Demo Screen Shots:

Pathways Explorer

CCR-to-NCI Thesaurus

VectorC, LLC

Pathways Explorer Demo

Pathways Explorer - Mozilla Firefox

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http://localhost:8889/com.vectorc.ssb.mule.example.transhealth.ui.TranslationalHealthApplication/TranslationalHealthApplication.html

Pathways Explorer | Patient Cohort Selector

Pathways Explorer

Enter Search Criteria

Pathway: wnt signaling pathway [p

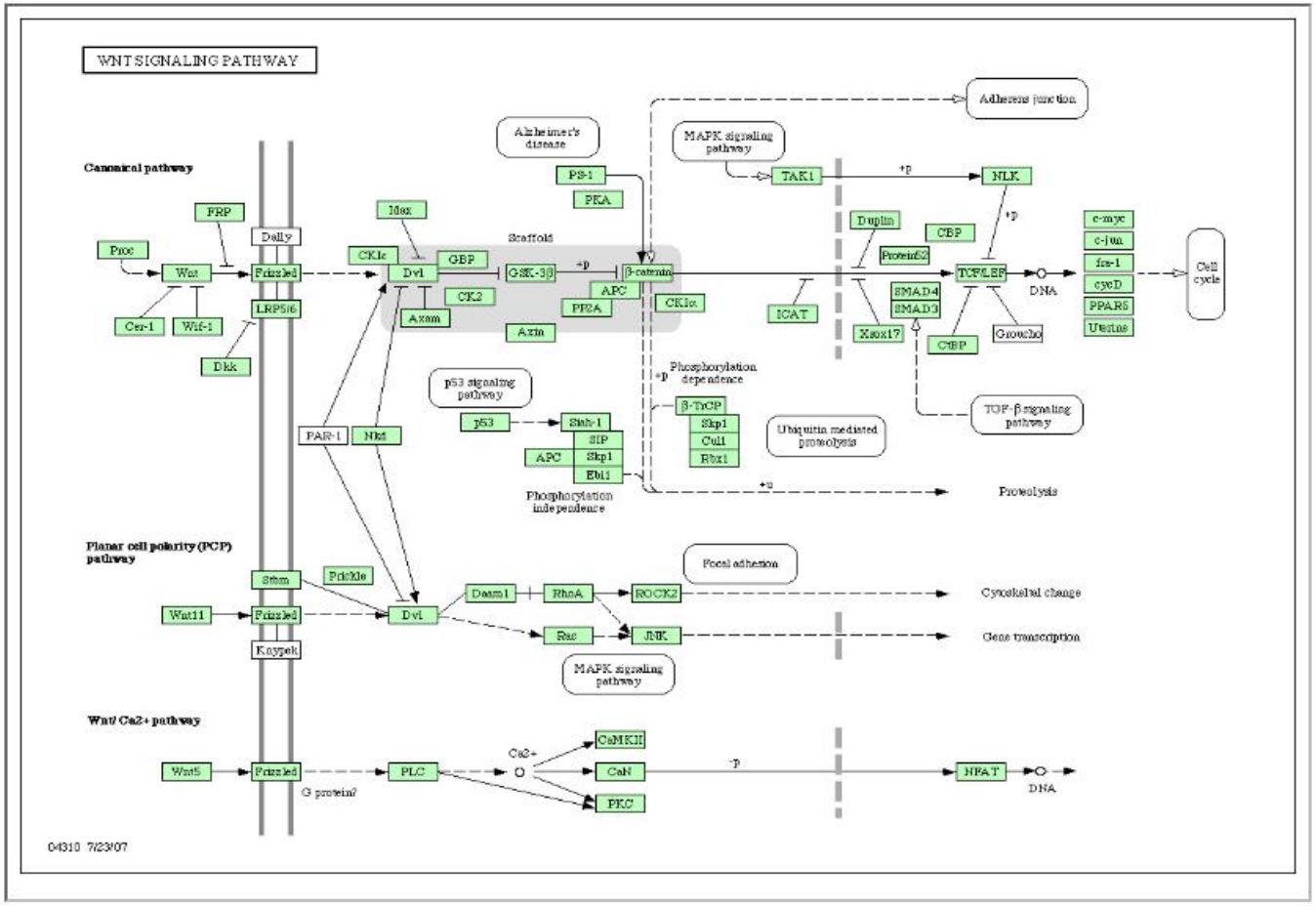
Genes:

Jun oncogene (Jun) [geneid:16476]
casein kinase 1, epsilon (Csnk1e) [ge
catenin (cadherin associated protein)
fos-like antigen 1 (Fosl1) [geneid:142
glycogen synthase kinase 3 beta (Gs
matrix metallopeptidase 7 (Mmp7) [g
myelocytomatosis oncogene (Myc) [
nemo like kinase (Nlk) [geneid:18099
peroxisome proliferator activator rece
presenilin 1 (Psen1) [geneid:19164]

Search

Pathway Researchers Publications Clinical Trials

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



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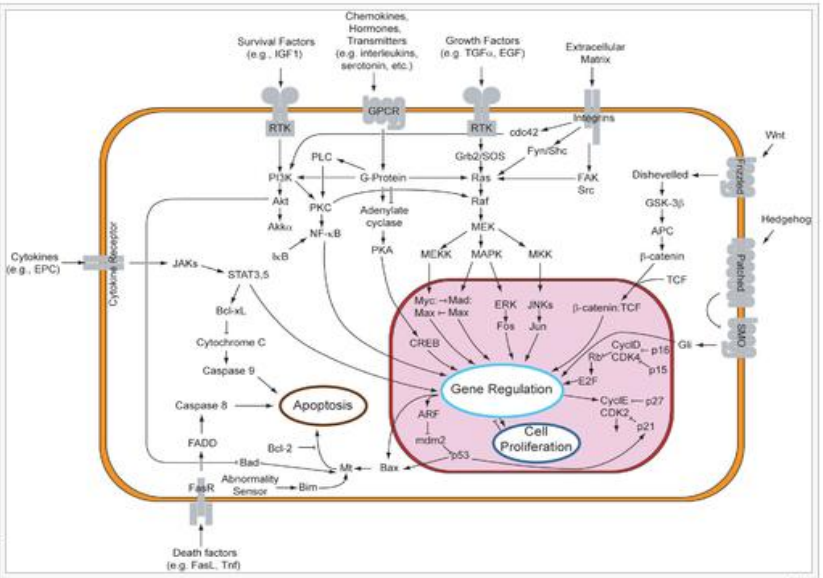
Wnt signaling pathway

