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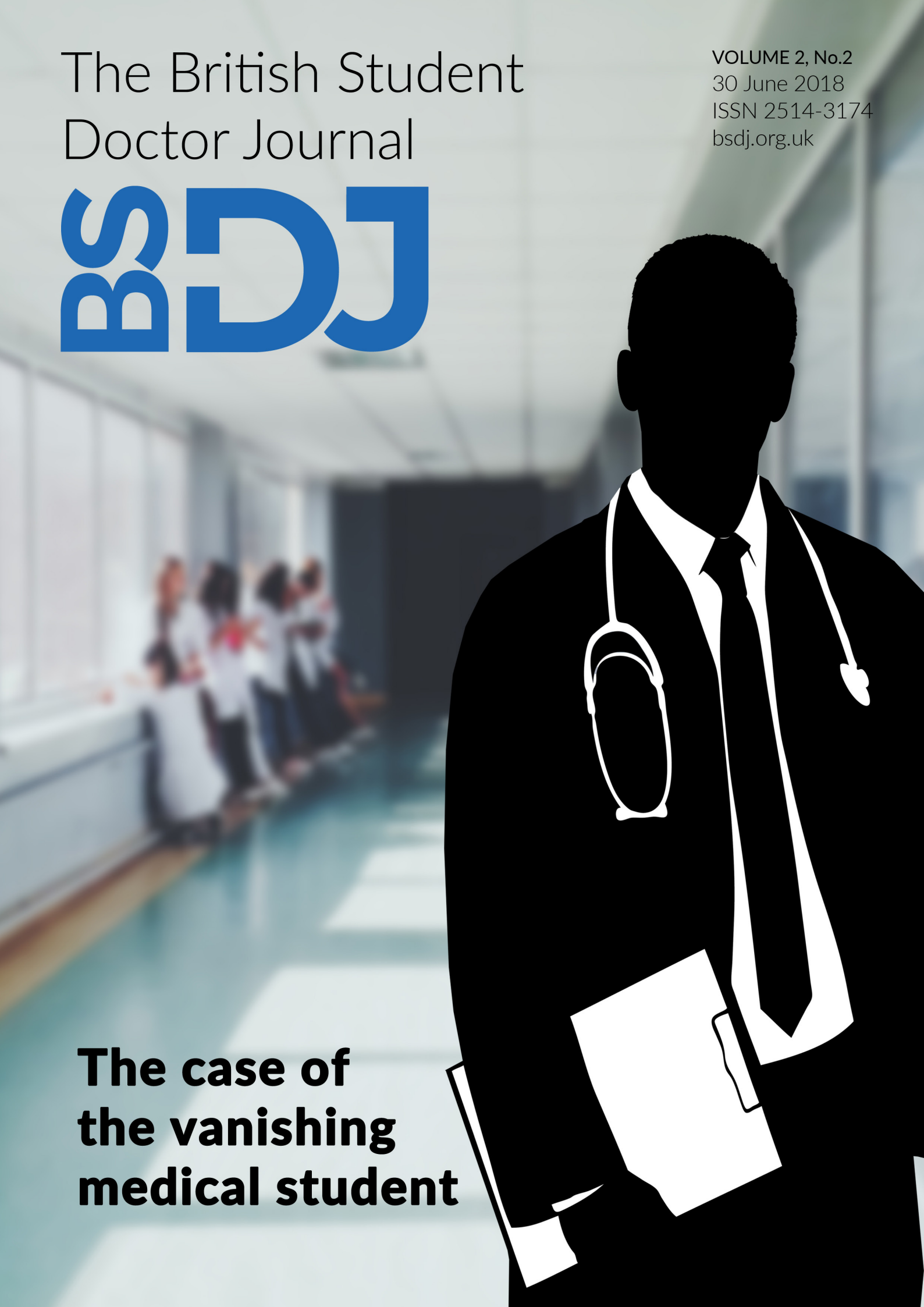

The case of the vanishing medical student

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Editorial

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Welcome to the fourth issue of *The British Student Doctor*.

In this issue, we feature '*The case of the vanishing medical student*' by Henry Dunn, a medical student at King's College London, and his colleagues Thomas Taylor and Amanda Bacon, medical students at the University of Oxford. In this reflective piece, they discuss how medical students are failing to take full opportunity of clinical placements. The authors believe that this is due to a number of factors, including the limited availability of bedside teaching, a culture of not wishing to overburden busy junior doctors, and a perception that there is little to gain from so called '*ward work*'.

As medical students, we also experienced the same challenges during our clinical attachments; being encouraged to study textbooks rather than to spend time on clinical placements or to '*take advantage*' of our free time whilst we could before facing the pressures of life in the NHS. Now as junior doctors at the end of our foundation year, reflecting back on the issues raised by Dunn et al., we understand these perceptions. However, we also realise with new insight the importance of using clinical practice to gain as much preparation as possible before the transition from medical student to junior doctor. Having passed through this phase recently, the change in responsibility that this passage entails cannot be underestimated.

Sir William Osler, a Canadian physician who founded John Hopkins Hospital in Baltimore, Maryland, and was the pioneer of bedside teaching, wrote in his book *Aequanimitas* in 1904:

"*He who studies medicine without books sails an uncharted sea, but he who studies medicine without patients does not go to sea at all.*"

When under pressure as junior doctors, often during the middle of the night on a busy ward cover shift, it is not the textbooks that we remember, but the patients that we have encountered as medical students on clinical attachments. These experiences serve as the guidance that we need to manage the most difficult of situations, and even to find solace in during the most stressful of times. However, we also realise that we now hold a responsibility to optimise the experience of medical students in our hospitals, to ensure that they do not feel undervalued or burdensome. It is also our responsibility to act as role models for the students that we teach, and stress the value of clinical attachments.

In this issue of *The British Student Doctor*, we also feature research by Andy Cheng and his colleagues on '*Analysing ethnic differences in compulsory admissions for psychiatric disorders in the UK*'. This pertinent work reveals worse outcomes for persons of ethnic minority backgrounds.

This issue also features a number of education articles, including '*Cannabis – current trends and methods of bioavailability*' by Sarah Gritis, '*The Ponseti method for the treatment of congenital talipes equinovarus*', '*Recent advances in the understanding of non-coding RNA*' by

Jernej Zorman and '*Skin picking disorders – what can we learn from such a topical issue?*' by George Johnson and colleagues.

Finally, we also publish an article by Luke Roberts and his colleagues at the Evelina London Children's Hospital on the topic of clinical coding. This article provides a rare insight into a process which is considered esoteric by most junior doctors, but is a process in which they play an important role, and is vital for NHS funding.

We hope that you enjoy reading this issue of *The British Student Doctor*. We would like to extend our gratitude to the medical students, junior doctors and faculty staff that have contributed to this issue of the journal. If any of the articles in this issue raise discussion, we would be pleased to consider a correspondence piece in response. Our author guidelines can be found on our website and offer all of the necessary guidance for submitting manuscripts for consideration for publication in our next issue.

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Original Research

Ethnic differences in compulsory admissions for psychiatric disorders in the UK: a systematic review and meta-analysis

ORIGINAL RESEARCH

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ABSTRACT

It is postulated by Mann et al. that some ethnic minorities in the United Kingdom (such as Asian and Black patients) experience more difficulty in being conventionally admitted for psychiatric conditions, and therefore present later with severe symptoms necessitating compulsory admission. (1) This systematic review evaluates whether compulsory admission rates differ between ethnicities in the UK and whether patients are accessing psychiatric healthcare through conventional systems (i.e. referral from primary care).

In our meta-analysis of 10 studies, ethnic minorities were more likely to be compulsorily admitted. Non-white patients were 2.38 times (95% CI: 1.55 – 3.65) more likely to be compulsorily admitted than White patients. Black patients were 2.77 times (95% CI: 1.84 – 4.18) more likely to be compulsorily admitted than White patients. Interestingly, there was no significant difference in compulsory admittance rate between Asian patients and White patients, with Asian patients being 0.96 times (95% CI: 0.78 – 1.17) more likely to be compulsorily admitted than White patients. Although admission rates differed between races, there was no clear consensus on an increased prevalence of psychiatric disorders amongst minorities based on 32 studies in the United Kingdom.

The systematic review discusses possible causes for the greater likelihood for compulsory admissions in Black patients compared to White patients, as well as the non-significant result in Asian patients compared to White patients. The possible causes encompass social stigma associated with psychiatric disorders, and cultural and language barriers to conventional psychiatric healthcare in ethnic minorities.

Introduction

Compulsory admission is the authorised admission of a patient in the UK to hospital after a formal mental health assessment under the UK's Mental Health Act 1983, (2) which encompasses any psychiatric disorder except for learning disabilities. Criteria for detainment, for either assessment or treatment purposes, include: if the patient is at risk of harming themselves or the public; if the nature and severity of the psychiatric disorder necessitate the detainment; or to ensure that the patient receives the appropriate treatment if they lack capacity to consent. (3)

Black patients in the UK have been shown to have higher rates of compulsory admission compared to White patients. (1) The higher detention rates have been suggested as a manifestation of racial discrimination and institutional racism within a healthcare system. (4) Lewis G et al. conducted a study where 220 British psychiatrists were given a questionnaire regarding a case vignette of a patient, whose sex and race were changed when given to each psychiatrist. (5) This study showed that psychiatrists, of which 79% were educated in the UK, view Black male patients as more violent compared to their White counterparts, implying the presence of an inherent prejudice towards Black patients. Other factors leading to higher compulsory admission rates amongst BME (Black and Minority Ethnic) groups include the lack of access for help through a conventional route, as one study found that African-Caribbean families were more likely to access help through the criminal system instead of through the medical system. (6) Furthermore, according to the 2011 National Census for English Proficiency, 7.3% of minorities consisting of non-Whites could not speak English or could not speak English well, compared to only 0.7% in the White population, (7) this could potentially lead to language barriers should they require mental health services. (8) All of the above factors collectively contribute to patients presenting later with more severe symptoms and necessitating compulsory admission.

The primary aim of this systematic review was to investigate the relationship between compulsory admission and ethnicity, amongst the minority groups of Blacks and Asians, regardless of clinical presentation. There was a secondary focus on the ethnic disposition to certain psychiatric illnesses, and hence the prevalence of psychiatric illnesses within each ethnic group. From the results obtained, we discuss the underlying social determinants within the field of mental health, and the need for improvement to deliver equal treatment regardless of ethnicity in the UK. In addition to the literature review, the study also aimed to conduct a meta-analysis using data from the studies that meet our inclusion criteria.

Method

Literature search

Search strategies for the systematic review focused on this primary question: *is there variation between ethnic groups in terms of compulsory hospital admission rate?* The data for meta-analysis was also generated from this question. There was a secondary question: *is there an increased prevalence of psychiatric disorders amongst ethnic minorities?*

The search was confined to studies published between 1983 to 2016, due to the establishment of the Mental Health Act in 1983. The primary bibliographic database was PubMed and Medical Subject Headings (MeSH) terms were grouped as search terms, which included: (a) GREAT BRITAIN, HOSPITALISATION, ETHNIC GROUPS; (b) MENTAL HEALTH, ETHNIC GROUPS, GREAT BRITAIN; (c) COMMITMENT OF MENTALLY ILL, GREAT BRITAIN, ETHNIC GROUPS; (d) GREAT BRITAIN, HOSPITALISATION, ETHNIC GROUPS, PSYCHIATRY; (e) GREAT BRITAIN, INDIGENOUS GROUPS, HOSPITALISATION; (f) COMMITMENT OF MENTALLY ILL, (ETHNICITY or ETHNIC GROUPS or MINORITIES), (GREAT BRITAIN or UNITED KINGDOM). [Figure 1]

Our inclusion criteria for the systematic review were studies looking at: (i) psychiatric compulsory admission (ii) comparison between ethnicities including Whites v non-Whites, Whites v Blacks, or Whites v Asians, and (iii) studies looking at the prevalence of psychiatric disorders within different ethnic groups.

Our exclusion criteria were: (i) topics unrelated to psychiatric compulsory admissions to hospitals, (ii) lack of access to full article, (iii) non-primary data, (iv) studies based outside the UK and (v) lack of comparison to a control White group. The PRISMA diagram (Figure 1) demonstrates the papers included. In addition to these criteria, only papers which had quantitative data involving odds ratios between comparison groups were included in the meta-analysis.

Quality ratings

The quality of published studies was assessed using the criteria from Bhui et al. that evaluated ethnic variation in pathways to specialist care (Table 1). (9) The criteria focused on three domains: sample source and size, adjustment for confounding variables and quality of method of ethnic group classification.

Studies were rated on the size of the sample (0-3), adjustment for confounding factors (0-5) and appropriate classification of ethnic groups (0-3). Studies were then categorised into high-rated (8-11), medium rated (4-7) and low rated (0-3). Each published study was independently evaluated by two reviewers in order to improve the reliability of inclusion and data-extraction. Where differences existed, consensus was achieved by means of discussion. (Table 1)

Data analysis

Meta-analysis and figures were generated using Review Manager (v5.3, The Cochrane Collaboration, London) to pool odds-ratio for

compulsory admission. Studies included in the meta-analysis did not adjust odds ratio for confounding factors, with analysis being based on uncorrected data.

Random-effects model was used when heterogeneity (I2) was found to be greater than 0.5. Fixed-effects model was used when heterogeneity (I2) was less than 0.5.

Ethical approval

Ethical approval was not necessary as there was no intervention conducted for this study.

Results

Identifying and rating primary studies

Preliminary search terms returned 294 potentially relevant titles, of which 215 were unique articles. After evaluating abstracts and data, we found that 32 studies met the inclusion and exclusion criteria. These papers were subsequently rated (summarised in Table 2).

Compulsory admissions

From the selected 15 studies that involved comparisons of compulsory admission rates with ethnicity (5 high-rated, (6,10–13), 9 medium-rated (9,14–20) and 1 low-rated (21)), all studies found that Black patients are more likely to be compulsorily admitted. From these 15 studies, 7 studies included odds ratios, and were thus included in our meta-analysis. Black patients are 2.77 times (95% CI: 1.84 - 4.18) more likely to be compulsorily admitted than White patients (Figure 3).

Our meta-analysis of 2 studies that compared compulsory admissions of non-White patients compared to White patients suggested non-White patients are 2.38 times (95% CI: 1.55 - 3.65) as likely as White patients to be compulsorily admitted (Figure 2).

Most studies suggest Asian patients are less likely to be admitted compared to White patients (2 high-rated (10,11), 2 moderately-rated (14,18)), while 1 moderately-rated study (22) found that Asian patients are more likely to be compulsorily admitted. From these 5 studies, 3 studies included odds ratios, and were thus included in our meta-analysis. Our meta-analysis (Figure 4) of odds ratio in compulsory admission of Asian patients against White patients was inconclusive (95% CI: 0.78 - 1.17) and therefore no significant difference was observed.

One primary study (23) found that ethnic minorities (Asian and Black patients) experience difficulties going through conventional admission pathways for psychiatric conditions. The study proposed that it could potentially deter patients from seeking help earlier, thereby resulting in untreated psychiatric disorders.

Prevalence of psychiatric disorders

When comparing regular GP admissions and prescription data, studies varied on whether psychotic conditions are more or less prevalent amongst minority groups in the UK. 3 high-rated (6,24,25) and 1 medium-rated study (22) concluded that psychotic-based admissions or prescription rates in minorities were not different to the White population. Contrastingly, 4 low-rated (19,26–28) and 2 medium-rated studies (14,29) found that psychotic conditions were more frequent among the Black population. Therefore, it appears that the prevalence of psychotic disorders is inconclusive.

Similarly, there was no consensus on whether the prevalence of psychotic conditions in the Asian minority is different to the White population. Among the medium-rated studies, 1 found decreased prevalence of psychotic conditions within the Asian population, (30) 1 found that prevalence differences amongst Asians and Whites were non-significant (22) and 3 found prevalence to be higher amongst Asians. (14,29,31) (Table 2)

Discussion

The inequality in compulsory admissions rates in different ethnicities could be attributed to a variety of reasons. Firstly, stigma within certain ethnic groups against psychiatric disorders can lead to individuals avoiding treatment and refraining from discussing their symptoms. Secondly, greater compulsory admission rates could also reflect a greater prevalence of severe psychiatric disorders in particular ethnic groups. Thirdly, minorities also encounter barriers due to cultural and language differences which impede obtaining a diagnosis or treatment plan from their clinician.

Stigma

Social stigma within ethnic groups against psychiatric disorders may affect the number of compulsory admissions observed. Anglin et al. (42) suggested that this may be the case for Black patients, who have a higher odds-ratio than other ethnic groups for compulsory admissions. This can be attributed to the lack of understanding of psychiatric disorders, and cultural beliefs within their group, that those with psychiatric disorders can cause harm to themselves or inflict harm on those around them through violence. (42) Such perceptions within the Black group can lead to people with psychiatric problems refraining from voluntarily seeking help, in order to avoid labelling, prejudice and segregation. (43,44)

In addition, reduced help-seeking behaviour may exacerbate an individual's psychiatric disorders and increase the likelihood of an event triggering compulsory admission. (5) Valmaggia et al. found that a longer duration of untreated psychosis (DUP), defined to be the time between the onset of the first psychotic symptom and the initiation of treatment, may have an association with worse prognosis and may also decrease the likelihood of remission from psychosis. (45) Furthermore, engagement of health services for psychosis in the prodromal phase may result in improved short-

term prognosis, decreasing the likelihood of an event which would require compulsory admission. (46)

Similarly, in Asian cultures, individuals with psychiatric health disorders are perceived to be dangerous; (47) however, the rate of compulsory admissions was found to be similar to the White population. This suggests that stigma may not necessarily have a consistent effect of increasing compulsory admission rates in different ethnic groups, suggesting the involvement of other sociological factors, which will be addressed below.

Ethnic disposition to compulsory admissions-related conditions

Inherent differences in prevalence between ethnicities could contribute to the varying rates of compulsory admissions observed between ethnicities. In this study, compulsory admission rates were not normalised to prevalence rates of psychiatric conditions, as it was inconclusive from our systematic review whether non-White individuals were more predisposed to psychiatric disorders compared to White individuals. We suggest further validation of our findings by adjusting compulsory admission rates to respective prevalence rates of psychiatric conditions.

The aetiology of schizophrenia and the difference in its prevalence in Black and White populations is widely reported. Members of the Black group in the UK were reported in studies carried out in the past decades to have higher rates of schizophrenia than White members. (48–50) A study looking at the family history of affected patients has found there was a seven-fold increase in risk for schizophrenia in second-generation African-Caribbean siblings of affected individuals compared to their White counterparts. This result suggests non-genetic factors, such as the environment, and a complex epigenetic interaction between environmental and genetic factors selectively affecting second generation Black patient groups. (51) Burnett et al. found that a greater proportion of Black patients with psychosis live alone, are unemployed and live in public housing compared to White and Asian patients. (23) It is possible that such social factors are associated with increased risk of developing or aggravating psychiatric illnesses. (52–54) However, more definite evidence is required to determine the significance of the associative roles of these social factors in development of psychiatric disorders amongst Black patients.

