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The Effect of Antibiotics on *S pneumoniae* Carriage—Hit 'Em Hard, Get In and Out

ABSTRACT & COMMENTARY

Synopsis: *Short-course, high-dose antibiotic therapy was associated with lower rates of subsequent carriage of resistant pneumococci than was longer-course, lower-dose therapy.*

Source: Schrag SJ, et al. *JAMA.* 2001;286:49-56.

From the dr. robert reid cabral public hospital in santo Domingo, Dominican Republic, comes a study of 795 children (aged 6-59 months) who received amoxicillin for a respiratory infection in order to test the hypothesis that short-course, high-dose therapy reduces the risk of post-treatment resistant pneumococcal carriage. To this end, patients were randomized to receive either 90 mg/kg/d for 5 days or 40 mg/kg/d for 10 days, each with twice-daily dosing. Nasopharyngeal cultures for *Streptococcus pneumoniae* (SP) were collected at baseline, 5 days, 10 days, and 28 days. The clinical response to therapy was not evaluated.

Compliance with the briefer regimen was higher with rates of 82% compared with 74% ($P = 0.02$). At the initial visit, 73% of the kids were found to have SP while 26% had penicillin nonsusceptible *S pneumonia* (PNSP). The rate of PNSP carriage was found to correlate with recent receipt of antibiotics, the presence of 3 or more children in the household, and attendance at school or day care. By day 5 of therapy, the isolation rate of SP had dropped to 25% and that of PNSP to 20%. By day 10, the recovery rate went up to 31% for SP and 21% for PNSP.

On day 28 following the initiation of therapy, the rate of SP carriage went back up to about 52% for both regimens, but, however, the rate of isolation of PNSP rose to only 24% in the short-course group compared with 32% in the 10-day course group. The differences were significant at the $P = 0.03$ level with a relative risk of 0.77 and 95% confidence intervals of 0.60-0.97. The incidence of adverse effects was low and similar in intensity. Thus, high-dose short-course therapy appears to be associated with a lesser effect on carriage of antibiotic

Volume 20 • Number 20 • July 15, 2001 • Pages 153-160

INSIDE

resistance than is low-dose, long-course therapy.

COMMENT BY ALAN D. TICE, MD, FACP

This study shows what kind of clinical studies can be done in developing countries with good investigators and a little help from the CDC. This collaboration provided methodology and microbiology along with excellent ready access to a large number of compliant patients.

The concept of trying to shorten the course of therapy is an important one, especially with the growing problem of antimicrobial resistance. How to limit this resistance is particularly important with the pathogen studied. It makes sense that shorter courses of intense therapy would be less likely to encourage the development and spread of resistant strains, but little has been done to prove it.

With the rising concerns about antimicrobial resistance, a new look at duration of therapy is in order. It is not easy, however, to shorten courses of therapy in the United States where any failure may end up in court if the textbook recommendations are not followed.

Short-course therapy is an important consideration for a number of infections and has been proven effective in careful studies of uncomplicated cystitis, gonorrhea, and chlamydia. It may also be possible to provide parenteral therapy as an alternative to oral antibiotics and achieve results with a single dose compared to a week's worth of oral antibiotics, as has been demonstrated with ceftriaxone therapy of otitis media. Opportunities for single-dose intramuscular or intravenous therapy also appear possible with streptococcal pharyngitis.

The results of this investigation confirm the benefit of shorter, higher-dose courses of therapy in reducing PNSP carriage relative to that of lower-dose, shorter duration therapy, but also raise a number of additional questions. Of particular interest is why it is that the sensitive strep are not eliminated after 5 or even 10 days of therapy? Why does the difference in PNSP carriage between short and long course therapies not appear before 28 days of therapy? The apparent protective effect of siblings is interesting but may well relate to a reservoir of relatively susceptible strains in the home.

Should we really be telling patients to stop antibiotics when they are afebrile, they feel better, rather than to continue them for some often arbitrarily chosen duration? Most short-course therapies entail high doses of antibiotics and/or only the most potent drugs. Shortening the course of intravenous antibiotic therapy in the hospital is actively being studied. Should we not look at outpatient and oral regimens as well? At any rate, the results discussed here provide fodder to the argument: "Hit 'em hard, and then get out." ❖

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Questions & Comments

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Antibiotic Use in the European Union

ABSTRACT & COMMENTARY

Synopsis: *Data obtained during 1997 showed that sales of antibiotics for nonhospital use varied 4-fold among the 15 member states of the European Union during 1997 with France, Spain, Portugal, and Belgium being the biggest consumers and Sweden, Denmark, Germany, and The Netherlands being the lowest consumers.*

Source: Cars O, et al. *Lancet.* 2001;357:1851-1853.

