Correspondence

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Response to Letter to the Editor

I thank Dr Cummings for her correspondence and for offering me an opportunity to clarify a number of points in relation to the Ultra High Risk (UHR) research.

I share Dr Cummings concern about the perils of disease mongering in psychiatry and also in medicine more widely. However, I disagree that UHR research is primarily motivated by a desire to expand markets for those who sell and deliver treatments, rather I believe it to be motivated by a desire to improve prognostic outcomes for one of the most debilitating chronic illnesses in medicine; schizophrenia. As such, I do not accept her suggestion that that the UHR field is disease mongering. I also disagree that as doctors we must avoid 'the appearance of' disease mongering. Appearances can be deceiving and if we rely on an enquiring mind, a systematic approach and the application of scientific rigour we can ensure our practice and our research stands up to scrutiny.

Dr Cummings suggests that 'any potential clinical utility' of the UHR criteria 'expires' on the basis that they identify 'potential risk' rather than just those who are certainly going to develop psychosis. While it would be ideal that only true positives were identified by the UHR criteria, false positives are tolerated in many other areas of preventative medicine and the weighing of the potential risk against the benefit is often difficult to 'finely balance'. In the primary prevention of cardiovascular events about 100 people need to be treated with a statin to prevent one heart attack or one stroke (Bandolier, 2004; Taylor *et al*. 2011). Conversely, for every 136 people treated with a statin, one will develop severe liver dysfunction, one will develop moderate-severe myopathy and four will develop cataracts as a consequence of treatment with the drug (Hippisley-Cox & Coupland, 2010). One in every three women found to have a tumour on breast cancer screening who are treated with surgery ± chemo-radiotherapy have tumours which would not have progressed (Bleyer & Welch, 2012). They are treated unnecessarily.

The debate associated with the proposed inclusion of a psychosis risk syndrome into DSM-5 was indeed lively and at times dramatic. However, the core themes being debated in the scientific literature (Woods *et al*. 2010; Nelson and Yung, 2011), at conferences (Lunchtime Debate, 2010), and in the online scientific forums (Schizophrenia Research Forum, 2013) were nuanced and not merely defined by whether the UHR concept/research was convincing or not. Many of the most vehement opponents to the psychosis risk syndrome's inclusion into DSM-5 were themselves leading researchers in the UHR field, for example Prof. Alison Yung, Prof. Shon Lewis, Prof. Anthony Morrison and Dr Barbara Cornblatt (Lunchtime debate, 2010; Schizophrenia Research Forum, 2013). Those in the scientific community who opposed the inclusion of a risk syndrome in DSM-5 did not dispute the worthiness of early intervention. However, like Dr Cummings they highlighted the issue of the potentially high number of 'false positives' diagnosed with the syndrome.

While identifying false positives may be acceptable in other areas of medicine, those who opposed the inclusion of the risk syndrome argued that the risk–benefit ratio was not favourable with regards to the risk syndrome due to a number of additional consequences associated with the identification of risk in this sample. These include the high risk of stigma and discrimination, the possibility of unintentionally sanctioning the use of antipsychotic medications for patients with attenuated psychotic symptoms in the absence of good evidence, and the lack of a clear evidence base for effective interventions and as such the low benefits resulting from case identification. In April 2012, it was announced that due to the nascency of the UHR research and the lack of substantive field trials, a psychosis risk syndrome would not be included in the main text of DSM-5. However 'Attenuated Psychotic Syndrome', is included in Section III for conditions being recommended for further study.

I agree with Dr Cummings that the basis for intervention of any kind in the UHR population is sparse. However, to state that the question of any intervention in this cohort has been 'asked and answered' is an over statement. The Cochrane review referenced in my paper and by Dr Cummings highlights some of the methodological difficulties with the existing literature and recommends further evidence is needed before recommendations on treatment in this cohort can be made (Marshall & Rathbone, 2011).

With regards to the ω fatty acids clinical trial that Dr Cummings referred to in her correspondence. This trial is part of a large multicentre double blind randomised placebo controlled trial called the NEURAPRO (North America, EURope, Australia PROdrome) study. The Australian site completed recruitment in August 2012 (Nelson, 2013) and the European site in Vienna only recently completed recruitment (Mossaheb, 2013), as such it will be some time before data from these trials will be analysed and the results published.

In conclusion, I too welcome the decision made by authors of DSM-5 to not include a psychosis risk syndrome in the latest edition. Such an inclusion would have been premature. However, I await with anticipation the data emerging from the large prospective multisite UHR studies due to be completed and published in the coming months and years.

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