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ORIGINAL PAPER

A Taxometric Analysis of Problem Gambling Data from a South African National Urban Sample

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Abstract We investigate the question whether problem gambling (PG) in a recent South African sample, as measured by the Problem Gambling Severity Index (PGSI), is dimensional or categorical. We use two taxometric procedures, Mean Above Minus Below A Cut (MAMBAC) and Maxim Covariance (MAXCOV), to investigate the taxonic structure of PG as constructed by the PGSI. Data are from the 2010 South African National Urban Prevalence Study of Gambling Behavior. A representative sample of the urban adult population in South Africa (N = 3,000). Responses are to the 9 item PGSI. MAMBAC provided positive but modest evidence that PG as measured by the PGSI was taxonic. MAXCOV pointed more strongly to the same conclusion. These analyses also provide evidence that a PGSI cutoff score of 10 rather than the standard 8 may be called for. PG as constructed by the PGSI may best be thought of as categorical, but further studies with more theory based measurements are needed to determine whether this holds in a wider range of samples and for other screens. A higher cutoff score may be called for on the PGSI when it is used for research purposes to avoid false positives.

Keywords Taxometrics · Canadian Problem Gambling Index · Problem Gambling Severity Index · Categorical versus dimensional · Prevalence

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Introduction

The current deliberations leading up to the forthcoming publication of DSM-V have called into question the general presumption in DSM-IV that mental and substance abuse disorders are categorical (Widiger and Clark 2000; Helzer et al. 2006; Muthén 2006; Saunders and Schukitt 2006; Helzer et al. 2007). Reasons for this include recurrent observations of high rates of co-occurrence of disorders and high frequencies of non-specific diagnoses. Problem gambling (PG) is not classified with substance abuse disorders in DSM-IV and has not featured in the debates cited above. However, the current working group proposal for DSM-V would group *pathological* gambling with substance abuse among a reintroduced category of 'addictions', reflecting substantial convergent evidence that such gambling shares core neurobiological, behavioral, and cognitive characteristics with the forms of substance abuse traditionally associated with addiction (Ross et al. 2008). Thus there are grounds for interest in the question of whether PG is best assessed on a presumption of a dimensional or a categorical latent structure.

The difference between categorical or dimensional views of disorders turns on whether there is a qualitative difference between disordered behaviors and other behaviors. On a dimensional view disordered behaviors are distributed across the population in an approximately continuous fashion in terms of severity. On this view gambling frequency, incidence of loss of control, extent of gambling related problems, and so on, would constitute a spectrum with different individuals ranking from to low to high on these attributes. On a categorical view disorders constitute distinct entities with characteristic causes, traits, and outcomes. Medical models of psychopathology are generally categorical.

There are multiple reasons for wanting to know whether PG is best conceived of as dimensional or categorical. In the clinical context, presumptions of categorical structure where dimensional conceptions are closer to reality may lead to over-emphasis on shifting patients' phenotypic status from positive to negative diagnoses, rather than reducing severity of continuous symptoms (Slade et al. 2009). Knowing whether PG is best understood as a dimension or as a category is important for several other reasons. If it is dimensional, then treating it as categorical sacrifices information and reduces statistical power (Blaszczynski and Nower 2002) and studies of its causes and course would benefit from using the full range of scores on valid gambling screens. Similarly, treating a categorical disorder as continuous also reduces statistical power (Ruscio et al. 2006). If there really is a distinct group of gamblers who have serious addictions, then lumping them together with those individuals who have various transitory and diverse consequential problems associated with gambling is likely to make it harder to understand the causes and the treatment needs of those who may suffer from continuous preoccupation with gambling as an overwhelmingly dominant source of reward. Additionally, if there is support for a categorical classification, it would be natural to ask how well the current scoring protocol of various screens identify category members.

The aim of the current paper is to provide a taxometric analysis (Meehl 1992, 1995, 2004; Meehl and Yonce 1994; Meehl and Yonce 1996; Ruscio et al. 2006) of the Problem Gambling Severity Index (PGSI), the scored module of the Canadian Problem Gambling Index (CPGI) (Ferris and Wynne 2001), in response to data from the South African National Urban Prevalence Study of Gambling Behaviour (NUPSGB ref deleted). In light of the motivations above, we investigate whether these data are most consistent with a categorical or a dimensional representation of PG (Table 1).

Though by no means without its critics, the PGSI has largely become the "screen of choice" for general population studies (Holtgraves 2009). For example, all longitudinal

Table 1 Problem Gambling Severity Index (PGSI) questions

Question	Response category			
	Never	Sometimes	Most of the time	Almost always
Thinking about the last 12 months…				
1. How often have you bet more than you could afford to lose?	0	1	2	3
2. How often have you needed to gamble with larger amounts of money to get the same feeling of excitement?	0	1	2	3
3. How often have you gone back another day to try to win back the money you lost?	0	1	2	3
4. How often have you borrowed money or sold anything to get money to gamble?	0	1	2	3
5. How often have you felt that you might have a problem with gambling?	0	1	2	3
6. How often have people criticized your betting or told you that you had a gambling problem, regardless of whether or not you thought it was true?	0	1	2	3
7. How often have you felt guilty about gambling or what happens when you gamble?	0	1	2	3
8. How often has your gambling caused you any health problems, including stress or anxiety?	0	1	2	3
9. How often has gambling caused any financial problems for you or your household?	0	1	2	3

studies of PG conducted to date have used it, and this is not true of any other screen. A major reason for the PGSI's popularity is its articulation in terms of several thresholds ("No risk/Low risk/Moderate risk/High risk for PG"), crossings of which can be used as a main dependent variable in a longitudinal analysis. Several studies report good psychometric properties for it (Ferris and Wynne 2001; Maitland and Adams 2007; Orford et al. 2010; Sharp et al. 2012). In light of this, and for the sake of inter-study comparability, it was adopted for use in the South African National Urban Prevalence Study of Gambling Behaviour (NUPSGB) (Ross et al. 2010). More recent studies have continued to find good reliability and validity in diverse populations. Loo et al. (2011) looked at a Chinese population and found that a unifactorial model fit the data well in exploratory and confirmatory factor analyses, correlating significantly with other screens, gambling frequency, comorbidities and other external variables.