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]

Contents [hide]

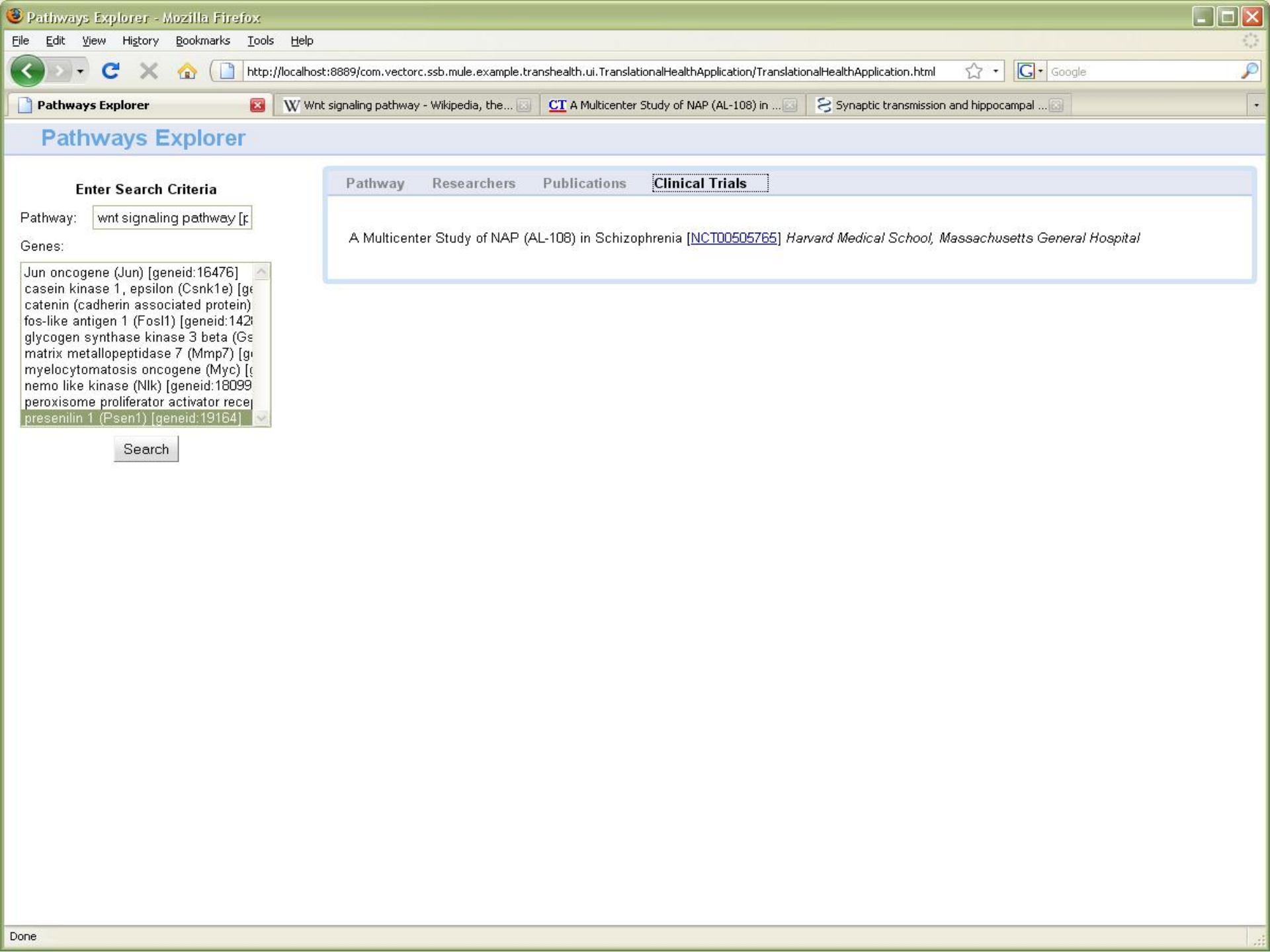


Overview of signal transduction pathways. On the upper right hand side of the cell, a Wnt signaling protein is shown to bind to a frizzled receptor.

Discovery [edit]

The name **Wnt** was coined as a combination of **Wg** (wingless) and **Int** [2] and can be pronounced as 'wint'. The *wingless* gene had originally been identified as a segment polarity gene in *Drosophila melanogaster* that functions during embryogenesis[3] and also during adult limb formation during metamorphosis.[4] The INT genes were originally identified as vertebrate genes near several integration sites of mouse mammary tumor virus (MMTV).[5] 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.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http://localhost:8889/com.vectorc.ssb.mule.example.transhealth.ui.TranslationalHealthApplication/TranslationalHealthApplication.html
Google
Pathways Explorer
Wnt signaling pathway - Wikipedia, the...
A Multicenter Study of NAP (AL-108) in ...
Synaptic transmission and hippocampal ...
Pathways Explorer
Enter Search Criteria
Pathway:
wnt signaling pathway [r
Genes:
Jun oncogene (Jun) [geneid:16476]
casein kinase 1, epsilon (Csnk1e) [g
catenin (cadherin associated protein)
fos-like antigen 1 (Fosl1) [geneid:142
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myelocytomatosis oncogene (Myc) [
nemo like kinase (Nlk) [geneid:18099
peroxisome proliferator activator rece
presenilin 1 (Psen1) [geneid:19164]
Search
Pathway
Researchers
Publications
Clinical Trials
A Multicenter Study of NAP (AL-108) in Schizophrenia [NCT00505765] Harvard Medical School, Massachusetts General Hospital
Done

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 27820044 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 atypical 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.

