猪流感(Swine Influenza) 是甲型（A 型）流感病毒引起的猪或人的一种急性、人畜共患呼吸道传染性疾病。这种病在猪中经常发生，很少导致猪的死亡（猪的病死率为 1-4%）。人类很少感染猪流感病毒，但也发现一些人类感染猪流感的病例，大多数是与病猪有过直接接触的人。2009 年 3 月，墨西哥和美国等先后发生人感染猪流感病毒，为 A 型流感病毒，H1N1 亚型猪流感病毒毒株，该毒株包含有猪流感、禽流感和人流感三种流感病毒的基因片断，是一种新型猪流感病毒，可以人传染人。人感染猪流感后的临床早期症状与流感类似，有发烧、咳嗽、疲劳、食欲不振等，还可以出现腹泻或呕吐等症状。病情可迅速进展，突然高热、肺炎，重者可以出现呼吸衰竭、多器官损伤，导致死亡。

一、病原学

猪流感病毒属于正粘病毒科(Orthomyxoviridae)，甲型流感病毒属(Influenza virus A) 。典型病毒颗粒呈球状，直径为 80 nm~120 nm，有囊膜。囊膜上有许多放射状排列的突起糖蛋白，分别是血凝素 HA、神经氨酸酶 NA 和 M2 蛋白。病毒颗粒内为核衣壳, 呈螺旋状对称, 直径为 10nm。猪流感病毒为单股负链 RNA 病毒，基因组约为 13.6 kb，由大小不等的 8 个独立片段组成。尽管不同亚型之间可以组成很多种流感病毒血清型，但是可造成人感染猪流感病毒的血清型主要有 H1N1、H1N2 和 H3N2。

猪流感病毒为有囊膜病毒，故对乙醚、氯仿、丙酮等有机溶剂均敏感，200 mL/ L 乙醚 4℃过夜，病毒感染力被破坏；对氧化剂、卤素化合物、重金属、乙醇和甲醛也均敏感，10 g/ L 高锰酸钾、1 mL/ L 升汞处理 3min，750 mL/ L 乙醇 5min，1 mL/ L 碘酊 5min，1 mL/ L 盐酸 3min 和 1mL/ L 甲醛 30min，均可灭活猪流感病毒。猪流感病毒对热敏感，56 ℃条件下，30min 可灭活；对紫外线敏感，但用紫外线灭活猪流感病毒能引起病毒的多重复活。

二、流行病学

在 1976 年美国发生所谓的“新泽西事件”中, 大约 500 人感染了猪流感 H1N1 亚型病毒, 该病毒与当时从猪体内分离的病毒相同,首次证实了在自然条件下, 猪流感病毒可从猪传播给人。1999 年 10 月，香港 1 名 10 月龄女婴感染了猪流感病毒 H3N2，现已完全康复。这些年来，世界各地都有人感染猪流感病毒不同病毒株的报道，但并没有大规模流行。近日墨西哥及美国等部分地区暴发了人感染猪流感疫情。世界卫生组织指出，墨西哥和美国感染的病例属于 H1N1 亚型猪流感病毒的一个相同毒株。

（一）传染源。

主要为病猪和携带病毒的猪，感染猪流感病毒的人也被证实可以传播病毒。感染这种病毒的动物均可传播。

（二）传播途径。

主要为呼吸道传播，也可通过接触感染的猪或其粪便、周围污染的环境或气溶胶等途径传播。某些毒株如 H1N1 可在人与人之间传播，其传染途径与流感类似，通常是通过感染者

