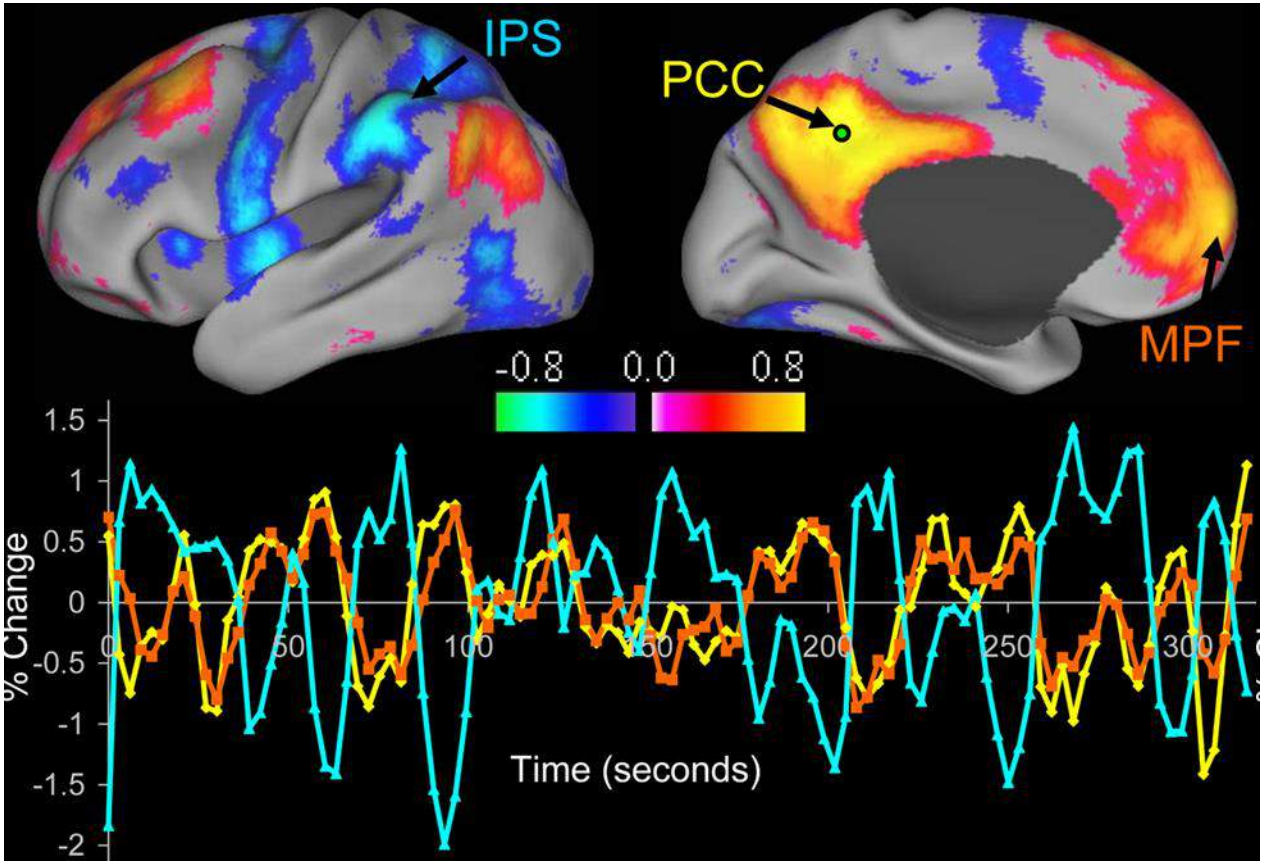
Figure 1: **The pathophysiological framework of Alzheimer's disease: connectivity as an intermediate phenotype between pathology and symptoms**
*Evidence that intermediate phenotypes are associated with pathological or clinical phenotypes.

	Alzheimer's disease	Frontotemporal degeneration (behavioural variant)	Parkinson's disease	Dementia with Lewy bodies
Functional connectivity				
Resting-state functional MRI	Reduced connectivity—default mode network	Reduced connectivity—salience network	Increased connectivity—basal ganglia-thalamocortical loops; normalisation after levodopa administration	Insufficient evidence
Resting-state EEG/MEG	Reduced connectivity—alpha and beta (8–30 Hz) range between long-distance fronto-parietal and fronto-temporal regions	Insufficient evidence	Increased connectivity—alpha and beta (8–30 Hz) range locally and globally	Reduced connectivity—alpha (8–13 Hz) range locally and globally
Structural connectivity (diffusion tensor imaging)	Reduced connectivity—posterior and limbic white-matter tracts	Reduced connectivity—anterior white-matter tracts	No change in the major white-matter tracts	Reduced connectivity—visual pathway
Network organisation	Change towards a different topology—small-world to random; hub vulnerability	Change towards a different topology—small-world to regular	Insufficient evidence	No evidence

EEG=electroencephalography. MEG=magnetoencephalography.

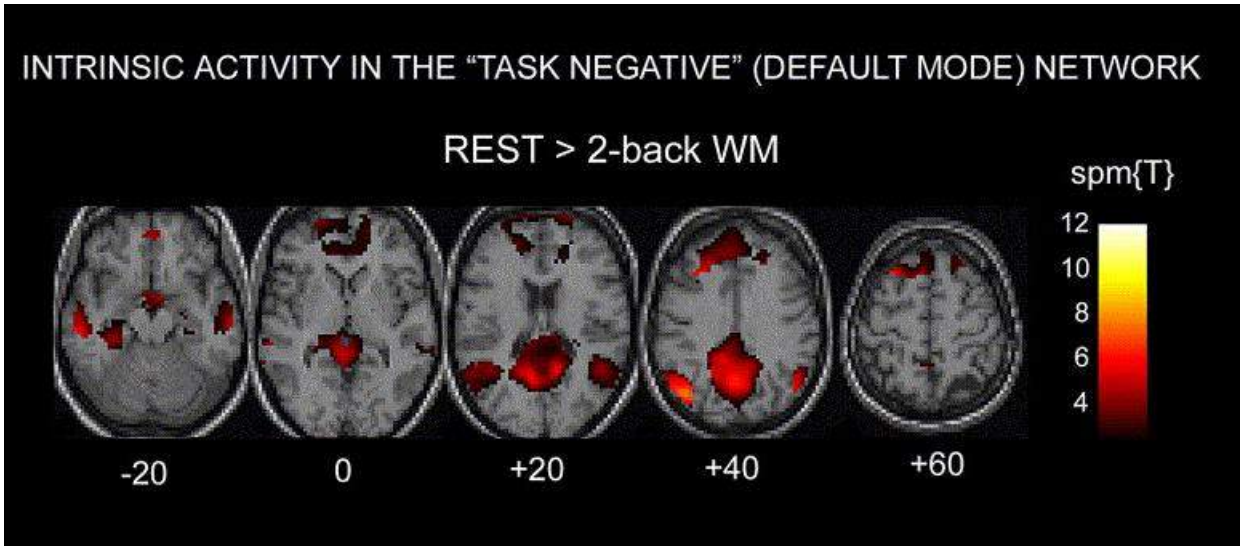
Table 2: **Connectivity disruption in the degenerative dementias**

Resting state functional connectivity reveals correlations (red) and anticorrelations (blu) with the default mode network.



Michael D. Fox and Michael Greicius

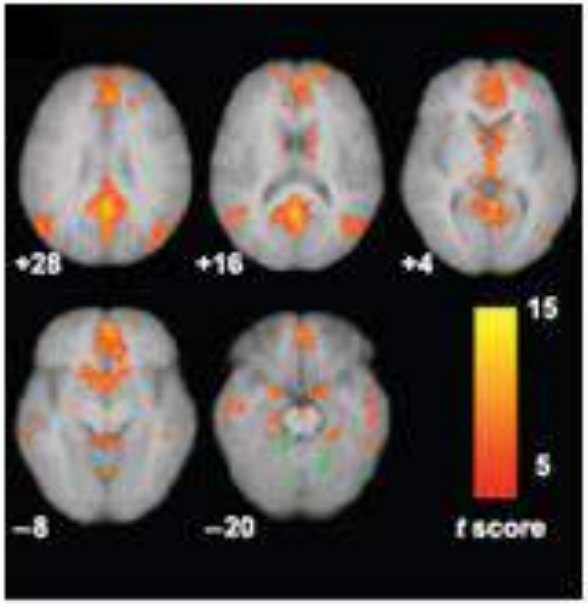
Clinical applications of resting state functional connectivity. Review.



