



## Antisocial features and “faking bad”: A critical note<sup>☆</sup>



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### ABSTRACT

We critically review the literature on antisocial personality features and symptom fabrication (i.e., faking bad; e.g., malingering). A widespread assumption is that these constructs are intimately related. Some studies have, indeed, found that antisocial individuals score higher on instruments detecting faking bad, but others have been unable to replicate this pattern. In addition, studies exploring whether antisocial individuals are especially talented in faking bad have generally come up with null results. The notion of an intrinsic link between antisocial features and faking bad is difficult to test and research in this domain is sensitive to selection bias. We argue that research on faking bad would profit from further theoretical articulation. One topic that deserves scrutiny is how antisocial features affect the cognitive dissonance typically induced by faking bad. We illustrate our points with preliminary data and discuss their implications.

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### 1. Introduction

Terms like malingering, symptom exaggeration, feigning, simulation, and faking bad are often used as loose equivalents. The Diagnostic and Statistical Manual of Mental Disorders, fourth edition (DSM-IV TR) defines malingering as “the intentional production of false or grossly exaggerated physical or psychological symptoms, motivated by external incentives” (American Psychiatric Association, 2000; p. 739). It stresses that clinicians should suspect malingering when two or more of the following conditions are present: The symptoms are reported within a forensic context, they contrast sharply with objective findings, there is lack of cooperation during diagnostic evaluation, and/or the patient meets criteria for antisocial personality disorder (ASPD). The new edition of the DSM (i.e., the DSM-V) does not contain substantial revisions of how it portrays malingering (American Psychiatric Association, 2013; p. 726–727; see for a critical analysis: Rogers, 2008; Berry & Nelson, 2010; Bass & Halligan, 2014). The DSM’s description of malingering has been characterized as a criminological model, because it assumes that malingering is “an antisocial act that is likely to be committed by antisocial persons” (Rogers, 2008; p. 9). Given that the DSM is a widely used and highly influential source, the conceptual and empirical underpinnings of its criminological typology of malingering warrant critical

reflection, which is the aim of the current article. We will employ the term faking bad rather than malingering because the latter term assumes the presence of independent evidence that exaggerated symptom reports are motivated by external incentives (Bass & Halligan, 2014). Yet, such evidence is not always available.

The detection of faking bad is a challenge for clinicians. Unstructured interviews generally yield low detection rates, meaning that many cases will be missed if clinicians solely rely on their subjective judgment (e.g., Rosen & Phillips, 2004). Indeed, intuitive clinical judgment yields detection rates of faking bad that are comparable to the disappointingly low hit rates (i.e., <60%) found for intuitive judgment in the broader deception–detection literature (Vrij, 2000). Against this backdrop, a wide array of tests has been developed that intend to provide an indication of the credibility of symptom reports. When employing these instruments, empirically based cut-offs aid in determining whether symptoms are likely to be genuine or not (Merten & Merckelbach, 2013). A reasonably high diagnostic accuracy can be obtained when multiple detection tests are combined. Two response styles have been identified as targets of these dedicated detection tools: Exaggeration of symptoms and intentional underperformance (Dandachi-FitzGerald, Ponds, Peters, & Merckelbach, 2011; Iverson, 2006; Van Oorsouw & Merckelbach, 2010). Thus, patients who engage in faking bad may claim an abundance of atypical symptoms on specialized self-report questionnaires such as the Structured Inventory of Malingered Symptomatology (SIMS; Smith & Burger, 1997; see for other examples Table 1), and/or they may tend to perform extremely poorly on simple cognitive tasks such as the Test of Memory Malingering (TOMM; Tombaugh, 1996; see for other examples Table 1).

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**Table 1**  
Summary of studies examining psychopathy, ASPD and faking.