咳嗽或打喷嚏等。

（三）易感人群。

普遍易感。患者多数年龄在 25 岁至 45 岁之间，目前报道以青壮年为主，应注意老人和儿童。

（四）高危人群。

从事养猪业者、在发病前 1 周内去过养猪、销售及宰杀等场所者以及接触猪流感病毒感染材料的实验室工作人员为高危人群。

人感染猪流感常发生在冬春季节，猪感染猪流感一般发生在夏秋季节。

三、临床表现

潜伏期一般 1 至 7 天左右，较流感、禽流感潜伏期长。

（一）临床症状。

人感染猪流感后的早期症状与普通人流感相似，包括发热、咳嗽、喉痛、身体疼痛、头痛、发冷和疲劳等，有些还会出现腹泻或呕吐、肌肉痛或疲倦、眼睛发红等。

部分患者病情可迅速进展，来势凶猛、突然高热、体温超过 39℃，甚至继发严重肺炎、急性呼吸窘迫综合征、肺出血、胸腔积液、全血细胞减少、肾功能衰竭、败血症、休克及 Reye 综合征、呼吸衰竭及多器官损伤，导致死亡。

（二）体征。

肺部体征常不明显，部分患者可闻及湿罗音或有肺部实变体征等。

（三）预后。

人感染猪流感的预后与感染的病毒亚型有关，大多预后良好；而感染 H1N1 者预后较差，病死率约为 6%。

（四）实验室检查。

1．外周血象：白细胞总数一般不高或降低。重症患者多有白细胞总数及淋巴细胞减少，并有血小板降低；

2．血清学诊断：可使用间接 ELISA、抗原捕捉 ELISA、荧光免疫法等；

3．反转录-聚合酶链式反应（RT-PCR）：由于 PCR 技术具有简便、快速、灵敏、特异性强等特点,已用于猪流感病毒基因的检测和分子流行病学调查等；

4．病毒分离：从患者呼吸道标本中（咽拭子、口腔含漱液、鼻咽或气管吸出物、痰或肺组织）分离猪流感病毒。常用的方法有鸡胚接种法和细胞培养法。现有的诊断方法中，病毒分离法是较敏感的，但需要 2-3 周时间。

（五）胸部影像学。

合并肺炎时肺内可见片状影像。严重病例片状影像范围广泛。

四、诊断

人感染猪流感的诊断主要结合流行病学史、临床表现和病原学检查等，临床上早发现、早诊断是治疗的关键。

（一）人感染猪流感的诊断标准。

1．医学观察病例：曾到过猪流感疫区，或与病猪及猪流感患者有密切接触史，1 周内出现流感临床表现者。列为医学观察病例者，对其进行 7 天医学观察（根据病情可以居家或医院隔离）。

2．疑似病例：曾到过疫区，或与病猪及猪流感患者有密切接触史（也可流行病学史不详），1 周内出现流感临床表现，呼吸道分泌物、咽试子、痰液、血清 H 亚型病毒抗体阳性或核酸检测阳性。

3．临床诊断病例：被诊断为疑似病例，且与其有共同暴露史的人被诊断为确诊病例者。

4．确诊病例：从呼吸道标本或血清中分离到特定病毒；RT-PCR 对上述标本检测，有猪流感病毒 RNA 存在，经过测序证实，或两次血清抗体滴度 4 倍升高，可确诊为人感染猪流感。

人感染猪流感诊疗流程见附件

（二）人感染猪流感的鉴别诊断。

人感染猪流感应注意与流感、禽流感、上感、肺炎、SARS、传染性单核细胞增多症、巨细胞病毒感染、军团菌肺炎、衣原体、支原体肺炎等鉴别。

五、治疗

（一）对症支持。

对疑似和确诊患者应进行就地隔离治疗，强调早期治疗。

对人感染猪流感目前主要是综合对症支持治疗。注意休息、多饮水、注意营养，密切观察病情变化；发病初 48 小时是最佳治疗期，对高热、临床症状明显者，应拍胸片，查血气。

（二）药物治疗。

1．抗病毒治疗：应及早应用抗病毒药物，可试用奥司他韦（oseltamivir 达菲）。达菲是一种神经氨酸酶抑制剂，对猪流感病毒可能有抑制作用，剂量 75mg Bid, 疗程 5 天，儿童慎用。从美国最近的猪流感病毒感染者中分离出的病毒对奥司他韦和扎那米韦（zanamivir）是敏感的，对金刚烷胺和金刚乙胺耐药。

