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Structure Match

As Drawn

Substructure (36K)

Filter by

- Substance Role
- Yield
- Number of Steps
- Experimental Protocols
- Reaction Type
- Reagent
 - Carbon monoxide (1)
 - (Dimethyl sulfide)trihydroboron (1)
 - Potassium carbonate (1)
 - Sodium borohydride (1)
- Catalyst
- Solvent

Reactions (6)

References

Scheme 1 (1 Reaction) View

Suppliers (88)

Expand Scheme

Scheme 2 (2 Reactions) View

Suppliers (7)

Suppliers (11)

Suppliers (4)

Experimental Protocols

MethodsNow™

Products: N-(1-Methylethyl)-N-phenylmethylbenzenemethanamine, Yield: 89%

Reactants: Acetone, Dibenzylamine

Reagents: Carbon monoxide

Catalysts: Rhodium trichloride

Solvents: Acetone

Procedure

1. Dissolve RhCl₃·4H₂O (1.1 mg, 2 mol %, 0.004 mmol) and dibenzylamine (0.2 mmol) in acetone (2.0 mmol) with 4Å molecular sieves (12 mg) in a 10 mL stainless steel autoclave.
2. Seal the autoclave.
3. Flush the autoclave three times with 10 atmosphere of CO.
4. Charge the autoclave with 50 atmosphere CO.
5. Place the reactor into a preheated oil bath to 160 °C.
6. After 48 hours, cool the reactor to room temperature.
7. Depressurize the reactor.
8. Transfer the reaction mixture into a flask.
9. Wash the autoclave with dichloromethane (2×1 mL).
10. Remove the combined solvent on a rotary evaporator.
11. Purify the residue by thin-layer chromatography (eluent: hexane/ethyl acetate/triethylamine (6/10/13)).

Transformation: Reductive Alkylation of Ammonia or Amines

Scale: milligram

Step-by-step synthetic procedures, extracted and summarized

- Save time with procedures summarized by CAS scientists
- Take step-by-step instructions directly to the lab
- Key chemical components of reactions are identified and linked to additional information

PATENTPAK A CAS SOLUTION

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ZOOM

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Key Substances in Patent

CAS RN 934660-93-2

CAS RN 934660-93-2

CAS Name Cobimetinib

Substance Detail

Reactions (47)

Suppliers (52)

References (234)

Edit Structure

Bcl-2 inhibitor are co-formulated.

10. The method of claim 10 wherein the MEK inhibitor and the selective Bcl-2 inhibitor are co-formulated in a pharmaceutical composition further comprising a pharmaceutically acceptable excipient.
11. The method of any of claims 1 through 7 wherein the MEK inhibitor is administered sequentially with the selective Bcl-2 inhibitor.
12. The method of claim 11 wherein the MEK inhibitor and the selective Bcl-2 inhibitor are formulated in separate orally available dosage forms.
13. The method of any of claims 1 through 12 wherein the MEK inhibitor inhibits MEK1, MEK2, or both MEK1 and MEK2.
14. The method of any of claims 1 through 13 wherein the MEK inhibitor is [3,4-difluoro-2-(2-fluoro-4-iodoanilino)phenyl]{3-hydroxy-3-[(2S)-piperidin-2-yl]azetidin-1-yl}methanone (cobimetinib) or a pharmaceutically acceptable salt thereof.
15. The method of any of claims 1 through 14 wherein the selective Bcl-2 inhibitor is 4-(4-{2-(4-chlorophenyl)-4,4-dimethylcyclohex-1-en-1-yl}methyl)piperazin-1-yl)-N-({3-nitro-4-[(tetrahydro-2H-pyran-4-ylmethyl)amino]phenyl}sulfonyl)-2-(1H-pyrrolo[2,3-b]pyridin-5-yloxy)benzamide (ABT-199) or a pharmaceutically acceptable salt thereof.
16. The method of any of claims 1 through 15 wherein the MEK inhibitor is cobimetinib or

Go straight to the chemistry in patents

- Patent chemistry is fully annotated with structures, nomenclature and more
- CAS expert scientists have identified chemistry locations, so SciFinder[®] reveals what patents usually obscure

Citation Map

Hepatocyte-specific deletion of DDB1 induces liver regeneration and tumorigenesis

By: Yamaji, Sachie; Zhang, Mingjun; Zhang, Jing; Endo, Yoko; Bibikov, Elena; Goff, Stephen P.; Cang, Yong
 Proceedings of the National Academy of Sciences of the United States of America (2010), 107(51), 22237-22242, S22237/1-S22237/5 | Language: English, Database: CAlplus

Abstract: Etiol. risk factors for hepatocellular carcinoma can be involved in the transformation process by directly targeting intracellular signaling pathways or by indirectly stimulating chronic cycles of hepatocyte destruction and regeneration. However, the contribution of these two routes to hepatocarcinogenesis has not been determined, partly because of the difficulty in distinguishing damaged and regenerated hepatocytes. Here we report that induced deletion of the damaged DNA binding protein 1 (DDB1) abrogates the self-renewing capacity of hepatocytes, resulting in compensatory proliferation of DD...

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