Table. Cost-effectiveness analysis of alternative human papillomavirus vaccination strategies*

Strategy	Discounted		Incremental†		
	Cost	QALY	Cost	QALY	ICER ($/QALY)‡
No vaccination	$72,659,302	2,698,711	–	–	–
12-y-old girls	$74,042,990	2,699,178	$1,383.688	467	Dominated
18-y-old women + 18–24-y-old female catch-up	$73,553,847	2,699,192	$894,545	481	$1,860
15-y-old girls + 15–24-y-old female catch-up	$73,895,046	2,699,214	$341,199	22	$15,509
12-y-old girls and boys	$78,707,825	2,699,327	$4,812,779	113	Dominated
12-y-old girls + 12–24-y-old female catch-up	$74,815,667	2,699,343	$920,621	129	$7,137
18-y-old women and men + 18–24-y-old female and male catch-up	$77,535,383	2,699,385	$2,719,716	42	$64,755
15-y-old girls and boys + 15–24-y-old female and male catch-up	$78,455,750	2,699,404	$920,367	19	$48,440
12-y-old girls and boys + 12–24-y-old female catch-up	$79,746,357	2,699,461	$1,290,607	57	$22,642
12-y-old girls and boys + 12–24-y-old female and male catch-up	$81,761,210	2,699,506	$2,014,853	45	$44,775

*QALY, quality-adjusted life year; ICER, incremental cost-effectiveness ratio; $, US dollars.
†Based on discounted costs reported by Elbasha et al. (*1*).
‡Compared with the preceding nondominated strategy. Strategy A is dominated if there exists another strategy, B, that is more effective and less costly than strategy A.

12-year-old girls only is dominated by the vaccination of 18-year-old women plus a catch-up strategy (women 18–24 years of age), although older groups have lower coverages.

In addition, I point out 2 particulars. First, epidemiology of HPV varies between countries (*2*), probably because of differences in culture and sexual habits. Thus, vaccination at older ages should be considered in countries in which prevalence of adolescent sexual activity or HPV is low. Second, higher vaccine coverage in older groups would decrease ICERs of these strategies (*1*). Both facts could reflect the real situation in some countries, e.g., Spain (*2*,*3*).

In conclusion, economic evaluations of HPV vaccination strategies should have broader sensitivity analysis to include as many country-specific realities as possible. To avoid misunderstandings that could lead policymakers to misallocate funds, these results should be evident to readers.

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Letters

Letters commenting on recent articles as well as letters reporting cases, outbreaks, or original research are welcome. Letters commenting on articles should contain no more than 300 words and 5 references; they are more likely to be published if submitted within 4 weeks of the original article's publication. Letters reporting cases, outbreaks, or original research should contain no more than 800 words and 10 references. They may have one Figure or Table and should not be divided into sections. All letters should contain material not previously published and include a word count.

Distemper in a Dolphin

To the Editor: Deaths caused by new members of the genus *Morbillivirus*, family Paramyxoviridae (*1*), have occurred in recent decades among phocine and cetacean species, particularly harbor seals (*Phoca vitulina*) in 1988 (*2*) and 2002 (*3*). Endangered Mediterranean striped dolphins (*Stenella coeruleoalba*) died in 1990 and 1991 (*4*), and common dolphins (*Delphinus delphis ponticus*) from the Black Sea died in 1994 because of infection with dolphin morbillivirus (DMV) (*5*). A similar virus caused deaths in bottlenose dolphins (*Tursiops truncatus*) in the Gulf of Mexico from 1987 through 1994 (*6*). Closely related morbilliviruses caused deaths in harbor porpoises (*Phocoena phocoena*) in European waters in 1988 (*7*) (*Porpoise morbillivirus*) and endangered Mediterranean monk seals (*Monachus monachus*) in 1997 (*8*) (*Monk seal morbillivirus*). After these epidemics, the viruses disappeared and no marine or terrestrial reservoirs have been identified.

In January 2007, a moribund, subadult, white-beaked dolphin (*Lagenorhynchus albirostris*) was found stranded on the North Friesian coast of Germany. The animal was humanely

killed and a complete necropsy was performed. The main lesion was a nonsuppurative meningoencephalitis with neuronal degeneration and few eosinophilic cytoplasmic inclusion bodies characteristic of a viral disease. Lungs showed suppurative and interstitial pneumonia. Paraffin-embedded sections of brain were examined for morbillivirus antigen by using an immunoperoxidase technique. We used various monoclonal antibodies that recognize different morbilliviruses. Tissues from a seal infected with phocine distemper virus and a dog with canine distemper were used as positive controls. Tissues from a white-beaked dolphin that underwent an autopsy in 2006 were used as negative controls. In the diseased dolphin, morbillivirus antigen was found exclusively in neurons and glial cells of the brain (Figure, panel A).