Aside from the PGSI, widely used screens include the South Oaks Gambling Screen (SOGS) (Lesieur and Blume 1987), the Gamblers Anonymous screen (Custer and Custer 1978), and the National Opinion Research Center DSM Screen for Gambling Problems (NODS) screen based most directly on DSM-IV criteria (Hodgins 2004). The PGSI was developed for use in nonclinical settings, motivated by evidence that the SOGS has a false positive rate in general nonclinical populations that is uncomfortably high for research uses. In addition, the PGSI is intended to reflect a dimensional conception of disordered gambling that occurs along a continuum ranging from social or recreational gambling with no adverse effects, through problem gambling with adverse effects for the individual, family, friends, colleagues, and the community, and through to pathological gambling involving severe negative consequences and meeting diagnostic criteria (Neal et al. 2004). This contrasts with the DSM-IV's categorical conception of pathological gambling,

according to which pathological gamblers are separated from normality by the presence of characteristic symptoms.

Another motivation for the current study is to evaluate the PGSI itself, in two respects. First, the PGSI uses a cutoff score of eight or more to categorize PGs. However, there are worries that at this cutoff the screen overdiagnoses (Ladouceur et al. 2005). These worries might be addressed by examining the taxonic status of PG as we will detail below. Second, the PGSI incorporates an amalgam of two different views about PG (Svetieva and Walker 2008): the idea that it should be thought of as an addiction where diminished control is present, and the idea that it simply labels a cluster of harmful social and behavioral effects encountered transiently but recurrently by some individuals. The PGSI contains items derived from both conceptions and has consequently been criticized on grounds of inconsistency (Svetieva and Walker 2008). What is labeled PG may express, at the highest level of abstraction, two different but statistically associated phenomena: on the one hand an addiction that is characterized by repeated failed attempts at self-control and has an hypothesized distinctive neurological signature in a dysfunction in the dopamine reward circuit, and on the other hand a transitory condition that largely involves problems in living and should best be treated as continuous with the goal of better managed enjoyment of gambling.

Taxometric methods of the sort used here to investigate the taxonic or dimensional nature of PG have been extensively used in the study of other psychopathological conditions such as ADHD (Haslam et al. 2006), schizophrenia (Rawlings et al. 2008), depression (Solomon et al. 2006), alcohol dependence (Slade et al. 2009; Walters et al. 2010), nicotine dependence (Ginestet et al. 2008), and cannabis dependence (Denson and Earleywine 2006). The widespread use of these methods in studying psychopathology is motivated by their ability to provide persuasive tests for taxonicity or dimensionalilty compared to other methods such as factor analysis that presuppose dimensionality from the outset (Ruscio and Ruscio 2004; Reise and Waller 2009).

To our knowledge there has been only one previous application of taxometric methods to investigate disordered gambling. Braverman et al. (2011) applied taxometric analysis to online sports betting behavior data. Their results were ambiguous and did not clearly support a categorical or dimensional approach, thus motivating further research into the issue. As far as we know no commonly used gambling screen has been subject to taxometric analysis.

The taxometric methods stem from the initial work of Meehl and others (Meehl and Yonce 1994, 1996; Meehl 2004; Ruscio et al. 2006; Grove 2004; Waller and Meehl 1998) and have been implemented in a suite of functions in the R programming language (Ruscio et al. 2006). The basic idea behind the procedures is to look at sets of indicators in different subsamples of data and to search for relations between those indicators that would obtain as the subsamples approached an even split between individuals in a taxon and those in its complement. Failure to find such subsamples is then an argument for dimensionality. To give an intuitive example, if we randomly sample from a population consisting of both men and women and regress their heights on baldness measures, we expect to see slopes at their maximum in subsamples composed of equal numbers of men and women and slopes of zero in subsamples consisting in only men or women (see Fig. 1). No such difference between equally mixed and skewed samples would be seen if gender were continuously distributed.

Making a strong case that a sample contains a taxon and a complement or, alternatively, that no taxon exists, involves two steps. First, one must verify that data are suitable for taxometric analysis. Indicators of a taxon that are highly correlated will not produce

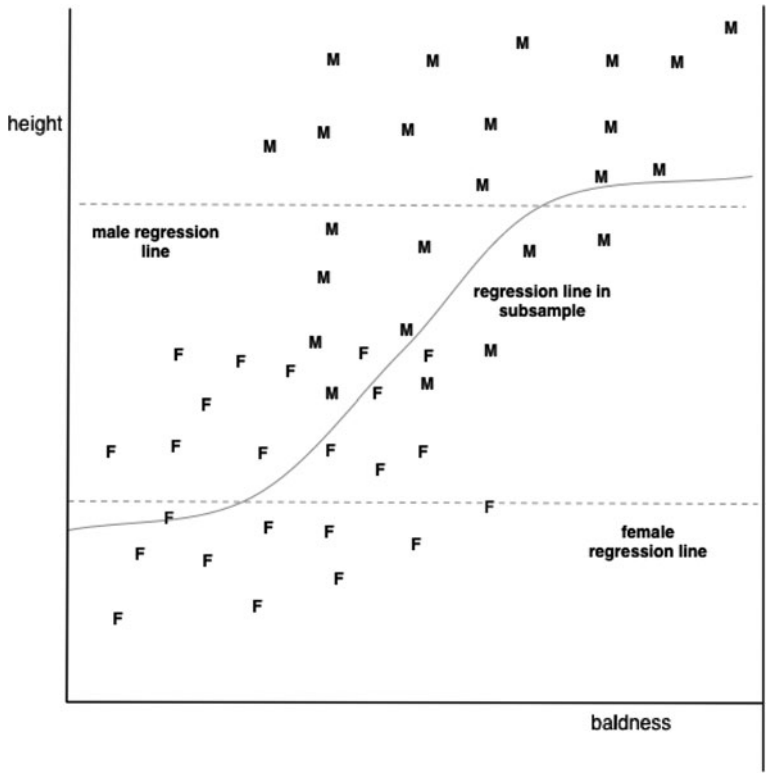

Fig. 1 The slope of the regression curve in different subsamples of the data varying from pure male and pure female samples to equally mixed samples. In the pure samples the regression lines are flat but they take on positive values in unequal samples and reach their peak in equally divided samples

