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Unloading the trunk: neurasthenia, CFS and race

A. Luthra, S. Wessely*

King's College School of Medicine, Institute of Psychiatry, 103 Denmark Hill, London SE5 8AZ, UK

Abstract

The aetiologies of both chronic fatigue syndrome (CFS) and its predecessor neurasthenia, have been linked to technological advances in 'developed' countries. This paper discusses how this has led to a form of race thinking within discussions about fatigue which has persisted for more than a century. We review the historical development of this race thinking from neurasthenia to CFS and describe how it is manifested in both the lay- and medical literature. We also review the epidemiological literature on CFS and ethnicity to better understand the relatively low percentage of non-white patients seen in tertiary referral clinics for CFS. The aim of this paper is to act as a starting point for a debate on race and CFS .

Keywords: Neurasthenia; Chronic fatigue; Ethnicity; Non-white; Epidemiology; 'Developed' countries

Introduction

The ever increasing complexity of modern technology is mirrored by an increasingly complex relationship between technology and health. A feature of this relationship is a gap in understanding between the medical profession and those it aims to serve. This gap frequently manifests to healthcare providers as an array of undefined, poorly understood symptoms—tiredness, fatigue, non-specific pain, sore throat, etc.—which are often interpreted differently by patients and doctors. Many possible causes related to modern technology have been implicated: keyboards (Ferguson, 1987), electricity (Hillert & Kolmodin-Hedman, 1997), electricity pylons (Jauchem, 1992), mercury fillings (Sternman & Grans, 1997), world radiation (Vyner, 1983), office buildings (Bardana, 1997), visual display units (Goethe, Odont, & Nilsson, 1995) and wood preservatives (Gupta, Perharic, Volans, Murray, & Watson, 1997).

The history of neurasthenia, the predecessor of the modern chronic fatigue syndrome (CFS), suggests that anxiety about technology is not only a modern phenomenon (Wessely, 1997). George M. Beard, the so-called 'Godfather of neurasthenia', argued that the steam engine, the telegraph, newspapers, and the sciences were essential aetiological factors in the development of the disease (Kim, 1994). Such attribution can be traced back to the Renaissance when, due to the nature of their work, courtiers and scholars were described as more liable to 'nervousness' than labourers and yeomen (Martensen, 1994). For the pioneers of neurasthenia therefore (Beard, 1869), it was not the men putting up the telegraph poles or building the steam engines who were at risk—it was those using these inventions. Intrinsic to neurasthenia was a class bias which characterised it, at least initially, as a disease of the 'civilised'. Vulnerability to modern technology was thought to arise from the excessive demands placed upon the users' sensitive nervous systems (Sicherman, 1997). Alongside this class bias existed a gender and race bias. During the late 19th century hypotheses about the causes of neurasthenia in women included: over-education which overtaxed their mental powers; the business world which attacked their 'sensitive organisations'; childbearing which was thought to have become harder for the urban woman; and modern clothing because it was designed more for display than for comfort (Haller, 1971).

*Corresponding author. Tel.: +44-20-7848-0796; fax: +44-20-7848-5408.

E-mail address: s.wessely@iop.kcl.ac.uk (S. Wessely).

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Race and neurasthenia

The explicit race thinking around neurasthenia during the early 20th century was largely based on contemporary evolutionary theory and social Darwinism.

"…neurasthenia is mostly a disease of brain workers.. men.. and civilised races"–(Mitchell Clarke, 1905)

"……the American Negro never suffered from neurasthenia till recently. But now it is of not infrequent occurrence, especially in the half breeds. Alcohol, syphilis and moral degeneration partly account for this, but more important is the fact that a lower race is trying to compete and is unfit to do so….. the mere struggle of the best specimens of an inferior race to attain the plane of a superior leads often to their downfall" (Burr, 1910).

Beard was heavily influenced by the theories of Spencer and Darwin (Beard, 1882) and race thinking was intrinsic to his initial formulations of neurasthenia . He used such race thinking to argue that Americans were more susceptible than Europeans because they were more developed (Haller, 1970; Veith, 1968). He also applied it to the 'lower races' as shown by the following anecdote from his book 'American Nervousness' : A black man was struggling up some stairs with a trunk when a white man came to his aid by quickly lifting it to the top. Beard argued that although white people could not work all day like black people, they could channel their energy into specific tasks and had a sophistication that black people lacked. He felt it was this lack of sophistication combined with an immature nervous system that explained why black people did not suffer from neurasthenia (Haller, 1970).

