

Multiplexed Biomarkers for Alzheimer's Disease (AD) Diagnosis

Inventors



Dr. Kelvin Lee
Professor of Chemical Engineering

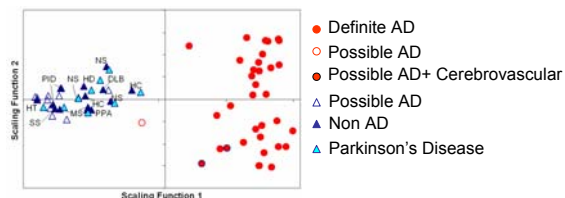


Dr. Norman Relkin
Professor of Clinical Neurology and Neuroscience

Technology

23 protein CSF biomarkers

- Segregate definite AD and nonAD (normal and demented) subjects
- 94% sensitivity and 94% specificity .



Protein Panel Composition

Inflammation:

Immunoglobins*, Plasminogen*, Fibrinogen beta, Complement component 3*

Proteolytic Inhibition:

α -1-antitrypsin*, proSAAS

Protein Transport:

Albumin, Vitamin D-binding protein, Transthyretin*, Retinol binding protein, Apolipoprotein E*, Apolipoprotein J*

Neuronal Membrane:

Contactin, Neuronal pentraxin receptor

*: implicated in AD

Products

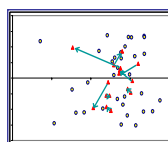
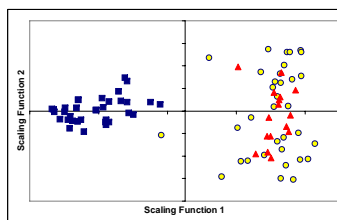
Assay kits and/or services for AD diagnosis and treatment monitoring.

Customers

- Hospitals
- Diagnostic companies
- Drug companies with AD therapeutics

Clinical Validation

- Cornell AD marker panel was used to evaluate patients in a Phase I clinical trial for a drug to treat AD, together with traditional clinical diagnostic method for AD.
- Results from Cornell AD marker panel are consistent with those of the clinical diagnosis.
- Analysis for the samples from the Phase II clinical trial is ongoing.
- Cornell AD marker panel is the first clinically validated diagnostic system for AD other than the traditional methods based on subjective review of patient history, brain imaging, and neuropsychological testing.



Market Opportunity:

- 2005: 24 million people worldwide have been diagnosed with AD. 2040 Projection: 80 million.
- Biomarkers can increase patients/ drug-treatment rate and revenues.
- Success of AD therapy hinges on the availability of biomarkers.

Currently

Duration of therapy ~ 9 years

Biomarkers with symptomatic treatment

Duration of therapy ~ 11 years

Biomarkers with disease-modifying therapy

Duration of therapy ~ 14 years

(Source: Decision Resources, Inc.)

Competition

Current method

- Current diagnosis is based on subjective review of patient history and exclusion of other neurological disorders.
- Most patients are not diagnosed until neuronal damage are severe with observable symptoms.

Other Biomarkers

Genetic markers

ApoE ϵ 4, Presenilin 1&2

Amyloid markers

sAPP α , sAPP β , A β -42/ A β -40, Amyloid-derived ligands (ADDLs).

Tau markers

Total, Tau, P-Tau, p-Tau/ A β -42

Other markers

CAT, AchE, NTP

These markers are generally not specific and sensitive enough for effective early diagnostics.

Imaging Agents

Pittsburgh Compound B, F-FDDNP, BX-471, etc.

No agents are available in the market yet. Have to be associated with large imaging devices.