No significant difference was found between compulsory admission rates for Asian patients compared to White patients. Despite Asians being a minority group, our findings could be explained by under-utilisation of healthcare services by Asians. For example, language barriers and cultural differences in referral networks and traditional treatment methods, may lead to under-utilisation of healthcare services. (55) Moreover, Saint et al. reported that Asian patients with psychological disorders, such as depression, tend to report more somatic symptoms than White patients, which could lead to misdiagnosis of conditions and contribute to the lower number of psychiatric admissions. (56) Therefore, the lack of detection of psychiatric disorders and the failure to meet the compulsory admission criteria for Asian groups may have led to no observable difference in compulsory admission rates between Asians and Whites.

Furthermore, a limitation of our study is that Asian patients are often under-represented in mental disorder prevalence studies, as the sample sizes are relatively small compared to other ethnicities. (30,34) In addition, many studies have not categorised Asians into subgroups despite significant cultural differences within the racial group. (10,11,14,18,27,33) Therefore, no conclusions can be drawn from a generalised grouping of all Asian patients, providing a possible direction for future investigations. (30)

Ethnic stereotypes and compulsory admissions

In the past, young Black men and adolescents in the UK have often been associated with violent crimes (57) and being '*troublemakers*' (58) by the media. Singh et al. (13) suggested that such stereotyping of the Black population has an effect on emergency decision-making in psychiatric wards, perhaps leading to more patients being compulsorily admitted. (5) It is not certain whether Black patients are themselves more violent in nature, with conflicting data on the matter, (41,59) whilst others suggest that it is the stereotyping of Black adolescents affecting the judgement of psychiatrists, resulting in greater risk of detainment. (5)

Boundaries leading to unmet needs

Under-utilisation of mental health support services at an earlier stage may cause patients to present with a more severe form of their condition which necessitates compulsory admissions. Mclean et al. suggest that the under-utilisation in the Black group could be attributed to the expectation of discrimination in the form of misdiagnosis, mistreatment and over-prescription which could deter them from using these services. (60) Therefore, as mentioned previously, institutionalised prejudice may not only lead to misdiagnosis, but the perception of such discrimination may also act as a barrier preventing early access to mental health services, aggravating their existing mental conditions.

Additionally, ethnic minorities may have subtly different presentations of psychiatric health conditions due to cultural variation, which can be missed by culturally unaware clinicians. (61) For example, psychiatrists in Chinese settings found some patients to report somatic symptoms in the place of dysphoria when presenting with depression. (62) Furthermore, Fabrega et al. suggest that criteria for psychiatric conditions such as the DSM-IV (Diagnostic and Statistical Manual of Mental Disorders) have a Western bias and assume that psychiatric conditions present universally. (63)

Sentell et al. found poor English proficiency to be a significant contributing factor towards missed diagnoses and unmet healthcare needs in the US. This was particularly apparent during consultations and treatment, which rely heavily on verbal communication, leading to a significant difference in outcome found between English-speaking minorities, and minorities with limited English proficiency (LEP). (64) Furthermore, patients with LEP without interpreters may incompletely disclose their true presenting symptoms and hence limit the efficacy of diagnosis and subsequent treatment. (65) In the United Kingdom, this may be a bigger issue within the Asian population, as a government census from 2011 found that 2.6% of the Black population could not speak English or could not speak English well, whereas 10.5% of the Asian population could not speak English at all or well. (7)

However, the result of our meta-analysis showed fewer compulsory admission rates within the Asian population, suggesting that there is a complex interaction of language proficiency with other factors. A detailed re-examination of the inadequacy in the mental health system, culturally conscious training and the use of interpreters during consultation for patients with LEP could be useful to tackle the unmet needs among ethnic minorities. (64)

Limitations arising from Classification

The statistical conclusions on racial disparities were influenced greatly by the data collection methods. This study used the racial categories WHITES, BLACKS (African-Caribbean), and ASIANS to enable a statistical comparison of different ethnic groups. However, inclusion and exclusion to each category vary widely in different studies, hence masking heterogeneity within the groups. (61) In addition, such categories evolve from social constructs, which are inconsistent and based largely on subjective classification constructed on geopolitical divides.

Self-identification and its influence on data

All studies in this meta-analysis rely on self-identification of ethnicity by the individual. While this approach is a conventional and safe approach in social demographic analyses, the overall identification of ethnicity in genetic analyses stems from an individual's ancestry informative markers, which are based on genomic data. Mersha et al. show that self-reporting African Americans can have drastically varying African and European ancestries. Since a similar deviation may be prevalent in the UK, associations between data and genetic disposition are limited. (66)

Scope of Study Selection

The studies used in this analysis were obtained from PubMed©, which includes the MEDLINE© database. 4 studies were included in the meta-analysis between Whites and non-Whites (Figure 2). 2 studies were included in the meta-analysis between Asians and Whites (Figure 4). This can contribute to the distortion of data. Therefore, additional search terms and databases should be utilised to obtain further relevant studies.

Follow-up studies could analyse additional databases such as EMBASE (medical and pharmacologic database by Elsevier publishing), CINAHL (cumulative index to nursing and allied health literature), CANCERLIT (cancer literature research database), and the Cochrane Collaborative, allowing for a wider net of studies to add to the present body of evidence.

Conclusion

This meta-analysis determined that the likelihood of compulsory admission is greater in Black compared to White populations and inconclusive in White compared to Asian populations in the UK. This systematic review suggested that Asian and Black patients have difficulties accessing conventional admission pathways for psychiatric conditions due to poor cultural awareness and the existence of social stigma within these ethnic groups. Stigma towards psychiatric disorders may also reduce help-seeking behaviour and lead to under-utilisation of mental health facilities. Possible reasons preventing ethnic minorities from receiving adequate treatment include language barriers in consultations, cultural variations in the presentation of psychiatric conditions, and institutionalised discrimination leading to mistreatment and misdiagnosis.

This study shows a clear presence of ethnic differences in unmet needs and a complex interplay of factors affecting compulsory admission rates for psychiatric disorders. Based on our findings, we suggest improving consultations through culturally conscious training for clinicians with interpreters for patients with LEP, as well as increasing awareness of psychiatric disorders within the ethnic groups. It was unclear whether the difference in compulsory admission rates between ethnic minorities and the White population was due to discrimination against ethnic minorities and psychiatric conditions.

References

1. Mann F, Fisher HL, Major B, Lawrence J, Tapfumaneyi A, Joyce J, et al. Ethnic variations in compulsory detention and hospital admission for psychosis across four UK Early Intervention Services. BMC psychiatry. 2014;14(1):256.

https://doi.org/10.1186/s12888-014-0256-1

PMid:25214411 PMCid:PMC4173060

2. HM Government. Mental Health Act 1983. 2016.

3. Salize H, Dreßing H, Peitz M. Compulsory admission and involuntary treatment of mentally ill patients-legislation and practice in EU-member states. Central Institute of Mental Health

Research Project Final Report. 2002;15(15).

4. Singh S, Burns T. Race and mental health: there is more to race than racism. BMJ. 2006;333:648–51.

https://doi.org/10.1136/bmj.38930.501516.BE

PMid:16990327 PMCid:PMC1570848

5. Lewis G, Croft-Jeffreys C, David A. Are British psychiatrists racist? The British Journal of Psychiatry. 1990;157(3):410–5.

https://doi.org/10.1192/bjp.157.3.410

PMid:2245273

6. Morgan C, Mallett R, Hutchinson G, Bagalkote H. Pathways to care and ethnicity. 1: Sample characteristics and compulsory admission Report from the ÆSOP study. The British Journal of Psychiatry. 2005;186(4):281–9.

https://doi.org/10.1192/bjp.186.4.281

PMid:15802683

7. Office for National Statistics. English language proficiency by ethnic group. England and Wales; 2011.

8. Office for National Statistics. 2011 Census: Detailed analysis – English language proficiency in England and Wales, Main language and general health characteristics. 2016.

9. Bhui K, Stanfield S, Hull S, Priebe S, Mole F, Feder G. Ethnic variations in pathways to and use of specialist mental health services in the UK. The British Journal of Psychiatry. 2003;182(2):105–16.

https://doi.org/10.1192/bjp.182.2.105

PMid:12562737

10. Singh S, Burns T, Tyrer P, Islam Z. Ethnicity as a predictor of detention under the Mental Health Act. Psychological Medicine; 2014;44(5):997–1004.

https://doi.org/10.1017/S003329171300086X

PMid:23795603

11. Bennewith O, Amos T, Lewis G, Katsakou C. Ethnicity and coercion among involuntarily detained psychiatric in-patients. The British Journal of Psychiatry. 2010;196(2):75–6.

https://doi.org/10.1192/bjp.bp.109.068890

PMid:20044667

12. Morgan C, Mallett R, Hutchinson G, Bagalkote H. Pathways to care and ethnicity. 2: source of referral and help-seeking report from the ÆSOP study. The British Journal of Psychiatry. 2005;186(4):290–6.

https://doi.org/10.1192/bjp.186.4.290

PMid:15802684

13. Singh S, Croudace T, Beck A, Harrison G. Perceived ethnicity and the risk of compulsory admission. Social Psychiatry and Psychiatric Epidemiology. 1997;33(1):39–44.

https://doi.org/10.1007/s001270050020

14. Corrigall R, Bhugra D. The role of ethnicity and diagnosis in rates of adolescent psychiatric admission and compulsory detention: a longitudinal case-note study. Journal of the Royal Society of Medicine. 2013;106(5):190–5.

15. Borschmann R, Gillard S, Turner K. Demographic and referral patterns of people detained under Section 136 of the Mental Health Act (1983) in a south London Mental Health Trust from 2005 to 2008. Medicine, Science and the Law. 2010;50(1):15–8.

https://doi.org/10.1192/bjp.182.2.105

PMid:12562737

16. Singh S, Greenwood N, White S. Ethnicity and the mental health act 1983. The British Journal of Psychiatry. 2007;191(2):99–105.

https://doi.org/10.1192/bjp.bp.106.030346

PMid:17666492

17. Simmons P, Hoar A. Section 136 use in the London Borough of Haringey. Medicine, Science and Law. 2001;41(4):342–8.

https://doi.org/10.1177/002580240104100412

PMid:11693231

18. Coid J, Kahtan N, Gault S, Jarman B. Ethnic differences in admissions to secure forensic psychiatry services. The British Journal of Psychiatry. 2000;177(3):241–7.

https://doi.org/10.1192/bjp.177.3.241

PMid:11040885

19. Koffman J, Fulop N, Pashley D, Coleman K. Ethnicity and use of acute psychiatric beds: one-day survey in north and south Thames regions. The British Journal of Psychiatry. 1997;171(3):238–41.

https://doi.org/10.1192/bjp.171.3.238

PMid:9337976

20. McGovern D, Cope R. Second generation Afro-Caribbeans and young whites with a first admission diagnosis of schizophrenia. Social Psychiatry and Psychiatriv Epidemiology. 1991;26(2):95–9. https://doi.org/10.1007/BF00791535

21. Davies S, Thornicroft G, Leese M, Higgingbotham A.

Ethnic differences in risk of compulsory psychiatric admission among representative cases of psychosis in London. BMJ. 1996;312(7030):533–7.

https://doi.org/10.1136/bmj.312.7030.533

PMid:8595280 PMCid:PMC2350333

22. Ineichen B, Harrison G, Morgan H. Psychiatric hospital admissions in Bristol. I. Geographical and ethnic factors. The British Journal of Psychiatry. 1984;145(6):600–4.

https://doi.org/10.1192/bjp.145.6.600

PMid:6509269

23. Burnett R, Mallett R, Bhugra D. The first contact of patients with schizophrenia with psychiatric services: social factors and pathways to care in a multi-ethnic population. Psychological medicine. 1999;29(2):475–83.

https://doi.org/10.1017/S0033291798008125

PMid:10218939

24. Connolly A, Taylor D, Sparshatt A. Antipsychotic prescribing in Black and White hospitalised patients. Journal of Psychopharmacology. 2011;25(5):704–9.

https://doi.org/10.1177/0269881109387841

PMid:21511740

25. Callan A. Schizophrenia in Afro-Caribbean immigrants. Journal of the Royal Society of Medicine. 1996;89(5):253–6.

https://doi.org/10.1177/014107689608900505

PMid:8778431 PMCid:PMC1295775

26. Chowdhury N, Whittle N, McCarthy K. Ethnicity and its relevance in a seven-year admission cohort to an English national adolescent medium secure health service unit. Criminal Behaviour and Mental Health. 2005;15(4):261–72.

https://doi.org/10.1002/cbm.29

PMid:16575847

27. Tolmac J, Hodes M. Ethnic variation among adolescent psychiatric in-patients with psychotic disorders. The British Journal of Psychiatry. 2004;184(5):428–31.

https://doi.org/10.1192/bjp.184.5.428

PMid:15123507

28. Suhail K, Cochrane R. Seasonal variations in hospital admissions for affective disorders by gender and ethnicity. Social Psychiatry and Psychiatric Epidemiology. 1998;33(5):211–7.

https://doi.org/10.1007/s001270050045

PMid:9604670

29. Thomas C, Stone K, Osborn M, Thomas P. Psychiatric morbidity and compulsory admission among UK-born Europeans, Afro-Caribbeans and Asians in central Manchester. The British Journal of Psychiatry. 1993;163(1):91–9.

https://doi.org/10.1192/bjp.163.1.91

PMid:8353706

30. Chang S, Steeg S, Kapur N. Self-harm amongst people of Chinese origin versus White people living in England: a cohort study. BMC psychiatry. 2015;15(1):1.

https://doi.org/10.1186/s12888-015-0467-0

PMid:25880647 PMCid:PMC4409751

31. Glover G. The use of inpatient psychiatric care by immigrants in a London borough. International Journal of Social Psychiatry. 1991;37(2):121–34.

https://doi.org/10.1177/002076409103700207

PMid:1917369

32. Lawlor C, Johnson S, Cole L. Ethnic variations in pathways to acute care and compulsory detention for women experiencing a mental health crisis. International Journal of Social Psychiatry. 2012;

https://doi.org/10.1177/0020764010382369

PMid:21059630 PMCid:PMC3257000

33. Tulloch A, Fearon P, David A. The determinants and outcomes of long-stay psychiatric admissions. Social psychiatry and psychiatric epidemiology. 2008;43(7):569–74.

https://doi.org/10.1007/s00127-008-0332-2

PMid:18347750

34. Ali S, Dearman S, McWilliam C. Are Asians at greater risk of compulsory psychiatric admission than Caucasians in the acute general adult setting? Medicine, Science and the Law. 2007;47(4):311–4.

https://doi.org/10.1258/rsmmsl.47.4.311

PMid:18069536

35. Gudjonsson G, Rabe-Hesketh S, Szmukler G. Management of psychiatric in-patient violence: patient ethnicity and use of medication, restraint and seclusion.; 2004;184(3):258–62.

https://doi.org/10.1192/bjp.184.3.258

PMid:14990525

36. Webber M, Huxley P. Social exclusion and risk of emergency compulsory admission. A case-control study. Social Psychiatry and

Psychiatric Epidemiology. 2004;39(12):1000–9.

https://doi.org/10.1007/s00127-004-0836-3

PMid:15583909

37. Riordan S, Donaldson S, Humphreys M. The imposition of restricted hospital orders: potential effects of ethnic origin. International journal of law and psychiatry. 2004;27(2):171–7.

https://doi.org/10.1016/j.ijlp.2004.01.007

PMid:15063641

38. Oluwatayo O, Gater R. The role of engagement with services in compulsory admission of African/Caribbean patients. Social psychiatry and psychiatric epidemiology. 2004;39(9):739–43.

https://doi.org/10.1007/s00127-004-0794-9

PMid:15672295

39. Commander M, Odell S, Surtees P. Characteristics of patients and patterns of psychiatric service use in ethnic minorities. International journal of social psychiatry. 2003;49(3):216–24.

https://doi.org/10.1177/00207640030493007

PMid:14626364

40. Audini B, Lelliott P. Age, gender and ethnicity of those detained under Part II of the Mental Health Act 1983. The British Journal of Psychiatry. 2002;180(3):222–6.

https://doi.org/10.1192/bjp.180.3.222

PMid:11872514

41. Dunn J, Fahy T. Police admissions to a psychiatric hospital. Demographic and clinical differences between ethnic groups. The British Journal of Psychiatry. 1990;156(3):373–8.

https://doi.org/10.1192/bjp.156.3.373

PMid:1971766

42. Anglin DM, Link BG, Phelan JC. Racial differences in stigmatizing attitudes toward people with mental illness. Psychiatric Services. 2006;

https://doi.org/10.1176/ps.2006.57.6.857

PMid:16754764

43. Corrigan P, Green A, Lundin R. Familiarity with and social distance from people who have serious mental illness. Psychiatric services. 2001;

https://doi.org/10.1176/appi.ps.52.7.953

44. Corrigan P, Edwards A, Green A. Prejudice, social distance, and familiarity with mental illness. Schizophrenia bulletin. 2001;27(2):219.

https://doi.org/10.1093/oxfordjournals.schbul.a006868

PMid:11354589

45. Marshall M, Lewis S, Lockwood A, Drake R, Jones P, Croudace T. Association Between Duration of Untreated Psychosis and Outcome in Cohorts of First-Episode Patients: A Systematic Review. Arch Gen Psychiat. 2005;62(9):975–83.

46. Valmaggia L, Byrne M, Day F, Broome M, Johns L, Howes O, et al. Duration of untreated psychosis and need for admission in patients who engage with mental health services in the prodromal phase. Br J Psychiatry. 2015;207(2):130–4.

https://doi.org/10.1192/bjp.bp.114.150623

PMid:26045348 PMCid:PMC4655441

47. Whaley A. Ethnic and racial differences in perceptions of dangerousness of persons with mental illness. Psychiatric services. 1997;48(10):1328–30.

https://doi.org/10.1176/ps.48.10.1328

PMid:9323754

48. Bebbington P, Hurry J, Tennant C. Psychiatric disorders in selected immigrant groups in Camberwell. Social Psychiatry. 1981;16(1):43–51.

https://doi.org/10.1007/BF00578068

49. Cochrane R, Bal S. Mental hospital admission rates of immigrants to England: a comparison of 1971 and 1981. Social psychiatry and psychiatric epidemiology. 1989;24(1):2–11.

https://doi.org/10.1007/BF01788193

PMid:2496474

50. McGovern D, Cope R. First psychiatric admission rates of first and second generation Afro Caribbeans. Social Psychiatry 1987;22(3):139–49.

https://doi.org/10.1007/BF00583848

PMid:3498221

51. Hutchinson G, Takei N, Fahy T, Bhugra D. Morbid risk of schizophrenia in first-degree relatives of white and African-Caribbean patients with psychosis. The British Journal of Psychiatry. 1996;169(6):776–80.

https://doi.org/10.1192/bjp.169.6.776

PMid:8968638

52. Sharpley M, Hutchinson G, Murray R, Kenzie K. Bringing in the social environment. The British Journal of Psychiatry. 2001;178(40):60–8.

https://doi.org/10.1192/bjp.178.40.s60

53. Lomas J. Social capital and health: implications for public health and epidemiology. Social science & medicine. 1998;47(9):1181–8.

https://doi.org/10.1016/S0277-9536(98)00190-7

54. Nazroo J. Rethinking the relationship between ethnicity and mental health: the British Fourth National Survey of Ethnic Minorities. Social psychiatry and psychiatric epidemiology. 1998;33(4):145–8.

https://doi.org/10.1007/s001270050036

PMid:9567663

55. Brewin C. Explaining the lower rates of psychiatric treatment among Asian immigrants to the United Kingdom: A preliminary study. Social Psychiatry. 1980;15(1):17–9.

https://doi.org/10.1007/BF00577957

56. Arnault SD, Kim O. Is there an Asian idiom of distress?: Somatic symptoms in female Japanese and Korean students. Archives of psychiatric nursing. 2008;22(1):27–38.

https://doi.org/10.1016/j.apnu.2007.10.003

PMid:18207054 PMCid:PMC2239242

57. Moore K, Jewell J, Cushion S. Media representations of black young men and boys: Report of the REACH media monitoring project. 2011.

