Dear Editor,

People with schizophrenia have a two to three-fold increased risk to die prematurely than those without schizophrenia and this excess of mortality is accounted for by a combination of increased risk factors such as patients’ life style, suicide (in particular in young male patients soon after diagnosis), premature development of cardiovascular disease, high prevalence of metabolic syndrome, carbohydrate and lipid metabolic disorders and equally important but not so often mentioned is sudden unexpected death.1 The exact pathophysiological cause of sudden unexpected death in schizophrenia (SUDS) is unknown, but it is probable that cardiac arrhythmia plays a potential role.2 Because cardiac abnormalities are an important cause in sudden death we consider whether it is possible that winter temperatures may facilitate cardiac abnormalities and hence SUDS. Exposure to winter temperatures is considered to be one of the main factors influencing morbidity and morality from cardiovascular diseases, including sudden death.3 Therefore, it is interesting to review some findings4 that explain an increase in cardiac events to cold temperatures: there is 53% more cases of acute myocardial infarction reported during the winter compared with the summer. During the winter, increases in hemoconcentration (erythrocyte count, plasma cholesterol and plasma fibrinogen levels) have been reported, which could contribute to arterial thrombosis. Cold weather can induce a higher systemic vascular resistance with an increase in the blood pressure (thus increasing oxygen demand). Winter temperatures may be associated with flu season and an increase in upper respiratory tract infections could place stress on the heart. According to these lines of evidence, we believe that cold weather could also be considered a new potential risk factor of sudden cardiac death in patients with schizophrenia.

At the moment, there is no information in the literature that describes the relationship between winter temperatures and SUDS. In accordance with this reasoning, we are totally in agreement with Davidson’s perspectives:4 “Because the modifiable risk factors for coronary atherosclerosis and sudden death are so prevalent within the schizophrenic population, it is important for clinicians treating patients with schizophrenia to know what these risks are and understand how they can contribute to increased mortality in these patients”.

In the mean time, some actions (medical or non-medical therapies) may help to prevent SUDS. For that, Kloner5 has described some very interesting commonsense and prudent tactics that the physician should consider during the winter time (called “Merry Christmas Coronary” and “Happy New Year Heart Attack”), especially for patients with established cardiac disease or for those with known risk factors for cardiac disease: 1) Instruct patients to avoid delay in seeking medical attention, should cardiac symptoms occur; 2) Instruct patients to avoid the known triggers for acute myocardial infarction, as excess physical exertion (especially shoveling snow), overeating, lack of sleep, emotional stress, illegal drugs, and anger. Avoid excess salt and alcohol intake. (Alcohol can also precipitate arrhythmias such as atrial fibrillation – the “holiday heart syndrome” and can depress cardiac contractility; 3) Modify and treat known cardiovascular risk factors (e.g., hypertension, smoking, diabetes, dyslipidemia); 4) Consider aspirin or β-blockers, or both, if appropriate; 5) Instruct patients to avoid exposure to severely cold temperatures; 6) Consider flu shots where appropriate.

Finally, the next logical steps to us, neuroscientists, are to understand and associate the mechanisms by which cold weather could influence the cardiovascular system of patients with schizophrenia. These mechanisms are likely to be important for developing new strategies in the prevention of SUDS.

Fulvio A Scorza, Roberta M Cysneiros, Esper A Cavalheiro
Laboratory of Experimental Neurology, Universidade Federal de São Paulo (Unifesp), São Paulo (SP), Brazil

Ricardo M Arida
Department of Physiology, Escola Paulista de Medicina, Universidade Federal de São Paulo (Unifesp), São Paulo (SP), Brazil

Wagner F Gattaz
Laboratory of Neuroscience (LIM-27), Department and Institute of Psychiatry, School of Medicine, Universidade de São Paulo (USP), São Paulo (SP), Brazil

References