2．抗生素：如出现细菌感染可使用抗生素。

（三）中医辨证治疗。

1．毒袭肺卫。

症状：发热、恶寒、咽痛、头痛、肌肉酸痛、咳嗽。

治法：清热解毒，宣肺透邪。

参考方药：炙麻黄、杏仁、生石膏、柴胡、黄芩、牛蒡子、羌活、生甘草。

常用中成药：莲花清瘟胶囊、银黄类制剂、双黄连口服制剂。

2．毒犯肺胃。

症状：发热、或恶寒，恶心、呕吐、腹痛腹泻、头身、肌肉酸痛。

治法：清热解毒，化湿和中。

参考方药：葛根、黄芩、黄连、苍术、藿香、姜半夏、苏叶、厚朴。

常用中成药：葛根芩连微丸、藿香正气制剂等。

3．毒壅气营。

症状：高热、咳嗽、胸闷憋气、喘促气短、烦躁不安、甚者神昏谵语。

治法：清气凉营。

参考方药：炙麻黄、杏仁、瓜蒌、生大黄、生石膏、赤芍、水牛角。

必要时可选用安宫牛黄丸以及痰热清、血必净、清开灵、醒脑静注射液等。

六、预防

（一）控制传染源。

开展人间和猪类流感疫情监测。一旦发现猪类或其它动物感染猪流感病毒，应按照《动物检疫法》有关规定，对疫源地进行彻底消毒，对病人及疑似病人进行隔离。

（二）切断传播途经。

对发现有病猪的养殖场、曾销售病猪肉的摊档、患者所在单位、家庭等进行消毒，对病死猪等废弃物应立即就地销毁或深埋；收治病人的门诊和病房按禽流感、SARS 标准做好隔离消毒；标本按照不明原因肺炎病例要求进行运送和处理。

（三）保护健康人群。

养成良好的个人卫生习惯，充足睡眠、勤于锻炼、减少压力、足够营养；避免接触流感样症状（发热，咳嗽，流涕等）或肺炎等呼吸道病人；注意个人卫生，经常使用肥皂和清水洗手，尤其在咳嗽或打喷嚏后；避免接触生猪或前往有猪的场所；避免前往人群拥挤场所；咳嗽或打喷嚏时用纸巾遮住口鼻，然后将纸巾丢进垃圾桶；如在境外出现流感样症状（发热，咳嗽，流涕等），应立即就医（就医时应戴口罩），并向当地公共卫生机构和检验检疫部门说明。

（四）加强院感控制措施。

对于疑似病人或确诊病人进行隔离并佩戴外科口罩；医务人员要做好个人防护，加强手卫生，使用快速手消毒剂进行手消毒；发热门诊和感染性疾病科等重点部门的医务人员应佩戴外科口罩，必要时佩戴护目镜或防护口罩；对发热门诊和感染性疾病科等重点部门应当加强室内通风。

