Demo Screen Shots:

*Pathways Explorer*

*CCR-to-NCI Thesaurus*

VectorC, LLC
Pathways Explorer Demo
The Wnt signaling pathway describes a complex network of proteins most well known for their roles in embryogenesis and cancer, but also involved in normal physiological processes in adult animals. [wikipedia]
A Thank You from Wikipedia Founder Jimmy Wales

Wnt signaling pathway
From Wikipedia, the free encyclopedia

The Wnt signaling pathway describes a complex network of proteins most well known for their roles in embryogenesis and cancer, but also involved in normal physiological processes in adult animals.[1]

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Discovery

The name Wnt was coined as a combination of Wingless and Int[2] and can be pronounced as wint.[3] The Wingless gene had originally been identified as a segment polarity gene in Drosophila melanogaster that functions during embryogenesis[4] and also during adult limb formation during metamorphosis.[5]

The INT genes were originally identified as vertebrate genes near several integration sites of mouse mammary tumor virus (MMTV).[6] The Int-1 gene and the wingless gene were found to be homologous, with a common evolutionary origin evidenced by similar amino acid sequences of their encoded proteins.

Mutations of the wingless gene in the fruit fly were found in wingless flies, while tumors caused by MMTV were found to have copies of the virus integrated into the genome forcing overproduction of one of several Wnt genes. The ensuing effort to understand how similar genes produce such different effects has revealed that Wnts are a major class of secreted morphogenic ligands of profound importance in establishing the pattern of development in the bodies of all multicellular organisms studied.
A Multicenter Study of NAP (AL-108) in Schizophrenia

This study is currently recruiting participants.
Verified by University of California, Los Angeles, June 2008

Sponsors and Collaborators:
University of California, Los Angeles
University of Maryland
Washington University School of Medicine
Massachusetts General Hospital
Nathan Kline Institute for Psychiatric Research
Columbia University
Duke University
Beth Israel Deaconess Medical Center

Information provided by: University of California, Los Angeles
ClinicalTrials.gov Identifier: NCT00505765

Purpose

The TURNS is a NIMH-funded contract for the evaluation of new compounds for the treatment of cognitive impairments in schizophrenia (HHSN 27620044 1003C; P.I.: Steve Marder, M.D.).

Despite advances in the safety, tolerability, and effectiveness of antipsychotic medications for the treatment of schizophrenia, many patients continue to be plagued by impairments in social and work functioning. Persons with schizophrenia commonly show deficits in a number of areas of cognition that include impairments in attention, memory, and executive functioning (the ability and organize one's behavior). Importantly, a large body of literature now shows a link between cognition and community functioning in schizophrenia. It is believed that treatments that improve cognitive deficits may lead to improvements in work and social functioning.

One approach to improve the community functioning of patients with schizophrenia is to develop new agents that treat the cognitive deficits of the illness. A promising agent is called AL-108. This drug is administered as a nasal spray. Studies in animals suggest that this drug may protect neurons and may improve cognition in schizophrenia. The current study is a twelve-week multicenter, double-blind, randomized clinical trial of two doses of AL-108 (5 and 30 mg/day intranasally) versus placebo in the treatment of persistent cognitive dysfunction in schizophrenia. The study medication will be added to patients' current antipsychotic medication or to their current injectable first-generation antipsychotic medication. The primary outcome measure will consist of the composite score of the MATRICS neuropsychological battery. Secondary outcome measures will include scores on symptoms, functional outcome, and safety measures. Sixty clinically stable patients with schizophrenia, drawn from eight sites, will participate in the study. Twenty-five patients will be enrolled at UCLA.