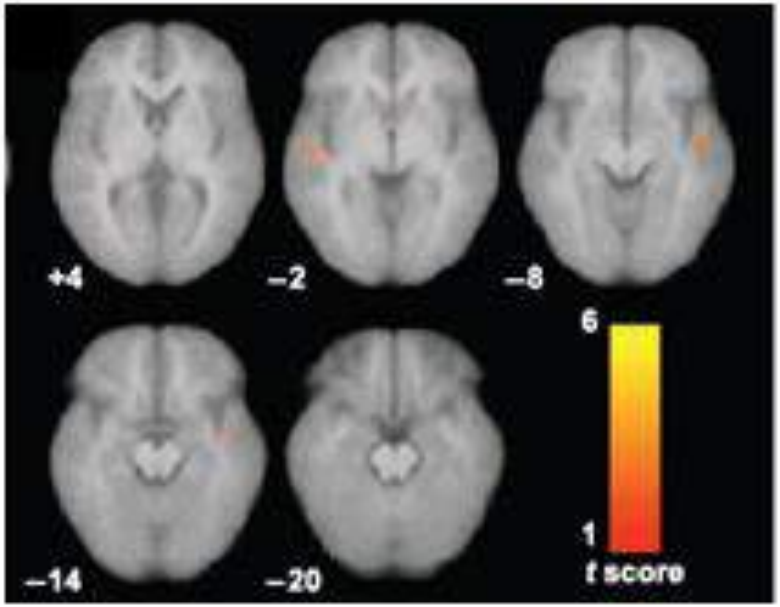
A direct comparison of activity in DMN during rest and sustained performance of the two-back working memory (WM) task showed decrease of intrinsic activity during WM compared to rest in most default-mode regions, including the medial prefrontal cortex, precuneus, posterior cortex cinguli, angular gyrus, parahippocampal gyrus and inferior temporal cortex. No significant increases in intrinsic activity during the WM task compared to rest were observed in the default-mode network.

Fransson, 2006.

Default mode activity during rest.

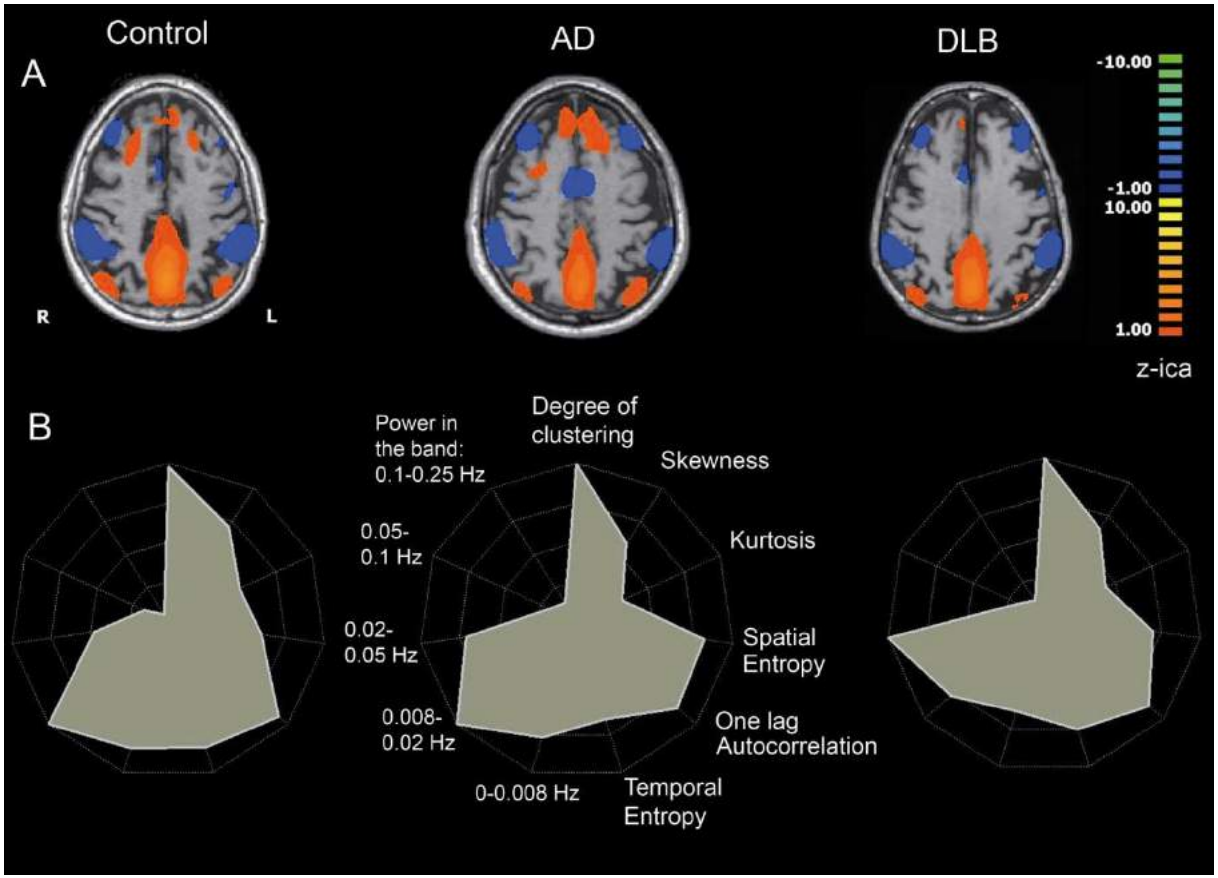


Suppression of DMN activity during a demanding task.



Analysis of DMN activity provides a measure of the degree to which a task engages a subject and whether it is sufficient to interrupt the processes, presumably cognitive, internally generated mediated by the DMN.

Greicius and Menon, 2004



A. Spatial maps of ICA for DMN in the control, AD and DLB group. Yellow-red and green-blue areas indicate positive and negative correlation with the IC waveform.

B. Fingerprints used to exclude ICs derived from artefacts.

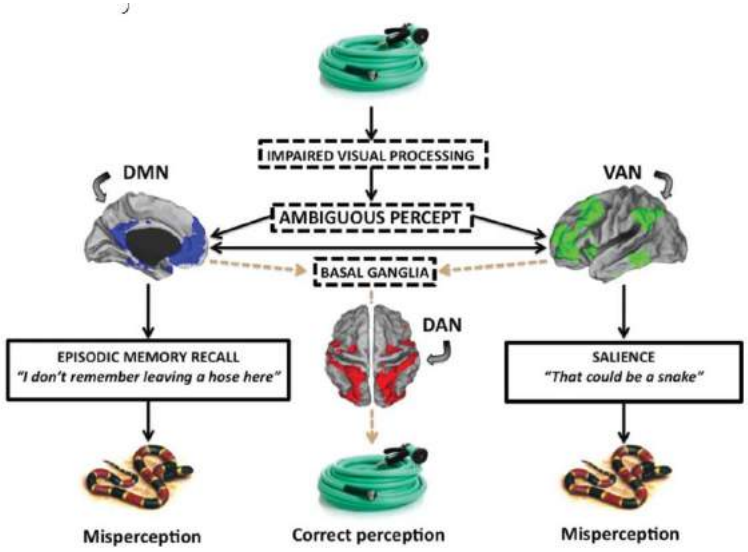
Visual Misperceptions and Hallucinations in Parkinson's Disease: Dysfunction of Attentional Control Networks?

