は2010年春に開始予定である。

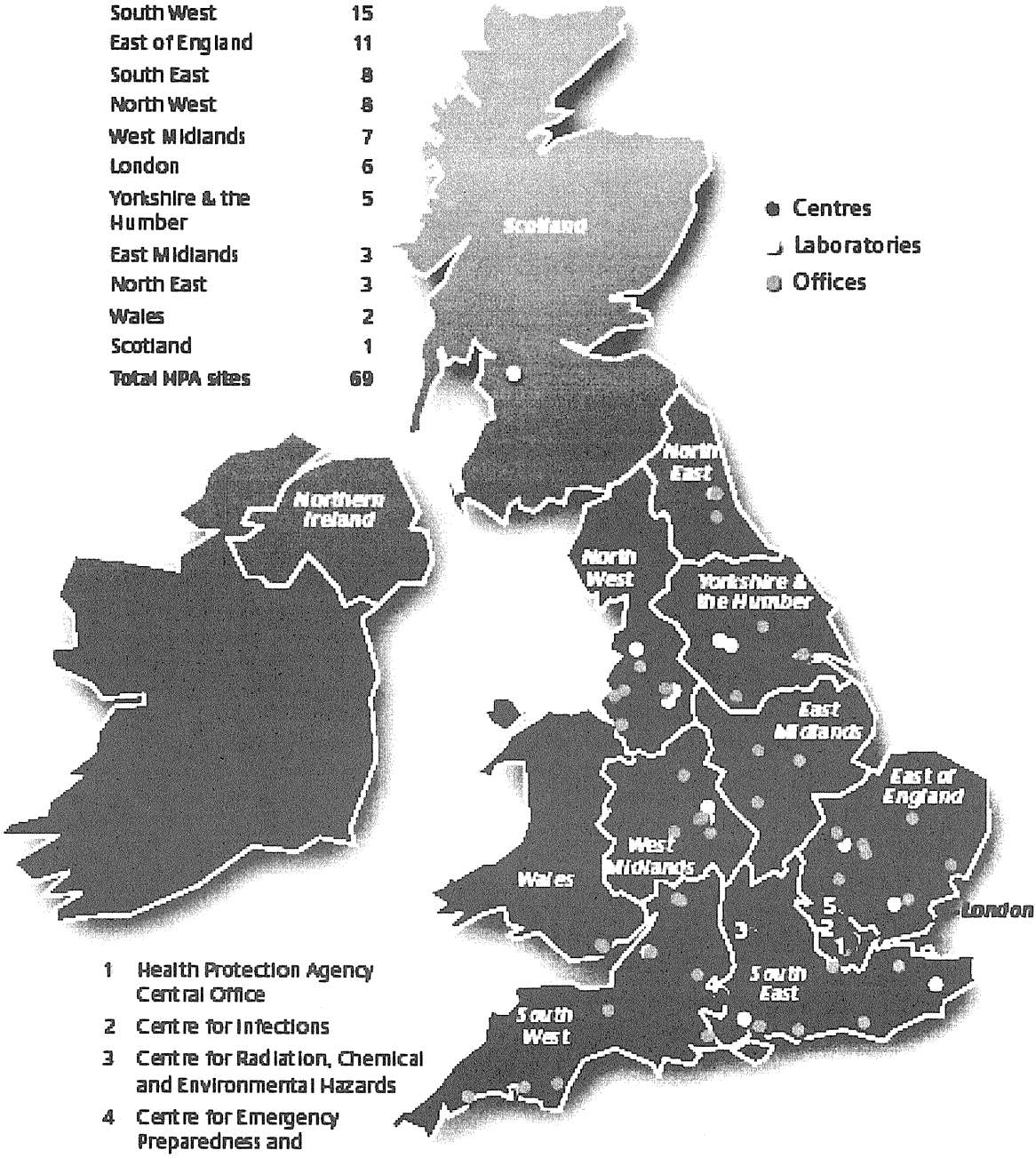
- 年間離職率は減少傾向にあり、2008/09年は12.05%であったのに対し、2009/10年は11.03%であった。自主退職率の減少が主な要因であり、2008/09年は7.6%であったのに対し、2009/10年は5.70%であった。
- 理事メンバー及び幹部職員200名に対する個別研修、人材育成担当職員への研修、100名を超える職員に対する平等影響アセスメント研修、職員向けのEラーニングパッケージの開発等、様々な種類の人材育成プログラムを実施した。
- 病欠率においては顕著な増加傾向にあり、前年の平均労働損失日数9.2日から9.76日に増えた。この増加傾向へ取り組むための計画が実施されているところである。

