

Psychological effects of chemical weapons: a follow-up study of First World War veterans

E. Jones*, B. Everitt, S. Ironside, I. Palmer and S. Wessely

Institute of Psychiatry and King's Centre for Military Health Research, Weston Education Centre, London, UK

Background. Chemical weapons exercise an enduring and often powerful psychological effect. This had been recognized during the First World War when it was shown that the symptoms of stress mimicked those of mild exposure to gas. Debate about long-term effects followed the suggestion that gassing triggered latent tuberculosis.

Method. A random sample of 103 First World War servicemen awarded a war pension for the effects of gas, but without evidence of chronic respiratory pathology, were subjected to cluster analysis using 25 common symptoms. The consistency of symptom reporting was also investigated across repeated follow-ups.

Results. Cluster analysis identified four groups: one ($n=56$) with a range of somatic symptoms, a second ($n=30$) with a focus on the respiratory system, a third ($n=12$) with a predominance of neuropsychiatric symptoms, and a fourth ($n=5$) with a narrow band of symptoms related to the throat and breathing difficulties. Veterans from the neuropsychiatric cluster had multiple diagnoses including neurasthenia and disordered action of the heart, and reported many more symptoms than those in the three somatic clusters.

Conclusions. Mild or intermittent respiratory disorders in the post-war period supported beliefs about the damaging effects of gas in the three somatic clusters. By contrast, the neuropsychiatric group did not report new respiratory illnesses. For this cluster, the experience of gassing in a context of extreme danger may have been responsible for the intensity of their symptoms, which showed no sign of diminution over the 12-year follow-up.

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Introduction

Because chemical weapons inspire fear disproportionate to their destructive power, they appeal to terrorists or militia groups conducting asymmetric warfare. Sarin attacks on civilians at Matsumoto City in 1994 and Tokyo in 1995, together with the gassing of Kurds in Halabja by Saddam Hussein in 1988, were designed to exploit such fears. However, the most significant use of chemical weapons remains the First World War, when they were used in increasingly large quantities by the major combatants. At the time, medical authorities were concerned by two questions, both of which have contemporary relevance: why did gas inspire such terror on the battlefield? And, to what extent were post-war symptoms in exposed veterans a product of these fears rather than the consequence of irreversible physical effects?

In a secret report compiled at the end of the First World War, Lt Colonel C. G. Douglas, a specialist

advisor in the physiological effects of chemical weapons, concluded that 'the particular value of the poison [mustard gas] is to be found in its remarkable casualty producing power as opposed to its killing power' (Douglas, 1919, p. 30). Although only 1% of British troops died as a result of chemical weapons during the conflict, an estimated 181 000 gas casualties were admitted to military hospitals (Douglas, 1918a; Haber, 1986; Spiers, 1986). At first, their symptoms were categorized as 'respiratory, gastro-intestinal and nervous', but following the introduction of phosgene in December 1915, a fourth group, 'cardiac' (rapid heartbeat and chest pain), was added (Anon, 1917, p. 527). Douglas observed that 'some form of bronchitis and cough' were among the most common acute symptoms of a non-lethal exposure to mustard gas (Douglas, 1917, p. 2). Meakins & Walker (1918) identified shortness of breath, cough, tachycardia and chest pain as residual symptoms.

Contemporaries were divided as to the cause of enduring symptoms. Some thought that both choking and vesicant gases produced a deficiency of oxygen in the blood, which in turn created a syndrome similar to disordered action of the heart (DAH) (Anon, 1918a,

* Address for correspondence: Professor E. Jones, Institute of Psychiatry and King's Centre for Military Health Research, Weston Education Centre, 10 Cutcombe Road, London SE5 9RJ, UK.
(Email: edgar.jones@iop.kcl.ac.uk)

p. 138). Others believed that symptoms were functional in origin, classifying such cases as 'gas neurosis' because of their similarity to DAH, 'but with a predominance of respiratory symptoms' (Ireland, 1926, p. 837; Abrahams, 1922). Functional photophobia, aphonia, hysterical cough, vomiting and palpitations were identified by Colonel A. B. Soltau, consultant physician in France for gassed cases, as a consequence of exposure to mustard gas (Southborough, 1922, p. 74). Hulbert (1920) thought 'gas neurosis' akin to shell-shock of a non-concussive type because the severity of symptoms bore no relation to 'the amount of gas inhaled' but arose 'in proportion to the individual's mental and emotional make-up and instability'.