In a comparison of antibiotic use in countries of the European Union (EU), the amount of antibiotics

sold in each country was converted to the defined daily dose (DDD), which is a unit based on the average daily dose used for the main indication of the drug. Most of the data on national sales were purchased from a private company that relies on various sources including manufacturers, wholesalers, pharmacies, prescribers, and hospitals and makes national estimates. The number of inhabitants in each country was obtained from EUROSTAT, the agency that collects such data for the EU. France consumed the most antibiotics, 4 times more than The Netherlands, which consumed the least. Broad-spectrum penicillins accounted for 39% of all sales followed by macrolides and lincosamides (17%), tetracyclines (14%), and cephalosporins (10%) and together made up 80% of all sales in th EU. The broad-spectrum penicillins were also the most frequently prescribed in each country except for Finland and Germany which consumed more tetracyclines, Sweden and Denmark which preferred narrow spectrum beta-lactam antibiotics (data not shown on the table), and Austria in which the macrolides were most frequently prescribed. There was also marked variation between countries in the consumption across every class of antibiotics. For instance, cephalosporins accounted for 1 in 5 antibiotics prescribed in Greece but were rarely used in The Netherlands, while quinolones were most widely used in Portugal. Cars and colleagues also remarked on how different the pattern of antibiotic uses was in 2 neighboring countries—Belgium and the Netherlands—that share a common language. The variation in outpatient antibiotic use in the EU was thought not to reflect widely differing patterns of bacterial infection but rather different historical, cultural, and social factors as well as disparities in health care systems between the 15 countries. Cars et al concluded with a challenge to member states to perform proper epidemiological studies on antibiotic prescribing and resistance.

COMMENT BY J. PETER DONNELLY, PhD

The differences noted in antibiotic use in the EU for a single year are remarkable and defy explanation but are not surprising. The EU aspires to harmonize everything from the quality of cucumbers to standardizing sausage, but on the ground each country still maintains its traditions, beliefs, culture, and habits—all of which exert a much greater influence on all aspects of daily life including, apparently, antibiotic preference. This is even tactily acknowledged by multinational pharmaceutical companies who would like to treat the EU as a single sector of the globe but still maintain offices in each country to deal with the local market. The differences between Belgium and The Netherlands are indeed noteworthy. They are even reflected in the trade names given to some drugs such as itraconazole, which is known in Belgium (and indeed everywhere else) as Sporanox. In The Netherlands, it is called Trisporal. Similarly, ceftazidime is known as Fortaz in The Netherlands, but across the border in Belgium it is known as Glazidim. However, only part of Belgium speaks Dutch (or Flemish). French is spoken in the

Table

Antibiotic Use in the European Union

			Proportion of drugs sold per country				
	DDD per 1000 inhabitants	DDD relative to The Netherlands	Broad-spectrum penicillins	Macrolides & lincosamides	Tetracyclines	Cephalosporins	Proportion of all drugs sold
France	36.51	4.1	52%	16%	9%	10%	87%
Spain	32.44	3.6	56%	18%	4%	8%	86%
Portugal	28.83	3.2	42%	13%	9%	11%	75%
Belgium	26.72	3	41%	15%	19%	11%	86%
Luxembourg	25.58	2.9	41%	18%	16%	11%	86%
Italy	23.99	2.7	47%	21%	2%	13%	83%
Greece	22.69	2.5	34%	20%	12%	21%	87%
Finland	19.34	2.2	20%	10%	28%	11%	69%
Ireland	18.34	2	44%	14%	19%	7%	84%
United Kingdom	18.04	2	38%	18%	20%	5%	81%
Austria	13.8	1.5	23%	26%	13%	9%	71%
Germany	13.58	1.5	20%	19%	24%	7%	70%
Sweden	13.51	1.5	10%	7%	22%	4%	43%
Denmark	11.35	1.3	21%	17%	9%	0%	47%
The Netherlands	8.96	1	32%	14%	26%	1%	73%

other part, which may well have as much influence on antibiotic use as it does on cuisine and couture.

