Correspondence

EDITED BY MATTHEW HOTOPF

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Decreasing suicide in Hungary

We read with interest McClure’s (2000) report of a decrease in suicide in England and Wales between 1990 and 1997. Although the conclusion that improvements in the national economy contributed to this change is unproven, there is a large amount of evidence linking suicide and unemployment. It is, therefore, worth highlighting recent changes in Hungary, where in the 1980s the suicide rate was the highest in the world, four times the rate in England and Wales.

The suicide rate in Hungary has shown a steady decline from 45.9 per 100 000 in 1984 to 31.7 in 1997, a fall of more than 30%. This decline was greater after 1990 when the rate was 39.9 per 100 000 (Rihmer, 1997; Central Statistical Office, 1998) and when the political and economic changes in Eastern Europe began. During the same period there was a six-fold rise in unemployment, a four-fold rise in the number of people earning below the official minimum income, a 25% rise in official estimates of alcoholism rates, and a 21% rise in divorce. Other former Communist countries showed either no substantial change or a marked increase in their suicide rates after 1989/1990 (Sartorius, 1995; further details available from Z. Rihmer upon request).

It is not known why suicide in Hungary has fallen despite an adverse change in key risk factors but it is intriguing to note changes in mental health care. The number of out-patient psychiatric departments increased from 95 in 1982 to 136 in 1997, and the number of out-patient consultations annually increased from 731 000 in 1980 to 1 190 000 in 1997. The number of psychiatrists increased from 550 in 1986 to 800 in 1997. More extensive medical training on depression and suicide was followed by an increase in the use of antidepressants from 3.7 defined doses/1000 persons/day in 1990 to 12.0 in 1998. Between 1984 and 1997, the number of emergency telephone services also increased from 5 to 28 (further details available from Z. Rihmer upon request).

We are now conducting a psychological autopsy study of suicide in Hungary with the aim of identifying possible protective factors, both social and clinical.


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Drug dependence and child abuse

In response to Marsden et al (2000) we were interested to note the prevalence of psychiatric symptoms in their group of clients seeking treatment for drug dependence, particularly the fact that female drug users reported higher levels across all scales, as this mirrors our experience in treating intravenous drug misusers. We would suggest that such symptoms may be sequelae of child abuse.

There is an established link between childhood trauma and psychiatric symptoms in adulthood (e.g. Briere & Runtz, 1993), and recent evidence to suggest a link between such early life experiences and substance misuse. Contemporary psychoanalytic theories of addiction also emphasise the relationship between experience of early life trauma and later substance misuse (e.g. Wurmser, 1984), proposing that drug use is an attempt at self-medication (Khantzian, 1997), or a chemical means of inducing a dissociative state, so often apparent in victims of child abuse. McDougall (1989) suggests that substance misuse offers an external means of discharging painful internal states, through immediate action.

Our work in Cornwall indicates that of 111 randomly selected patients seen by the drug team, 46% of males and 73% of females reported childhood abuse to a degree that would have placed them on the at-risk register. Child sexual abuse was reported by 1.85% of males and 43% of females. This work provides evidence that there is a high incidence of early life trauma and abuse in the drug-misusing population. The figures account for the larger proportion of women presenting with drug dependence problems reporting abuse issues, which fits with Marsden et al’s report of a higher incidence of psychiatric symptomatology among their female clients.

We feel this is a very important area that needs to be evaluated further, as we find that the degree of trauma suffered by the patient can predict therapeutic needs and types of intervention required, including substitute prescribing, residential treatment and/or in-depth psychotherapeutic treatment. This is an aspect in the future that may enable drug services to tailor treatment packages to meet the individual’s needs and to target appropriate interventions.


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Relevance of Schneider’s first-rank symptoms

Peralta & Cuesta (1999) accurately criticise the significant bias of prior studies, namely the inclusion of patients in whom schizophrenia was diagnosed using criteria that strongly rely upon first-rank symptoms. In their study the authors clearly showed the high prevalence of first-rank symptoms in schizophrenia and non-schizophrenic psychosis (diagnosed mainly by Feighner’s criteria). They conclude that first-rank symptoms are not useful in differentiating schizophrenia from other psychotic disorders. This obviously seems correct, since Schneider (1959) himself never intended to differentiate schizophrenia from other psychoses (mainly schizophreniform disorder, schizoaffective disorder and atypical psychosis), as these disorders were part of the Schizophrener For menkreis in Schneider’s system. Therefore, we strongly disagree with Peralta & Cuesta that the diagnostic relevance of first-rank symptoms should be to differentiate schizophrenia from other psychotic disorders.

