ILTCI

CONSIDERATIONS IN COGNITIVE UNDERWRITNG:
An Introduction to Two Concepts

Cognitive Reserve &
Vascular Cognitive Impairment

Practical Observation

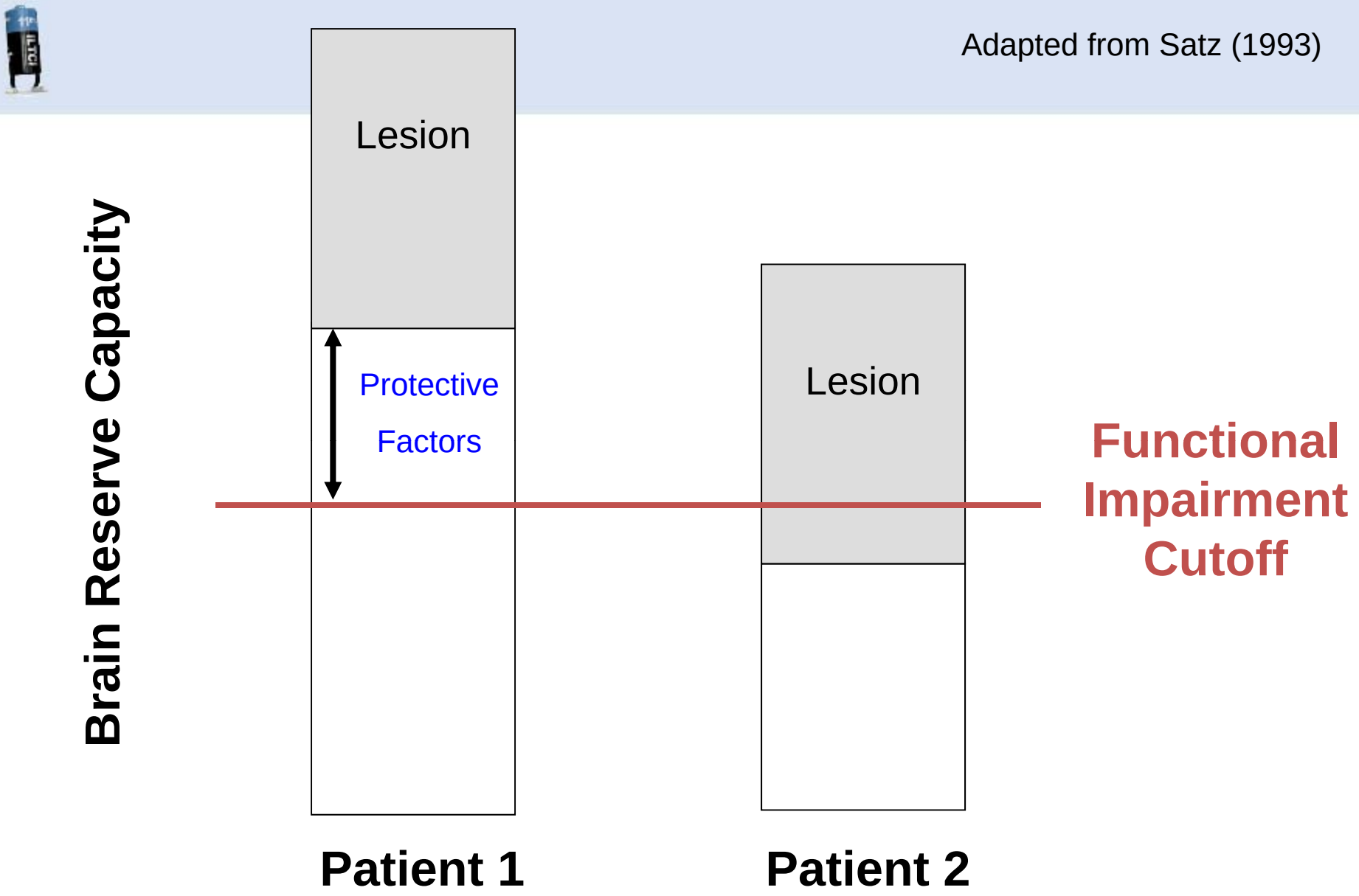
We don’t always see the cognitive impairment that we would expect given the amount of brain pathology present.