58. Cottle G, Croft-Jeffreys C. Ethnic Minorities & The Media: Changing Cultural Boundaries. McGraw-Hill Education (UK). 2000.

59. Owens D, Harrison G, Boot D. Ethnic factors in voluntary and compulsory admissions. Psychological medicine. 1991;21(1):185–96.

https://doi.org/10.1017/S003329170001477X

PMid:2047495

60. Mclean C, Campbell C, Cornish F. African-Caribbean interactions with mental health services in the UK: experiences and expectations of exclusion as (re) productive of health inequalities. Social Science & Medicine. 2003;56(3):657–69.

https://doi.org/10.1016/S0277-9536(02)00063-1

61. Cohen B, Bulatao R, Anderson N. Critical perspectives on racial and ethnic differences in health in late life. National Academics Press. 2004.

62. Kleinman A. Depression, somatization and the "new cross-cultural psychiatry."Social Science & Medicine. 1967;11(1):3–9. https://doi.org/10.1016/0037-7856(77)90138-X

63. Horacio F. International Systems of Diagnosis in Psychiatry. Journal of nervous and mental disease. 1994;182(5):256–63.

https://doi.org/10.1097/00005053-199405000-00002

64. Sentell T, Shumway M, Snowden L. Access to mental health treatment by English language proficiency and race/ethnicity. Journal of General Internal Medicine. 2007;22(2):289–93.

https://doi.org/10.1007/s11606-007-0345-7

PMid:17957413 PMCid:PMC2150610

65. Bauer A, Alegría M. Impact of Patient Language Proficiency and Interpreter Service Use on the Quality of Psychiatric Care: A Systematic Review. Psychiatric Services. 2010;61(8).

https://doi.org/10.1176/ps.2010.61.8.765

66. Mersha T, Abebe T. Self-reported race/ethnicity in the age of genomic research: its potential impact on understanding health disparities. Human genomics. 2015;9(1):1.

https://doi.org/10.1186/s40246-014-0023-x

PMid:25563503 PMCid:PMC4307746

Figures

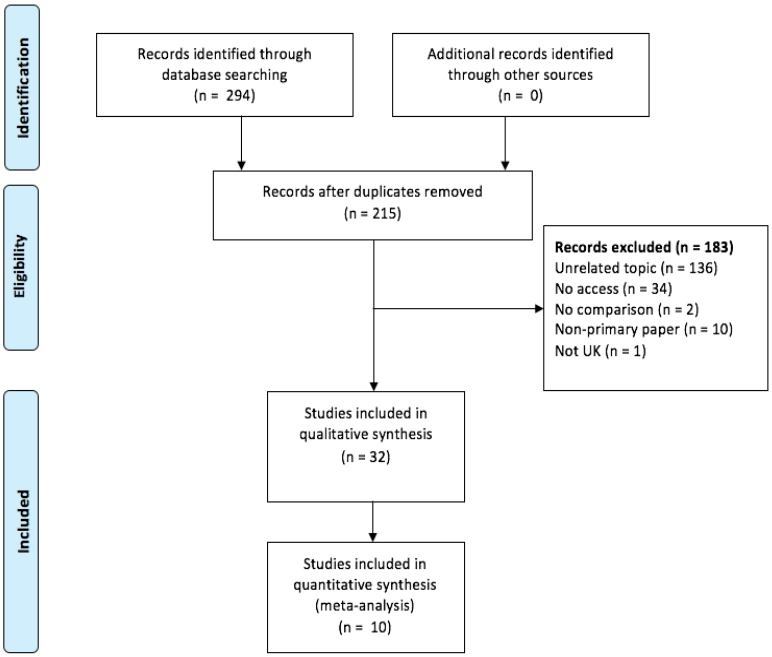

Figure 1 The Flow diagram of our systematic review

Source of sample and sample size	Score	Adjustment for confounding variables	Score	Ethnicity categorisation			
				Quality	Score	Use in analysis	Score
Routine data (e.g. health authority, GP list data)	0	None	0	Third-party report (e.g. ward staff categorisation, name-based methods, skin colour methods)	0	Inappropriate ethnic groups combined for major study	0
Project specific data: <30 cases in ethnic groups for major outcomes	1	Age and/or sex	1	Self-reported ethnicity or use of census categories	1	reasonable amalgamation of groups collected by census/self-report method	1
Project specific data: <30 cases in ethnic groups for major outcomes	2	Diagnosis or the severity of disease Give 1 point if this sample is selected by diagnosis	1			All analysis done on ethnic groups without combination, and self-report/ census categories for categorisation	2
Project specific data and total smaple size >500	3	Comorbidity and risk factors for outcome of interest	1-3				
Maximum possible score	3		5		1		2

Table 1 presents the rating system used. Papers are categorized into high-rated (8-11), moderate-rated (4-7) and low-rated (0-3)

Author	Title	Rating
(Chang, Steeg, & Kapur, 2015) (30)	Self-harm amongst people of Chinese origin vs White people living in England: a cohort study	Moderate
(Singh, Burns, Tyrer, & Islam, 2014) (10)	Ethnicity as a predictor of detention under the Mental Health Act	High
(Corrigall & Bhugra, 2013) (14)	The role of ethnicity and diagnosis in rates of adolescent psychiatric admission and compulsory detention: a longitudinal case-note study.	Moderate
(Lawlor, Johnson, & Cole, 2012) (32)	Ethnic variations in pathways to acute care and compulsory detention for women experiencing a mental health crisis	Moderate
(Connolly, Taylor, & Sparshatt, 2011) (24)	Antipsychotic prescribing in Black and White hospitalised patients.	High
(Borschmann, Gillard, & Turner, 2010) (15)	Demographic and referral patterns of people detained under Section 136 of the Mental Health Act (1983) in a south London Mental Health Trust from 2005 to 2008.	Moderate
(Bennewith, Amos, Lewis, & Katsakou, 2010) (11)	Ethnicity and coercion among involuntarily detained psychiatric in-patients.	High
(Tulloch, Fearon, & David, 2008) (33)	The determinants and outcomes of long-stay psychiatric admissions: a case-control study.	Low
(Ali, Dearman, & McWilliam, 2007) (34)	Are Asians at greater risk of compulsory psychiatric admission than Caucasians in the acute general adult setting?	Moderate

(Morgan, Mallett, Hutchinson, & Bagalkote, 2005) (12)	Pathways to care and ethnicity. 2: Source of referral and help-seeking.	High
(Morgan, Mallett, Hutchinson, & Bagalkote, 2005a) (6)	Pathways to care and ethnicity. 1: Sample characteristics and compulsory admission.	High
(Chowdhury, Whittle, & McCarthy, 2005) (26)	Ethnicity and its relevance in a seven-year admission cohort to an English national adolescent medium secure health service unit.	Low
(Tolmac & Hodes, 2004) (27)	Ethnic variation among adolescent psychiatric in-patients with psychotic disorders.	Low
(Gudjonsson, Rabe-Hesketh, & Szmukler, 2004) (35)	Management of psychiatric in-patient violence: patient ethnicity and use of medication, restraint and seclusion	Moderate
(Webber & Huxley, 2004) (36)	Social exclusion and risk of emergency compulsory admission, A case-control study	High
(Riordan, Donaldson, & Humphreys, 2004) (37)	The imposition of restricted hospital orders: potential effects of ethnic origin.	Low
(Oluwatayo & Gater, 2004) (38)	The role of engagement with services in compulsory admission of African/Caribbean patients	Moderate
(Commander, Odell, & Surtees, 2003) (39)	Characteristics of patients and patterns of psychiatric service use in ethnic minorities	Moderate
(Audini & Lelliott , 2002) (40)	Age, gender and ethnicity of those detained under Part II of the Mental Health Act 1983	Moderate

(Simmons & Hoar, 2001) (17)	Section 136 use in the London borough of Haringey	Moderate
(Coid, Kahtan, Gault, & Jarman, 2000) (18)	Ethnic differences in admissions to secure forensic psychiatry services	Moderate
(Burnett, Mallett, & Bhugra, 1999) (23)	The first contact of patients with schizophrenia with psychiatric services: social factors and pathways to care in a multi-ethnic population	Moderate
(Suhail & Cochrane, 1998) (28)	Seasonal variations in hospital admissions for affective disorders by gender and ethnicity.	Low
(Singh, Croudace, Beck, & Harrison, 1997) (13)	Perceived ethnicity and the risk of compulsory admission.	High
(Koffman J, 1997) (19)	Ethnicity and use of acute psychiatric beds: one-day survey in north and south Thames regions.	Low
(Davies, Thornicroft, Leese, & Higgingbotham, 1996) (21)	Ethnic differences in risk of compulsory psychiatric admission among representative cases of psychosis in London	Low
(Callan, 1996) (25)	Schizophrenia in Afro-Caribbean immigrants	High
(Thomas, Stone, Osborn, & Thomas, 1993) (29)	Psychiatric morbidity and compulsory admission among UK-born Europeans, Afro-Caribbeans and Asians in central Manchester.	Moderate
(Glover, 1991) (31)	The use of inpatient psychiatric care by immigrants in a London Borough	Moderate
(Dunn & Fahy, 1990) (41)	Police admissions to a psychiatric hospital. Demographic and clinical differences between ethnic groups.	Moderate

(McGovern & Cope, 1991) (20)	Second generation Afro-Caribbeans and young whites with a first admission diagnosis of schizophrenia	Moderate
(Ineichen, Harrison, & Morgan, 1984) (22)	Psychiatric hospital admissions in Bristol. I. Geographical and ethnic factors.	Moderate

Table 2 shows the papers we included in our systematic review

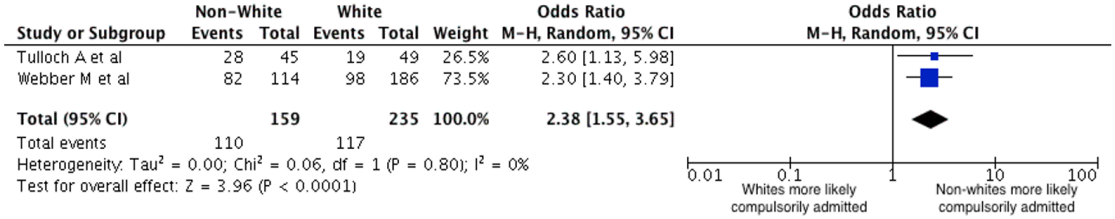

Figure 2: Whites v non-Whites. Heterogeneity = 0%. Pooled results suggest non-White patients are 2.38 (1.55-3.65) times more likely to be compulsorily admitted compare to White patients.

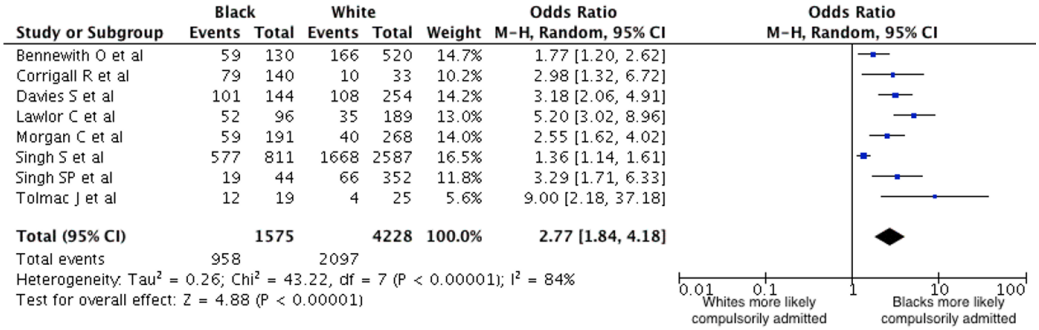

Figure 3: Whites v Blacks. Heterogeneity = 84%. Pooled results suggest Black patients are 2.77 (1.84-4.18) times more likely to be compulsorily admitted compare to White patients.

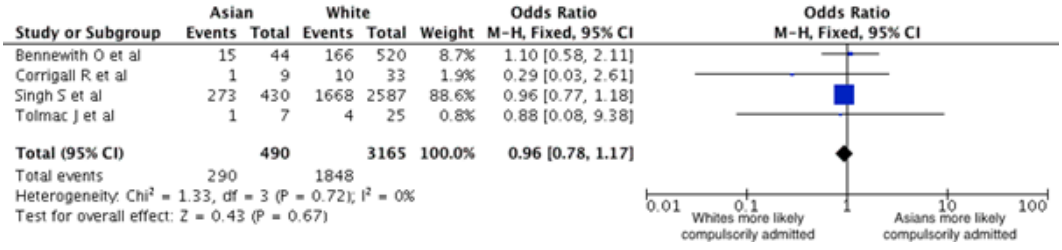

Figure 4: Whites v Asians. Heterogeneity = 0%. Pooled results suggest Asian patients are 0.96 (0.78-1.17) times more likely to be compulsorily admitted compare to White patients.

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Education

The Ponseti method for the treatment of congenital talipes equinovarus (CTEV)

EDUCATION

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ABSTRACT

Summary
Congenital talipes equinovarus (CTEV) is the most common musculoskeletal condition in newborns, affecting one-to-two per thousand babies. The Ponseti method is a treatment regime for the management of CTEV which describes a detailed method of manipulation, casting and bracing. Despite its initial publication in 1963, it only started to become popular in the 1990s, coming to UK shores in 1997 when NHS Physiotherapist Steve Wildon widely popularized the method around the country, including in Morecambe Bay. Patients who are treated with the Ponseti method report high levels of satisfaction many years after treatment. The body of literature evaluating the steps of the Ponseti method has grown in the last decade as the technique has become the gold standard treatment for clubfoot. Future research will look at how the Ponseti method can be improved in the management of CTEV.

Relevance
Medical students should be aware of CTEV and understand the steps in management of this common musculoskeletal condition in newborns.

Take Home Messages
• CTEV is the most common musculoskeletal condition in newborns and the current gold standard management is the Ponseti method.
• Ponseti method enables us to correct the majority of clubfeet with its conservative approach.
• Relapses which occur have a good prognosis when treated with surgery in the form of a tibialis anterior tendon transfer.

Introduction

Treatment of Congenital Talipes Equinovarus (CTEV), also known as clubfoot, has changed dramatically over the last twenty years with the introduction of the Ponseti method. This conservative mode of treatment, which involves manipulating joints using casts, was first described by Dr. Ponseti in 1963 (1) at the University of Iowa and is considered to be the most effective method of treating clubfoot worldwide. Ponseti published a further detailed description of his method in 1972 (2) and reported the results of his method in a series of long term studies. (3-5) The recommendations which he made in his first two papers are still valid today with only minor adjustments being made by himself or his colleagues.

Prior to the introduction of the Ponseti method, a number of conservative techniques were used to treat clubfoot. (6) The first documented treatment for clubfoot by Hippocrates was similar to the Ponseti method used today. As early as 400 BC, Hippocrates described managing clubfoot with manipulations and bandages as if the foot was a 'wax model' which needed to be manipulated gently. Treatment options then varied between surgical and conservative management, with popular methods including the Kite method, French method and surgery. (6)

The Ponseti method initially had a slow uptake and has only been accepted widely within the past 15 years, despite its original description 52 years ago. The method now, however, is firmly the choice of management. The Ponseti method was found to be the treatment of choice among members of the Paediatric Orthopaedic Society of North America (POSNA), with 96.7% stating that it was their preferred method for the treatment of idiopathic clubfoot. (7) A series of reviews from around Europe and beyond have also yielded results showing the superiority of the Ponseti method. (8-10) When compared directly to surgical treatment, the Ponseti method has reduced stiffness and increased range of motion of the ankle joint, in addition to not being exposed to the risks of surgery. (11)

Congenital Talipes Equinovarus (CTEV/Clubfoot)

Clubfoot (CTEV) is the most common musculoskeletal condition in newborns, affecting one-to-two per thousand babies. (12) In most cases, the aetiology is unclear, and as such termed idiopathic CTEV. In other cases, it may be associated with a genetic syndrome such as spina bifida. (13) There are a number of hypotheses for the cause of CTEV, such as neuromuscular disease, (14) lack of foetal movement in utero (15) and genetic microduplication. (16) Boys are more affected than girls (2.5:1) (17) and the condition has a 50% chance of being bilateral. (18) It characteristically has four deformities, with forefoot cavus and adductus and hindfoot varus and equinus. (6) This leaves the foot looking like it has been internally rotated with the toes almost facing upwards towards the midline of the body.

From an anatomical point of view, the appearance is due to the talus being distorted and focussed laterally and the navicular being subluxated off the talar head so that it may articulate with the medial malleolus. The ankle and subtalar joints are in a fixed equinus, the calcaneus is in equinus, varus and rotated internally (Figure 1). These changes in the shape and position of the tarsal bones appear to have an excessive pull on the tibialis posterior, gastrocnemius, soleus, tibialis anterior and long toe flexors. The size of the leg muscles is negatively correlated with the severity of the clubfoot and the ligaments in the affected clubfeet are very thick and taut.

Casting and Manipulation

The Ponseti method to correct clubfoot is started within the first couple of weeks from birth. The method involves five-to-eight weeks of leg plaster casts, which run from the toe to the groin, which are changed once a week. Short leg casts are not used as they cannot hold the abduction and have the potential to slip off, recognised to be an important factor in the development of complex clubfoot (feet which significantly shortened with a rigid equinus and severe plantar flexion of all metastarsals). (19) During this time a number of stretches are used to help correct the deformity, which is recommended to be performed prior to casting (Figure 2). Firstly, the cavus is corrected with forefoot supination with pressure applied to the first metatarsophalangeal joint in order to raise it, meaning that the forefoot must be supinated to align it with the hindfoot in order to decrease cavus. The varus is then corrected by rotating the calcaneus and forefoot around the talus, with the head of the talus acting as a fulcrum, so that the foot is pointing now pointing outwards. It is important not to touch the calcaneus as it may block the motion of calcaneus which must be able to move from underneath talus. Finally, the equinus is corrected with stretches to dorsiflex the foot. It is important not to actively dorsiflex the foot prior to subtalar joint correction.