reliable results under taxometric examination. Highly skewed data can produce results that falsely mimic the presence of a taxon. Studies done in populations where the expected number of taxon members—the base rate—is very small (or large) make it hard to test the dimensional versus categorical hypothesis.

The second step is accurately interpreting the results of taxometric analyses, which does not involve traditional hypothesis testing. There are three main ways of supporting an interpretation of dimensionality or taxonicity. Taxometric analyses produce graphs with input indicators on the x axis and output on the y axis, using all pairs of indicators. An exhaustive taxometric study sequentially examines all indicators in each variable role. A characteristic unimodal peaked curve signifies taxonicity and a dish shaped graph suggests dimensionality. Curves based on actual data are compared with curves based on simulated data that share key parameters of the actual data (e.g., number of indicators, skewness, validity) but in which taxonic and dimensional latent structures, respectively, are built in by the simulator. Conclusions of dimensionality or taxonicity with respect to the real data should be supported by similar curve shapes in, respectively, the dimensional or taxonic simulated data.

Another well-established method for interpreting taxometric results is a goodness of fit indicator called the Comparison Curve Fit Index (CCFI) that can compare research data to simulated dimensional and categorical comparison data to provide an objective measure of

curve similarity. The CCFI is based on the root mean squared residuals of y values. Recent research has shown that the CCFI quite reliably picks out taxons and can do so in situations where visual evidence is ambiguous. In simulations Ruscio et al. (2007) showed that the CCFI picked out taxons with a 78 % specificity and 91 % sensitivity (see also Ruscio et al. 2010). A final means of supporting a particular interpretation involves looking at the base rate estimates for a possible taxon across indicators and individual indicator pair curves. If the base rate estimates are similar across indicators, then a taxon may be suggested. However this is the weakest of the three kinds of evidence (Ruscio 2007).

The existence of taxonic groups does not preclude individuals within the groups differing along some continuous dimensions. For example, a given person either has or doesn't have a gene for Huntington's disease, but the severity of the disease varies with number of gene copies. Individuals falling into a putative taxon of pathological gamblers might likewise differ in various indicators of severity. These differences might provide further useful information over and above that given by taxon status. For example in the case of depression, Ahmed et al. (2011) recently found taxometric evidence for a depressive taxon, but then also found that severity differences within the category predicted external values such as help seeking, family history and duration.

Methods

Sample and Diagnostic Interview

The NUPSGB is based on face to face interviews, conducted in November and December 2008, with 3,000 adults, randomly drawn from the census of households, in the metropoles of Johannesburg, Tshwane, Cape Town and eThekweni (Durban), South Africa. In the sample, 43.3 % had never gambled, 35.8 % were at no risk for problem gambling, 10.33 % were at low risk for problem gambling, 7.60 % at moderate risk for problem gambling, and 3.23 % were problem gamblers as measured by the PGSI.[1]

The study methods described below were applied to a sub-sample of 619 from the original study. Those who reported having not gambled in the past year were excluded in order to avoid recall bias problems as were those individuals who had gambled in the past year but recorded no positive score on the PGSI (indicating no risk for gambling problems). Note that the methods employed here do not involve the usual sampling framework characteristic of frequentist statistics. Thus considerations of demographic representativeness are unmotivated in taxometric analysis. Our final sample contained more men than women, for example, because in general men show more gambling problems. We also note that looking for taxons in samples where the base rate of the outcome variable is low, as with PG, biases the results in favor of dimensionality, with taxons typically becoming invisible even where they exist for base rates below 10 % (Ruscio et al. 2006). Thus finding evidence of taxonic structure despite a low base rate makes for compelling evidence.

Due to the presence of "Don't Know" and "Refuse" response options in the NUPSGB questionnaire for certain variables of interest, missing data arises. The algorithms used in

[1] This rate of severe gambling problems in our population sample is higher than in studies in other countries. Prevalence of disorders related to self-control in general are thought to be abnormally high in South Africa by comparison with global prevalence estimates, most of which are derived from much wealthier countries with superior social support infrastructure. See Ellis et al. (2011) for an overview.

taxometric analysis cannot accommodate missing values. We therefore imputed values for these responses using a single well-supported univariate imputation method. The variables imputed included the amount of money spent on gambling in the last month (211 observations missing, or 12 % of the total sample), and categorical variables indicating the severity of the gambling problem (in section E of the questionnaire). For the 9 categorical variables, the number of missing observations was never greater than ten (or 0.5 % of the total sample).

Royston's (2007) Imputation by Chained Equations (ICE) algorithm implemented in Stata version 8.2 was used for this exercise. This algorithm is similar in principle to Van Buuren et al. (1999) Multiple Imputation by Chained Equations (MICE) algorithm (see Royston 2004, 2007, 2009 for discussion).[2] Due to the small fraction of missing data in the sample, we perform single rather than multiple imputations. Imputed values for the missing data are drawn from the conditional distributions of the outcome variables of interest. The estimators used in the algorithm are ordinary least squares to impute for the log of the amount of money spent on gambling and ordered logistic regression to impute for the Likert-scale categorical variables.