Whatever the reasons for the variation in antibiotic use in the EU, it is tempting to speculate about this being the explanation for differences in resistance rates since the northern countries like The Netherlands, Sweden, and Denmark have the lowest rates of antibiotic resistance in the EU. Countries like France, Greece, Italy, and Spain have some of the highest rates. These differences might simply reflect a North-South divide or even division between the Protestant and Catholic/Orthodox countries. Certainly these data are tantalizing and should encourage more research to explore further the link between antibiotic resistance and both higher consumption and the preference for certain antibiotic classes. I am also curious to know what a similar investigation into the pattern of antibiotic use between the states of the United States. The union is older than the EU and governed by federal regulations rather than a council. If there was greater uniformity between the states than that seen in the EU, central regulation could alter antibiotic use. However, if there was similar variation, other means would have to be sought to ensure a more rational use of the precious resources of antibiotics. ❖

Special Feature

A Wake-Up Call: African Trypanosomiasis Strikes Back

By Stan Deresinski, MD, FACP

Two cases of east african trypanosomiasis occurring in travellers who had briefly visited the Serengeti Park in Tanzania were reported to the annual meeting of the American Society of Tropical Medicine and Hygiene last autumn in Houston, Tex. These reports were disturbing because, according to David Freedman, the GeoSentinel network database covering 26 global sites had had no reports of East African trypanosomiasis in the previous 3 years. These occurrences were not totally unexpected, however, since trypanosomiasis is present throughout Tanzania and, within that country, trypanosomiasis is most prevalent in a region from Kigoma at Lake Tanganyika to Arusha in the northern part of the country, areas commonly visited by tourists. Furthermore, the incidence of African trypanosomiasis has been increasing for decades. Thus, it is not surprising that new cases of trypanosomiasis in travelers continue to be reported with the recent total now reaching at least 8.[1]

Seven of the 8 were Europeans, including residents of Holland, Italy, Norway, and the United Kingdom. Most had been in or near the Serengeti and most reported tsetse bites, although the infection may have been acquired in other areas visited. The interval from onset of symptoms to diagnosis appears to have ranged from 2 to 4 days in most cases. At least 1 patient, who did not receive therapy until 10 days after the onset of symptoms, died.

These cases in western travellers are, of course, of concern and should alert physicians to consideration of this infection in febrile individuals returning from endemic areas. However, it is more a reflection of another in a long line of African tragedies—the resurgence of a once almost eradicated disease that causes uncounted economic and human devastation.

Human African trypanosomiasis (HAT) is caused by 1 of 2 subspecies of the hemoflagellate protozoan, *Trypansoma brucei*, that are endemic in areas of sub-Saharan between 14º north and 15º south latitudes.[2,3] *T b gambiense* is transmitted in central and west Africa and in general causes chronic disease with an incubation period in indigenous people of months to years. *T b rhodesiense* is acquired in southern and east Africa and causes acute infection after a relatively briefer incubation period. Uganda is currently the only country in which both subspecies are reportedly present.

HAT is transmitted by the bite of the tsetse, a word meaning “fly” in Tswana, the language of the former Bechuanaland and, since 1966, Botswana. The tsetse is 6-14 mm in length, with a proboscis projecting straight from the head (*see Figure 1*).[4] Tsetse are found in vegetation near rivers and lakes, gallery-forests, and wooded savannah. The flies that transmit *T b gambiense*, for which there is no known animal reservoir, usually reside along the banks of shaded streams in proximity to human habitation. In contrast, the flies that transmit *T b rhodesiense* dwell on lightly covered bush land and feed on game animals as well as domesticated animals. In cattle, the disease is called “nagana.” The presence of an animal reservoir makes eradication potentially more difficult for this subspecies.

