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Committee to Advise on Tropical Medicine and Travel (CATMAT)*

STATEMENT ON POLIOMYELITIS VACCINATION FOR INTERNATIONAL TRAVELLERS

(Evidence-based medicine recommendations)[1]

The clinical manifestations of poliomyelitis may range from an inapparent infection to severe neurologic involvement with paralysis and death. The ratio of inapparent infection to clinical disease due to polio is estimated to be 60:1 to 1000:1[2,3,4]. Polio infection is caused by any of the following three antigenic types of enteroviruses: polioviruses 1, 2 and 3. Fecal-oral is the major route of transmission where sanitation is poor.

In Canada, the last major epidemic occurred in 1959 when 1,887 paralytic cases occurred. Following the introduction of the inactivated polio vaccine (IPV) in 1955, and the oral polio vaccine (OPV) in 1962, the indigenous disease has been virtually eliminated. Rarely, outbreaks have been reported in populations that have declined immunization on religious grounds[5,6]. From 1980 to 1992, 10 cases of polio were reported in Canada[7]. Nine of these were felt to be vaccine-related[5].

Since wild polio viruses no longer circulate in Canada, routine immunization or booster doses of polio vaccine for adults are no longer recommended in Canada, except in special circumstances[5]. One of these circumstances may be international travel to areas with endemic or epidemic-polio virus activity.

In a recent serologic survey of 233 American international travellers, 12% were found to be lacking antibodies to polio viruses 1 or 3[8]. All had antibodies to polio virus 2. All travellers in this survey who had received polio vaccination within the previous 5 years had antibodies to all three types. Only 84% of those who had received a polio booster > 5 years earlier were seropositive to all three types. Although these data may not be generalizable to Canadians, they do suggest that many travellers may not have protective antibodies against the polioviruses.

Because of the small number of polio cases reported in Canada, none of which appear to be travel-related, it will be easier to say who should not be immunized against polio for international travel. This approach will become easier as we move toward the goal of global eradication of polio.

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A World Health Organization report on the status of the Expanded Programme on Immunization (EPI) summarizes the incidence of reported cases of polio by world region as of 1992[6]. In the Americas, the last case of indigenous polio caused by a wild polio virus was detected in Peru on 23 August, 1991[7]. Despite intensive surveillance for cases of flaccid paralysis in the region, no new cases have been detected. Reporting is 100% complete in the region. Forty-two of the 47 countries or regions (89%) have reported no cases of polio for at least 3 years[6]. When no new cases are reported from a region for 3 years, the region is considered to be free of polio transmission. In October 1994, the World Health Organization reported that poliomyelitis had been eradicated from the Americas[9].

Unfortunately, for parts of Africa, the Middle East, and Asia the rates of poliomyelitis continue to range from 1 to 10 cases per 100,000 population (Figure 1). India contributes three of every four reported cases of polio[6].

These data do not necessarily translate into a risk of infection or disease for the international traveller. In endemic regions, or in areas experiencing epidemics of polio, a careful pre-travel history of activities that may pose an increased risk of exposure to contaminated water may indicate a need for primary vaccination or for a booster dose for adults. These risk characteristics in a geographic area of polio transmission may include duration of travel, expected exposure to unsafe water, and activities where increased exposure to unsanitary conditions may occur such as back packing, providing international aid or health care, or participating in a cultural exchange program.

RECOMMENDATION 1

***Primary immunization of children and, if indicated, primary immunization of adults or a booster dose for adults is recommended in accordance with the recommendations of the National Advisory Committee on Immunization*[5]** [strength of recommendation: category A; quality of evidence: grade III[1]] (see also Appendix I).

Recommended Usage

Infants and Children: immunization against poliomyelitis may be undertaken with IPV or OPV. The recommended schedule is to immunize at 2, 4, 6 (omit this dose if OPV is used exclusively) and again at 18 months of age, with a booster at 4 to 6 years and 14 to 16 years (omit this dose if OPV is used exclusively). **Following primary immunization, the only indication for revaccination is a one-time booster as an adult, at least 10 years after primary immunization, for travellers to foreign countries where poliomyelitis is endemic**.