Study	Year	Subjects	Psychopathy & ASPD instruments	Faking bad/good instruments	Statistics	Findings/conclusion	Link yes/no
<i>Prevalence studies for faking in psychopathy (n = 5)</i>							
Kucharski et al.	2006	N = 188 male criminal defendants	PCL-R	MMPI-II, PAI, SIRS	ANOVA	High psychopathy group scored higher on MMPI-II subscales than low/medium groups; MMPI-II F: $F(2,189) = 8.43, p < .01$ , MMPI-II F-K: $F(2,189) = 10.20, p < .01$ , MMPI-II Fb: $F(2,189) = 5.99, p < .01$ , MMPI-2 F(p): $F(2,189) = 7.19, p < .01$ ; the PAI Negative Impression-scale: $F(2,164) = 6.63, p < .01$ ; and the sum of SIRS scales: $F(2,107) = 6.18, p < .01$ .	Yes
Cima et al.	2008	N = 118 controls and 34 prison inmates	PPI	SS-R	Pearson correlations	Psychopaths did not exhibit more faking good than non-psychopaths.	No
Freeman and Samson	2012	N = 300 non-incarcerated community members	SRP-III	IM subscale	Chi-square	Higher psychopathy was associated with lower faking good, $r = -.55, p < .01$ .	No
Heinze and Vess	2005	N = 392 male hospitalized forensic patients	PCL-R	MMPI-II	Chi square	Those scoring high on the PCL-R more often engaged in faking bad than those scoring medium or low on the PCL-R, $\chi^2 = 6.95, df = 2, p = .03$ .	Yes
Cima and Van Oorsouw	2013	N = 31 male prison inmates	PPI (Factor 1 and 2)	SIMS	Correlations	PPI-1 was unrelated to faking bad, while PPI-2 was related to faking bad, $r = .44, p < .05$ .	Yes/No
<i>Prevalence studies for faking in ASPD (n = 4)</i>							
Grillo et al.	1994	N = 90 personal injury claimants	MCMI-II	MMPI-II	Correlations	The antisocial subscale was correlated with several MMPI-II subscales. MMPI-II F: $r = .26, p < .01$ , MMPI-II K: $r = -.44, p < .001$ , MMPI-II L: $r = -.30, p < .01$ , MMPI-II F-K: $r = .42, p < .01$ , MMPI O-S: $r = .35, p < .001$ .	Yes
Delain et al.	2003	N = 64 criminal forensic participants	RRF	TOMM	Chi square	Those who scored below the cut-off of the TOMM ( $n = 25$ ) more often met ASPD criteria than controls ( $n = 31$ ), $\chi^2 = 3.86, df = 1, p = .05$ .	Yes
Sumanti et al.	2006	N = 233, compensation claimants	PAI (ANT-subscale)	Rey 15-item test, Dot-counting test, PAI-NIM, PAI-MAL, PAI-RDF	Correlations t-tests	Only significant for PAI-NIM, $t = 50.28, p < .05$ , indicating that subjects who scored above the PAI-NIM cut-off, and thus engaged in faking bad, also scored higher on antisociality.	Yes/No
Pierson et al.	2011	N = 71 forensic patients with/without ASPD	SCID-II	SIRS	Chi square	ASPD individuals did not score higher on SIRS than those without ASPD.	No
<i>Deceptive ability studies (n = 6)</i>							
Boone et al.	1995	N = 154 litigation subjects	MCMI	Rey 15-item test/DOT-counting test	Kruskal–Wallis analyses	No difference in antisocial scores between those failing and passing faking tests.	No
Edens et al.	2000	N = 143 students tested twice: once instructed to fake bad and once honest	PPI	MMPI-psychoticism scale, DPS, Validity scales of the PPI	Group comparisons were difficult due to skewness of data.	Psychopathic traits were not associated with passing fake bad subscales.	No
Poythress et al.	2001	N = 55 Male prison inmates Half instructed to fake bad. Other half was clinically judged to be malingering	PPI	SIMS, SIRS, PAI	Correlations	Psychopathy was unrelated to successfully faking bad.	No
Book et al.	2006	N = 201 students instructed to fake good ( $n = 96$ ) or fake bad ( $n = 105$ )	LSRP	HPSI	ANOVA	Psychopathic traits were unrelated to faking bad. Those who were caught faking good did display lower total psychopathy scores, $F(1,92) = 8.72, p < .01$ .	Yes/No
MacNeil and Holden	2006	N = 200 students instructed to fake bad/good	PPI	HPSI, BIDR, PRF-D	t-tests	Most findings were not significant. However, higher scores on the PPI subscale Machiavellian Egocentricity were related to faking good on, HPSI: $t = 2.78, p < .01$ ; IM: $t = 2.56, p < .05$ ; DFA: $t = -2.17, p < .05$ , while higher scores on PPI Blame Externalization were related to faking good on, HPSI: $t = 3.96, p < .001$ ; IM: $t = 2.06, p < .05$ ; DFA: $t = -1.98, df = 198, p < .05$ .	Yes/No
Marion et al.	2012	N = 465 undergraduates and 122 male criminal defendants	PPI-R, TriPM, LSRP, PCL-R	MMPI-2-RF, SIRS	Hierarchical regression analysis	Those high on psychopathy were not better at faking bad than those low in psychopathy. In contrast, individuals high on callous–unemotional–aggressive–traits were worse at avoiding detection.	No

Notes. ASPD = Antisocial Personality Disorder. Instruments to assess psychopathy/ASPD: MCMI-II = Millon Clinical Multiaxial Inventory-II. RRF = Standardized Record Review Form (for current DSM diagnoses. PCL-R = Psychopathy Checklist-Revised. PAI = Psychological Assessment Inventory (Antisocial subscale). PPI (-R) = Psychopathic Personality Inventory (Revised). SCID-II = Structured Clinical Interview for DSM-Axis II diagnoses. SRP-III = Self-Report Psychopathy Scale-III. LSRP = Levenson Self-Report Psychopathy scale. TriPM = Triarchic Psychopathy Measure. Measures to detect faking: MMPI-II = Minnesota Multiphasic Personality Inventory-II, MMPI-2-RF = Restructured Form. TOMM = Test of Memory Malingering. PAI = Psychological Assessment Inventory, PAI-NIM = Negative Impression Scale, PAI-MAL = Malingering Index, PAI-RDF = Rogers Discrimination function. SIRS = Structured Interview of Reported Symptoms. SS-R = Supernormality Scale-Revised. IM = Paulhus Deception Scales; Impression Management Scale. SIMS = Structured Inventory of Malingered Symptomatology. DPS = Dissimulation Potential Scale. HPSI = Holden Psychological Screening Inventory. BIDR = Balanced Inventory of Desirable Responding. PRF-D = Personality Research Form Desirability Scale.

Studies administering dedicated detection instruments to criminal forensic samples have reported prevalence estimates of faking bad of up to 65% (e.g., Alwes, Clark, Berry, & Granacher, 2008; Ardolf, Denney, & Houston, 2007; Denney, 2007; McDermott, Dualan, & Scott, 2013). Such impressive statistics are not surprising, given that the stakes are often high in the forensic arena. For example, defendants may reason that it helps their legal case when triers of fact think that they suffer from a mental disorder. Or sentenced inmates may fake symptoms because they want to be transferred to a different ward or want to be prescribed stimulant medication (McDermott et al., 2013). However, incentives for faking bad may also be present in non-forensic samples (Van Egmond & Kummeling, 2002). Direct comparisons of faking bad rates between non-forensic and forensic samples have not been reported in the literature. Still, faking bad estimates of expert respondents in Mittenberg, Patton, Canyock, and Condit (2002) were considerably lower for non-forensic samples than for forensic samples (i.e., 7%–12% versus 20%–30%, respectively).