<table>
<thead>
<tr>
<th>Condition</th>
<th>Intervention</th>
<th>Phase</th>
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<tbody>
<tr>
<td>Schizophrenia</td>
<td>Drug AL-108</td>
<td>Phase II</td>
</tr>
<tr>
<td>Pathway</td>
<td>Researchers</td>
<td>Publications</td>
</tr>
<tr>
<td>---------</td>
<td>-------------</td>
<td>--------------</td>
</tr>
<tr>
<td>Wnt signaling pathway</td>
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- Synaptic transmission and hippocampal long-term potentiation in transgenic mice expressing FAD-linked presenilin 1. [pubmed:10078973]
- A presenilin-1-dependent gamma-secretase-like protease mediates release of Notch intracellular domain. [pubmed:10200545]
- Transgenic animals in Alzheimer's disease research. [pubmed:10356989]
- Biology of presenilins as causative molecules for Alzheimer disease. [pubmed:10401981]
- Differentiation of the macrophage phagocyte system during mouse embryogenesis, the role of transcription factor PU.1. [pubmed:10931506]
- Accumulation of murine amyloid-beta in a gene-dosage-dependent manner in PS1+7 'knock-in' mice. [pubmed:10383647]
- Presenilin-1 deficiency leads to loss of Cajal-Retzius neurons and cortical dysplasia similar to human type 2 lissencephaly. [pubmed:10421573]
- Amyloid phenotype characterization of transgenic mice overexpressing both mutant amyloid precursor protein and mutant presenilin 1 transgenes. [pubmed:10440156]
- Presenilin 2 deficiency causes a mild pulmonary phenotype and no changes in amyloid precursor protein processing but enhances the embryonic lethal phenotype of presenilin 1 deficiency. [pubmed:10615853]
- Dietary restriction protects hippocampal neurons against the death-promoting action of a presenilin-1 mutation. [pubmed:10526115]
- Alzheimer's presenilin 1 mutations impact kinesin-based axonal transport. [pubmed:12065290]
- Presenilin redistribution associated with aberrant cholesterol transport enhances beta-amyloid production in vivo. [pubmed:12863267]
- Presenilins mutated at Asp-257 or Asp-365 restore Pen-2 expression and Nicastrin glycosylation but remain catalytically inactive in the absence of wild type Presenilin. [pubmed:12865766]
- Triple-transgenic model of Alzheimer's disease with plaques and tangles: interstitial Abeta and synaptic dysfunction. [pubmed:12895417]
- Notch1 competes with the amyloid precursor protein for gamma-secretase and down-regulates presenilin 1 gene expression. [pubmed:12900156]
- A presenilin dimer at the core of the gamma-secretase enzyme: insights from parallel analysis of Notch 1 and APP proteolysis. [pubmed:14566526]
- Presenilin-1 deficiency impairs glutamate-evoked intracellular calcium responses in neurons. [pubmed:14980721]
- Binding sites of gamma-secretase inhibitors in rodent brain distribution, postnatal development, and effect of desaturation. [pubmed:15044533]
- Loss of presenilin function causes impairments of memory and synaptic plasticity followed by age-dependent neurodegeneration. [pubmed:15081626]
- Notch activation induces apoptosis in neural progenitor cells through a p53-dependent pathway. [pubmed:15081626]
- Notch oncogenes depend on gamma-secretase/presenilin activity for processing and function. [pubmed:15123652]
Synaptic transmission and hippocampal long-term potentiation in transgenic mice expressing FAD-linked presenilin 1.

Parent, A.; Linden, D.J.; Sisodia, S.S.; Borchelt, D.R.

Department of Neuroscience, Johns Hopkins University School of Medicine, Baltimore, Maryland 21205, USA. aparant@helix.nih.gov

Mutations in two related genes, presenilin 1 and presenilin 2 (PS1 and PS2), cause a subset of early-onset familial Alzheimer's disease (FAD). PS1 is expressed in a variety of neuronal and peripheral tissues, including neuronal populations known to be at risk in Alzheimer's disease such as CA1 hippocampal neurons. To examine whether FAD-linked mutations in PS1 directly influence the physiology of learning and memory, we measured the field excitatory postsynaptic potential (EPSP) at the Schaffer collateral-CA1 synapse in hippocampal slices. Basal synaptic transmission and long-term potentiation (LTP) were examined in neurons of transgenic mice expressing wild-type human PS1 (WTg) and FAD-linked A426E PS1 variant (MTg) and in neurons of nontransgenic littermates (NTg). Several measures of basal synaptic transmission were unaltered in WTg and MTg compared to NTg mice, including maximum EPSP slope, maximum EPSP amplitude, maximum fiber volley amplitude, and the function relating fiber volley amplitude to EPSP slope, an index of basal synaptic strength. In addition, paired-pulse facilitation was not changed. However, upon theta burst stimulation or high-frequency stimulation, input-specific LTP in MTg animals had a larger initial amplitude and was more persistent than that in WTg or NTg animals. These data suggest that the FAD-linked A426E variant of PS1 leads to higher degree of LTP induction in mice.

