

# Detection of telomerase activity and hTERT mRNA expression and its application for the diagnosis of gynecologic tumors

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# 2000 Fiscal Year Final Research Report Summary

## Detection of telomerase activity and hTERT mRNA expression and its application for the diagnosis of gynecologic tumors

Research Project

### Project/Area Number

11671604

### Research Category

Grant-in-Aid for Scientific Research (C)

### Allocation Type

Single-year Grants

### Section

一般

### Research Field

Obstetrics and gynecology

### Research Institution

Kanazawa University

### Principal Investigator

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### Project Period (FY)

1999 – 2000

### Keywords

Telomerase / hTERT / Diagnosis / Screening / Gynecologic tumor

### Research Abstract

Telomerase is a cellular reverse transcriptase which catalyzes the synthesis and extension of telomeric DNA. This enzyme is specifically activated in most malignant tumors, but is usually inactive in normal somatic cells, and within which telomeres therefore progressively shorten with cell division. A mechanism to maintain telomere stability is required for the cells to overcome replicative senescence, and telomerase activation may therefore be a rate-limiting or critical step in cellular immortality and oncogenesis. Human telomerase reverse transcriptase (hTERT) is a catalytic subunit protein of telomerase and is a critical determinant of its enzymatic activity. In the present study, we examined the utility of detecting telomerase activity and hTERT mRNA expression for the diagnosis and screening of gynecologic tumors. We found that most gynecologic cancers express telomerase activity while the corresponding normal tissues do not. Cervical and endometrial exfoliated cells were collected

from the patients with or without cervical or endometrial lesions and telomerase activity and hTERT mRNA expression were detected by the TRAP assays or RT-PCR assays. Approximately 90% of the patients with cervical or endometrial cancer were positive for telomerase activity or hTERT mRNA expression, while less than 10% of those without any lesions were so. 50 to 60% of the patients with premalignant lesions, such as squamous intraepithelial lesions (SIL) expressed telomerase activity or hTERT mRNA. These findings suggest that detection of telomerase activity or hTERT mRNA expression may be an useful tool for cancer diagnosis and screening.

## Research Products (8 results)

All Other

All Publications

[Publications] Takakura M, Kyo S et al: "Cloning of hTERT gene promoter and identification of proximal core promoter essential for transcriptional activation in immortalized and cancer cells" *Cancer Research*. 59. 551-557 (1999) ▼

[Publications] Kyo S. et al: "Estrogen activates telomerase" *Cancer Research*. 59. 5917-5921 (1999) ▼

[Publications] Kyo S. et al: "Sp1 cooperates with c-Myc to activate transcription of the human telomerase reverse transcriptase gene (hTERT)" *Nucleic Acids Research*. 28. 669-677 (2000) ▼

[Publications] Wang Z, Kyo S et al: "Progesterone regulates hTERT gene expression via activation of MAP Kinase signaling pathway" *Cancer Research*. 60. 5376-5381 (2000) ▼

[Publications] Takakura M, Kyo S, Kanaya T, Takeda J, Yutsudo M, Hirano H, Inoue M.: "Cloning of human telomerase reverse transcriptase gene promoter and identification of proximal core promoter essential for transcriptional activation in immortalized and cancer cells." *Cancer Res*. 59. 551-557 (1999) ▼

[Publications] Kyo S, Takakura M, Kanaya T, Wang Zhuo, Fujimoto K, Nishio Y, Orimo A, Inoue M.: "Estrogen activates telomerase." *Cancer Res*. 59. 5917-5921 (1999) ▼

[Publications] Kyo S, Takakura M, Kanaya T, Taira T, Kanaya T, Itoh H, Yutsudo M, Ariga H, and Inoue M.: "Sp1 cooperates with c-Myc to activate transcription of human telomerase reverse transcriptase (hTERT) gene." *Nucleic Acids Res.* 28. 669-677 (2000) ▼

[Publications] Wang Z, Kyo S, Takakura M, Tanaka M, Yatabe N, Maida Y, Fujiwara M, Hayakawa J, Ohmichi M, Koike K and Inoue M.: "Progesterone regulates human telomerase reverse transcriptase (hTERT) gene expression via activation of MAP kinase signaling pathway." *Cancer Res*. 60. 5376-5381 (2000) ▼

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