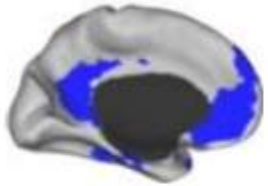
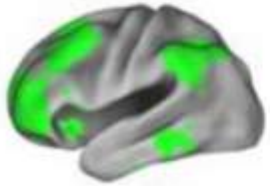
James M. Shine, BSc, MBBS,[1] Glenda M. Halliday, BSc, PhD,[2] Sharon L. Naismith, MAPS, CCN,[1] and Simon J.G. Lewis, MBBCh, BSc, MRCP, FRACP, MD[1]*

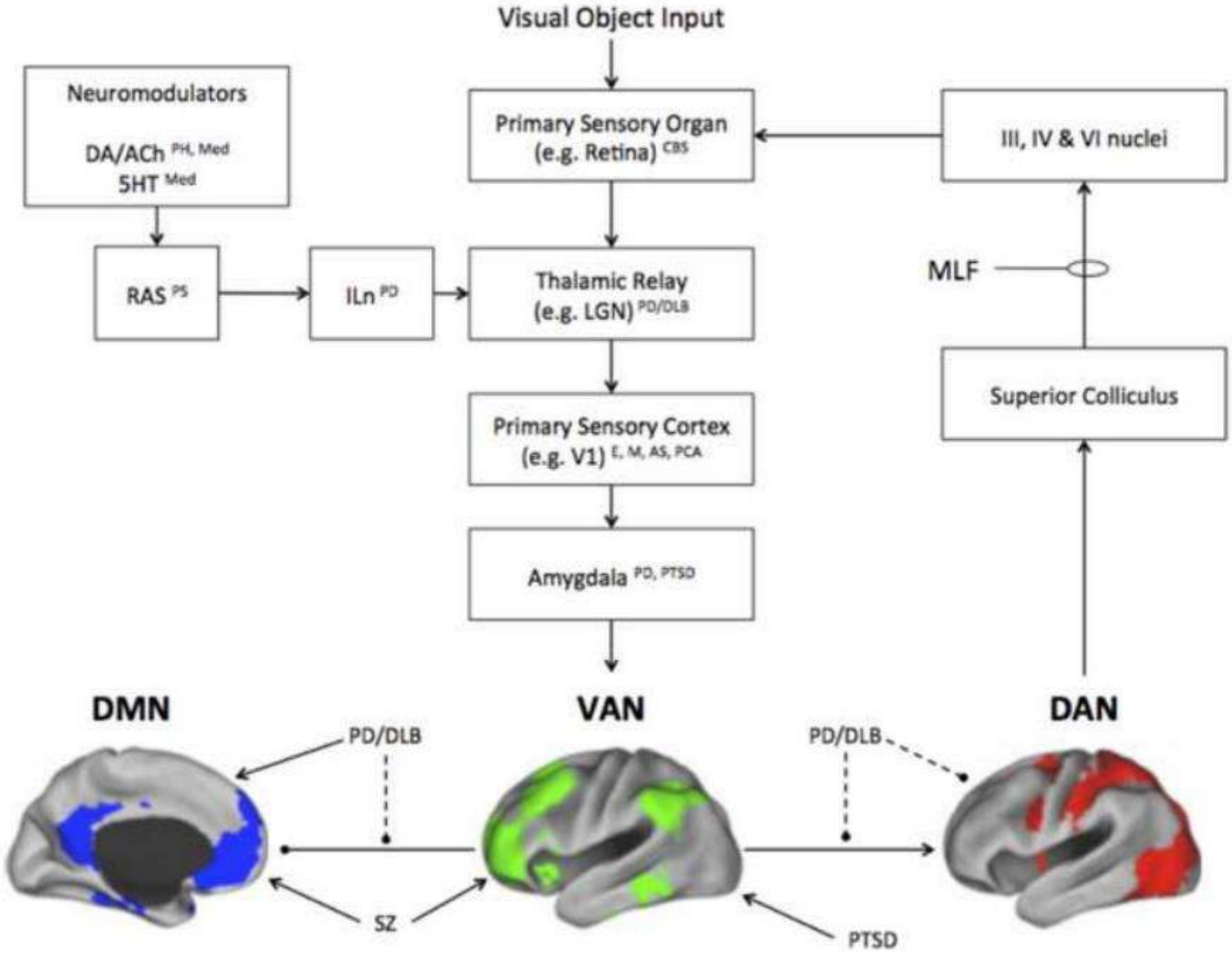
Movement Disorders, Vol. 26, No. 12, 2011

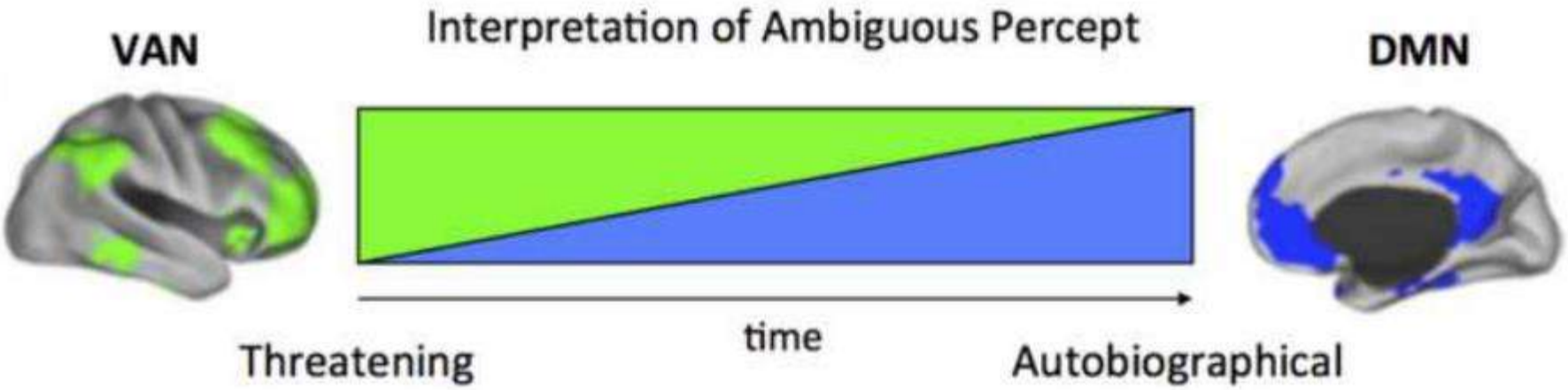
TABLE 1. Supportive evidence for dysfunction in attention control networks in hallucinations and misperceptions

Network	Function	Brain areas involved	Pathology in PD	Neuroimaging findings
Default mode (DMN)	Task-independent introspection	Posterior cingulate cortex/ precuneus	Limited[a]	Increased DMN during cognition[26b]
	Self-referential tasks	Medial prefrontal cortex	Significant with progression[18a]	
		Medial temporal lobe	Significant with progression[18]	
Ventral attention (VAN)	Engages attention to salient stimuli	Right basolateral amygdala	Significant[19,35a]	Reduced BOLD in frontal regions[16a]
	Mediates activation of other networks	Ventral frontal cortex	Variable	Atrophy in frontal regions[15–18a]
		Right temporoparietal junction	Limited	
		Ventral striatum	Limited	
Dorsal attention (DAN)	Voluntary orienting	Striatum (esp. head of caudate)	Deafferentation	Reduced BOLD in frontal regions[16a]
	Cognitive information processing	Dorsolateral prefrontal cortex	Limited	Atrophy in frontal regions[15–18a]
		Posterior parietal cortex	Limited	
		Frontal eye fields	Limited	
Visual	Transmission of visual information	Retina	Significant (with progression)[17a]	Altered cortical visual processing[15,16a]
	Perception of lower-order visual stimuli	Thalamic lateral geniculate nucleus	Limited	
		Occipital cortices	Limited	Atrophy in occipital regions[41c]



Network	Anatomical Areas	Function
Default Mode Network 	• Medial temporal cortex • Medial prefrontal cortex • Posterior cingulate cortex	• Task-independent introspection • Self-referential tasks
Dorsal Attentional Network 	• Dorsolateral prefrontal cortex • Posterior parietal cortex • Corpus striatum	• Voluntary orienting • Cognitive information processing
Ventral Attentional Network 	• Basolateral amygdala • Lateral and inferior prefrontal cortex • Temporoparietal junction • Ventral striatum	• Mediate activation of other networks • Engages attention to salient stimuli


Visual Object Input
Neuromodulators
DA/ACh PH, Med
5HT Med
Primary Sensory Organ
(e.g. Retina) CBS
III, IV & VI nuclei
RAS PS
ILn PD
Thalamic Relay
(e.g. LGN) PD/DLB
MLF
Primary Sensory Cortex
(e.g. V1) E, M, AS, PCA
Superior Colliculus
Amygdala PD, PTSD
DMN
VAN
DAN
PD/DLB
PD/DLB
SZ
PTSD