今後の取り組み：

- 2010年3月に合意された新しい人材計画に沿い、開発専門家グループの主導のもと、人材開発プログラムを効果的に実施する。
- 毒物学における管理専門家研修を皮切りに、専門性の養成と管理開発の両面を習得可能な訓練学校を設立する。
- 黒人や少数民族グループの上級職員が少ないことに対応するための行動計画を実施する。

＜HPA施設所在地＞

Region	HPA sites
South West	15
East of England	11
South East	8
North West	8
West Midlands	7
London	6
Yorkshire & the Humber	5
East Midlands	3
North East	3
Wales	2
Scotland	1
Total HPA sites	69



1 Health Protection Agency Central Office
2 Centre for Infections
3 Centre for Radiation, Chemical and Environmental Hazards
4 Centre for Emergency Preparedness and Response
5 National Institute for Biological Standards and Control

For more information see www.hpa.org.uk

www.hpa.org.uk

Ⅲ. 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

著者氏名	論文タイトル名	書籍全体の編集者名	書　籍　名	出版社名	出版地	出版年	ページ
山本	疫学統計ソフト Epi Info 日本語版		食中毒の疫学研修講座	日本食品衛生協会		2012	107-130

雑誌

発表者氏名	論文タイトル名	発表誌名	巻号	ページ	出版年
太田晶子,永井正規,川戸美由紀,橋本修二,村上義孝,多田有希,重松美加,安井良則,谷口清州.	感染症発生動向調査に基づく検討　第1報　インフルエンザA(HINI)2009流行の特徴.	日本公衆衛生雑誌	58(10)	特別付録:401	2011
川戸美由紀,橋本修二,太田晶子,永井正規,村上義孝,多田有希,重松美加,安井良則,谷口清州.	感染症発生動向調査に基づく検討　第2報　インフルエンザの定点の現状.	日本公衆衛生雑誌	58(10)	特別付録:401	2011
橋本修二,川戸美由紀,太田晶子,永井正規,村上義孝,多田有希,重松美加,安井良則,谷口清州.	感染症発生動向調査に基づく検討　第3報　インフルエンザの定点設計.	日本公衆衛生雑誌	58(10)	特別付録:401	2011
Otsuka N, Han HJ, Toyoizumi-Ajisaka H, Nakamura Y, Arakawa Y, Shibayama K, Kamachi K	Prevalence and genetic characterization of pertactin-deficient *Bordetella pertussis* in Japan	PLoS ONE	7(2)	e31985	2012
Suzuki T, Kataoka H, Ida T, Kamachi K, Mikuniya T	Bactericidal activity of topical antiseptics and their gargles against *Bordetella pertussis*	J Infect Chemother			in press
Kenri, T. Horino,A., et.al.	Complete Genome Sequence of Mycoplasma pneumoniae Type 2a Strain 309, Isolated in Japan	J. Bacteriol.	194	1253-1254	2012
Yamaguchi M, Sano Y, Dapat IC, Saito R, Suzuki Y, Kumaki A, Shobugawa Y, Dapat C, Uchiyama M, Suzuki H.	High frequency of repeated infections due to emerging genotypes of human respiratory syncytial viruses among children during eight	J Clin Microbiol.	49(3)	1034-40.	2011
中野貴司	インフルエンザウイルス	小児科	52巻8号	p1101-1109	2011年7月

Ⅳ．研究成果の刊行物・別刷

Jpn. J. Infect. Dis., 64, 493-498, 2011

Original Article

Molecular Characterization of Human Adenovirus Type 8 (HAdV-8), including a Novel Genome Type Detected in Japan

Arun Kumar Adhikary[1,3], Urmila Banik[2], Nobuhiko Okabe[3], and Tsuguto Fujimoto[3]*

[1]*Department of Microbiology, BGC Trust Medical College, Chandanaish, Chittagong;*
[2]*Department of Pathology, Chittagong Medical College, Chawkbazar, Bangladesh; and*
[3]*Infectious Disease Surveillance Center, National Institute of Infectious Diseases, Tokyo 162-8640, Japan*