As long as faking bad goes undetected, it represents a threat to decision making. That is, faking bad may compromise the integrity of clinical data underlying evaluations with regard to diagnosis or therapy progress (Dandachi-FitzGerald et al., 2011; Merten & Merckelbach, 2013; Rosen, 2006), and sometimes it may even impact judicial decisions about competency to stand trial or criminal responsibility (Van Oorsouw & Merckelbach, 2010).

## 2. Faking bad: shortcomings of the criminological model

The DSM contains a disclaimer cautioning against its use within the forensic setting. Specifically, it stresses that diagnostic information may be misused in such a setting due to the imperfect fit between the fields of law and clinical diagnostics (American Psychiatric Association, 2013; p. 25). Nonetheless, its often-cited criminological typology of malingering may lure clinicians into believing that the criminological model is a valuable starting point whenever the suspicion of faking bad is raised. The assumption of the criminological model that potential incentives increase the probability of faking bad rests on an empirical basis (Bianchini, Curtis, & Greve, 2006; McDermott et al., 2013). Yet, in other respects, the criminological model is problematic (e.g., Berry & Nelson, 2010; Otto, 2008; Rogers, 1990). In this article, we discuss three of its shortcomings that may have dire consequences for clinical practice. First, the model assumes an intimate link between antisocial personality features and faking bad, but it fails to elaborate on the details of this link (e.g., Salekin, Kubak, & Lee, 2007). Are antisocial individuals more likely than others to engage in faking bad, as DSM's criminological model seems to imply? Or do they have better deceptive abilities such that they are superior in evading detection, as some clinical accounts of psychopathy seem to suggest (see, for a critical analysis, Klaver, Lee, Spidel, & Hart, 2009)? Below, we review the relevant literature and conclude that findings on the prevalence of faking bad among antisocial individuals are inconsistent, and that there is little support for the notion that individuals with psychopathy or ASPD possess superior faking skills.

A second limitation is that the criminological model focuses on one form of faking, namely faking bad (Otto, 2008). However, particularly in a forensic context, faking good might be just as important. Faking good refers to the exaggeration of virtues and good qualities, while simultaneously downplaying less favorable characteristics or symptoms (Cima et al., 2003). Forensic patients who engage in faking good may pretend to no longer suffer from symptoms that they, in reality, still have. At first glance, faking bad and faking good seem to be behavioral opposites. However, they may be interrelated in a dynamic way. Below, we present pilot data illustrating that both faking bad and faking good are, indeed, relevant dimensions to consider in a forensic population.

A third limitation of the criminological model of faking bad is that it ignores the possibility that faking may go along with genuinely felt symptoms. We review literature and data suggesting that unless people

have antisocial features, faking bad can produce somatoform-like symptoms that the person comes to experience as real. Thus, antisocial features (including psychopathy) might be more relevant to the consequences than to the mere occurrence of faking bad, an issue to which we will return below.

## 3. Psychopathy, ASPD, and faking: a qualitative review

ASPD is a DSM diagnosis, whereas psychopathy is not. Although there is overlap between these conditions, most individuals with ASPD are not psychopathic (Hare, 2003), and the conditions differ in important respects. Briefly, psychopathy requires the presence of personality traits such as superficial charm, a glib interpersonal style, guiltlessness, egocentricity, and a lack of empathy, whereas ASPD primarily refers to a chronic pattern of norm violation (Hare & Neumann, 2006). For both concepts the presumed association with faking bad has much *prima facie* plausibility. In the case of psychopathy, inherent features such as conning, manipulation, and exploitation of others can be easily interpreted as ingredients of faking bad. In the case of ASPD, plausibility stems from the element of social transgression that is common to both ASPD and faking bad. Plausibility aside, is it, indeed, the case that individuals with psychopathy or ASPD engage in faking bad more often than others?

In what follows, we present a *qualitative* review of the extant empirical literature. Two approaches have been employed to examine the relationship between faking and antisocial features. The first approach (prevalence research) tests whether faking is more likely to occur among those diagnosed with either psychopathy or ASPD, while the second (deceptive ability research) evaluates whether these individuals are well-versed in faking.

We extensively searched three databases (PsycInfo, PubMed, and GoogleScholar) with a variety of search terms, including *psychopathy*, *antisocial personality disorder*, *psychopath(s)*, and *antisocial(s)* combined with terms that refer to dishonest reporting, namely *malingering*, *feigning*, *faking (bad and good)*, *dissimulation*, *simulation*, *over/underreporting*, and *response styles*. We limited our search to peer-reviewed studies published in English journals between 1990 and 2013 that employed standardized measurements to tap into psychopathy/ASPD and independent (i.e., stand alone) measures of faking bad/good, and that relied on samples of more than 20 participants. We found 15 studies that met these criteria. Table 1 summarizes their designs and main findings. As can be seen, studies differed in their approach (testing prevalence versus testing deceptive ability), in the type of faking that was measured (faking bad versus faking good), the type of sample that was studied (analogue samples versus forensic patients or injury claimants), and the potential incentives that might have been involved. For forensic inmates, incentives related to evaluations of competency to stand trial and criminal responsibility, whereas for injury and compensation claimants, incentives related to psychiatric evaluations requested by insurance carriers. Importantly, not all studies specified incentives and several studies, mostly in the domain of deceptive ability research, were carried out using instructed simulation paradigms that generally do not involve any incentives.

