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2. 学会発表

H. 知的財産権の出願・登録状況

(予定を含む。)

1. 特許取得 なし
2. 実用新案登録 なし
3. その他 特になし

婦人科腫瘍、胎児組織の移植維持系の確立

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研究要旨：婦人科癌における腫瘍増殖のメカニズムの解明に向けて上皮-間質相互作用を解析する移植モデルを確立した。その研究システムにおいて腫瘍間質が HB-EGF 蛋白をオートクリンあるいはパラクリンの分泌し、子宮頸癌進展に重要な役割を持つことを示した。

A. 研究目的

抗癌剤耐性のメカニズムを解明するための Key として上皮-間質相互作用に着目した。上皮、間質を個別あるいはともに培養する婦人科癌の移植系を確立し、上皮-間質相互作用に関わる分子を同定することを目的とした。

B. 研究方法

1. 子宮頸癌組織のうち上皮成分と間質を個々に培養し、移植モデルを確立し上皮-間質相互作用が腫瘍増殖に与える影響を検討した。

2. HB-EGF に注目し、子宮頸癌細胞株を用いてノックダウンあるいは過剰発現させることにより腫瘍の増殖能の違いを解析する。

3. 手術摘出標本のパラフィン包埋切片を HB-EGF 抗体を用いて免疫染色を行い発現の有無・程度を解析する。

（倫理面への配慮）

研究に際し、細胞株以外に臨床検体の採取・利用も不可欠であるが、インフォームドコンセントを得たうえで、手術で摘出された組織を用いるため、患者に危害が及ぶことはなかった。

C. 研究結果

1. *in vitro* 実験系およびマウスへの Xenograft 移植モデル実験より、子宮頸癌において上皮-間質共培養した腫瘍は、上皮単独培養の腫瘍よりも有意に増殖能が有意に高いことが明らかになった。そのメカニズムのひとつとして HB-EGF のオートクリン機構が重要であることを証明した。実際に子宮頸癌組織の免疫染色の結果からその間質に高発現することが証明された。(Murata T. et al, Cancer Res. 2011 Nov 1;71(21):6633-42. Epub 2011 Oct 18)。

D. 考察

上皮-間質相互作用は子宮頸癌の発生・進展に重

要な役割を果たしており、治療の標的になりうると考えられる。HB-EGF はその候補分子のひとつであることが考えられる。

E. 結論

上皮-間質相互作用を破壊することが腫瘍の進展を阻止する可能性がある。これらの先駆的情報は今後、実際の臨床への応用の基盤となるものである。

F. 健康危険情報

G. 研究発表

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1. 特許取得 なし
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3. その他 特になし

II. 研究成果の刊行に関する一覧表

別紙4

研究成果の刊行に関する一覧表

書籍

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