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SUMMARY: Human adenovirus type 8 (HAdV-8) is a common agent of severe epidemic keratoconjunctivitis (EKC). Twenty-six strains were isolated from sporadic cases of EKC in the southern part of Japan between 1998 and 1999 and were identified as HAdV-8 by the neutralization method using type-specific antiserum against HAdV-8. A comparative analysis of different HAdV-8 genome types was performed using various molecular methods. Restriction enzyme analyses of genomic DNA were performed with *Bam*HI, *Hind*III, *Pst*I, *Sac*I, *Sal*I, and *Sma*I and identified 25 isolates as HAdV-8E and 1 isolate as HAdV-8J, a novel genome type. The genetic relatedness between HAdV-8J and the other genome types was calculated by pairwise comigrating restriction fragments. The new genome type was most genetically related to HAdV-8E. In a phylogram of both the hexon and fiber, HAdV-8J formed a monophyletic cluster with other genome types of HAdV-8. Although HAdV-8J was identified from a sporadic case of EKC, this strain may cause future outbreaks and thus warrants further monitoring.

INTRODUCTION

Several types of human adenoviruses (HAdVs), such as HAdV-1, HAdV-2 (species C), HAdV-3, HAdV-7, HAdV-11 (species B), HAdV-4 (species E), HAdV-8, HAdV-19a, HAdV-37, HAdV-53, HAdV-54, and HAdV-56 (species D), can cause ocular infections. Among them, HAdV-8 is an important causative agent of a severe form of viral conjunctivitis, known as epidemic keratoconjunctivitis (EKC) (1,2). Since its discovery in 1955 (3), HAdV-8 has been isolated throughout the world and continues to be the most common agent of EKC (4,5).

Until now, 4 genome types, namely HAdV-8A, HAdV-8B, HAdV-8E, and HAdV-8I have been identified in Japan using the restriction enzyme analysis (REA) (6–14). However, recent genome-sequence analysis suggests that HAdV-8I is actually a different type (HAdV-54) (18) that cross-reacts with HAdV-8 in serological tests and, therefore, has been misidentified as HAdV-8. Most of the genome types of HAdV-8 were isolated from outbreaks of EKC; afterwards, these strains were also found to cause sporadic cases of EKC. Therefore, it is crucial to molecularly examine isolates from sporadic cases in order to detect the emergence of new strains that may cause future outbreaks.

In the present retrospective study, a total of 26 HAdV-8 strains were isolated from sporadic cases of EKC between 1998 and 1999 from an eye clinic in the southern part of Japan. The isolates were analyzed with 6 endonucleases (*Bam*HI, *Hind*III, *Pst*I, *Sac*I, *Sal*I, and *Sma*I). One of the isolated strains revealed a new restriction pattern. This isolate was identified as a new genome type and was termed HAdV-8J based on the Asia-Pacific system of genome type classification. We performed phylogenetic analysis of the fiber knob and hypervariable regions of the hexon in order to uncover its evolutionary relationship with other oculopathogenic HAdVs.

*Corresponding author: Mailing address: Infectious Disease Surveillance Center, National Institute of Infectious Diseases, 1-23-1 Toyama, Shinjuku-ku, Tokyo 162-8640, Japan. Tel: +81-3-5285-1111, Fax: +81-3-5285-1129, E-mail: fujimo-t@nih.go.jp

MATERIALS AND METHODS

Viral strains: Twenty-six HAdV-8 strains were isolated from conjunctival scrapings from sporadic cases of EKC at an eye clinic in Miyakonojo, of the southern part of Japan, between 1998 and 1999. Of the 26 strains, 12 were isolated in 1998, and 14 were isolated in 1999. All strains were isolated for surveillance purposes and were obtained with informed consent from the patients. All strains were identified as HAdV-8 in a neutralization test using antisera obtained from American Type Culture Collection (ATCC; Rockville, Md., USA).