The usually prolonged incubation period seen in native populations in Gambian HAT is generally much briefer in non-natives. The initial symptoms include intermittent fevers that correspond to successive surges of antibodies produced in response to the remarkable antigenic variation of surface proteins of

Figure 1

The Tsetse Fly

the parasite as it attempts to escape immune control. Lymphadenopathy is commonly present (Winterbottom's sign is occipital adenopathy seen in this infection), and a trypanosomal chancre may still be detectable in those who present after shorter incubation periods. The episodes of fever generally last several days and recur within several weeks. They may be accompanied by a multiform skin eruption and pruritus and occasionally edematous swelling, particularly of the face. After months of chronic illness accompanied by weight loss, symptoms of central nervous system disease appear, with somnolence developing in most. In Rhodesian HAT, the time course is more compressed and the patients more acutely ill. The diagnosis of HAT is made by the demonstration of trypanosomes in lymph node "juice," blood, or cerebrospinal fluid (CSF). All patients with HAT should have examination of CSF.

Treatment depends on both the infecting subspecies (identified by geographic origin of the infection) and the stage of the disease, with Phase II reflecting evidence of neurological involvement (*see Tables 1 and 2*). However, treatment is associated with significant adverse effects as well as increasing evidence of inefficacy suggesting the development of resistance in the organism. For instance, treatment with melarsoprol, an arsenical discovered in 1949 and the only available drug that has been effective in the presence of neurological involvement by either subspecies, is often accompanied by severe side effects, including a reactive encephalopathy reported to be fatal in 3-10% of cases. In addition, it has recently been reported that the relapse rate after treatment in patients with *T b gambiense* infection in northwestern Uganda after treatment with melarsoprol is > 25%.[5]

The therapeutic picture has been further muddied as the result of drug shortages resulting from economic decisions of pharmaceutical companies. Perhaps the most disgusting story, albeit one with a favorable outcome, was that of eflornithine, the only available drug for those who fail treatment with melarsoprol. The pharmaceutical company with the rights to eflornithine abandoned production of this drug in 1994 due to low profitability. In 2000, however, the company resumed production of the drug—not out of the goodness of their hearts and concern for their fellow man, but because they were now marketing it as a topical agent for removal of unwanted hair.

There is clearly a need for new therapeutic approaches. In the absence of interest from the pharmaceutical industry, WHO has identified a number of rather limited therapeutic research priorities. These include the use of nifurtimox as a single agent or in combination as well as the evaluation of other drug combinations, the evaluation of a short pentamidine protocol, and the evaluation of efficacy of 2 newer drugs, berenil and megazol.

Control of the disease involves low-altitude spraying, trapping, and case finding with treatment. The only

Table 1

WHO Case Definitions

- **Confirmed:** Trypanosomes observed in blood, lymph gland fluid, or CSF.
- **Stage I:** Trypanosomes in blood or lymph gland fluid, but not CSF and with ≤ 5 WBC.
- **Stage II:** One or more of the following: Trypanosomes in CSF and/or CSF WBC > 5.
- **Suspected relapse:** after appropriate treatment of confirmed disease, develops either/or an increase in CSF WBC of ≥ 2-fold with a total CSF WBC > 5.
- **Confirmed relapse:** After appropriate treatment of confirmed infection has trypanosomes observed in CSF, blood, or lymph gland fluid within a 2-year post-treatment follow-up.

Table 2

WHO Treatment Recommendations

Stage I:

T b gambiense—pentamidine

T b rhodesiense—suramin

Stage II:

T b gambiense—melarsoprol or eflornithine hydrochloride

T b rhodesiense—melarsoprol

means by which travellers can avoid infection is to avoid the bite of the tsetse. Areas of heavy infestation tend to be sporadically distributed and are usually well known to local inhabitants. Avoidance of such areas is the best means of protection. These flies are attracted to moving vehicles and dark, contrasting colors. They are not affected by insect repellents and can bite through lightweight clothing. Travellers at risk should wear clothing of wrist and ankle length that is made of medium-weight fabric in neutral colors that blend with the background environment.[6]

Three major epidemics of HAT have occurred over the last century. The first lasted a decade, ending in 1906, the year after the first therapeutic agent, the arsenical Atoxyl had been introduced. In the same year that the epidemic ended, Colonial Undersecretary Winston Churchill reported to the British House of Commons that "the disease had reduced the population of Uganda from 6.5 million to 2.5 million."[7]