Condition	Intervention	Phase
Schizophrenia	Drug: AL-108	Phase II

Pathways Explorer

Enter Search Criteria

Pathway: wnt signaling pathway [r

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- nemo like kinase (Nlk) [geneid:18099
- peroxisome proliferator activator rece
- presenilin 1 (Psen1) [geneid:19164]

Search

Pathway | Researchers | **Publications** | Clinical Trials

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:10206645

Transgenic animals in Alzheimer's disease research. pubmed:10356989

Biology of presenilins as causative molecules for Alzheimer disease. pubmed:10361981

Differentiation of the mononuclear phagocyte system during mouse embryogenesis: the role of transcription factor PU.1. pubmed:10381505

Accumulation of murine amyloidbeta42 in a gene-dosage-dependent manner in PS1 '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:10448051

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:10518543

Dietary restriction protects hippocampal neurons against the death-promoting action of a presenilin-1 mutation. pubmed:10526115

Alzheimer's presenilin 1 mutations impair kinesin-based axonal transport. pubmed:12805290

Presenilin redistribution associated with aberrant cholesterol transport enhances beta-amyloid production in vivo. pubmed:12843267

Presenilins mutated at Asp-257 or Asp-385 restore Pen-2 expression and Nicastrin glycosylation but remain catalytically inactive in the absence of wild type Presenilin. pubmed:12885769

Triple-transgenic model of Alzheimer's disease with plaques and tangles: intracellular Abeta and synaptic dysfunction. pubmed:12895417

Notch1 competes with the amyloid precursor protein for gamma-secretase and down-regulates presenilin-1 gene expression. pubmed:12960155

A presenilin dimer at the core of the gamma-secretase enzyme: insights from parallel analysis of Notch 1 and APP proteolysis. pubmed:14566063

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 deafferentation. pubmed:15044533

Loss of presenilin function causes impairments of memory and synaptic plasticity followed by age-dependent neurodegeneration. pubmed:15066262

Notch activation induces apoptosis in neural progenitor cells through a p53-dependent pathway. pubmed:15081359

Notch oncoproteins depend on gamma-secretase/presenilin activity for processing and function. pubmed:15123653

1: Neurobiol Dis. 1999 Feb;6(1):56-62.

Related Articles, Links

ELSEVIER FULL-TEXT ARTICLE

Synaptic transmission and hippocampal long-term potentiation in transgenic mice expressing FAD-linked presenilin 1.

Parent A, Linden DJ, Sisodia SS, Borchelt DR.

Department of Neuroscience, Johns Hopkins University School of Medicine, Baltimore, Maryland 21205, USA. aparent@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 (fEPSP) 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 (WtTg) and FAD-linked A246E PS1 variant (MTg) and in neurons of nontransgenic littermates (NTg). Several measures of basal synaptic transmission were unaltered in WtTg and MTg compared to NTg mice, including maximum fEPSP slope, maximum fEPSP amplitude, maximum fiber volley amplitude, and the function relating fiber volley amplitude to fEPSP 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 WtTg or NTg animals. These data suggest that the FAD-linked A246E variant of PS1 leads to higher degree of LTP induction in mice.

Publication Types:

- Research Support, Non-U.S. Gov't
- Research Support, U.S. Gov't, P.H.S.

PMID: 10078973 [PubMed - indexed for MEDLINE]

