Associazione Geriatri Extraospedalieri
GERIATRIA ITALIANA TERRITORIALE
sezione CAMPANIA

con il patrocinio di:
ASL Caserta, OMCEO Caserta, SICG, AIP

Giovedì, 16 ottobre 2014

GIORNATA GERIATRICA REGIONALE

Ordine dei Medici di Caserta
Via Bramante, 19 Parco Gabriella

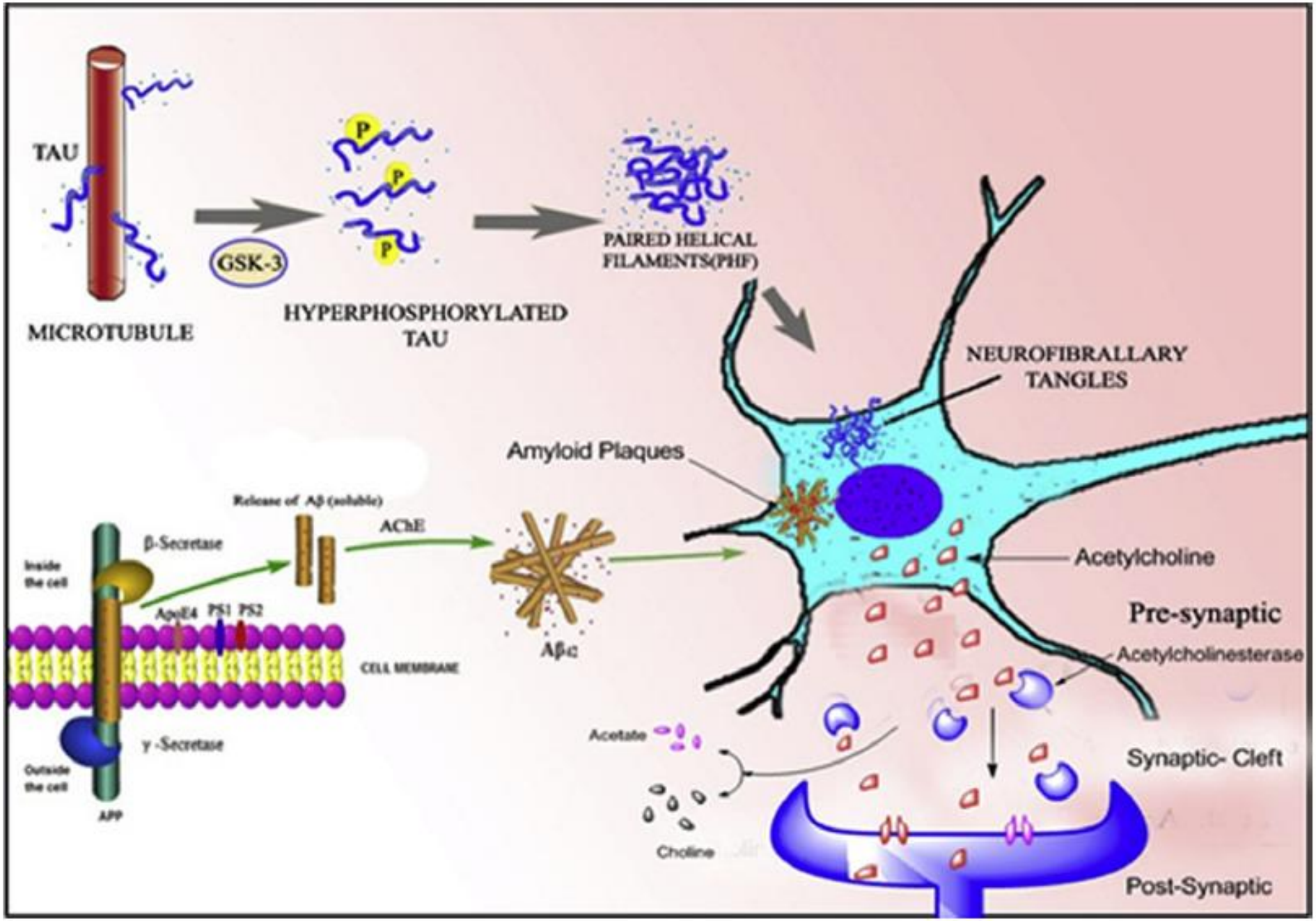
ASSOCIAZIONE ITALIANA PSICOGERIATRIA
Sezione Regionale Campana

Azienda Ospedaliera Universitaria Federico II
Integrata con il Servizio Sanitario Regionale

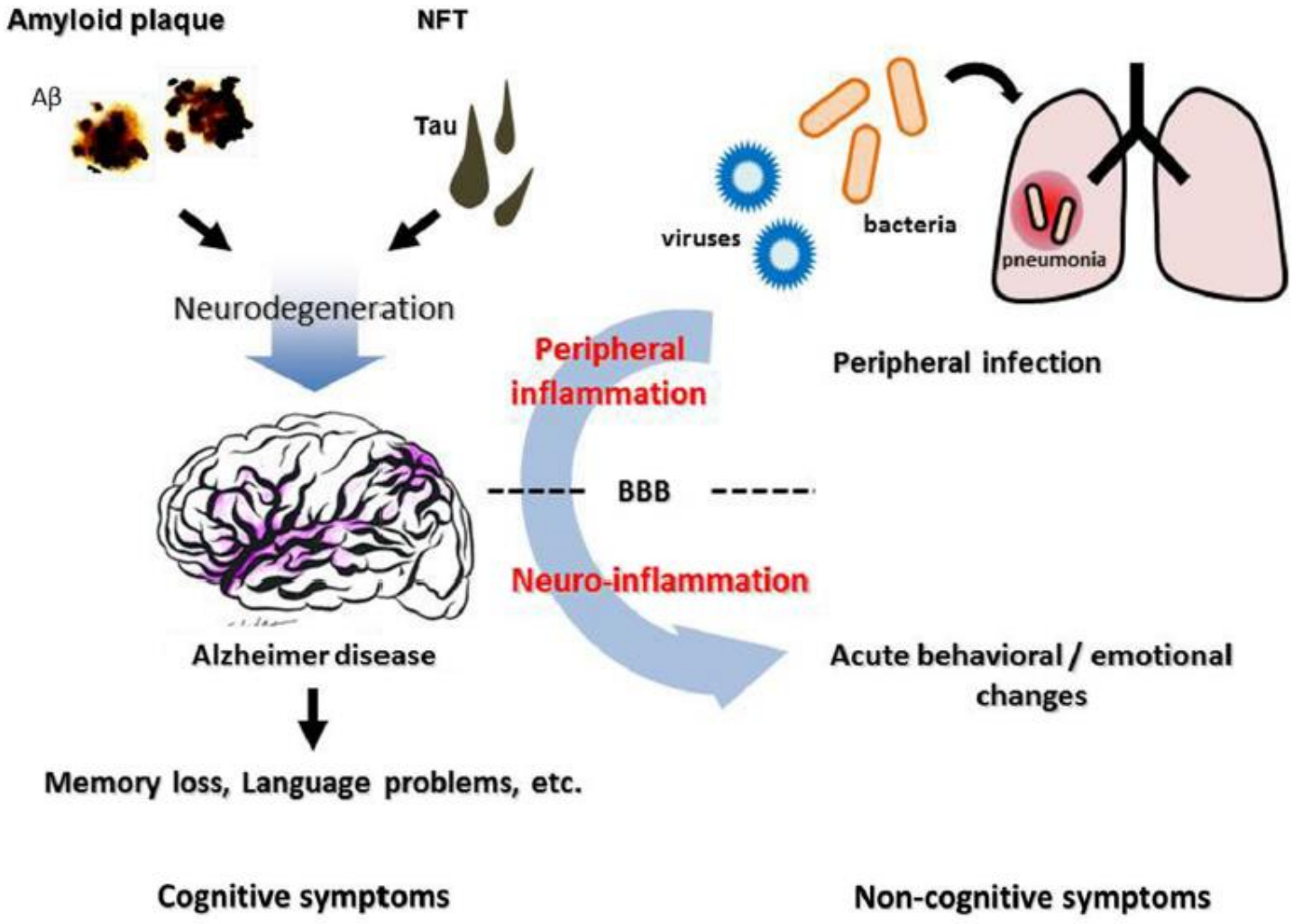
Terapia farmacologica e non farmacologica della demenza: lo stato dell'arte

Vincenzo Canonico

Unità di Valutazione Alzheimer
Cattedra di Geriatria
Università "Federico II" Napoli

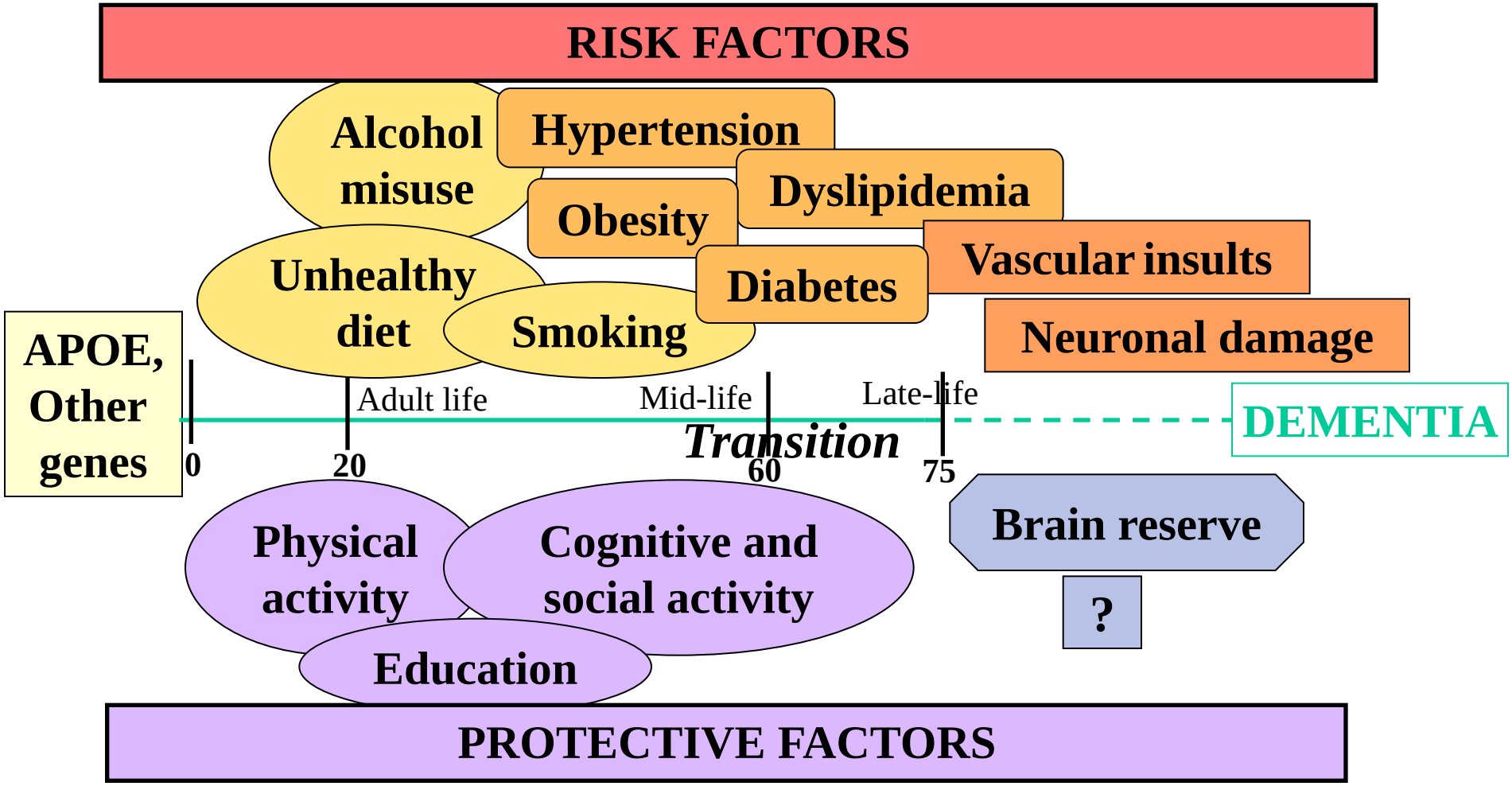

TAU
MICROTUBULE
GSK-3
P
P
P
HYPERPHOSPHORYLATED TAU
PAIRED HELICAL FILAMENTS(PHF)
NEUROFIBRALLARY TANGLES
Amyloid Plaques
Release of Aβ (soluble)
AChE
β-Secretase
Inside the cell
ApoE4 PS1 PS2
CELL MEMBRANE
γ -Secretase
Outside the cell
APP
Aβ42
Acetylcholine
Pre-synaptic
Acetylcholinesterase
Acetate
Choline
Synaptic- Cleft
Post-Synaptic