In 1928, Colonel H. L. Gilchrist, physician to the US Army Chemical Warfare Service, asked the key question 'to what extent gas should be held responsible for the great train of symptoms of which so many ex-soldiers complain?' (1928, p. 31). As early as 1918, two French military physicians, Marcel Pinard and M. Rendu, had hypothesized that gassing triggered 'latent tuberculosis, which might otherwise have remained dormant' (Anon, 1918*b*, p. 272; Anon, 1918*c*, p. 852). However, post-war studies suggested that the relationship between gas poisoning and subsequent respiratory disorder was far from clear-cut. In 1920, somewhat surprisingly, a report from the US Surgeon General found that the incidence of tuberculosis among troops who had been gassed was almost half that of soldiers who had not been exposed to chemical weapons (Anon, 1920, p. 103). A 4-year follow-up study of 700 UK servicemen exposed to chlorine in 1915 found that less than 5% had bronchitis and associated pulmonary conditions, while a study of 4575 mustard-gas cases discharged from UK hospitals showed that only 0.3% had been invalidated from the forces as permanently unfit for duty (Meakins & Priestley, 1918). Although many physicians believed that pulmonary tuberculosis was a common consequence of gassing (Vallow, 1922), Sandall found that only one veteran had any evidence of the disease in a sample of 83 pensioners with an award for the effects of gas, although 38 cases (46%) showed evidence of emphysema or bronchitis (Sandall, 1922). A 14-year follow-up study of 89 US servicemen exposed to mustard gas found that 62 (70%) had no organic pathology. Potential confounders, such as smoking, were not explored in the cases of the 27 with bronchitis, asthma or emphysema (Gilchrist & Matz, 1933). These findings ran counter to popular perception that the effects of poison gas were permanent and therefore likely to be the primary cause of any subsequent respiratory problems.

Using war pension files, this study explores enduring symptoms of veterans exposed to mild gassing.

Evidence from post-war medical boards may provide clues as to why beliefs about chemical weapons became so entrenched and influential. Was it that veterans suffered from chronic respiratory disorders, or were the psychological effects of gas more than merely transient? This investigation tests the null hypothesis that, over the medium and long term, symptom reporting was not consistent or even focused on the respiratory system.

Method

The 7800 First World War case files held by the War Pensions Agency provided a random sample of British veterans who had been exposed to gas. The records relate to all diagnoses and referred to the longest-lived veterans. The files are, in fact, the only source of detailed follow-up data for gassed servicemen in the UK. The case-notes contain service records, details of treatment received while in the forces and the findings of subsequent medical investigations, and are ordered by date of application within the 13 administrative regions created by the Ministry of Pensions in June 1919. By taking consecutive files for 'effects of gas' from each of the regions in proportion to the denominator, a random sample of 103 subjects was extracted. Although not a representative gassed population, the records for these veterans provided an extended period of assessment and included death certificates. Regular medical boards held over periods of up to 60 years enabled us to assess whether these pensioners had suffered severe or lasting harm from chemical weapons.

Veterans with severe respiratory illness were excluded from the study to focus on those whose ideas and beliefs were not grounded in objective pathology directly related to the war. Pensioners who had occasional or mild episodes of bronchitis were included in the study because these were recovering conditions that may have been related to other effects such as smoking, industrial pollution or poor housing. Because the search exercise was wide-ranging, the 103 cases in the sample constitute almost all of the gassed cases in the archive who did not suffer from long-term or severe respiratory illness. Repeated follow-ups during the interwar period enabled us to exclude veterans who suffered from tuberculosis, chronic bronchitis or emphysema associated with gassing. A total of 23 pensioners were rejected for this reason or because it was clear from hospital records that the soldier's account of having been gassed was false.