Taxometric Procedures

The underlying rationale for taxometric procedures is the General Covariance Mixture Theorem (Meehl 1973), which partitions total covariance between two indicators into the covariance within a taxon and the covariance within its complement, and—importantly—the covariances resulting from different group mixtures. The methods are sometimes called coherent cut kinetics because they look for predictable results as a cutoff point is varied over a distribution of scores for different mixtures of individual scores on indicators. If there is a taxon, then there should be an optimal score on an indicator that separates the taxon from the complement. Lack of such a score suggests dimensionality. The two procedures we used follow this general logic, but provide mathematically distinct algorithms. Recent empirical simulations have shown that the algorithms provide independent lines of evidence (Walters and Ruscio 2009b).

MAMBAC

The first procedure, Mean Above Minus Below A Cut (MAMBAC), considers all combinations of pairs of PSGI response variables. For each pair, one indicator is used as input variable and another indicator as an output variable. Repeated cuts are made at regular intervals along the indicator variable to divide the sample into subsets, starting with low values of the indicator variable and making cuts at increasing values of the indicator variable (Fig. 2a). For each cut, MAMBAC then calculates the mean score on the output indicator for those falling below the cut score and the mean score for those above the cut score, and the difference is plotted on the y axis against cases sorted by cutting scores on the x axis (Fig. 2b). If a taxon is present (and skew is minimal as is indicator intercorrelation in the taxon), then the greatest y output value will occur in the sub-group that is closest to a 50–50 split between taxon members and complement members. y values will be correspondingly lower as sub-groups approach mostly taxon members on one end of the distribution and mostly complement members on the other end. Presence of a taxon is then indicated by a peaked convex curve, while dimensionality produces a concave curve.

[2] Further details of the specification of the algorithm and the results obtained are available from the authors upon request.

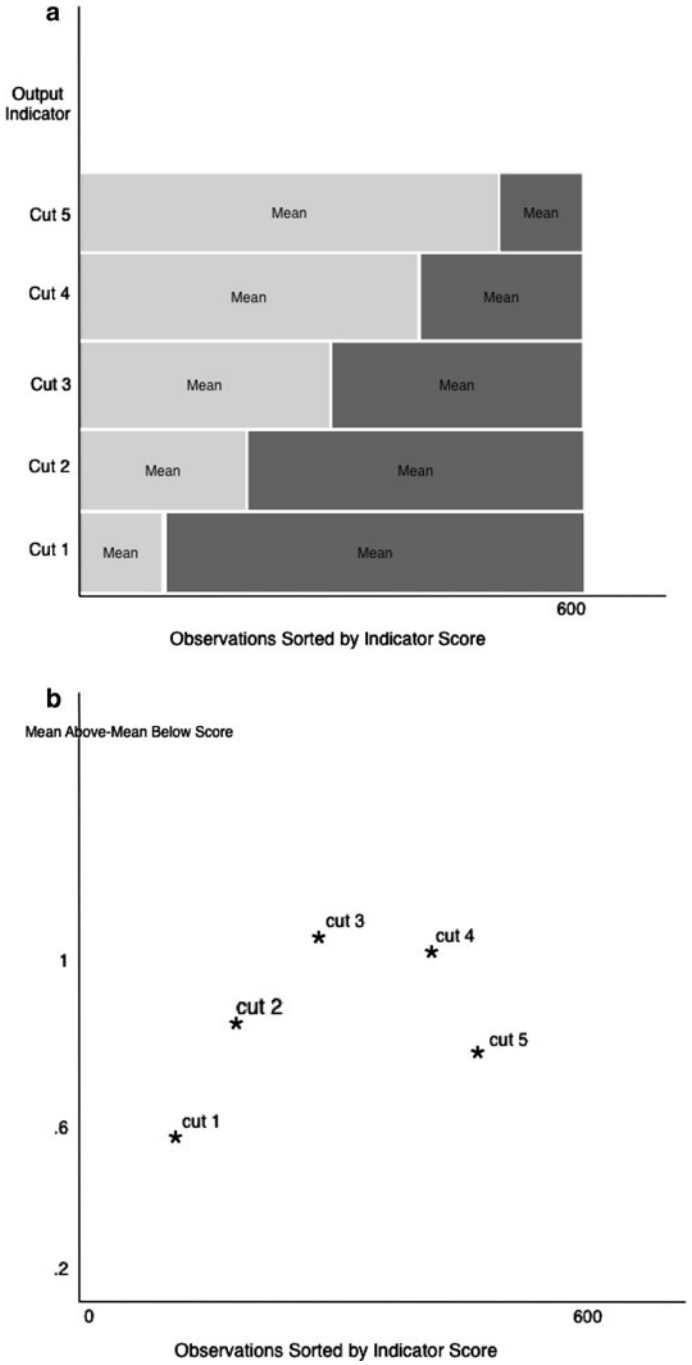

Fig. 2 **a** The algorithm first divides the total observations into two subsets of different sizes based on input indicator scores and calculates the mean value of the output value in the larger and the smaller subsets for each division. Here five cuts have been made for illustration purposes, but normally the algorithms are set to make more cuts. **b** The differences between means for each cut (five in this case) are then plotted on the y axis