Systemic inflammation, blood-brain barrier vulnerability and cognitive/non-cognitive symptoms in Alzheimer disease: relevance to pathogenesis and therapy



Takeda S Frontiers in Aging Neuroscience 2014; 6, 1-8

A multi-factorial disease



Mangialasche, Kivipelto et al., Alzheimers Res Ther 2012

Terapia farmacologica delle demenze

Terapia non farmacologica delle demenze

TERAPIA DELLE DEMENZE

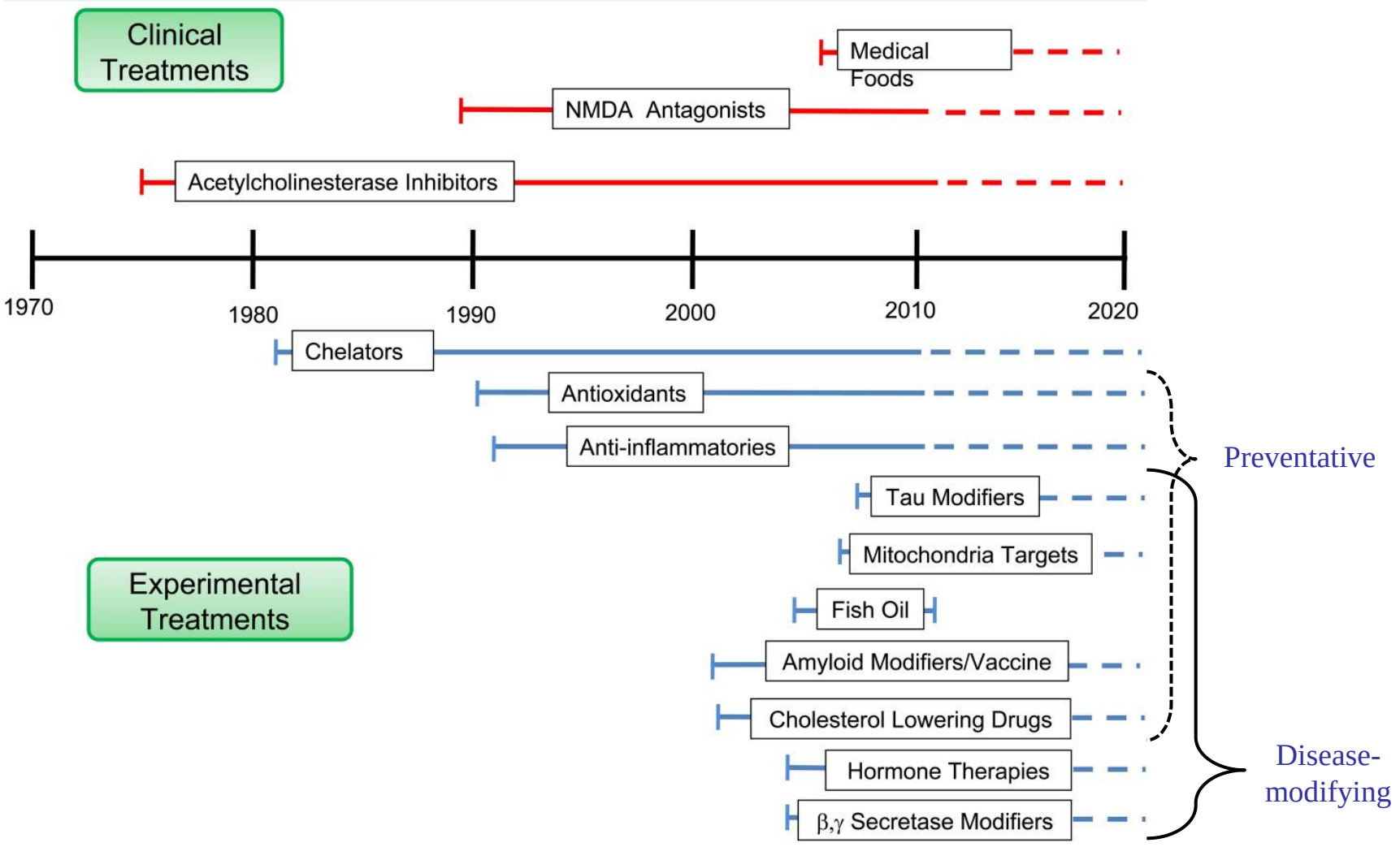
Terapia delle demenze

- **Deficit cognitivo**
- **BPSD**
- **Complicanze**

Lo stato dell’arte

?

Pharmacological treatment of AD



Stone JG, Casadesus G, Gustaw-Rothenberg K, et al. Frontiers in Alzheimer's disease therapeutics. Ther Adv Chronic Dis 2011; 2 (1): 9-23

I farmaci di oggi

Anticolinesterasici

- Donepezil (5-10 mg)
- Galantamina (8-16-24 mg)
- Rivastigmina (1.5-3.0-4.5-6.0, 4.6-9.5-13.3mg)

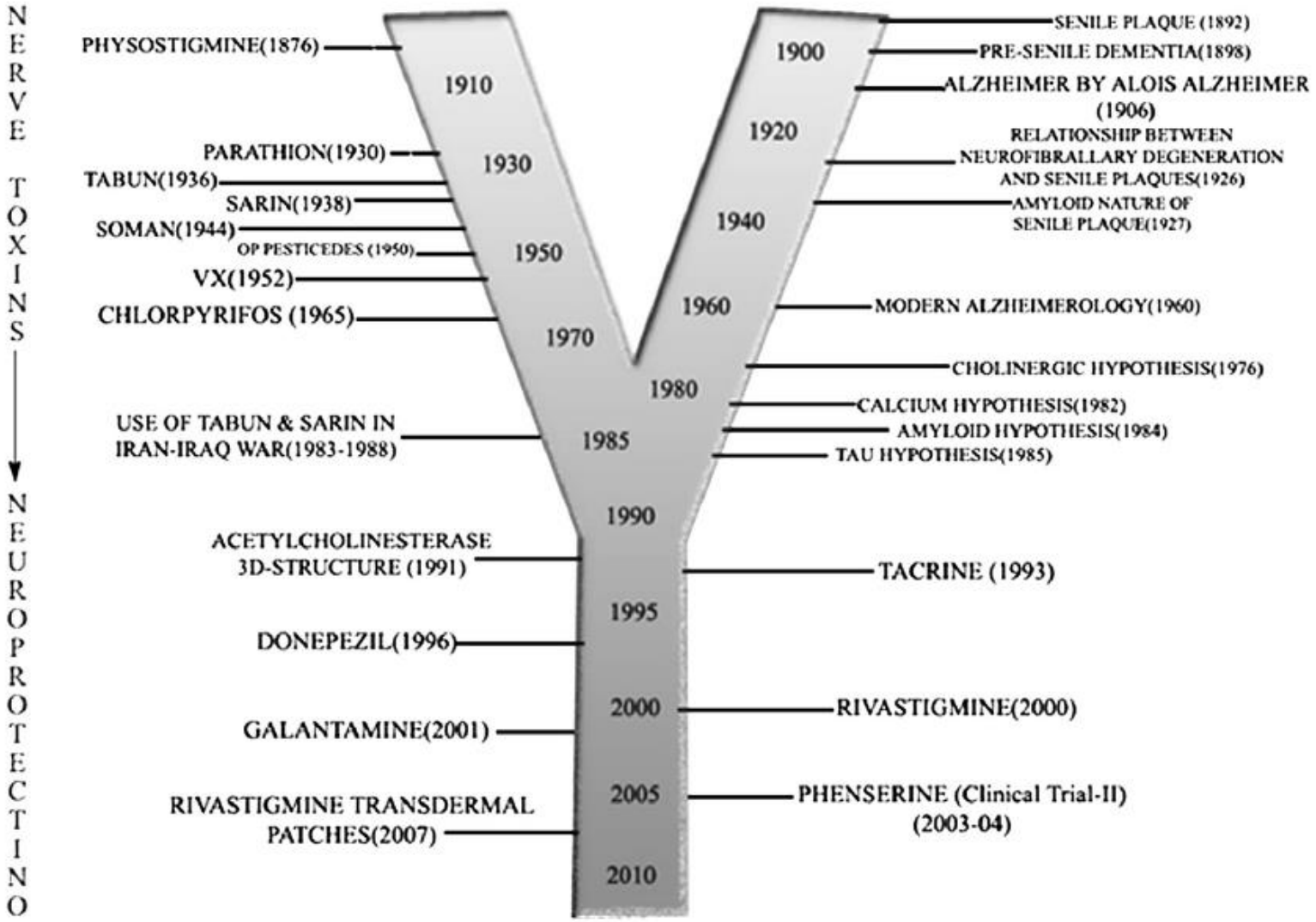
Agenti glutammatergici

- Memantina (10-20mg, soluzione)

Terapia dei BPSD (antipsicotici, stabilizzanti dell'umore, antidepressivi)

Terapia delle complicanze (allettamento, disfagia, disidratazione, inappetenza etc)

Acetylcholinesterase inhibitors as Alzheimer therapy: From nerve toxins to neuroprotection



Pharmacological Trials for AD

New pharmacological strategies for treatment of Alzheimer's disease: focus on disease modifying drugs

Drug	Mechanism of action relevant for AD	Phase of study	Result of study	Caveat of study
Rosiglitazone	β-secretase inhibition (?)	3	Ineffective	Lack of biomarker
Semagacestat	γ-secretase inhibition	3	Premature end	Severe adverse drug reaction
Tarenflurbil	γ-secretase modulation	3	Ineffective	Low potency, blood–brain barrier passage
Tramiprosate	Inhibition of Aβ oligomerization	3	Ineffective	–
Scyllo-inositol	Inhibition of Aβ oligomerization	2	Ineffective	Biomarker change
Bapineuzumab	Aβ clearance	3	Ongoing	Vasogenic oedema, amyloid angiopathy
Solaneuzumab	Aβ clearance	3	Ongoing	–
Lithium	Inhibition of tau phosphorylation	2	Clinical improvement Decrease of P-tau in CSF	–
Methylthioninium chloride	Inhibition of tau aggregation	2	Clinical improvement with 60 mg day^{-1}	Lack of biomarker
Nilvadipine	Aβ clearance	Open label	Clinical improvement	Lack of biomarker
Latrepirdine	Mitochondrial protection	3 3	Ineffective Ongoing (in association with other drugs)	– –

Salomone S, Caraci F, Leggio GM, et al. New pharmacological strategies for treatment of Alzheimer's disease: focus on disease modifying drugs. Br J Clin Pharmacol 2012; 73 (4): 504-17

Latest treatment options for Alzheimer's disease, Parkinson's disease dementia and dementia with Lewy bodies

Martin Broadstock, Clive Ballard† & Anne Corbett
King's College London, Wolfson Centre of Age-Related Diseases, London, UK

Introduction: Alzheimer's disease (AD), Parkinson's disease dementia (PDD) and dementia with Lewy bodies (DLB) together account for the vast majority of individuals with dementia. Approximately 35 million people worldwide are affected with this condition, and despite decades of research, effective therapies that slow or reverse disease progression have not yet been developed. The recent failure of several large-scale clinical trials is beginning to challenge the magnitude of focus on amyloid-related therapies for AD, and newer drug targets that have shown promise in the laboratory are being investigated in clinical trials.