One of the most common errors which occurs during casting and stretching is the inadequate counter pressure on the talus. (20) Talus is very small in infants and is often more superior and anterior than would be expected. If counter pressure is applied too inferiorly or the pressure is over too broad of an area, calcaneus can be blocked and cannot move out from underneath the talus. When this happens, the abducting forces act on Lisfranc joint (the articulation between the midfoot and the forefoot) and the Chopart joint (transverse tarsal joint) causing abduction of the midfoot and possibly the formation of a lateral crease. This is regarded as a red flag during casting. (21)

In terms of the types of cast, Ponseti recommended a thin cast with little padding which should be moulded to the foot. The risk of the cast slipping can be prevented by using a cast with a well moulded heel and one which is high enough to reach the groin with the knee in at least 90® degrees of flexion. The second and third casts carry

the highest risk of slipping off, particularly if patients have a severe equinus and cavus. (21) Casts should only be removed just before the new cast is applied. Premature removal of casts the night before a new one is applied has been shown to result in a higher number of casts being required for correction. (22) The casts are usually changed once a week but accelerated protocols have also been reported; Morcudende et al. (5) found similar success rates when changing the casts every five days and Harnett et al. (23) found similar results when changing casts three times per week compared to a weekly change group. Shorter intervals between cast changes, however, may not be preferable. Pirani et al. suggested that tissues may need a certain amount of time in the corrected position within the cast in order to adapt. (24) This is due to changes in growth as a result of different mechanical loading in fast-growing tissues. (24)

CTEV severity and treatment progress using the Ponseti method is measured using the Pirani score. (25) The score shows whether the deformity is correcting normally or whether there is a problem. Scoring involves looking at six signs, three in the midfoot (such as the lateral head of the talus) and three in the hindfoot, (such as the posterior crease) and a score of 0, 0.5 or 1 is given depending on severity of the deformity. (25) A score of four or more means that the patient is likely to require at least four casts, whereas a purely hindfoot score of 2.5 has a high chance of requiring a tenotomy. (25)

Percutaneous Achilles tenotomy

Following casting, many patients will require a Percutaneous Achilles tenotomy (pAT) to relieve the remaining equinus. A pAT involves releasing the Achilles tendon from its insertion on the calcaneum. Ponseti originally reported that pAT was required in 79% of cases, (1) with subsequent studies reporting rates similar to this (80-90%). (5, 26) Timing of the procedure is crucial to the success of the procedure. Ponseti recommended pAT when there was less than 15-20 degrees of dorsiflexion and when the foot had been abducted to at least 60 degrees. (20) This is necessary as to allow the calcaneus to be able to move out completely from underneath talus, correcting the subtalar misalignment. If pAT is attempted before 60-70 degrees of abduction and before the correction of the subtalar alignment the hindfoot will likely stay uncorrected. After the operation, a post tenotomy cast should be applied and moulded in maximum abduction and dorsiflexion to achieve good correction. If the foot is not dorsiflexed after the tenotomy, this may result in insufficient dorsiflexion once the cast has been removed.

The tenotomy was originally recommended to be performed under local anaesthetic by Ponseti, however, a number of studies have used either local or general anaesthetic with both being found to be safe and effective. (27, 28) A recent review article concluded that pAT can be potentially performed under either local or general anaesthesia with the choice being mostly dependent on the setting and experience of the staff involved. (21)

Boots and braces

Following the casting and manipulation phases of treatment, the affected feet are fitted with open-toed boots attached to, and connected to one another, via a "Denis Browne bar". The bar-connected brace maintains the corrected foot in 60 to 70 degrees of external rotation on the affected side and in 30 to 40 degrees of external rotation on the normal side. The bar should be bent from 5 to 10 degrees and hold the feet in a valgus position with sufficient length so that the heels of the shoes are the same as the width of the shoulders. (29) Whilst wearing the brace, the shoes maintain the foot in 10 to 15 degrees of dorsiflexion. (30) Although the foot appears to be 'over-corrected' into abduction whilst in the brace, the result is not a real overcorrection but full abduction. (30)

Initially, Ponseti had described the use of such a foot abduction orthosis (FAO) following three months of a full time brace for an additional twenty-one months (average mean duration). (1) Ponseti found that this yielded a high recurrence rate and recommended that the FAO to be used at night for a number of years in his second paper. (2) These shoes are required to be worn for twenty-three hours a day for the following three months and then during the night for approximately four years. Following successful management with the Ponseti method, the patient has a pain-free, functional foot with good mobility. They are able to wear non-modified shoes.

One of the most significant risk factors for relapse of clubfoot after correction is non-compliance with the boots, with parental educational level being an important factor. (31) Studies have looked into ways of improving compliance, with the use of a dynamic brace (32) and using strategies to educate the parents and provide written instructions (33) both resulting in a higher level of compliance.

Results of treatment

The Ponseti method has been found to yield very good results for correcting clubfoot with an initial success rate of 90% (1) and 98% reported. (5) 90% of a group of patients Ponseti treated with his method were also found to be satisfied with the function and appearance of their feet when followed up nineteen years later. (3) Some of these patients were followed up a further eleven years later showed no deterioration of the appearance or function of their feet. (4)

Despite the high success rate using the Ponseti method, complications may arise. Overcorrection has been reported as a common complication with one study reporting 12.2% of feet being left with a valgus overcorrection. Overcorrection was also found to be a significant predictor of pain complaints following Ponseti therapy ($p=<0.001$). (34)

For patients who relapse, a tibialis anterior tendon transfer is recommended. This procedure was found to be performed in 21% of feet treated by Ponseti in a follow-up study. (35) A similar figure has been found in other studies. (36, 37) The procedure is especially recommended if the relapse is mostly dynamic supination and adduction. In this procedure it is recommended that a full transfer to the third cuneiform is performed. (1, 2) Relapse may occur after this procedure though; a recent study reviewed patients treated by tibialis anterior tendon transfer after initially having successful treatment from the Ponseti method. They found that relapse after tibialis anterior tendon transfer occurred in 15% of feet. (38)

Finding its feet in the UK

With over fifty years of use in the United States, the Ponseti method was popularised on British shores in 1997 in the North West with NHS Physiotherapist Steve Wildon. Mr Wildon approached Mr Paul Marshall, the current lead clinician for the Department of Orthopaedics and Trauma at the Royal Lancaster Infirmary, to start this form of treatment in the UK. The method has only grown in its use since then with a centre set up in Manchester dedicated to the treatment of clubfoot with the Ponseti method by Miss Naomi Davis, a Consultant Paediatric Orthopaedic Surgeon in Manchester. Miss Davis is a former registrar of Mr Paul Marshall. Miss Davis set up the Ponseti Clinic for Clubfoot Management in Manchester in early 2002.

There are, however, a number of variations in how the Ponseti method is used depending on the part of the country. Comparing the University Hospitals of Morecambe Bay Trust method of implementing the Ponseti method to the method at the Manchester clinic yields a couple of differences. Firstly, in Manchester, the stretches which are used during the casting phase are not performed. Secondly, in Manchester a Plaster of Paris is used compared to a soft cast which is used within the Morecambe Bay trust. Plaster of Paris is a cheaper alternative to the soft cast but it is harder to remove. This may be to do with the number of patients seen at the respective sites, as the Manchester site sees many more clubfeet due to the area it covers compared to the Morecambe Bay site, so more staff are available to remove the cast and having a cheaper cast makes treating these patients more economically viable.

Conclusion

Despite taking a number of decades to become the mainstay of treatment, the Ponseti method enables us to correct the majority of clubfeet with its conservative approach. In addition, the method is popular amongst patients, with follow-up studies from Ponseti showing high rates of satisfaction and no deterioration of appearance of function of the feet. The last decade has highlighted the popularity of the regime, reflected by the increasing body of literature analysing the Ponseti method. Research is now focusing on making the Ponseti method more effective by aiming for a shorter casting time or a reduced rate of relapse, using techniques such as an accelerated method, (23) adjuvant therapy with Botulinium A toxin (39) and different casting methods. (40)

References

1. Ponseti IV SE. Congenital club foot: the results of treatment. J Bone Joint Surg Am 1963;43(A):261-344.

2. Ponseti IV, Campos J. Observations on pathogenesis and treatment of congenital clubfoot. Clinical orthopaedics and related research. 1972;84:50-60.

https://doi.org/10.1097/00003086-197205000-00011

PMid:5032850

3. Laaveg SJ, Ponseti IV. Long-term results of treatment of congenital club foot. The Journal of bone and joint surgery American volume. 1980;62(1):23-31.

https://doi.org/10.2106/00004623-198062010-00004

PMid:7351412

4. Cooper DM, Dietz FR. Treatment of idiopathic clubfoot. A thirty-year follow-up note. The Journal of bone and joint surgery American volume. 1995;77(10):1477-89.

https://doi.org/10.2106/00004623-199510000-00002

PMid:7593056

5. Morcuende JA, Abbasi D, Dolan LA, Ponseti IV. Results of an accelerated Ponseti protocol for clubfoot. Journal of pediatric orthopedics. 2005;25(5):623-6.

https://doi.org/10.1097/01.bpo.0000162015.44865.5e

PMid:16199943

6. Miedzybrodzka Z. Congenital talipes equinovarus (clubfoot): a disorder of the foot but not the hand. Journal of anatomy. 2003;202(1):37-42.

https://doi.org/10.1046/j.1469-7580.2003.00147.x

PMid:12587918 PMCid:PMC1571059

7. Zionts LE, Sangiorgio SN, Ebramzadeh E, Morcuende JA. The current management of idiopathic clubfoot revisited: results of a survey of the POSNA membership. Journal of pediatric orthopedics. 2012;32(5):515-20.

https://doi.org/10.1097/BPO.0b013e318259ff79

PMid:22706469

8. Chotel F, Parot R, Durand JM, Garnier E, Hodgkinson I, Berard J. [Initial management of congenital varus equinus clubfoot by Ponseti's method]. Revue de chirurgie orthopedique et reparatrice

de l'appareil moteur. 2002;88(7):710-7.

9. Tindall AJ, Steinlechner CW, Lavy CB, Mannion S, Mkandawire N. Results of manipulation of idiopathic clubfoot deformity in Malawi by orthopaedic clinical officers using the Ponseti method: a realistic alternative for the developing world? Journal of pediatric orthopedics. 2005;25(5):627-9.

https://doi.org/10.1097/01.bpo.0000164876.97949.6b

PMid:16199944

10. Goksan SB, Bursali A, Bilgili F, Sivacioglu S, Ayanoglu S. Ponseti technique for the correction of idiopathic clubfeet presenting up to 1 year of age. A preliminary study in children with untreated or complex deformities. Archives of orthopaedic and trauma surgery. 2006;126(1):15-21.

https://doi.org/10.1007/s00402-005-0070-9

PMid:16283342

11. Švehlík M, Floh U, Steinwender G, Sperl M, Novak M, Kraus T. Ponseti method is superior to surgical treatment in clubfoot – Long-term, randomized, prospective trial. Gait & Posture. 2017;58:346-51.

https://doi.org/10.1016/j.gaitpost.2017.08.010

PMid:28866453

12. Dobbs MB, Nunley R, Schoenecker PL. Long-term follow-up of patients with clubfeet treated with extensive soft-tissue release. The Journal of bone and joint surgery American volume. 2006;88(5):986-96.

https://doi.org/10.2106/JBJS.E.00114

https://doi.org/10.2106/00004623-200605000-00009

PMid:16651573

13. Swaroop VT, Dias L. Orthopaedic management of spina bifida—part II: foot and ankle deformities. Journal of children's orthopaedics. 2011;5(6):403-14.

https://doi.org/10.1007/s11832-011-0368-9

PMid:23205142 PMCid:PMC3221758

14. Lovell ME, Morcuende JA. Neuromuscular disease as the cause of late clubfoot relapses: report of 4 cases. Iowa Orthop J. 2007;27:82-4.

PMid:17907435 PMCid:PMC2150663

15. Hester TW, Parkinson LC, Robson J, Misra S, Sangha H, Martin JE. A hypothesis and model of reduced fetal movement as a common pathogenetic mechanism in clubfoot. Med Hypotheses. 2009;73(6):986-8.

https://doi.org/10.1016/j.mehy.2009.04.056

PMid:19786327

16. Peterson JF, Ghaloul-Gonzalez L, Madan-Khetarpal S, Hartman J, Surti U, Rajkovic A, et al. Familial microduplication of 17q23.1-q23.2 involving TBX4 is associated with congenital clubfoot and reduced penetrance in females. Am J Med Genet A. 2014;164A(2):364-9.

https://doi.org/10.1002/ajmg.a.36238

PMid:24592505

17. Wallander H, Hovelius L, Michaelsson K. Incidence of congenital clubfoot in Sweden. Acta orthopaedica. 2006;77(6):847-52.

https://doi.org/10.1080/17453670610013123

PMid:17260191

18. Huntley JS. Optimising the management of congenital talipes. The Practitioner. 2013;257(1765):15-8, 2.

19. Ponseti IV, Zhivkov M, Davis N, Sinclair M, Dobbs MB, Morcuende JA. Treatment of the complex idiopathic clubfoot. Clinical orthopaedics and related research. 2006;451:171-6.

https://doi.org/10.1097/01.blo.0000224062.39990.48

PMid:16788408

20. Ponseti IV. Common errors in the treatment of congenital clubfoot. International orthopaedics. 1997;21(2):137-41.

https://doi.org/10.1007/s002640050137

PMid:9195271 PMCid:PMC3616653

21. Radler C. The Ponseti method for the treatment of congenital club foot: review of the current literature and treatment recommendations. International orthopaedics. 2013;37(9):1747-53.

https://doi.org/10.1007/s00264-013-2029-8

https://doi.org/10.1007/s00264-013-2031-1

PMid:23928728 PMCid:PMC3764299

22. Terrazas-Lafargue G, Morcuende JA. Effect of cast removal timing in the correction of idiopathic clubfoot by the Ponseti method. The Iowa orthopaedic journal. 2007;27:24-7.

PMid:17907426 PMCid:PMC2150656

23. Harnett P, Freeman R, Harrison WJ, Brown LC, Beckles V. An accelerated Ponseti versus the standard Ponseti method: a prospective randomised controlled trial. The Journal of bone and joint surgery British volume. 2011;93(3):404-8.

https://doi.org/10.1302/0301-620X.93B3.24450

PMid:21357965

24. Pirani S, Zeznik L, Hodges D. Magnetic resonance imaging study of the congenital clubfoot treated with the Ponseti method. Journal of pediatric orthopedics. 2001;21(6):719-26. https://doi.org/10.1097/00004694-200111000-00004

https://doi.org/10.1097/01241398-200111000-00004

PMid:11675543

25. Dyer PJ, Davis N. The role of the Pirani scoring system in the management of club foot by the Ponseti method. The Journal of bone and joint surgery British volume. 2006;88(8):1082-4.

https://doi.org/10.1302/0301-620X.88B8.17482

PMid:16877610

26. Herzenberg JE, Radler C, Bor N. Ponseti versus traditional methods of casting for idiopathic clubfoot. Journal of pediatric orthopedics. 2002;22(4):517-21.

https://doi.org/10.1097/01241398-200207000-00019

https://doi.org/10.1097/00004694-200207000-00019

PMid:12131451

27. Lebel E, Karasik M, Bernstein-Weyel M, Mishukov Y, Peyser A. Achilles tenotomy as an office procedure: safety and efficacy as part of the Ponseti serial casting protocol for clubfoot. Journal of pediatric orthopedics. 2012;32(4):412-5.

https://doi.org/10.1097/BPO.0b013e31825611a6

PMid:22584844

28. Parada SA, Baird GO, Auffant RA, Tompkins BJ, Caskey PM. Safety of percutaneous tendoachilles tenotomy performed under general anesthesia on infants with idiopathic clubfoot. Journal of pediatric orthopedics. 2009;29(8):916-9.

https://doi.org/10.1097/BPO.0b013e3181c18ab5

PMid:19934709

29. Zhao D, Liu J, Zhao L, Wu Z. Relapse of clubfoot after treatment with the Ponseti method and the function of the foot abduction orthosis. Clinics in orthopedic surgery. 2014;6(3):245-52.

https://doi.org/10.4055/cios.2014.6.3.245

PMid:25177447 PMCid:PMC4143509

30. Staheli L. Ponseti Management. Seattle, WA.: Global HELP; 2009 [accessed 22 June 2018]. Available from: http://www.global-help.org/publications/books/book_cfponseti.html.

31. Dobbs MB, Rudzki JR, Purcell DB, Walton T, Porter KR, Gurnett CA. Factors predictive of outcome after use of the Ponseti method for the treatment of idiopathic clubfeet. The Journal of bone and joint surgery American volume. 2004;86-A(1):22-7.

https://doi.org/10.2106/00004623-200401000-00005

PMid:14711941

32. Garg S, Porter K. Improved bracing compliance in children with clubfeet using a dynamic orthosis. Journal of children's orthopaedics. 2009;3(4):271-6.

https://doi.org/10.1007/s11832-009-0182-9

PMid:19495824 PMCid:PMC2726866

33. Zionts LE, Dietz FR. Bracing following correction of idiopathic clubfoot using the Ponseti method. The Journal of the American Academy of Orthopaedic Surgeons. 2010;18(8):486-93. https://doi.org/10.5435/00124635-201008000-00005

PMid:20675641

34. Hayes CB, Murr KA, Muchow RD, Iwinski HJ, Talwalkar VR, Walker JL, et al. Pain and overcorrection in clubfeet treated by Ponseti method. Journal of pediatric orthopedics Part B. 2018;27(1):52-5.

35. Bor N, Coplan JA, Herzenberg JE. Ponseti treatment for idiopathic clubfoot: minimum 5-year followup. Clinical orthopaedics and related research. 2009;467(5):1263-70.

https://doi.org/10.1007/s11999-008-0683-8

PMid:19130158 PMCid:PMC2664421

36. Eberhardt O, Peterlein CD, Fernandez FF, Wirth T. [Mid-term results of idiopathic clubfeet treated with the Ponseti method]. Zeitschrift fur Orthopadie und Unfallchirurgie. 2012;150(2):190-7.

https://doi.org/10.1055/s-0031-1298271

PMid:22354441

37. Church C, Coplan JA, Poljak D, Thabet AM, Kowtharapu D, Lennon N, et al. A comprehensive outcome comparison of surgical and Ponseti clubfoot treatments with reference to pediatric norms. Journal of children's orthopaedics. 2012;6(1):51-9.

https://doi.org/10.1007/s11832-012-0387-1

PMid:23449014 PMCid:PMC3303018

38. Masrouha KZ, Morcuende JA. Relapse after tibialis anterior tendon transfer in idiopathic clubfoot treated by the Ponseti method. Journal of pediatric orthopedics. 2012;32(1):81-4.

https://doi.org/10.1097/BPO.0b013e31823db19d

PMid:22173393

39. Cummings RJ. The effectiveness of botulinum A toxin as an adjunct to the treatment of clubfeet by the Ponseti method:

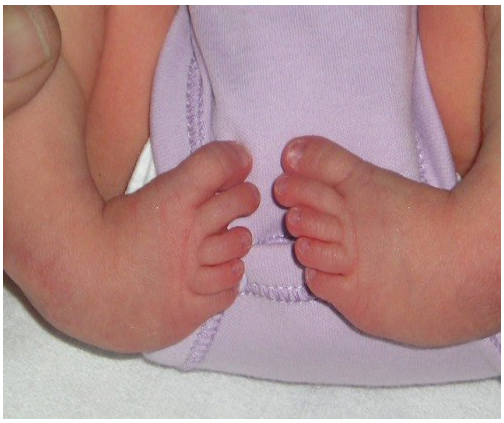
a randomized, double blind, placebo controlled study. J Pediatr Orthop. 2009;29(6):564-9.