### 3.1. Prevalence testing

A total of nine eligible studies examined the relationship between faking and psychopathy ( $n = 5$ ) or ASPD ( $n = 4$ ). Three studies found some support for the idea that psychopathy is related to a higher probability of faking (Cima & van Oorsouw, 2013; Heinze & Vess, 2005; Kucharski, Duncan, Egan, & Falkenbach, 2006) and two studies did so for ASPD (Delain, Stafford, & Ben-Porath, 2003; Grillo, Brown, Hilsabeck, Price, & Lees-Haley, 1994). For example, Kucharski et al. (2006) examined faking bad and psychopathy among 188 male criminal defendants. Faking bad was assessed with multiple measures, including the Minnesota Multiphasic Personality Inventory-II (MMPI-II; Butcher, Dalstrom, Graham, Tellegen, & Kraemmer, 1989), the Psychological Assessment Inventory (PAI; Morey, 1996), and the Structured Interview of Reported

Symptoms (SIRS; Rogers, Gillis, Dickens, & Bagby, 1991). The Psychopathy Checklist-Revised (PCL-R; Hare, 2003) was employed as an index of psychopathic traits. PCL-R total scores were divided into low (<20), moderate (20–29), and high (>29) psychopathy. Furthermore, both PCL-R Factor 1, which covers interpersonal and affective traits (e.g., manipulation and callousness), and PCL-R Factor 2, which reflects the antisocial component of psychopathy and closely resembles ASPD (Hare, 2003), were taken into account. Relative to the other groups, individuals in the high PCL-R group had raised scores on the fake bad validity scales of the MMPI-II, the PAI Negative Impression-scale, and the SIRS. Importantly, the authors found that Factor 1, rather than Factor 2, best predicted faking bad. These results lend some support to the idea that due to their manipulative traits (Factor 1), psychopathic individuals are more likely to engage in faking bad. However, the authors also noted that many psychopathic individuals in their study did not exhibit any signs of faking (see also Heinze & Vess, 2005), indicating that using psychopathy as a proxy for faking bad would produce many false positives.

Cima and van Oorsouw (2013) examined the relationship between psychopathy and faking bad in a sample of 31 prison inmates. The Psychopathic Personality Inventory (PPI; Lilienfeld & Andrews, 1996) was employed to assess psychopathy and the SIMS was administered to detect faking bad. In contrast to Kucharski et al.'s (2006) findings, these authors observed that faking bad was significantly related to PPI Factor 2 (impulsive antisociality/selfishness), but not to PPI Factor 1 (fearless dominance). This demonstrates that even studies that do find a link between psychopathy and faking bad are far from consistent when it comes to the dimensions that underlie this link: While some studies conclude that such a link is predominantly carried by trait-based dimensions (e.g., manipulative tendencies), others suggest that the behavioral dimension (i.e., norm violation) is the primary driver.

Not all studies have replicated the link between psychopathy or ASPD and faking. For example, relying on a sample of forensic patients in a maximum-security setting, Pierson, Rosenfeld, Green, and Belfi (2011) administered the Structured Clinical Interview for DSM-Axis II Disorders (SCID-II; First, Gibbon, Spitzer, Williams, & Benjamin, 1997) to assess the presence of ASPD, and the SIRS (Rogers et al., 1991) to establish the presence of faking bad. Of the 71 patients, 28 met the criteria for ASPD. The ASPD and non-ASPD groups did not differ with respect to their SIRS scores. As a matter of fact, many individuals diagnosed with ASPD seemed to be genuine in their symptom presentation. This shows that the presence of a full-blown ASPD diagnosis does not necessarily go hand in hand with faking bad tendencies. Sumanti, Boone, Savodnik, and Gorsuch (2006) obtained similar results in a sample of compensation seeking individuals.

Cima, van Bergen, and Kremer (2008) examined psychopathy and faking good in healthy controls ( $n = 115$ ) and forensic patients ( $n = 32$ ). Participants completed the PPI (Lilienfeld & Andrews, 1996) as an index of psychopathy and the Supernormality Scale-Revised (SS-R; Cima et al., 2003) as an index of faking good. The authors found that higher psychopathy scores were related to less faking good on the SS-R. However, Freeman and Samson (2012) administered the Balanced Inventory of Desirable Responding (BIDR; Paulhus, 1991) and the Self-Report Psychopathy Scale-III (SRP-III; Paulhus, Hemphill, & Hare, 2012) to a sample of 300 non-incarcerated community members and failed to obtain a significant association between the Impression Management (IM) subscale of the BIDR – which can be regarded as an index of faking good – and psychopathy.

Considering the studies summarized in the upper part of Table 1, it is difficult to escape the conclusion that the criminological model of faking bad is unable to accommodate the complexities reported in the extant empirical literature.

### 3.2. Deceptive ability testing

Some authors have argued that antisocial or psychopathic individuals are good liars because they do not feel guilty when lying (Porter,

ten Brinke, & Wallace, 2012). Following this line of reasoning one would expect that these individuals are superior in faking symptoms or in falsely denying their absence. However, studies that examined deceptive abilities in individuals with psychopathy ( $n = 5$ ) or ASPD ( $n = 1$ ) are consistent in their null findings (see lower part Table 1). That is, the majority of studies failed to find any support for the clinical lore that psychopathic individuals are superior fakers who are versed in evading detection (Boone et al., 1995; Edens, Buffington, & Tomicic, 2000; Marion et al., 2012; Poythress, Edens, & Watkins, 2001). Neither were antisocial traits found to be related to successfully passing a fake bad test (Boone et al., 1995). The few studies that did find indications for psychopaths' superior deception capacities came up with weak and only partially confirming results (Book, Holden, Starzyk, Wasylkiw, & Edwards, 2006; MacNeil & Holden, 2006).