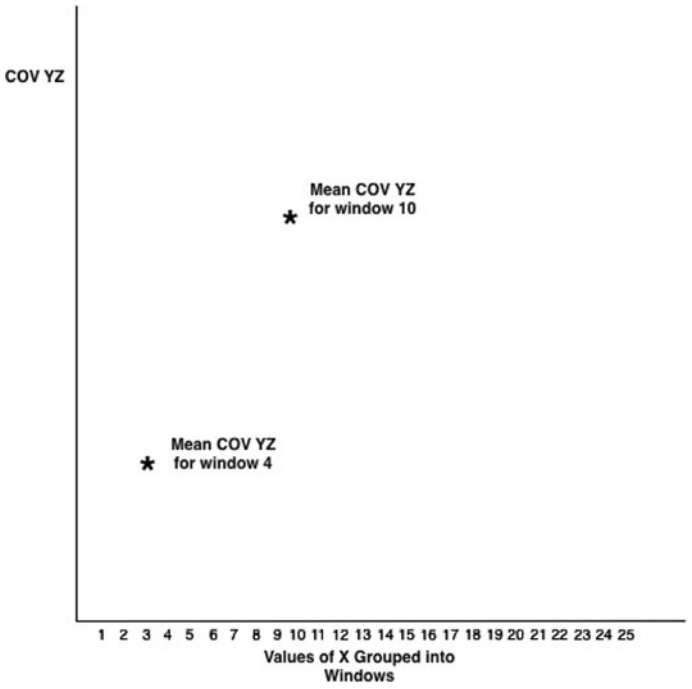

Fig. 3 MAXCOV works by arranging the indicator observations along the x axis in increasing order, dividing those cases into equally numbered subsets of windows or cuts, calculating the average covariance for the two output indicators, and plotting that value on the y axis. Here the algorithm has calculated the average covariance of the two output indicators in windows 5 and 10

This procedure is implemented using all indicators in all possible input combinations, producing a set of individual curves and an average curve. Comparisons of simulated dimensional and categorical data with the empirical characteristics of the research data are calculated by the R algorithm that implements MAMBAC. Curves were examined visually to judge whether the data most resemble the taxonic or dimensional simulation, and the CCFI index provides an objective and reliable method of testing these judgments.

MAXCOV

Maximum Covariance (MAXCOV) uses the covariance between two indicators in subsets of the values of a third indicator. The procedure sorts the sample into sub-groups along equally spaced cuts, creating "windows" along the line of indicator scores on the x axis. The average covariance of the two output indicators for the values of input indicators in the window is plotted on the y axis against sub-groups (windows) of the values of an indicator variable on the x axis (Fig. 3). As with MAMBAC, greater y scores will be observed as the sub-groups approach an equal split between taxon and complement members. If there is no taxon, then the covariance between output indicators should remain the same across sub-groups. This procedure is repeated using all possible combinations of indicators, again producing a set of individual curves and an averaged curve. Comparisons of simulated dimensional and categorical data with the actual data are calculated, and the curves

compared by visual inspection and by the CCFI generated by the R algorithm that implements MAXCOV.

Results

Indicator Validity and Sample Characteristics

The sample for analysis consisted of 619 individuals. Applying the standard PGSI cutoff score of 8 for identifying subjects at high risk for PG, 96 individuals in the sample would fall into the possible taxon if the PGSI were a perfectly reliable instrument. Thus the sample size and estimated taxon base rate meet the standard recommendation (Ruscio et al. 2006) that taxometric analysis involve a minimum sample size of 300 and minimum estimated base rate of 10 %.

Indicator correlations in the putative taxon were all well below the recommended value of 0.3 except indicators for the first two items. These items were combined for the analysis, making all the indicators used suitable for taxometric analysis. Cohen's d values (the standardized mean difference between the putative taxon and its complement) are listed in Table 2. All but indicator 1 are well above the recommended value of 1.3 to ensure data suitable for taxometric analysis (Ruscio et al. 2006). Thus the sample meets the criteria necessary for identifying a taxon if one exists.

The data did show positive skew, which can make it difficult to visually interpret MAMBAC and MAXCOV curves. The skew characteristic of the data was taken into account in the bootstrap procedure that created the simulated comparison data.

MAMBAC Results

The MAMBAC analysis was done using 10 replications and 100 comparison samples for the simulated data, in order to ensure representative results. Fifty evenly spaced cuts were performed beginning at 25 cases from either end of the distribution, a procedure recommended for reliability on the basis of simulation studies (Walters and Ruscio 2009a, b). Using summed indicators is a common practice, motivated in part by data from screens that have limited response item numbers. However, evidence from simulations shows that analyses using summed indictors are less reliable than those that use each individual

Table 2 Cohen's d measure of size of difference between indicators in the putative taxon and complement

Indicator	Value	Cohen's d
1	0.848	0.880
2	1.684	1.994
3	1.735	2.082
4	1.849	2.287
5	1.660	1.955
6	2.049	2.705
7	1.774	2.149
8	1.426	1.599
Mean	1.628	1.956
SD	0.361	0.536

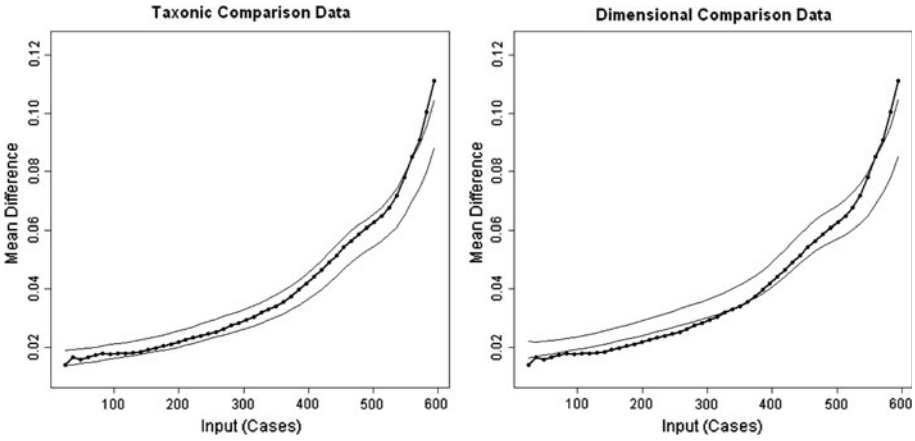

Fig. 4 MAMBAC comparison curves for simulated taxonic and dimensional data sets

indicator as input and output variables (Walters and Ruscio 2009a, b). We therefore followed this approach. Studies also suggest that having more than three response categories is nearly as conducive to reliability as having 20 (Walters and Ruscio 2009a, b).