Figure 1
Global incidence of poliomyelitis, 1995

For children < 7 years, but not immunized in infancy: immunization against poliomyelitis may be undertaken with IPV or OPV. The recommended schedule is as follows: first visit, 2 months later, 2 months following the second dose (omit this dose if OPV is used exclusively), 6 to 12 months later, at 4 to 6 years (omit this dose if the previous doses given after the fourth birthday) and 14 to 16 years (omit this dose if OPV is used exclusively) and, if required, every 10 years thereafter.

Routine immunization schedule for children > 7 years, or for adults, not immunized in infancy: immunization against poliomyelitis may be undertaken with IPV or OPV. **Caution is advised in using OPV in a previously unimmunized adult, or in an individual who may expose an unimmunized adult to the vaccine strain, due to the risk of vaccine-associated disease**. The vaccine is given at the first visit, 2 months later, 6 to 12 months later and, if required, every 10 years thereafter.

RECOMMENDATION 2

Booster immunization against polio is not required for travel to the Americas[6] (strength of recommendation: category E; quality of evidence: grade II).

RECOMMENDATION 3

International travellers to other polio-endemic or epidemic regions of the world should be considered for a primary series or booster vaccination, depending upon an evaluation of their risk of exposure to the virus[6,8] (strength of recommendation: category A; quality of evidence: grade II).

References

1. MacPherson DW. *Evidence-based medicine.* CCDR 1994;20: 145-47.
2. Centers for Disease Control. *Neurotropic diseases surveillance: poliomyelitis summary, 1974-1976.* CDC, Atlanta, 1977.
3. Nathanson N, Martin JR. *The epidemiology of poliomyelitis. Enigmas surrounding its appearance, periodicity, and disappearance.* Am J Epidemiol 1979;110:672-92.
4. Melnick JL, Ledinko N. *Social serology: antibody levels in a normal young population during an epidemic of poliomyelitis*. Am J Hyg 1951;54:354-82.
5. National Advisory Committee on Immunization. *Canadian immunization guide.* 4th ed. Ottawa, Ont: Health Canada, 1993 (Supply and Services Canada, Cat. No. H49-8/1993E).
6. *Expanded programme on immunization: poliomyelitis in 1992.* Wkly Epidemiol Rec 1993;68:225-30.
7. *Expanded programme on immunization: update: eradication of paralytic poliomyelitis in the Americas.* Wkly Epidemiol Rec 1992;67:325-26.
8. Hilton E, Singer C, Kozarsky P et al. *Status of immunity to tetanus, measles, mumps, rubella and polio among U.S. travelers.* Ann Intern Med 1991;115:32-3.
9. *Expanded programme on immunization: certification of poliomyelitis eradication - the Americas, 1994.* Wkly Epidemiol Rec 1994;69:293-95.

Committee to Advise on Tropical Medicine and Travel (CATMAT)

TRAVEL STATEMENT ON JET LAG

Definition

The term "jet lag" describes the constellation of physical and psychologic symptoms associated with rapid crossing of multiple time zones (meridians). These symptoms are due primarily to disturbance of the physiologic circadian rhythm and sleep cycle.

Control of the circadian rhythm in mammals and the "endogenous clock" appears to be centred in the suprachiasmatic nuclei of the hypothalamus[1]. Circadian rhythms affect a wide range of biologic measures and functions,such as body temperature, blood pressure and hormone secretion,and have even been demonstrated at the cellular level. The endogenous circadian rhythm is usually not exactly 24 hours so that, under normal, stable conditions, an individual's "endogenous clock" is being readjusted by exogenous, environmental cues (zeitgebers) on a daily basis. Social cues as well as exposure to light, particularly bright light, appear to be important both in the regulation of a stable circadian rhythm and in adjustment to a rapid change in time zone. Melatonin contributes to the physiologic regulation of circadian rhythm[2].