40. Pittner DE, Klingele KE, Beebe AC. Treatment of clubfoot with the Ponseti method: a comparison of casting materials. J Pediatr Orthop. 2008;28(2):250-3.

https://doi.org/10.1097/BPO.0b013e318164f8e7

PMid:18388724

Figures



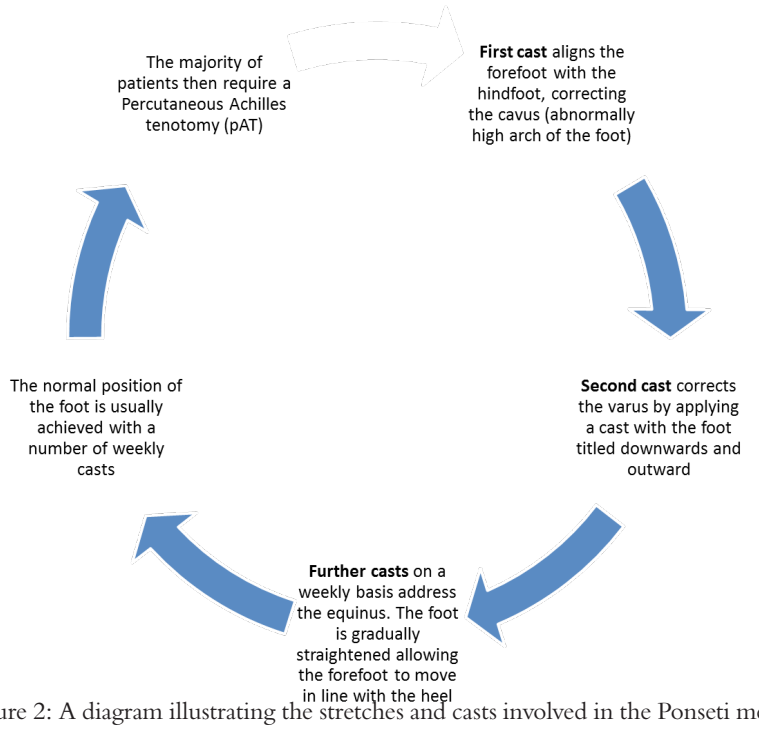


Figure 2: A diagram illustrating the stretches and casts involved in the Ponseti method

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Education

Cannabis - Current Trends and Methods of Bioavailability

EDUCATION

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ABSTRACT

Summary
There is a growing global trend towards the decriminalisation of cannabis use. Several countries have already legalised this drug for medicinal and recreational use, and more are expected to continue this trend in the future. Inhalation and ingestion of cannabis are the two basic methods of consumption, and each provides unique bioavailability mechanisms. Anticipating the increasing social and legal acceptance of cannabis, this article provides a basic understanding of the geographic acceptance, methods of use, bioavailability of active ingredients, and mechanisms of absorption in the human body.

Relevance
The growing acceptance of cannabis increases the probability that current medical students will encounter patients using cannabis in some form. This is a significant trend especially considering the psychoactive effects of the drug and the medicinal benefits. Having a basic knowledge of the methods of consumption and the longevity of cannabinoid compounds in the human body will help students and practicing physicians better diagnose patients and implement optimum wellness plans.

Take Home Message
As societies and lawmakers increasingly accept the use of cannabis, it's important that medical professionals have a basic knowledge of the various delivery methods and the impact of cannabinoid compounds in the human body. Patient care can be enhanced through a better understanding of how the drug is used and how it affects the human body. This educational piece provides a step in that direction.

Introduction

The global decriminalisation of cannabis is increasingly hitting the newswires these days. With a long-recorded history of human use, this drug contains approximately 60 pharmacologically active cannabinoid compounds including tetrahydrocannabinol (THC) and cannabidiol (CBD), which are known for their psychoactive and non-psychoactive effects respectively. (1, 2) While the popularity of cannabis as a recreational drug is rooted in its ability to alter sensory perception, a growing body of research also points to several medical benefits, including the relief of chronic and neuropathic pain, and as a treatment for insomnia, anxiety, epileptic seizures and nausea. (3, 4, 5, 6, 7, 8)

The Growing Acceptance of Cannabis

The attractiveness of cannabis is growing worldwide. According to the United Nations Office on Drugs and Crime, about 3.8 percent of the global population, or an estimated 183 million people, use cannabis making it the third most popular recreational drug in the world, behind alcohol and tobacco. (9) More importantly, while it is still an illicit substance in many parts of the world, there is a growing trend towards legalisation for medical and recreational use. (10, 11) For example, in Uruguay and Portugal, cannabis is now completely legal, while in countries such as the Czech Republic, Finland, Norway, the Netherlands, Spain and Greece, cannabis is legal for medicinal use or decriminalised in some form. (12, 13) Across the pond in North America, there are 29 U.S. states that have legalised medicinal cannabis and 9 of these have also legalised it for recreational use. (14) This global momentum towards acceptance will continue with the Canadian government slated to become the first G7 country planning to fully decriminalise marijuana in 2018, while further U.S. legalisation is also underway in 8 additional states. (15-17) Although cannabis is still illegal in the United Kingdom, attitudes are changing with roughly half of the public (47%) supporting sale of the drug through licensed shops. (18)

Bioavailability and Longevity of Cannabinoid Compounds

With this growing acceptance, it is especially useful for medical professionals to know how the drug is consumed and the bioavailability of cannabinoid compounds upon entering the body. Firstly, we know that the drug is typically inhaled or ingested, and that smoking is the most common method of inhalation. The bioavailability of the active ingredients and how quickly the psychotropic effects are experienced are affected by several factors, including potency, ratio of THC to CBD, method of delivery, stomach contents, metabolism, biochemistry and interaction with other medications. The act of smoking cannabis provides the highest bioavailability and involves the decarboxylation of the naturally existing tetrahydrocannabinolic acid (THCA) into THC (delta-9 THC). These active ingredients then travel through the airway and into the lungs where they are absorbed into the bloodstream resulting in maximum plasma concentration within minutes. One study found that inhalation resulted in bloodstream absorption rates of 10 to 45 percent, with bioavailability peaking 3 to 10 minutes after inhalation. (19) Psychoactive effects and increased heart rate are experienced between 30 seconds and 2 minutes after inhalation once the THC compounds reach the brain – and taper off within 2 to 3 hours. (20) In contrast, when cannabis is ingested within foods or drinks it is processed in the highly acidic environment of the stomach, which damages the cannabinoid molecules and reduces bioavailability. Nevertheless, remaining molecules are absorbed in the gastrointestinal tract and liver, which metabolise the THC into 11-hydroxyl-delta-9-THC, a smaller lipophilic molecule able to cross the blood-brain barrier. This results in a more prolonged psychotropic effect compared to inhalation. In two different studies, oral administration resulted in an absorption rate between 4 to 20 percent - the effects are experienced 30 minutes to 2 hours after ingestion and can last up to 8 hours. (21, 22) As an alternative delivery method, there are also cannabis-infused balms, oils and lotions that are absorbed through the skin. This approach activates local cannabinoid receptors and is often used for localized pain relief without the broader psychoactive effects. With all of these methods, the external cannabinoids act on the body's own endocannabinoid system and stimulate cannabinoid CB1 and CB2 receptors, which are involved in the homeostasis of biological functions. For example, the anti-nausea effects of THC are mediated by the interactions of THC with CB1 receptors in the dorsal vagal complex. (23) In contrast, CBD, the second most common cannabinoid, exerts antiemetic properties through other mechanisms and different pathways in the brain resulting in few psychotropic effects.

Conclusion

Societies and lawmakers are increasingly accepting the use of cannabis, especially in light of a growing body of research pointing to various medical benefits. This has resulted in a global trend towards decriminalization, which is expected to encourage future use. Understanding the basic delivery methods of inhalation and ingestion, and the corresponding bioavailability and longevity of cannabis compounds is important in order to better diagnose patients and implement optimum wellness plans.

References

1. Dewey WL. Cannabinoid pharmacology. Pharmacological Review. 1986;38:151–178.

PMid:3529128

2. Russo EB, Marcu J. Cannabis Pharmacology: The Usual Suspects and a Few Promising Leads. Advances in Pharmacology. 2017;80:67-134.

https://doi.org/10.1016/bs.apha.2017.03.004

PMid:28826544

3. National Academies of Sciences, Engineering and Medicine. The Health Effects of Cannabis and Cannabinoids: The Current State of Evidence and Recommendations for Research. 2017 [accessed 20 Feb 18]. Available from: https://www.nap.edu/read/24625/chapter/6.

4. Hill KP. Medical Marijuana for Treatment of Chronic Pain and Other Medical and Psychiatric Problems: A Clinical Review. JAMA. 2015;313(24):2474-83.

https://doi.org/10.1001/jama.2015.6199

PMid:26103031

5. Abrams DI, Jay CA, Shade SB, et al. Cannabis in Painful HIV-Associated Sensory Neuropathy: A Randomized Placebo-Controlled Trial. Neurology. 2007;68(7).

https://doi.org/10.1212/01.wnl.0000253187.66183.9c

PMid:17296917

6. Cooper ZD, Comer SD, Haney M. Comparison of the Analgesic Effects of Dronabinol and Smoked Marijuana in Daily Marijuana Smokers. Neuropsychopharmacology. 2013;38(10):1984-92.

https://doi.org/10.1038/npp.2013.97

PMid:23609132 PMCid:PMC3746706

7. Russo EB, Guy GW, Robson PJ. Cannabis, Pain, and Sleep: Lessons from Therapeutic Clinical Trials of Sativex, a Cannabis-based Medicine. Chemistry & Diodiversity. 2007;4(8):1729-1743.

8. Porcari GS, Fu C, Doll ED, et al. Efficacy of artisanal preparations of cannabidiol for the treatment of epilepsy: Practical experiences in a tertiary medical center. Epilepsy & Behavior. 2018;80:240-246.

https://doi.org/10.1016/j.yebeh.2018.01.026

PMid:29429908

9. UNODC - United Nations Office on Drug and Crime. World Drug Report. 2017 [accessed 20 Feb 2018]. Available from: https://www.unodc.org/wdr2017/field/Booklet_2_HEALTH.pdf

10. Gupta S. Why I Changed my Mind on Weed, CNN. 2013 [accessed 20 Feb 2018]. Available from: https://www.cnn.com/2013/08/08/health/gupta-changed-mind-marijuana/index.html.

11. Ingraham C. Just how Mainstream is Marijuana? There is a Congressional Cannabis Caucus. The Washington Post. 2016 [accessed 20 Feb 2018]. Available from: http://www.washingtonpost.com/news/wonk/wp/2017/02/17/just-how-mainstream-is-marijuana-theres-now-a-congressional-cannabis-caucus/?utm_term=.9dab402901c6.

12. Goni U. Uruguay, the First Country where you can Smoke Marijuana wherever you like. The Guardian. 2017 [accessed 20 Feb 2018]. Available from: https://www.theguardian.com/society/2017/may/27/marijuana-legalisation-uruguay-seen-half-measure-users.

13. Masters S. Greece becomes sixth EU country to legalise cannabis for medicinal purposes. 2017 [accessed 20 Feb 2018]. Available from: https://www.express.co.uk/news/world/824491/Greece-legalises-cannabis-for-medical-purposes.

14. Barcott, B. Marijuana Goes Main Stream. New York; Time. 2017 [accessed 20 Feb 2018]. Available from: https://shop.time.com/storefront/books/time-marijuana-goes-main-street/prodTDSHOPMARIJUANABZ.html.

15. Government of Canada. Legalization and Regulation of Cannabis. 2018 [accessed 20 Feb 2018]. Available from: https://www.canada.ca/en/services/policing/justice/legalization-regulation-marijuana.html.

16. Power M. The wild west of weed: will legalization work for Canada. The Guardian. 2017 [accessed 20 Feb 2018]. Available from: https://www.theguardian.com/society/2017/mar/29/the-wild-west-of-weed-will-legalisation-work-for-canada.

17. Sanders L. 2018. Marijuana Legalization 2018: Which States Might Consider Cannabis Laws this Year? Newsweek. 2018 [accessed 20 Feb 2018]. Available from: http://www.newsweek.com/marijuana-legalization-2018-which-states-will-consider-cannabis-laws-year-755282.

18. Grice A. Cannabis legalization: 47% support sale of drug through licenced shops, poll reveals. The Independent. 2016 [accessed 20 Feb 2018]. Available from: http://www.independent.co.uk/news/uk/home-news/legalising-cannabis-47-support-sale-of-drug-through-licensed-shops-poll-reveals-a6976796.html

19. Grotenhermen F.Pharmacokinetics and Pharmacodynamics of Cannabinoids. Clinical Pharmacokinetics. 2003;42(4):327-60.

https://doi.org/10.2165/00003088-200342040-00003

PMid:12648025

20. Wolf L., Wolf M. The Medical Marijuana Dispensary. USA: Althea Press; 2016.

21. Brenneisen R, Egli A, Elsohly MA, et al. The Effect of Orally and Rectally Administered Delta 9-Tetrahydrocannabinol on Spasticity: A Pilot Study with 2 Patients. International Journal of Clinical Pharmacology and Therapeutics. 1996;34(10).

PMid:8897084

22. Martin BR et al. Chemistry and Pharmacology of Cannabis. In: Kalant, H., Corrigall WA., Hall W., Smart RG., editors. The Health Effects of Cannabis. Toronto: CAMH; 1999. p.19-68.

23. Pacher P, Batkai S, Kunos G. The Endocannabinoid System as an Emerging Target of Pharmacotherapy. Pharmacological Reviews 58. 2006;3:389-462.

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Education

Recent advances in our understanding of the pathobiology of non-coding RNA

EDUCATION

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ABSTRACT

Summary

The central dogma of molecular biology recognised RNA as a mere intermediate between DNA sequence and proteins, the main protagonists of cellular functions. The shadow of a wrinkle in this concept appeared when the Human Genome Project revealed that protein-coding genes only account for 2% of our genome and the remaining 98% was originally dismissed as elusive "junk DNA."

Relevance

Recent advances in next-generation sequencing provided tantalising evidence that 70-90% of human genome is actively transcribed(1). Numerous studies started discerning the functional importance of long and short (>200 and <200 nucleotides, respectively) non-coding transcripts far beyond the initial textbook-engraved infrastructural and information-transmitting role of rRNA, tRNA and mRNA.

Take Home Messages

By "rediscovering the junk," this essay reviews a selection of recent disease-associated advances in the field of small non-coding RNAs before extending the discussion to long non-coding RNAs and finally to the overarching concepts of competing endogenous RNAs (ceRNAs) and 'social' RNA.

RNA interference (RNAi)

RNA interference is a process whereby micro RNA (miRNA) and small interfering RNA (siRNA) molecules inhibit target gene expression by acting on mRNA. The first miRNA to be identified, lin-4, was discovered as a short non-coding RNA required for repression of lin-14 gene and subsequent maturation of C. elegans larvae. (2) The sequence of lin-4 displayed complementarity to the 3' untranslated region (UTR) of lin-14 mRNA, (2) suggesting that lin-4 regulates lin-14 expression through an RNA-RNA interaction. The discovery of let-7 miRNA (3) and its conservation from worms to humans (4) first underscored the prevalence and functional importance of miRNA-dependent gene regulation. Although estimated to represent less than one percent of our genome, miRNAs have been proposed to regulate as many as 92% of human genes. (5)

What is the mechanism of gene regulation by miRNAs? The process of miRNA biogenesis typically starts with RNA polymerases transcribing miRNA genes into primary miRNAs, which fold to form hairpin-loop structures (Figure 1). These are recognised and cleaved by the Microprocessor complex to form precursor miRNAs (pre-miRNAs). Following nuclear export, pre-miRNAs are cleaved by a protein complex involving Dicer endonuclease and one of the resulting 21-23bp strands assembles with Argonaute proteins into an RNA-induced silencing complex (RISC). Using miRNA as a guide, RISC binds to complementary miRNA-response elements (MREs) in 3' untranslated region (UTR) of target mRNAs leading to mRNA cleavage or translational inhibition. While miRNAs are endogenous, siRNAs are exogenous RNA duplexes that enter the cell, are cleaved by Dicer and follow the same processing and functional path as miRNAs.

About 20 years since their discovery and landmark observations, miRNAs are recognised as regulators of most cellular processes, with their derangements reported in a host of human disease.

miRNAs in tumorigenesis

Tumorigenesis is generally associated with gain-of-function mutations or amplification of oncogenes and/or loss-of-function mutations in tumour suppressor genes (TSG). Oncomirs, i.e. miRNAs that promote cancer, typically repress TSGs. The polycistronic cluster miR-17-92 is located in a region of chromosome 13q and is frequently amplified in tumours. (6) The binding of oncprotein MYC to this cluster drives the expression of six miRNAs with reported roles in cell proliferation, apoptosis and angiogenesis. (7) Overexpression of this cluster in MYC-driven mouse model of lymphoma accelerated tumorigenesis by suppressing apoptosis, (8) identifying miR-17-92 as an oncomir normally acting downstream of MYC.

The first evidence for altered miRNA expression in cancer came from genome-wide studies in patients with B-cell chronic lymphocytic leukaemia (B-CLL), the most prevalent type of adult leukaemia. Using somatic cell hybridisation, 68% of B-CLL patients were found to carry a deletion in the gene cluster encoding miR-15 and miR-16 miRNAs. (9) Prolonged survival of B cells in B-CLL corresponded to increased expression of the anti-apoptotic protein Bcl-2 (among other targets), a process successfully reversed by forced expression of miR-15 and miR-16 into cultured B cells of B-CLL patients. (10) This demonstrated the tumour-suppressing function of miR-15 and miR-16, which was confirmed in mouse xenograft experiments: human prostatic tumour grafts transfected with miR-15 and mir-16 displayed increased regression compared to untransfected controls. (11) Hence, miRNAs can function as TSGs and their restoration bears therapeutic promise.

Human cancers typically show a global decrease in miRNA expression, (12) which could reflect the undifferentiated state of tumours or causally contribute to the transformed phenotype. To answer this question, Kumar and co-workers generated mouse and human lines deficient in miRNA processing proteins Drosha or DGCR8 (components of the microprocessor complex) and Dicer1. These lines displayed a substantial decrease in steady state miRNA levels, increased growth in soft agar cultures and more malignant behaviour when injected into nude mice compared to controls, (12) demonstrating that miRNA pathways tend to act as an overall brake on cancer growth rather than simply reflecting the undifferentiated state. Interestingly, overexpression of let-7 miRNA alone reversed the in vitro effects of Dicer1 knockdown (12), implying that let-7 may be the key TSGs among miRNAs. Let-7 miRNAs target the KRAS oncogene, (13) whose properties have to date thwarted all therapeutic efforts using small molecule inhibitors. Hence, overexpression of let-7 miRNA provides an alternative approach to target KRAS, which is frequently mutated in human cancer. (14)

miRNAs in immunity

One caveat in therapeutic application of miRNAs is that a single miRNA typically induces pleiotropic effects in cell types with different transcriptional profiles. While let-7 suppresses tumorigenesis in cancer cells, (13) it was demonstrated to play a diametrically opposite role in tumour-associated macrophages (TAMs). (15) TAMs typically exist as two populations: tumour-reactive (M1) macrophages attack tumour cells whereas tumour-tolerant (M2) macrophages supress anti-tumour immunity. Global ablation of miRNAs by deletion of Dicer1 in mice resulted in polarisation of M2 towards M1 population and boosted anti-tumour reactivity. (15) This process was rescued by forced expression of let-7 miRNA, (15) suggesting that the tumour-tolerant M2 phenotype is chiefly maintained by let-7 miRNA.