Areas covered: This review summarises the current understanding of the underlying biology of AD, PDD and DLB and outlines the most recent drug candidates in advanced clinical trials.

Expert opinion: The lack of success in drug discovery for disease-modifying therapies for AD, PDD and DLB can be attributed to limitations in the design of clinical trials and the narrow focus of molecular targets for treatment. New avenues for drug discovery including repositioning and novel target identification may now provide opportunities for success, provided a critical mass of clinical trials is achieved through increased investment.

Keywords: Alzheimer's disease, clinical trials, dementia, dementia with Lewy bodies, Parkinson's disease dementia, treatment

Expert Opin. Pharmacother. (2014) 15(13):1797-1810

*

Table 1. Ongoing advanced clinical trials for Alzheimer's disease.

Drug	Mechanism	Phase and location	Study description	Trial completion date	Clinical trial number
Solaneuzemab (LY2062430)	Anti-Aβ passive immunotherapy	III – US	18-month, placebo-controlled RCT in 2100 people with mild AD investigating the progression of AD	December 2016	NCT01900665 (NCT01760005 with autosomal dominant AD mutations), NCT02008357 (people at risk of developing AD)
Crenezumab	Anti-Aβ passive immunotherapy	II – US	5-year, placebo-controlled RCT in 300 people with a presenilin1 E280A mutation who are also asymptomatic	September 2020	NCT01998841 (NCT01723826 as an extension of previous studies).
Gantenerumab	Anti-Aβ passive immunotherapy	III – Worldwide	24-month, placebo-controlled RCT in 1000 people with mild AD	July 2018	NCT02051608, NCT01224106 (prodromal AD)
MK-8931	Î -secretase inhibitor	III – worldwide	24-month, placebo-controlled RCT in 1500 people with prodromal AD	March 2018	NCT01953601, NCT01739348
TRx0237	Tau aggregation inhibitor	III – US and EU	18-month, placebo-controlled RCT in 700 people with mild AD	January 2016	NCT01689233, NCT01689246 (15-month study)
Intranasal insulin, Humulin®	Insulin signalling pathways	II/III – US	18-month, placebo-controlled RCT in 240 people with mild AD or MCI	February 2015	NCT01767909

Table 1. Ongoing advanced clinical trials for Alzheimer's disease (continued). *

Drug	Mechanism	Phase and location	Study description	Trial completion date	Clinical trial number
Exenatide (Exendin–4)	Glucagon- like peptide-1 agonist	II – US	36-month RCT of 230 people with AD	December 2015	NCT01255163
Liraglutide	Glucagon- like peptide-1 agonist	IIb – UK	12-month, placebo-controlled RCT in 206 people with mild AD	January 2017	NCT01843075
Metformin	Antidiabetic, protein phosphatase 2A agonist	Completed	12-month, placebo-controlled RCT in 80 people with AD	February 2012	NCT00620191
Nilvadipine	Calcium channel antagonist	III – EU	18-month, placebo-controlled RCT in 500 people with AD across 18 European sites funded by the European Union	December 2017	NCT02017340
TTP488	Antagonist at RAGE	III – US	18-month, placebo-controlled RCT in 800 people with mild AD, receiving AChE inhibitor and/or memantine	March 2017	NCT02080364
EVP-6124	Nicotinic α7 partial agonist	III – US	26-week, placebo-controlled RCT in 790 people with mild-to-moderate AD currently or previously receiving AChEIs	January 2017	NCT01969123, NCT01969136, NCT02004392
Sunphenon EGCg (Epigallocatechin-Gallate)	Antioxidant and signal modulator	II/III – Germany	18-month, placebo-controlled RCT in 50 people with mild AD	June 2015	NCT00951834
AC-1204	Caprylic triglyceride	II/III – US	6-month, placebo-controlled RCT in 480 people with mild/ moderate AD	January 2015	NCT01741194
Coconut oil	Coconut oil and medium chain triglycerides	II/III – US	6-month, placebo-controlled RCT in 65 people with mild/ moderate AD	June 2015	NCT01883648
Zydena (Udenafil)	PDE5 inhibitor	III – Korea	6-month, placebo-controlled RCT in 210 people with AD	August 2015	NCT01940952

Table 1. Ongoing advanced clinical trials for Alzheimer's disease (continued).

Drug	Mechanism	Phase and location	Study description	Trial completion date	Clinical trial number
Lu AE58054	5-HT_6 receptor antagonist	III – US	6-month, placebo-controlled RCT in 840 people with AD treated with donepezil	January 2016	NCT02006641, NCT02006654 (with other AChEIs), NCT01955161 (mild-to-moderate AD), NCT02079246 (not yet open for recruitment)
Mastinib	Mast cell inhibitor	III – EU	6-month, placebo-controlled RCT in 396 people with mild-to-moderate AD	December 2016	NCT01872598
Plasmapheresis with infusion of Flebogamma®, human albumin combined with intravenous immunoglobulin	Intravenous immunoglobulin	II/III – US and Spain	14-month, placebo-controlled RCT in 350 people with AD	December 2016	NCT01561053
Selenium and vitamin E	PP2A agonist, antioxidant	III – US, Canada and Puerto Rico	72- to 120-month, placebo-controlled prevention trial of 10,400 elderly men	August 2014	NCT00040378
RO4602522	Monoamine oxidase B inhibitor	II – Worldwide	12-month, placebo-controlled RCT in 495 people with moderate AD undergoing therapy with either and AChEI or memantine	May 2015	NCT01928420
Riluzole	Glutamate modulator	II – US	6-month, placebo-controlled RCT in 48 people with AD undergoing therapy with donepezil	November 2017	NCT01703117

Table 2. Ongoing advanced Parkinson's disease dementia and dementia with Lewy bodies therapeutics in clinical trials.

Drug	Mechanism	Phase and location	Study description	Trial completion date	Clinical trial number
Donepezil	Anticholinesterase	III – UK Multicentre	24-month, placebo-controlled RCT in 500 people with PD and mild dementia	May 2017	NCT01014858
Atomoxetine	Selective noradrenalin reuptake inhibitor	II – US	4-month study of 30 people with PD and cognitive impairment	May 2015	NCT01738191

Alzheimer's disease drug-development pipeline: few candidates, frequent failures

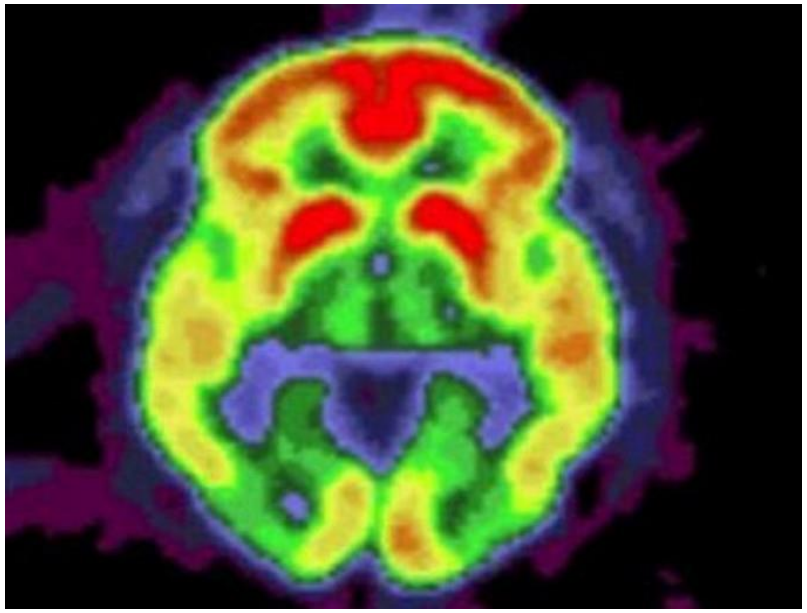
Jeffrey L Cummings[1*], Travis Morstorf[2] and Kate Zhong[1]

Table 1 Overview of Alzheimer's disease clinical trials from clinicaltrials.gov

Year registered	Phase 1	Phase 2	Phase 3	Total
2002	0	2	3	5
2003	0	5	7	12
2004	1	9	4	14
2005	4	19	9	32
2006	5	14	6	25
2007	16	22	8	46
2008	25	27	9	61
2009	28	30	14	72
2010	16	24	11	51
2011	15	26	4	45
2012	14	28	8	50
Total	124	206	83	413

Alzheimer's Research & Therapy 2014, 6:37

Svolta nella ricerca sull'Alzheimer: create cellule 'malate' in provetta



Creato per la prima volta l'Alzheimer in provetta per studiare nuovi farmaci. Per scoprire se un farmaco può curare la demenza si impiegano in media 12 anni, almeno 10 per sperimentarlo sui malati. Otto anni per individuare sui topi la molecola più promettente. E via così, un tentativo alla volta. Da oggi non è più così. A partire da staminali di embrioni umani è stato creato un microcervello malato di Alzheimer, moltiplicato in molti esemplari, e si andrà a verificare l'azione di 1.200 farmaci già in uso e di altri 5000 che si stavano selezionando sui topi.

La Repubblica Salute 15 ottobre 2014

Original Investigation | LESS IS MORE

*

Use of Medications of Questionable Benefit in Advanced Dementia

Jennifer Tjia, MD, MSCE; Becky A. Briesacher, PhD; Daniel Peterson, MA; Qin Liu, MD, PhD; Susan E. Andrade, ScD; Susan L. Mitchell, MD, MPH

OBJECTIVES To estimate the prevalence of medications with questionable benefit used by nursing home residents with advanced dementia, identify resident- and facility-level characteristics associated with such use, and estimate associated medication expenditures.

DESIGN, SETTING, AND PARTICIPANTS Cross-sectional study of medication use by nursing home residents with advanced dementia using a nationwide long-term care pharmacy database linked to the Minimum Data Set (460 facilities) between October 1, 2009, and September 30, 2010.

MAIN OUTCOMES AND MEASURES Use of medication deemed of questionable benefit in advanced dementia based on previously published criteria and mean 90-day expenditures attributable to these medications per resident. Generalized estimating equations using the logit link function were used to identify resident- and facility-related factors independently associated with the likelihood of receiving medications of questionable benefit after accounting for clustering within nursing homes.

