

# 細胞核と細胞質間の輸送の分子機構： インフルエンザウイルスによる研究

著者	福田 龍二
著者別表示	Fukuda Ryuji
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# 1994 Fiscal Year Final Research Report Summary

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## Studies on the mechanism of nuclear-cytoplasmic transport -From the studies of the influenza virus assembly

Research Project

### Project/Area Number

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05680589

### Research Category

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Grant-in-Aid for General Scientific Research (C)

### Allocation Type

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Single-year Grants

### Research Field

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Molecular biology

### Research Institution

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Kanazawa University

### Principal Investigator

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**FIKUDA Ryuji** Kanazawa Univ., School of Medicine, Professor, 医学部, 教授 (60027331)

### Co-Investigator(Kenkyū-buntansha)

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TAKIZAWA Takenori Kanazawa Univ., School of Medicine, Assistant Professor (joined in the project A, 医学部, 講師 (40192158)

HATADA Eriko Kanazawa Univ., School of Medicine, Instructor, 医学部, 助手 (70228469)

### Project Period (FY)

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1993 - 1994

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Nuclear-cytoplasmic Transport / Influenza Virus / Nuclear Pore / M1 Protein / NA Protein / Virus Assembly / Reverse Genetics / ウイルス粒子形成

### Research Abstract

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We have realized that the studies of the influenza virus growth provide several interesting problems concerning nuclear-cytoplasmic transport. In the early stage of virus assembly, the virus genomic RNA produced in the nucleus immediately with virus core proteins, and the ribonucleoprotein complex (vRNP) thus formed is transported through the nuclear pore into the cytosol, and then into budding virus particles on the plasma membrane. In the first part of this study, nuclear-cytoplasmic transport of vRNPs was studied by analysing the distribution of vRNAs employing an in situ hybridization technique. The analyzes were performed using wild-type virus as well as a ts mutant virus, ts-51, which harbors a mutation in the segment 7, and has a defect in the late

phase of virus growth. Nucleotide sequence analysis revealed a single amino acid change in the M1 protein. In the ts-51 virus-infected cells at a nonpermissive temperature, more than 95% of the vRNAs and the M1 protein remained in the nucleus, even at 6 hrpi and thereafter, when about 50% of them moved to the cytoplasm for the wild-type virus. These observations indicated that the M1 protein participated in the nuclear-cytoplasmic transport of the vRNAs. One hypothesis was that the M1 was associated with vRNPs in the nucleus forming possible M1-vRNP complexes, which were then transported into the cytosol. In the second part of this study, the virus assembly process in plasma membrane was investigated on the significance of the conserved sequence of the NA protein (its cytoplasmic domain and a successive sequence of the transmembrane domain) by the reverse genetic technique. It was indicated that both successive regions played important roles in the formation of the infective virus particles.

## Research Products (8 results)

All Other

All Publications (8 results)

- [Publications] K.Enami 他3名: "An influenza virus temperature-sensitive mutant defective in the nuclear-cytoplasmic transport of the negative-sense-viral RNAs" *Virology*. 194. 882-827 (1993) ▼
- [Publications] T.Takizawa 他5名: "Induction of programmed cell death(apoptosis)by influenza infection in tissue culture cells" *J.General Virology*. 74. 2347-2355 (1993) ▼
- [Publications] T.Takizawa 他4名: "Activation of the apoptotic Fas antigen-encoding gene upon influenza virus infection involuing spontaneously produced beta-interferon" *Virology*. (in press). (1995) ▼
- [Publications] H.Ohmori 他4名: "dinP,a new gene in Escherichia coli,whose product shows similarities to UmuC and its homolegues" *Mutation Research Letters*. (in press). (1995) ▼
- [Publications] K.Enami, Y.Qiao, R.Fukuda, and M.Enami: "An influenza virus temperature-sensitive mutant defective in the nuclear-cytoplasmic transport of the negative-sense viral RNAs." *Virology*. 194. 822-827 (1993) ▼
- [Publications] T.Takizawa, S.Matsukawa, Y.Higuchi, S.Nakamura, Y.Nakanishi and R.Fukida: "Induction of programd cell death (apoptosis) by influenza virus infection in tissue culture cells." *J.Gen.Virol*.74. 2347-2355 (1993) ▼
- [Publications] T.Takizawa, R.Fukuda, T.Miyawaki, K.Ohashi and Y.Nakanishi: "Activation of the apoptotic Fas antigen-encoding gene upon influenza virus infection involving spontaneously produced bera-interferon" *Virology*. (in press). (1995) ▼
- [Publications] H.Ohmori, E.Hatada, Y.Qiao, M.Tsuji and R.Fukuda: "dinP,a new gene in Escherichia coli, whose product shows similarities to UmuC and its homologues" *Mutation Research Letters*. (in press). (1995) ▼

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