Using a standard form, biographical and military details were recorded, together with 94 possible symptoms. Casualty cards and hospital records were used to verify exposure to chemical weapons. The 25

most common symptoms were identified and used as the basis of the cluster analysis. Because terms, both diagnostic and symptom, may have changed their meaning subtly over the 90 years since the end of the First World War, no attempt was made to reclassify them according to modern criteria.

Cluster analysis is a generic term for a wide range of numerical techniques that try to discover homogeneous groups or clusters of individuals in a sample of data. There are many such techniques available and most are described in Everitt *et al.* (2001). In this study we used a model-based method, suggested by Banfield & Raftery (1993) and extended by Fraley & Raftery (1999, 2002). This statistically respectable method has the advantage of an associated test for number of clusters known as the Bayesian Information Criterion (BIC) (see Fraley & Raftery, 1999). The chosen clustering technique (which is implemented in the MCLUST package in R) was applied to the first 10 principal component scores derived from a principal components analysis of the original 25 symptoms. These 10 components accounted for over 70% of the variation in the data set.

Results

Pensioners

The mean age of the sample was 82 years with a range from 54 to 102 years. They were a particularly long-lived and healthy group. Only three of the 103 were not from combat units, principally infantry and artillery. Most of the sample was composed of volunteers (62.1%), although 25.2% were conscripts. Only 13 (12.7%) were regular soldiers or reservists. As regards rank, the distribution of the sample followed the organization of troops on the ground: one officer (1%), two warrant officers (1.9%), five sergeants (4.9%), 14 corporals (13.6%) and 81 private soldiers (78.6%). Typically, an infantry platoon, commanded by a junior officer, consisted of 50 to 60 men, commonly in a ratio of one non-commissioned officer (NCO) to 10 privates, which suggests that NCOs (20%) are slightly over-represented. As experienced soldiers they ought to have been proficient in taking anti-gas measures. Because they had to check that men under their command had put on their respirators correctly, NCOs may have neglected their own protection. It was also their responsibility to assess the nature of the threat and to judge when gas had dispersed and it was safe to remove masks.

Cluster groups

The BIC selected a four-group solution as optimal. The largest group ($n=56$), defined by respiratory and

cardiovascular symptoms, we labelled as the somatic cluster. Prominent were persistent cough, shortness of breath, chest pain, irregular heartbeat and difficulty undertaking tasks (Table 1). With the exception of headache, anxiety and dizziness, neuropsychiatric symptoms (such as repeated fears, jumpiness and depression) were largely, though not entirely, absent. The cluster revealed an emphasis on the respiratory and cardiovascular systems.

The second group ($n=30$) was dominated to a far greater extent by respiratory symptoms, principally cough, shortness of breath and chest pain. This was labelled the respiratory cluster. When expressed as a proportion of all reports over the period 1919 to 1931, the three accounted for 68% of the total. Less prominent were: heavy sweating, difficulty performing tasks, fatigue, sleep problems, shortness of breath, chest pain, dizziness, tightness of chest and headaches. Almost non-existent were: anxiety, repeated fears, depression and jumpiness.

Cluster three ($n=12$) stands apart from the other groups in that psychological symptoms predominate. Persistent anxiety, repeated fears, sleep difficulties, dizziness and tremor were characteristic of this group. Veterans in this neuropsychiatric group also reported many more symptoms (41 per subject) than the other three groups (23, 16 and 17 respectively).

Group four members ($n=5$) have a tight cluster of symptoms related to the throat and breathing difficulties. They are differentiated from the somatic and respiratory clusters by having difficulty producing sounds and suffering from persistent sore throats. Neuropsychiatric symptoms hardly feature in this group.

The composition of the four groups did not meet predictions. We expected to find a single homogeneous group united by the powerful experience of having been gassed. It was hypothesized that pensioners would exhibit a narrow pattern of symptoms based around the respiratory system. This was not confirmed.