The comparison curves for the simulated taxonic and dimensional data are shown in Fig. 4, plotted against curves representing 1 SD of the mean of the simulated data. The research data do not appear to more strongly visually resemble either the dimensional or categorical simulations. The base rate estimates across indicators, listed in Table 3, have a mean of 0.126 and a standard deviation (SD) of 0.043, providing some evidence of taxonicity. The CCFI value, which has been shown to be more reliable than the SD of the individual curves (Walters and Ruscio 2009b) is 0.58. Fits above 0.5 are evidence of taxonicity, but values between 0.4 and 0.6 must be treated with caution. Thus the MAMBAC results are suggestive but not strong evidence for taxonicity over dimensionality of PG as measured by the PGSI in our sample.

Because of the concerns mentioned in the introduction that the PGSI merges signs of addiction such as loss of control with more situational factors such as criticism by others, we reran the MABAC analysis with the latter items excluded, using PGSI items 2, 3, 4, and 8. The CCFI increased from 0.58 to 0.758. The latter score is strong evidence of taxonicity.

Table 3 MAMBAC: estimated taxon base rates for each indicator

Indicator	Rate
1	0.125
2	0.070
3	0.132
4	0.139
5	0.198
6	0.076
7	0.107
8	0.164
Mean[a]	0.126
SD[a]	0.0434

[a] Summary of base rates across indicators

MAXCOV Results

The MAXCOV analysis showed more decisive results. Again 10 replications and 100 samples were used. All individual indicators were used as triplets, a procedure shown to be superior to using summed indicators as input variables (Walters and Ruscio 2009a, b). Twenty-five windows were used with 0.90 overlap, parameters shown by simulation evidence to be the most reliable. Skew was again taken into account in generating the simulated comparison data. The comparison curves for the simulated taxonic and dimensional data are shown in Fig. 5 plotted against 1 SD of the mean of the comparison curves. The research data lie entirely within the taxon comparison curves. The range of estimates of base rates across indicators (Table 4) is considerably narrower than those produced by the MAMBAC analysis and range from 0.142 to 0.09, thus providing some support for taxonicity. The comparison fit index is 0.735, strongly supporting taxonicity over dimensionality. This fit level provides independent support for the conclusion reached more tentatively on the basis of the MAMBAC CCFI analysis.

PGSI Cutoff Points

We used the above results to interrogate the standard PGSI cutoff score of 8. Cases can be classified using the estimated base rate of all indicators produced in the MAMBAC and

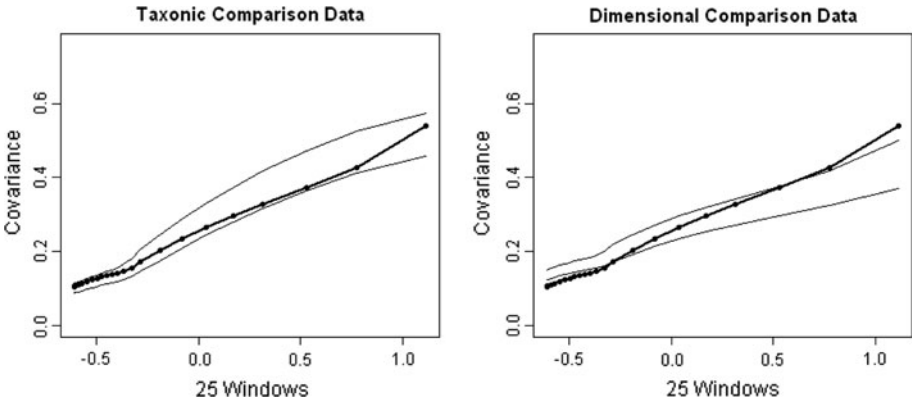

Fig. 5 MAXCOV curves for taxonic and dimensional comparison data

Table 4 Base rate estimates by indicator for MAXCOV analysis

Indicator	Rate
1	0.142
2	0.128
3	0.0965
4	0.117
5	0.102
6	0.090
7	0.106
8	0.137
Mean[a]	0.115
SD[a]	0.019

[a] Summary of base rates across indicators

MAXCOV analyses using the taxometric program "P.Classify" in Ruscio's R implementation. Extensive simulation studies (Ruscio 2009) have shown this to be an effective method for determining which individuals should be classified as members of a taxon.

The average estimated base rate of 12 % across the analyses was used to classify the cases in our data set. Since this base rate is notably lower than the 15.5 % rate that the cutoff of 8 produces, it is not surprising that fewer cases were counted as PGs by the taxometric algorithm. 65 cases were assigned PG status versus 96 using the standard PGSI cutoff. PGs were only found at a PGSI cutoff of 10 and higher. This may motivate revisiting other PGSI samples reported in the literature using this cutoff, with the aim of investigating a possible basis for the recurrent suggestions that the PGSI is biased in favor of false positive identifications of PG. While false positives may be preferable in a clinical setting, their presence in *research* on the predictors of disordered gambling will make it more difficult to find significant correlations.

Discussion

One taxometric procedure, MAMBAC, provided weak positive support for the conclusion that PG as measured by the PGSI is categorical in our study sample. A second taxometric procedure, MAXCOV, provided stronger evidence for the existence of a taxon. These are the first reported results bearing on the question of whether PG as measured by the currently most popular screen in general prevalence studies has a categorical or dimensional latent structure. Further research is needed using alternative screens, ideally designed on the basis of explicit models of the behavioral elements of addictive gambling.

