Review

Why are programmes for offenders with personality disorder not informed by the relevant scientific findings?

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This paper examines the evidence to justify intervening in those with personality disorder, specifically antisocial personality disorder (ASPD) as defined by the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV, American Psychiatric Association 1994). The evidence from randomized controlled trials in the mental health literature is reviewed and found to be deficient with only five trials satisfying Cochrane criteria, all of which had a reduction in substance misuse as their primary outcome, rather than a change in the personality disorder *per se*.

Next, I consider the contribution of Thomas Kuhn to explain why it is difficult to develop a scientific basis in forensic mental health. I argue that, because forensic mental health is inclusive in its purpose (interacting with the law, social services and the penal system, all of which have different rules and agendas), it is difficult to develop a consensus on fundamentals, this consensus being a hallmark of a science.

Finally, I argue that despite the absence of evidence from mental health, providers for ASPD are in a fortunate position in being able to draw upon the correctional literature. This is relevant, provided that we agree that a reduction in offending is the primary outcome. While mental health can learn much from correctional practice, it can also enhance the efficacy of the latter by, for instance, drawing attention to the specific vulnerabilities of the personality structure that might impede programme delivery in correctional settings. Means of achieving a conjunction of mental health and correctional practice are urgently required as this would be beneficial to both.

Keywords: antisocial personality disorder; treatment; correctional programmes; systematic reviews

It is a characteristic of the design of scientific research that exquisite attention is devoted to methodological problems that can be solved, while the pretense is made that the ones that cannot be solved are really nothing to worry about.

(Lewontin 1995, p. 25)

1. INTRODUCTION

One of the most striking memories from this meeting was the discontinuity between the sophistication of many of the scientific papers, especially those from a developmental perspective, and the failure to translate these findings into clinical practice. This was best illustrated by one of the speakers who, after a very erudite exposition on the developmental origins of psychopathy, seemed at a loss to explain how his findings might inform the management of a difficult, out of control, teenager when questioned by a clinician.

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One contribution of 11 to a Discussion Meeting Issue 'The neurobiology of violence: implications for prevention and treatment'.

The rhetorical nature of the title underscores this dissonance between the research evidence and clinical practice. Clearly, as the above example demonstrates, there is a significant gap between what is known and what we need to know if we are to conduct clinical practice based on scientific evidence in this area. Despite this gap, clinicians will still have to deliver a service even though this will always be playing 'catch up' with the scientific evidence. This paper has two purposes: (i) to answer the question posed in the title and (ii) to come up with some practical recommendations to fill the 'gap' between the scientific evidence and clinical practice.

Returning to the title, even if the proposition implied is true, it poses a question that can be answered at a number of levels. In this paper, each of the following will be considered in turn:

- (i) What is the nature of the scientific evidence for effective interventions in offenders with personality disorder?
- (ii) If such evidence exists, under which conditions can mental health professionals ignore such evidence?

- (iii) Even when such evidence exists and is accepted by the professionals, can it still be ignored by policy makers and politicians?

In order to focus this endeavour, I will consider these questions in relation to antisocial personality disorder (ASPD)—specifically, as it is defined in the *Diagnostic and Statistical Manual of Mental Disorders* (DSM). Apart from a self-evident relationship between ASPD and violent offending, there may be surprise that I have chosen to concentrate on ASPD rather than psychopathy. After all, psychopathy has been the focus of many of the papers at this meeting. It is also much more sharply defined than ASPD and has been studied at much greater depth with much neuropsychological evidence identifying one or more of the mechanisms that might explain it. By contrast, ASPD is a much less credible diagnostic construct as it is poorly researched and has little theoretical underpinning (see Hare *et al.* (1991) and Ogloff (2006) for a review of the overlap between these constructs). Nonetheless, I believe that this focus on ASPD, rather than on psychopathy, is justified for the following reasons.

2. REASONS FOR TAKING ASPD SERIOUSLY

(a) *Epidemiology*

Estimates of the lifetime prevalence of ASPD in the general population vary with rates in North America of 4.5% in men and 0.8% in women (Robins *et al.* 1991), 6.8% in men and 0.8% in women (Swanson *et al.* 1994) both of these being significantly higher than in Europe that has corresponding figures of 1.3% in men and 0% in women (Torgensen *et al.* 2001) and 1% in men and 0.2% in women (Coid *et al.* 2006). Whether these differences are real differences in rates or a consequence of different methodologies is unclear; nonetheless, we can draw two conclusions. First, despite these relative differences, the rates of ASPD reported suggest that it is a prevalent personality disorder whereas psychopathy (as defined by the *Psychopathy Checklist-Revised*—PCL-R) is much less prevalent. For instance, while 50% of those in prison will meet criteria for ASPD, the prevalence of psychopathy among UK prisoners is only 4.5% using a PCL-R score greater than 30 and 13% using a score greater than 25 (Hare *et al.* 2000). Hence, the greater prevalence of ASPD in the population indicates that it is much more important than psychopathy, from a public health perspective.

Second, even with the most conservative estimates, ASPD in men has the same prevalence as major mental illnesses such as schizophrenia and bipolar disorder. However, these conditions receive the greatest attention and resources from mental health professionals while those with ASPD receive very little. I make this point, not in anyway to downplay the oftentimes devastating consequences of these disorders; rather it is to highlight that ASPD's high prevalence suggests that it deserves our attention, especially if it is associated with some impairment for the individual. Moreover, even for those with schizophrenia, it is often their comorbid ASPD that makes them difficult to manage, particularly as regards their violent behaviour (Bloom *et al.* 2000).