Publication Types:
- Research Support, Non-U.S. Govt.
- Research Support, U.S. Govt, P.H.S.

PMID: 10078973 [PubMed - indexed for MEDLINE]
CCR-to-NCI Thesaurus Demo
### Patient Demographics

<table>
<thead>
<tr>
<th>Condition</th>
<th>Min</th>
<th>Max</th>
<th>Medication</th>
<th>Min</th>
<th>Max</th>
</tr>
</thead>
<tbody>
<tr>
<td>cardiovascular disorder</td>
<td>1</td>
<td>3</td>
<td>enzyme inhibitor</td>
<td>1</td>
<td>3</td>
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</table>

[Generate 100 Records]

### Patient Population

<table>
<thead>
<tr>
<th>Name</th>
<th>Conditions</th>
<th>Medications</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.Patients</td>
<td>Paroxysmal Ventricular Tachycardia</td>
<td>Pentoxyfilline, Pirarubicin, Armsacrine</td>
</tr>
<tr>
<td>1.Patients</td>
<td>Patent Foramen Ovale</td>
<td>Efavirenz</td>
</tr>
<tr>
<td>2.Patients</td>
<td>Thromboembolism, Hypertensive Encephalopathy, Acute Myocardial Infarction</td>
<td>Tenofivir Disopilx Fumarate, Gossypol</td>
</tr>
<tr>
<td>3.Patients</td>
<td>Coronary Atherosclerosis, Cerebrovascular Accident, Raynaud's Disease</td>
<td>Benazepril Hydrochloride, Indometacin</td>
</tr>
<tr>
<td>4.Patients</td>
<td>Chronic Pulmonary Heart Disease, Transient Retinal Arterial Occlusion</td>
<td>Indinavir Sufate, Piroxicam</td>
</tr>
<tr>
<td>5.Patients</td>
<td>Isorythmic Atriointricular Dissociation, Acute Coronary Syndrome</td>
<td>Lisinopril, Pyridostigmine Bromide</td>
</tr>
<tr>
<td>6.Patients</td>
<td>Isorythmic Atriointricular Dissociation, Wolf-Parkinson-White Syndrome</td>
<td>Flurbiprofen</td>
</tr>
<tr>
<td>7.Patients</td>
<td>Atriointricular Nodal Reentry Tachycardia, Acute Coronary Syndrome</td>
<td>Naproxen, Sulindac, Formestane</td>
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<td>8.Patients</td>
<td>Functional Tachycardia</td>
<td>Clofarabine</td>
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<tr>
<td>Patient</td>
<td>Condition</td>
<td>Medication</td>
</tr>
<tr>
<td>--------</td>
<td>---------------------------------------------------------------------------</td>
<td>------------</td>
</tr>
<tr>
<td>6</td>
<td>Isorhythmic Atrioventricular Dissociation</td>
<td>Flurbiprofen</td>
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<td>Wolff-Parkinson-White Syndrome</td>
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<td>7</td>
<td>Atrioventricular Nodal Reentry Tachycardia</td>
<td>Naproxen</td>
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<tr>
<td></td>
<td>Acute Coronary Syndrome</td>
<td>Sulindac</td>
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<td>Formestane</td>
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<td>8</td>
<td>Junctional Tachycardia</td>
<td>Clofarabine</td>
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<td></td>
<td>Transient Retinal Arterial Occlusion</td>
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<tr>
<td>9</td>
<td>Nevius Flammeus</td>
<td>Forodesine Hydrochloride</td>
</tr>
</tbody>
</table>

**Cohort Definition**

- **Condition**: myocardial infarction
- **Medication**: rofecoxib

**Cohort Results**

<table>
<thead>
<tr>
<th>Name</th>
<th>Conditions</th>
<th>Medications</th>
</tr>
</thead>
<tbody>
<tr>
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<tr>
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</tbody>
</table>