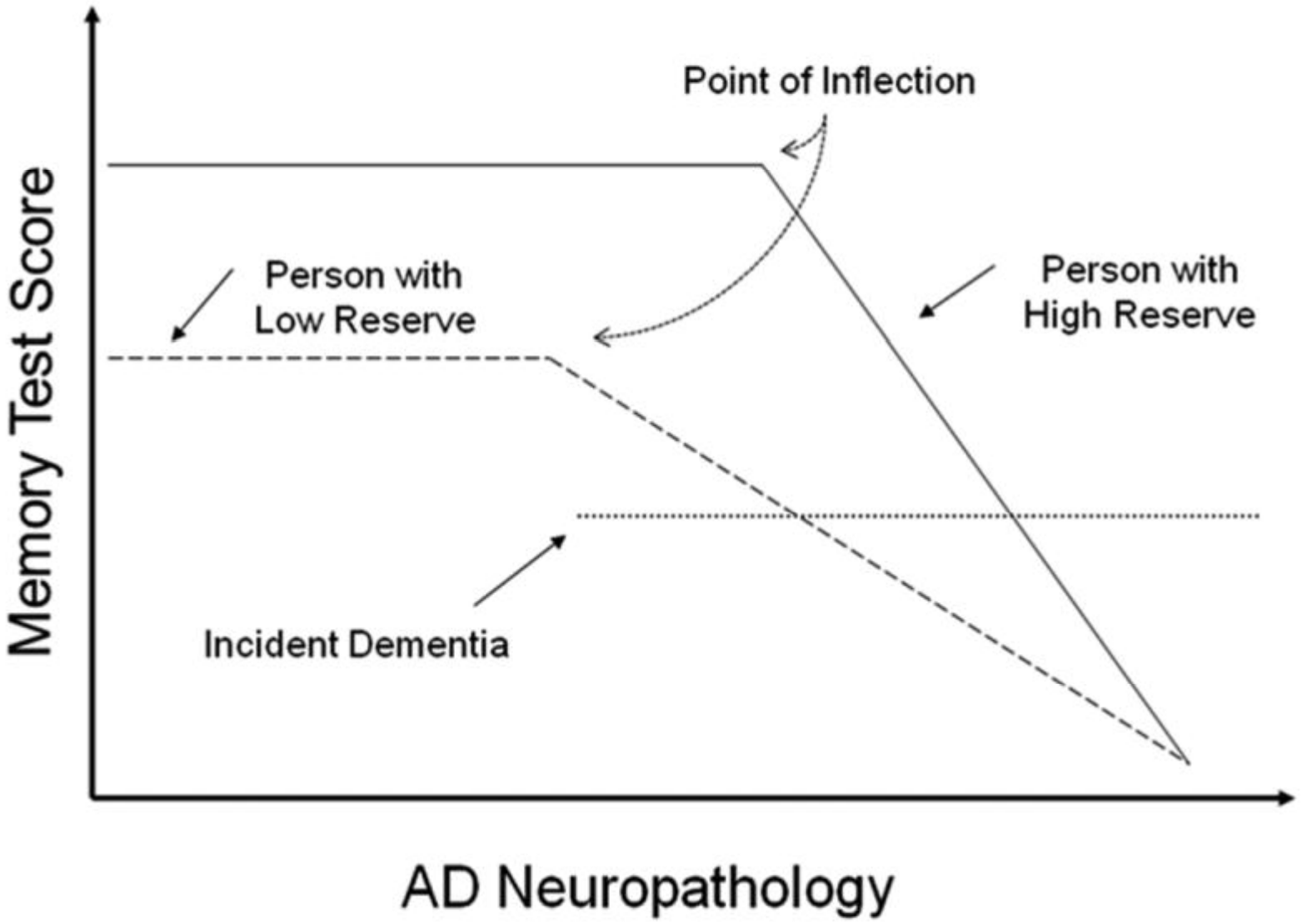
Cognitive Reserve

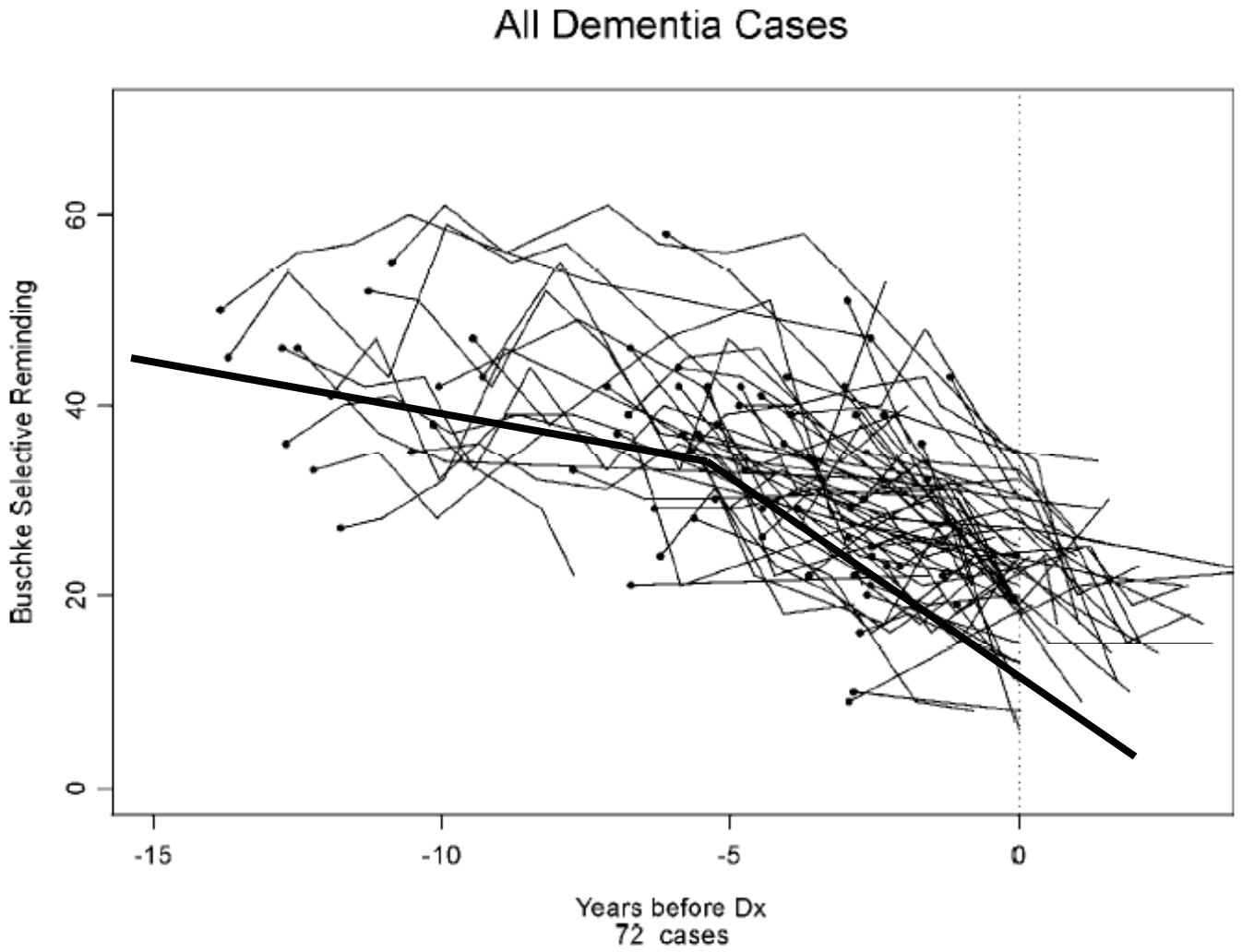
- **ReserveThreshold** Summarized by Satz (1993)
- Hypothetical construct: “Brain Reserve Capacity” (BRC)
- Concrete examples of BRC: neuronal count
- There is individual variation in BRC
- Education is one index of BRC
- There is a critical threshold of BRC. Once depleted past this critical point, specific clinical or functional deficits emerge.