RESULTS Of 5406 nursing home residents with advanced dementia, 2911 (53.9%) received at least 1 medication with questionable benefit (range, 44.7% in the Mid-Atlantic census region to 65.0% in the West South Central census region). Cholinesterase inhibitors (36.4%), memantine hydrochloride (25.2%), and lipid-lowering agents (22.4%) were the most commonly prescribed. In adjusted analyses, having eating problems (adjusted odds ratio [AOR], 0.68; 95% CI, 0.59-0.78), a feeding tube (AOR, 0.58; 95% CI, 0.48-0.70), or a do-not-resuscitate order (AOR, 0.65; 95% CI, 0.57-0.75), and enrolling in hospice (AOR, 0.69; 95% CI, 0.58-0.82) lowered the likelihood of receiving these medications. High facility-level use of feeding tubes increased the likelihood of receiving these medications (AOR, 1.45; 95% CI, 1.12-1.87). The mean (SD) 90-day expenditure for medications with questionable benefit was $816 ($553), accounting for 35.2% of the total average 90-day medication expenditures for residents with advanced dementia who were prescribed these medications.

CONCLUSIONS AND RELEVANCE Most nursing home residents with advanced dementia receive medications with questionable benefit that incur substantial associated costs.

JAMA Intern Med. doi:10.1001/jamainternmed.2014.4103
Published online September 8, 2014.

Table 3. Prevalence of Questionably Beneficial Medication Use Among 5406 Nursing Home Residents With Advanced Dementia During First 90 Days of Observation, 2009

Drug Class	No. (%)
Cholinesterase inhibitor	1966 (36.4)
Memantine hydrochloride	1362 (25.2)
Lipid-lowering agent	1213 (22.4)
Antiplatelet agent[a]	389 (7.2)
Hormone antagonist	62 (1.1)
Leukotriene inhibitor	61 (1.1)
Sex hormone	65 (1.2)
Cytotoxic chemotherapy	29 (0.5)
Immunomodulator	4 (0.007)

[a] Excluding aspirin.

JAMA Intern Med. doi:10.1001/jamainternmed.2014.4103
Published online September 8, 2014.

Treatment of Dementia With Lewy Bodies *

Brendon P. Boot, MBBS[1,*]

Opinion statement

Dementia with Lewy bodies (DLB) is a multisystem disorder with diverse disease expression. A treatment regime restricted to the cognitive aspects of the disease does no favor to patients. Instead, patients should be educated to recognize the symptoms of this multisystem involvement. There are no treatments that slow the progression of disease, but symptomatic treatments can be effective. When thinking about treatment, we find it useful to divide the symptoms and signs into five categories: (a) cognitive features, (b) neuropsychiatric features, (c) motor dysfunction, (d) autonomic dysfunction, and (e) sleep dysfunction. Clinicians, funding bodies and industry are increasingly recognizing the importance of this common and debilitating disease.

Current Treatment Options in Neurology (2013) 15:738–764

Table 1. Revised criteria for the clinical diagnosis of Dementia with Lewy bodies (DLB)

1. *Central feature*
   Progressive cognitive decline, usually with deficits in attention, executive function, and visuospatial ability
2. *Core features*
   Fluctuating cognition
   Recurrent visual hallucinations
   Parkinsonism
3. *Suggestive features*
   REM sleep behavior disorder
   Severe neuroleptic sensitivity
   Low dopamine transporter uptake in basal ganglia demonstrated by SPECT or PET imaging
4. *Supportive features*
   Repeated falls and syncope
   Transient, unexplained loss of consciousness
   Severe autonomic dysfunction
   Hallucinations in other modalities
   Systematized delusions
   Depression
   Relative preservation of medial temporal lobe structures on CT/MRI scan
   Generalized low uptake on SPECT/PET perfusion scans, with reduced occipital activity
   Abnormal (low uptake) MIBG myocardial scintigraphy

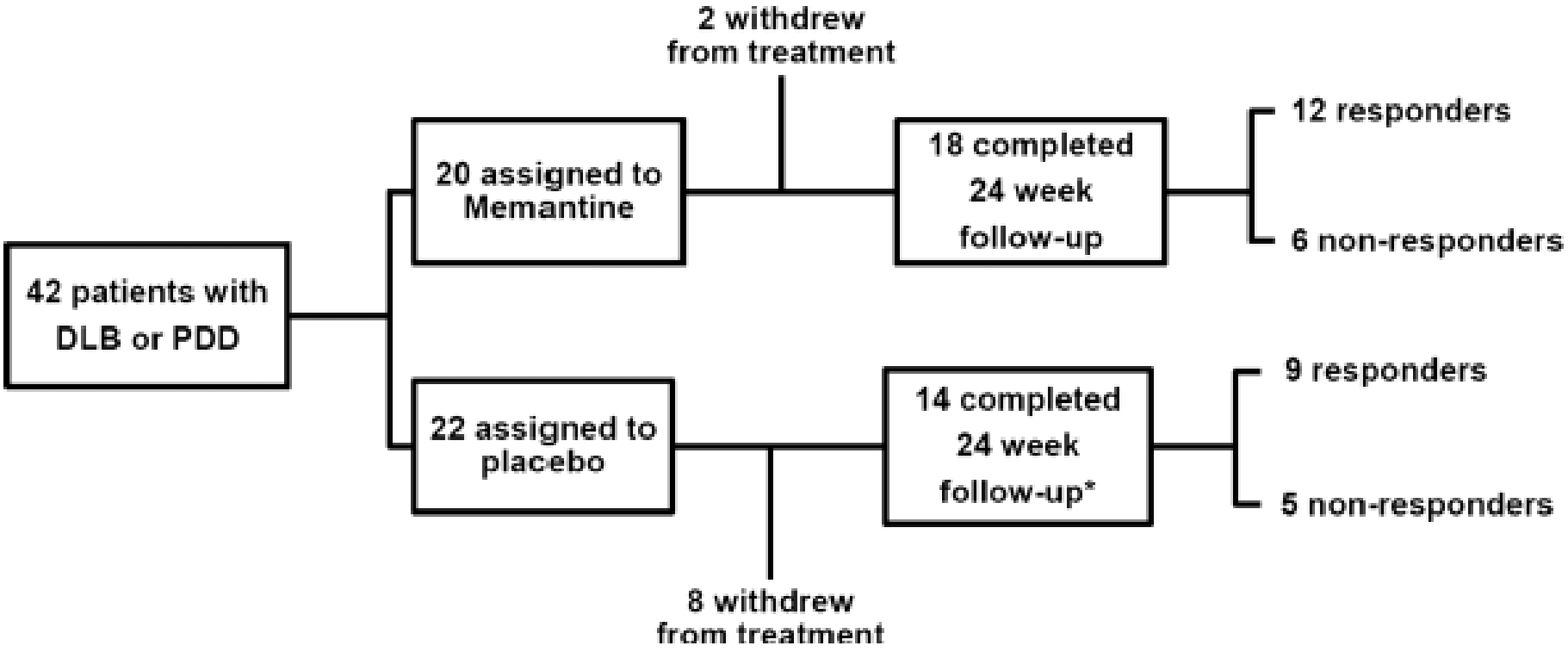
Current Treatment Options in Neurology (2013) 15:738–764

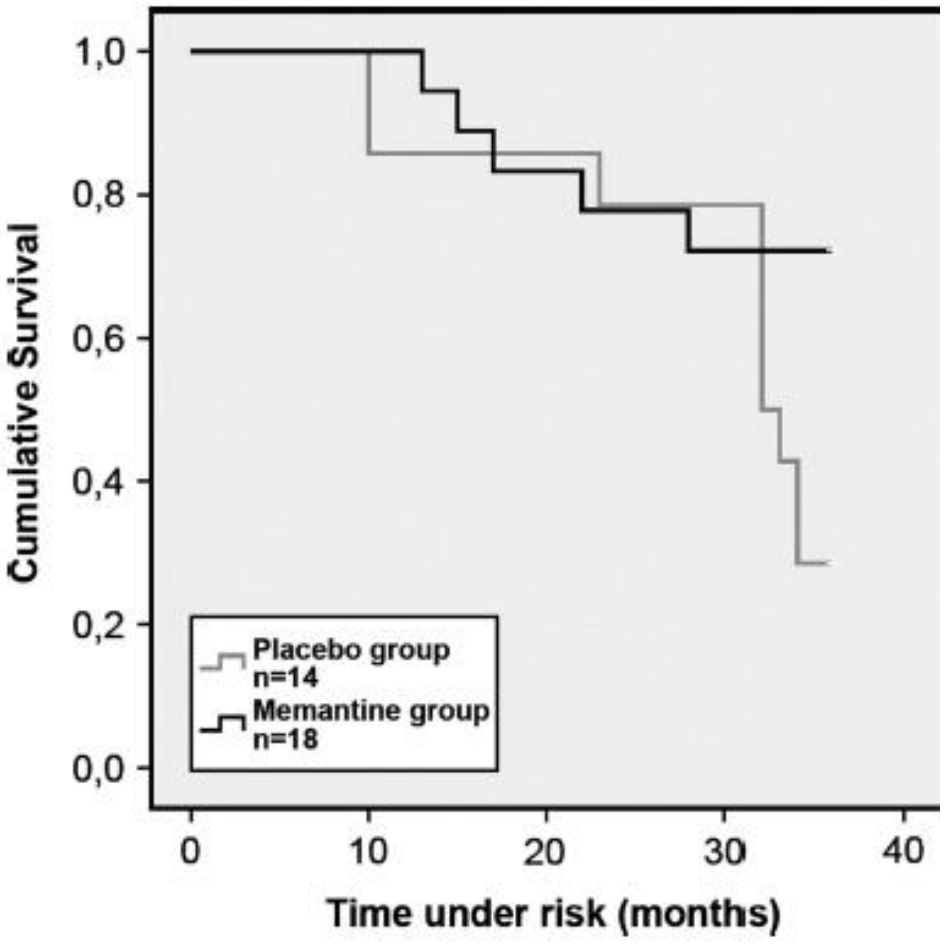
Treatment effect of memantine on survival in dementia with Lewy bodies and Parkinson's disease with dementia: a prospective study *

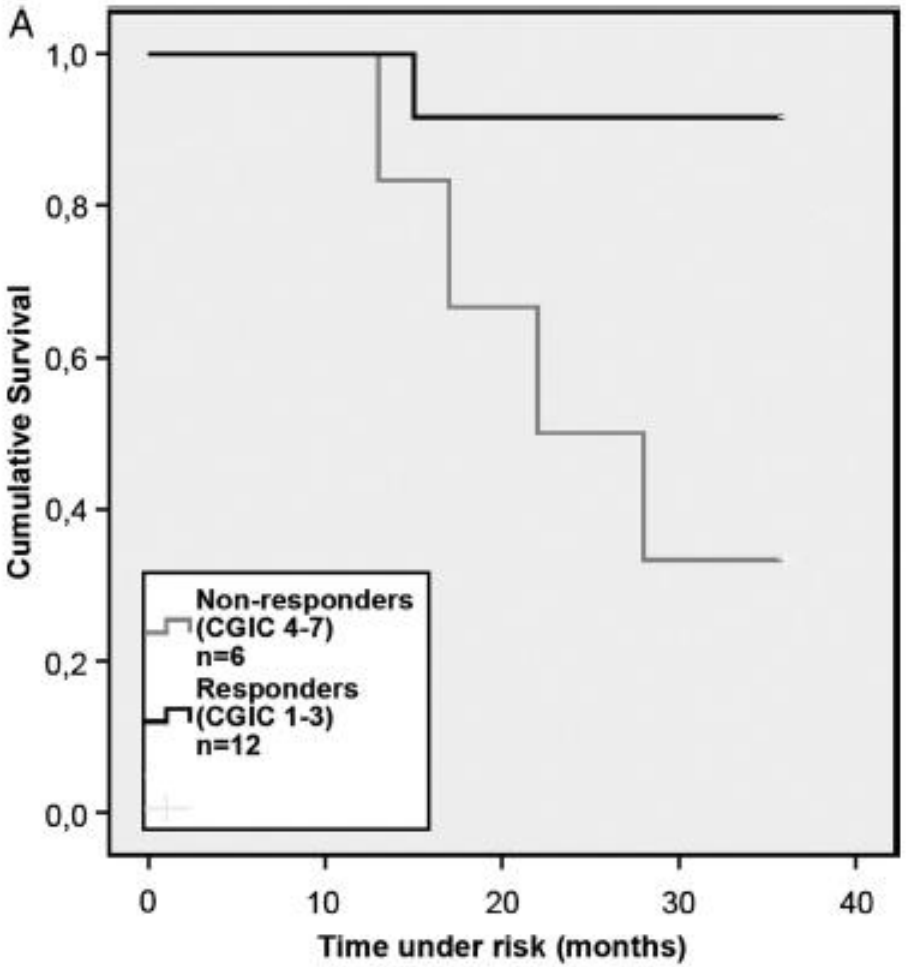
Kajsa Stubendorff,[1,2] Victoria Larsson,[1] Clive Ballard,[3] Lennart Minthon,[1] Dag Aarsland,[4,5] Elisabet Londos[1]

Conclusions: Early treatment with memantine and a positive clinical response to memantine predicted longer survival in patients with DLB and PDD. This suggests a possible disease-modifying effect and also has implications for health economic analysis. However, owing to the small study sample, our results should merely be considered as generating a hypothesis which needs to be evaluated in larger studies.