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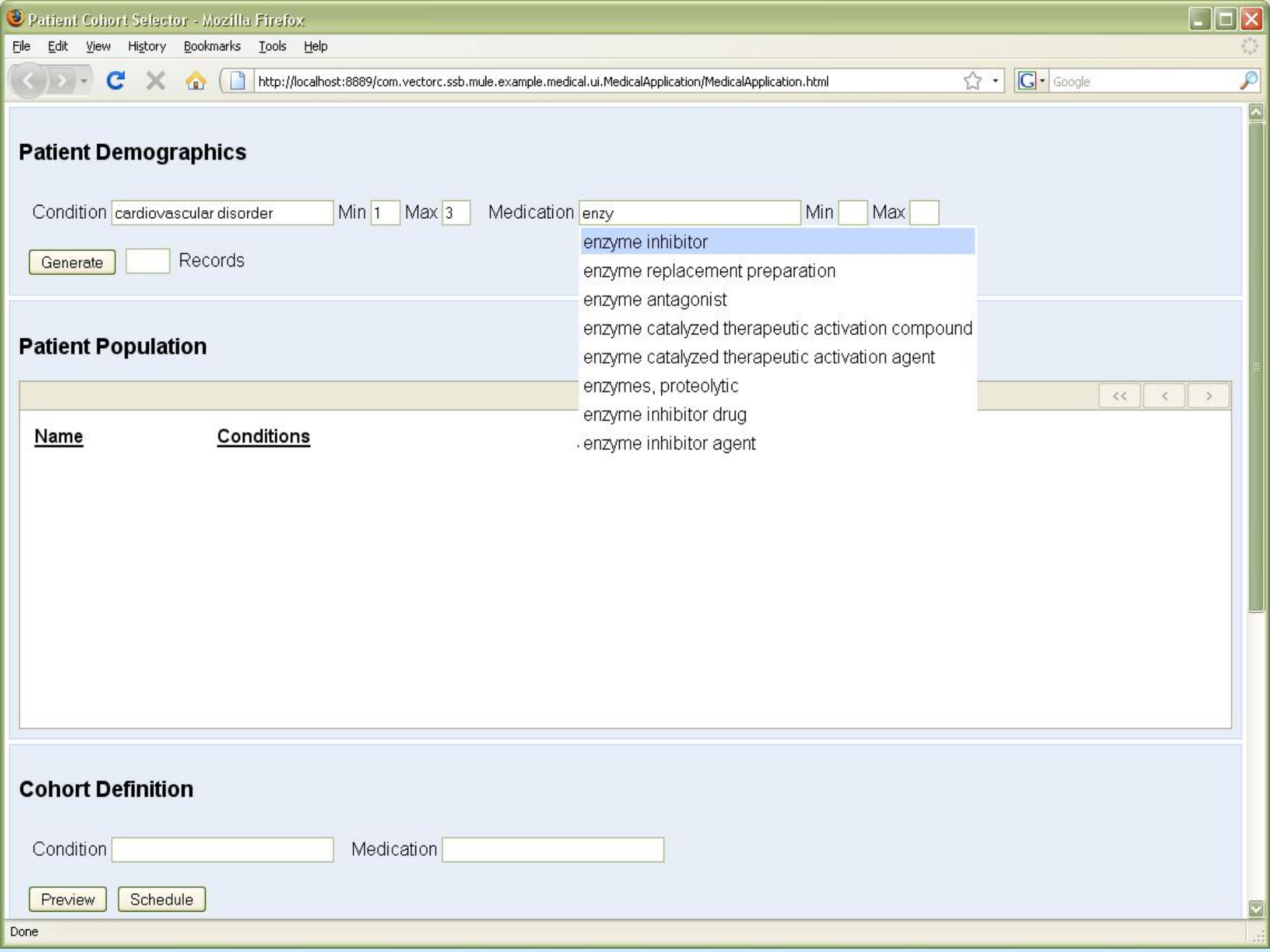
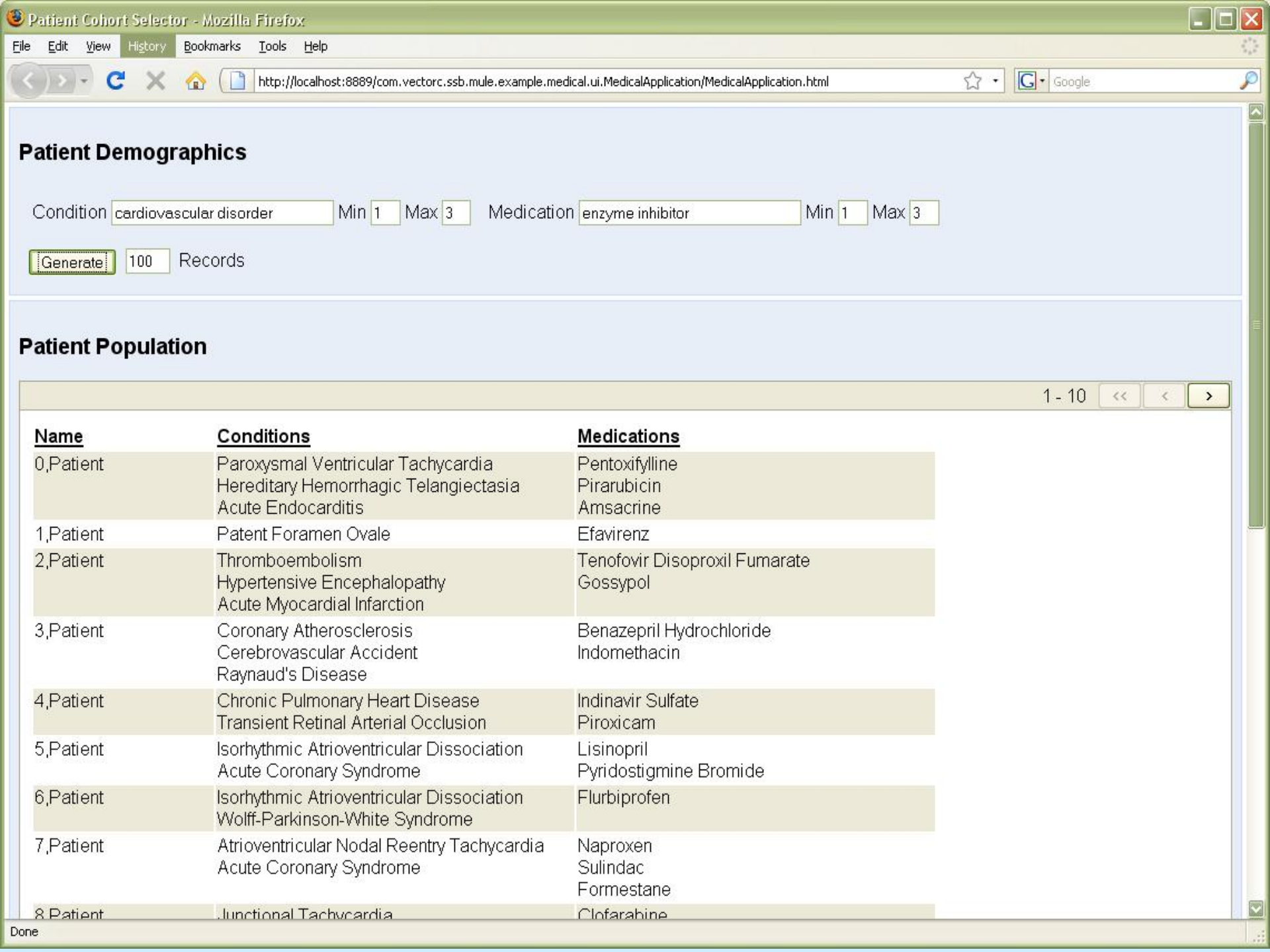
CCR-to-NCI Thesaurus Demo