Virus propagation and extraction of viral DNA: Viral DNA was extracted from infected A549 cells (6). Once a cytopathic effect (CPE) greater than 75% was observed, cells were dislodged and pelleted by low-speed centrifugation. Cells were washed with phosphate-buffered saline (PBS) and resuspended in lysis buffer (10 mM Tris-HCl [pH 7.4], 10 mM EDTA, 1% SDS) for 15 min at room temperature. The suspension was then incubated with 200 μg/ml proteinase K (Sigma Chemical, St. Louis, Mo., USA) at 37°C for 1 h. NaCl was added to a final concentration of 1 M and further incubated at 4°C overnight to precipitate cellular DNA. The suspension was centrifuged at 15,000 g for 30 min. The supernatant was incubated with 30 μg of RNase A (Sigma Chemical) for 1 h and extracted twice in phenol/chloroform. The

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Virology

journal homepage: www.elsevier.com/locate/yviro

Letter to the Editor

Human adenovirus species C (HAdV-C) fiber protein

The published paper in your journal "Characterization of species C human adenovirus serotype 6 (AdV6)"; Virology 412 (2011) 19–27, has come into my attention. The fiber protein sequence alignment of the human adenovirus species C (HAdV-C) described in the paper was already done by us in the year 2004 (Adhikary et al. Journal Clin. Pathol 2004;57:612–17). However, we have found some dissimilarities in this article that we would like to bring to your attention and state them as follows:

Number of motifs in HAdV-1 (human adenovirus type 1), HAdV-2, HAdV-5, HAdV-6 fiber shaft

The primary sequence of the HAdV shaft consists of 15-residue pseudorepeats or motifs. The repeats are characterized by conserved hydrophobic residues at positions 1, 3, 9 and 11, and a conserved proline or glycine at position eight of the motif. Green et al. (EMBO Journal 1983;2:1357–65) predicted a secondary structure corresponding to these repeats comprising two three-residue β-strands separated by a five-residue turn containing the conserved proline or glycine, and at the end a four-residue turn. In the above mentioned research paper by Eric et al. 21 motifs were shown in case of HAdV-C1, HAdV-C2 and HAdV-C5 and 18 motifs in case of HAdV-C6. But this should have been 22 in case of HAdV-C1, HAdV-C2 and HAdV-C5 and 19 in HAdV-C6. We do not understand whether the authors have made any error in organizing and counting the motifs. It is well known that the knob of HAdV-C starts from the **WTTPQP** residue (Green et al. EMBO Journal 1983;2:1357–65, Signas et al. Journal Virol 1985;53:672–78, Adhikary et al. Journal Clin. Pathol 2004;57:612–17). But Eric et al. described that the knob started 15 residues earlier at the **GAIT** residue. As a result there was reduction of 1 motif, that is, 21 motifs in HAdV-1, HAdV-2 and HAdV-5 and 18 motifs in HAdV-6 instead of 22 and 19 motifs, respectively.

Total amino acid (AA) residue count in HAdV-6 fiber gene

Total AA residue count in case of HAdV-C6 was shown to be 547 residues. But this should have been 528 residues long. Alignment figure of fiber protein given in the article also revealed 528 AA residues. We do not understand whether they have applied a counting system which is very different from the usual.

In the year 2004, we published the research article "Heterogeneity of the fiber sequence in subgenus C adenoviruses" in the Journal of Clinical Pathology; 2004; 57:612–617. In that study we described a PCR method that amplified fiber genes of all the HAdV-C prototypes in a single tube reaction. Our result showed that the size of amplicon of HAdV-C6 was smaller than the other members of HAdV-C. In order to elucidate the smaller size of the amplicon we sequenced the fiber gene of prototype HAdV-C1 (adenoid 71) and HAdV-C6 (tonsil 99) which was deposited in the GenBank under the accession number AB125750 and AB125751 respectively. Alignment of predicted amino acid of fiber protein of HAdV-C revealed that the shaft of HAdV-C1, HAdV-C2 and HAdV-C5 were 22 motifs long whereas motif 15 to 17 were absent in case of HAdV-C6. It was revealed that smaller size of the amplicon was due to a shorter (19 motifs) shaft in HAdV-C6. We concluded in our study that the shorter shaft of HAdV-C6 may be responsible for a lower rate of infection.

Arun Kumar Adhikary
Department of Microbiology,
BGC Trust Medical College,
Chittagong, Bangladesh
Corresponding author at: Department of Microbiology, BGC Trust Medical College, Chandanaish, Chittagong, Bangladesh.
E-mail address: arun_adhikary@msn.com.