Frozen tissue samples and blood were examined for morbillivirus nucleic acid by reverse transcription–PCR with a set of universal morbillivirus primers that are specific for highly conserved regions of virus nucleoprotein (N) (*9*) and phosphoprotein (P) (*10*). A 457-bp amplicon of the P gene (GenBank accession no. EF451565) and a 287-bp amplicon of the N gene (GenBank accession no. EF469546) were detected in brain tissue. Our isolate, DMV/DE/2007, showed homologies of 99% with the N gene and 98% with the P gene of DMV isolated from Mediterranean striped dolphins. Phylogenetic analysis showed that isolate DMV/DE/2007 is closely related to DMV (Figure, panel B), porpoise morbillivirus, and monk seal morbillivirus (*8*).

Histologic changes in the dolphin resembled those of distemper in seals (*3*), porpoises (*7*), and other dolphins (*4*–*6*). Identification of morbillivirus antigen in diseased tissues and isolation of genome fragments of a morbillivirus provide conclusive evidence for a primary etiologic role of this virus. Sequencing of the virus and phylogenetic comparison showed that the virus is closely related to previously described dolphin morbillivirus and porpoise and monk seal morbilliviruses (*8*). To our knowledge, this is the first report of morbillivirus infection in a white-beaked dolphin in German waters and in a marine mammal since the last epidemic among harbor seals in northern Europe in 2002. Isolation of DMV has not been reported since 1994.

Our findings indicate that DMV is still circulating in some marine mammals. Similar to infections in terrestrial hosts, morbillivirus infections may occur in marine mammals in cycles without overt clinical disease in susceptible animals, as documented for harbor seals (*2*,*3*). Serum samples collected from 1995 through 1999 from cetacean species in various regions were positive for DMV, but porpoises and striped dolphins showed a decrease in humoral immunity, making them vulnerable to new epidemics. No data exist on seroprevalence of morbillivirus-specific antibodies

