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**Project:** Somatisation disorders in people with childhood sexual abuse

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**Introduction:**

When patients develop medical symptoms that are thought to have a psychological or emotional cause, a diagnosis of 'somatisation disorder' is made. Somatisation is very common, and is thought to account for more than 20% of referrals to neurology clinics, for example. It has been shown that patients who have somatisation disorder are more likely to recall childhood sexual abuse (CSA) than those with comparable symptoms with an identified medical cause. Multiple symptomatology and greater healthcare utilisation have also been associated with CSA. However, the literature lacks a population-based study of somatisation and healthcare utilisation in patients with CSA.

**Aim:**

To improve our understanding of the relationship between CSA and somatisation and to determine healthcare utilisation in a cohort of patients with CSA, comparing with a cohort of patients without CSA.

**Method:**

The Christchurch Health and Development Study (CHDS) is a longitudinal study of a birth cohort of 1265 children born in Christchurch in mid-1977. This covered 97% of all births at this time in Christchurch. This cohort has now been studied on 23 occasions from birth, most recently in 2012 at the age of 35. Over the course of the CHDS, the study has produced over 400 scientific papers, books and reports and has been cited in peer-reviewed literature over 15,000 times. Retrospective reports of exposure to childhood sexual abuse prior to age 16 were obtained from cohort members at ages 18 and 21 years. Participants were classified into one of four exposure groups reflecting the extent/severity of CSA reports. These groups were: (a) no sexual abuse (b) non-contact sexual abuse only (involving indecent exposure, public masturbation or unwanted sexual propositions); (c) contact sexual abuse not involving attempted or completed sexual penetration; (d) attempted or completed sexual penetration including vaginal, oral and anal intercourse. For simplicity we grouped (b) and (c) together so we could compare severe CSA with less-severe CSA, and no CSA. For reasons of availability of electronic medical records, we sampled the period from 1st January 2008 until 31st December 2015. We had permission from 408 CHDS study participants – who have resided in the CDHB catchment area for the study period - to access their medical records and carry out an exploratory study. 9.1% of this sample had severe CSA (37 people); 8.8% had less-severe CSA (36 people); 82.1% had no CSA (335 people). The 'medically unexplained symptoms (MUS)' paradigm has been used to study somatisation in large populations. For the purposes of this study, MUS required either: having at least two presentations within the study period to secondary care for medical symptoms not explained by tests or examination, or, by receiving

a diagnosis of MUS (fibromyalgia, pain syndrome, IBS, etc.) from a secondary care physician. We recorded all secondary care medical contacts within the study period, including clinical investigations, laboratory tests, and radiological investigations for each of the 408 participants. Psychological and obstetric encounters were excluded.

### **Results:**

The sample was rated for likelihood of MUS by two investigators. The first investigator excluded those people who had no potential MUS. The second investigator then independently rated the 61 remaining people. The first 'iteration' revealed 21 'probable' MUS cases and 9 'possible' cases. The final iteration revealed 23 cases of MUS, or 5.6% of our total sample. The data was analysed using SPSS (a statistics program) together with the CHDS CSA rating data. Those who reported exposure to severe CSA were 8.65 times more likely to have MUS than those with no CSA. This relationship was highly significant (95% CI = 3.36-22.30  $p = 0.000$ ). Those with severe CSA were 5.46 times more likely to develop MUS than those exposed to less-severe CSA (95% CI = 1.09 – 27.39  $p = 0.039$ ). 24.3% of those exposed to severe CSA developed MUS (9/37). Conversely, 39% of patients with MUS had been exposed to severe CSA (9/23). People with severe CSA had an average of 20.27 more laboratory tests ( $p = 0.008$ ), 3.72 more radiological investigations ( $p = 0.003$ ), and 0.504 more clinical investigations ( $p = 0.021$ ) than those with no CSA, over the eight year study period. People with less-severe CSA had an average of 17.27 more lab tests than those with no CSA ( $p = 0.020$ ). People with severe CSA had an average of 7.72 more hospital encounters than those with no CSA (95% CI 0.85-14.47,  $p = 0.028$ ).

### **Conclusion:**

Our results suggest that a report of severe CSA is associated with a 24% chance of having MUS. Conversely, a patient with MUS has a 39% chance of having reported CSA. CSA was associated with more frequent hospital visits, tests and investigations. CSA is likely to be implicated in the development of MUS. The correlation between severe CSA and MUS is unusually strong for a psychosocial variable. It should be emphasised that because we only studied secondary care contact (which excludes GP visits), within a study period of 8 years, our findings may underestimate the strength of the relationship. This project has important clinical implications, in confirming and quantifying a strong relationship between CSA and MUS. This will help guide doctors in determining appropriate investigation and management of their patients. The research implications are also wide. Not all patients who report CSA have MUS and not all people with MUS have CSA: studying differences between subgroups with and without CSA and MUS has potential to provide important insight into how MUS and somatisation develop.