Tsuguto Fujimoto
Nobuhiko Okabe
National Institute of Infectious Diseases,
1-23-1 Toyama Shinjuku-ku,
Tokyo 162-8640, Japan
E-mail addresses: fujimo-t@nih.go.jp (T. Fugimoto).
okabenob@nih.go.jp (N. Okabe).

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Surveillance of Adenovirus D in Patients With Epidemic Keratoconjunctivitis From Fukui Prefecture, Japan, 1995–2010

Masako Nakamura,[1] Eiko Hirano,[1] Kazuaki Kowada,[1] Fubito Ishiguro,[1] Zenya Yamagishi,[2] Arun Kumar Adhikary,[3] Nozomu Hanaoka,[4] Nobuhiko Okabe,[4] Kiyosu Taniguchi,[4] and Tsuguto Fujimoto[4]*

[1]*Fukui Prefectural Institute of Public Health and Environmental Science, Fukui, Japan*
[2]*Yamagishi Eye Clinic, Fukui, Japan*
[3]*Department of Microbiology, BGC Trust Medical College, Chittagong, Bangladesh*
[4]*National Institute of Infectious Diseases, Tokyo, Japan*

Human adenoviruses species D (HAdV-D) are known to cause severe epidemic keratoconjunctivitis. However, the isolation rate of HAdV-D is not high, because HAdV-D is usually slow to propagate. Although new types of HAdV-D have been reported, accurate surveillance has not been performed because of difficulties in culturing the viruses and lack of a practical identification method. In this study, HAdV-Ds were detected and identified from patients with epidemic keratoconjunctivitis in the Fukui Prefecture during 1995–2010 by PCR, loop-mediated isothermal amplification (LAMP) of DNA, and conventional virus isolation and neutralization tests. All samples were subjected to culture and PCR and LAMP. A total of 124 strains of HAdV-D were detected from 157 patients with epidemic keratoconjunctivitis. The strains consisted of the following types: D8 (n = 8), D19 (n = 4), D37 (n = 40), D53 (n = 5), D54 (n = 66), and D56 (n = 1). Among these, D53, D54, and D56 are new types that have been reported recently. The results of this study demonstrated that new types of HAdV-D caused epidemic keratoconjunctivitis during 1995–2010, and included an outbreak of keratoconjunctivitis caused by HAdV-D54. The LAMP method was able to detect and identify HAdV-D53 and HAdV–D54 in 1 hr, and may therefore be applicable for use at the bedside. ***J. Med. Virol.***

KEY WORDS: new type adenovirus; loop-mediated isothermal amplification; PCR-sequencing; outbreak

INTRODUCTION

Human adenoviruses (HAdVs) cause a wide range of infections, including respiratory infections, eye diseases, and gastroenteritis [Wold and Marshall, 2007]. HAdVs are divided into seven species, A to G [Jones et al., 2007]. Among these species, human species D adenovirus (HAdV-D) includes several types that cause epidemic keratoconjunctivitis. HAdV-D type D8, -D19a, and -D37 are well known to cause severe epidemic keratoconjunctivitis, whereas the other types of HAdV-D usually do not [Aoki and Tagawa, 2002].

Recently, new types of HAdV-D have emerged as causative agents of severe epidemic keratoconjunctivitis. These include HAdV-D53 [Aoki et al., 2008; Walsh et al., 2009; Kaneko et al., 2011a], -D54 [Ishiko et al., 2008; Ishiko and Aoki, 2009; Kaneko et al., 2011b], and -D56 [Kaneko et al., 2011c; Robinson et al., 2011]. Like HAdV-D8, some types of HAdV-D are also fastidious and difficult to isolate from clinical samples [Kaneko et al., 2011b]. Moreover, HAdV-D types have cross-neutralizing reactions with other HAdV-D [Hierholzer et al., 1991]. Therefore, in this study, gene-based methods such as polymerase chain reaction (PCR) [Miura-Ochiai et al., 2007; Robinson et al., 2011] and loop-mediated isothermal amplification

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*Correspondence to: Tsuguto Fujimoto, Infectious Disease Surveillance Center, National Institute of Infectious Diseases, 1-23-1 Toyama, Shinjuku-ku, Tokyo 162-8640, Japan.
E-mail: fujimo-t@nih.go.jp

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Laboratory and Epidemiology Communications