(b) *Evidence of an associated biological disadvantage for those with ASPD*

Identifying a disorder as being prevalent is not sufficient to justify the allocation of scarce human resources unless it is also associated with obvious disadvantages. The respiratory physician Scadding (1967), for instance, defined a disease in biomedical terms as a condition that would place the individual at a biological disadvantage when compared with other members of the species. The two obvious criteria by which this could be judged are either an increase in mortality or morbidity in the individual being so affected. As regards the former, Martin *et al.*'s (1985) follow-up of 500 psychiatric outpatients with a range of psychiatric conditions in St Louis found that those with ASPD had the second highest standardized mortality rate (SMR=8.57, $p=0.01$) exceeded only by those with drug addiction. Not only was this increased mortality due to an increased rate of suicide but it was also a consequence of reckless behaviour such as drug misuse and aggression. An even more striking finding was provided by Black *et al.* (1996) in their follow-up of men with ASPD. They found that young men with ASPD in particular had a high rate of premature death with those under the age of 40 having an SMR of 33 with the risk diminishing with increasing age.

As regards an increase in morbidity, ASPD is often comorbid with several other Axis I disorders, with the Swanson *et al.* (1994) community study showing that those with ASPD had an increased prevalence of '...nearly every other psychiatric disorder ... with 90.4% having at least one other psychiatric disorder.' Comorbidity with substance misuse is especially common. For instance, in the Epidemiological Catchment Area study, when men with and without ASPD were compared, those with ASPD were three and five times more likely to abuse alcohol and illicit drugs (Robins *et al.* 1991). It is also important to note that, while women have a significantly lower prevalence of ASPD than men, those women with ASPD have an even higher prevalence of substance misuse as compared with the men (Robins *et al.* 1991; Compton *et al.* 2005). In addition, ASPD has been found to co-occur with anxiety disorders in 54% (Goodwin & Hamilton 2003) and 47.5% (Lenzenweger *et al.* 2007) of the cases examined. The latter also found that 28% of those with ASPD also suffered from any mood disorder. One point that I will return to later is that the presence of ASPD is often a negative moderator when these comorbid conditions are treated with conventional approaches. As ASPD has also been shown to frequently co-occur with other Axis II conditions (Moran 1999), it is reasonable to conclude that ASPD is a condition with high morbidity.

(c) *ASPD is associated with increased costs, not only to the individual but also to society more generally*

ASPD is associated with fractured families, low educational attainment, a poor occupational history and a significant association with criminality. Hence, as the adverse consequences of ASPD extend well beyond the individual who is affected, their impact has a high societal cost. These costs arise directly as

increased costs to public services but also indirectly as losses in economic productivity. An example of the former is provided by Scott *et al.*'s (2001) comparison of the lifetime public costs of three groups of 27-year olds (those who were normal in childhood, those with some conduct disorder (CD) traits and those with CD in childhood). They found a 10-fold difference in the costs between those adults with and without CD in childhood. This difference will be important when I argue later that these high costs provide a justification for using expensive interventions in this group.

(d) *Early prevention*

ASPD is unique in that it is the only personality disorder (at least within the DSM) that requires an antecedent criterion from childhood (i.e. CD) in order to make a diagnosis. The criticism of this requirement has been made sharply by Millon *et al.* (1998, p. 8) in an often-cited quotation that '... the DSM-III Task Force voted to base its diagnostic guidelines on this single, albeit well-designed, follow-up study of delinquent cases referred to one child guidance clinic in a large mid-western city'. In addition, it has been difficult to distinguish those with adult criteria for ASPD only (so called 'late bloomers') from those with CD and adult criteria when they are compared across a range of characteristics independent of the diagnosis (Black & Braun 1998; Perdikouri *et al.* 2007).

Despite these criticisms, Robins' earlier finding has now been replicated by several prospective longitudinal follow-up studies identifying a substantial number of children where their CD with aggressive behaviour persists into adulthood (Robins *et al.* 1991; Moffit *et al.* 2001; Loeber *et al.* 2002; Simonoff *et al.* 2004; De Brito & Hodgins *in press*). Moreover, males with CD go on to commit more violent crimes in adulthood (Loeber *et al.* 2002). The important implication of the presence of this childhood criterion in the diagnosis of adult ASPD is that prevention at an early stage may be possible. Indeed, I take this to be the leitmotiv of this meeting with several of the papers (Hodgins; Loeber & Pardini) emphasizing (and providing evidence) that early identification and intervention is both possible and desirable.

(e) *ASPD and criminality are not the same (or are they?)*

One of the major criticisms of ASPD is its apparent emphasis on criminal behaviour in its definition. This has led to the belief that ASPD and its variants may be overdiagnosed in certain settings, such as prison, and underdiagnosed in the community (Lilienfeld 1998; Ogloff 2006). However, if ASPD was synonymous with criminal behaviour and vice versa, this would imply that all of those in custodial settings would meet criteria for ASPD and that ASPD would be rare in those without a criminal history. However, this is not the case. For instance, the prevalence of ASPD among prisoners is slightly less than 50% (Hart & Hare 1989; Singleton *et al.* 1998; Fazel & Danesh 2002). Even Robins, who has emphasized aberrant behaviour as a key to the diagnosis of ASPD, pointed out that only 47% of those who met DSM-III ASPD in the Epidemiological Catchment Area study (i.e. in the community) had significant arrest records with aggression, job problems and

promiscuity being more common than serious crimes (Robins 1987; Robins *et al.* 1991). Thus, while there is an important relationship between ASPD and criminal behaviour, this relationship is not straightforward with half of those convicted not meeting criteria for ASPD and half of those in the community with ASPD never having had a conviction.

Moreover, the majority of mental health practitioners do not regard the criminal focus for ASPD as defined by DSM-IV or one of the other diagnostic systems as being especially helpful in their clinical practice. Rather, they focus on unstable interpersonal relationships, disregard for the consequences of one's behaviour, a failure to learn from experience, egocentricity and a disregard for the feelings of others (Livesley *et al.* 1987; Tennant *et al.* 1990). Clearly, therefore, there is more to ASPD than criminal behaviour; the components of the underlying personality construct will be discussed later.

