depolarizations, decreased K⁺ conductance and enhancement of excitatory signals such as Glutamate [12]. Serotonin plays a dual (may be synergistic effect) role in increasing susceptibility to seizures. Firstly through 5-HT2A receptors (as mentioned above) and secondly through its effects on sleep; on the one hand, it promotes NREM sleep (NREM increases susceptibility to seizures), while on the other hand it exerts inhibitory role in actual REM initiation and PGO wave generation, the latter two supposed to be potent anti-epileptic.

Thus, the evidences put forward do support the possibility of risk of epileptogenesis during meditation as hypothesized in my article at the same time refuting the objections and controversies raised in above comment-letters.

Finally, funded by the Indian government, a ten-year study by Desiraju [13] could yield no beneficial positive effects of meditation as claimed by its proponents. Lazarus [14] and Otis [15] have also on the contrary reported adverse outcomes and effects of meditation.

References


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Salmeterol and paroxetine: More evidence for paradoxical medicine

Lurie and Wolfe of Public Citizen recently suggested in the Lancet that Glaxo Smithkline (GSK) bundled post-trial data with trial data that effectively downplayed the risk of death with use of salmeterol [1]. Similar claims were recently levied against GSK only last year with respect to studies of paroxetine in children and adolescents in which data on suicide was not initially fully disclosed [2,3]. While these discussions have focused on incomplete disclosure, we remain intrigued by the possibility that chronic use of these drugs can produce an exacerbation of the underlying condition. In the case of paroxetine, the phenomenon of serotonin withdrawal syndrome has become increasingly recognized, and, as such may have contributed to an increased risk of morbidity and mortality. In the case of salmeterol, although
the beta-agonist may have directly produced some of the witnessed sequelae, this putative risk may actually be less than what has been traditionally presumed [4]. However, an additional explanation may merit consideration—the idea that paradoxical effects caused from interval withdrawal between times of administration may have caused autonomic remodeling and patient deaths. If so, beta-agonists may potentially represent the fourth class of drugs to show paradoxical responses that did not emerge initially with shorter term trials and that only became revealed with chronic use. Each of these drug categories has an entirely independent mechanism of action—we have previously discussed cyclooxygenase-2 inhibitors [5] and more recently the PPAR agonist muraglitazar [6]. Given such a consistent pattern of behavior for different types of medications, we believe that this phenomenon warrants a more fundamental explanation—namely that we are witnessing paradoxical medicine in action [7], but as a detriment rather than as a benefit.

References


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Tumor-causing plant bacteria may infect animals

Agrobacterium tumefaciens is a well known phytopathogen; it is a soil bacterium that causes plant’s tumors disease called Crown Galls [1]. The microbe contains a DNA plasmid called DNA Ti (tumor inducing) [7]. The bacterium is introduced in a wounded plant and the plasmid is then integrated in to the cell plant genome and overproduces growth regulators (auxins and cytokines) that can initiate the Crown Gall [6] disease. The growth regulators are also present in mammal’s metabolism. Discovered by Erwin Smith (1854–1927) [2–5], we know now that the soil bacterium may jump Kingdoms [8]. In 2001, Vitaly Citovsky from the State University of New York and colleagues found that the plant bacterium was able to attach human cells in vitro and insert its DNA as it does with plant cells [9]. Recently, the soil microbe has been isolated from blood samples of cancer patients [10–12]. We are studying the potential infectivity and oncogenetic flow between Agrobacterium and animals in vivo [13].

After 120 days of wild Agrobacterium injection on wounded Chrysanthemum maximum, galls are evident and pathologically confirmed. Then, we treated 40 SWISS mice, 8 mice each group, with A. tumefaciens cultured: Orally, SC and intraperitoneal injections weekly shaved skin and topicated with crown galls bacteria and only shaved as control. After 11 weeks, skin lesions and regionally nodes are evident in 6–8 mice in shaved and topicated group. At optical microscope, H–E, lymphoid hyperplasia, neoangiogenesis and neofibrinogenesis are confirmed. After 18 weeks, multiple tumors and distant metastasis are seen, due to hyperplasia and mesenchymal metaplasia.

Our observations confirmed in vivo the pathogenicity of A. tumefaciens on mammals and we think...