There were, not surprisingly, many criticis of Beard's theories. Some argued that it could not be an American disease since Europeans also seemed particularly susceptible. Doctors on both sides of the Atlantic attacked his scholarship (Hammond, 1881)—many themselves offended at being labelled as vulnerable (Spitzka, 1881). Despite this, the concept of neurasthenia affecting only upper class whites held sway for at least two decades till the turn of the 20th century. The first to be challenged was the class bias. In 1889, Charcot pointed out that neurasthenia was no longer a disease of the upper classes (Levillain, 1891). By 1906, in a reworking of the original theories, the working class were now seen as the main victims of the disease. At the same time, the perspective on race also began to shift although never to the same degree as that on class. This resulted from the discovery around 1895, of European and American Jewish patients with neurasthenia (Wessely, 1994; Drinka, 1984a, Chapter 8).

A notable effect of all these changes was a clear theoretical shift in aetiological hypotheses from Beard's 'reaction-and-shock' on a healthy nervous system, to include hereditary degeneracy as a second hypothesis. The latter was seen as the most likely cause of neurasthenia amongst the working-class, the mechanism being a social drift downwards into the lower classes due to bad genes, with reinforcement at that position by a combination of genes and environment (Drinka, 1984b, Chapter 9). At the time such a hypothesis was not unique to neurasthenia. Other disorders such as hysteria, schizophrenia and even 'eccentricity' were labelled as due to 'hereditary theory'. Despite the genetic basis to these theories, they were not applied to ethnic groups. One can only hypothesise that neurasthenia was not felt to be worthy of further examination in non-whites and that Beard's explanation—that the 'lower races' possessed nervous systems which were too immature—was seen as sufficient.

The decline, fall and rise of neurasthenia

Several researchers have mapped the shift of neurasthenia from a neurological entity in the 19th century to a psychological one in the 20th century (Kim, 1994; Wessely, 1990; Abbey & Garfinkel, 1991). Many reasons have been put forward for this. Abbey and Garfinkel (1991) argue that during the late 19th century, neurasthenia had a social role which enabled patients to cope with modern stressors. When these stressors changed in the early 20th century, so did the role of neurasthenia. It has also been argued that the demise of neurasthenia resulted from the combination of changes in psychiatric nosology, a shift in the social class bias and a change in the accepted aetiology of the illness from organic to psychological (Wessely, 1994).

Whatever the exact reason, societal change and changes in illness behaviour were undoubtedly important in the shift to a fallow period between the early 1900s and 1934. During this period, fatigue like illnesses were remarkably quiet in the literature. Their reemergence in the 20th century took several forms. One was the recognition of epidemic- or cluster illnesses which, at the time, were attributed to viral epidemics (Kim, 1994). Such clusters manifested as motor and sensory symptoms with negative laboratory results and occurred at Los Angeles County Hospital (1934), Akureyri in Iceland (1948), the Royal Free Hospital (1955) and at Punta, Georgia (1955). The exact aetiology of these 'epidemics' remains unclear and a psychogenic origin is plausible for at least some of these illnesses (McEvedy & Beard, 1970). Their significance from a historical point of view is that these cluster episodes captured the interest of organic researchers and set the ground for much modern work into the physiological-, infectious- and immunological causes of fatigue. In this new framework, immunology replaced neurology and viruses were seen as the scourge on susceptible immune systems. A second line of descent was via the steady stream of 'new' illnesses blamed on unwelcome features of the

environment. By the 1980s a variety of new hypotheses had appeared on the aetiology of fatigue.These included chronic hypoglycemia, 'total allergy syndrome', chronic candidiasis, dental amalgam, Epstein Barr Virus infection and others. These were seen as specific illnesses and as potential causes of CFS. Multidisciplinary research, a growing lay interest, and the sheer breadth of postulated aetiologies meant that in 1988 the Centre for Disease Control produced a case definition of CFS in order to ensure theoretical consistency. Subsequent work has redefined the criteria for diagnosing CFS and findings within psychiatry (Wessely, Hotopf, & Sharpe, 1998a, Chapter 10; Lawrie, Manders, Geddes, & Pelosi, 1997) infectious diseases (Levy, 1994) and immunology (Wessely, Hotopf, & Sharpe, 1998b, Chapter 9) produce a picture of a complex illness which functions at many levels.

