

SUMMARY

[0005a] In one aspect, the present disclosure refers to a method of predicting susceptibility of a subject suffering from head and neck cancer to a treatment with an anti-cancer drug, wherein the method comprises detecting the presence or absence of a genetic alteration in a long non-coding RNA (lncRNA) that resides in an antisense strand of an oncogene; wherein the oncogene is EGFR, and the genetic alteration is a homozygous G>A mutation at Q787Q position in exon 20 of EGFR; wherein in case the homozygous genetic alteration is present, the subject is predicted to show improved susceptibility to the treatment with the anti-cancer drug compared to a subject not carrying the genetic alteration; and wherein the anti-cancer drug is an EGFR tyrosine kinase inhibitor. In another aspect, the present disclosure refers to method of predicting the susceptibility of a subject suffering from head and neck cancer to a treatment with an EGFR tyrosine kinase inhibitor, the method comprising determining whether either one or both of the following is given: i) the subject has a homozygous silent G>A genetic mutation at Q787Q position in exon 20 of EGFR; ii) the subject has lower EGFR-AS1 or EGFR-AS1 lncRNA expression level compared to subject that does not respond to treatment with an EGFR tyrosine kinase inhibitor; wherein in case one or both of i) to ii) is given, the subject is predicted to show improved susceptibility to a treatment with an EGFR tyrosine kinase inhibitor compared to a subject not showing any of i) to ii). In yet another aspect, the present disclosure refers to the use of an anti-cancer drug in the manufacture of a medicament for treating head and neck cancer in a subject, wherein the subject has been determined to have one or both of: i) a homozygous silent G>A mutation at Q787Q position in exon 20 of EGFR; and ii) lower EGFR-AS1 or EGFR-AS1 lncRNA expression level compared to subject that does not respond to treatment with an EGFR tyrosine kinase inhibitor; and wherein the anti-cancer drug is an EGFR tyrosine kinase inhibitor.

[0006] In one example, the present disclosure refers to a method of predicting susceptibility of a subject suffering from cancer to a treatment with an anti-cancer drug, wherein the method comprises detecting the presence or absence of a genetic alteration in long non-coding RNA (lncRNA) that resides in an antisense strand of an oncogene, wherein the genetic alteration alters or disrupts expression of the oncogene; wherein in case the

Claims

1. A method of predicting susceptibility of a subject suffering from head and neck cancer to a treatment with an anti-cancer drug, wherein the method comprises detecting the presence or absence of a genetic alteration in a long non-coding RNA (lncRNA) that resides in an antisense strand of an oncogene; wherein the oncogene is EGFR, and the genetic alteration is a homozygous G>A mutation at Q787Q position in exon 20 of EGFR; wherein in case the homozygous genetic alteration is present, the subject is predicted to show improved susceptibility to the treatment with the anti-cancer drug compared to a subject not carrying the genetic alteration; and wherein the anti-cancer drug is an EGFR tyrosine kinase inhibitor.
2. The method of claim 1, wherein the head and neck cancer is head and neck squamous cell carcinoma (HNSCC) or oral squamous cell carcinoma (OSCC).
3. The method of claim 1 or 2, further comprising measuring of either one or both of the following for predicting the susceptibility: i) whether the subject has lower EGFR-AS1 or EGFR-AS1 lncRNA expression level compared to a subject that does not respond to treatment with the anti-cancer drug; ii) whether the subject has higher EGFR isoform D/isoform A ratio compared to a subject that does not respond to treatment with the EGFR tyrosine kinase inhibitor.
4. The method of any one of claims 1 to 3, wherein the EGFR tyrosine kinase inhibitor is selected from the group consisting of gefitinib, erlotinib and afatinib.
5. A method of predicting the susceptibility of a subject suffering from head and neck cancer to a treatment with an EGFR tyrosine kinase inhibitor, the method comprising determining whether either one or both of the following is given:
 - i) the subject has a homozygous silent G>A genetic mutation at Q787Q position in exon 20 of EGFR;
 - ii) the subject has lower EGFR-AS1 or EGFR-AS1 lncRNA expression level compared to subject that does not respond to treatment with an EGFR tyrosine kinase inhibitor;

wherein in case one or both of i) to ii) is given, the subject is predicted to show improved susceptibility to a treatment with an EGFR tyrosine kinase inhibitor compared to a subject not showing any of i) to ii).

- 5 6. Use of an anti-cancer drug in the manufacture of a medicament for treating head and neck cancer in a subject, wherein the subject has been determined to have one or both of: i) a homozygous silent G>A mutation at Q787Q position in exon 20 of EGFR; and ii) lower EGFR-AS1 or EGFR-AS1 lncRNA expression level compared to subject that does not respond to treatment with an EGFR tyrosine kinase inhibitor; and
10 wherein the anti-cancer drug is an EGFR tyrosine kinase inhibitor.
7. The method of claim 5 or use of claim 6, wherein the head and neck cancer is head and neck squamous cell carcinoma (HNSCC) or oral squamous cell carcinoma (OSCC).
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8. The method of claim 5, the use of claim 6, or the method or use according to any of claim 7, wherein the EGFR tyrosine kinase inhibitor is selected from the group consisting of gefitinib, erlotinib and afatinib.
- 20 9. The method or use according to claim 8, wherein the EGFR tyrosine kinase inhibitor is gefitinib.