Moreover, the authors conclude that until more evidence is available, first-rank symptoms ‘‘should not receive particular emphasis in the ICD–11 and DSM–V diagnostic criteria for schizophrenia’’. Peralta & Cuesta omit a second possible conclusion: that the development of a non-schizophrenic psychosis (which, in fact, includes many of the diagnostic criteria of schizophrenia in any psychiatric classification) seems an artificial division of one clinical entity and this group should be considered as a schizophrenia subgroup in the ICD–11 and DSM–V.


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Authors’ reply: Drs Ortuño and Bonelli raise interesting questions regarding the relationship between our finding of lack of diagnostic value of first-rank symptoms for schizophrenia and Schneider’s concept of schizophrenia. One of the major problems in interpreting the work of Schneider (1987) is that he described his concept of schizophrenia very succinctly. He mentioned clearly the pathognomonic value of first-rank symptoms for schizophrenia in the absence of organic illness (p. 65). However, the presence of first-rank symptoms, although sufficient, is not necessary for the diagnosis, which may be also done on the basis of ‘second-rank’ symptoms. Unfortunately, Schneider tells us nothing about how to diagnose the disorder on the basis of these symptoms, and as a consequence, beyond the presence of first-rank symptoms, the diagnosis of schizophrenia is only vaguely described. On the other hand, Schneider gives us a precise description of the boundaries of the concept when he states that ‘‘since in comparison with cyclothymia the diagnostic frame of schizophrenia is very broad and vague, we tend to include atypical cases within the varied clinical picture of schizophrenia’’ (Schneider, 1987, p. 8). It seems, therefore, that within the endogenous psychoses, for Schneider all that does not fit the diagnosis of cyclothymia is schizophrenia. His own data on the differential prevalence of endogenous psychosis (Schneider, 1987, p. 8) appear to support this assertion: 84% for schizophrenia, 15% for cyclothymia and 1% for mixed psychoses.

We agree that our study results, by showing that first-rank symptoms spread across the full spectrum of psychoses, do not necessarily contradict Schneider’s schizophrenia concept, since DSM–III–R psychotic disorders and mood disorders with first-rank symptoms would correspond to Schneider’s Schizophrener For menkreis, which is very similar to the current notion of ‘schizophrenia spectrum disorders’. Our statement that first-rank symptoms ‘‘should not receive particular emphasis in the ICD–11 and DSM–V diagnostic criteria for schizophrenia’’ refers to the way in which first-rank symptoms are used by these criteria, that is as symptoms having higher diagnostic prominence for schizophrenia than for non-schizophrenic psychoses. We agree that the division of psychotic disorders into schizophrenic and non-schizophrenic psychoses is an artificial one (our data regarding first-rank symptoms support this) as it is the radical separation between schizophrenia and manic–depressive illness made by Schneider. Notwithstanding, we do not agree with the contention that all psychotic disorders must be diagnosed as schizophrenia. If the schizophrenia concept makes any sense (at least in regard to historical and clinical reasons), it is to differentiate a subgroup of psychotic disorders with poor outcome; but precisely here is where the first-rank symptoms (and by extension, Schneider’s schizophrenia concept) fail. Schneider held with Kraepelin’s nosological distinction between schizophrenia and manic–depressive illness, and like him assumed a poorer prognosis for schizophrenia than for cyclothymia. He seems to think of first-rank symptoms when he mentions ‘‘...the task so important for clinical and social psychiatry, to search for symptoms, which from experience will permit predictions regarding the future course and outcome. This will after all be the question asked of us’’ (Schneider, 1925). The attribution of first-rank diagnostic value for schizophrenia to certain symptoms could be interpreted in the sense that their presence conveys poor outcome. However, the link between first-rank symptoms and poor outcome implicit in Schneider’s writings is at odds with existing data indicating that first-rank symptoms are of no prognostic relevance (Mason et al, 1997). Therefore, given that (a) first-rank symptoms are present with a similar prevalence across the full spectrum of psychotic disorders, and (b) that first-rank symptoms are unrelated to the prognosis of schizophrenia, it seems fair to conclude that they are useless for the diagnosis of schizophrenia. In fact, our data support Crow’s (1995) contention that they are not disease entities but continua of variation where schizophrenia and manic–depressive illness represent the extreme forms of the psychotic continuum.