3. WHAT IS THE NATURE OF THE SCIENTIFIC EVIDENCE FOR EFFECTIVE INTERVENTIONS IN OFFENDERS WITH ASPD?

There is now general agreement that there is a hierarchy of evidence that one can call upon in deciding which intervention to use in a clinical case. This hierarchy places meta-analyses that summarize the results of relevant randomized controlled trials at the peak, followed by the results of well-conducted individual randomized controlled trials (RCTs) that in turn are followed by other forms of evidence (e.g. controlled studies, cohort studies, case series, etc.). While these weaker forms of design are regarded as less persuasive, their contribution ought not to be dismissed out of hand as RCTs do not arise *de novo*. In addition, it has been argued that, as interventions in this group are what have been termed as 'complex interventions', different criteria apply (Anon. 2000).

This has been ably reviewed for offender programmes by Hollin (2006) who points out that, as many of the interventions for offenders (and for those with ASPD) are complex interventions, it would be foolhardy to rely on any one methodology to provide the necessary evidence in this field. Hollin concludes with a quotation from Slade & Priebe (2001) that provides an argument for a diverse set of methodologies given the heterogeneity in the area:

Mental health research needs to span both the natural and social sciences. Evidence based on RCTs has an important place, but to adopt concepts from only one body of knowledge is to neglect the contribution that other, well-established methodologies can make... RCTs can give better evidence about some contentious research questions, but it is an illusion that the development of increasingly rigorous and sophisticated RCTs will ultimately provide a complete evidence base.

(Slade & Priebe 2001, p. 287)

While one might accept the trust of Slade and Priebe's argument, this carries a danger. If, for instance, one assumes that interventions in mental health are somehow different and more complex than other health

Table 1. Results from a systematic review of randomised controlled trials into the treatment of those with ASPD. (RR is relative risk, WMD is weighted mean difference, ASI is addiction severity index.)

type of analysis	study	comparison	outcome measure	results	favours
<i>antisocial personality disorder: psychological interventions</i>					
individual study	Brooner <i>et al.</i> (1998)	contingency management intervention (CMI) versus standard methadone substitution	transferred to routine care after 3 months	0.50 (0.26, 0.97) (RR)	CMI
individual study	Brooner <i>et al.</i> (1998)	contingency management intervention (CMI) versus standard methadone substitution	treatment dropout	1.38 (0.50, 3.84) (RR)	n.s.
individual study	Messina <i>et al.</i> (2003)	contingency management plus methadone maintenance (CM + MM) versus cognitive behavioural therapy plus contingency management plus methadone maintenance (CBT + CM + MM)	cocaine-free urine sample; during treatment (16 weeks)	1.70 (-9.72, 13.12) (WMD)	n.s.
skewed data	Messina <i>et al.</i> (2003)	CBT + MM versus CM + MM	substance use: cocaine-free urine sample; post treatment at 16 weeks	$p < 0.05$	CM + MM
skewed data	Messina <i>et al.</i> (2003)	CBT + MM versus CBT + CM + MM	substance use: cocaine-free urine sample; post treatment at 16 weeks	n.s.	n.s.
skewed data	Messina <i>et al.</i> (2003)	CBT + MM versus MM	substance use: cocaine-free urine sample; post treatment at 16 weeks	$p < 0.05$	CBT + MM
skewed data	Messina <i>et al.</i> (2003)	CM + MM versus MM	substance use: cocaine-free urine sample; post treatment at 16 weeks	$p < 0.05$	CM + MM
skewed data	Messina <i>et al.</i> (2003)	CBT + CM + MM versus MM	substance use: cocaine-free urine sample; post treatment at 16 weeks	$p < 0.05$	CBT + CM
<i>antisocial personality disorder: pharmacological interventions</i>					
meta-analysis	Leal <i>et al.</i> (1994) and Powell <i>et al.</i> (1995)	dopaminergic (amantadine) versus placebo	leaving the study early	1.18 (0.72, 1.94) (RR)	n.s.
meta-analysis	Leal <i>et al.</i> (1994) and Powell <i>et al.</i> (1995)	tricyclic (desipramine) versus dopaminergic (amantadine)	leaving the study early	0.74 (0.45, 1.23) (RR)	n.s.
meta-analysis	Leal <i>et al.</i> (1994) and Powell <i>et al.</i> (1995)	tricyclic (desipramine) versus placebo	leaving the study early	0.90 (0.52, 1.55) (RR)	n.s.
individual study	Arndt <i>et al.</i> (1992, 1994)	desipramine + standard methadone treatment versus placebo + standard methadone treatment	days medical problems in past 30 days: ASI: at 12 weeks	0.67 (0.21, 2.13) (RR)	n.s.
individual study	Arndt <i>et al.</i> (1992, 1994)	desipramine + standard methadone treatment versus placebo + standard methadone treatment	days worked in past 30 days: ASI: at 12 weeks	0.90 (0.43, 1.90) (RR)	n.s.
individual study	Arndt <i>et al.</i> (1992, 1994)	desipramine + standard methadone treatment versus placebo + standard methadone treatment	days opiate use in past 30 days: ASI: at 12 weeks	1.00 (0.15, 6.64) (RR)	n.s.
individual study	Arndt <i>et al.</i> (1992, 1994)	desipramine + standard methadone treatment versus placebo + standard methadone treatment	days cocaine use in past 30 days: ASI: at 12 weeks	1.13 (0.50, 2.52) (RR)	n.s.
individual study	Arndt <i>et al.</i> (1992, 1994)	desipramine + standard methadone treatment versus placebo + standard methadone treatment	days illegal activity in past 30 days: ASI: at 12 weeks	2.00 (0.40, 10.11) (RR)	n.s.

(Continued.)