Post-war effects

A detailed survey of the subjects' medical histories showed that 31 (55%) of the 56 veterans who fell in the somatic cluster and 19 (63%) of the respiratory cluster had reported an acute respiratory infection in the immediate post-war period. All five (100%) in the throat cluster had reported a post-war illness of this nature. By contrast, no one from the neuropsychiatric group reported a new respiratory disorder. Given that upper respiratory tract infections (URTIs) are common throughout the population, this implies that veterans in the somatic and respiratory clusters ascribed greater

Table 1. Symptoms by subjects

Symptoms	Cluster 1 (n = 56)	Cluster 2 (n = 30)	Cluster 3 (n = 12)	Cluster 4 (n = 5)
Persistent cough	235 (84)	145 (100)	21 (42)	25 (100)
Shortness of breath	194 (88)	125 (97)	26 (67)	16 (100)
Chest pain	110 (63)	50 (77)	31 (75)	9 (80)
Rapid or irregular heartbeat	80 (44)	26 (57)	27 (75)	0
Difficulty undertaking tasks	69 (73)	13 (27)	20 (75)	1 (20)
Persistent anxiety	33 (34)	1 (3)	60 (100)	0
Tremor	33 (30)	4 (10)	56 (100)	0
Dizziness	53 (46)	11 (27)	24 (92)	0
Headaches	39 (34)	11 (23)	33 (83)	1 (20)
Heavy sweating	42 (23)	24 (33)	17 (67)	0
Fatigue	51 (54)	6 (13)	20 (67)	1 (20)
Weakness	50 (48)	12 (3)	11 (67)	0
Sleep difficulties	29 (30)	3 (10)	37 (92)	0
Tightness of chest	37 (21)	22 (30)	1 (8)	2 (40)
Exhaustion	42 (39)	3 (7)	16 (58)	0
Vomiting	48 (39)	8 (13)	2 (17)	0
Repeated fears	7 (9)	0	37 (100)	0
Difficulty producing sounds	15 (14)	0	6 (25)	18 (100)
Stomach pain	29 (21)	2 (7)	3 (25)	0
Sore throat	19 (13)	2 (7)	2 (8)	9 (100)
Back pain	24 (30)	0	3 (17)	2 (20)
Depression	5 (7)	0	23 (83)	0
Tenderness	19 (23)	1 (3)	1 (8)	0
Itchy or painful eyes	19 (16)	0	2 (8)	0
Jumpiness	4 (5)	1 (3)	15 (83)	0
Total	1286	470	494	84

Figures in parentheses indicate the percentage in each cluster who reported the symptom. The totals include multiple reports of the same symptom by subjects.

significance to these illnesses than did those in the neuropsychiatric cluster. Barsky *et al.* (1988) and Lane *et al.* (1988) have proposed that somatic symptoms can be influenced in two ways: by amplification or by dysphoria (a range of negative emotions including guilt, depression and anxiety). Amplification in a context of URTIs, they proposed, influenced the report of localized, rather than systemic, symptoms whereas dysphoria made people feel sicker in a global and non-specific way. It is possible, therefore, that veterans in the neuropsychiatric cluster (characterized by high scores for both depression and anxiety) experienced a general feeling of ill health and that those in the three somatic clusters tended to amplify specific symptoms.

Co-morbidity

Of the 12 First World War veterans in the neuropsychiatric cluster, all had a secondary diagnosis of shell-shock, neurasthenia, DAH, or other functional

somatic disorder. By comparison, the other three clusters had significantly lower levels of co-morbidity: 38% in group 1, 0.7% in group 2 and 0% in group 4. Contemporaries had struggled to define the residual effects of gassing. Soltau, a military specialist who examined the files of 150 gas pensioners, concluded that 30% reported a range of symptoms that could equally well meet the criteria for neurasthenia or shell-shock, while a further 25% could be reclassified as DAH, formerly known as soldier's heart (Elliott & Soltau, 1923). In this context, all of these diagnoses referred to a stress-related disorder.

