Sleep Deprivation among Medical Residents: Different Perspectives

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The importance of adequate sleep is well established. The recommended sleep duration is 5–9 hours; among the elderly less than 5 hours or more than 9 hours sleep is associated with high mortality [1]. Sleep deprivation contributes to a number of molecular, immune and neural changes that play a role in disease development independent of primary sleep disorders. These changes in biological processes in response to chronic sleep deficiency may serve as etiological factors for the development and exacerbation of cardiovascular and metabolic diseases and, ultimately, a shortened lifespan [2].

Even a short reduction in sleep time may lead to deteriorated functioning. Sleeplessness accounts for impaired perception, difficulty concentrating, vision disturbances, slower reactions, as well as the occurrence of micro-episodes of sleep during wakefulness that lead to lower capabilities and efficiency in task performance. The impairment of performance caused by 20–25 hours of sleeplessness is comparable to that following ethanol intoxication at the level of 0.10% blood alcohol concentration [3].

The possibility of increased breast cancer morbidity among shift workers has been under continuous debate in recent years [4]. Meta-analysis of the studies reveals weak evidence of this occurrence among long-term night-shift workers, suggesting that only flight attendants with international or overnight work and nurses working night shifts long-term (more than 8 years) were at increased risk of breast cancer [5]. Exposure to light during the night has been suggested as the major factor leading to the excessive breast cancer morbidity [6], based on the recognition in recent years of the role of melatonin as an inhibiting factor in the growth of some human tumors, especially those that are hormone dependent [7].

In the current issue of *IMAJ*, Pikovsky and co-workers [8] present their findings of a study comparing medical students to medical residents with regard to sleep. They noted an excessive sleepiness and weight gain among residents, but no difference in inflammatory markers. The comparison of these two groups is inadequate since one group was fully employed while the other group, the medical students, was not. The preferred group for comparison with the young residents should be of similar age and fully employed. The study was based on self-administered questionnaires on sleepiness and self-reported professional performance with no support of objective tests.

Inadequate sleep and long work hours are longstanding traditions in the medical profession and work schedules are especially intense for resident physicians. The debate on duration of night shifts and the number of working hours per week among residents has intensified in recent years, but there are no conclusive results since a reduction in work hours may lead to less clinical exposure, erosion of professionalism, and inadequate preparation for independent practice [9].

Another issue of concern is the level of performance in a sleep-deprived state. Yet, a study comparing sleep-deprived surgeons to non-sleep-deprived surgeons revealed similar surgical outcomes in the patients of the two groups, without any increased risk in the patients of the sleep-deprived group [10].

In recent years some countries established a restricted work-hour schedule for residents, with limitation of on-call duties (night or day) to 14 hours and a decrease in the number of working hours per week, ranging from 48 to 63 among the various countries. The effect of this change is controversial. A recent study in Canada examined the quality of life among residents following implementation of the new restriction. The majority of residents reported a poorer quality of life and inability on their part to provide continuous and safe patient care [11]. In contrast, a study among pediatric residents in the United States found that self-reported motor vehicle accidents and/or near-misses of accidents significantly decreased and resident satisfaction increased with the new schedule [12].

Professional performance, morbidity and life quality of residents and senior physicians should be examined and evaluated with the aim of finding the golden mean: balancing the need for treatment continuity of patients, clinical education, and the physician's health and quality of life.

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Capsule

Antigen specific B cell receptor sensitizes B cells to infection by influenza virus

Influenza A virus-specific B lymphocytes and the antibodies they produce protect against infection. However, the outcome of interactions between an influenza hemagglutinin-specific B cell via its receptor (BCR) and virus is unclear. Through somatic cell nuclear transfer Dougan and colleagues generated mice that harbor B cells with a BCR specific for the hemagglutinin of influenza A/WSN/33 virus (FluBI mice). Their B cells secrete an immunoglobulin gamma 2b that neutralizes infectious virus. Whereas B cells from FluBI and control mice bind equivalent amounts of virus through interaction of hemagglutinin with surface-disposed sialic acids, the A/WSN/33 virus infects only the hemagglutinin-specific B cells. Mere binding of virus is not sufficient for infection of B cells: this requires interactions of the BCR with hemagglutinin, causing both disruption of antibody secretion and FluBI B-cell death within 18 hours. In mice infected with A/WSN/33, lung-resident FluBI B cells are infected by the virus, thus delaying the onset of protective antibody release into the lungs, whereas FluBI cells in the draining lymph node are not infected and proliferate. The authors propose that influenza targets and kills influenza-specific B cells in the lung, thus allowing the virus to gain purchase before the initiation of an effective adaptive response.

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Capsule

Integrin-modulating therapy prevents fibrosis and autoimmunity in mouse models of scleroderma

In systemic sclerosis (SSc), a common and etiologically mysterious form of scleroderma (defined as pathological fibrosis of the skin), previously healthy adults acquire fibrosis of the skin and viscera in association with autoantibodies. Familial recurrence is extremely rare and causal genes have not been identified. Although the onset of fibrosis in SSc typically correlates with the production of autoantibodies, whether they contribute to disease pathogenesis or simply serve as a marker of disease remains controversial and the mechanism for their induction is largely unknown. The study of SSc is hindered by a lack of animal models that recapitulate the etiology of this complex disease. To gain a foothold in the pathogenesis of pathological skin fibrosis, Gerber and co-scientists studied stiff skin syndrome (SSS), a rare but tractable Mendelian disorder leading to childhood onset of diffuse skin fibrosis with autosomal dominant inheritance and complete penetrance. They showed previously that SSS is caused by heterozygous missense mutations in the gene (*FBN1*) encoding fibrillin-1, the main constituent of extracellular microfibrils. SSS mutations all localize to the only domain in fibrillin-1 that harbors an Arg-Gly-Asp (RGD) motif needed to mediate cell-matrix interactions by binding to cell surface integrins. Here they show that mouse lines harboring analogous amino acid substitutions in fibrillin-1 recapitulate aggressive skin fibrosis that is prevented by integrin-modulating therapies and reversed by antagonism of the pro-fibrotic cytokine transforming growth factor-beta (TGFβ). Mutant mice show skin infiltration of pro-inflammatory immune cells including plasmacytoid dendritic cells, T helper cells and plasma cells, and also autoantibody production; these findings are normalized by integrin-modulating therapies or TGFβ antagonism. These results show that alterations in cell-matrix interactions are sufficient to initiate and sustain inflammatory and pro-fibrotic programs and highlight new therapeutic strategies.

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