Table 1. (Continued.)

type of analysis	study	comparison	outcome measure	results	favours
individual study	Arndt <i>et al.</i> (1992, 1994)	desipramine + standard methadone treatment versus placebo + standard methadone treatment	days family/social problems in past 30 days: ASI: at 12 weeks; days psychological problems in past 30 days: ASI: at 12 weeks	1.00 (0.54, 1.86) (RR)	n.s.
individual study	Arndt <i>et al.</i> (1992, 1994)	desipramine + standard methadone treatment versus placebo + standard methadone treatment	days psychological problems in past 30 days: ASI: at 12 weeks	1.25 (0.37, 4.21) (RR)	n.s.
individual study	Leal <i>et al.</i> (1994)	desipramine versus placebo	leaving the study early	1.14 (0.15, 8.99) (RR)	n.s.
individual study	Leal <i>et al.</i> (1994)	amantadine versus placebo	leaving the study early	2.50 (0.42, 14.83) (RR)	n.s.
individual study	Leal <i>et al.</i> (1994)	desipramine versus amantadine	leaving the study early	0.46 (0.13, 1.66) (RR)	n.s.
individual study	Powell <i>et al.</i> (1995)	nortriptyline versus bromocriptine	leaving the study early	0.85 (0.49, 1.46) (RR)	n.s.
individual study	Powell <i>et al.</i> (1995)	nortriptyline versus bromocriptine	substance use: not abstinent from alcohol: by 6 months	0.79 (0.57, 1.10) (RR)	n.s.
individual study	Powell <i>et al.</i> (1995)	nortriptyline versus placebo	leaving the study early	0.88 (0.50, 1.53) (RR)	n.s.
individual study	Powell <i>et al.</i> (1995)	nortriptyline versus placebo	substance use: not abstinent from alcohol: by 6 months	0.72 (0.53, 0.97) (RR)	nortriptyline
individual study	Powell <i>et al.</i> (1995)	bromocriptine versus placebo	leaving the study early	1.03 (0.62, 1.72) (RR)	n.s.
individual study	Powell <i>et al.</i> (1995)	bromocriptine versus placebo	substance use: not abstinent from alcohol: by 6 months	0.91 (0.75, 1.10) (RR)	n.s.
skewed data	Coccaro & Kavoussi (1997)	fluoxetine versus placebo	mental state: anxiety score: HARS-14: by 12 weeks	$F=5.5$ ANCOVA $p=0.03$	fluoxetine
skewed data	Coccaro & Kavoussi (1997)	fluoxetine versus placebo	behaviour: depression: HDRS-21: by 12 weeks	n.s.	n.s.
skewed data	Coccaro & Kavoussi (1997)	fluoxetine versus placebo	quality of life: HASSLES score: by 12 weeks	n.s.	n.s.
skewed data	Coccaro & Kavoussi (1997)	fluoxetine versus placebo	quality of life: uplift scores: HUS: by 12 weeks	n.s.	n.s.
skewed data	Leal <i>et al.</i> (1994)	desipramine versus placebo	drug use: \$ spent per week on cocaine: by 12 weeks	n.s.	n.s.
skewed data	Leal <i>et al.</i> (1994)	amantadine versus placebo	drug use: \$ spent per week on cocaine: by 12 weeks	n.s.	n.s.
skewed data	Leal <i>et al.</i> (1994)	amantadine versus desipramine	drug use: \$ spent per week on cocaine: by 12 weeks	n.s.	n.s.

areas so that alternative criteria in assessing their efficacy apply, this could easily result in them being treated differently (i.e. more disadvantageously) than other areas of health care where its arguments may be seen as a form of 'special pleading'. In the next section, therefore, I shall concentrate exclusively on describing those RCTs that met the Cochrane criteria for those with ASPD.

(a) Systematic reviews of treatments for ASPD

Similar scientific imperatives apply in conducting a systematic review as designing a RCT (i.e. to avoid bias and to have a methodology that can be replicated). This is achieved by specifying in advance of reviewing any of the literature of the following: the criteria by which trials are to be included/excluded on the basis of the Populations studied; the Interventions offered; the Control population against which the intervention is compared; and finally, the Outcomes that are relevant. It needs to be understood that making decisions on each of the components of this PICO acronym are based on common-sense assumptions and not on the science itself. Of especial relevance to this discussion is whether or not to include the offending literature in evaluating the effectiveness of interventions for those with ASPD?

There is an argument both for and against considering this literature. The argument in favour is that, unless there is a specific process that might exclude them, it is probable that half of those of inmates involved in correctional programmes will meet criteria for ASPD (i.e. it is unlikely that they would be specifically excluded as an ASPD diagnosis is not an inclusion/exclusion criterion in such programmes). Hence, there is an argument for including the results from this offending literature. The argument against is that since a diagnosis of ASPD has not been made, it is impossible to know who is being treated in these trials so that it would be foolhardy to draw any inferences as to their relevance for the treatment of ASPD.

In the systematic reviews to be described, we took a conservative position and restricted the review to studies in which an explicit diagnosis of ASPD had been made, a decision that effectively excluded the criminological literature. We leave it to others as to whether or not this was the correct decision but the point to note here was that this could not be regarded as a decision based on scientific evidence alone, rather it depended on value judgements and common sense. The reasons that guided our reviews are discussed at length in our two companion papers (Duggan *et al.* 2007, *in press*).

(b) Findings from the review

This review largely supported the findings of the two earlier reviews by Dolan & Coid (1993) and Warren *et al.* (2003) (i.e. that there was little evidence to support intervening in those with ASPD), while extending the previous searches to 31 December 2006. In this review, we were able to include only five trials on ASPD that meet Cochrane criteria (table 1). Although overall, there were as many trials for personality disorder of any kind between our previous review up to 2002 (Duggan *et al.*

2006) and between 2002 and 2006, it was noteworthy that there had been no new trials for ASPD between 2002 and 2006.

The second significant finding was that all the five trials (of which two involved a psychological and three a drug intervention) used, as their outcome measure, a reduction in substance misuse. Granted substance misuse is a serious problem in those with ASPD, yet none of these trials addressed the core psychological problem in those with ASPD. However, since I have previously mentioned that this core disturbance is difficult to describe, perhaps this is not too surprising.