Cognitive Reserve

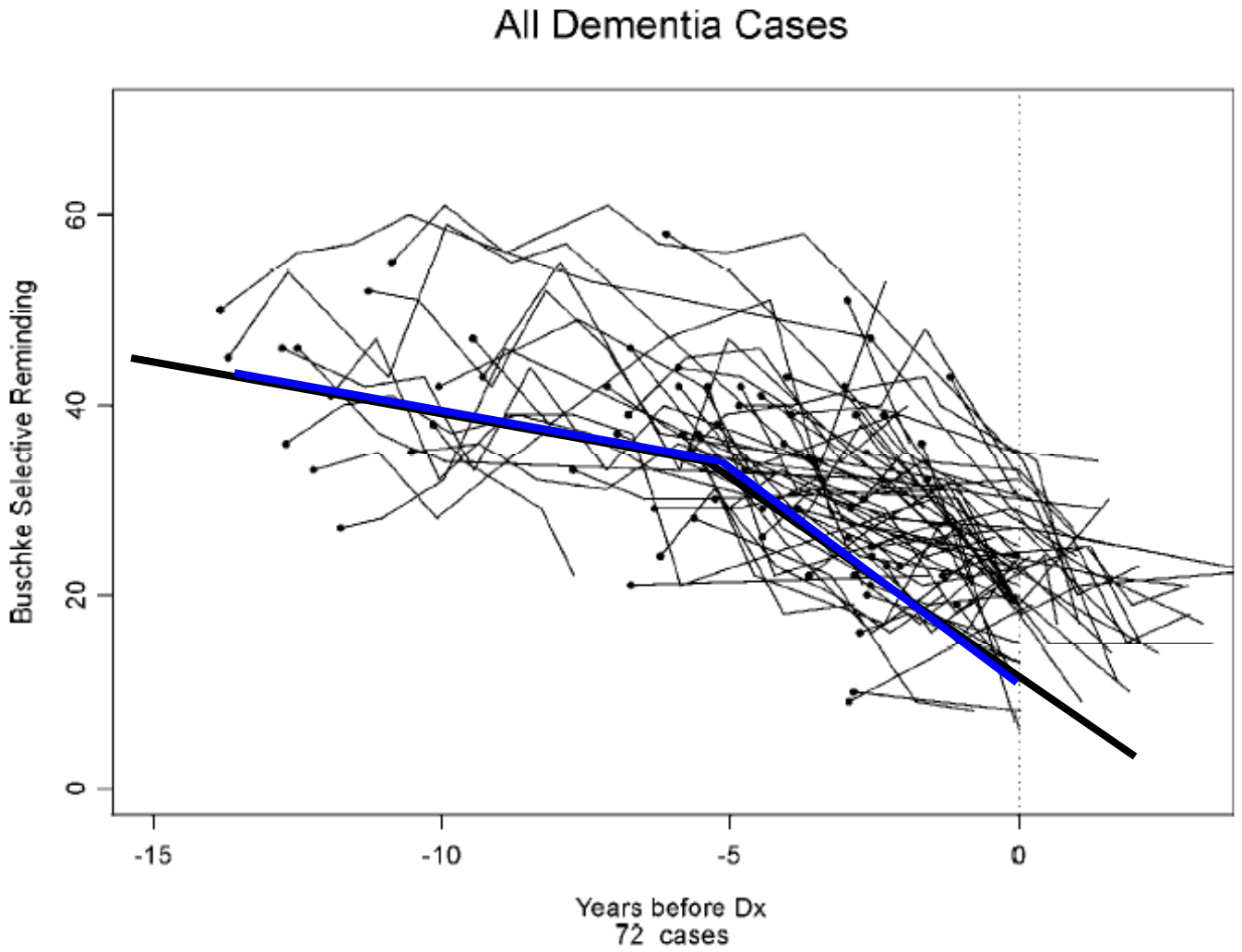
- Two individuals who appear the same clinically, whether demented or non-demented, can have widely divergent levels of underlying age-related neural changes or AD pathology.
- Thus, the clinical diagnosis of normal aging, MCI or AD may be accompanied by very minimal pathology or more than enough to meet pathological criteria for AD.
- Understanding CR therefore becomes an important component of diagnosing and characterizing aging and dementia.


Adapted from Satz (1993)
Brain Reserve Capacity
Lesion
Protective
Factors
Lesion
Functional
Impairment
Cutoff
Patient 1
Patient 2


Point of Inflection
Person with
Low Reserve
Person with
High Reserve
Incident Dementia
Memory Test Score
AD Neuropathology

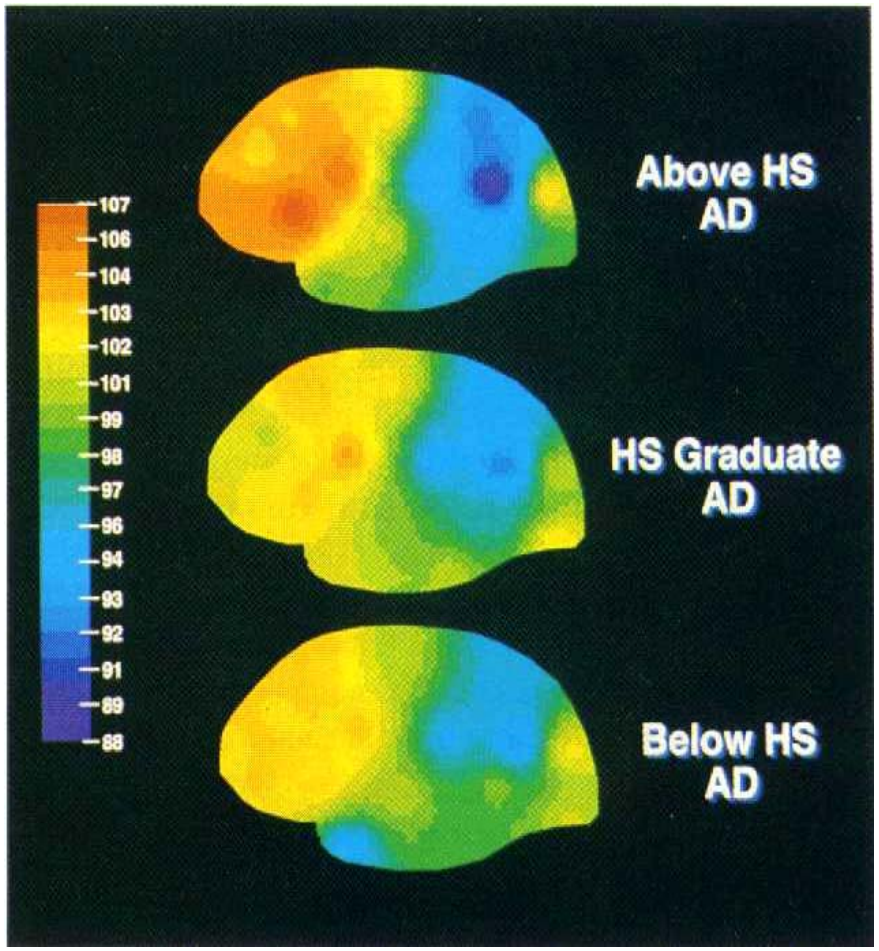


Rate of memory decline increases 5.1 years before dementia diagnosis (Hall et al, 2000)



Rate of memory decline increases 5.1 years before dementia diagnosis (Hall et al, 2000)