Patient Cohort Selector - Mozilla Firefox
File Edit View History Bookmarks Tools Help
http://localhost:8889/com.vectorc.ssb.mule.example.medical.ui.MedicalApplication/MedicalApplication.html
Google
Patient Demographics
Condition cardiovascular disorder Min 1 Max 3 Medication enzy Min Max
enzyme inhibitor
enzyme replacement preparation
enzyme antagonist
enzyme catalyzed therapeutic activation compound
enzyme catalyzed therapeutic activation agent
enzymes, proteolytic
enzyme inhibitor drug
enzyme inhibitor agent
Generate Records
Patient Population
Name
Conditions
Cohort Definition
Condition Medication
Preview Schedule
Done



Patient Demographics

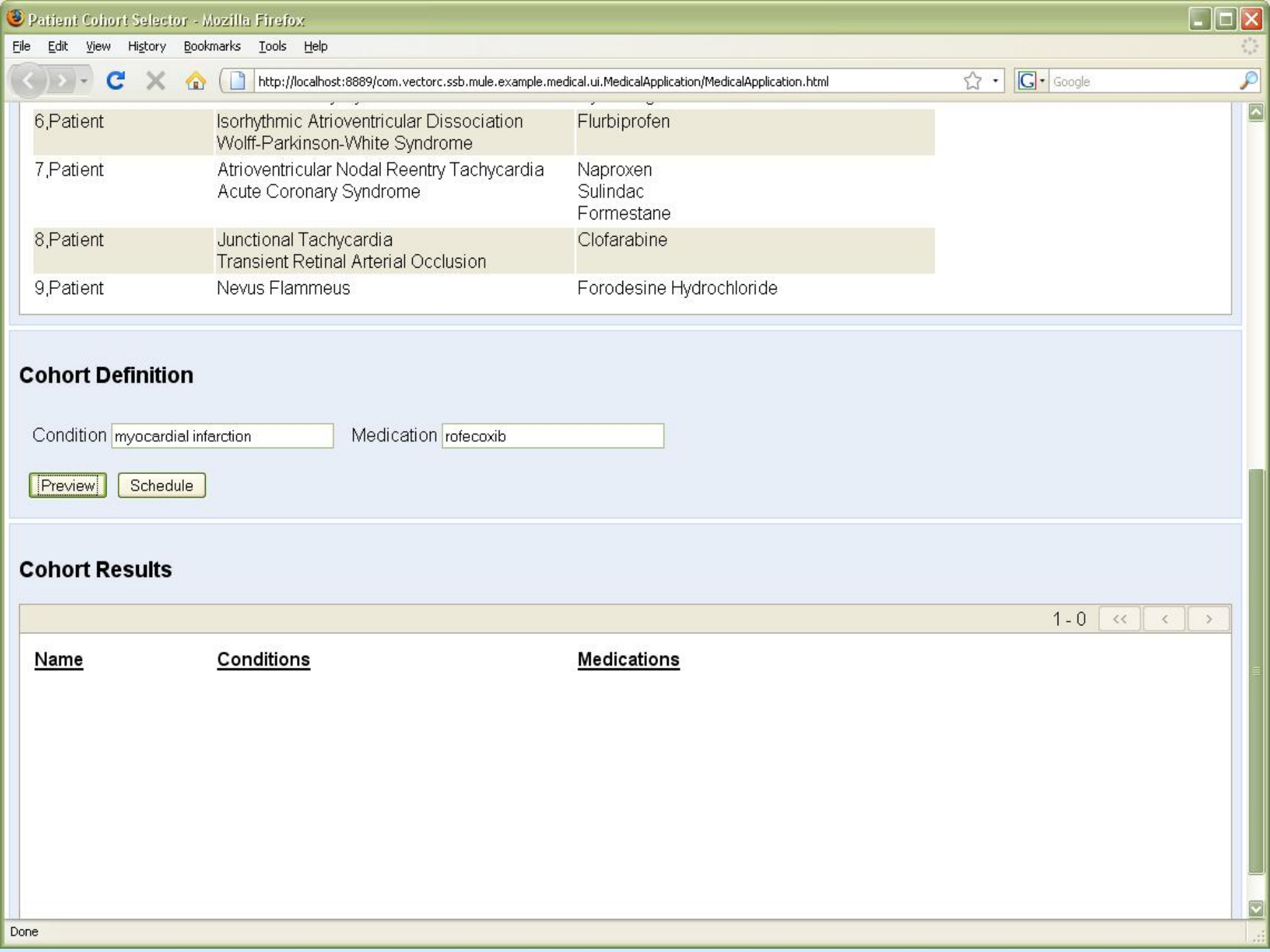
Condition: cardiovascular disorder | Min: 1 | Max: 3 | Medication: enzyme inhibitor | Min: 1 | Max: 3