Symptom reporting over time

Because veterans were followed up regularly to assess their pension status, consistency of symptom reporting could be tested over the 12 years to 1931 when the Ministry of Pensions reduced the frequency of medical boards. Changes in the reporting of symptoms were

plotted but because of annual variations few statistically significant trends were identified. For the neuropsychiatric cluster, repeated fears were reported more often over time (rising from 4.4% of total symptoms reported in 1919–21 to 12.7% in 1928–31), as was difficulty completing tasks (rising from 1.2% in 1919–21 to 12.7% in 1928–31), while the incidence of tremor fell (from 13.7% in 1919–21 to 2.8% in 1928–31). The relationship between reporting and time was examined further for these three symptoms using a simple linear correlation and the results suggested an association rather than a causal relationship for repeated fears ($r^2=0.77$), difficulty completing tasks ($r^2=0.87$) and incidence of tremor ($r^2=0.7$).

Discussion

Military context

Associated with 'violent irritant or choking sensations' (Douglas, 1923, p. 292), chemical weapons were designed to inspire terror. Some, such as phosgene, were difficult to detect, having little odour or colour. To distinguish between the unpleasant (tear gas and the chlorarsines) and the lethal (chlorine, phosgene and mustard gas) took nerve and training. Indeed, chlorarsines, used at the very end of the war, caused short-term but intense respiratory distress sufficient in some cases 'to cause a man to lose his mental control' (Douglas, 1918*b*, p. 1). Soltau argued that gas frightened soldiers because 'there is nothing more liable to cause panic than the idea of being choked' (Southborough, 1922, p. 73), an understandable observation. It was scarcely surprising that the use of chemical agents during the First World War led to misinterpretation. Exposed to a variety of terrifying explosions and smells, infantrymen in the trenches sometimes mistakenly concluded that they had been gassed, developing coughs, shortness of breath, chest pain and palpitations. In such cases, medical inquiries showed that these symptoms could not be explained by the presence of toxins and these servicemen were dismissed as cases of 'war neurosis'.

A diagnostic problem encountered by military physicians was that the symptoms of anxiety often mimicked the physical effects of mild exposure to gas (Palmer, 2004), a similarity sometimes exploited by front-line troops seeking a medical route from the battlefield (Ireland, 1926). In part, problems of classification arose because medical science struggled to keep pace with the rapid development of chemical warfare (Herringham, 1920). New gases were introduced to circumvent protective measures but also to take soldiers and their doctors by surprise, thereby maintaining their terrifying reputation. Every time a

novel agent was used, urgent study was needed to identify its type, properties and what could be done to neutralize its effects. The dissemination of technical information among a vast army proved problematic given only rudimentary communications. As a result, as W. J. Forshaw based at No. 2 Australian General Hospital observed, 'many regimental medical officers have no knowledge of the after effects and receive no information and scanty supplies of material for treatment' (Forshaw, 1918). Left in relative ignorance, Douglas concluded, 'I really believe that nearly all medical officers are terrified of the mere mention of gas poisoning' (Douglas, 1918*c*). Consequently, doctors often practised defensive medicine and referred patients to base hospitals whether or not this caution was indicated.

Contemporary relevance

Despite international conventions, chemical weapons have continued to be used on the battlefield, although the major combatants refrained from their deployment during the Second World War possibly because of fear of retaliation. During the Iraq–Iran War, however, Saddam Hussein used mustard gas against the Kurds (Hay & Roberts, 1990). In the 1991 Gulf War, when Coalition forces sought to restore independence to Kuwait, this earlier willingness to use chemical weapons raised the level of threat. As a result, considerable attention was given to monitoring in the field, while personnel were required to take medical countermeasures against possible attacks.

