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COMMENTARY TO SPECIAL ARTICLE

Heuristic model of depressive disorders as systemic chronic disease

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The essay by Professor Patten on the various paradigms for depressive disorders has the merits of inviting an epistemological reflection or simply encouraging thinking out of the box. Eight possible models are proposed for depressive disorders: chemical imbalances, degenerative conditions, toxicological syndromes, injuries, obsolete category, medical mysteries and evolutionary vestiges. Medical disease models have been important in shaping the reflection, especially for an international journal such as *Epidemiology and Psychiatric Sciences.* In the following comments, I would like to enrich and link some of the models with the public health perspective, provide further epidemiological findings often drawn from Canada, and propose the heuristic model of depressive disorders also as a systemic chronic disease.

Public health recognises four determinants of diseases: genetic, environmental (social or physical), health habits and services. In its classical context, epidemiology as a discipline has provided public health with data on prevalence and methods to test aetiology and provides a description of service utilisation.

Epidemiological studies on depression have provided a picture of depression both as a short-lived episodic disorder that is apparently very reactive to life events and situations, and as a chronic disorder that is also reactive to life events and chronic difficulties. For example, as early as the end of the 1980s, the Epidemiological Catchment Area study in the USA found a 40% complete remission of major depression after 1 year for cases identified in the course of community surveys (Regier *et al.* 1984; Robins & Regier, 1991). With the emergence of the monstrous DMS-5, there has been a continuous debate between 'lumpers' and 'splitters' in many fields of epidemiology, including psychiatry. Genetic epidemiology has led Kenneth Kendler to the terrible verdict that 'DSM does not carve nature at its joints' (Kendler & Gardner, 1998; Kendler, 2013). It underlines the failure to distinguish between clinically severe formal depression, major depression and 'sub-clinical syndromes' (Kessler *et al.* 1997). Sir David Goldberg was more blunt as a 'lumper' at a public conference about anxio-depressive disorders when he remarked: 'It would be presumptuous to call them separate diseases when they share the same genes.'

What may be the nature of the beast? Certainly not the limited theory of the anomaly of neurotransmitters as Patten points out. I believe it is better represented as an anomaly of stress mechanisms. Coincidentally, the general theory of stress was developed in Montreal by Professor Hans Selye (1907–1982). And more recently, also from Montreal, Michael Meaney, using animal models, and Gustavo Turecki, using post-mortem depressed suicides, have illuminated epigenetic pathways in showing the effects of the social environment (poor parental care, childhood trauma) and stress on glucocorticoid receptor genes (Turecki & Meaney, 2014). Recent Canadian Surveys have confirmed the association between childhood trauma, a diversity of mental disorders and suicidal behaviours (Afifi *et al.* 2014). Can this theory be put to the test? Would the reduction of childhood trauma or impoverished parental guidance improve child attachment and break the cycle of reproduction of anxiety and depression, as Robins & Rutter (1990) put it? The longitudinal study of children of depressed mothers in underprivileged areas together with the establishment of a universal day care program in Quebec made it possible to test the theory. Herba *et al.* (2013) found that for mothers with symptoms of high mood disorders, regulated early childcare services reduced the risks of problem

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internalisation by their children. They suggested that regulated child care services may be used as a public health intervention to buffer the negative effect of maternal depression on the internalisation of problems by children.

Childhood trauma has been associated not only with mental disorders but with other chronic diseases and lower life expectancy (Cicchetti, 2010). The allostatic load model, a more general theory of the hypopituary-glucocorticoid pathway, was developed by Bruce McEwen (1998, 2003). This 'wear and tear' model stipulates that with the early developmental mechanisms described above (Turecki & Meaney, 2014), and with continuous exposure to psycho-social and normal life stress, a maladaptative overreaction occurs that wears and tears different circulatory, inflammatory, immune and psychological systems and, with other genetic predispositions, leads to premature chronic diseases like cardiovascular disease, diabetes, hypertension and depression. Epidemiological studies of depression have regularly found associations between depression and chronic diseases to an extent that up to 60% of depressed individuals may also have some common chronic diseases (Schmitz *et al*. 2007). These chronic diseases are therefore linked by aetiological pathways and respond to undue psychosocial stress with flare-ups of diseases, some very serious. In the workplace for example, where common mental disorders have become the primary sources of disability (Jacobs *et al*. 2010), the same undue psycho-social stress is associated with increased cardiovascular diseases and burn-out, as shown by the now half-century-old Whitehall studies and their replications in Canada (Marmot *et al*. 1984; Stansfeld *et al*. 1999; Vézina *et al*. 2004; Aboa-Éboulé *et al*. 2011). Again, can it be prevented by environmental intervention? In Quebec City, Bourbonnais *et al*. (2011) showed the long-term positive effects of an intervention on psychosocial work factors among healthcare professionals in a hospital setting. Continuous improvements of work environment practices (e.g., the Quebec 'healthy enterprise standards' (BNQ, 2008)) have been embedded in work and safety standards to improve both somatic and mental health. It remains to be tested if large-scale implementation is feasible, should changes occur in organisational practices that result in better cardiovascular and mental disorder outcomes.

Less has been said by Patten about the health services epidemiology in relation to depression and models of chronic disorders. The vast majority of people with common mental disorders and anxiety depressive disorders are identified and treated by primary care providers, i.e., general practitioners. Primary care has been identified by the World Health Organisation in both developing and industrialised countries as the fulcrum for the therapeutic management of mental disorders. One Canadian population-based and administrative database study in a public managed care system that covers over 99% of the population showed that 75% of the population had consulted a general practitioner at least once in the preceding year; that one in five were identified or seen for mental disorders in the preceding year; that those identified with a mental disorder accounted for 3–4 times more medical visits than those without; and that two-thirds of their visits were for other common chronic diseases rather than for common mental disorders like anxiety or depression (Ouadahi *et al*. 2009).

In industrialised countries, primary care has been moving from the treatment of infectious diseases to very common chronic diseases like diabetes, hypertension, cardiovascular diseases, chronic pain and asthma. Effective clinical chronic diseases management calls for a re-organisation of healthcare in favour of patient education and self-management, monitoring of symptoms and treatment adherence, decision support for medication management, a patient registry and supervision by care managers (Wagner, 1998; Kates & Mach, 2007). The work of Katon and Seelig (2008) has shown that the joint treatment and management of depression and chronic diseases like diabetes has synergistic effects on both conditions and on general social functioning, confirming the epidemiological studies that have shown a synergistic effect on the social functioning of patients with depression in the presence of common chronic diseases like diabetes.

Professor Patten has stimulated the present comment by inviting us to think outside the box about the aetiology and research agenda in the clinical and public health areas. The heuristic model proposed is that some depressive disorders share common aetiological pathways with other common chronic disorders. Prevention strategies are possible for all these chronic disorders – from regulated childcare to better workplace organisational practices – and better clinical outcomes could be achieved in primary care with the support of specialist care within a chronic disease disorders management model.

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