VAN
Interpretation of Ambiguous Percept
DMN
time
Threatening
Autobiographical

Movement Disorders
Vol. 20, No. 2, 2005, pp. 130–140
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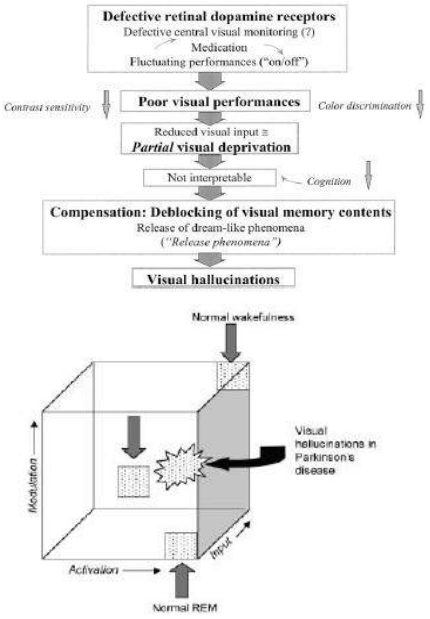
Review

Repeated Visual Hallucinations in Parkinson's Disease as Disturbed External/Internal Perceptions: Focused Review and a New Integrative Model

Nico J. Diederich, MD,[1,2]* Christopher G. Goetz, MD,[2] and Glenn T. Stebbins, PhD[2]



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Journal of the Neurological Sciences xx (2006) xxx – xxx

Journal of the Neurological Sciences

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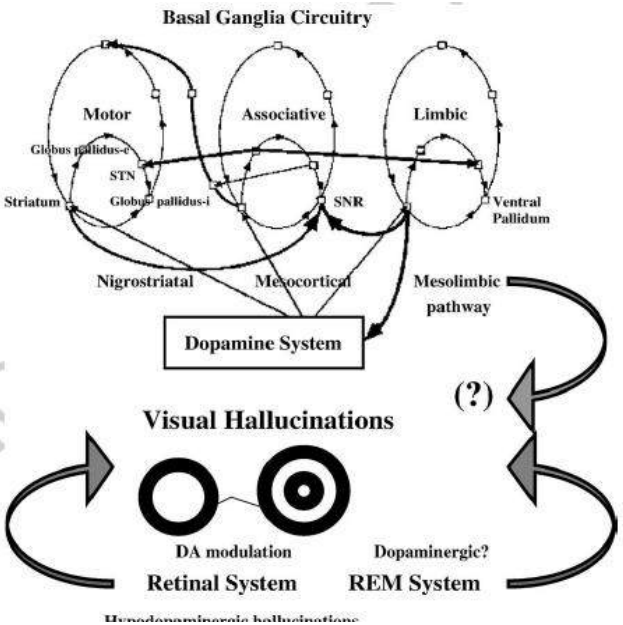
Visual hallucinations in Parkinson's disease: Clues to separate origins

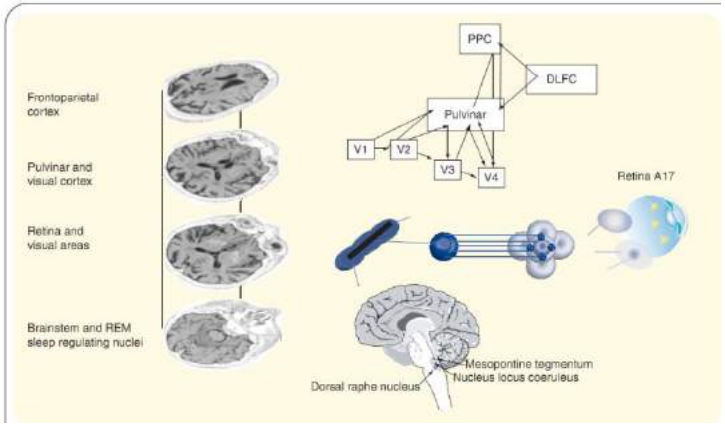
M. Onofrj [a,b,*], L. Bonanni [a,b], G. Albani [c], A. Mauro [c], D. Bulla [d], A. Thomas [a,b]



New approaches to understanding hallucinations in Parkinson's disease: phenomenology and possible origins

Marco Onofrj†*, Astrid Thomas and Laura Bonanni*

Expert Rev. Neurotherapeutics 7(12), 1731–1750 (2007)



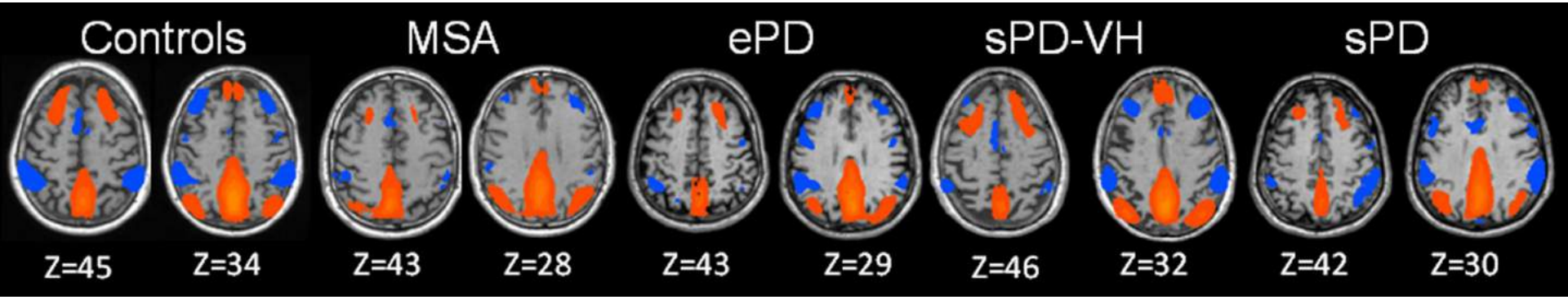
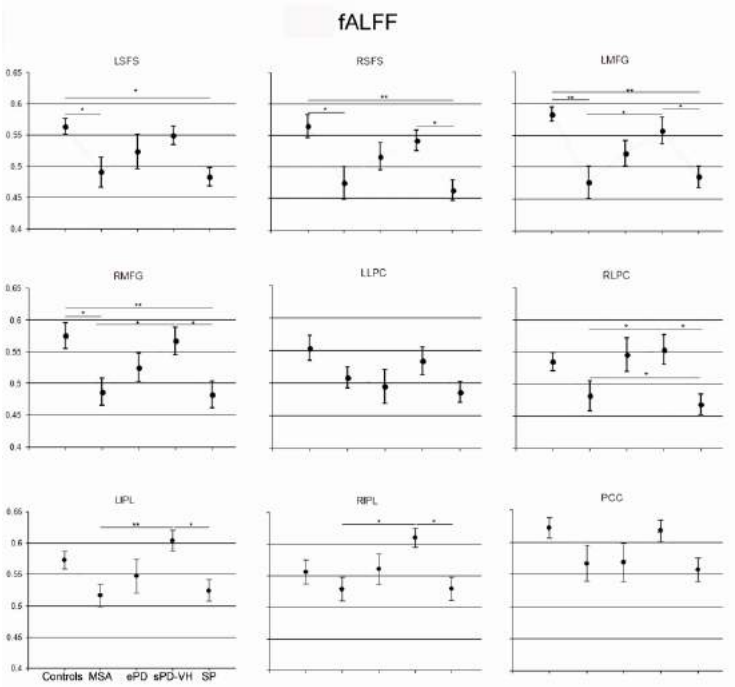
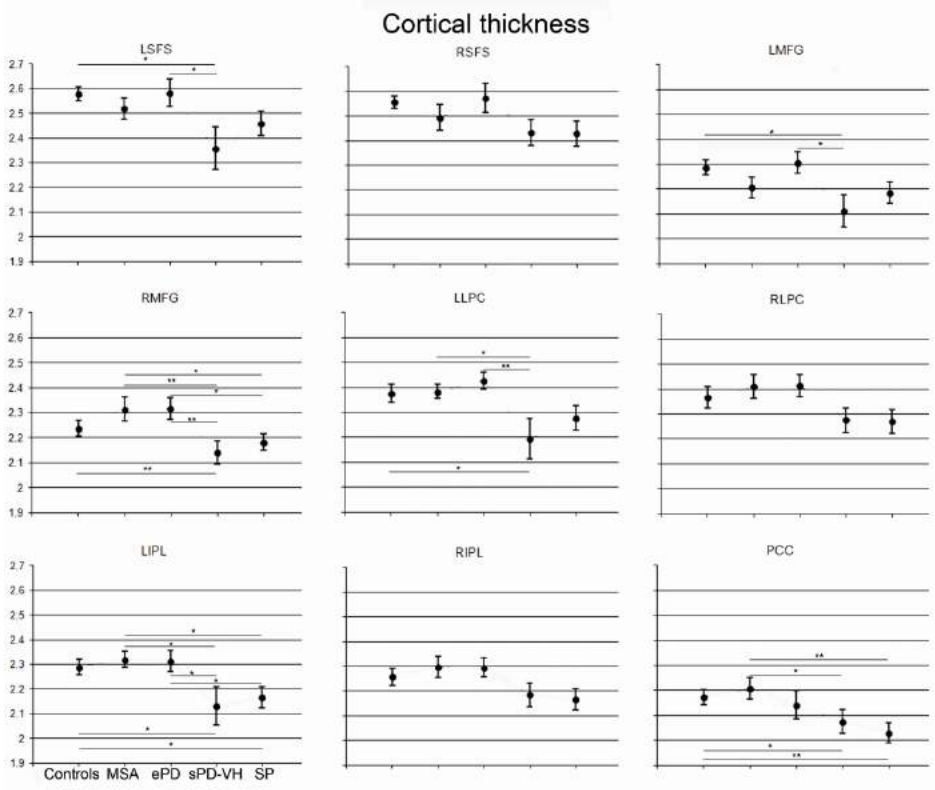