Race and CFS

It is impossible to be certain that the neurasthenia of the 19th century and the CFS of the 20th century are the same illness. However, clinical descriptions are persuasive and there has been no scholarship to suggest otherwise. If CFS represents a 20th century reemergence of neurasthenia, then the question arises: what has happened to the aforementioned class, gender and race biases intrinsic to 19th century neurasthenia? Did these re-emerge with CFS in the 20th century and, if so, in what form? Whilst an examination of all three biases would be justified, the focus of this paper is race. What follows is a review of the lay- and medical literature as well as more recent epidemiological studies to examine the construction of race in the discourse around CFS.

Lay literature on race and CFS

The lay- and medical literature produced around the time of the emergence of CFS is an important starting point in our examination of how race is constructed within CFS. From the lay literature (where CFS is more commonly referred to as myalgic encephalomyelitis (ME)), we have chosen a sample of publications representing some of the most active patient groups in the United Kingdom. These include self-help books, newsletters and journals such as 'Interaction—the journal for Action for ME' (AfME).

Overall, CFS (or ME) is represented as a Western disease. Western patients with CFS are regarded as particularly vulnerable to allergens and the increased prevalence of CFS results from a combination of underexposure to viruses and overexposure to allergens and pollutants. Dawes and Downing (1989) argue that in post-industrial societies the scourge of infection has been replaced by allergens. In non-Western countries—variously referred to as 'less civilised' (Hoskins, 1993) or 'primitive cultures' (Shepherd, 1993)—infections are still thought to play a major role in day to day life. This is ascribed to the universally poor sanitation present across 'tropical countries' (Dowsett, 1988) which exposes patients to viruses not as prevalent in the West. This overexposure to viruses is said to strengthen the non-Western immune system, so preventing CFS in these cultures. Fieden and Bill (1992) take this argument further and suggest that the racial discrepancy in CFS seen in Atlanta, USA is also due to the poor sanitation. Despite this being a Western society, the implication is that the black population are—irrespective of class or economic status—recreating a third world environment which exposes them to viruses and so protects them from CFS.

Although scientific evidence is rarely presented to support these arguments, a growing body of serious research has emerged which suggests that early allergen exposure can be linked to the onset of asthma via a mechanism similar to the above (Svanes, Jarvis, Chinn, & Burney, 1999; Von Mutius, Weiland, Fritzsch, Duhme, & Keil, 1998; Pin, Pilenko-McGuigan, Cans, Gousset, & Pison, 1999). Although this evidence may provide support for the above-mentioned lay hypotheses about CFS, one must remember that the pathophysiology, course, aetiology and treatment of asthma differs dramatically from CFS and that it is extremely difficult to generalise from the one condition to the other.

The lay literature on race and CFS/ME warrants further discussion from a historical point of view. We would argue that the descriptions of race in the CFS/ME lay literature represents a 20th century re-emergence of the 19th century race bias around neurasthenia. Clearly the crude racism of the 19th century has disappeared and those who have advanced some of the modern theories quoted above would undoubtedly find such views abhorrent. Nevertheless, there are parallels between the 19th and 20th century writings in that both eschew social explanations to argue that neurasthenia/CFS is caused by new technology. Differential exposure and patient vulnerability to such new technologies is used to explain the racial differences in CFS. The exact nature of this patient vulnerability has also evolved—neurasthenia patients were seen as 'neurologically vulnerable', whereas CFS patients are 'immunologically vulnerable'. As a result, ME theorists focus on sanitation rather than on biology as the cause of the low rates of ME in non-white populations. We no longer hear about Beard's 'neurological immaturity' keeping black patients free of illness but now read about the poor sanitation of 'primitive cultures' being protective. We conclude from all this therefore, that cultural/racial biases remain alive and well and that this discourse still

harbours traces of the race thinking in Beard's theories of neurasthenia.

Medical literature on race and CFS: a professional silence?

The re-emergence of Beard's thinking in the medical literature was evident as early as 1965 when Holt described the presence of sporadic CFS as 'a new disease sweeping the civilised world' (Holt, 1965). One of the first accounts of doctors' experiences of black people with CFS was from Atlanta in 1984: 'I can count the number of black patients I have seen with CFS on the fingers of one hand' (Dubois et al., 1984).