Generate | 100 | Records

Patient Population

1 - 10 << < >

Name	Conditions	Medications
0,Patient	Paroxysmal Ventricular Tachycardia Hereditary Hemorrhagic Telangiectasia Acute Endocarditis	Pentoxifylline Pirarubicin Amsacrine
1,Patient	Patent Foramen Ovale	Efavirenz
2,Patient	Thromboembolism Hypertensive Encephalopathy Acute Myocardial Infarction	Tenofovir Disoproxil Fumarate Gossypol
3,Patient	Coronary Atherosclerosis Cerebrovascular Accident Raynaud's Disease	Benazepril Hydrochloride Indomethacin
4,Patient	Chronic Pulmonary Heart Disease Transient Retinal Arterial Occlusion	Indinavir Sulfate Piroxicam
5,Patient	Isorhythmic Atrioventricular Dissociation Acute Coronary Syndrome	Lisinopril Pyridostigmine Bromide
6,Patient	Isorhythmic Atrioventricular Dissociation Wolff-Parkinson-White Syndrome	Flurbiprofen
7,Patient	Atrioventricular Nodal Reentry Tachycardia Acute Coronary Syndrome	Naproxen Sulindac Formestane
8,Patient	Junctional Tachycardia	Clofarabine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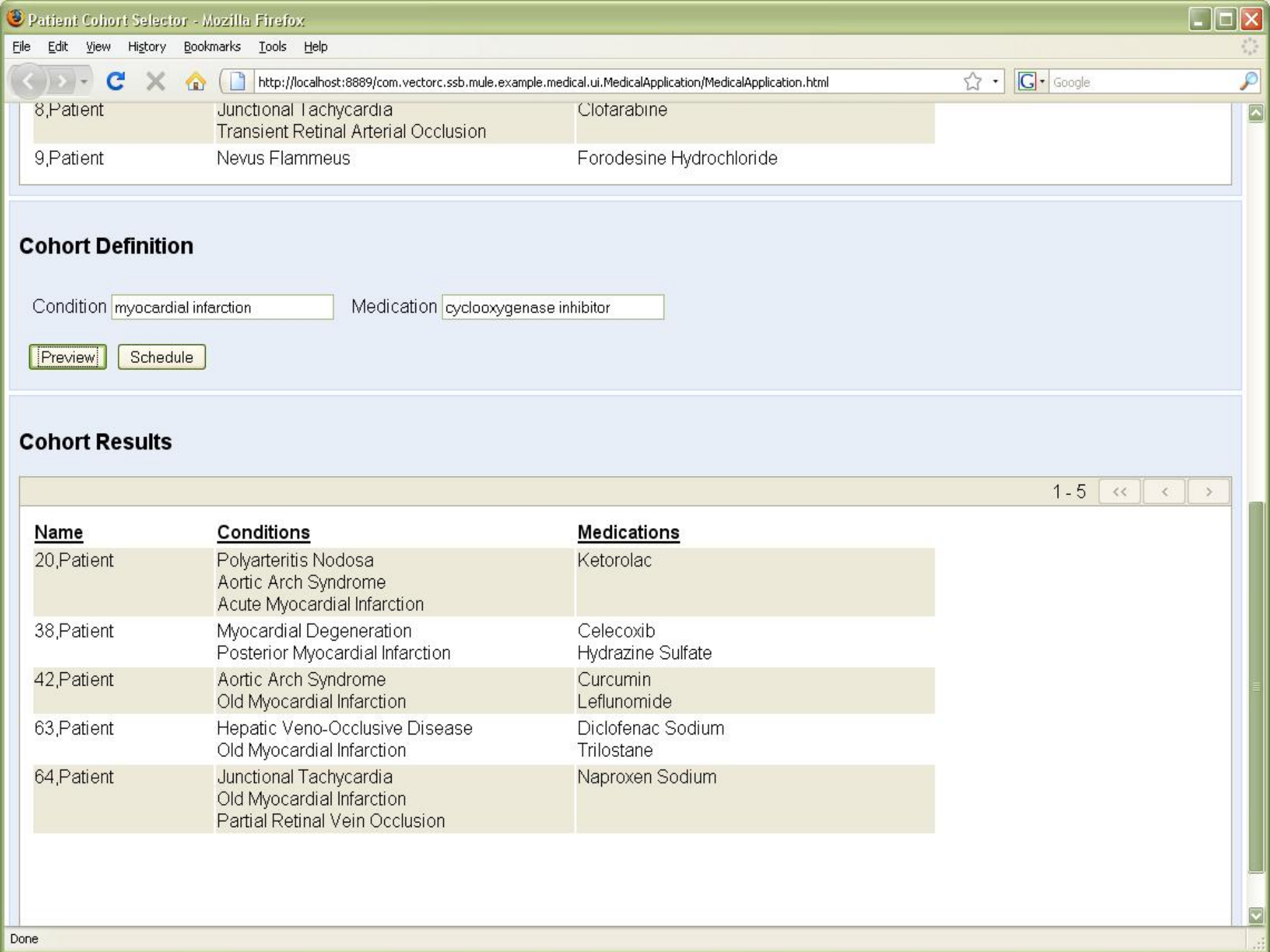
Google
6,Patient
Isorhythmic Atrioventricular Dissociation
Wolff-Parkinson-White Syndrome
Flurbiprofen
7,Patient
Atrioventricular Nodal Reentry Tachycardia
Acute Coronary Syndrome
Naproxen
Sulindac
Formestane
8,Patient
Junctional Tachycardia
Transient Retinal Arterial Occlusion
Clofarabine
9,Patient
Nevus Flammeus
Forodesine Hydrochloride
Cohort Definition
Condition myocardial infarction
Medication rofecoxib
Preview
Schedule
Cohort Results
1 - 0
Name
Conditions
Medications
Done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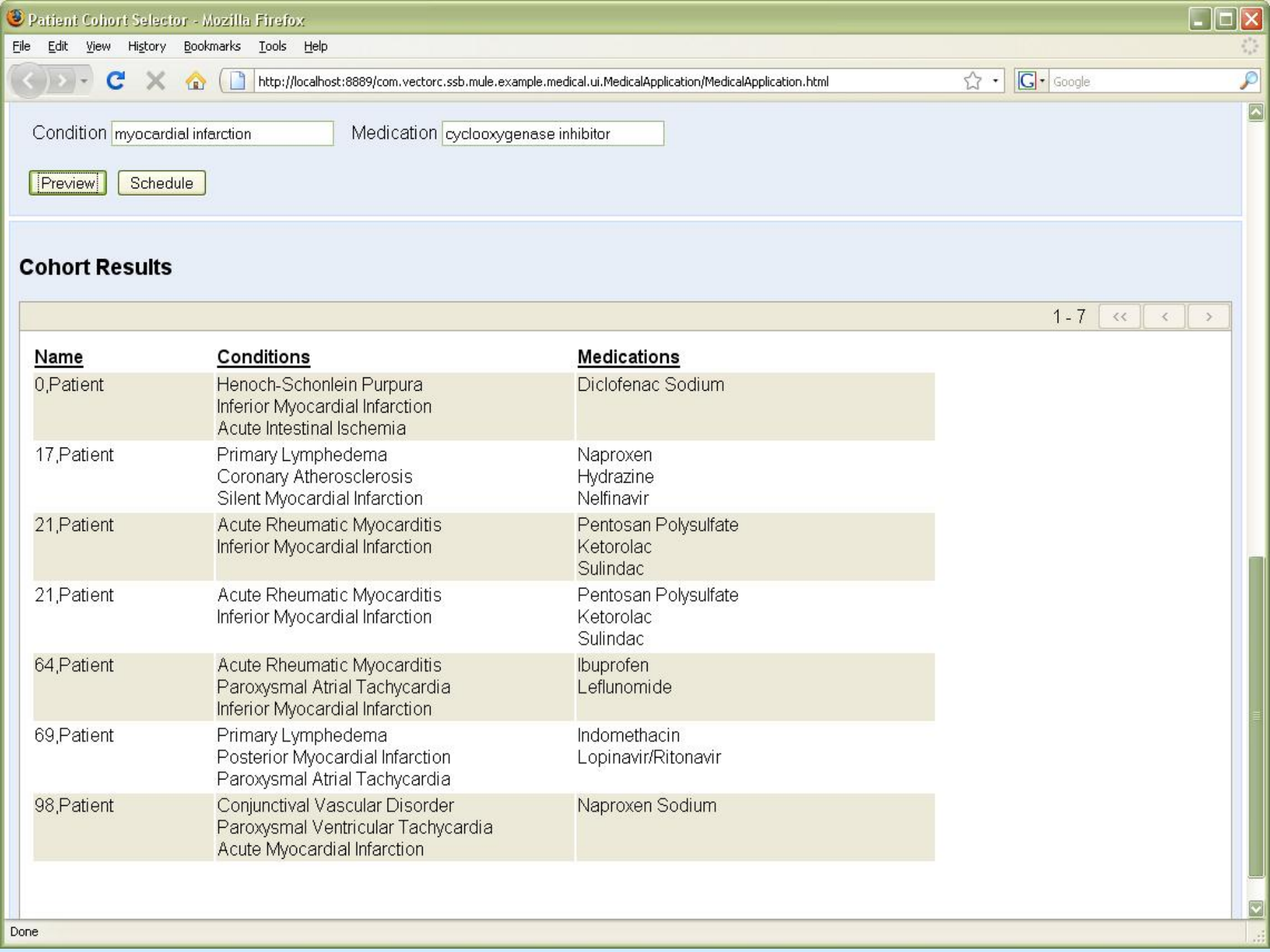
Google
8,Patient
Junctional Tachycardia
Transient Retinal Arterial Occlusion
Clofarabine
9,Patient
Nevus Flammeus
Forodesine Hydrochloride
Cohort Definition