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Social anxiety in patients with facial disfigurement

Newell & Marks (2000) highlight an important, under-researched area. They suggest treatment using cognitive–behavioural therapy, concentrating on exposure to avoided situations. However, their conclusions and recommendations appear broader than the data support.

They recruited from dermatology clinics, ex-surgery lists and media adverts. Their sample might therefore be expected to include subjects with less severe disfigurement or even a primary diagnosis of dysmorphophobia. The preponderance of women requires explanation when many conditions causing facial disfigurement affect both genders equally. The nature and severity of disfigurement should be described. Facial disfigurement can result from bone deformity, scarring, muscular paralysis or abnormal movement. It may be congenital or acquired and onset can be sudden or gradual at any age. Aetiology includes trauma, surgery, neoplasms and infections and likelihood of recovery varies greatly. These factors are likely to influence the psychological difficulties experienced, including those manifest in social settings. Exposure would only be expected to help the phobic components of these problems.

Impaired control of functions important in social situations – including eating, drinking, speaking and facial expression – and altered self-image and differences in the reactions of others are likely to require changes to the routine advice given to people with social phobia. Repeated exposure to distressing events such as dribbling may reinforce negative thoughts about the self rather than minimise anxiety, as in typical phobic states. Specialist advice regarding make-up has improved patients’ confidence and mood (Kanzaki et al., 1998), which would be expected to aid social interaction despite helping patients ‘avoid’ their true appearance.

Newell & Marks’ study, therefore, does not support the conclusion that all social anxiety in patients with all types of facial disfigurement has the same psychopathology as social phobia. Cognitive–behavioural interventions probably need to address more than the avoidance or beliefs typical of social phobia. The need for exposure, a range of cognitive techniques, grief work, specialist physiotherapy and speech therapy is likely to vary. Future research should describe the type, course and severity of disfigurement and associated difficulties and clarify specific concerns occurring in social settings.


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Authors’ reply: We welcome Dr Butler’s comments and endorse the call for further research in the area of psychological difficulties following disfigurement. While we accept the call for caution, given the heterogeneous nature of our sample, we believe our conclusions are appropriately modest. We do not suggest either that phobic avoidance is the sole element of psychological distress following disfigurement, or that “all social anxiety” in such people has the same psychopathology as social phobia”. Indeed, the role of multiple contributing factors to such distress has been emphasised by one of us (Newell, 1991), and we noted our awareness of, but inability to investigate, the role of stigma. Moreover, Newell (1991) stresses the importance of sensitivity when advising of exposure exercises, precisely to reduce the potential for reinforcing negative thoughts and increasing anxiety, as Dr Butler suggests. More generally, the need for individualised treatment has been repeatedly stressed in behaviour therapy and cognitive therapy, although self-help methods (which, of necessity, give general prescriptions of advice which clients modify themselves) show promise. In the context of disfigurement, a simple self-help leaflet (Newell & Clarke, 2000) produced modest benefits relative to untreated controls and of a level roughly similar to those found in a group social skills intervention (Robinson et al., 1996).

Although the nature of the sample is important, it is difficult to obtain participants from this group, as previous studies have found. However, findings regarding gender differences among disfigured people with respect to psychological disturbances have been equivocal, and findings tend to suggest that level of disfigurement is a poor predictor of psychological adjustment.

Exposure therapy is obviously not a panacea, but rather a promising approach to psychological disturbance following disfigurement where social anxiety is present.

Dr Butler rightly draws attention to the need for flexible, individually tailored treatment, although this is questioned by some results (Schultz et al., 1992). There is likewise a need to avoid the inclusion of poorly supported interventions, and to build an appropriate evidence base. For example, we know of no studies that demonstrate the effectiveness of grief work among people with disfigurement, and there is likewise little evidence of the effectiveness of other interventions for psychological difficulties following disfigurement, despite the size of the problem.