There are several reasons to think that our data, despite providing moderate support that PG is taxonic, are not ideally suited for detecting taxonicity. Three key items of the DSM IV definition of PG are not present in the PGSI: preoccupation with gambling, repeated unsuccessful efforts to cut back or stop, and gambling to escape dysphoric mood. These arguably involve problems in self control, which is widely viewed as a crucial construct implicated in addiction (West 2006). The moderate evidence for taxonicity found in this study might result from the exclusion of these elements from the PGSI. Moreover, the components of the PGSI that concern financial and relationship problems may be secondary to addictive behavior rather than constitutive of it, and might vary with extraneous factors such as income levels. Individuals in lower South African socioeconomic groups are overrepresented in the set of pathological gamblers identified by the PGSI in our study. Contextual and variable factors such as these may be adding noise that explains why our MAMBAC analysis produces a less decisive result than our MAXCOV analysis. Our finding that the MAMBAC CCFI increased to 0.758, strong evidence for taxonicity, when items less directly related to loss of control were dropped supports this contention. In addition, the base rate produced by our analysis of cutoff scores suggest that the true base it is just above the minimum 10 % needed to find a taxon if one exists, so our sample may be biased against finding taxonicity. On the other hand, this increases the suggestiveness of our finding based on MAXCOV. So while a limitation of this study is that the results are not completely decisive, a real strength is that it produced significant evidence for taxonicity despite less than ideal data and screening instrument.

The findings from this study are also relevant to criticisms of the cutoff score of 8 that is standardly used for diagnoses and prevalence findings based on the PGSI. We have provided some evidence that this score may be too permissive if the goal is to identify a distinct group of gamblers with addiction-like characteristics. Further evaluation of the best

cutoff for this purpose would come from determining whether using the higher suggested cutoff of 10 allows for better prediction of variables associated with neural and clinical correlates of chronic inability to control gambling, a topic for future research. In the clinical setting, this result suggests caution in interpreting the meaning of scores of 8 and 9.

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Conflict of interest Don Ross is formerly Director of Research, and Jacques Rousseau and Andrew Dellis are former paid consultants, for the National Responsible Gambling Programme, South Africa, which funded the collection of the data used in the reported study. This agency receives money derived from a levy on casinos. Neither the agency nor the industry have any influence on design or reporting of research, which is quality assured and governed by the policies of the University of Cape Town.

References

Ahmed, A. O., Green, B. A., Clark, C. B., Stahl, K. C., & McFarland, M. E. (2011). Latent structure of unipolar and bipolar mood symptoms. *Bipolar Disorders, 2011*(13), 522–536.

Blaszczynski, A., & Nower, L. (2002). A pathways model of problem and pathological gambling. *Addiction, 97*(5), 487–499.

Braverman, J., LaBrie, R. A., & Shaffer, H. J. (2011). A taxometric analysis of actual internet sports gambling behavior. *Psychological Assessment, 23*(1), 234–244.

Custer, R. L., & Custer, L. F. (1978). *Characteristics of the recovering compulsive gambler*. Paper presented at the Fourth Annual Conference on Problem Gambling, Reno, Nevada.

Denson, T., & Earleywine, M. (2006). Pothead or pot smoker? A taxometric investigation of cannabis dependence. *Substance Abuse Treatment, Prevention and Policy, 1*, 22–27.

Ellis, G., Stein, D., Thomas, K., & Meintjes, E. (2011). *Substance use and abuse in South Africa: Insights from brain and behavioral sciences*. Cape Town: UCT Press.

Ferris, J., & Wynne, H. (2001). *The Canadian Problem Gambling Index: Final report*. Ottawa: Canadian Centre on Substance Abuse.

Ginestet, C., Mitchell, K., & Wellman, N. (2008). Taxometric investigation of the latent structure of nicotine dependence: An epidemiological sample. *Nicotine & Tobacco Research, 10*, 833–841.

Grove, W. M. (2004). The MAXSLOPE taxometric procedure: Mathematical derivation, parameter estimation, consistency tests. *Psychological Reports, 95*, 517–550.

Haslam, N., Williams, B., et al. (2006). The latent structure of attention-deficit/hyperactivity disorder: A taxometric analysis. *Australian and New Zealand Journal of Psychiatry, 40*(8), 639–647.

Helzer, J. E., Bucholz, K. K., et al. (2007). A dimensional option for the diagnosis of substance dependence in DSM-V. *International Journal of Methods in Psychiatric Research, 16*(S1), S24–S33.

Helzer, J. E., Van Den Brink, W., & Guth, S. E. (2006). Should there be both categorical and dimensional criteria for the substance abuse disorders in DSM-V? *Addiction, 101*, 17–22.

Hodgins, D. (2004). Using the NORC DSM Screen for Gambling Problems as an outcome measure for pathological gambling: Psychometric evaluation. *Addictive Behaviors, 29*, 1685–1690.

Holtgraves, T. (2009). Evaluating the Problem Gambling Severity Index. *Journal of Gambling Studies, 25*, 105–120.

Ladouceur, R., Jacques, C., Chevalier, S., Sevigny, S., & Hamel, D. (2005). Prevalence of Pathological Gambling in Quebec in 2002. *Canadian Journal of Psychiatry, 50*, 451–456.

Lesieur, H., & Blume, S. (1987). The South Oaks Gambling Screen (SOGS): A new instrument for the identification of pathological gamblers. *American Journal of Psychiatry, 144*(9), 1184–1188.

Loo, J. M., Oei, T. P., & Raylu, N. (2011). Psychometric evaluation of the Problem Gambling Severity Index-Chinese version (PGSI-C). *Journal of Gambling Studies, 27*(3), 453–466.

Maitland, S., & Adams, G. (2007). *Replication and generalizability of the Problem Gambling Severity Index: Are results consistent and comparable across studies? Final report*. Ontario: Ontario Problem Gambling Research Centre.

Meehl, P. E. (1973). MAXCOV-HITMAX: A taxometric search method for loose genetic syndromes. In P. E. Meehl (Ed.), *Psychodiagnosis: Selected papers* (pp. 200–224). Minneapolis: University of Minnesota Press.