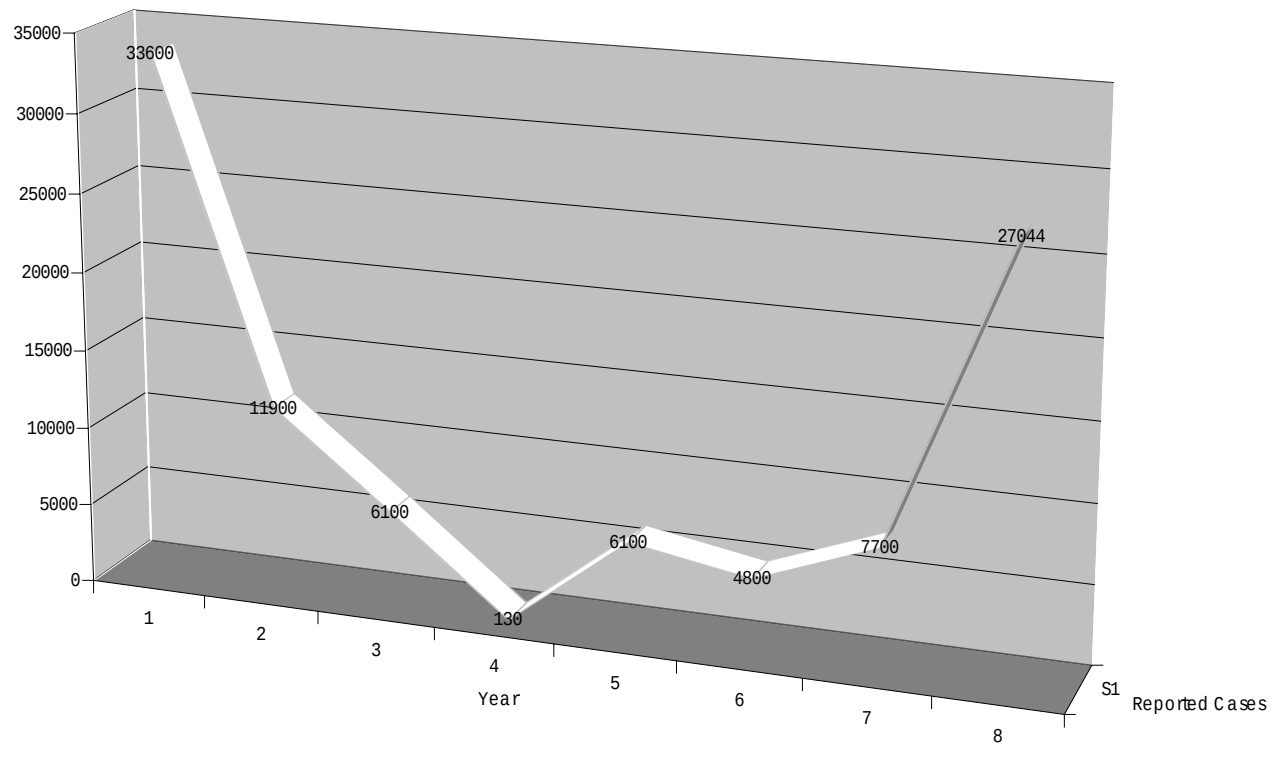
The second epidemic, in 1920, was rapidly controlled as the result of systematic screening of millions at risk by mobile teams and treatment with an arsenical. In 1930, the French publication "L'illustration" reported that "Our doctors have vanquished the tsetse fly."[8] In a common theme of political and public health failure (think, eg, of malaria, TB, etc), the disease became infrequent between 1960 and 1965, resulting in dismantling of control programs. The result: a third epidemic starting in 1970, which continues at this time and shows no signs of abatement. The failure of public health can be seen by the fact that only 3-4 million of the more than 60 million people at risk are currently under surveillance. While 45,000 cases were reported in 1999, it is believed that the true case incidence is 300,000 to 500,000. Despite inadequate surveillance, the WHO indicates that the prevalence of trypanosomiasis in some villages in Angola, the Democratic Republic of Congo, and southern Sudan is between 20% and 50%.[9] For instance, screening of 1400 persons in 16 villages of Tambura County, Western Equatoria, Sudan in 1997 found prevalences as high as 45%, with a mean prevalence of 19.3%.[2] The mean prevalence in this same area in 1988 had been only 0.3%.