An illustrative study is provided by Poythress et al. (2001), who examined deceptive ability and psychopathy in a mixed sample. Malingers recruited from the general population were labeled as General Population Malingers (GM;  $n = 29$ ). Participants recruited from a forensic mental health unit, who had been determined to be malingers by staff psychiatrists using SIRS items, were labeled as Clinical Malingers (CM;  $n = 26$ ). Both groups completed the PPI under standard instructions, meaning that they were instructed to answer honestly. Next, the SIRS, PAI, and SIMS were administered. The GM group was asked to provide answers that would lead experts to assume that genuine complaints were presented. The CM group received a standard instruction (i.e., honest reporting) prior to completion of the measures, but was also informed that some of their test scores would be accessible for the mental health unit staff. This was done to provide the forensic subsample with a motive to engage in faking bad. For the aggregated sample ( $N = 55$ ), no associations were evident between PPI scores and dichotomous pass-fail scores on the faking indexes. Similar null results have been reported by other researchers using student and injury claimant samples (e.g., Boone et al., 1995; Marion et al., 2012).

Another study that investigated the association between psychopathy and deceptive abilities was conducted by Book et al. (2006). In a student sample, participants were instructed to fake good or bad on the Holden Psychological Screening Inventory (HPSI; Holden, 1996). Depending on their score, the authors classified participants into two groups for faking good: With a score above the cut-off of 20, participants were classified as 'not caught faking', while a score below 20 was taken as proof of faking good, which would lead to a classification of 'caught faking'. A similar approach was followed for the faking bad condition: A score below the cut-off score of 80 was labeled as 'not caught faking', whereas a score above 80 was labeled as 'caught faking'. Psychopathy was assessed using the Levenson Self-Report Psychopathy scale (LSRP; Levenson, Kiehl, & Fitzpatrick, 1995). Compared to participants who evaded detection of faking good, individuals caught faking good were characterized by lower scores on the psychopathy measures. However, for faking bad, no group differences were found between successful and unsuccessful fakers. Using the PPI rather than the LSRP as a measure of psychopathy, MacNeil and Holden (2006) conducted a similar study and by and large, replicated this pattern. That is, a link between psychopathy and successful faking was found for faking good, but not for faking bad. Yet, the positive findings were only apparent for some of the PPI subscales (i.e., Machiavellian Egocentricity and Blame Externalization). To sum up, the idea that psychopathy or ASPD is related to a superior capacity to evade detection does not have a strong empirical underpinning.

### 3.3. Conceptual issues

Why is the empirical literature on psychopathy, ASPD and faking inconsistent? We believe that this domain is plagued by conceptual and methodological problems. Consider studies that did not find a raised prevalence of faking bad in psychopathy or ASPD (e.g., Pierson et al.,

2011; Table 1). One could maintain that such null results simply reflect antisocial individuals' ability to fake and yet to avoid detection. On the other hand, when studies on deceptive abilities find that antisocial individuals are *not* superior in evading detection, authors may conclude that these individuals simply did not bother enough about being caught, and that if stakes would have been higher, they would have displayed excellent faking abilities (i.e., undetected faking). Our point is that the meaning of passing a fake bad or fake good measure is ambiguous. It may imply that the person is not engaging in faking bad or good (i.e., true negatives), but it may also indicate that the person is well versed in escaping detection while faking (i.e., false negatives). It is because of this ambiguous information that the key assumption of the criminological model, namely that there is an intrinsic link between antisocial features and faking, is difficult to falsify.

Another reason for the lack of empirical consistency is that many studies presented in Table 1 relied on a cross-sectional methodology. This type of study would only detect a correlation between faking and antisocial features 1) if faking bad were to have trait-like properties, and 2) if antisocial individuals were to possess higher levels of these traits. There are two problems with this line of reasoning. First, individuals do not engage in faking bad all the time. Faking bad is a contextual phenomenon, as was shown by Rogers et al. (2002), who instructed juvenile offenders to play a socially desirable or a nonconformist role and then administered psychopathy measures. The socially desirable role decreased self-reported psychopathic trait scores (both Factor 1 and 2), while the nonconformist role increased these scores. Rogers et al.'s (2002) study illustrates an important point: The criminological model is preoccupied with how antisocial features impact faking tendencies, but the reversed causal chain – context dependent roles that affect measures of antisocial features (psychopathy and ASPD) – is as much, and perhaps even more interesting.

Second, as pointed out by Rogers (1990) and Berry and Nelson (2010) the criminological model of faking bad fosters a highly selective use of detection tools. That is, tools may be overemployed when psychopathy or ASPD features are present and underemployed in their absence. Such practice may introduce confirmation bias. Thus, a strong emphasis on the trait-like properties of faking ignores the situational specificity of faking, and in doing so may promote an increase in both false positives (i.e., those with psychopathy/ASPD erroneously assumed to be faking) and false negatives (i.e., those without psychopathy/ASPD erroneously assumed *not* to be faking).

#### 4. Empirical intermezzo 1: detection of faking in a forensic sample

The literature summarized in Table 1 makes clear that, inspired by the criminological model, empirical studies have been preoccupied with antisocial features and their link with faking bad, while mostly disregarding another dimension of faking, namely faking good. Faking bad and faking good are not mutually exclusive categories. For example, during the pre-trial phase, defendants may fake psychiatric symptoms and cognitive deficits in an attempt to reduce their criminal responsibility. Yet, once convicted, these same individuals may engage in faking good so as to acquire privileges, including parole (e.g., Cima et al., 2003). As another example, plaintiffs involved in a civil compensation procedure may feign certain symptoms (e.g., post-traumatic stress symptoms), but at the same time emphasize their virtues (i.e., faking good) to impress as a decent and reasonable person in the eyes of judicial decision makers (e.g., Merckelbach, Smeets, & Jelicic, 2007).

We conducted an exploratory study on faking bad and faking good in a sample of 84 male criminal offenders from six maximum security forensic institutions and one prison, all located in the Netherlands (for a more detailed description of the sample, see Nentjes, Bernstein, Arntz, van Breukelen, & Slaats, 2015). The study was approved by the standing ethical committee of the Faculty of Psychology and Neuroscience of Maastricht University. Written informed consent was obtained from all participants. Based on the literature summarized earlier, we

expected at most only modest associations of faking with psychopathy or ASPD. We also anticipated more faking in prisons than in forensic hospitals because external incentives are more prominent in the first than in the latter (McDermott et al., 2013).