Education and rCBF

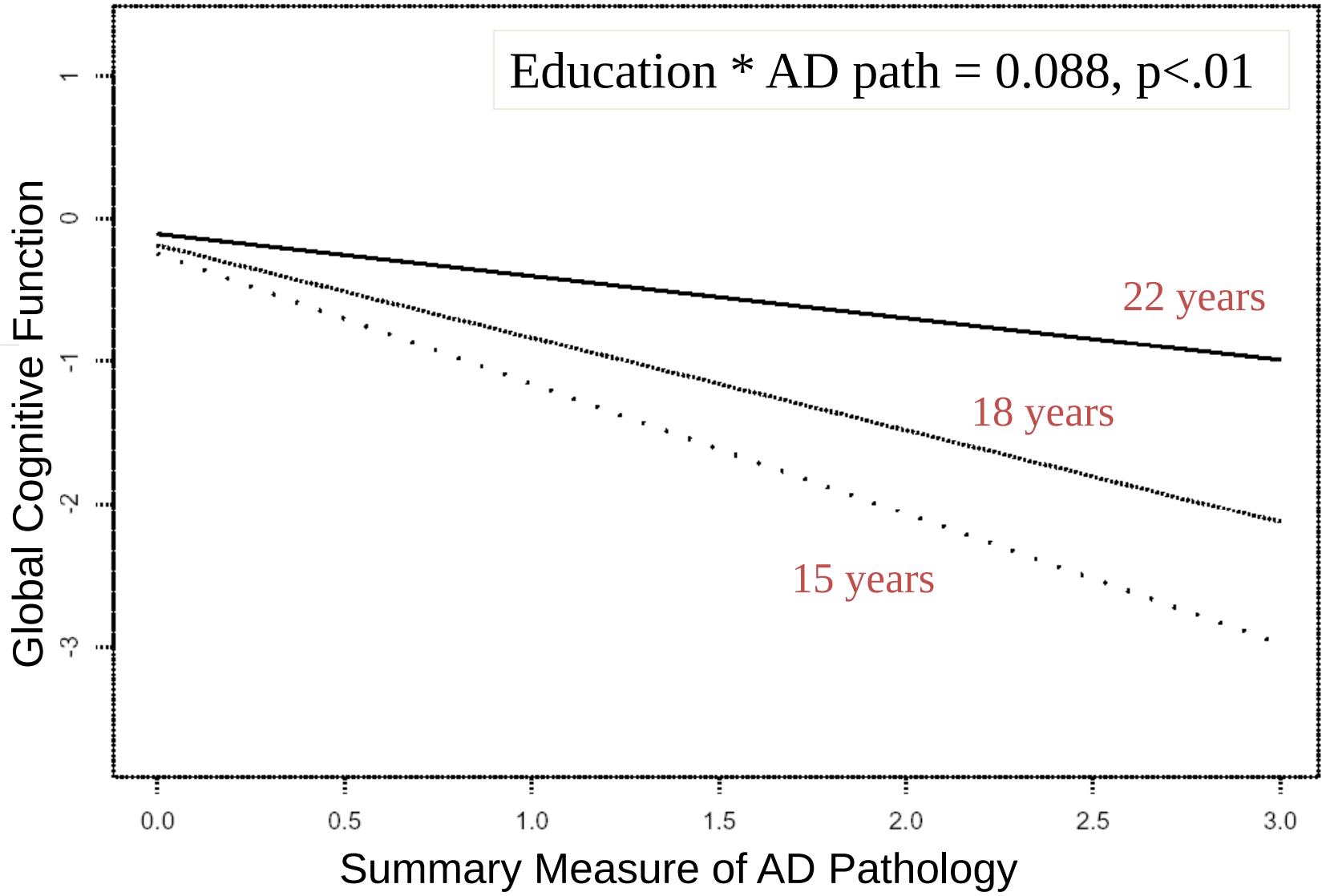


Controlling for clinical disease severity, there is an inverse relationship between education and a functional imaging proxy for AD pathology