Given the persistent problems in trying to produce the relevant evidence and our failure to do so, this begs the question as to why this is so. The reasons are not difficult to determine. Returning to the PICO acronym, one can see difficulties in each of these areas that are relevant to ASPD. For the population (*P*) component, there is no unanimity of approach, with many questioning the whole construct of ASPD, in particular seeing it in the DSM as being too wedded to criminality (Widiger & Corbitt 1993). Similarly, there are problems for the selection of the intervention (*I*) as, in the absence of a good theory of personality disorder (Epstein 1987), there is no clear steer as to which of a wide range of interventions might be used. As a consequence, it is difficult to make comparisons across studies as few use the same intervention. For the comparator (*C*), those trials that we reviewed were frequently underpowered. For instance, the mean number of participants in the pharmacological trials was 22, far too low a number to have any confidence in the findings. An additional caveat is the choice of the comparator, as in many trials this choice was likely to favour the experimental treatment. This has been a problem in other areas of mental health where attempts at replicating the findings from earlier trials, where a large effect size favouring the experimental treatment was found, failed when a more effective comparator was used in subsequent trials. For instance, when assertive outreach services were first compared against conventional services for those with serious and enduring mental illness in the USA, a large effect size was found in favour of the assertive outreach service (Stein & Test 1980). However, when an attempt was made to replicate this in the UK, it failed (Burns *et al.* 1999). One of the reasons to explain this discrepancy was that standardized or treatment as usual services in the UK were already reasonably effective, so that the magnitude of the benefit of an enhanced (and more expensive) service was unlikely to match the earlier USA results where conventional services were poorly resourced and organized (Tyrrer 2000).

Finally, let us consider the outcomes (*O*). A major criticism of existing trials in personality disorder in general is that there are far too many outcome measures used, a feature that makes cross-study comparison difficult, if not impossible. However, the criticism goes deeper than this technical consideration in systematic reviews as a recent review of outcomes in psychiatric practice condemned the many and varied outcome measures used as being useful neither to clinical practitioners nor to patients (Gilbody *et al.* 2003). Here, the excessive number of outcomes in mental

health contrasted unfavourably with the minimalist approach of rheumatology and oncology. Although mental health can legitimately claim to have conditions that are more complex in their outcome than, say, oncology, there is nonetheless a sense in which mental health trialists are self-indulgent in measuring too many outcomes, and that exercising greater restraint would serve themselves (and those who attempt to interpret their data) well.

In summary, I believe that any fair reading of the current literature on the efficacy of intervening in those with ASPD from a mental health perspective is that we have very little evidence to justify any intervention. However, there is evidence (described below) from the criminological literature that may justify intervening provided that the outcome is a reduction in re-offending.

Currently, therefore, we find ourselves in a curious predicament: namely, when ASPD is diagnosed in a mental health context, there is little evidence to justify intervening from the mental health literature. Conversely, when ASPD is not diagnosed, there may be some justification in providing interventions from the criminological or correctional literature. I will return to identifying the conditions required to 'square this circle' towards the end of this paper but I first make a brief digression to examine scientific evidence more generally from a philosophical or, perhaps, more accurately from a historiographical perspective.

(i) *If such evidence exists, under which conditions can mental health professionals ignore such evidence?*

Answering this question requires us to examine the nature of scientific inquiry itself. In the early to mid part of the last century, the nature of scientific inquiry was dominated by the Viennese logical positivist inductivist tradition suggesting that science advanced through an accumulation of valid facts (e.g. Carnap 1962). This view of scientific advance was subsequently challenged by a number of philosophers including Hanson (1958) and Polanyi (1958) who saw this view as being too simplistic as these scientific 'facts' were always viewed through a prism of an assumptive world that directed their inquiry.

For brevity, and to summarize this tradition, I shall focus on the work of Thomas Kuhn who wrote an influential work entitled *The structure of scientific revolutions* (Kuhn 1970). Kuhn was a student of theoretical physics who, during his late graduate career, shifted to the history, sociology and philosophy of science. In his work, Kuhn also challenged the accepted notion that advancement in science was a result of an incremental process of accumulating more and more valid 'facts'—the so-called '...scientific development as a process of accretion' (Kuhn 1970, p. 3).

In his exploration of scientific discovery, Kuhn observed that the kind of disagreements on fundamentals that appeared to pervade the social and psychological sciences (and I suspect that of mental health also) were not evident in such sciences as astronomy, physics or biology. To explain this difference in approach, Kuhn introduced the notion of the 'paradigm' in scientific research. This, now much overused term in general discourse, is defined by him as '...universally recognized

scientific achievements that for a time provide model problems and solutions to a community of practitioners.' (Kuhn 1970, p. viii). As a paradigm implicitly defined the legitimate problems and methods of a research field for succeeding generations of practitioners, 'normal science' was understood as scientists working on puzzles generated by the paradigm, without having to consider fundamentals. Thus, for Kuhn, (normal) science is '...like an accepted judicial decision in the common law, it is an object for further articulation and specification under new or more stringent conditions.' (Kuhn 1970, p. 23).

Kuhn (1970, p. 5) stresses—and this will surprise many scientists—that science is not a process of discovery, rather it is a process to confirm through 'puzzle solving' that which might already be anticipated. Because the questions that might be answered are already predefined by the paradigm, they need to be '...firmly embedded in the educational initiation that prepares and licenses the student for professional practice. Because that education is both rigorous and rigid, these answers come to exert a deep hold on the scientific mind'.

This 'deep hold on the scientific mind' plays a crucial role in determining the kinds of questions asked, the methods used to answer them, the answers that might be deemed acceptable, etc. But the crucial aspect is that practitioners buy into these assumptions in their training and by doing so they no longer have to concern themselves with fundamentals in the field (that are now taken as a 'given'). Hence, the scientist can focus on precise work that ultimately may generate anomalies. This ability to disregard 'fundamental questions' has an enormous advantage for the practitioner as it provides a consensual view of the world so that his/her function as a scientist is to confirm this through a series of more and precise measurements and technical accomplishments.