*





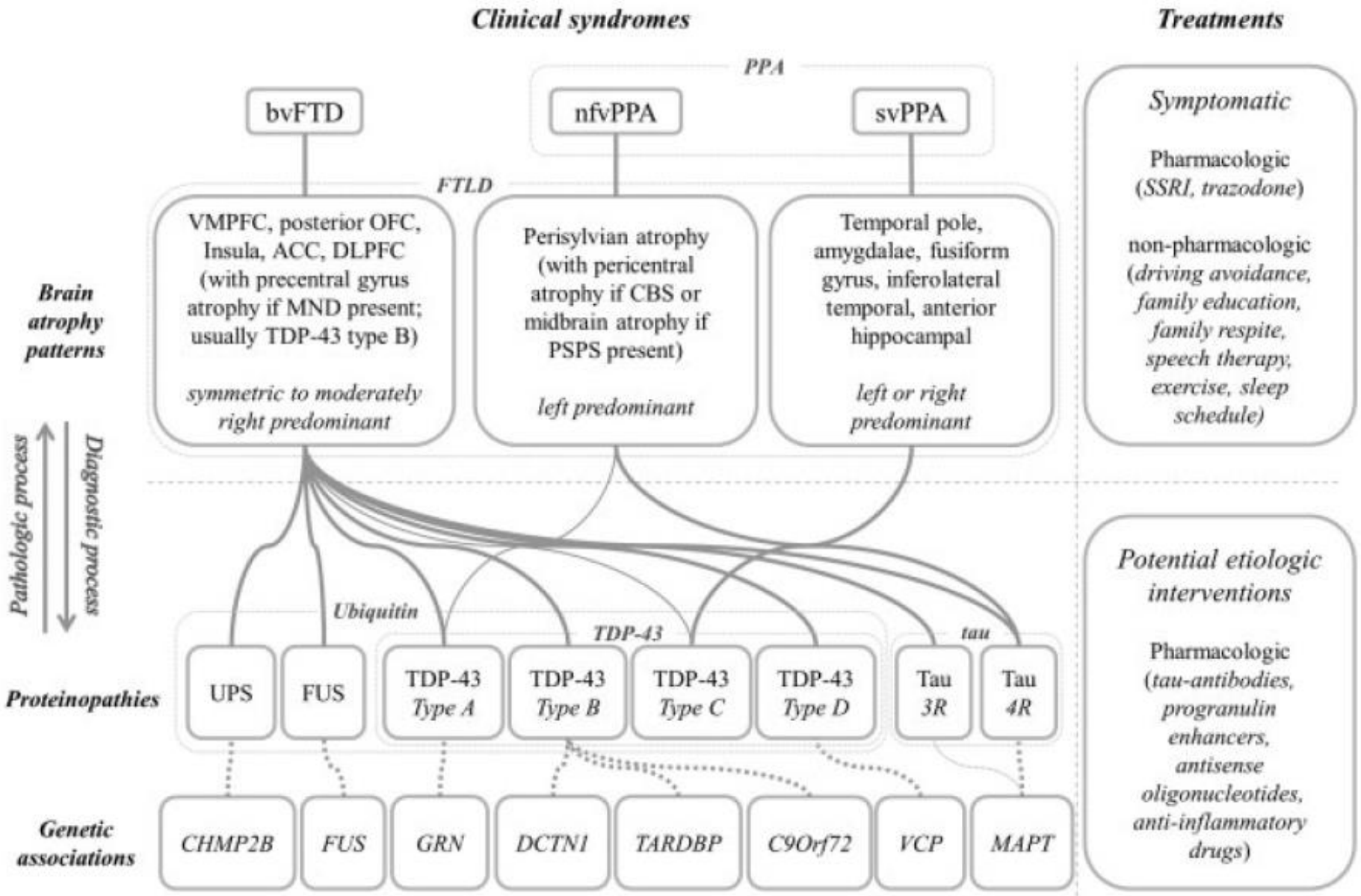


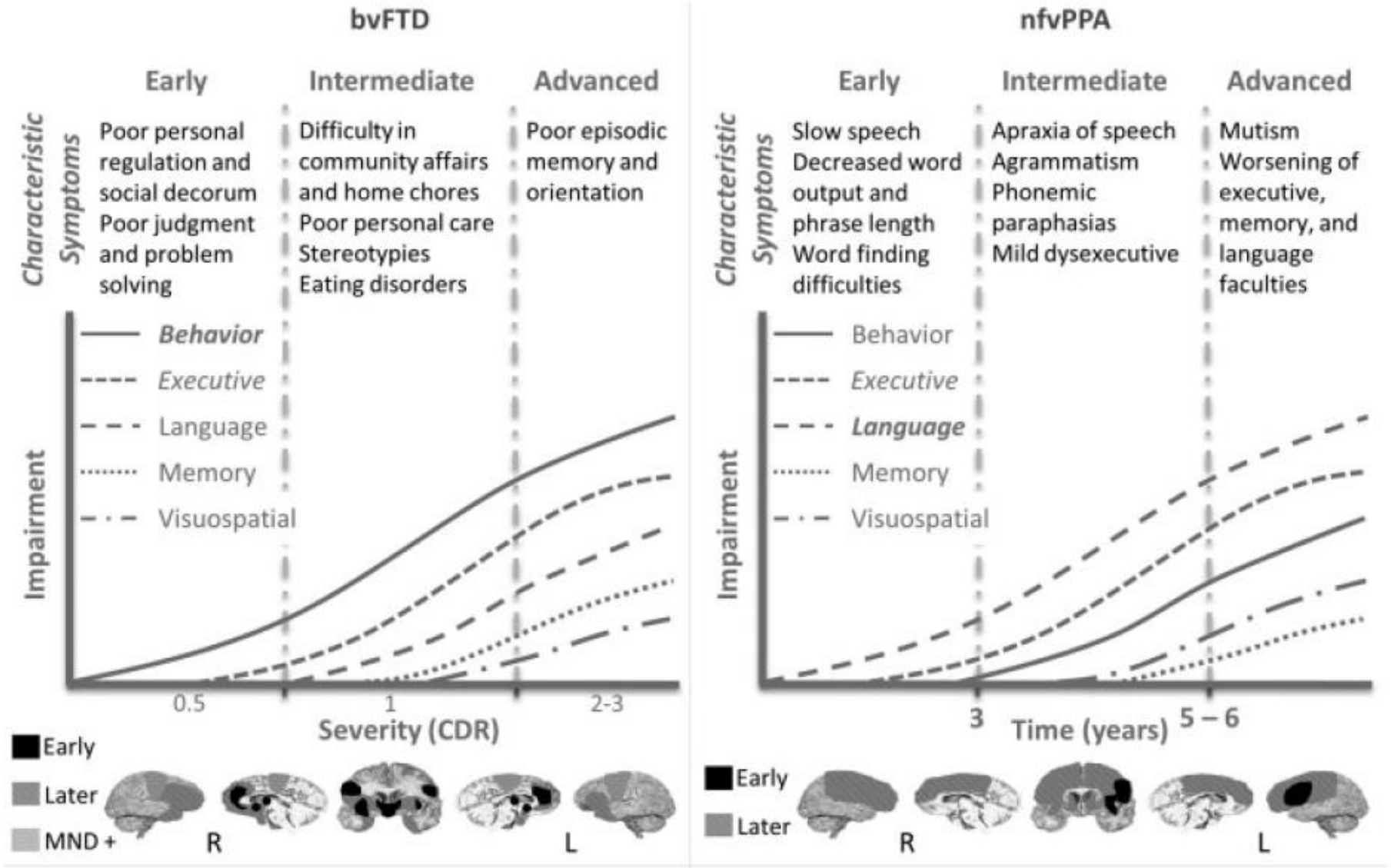
Frontotemporal Lobar Degeneration: A Clinical Approach

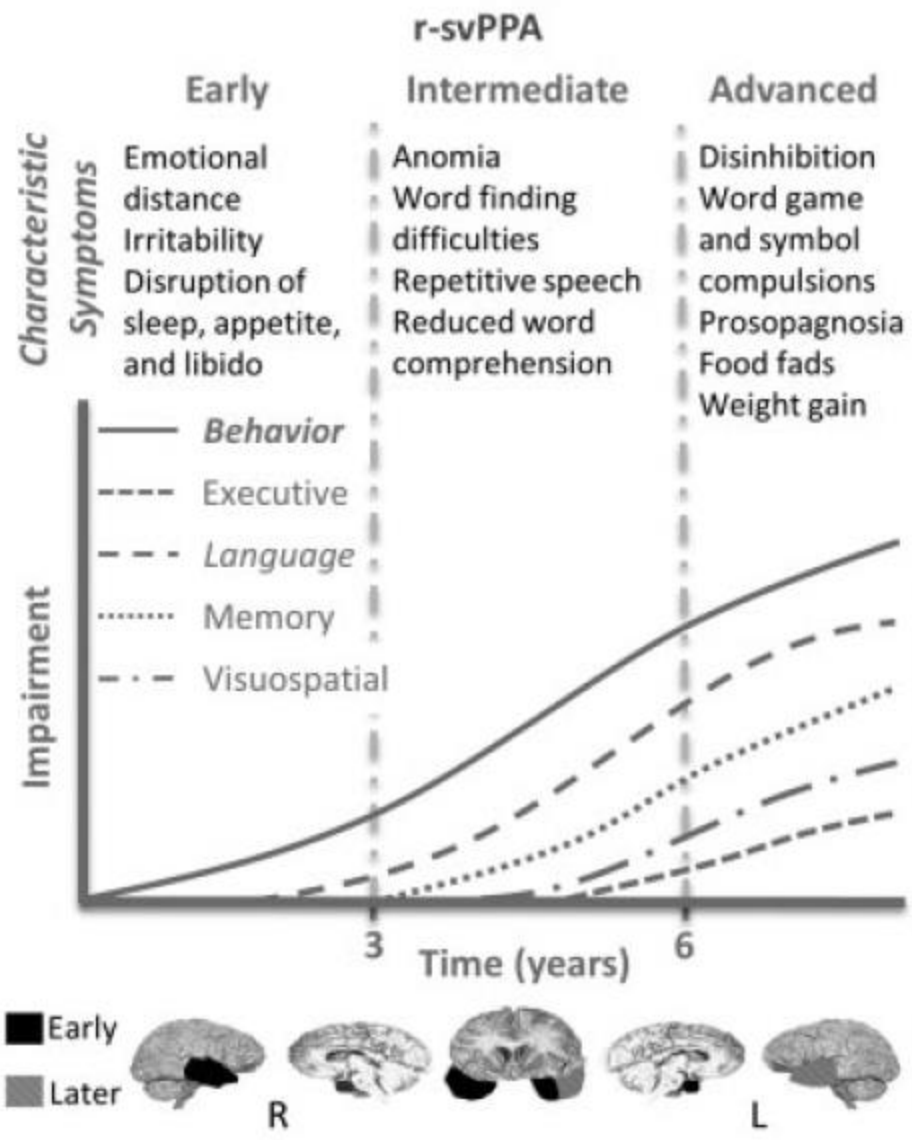
Elissaios Karageorgiou, MD[1,2] Bruce L. Miller, MD[1]