Similar findings have been noted for occupational attainment

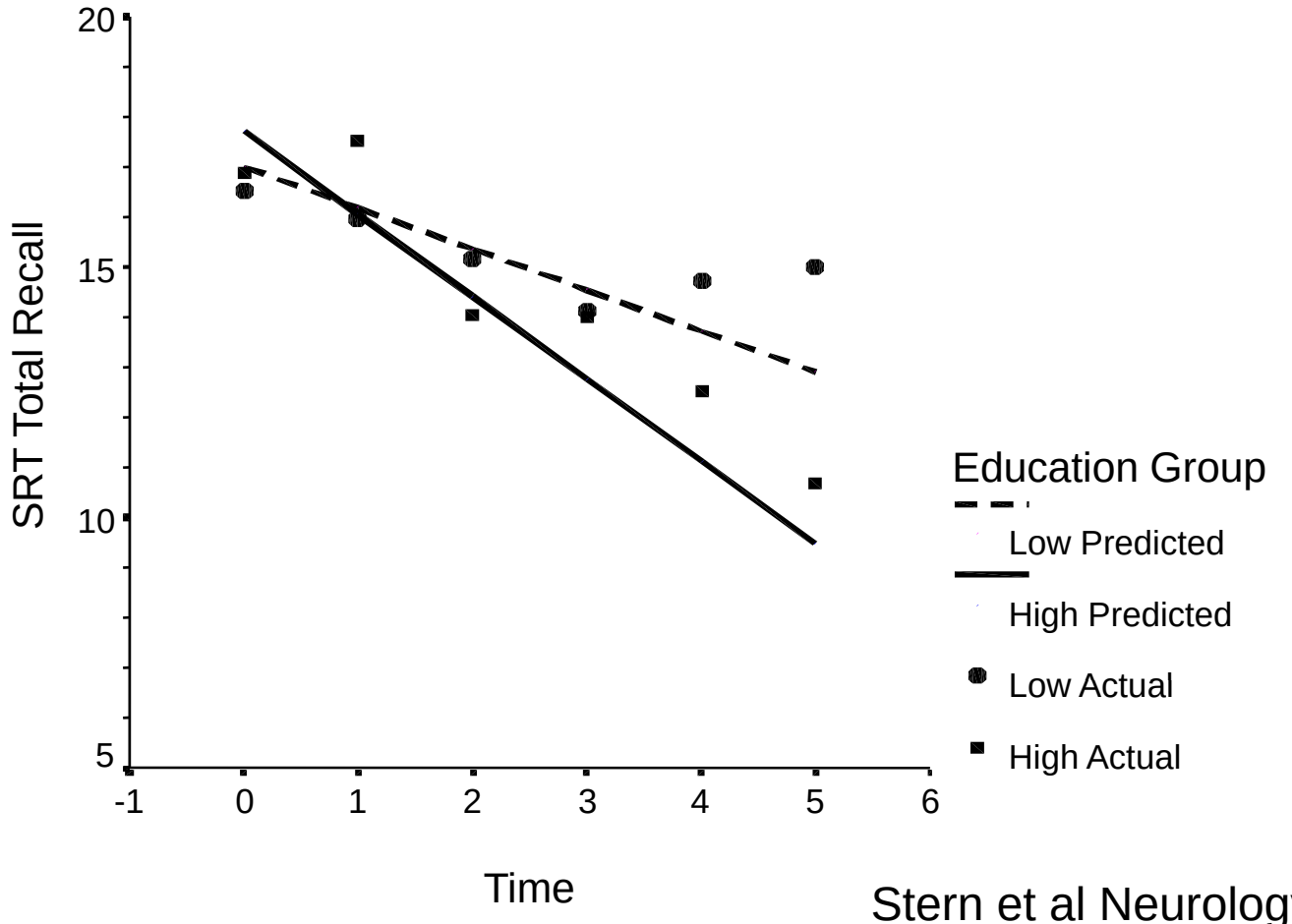
Stern et al, Ann Neurol 2004

Interaction of AD Pathology and Education



Bennett DA et al. Neurology 2003

More rapid memory decline in AD patients with higher educational attainment

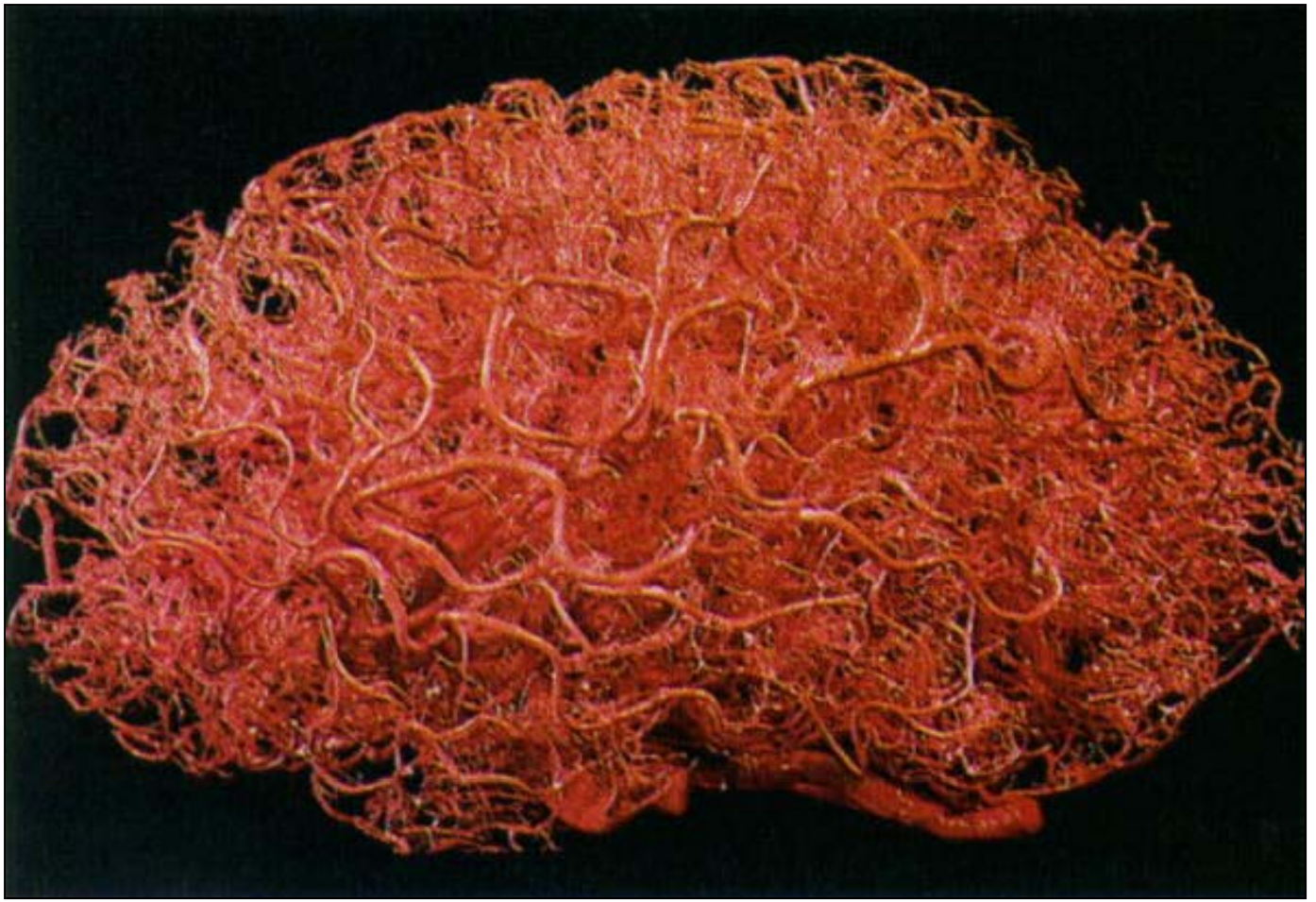


Stern et al Neurology 1999

Practical Observation

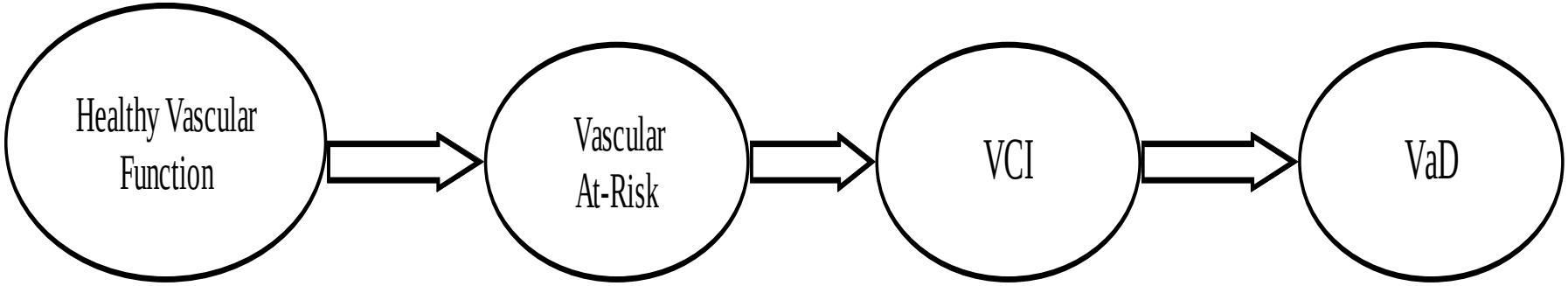
Not all cognitive impairment relates to Dementia of the Alzheimer's Type.

