Title (en)

INHIBITION OF THE FORMATION OF VASCULAR HYPERPERMEABILITY

Title (de)

VERHINDERUNG VON VASCULARER HYPERPERMEABILITÄT

Title (fr)

INHIBITION DE LA FORMATION D'UNE HYPERPERMEABILITE VASCULAIRE

Publication

EP 1126842 A2 20010829 (EN)

Application

EP 99962685 A 19991103

Priority

- US 9925903 W 19991103
- US 10746298 P 19981106

Abstract (en)

[origin: WO0027414A2] Vascular hyperpermeability in individuals is a prelude to a number of physiological events that are often deleterious. Among these events is the formation of edema, diapedesis, aberrant trans-endothelial exchange, extravasation, exudation and effusion, matrix deposition (often with abnormal stromal proliferation) and vascular hypotension. Vascular hyperpermeability and the subsequent events can be inhibited by the administration of a compound that inhibits the enzyme activity of the VEGF tyrosine kinase receptor known as KDR tyrosine kinase. Preferred administered compounds selectively inhibit the function of KDR tyrosine kinase but do not block the activity of Flt-1 tyrosine kinase which is another VEGF tyrosine kinase receptor.

[origin: WO0027414A2] Vascular hyperpermeability and the subsequent events such as macular edema, retinoblastoma, ocular ischemia, ocular inflammatory disease or infection, choroidal melanoma, edematous side-effects induced by iron chelation therapy, pulmonary edema, myocardial infarction, rheumatoid diseases, anaphylaxis, allergies, hypersensitive reactions, cerebral edema, brain tumor fluid-filled cysts, communicating hydrocephalus, carpal tunnel syndrome, organ damage resulting from a burn, irritation or infection, erythema multiforme, edematous macules and other disorders, brain tumors, tumor effusions, lung or breast carcinomas, ascites, pleural effusions, pericardial effusions, high altitude "sickness", radioanaphylaxis, radiodermatitis, glaucoma, conjunctivitis, choroidal melanoma, adult respiratory distress syndrome, asthma, bronchitis, ovarian hyperstimulation syndrome, polycystic ovary syndrome, menstrual swelling, menstrual cramps, stroke, head trauma, cerebral infarct or occlusion, hyotension, ulcerations, sprains, fractures, effusions associated with synovitis, diabetic complications, hyperviscosity syndrome, liver cirrhosis, microalbuminuria, proteinuria, oliguria, electrolyte imbalance, nephrotic syndrome, exudates, fibroses, keloid, can be inhibited by the administration of a compound that inhibits the enzyme activity of the VEGF tyrosine kinase receptor known as KDR tyrosine kinase. The preferred compound 4,5-dihydro-3-pyridin-4-yl-1(2)H-benzo[g]indazole selectively inhibits the function of KDR tyrosine kinase but do not block the activity of Flt-1 tyrosine kinase which is another VEGE tyrosine kinase receptor.

IPC 1-7

A61K 31/415

IPC 8 full level

A61K 45/00 (2006.01); **A61K 31/7105** (2006.01); **A61K 38/00** (2006.01); **A61K 38/17** (2006.01); **A61K 38/19** (2006.01); **A61K 39/395** (2006.01); **A61K 48/00** (2006.01); **A61P 1/04** (2006.01); **A61P 1/16** (2006.01); **A61P 3/10** (2006.01); **A61P 7/10** (2006.01); **A61P 9/00** (2006.01); **A61P 9/02** (2006.01); **A61P 9/10** (2006.01); **A61P 11/06** (2006.01); **A61P 11/16** (2006.01); **A61P 13/00** (2006.01); **A61P 13/02** (2006.01); **A61P 15/00** (2006.01); **A61P 17/02** (2006.01); **A61P 19/00** (2006.01); **A61P 27/00** (2006.01); **A61P 27/06** (2006.01); **A61P 27/12** (2006.01); **A61P 29/00** (2006.01); **A61P 35/00** (2006.01); **A61P 37/08** (2006.01); **A61P 43/00** (2006.01); **C07K 14/71** (2006.01)

CPC (source: EP KR)

A61K 39/395 (2013.01 - KR); **A61K 39/39533** (2013.01 - EP); **A61K 45/06** (2013.01 - EP); **A61P 1/04** (2017.12 - EP); **A61P 1/16** (2017.12 - EP); **A61P 3/10** (2017.12 - EP); **A61P 7/10** (2017.12 - EP); **A61P 9/00** (2017.12 - EP); **A61P 9/02** (2017.12 - EP); **A61P 9/10** (2017.12 - EP); **A61P 9/14** (2017.12 - EP); **A61P 11/06** (2017.12 - EP); **A61P 11/16** (2017.12 - EP); **A61P 13/00** (2017.12 - EP); **A61P 13/02** (2017.12 - EP); **A61P 15/00** (2017.12 - EP); **A61P 17/02** (2017.12 - EP); **A61P 19/00** (2017.12 - EP); **A61P 27/00** (2017.12 - EP); **A61P 27/06** (2017.12 - EP); **A61P 27/12** (2017.12 - EP); **A61P 29/00** (2017.12 - EP); **A61P 35/00** (2017.12 - EP); **A61P 37/08** (2017.12 - EP); **A61P 43/00** (2017.12 - EP); **C07K 14/71** (2013.01 - EP); **C12N 15/1137** (2013.01 - EP); **C12N 15/1138** (2013.01 - EP); A61K 38/00 (2013.01 - EP); A61K 2039/505 (2013.01 - EP); C12N 2310/11 (2013.01 - EP); C12N 2310/12 (2013.01 - EP)

C-Set (source: EP)

A61K 39/39533 + **A61K 2300/00**

Citation (search report)

See references of WO 0027414A2

Designated contracting state (EPC)

AT BE CH CY DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE

DOCDB simple family (publication)

WO 0027414 A2 20000518; **WO 0027414 A3 20000908**; AR 023912 A1 20020904; AU 1908000 A 20000529; BG 105476 A 20020228; BR 9915139 A 20010807; CA 2347916 A1 20000518; CN 1342077 A 20020327; CO 5150183 A1 20020429; CZ 20011564 A3 20020417; EP 1126842 A2 20010829; HU P0104302 A2 20020328; HU P0104302 A3 20021128; ID 29063 A 20010726; IL 142583 A0 20020310; JP 2002529421 A 20020910; KR 20010080952 A 20010825; NO 20012218 D0 20010504; NO 20012218 L 20010618; PL 348163 A1 20020506; SK 5052001 A3 20021008; TR 200102278 T2 20011221

DOCDB simple family (application)

US 9925903 W 19991103; AR P990105608 A 19991105; AU 1908000 A 19991103; BG 10547601 A 20010425; BR 9915139 A 19991103; CA 2347916 A 19991103; CN 99812924 A 19991103; CO 99070343 A 19991108; CZ 20011564 A 19991103; EP 99962685 A 19991103; HU P0104302 A 19991103; ID 20011000 A 19991103; IL 14258399 A 19991103; JP 2000580643 A 19991103; KR 20017005724 A 20010507; NO 20012218 A 20010504; PL 34816399 A 19991103; SK 5052001 A 19991103; TR 200102278 T 19991103