Molecular evaluation of metastatic prostatic cancer and the study on enhancement of their effects treating for non-tratment and refractory prostatic cancer.

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Research Project

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Research Category		
Grant-in-Aid for Scientific Research (C)		
Allocation Type		
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Kanazawa University		
Principal Investigator		
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Project Status		
Completed (Fiscal Year 1998)		
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Keywords

prostatic cancer / AR / ER / interferon / P16 / 癌抑制遺伝子 / LNCaP / PC-3 / DU-145 / 分化誘導 / 5-azacytidine / retinoic acid / p16

Research Abstract

1)Molecular evaluation of hematogenous micro-metastasis from prostatic cancer

Statistically we have selected patients according to their clinical stage, these patient had been preserved data such as PSA values, PSA mRNA from peripheral blood cells in advance. We are supposed to evaluate the relations between PSA value and the PSA expression using RT PCR.Now we are observing the clinical course these patients.

2)Induction of expression in androgen receptor (AR) and estrogen receptor (ER) for four prostatic cancer cell lines.

In our study, we evaluated an induction of AR and ER in AR-negative prostatic cell line (PC3, DU-145, TSUPr-1) and AR-positive cell line (LNCap) exposing agents such as interferon (INF), 5-azacytidine, all-trans retinoic acid (ATRA) which have an activity of potential cell differentiation.

(1)There are no effects suppressing cell proliferation any cell lines using INF and ATRA.On the other hand, there are some effect suppressing cell proliferation using 5-azacytidine in these four prostatic cancer cell lines every concentrations. This results suggested that this induction activity is depend on cell cycle or promoter of transcription mechanism.

(2)A cell cycle point of view, this system is related to p16 which is known to cancer suppressor gene. The p16 mRNA express high in DU145, low in LNCap. Furthermore TSUPr-1 and PC-3 which never express p16 protein in themselves induce the expression of p16 using 5-azacytidine. This means that the promoter region of these four prostatic cell line is demethylated. This result shows sate possibility that suppression of cell proliferation result in hypemethylation exposing 5-azacytidine to prostatic cancer cell lines Next step we plan to evaluate the expression of genes which are related to controlling cell cycle, to demonstrate the telomerase activity as one of markers as proliferation and cell differentiation.

Report (3 results)

1998 Annual Research Report

Final Research Report Summary

1997 Annual Research Report

Research Products (13 results)

All Publications (13 results)

[Publications] 並木 幹夫・高 栄哲: "前立腺癌 ホルモン療法" 臨床科学. 33(12). 1576-1585 (1997)

[Publications] Iguchi K,Uchibayashi T: "Induction of necrosis by zinc in prostate carcinoma cells and identification of proteins increased in association with this induction." Eur.J.Biochem.253. 766-770 (1998)

[Publications] Kobayashi T.Uchibayashi T.: "A chick embryo model for mefastatic human prostate cancer" Eur.Urol.34. 154-160 (1998)

[Publications] Kitagawa Y.Uchibayashi T.: "Expression and tissue localization of membrane-types 1,2 and 3 matrix matalloproteinase in human uruthelial carcinoma" J.Urol. 160. 1540-1545 (1998)

[Publications] Namiki, M., Koh, E., Kunimi, K.: "Prostatic Cancer (2) Hormonal therapy" Rinshou Kagaku. 33(12). 1576-1585 (1997)

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[Publications] Kobayashi T., Koshida K., Endo Y., Imao T., Uchibayashi T., Sasaki T., Namiki M.: "A chick embryo model for metastatic human prostate cancer." Eur. Urol. 34. 154-160 (1998)

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[F	rublications] 高 栄哲・並木幹夫: "性腺・男子" Annual Review内分泌・代謝1998. 254-256 (1998)	~	
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_	rublications] 横山 修・他: "Change in bladder contractility associated with bladder over activity in Rats with cerebral infarction." J.Urol. 159. 577-580 998)	~	
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