One of the starting points for this paper was our observation that non-white patients are under-represented at the Chronic Fatigue clinic at King's College Hospital, London. An analysis of the unit database of all patients seen from April 1997 to April 1999 confirmed this. Out of 280 new patients seen over this period, data on ethnic status was available for 246. Of these, only 10% (25) were non-white, 20 African-Caribbean and 5 of other racial origin. This is despite the hospital serving a population which is 30–40% white. There are two possible explanations for this—either that non-white patients may be less vulnerable to CFS or that biases in their labelling, diagnosis or referral preclude them from attending the CFS referral centre.

Buchwald, Manson, Pearlman, Umali, and Kith (1996) report no significant differences between white and non-white patients with chronic fatigue seen in a tertiary referral clinic in Seattle. The features of the illness (demographic, clinical and psychosocial) were very similar in both populations—evidence against any intrinsic ethnic difference. They also found a disproportionately low number of ethnic-minority patients relative to the local population in their tertiary clinics. They ascribe this to cultural differences in health-seeking behaviour, accessibility to care, the perception of symptoms as a problem and the perception of the need for care itself (see Table 1).

Several other studies support these findings. In the so-called 'CDC referral study', 900 sentinel physicians recorded CFS-like illnesses in four American cities to document the administrative prevalence of CFS in health seeking populations. The prevalence rate of CFS amongst whites was 7.6 per 100 000 compared to fewer than 1 per 100 000 for non-whites. Again, researchers conclude that this ethnic difference may be due to referral bias or differences in health seeking behaviour (Gunn, Connell, & Randall, 1993).

The above-mentioned tertiary referral bias has been seen to operate not only with race, but also with sex (Lawrie, & Pelosi, 1995, 1994; Lawrie et al., 1997;

Table 1
Epidemiological studies which look at CFS and race

Studies	Date published	Findings about race
Hospital-based studies		
CDC referral study—4 centres, 900 physicians (Gunn et al., 1993)	1993	Prevalence of CFS in non-whites 7 times less than whites
Seattle, Tertiary referral clinic (Buchwald et al., 1995, 1996)	1996	Features of CFS same regardless of ethnicity. Lower prevalence of CFS in black patients than would be expected from the local demographics? Possibly due to differences in perception of CFS symptoms and access to health care
Community-based studies		
Seattle 4000 patients (Buchwald et al., 1995, 1996)	1995	Black patients 17% of CF patients despite being only 10% of local population
California office workers (Shefer et al., 1997)	1997	Increased prevalence of fatigue lasting more than 1 month in Hispanic and Native-American office workers compared to whites
San Francisco—cross sectional telephone survey (Steele et al., 1998)	1998	Increased fatigue in African-Americans and Native Americans survey compared to whites, but reduced levels in Asians
Chicago. Community-based: initial screening via telephone interview with subsequent clinical assessment (Jason et al., 1999)	1999	Latino and African Americans higher prevalence than whites. Latinos were the highest—due to factors associated with being a member of an ethnic group, e.g. psychosocial stress, barriers in access to health care, more hazardous occupations. Or factors particular to the ethnic group e.g. how long community has been established, or perceptions of ill-health within that group

Buchwald et al., 1995) and social class (Euba, Chalder, & Deale, 1996, Wallace, 1991). In response to this, recent studies have moved away from the clinic and into the community and the results with respect to ethnicity have suggested that biases are indeed operating.

Such a study of 4000 patients in a health maintenance organisation in Seattle contradicted the hospital-based studies—black patients accounted for 9.5% of CFS patients despite representing only 4% of the local population (Buchwald et al., 1995) . Another study which examined fatigue in office workers in California (Shefer et al., 1997) found increased levels of fatigue in Native Americans and Hispanics. Similarly, a cross-sectional telephone survey in San Francisco found increased numbers of African-Americans and Native Americans but lower numbers of Asians reporting CFS-like illness (Steele et al., 1998) . The most comprehensive epidemiological study to look at ethnic differences in CFS examined a multi-ethnic urban community sample in Chicago. This study aimed specifically to address the issue of under-representation of CFS in non-white groups (Jason et al., 1999). They found a higher prevalence of CFS in the Latino- and African-American ethnic groups than in whites, with the highest prevalence amongst Latinos. The authors suggest a number of reasons for this: psychosocial stress, behavioural risk factors, differences in seeking and accessing health care, differences in housing or unemployment and the particulars of how distress is expressed in different ethnic groups (Jason et al., 1999). Interestingly, a further analysis of this data revealed that ethnicity alone was not sufficient to explain differences in fatigue severity (Song, Jason, & Taylor, 1999). The severity of fatigue within the Chicago Latino population was very dependent upon other sociodemographic variables (age, gender, socioeconomic status) which appear to interact with ethnicity in a complex fashion.

