Correspondence

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Letter to the Editor

Dear Editor,

Disease mongering is the widening of the boundaries of treatable illness in order to expand markets for those who sell and deliver treatments (Moynihan *et al.* 2002). Psychiatry should be at pains both to avoid disease mongering and to avoid the appearance of it. I write unconvinced by the 'ultra-high risk of psychosis'/ 'psychosis risk syndrome' category (O'Connor, 2013), having followed with interest the debate about the psychosis risk syndrome, offering as it did the dramatic (for academic psychiatry) scenes of Allen Frances, Chair of DSM-IV, nominating it as 'the worst DSM-5 proposal' (Frances, 2010), and the excoriation of Dr Patrick McGorry in Australian parliament (Whitely, 2012).

Those supporting the use of the 'ultra-high risk' term submit that it is 'possible to identify individuals who may be at risk of developing psychosis'. Clearly this is in marked distinction to being able to identify individuals who are going to develop psychosis, and it is here that any potential clinical utility of the diagnosis expires. There is a simple explanation for the decline in the predictive value of the 'ultra-high risk' criteria (Yung et al. 2007). The initial high positive predictive value was achieved by means of post-hoc selection of scale items and the definition of 'transition' to psychosis was not determined by DSM-IV or ICD-10 diagnoses, but by an arbitrary cut off on the BPRS defining 'essentially the threshold at which neuroleptic medication would be commenced in common clinical practice' (Yung et al. 2003). New prospective samples were therefore highly unlikely to replicate the initial findings, as predicted by Warner (2005).

There are circumstances where treating risk is appropriate, for example cholesterol-lowering medications to reduce risk of heart attacks. This is down to finely weighing the potential risk against the benefit. CBT, while not an intervention associated with considerable harm, is costly and uses finite resources. While omega fish oils are not generally thought to be harmful, the entirety of the evidence base supporting their delaying the onset of psychosis is an RCT involving 81 individuals (Amminger *et al.* 2010). Three years have passed without positive replication. I note that a larger prospective replication study was registered with the Australia New Zealand Clinical Trials Register in 2008 (Trial ID: ACTRN

12608000475347), and another in 2010 (Trial ID: EUCTR2008-005004-13-AT), with a more restricted age profile, with the EU clinical trials register. No results have been published, and one is tempted to infer that these are negative. Omega fish oils have impressed previously as an intervention desperately seeking an indication, with initial success followed by larger meta-analyses showing no clinically meaningful effect on, for example, mortality, cardiovascular disease, cancer (Hooper *et al.* 2006), dementia (Sydenham *et al.* 2012), depression (Bloch & Hannestad, 2012), ADHD (Gillies *et al.* 2012).

The greater fear of course was of widespread lowering of thresholds for prescribing long-term antipsychotic medication. There is a superficial appeal to the idea that antipsychotic medication could reduce the rate of conversion to major psychotic disorders, and the attendant impairment of functioning, but this should not override the need for evidence-based practice. The question has been asked and answered - there is no evidence of improved outcome with use of medication in this group (Marshall & Rathbone, 2011), and yet in centres where the term 'ultra-high risk' denotes a patient cohort, treatment with antipsychotic medication occurs (Nieman et al. 2009; McFarlane et al. 2010). This is deeply discomfiting. While DSM-5 may well not be the 'paradigm shift' anticipated at its inception, the rejection of the 'ultra-high risk' diagnosis reflects some scientific rigor in the process and is to be welcomed.

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