Done
### Cohort Definition

- **Condition**: myocardial infarction
- **Medication**: cyclooxygenase inhibitor

### Cohort Results

<table>
<thead>
<tr>
<th>Name</th>
<th>Conditions</th>
<th>Medications</th>
</tr>
</thead>
</table>
| 20.Pat | Polyarteritis Nodosa  
Aortic Arch Syndrome  
Acute Myocardial Infarction | Ketorolac           |
| 38.Pat | Myocardial Degeneration  
Posterior Myocardial Infarction | Celecoxib  
Hydrazine Sulfate |
| 42.Pat | Aortic Arch Syndrome  
Old Myocardial Infarction | Curcumin  
Leflunomide |
| 63.Pat | Hepatic Veno-Occlusive Disease  
Old Myocardial Infarction | Diclofenac Sodium  
Trilostane |
| 64.Pat | Junctional Tachycardia  
Old Myocardial Infarction  
Partial Retinal Vein Occlusion | Naproxen Sodium |
<table>
<thead>
<tr>
<th>Name</th>
<th>Conditions</th>
<th>Medications</th>
</tr>
</thead>
</table>
| 0, Patient | Henoch-Schonlein Purpura  
Inferior Myocardial Infarction  
Acute Intestinal Ischemia | Diclofenac Sodium      |
| 17, Patient | Primary Lymphedema  
Coronary Atherosclerosis  
Silent Myocardial Infarction | Naproxen  
Hydrazine  
Nefilnavir |
| 21, Patient | Acute Rheumatic Myocarditis  
Inferior Myocardial Infarction | Pentosan Polysulfate  
Ketorolac  
Sulindac |
| 21, Patient | Acute Rheumatic Myocarditis  
Inferior Myocardial Infarction | Pentosan Polysulfate  
Ketorolac  
Sulindac |
| 64, Patient | Acute Rheumatic Myocarditis  
Paroxysmal Atrial Tachycardia  
Inferior Myocardial Infarction | Ibuprofen  
Levofloxacin |
| 69, Patient | Primary Lymphedema  
Posterior Myocardial Infarction  
Paroxysmal Atrial Tachycardia | Indomethacin  
Lopinavir/Ritonavir |
| 99, Patient | Conjunctival Vascular Disorder  
Paroxysmal Ventricular Tachycardia  
Acute Myocardial Infarction | Naproxen Sodium       |
Diclofenac Sodium (Link To Concept in BioPortal)

**Details**
- **Class Name:** Diclofenac Sodium
- **ID:** Diclofenac_Sodium
- **Cas Registry:** 15307-79-8
- **Fda Unii Code:** DTG1282970
- **Code:** C47304
- **Label:** Diclofenac Sodium
- **Has Free Acid Or Base Form:** Diclofenac

**Synonym:**
- GP 45840, Diclofenac Sodium, DICLOFENAC SODIUM, Voltaren, 2-[2,6-Dichlorophenyl]amino benzeneacetic Acid Monosodium Salt
- Umls Cui: C0700593
- Full Syn: 2-[2,6-Dichlorophenyl]amino benzeneacetic Acid Monosodium Salt SNNCI, DICLOFENAC SODIUMPTDFADQTG128297Q, Diclofenac SodiumPTDCP09109, Diclofenac SodiumPTNCI, VoltarenERNCI, GP 45840ChNCCI

**Rdf Type:** Pharmacologic Substance

**Definition:**
Diclofenac sodium is a non-selective reversible and competitive inhibitor of cyclooxygenase, a sodium salt form of diclofenac, a benzene acetic acid derivative and nonsteroidal anti-inflammatory drug (NSAID) with analgesic, antipyretic and anti-inflammatory activity.
Cohort Update

vectorc.demo@gmail.com

Patients with Myocardial Infarction taking Cyclooxygenase Inhibitor

Name: Patient 0
Name: Patient 17
Name: Patient 21
Name: Patient 21
Name: Patient 64
Name: Patient 69
Name: Patient 98

2:27 PM (1 minute ago)