猪流感疫苗：目前只有用于猪的猪流感疫苗，还没有专门用于人类的。就目前情况看，普通的流感疫苗对预防人类猪流感没有明显效果。

附件:人感染猪流感诊疗流程.doc

猪流感病毒基因序列：
http://www.ncbi.nlm.nih.gov/genomes/FLU/SwineFlu.html

科技文献

目　录

最新猪 H1N1 流感病毒序列.....6

1．2009 年 3 至 4 月美国南加州两儿童感染 A 型(H1N1)猪流感病毒.....9

2．美国出现对奥司他韦有耐药性的 A 型(H1N1)流感病毒感染.....9

3．中国从猪体中检测的人源 H1N1 和 H3N2 流感病毒分离和基因序列分析.....11

4．美国威斯康星州出现人感染三源重配株 A 型（H1N1）猪流感病毒病例 11

5．对奥司他韦有耐药性的 A 型(H1N1)流感病毒院内感染的发病率和死亡率.....12

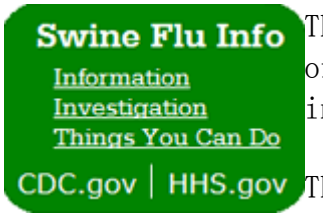
6．欧洲禽和猪 A 型 H1N1 流感病毒不同的演化轨迹 13

7．2008 年 11 月西班牙 Aragon 地区出现人感染猪流感 A 型(H1N1)病毒病例 14

最新猪 H1N1 流感病毒序列

Newest swine influenza A (H1N1) sequences

Swine Flu 2009 Outbreak



The Centers for Disease Control and Prevention (CDC) and other health officials are actively tracking the recent emergence of human cases of swine influenza A (H1N1) virus infection.

The following swine influenza A (H1N1) sequences were submitted to NCBI and are available in GenBank:

April 29, 2009, 1 submitted by University of Regensburg, Germany; 3 by CDC:

	PB2	PB1	PA	HA	NP	NA	MP	NS
Influenza A virus (A/Regensburg/Germany/01/2009(H1N1))							FJ970928	
Influenza A virus (A/California/06/2009(H1N1))						FJ971075		FJ971074
Influenza A virus (A/California/08/2009(H1N1))				FJ971076				

April 28, 2009, 34 submitted by CDC:

	PB2	PB1	PA	HA	NP	NA	MP	NS

Swine Flu 2009 Outbreak

Influenza A virus (A/New York/19/2009(H1N1))				FJ969509				
Influenza A virus (A/New York/20/2009(H1N1))				FJ969542		FJ969541		
Influenza A virus (A/Kansas/03/2009(H1N1))				FJ969523				
Influenza A virus (A/Ohio/07/2009(H1N1))				FJ969521 FJ969535		FJ969520 FJ969534		
Influenza A virus (A/California/04/2009(H1N1))	FJ969516		FJ969515		FJ969512	FJ969517	FJ969513	FJ969514
Influenza A virus (A/California/07/2009(H1N1))	FJ969530	FJ969531	FJ969529 FJ969539	FJ969540	FJ969536		FJ969527 FJ969537	FJ969528 FJ969538
Influenza A virus (A/California/08/2009(H1N1))							FJ969518 FJ969532	FJ969519 FJ969533
Influenza A virus (A/California/10/2009(H1N1))				FJ969511			FJ969510	
Influenza A virus (A/Texas/04/2009(H1N1))	FJ969525	FJ969526	FJ969524					
Influenza A virus (A/Texas/05/2009(H1N1))		FJ969522						

April 27, 2009, 40 submitted by CDC:

Swine Flu 2009 Outbreak

	PB2	PB1	PA	HA	NP	NA	MP	NS
Influenza A virus (A/California/04/2009(H1N1))	FJ966079	FJ966080	FJ966081	FJ966082	FJ966083	FJ966084	FJ966085	FJ966086
Influenza A virus (A/California/05/2009(H1N1))	FJ966955	FJ966958	FJ966957	FJ966952	FJ966953	FJ966956	FJ966954	
Influenza A virus (A/California/06/2009(H1N1))	FJ966963	FJ966965	FJ966964	FJ966960	FJ966961		FJ966962	
Influenza A virus (A/California/07/2009(H1N1))	FJ966976	FJ966978	FJ966977	FJ966974			FJ966975	
Influenza A virus (A/California/09/2009(H1N1))				FJ966971		FJ966973	FJ966972	
Influenza A virus (A/Texas/04/2009(H1N1))				FJ966982	FJ966979	FJ966981	FJ966980 FJ966983	
Influenza A virus (A/Texas/05/2009(H1N1))			FJ966970	FJ966959	FJ966967	FJ966969	FJ966968	FJ966966

摘自 http://www.ncbi.nlm.nih.gov/genomes/FLU/SwineFlu.html

1．2009 年 3 至 4 月美国南加州两儿童感染 A 型(H1N1)猪流感病毒

MMWR Morb Mortal Wkly Rep. 2009 Apr 24;58(15):400-2.