Of the offenders, 83% were diagnosed with ASPD using the Structured Interview for DSM-IV Personality Disorders (SIDP-IV; Pfohl, Blum, & Zimmerman, 1995). Using cut-offs of 25 and 30 on the PCL-R (Cooke & Michie, 1999; Hare, 2003), 51% ( $n = 43$ ) and 24% ( $n = 20$ ), respectively, qualified for a diagnosis of psychopathy. We had offenders fill out the following three measures:

- The Paulhus (1991) Balanced Inventory of Desirable Responding (BIDR) with its two subscales of Self-Deceptive Enhancement (SDE) and Impression Management (IM). Both subscales measure exaggeration of positive qualities, with the SDE scale being more geared towards denial of psychologically threatening thoughts and the IM scale being more sensitive to intentional overreporting of positive behavior.
- The Supernormality Scale-Revised (SS-R; Cima et al., 2008) that intends to measure the tendency to deny common symptoms (e.g., intrusive thoughts). Like the SDE and IM, it is a measure of faking good, albeit in another domain (i.e., denial of common psychological symptoms).
- The SIMS (Smith & Burger, 1997) that measures overreporting (i.e., faking bad) of rare and bizarre symptoms.

Pearson product-moment correlations between faking indices and PCL-R scores are displayed in Table 2. As can be seen, psychopathy was negatively associated with faking good as measured by the IM subscale of the BIDR (see also Freeman & Samson, 2012), yet showed a positive association with the tendency to fake bad. Significant correlations between faking and psychopathy were carried by PCL-R Factor 2 (antisocial behavior), which is in line with Cima and van Oorsouw (2013). In contrast to what one might expect (see also, Kucharski et al., 2006), PCL-R Factor 1 (interpersonal/affective traits) was not associated with any of the faking measures. The relationship between Factor 2 and faking good was not apparent for the SDE scale, whereas it was only marginally significant for the SS-R. Most importantly, the effect sizes associated with the significant relationships between Factor 2 and faking remained small, with the proportions of variance explained ( $r^2$ ) being as low as 7% and 8% for faking good and bad, respectively. When Bonferroni corrections were applied, the association between the SIMS and PCL-R Factor 2 attained significance, while the association between the IM subscale and PCL-R Factor 2 reached borderline significance (two-tailed  $p = .014$ ).

We supplemented our correlation analyses with a categorical approach to the data. Employing the standard cut-offs for the PCL-R,

**Table 2**

Correlations, means, and standard deviations for scores on faking good (SDE, IM, SS-R) and faking bad (SIMS).<sup>a</sup>

	PCL-R total	PCL-R Factor 1	PCL-R Factor 2	<i>M</i>	<i>SD</i>
SDE	-.16	-.08	-.14	87.9	11.5
IM	-.25*	-.04	-.27*	74.7	18.1
SS-R total <sup>b</sup>	.18	.07	.19	55.9	8.9
SIMS total <sup>c</sup>	.29**	.06	.28**	7.8	5.6

Notes. PCL-R = Psychopathy Checklist-Revised; Factor 1 = Interpersonal/affective traits; Factor 2 = Antisocial behavior/deviant lifestyle; SS-R = Supernormality Scale-Revised; SIMS = Structured Inventory of Malingering Symptomatology; SDE = Self-Deceptive Enhancement; IM = Impression Management.

\* $p < .05$ . \*\* $p < .01$ , two-tailed.

<sup>a</sup> Correlation coefficients did not appreciably change direction or size after partialling out the effects of participants' age and IQ, or after controlling for the shared variance between PCL-R Factor 1 and 2 using a regression approach.

<sup>b</sup> High scores on the SS-R indicate low levels of faking good.

<sup>c</sup>  $n = 82$ ; SIMS total scores were log-transformed to reduce positive skewness.

SS-R, and SIMS (25, 60, and 16, respectively), we found that 7% of the non-psychopathic inmates ( $n = 3/41$ ) and 5% of the psychopathic inmates ( $n = 2/43$ ) exhibited faking good, whereas 3% of the non-psychopathic inmates ( $n = 1/40$ ) versus 12% of the psychopathic inmates ( $n = 5/42$ ) exhibited faking bad. These group differences did not reach significance (Fisher's exact tests:  $p$ 's  $> .10$ ).

To examine situational specificity, we compared prisoners and forensic patients with regard to faking. The two groups did not differ in average PCL-R scores,  $t(83) = -1.28$ ,  $p = .20$ . Employing the standard cut-offs of the SIMS and the SS-R, 4% of the forensic patients ( $n = 3/70$ ) versus 25% of the prisoners ( $n = 3/12$ ) engaged in faking bad, while faking good was displayed by  $<1\%$  of the patients ( $n = 1/72$ ) versus 25% of the prisoners ( $n = 3/12$ ). Here, group differences did reach borderline significance taking Bonferroni corrections into account (two-tailed Fisher's exact  $p$  for faking bad:  $= .04$ ; two-tailed Fisher's exact  $p$  for faking good:  $= .02$ ).

The majority of the offenders in our sample fulfilled the diagnostic criteria for ASPD, yet did not display faking (either good or bad). In addition, psychopathic offenders did not fake more than their non-psychopathic counterparts. Thus, in keeping with a number of other studies listed in Table 1 (e.g., Cima et al., 2008; Pierson et al., 2011), our data indicate that the tendency to fake is not sufficiently explained by constructs like psychopathy or ASPD. Furthermore, our data illustrate that in forensic participants, faking good might be as common as faking bad. They also demonstrate that context makes a difference, in that faking seems to be more common in a prison setting than in a forensic psychiatric setting (see also McDermott et al., 2013). Thus, the criminological model's trait-like view of faking should be replaced by a more context-based and motivational approach that also takes into account faking good (see for an elaborated discussion of this point, Rogers, 1990).