Cerebrovascular Changes



Zlokovic and Apuzzo, 1998

Vascular Cognitive Impairment Continuum



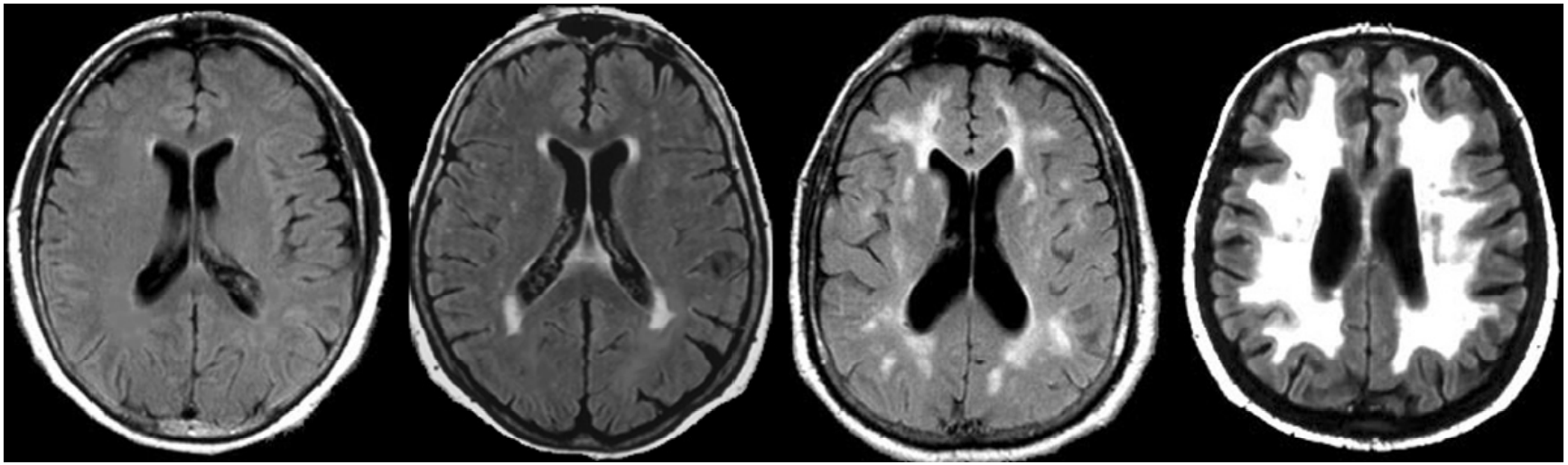
Practical Observation

- Vascular changes can result in unique patterns of cognitive impairment.
- Vascular changes also account for one third of the risk for Dementia of the Alzheimer's Type.
- Vascular changes may be associated with no impairment (yet).

Terms

- White matter abnormalities
- White matter lesions
- White matter changes
- Small vessel disease
- Small vessel ischemic disease
- Microangiopathy
- Leukoariosis
- Very small infarcts
- T2 flair hyperintensities

LA on Magnetic Resonance Imaging



Normal Mild Moderate Severe

From Malloy et al, 2006

Comparison of stroke & brain microvascular disease

Characteristic	**Stroke (arterial)**	**Microvascular disease (arteriolar)**
Onset/progression	sudden/brief if any	ill-defined/gradual over years
Manifestations	focal neurologic deficit	*functional* impairment
Location	vascular distribution	grow from head/tail-lateral ventricles
Size	stroke(cm)→lacune (mm)	<1 mm
Vessel	large to small artery	arteriolar
Pathogenesis - mechanism of tissue damage	vascular disease risk factors - ischemic	vascular disease risk factors - unclear

Small Vessel Disease

Small vessel disease (SVD) or microangiopathy is mediated by risk factors such as hypertension and diabetes.

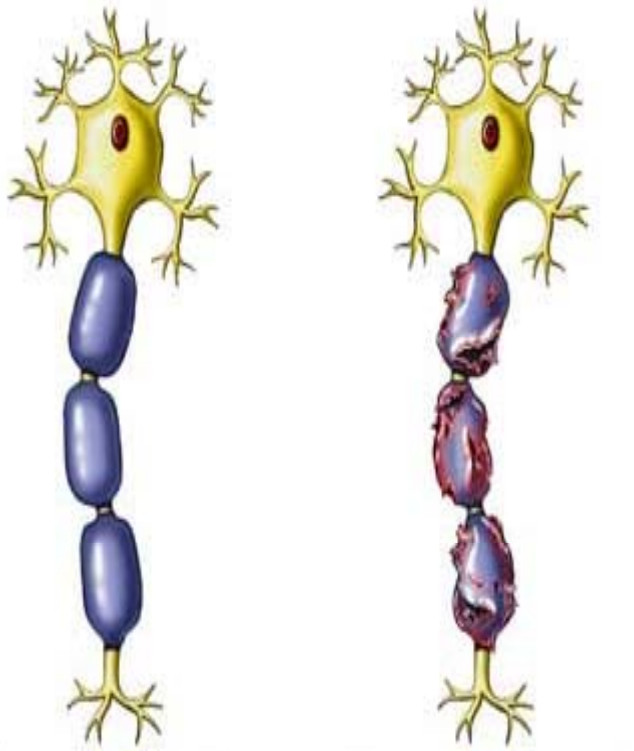
Related to ischemic damage of varying degrees that has been caused by injury to the penetrating vessels that supply the basal ganglia, thalamus, putamen, caudate subcortical white matter and internal capsule.

This is cerebrovascular disease

Pathology

Originally it was suggested that selective demyelination might occur as a result of ‘‘incomplete’’ ischemia,

but a single electron microscopy study showed that pallor was largely due to loss of nerve fibres in their entirety.



Neuron with myelin sheath

Neuron with damaged myelin sheath

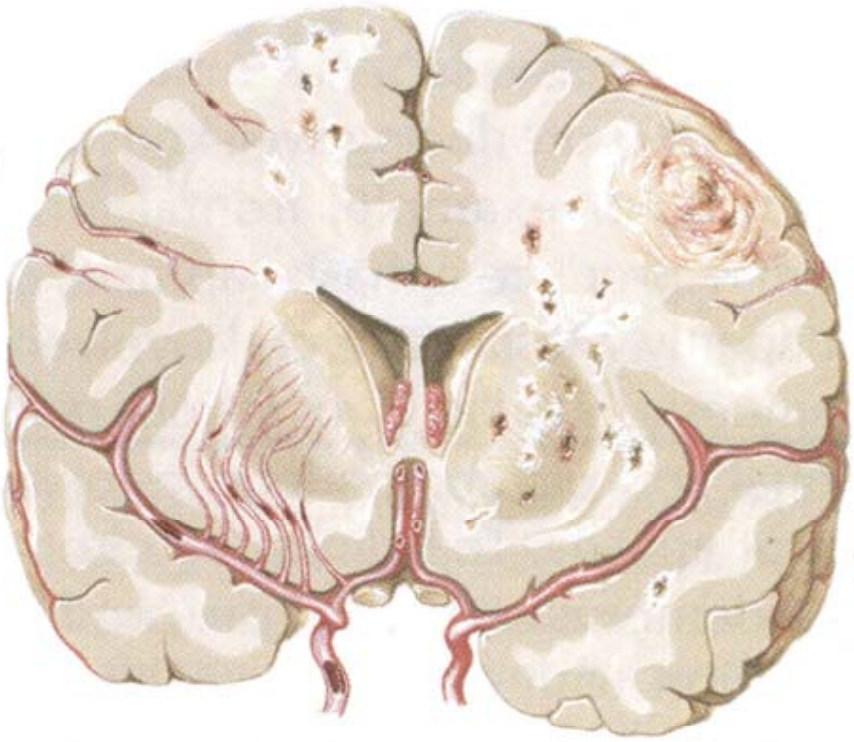
White Matter Changes with Age

- The majority of age-related prefrontal brain volume losses are associated with reductions in white matter not gray matter
- Myelin volume and integrity reaches a peak by mid-age and then declines with advancing age
- Raz (2000) proposed that age-related vulnerability in frontal areas may reflect the distributed pattern of white matter during development and follows the rule of “last in, first out”