Condition myocardial infarction
Medication cyclooxygenase inhibitor
Preview
Schedule
Cohort Results
1 - 5
Name
Conditions
Medications
20,Patient
Polyarteritis Nodosa
Aortic Arch Syndrome
Acute Myocardial Infarction
Ketorolac
38,Patient
Myocardial Degeneration
Posterior Myocardial Infarction
Celecoxib
Hydrazine Sulfate
42,Patient
Aortic Arch Syndrome
Old Myocardial Infarction
Curcumin
Leflunomide
63,Patient
Hepatic Veno-Occlusive Disease
Old Myocardial Infarction
Diclofenac Sodium
Trilostane
64,Patient
Junctional Tachycardia
Old Myocardial Infarction
Partial Retinal Vein Occlusion
Naproxen Sodium
Done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http://localhost:8889/com.vectorc.ssb.mule.example.medical.ui.MedicalApplication/MedicalApplication.html
Google
Condition myocardial infarction
Medication cyclooxygenase inhibitor
Preview
Schedule
Cohort Results
1 - 7
Name
Conditions
Medications
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Henoch-Schonlein Purpura
Inferior Myocardial Infarction
Acute Intestinal Ischemia
Diclofenac Sodium
17,Patient
Primary Lymphedema
Coronary Atherosclerosis
Silent Myocardial Infarction
Naproxen
Hydrazine
Nelfinavir
21,Patient
Acute Rheumatic Myocarditis
Inferior Myocardial Infarction
Pentosan Polysulfate
Ketorolac
Sulindac
21,Patient
Acute Rheumatic Myocarditis
Inferior Myocardial Infarction
Pentosan Polysulfate
Ketorolac
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Inferior Myocardial Infarction
Ibuprofen
Leflunomide
69,Patient
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Posterior Myocardial Infarction
Paroxysmal Atrial Tachycardia
Indomethacin
Lopinavir/Ritonavir
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Conjunctival Vascular Disorder
Paroxysmal Ventricular Tachycardia
Acute Myocardial Infarction
Naproxen Sodium
Done