## 5. The consequences of faking bad

The previous sections focused on the detection of faking and its prevalence among those with antisocial features. A more fundamental, yet largely ignored issue is whether faking bad has different consequences in individuals with and without such features. The criminological model assumes that there are strict demarcation lines between faking bad and genuine somatoform symptoms. The idea is that faking bad is under intentional control, while somatoform complaints result from the unconscious production of symptoms. There are, however, reasons to question this distinction. For example, simulation research in our lab (Merckelbach, Dandachi-FitzGerald, van Mulken, Ponds, & Niesten, 2013; Merckelbach, Jelicic, & Pieters, 2011) suggests that faking bad produces residual symptoms. Undergraduates were provided with a forensic scenario and then instructed either to fake bad or to respond honestly while completing self-reports of symptoms. After approximately an hour, the self-report scales were administered again, with the instruction that all participants should now answer honestly. At follow-up, participants who had initially engaged in faking maintained elevated symptom levels compared to control participants. Not only laboratory findings, but also clinical data (summarized in Merckelbach & Merten, 2012) indicate that faking bad can result in vague symptoms that the person may come to experience as real. This indicates that faking is more than simply a complication during diagnostic routines: It represents a phenomenon with psychopathological potential.

Merckelbach and Merten (2012) and Bayer (1985) have argued that faking bad produces cognitive dissonance because people generally find the inconsistency between their faking behavior and their moral standards aversive. They typically resolve this dissonance by convincing themselves that, to some extent, they do actually suffer from the symptoms that they had initially only faked. Rodriguez and Strange (2014) have recently found that dissonance-inducing events, such as writing a counter-attitudinal essay, can lead to attitude change

accompanied by memory distortions for the initial attitude. Similarly, with respect to faking bad, dissonance may create self-deceptive effects that amount to the belief that one does have genuine symptoms.

The cognitive dissonance framework has an intriguing implication. Specifically, residual effects of faking bad are only to be expected when an individual experiences cognitive dissonance in the first place. Whereas many individuals may at least feel some conflict after engaging in morally unacceptable behavior such as faking bad, this may not apply to those with antisocial or psychopathic features. In fact, there are clear indications that these individuals are rather insensitive to cognitive dissonance. For example, Murray, Wood, and Lilienfeld (2012) instructed undergraduates to deceive fellow students into believing a task was enjoyable when in reality it was not. Those with low psychopathy scores, as measured by the LSRP, were sensitive to this cognitive dissonance induction, whereas those with high psychopathy scores were not. Thus, it is reasonable to assume that, relative to controls, individuals with psychopathic or antisocial features feel less dissonance when they engage in faking bad. In this way, psychopathy may immunize against the residual effects of faking.

## 6. Empirical intermezzo 2: faking bad, antisocial features, and dissonance

We explored these predictions in a preliminary study that was approved by the standing ethical committee of the Faculty of Psychology and Neuroscience of Maastricht University. Written informed consent was obtained prior to participation. In this study, sixty students (22 men) indicated on a 100 mm visual analogue scale (VAS) to what extent they experienced somatic complaints at the moment of testing. Next, they were asked to write a brief sick note to their professor, in which they fabricated that they were ill and could therefore not attend classes. Several authors have suggested that perceived free choice is an important condition for dissonance to occur (e.g., Brehm & Cohen, 1962). We therefore made students aware of their freedom to choose whether or not to write the sick note. All students decided to write the note. Following this, participants indicated on a single 100 mm VAS how unpleasant (i.e., dissonant; 0 = not unpleasant at all; 100 = very unpleasant) it was to write the note. Dissonance is characterized by an unpleasant feeling state that dissolves as soon as reduction strategies (e.g., internalization of symptoms) are successfully applied (Festinger, 1957). Thus, it is important to capture dissonance during or soon after its activation. With this consideration in mind, we relied on a single item that was administered immediately after the manipulation. Analyses revealed that writing the note about faked illness resulted in unpleasantness ratings that deviated significantly from zero (not unpleasant at all),  $t(58) = 6.82$ ,  $p < .01$ , Cohen's  $d = 1.79$ . Next, participants once again indicated on a 100 mm VAS to what extent they experienced somatic complaints. At a later point in time, participants were administered the LSRP as an index of psychopathic traits. As predicted, higher dissonance levels were moderately associated with stronger residual symptom effects ( $r = .37$ ,  $p < .01$ ; two-tailed). In total, 40 participants completed the LSRP during a post-test. The correlation between dissonance and the LSRP total score in this group was  $r = -.32$  ( $p = .04$ ; two-tailed), indicating that higher psychopathic trait scores were, indeed, accompanied by lower dissonance levels. The correlations between psychopathy scores and residual symptoms remained non-significant ( $r = .02$ ). Thus, the data are consistent with the interpretation that high psychopathy scores may moderate, and therefore obscure, the link between dissonance and residual symptoms.

One could argue that our method of data collection was relatively transparent and may have induced participants to engage in hypothesis affirming behavior. Future studies may control for this potential source of confounding by using more sophisticated versions of this paradigm

(e.g., with a stronger cover story). Given that the paradigm employed has not been used before and needs optimization, our findings should be perceived as a tentative illustration of how researchers may study the *consequences* of faking, a topic that has been largely ignored by the criminological model.