Outbreak of Epidemic Keratoconjunctivitis Caused by Adenovirus Type 54 in a Nursery School in Kobe City, Japan in 2008

Kyoko Akiyoshi, Tomoko Suga, Kiyoka Fukui[1], Kiyosu Taniguchi[2], Nobuhiko Okabe[2], and Tsuguto Fujimoto[2]*

Kobe Institute of Health, Kobe 650-0046; [1]Fukui Clinic, Kobe 653-0836; and [2]National Institute of Infectious Diseases, Tokyo 162-8640, Japan

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Human adenovirus type 54 (HAdV-54) is a novel type of adenovirus (1) that has so far been detected only in Japan, and causes epidemic keratoconjunctivitis (EKC) (1–3). HAdV-54 was identified as the HAdV-8 variant strain before 2008, because antibodies to HAdV-54 showed cross-reactivity with those to HAdV-8 in a neutralization test (NT) (1,3). In this report, we describe an outbreak of HAdV-54 conjunctivitis in a nursery school in Japan in 2008.

An outbreak of EKC occurred from 5 June to 13 August 2008 at a nursery school in Kobe City, Japan. On 5 June 2008, a 1-year-old nursery school child was referred to a local ophthalmologist for conjunctival infection, conjunctival swelling, and eye discharge. The patient was clinically diagnosed with acute allergic conjunctivitis. Since the disease is non-infectious, the child continued to attend nursery school (total number of students = 106). On 10 June, 5 students of the same class showed similar symptoms of conjunctivitis. Their parents (n = 4) also showed similar clinical manifestations on 17 June. Gradually, the conjunctivitis spread among the 3–5-year-old children in the other classes. For the purpose of investigation, an ophthalmologist (consultant) for the school interviewed several students and teachers on 30 June. After clinical examinations, the ophthalmologist diagnosed the disease as EKC. The patients were advised to stay in their homes to prevent further spread of the disease. The school authorities prevented the students and the teachers who were suspected to have EKC from attending school. However, many students who were incorrectly diagnosed with acute allergic conjunctivitis in the early stages of EKC at two ophthalmologic clinics continued to attend school. The number of patients increased daily, and at least 30 students of the nursery school (30/106, 28%) developed conjunctivitis. The incidence of EKC gradually decreased and finally terminated in mid-August, before the school closed for the Japanese summer holiday. In addition to the usual symptoms and signs of EKC, some students presented with fever. The acute symptoms of EKC waned within a month in all patients. However, 10–14 days after the onset of the disease, 8 patients (53.3%) developed corneal opacity. The corneal opacity persisted for a prolonged period in 2 patients, and these patients were still being treated as of 2010.

From 27 June to 30 July 2008, the ophthalmologist for the school collected conjunctival scrapings from 15 EKC patients: school students (n = 6), their family members (n = 6), school staff (n = 2), and others (n = 1).

In 2000, we identified HAdV-8 in 2 isolates using NT (4), but these 2 isolates were observed to be HAdV-54 by Ishiko et al. who sequenced their complete genome (1). The complete genome sequence of HAdV-54 (Kobe-H strain) was deposited in GenBank (accession no. AB333801).

In 2008, 15 EKC samples were inoculated in FL, HEp-2, and Vero-E6 cells. Human adenovirus was isolated from 4 EKC patients. We sequenced the hexon-coding genes of the 4 strains. A PCR-sequencing method (5) was used with primers AdnU-S′2 and AdnU-A2. The primer sequences were as follows: AdnU-S′2, 5′-TTC CCC ATG GCN CAC AAY AC-3′, where N = A/T/C/G and Y = C/T and AdnU-A2, 5′-TGC CKR CTC ATR GGC TGR AAG TT-3′, where K = G/T and R = A/G. An aliquot (2 μL) of the extracted DNA was used as the template. Amplification reactions were conducted using 50 μl of the reaction mixture that contained the following: each of the primer pairs, 0.5 μM; each dideoxynucleotide, 200 μM; SpeedSTAR™ HS DNA Polymerase (TaKaRa Shuzo, Shiga, Japan), 1 U; Tris-HCl (pH 8.0), 10 mM; and KCl, 50 mM. Forty cycles of PCR were performed, and each cycle consisted of the following incubations: 95°C for 5 s, 50°C for 15 s, and 72°C for 10 s. PCR products (554 bp) were purified using a QIAquick PCR purification kit (Qiagen, Hilden, Germany) and used as templates for DNA sequencing reactions. Partial hexon gene sequences (350 bp) were determined and used to detect human adenoviruses types. The 4 isolates were identified as HAdV-54 because the sequences of the 4 strains were 100% identical to that of AdV-D54 (accession no. AB333801). Therefore, the PCR sequencing test is considered necessary for accurate identification of virus types.