In adaptive immunity, tolerance mechanisms including regulatory T (Treg) cells prevent other T cells from reacting against self-proteins, thereby limiting autoimmune disease. Tregs exist in two

populations: natural nTregs differentiate in the thymus whereas induced iTregs are induced by IL-2 and TGFβ stimulation of naïve CD4 T cells in the periphery. nTregs and iTregs differ epigenetically (16) and iTregs were shown to lose Treg markers (and immunosuppressive function) and differentiate into exTreg cells (16) (Figure 2). The latter recognise self-antigens and can, instead of suppressing autoimmunity, actually attack self-antigens themselves. (17) This raises the question of what causes iTregs to become exTregs? Immunosuppressive Treg cells and immunoreactive TH17 cells have diametrically opposite functions, yet share many differentiation factors. In the presence of IL-1β (from activated monocytes) and IL-2, iTregs downregulate the canonical Treg transcription factor Foxp3 (i.e. become exTregs) and start producing pro-inflammatory IL-17, thus functionally resembling TH17 cells. (18) Interestingly, the transition from iTreg cells to TH17-like exTreg cells has been shown to involve miRNAs. Peripheral CD4 T cells from humans with systemic lupus erythematosus and Crohn's disease as well as autoimmune mouse models demonstrated downregulation of miR-125a, which stabilises both the commitment and regulatory capacity of Treg cells. (19) Mice deficient in miR-125a spontaneously developed autoimmune disease, which was successfully reversed using synthetic miR-125a analogues. (19) Hence, miR-125a serves as a transcriptional regulator of Treg cells whose forced re-expression can re-wire aberrant Treg cells back to normal physiological function and prevent autoimmunity.

tRNA fragments in transgenerational epigenetic inheritance

Besides the conventional role of tRNAs in translation, striking new evidence suggests an active role of tRNA fragments (tRFs) in transgenerational inheritance independent of changes in DNA sequence. Using in vitro fertilisation, offspring of males consuming a low-protein diet exhibited significant hepatic up-regulation of squalene epoxidase (involved in cholesterol biosynthesis) compared to controls, implying that paternal diet can affect offspring metabolism via information located in sperm. (20) Deep sequencing of small RNAs in sperm of low-protein diet versus control males demonstrated the former to have increased abundance of tRFs, whose injection into normal zygotes recapitulated the metabolic phenotype of tRF donors. (21) Locked nucleic acid (LNA) oligonucleotides blocking tRFs derived from the 5' end of glycine tRNA resulted in dramatic up-regulation of about 70 genes, (22) demonstrating that tRFs can profoundly influence the transcriptional profile of the zygote. Hence, sperm tRFs present a paternal epigenetic factor mediating transgenerational inheritance of diet-induced metabolic disorders.

Long non-coding RNAs

While short non-coding RNAs mainly affect mRNA stability and translation, the functional repertoire of long non-coding (lnc) RNAs is considerably more versatile. This includes chromatin binding and epigenetic regulation, protein scaffolding and "sponging" of miRNAs as discussed below.

Selective targeting of mitochondrial metabolism in melanoma through the lncRNA SAMMSON

The survival of several tumours requires oxidative phosphorylation, which stimulated the development of metabolism-targeting anti-cancer agents. Single nucleotide polymorphism (SNP) array data revealed that focal amplifications of chromosome 3p characteristic of melanoma invariably encompass a newly annotated lncRNA gene SAMMSON, which was found to be selectively expressed in more than 90% of human primary and metastatic skin melanomas as demonstrated by RNA sequencing. (23) Prognosis and clinical success of current anti-melanoma therapies is associated with mutational status of BRAF, NRAS and TP53 of the tumour while chronic treatment with novel BRAF inhibitors, e.g. vemurafenib, invariably leads to drug resistance. (24) Promisingly, silencing SAMMSON using antisense oligonucleotides reduced proliferation of all melanoma cultures independently of BRAF, NRAS and p53 status and preserved sensitivity to BRAF inhibitors. (23) RNA pull down and mass spectroscopy identified a SAMMSON-associated protein, p32, known for its role in maintenance of mitochondrial membrane potential and oxidative phosphorylation. (25) Antisense nucleic acid-induced silencing of SAMMSON decreased mitochondrial membrane potential and triggered apoptosis, and the effect was rescued by reintroduction of p32. (23) Hence, SAMMSON silencing inhibits the survival of melanoma at least partly by disrupting p32-mediated mitochondrial functions.

How does impaired mitochondrial function hinder cell proliferation? Collapsed mitochondrial membrane potential impairs the import of cytoplasmic proteins into mitochondria, which leads to toxic accumulation of mitochondrial precursors in the cytosol, stalling of the cell cycle and/or apoptosis. (26) The relevance of this mechanism was confirmed by exposure of human melanoma cells to inhibitors of translation, which abolished the lethal effect of SAMMSON silencing. (23) Importantly, intravenous treatment with SAMMSON-silencing antisense nucleic acids significantly suppressed the growth of human melanoma tumours injected into mice, (23) identifying SAMMSON as an attractive therapeutic target for the disruption of mitochondrial metabolism selectively in melanoma.

Competing endogenous RNA (ceRNA)

Specific miRNA molecules can be repressed experimentally using chemically modified antisense oligonucleotides or artificial miRNA sponges. The latter are synthetic RNA constructs harbouring multiple miRNA-binding elements (MREs). The competition for shared miRNAs between decoy MREs of the sponge and functional MREs of natural miRNA target transcripts titrates the amount of functional miRNA within the cell. Growing

understanding of human transcriptome led to postulating the existence of natural RNA sponges, whereby all (co-localised) RNA transcripts that contain MREs can communicate with and regulate each other by competing for shared miRNAs, thus acting as competing endogenous RNAs (ceRNAs) (27). In this way, protein-coding mRNAs may possess an additional non-coding function as ceRNAs. Though ceRNA research is still in infancy, the ceRNA role of lncRNAs, (28) mRNAs (29) and circular RNAs (30) has already been reported. For example, the non-coding PTENP1 pseudogene has been reported to regulate the levels of its cognate gene, the tumour suppressor PTEN, by competing for shared miRNAs. (31) PTENP1 pseudogene is frequently lost in human cancers, (31) which results for uncontrolled repression of PTEN protein by miRNAs. Intriguingly, comparison of cultured human transformed versus untransformed cells revealed that the former express mRNAs with shorter 3' UTR regions. (32) While reducing their regulation by miRNA, this property also diminishes their ceRNA role. The functional consequence of this, if any, still remains to be elucidated.

RNA trafficking and the controversy of 'social' RNA

Exosomes are plasma-membrane-derived vesicles that transfer proteins, mRNAs, miRNAs and other ncRNAs between cells. Returning to the study of sperm tRNA fragments (tRFs) in transgenerational inheritance: deep sequencing of sperm cells at different locations along the epididymis demonstrated that all sperm cells contained comparable amounts of tRNAs, yet the abundance of tRFs increased distally along the epididymis. (22) This suggests that tRFs are not the product of tRNA degradation within the sperm. Instead, deep sequencing showed the sperm tRFs to be derived from exosome-like vesicles released by epididymis termed epididymosomes, (22) suggesting that functional ncRNAs can participate in intracellular communication.

miRNAs are trafficked around the body in high-density lipoprotein (HDL) particles. (33) The HDL-miRNA profile of healthy human subjects was found to be significantly different from patients with familial hypercholesterolemia and the two types of HDL induced different transcriptional profiles in cultured human hepatocytes as demonstrated by miRNA microarrays. (33) This demonstrated that HDL-transported miRNAs are functional, differ in disease states and can alter the transcriptome of target tissues.

Lastly and most controversially, the 'social' RNA hypothesis posits that the RNA world extends beyond the boundaries of an organism. (34) Within numerous limitations, RNA-containing complexes in the environment could influence gene expression profiles between animals of the same species as well as across species. Using miRNA microarray profiling of HDL-miRNA complexes, healthy human subjects in regular contact with different domesticated animals were demonstrated to possess different HDL-miRNA profiles that clustered based on the type of animal they were in contact with. (35) Furthermore, the differentially abundant miRNA species were shown to differentially regulate the pro-inflammatory NFκB pathway in cultured human macrophages, (35) contentiously suggesting that the type of animal one regularly contacts could influence one's susceptibility to inflammation.

Conclusion

With comparable numbers of protein coding genes spanning the evolutionary tree from simple worms to humans, complexity is now believed to reside largely in the non-coding part of the genome previously dismissed as junk DNA or transcriptional noise. Unravelling the intricacies of this vast RNA underworld has already revealed numerous pathophysiological mechanisms, only a handful of which are presented here. To date, the role of some newly defined ncRNA classes, e.g. agotrons, (36) extra-coding RNAs (37) and enhancer RNAs (38) remains poorly characterised, which, together with the likely prospect of more ncRNA species still to be defined, heralds a profuse expansion of the newly (re)discovered layer of gene regulation and potential therapeutic applications.

References

1. Wilhelm BT, Marguerat S, Watt S, Schubert F, Wood V, Goodhead I, et al. Dynamic repertoire of a eukaryotic transcriptome surveyed at single-nucleotide resolution. Nature. 2008;453(7199):1239–43.

https://doi.org/10.1038/nature07002

PMid:18488015

2. Lee RC, Feinbaum RL, Ambros V. The C. elegans heterochronic gene lin-4 encodes small RNAs with antisense complementarity to lin-14. Cell. 1993;75(5):843–54.

3. Reinhart BJ, Slack FJ, Basson M, Pasquinelli AE, Bettinger JC, Rougvie AE, et al. The 21-nucleotide let-7 RNA regulates developmental timing in Caenorhabditis elegans. Nature. 2000;403(6772):901–6.

4. Pasquinelli AE, Reinhart BJ, Slack F, Martindale MQ, Kuroda MI, Maller B, et al. Conservation of the sequence and temporal expression of let-7 heterochronic regulatory RNA. Nature. 2000;408(6808):86–9.

5. Miranda KC, Huynh T, Tay Y, Ang Y-S, Tam W-L, Thomson AM, et al. A Pattern-Based Method for the Identification of MicroRNA Binding Sites and Their Corresponding Heteroduplexes. Cell. 2006;126(6):1203-17.

https://doi.org/10.1016/j.cell.2006.07.031

6. Ota A, Tagawa H, Karnan S, Tsuzuki S, Karpas A, Kira S, et al. Identification and characterization of a novel gene, C13orf25, as a target for 13q31-q32 amplification in malignant lymphoma. Cancer Res. 2004;64(9):3087–95.

7. Olive V, Jiang I, He L. mir-17-92, a cluster of miRNAs in the midst of the cancer network. Int J Biochem Cell Biol. 2010;42(8):1348–54.

8. He L, Thomson JM, Hemann MT, Hernando-Monge E, Mu D, Goodson S, et al. A microRNA polycistron as a potential human oncogene. Nature. 2005;435(7043):828–33.

9. Calin GA, Dumitru CD, Shimizu M, Bichi R, Zupo S, Noch E, et al. Frequent deletions and down-regulation of micro- RNA genes miR15 and miR16 at 13q14 in chronic lymphocytic leukemia. Proc Natl Acad Sci U S A. 2002;99(24):15524–9.

10. Cimmino A, Calin GA, Fabbri M, Iorio M V., Ferracin M, Shimizu M, et al. miR-15 and miR-16 induce apoptosis by targeting BCL2. Proc Natl Acad Sci. 2005;102(39):13944–9.

https://doi.org/10.1073/pnas.0506654102

11. Calin GA, Cimmino A, Fabbri M, Ferracin M, Wojcik SE, Shimizu M, et al. MiR-15a and miR-16-1 cluster functions in human leukemia. Proc Natl Acad Sci. 2008;105(13):5166–71.

https://doi.org/10.1073/pnas.0800121105

12. Kumar MS, Lu J, Mercer KL, Golub TR, Jacks T. Impaired microRNA processing enhances cellular transformation and tumorigenesis. Nat Genet. 2007;39(5):673–7.

13. Johnson SM, Grosshans H, Shingara J, Byrom M, Jarvis R, Cheng A, et al. RAS is regulated by the let-7 microRNA family. Cell. 2005;120(5):635–47.

14. Cully M. Closing the door on KRAS-mutant lung cancer. Nat Rev Drug Discov. 2016;15(11):747–747.

15. Baer C, Squadrito ML, Laoui D, Thompson D, Hansen SK, Kiialainen A, et al. Suppression of microRNA activity amplifies IFN-γ-induced macrophage activation and promotes anti-tumour immunity. Nat Cell Biol. 2016;18(7):790–802.

16. Lin X, Chen M, Liu Y, Guo Z, He X, Brand D, et al. Advances in distinguishing natural from induced Foxp3(+) regulatory T cells. Int J Clin Exp Pathol. 2013;6(2):116–23.

17. Zhou X, Bailey-Bucktrout SL, Jeker LT, Penaranda C, Martínez-Llordella M, Ashby M, et al. Instability of the transcription factor Foxp3 leads to the generation of pathogenic memory T cells in vivo. Nat Immunol. 2009;10(9):1000–7.

https://doi.org/10.1038/ni.1774

18. Deknuydt F, Bioley G, Valmori D, Ayyoub M. IL-1β and IL-2 convert human Treg into TH17 cells. Clin Immunol. 2009;131(2):298–307.

https://doi.org/10.1016/j.clim.2008.12.008

PMid:19211307

19. Pan W, Zhu S, Dai D, Liu Z, Li D, Li B, et al. MiR-125a targets effector programs to stabilize Treg-mediated immune homeostasis. Nat Commun. 2015;6:7096.

20. Carone BR, Fauquier L, Habib N, Shea JM, Hart CE, Li R, et al. Paternally Induced Transgenerational Environmental Reprogramming of Metabolic Gene Expression in Mammals. Cell. 2010;143(7):1084–96.

https://doi.org/10.1016/j.cell.2010.12.008

PMid:21183072 PMCid:PMC3039484

21. Chen Q, Yan M, Cao Z, Li X, Zhang Y, Shi J, et al. Sperm tsRNAs contribute to intergenerational inheritance of an acquired metabolic disorder. Science. 2016;351(6271):397–400.

22. Sharma U, Conine CC, Shea JM, Boskovic A, Derr AG, Bing XY, et al. Biogenesis and function of tRNA fragments during sperm maturation and fertilization in mammals. Science. 2016;351(6271):391–6.

23. Leucci E, Vendramin R, Spinazzi M, Laurette P, Fiers M, Wouters J, et al. Melanoma addiction to the long non-coding RNA SAMMSON. Nature. 2016;531(7595):518–22.

https://doi.org/10.1038/nature17161

24. Flaherty KT, Puzanov I, Kim KB, Ribas A, McArthur GA, Sosman JA, et al. Inhibition of Mutated, Activated BRAF in Metastatic Melanoma. N Engl J Med. 2010;363(9):809–19.

https://doi.org/10.1056/NEJMoa1002011

25. Fogal V, Richardson AD, Karmali PP, Scheffler IE, Smith JW, Ruoslahti E. Mitochondrial p32 Protein Is a Critical Regulator of Tumor Metabolism via Maintenance of Oxidative Phosphorylation. Mol Cell Biol. 2010;30(6):1303–18.

https://doi.org/ 10.1128/MCB.01101-09

26. Richter-Dennerlein R, Dennerlein S, Rehling P. Integrating mitochondrial translation into the cellular context. Nat Rev Mol Cell Biol. 2015;16(10):586–92.

https://doi.org/10.1038/nrm4051

27. Salmena L, Poliseno L, Tay Y, Kats L, Pandolfi PP. A ceRNA Hypothesis: The Rosetta Stone of a Hidden RNA Language? Cell. 2011;146(3):353–8.

https://doi.org/10.1016/j.cell.2011.07.014

PMid:21802130 PMCid:PMC3235919

28. Wang J, Liu X, Wu H, Ni P, Gu Z, Qiao Y, et al. CREB up-regulates long non-coding RNA, HULC expression through interaction with microRNA-372 in liver cancer. Nucleic Acids Res. 2010;38(16):5366–83.

https://doi.org/10.1093/nar/gkq285

29. Karreth FA, Tay Y, Perna D, Ala U, Tan SM, Rust AG, et al. In vivo identification of tumor- suppressive PTEN ceRNAs in an oncogenic BRAF-induced mouse model of melanoma. Cell. 2011;147(2):382–95.

30. Hansen TB, Jensen TI, Clausen BH, Bramsen JB, Finsen B, Damgaard CK, et al. Natural RNA circles function as efficient microRNA sponges. Nature. 2013;495(7441):384–8.

31. Poliseno L, Salmena L, Zhang J, Carver B, Haveman WJ, Pandolfi PP. A coding-independent function of gene and pseudogene mRNAs regulates tumour biology. Nature. 2010;465(7301):1033–8.

32. Mayr C, Bartel DP. Widespread shortening of 3'UTRs by alternative cleavage and polyadenylation activates oncogenes in cancer cells. Cell [Internet]. 2009 Aug 21 [cited 2016 Aug 28];138(4):673–84. Available from: http://www.ncbi.nlm.nih.gov/pubmed/19703394

33. Vickers KC, Palmisano BT, Shoucri BM, Shamburek RD, Remaley AT. MicroRNAs are transported in plasma and delivered to recipient cells by high-density lipoproteins. Nat Cell Biol. 2011;13(4):423–33.

https://doi.org/10.1038/ncb2210

34. Sarkies P, Miska EA, Fire A, Hamilton AJ, Baulcombe DC, Brosnan CA, et al. Molecular biology. Is there social RNA? Science. 2013;341(6145):467–8.

35. Vickers KC. Lipoprotein transport of small non-coding RNAs in cardiometabolic disease. Keynote Lect Int Student Congr Graz Med Univ. 28 05 2016.

36. Hansen TB, Venø MT, Jensen TI, Schaefer A, Damgaard CK, Kjems J, et al. Argonaute-associated short introns are a novel class of gene regulators. Nat Commun. 2016;7:11538.

https://doi.org/10.1038/ncomms11538

37. Savell KE, Gallus NVN, Simon RC, Brown JA, Revanna JS, Osborn MK, et al. Extra-coding RNAs regulate neuronal DNA methylation dynamics. Nat Commun. 2016;7:12091.

https://doi.org/10.1038/ncomms12091

38. Li W, Notani D, Rosenfeld MG. Enhancers as non-coding RNA transcription units: recent insights and future perspectives. Nat Rev Genet. 2016;17(4):207–23.

https://doi.org/10.1038/nrg.2016.4

39. Schratt G. microRNAs at the synapse. Nat Rev Neurosci. 2009;10(12):842–9.

https://doi.org/10.1038/nrn2763

40. Weaver CT, Hatton RD. Interplay between the TH17 and TReg cell lineages: a (co-)evolutionary perspective. Nat Rev Immunol. 2009;9(12):883–9.

https://doi.org/10.1038/nri2660

Figures

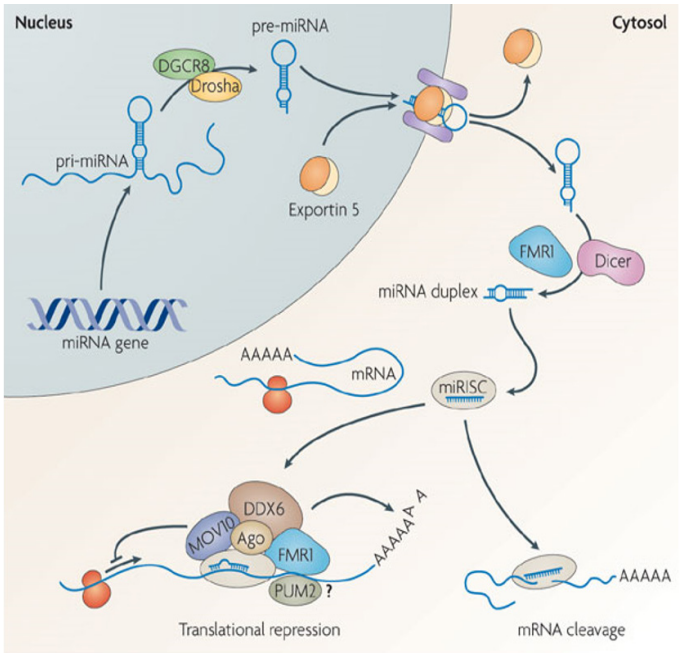

Figure 1: miRNA biogenesis and mode of action(39).