Symptoms of Jet Lag

Symptoms of jet lag are common with time zone changes ≥ 5 hours. While nearly all travellers will experience some symptoms with large time zone shifts, there is considerable individual variation in severity and time to recovery. Problems may increase with age[3]. The time to re-establishment of circadian equilibrium is generally greater with eastward than westward flights.

The symptoms of jet lag are quite familiar to most trans-meridian travellers. Sleep deprivation, difficulty sleeping, and a delay in cycling the sleep pattern to match that of the destination occur, resulting in fatigue and associated symptoms. Mood disturbance, anorexia, and gastrointestinal symptoms are common. Subjective effects are usually prominent for only a few days, but objective measurements of sleep patterns, body temperature, and hormone levels show that a complete physiologic phase shift may take up to 14 days. Jet lag can adversely affect performance, including athletic performance and manual and cognitive skills[4].

Possible Measures for the Prevention and Management of Jet Lag

I. Adequate intake of fluids and limited alcohol intake on the flight may be expected to reduce the possibility of dehydration and may improve well-being for reasons not directly related to jet lag.

Specific types of food, i.e., carbohydrate versus protein, have been suggested but not demonstrated to have an impact on symptoms of jet lag. Timing of meals may contribute to re-adjustment to a new zone.

Benzodiazepines facilitate adjustment of the circadian rhythm in hamsters, but this effect has not been demonstrated in humans[5]. The only known benefit of their use is symptomatic in facilitating sleep according to a new schedule, for the first day or few days after arrival. Caffeine similarly has been used to delay sleep to accommodate the schedule of a new time zone, but any possible benefit for travellers is likely to be modest.

RECOMMENDATION 1

Travellers crossing multiple time zones by air should be advised of the likely occurrence and implications of jet lag. Travellers should consider that they may wish to plan important physical or intellectual activities, such as competitive sports or critical negotiations, for 48 hours or more after arrival in a new time zone.

Travellers should be encouraged to be well-rested and not sleep-deprived at the commencement of a trans-meridian journey.

The traveller who intends to stay in a new time zone for other than a very brief period of time should attempt immediately on arrival to adjust his or her cycle of sleeping, eating, and activity to that of the destination. Where practicable, this adjustment may be initiated during, or even prior to, the journey. Outdoor light exposure at the destination may be particularly important (see Recommendation 3)***.***

Short-acting benzodiazepines may be used to facilitate sleep for the first night or few nights in a new time zone. Travellers should be aware of their potential effect on cognitive and manual skills [strength of recommendation: category B; quality of evidence: grade III[6] (see also Appendix I)].

II. Melatonin is secreted by the pineal gland and melatonin receptors are present in the suprachiasmatic nucleus. Melatonin secretion is inhibited by light exposure. Melatonin is involved in regulation of cyclic activity in other species and exogenously administered melatonin affects sleep and EEG activity in humans. Exogenous melatonin influences entrainment of circadian rhythm in animal models.

Three small randomized controlled studies in human air travellers, each using somewhat different regimens of melatonin administration, found small and inconsistent reductions in jet lag symptoms[7-9]. One human study in a controlled laboratory environment showed small but significant differences in adjustment of temperature and other physiologic cycles[10].

The safety of melatonin has not been established. It is an investigational drug in Canada and the United States, although it is readily available in health food stores.

RECOMMENDATION 2

Melatonin is not presently recommended for the prevention or management of jet lag (strength of recommendation: category C; quality of evidence: grade I).

III. Bright light, but probably not usual indoor illumination, appears to be one important exogenous cue for resetting of the circadian rhythm[11]. Bright light for 7½ hours/day for four nights markedly improved adaptation to a simulated night shift routine[12]. A single 4-hour exposure to bright light enhanced phase shift to a moderate degree and enhanced alertness at work[13]. Adequate studies using light manipulation in travellers have not been published.