These more recent epidemiological findings therefore contradict lay theories which argue that CFS is uncommon amongst ethnic minorities. The overall picture with respect to race appears to be highly variable and more dependant upon the specific ethnic minority in question rather than environmental or infectious exposures.

This variability of CFS within ethnic groups highlights the danger of over-simplifying racial categories to white/non-white or black. Racial categorisation varied widely between the epidemiological studies examined. Several studies collected data on race but did not present it in the results or discussion. A study of 202 nurses (Jason et al., 1998) used racial categories solely to compare demographics between nurses with and without CFS-like illnesses. Similarly, Fukuda et al. (1997) and Price, North, Wessely, and Fraser (1992) present the racial demographics of their samples but without further comment or evaluation. A far greater number of papers did not collect data on race despite, invariably, employing some form of categorisation for social class or gender (Ho-Yen, 1988; Lloyd, Hickie, Boughton, Spencer, & Wakefield, 1990; Bazelmans et al., 1997; Bates et al., 1993; Buchwald, Sullivan, & Komaroff, 1987; Chester, 1997, Euba et al., 1996; Lawrie & Pelosi, 1995; Lawrie et al., 1997; Minowa et al., 1996; Wessely et al., 1997).

We further investigated the use of racial categories by re-examining 121 reviews on CFS from the sample of review papers described by Joyce, Rabe-Hesketh, and Wessely (1998). We found that only 6% of these review papers mentioned race at all, whilst only 13% of a further sample of 221 original data-based papers included ethnicity as a demographic variable.[1] Given that most research is done by clinicians, it may be possible to generalise from this about the perception of CFS by clinicians. Further evidence is provided by a recent qualitative study of non-white CFS patients (Samec Trust, 2000). In this study ethnic minority patients report that doctors are more likely to diagnose 'black illnesses' before thinking of CFS. This study also points out that patient information and support groups tend to be Eurocentric in nature leading to further exclusion of ethnic minorities once the diagnosis of CFS is made.

In summary, the dramatic under-representation of ethnic minorities in our CFS clinic is more likely to represent a combination of diagnostic and referral bias by clinicians and selection bias from health-seeking behaviour rather than any lack of vulnerability to CFS. Just as the myth of the 'happy savage' contributed to under-reporting of depression in the African populations until the 1950s (Bhugra, 1996), so is there now evidence of race thinking in the literature on CFS. The situation is analogous to the early formulations of CF/ME as 'yuppie flu' leading many commentators to speculate on what aspect of upper class professional lifestyles made people vulnerable to the condition, until later epidemiological research suggested that a variety of biases were more likely explanations (Wessely, Hotopf, & Sharpe, 1998c, Chapter 15).

Conclusion

This paper aimed to show how the 19th century racial thinking intrinsic to neurasthenia has developed over the last hundred years with CFS/ME. We present epidemiological evidence that the racial differences in CFS presentation to specialist referral clinics are likely to be

[1] This included papers from a completed systematic review of CFS immunology. Search strategies are detailed in this review (Lyall, Wessely, & Peakman, 2003) and are available on request.

the result of perceptions of CFS by doctors as well as patients, rather than any specific aspect of the illness. This perception appears to be shaped by both the lay- and professional literature. An examination of these literatures has revealed how they reinforce ideas about race that originated in the theories about neurasthenia a century ago. In particular, the 19th century belief that non-whites are protected from neurasthenia is clearly seen to re-emerge in the literature on CFS. Recent CFS research suggests, however, that the relationship between ethnicity and CFS is far more complex than the theories proposed in the lay literature.

We have not attempted a comprehensive review of the construction of CFS but chose to focus on specific areas of interest. The field of CFS and ethicity is rich with possibilities for further research, both quantitative and qualitative. In particular, broader community-based epidemiological studies focusing on distinct ethnic groups would be invaluable. Further detailed examination of the interaction of race and class is essential if we are to fully understand the biases inherent in the diagnosis of CFS. This paper hopes to be a starting point.

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