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Big data: little difference

The literature on racial and cultural variability and the experience of pain is relatively small, but often contentious and important for reasons of policy and equity of access to health care. This is well documented in the USA (Green et al., 2003; Tait and Chibnall 2014) and European countries (Dixon et al., 2011). Some early experimental studies (e.g., Sternbach and Tursky 1965) suggested that while there were no differences between racial groups in pain thresholds, differences could be observed when tolerance was measured. In contrast, other studies suggested that black Americans had lower thresholds than their white counterparts (Zatzick and Dimsdale 1990). Unsurprisingly, establishing differences between groups in the apparently simple relationship between a controlled physical stimulus and subjective report and behavioural activity associated with the stimulus reveals that relationships are subtly affected by factors such as the social context of testing (Hsieh et al., 2011). In a recent review Tait and Chibnall (2014, pp135-136) précis the findings as follows: ‘mechanisms underlying these differences, however, remain elusive ...’.

In this issue of the journal John Robbins and his colleagues (Robbins et al., 2014) report an analysis which tested for an association between the degree of African ancestry in American women and the report of pain. Robbins and his colleagues rightly note that in most previous research ethnicity has been self-defined. They note that ‘to date, no specific genetic link between African ancestry and pain has been established’ and their contribution is to rectify this by assessing genetic markers of ancestry. They used a set of 92 single nucleotide polymorphisms (SNPs) to estimate the proportion of African vs. European ancestry in a large sample (N > 11,000) of self-declared African American women recruited to several studies

between 1993 and 1998. Data were also available on several measures (pain, depression – CES-D, and socio-economic status) as well as information on age, education, diabetic status and self-reported health status. Pain was measured as a composite of two items from the SF-36, each rated on a 0-5 scale. The items were *how much bodily pain had been experienced* and *how much pain interfered with normal work*. Both items were rated over the previous 4 weeks and the items were combined and recoded to a 0-100 scale. As is the convention with the SF-36, lower scores represented more pain. The correlation between African ancestry and the composite pain variable was $r = -0.0175$, i.e., the greater the proportion of African ancestry the higher the self-reported pain. With such a large sample this correlation is significant (approximate p-value = $3.7E-15$) as were several other correlations. P-values, however, can be notoriously misleading (Ziliak and McCloskey 2008). In the present study it might be helpful to compare the magnitude of the correlation between African ancestry and pain with the correlation between other variables and pain. The authors report correlations of $r = -0.132$ for social economic status and $r = -0.255$ for depression. (To put these correlations into context, the relationship between depression and pain corresponds to a Cohen's effect size value, $d = 0.53$, which is much larger than the difference observed between treatments and controls in many RCTs in the field of chronic pain.) These two correlations account for 1.74% and 6.5% of the variance, while African ancestry accounts for 0.03%. To express this rather crudely, depression accounts for 216 times more variance and the measure of neighbourhood SES for more than 50 times more variance than does African ancestry. Big data sets have many merits and their propensity to generate significance effects is seductive (Mayer-Schönberger and Cukier 2013).

Robbins and his colleagues have undoubtedly made an advance in using a biological measure of ethnicity, but we should be cautious about the interpretation of

this. They rightly comment on the relative magnitude of the relationships observed in this study. The observed relationships are not surprising and are consistent with many other observations. Depression might be expected to correlate with the present measure of pain given that one of the two items contributing to the scale refers to interference (lowered behavioural activity). The need for better controlled studies (better definition and improved measurement of pain) and to apply the lessons learned from the earlier generation of studies (Tait and Chibnall 2014) is an easy statement to make. But perhaps we might ask whether such an enterprise should be high on the research agenda. Can we seriously entertain a hypothesis which broadly suggests that in the evolution of *homo sapiens* something as fundamental as the protective mechanisms of pain would be subjected to differential selective pressures associated with race? I suspect that if such differences do exist then Tait and Chibnall are right: the differences will be small. They are also right when they state: 'It is important to recognize, however, that the reported differences are small and may have little implication for an individual patient...'. Whatever differences do exist, they should have *no* implication for an individual patient as far as the delivery of care is concerned. 'I will not permit considerations of age, disease or disability, creed, ethnic origin, gender, nationality, political affiliation, race, sexual orientation, social standing or any other factor to intervene between my duty and my patient. (The Declaration of Geneva, <http://www.wma.net/en/30publications/10policies/g1/>).

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