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A proof-of-concept study applying machine learning methods to putative risk factors for eating disorders: results from the multi-centre European project on healthy eating

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Abstract

Background. Despite a wide range of proposed risk factors and theoretical models, prediction of eating disorder (ED) onset remains poor. This study undertook the first comparison of two machine learning (ML) approaches [penalised logistic regression (LASSO), and prediction rule ensembles (PREs)] to conventional logistic regression (LR) models to enhance prediction of ED onset and differential ED diagnoses from a range of putative risk factors.

Method. Data were part of a European Project and comprised 1402 participants, 642 ED patients [52% with anorexia nervosa (AN) and 40% with bulimia nervosa (BN)] and 760 controls. The Cross-Cultural Risk Factor Questionnaire, which assesses retrospectively a range of sociocultural and psychological ED risk factors occurring before the age of 12 years (46 predictors in total), was used.

Results. All three statistical approaches had satisfactory model accuracy, with an average area under the curve (AUC) of 86% for predicting ED onset and 70% for predicting AN ν . BN. Predictive performance was greatest for the two regression methods (LR and LASSO), although the PRE technique relied on fewer predictors with comparable accuracy. The individual risk factors differed depending on the outcome classification (EDs ν . non-EDs and AN ν . BN).

Conclusions. Even though the conventional LR performed comparably to the ML approaches in terms of predictive accuracy, the ML methods produced more parsimonious predictive models. ML approaches offer a viable way to modify screening practices for ED risk that balance accuracy against participant burden.

Introduction

Eating disorders (ED) are psychiatric conditions characterised by high rates of comorbidity and relapse (e.g. Klump, Bulik, Kaye, Treasure, & Tyson, 2009). Decades of retrospective and longitudinal studies have sought to elucidate the key factors that contribute to EDs (e.g. Striegel-Moore & Bulik, 2007). Converging evidence across studies has identified various socio-cultural (Stice, 2002), demographic, family history, negative life events, personality, and genetic factors to play a role in influencing the onset and progression of EDs (Jacobi, Hayward, de Zwaan, Kraemer, & Agras, 2004). The large number of potential risk factors suggests that the pathway to an ED is complex and heterogeneous.

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Most studies that have aimed to identify ED risk factors have done so using a set of commonly used statistical methods (e.g. general, or generalised linear models) applied to a single dataset, without sufficient consideration of model parsimony or replicability of results. Although these studies have significantly advanced knowledge around the aetiology of EDs and have led to effective prevention programmes (Stice, Marti, Shaw, & Rohde, 2019), conventional statistical approaches have limitations that can hinder our ability to fully understand and more accurately predict ED onset and progression.

Conventional logistic regression (LR)-based approaches are best suited for the examination of smaller subsets of predictor variables that are theoretically justified. This is because the blind inclusion of many predictors can be one cause of overfitting, an artefactual increase in the predictive accuracy of a model simply because it contains more predictors than important predictors. Moreover, by focusing on sets of predictors that are unique to each study, overfitting can occur as there is rarely an opportunity to evaluate the performance of a predictive model in new datasets, affecting the generalisability of findings. The consequence of these practices is reduced ability to identify parsimony within a large/ complex set of risk variables and can mean that the predictive models have very low cross-sample replicability (Ranganathan, Pramesh, & Aggarwal, 2017). Thus, although previous studies have appropriately utilised conventional LR approaches on smaller subsets of predictors within a single dataset (e.g. Krug et al., 2009), it is likely necessary to incorporate the breadth of putative risk factors to establish which factors are most important and, in turn, devise the best models to accurately predict ED outcomes (Ali et al., 2019).

Moreover, although several ED-relevant models incorporate interaction terms within their models (e.g. Bardone-Cone, Abramson, Vohs, Heatherton, & Joiner, 2006), the list of proposed interactions is typically short, and there is seldom exploration of interactions that may be useful, yet not anticipated *a priori* (Stice & Desjardins, 2018). In light of evident heterogeneity in risk factor predictive value across samples, hitherto unexplored yet complex interactions among risk factors may enhance prediction. Traditional general(ised) linear models are ill-suited to searches for interaction terms as they are reliant on user anticipation of relevant variables to model in this way.

