identification and monitoring of cardiovascular abnormalities in this group of patients require sensitive and efficient techniques. In another P^2C^2 HIV report, there was unacceptable variability of many M-mode cardiac measurements, including fractional shortening, between the local and central institutions. The 95% predictive interval for fractional shortening was "–10% to 8% indicating that a fractional shortening of 32% measured centrally could be anywhere between 22% and 40% when measured locally".[5] A less variable method of measuring cardiac function should be identified and used in future studies that attempt to evaluate early treatment of HIV-associated cardiac depression with novel therapeutic approaches.

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Scientific recommendations and human behaviour: sitting out in the sun

Changing human behaviour is not easy, as all those involved in health prevention know. A major risk factor for skin cancer is sun exposure during childhood; preventive strategies include avoiding or minimising exposure to the sun, wearing protective clothes, and use of sunscreen. During their early years, children's behaviour, at least for exposure to the sun, is largely determined by their parents or carers. Gianluca Severi and colleagues[1] recently studied European children aged 1–6 years old and their sun protection practices during summer holidays. The investigators found that as the children aged, sunburn incidence increased from 1% to 23%. However, use of protective clothing decreased from 46% in the first year to 19% in the sixth year. Sunscreen use remained constant and was the most commonly used sun-protection method. Other recent US studies[2,3] in different settings with different age ranges found similar results. So why is sun protection practised at less-than-ideal rates? Theories of behaviour change may offer some insight.

Theories about behavioural science share several fundamental principles.[4–6] First is that human behaviour is often irrational when judged by logic or scientific fact. Second, behaviour is influenced by the likely outcomes of a behaviour combined with the magnitude of value attached to each outcome. For example, wearing a hat (behaviour) keeps the sun out of the eyes (a very likely outcome), which is good (high positive value). Third, these values are from the perspective of the person and based subjectively on the individual's beliefs and experiences. Fourth, a single behaviour typically has many perceived outcomes, both positive and negative—eg, wearing a hat is fashionable, but causes sweating and messy hair. Measuring the likelihood of positive and negative outcomes and their associated values will help predict the direction of the behaviour. In general, tangible immediate outcomes are more salient, and tend to have a greater influence on behaviour than theoretical long-term outcomes, such as skin cancer.

The fifth principle is of particular relevance to sun protection. The more complex the behaviour, the more difficult it is to change. The complexity can be determined by looking at three different dimensions of a behaviour: its target, time, and context. Target refers to the behaviour of interest. For sun protection, the behaviour is actually many different sets of behaviours (avoiding exposure to the sun during peak hours, wearing protective clothing, seeking shade, and using sunscreen). Each of these behaviours can be and should be specified further (eg, wearing a wide-brimmed hat *vs* a cap). Time refers to the timing (applying sunscreen before *vs* after going outdoors), as well as frequency (rarely *vs* always) and duration of the behaviour (wearing a hat sometimes *vs* the entire time outdoors). Context refers to the setting and circumstances in which the behaviour occurs. An uncomfortably hot day may prompt avoidance of peak sun exposure, whereas the cool or cloudy day may result in no sun protection. In addition, the same context may result in different influences on behaviour. While one individual sees the summer holiday as a time to obtain a desired tan, another may be extra careful about sun protection.

The sixth and final principle involves the degree of control a person has over the behaviour. This includes both the internal skill and control of the individual, as well as external environmental factors that can affect individual control. For the adult, internal control and skills needed for sun protection are not generally an issue. Avoiding exposure, wearing protective clothing, or applying lotions are common skills and can be exercised if desired. However, internal control and skill become more complicated when the complexity of the task increases (eg, diabetes management) or there is interpersonal interaction involved. Although a parent has the skills and control needed to protect an infant from the sun, control lessens as the child ages, which may help explain why sun protection of children decreases with age. External control—the degree to which the social and physical environment facilitates or impedes the desired behaviour—can also be a major factor for sun protection. Examples include: tanned skin as the social norm, outdoor activities scheduled during peak hours, or shade being unavailable.

Applying these theoretical principles to sun protection reveals the challenge, especially when compared with single, less frequent, and less complex behaviours, such as having routine blood tests, immunisations, or screening. So what needs to be done? Behavioural science theories suggest that support and education for sun protection are necessary from all aspects of society: families, health-care systems, schools, worksites, community organisations, and the mass media.[7] Such support and education would ideally be combined with supportive environmental norms and policies that facilitate sun protection, rather than impede it. Lastly, theory suggests that behavioural change takes time and persistence.