Figure 1. Group-level ICA results representing DMN pattern for control, MSA, ePD, sPD-VH and sPD group. Maps were overlaid onto Talairach-transformed T1 image of a representative control, MSA, ePD, sPD-VH and sPD patient. Notice that DMN was clearly identified in each group.
Abbreviations: ePD = early Parkinson's Disease; MSA = Multiple System Atrophy; sPD = severe Parkinson's Disease without visual hallucinations; sPD-VH = severe PD with visual hallucinations.


fALFF
LSFS
RSFS
LMFG
RMFG
LLPC
RLPC
LIPL
RIPL
PCC
Controls MSA ePD sPD-VH SP


Cortical thickness
LSFS
RSFS
LMFG
RMFG
LLPC
RLPC
LIPL
RIPL
PCC
Controls MSA ePD sPD-VH SP

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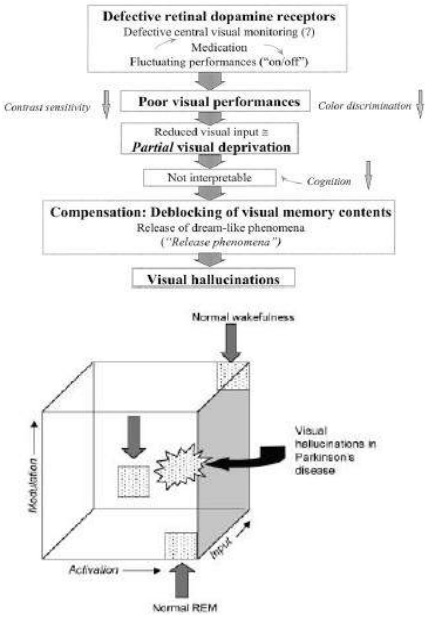

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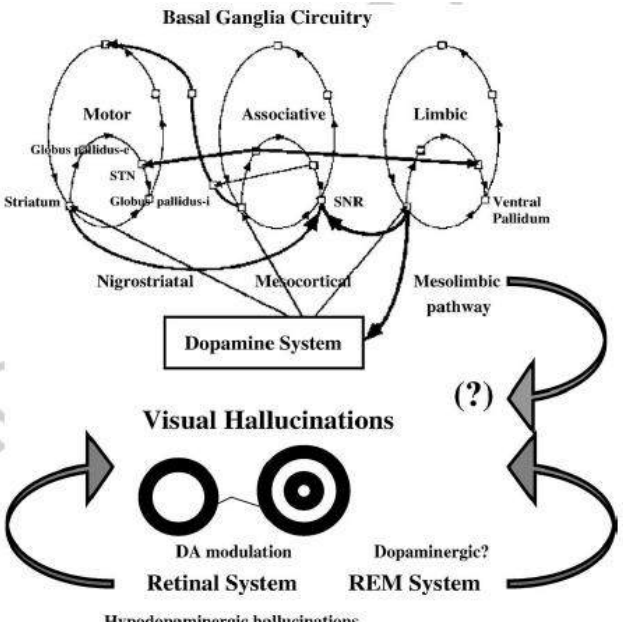

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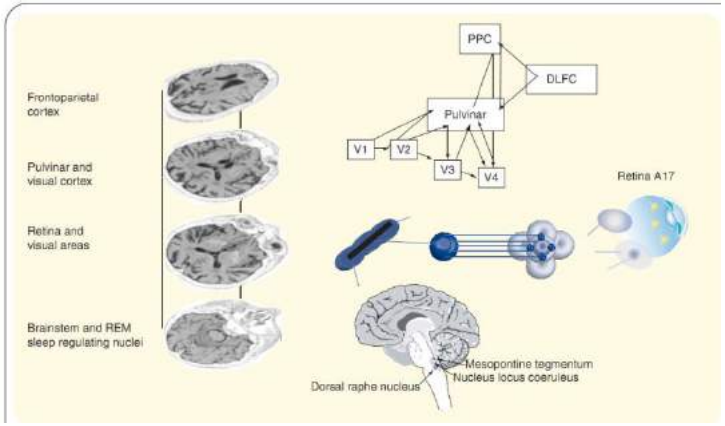

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Clinical neuroanatomy

Relevance of subcortical visual pathways disruption to visual symptoms in dementia with Lewy bodies

Stefano Delli Pizzi [a,b,c], Valerio Maruotti [b], John-Paul Taylor [d], Raffaella Franciotti [a,b,c], Massimo Caulo [a,c], Armando Tartaro [a,c], Astrid Thomas [a,b], Marco Onofrj [a,b] and Laura Bonanni [a,b,*]

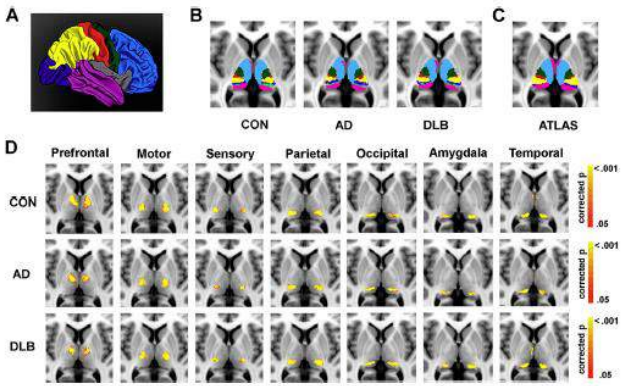

Fig. 1 – Thalamic structural connectivity. (A) Cortical rendering of target regions used for thalami parcellation (colours were in agreement with thalamic connectivity). (B) Grand averages of connectivity-based subdivision of thalami are shown for controls, dementia with Lewy bodies (DLB) and Alzheimer's Disease (AD). Within-group probabilistic tractography outputs for each cortical target region were performed with "find the biggest" command line. Thalamic voxels are classified and coloured according to the highest probability of connection to a specific cortical region. Blue = connectivity-defined sub-region (CDR) that projects from thalamus to prefrontal cortex; dark green = CDR that projects from thalamus to motor cortex; red = CDR that projects from thalamus to primary and secondary somato-sensory cortex; yellow = CDR that projects from thalamus to parietal cortex; dark blue = CDR that projects from thalamus to occipital cortex; green = CDR that projects from thalamus to amygdala; fuchsia = CDR that projects from thalamus to temporal cortex. (C) Thalami regions defined by Oxford Thalamic Connectivity Atlas. (D) Within-group probabilistic tractography maps for each cortical target region. The significant results are displayed by voxels rating from red to yellow ($p < .05$, FWE-corrected). No significant differences were observed among groups.