The isolation rate of HAdV-54 was not high when compared to immunochromatographic test (ICT) kit,

*Corresponding author: Mailing address: Infectious Disease Surveillance Center, National Institute of Infectious Diseases, 1-23-1 Toyama, Shinjuku-ku, Tokyo 162-8640, Japan. Tel: +81-3-5285-1111, Fax: +81-3-5285-1129, E-mail: fujimo-t@nih.go.jp

Journal of Clinical Microbiology

High Frequency of Repeated Infections Due to Emerging Genotypes of Human Respiratory Syncytial Viruses among Children during Eight Successive Epidemic Seasons in Japan

Masahiro Yamaguchi, Yasuko Sano, Isolde C. Dapat, Reiko Saito, Yasushi Suzuki, Akihiko Kumaki, Yugo Shobugawa, Clyde Dapat, Makoto Uchiyama and Hiroshi Suzuki

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小 児 科

特集：*抗ウイルス薬の進歩とその使い方*

インフルエンザウイルス

中 野 貴 司

金 原 出 版 株 式 会 社

パンデミックインフルエンザA（H1N1）2009の特徴

中野貴司*

はじめに

パンデミックインフルエンザA（H1N1）2009による病像の特徴と，血清疫学データから考察した感染伝播様式，不顕性感染の頻度について述べる。

I パンデミックインフルエンザA（H1N1）2009の病像

1．重症度

パンデミックインフルエンザA（H1N1）2009（以下2009 pdm）が発生した当初，メキシコでの致命率を計算したところ2%近い値が出た。もし同じ病気に100人が罹って，そのうち2人が亡くなれば恐るべき重症疾患である。1918年にパンデミックを起こしたスペインインフルエンザの致命率が2～3%といわれ，それに匹敵する死者が出ることになる。しかし，その後各地域で調査した結果，そのような死亡者の多発はないことがわかってきた。

メキシコでは流行が始まってしばらくしてから，「肺炎の入院が多い」，「呼吸器感染症の患者が多い」ということから新しい疾患の発生がわかったのであろう。すなわち，医療機関を受診しない軽症者も実際にはたくさんいて，彼らは患者として報告されなかった。したがって，流行が始まった当初の致命率が，計算上は高く見積もられた可能性が高い。現在では，季節性のインフルエンザと同程度か，あるいはより軽症と考えられている。

ただし，2009 pdmと季節性インフルエンザの予後を単純に比較することはなかなか難しい。新型インフルエンザ騒動のなかで多くの患者が早期に医療機関を受診し適切な治療を受けた可能性や，2009 pdmでは高齢者の患者が少なかったことも死者減少に影響したであろう。

2．小児における2009 pdm

1）小児入院患者の特徴

小児二次救急基幹病院における，地域での流行開始当初の入院患者の概要[1]を紹介する。国立病院機構三重病院では，最初の患者が8月28日に入院し，その後11月30日までの約3か月間に93名が入院した。11月が流行のピークで65名が入院し，12月は入院患者数25名と減少に転じ，年明けには患者発生は終息に向かった。過去3シーズンの季節性インフルエンザでは，同じく約3か月間のそれぞれの流行期に約50名が入院していた。すなわち，2009 pdmによる入院患者数は，季節性インフルエンザよりはるかに多数であった。

短期間に多数の入院患者が集積したことについて，病状が中等症以上の患者が多かった可能性はもちろんあるが，パンデミック騒動による家族や外来診療医の不安のために，入院に至った例もあったかもしれない。それを裏づける理由として，入院患者の平均入院期間は3.93日と決して長くはなかった

入院患者の内訳は，男児63例（68%）・女児30

Nakano Takashi
* 川崎医科大学附属川崎病院小児科
〒700-8505 岡山市北区中山下2-1-80
TEL 086-225-2111 FAX 086-232-8343