In this review, the authors outline a clinical approach to frontotemporal lobar degeneration (FTLD), a term coined to describe a pathology associated with atrophy of the frontal and temporal lobes commonly seen with abnormal protein aggregates. It accounts for ~10% of pathologically confirmed dementias. The three clinical syndromes associated with FTLD are jointly classified as frontotemporal dementia (FTD) and include behavioral variant frontotemporal dementia (bvFTD), nonfluent-agrammatic primary progressive aphasia (nfvPPA), and semantic variant PPA (svPPA; left: l-svPPA and right: r-svPPA). All syndromes have differential impairment in behavioral (bvFTD; r-svPPA), executive (bvFTD; nfvPPA), and language (nfvPPA; svPPA) functions early in the disease course. With all three there is relative sparing of short-term memory and visuospatial abilities early on, and with the two language syndromes, nfvPPA and svPPA, behavior is also intact. Symptoms are associated with specific atrophy patterns, lending unique imaging signatures to each syndrome (frontal: bvFTD and nfvPPA; temporal: svPPA). Common proteinopathies involve accumulation of tau, transactive response DNA binding protein 43, and fusion in sarcoma protein. Parkinsonism presents in all syndromes, especially cases with tau pathology and *MAPT* or *GRN* mutations. nfvPPA often has corticobasal degeneration or progressive supranuclear palsy as the underlying neuropathological substrate. bvFTD co-occurs with motor neuron disease in ~15% of cases, and many such cases are due to *C9Orf72* mutations. Other common genetic mutations in FTLD involve *GRN* and *MAPT*. Behavioral symptoms are best managed by selective serotonin reuptake inhibitors, while atypical antipsychotics should be used with caution given side effects. Promising etiologic treatments include anti-tau antibodies, antisense oligonucleotides, and progranulin enhancers.

Semin Neurol 2014;34:189–201


Clinical syndromes
Treatments
PPA
bvFTD
nfvPPA
svPPA
FTLD
Brain atrophy patterns
VMPFC, posterior OFC, Insula, ACC, DLPFC (with precentral gyrus atrophy if MND present; usually TDP-43 type B)
symmetric to moderately right predominant
Perisylvian atrophy (with pericentral atrophy if CBS or midbrain atrophy if PSPS present)
left predominant
Temporal pole, amygdalae, fusiform gyrus, inferolateral temporal, anterior hippocampal
left or right predominant
Symptomatic
Pharmacologic (SSRI, trazodone)
non-pharmacologic (driving avoidance, family education, family respite, speech therapy, exercise, sleep schedule)
Pathologic process
Diagnostic process
Ubiquitin
TDP-43
tau
Proteinopathies
UPS
FUS
TDP-43 Type A
TDP-43 Type B
TDP-43 Type C
TDP-43 Type D
Tau 3R
Tau 4R
Potential etiologic interventions
Pharmacologic (tau-antibodies, progranulin enhancers, antisense oligonucleotides, anti-inflammatory drugs)
Genetic associations
CHMP2B
FUS
GRN
DCTN1
TARDBP
C9Orf72
VCP
MAPT


bvFTD
Early
Intermediate
Advanced
Characteristic Symptoms
Poor personal regulation and social decorum
Poor judgment and problem solving
Difficulty in community affairs and home chores
Poor personal care
Stereotypies
Eating disorders
Poor episodic memory and orientation
Behavior
Executive
Language
Memory
Visuospatial
Impairment
0.5
1
2-3
Severity (CDR)
Early
Later
MND +
R
L
nfvPPA
Early
Intermediate
Advanced
Characteristic Symptoms
Slow speech
Decreased word output and phrase length
Word finding difficulties
Apraxia of speech
Agrammatism
Phonemic paraphasias
Mild dysexecutive
Mutism
Worsening of executive, memory, and language faculties
Behavior
Executive
Language
Memory
Visuospatial
Impairment
3
Time (years)
5 – 6
Early
Later
R
L


r-svPPA
Early
Intermediate
Advanced
Characteristic Symptoms
Emotional distance
Irritability
Disruption of sleep, appetite, and libido
Anomia
Word finding difficulties
Repetitive speech
Reduced word comprehension
Disinhibition
Word game and symbol compulsions
Prosopagnosia
Food fads
Weight gain
Behavior
Executive
Language
Memory
Visuospatial
Impairment
3
6
Time (years)
Early
Later
R
L

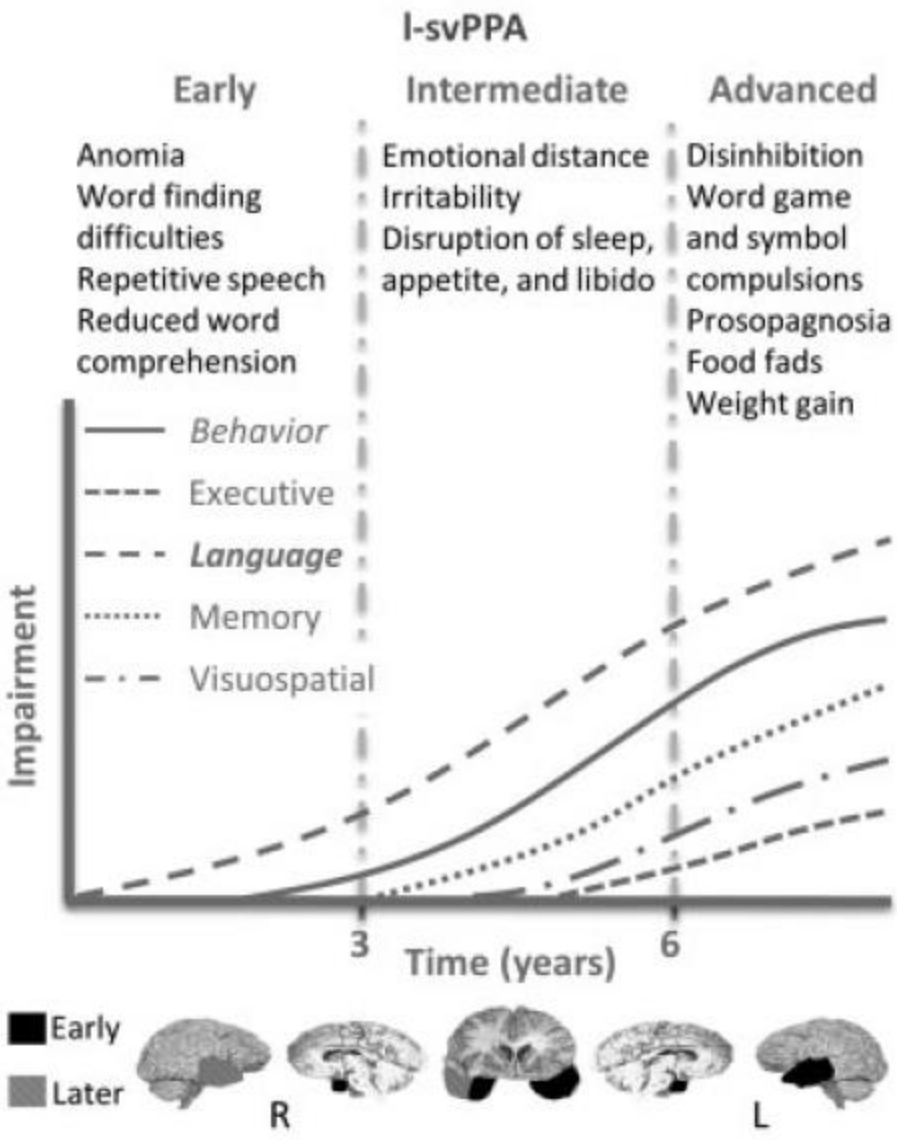

l-svPPA
Early
Intermediate
Advanced
Anomia
Word finding difficulties
Repetitive speech
Reduced word comprehension
Emotional distance
Irritability
Disruption of sleep, appetite, and libido
Disinhibition
Word game and symbol compulsions
Prosopagnosia
Food fads
Weight gain
Behavior
Executive
Language
Memory
Visuospatial
Impairment
3
6
Time (years)
Early
Later
R
L

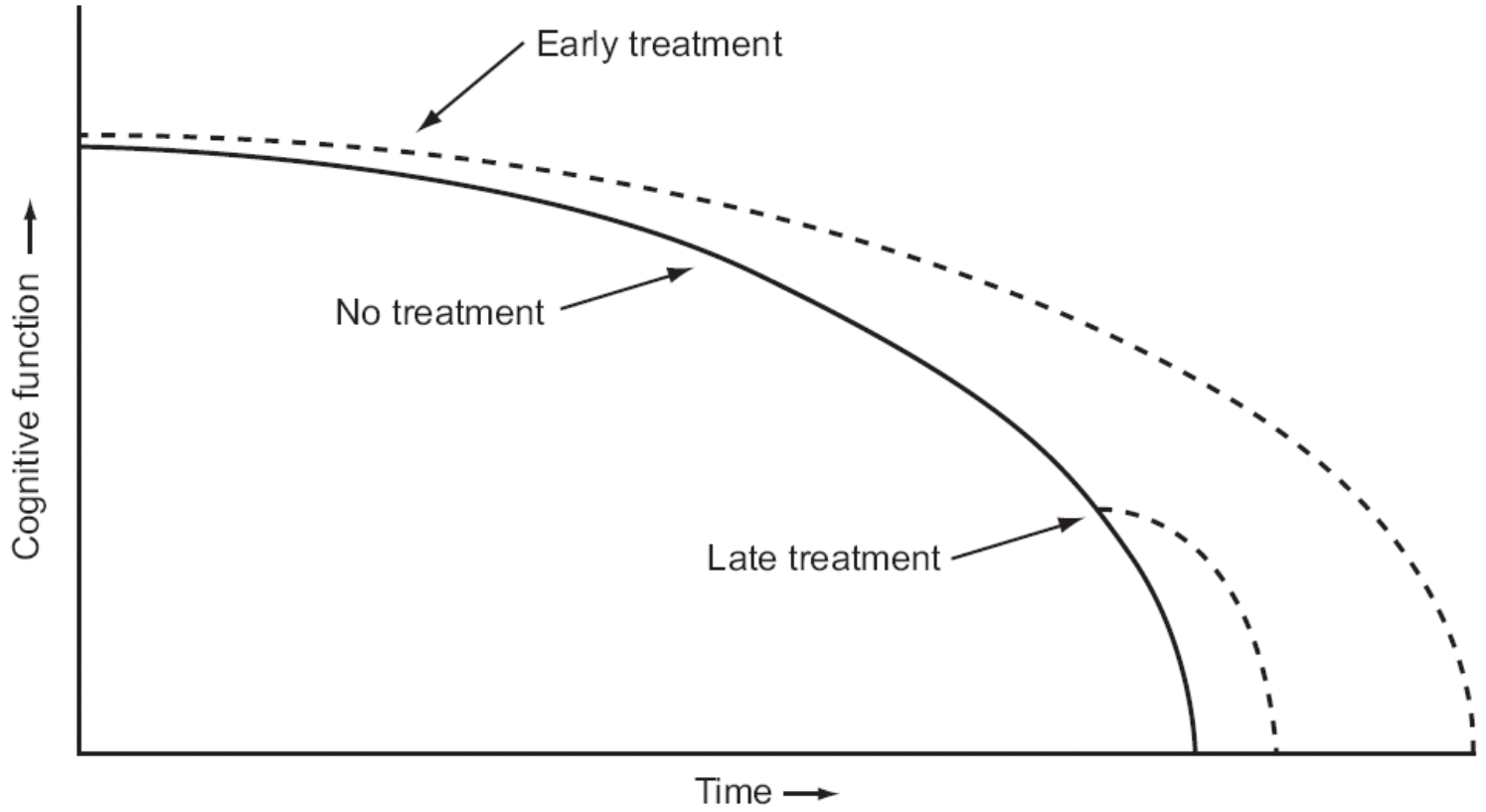
Table 5 Pharmacological treatments[64–82]