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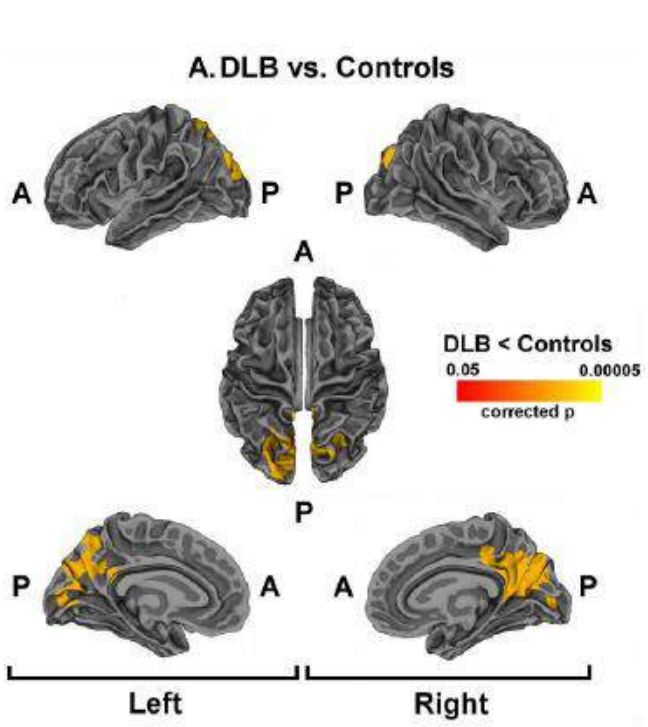
PLOS | ONE

Structural Alteration of the Dorsal Visual Network in DLB Patients with Visual Hallucinations: A Cortical Thickness MRI Study

Stefano Delli Pizzi[1,2,3], Raffaella Franciotti[1,2,3], Armando Tartaro[1,3], Massimo Caulo[1,3], Astrid Thomas[1,2], Marco Onofrj[1,2], Laura Bonanni[1,2]*

1 Department of Neuroscience and Imaging, "G. d'Annunzio" University, Chieti, Italy, **2** Aging Research Centre, Ce.S.I., "G. d'Annunzio" University Foundation, Chieti, Italy, **3** Institute for Advanced Biomedical Technologies (ITAB), "G. d'Annunzio" University Foundation, Chieti, Italy



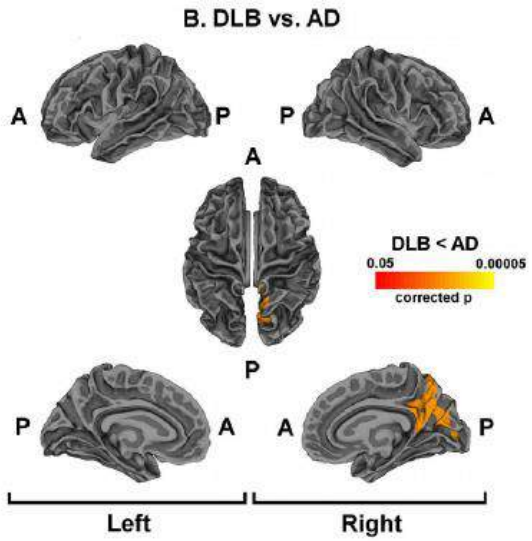


Figure 1. Cortical thinning in DLB patients. Statistical maps showing significant difference between DLB patients and controls (panel A) and between DLB patients and AD (panel B). Areas with cluster wise probability below p = 0.05 are shown, according to Monte Carlo correction for multiple comparisons. Panel A: Colour rating from red to yellow indicates local cortical thickness reduction in DLB patients compared to controls. Panel B: Colour rating from red to yellow indicates local cortical thickness reduction in DLB patients compared to AD. Abbreviations: A = Anterior; P = Posterior.
doi:10.1371/journal.pone.0086624.g001

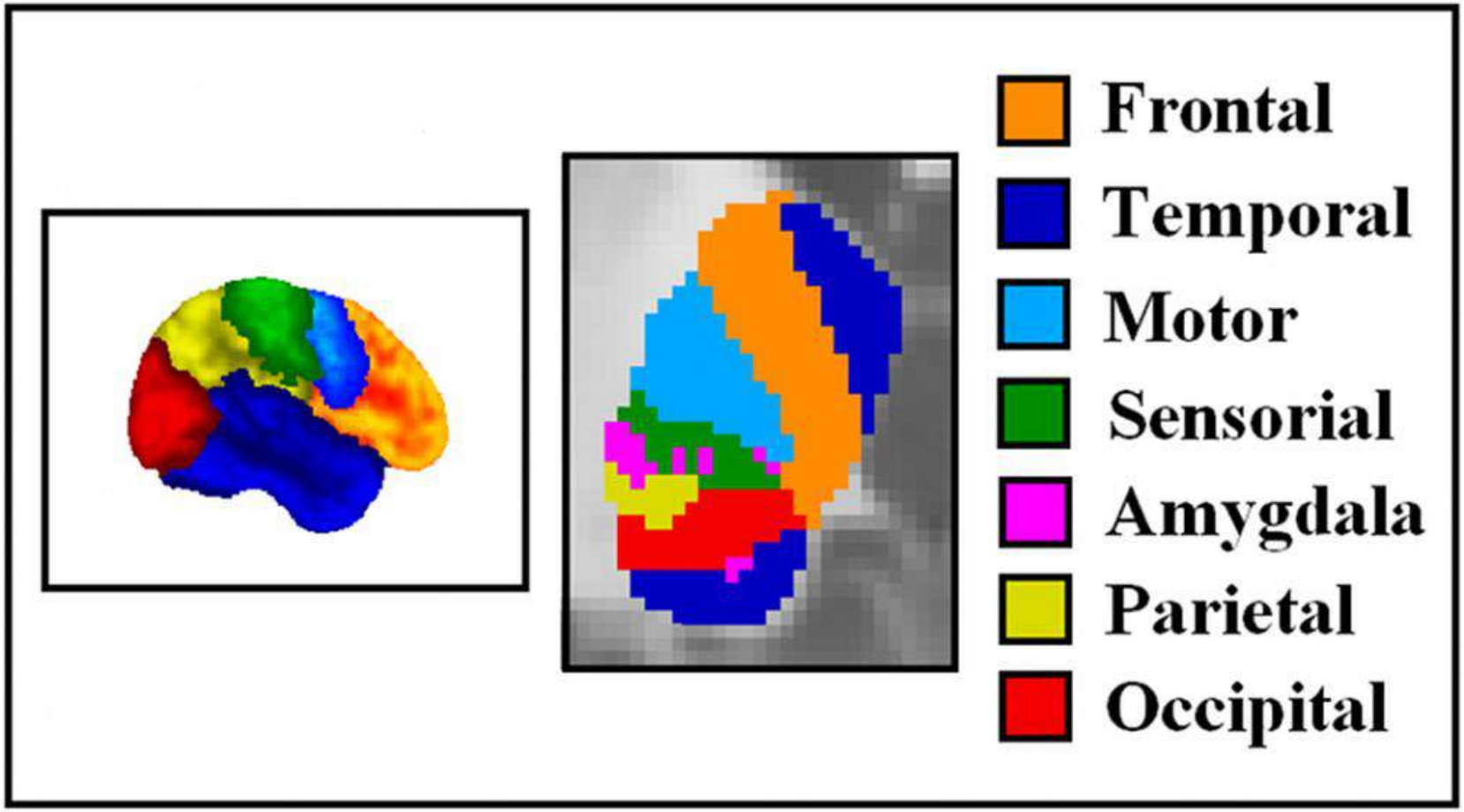


Figure 1. Representative connectivity-based subdivision of thalami in a single subject. Thalamic voxels are classified and coloured according to the regions of the cortex which they showed the highest connection probability. Thalamic connectivity-definited regions (CRDs). Orange=CDR that projects from thalamus to frontal cortex; blue=CDR that projects from thalamus to motor cortex; green=CDR that projects from thalamus to primary and secondary somato-sensory cortex; yellow=CDR that projects from thalamus to parietal cortex; pink=CDR that projects from thalamus to amygdala; red= CDR that projects from thalamus to occipital; dark blue=CDR that projects from thalamus to temporal. Images are oriented according to radiological convention (i.e., the right hemisphere of the brain corresponds to the left side of the image).