Machine-learning (ML) methods can overcome the limitations associated with conventional statistical approaches and are optimally suited to enhance the prediction of ED onset and progression. ML involves a constellation of data-driven techniques that enable computer algorithms to identify and iteratively refine the ideal parameters to fit complex patterns between variables (Bzdok & Meyer-Lindenberg, 2018). ML approaches are well suited to circumstances where there is a large amount of data to model and the best combination of predictors is uncertain a priori, as some ML approaches [such as prediction rule ensembles (PREs)] are designed to facilitate the detection of complex (nonlinear) high-dimensional interactions that might inform predictions at the individual patient level (Dwyer, Falkai, & Koutsouleris, 2018). Accumulating research has demonstrated the advantages of ML over conventional statistical approaches in predicting the course, trajectory, and treatment outcomes of severe psychiatric conditions (e.g. Chekroud et al., 2016).

In contrast, the application of ML methods to predict ED onset and progression is limited. Some studies have used ML-based classification-tree analyses to establish empirically derived cutpoints on various baseline measures for identifying individuals most at risk for an ED (for review, see Stice, 2016). Results from these studies suggest that there may be many transdiagnostic and diagnostic-specific interactions between putative risk factors that increase the probability of future illness onset and progression. However, it is noteworthy that each investigated a small number of putative risk factors that feature in prominent theoretical models, and as such did not capitalise on ML capabilities of being able to handle and detect complex patterns between many variables. Furthermore, these studies have not tested for competing ML analyses to conventional LR models, suggesting that we do not know the improved accuracy and model parsimony of ML methods over commonly used methods for predicting ED risk outcomes.

Two recent prospective studies examined the progression of EDs capitalised on the strengths of ML-methods. The first study by Haynos et al. (2020) used ML-techniques (elastic net, random forests) to predict ED behaviour and diagnostic persistence from more than 50 self-reported sociodemographic, clinical, and patient history variables in 320 participants at Year 1 and 277 individuals at Year 2 follow-up assessments. They found that the ML models provided consistently higher prediction accuracy over 1 and 2 years than the conventional LR models (with 19% greater classification accuracy). Another recent study by Espel-Huynh et al. (2021) compared ML approaches [support vector machine (SVM) and k-nearest neighbours] to conventional LR models to generate personalised predictions of symptom trajectories among 333 ED patients during the first two weeks of residential treatment. Contrary to Haynos et al.'s (2020) findings, this study revealed that the ML models did not improve predictive power beyond the one achieved by the LR analyses.

The current study

The present retrospective study builds on those two prior studies by applying ML-techniques to several putative ED risk factors on two outcomes: the presence v. absence of an ED, and the type of ED diagnosis (AN v. BN). The first outcome was selected because it is more important to identify those at elevated risk for any ED. This is because prevention programmes should ideally target all EDs, rather than just one subtype (Stice et al., 2019). The second outcome was selected as it enabled evaluation of whether this predictor set could also differentiate participants at the level of the ED subtype, thus informing treatment planning efforts.

The aims of the present study were as follows. First, we sought to compare two different statistical ML approaches against the traditional LR approach for accuracy in the prediction of our key outcomes. Second, we aimed to explore which predictors were identified as important in these competing models.

Methods

Participants and procedure

Data were drawn from six ED centres from five different European countries. The current sample of 1402 participants comprised 588 ED patients [333 with AN (174 with AN-restrictive subtype (AN-R), 159 with AN-binge purging (AN-BP) subtype) and 255 with BN (224 with BN purging subtype and 31 with BN non-purging subtype) and 760 healthy controls (54 had missing data on diagnosis). Diagnoses (DSM-IV-R, APA, 2000) were derived through a semi-structured clinical interview carried out by experienced psychologists and psychiatrists. Most of the ED participants were recruited from the participating clinical sites. The healthy control group was recruited from community sources from the same catchment areas. The exclusion criterion for the control group was a lifetime history of any health or mental illness (including EDs), screened by the General Health Questionnaire-28 (Goldberg, 1981). Ethical approval for the study was obtained from all study sites.