Medication	Dose	Population	Study designs	Combined study outcome	Side effects
Trazodone	Up to 300 mg daily	bvFTD	DB-CO-RCT	Improved behavior[a]	Fatigue, dizziness, hypotension
Fluvoxamine	50–150 mg daily	bvFTD, svPPA	OL	Improved stereotypies	Appetite loss
Paroxetine	Up to 40 mg daily	bvFTD	OL, OL-RCT, DB-CO-RCT	No definite behavioral benefit Improved mood, compulsions, and eating disorders	Well tolerated
Fluoxetine	20 mg daily	bvFTD	OL	Improved mood, compulsions, and eating disorders	Well tolerated
Sertraline	50–125 mg daily	bvFTD	OL-CT, OL	Improved stereotypies	Well tolerated
Citalopram	40 mg daily	bvFTD	OL	Improved behavior	Well tolerated
Donepezil	Up to 10 mg daily	bvFTD	OL, DC	No benefit	Worse behavioral symptoms
Galantamine	Up to 24 mg daily	bvFTD, PPA	OL to DB-RCT	No benefit	Mild GI symptoms
Rivastigmine	Up to 9 mg daily	bvFTD	OL-CT	Improved behavior	Well tolerated
Quetiapine	Up to 150 mg total daily dose	bvFTD, nfvPPA, svPPA	DB-CO-RCT	No definite benefit	Somnolence
Olanzapine	Up to 10 mg daily	bvFTD	OL	Improved agitation and anxiety	Somnolence, mild GI symptoms
Bromocriptine	Up to 7.5 mg 3 times daily	PPA	DB-CO-RCT	No benefit	Rare frustration intolerance
Methylphenidate	40 mg once	bvFTD	DB-CO-CT	Improved decision making within a few hours	Non-significant blood pressure increase
Dextroamphetamine	20 mg total daily dose	bvFTD, nfvPPA, svPPA	DB-CO-RCT	Improved behavior	Well tolerated
Memantine	Up to 20 mg daily	bvFTD, nfvPPA, svPPA	OL, DB-RC	No benefit	Well tolerated

Lo stato dell’arte

Curare con i farmaci disponibili

INIZIARE PRESTO LA CURA



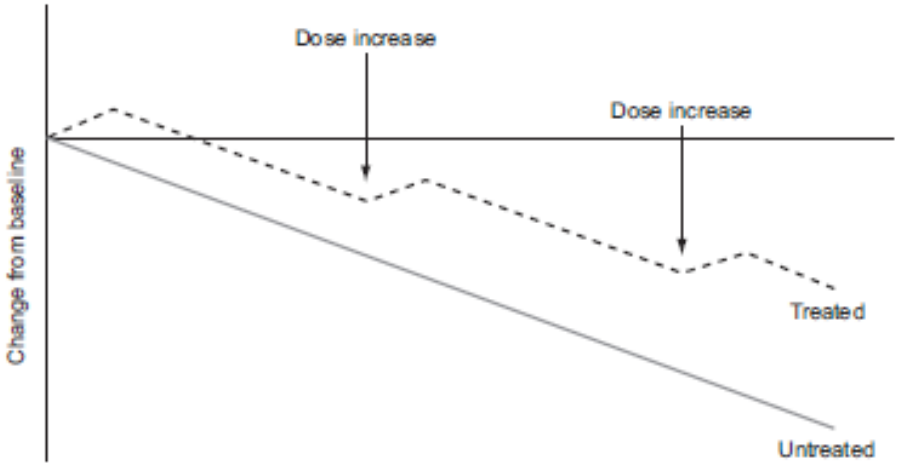
Alzheimer's & Dementia 7:177-184, 2011

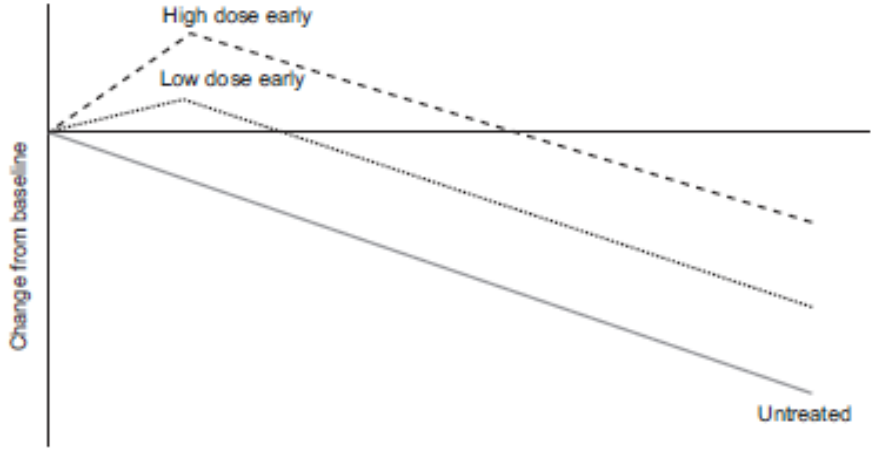
RAGGIUNGERE I MASSIMI DOSAGGI

Review Article

Defining optimal treatment with cholinesterase inhibitors in Alzheimer's disease

Gary Small[a]*, Roger Bullock[b]





Alzheimer's & Dementia 7: 177-184, 2011

SOSTITUIRE I FARMACI

BAP Guidelines

Clinical practice with anti-dementia drugs: a revised (second) consensus statement from the British Association for Psychopharmacology

John T O'Brien[1], Alistair Burns[2],

on behalf of the BAP Dementia Consensus Group

Psychopharm

Journal of Psychopharmacology
25(8) 997–1019

DOI: 10.1177/0269881110387547
jop.sagepub.com
SAGE

Table 3. Summary box: Alzheimer's disease

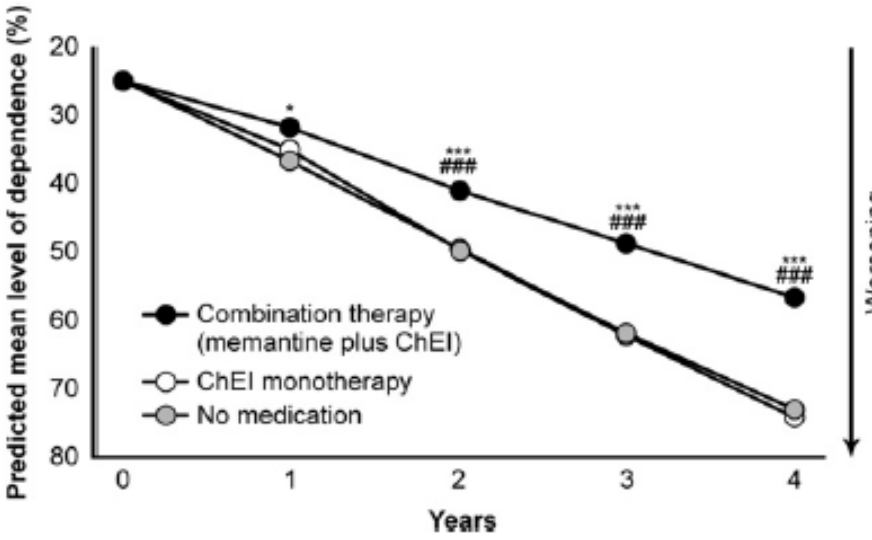
Intervention	Level of evidence	Recommendation
Treatment with cholinesterase inhibitors and memantine	There is type I evidence for the efficacy of cholinesterase inhibitors in the treatment of mild to moderate Alzheimer's disease and type I evidence for memantine in moderate to severe Alzheimer's disease.	A
Switching between cholinesterase inhibitors	There is type II evidence to support the switching of one cholinesterase inhibitor to another if the first is not tolerated or effective.	B
Combination therapy	There is type II evidence for adding memantine to a cholinesterase inhibitor, but also a negative type 1b study. Until further studies are available the benefits of combination therapy is unclear.	B

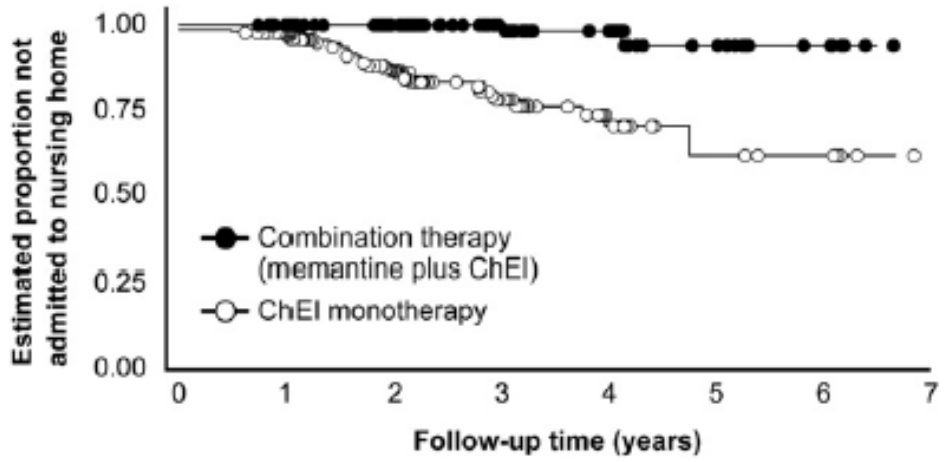
O'Brien JT, Burns A. Clinical practice with anti-dementia drugs: a revised (second) consensus statement from the British Association for Psychopharmacology. J Psychopharmacol 2011; 25: 997-1019

ASSOCIARE LA MEMANTINA

Benefits of combined cholinesterase inhibitor and memantine treatment in moderate–severe Alzheimer's disease

Serge Gauthier[a,*], José L. Molinuevo[b]