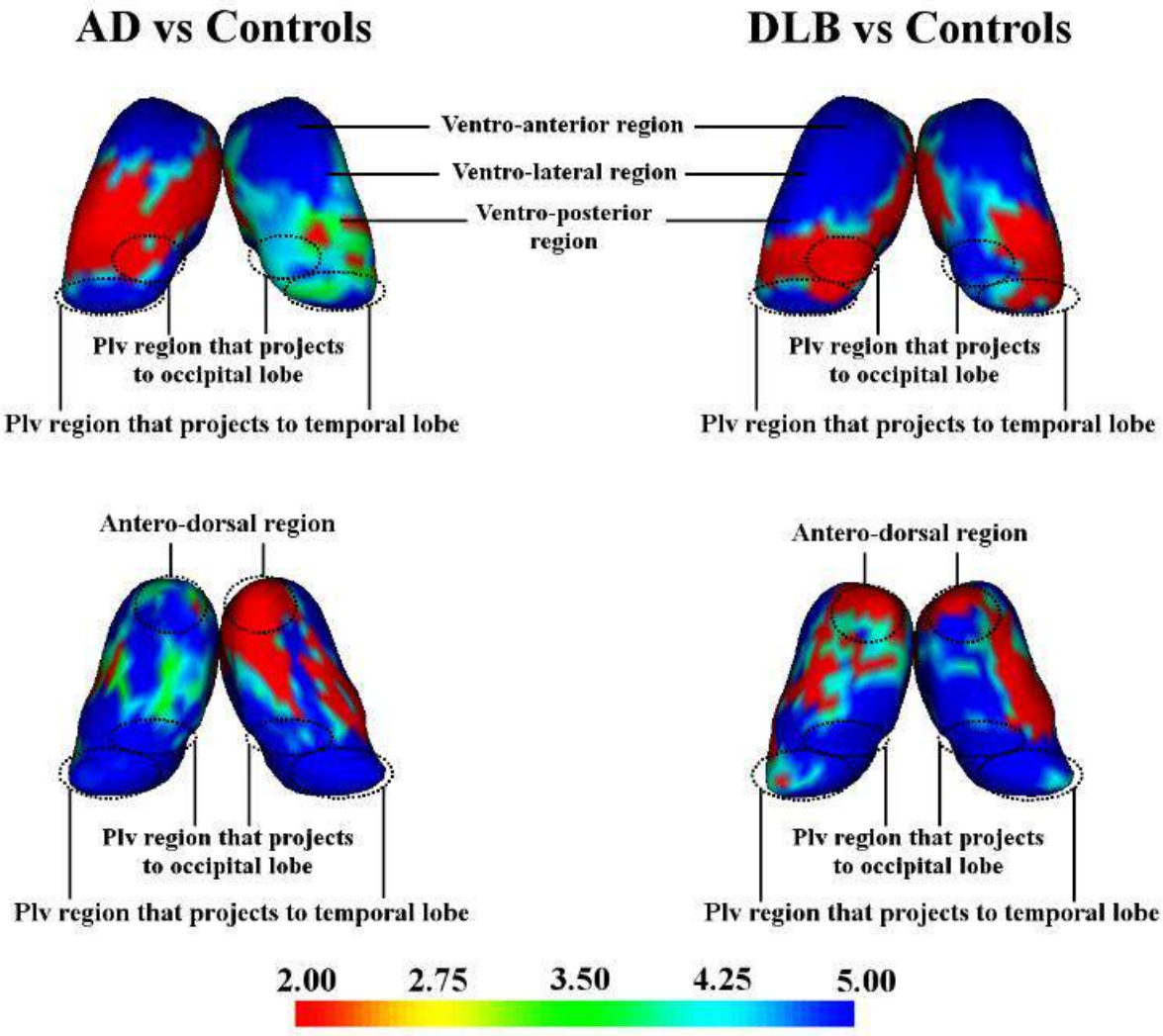
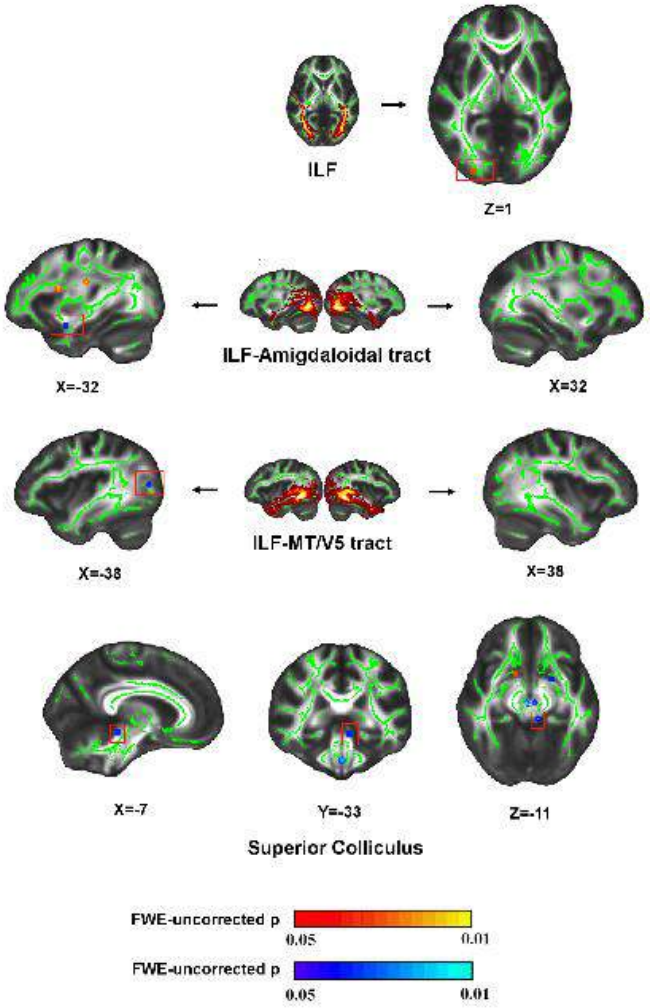


Figure 2. Vertex-wise comparison of the thalami in AD and DLB patients respect to control subjects. Thalamic regions are discriminated according with the connectivity-based subdivision of thalami. Colours rating from red to dark blue show the strength of local shape changes. For vertex-wise shape analysis, F-statistics giving p=0.15 (F=2), p=0.08 (F=2.75), p=0.04 (F=3.5), and p=0.01 (F=5). The thalamic regions of interest are bounded by the dashed or indicated by black arrows. Comparing spatially the different pattern of GM atrophy between the two maps, GM loss is more evident in AD vs controls comparison respect to DLB vs controls comparison in the pulvinar and in the antero-ventral regions of the thalamus that both projects to temporal lobe. Contrary to AD vs controls map, in the DLB vs controls comparison, thalamic sites of GM atrophy are localized in the medio- and posterior- ventral portion that project to sensori-motor cortex and parietal cortex and in the regions that project to occipital lobe. Images are oriented according to neurological convention (i.e., the right hemisphere of the brain corresponds to the right side of the image).