Swine Influenza A (H1N1) infection in two children--Southern California, March-April 2009.

Centers for Disease Control and Prevention (CDC).

On April 17, 2009, CDC determined that two cases of febrile respiratory illness occurring in children who resided in adjacent counties in southern California were caused by infection with a swine influenza A (H1N1) virus. The viruses from the two cases are closely related genetically, resistant to amantadine and rimantadine, and contain a unique combination of gene segments that previously has not been reported among swine or human influenza viruses in the United States or elsewhere. Neither child had contact with pigs; the source of the infection is unknown. Investigations to identify the source of infection and to determine whether additional persons have been ill from infection with similar swine influenza viruses are ongoing. This report briefly describes the two cases and the investigations currently under way. Although this is not a new subtype of influenza A in humans, concern exists that this new strain of swine influenza A (H1N1) is substantially different from human influenza A (H1N1) viruses, that a large proportion of the population might be susceptible to infection, and that the seasonal influenza vaccine H1N1 strain might not provide protection. The lack of known exposure to pigs in the two cases increases the possibility that human-to-human transmission of this new influenza virus has occurred. Clinicians should consider animal as well as seasonal influenza virus infections in their differential diagnosis of patients who have febrile respiratory illness and who 1) live in San Diego and Imperial counties or 2) traveled to these counties or were in contact with ill persons from these counties in the 7 days preceding their illness onset, or 3) had recent exposure to pigs. Clinicians who suspect swine influenza virus infections in a patient should obtain a respiratory specimen and contact their state or local health department to facilitate testing at a state public health laboratory.
PMID: 19390508 [PubMed - indexed for MEDLINE]

2．美国出现对奥司他韦有耐药性的 A 型(H1N1)流感病毒感染

JAMA. 2009 Mar 11;301(10):1034-41. Epub 2009 Mar 2.
Comment in:

JAMA. 2009 Mar 11;301(10):1066-9.

Infections with oseltamivir-resistant influenza A(H1N1) virus in the United States.

Dharan NJ, **Gubareva LV**, **Meyer JJ**, **Okomo-Adhiambo M**, **McClinton RC**, **Marshall SA**, **St George K**, **Epperson S**, **Brammer L**, **Klimov AI**, **Bresee JS**, **Fry AM**; **Oseltamivir-Resistance Working Group**.Collaborators (44)

Ahmed F, Nalluswami K, Bascom SD, Berisha V, Boulton RB, Cohen J, Corkren E, Crockett M, Dao C, Deyde VM, Hall H, Patton M, Sheu TG, Wallis TR, Hales C, Sunenshine R, Erhart LM, Komatsu K, Sunenshine R, Goodin K, Hanson M, Koepsell J, Rietberg K, Haupt T, Laplante JM, Mingle L, Linchangco P, Louie J, Moore A, McHugh L, Moore Z, Najera R, Park S, Rajan R, Person CJ, Yousey-Hindes K, Powell RJ, Reisdorf E, Shult PA, Van TT, Richards SM, Siston A, Stoute A, Van Houten C Jr.