In the event, it seems that Iraqi forces did not use their chemical arsenal during the conflict. However, in March 1991, shortly after the ceasefire, US forces destroyed an ammunition dump at Khamisiyah, which included rockets containing the nerve agent sarin. This led some to suggest that so-called 'Gulf War syndrome' was in fact an expression of chemical exposure (Haley & Kurt, 1997). Although there is little or no scientific evidence to suggest that Coalition forces were exposed to damaging levels of toxins during their deployment (US Department of Defense, 2000; Riddle *et al.* 2003; Brown, 2006), 100 000 US servicemen were formally notified by the government that they 'might' have been exposed to sarin from the Khamisiyah plume. As in the First World War, fear and uncertainty helped to shape both beliefs and symptoms.

In 2006, it was reported that 64% of a sample of 335 US veterans of the Gulf War believed that they had been subjected to biological or chemical weapons compared with 6% of 269 service controls who had not been deployed to the conflict (Brewer *et al.* 2006). In support of this assertion, servicemen cited the experience of an alert while in theatre and enduring

physical symptoms. Furthermore, a study of 2246 US Gulf War veterans who believed that they had been exposed to nerve or mustard gas found that they had also reported more symptoms while serving in the Gulf, were more likely to be diagnosed with a mental disorder and reported poorer current health status (Stuart *et al.* 2003). In other words, those Gulf War veterans who believe that they had been exposed to chemical weapons were currently more likely to be symptomatic and disabled than colleagues who did not hold these convictions (Riddle *et al.* 2003; Stuart *et al.* 2003).

In addition, psychological distress prospectively increases the recall of traumatic events and hazards over time. A study of Gulf War veterans showed that a worsening health perception increased the likelihood of reported adverse exposures (Wessely *et al.* 2003).

Most recently, the publication of data from a small, non-random neuroimaging study of US veterans who had been within the Khamisiyah plume reignited the public debate about the long-term effects of poison gas (Heaton *et al.* 2007). It raised the question of whether medically unexplained symptoms observed in Gulf War veterans were psychological in origin or the product of neurological changes caused by toxins, a debate familiar to the First World War generation of physicians. At one stage, for example, Mott had hypothesized that shell-shock was caused by toxins, such as carbon monoxide, released by the incomplete detonation of ordnance (Mott, 1919, p. 228).

Beliefs and symptoms

Studies of US Gulf War veterans who believe that they have been gassed resonate with our group of war pensioners for whom evidence of exposure to chemical weapons is far stronger. For those in the three somatic clusters the experience of being gassed coupled with URTIs contracted after demobilization served to maintain a broad range of symptoms throughout their long lives. It appears that respiratory infection acquired in peacetime may have reinforced any ideas about the potency of gassing rather than providing a model of healing and recovery. Data from the Gulf War suggest that pensioners in the neuropsychiatric cluster, who reported many symptoms of distress, were likely to have heightened sensitivity to memories of being gassed or having experienced chemical warfare threats and alarms.

Statements provided by gassed veterans in support of their pension claim revealed that most believed they had suffered irreversible physical damage (Jones *et al.* 2007). For example, one subject wrote in April 1924, 'symptoms began after gassing ... since then my disability has gradually increased. Now I am unable to follow any occupation whatever', while another

commented in March 1930 that 'I have been getting gradually worse every year ... my system is full of gas'. The tendency to interpret subsequent illnesses and symptoms in terms of gas exposure was recognized by Gilchrist: 'the blame for every conceivable sort of ailment has been placed on gas; in fact, there is scarcely a functioning organ of the body whose disturbed action either during or since its participation in [the war] has had the blame for its erratic performance laid to the door of poison gas' (1928, p. 31). It appears, therefore, that both events and symptoms determine attribution and that new ailments can reinforce existing interpretations, whether or not actual organic damage had occurred.

Limitations

The study is limited by the relatively small sample. However, the destruction or loss of records has resulted in this being the largest group of gassed veterans for which follow-up data were available. Although we had 3084 reported symptoms, the small number of subjects may restrict the extent to which the results can be generalized.