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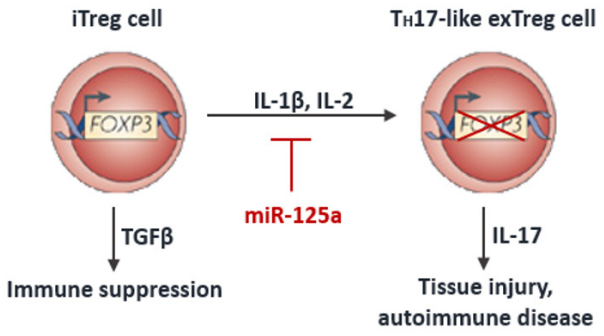

Figure 2: miR-125a stabilises iTreg cells and prevents their transition into pro-inflammatory TH17-like exTreg cells (adapted from(40)).

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Education

Skin picking disorder: what can we learn from such a topical issue?

EDUCATION

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ABSTRACT

Summary

Skin picking disorder (SPD) is a psychodermatological condition characterised by repeated pathological picking of the skin, creating recalcitrant excoriated skin lesions. As well as the physical manifestations of skin picking, SPD carries with it a host of debilitating social, psychiatric and medical sequelae, which greatly impairs quality of life in sufferers. It is clinically challenging to make an accurate diagnosis of SPD and, in our experience, successful management requires a sensitive and holistic biopsychosocial approach involving a psychodermatology multidisciplinary team.

Relevance

The prevalence of SPD is ~2-4%, yet fewer than 20% of sufferers feel that their clinician 'knew much' about their condition. This must be immensely frustrating for sufferers, and it chimes with research suggesting that medical students, and by extrapolation doctors, have multiple gaps in their knowledge of psychiatric disorders, patients and treatments. We believe that a greater emphasis on psychiatry in medical training is long overdue, particularly to highlight the crucial importance of addressing psychosocial factors alongside physical symptoms in all patients.

Take Home Messages

1. SPD is an under-diagnosed and often poorly managed condition that is associated with clinically significant distress or impairment in multiple areas of day-to-day functioning.

2. Given the wide differential diagnosis for SPD, a careful history, physical and mental state examination is critical in making an accurate diagnosis.

3. SPD lacks a standardised treatment protocol, but successful management can be achieved through involvement of a psychodermatology multidisciplinary team.

4. Psychosocial illness is poorly understood by the typical medical undergraduate. Medical schools must strive to remedy this by placing a greater emphasis on the importance of holistic assessment and treatment in all patients.

Skin picking disorder (SPD) is a psychodermatological condition reported in medical literature from as early as 1875. (1) The disorder is characterised by repeated pathological picking of the skin to the extent where lesions are apparent across accessible sites such as the face, neck, arms and scalp. SPD carries with it a host of extremely debilitating social, psychiatric and medical sequelae, and as such, greatly impedes quality of life in sufferers.

Despite historical recognition of SPD and its serious complications, the disorder only became officially recognised in the Diagnostic and Statistical Manual of Mental Disorders (DSM-V) in 2013. The exact aetiopathogenesis underpinning SPD remains unclear, but the evidence implicates both genetic and environmental components. (2) Rodent models have identified some candidate genes, which appear to converge on dysregulation in the neural circuitry involved in habit generation and top-down motor inhibitory control processes. (1)

Picking behaviours vary within and between sufferers but are typically highly ritualised, and triggered by stress, anxiety or boredom. Patients also report feelings of mounting tension prior to picking, with relief and even pleasure experienced during or immediately afterwards. The psychosocial component of SPD is severe, with many sufferers avoiding daily activities that may expose their skin such as work, sport and social commitments. The isolation that follows compounds the problem further by allowing more time for skin hypervigilance, picking and unfortunately frequent suicidal ideation. Medical sequelae include bleeding, localised infection, septicaemia, scarring and, in the most severe cases, an increased risk of mortality warranting neurosurgical intervention. (3)

From the clinician's point of view, the priority in assessing patients with possible SPD is to actively exclude a primary organic disorder. Systemic causes of pruritus must be ruled out, such as iron deficiency anaemia, diabetes mellitus and coeliac disease. Primary organic dermatoses such as scabies, as well as other related yet distinct psychodermatological conditions e.g. dermatitis artefacta, must also be distinguished. There is also the challenge of separating SPD from common psychiatric co-morbidities that may better explain the skin picking. These include body dysmorphic disorder and psychotic disorders involving skin-related delusions and tactile hallucinations. In light of this, a mental state examination is crucial to making an accurate diagnosis.

SPD currently lacks a standardised approach to management. Evidence in the literature demonstrates a significant benefit for behavioural, but not pharmacological therapies, as compared to placebo. (4) Skin directed therapies should always be employed in conjunction with psychiatric or psychological treatments for dual purpose: to treat infection or dry skin if indicated, but also to reassure patients unwilling to consider psychosocial factors that their problem is being taken seriously. In our experience, successful management requires a sensitive and holistic approach that, looking forward, may best be delivered in a specialist multidisciplinary psychodermatological clinic.

The prevalence of SPD is ~2-4%, however, studies have revealed that less than 20% of afflicted patients felt that their clinician 'knew much' about their condition. (4,5) Whilst this must be frustrating for sufferers, it also raises a myriad of questions surrounding medical education.

Medical students are all too familiar with the endless lists of rare mutations that lead to conditions never-to-be-seen on the wards. Studies have found that medical students have multiple lacunae in their knowledge toward psychiatric disorders, patients and treatments. (6) This both fits with the aforementioned issues with SPD and is something that medical students experience throughout their studies.

There is a complex interplay between psychiatry and the medical and surgical specialties that all practitioners need to be aware of. Failing to understand this dynamic is a compromise of one's duty of care, and creates a potential hazard for our patients. A simple example of this lies with individuals who attend A&E with self-harm injuries. Patients are often labelled as 'frequent flyers' and it is unfortunately accepted throughout the literature that analgesia is rarely offered, with one patient being told 'I thought you liked pain'. (7) This represents a physical manifestation of psychiatric disease and the poor treatment of these patients likely represents poor understanding of the full clinical picture, as discussed above with SPD.

Medical students depend on their education to build a career that needs to be fundamentally safe and improve the lives of patients. It is the joint responsibility of both students and their seniors to acknowledge the importance of these newly classified disorders. We therefore deserve autonomy over the content of our education. Whilst we acknowledge that there are mounting pressures on medical education, we suggest the following moving forward:

- A greater emphasis on psychiatry, including niche disorders that are commonly overlooked in typical undergraduate programs.
- Considering the feasibility of psychiatry as a compulsory component in the foundation program.
- Medical schools to place greater emphasis on the uses and benefits of cognitive and behavioural therapies.

It is our hope that consideration of the above will raise awareness of some of the pitfalls that we face as the new generation of medical professionals. It must be frustrating for patients who often have no choice through which lens we view their disease. We believe a holistic biopsychosocial approach should be promulgated in all patients to a greater or lesser extent, to ensure that organic disease is not missed, and that psychosocial illness is given the credence it

deserves.

References

1. Grant JE, Odalug BL, Chamberlain SR, Keuthen NJ, Lochner C, Stein DJ. American Journal of Psychiatry. 2012; 169(11): 1143-1149.

https://doi.org/10.1176/appi.ajp.2012.12040508

PMid:23128921

2. Monzani B, Rijsdijk F, Cherkas L, Harris J, Keuthen N, Mataix-Cols D. Prevalence and heritability of skin picking in an adult community sample: a twin study. Am J Med Genet B Neuropsychiatr Genet. 2012;159(5):605-610.

3. Kim DI, Garrison RC, Thompson G. A near fatal case of pathological skin picking. Am J Case Rep. 2013;14:284–287.

https://doi.org/10.12659/AJCR.889357

PMid:23919102 PMCid:PMC3731172

4. Schumer MC, Bartley CA, Bloch MH. Systematic Review of Pharmacological and Behavioural Treatments for Skin Picking Disorder. Journal of Clinical Psychopharmacology. 2016;36(2):147-152.

https://doi.org/10.1097/JCP.0000000000000462

PMid:26872117 PMCid:PMC4930073

5. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, 5th edn. Arlington VA: American Psychiatric Association; 2013.

6. Chawla JM. Balhara YPS. Sagar R. Shivaprakash. Undergraduate medical students' attitude towards psychiatry: a cross-sectional study. Indian Journal of Psychiatry. 2012;54(1):37-40.

https://doi.org/10.4103/0019-5545.94643

PMid:22556435 PMCid:PMC3339216

7. BMJ. Self Harm and the Emergency Department. BMJ. 2016;353 [accessed 29 June 2018]. Available from: https://www.bmj.com/content/353/bmj.i1150/rapid-responses

https://doi.org/10.1136/bmj.i1150

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Education

Clinical Coding - an insight into healthcare data

EDUCATION

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ABSTRACT

Summary

Clinical coding is an important function within healthcare informatics. Coding datasets have various statistical and clinical uses: a high level of accuracy, completeness and specificity is paramount. Preventable errors can accumulate along the coding pathway and may have significant implications for health care management worldwide.

Relevance

Amongst other functions, clinical coding datasets are used for policy creation, clinical audit and finance. Both clinical staff and coding professionals play an equally important role in ensuring accurate reporting of hospital activity, but the nature and importance of the clinician's role is often misunderstood and underappreciated. The coding pathway is highly susceptible to human error with clinician-based errors ranging from incorrectly defining a clinical entity to underreporting comorbidities. Clinical staff should be aware of the importance of clinical coding, its uses and the role they play in ensuring accuracy: a hospital is measured by its output data. High-quality data leads to well-informed service provision, resource allocation and business planning which cumulates into enhanced patient outcomes.

Take Home Messages

Errors occurring along the coding pathway can have significant financial and statistical ramifications. Clinicians and coding professionals must work collaboratively to improve the quality of healthcare data flows. Regular engagement between all influential parties along the coding pathway is vital, with frequent reviews of clinical documentation and coding reports a firm foundation for continual improvement. Accurate and reliable healthcare data supports the optimisation of patient care.

Introduction

Effective healthcare management requires versatile and pre-emptive strategic planning. For this to be possible, hospitals depend on an effective mechanism for data collection, collation and analysis: clinical coding. Clinical coding is the translation of clinical statements into alphanumeric codes, typically four characters in length. (1) Its purpose is to standardise the recording of clinical information to enable datasets to be analogous. (2) There are numerous ways to describe the same clinical entity, so without a standardised system these cases could be coded differently, creating a barrier to comparable analyses. In the United Kingdom (UK), two classifications are currently used for clinical coding: the 10th International Statistical Classification of Diseases and Related Health Problems (ICD-10) and the Office of Population Censuses and Surveys Classification of Interventions and Procedures version four (OPCS-4). (3)

The quality of coding depends on a collaborative approach being championed by coders and clinical staff alike. (4) The value and accuracy of clinical coding is particularly important in England as coding underpins the National Tariff Payment System (NTPS) (formally known as Payment by Results (PbR)). (3) Under this system, provider income flows are directly related to clinical activity. As clinical coding is the nationally adopted method to document such activity, it is essential that coding datasets are complete, accurate and hold the highest possible level of specificity to circumnavigate potential financial pitfalls.

Despite its importance, awareness of clinical coding is low amongst junior doctors. (5) It is essential that all clinical staff understand the importance of the role they have to play in refining healthcare data flows, as ultimately they determine the usefulness of such data for all secondary purposes.

Uses of clinical coding

Clinical coding allows for reliable collation, aggregation and comparison of healthcare data on local, national and international platforms.

Local uses

Coding data is useful for clinical audit, tracking patient outcomes and measuring treatment effectiveness. It is a means for easily identifying specific patient cohorts, tracking complication rates and linking outcomes to a defined criteria, such as the surgeon performing a procedure or procedural technique adopted.

Management in a provider setting use coding data for monitoring and planning. Casemix analyses facilitate appropriate resource distribution, policy derivation and growth prediction. Reliable data aids clinical governance and encourages good practice. (6)

Under the NTPS, coding datasets are used as a means to reimburse providers for the care that they provide. Inaccuracies can lead to signficant financial losses with any one error having the potential of being valued in the thousands of pounds. (4) Coding datasets are therefore a key component in cost analyses and business planning. (7)

National uses

Monitoring patterns in activity and trends over time allows for tailored, region-specific resource planning and allocation. Coding data is widely used by governmental and healthcare bodies to monitor, develop and evaluate healthcare policies across the UK. (8) National indicators of clinical quality and best practice are tracked and reimbursed using specific coding derivatives. (9)

National benchmarking and comparison highlights areas where improvement is needed and provides a resource for patients to make greater-informed choices about where and how they access care. (10, 11) Discrepancies in coding data help ascertain 'dead zones' for particular types of care and provide a means for assessing fair access to healthcare.

International uses

All World Health Organisation (WHO) members use the ICD. (12) This permits monitoring of the aetiology, incidence and prevalence of diseases globally and helps identify at risk populations, on the basis of diagnostic, demographic or environmental factors. (13) International learning models can be derived from shared data pools, permitting global collaboration on policy creation and resource forecasting.

The clinical coding pathway

From admission to secondary user

Figure 1 outlines the inpatient coding pathway from admission to secondary user. This pathway is specific to secondary and tertiary care in England.

The pathway begins when a patient is admitted to a hospital and undergoes treatment or investigation (step 1). Clinical staff detail all of the patient events that occurred throughout the hospital spell in the medical record.

Following discharge, clinical coders translate the clinical information into codes using ICD-10 and OPCS-4 (step 2). Providers work towards having all activity coded by an initial deadline known as 'flex,' which gives providers and commissioners the opportunity to assess the data and make corrections where appropriate. (14)

Quality assurance analyses are performed to ensure coding accuracy and abidance by national standards (step 3). The information governance toolkit is a framework for clinical coding audit. Providers must ensure that they have carried out internal audits on whichever is smaller of 200 or 2% of total finished

consultant episodes (FCEs). (15) Continual assurance activity helps safeguard a precise position for contract monitoring and performance regulation (step 4a).

In line with a final deadline known as 'freeze,' (14) providers submit their coding data to the Secondary Uses Services (SUS+) via commissioning datasets (step 4b). (10) SUS+ is a UK information warehouse that stores healthcare data in adherence to national policies. (10) There are different commissioning datasets depending on the type of care provided (admitted patient care (APC), outpatients (OP), accident & emergency (A&E) etc.).

Before the data can be accepted by SUS+, it is validated against business rules and if this is successful, the data is then passed through a grouper software (step 5). (16) The grouper uses a sophisticated algorithm, created by the National Casemix Office, to group clinically meaningful clusters of activity. (3) These groups are known as Healthcare Resource Groups (HRGs). (3) HRGs are the currency for remuneration in England and carry a defined tariff representative of the care provided with consideration of demographic and provider information. (17)

If processing has been efficacious, the data is made available to approved SUS+ users. There are two data marts: defined layers of the data warehouse aligned with user requirements (step 5). The Standard Extract Mart (SEM) view shows completed hospital episodes without any additional payment-related additions. (16) SEM is used to derive Hospital Episode Statistics (HES) (step 6a). HES is an anonymised database containing information of all admissions, outpatient appointments and A & E attendances in England. (8) The PbR view shows spell based activity with additional payment related derivations (step 6b). (16) It is used for derivation of NTPS policy, monitoring and reimbursement.

Key Roles

The number of 'influencers' along the pathway varies with the size of a setting and local practice. Some influential roles are outlined below.

Clinical Coders

Clinical coders perform the act of coding, working in accordance with local, national and international guidance to ensure uniformity. (13, 18) Clinical coders have a responsibility to stay up to date with changes to these standards as well as local recording protocols. They are encouraged to work towards professional registration (19) to prove a standard of accuracy.

Clinical Coding Auditors

Coding auditors assure the quality of clinical coding against national standards and reporting requirements. (13, 15) This may be done internally within a trust or on behalf of external commissioning providers.

Clinical Staff

All clinical staff have a legal requirement to ensure documentation is complete and accurate. (20) Junior doctors are typically tasked with completing discharge summaries, which are a primary source of information for coding purposes.

Data Analysts

Data analysts utilise coding datasets for strategic planning, contract monitoring and identification of areas for change.

Health Informatics Managers

Informatics managers are tasked with aggregating and submitting all commissioning data sets to SUS+. They work extensively with commissioners and other third parties to ensure the correct processes, outlined within the NHS standard contract are adhered to. (21)

Sources of coding errors

Estimates on coding accuracy vary drastically, with systematic reviews suggesting a median accuracy of around 80%. (22)

Coder errors

Errors by clinical coders are well reported in the literature and are often multifactorial. (17, 22, 23) The experience of a coder directly influences the quality of coding. (23) A coder's knowledge about the diseases and procedures that they are coding can make the difference between separating a symptom of a condition from a new finding. (24) A coder could 'under-code' by being unable to differentiate between separate clinical entities. Time restraints, the level of coder-clinician communication and departmental leadership styles can all affect coding practice. (25) Failure to clarify queries or request missing subsidiary information, such as external causes or infection aetiology, can all lead to paucity in coding datasets. (17, 26)

Clinician errors

The eventual value of clinical coding is limited by the quality of clinical documentation; electronic discharge summaries have been shown to often be suboptimal for coding purposes. (17) Junior doctors receive minimal education on clinical coding, (27) and are typically first introduced to the topic at local hospital inductions. In general, this is ineffective and, despite a willingness to engage with the field, clinicians often fail to adequately meet all coding requirements when recording clinical events. (5) A lack of a robust educational programme has meant that misconceptions on the uses and importance of clinical coding are rife, and many clinical staff members are uncertain as to what is required from them. (5, 26)

Lack of engagement, between clinical staff and coding professionals, has been correlated with significant financial repercussions and omission of noteworthy information. (4, 26) A lack of specificity frequently causes difficulties with different codes available as subcategories of the same disease or morbid entity. An example of this is the coding of obesity. In ICD-10, obesity (with no further modifiers) is coded as E66.9 – Obesity, unspecified whereas morbid obesity and drug-induced obesity are coded with E66.8 – Other obesity and E66.1 – Drug-induced obesity respectively. (12) In some cases, the clinical management of the conditions defined by ICD-10 subcategories does not change, so from a medical perspective the 'unspecified' description of a condition may be deemed sufficient to ensure continuity of care. (28) Idiosyncratic use of abbreviations, inconsistencies between the ways clinicians document patient events and failure to record information in a predictable structure, all create interpretational dilemmas for the coder. (4)

Under the current version of the NTPS, inpatient tariffs can be affected by a patient's summed 'comorbidity score'; correct financial reimbursement is reliant on all relevant secondary diagnoses and comorbidities being coded. (3) Failure to record that conditions have been fully treated or resolved can inhibit the correct 'history of' codes being assigned and requires the coder to dedicate significant resources towards determining whether a condition is current. Coders cannot infer test results and require a definitive interpretation of radiological, biochemical, histological and microbiological results. (13) This interpretation must be made by the clinician and documented in the medical record, especially in cases where the patient is treated on the basis of these investigations. The nature of acute injuries should be recorded to ascertain the circumstances in which the injury arose, to permit injury surveillance. (29)

Methods for reducing clinical coding errors

The first step in reducing the likelihood of coding errors occurring is raising awareness of regularly reported problems. With increased awareness, it is possible to introduce subtle changes in local practice to best serve both clinical and administrative functions. Table 1 outlines a framework for mitigating the effects of common pitfalls.

