

(n=3). Ten NPWT patients did not heal and were assessed within the study window on or before day 112. Likewise, 15 control patients had their final visits on days 113 (n=4) and 114 (n=11).

The rationale for presenting the table beneath figure 2 was to provide the exact count of patients remaining on study with open wounds as of a specific study day; given the application of an acceptable interval of time surrounding a study office visit (eg, ± 7 days), the values might not coincide. The legend for figure 2 described "at risk" patients after the last NPWT patient healed (at day 106). At that time, there were 12 NPWT patients with open wounds. Seven had their last visit on or before day 112, and five after day 112, as described above. In the control group, the last patient healed at day 112, therefore the data are identical to the numbers displayed in the text of the figure.

Regarding decisions for NPWT application, the VAC device was used according to product labelling. Such use allowed clinicians to change therapy on the basis of changes in the wound. The decision for surgical closure was also based on clinical judgment. This method allowed a real-world assessment of a constantly evolving wound environment within the context of a randomised trial. The primary objective was to assess complete wound closure with or without surgical intervention.

We appreciate these queries and believe that continued robust inquiries into this fascinating discipline can do nothing but reduce the burden of lower extremity amputation in high-risk patients.

We declare that we have no conflict of interest other than that stated in the original paper.

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Speciation and schizophrenia: literature anticipates science

Simon Wessely's perceptive analysis (Nov 19, p 1765)¹ of Sebastian Faulks' novel *Human traces* elucidates the uneasy relation between fiction and scientific concepts. "Literature expels science" was a view considered by Medawar, but Shelley's dictum that "all science is poetry" implies that they answer to the same rules.

I claim originality for the hypothesis that the genetic variation relating to psychosis had its origin in the speciation event that gave rise to modern *Homo sapiens*, and was associated with cerebral asymmetry, the neural basis of the capacity for language.² At his invitation, I corresponded with Sebastian Faulks about evolutionary accounts of schizophrenia while he was writing the novel. When he sent a draft, I read an able summary of the above theory expounded by the character Dr Thomas Midwinter in a lecture dated 1910. We agreed on the significance of the concept but to differ on its origin.

But interesting questions are raised. First, is the theory correct? Other researchers³ reach contrary conclusions about the genetics of psychosis. I am gratified that Faulks saw merit in it but am chagrined at my inability to persuade the relevant scientific community of the cogency of the hypothesis. Second, could it have been arrived at in 1910? Simon Wessely implies that evolutionary theory was undeveloped at that time, but this provokes the question: what was missing? Faulks argued: "the basic parts of the syllogism were in the public domain and I think that a rather broad brush thinker, steeped in Shakespeare as much as in the chemistry of cells, could have put together a theory along these lines, since the basis of Thomas's argument 'Only man is mad; what makes madness therefore must be what makes man'

is so simple as to be almost tautological."

I responded "It's easy when you know how!" but one can test whether anyone came near by a literature search in the Web of Science database on "speciation and schizophrenia". Ten of 12 references including the first five are to papers by myself, with the first in 1995.² The bibliometric approach also reveals the probable missing element: "speciation and sapiens" apparently did not occur as concurrent title or keyword terms before 1992.⁴ The notion of a discrete genetic event, somewhat at odds with the "evolutionary synthesis" of Mendelian genetics and Darwinian gradualist theory forged in the 1940s, perhaps required the formulation of the out of Africa hypothesis of modern *Homo sapiens*⁵—the concept of a single species with a temporally defined origin.

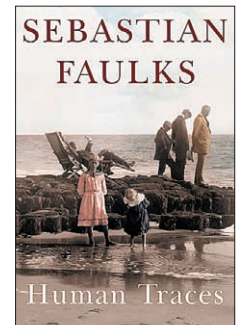
Thus the salient fact is not that Thomas Midwinter in 1910 could not have conceived the notion that the genetics of schizophrenia and the speciation of modern *Homo sapiens* are related, but that he did not do so. And if he had done so would he have been right? In 2006, the era of the chimpanzee genome, the hypothesis invites refutation.

I declare that I have no conflict of interest.

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Human Traces
Sebastian Faulks