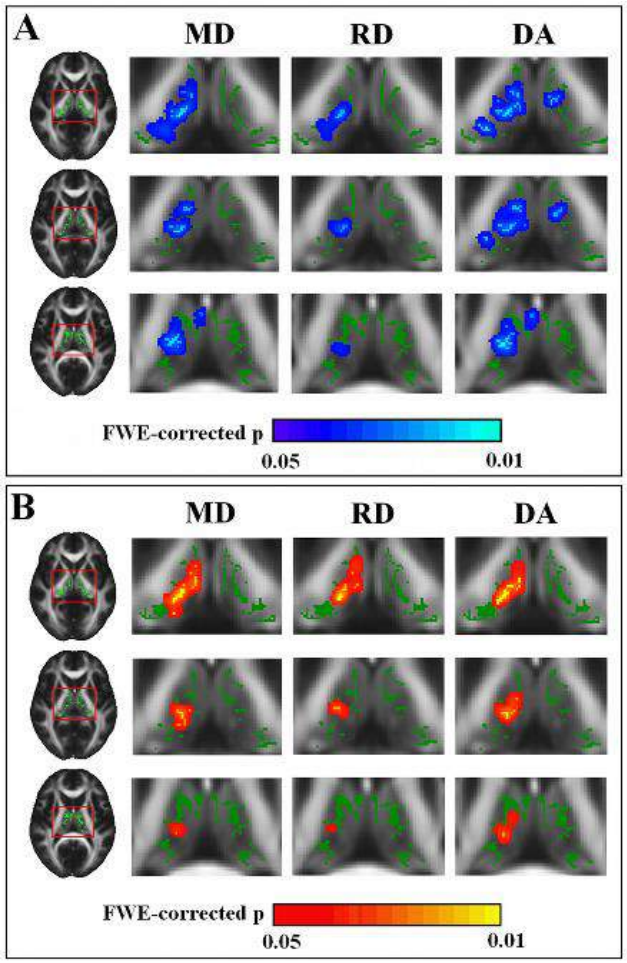
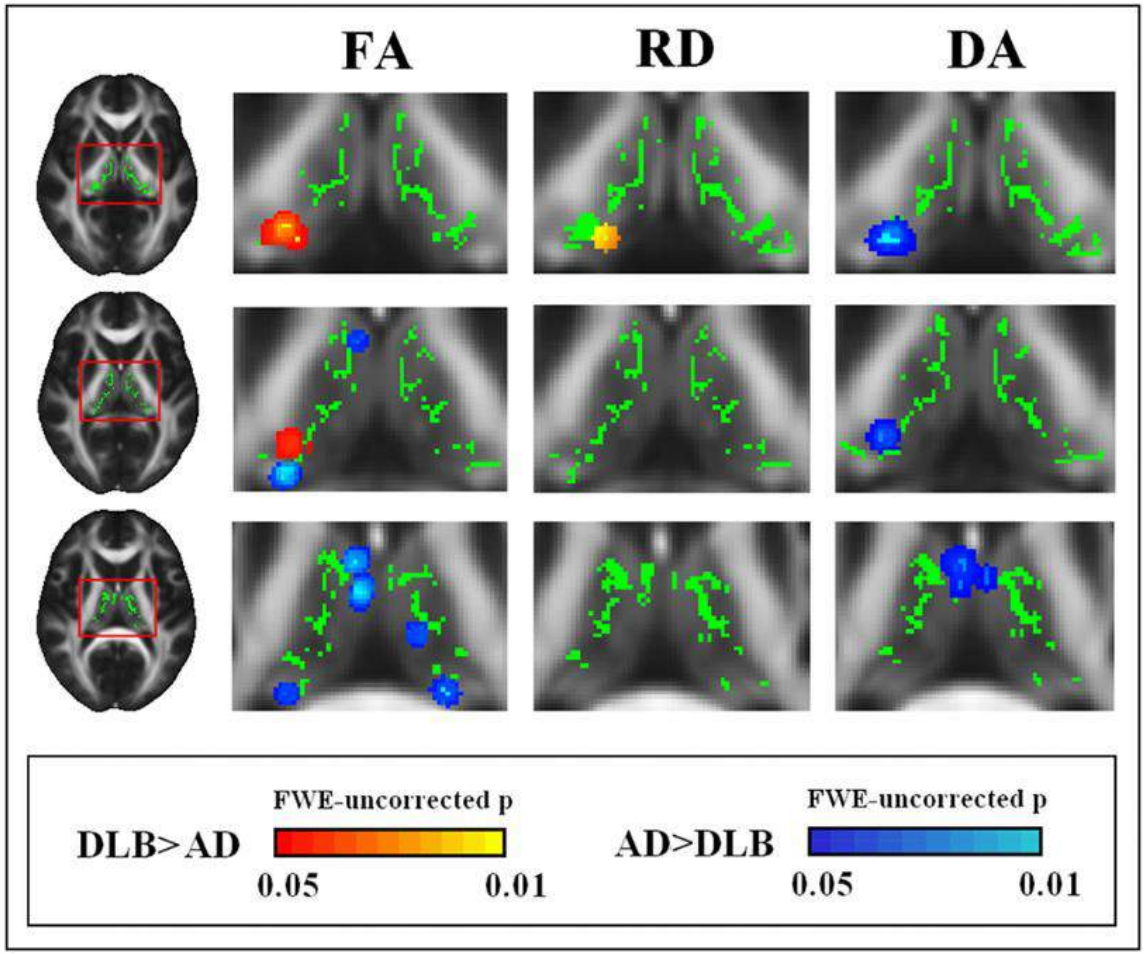


Figure 3. Spatial maps of WM integrity changes in the inferior longitudinal fascicle (ILF), amygdaloid and superior colliculus tracts. TBSS

Spatial maps of FA, MD, RD and DA changes in the white matter of thalami. ROI TBSS results are overlaid on the MNI152 standard brain.



Spatial maps of FA, MD and DA changes in the WM of thalami. ROI TBSS results are overlaid on the MNI152 standard brain.

Cerebral Cortex
doi:10.1093/cercor/bhu220

Thalamic Involvement in Fluctuating Cognition in Dementia with Lewy Bodies: Magnetic Resonance Evidences

Stefano Delli Pizzi[1,2,3], Raffaella Franciotti[1,2,3], John-Paul Taylor[4], Astrid Thomas[1,2], Armando Tartaro[1,3], Marco Onofrj[1,2] and Laura Bonanni[1,2]

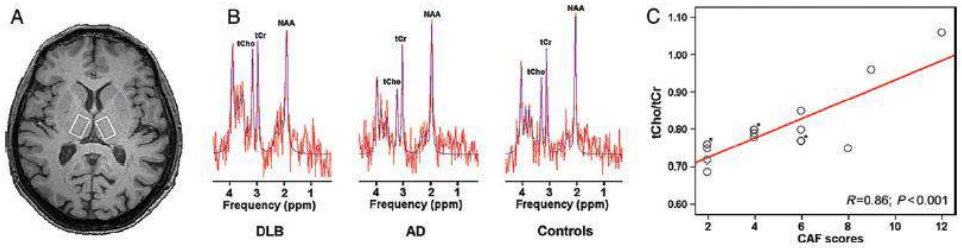


Figure 1. Proton magnetic resonance spectroscopy (^{1}H-MRS). (*A*) Two voxels of $1.5 \times 1.0 \times 1.5$ mm^3 were, respectively, placed on right and left thalami by using T_1-weighted image as anatomical reference. (*B*) Representative spectra for DLB, AD, and controls. Estimated signals (violet) were reported on original signals (red). NAA, *N*-acetyl-aspartate (2.02 ppm); tCr, total creatine (3.03 ppm); tCho, total choline (3.22 ppm). (*C*) Scatterplot expresses the linear regression between CAF scores and tCho/tCr values in the right thalamus. AD, Alzheimer's disease; DLB, dementia with Lewy bodies. Values marked with an asterisk are overlapped.

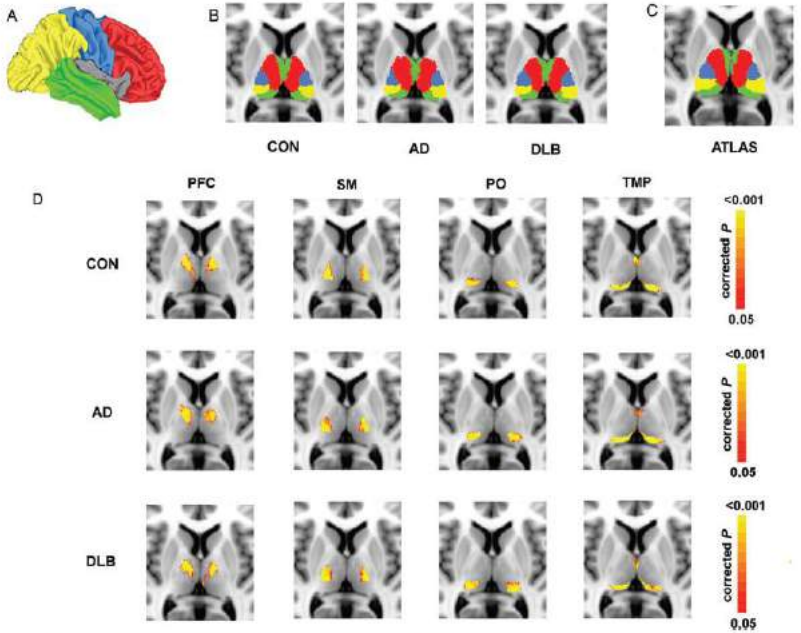


Figure 2. Structural connectivity. (*A*) Cortical rendering of target regions used for thalami parcellation (colors were in agreement with thalamic connectivity). (*B*) Connectivity-based subdivision of thalami for controls, dementia with Lewy bodies (DLB), and Alzheimer's disease (AD). Thalamic voxels are classified and colored according to the highest probability of connection to specific cortical regions. Red, connectivity-defined subregion (CDR) that projects from thalamus to prefrontal cortex; blue, CDR that projects from thalamus to motor and sensory cortices; yellow, CDR that projects from thalamus to parieto-occipital cortex; green, CDR that projects from thalamus to temporal cortex. (*C*) Thalami regions defined by Oxford Thalamic Connectivity Atlas. (*D*) Within-group probabilistic tractography maps for each cortical target region. The significant results are shown by voxels rating from red to yellow ($P < 0.05$, FWE-corrected). No significant differences were found among groups. PFC, prefrontal; SM, sensorimotor; PO, parieto-occipital; TMP, temporal.

The Neuroscience of Visual Hallucinations

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5

The clinical associations of visual hallucinations

Marco Onofrj, Astrid Thomas, Giovanni Martinotti, Francesca Anzellotti, Massimo Di Giannantonio, Fausta Ciccocioppo, and Laura Bonanni

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