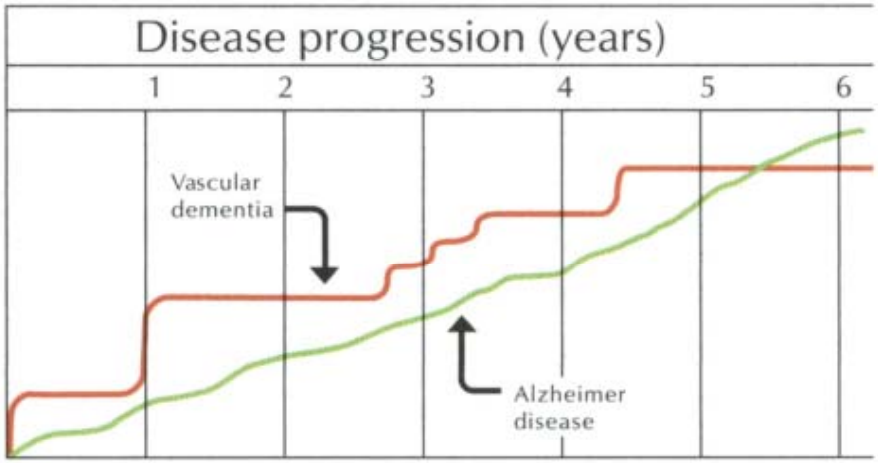
WMH Changes with Age and Vascular Disease

- WMH in the frontal lobe are present in healthy middle aged and elderly
- WMH volume is related to vascular health
- Over 5 years WMH loads increased:
  - 2.6% year in healthy adults
  - 9.6% year in adults with VD
- Lesion load may not correlate with funtional (cognitive) impairment

Raz et al., Neuropsychology, 2007;21:49



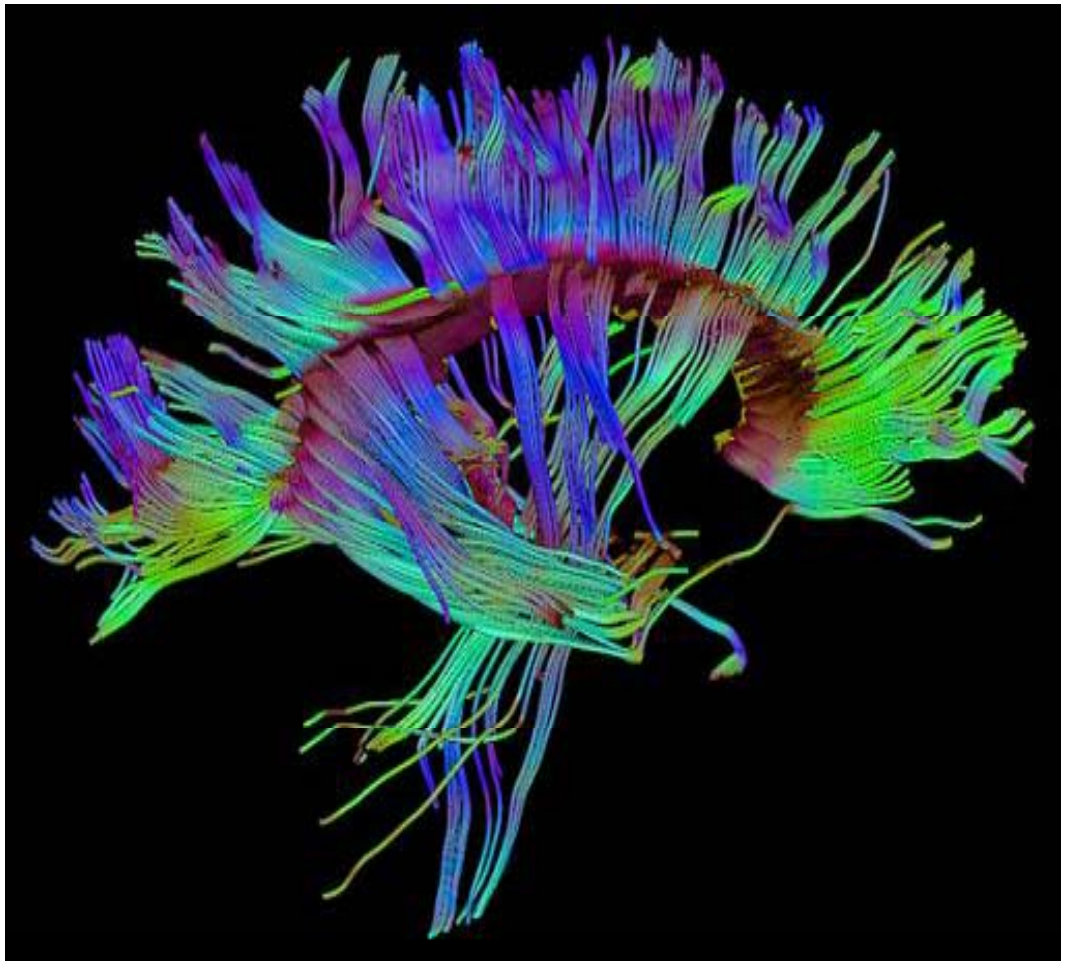
Cerebrovascular disease results in multiple small cortical and subcortical infarcts.