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Outcome of hospital-treated depression

“Then you should say what you mean”, the March Hare went on. ‘I do’, Alice hastily replied; ‘at least – I mean what I say – that’s the same thing, you know.” (Lewis Carroll, Alice in Wonderland).

Tuma (2000) gives a ‘recovery’ rate of 24% for depressed elderly patients but this figure actually refers to his category ‘lasting recovery’. If the term recovery may be used to include those who have relapsed but then recovered for a (specified) period of time, the rate from Tuma’s study is 44%, or 66% once natural deaths have been removed. Tuma appears to be following Murphy’s (1987) view of recovery in depression as being a pint-pot only half-full.

This is not to disagree with the conclusion drawn from the study’s data that elderly patients with depression have a poorer prognosis than younger adults, but there is a need to respond to the call for more clarity, if not unanimity, in what terms mean (Frank et al., 1991). Low detection and treatment rates for depression in
older patients in the community are not likely to be improved by ‘term’-inally induced therapeutic nihilism. If we are to avoid such confusion, we should heed the words of another Lewis Carroll character:

“..When I use a word, ‘Humpty Dumpty said in a rather scornful tone, it means just what I choose it to mean – neither more nor less.”


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Anorexia nervosa: treatment with olanzapine

Anorexia nervosa is a multiply determined disorder of unknown aetiology. Restriction of food intake culminating in profound emaciation is considered to be pathognomonic (Kaye et al, 1999). One of the diagnostic criteria for anorexia nervosa is body image disturbance that is characterised by seeing and judging oneself to be fat and by claiming to ‘see’ oneself as fat despite being underweight. The bizarre body self-image in anorexia nervosa can be regarded as a psychotic way of thinking. We tried to treat anorexia nervosa as a psychotic disorder with olanzapine. Hansen (1999) has previously reported a case of treatment of anorexia nervosa with olanzapine; here we report three further cases.

A 30-year-old woman with anorexia nervosa since she was 18 years old additionally developed bulimia nervosa 5 years before presentation to our clinic. She was treated traditionally for many years without significant positive results. When she applied to our clinic her weight was 44 kg, height 167 cm. Mental state examination revealed severe body image disturbance. We commenced her on 5 mg olanzapine daily. In 9 months her weight was 53 kg, and she recovered from symptoms of both anorexia nervosa and bulimia nervosa. According to her drawings her body self-image changed from a fat little girl to an attractive grown-up woman. She still receives 5 mg olanzapine daily.

A 34-year-old woman had been suffering from anorexia nervosa and borderline personality disorder since puberty. When she came to our clinic her weight was 60 kg, height 180 cm. She complained of confusion, having too many thoughts in her head at one time. She had a seriously disturbed body image – in her drawings she looked like a little fat girl without hands and secondary sexual signs. She was started on olanzapine 5 mg, and after 2 months her body image has improved (she now sees herself as a grown-up woman) and she feels healthy.

In all three cases olanzapine was well tolerated. Our patients restored their body weight and appetite as well as their body self-image. They now think of themselves as normal, mature adults. The problem is to convince the patient to start and to continue with olanzapine therapy within the first 2 months, because it takes a few weeks before a full antipsychotic effect is achieved. We think that further investigations in this area should take place.


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Nicotine reduction: effectiveness of bupropion

Hayford et al (1999) and others have found sustained-release bupropion useful as part of nicotine reduction treatment programmes. However, our clinical experience suggested the primary benefit of sustained-release bupropion (300 mg/day) occurred within the first month of treatment and the recommended second month of medication was probably not helpful.

We evaluated the treatment progress of 74 (30 one-month, 44 two-month) research volunteers (further details available from the first author upon request). Treatment condition assignment was random but one-month patients had the option of an additional two months of medication if they were not successful at quitting at immediate follow-up, thus decreasing the number of one-month participants for this study. Volunteers had a mean age of 56 and were primarily male (91%), cigarette smokers (96%) and White (92%). One- and two-month groups did not differ significantly on a number of treatment motivation measures, demographic variables and self-reported health variables.