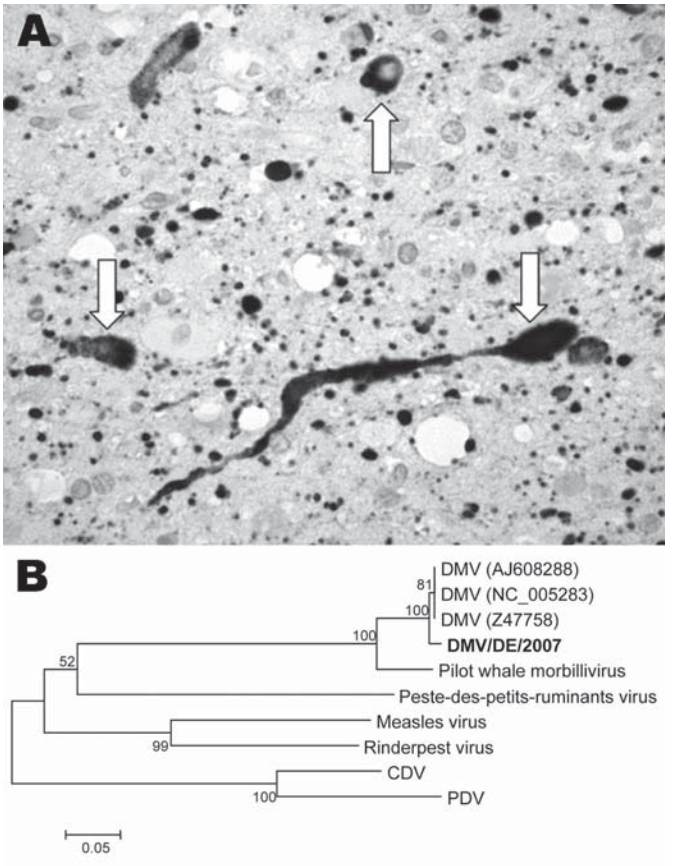

Figure. A) Immunohistologic demonstration of morbillivirus antigen in cytoplasm and nuclei of neurons (arrows) and glial cells in the brain of a white-beaked dolphin, using a monoclonal antibody (GenWay, San Diego, CA, USA) against nucleoprotein of canine distemper virus (CDV)/phocine distemper virus (PDV) visible as numerous black dots (magnification ×630). B) Unrooted neighbor-joining phylogenetic tree constructed by using 353 nt from the gene coding for the morbillivirus phosphoprotein. Alignments were calculated with ClustalX version 1.83 (http://bips.u-strasbourg.fr/fr/documentation/ClustalX). Bootstrapping (values indicated in %) was performed with 1,000 replicates using MEGA 3.1 software (www.megasoftware.net/mega.html). The new isolate from this study is shown in **boldface**. The following sequences were included: dolphin morbillivirus (DMV) (GenBank accession nos. NC_005283, Z47758, AJ608288), pilot whale morbillivirus (AF200817), Peste-des-petits-ruminants virus (NC_006383), measles virus (NC_001498), Rinderpest virus (NC_006296), CDV (NC_001921), and PDV (D10371). Scale bar shows nucleotide substitutions per site.

in white-beaked dolphins. We do not know how the dolphin contracted the infection and whether this remains an isolated case or the beginning of a new zoonosis.

White-beaked dolphins are found in moderate and subarctic waters of the Atlantic Ocean between the eastern coast of North America and northern Europe. They may migrate hundreds of kilometers within days. Therefore, these dolphins may play a role as a reservoir and vector for this morbillivirus, which is infectious for harbor porpoises, bottlenose dolphins, and other cetacean species (*10*). The reappearance of a morbillivirus represents a serious threat to susceptible marine mammals in northern European and American waters, with potentially devastating consequences and possibly the beginning of a new epidemic.

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Bartonella australis sp. nov. from Kangaroos, Australia

To the Editor: During April–May 1999, 3 *Bartonella* isolates (AUST/NH1, AUST/NH2, AUST/NH3) were cultivated and established from the blood of 5 *Macropus giganteus* gray kangaroos from central coastal Queensland, Australia. We used multigene sequencing to evaluate whether these *Bartonella* isolates fulfill the minimum requirements for classification as a new species.

DNA from each *Bartonella* isolate was extracted by using the QIAamp tissue kit (QIAGEN, Hilden, Germany) according to the manufacturer's instructions. Partial PCR amplification and sequencing of the genes encoding the 16S rDNA (*rrs*), citrate synthase (*gltA*), β-subunit of the RNA polymerase (*rpoB*), and cell division protein (*ftsZ*), as well as for the 16S–23S rDNA intergenic spacer (ITS) were attempted by using previously described primers and conditions (*1*). *Bartonella* sp. isolates AUST/NH1 to AUST/NH3 exhibited identical sequences for all 4 genes and the spacer studied, and isolate AUST/NH1 was selected as type strain among kangaroo isolates. Similarity rates between strain Aust/NH1 and validated *Bartonella* species (online Appendix Table, available from www.cdc.gov/EID/content/13/12/1961-appT.htm) ranged from 84.7% to 91.6%, from 97.5% to 98.5%, from 79.6% to 87.2%, from 85.4% to 95.0%, and from 83.5% to 87.1% for the ITS and *rrs*, *gltA*, *rpoB,* and *ftsZ* genes, respectively. Therefore, for each of these 4 genes or the spacer, strain AUST/NH1 exhibited similarity rates with all other species lower than the cutoffs published to classify *Bartonella* isolates within a validated species (*1*). It may thus be regarded as a new species.

To estimate the genomic G+C content of strain AUST/NH1, we amplified and sequenced its *ftsY* gene as described (*2*) by using the BartftsyF (5′-ATGACAAAAYCYTTTATMAA-3′) and BartftsyR (5′-TCATGAGTGTCTTCCTGC-3′) primers. The *ftsY* G+C content was 37.7%; the calculated genomic G+C content was 39.51%. The *ftsY* sequence was deposited in GenBank under accession no. DQ538398.

The phylogenetic relationships among the studied bartonellae were inferred from sequence alignments of each gene and from concatenated gene sequences by using the maximum parsimony and neighbor-joining methods within the MEGA version 2.1 software package (*3*) and the maximum-likelihood method within the PHYLIP software package (*4*). Using *rrs*, *gltA*, and *rpoB* sequences, the phylogenetic position of strain AUST/NH1 was supported by bootstrap values <70%. In contrast, by using the ITS, *ftsZ,* and concatenated sequences, strain