Cultural shift

Gas in the form of chlorine inspired terror from its first use at Ypres in April 1915. The introduction of more deadly phosgene in December 1915 and the switch to mustard gas in July 1917 maintained an element of surprise and uncertainty. Although the capacity of chemical weapons to frighten has scarcely diminished over the past 90 years, popular conceptions have evolved (Fullerton & Ursano, 1990). At the beginning of the twentieth century, chemicals in general were not viewed with the suspiciousness that they engender today. Indeed, food delivered to the trenches was sometimes labelled 'chemically treated' as a sign of safety and freedom from bacteriological contamination. Such a description no longer inspires confidence in contemporary society characterized as 'chemophobic' and in which concerns about the toxic effects of chemicals are not restricted to weapons (Durodié, 2003). Thus, the psychological impact of gas may be greater in our current culture than during the First World War, and for that reason chemical weapons retain a potent appeal for modern terrorism (Alexander & Klein, 2003).

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References

- Abrahams A** (1922). The later effects of gas-poisoning. *Lancet* **2**, 933–934.
- Alexander DA, Klein S** (2003). Biochemical terrorism: too awful to contemplate, too serious to ignore. *British Journal of Psychiatry* **183**, 495–496.
- Anon** (1917). Cardiac disorders due to the new poisonous gases. *British Medical Journal* **2**, 527.
- Anon** (1918a). War gas poisoning. *British Medical Journal* **2**, 138–139.
- Anon** (1918b). Poison gas and tuberculosis. *Lancet* **1**, 272.
- Anon** (1918c). The effects of asphyxiating gas. *Lancet* **1**, 852.
- Anon** (1920). *Report of the Surgeon General of the Army*. US Government Printing Office: Washington, DC.
- Banfield JD, Raftery AE** (1993). Model-based Gaussian and non-Gaussian clustering. *Biometrics* **49**, 803–821.
- Barsky AJ, Goodson JD, Lane RS, Cleary PD** (1988). The amplification of somatic symptoms. *Psychosomatic Medicine* **50**, 510–519.
- Brewer NT, Lillie SE, Hallman WK** (2006). Why people believe they were exposed to biological or chemical warfare: a survey of Gulf War veterans. *Risk Analysis* **26**, 337–345.
- Brown M** (2006). Toxicological assessments of Gulf War veterans. *Philosophical Transactions of the Royal Society. Series B, Biological Sciences* **361**, 649–680.
- Douglas CG** (1917). Effects of gas shell bombardment [The National Archives (hereafter TNA), WO32/5176, 26 July 1917].
- Douglas CG** (1918a). Casualties caused in British forces by gas warfare (TNA, WO142/1090).
- Douglas CG** (1918b). Effects produced in the field by shell containing chlorarsines (TNA, WO142/108, 22 October 1918).
- Douglas CG** (1918c). Memo to Edkins (TNA, WO142/104, 14 April 1918).
- Douglas CG** (1919). Note on total casualties caused in the British forces by gas warfare, (TNA, WO142/100, 17 January 1919).
- Douglas CG** (1923). Development of gas warfare. In *History of the Great War based on Official Documents. Medical Services: Diseases of the War*, vol. 1 (ed. W. G. Macpherson, W. P. Herringham, T. R. Elliott and A. Balfour), pp. 271–310. HMSO: London.
- Durodié B** (2003). The true cost of precautionary chemicals regulation. *Risk Analysis* **23**, 389–398.
- Elliott TR, Soltau AB** (1923). Invalidism from gas poisoning. In *History of the Great War based on Official Documents. Medical Services: Diseases of the War*, vol. 1 (ed. W. G. Macpherson, W. P. Herringham, T. R. Elliott and A. Balfour), pp. 517–525. HMSO: London.
- Everitt BS, Landau S, Leese M** (2001). *Cluster Analysis*. Arnold: London.
- Forshaw WJ** (1918). Some remarks on mustard gas (TNA, WO142/107, p. 2).
- Fraley G, Raftery AE** (1999). MCLUST: software for model-based cluster analysis. *Journal of Classification* **16**, 297–306.