All patients signed consent forms and received the same behavioural treatment information in the first session. All were also instructed to return to a second meeting at which time their progress was evaluated and they received more behavioural strategy information and their assigned sustained-release bupropion. Participants were followed (a) as soon as convenient after nicotine quit date, (b) three-months after immediate follow-up, and (c) six-months after initial group meeting. About 80% of potential subjects volunteered for this project and follow-up return rate averaged 81%.

Use of one or two months of bupropion did not significantly affect self-reported quit rates at immediate, three-month or six-month follow-up periods. Self-reports of decreased nicotine intake among patients who did not quit entirely also did not differ between one- and two-month groups at immediate, three-month, or six-month follow-up periods. Only weight gain was associated with six-month treatment success. Participants who were successful reported more weight gain than those who were not. However, one- or two-month dosing schedule was not significantly associated with reported weight changes. Quitting success at six months was not related to age, gender, tobacco type, income category, race, combat exposure, years’ service, side-effect ratings, health problem ratings concerning breathing, heart, general medical, psychiatric, or substance dependence areas, religious behaviour, immediate weight gain, or initial self-ratings of treatment programme helpfulness/motivation/self-control/completion.
Paradoxical pattern of haematological risk with clozapine

I would be intrigued to hear further comments from Munro et al (1999) concerning the apparent paradox of the inverse relationship between dose and risk, both of neutropenia and of agranulocytosis.

A curious interaction of enzymes and metabolites, as briefly alluded too, is a fascinating possibility; other more banal explanations might also be entertained. One imagines the authors considered the possibility of an artefact. The agranulocytosis risk was the raisin d’être for the Clozaril Patient Monitoring Service (CPMS); so we may reasonably propose a tendency to (a) reduce the dose, and (b) fail to raise it, in those who exhibited lower white cell counts. Could such a mechanism produce these results? The data presented do not appear sufficient to rule out such an explanation.

Do the authors wish to comment on why the baseline white blood cell count should be associated with hazard of neutropenia but seemingly (from absence of specific data and comment) not with agranulocytosis?

Psychiatrists have, over the years, made minimal use of therapeutic drug monitoring and one presumes from this report that this was not incorporated in any way into the CPMS. Is there a lesson here and might that have elucidated the putative ‘ratio of metabolites’ question?


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Authors’ reply: We are grateful for the above comments and for the chance to cover the issue of clozapine dose and blood dyscrasias in more detail. Extensive analyses were done to explore this and the key message is that there is no evidence that risk of haematological discontinuation is related to increased dose.

The peak risk for both agranulocytosis and neutropenia on clozapine occurs in weeks 6–18 of treatment. It is likely that in these early stages of treatment, the dose of clozapine is still being titrated up to a typical maintenance level. The low doses of clozapine in patients with agranulocytosis or neutropenia reflect the overlap in time of peak risk for blood dyscrasias and the drug titration period. This is more likely to explain the finding, rather than the proposed tendency to reduce dose or fail to raise it in those who exhibit lower white cell counts. When it is noted that a patient’s white blood cell (WBC) count is falling, a course of action (e.g. repeat blood monitoring) is advised by the CPMS. This does not include any advice about reductions in or maintenance of clozapine dose, because the blood problems associated with the drug have been repeatedly reported as being dose-independent phenomena.

Low baseline WBC count was associated with increased hazard of neutropenia, but not agranulocytosis. It is likely that patients discontinuing clozapine for neutropenia have a number of different aetiologies behind the reduced WBC count. There is a natural variance in WBC count within the population. Those individuals tending to have a low count pre-treatment were more likely to be excluded for subsequent low counts coincidental to the clozapine treatment. This is shown by the expected finding that African–Caribbean patients have lower baseline WBC counts than Caucasians because of benign ethnic variation and, correspondingly, have higher rates of neutropenia on clozapine treatment. In sharp contrast, the rate of agranulocytosis in African–Caribbean patients is not increased. Frequency of agranulocytosis is clearly independent of the baseline WBC count, suggesting that different mechanisms exist to explain the neutropenia and agranulocytosis.