Figure 2, taken from data reported to Promed, demonstrates the incidence of trypanosomiasis in the Democratic Republic of Congo (formerly Zaire), at approximately 10-year intervals, over most of the last century (*see Figure 2*).[1]

The human and animal forms of African trypanosomiasis are a major obstacle to development of rural sub-Saharan Africa as a consequence of loss of cattle

Figure 2

Annual Incidence of Trypanosomiasis in the Democratic Republic of Congo (Zaire)



and human disease leading to abandonment of potentially fertile lands. While much of the resurgence of disease can be attributed to population displacement, political instability, civil war, and the collapse of health systems, the indifference of the developed world contributes mightily. ❖

References

1. http://www.promedmail.org.
2. African trypanosomiasis or sleeping sickness. WHO Fact Sheet No. 258, March 2001;1-5. http://www.who.int/inf-fs/en/fact259.html.
3. Pepin J, Donelson JE. African trypanosomiasis (sleeping sickness). In: Guerrant RL, Walker DH, Weller PF, eds. *Tropical Infectious Diseases*. Philadelphia, Pa: Churchill Livingstone; 1999:774-784.
4. http://www.roberth.u-net.com/tsetse.htm.
5. 3rd Meeting of the WHO Steering Committee of Human African Trypanosomiasis Treatment and Drug Resistance Network, Bruges, Belgium, May 28-30, 2000.
6. http://www.mdtravelhealth.com/infectious/who_trypanyogeo_4.html.
7. Zimmer C. http://www.discover.com/august_issue/sleep.html.
8. http://www.tulane.edu/~dmsander/WWW/224/Trypano.html.
9. West African trypanosomiasis resurging in southern Sudan. CDC/NCID FOCUS. 1997; 6/ September-October 1997. http://www.cdc.gov/ncidod/focus/vol6no5/trypan.htm.

Additional Reading

1. Sinha A, et al. African trypanosomiasis in two travelers from the United States. *Clin Infect Dis.* 1999;29: 840-844.

Correction

In the May 1, 2001, issue on page 120 in the Update, "Treatment of MDR-TB Meningitis," the sentence in the second paragraph, "intrathecal treatment via an Omaya reservoir with both levofloxacin (maximal dose, 1.5 mg) and azithromycin (maximal dose 5 mg)" should have read "amikacin." We regret any confusion this may have caused. ❖

CME Questions

5. **High-dose amoxicillin is more effective in eliminating *S pneumoniae* within 5 days than lower dose therapy for 10 days.**
   a. True
   b. False

6. **What percent of children treated for a respiratory infection in this study were at least colonized with *S pneumoniae* before antibiotics were begun?**
   a. Less than 25%
   b. Between 25 and 50%
   c. Between 50 and 75%
   d. More than 75%

7. **Which of the following is correct?**
   a. Use of common insect repellents containing DEET are highly effective in preventing tsetse bites.
   b. Human African trypanosomiasis is only acquired in parts of South Africa.
   c. Trypanosomes are hemoflagellate protozoans.
   d. The major clinical consequence of human African trypanosomiasis is the development of cardiomyopathy.

8. **Which of the following is correct?**
   a. Winterbottom's sign is bilateral ptosis seen in human African trypanosomiasis.
   b. The diagnosis of human African trypanosomiasis is made by the demonstraiton of trypanosomes in lymph node fluid, blood, or cerebrospinal fluid.
   c. Human African trypanosomiasis is treated with dapsone plus trimethoprim.
   d. Human African trypanosomiasis is inevitably manifested by "sleeping sickness."

9. **Short-course amoxicillin therapy during therapy reduced the presence of penicillin nonsusceptible *S pneumoniae* (PNSP).**
   a. True
   b. False

Readers are Invited

Readers are invited to submit questions or comments on material seen in or relevant to *Infectious Disease Alert*. Send your questions to: Neill Larmore—Reader Questions, *Infectious Disease Alert,* c/o American Health Consultants, P.O. Box 740059, Atlanta, GA 30374. For subscription information, you can reach the editors and customer service personnel for *Infectious Disease Alert* via the Internet by sending e-mail to neill.larmore@ ahcpub.com. We look forward to hearing from you. ❖

In Future Issues:

Are Patients Dissatisfied if They Do Not Receive an Antibiotic for a Respiratory Infection?

UPDATES

By Carol A. Kemper, MD, FACP

Children Mistakenly Receive Cattle Vaccine

Sources: *The Irish Times*, Ireland, July 1, 2001 & *The New York Times*, June 30, 2001.

