Claims

1. Use of a combination of (i) a CBP/p300 bromodomain inhibitor and (ii) an EGFR inhibitor for the manufacture of a medicament for the treatment of a patient suffering from non-small cell lung cancer (NSCLC), wherein the NSCLC exhibits an oncogenic alteration in the EGFR.

2. The use of a combination according to claim 1, wherein the oncogenic alteration in the EGFR results in overactivation of the EGFR.

3. The use of a combination according to claim 1 or 2, wherein the oncogenic alteration is caused by a deletion and/or insertion in exon 18 or in exon 19 or in exon 20 of the EGFR gene; a kinase domain duplication in the EGFR gene; an amplification of the EGFR gene; at least one base mutation in the EGFR gene resulting in an amino acid substitution in the EGFR selected from the group consisting of L858R, G719S, G719A, G719C, V765A, T783A, S768I, S768V, L861Q, E709X, L819Q, A750P and combinations thereof, wherein X indicates any amino acid; and combinations of any of the foregoing.

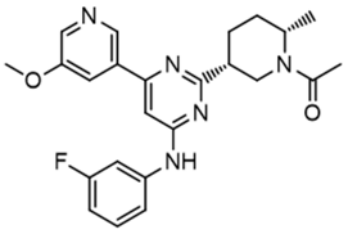
4. The use of a combination according to any one of claims 1 to 3, wherein the oncogenic alteration is caused by a deletion in exon 19 of the EGFR gene, preferably a deletion resulting in the deletion of E746-A750 or L747-E749 in the EGFR; at least one base mutation in the EGFR gene resulting in the amino acid substitution L858R or A750P in the EGFR; and combinations thereof.

5. The use of a combination according to any one of the preceding claims with the proviso that, if the NSCLC additionally exhibits a resistance alteration in the EGFR due to previous administration of an EGFR inhibitor, the EGFR inhibitor of the combination is not the EGFR inhibitor previously administered.

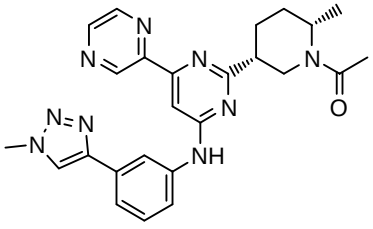
6. The use of a combination according to claim 5, wherein the resistance alteration in the EGFR is caused by at least one base mutation in the EGFR gene resulting in an amino acid substitution in the EGFR selected from the group consisting of T790M, C797X, L792X, G796X, L718Q, L718V, G724S, D761Y, V834L, T854A, and combinations thereof, wherein X indicates any amino acid.

7. The use of a combination according to claim 5 or 6, wherein the resistance alteration in the EGFR is caused by at least one base mutation in the EGFR gene resulting in the amino acid substitution T790M in the EGFR.

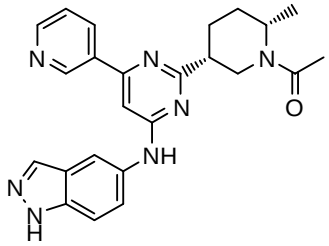
8. The use of a combination according to any one of the preceding claims, wherein the CBP/p300 bromodomain inhibitor is selected from the group consisting of



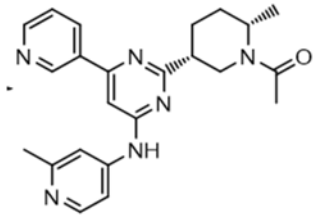
(Compound A),



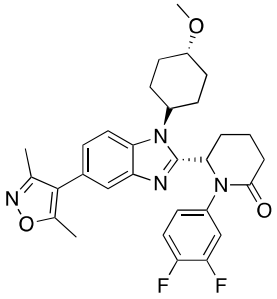
(Compound C),



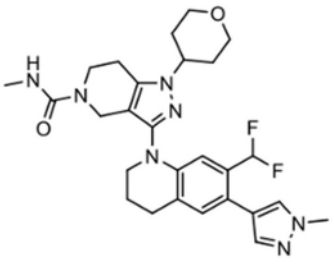
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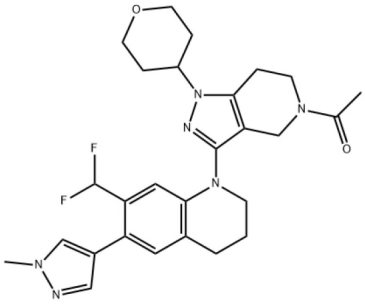
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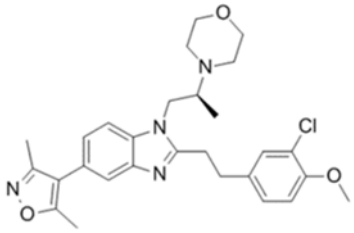
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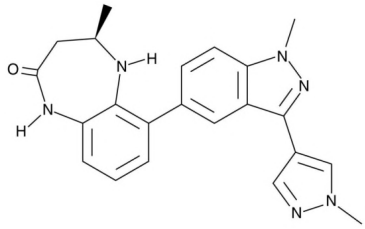
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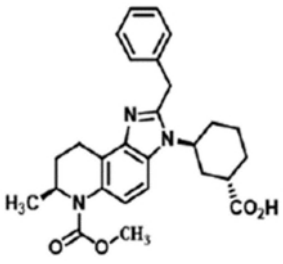
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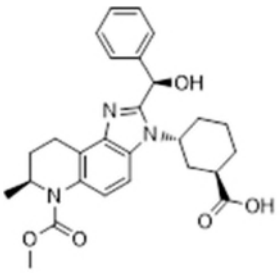
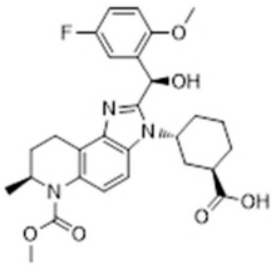
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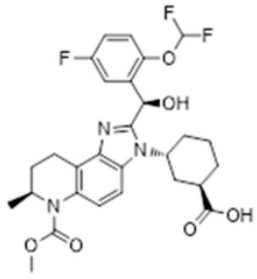


(CPI-637),



(FT-

6876),  (Compound 462),  (Compound 424)



and (Compound 515).

9. The use of a combination according to any one of the preceding claims, wherein the EGFR inhibitor is selected from the group consisting of ABBV-321, abivertinib, afatinib, alflutinib, almonertinib, apatinib, AZD3759, brigatinib, dacomitinib, DZD9008, erlotinib, gefitinib, icotinib, lapatinib, lazertinib, mobocertinib, nazartinib, neratinib, olafertinib, osimertinib, poziotinib, pyrotinib, rezivertinib, TAS6417, vandetanib, varlitinib, amivantamab, CDP1, cetuximab, GC1118, HLX07, JMT101, necitumumab, nimotuzumab, matuzumab, panitumumab, SCT200, SI-B001, zalutumumab, and combinations thereof.

10. The use of a combination according to any one of the preceding claims, wherein the combination is to be administered to the patient during each treatment cycle.

11. The use of a combination according to any one of the preceding claims, wherein (i) and (ii) are to be administered as separate dosage forms or comprised in a single dosage form.

12. The use of a combination according to claim 10, wherein the administration during each treatment cycle is concomitantly or sequentially if (i) and (ii) are to be administered as separate dosage forms.