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CONTEXT: During the 2007-2008 influenza season, oseltamivir resistance among influenza A(H1N1) viruses increased significantly for the first time worldwide. Early surveillance data suggest that the prevalence of oseltamivir resistance among A(H1N1) viruses will most likely be higher during the 2008-2009 season. OBJECTIVES: To describe patients infected with oseltamivir-resistant influenza A(H1N1) virus and to determine whether there were any differences between these patients and patients infected with oseltamivir-susceptible A(H1N1) virus in demographic or epidemiological characteristics, clinical symptoms, severity of illness, or clinical outcomes. DESIGN, SETTING, AND PATIENTS: Influenza A(H1N1) viruses that were identified and submitted to the Centers for Disease Control and Prevention by US public health laboratories between September 30, 2007, and May 17, 2008, and between September 28, 2008, and February 19, 2009, were tested as part of ongoing surveillance. Oseltamivir resistance was determined by neuraminidase inhibition assay and pyrosequencing analysis. Information was collected using a standardized case form from patients with oseltamivir-resistant A(H1N1) infections and a comparison group of patients with oseltamivir-susceptible A(H1N1) infections during 2007-2008. MAIN OUTCOME MEASURES: Demographic and epidemiological information as well as clinical information, including symptoms, severity of illness, and clinical outcomes. RESULTS: During the 2007-2008 season, influenza A(H1N1) accounted for an estimated 19% of circulating influenza viruses in the United States. Among 1155 influenza A(H1N1) viruses tested from 45 states, 142 (12.3%) from 24 states were resistant to oseltamivir. Data were available for 99 oseltamivir-resistant cases and 182 oseltamivir-susceptible cases from this period. Among resistant cases, median age was 19 years (range, 1 month to 62 years), 5

patients (5%) were hospitalized, and 4 patients (4%) died. None reported oseltamivir exposure before influenza diagnostic sample collection. No significant differences were found between cases of oseltamivir-resistant and oseltamivir-susceptible influenza in demographic characteristics, underlying medical illness, or clinical symptoms. Preliminary data from the 2008-2009 influenza season identified resistance to oseltamivir among 264 of 268 influenza A(H1N1) viruses (98.5%) tested. CONCLUSIONS: Oseltamivir-resistant A(H1N1) viruses circulated widely in the United States during the 2007-2008 influenza season, appeared to be unrelated to oseltamivir use, and appeared to cause illness similar to oseltamivir-susceptible A(H1N1) viruses. Circulation of oseltamivir-resistant A(H1N1) viruses will continue, with a higher prevalence of resistance, during the 2008-2009 season.

3．中国从猪体中检测的人源 H1N1 和 H3N2 流感病毒分离和基因序列分析

Biochem Biophys Res Commun. 2007 Apr 27;356(1):91-6. Epub 2007 Feb 27.

Isolation and genetic analysis of human origin H1N1 and H3N2 influenza viruses from pigs in China.

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Influenza A viruses of subtypes H1N1 and H3N2 have been reported widely in pigs, associated with clinical disease. These mainly include classical swine H1N1, avian-like H1N1, and human-like or avian-like H3N2 viruses. From 2005 to 2006, we carried out swine influenza virus surveillance in eight provinces of China. Here we report, for the first time, the isolation and genetic analysis of a human-like influenza H1N1 virus from a pig in a farm of Guangdong province of southern China, a host suspected to generate new pandemic strains through genetic reassortment. Each of the eight gene segments is of human origin. Phylogenetic analysis indicates that these genes form a human lineage, suggesting that this virus is the descendant of recent human H1N1 influenza viruses. In addition, four swine H3N2 viruses were also isolated. The three H3N2 viruses from Guangdong province are descendants of recent human viruses, while an H3N2 virus from Heilongjiang province derives from early human viruses. Isolation and genetic analysis of human H1N1 and H3N2 influenza viruses from pigs in China provides further evidence about the interspecies transmission of human influenza viruses to pigs and emphasizes the importance of reinforcing swine influenza virus surveillance.

4．美国威斯康星州出现人感染三源重配株 A 型（H1N1）猪流感病毒病例

Emerg Infect Dis. 2008 Sep;14(9):1470-2.

Human case of swine influenza A (H1N1) triple reassortant virus infection, Wisconsin.

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Zoonotic infections with swine influenza A viruses are reported sporadically. Triple reassortant swine influenza viruses have been isolated from pigs in the United States since 1998. We report a human case of upper respiratory illness associated with swine influenza A (H1N1) triple reassortant virus infection that occurred during 2005 following exposure to freshly killed pigs.