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Bacterial infection as a cause of multiple sclerosis

Multiple sclerosis is an inflammatory demyelinating disease in which the immune system of genetically susceptible individuals is inexplicably activated to attack the central nervous system. Epidemiological studies strongly suggest that environmental factors are involved on a background of genetic susceptibility.[1] The possible involvement of infectious pathogens, most often viruses, has been much studied.[2,3]

Multiple sclerosis has a unique geographic distribution—temperate zones have a low prevalence and more northerly areas have a prevalence more than ten times that in warmer climates.[4] Sanitation, climate, ultraviolet radiation, hours of sunshine, socioeconomic status, and other environmental factors have been examined with little success.[1] Much early research used case-control designs with potential recall bias.[5] More recently, seroepidemiological research has suggested the involvement of infectious pathogens in multiple sclerosis: specific antibody responses in cerebrospinal fluid and blood, isolation of the pathogen from tissue of patients with multiple sclerosis, or in-situ or ex-vivo pathogen detection. The results have rarely been harmonious. Laboratory markers cannot be easily studied at the population level because infection by some agents (eg, with human herpesvirus 6 or *Chlamydia pneumoniae*) does not result in identifiable clinical disease, or infection occurs in childhood and is not reliably reported by study subjects.

The convergence of epidemiology and seroepidemiology of research, however, is seen with Epstein-Barr virus.[6,7] Data from the Nurses' Health study,[8] for example, show a moderately increased risk of multiple sclerosis in nurses with a history of infectious mononucleosis (odds ratio 2·1, 95% CI 1·5–2·9). Taking only those nurses whose report of infectious mononucleosis was confirmed by a positive heterophil-antibody-test, the risk remained (2·3, 1·6–3·5). Although there was no association found between multiple sclerosis and reports of other common viral diseases before disease onset, there was an association with mumps after 15 years of age and with late age at measles infection. Whether Epstein-Barr virus is a necessary cause requiring additional triggers to produce disease or merely a marker for a true cause is unresolved.[9]

Infection with *Borrelia burgdorferi*, the spirochaete responsible for Lyme disease, can involve the central nervous system and the later stages of the disease may mimic the clinical symptoms of multiple sclerosis.[10] Seroepidemiological studies of *B burgdorferi* and multiple sclerosis have produced conflicting results. Chmielewska-Badora and colleagues[11] reported that ten of 26 (38%) patients with multiple sclerosis were seropositive for *B burgdorferi* compared with 149 of 743 (20%) patients with other neurological disorders (p=0·042). Yet others reported negative findings.[12,13] More recently, Ø Brorson and colleagues[14] studied the presence of the infectious agent, or at least its cystic structure, in the cerebrospinal fluid of ten patients with multiple sclerosis, in five controls who had lower back pain, and in one patient infected with *B burgdorferi*. Cystic structures were found in eight of the ten with multiple sclerosis with use of immuofluorescence before culture and in all the multiple sclerosis patients by transmission electron microscopy and acridine-orange staining. No cystic structures were found in the controls with any method. The investigators also reported a positive reaction to antispirochaetal antiserum, a similarity between the cystic structures with known cystic forms of spirochaetes, and the similarity between the cysts found in the multiple sclerosis patients and the patient with *B burgdorferi* infection. These results led the team to suggest that the multiple sclerosis patients were infected with a spirochaete, most likely *B burgdorferi*. Whether this infection really was *B burgdorferi* and whether it occurred before or after the onset of multiple sclerosis cannot be determined from this study and indeed, given current methodology, it is difficult to imagine how this could be determined.

Whether infection with *B burgdorferi* is a cause of multiple sclerosis or whether it is merely a result of heightened susceptibility of multiple sclerosis patients to infection due to damage to the blood-brain barrier remains one of the enigmas of multiple sclerosis research. Indeed, this caveat applies to all infectious pathogens that have been associated with multiple sclerosis. Current thinking on how infections could trigger the autoimmune/immunopathological manifestations of multiple sclerosis target the following mechanisms: molecular mimicry between the pathogen and myelin antigens, determinant spreading after injury to the central nervous system by the pathogen, and bystander inflammation caused by central nervous system infection.[3] It needs to be explained how a ubiquitous infection, such as that with Epstein-Barr virus, could be involved in the pathogenesis of multiple sclerosis. Moreover, several pathogens could be associated with multiple sclerosis and their presence in the central nervous system may not be a necessary requirement for disease initiation or perpetuation.

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