Training and membership of the scientific community is crucial to the functioning of paradigms. Kuhn writes, for instance, that 'The study of paradigms ...is what mainly prepares the student for membership in a particular scientific community with which he will later practice. Because he there joins men who learnt the bases of their field from the same concrete models, his subsequent practice will seldom evoke overt disagreement. Men whose research is based on shared paradigms are committed to the same rules and standards for scientific practice' (Kuhn 1970, p. 11). Moreover, it is exclusive, as professional researchers no longer write for the public 'Instead they (i.e. his research articles) will appear as brief articles addressed only to professional colleagues, the men whose knowledge of a shared paradigm can be assumed and who prove to be the only ones able to read the papers addressed to them' (Kuhn 1970, p. 20).

This commitment to greater precision demanded in the practice of normal science is critical in explaining how science advances, for it is this focused activity which inevitably leads to 'anomalies' that stretch the paradigm and eventually leads to its replacement (i.e. a scientific revolution). Hence, Kuhn challenged the progress of science as an incremental process of gathering valid facts, observing that science advanced through a series of paradigmatic shifts (or revolutions)

in which old phenomena (or facts) were viewed in new ways. Hence, 'progress' in science is best portrayed by a discontinuous process in which 'normal' scientific endeavours are punctuated by extraordinary change (i.e. revolutions). Examples are the Copernican revolution, the overthrow of the phlogiston theory, etc. However, according to Kuhn, these revolutions depended on scientists accepting the necessity of a shared paradigm in the first place.

While Kuhn's views have been criticized (e.g. Lakatos & Musgrave 1970), they do provide us with a possible answer as to why it is difficult to develop a science in the area of mental health in general and in forensic mental health in particular. Clearly, this latter task is difficult as it would require bringing together a broad range of disciplines—mental health, law, criminology, sociology, etc., all of which have a legitimate interest in the area but which also are likely to have different priorities and ways of working. Indeed, Kuhn himself acknowledges that 'History suggests that the road to a firm research consensus is extraordinarily arduous' (Kuhn 1970, p. 15) and '...it remains an open question what parts of social science have yet acquired such paradigms at all'. Unfortunately, without an agreement on fundamentals, it is easy for practitioners to choose whichever facts support their position and disregard those that contradict it. This, I believe, is one of the reasons why forensic mental health practice is slow to advance, *viz.*—that those who work within it are not working from a shared paradigm.

Therefore, I argue that the challenge facing mental health practitioners if they wish their area to become a science (à la Kuhn) is to achieve a consensus on fundamentals so that normal science can develop and prosper. And there is some evidence that this is beginning to occur. Consider, for instance, how mental health professionals altered their view on whether mental disorder was (or was not) associated with violent offending. In the 1970s, the prevailing view was that mental disorder was not associated with violent offending; yet, within a decade, this view was reversed and replaced by its opposite (i.e. the mental disorder was related to violent offending; Beck & Wechel 1998). This change of view was motivated by evidence that found increased rates of offending in those with mental disorder compared with the general population in several large cohorts (e.g. Hodgins *et al.* 1996), so that this evidence could not easily be discounted.

Once this association was accepted (I suggest that this represented a paradigmatic shift owing to the fundamental nature of the assumptions), it legitimized a large endeavour to develop more and more precise measurements of this association. Hence, over the past two decades, there has been a concentration on the development of new risk measures to detect the relationship between the mental disorder and an offending history. For instance, this has seen the development of Historical Clinical Risk-20, the violence risk scale, etc.,—this technological development being exactly the kind of activity that might be predicted by Kuhn's theory. Those who have developed these instruments were no longer concerned as to whether or not mental disorder was associated with offending. For them, the answer was clear and positive so that their role

was to articulate this with greater and greater precision by developing the appropriate technology to measure this association.

Hence, there is evidence—supported by much of the developmental and experimental neuropsychological presentation at this meeting—that 'science' is beginning to develop in this area. Notwithstanding these developments, as the first paragraph makes clear, there is a considerable gap between what the science is able to tell us and what we need to know to respond to clinical demands. As this response cannot wait until all the science is in, do we have anything sensible to recommend to those who are required to intervene in those with ASPD in their current day-to-day practice?

4. A POSSIBLE WAY FORWARD IN THE TREATMENT FOR THOSE WITH ASPD?

Although I have pointed out that there are very few RCT data to justify intervening in those with ASPD, this condition has two advantages over other types of personality disorder to legitimize intervening in this group. First, there is a very large correctional literature that may have some relevance to those with ASPD, given that 50% of those in correctional settings meet criteria for ASPD. Second, ASPD is associated, not merely with personal costs (although that is indeed the case, *vide supra*), but that it oftentimes has additional societal costs. These added societal costs need to be taken into account in any determination of the costs and benefits of intervening in this group.

Returning to the first point, I accept that the inclusion of the correctional literature reverses our earlier decision to exclude this in our systematic review; but I see this as being justified for the purposes of this paper. The correctional literature with interventions to reduce re-offending is considerable and, while it parallels that in the mental health, there is little overlap between them. This correctional or 'What works' literature has already been discussed at length elsewhere (Blackburn 2004; McGuire 2004; Andrews & Bonta 2006) so here I will merely refer to the summary table in figure 1 that shows the effect sizes of differing criminogenic programmes (taken from McGuire 2004). To aid interpretation, the outcome variable is further re-offending, with the vertical line being that of no effect, between the experimental and the control treatments. The bars to the right are those treatments where the experimental treatment increased the likelihood of further re-offending (i.e. had a negative effect) and those to the left are experimental treatments that reduced likelihood of further re-offending.