The future of clinical coding

The field of clinical coding is rapidly evolving and there is plenty of debate about future directions. (2) The implementation of Systematized Nomenclature of Medicine–Clinical Terms (SNOMED CT), which is a structured terminology within an Electronic Patient Record (EPR), (31) will permit ICD-10 and OPCS 4.8 to be cross mapped directly to clinical documentation rather than coded by a coding professional. As such, the roles of the clinician and coder will continue to transform over time.

Conclusion

It is essential that all influential parties, along the coding pathway, work coherently to ensure healthcare data is complete, accurate and reliable. Sources of error are plentiful but ill effects can be diminished or lessened with diligence and planning. Education is the key to improvement and more must be done to raise awareness if we are to continue to use clinical coding for essential secondary functions.

References

1. Eve-Jones S. Coding for clinicians. The Bulletin of the Royal College of Surgeons of England. 2014;96:286-287.

https://doi.org/10.1308/147363514X14042954768673

2. Tatham A. The increasing importance of clinical coding. British Journal of Hospital Medicine. 2008:69;372-373.

https://doi.org/10.12968/hmed.2008.69.7.30409

3. National Casemix Office. Casemix companion. Health and Social Care Information Centre:2018 [accessed 26 Feb 2018]. Available from:https://digital.nhs.uk/media/37396/HRG4-201718-Reference-Costs-Grouper-Casemix-Companion-v1-0/pdf/HRG4__201718_Reference_Costs_Grouper_Casemix_Companionv1-0.

4. Mahbubani K, Georgiades F, Goh E, Chidambaram S, Sivukamaran P, Rawson T et al. Clinician-directed improvement in the accuracy of hospital clinical coding. Future Healthcare Journal. 2018:5;47-51.

https://doi.org/10.7861/futurehosp.5-1-47

5. Sehjal R, Harries V. Awareness of clinical coding: a survey of junior hospital doctors. Sehjal, R and Harries, V. British Journal of Health Care Management. 2016;22:310-314.

https://doi.org/10.12968/bjhc.2016.22.6.310

6. Veenstra G, Ahaus K, Welker G, Heineman E, Van Der Laan M, Mutinghe F. Rethinking clinical governance: healthcare professionals' views: a Delphi study. BMJ Open. 2017;7.

https://doi.org/10.1136/bmjopen-2016-012591

7. Hof S, Fügener A, Schoenfelder J, Brunner J. Case mix planning in hospitals: a review and future agenda. Health Care Management Science. 2017;20:207-220.

https://doi.org 10.1007/s10729-015-9342-2

8. Waller P, Shaw M, Ho D, Shakir S, Ebrahim S. Hospital admissions for 'drug-induced' disorders in England: a study using the Hospital Episodes Statistics (HES) database. British Journal of Clinical Pharmacology. 2005;59:213-219.

https://doi.org/10.1111/j.1365-2125.2004.02236.x

9. Health and Social Care Information Centre. Hospital accident and emergency activity: support information, 2016-17. 2017 [accessed 26 Feb 2018]. Available from: https://digital.nhs.uk/media/33256/Hospital-Accident-and-Emergency-Activity-2016-17-Supporting-Information/default/acci-emer-atte-eng-2016-17-supp.

10. NHS England and Health and Social Care Information Centre. NHS hospital data and datasets: a consultation. 2013 [accessed 26 Feb 2018]. Available from: https://www.england.nhs.uk/wp-content/uploads/2013/07/hosp-data-consult.pdf.

11. Dr Foster Intelligence. Essential reading for smart spending: Dr Foster hospital guide 2013. 2013 [accessed 21 Mar 2018]. Available from: http://myhospitalguide.drfosterintelligence.co.uk/downloads/report/Report.pdf.

12. World Health Organization. International Statistical Classification of Diseases and Related Health Problems. 10th Revision. 5th ed. Geneva: WHO; 2016.

13. Clinical Classifications Service. National clinical coding standards ICD-10 5th Edition: accurate data for quality information. 2018 [accessed 22 Feb 2018]. Available from: https://hscic.kahootz.com/connect.ti/t_c_home/view?objectId=298579

14. Health and Social Care Information Centre. PbR data user guide. 2013 [accessed 26 Feb 2018]. Availabke from: http://content.digital.nhs.uk/media/12502/pbr-data-user-guide-v1/pdf/PbR_data_user_guide_v1.0.pdf.

15. Health and Social Care Information Centre. Information governance toolkit v14.0. [Accessed 01 Mar 2018]. Available from: https://www.igt.hscic.gov.uk/RequirementDocuments/IGT_Requirement_505_ACUTE_v14_0.pdf.

16. NHS Digital. SUS+ PbR reference manual: implementation of National Tariff/Payment by results (PbR) in the Secondary Uses Service v4.3. 2017 [accessed 01 Mar 2018]. Available from: http://content.digital.nhs.uk/media/25684/SUS-PbR-Reference-Manual-v43/pdf/SUS__PbR_Reference_Manual_v4.3.pdf.

17. Roberts L, Timmis J, Nagrecha R, Martinez-Alier N. Capturing causative agents in clinical coding. British Journal of Healthcare Management. 2016;22:461-468.

https://doi.org/10.12968/bjhc.2016.22.9.461

18. Clinical Classifications Service. National clinical coding standards OPCS-4: accurate data for quality information. 2018 [accessed 22 Feb 2018]. Available from: https://hscic.kahootz.com/gf2.ti/f/762498/33700805.1/PDF/-/NCCSOPCS420185.pdf.

19. IHRIM - National Clinical Coding Qualification. [Accessed]. Available from: http://www.ihrim.co.uk/index.php?option=com_content&view=article&id=109&Itemid=433.

20. General Medical Council. Good medical practice. 2013 [accessed 01 Mar 2018]. Available from: https://www.gmc-uk.org/static/documents/content/Good_medical_practice_-_English_1215.pdf.

21. NHS England. NHS standard contract 2017/18 and 2018/19: service conditions. 2016 [accessed 01 Mar 2018]. Available from: https://www.england.nhs.uk/wp-content/uploads/2018/01/2-nhs-standard-contract-1718-1819-service-conditions-full-length.pdf.

22. Burns E, Rigby E, Mamidanna R, Bottle A, Aylin P, Ziprin P et al. Systematic review of discharge coding accuracy. Journal of Public Health. 2012;34:138-148.

https://doi.org/10.1093/pubmed/fdr054

23. Zafirah S, Nur A, Puteh S, Aljunid S. Potential loss of revenue due to errors in clinical coding during the implementation of the Malaysia diagnosis related group (MY-DRG®) Casemix system in a teaching hospital in Malaysia. BMC Health Services Research. 2018;18:38.

https://doi.org/10.1186/s12913-018-2843-1.

24. Price E, Robinson K. The coding masterpiece: a framework for the formal pathways and processes of health classification. Health Information Management Journal. 2011;40:14-20.

https://doi.org/10.1177/183335831104000103.

25. Reid B, Ridoutt L, O'Connor P, Murphy D. Best practice in the management of clinical coding services: insights from a project in the Republic of Ireland, Part 2. Health Information Management Journal. 2017;46:105-112.

https://doi.org/10.1177/1833358317697470.

26. Roberts L, Cheung R. The aetiology of infections: the significance for hospital management. British Journal of Healthcare Management. 2017;23:271-280.

https://doi.org/10.12968/bjhc.2017.23.6.271.

27. UCL. Medicine MBBS BSc. [Accessed 01 Mar 2018]. Available from: http://www.ucl.ac.uk/prospective-students/undergraduate/degrees/medicine-mbbs-bsc/.

28. Nouraei S, Virk, J, Hudovsky A, Wathen C, Darzi A, Parsons D. Accuracy of clinician-clinical coder information handover following acute medical admissions: implication for using administrative datasets in clinical outcomes management. Journal of Public Health. 2016;38:352-362.

https://doi.org/10.1093/pubmed/fdv041.

29. Roberts, L. Clinical coding and external causes of injury: the importance of documentation. Journal of Plastic, Reconstructive and Aesthetic Surgery. 2016;69:1560-1561.

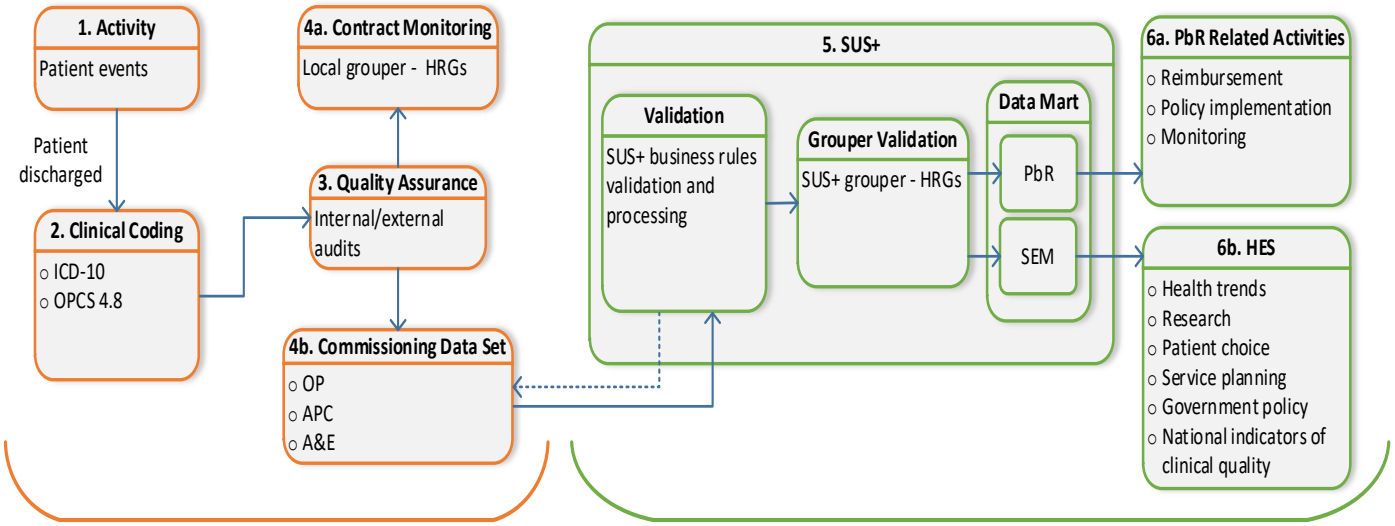
https://doi.org/10.1016/j.bjps.2016.08.027.

30. Clinical Classifications Service. Coding clinic. 2017 [accessed 22 Feb 2018]. Available from: https://hscic.kahootz.com/connect.ti/t_c_home/view?objectId=298579.

31. Al-Hablani, B. The use of automated SNOMED CT clinical coding in clinical decision support systems for preventive care. Perspectives in Health Information Management. 2017;14.

PMid:28566995 PMCid:PMC5430114

Figures



Legend

OP	Outpatient	PbR	Payment by Results
APC	Admitted Patient Care	SUS+	Secondary Uses Service
A&E	Accident and Emergency	SEM	Standard Extract Mart
HRG	Healthcare Resource Group	HES	Hospital Episode Statistics
ICD-10	10th International Classification of Diseases and Health Related problems		
OPCS-4	OPCS Classification of Interventions and Procedures		

Figure 1: The clinical coding pathway from admission to secondary user

Error	Owner	Potential solution(s)
Lack of specificity	Clinical staff	• Be as specific as possible when documenting diagnoses (e.g. link results, use multiple descriptors) and procedures (e.g. site, laterality, method of approach). • Liaise with coders to establish the descriptions required for accurate code assignment.
Use of abbreviations	Clinical staff	• Record diagnoses and procedures using the full ICD-10/OPCS 4 descriptions. • If an abbreviation must be used provide a reference key for clarification.
Omitted comorbidities	Clinical staff	• Record all comorbidities in all primary source documentation (e.g. clinic letters, discharge summaries). • Use service specific tick sheets to identify commonly missed comorbidities. • Engage in regular coding audits. • Develop and engage with new internal IT systems to promote comorbidity capture.
Unclear primary diagnoses or reason for admission	Clinical staff	• Record a patient's primary diagnosis clearly following any local pro forma standards. • If no definitive diagnosis can be made, record all of the patient's signs, symptoms or abnormal finding. • If there are no signs and symptoms, describe the reason for encounter (e.g. follow up after surgery or screening due to a family history of a condition).
Missing procedures/ interventions	Clinical staff	• Ensure that any procedures/interventions are fully described including those that may have been completed on the ward. • Record the dates procedures/interventions took place. • Engage in regular coding audits.
Ambiguous past medical history	Clinical staff	• Make a clear distinction between a condition that has been fully treated or resolved and one which is ongoing. • Follow local pro forma protocols.
Use of suboptimal language	Clinical staff	• Refrain from using terms such as "likely," "suggestive of" and "impression" that cannot be used for clinical coding. • Instead, use phrases such as "probable," "working diagnosis" and "treated as."

Absent linkages between results and disease status	Clinical staff	• Link all results to a condition (e.g. microbiology, histology, radiology etc.). • If results are not available at the time of discharge, document that they are pending. • Review test results and amend the discharge documentation where appropriate. • Engage in regular coding audits.
Unclear timelines	Clinical staff	• Record date and times of ward/clinician transfers. • Record dates when procedures were performed or complications arose.
Lack of engagement	Clinical staff/ Coder	• Participate in audits and assurance exercises. • Encourage wider partaking in coding events and workshops.
Inappropriate code assignment	Coder	• Attend frequent training to refresh knowledge of the national standards. (13, 18) • Clarify any ambiguous clinical information with responsible clinicians.
Not keeping up to date with new coding standards	Coder	• Read and update classifications with the latest coding standards and coding clinics. (13, 18, 30)
Lack of experience	Coder	• Undertake coding for different specialties • Attend all refresher courses and training workshops (where relevant).

Table 1: Methods for reducing coding errors

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Reflections

The case of the vanishing medical student

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ABSTRACT

Medical students are often hard to find on the wards after 3.00pm. In this reflective piece, using our own experiences of clinical teaching we describe the case of an archetypal, and stereotypical medical student who rarely attends a full day of timetabled clinical teaching. Using this case as an example we outline our insights of the reasons underpinning the fact many medical students leave clinical teaching placements well before 5.00pm, and in doing so miss out of learning opportunities. We also make two simple recommendations to medical students and teachers that could be employed to incentivise students to linger longer on their clinical placements.

Introduction

Medical school is a challenge, and developing the clinical skills and understanding to become an excellent doctor requires dedication, perseverance and lots of practice.

Being a doctor appears to be even more challenging, with long working hours, and without the luxuries of time, lack of accountability, and opportunities to go home at 2.30pm commonly afforded to medical students.

Students become increasingly aware of two contrasting choices whilst at clinical school: a choice to commit more time to clinical education in order to thrive as a junior doctor, and a choice to commit more time to social life and take all opportunities to avoid staying late in hospital whilst it is possible.

Here we describe the case of the vanishing medical student, analyse factors contributing to their regular disappearance and suggest strategies to incentivise students to commit more time to clinical education.

Case presentation

Mr X is a 22-year-old archetypal, stereotypical medical student in the prime of his student life. He presented insidiously over 2 years with afternoons off syndrome, which is now so severe he has only stayed on the wards after 2.30pm once in the last 3 months. This is on a background of feeling like a spare part on the ward, minimal formalised ward-based teaching sessions, and recommendations by ward staff to "enjoy being a student while you can".

He has an interesting past medical history with lots of exposure to clinical specialties during his work experience, volunteering in a hospitals abroad and had dreams of becoming a cardiac surgeon from a young age.

Currently he lives in a student flat, is a non-smoker and has a mild Netflix addiction.

Mr X is untroubled by his condition as he feels he will "learn most of it [clinical medicine] on the job". He also claims he uses his afternoons practicing for objective structural clinical exams (OSCEs) or reading in the library in order to prepare for exams, working on audits to develop his curriculum vitae (CV) as well as rewatching season one of Game of Thrones. He has attempted one cannula in the last two years and was unsuccessful – none of his flat mates have attempted one.

Discussion

This common case highlights many issues that contribute to the regular disappearance of many medical students from clinical training.

Importantly, junior doctors are becoming increasingly demoralised and this has direct implications for their own teaching and the teaching of medical students. (1) Not only are junior doctors workloads restricting the amount of time they can dedicate to teaching students, (1) observing exhausted, stressed, and busy junior doctors on the wards encourages some students to leave early and make the most of having minimal responsibility while they can. This is not helped by the fact students are regularly asked something along the lines of 'why are you here, its 3.00pm?' or told 'there is not much going on, I would just take the afternoon off.'

Students avoiding the wards creates a vicious cycle: by not attending, students fail to develop trusting relationships with the teams - this means when they are about they are given less responsibility and opportunities to practice, and often feel like spare parts – this means they are less likely to attend.

Additionally, as observed in the case above, examination systems at medical schools do not always incentivise students to spend more time on the wards.

Recommendations

We recommend two practical solutions for managing cases of vanishing students. Firstly, students should spend week one of a new rotation committing fully and getting to know the ward team, breaking the vicious cycle described above and increasing future learning opportunities and student satisfaction.

Secondly, as undertaken in some UK teaching hospitals, students should be assigned to individual patients on the ward. Students would then be responsible for presenting their patients on the morning ward rounds and assisting with any procedure and tests being performed. Furthermore, when other opportunities for learning are lacking, rather than escaping home, students can spend time talking to their selected patients. This should encourage students to spend more time on the wards, allow them to follow the elusive 'patient journey' and provide an opportunity for students to play a key role in making a patient feel better – which, as mentioned on all student's personal statements, is a motivating factor driving them to join the medical profession.

We believe using these steps, as well as encouraging professionalism among students, should help students to choose to commit more time to clinical education in order to thrive as a junior doctor.

References

1) General Medical Council. The State of Medical Education and Practice in the UK. 2016 [accessed 23 June 2016]. Available from: https://www.gmc-uk.org/-/media/documents/somep-2016-full-report-lo-res_pdf-68139324.pdf

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