High intensity light, delivered by a desktop light panel or through a visor device, has been used for treatment of seasonal affective disorder[14-16]. Concern has been expressed about the long-term safety of these modes of high intensity light exposure[17]. At least one company markets a kit containing a special visor which delivers high intensity light, dark glasses to control external light exposure, and advice for specific travel itineraries.

RECOMMENDATION 3

Travellers can be advised that exposure to bright light, particularly outdoor light in daylight, at the destination may speed readjustment of the circadian rhythm and could reduce symptoms of jet lag (strength of recommendation: category B; quality of evidence: grade II).

There is insufficient evidence regarding both safety and efficacy of artificial means of light administration as a method of jet lag prevention or treatment in travellers (strength of recommendation: category C; quality of evidence: grade III).

References

1. Minors DS, Waterhouse JM. *Circadian rhythms in general.* Occup Med (State of the Art Reviews in Occup Med) 1990; 5:165-82.
2. Shanahan TL, Czeilser CA. *Light exposure induces equivalent phase shifts of the endogenous circadian rhythms of circulating plasma melatonin and core body temperature in men*. J Clin Endocrinol Metab 1991;73:227-35.
3. Monk TH, Buysse DJ, Reynolds CF III et al. *Inducing jet lag in older people: adjusting to a 6-hour phase advance in routine.* Exp Gerontol 1993;28:119-33.
4. Comperatore CA, Krueger GP. *Circadian rhythm desynchronosis, jet lag, shift lag, and coping strategies.* Occup Med (State of the Art Reviews in Occup Med) 1990;5:323-41.
5. Turek FW, Van Reeth O. *Use of benzodiazepines to manipulate the circadian clock regulating behavioural and endocrine rhythm.* Horm Res 1989;31:59-65.
6. MacPherson DW. Evidence-based medicine. CCDR 1994;20: 145-47.
7. Petrie K, Conaglen JV, Thompson L et al. *Effect of melatonin on jet lag after long haul flights.* Br Med J 1989;298:705-07.
8. Claustrat B, Brun J, David M et al. *Melatonin and jet lag: confirmatory results using a simplified protocol.* Biol Psychiatry 1992;32:705-11.
9. Petrie K, Dawson AG, Thompson L et al. *A double-blind trial of melatonin as a treatment for jet lag in international cabin crew*. Biol Psychiatry 1993;33:526-30.
10. Samel A, Wegmann HM, Vejvoda M et al. *Influence of melatonin treatment on human circadian rhythmicity before and after a simulated 9-hour time shift*. J Biol Rhythms 1991;6:235-48.
11. Czeisler CA, Kronauer RE, Allan JS et al. *Bright light induction of strong (type 0) resetting of the human circadian pacemaker*. Science 1989;244:1328-33.
12. Czeisler CA, Johnson MP, Duffy JF et al. *Exposure to bright light and darkness to treat physiologic maladaptation to night work.* N Engl J Med 1990;322:1253-59.

13. Dawson D, Campbell SS. *Timed exposure to bright light improves sleep and alertness during simulated night shifts*. Sleep 1991;14:511-16.
14. Rosenthal NE. *Diagnosis and treatment of seasonal affective disorder*. JAMA 1993;270:2717-20.
15. Joffe TJ, Moul DE, Lam RN et al. *Light visor treatment for seasonal affective disorder: a multicenter trial*. Psychiatry Res 1993;46: 29-39.
16. Rosenthal NE, Moul DE, Hellekson CJ et al. *A multicenter study of the light visor for seasonal affective disorder: no difference in efficacy found between two different intensities.* Neuropsychopharmacology 1993;8:151-60.
17. Terman M, Remé CE, Rafferty B et al. *Bright light therapy for winter depression: potential ocular effects and theoretical implications*. Photochem Photobiol 1990;51:781-92.