Along with other studies (Merckelbach et al., 2011, 2013), our data show that faking induces cognitive dissonance, which in turn is known to foster attitude change. As Murray et al. (2012) noted, the absence of dissonance might explain the often reported lack of therapeutic progress in individuals with psychopathy. Thus, one could speculate that a lack of treatment effects among psychopaths reflects their failure to internalize faking good. A more extensive discussion on this topic can be found in Maruna and Mann (2006), who refer to literature showing that offenders who engage in excuse making for their crimes (i.e., a form of faking good and a means to reduce dissonance) at least show social awareness and have lower recidivism rates than those who do not engage in excuse making. Thus, dissonance theory provides a valuable framework to understand the consequences of faking.

## 7. Concluding remarks

The criminological model of faking bad suggests that ASPD or psychopathy is a red flag for faking bad. Our review of the empirical literature, however, makes clear that this view is too preoccupied with one form of faking, has a weak empirical basis, and is plagued by conceptual problems. In their thought-provoking review, Berg et al. (2013) recently listed misconceptions about psychopathy, such as the idea that therapy makes psychopaths worse. The authors could also have listed the misconception that psychopathy and ASPD are intimately linked with faking bad.

As said before, this misconception is not without consequences for clinical practice. It suggests that clinicians should preferably administer detection instruments in a forensic context, when in fact faking tendencies may occur wherever there are incentives. For example, Van Egmond and Kummeling (2002) interviewed a mixed group of psychiatric outpatients about their “hidden agendas”, a term that refers to the potential incentives (e.g., disability compensation, study privileges, stimulant medication) that individuals attribute to the patient status and of which their therapists are often not aware. The authors noted that 42% of the patients admitted to have such a hidden agenda. Van Egmond and Kummeling also observed that the treatment outcome for this group was worse than for patients without a hidden agenda. It would be an over-interpretation to argue that the patients with a hidden agenda all engaged in faking bad. However, it is safe to conclude that even outside the forensic domain, the potential for faking bad in patient samples is more sizeable than some clinicians may assume it to be on the basis of their understanding of the criminological model. As a further example, Dandachi-FitzGerald et al. (2011) administered two faking bad measures to a large group of psychiatric outpatients and found that 34% of them failed on one or both tests. Failing a faking bad test was related to inflated symptom reporting on standard clinical instruments. It is unlikely that these 34% were all patients with comorbid psychopathy or ASPD. A more sensible framework would be one that recognizes that there might be many circumstances and conditions in which patients use a response style that defeats a conventional checklist approach. Research examining the extent to which clinicians endorse the criminological model of faking bad and its implications, as well as their level of confidence in the link between faking bad and antisocial features, is needed to provide further insight in the degree to which this model interferes with clinical practice.

Psychopathy and ASPD are dimensional constructs (Hare, 2003). Likewise, faking is not an all-or-none phenomenon, but comprises several dimensions (e.g., denying symptoms, over-reporting desirable behavior, underperforming on cognitive tests, over-reporting rare symptoms). These dimensional aspects do not fit well with the categorical approach of the criminological model. Yet, recognizing the

dimensional nature of the key constructs provides an important starting point for future studies exploring the correlates of psychopathy and ASPD. For example, Young-Lundquist, Boccaccini, and Simpler (2012) examined how psychopathy relates to self-reported adaptive functioning in a forensic sample. The authors observed that PPI Factor 2 (impulsive antisociality/selfishness) predicted poor adaptive functioning, which makes sense if one assumes that antisocial behavior interferes with the ability to live a normal life. However, when the authors used a faking bad index as a covariate, the potential of PPI Factor 2 to account for poor adaptive functioning became less obvious. It is this type of approach that is informative because it allows for studying the correlates of psychopathy and ASPD (e.g., impaired everyday functioning) in a way that is not confounded by faking. Accurate information on such correlates is relevant for forensic practice because it allows for a more (cost-)efficient allocation of therapeutic resources.

After we had carried out our qualitative review of the literature on psychopathy and ASPD and faking, we became aware of the meta-analysis of Ray et al. (2013). Unlike our review that only included studies with dedicated and stand-alone measures of faking, their meta-analysis focused on embedded faking measures such as the response validity scales of the MMPI, the PPI, and the PAI. There was no overlap between the studies listed in our Table 1 and the 45 studies reviewed by Ray et al. (2013). Nevertheless, their overall conclusion parallels our results in that these authors found no convincing association between psychopathy and faking good, while a medium association was found between faking bad and the behavioral component (95% CI of weighted mean effect size [.23–.40]), but not the personality component (CI [.00–.14]) of psychopathy. The authors argued that this is good news, because it shows that self-report psychopathy measures are not necessarily compromised by faking good. Some caution is advised here: although the link between antisocial features and faking might, indeed, be small, an alternative explanation is that inventories and interviews that assess antisocial features are biased due to patients' minimization of such features. Psychopaths' tendency to minimize is exemplified by their exaggeration of the reactive elements in their crimes (Porter & Woodworth, 2007). If this type of response bias also occurs on psychopathy and ASPD measures, it may obscure true correlations with faking. Further research into the robustness of instruments assessing ASPD or psychopathy against response biases is therefore needed.

Faking bad may induce dissonance that fosters internalization of symptoms. Yet, individuals high in psychopathy are less sensitive to dissonance. Although our findings are at this stage preliminary, dissonance theory may prove a fruitful framework for unraveling mechanisms underlying both faking bad and good. It remains to be seen whether faking good induces dissonance and, in doing so, produces its own residual effects (i.e., residuals of desirable behavior). Such findings would be valuable for the articulation of innovative therapeutic strategies that focus on the benefits rather than the disadvantages of faking good.

As Berry and Nelson noted in their review article: “At a fundamental level, the categorical DSM criteria do not map on to the available objective data on the nature of the phenomenon” (2010; p. 296). It is this disparity between the criminological model of faking bad and the empirical literature on faking that needs to be resolved so that more fundamental issues can be addressed. The corpus of clinical knowledge would benefit from systematic research on such issues, rather than from studies that follow the narrow-minded view that antisocial features and faking bad are uniquely related.

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