Owing to the editorial requirements to shorten the original draft, the discussion around the ratio of drug metabolites had to be truncated. The excellent suggestion regarding therapeutic drug monitoring and elucidation of the metabolite ratio, as a possible key to discovering the mechanism, points to a possible lost opportunity. Therapeutic drug monitoring is not undertaken or required routinely. However, if requested, the parent drug and the major metabolites can be measured. Unfortunately, these easily measured metabolites are very unlikely to yield important information regarding toxic mechanism, although they are proving useful in assisting with a variety of specific clinical situations such as drug interaction and suspected compliance problems. The characteristics of these major metabolites, present almost invariably in every patient at generally constant ratios, simply does not explain the frequency and temporal patterns seen for agranulocytosis. Agranulocytosis is unlikely to be due to the direct toxicity of the parent drug or these major stable metabolites. A more promising possibility involves the formation of a short-lived reactive metabolite, a nitrenium ion which binds to neutrophil proteins. This may be the mediator of the toxicity by disruption of neutrophil function or by acting as a hapten to invoke immune destruction of the neutrophil. The explanation of why only 0.73% develop agranulocytosis still has to invoke multifactorial possibilities based on individual differences in bioactivation and detoxification, which may be genetically determined.

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Hospital Anxiety and Depression Scale for use with adolescents

I had difficulties in answering important questions about the support of some of the conclusions that White et al (1999) draw in their work on validation of the Hospital Anxiety and Depression Scale for use with adolescents.

(a) It is not clear whether the non-clinical sample used in the study can be considered independent since the method of selection is not mentioned.

(b) Of the 248 children (110 girls) who were tested first, 180 were re-tested. However, the girl/boy ratio in the latter group is not indicated. Moreover, the out-patient sample comprised 48 subjects (27 girls) and the deliberate self-harm (DSH) sample had 38 subjects (30 girls). Considering the disproportionate group sizes and gender distribution it is surprising to find that the variances in the different groups are homogeneous. However, this information cannot be deduced from the published data. As a result, it is very difficult to assess fundamental requirements for the F-test.

(c) Girls are 44% of the non-clinical sample, 56% of the out-patient sample and, more importantly, 79% of the DSH group. The authors conclude that there is a statistically significant gender difference, with girls scoring higher than boys in both depression and anxiety scales. Assuming that the F-test’s requirements are met, then it may not seem surprising to find an overall significant difference detected by the F-test because of the characteristics of the DSH group.

(d) As the authors do not report multiple comparisons between the groups, it is not possible to know whether the differences remain when the DSH group is excluded.

(e) The analysis does not include techniques to control for gender, which appears to be a very important confounder.

(f) The authors assessed test–retest reliability with Pearson’s correlation coefficient. As this technique does not take into account errors of measurement, it does not measure agreement and its results are not meaningful.


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Authors’ reply: We welcome the opportunity to clarify the points raised by D. Marchevsky.

(a) The non-clinical sample was selected by asking the head teachers of each school to choose a selection of mixed-ability classes from each of the school year groups that fitted the age range we had selected.

(b) Of the 180 adolescents re-tested, 77 (43%) were girls, almost identical to the ratio of the first test sample (44%). The variances in the different groups are indeed heterogeneous, but the results of the analysis hold when the analyses are corrected for this effect or when non-parametric analyses are carried out. Limited space precluded us from reporting the full analyses.

(c) The results remain the same whether or not the deliberate self-harm group is included in the analyses.

(d) Robust multiple comparisons show that, for the depression sub-scale, the out-patients depressed group scores higher than the other three groups, with the other two clinical groups not differing significantly from each other, but both scoring significantly higher than the school sample. For the anxiety sub-scale, the three clinical groups do not differ significantly from each other, but all score significantly higher than the school sample.

(e) Analyses for each gender separately produce the same results.

(f) We see no problems with using the Pearson product–moment correlation as a measure of test–retest reliability. There is a long history of using this correlation as a measure of reliability in the psychometric test theory literature. Note that we are not measuring agreement between raters here, for which a measure such as kappa would be appropriate.

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One hundred years ago

Medical fees for lunacy cases

The Devonport Board of Guardians consider that the sum of one guinea is too large a fee for medical men to be paid in pauper lunacy cases and recently approached the borough magistrates with a view to having the fee reduced. At the meeting of the guardians held on Oct. 12th it was reported that the magistrates did not share the opinion of the guardians. Eventually the board decided to write to the members of the medical profession in Devonport asking them if they would accept a fee of half a guinea for these cases, and the matter will be again brought before the magistrates.

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Decreasing suicide in Hungary
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