- Fraley G, Raftery AE** (2002). Model-based clustering, discriminant analysis and density estimation. *Journal of the American Statistical Association* **97**, 611–631.
- Fullerton CS, Ursano RJ** (1990). Behavioural and psychological responses to chemical and biological warfare. *Military Medicine* **155**, 54–59.
- Gilchrist HL** (1928). *A Comparative Study of World War One Casualties from Gas and other Weapons*. US Government Printing Office: Washington, DC.
- Gilchrist HL, Matz PB** (1933). *The Residual Effects of Warfare Gasses*. US Government Printing Office: Washington, DC.
- Haber LF** (1986). *The Poisonous Cloud: Chemical Warfare in the First World War*. Clarendon Press: Oxford.
- Haley RW, Kurt TL** (1997). Self-reported exposure to neurotoxic chemical combinations in the Gulf War. A cross-sectional epidemiological study. *Journal of the American Medical Association* **277**, 231–237.
- Hay A, Roberts G** (1990). The use of poison gas against the Iraqi Kurds: analysis of bomb fragments, soil and wool. *Journal of the American Medical Association* **263**, 1065–1066.
- Heaton KJ, Palumbo CL, Proctor SP, Killiany RJ, Yurgelun-Todd DA, White RF** (2007). Quantitative magnetic resonance imaging in US Army veterans of the 1991 Gulf War potentially exposed to sarin and cyclosarin. *Journal of NeuroToxicology* **28**, 460–470.
- Herringham WP** (1920). Gas poisoning. *Lancet* **1**, 423–424.
- Hulbert HS** (1920). Gas neurosis syndrome. *American Journal of Insanity* **77**, 213–216.
- Ireland MW** (1926). *The Medical Department of the US Army in the World War, Volume XIV, Medical Aspects of Gas Warfare*. Government Printing Office: Washington, DC.
- Jones E, Palmer I, Wessely S** (2007). Enduring beliefs about the effects of gassing in war: qualitative study. *British Medical Journal* **335**, 1313–1315.
- Lane RS, Barsky AJ, Goodson JD** (1988). Discomfort and disability in upper respiratory tract infection. *Journal of General Internal Medicine* **3**, 540–546.
- Meakins JC, Priestley JG** (1918). Report on the length of stay in hospital in the UK and disposal of gas casualties. MRC Chemical Warfare Medical Committee Report No. 16. HMSO: London.
- Meakins JC, Walker TM** (1918). Changes observed in the heart and circulation and the after-effects of irritant gas poisoning. MRC Chemical Warfare Medical Committee Report No. 7. HMSO: London.
- Mott F** (1919). *War Neuroses and Shell Shock*. Henry Froude and Hodder & Stoughton: London.
- Palmer I** (2004). The psychological dimension of chemical, biological, radiological and nuclear (CBRN) terrorism. *Journal of the Royal Army Medical Corps* **150**, 3–9.
- Riddle JR, Brown M, Smith T, Ritchie E, Brix KA, Romano J** (2003). Chemical warfare and the Gulf War: a review of

- the impact on Gulf veterans' health. *Military Medicine* **168**, 606–613.
- Sandall TE** (1922). The later effects of gas poisoning. *Lancet* **2**, 857–859.
- Southborough** (1922). *Report of the War Office Committee of Inquiry into 'Shell-Shock'*. HMSO: London.
- Spiers EM** (1986). *Chemical Warfare*. Macmillan: London.
- Stuart JA, Ursano RJ, Fullerton CS, Norwood AE, Murray K** (2003). Belief in exposure to terrorist agents: reported exposure to nerve or mustard gas by Gulf War veterans. *Journal of Nervous and Mental Disease* **191**, 431–436.
- US Department of Defense** (2000). *Special Oversight Board for Department of Defense Investigations of Gulf War Chemical and Biological Incidents, Final Report*. Presidential Advisory Committee on Gulf War Veterans Illnesses: Washington, DC.
- Vallow H** (1922). The later effects of gas poisoning. *Lancet* **2**, 985.
- Wessely S, Unwin C, Hotopf M, Hull L, Ismail K, Nicolaou V, David A** (2003). Stability of recall of military hazards over time. Evidence from the Persian Gulf War of 1991. *British Journal of Psychiatry* **183**, 314–322.