Meehl, P. E. (1992). Factors and taxa, traits and types, differences of degree and differences in kind. *Journal of Personality, 60*, 117–174.
Meehl, P. E. (1995). Bootstraps taxometrics: Solving the classification problem in psychopathology. *American Psychologist, 50*, 266–274.
Meehl, P. E. (2004). What's in a taxon? *Journal of Abnormal Psychology, 113*, 39–43.
Meehl, P. E., & Yonce, L. J. (1994). Taxometric analysis: I. Detecting taxonicity with two quantitative indicators using means above and below a sliding cut (MAMBAC procedure). *Psychological Reports, 74*, 1059–1274.
Meehl, P. E., & Yonce, L. J. (1996). Taxometric analysis: II. Detecting taxonicity using covariance of two quantitative indicators in successive intervals of a third indicator (MAXCOV procedure). *Psychological Reports, 78*, 1091–1227.
Muthén, B. (2006). Should substance use disorders be considered as categorical or dimensional? *Addiction, 101*(s1), 6–16.
Neal, P., Delfabbro, P. H., & O'Neill, M. (2004). *Problem gambling and harm: Towards a national definition*. Report prepared for the National Gambling Research Program Working Party, Melbourne.
Orford, J., Wardle, H., et al. (2010). PGSI and DSM-IV in the 2007 British Gambling Prevalence Survey: Reliability, item response, factor structure and inter-scale agreement. *International Gambling Studies, 10*(1), 31–44.
Rawlings, D., Williams, B., et al. (2008). Taxometric analysis supports a dimensional latent structure for schizotypy. *Personality and Individual Differences, 44*(8), 1640–1651.
Reise, S., & Waller, N. (2009). Item response theory and clinical measurement. *Annual Review of Clinical Psychology, 5*, 27–48.
Ross, D., Dellis, A., Hofmeyr, A., Kincaid, H., Sharp, C., & Vucinich, R. (2010). *South African National Urban Prevalence Study of Gambling Behaviour*. http://uct.academia.edu/DonRoss/Papers/1258256/National_Urban_Prevalence_Study_of_Gambling_Behaviour_in_South_Africa_NUPSGB_Summary_Data.
Ross, D., Sharp, C., Vuchinich, R., & Spurrett, D. (2008). *Midbrain mutiny: The behavioural economics and neuroeconomics of disordered gambling*. Cambridge, MA: MIT Press.
Royston, P. (2004). Multiple imputation of missing values. *Stata Journal, 4*(3), 227–241.
Royston, P. (2007). Multiple imputation of missing values: Further update of ice, with an emphasis on interval censoring. *Stata Journal, 7*(4), 445–464.
Royston, P. (2009). Multiple imputation of missing values: Further update of ice, with an emphasis on categorical variables. *Stata Journal, 9*(3), 466–477.
Ruscio, J. (2007). Taxometric analysis: An empirically-grounded approach to implementing the method. *Criminal Justice and Behavior, 34*, 1588–1622.
Ruscio, J. (2009). Assigning cases to groups using taxometric results: An empirical comparison of classification techniques. *Assessment, 16*, 55–70.
Ruscio, J., Haslam, N., & Ruscio, A. (2006). *Introduction to the taxometric method: A practical guide*. Hillsdale: Lawrence Erlbaum Associates.
Ruscio, J., & Ruscio, A. (2004). Clarifying boundary issues in psychopathology: The role of taxometrics in a comprhensive program of structural research. *Journal of Abnormal Psychology, 113*, 145–154.
Ruscio, J., Ruscio, A., & Meron, M. (2007). Applying the bootstrap to taxometric analysis: Generating empirical sampling distributions to help interpret results. *Multivariate Behavioral Research, 42*, 349–386.
Ruscio, J., Walters, G., Marcus, D., & Kaczetow, W. (2010). Grouping the relative fit of categorical and dimensional latent variable models using consistency tests. *Psychological Assessment, 22*(1), 5–21.
Saunders, J. B., & Schukitt, M. A. (2006). The development of a research agenda for substance abuse disorders in the diagnosis in the Diagnostic and Statistical Manual of Mental Disorders, fifth edition (DSM-V). *Addiction, 101*, 1–5.
Sharp, C., Steinberg, L., Yaroslavsky, I., Hofmeyr, A., Dellis, A., Ross, D., et al. (2012). An item response theory analysis of the Problem Gambling Severity Index in a national representative sample. *Assessment, 19*(2), 167–175.
Slade, T., Grove, R., & Teesson, M. (2009). A taxometric study of alcohol abuse and dependence in a general population sample: Evidence of dimensional latent structure and implications for DSM-V. *Addiction, 104*, 742–751.
Solomon, A., Ruscio, J., et al. (2006). A taxometric investigation of unipolar depression in a large community sample. *Psychological Medicine, 36*(07), 973–985.
Svetieva, E., & Walker, M. (2008). Inconsistency between concept and measurement: The Canadian Problem Gambling Index (CPGI). *Journal of Gambling Issues 1s*(22), 157–173.

van Buuren, S., Boshuizen, H., & Knook, D. (1999). Multiple imputation of missing blood pressure covariates in survival analysis. *Statistics in Medicine, 1999*(18), 681–694.

Waller, N. G., & Meehl, P. E. (1998). *Multivariate taxometric procedures: Distinguishing types from continua*. Thousand Oaks, CA: Sage.

Walters, G. D., Diamond, P., & Magaletta, P. R. (2010). What is the latent structure of alcohol use disorders? A taxometric analysis of the Personality Assessment Inventory Alcohol Problems Scale in male and female prison inmates. *Psychology of Addictive Behaviors, 24*(1), 26–37.

Walters, G. D., & Ruscio, J. (2009a). To sum or not to sum: Taxometric analysis with ordered categorical assessment items. *Psychological Assessment, 21*(1), 99–111.

Walters, G. D., & Ruscio, J. (2009b). Where do we draw the line? Assigning cases to subsamples for MAMBAC, MAXCOV, and MAXEIG taxometric analyses. *Assessment, 20*(10), 1–13.

West, R. (2006). *Theory of addiction*. Oxford: Blackwell.

Widiger, T. A., & Clark, L. A. (2000). Toward DSM-V and the classification of psychopathology. *Psychological Bulletin, 126*, 946–963.