This table allows us to draw two important conclusions. Criminogenic programmes (bars extending to the right) that have a punitive intent, either have no effect or increase the likelihood of subsequent re-offending. Conversely, criminogenic programmes (bars extending to the left) with a therapeutic ethos have a positive effect in reducing future re-offending. Many of these positive programmes are based on cognitive behavioural principles and on the risk-need-responsivity principle (Andrews & Bonta 2006). The principles of this programme are to match the programme with the offender's risk to re-offend (the *R* component), to assess

Figure 1. Variations in effect sizes (McGuire 2004). The figure shows effect sizes for selected types of interventions from meta-analytic reviews, to illustrate the range of findings obtained. In the graph, the vertical line represents zero change (no difference between experimental and control samples). Bars extending to the right represent *increases* in recidivism among experimental samples relative to controls. Bars to the left represent *reductions* in recidivism.

criminogenic needs and target these with treatment (the *N* component) and finally, to tailor the intervention to the capacity of the offender to benefit from them (the *R* component). It is interesting to note in passing that in their more recent work, Andrews & Bonta (2006) distinguish between a general and a specific responsivity. They describe the latter as a 'fine tuning' of the CBT approach so that it takes into account the personality characteristics of the individual—a point that we will discuss below. While criminogenic programmes have been criticized by mental health professionals as sometimes being too formulaic, this is often a result of inadequate resources rather than anything inherent to the programme as the 'responsivity' element makes clear.

5. THE IMPORTANCE OF ESTIMATING THE COSTS AND BENEFITS IN TREATMENTS FOR THOSE WITH ASPD

Although some of these correctional programmes are effective, compared with the (usually) no treatment control, the decision to invest in them also needs to

consider the costs of implementing the programme versus the money saved as a consequence. Cost effectiveness in studies is only now becoming commonplace in the evaluation of mental health interventions (for instance, only 4 out of the 27 trials (15%) reported a cost effectiveness component in our systematic review described earlier); these studies are more commonplace in a correctional setting, although these are again not without their critics (Welsh & Farrington 2000; McDougall *et al.* 2003). Nonetheless, I believe that correctional programmes lead the way in that they have a simple dichotomous outcome measure (re-offended or not?) and hence can provide data on costs that can usefully be interrogated.

As an example, I will briefly describe a study by Aos *et al.* (2001) who provided a report on the cost effectiveness of different programmes to reduce re-offending in Washington State. This systematically analysed different programmes to determine whether their benefits (as measured by the value to the tax payer and to the victim as a result of subsequent crime

Figure 2. Net economic benefits of programmes designed to reduce crime, with monetary values in 2000 \$ (adapted from Aos *et al.* 2001).

reduction) were likely to outweigh their costs. The purpose of this exercise was clear: '... to help decision makers in directing scarce public resources *towards* economically successful programs and *away from* unsuccessful programs ...' (my italics). Thus, the imperative to policy makers and those who controlled the public purse was clear: invest in those programmes that show a positive cost benefit and disinvest in programmes with a negative cost benefit.

The findings of Aos *et al.* are reported in figure 2 and table 2. Here, the vertical line of no effect separates programmes that have a net cost benefit (on the right) with those with a negative benefit on the left. The programme evaluations are sequenced by age with the earliest interventions in childhood at the top proceeding to interventions for juveniles and finally to those for adults. A summary of the conclusions from this table briefly is that the greatest cost benefit advantage is intervening in juveniles; the greatest cost benefit is on the victim (not surprisingly) rather than on the society at large; programmes of the same effect size (e.g.

quantum opportunities programme and multisystemic therapy, both of which had an effect size of -0.31) may have very different benefits when their costs are taken into account.

No doubt, there is much of Aos *et al.*'s analysis that is deficient and will be contested. The reason, however, for reporting it here is to illustrate that this is a level that mental health programmes need to attain if they are to remain competitive in attracting funding from the public purse to support their services.

In summary, I believe that one can draw the following conclusions from the Maguire and Aos *et al.*'s data: (i) that interventions that are therapeutic, rather than punitive, are more likely to be beneficial in reducing re-offending, (ii) that the magnitude of this effect is age dependent (i.e. the greatest impact is on juvenile offenders with a reduced effect on young children or adults), and (iii) for some crimes (especially those involving violence), the cost benefits in favour of the intervention are often considerable as the costs of these types of crimes are often very high.

Table 2. Cost benefits for different interventions to reduce offending. (after Aos *et al.* 2001.)

type of programme	effect size	cost of programme/ head	net benefit for tax payer	net benefit for tax payer and victim
quantum opportunities programme	-0.31	\$18 964	-\$8855	\$16 428
multisystemic therapy	-0.31	\$4743	\$31 681	\$131 918
in-prison therapeutic community	-0.05	\$2804	-\$899	\$5230
CBT sex offender treatment	-0.11	\$6246	-\$778	\$19 534
intensive supervision	-0.03	\$3296	-\$2250	-\$384
reasoning and rehabilitation	-0.07	\$308	\$2202	\$7104

While, overall, the effect of these criminogenic programmes in reducing re-offending is positive, their overall effect size is not massive. Losel (1998), for instance, has calculated that their mean effect size is 0.10–0.12 (i.e. a 10–12% reduction of the active intervention over the control). Further, despite the theoretical power of the RNR model, it is not without its critics who argue for its reconstruction (Ward *et al.* 2007). If we assume the following:

- (i) 50% of those in prisons have ASPD,
- (ii) those with ASPD are as probable as not to be treated in correctional programmes (as there is no explicit attempt to exclude them as the presence or absence of ASPD is not an entry criterion to treatment), and
- (iii) the presence of ASPD itself, but moreover, its co-occurrence with many other Axis I and II conditions is likely to have a detrimental moderating effect on the effectiveness of correctional programmes (Newton-Howes *et al.* 2006),

then these assumptions have the following therapeutic implications.