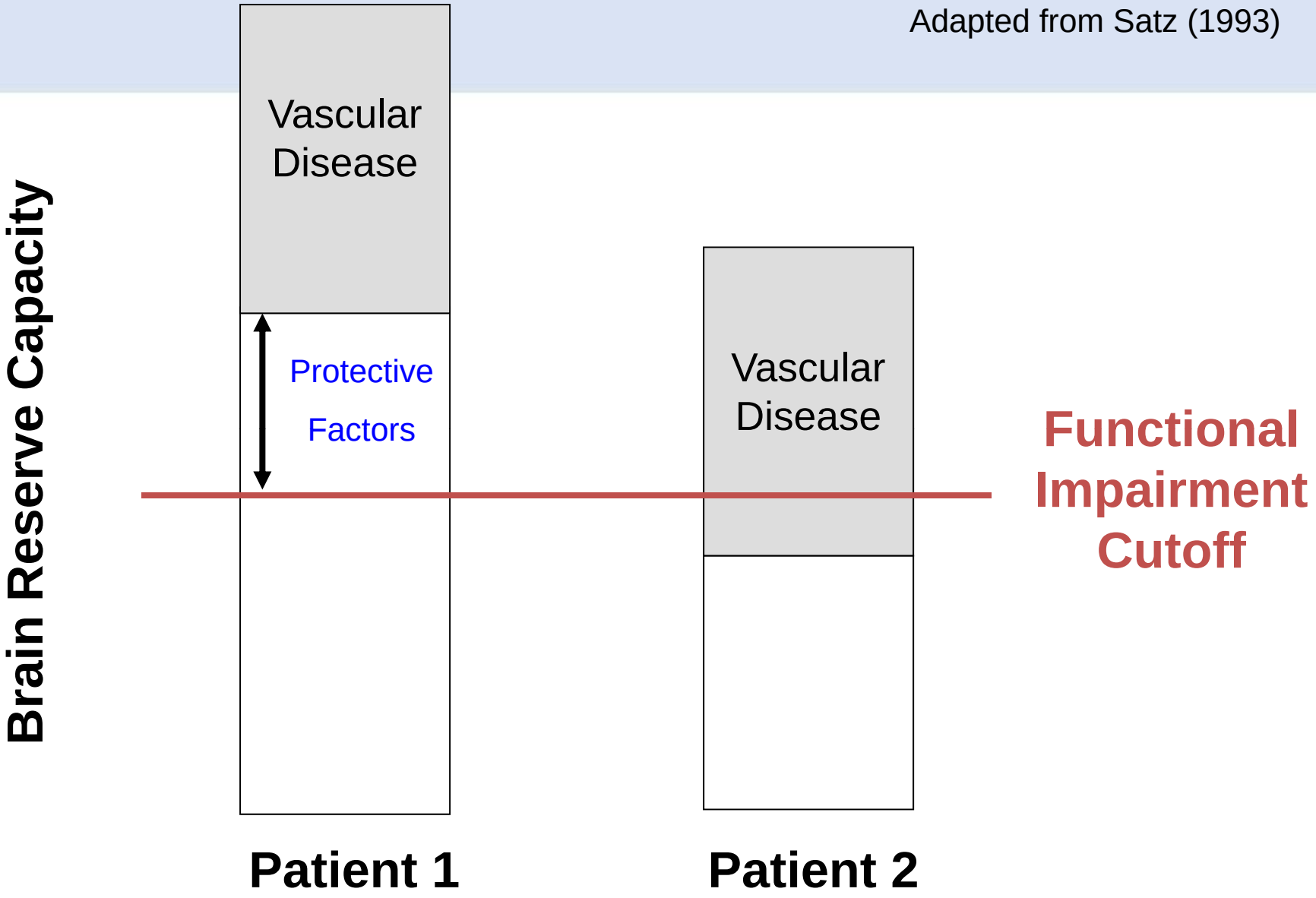
23% of lacunar infarct patients developed dementia within four years, which represented a 4-12 fold increase in risk relative to controls

Loeb et al., *Stroke*, 1992; 29: 1225

New Technology & New Findings: Diffusion Tensor Imaging

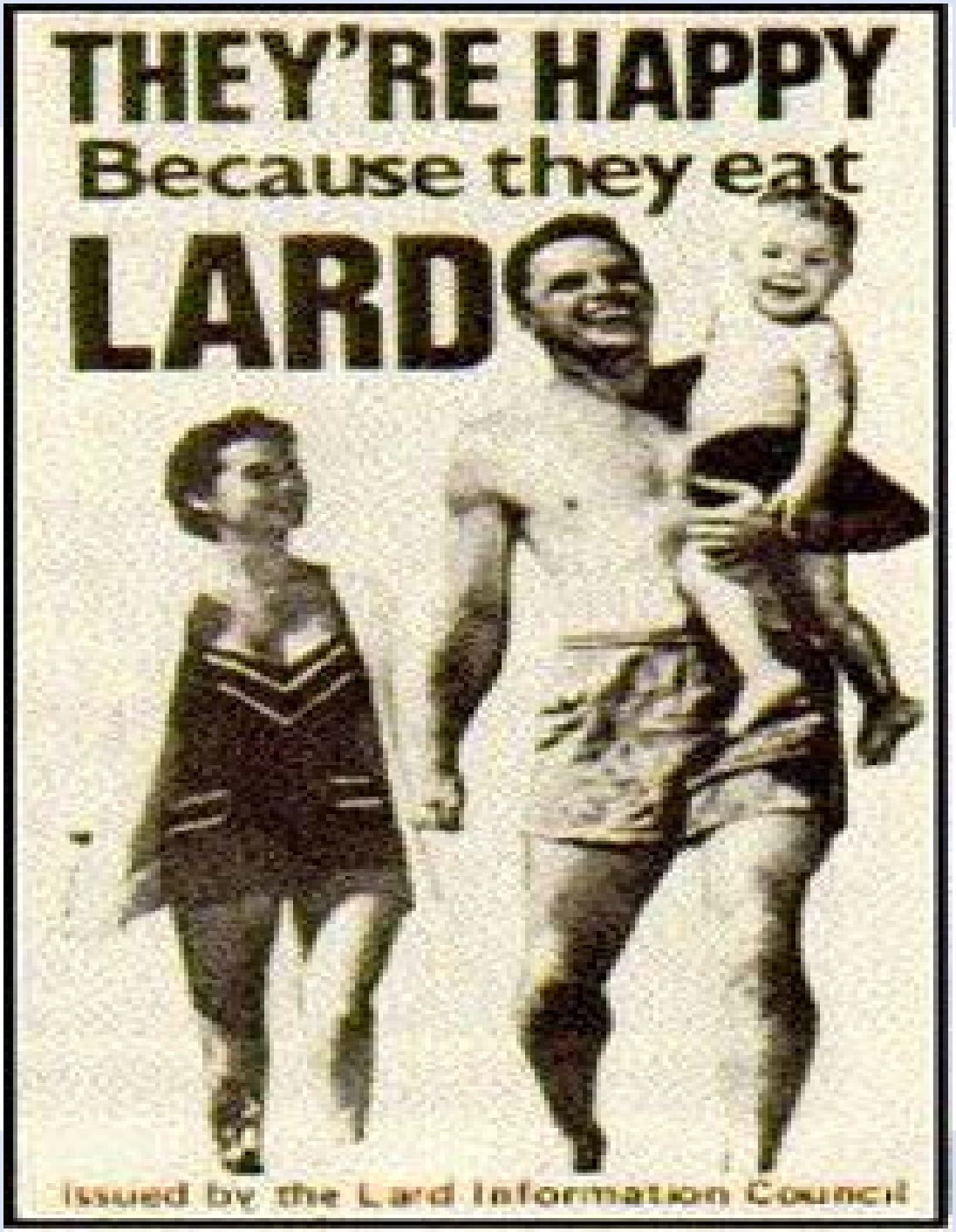


Adapted from Satz (1993)



Underwriting Considerations

- Cognitive reserve as a way to conceptualize cognitive risk at time of underwriting
- Pay attention to subjective complaints
- Consider age
- Obtain Medical Records
  - MRI reports (report body and reason for referral)
  - Consider location and extent of white matter lesions
  - Consider size of lesions
  - Consider presentation (punctate, linear, confluent)
  - Pay attention to mention of atrophy
  - Progression across MRI studies
  - Consider comorbid vascular risk factors and their severity
- Consider late life depression as a unique entity
- Consider a cognitive screen, but be familiar with its strengths and weaknesses
- Involve your medical resource


THEY'RE HAPPY
Because they eat
LARD
Issued by the Lard Information Council