5．对奥司他韦有耐药性的 A 型(H1N1)流感病毒院内感染的发病率和死亡率

JAMA. 2009 Mar 11;301(10):1042-6. Epub 2009 Mar 2.

Morbidity and mortality associated with nosocomial transmission of oseltamivir-resistant influenza A(H1N1) virus.

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CONTEXT: The sudden emergence and rapid spread of oseltamivir-resistant influenza A(H1N1) viruses with neuraminidase (NA) gene H274Y amino acid substitution is the hallmark of global seasonal influenza since January 2008. Viruses carrying this mutation are widely presumed to exhibit attenuated pathogenicity, compromised transmission, and reduced lethality. OBJECTIVE: To investigate nosocomial viral transmission in a cluster of patients with influenza A(H1N1) virus infection. DESIGN, SETTING, AND PATIENTS: Descriptive outbreak investigation of 2 hematopoietic stem cell transplant recipients and an elderly patient who developed hospital-acquired influenza A virus infection following exposure to an index patient with community-acquired H274Y-mutated influenza A(H1N1) virus infection in a medical ward at a Dutch university hospital in February 2008. The investigation included a review of the medical records, influenza virus polymerase chain reaction and culture, phenotypic oseltamivir and zanamivir susceptibility determination, and hemagglutinin chain 1 (HA(1)) gene and NA gene sequence analysis. MAIN

OUTCOME MEASURE: Phylogenetic relationship of patient cluster influenza A(H1N1) viruses and other 2007-2008 seasonal influenza A(H1N1) viruses. RESULTS: Viral HA(1) and NA gene sequence analysis from the 4 patients revealed indistinguishable nucleotide sequences and phylogenetic clustering of H274Y-mutated, oseltamivir-resistant influenza A(H1N1) virus, confirming nosocomial transmission. Influenza virus pneumonia (3 patients) and attributable mortality (2 patients) during active infection was observed in patients with lymphocytopenia at onset. CONCLUSION: Seasonal oseltamivir-resistant influenza A(H1N1) viruses with NA gene H274Y mutation are transmitted and retain significant pathogenicity and lethality in high-risk patients.

6．欧洲禽和猪 A 型 H1N1 流感病毒不同的演化轨迹

J Virol. 2009 Mar 18.

Different evolutionary trajectories of European avian-like and classical swine H1N1 influenza A viruses.

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In 1979 a lineage of avian-like H1N1 influenza A virus emerged in European swine populations independently from the 'classical' swine H1N1 virus lineage that had circulated in pigs since the 'Spanish' influenza pandemic of 1918. To determine whether these two distinct lineages of swine-adapted A/H1N1 viruses have evolved in similar ways, as might be expected given their common host species and origin from avian-like A/H1N1 ancestors, we compared patterns of nucleotide and amino acid change in whole genome sequences of both groups. An analysis of nucleotide compositional bias across all 8 genomic segments for the two swine lineages showed a clear lineage-specific bias, although a segment-specific effect was also apparent. As such, there only appears to be a relatively weak host-specific selection pressure. Strikingly, despite each lineage evolving in the same species host for decades, amino acid analysis revealed little evidence for either parallel or convergent changes. These findings suggest that although adaptation due to evolutionary lineages can be distinguished, there are functional and structural constraints on all gene segments, and that the evolutionary trajectory of each lineage of swine A/H1N1 virus has a strong historical contingency. Thus, in the context of emergence of an influenza A virus strain via a host-switch event, it is difficult to predict what specific polygenic changes are needed for mammalian adaptation.

7．2008 年 11 月西班牙 Aragon 地区出现人感染猪流感 A 型(H1N1)病毒病例

Euro Surveill. 2009 Feb 19;14(7). pii: 19120.

Human case of swine influenza A (H1N1), Aragon, Spain, November 2008.

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A human case of swine influenza A (H1N1) in a 50-year-old woman from a village near Teruel (Aragon, in the north-east of Spain), with a population of about 200 inhabitants, has been reported in November 2008.

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