One could argue, for instance, that this modest effect of current correctional programmes might be enhanced if the specific psychological vulnerabilities of the subset of ASPD offenders were addressed in programme development (as suggested by Andrews & Bonta 2006, above). This might include a need to address a high level of impulsivity, treating co-occurrent Axis I disorders, substance misuse, non-compliance with treatment, etc., many of which feature in those with ASPD. For instance, Tyrer *et al.* (2003) subdivides those with personality disorder into treatment seekers and treatment rejectors with those with ASPD having several features suggesting that they would reject treatment (e.g. hostility, poor education, etc.). Consequently, there is much that mental health services might be able to contribute to the already existing programmes for offenders.

For this to be meaningful, however, we need a mechanism that links convincingly the features of ASPD sufferers to their poor response to interventions. And, for this, we need a theory of personality disorder. Unfortunately, we currently lack such a theory for personality in general and for ASPD in particular. This echoes Epstein's (1987) observation made 30 years ago on the absence of a good theory for personality disorder. 'The joker in the deck is how to get a good theory. Clearly, the essence of a scientific enterprise is

a continuous interaction between observation and conceptualization or, expressed otherwise, between empiricism and theory construction.'

Although much of the Epstein's plea still remains the case, I believe that we are now in possession of a sufficient number of 'empirical' facts to begin to develop such a theory. For instance, this conference, and others like it, has identified a prototypic ASPD individual as someone with a development trajectory which, as a result of innate temperamental difficulties interacting with a harsh and inconsistent parenting style results in a '...child (that) never acquires the social skills and regulatory mechanisms necessary to navigate the world of adolescence. The child consistently fails to attend to relevant social cues, readily makes hostile attributions about peers and adults, accesses aggressive responses in social situations, and either impulsively performs these responses, without thinking about their consequences or evaluates their probable outcomes as acceptable and selects them.' (Dodge 2000).

The clinician dealing with those with ASPD will recognize many of these features (i.e. the lack of social skills, the paranoid thinking that leads to hostile attributions, aggression in social situations and impulsivity without thought as to the consequences, etc.). The problem with this description is that it is merely a collection of undesirable traits. What is required therefore is a unifying theory that brings these disparate elements together to produce a personality construct.

What then are the core psychological characteristics of those with ASPD that could aid theory development? I believe that these are best captured by Benjamin in the following quotation:

There is a pattern of inappropriate and unmodulated desire to control others, implemented in a *detached manner*. There is a strong need to be independent, to resist being controlled by others, who are usually held in contempt. There is a willingness to use untamed aggression to back up the need for control or independence. The ASP(D) usually presents in a friendly, sociable manner, but that friendliness is always accompanied by a baseline position of detachment. He or she doesn't care what happens to self or others.

(Benjamin 1993, p. 198) (my italics).

My interpretation of Benjamin's quotation is as follows: First, the core personality characteristics of ASPD are described as both (i) a need to be in control and (ii) also to be detached. (This resonates with the 'callous unemotional behaviour' ascribed

to the psychopath.) Second, there are the secondary consequences of these primary characteristics of (i) using 'untamed aggression' to maintain control and (ii) a carelessness (or thoughtlessness) about what happens to self or others, thereby leading to risky behaviour. Thus, while the surface phenomena of violent and irresponsible behaviour may be the most visible aspects of ASPD, Benjamin draws to our attention that this behaviour is a consequence of a deeper psychological need to be in control while, at the same time, to remain detached. This need to be in control explains why those with ASPD are largely 'treatment resisting' so that they are (i) difficult to engage in treatment and (ii) difficult to maintain in treatment once engaged. I suggest that correctional programmes for those with ASPD need to be informed by this thinking and enhanced accordingly if their current level of effectiveness is to be improved.

6. IS THE PROBLEM THAT THE EVIDENCE EXISTS BUT IT IS IGNORED BY POLICY MAKERS AND POLITICIANS?

In the light of the foregoing, it is difficult to argue that policy makers and politicians ignore evidence offered to them as it is clear that there is little evidence to start with. Muir-Gray (2001) indeed makes the point that politicians are often blamed unfairly for ignoring scientific evidence when, in fact, the real issue is that this evidence does not exist. He provides an example where Government made a decision to reject mass screening for carcinoma of the prostate on the evidence from two high-quality meta-analyses. The implication is clear: that there is an imperative for scientists and clinicians to provide decision makers with the appropriate evidence to allow the latter to arrive at the best decision.

Muir-Gray goes on to distinguish between 'decision making' and 'decision taking'. The former involves (and indeed ought to involve) contributions from scientists and clinicians so that those who ultimately have to make decisions on behalf of the public are properly informed. However, when these decisions are finally 'taken', additional considerations that go beyond the 'evidence' where professionals seek and ought to make a contribution come into play, as 'values' and 'resources', and have also to be considered by those who have charge of the public purse. As the trust of this contribution is that we have failed at the decision-making phase (i.e. in not being able to provide evidence for the reasons given above), we can hardly blame politicians and policy makers at the decision-taking phase if we believe that the correct decision has not been made.

7. CONCLUSION

In answering the question in the title, my response is that

- (i) We have very limited scientific evidence for effective interventions in offenders with ASPD from the mental health field. However, the evidence from correctional programmes does exist and it is probable that these interventions

are being offered to those with ASPD—both in custodial settings and in the community by probation services. The effectiveness of these interventions is likely to be enhanced if the core psychological characteristics of those with ASPD are taken into account.

- (ii) The failure to develop a consensus on some crucial aspects of ASPD and its interventions has severely hampered the field and limits progress. This failure to agree on fundamentals is partly a result of the several different disciplines in the field with an absence of consensus inhibiting its 'scientific' development. This needs to be overcome if the area is to produce high-quality scientific evidence.
- (iii) In the light of (i) and (ii), we are in a weak position to influence the political process in the allocation of funds so that unless and until these areas are addressed, interventions for those with ASPD are likely to continue to remain in a scientific limbo.

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