Appendix 1

Categories for strength of each recommendation	
CATEGORY	**DEFINITION**
A	Good evidence to support a recommendation for use.
B	Moderate evidence to support a recommendation for use.
C	Poor evidence to support a recommendation for or against use.
D	Moderate evidence to support a recommendation against use.
E	Good evidence to support a recommendation against use.

Categories for quality of evidence on which recommendations are made	
GRADE	**DEFINITION**
I	Evidence from at least one properly randomized, controlled trial.
II	Evidence from at least one well-designed clinical trial without randomization, from cohort or case-controlled analytic studies, preferably from more than one centre, from multiple time series, or from dramatic results in uncontrolled experiments.
III	Evidence from opinions of respected authorities on the basis of clinical experience, descriptive studies, or reports of expert committees.

International Notes

DENGUE AND DENGUE HEMORRHAGIC FEVER — VENEZUELA

From the beginning of the year to 15 July, 1995, 11,808 cases of dengue fever (DF) were reported, including 2,433 cases of dengue hemorrhagic fever (DHF) of which 13 died. The States of Amazonas, Aragua, Barinas, Lara, Mérida, Miranda, Táchira and Trujillo have reported morbidity rates above the country average. The number of cases of DF increased by 28% and DHF by 93% in the week ending 16 July compared with the preceding week.

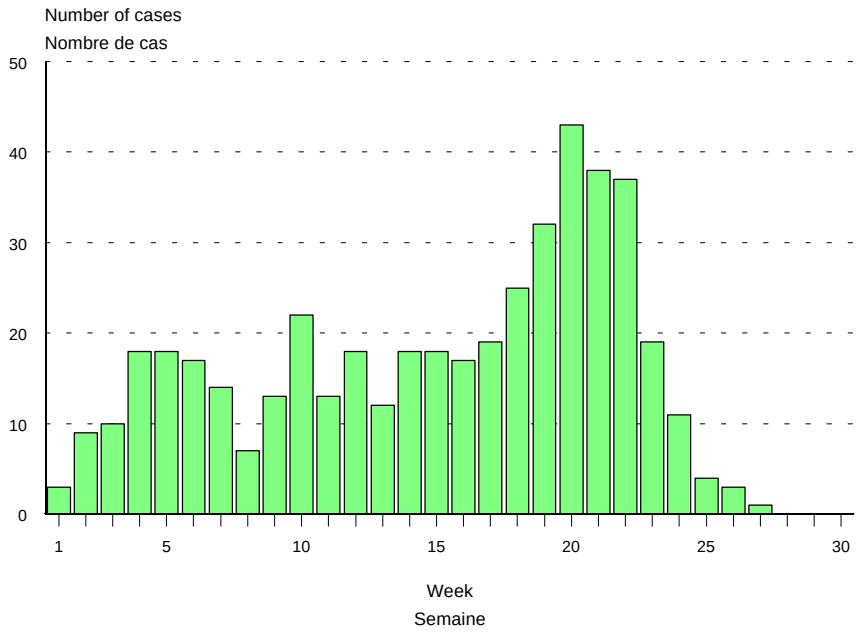
Dengue type 4 has been the predominant serotype. Control measures include public information and insecticide spraying and treatment of vector breeding sites.

Source: *WHO Weekly Epidemiological Record, Vol 70, No 32, 1995.*

YELLOW FEVER — PERU

An outbreak of yellow fever cased 440 cases from the first week of January 1995 up to the first week of July 1995, corresponding to an overall incidence rate of 1.87 per 100,000 population. Of the 440 cases, 169 (38.4%) died. The weekly number of cases was around 10 to 20 in the first 4 months of the year, then increased to a peak in the week ending 21 May and rapidly declined (Figure 1). Only 1 case was reported in the week ending 9 July and the 9 cases reported in the preceding 2 weeks were scattered over four districts in Huánuco Department, three districts in Junín and one district in San Martín Departments. No cases of urban transmission have been documented.

Figure 1
Yellow fever cases, Peru, January-July 1995



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