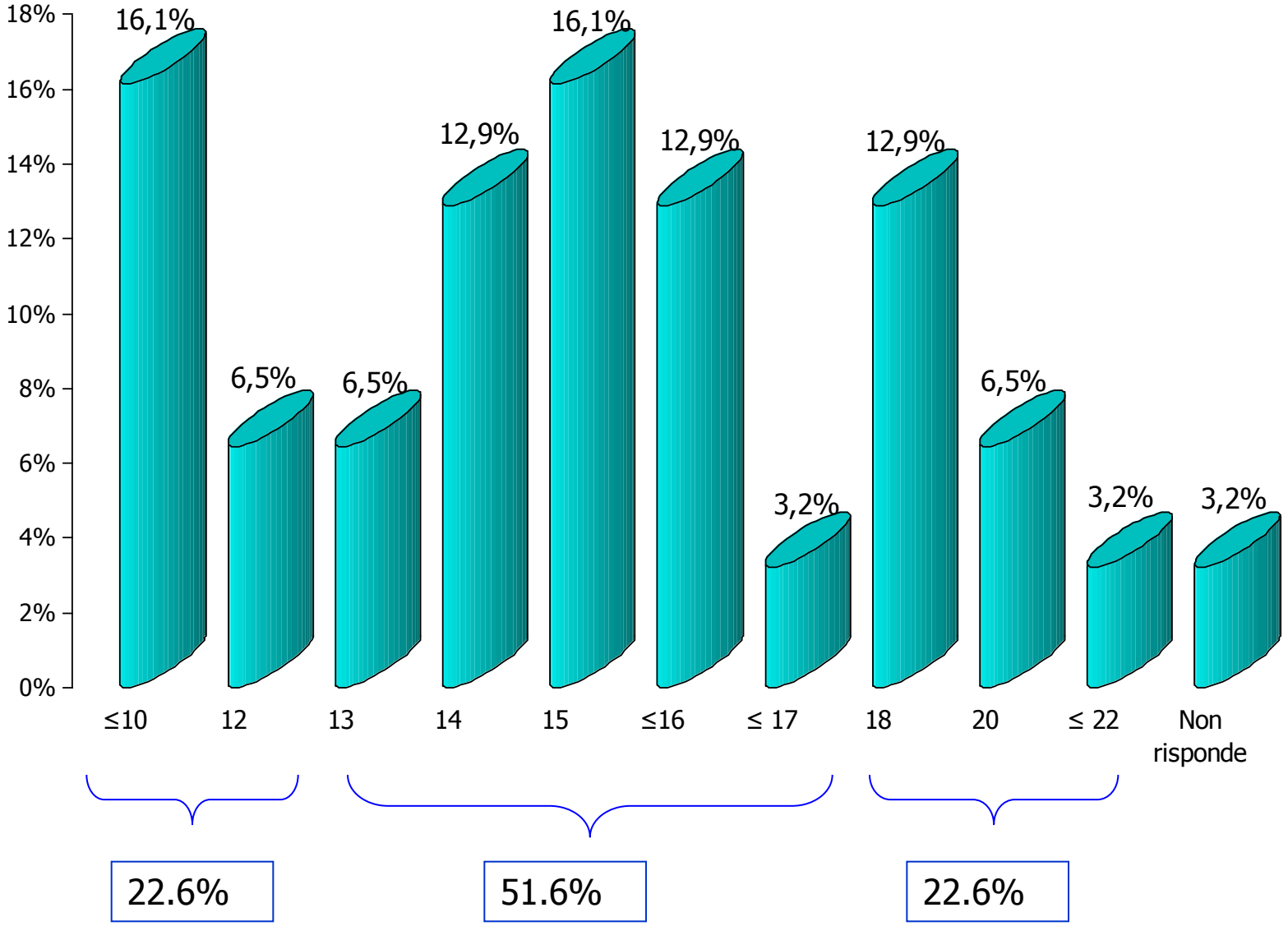




Alzheimer's & Dementia - (2012) 1–6

ASSOCIAZIONE ITALIANA PSICOGERIATRIA
Sezione Regionale Campana

A quale valore di MMSE è solito introdurre la Memantina?

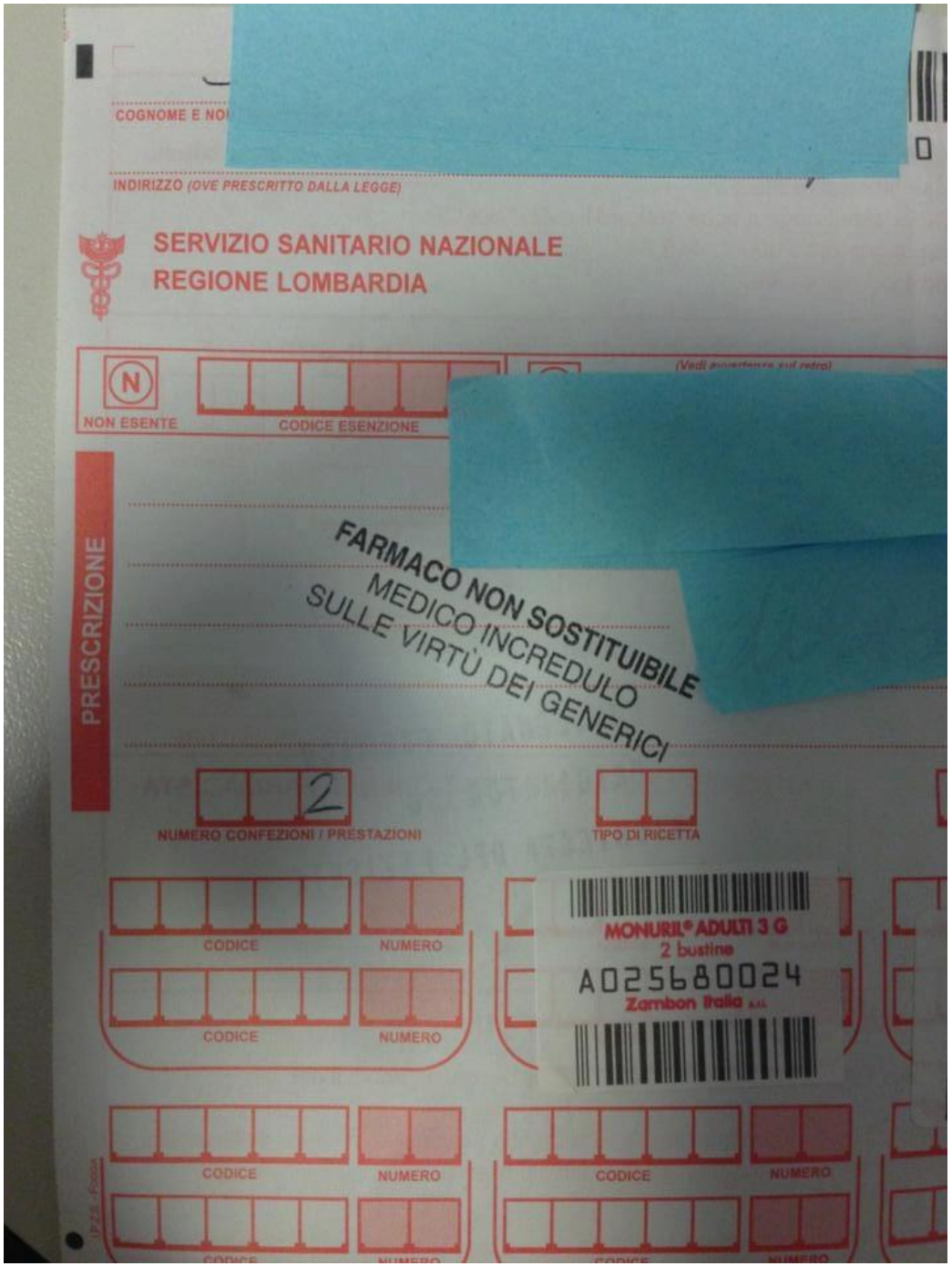


AntiChe e memantina:
prescrizione in Campania

- Napoli differenza tra i distretti
- Salerno e provincia interdetta la doppia fornitura
- Caserta segue regole di prescrizione off-label

VALUTARE LA RISPOSTA AL GENERICO

- Donepezil
- Rivastigmina
- Memantina
- Quetiapina
- Risperidone

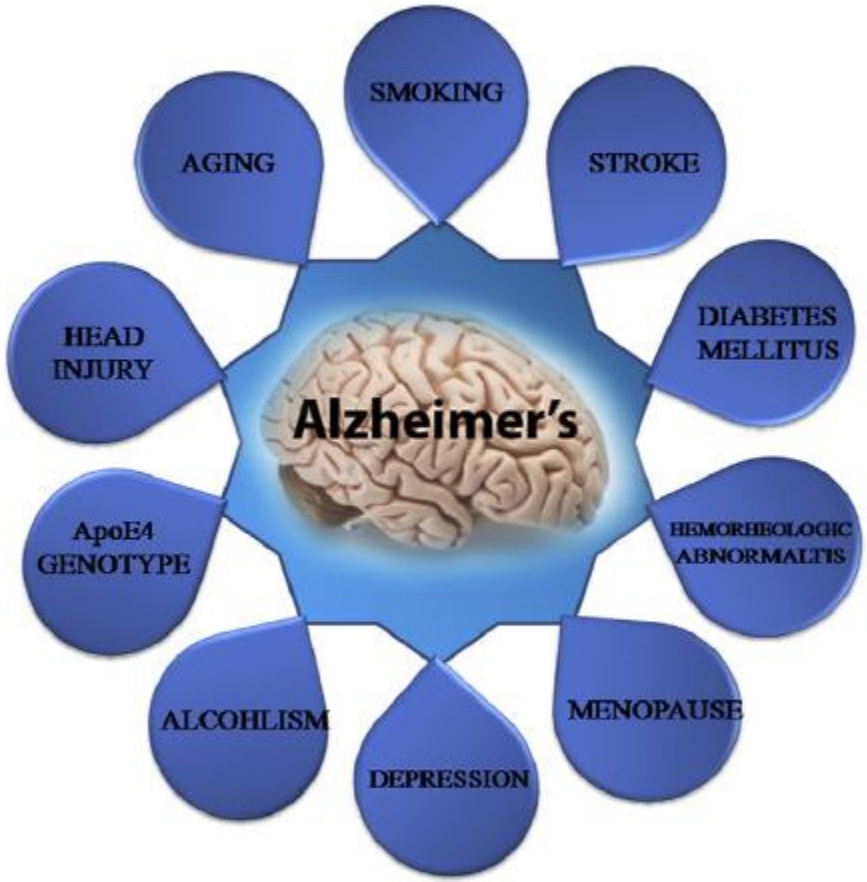

COGNOME E NO
INDIRIZZO (OVE PRESCRITTO DALLA LEGGE)
SERVIZIO SANITARIO NAZIONALE
REGIONE LOMBARDIA
N
NON ESENTE
CODICE ESENZIONE
PRESCRIZIONE
FARMACO NON SOSTITUIBILE
MEDICO INCREDULO
SULLE VIRTÙ DEI GENERICI
2
NUMERO CONFEZIONI / PRESTAZIONI
TIPO DI RICETTA
CODICE
NUMERO
MONURIL® ADULTI 3 G
2 bustine
A025680024
Zambon Italia s.r.l.

Conclusioni

Disturbo di memoria

Disturbo in altre aree cognitive e non cognitive

Riduzione attività di vita quotidiana



CATTEDRA DI GERIATRIA

ASSOCIAZIONE ITALIANA PSICOGERIATRIA
Sezione Regionale Campana

CATTEDRA DI NEUROLOGIA

La memoria: aspetti neuropsicologici e clinici

NAPOLI 18 DICEMBRE 2014

Aula Magna - Univ. Federico II
Via Partenope, 36

VI CORSO DI FORMAZIONE IN PSICOGERIATRIA