Irish authorities are attempting to sort out how children participating in a clinical drug trial in 1973 received a vaccine intended for animals. Although it was not immediately clear whether vaccine was administered inadvertently or as part of the study, the children apparently received Tribovax T in lieu of the similar-sounding Trivax vaccine for children, which contains vaccine against diptheria, tetanus, and pertussis. Tribovax T (Schering Plough) is a "4-in-1" clostridial vaccine used in sheep and cattle to prevent Blacleg, Braxy, Black Disease, and tetanus (http://www.farm-rite.co.uk/product).

Adding further confusion were reports suggesting that all reactions to vaccinations were reported to the manufacturer of Trivax, the Wellcome Foundation. If correct, it is possible that someone may have known about the mix-up. Thus far, no long-term effects have been reported, but health authorities promised to conduct a complete investigation. ■

Polio Revisits Eastern Europe

Source: *Eurosurveillance Weekly*, May 24, 2001.

Two cases of poliomyelitis have been reported in a 13-month-old child and a 2-year-old child in the Romany area of Bulgaria this spring—the first cases of poliovirus infection in Europe since 1998 when polio was reported in Turkey near the Iranian border. The first case occurred in Burgas on the Black Sea in March and, by April, authorities acted to step up vaccination of children in the area. Nevertheless, a second case occurred about 90 km west of Burgas in May, suggesting that polio had been circulating in the larger community. Because ~5% of Bulgarian children have never received poliovaccine, a national vaccination program had been planned for late May, but it was unfortunately postponed for lack of available vaccine.

Various laboratories have identified the causative strain as a wildtype poliovirus-1 and not a vaccine-derived strain, as recently occurred in the Dominican Republic and Haiti (Kemper CA. *Infectious Disease Alert.* 2001;20: 83-85). Most poliovirus-1 virus is presently believed to originate from the Indian subcontinent. ■

Couple Torches Home to Avoid Mildew

Source: Tresniowski A, et al. *People* Magazine, July 9, 2001:109-110; and www.latimes.com/health/news, May 25, 2001.

As an id expert, have you ever been asked to provide advice on environmental mildew and fungus? A California couple is spending more than $3,000,000 to create a "germ-free" home in Ventura County, California. The 11,000 square-foot home will be built almost entirely from steel, and coated in a ceramic powder impregnated with silver ions, which suppresses the growth of mold, fungus, and bacteria—much the same as silver impregnated catheters and other medical devices. Even the fixtures, fabrics, and appliances will be coated with the antimicrobial powder. The substance will also be used on almost everything else in the home, from cookware, to the carpets and mattress pads, to the racks in the 6000-bottle wine cellar. The project is a collaboration between the attorney-owner, architect David Martin, Ohio-based AK Steel, and AgION Technologies, as well as about 60 other companies whose products were being modified for the project.

On a similar note, fearing that the mildew in their home was either too costly or impossible to eradicate, another couple took the extraordinary step of having their home in Foresthill, Calif. burnt to the ground. The couple believed that black mold due to *Stachybotrys chartarum* in their home was causing numerous health problems in family members, such as chronic respiratory ailments and developmental delays in their infant son. Rather than spend the purported $85,000 to get rid of the mold in their home, they hired 40 volunteer firefighters to set it ablaze and sold the property.

Interestingly, some architects believe that the more modern building codes that require buildings be extra-insulated and even wrapped in plastic, ostensibly to keep moisture out, do exactly the opposite by not letting wood structures breath. ■

Plague Fells Chipmunk at Lake Tahoe

Source: ProMED-mail post, July 20, 2001; www.promedmail.org.

In quick follow-up to the most recent *Infectious Disease Alert* (quiz: Which animal may be infected with *Yersinia pestis* in New Mexico and Nevada?), a dead chipmunk found at the Lake Tahoe Forest Service Visitors Center in South Lake Tahoe has tested positive for bubonic plague. Health officials are posting warning to visitors to the area not to handle live or dead wild animals, not to rest, sleep, or camp near a rodent burrow (how do you know?), and make sure that any dogs or cats you bring into the area wear flea collars and don't mess with rodents. The last recorded death due to pneumonic plague in the area occurred in 1980 when a cat brought home a dead chipmunk and transmitted the infection to its owner. ■