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Zuschläge

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Patritumab

Cat. No.:	HY-P99275
CAS No.:	1262787-83-6
Target:	EGFR; Akt; ERK; PARP; Survivin
Pathway:	JAK/STAT Signaling; Protein Tyrosine Kinase/RTK; PI3K/Akt/mTOR; MAPK/ERK Pathway; Stem Cell/Wnt; Cell Cycle/DNA Damage; Epigenetics; Apoptosis
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.

BIOLOGICAL ACTIVITY

Description	<p>Patritumab (Human Anti-ERBB3 Recombinant Antibody) is a neutralizing monoclonal antibody to ERBB3. Patritumab shows a synergy with Cetuximab (HY-P9905), potently inhibits the phosphorylation of EGFR, HER2, HER3, ERK, and AKT. Patritumab also induces cell apoptosis and suppresses the growth of pancreatic, non-small cell lung cancer, and colorectal cancer xenograft tumors^[1].</p>								
In Vitro	<p>Patritumab targets to the extracellular domain (ECD) of HER3 and (10 µg/mL; 5 d) induces DiFi-HRG4 cells apoptosis^[1]. Patritumab (10 µg/mL; 6 h) markedly inhibits the phosphorylation of HER3 and AKT, without affecting that of ERK, in DiFi-HRG4 cells^[1].</p> <p>Patritumab (10 µg/mL; 48 h) also induces the cleavage of PARP accompanied with both up-regulation of BIM and down-regulation of survivin expression^[1].</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p> <p>Western Blot Analysis^[1]</p> <table border="1"> <tr> <td>Cell Line:</td> <td>DiFi-HRG cells</td> </tr> <tr> <td>Concentration:</td> <td>10 µg/mL</td> </tr> <tr> <td>Incubation Time:</td> <td>6 hours</td> </tr> <tr> <td>Result:</td> <td>Inhibited the phosphorylation of HER3 and AKT as well as down-regulated survivin expression.</td> </tr> </table>	Cell Line:	DiFi-HRG cells	Concentration:	10 µg/mL	Incubation Time:	6 hours	Result:	Inhibited the phosphorylation of HER3 and AKT as well as down-regulated survivin expression.
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In Vivo	<p>Patritumab (1 mg/mouse; i.p.; twice a week for 4 weeks) combines with 1 mg Cetuximab and restores Cetuximab sensitivity in DiFi-HRG tumor xenografts model in mice^[1].</p> <p>Heregulin produced by colorectal cancer tumors harboring wild-type KRAS induces Cetuximab resistance, and that combination therapy with cetuximab and patritumab overcomes such resistance in vivo^[1].</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p> <table border="1"> <tr> <td>Animal Model:</td> <td>Female athymic nude mice (BALB/c; 5-6 weeks old) with DiFi-Mock1 or DiFi-HRG4 (s.c.)^[1]</td> </tr> <tr> <td>Dosage:</td> <td>1 mg/body</td> </tr> <tr> <td>Administration:</td> <td>Intraperitoneal injection; twice a week for 4 weeks</td> </tr> </table>	Animal Model:	Female athymic nude mice (BALB/c; 5-6 weeks old) with DiFi-Mock1 or DiFi-HRG4 (s.c.) ^[1]	Dosage:	1 mg/body	Administration:	Intraperitoneal injection; twice a week for 4 weeks		
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Result:

Individual Patritumab treatment had little effect on the growth of tumors formed by either cell line.
Combination of Cetuximab and Patritumab induced substantial regression of DiFi-HRG4 xenografts.

REFERENCES

[1]. Kawakami H, et al. The anti-HER3 antibody patritumab abrogates cetuximab resistance mediated by heregulin in colorectal cancer cells. *Oncotarget*. 2014 Dec 15;5(23):11847-56.

Caution: Product has not been fully validated for medical applications. For research use only.

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