Diclofenac Sodium (Link To Concept in BioPortal)

Class Name: Diclofenac Sodium

ID: Diclofenac_Sodium

Cas Registry: 15307-79-6

Fda Unii Code: QTG126297Q

Code: C47984

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

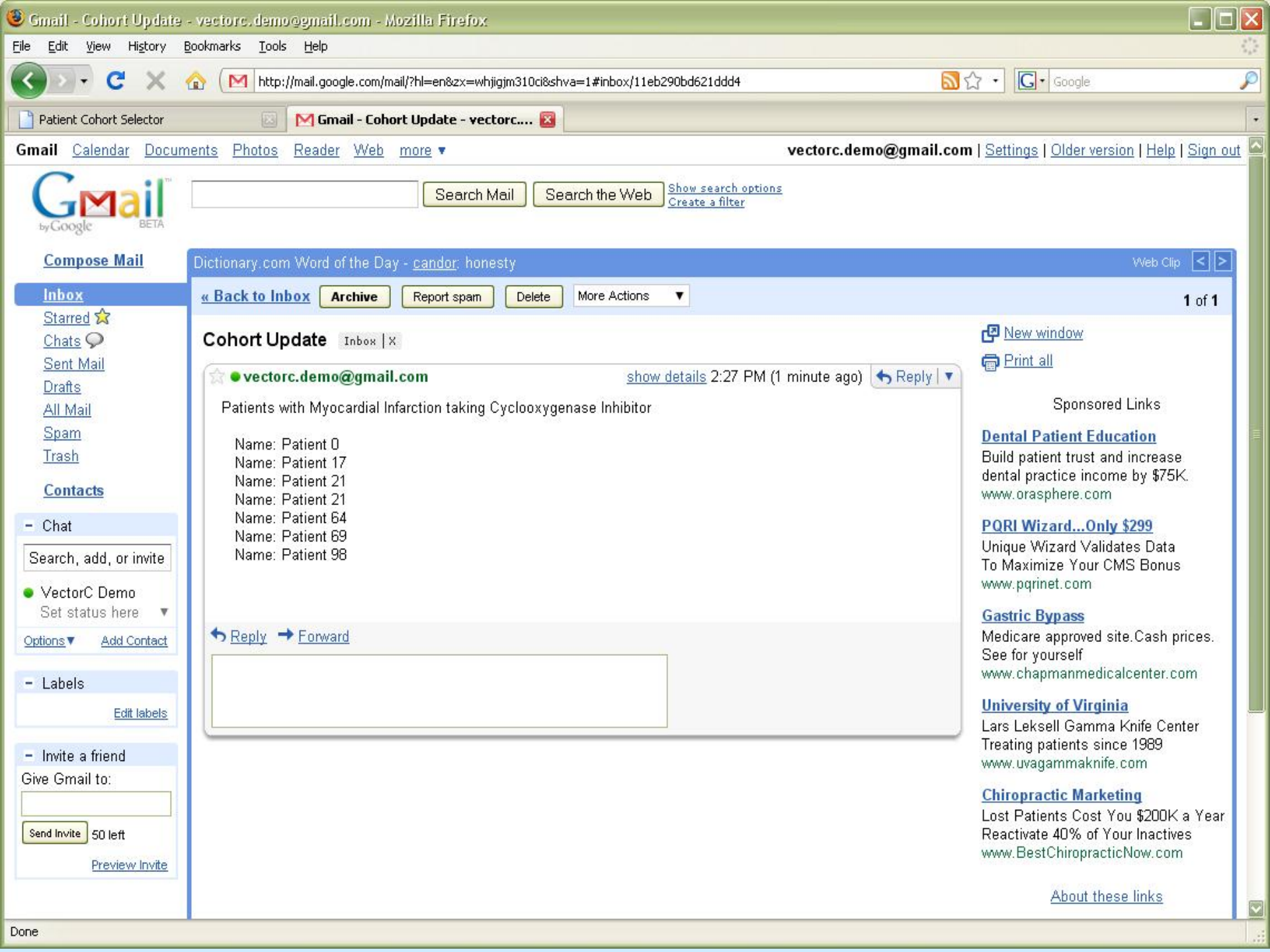
Umls Cui: C0700583

Full Syn: 2-[(2,6-Dichlorophenyl)amino]benzeneacetic Acid Monosodium SaltSNNCI , DICLOFENAC SODIUMPTFDAQTG126297Q , Diclofenac SodiumPTDCP09109 , Diclofenac SodiumPTNCI , VoltarenBRNCI , GP 45840CNNCI

Rdf Type:

Semantic Type: Pharmacologic Substance

Definition: NCIThe sodium salt form of diclofenac, a benzene acetic acid derivate and nonsteroidal anti-inflammatory drug (NSAID) with analgesic, antipyretic and anti-inflammatory activity. Diclofenac sodium is a non-selective reversible and competitive inhibitor of cyclooxygenase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Gmail - Cohort Update - vectorc....
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Gmail by Google BETA
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Search the Web
Show search options
Create a filter
Compose Mail
Inbox
Starred
Chats
Sent Mail
Drafts
All Mail
Spam
Trash
Contacts
Chat
Search, add, or invite
VectorC Demo
Set status here
Options ▾
Add Contact
Labels
Edit labels
Invite a friend
Give Gmail to:
Send Invite
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Preview Invite
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Web Clip
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Archive
Report spam
Delete
More Actions ▼
1 of 1
Cohort Update
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Reply
Patients with Myocardial Infarction taking Cyclooxygenase Inhibitor
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Name: Patient 17
Name: Patient 21
Name: Patient 21
Name: Patient 64
Name: Patient 69
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Reply
Forward
New window
Print all
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Dental Patient Education
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