The cross-cultural (environmental) risk factor questionnaire (CCQ)

This retrospective self-administered questionnaire entails a total of 51 items, divided into six sections, which assess a wide range of factors related to the development and maintenance of EDs. Information on how the CCQ items were developed can be found in our previous publications (e.g. Krug et al., 2009).

Only items asking about events before the age of 12 years that took place before the ED emerged were included. A full list of all the 46 risk factors split into the four sections (1) *Eating and weight concerns;* (2) *Individual and family eating patterns;* (3) *Family style, expectations, and lifestyle behaviours;* (4) *Social ideals of thinness*] can be found in Table 1 and a copy of the full CCQ questionnaire can be requested from the corresponding author. Most of these questions were provided on a YES/NO format or a five-point response scale ranging from 'Not at all' to 'Extremely'.

Participants were also asked to provide their demographics, including age, gender, level of education and employment. In Section 3, questions on lifestyle behaviours were included because the developmental phenotype of social difficulties (i.e. loneliness, shyness, inferiority, and low social support) commonly found in ED patients (e.g. Krug et al., 2013) could be assessed indirectly through these CCQ questions.

A previous study assessing the psychometric properties of the CCQ (including all 127 items; Penelo et al., 2011) provided satisfactory accuracy for discriminating between ED cases and controls (area under the ROC curve = 0.88). In previous publications, Cronbach alpha values ranged from 0.75 to 0.92 (Krug et al., 2009; Penelo et al., 2011).

Statistical analyses

Three modelling approaches were used for comparison: (1) conventional binary LR, (2) LASSO, and (3) PREs. The conventional binary LR included all 46 risk factors simultaneously and retained all these factors in the final model. By contrast, the LASSO approach aims to balance parsimony with an overall model performance by penalising predictors that have only small contributions to the model by shrinking their effect sizes towards zero, with the outcome being a model that retains only those predictors that are important (Tibshirani, 1996). Finally, interaction effects were examined using PREs. PRE is a relatively new statistical learning method that automatically identifies stratifications within a set of predictors that improve the prediction of an outcome. These stratifications (subgroups defined by multiple interacting variables) are defined by cut points that are estimated by the PREs and can be written as simple rules that define an important stratification of the form if [condition] then [prediction] (Fokkema & Strobl, 2020). The conditions could be a decision rule that spans multiple interacting variables (e.g. if an individual reports dieting and weight-related teasing occurred in childhood, then they are more likely to have an ED currently). Specific decision rules are ranked based on a measure known as importance which can take values >0 (higher values mean stronger performance of the prediction rule) and account for both the magnitude

of its regression coefficient and the extent to which the stratification varies in the sample (Fokkema & Strobl, 2020).

All analyses were conducted in R version 6.3. Standard and penalised LR were estimated using the glmnet package version 3.0.2 (Friedman, Hastie, & Tibshirani, 2010) and PREs were estimated using the pre package version (Fokkema & Strobl, 2020). Several analytic decisions were held constant across these methods. First, an imputed data set was created for the control v. ED and BN v. AN samples using the mice package version 3.8.0 (Van Buuren & Groothuis-Oudshoorn, 2011). For each comparison, a positive coefficient reflected a higher risk for EDs or AN, whereas negative coefficients indicated a higher risk for non-ED or BN. Continuous variables were standardised by dividing scores by 2 standard deviations, as recommended to improve alignment with binary indicators (Gelman, 2008). Second, the overall dataset was divided with an 80/20% split for training and test sets, respectively, to mitigate potential overfitting to the training set and improve out-of-sample performance. Reported results are based on the 20% test set. Third, the predictive performance of models was assessed using the area under the curve (AUC) of the receiver operator characteristic and F1 scores, which is a metric that incorporates both the positive predictive value (a.k.a precision) and sensitivity (a.k.a recall) of model predicted outcomes. In accordance with other ML studies (Haynos et al., 2020) we applied a value of AUC of 0.5 to suggest no discrimination, whereas a value of 1 was given to perfect discriminations. AUC scores were able to range from 'extremely poor' (0.5-0.59), 'poor' (0.60- 0.69), 'fair' (0.70-0.79), 'good' (0.80-0.89), to 'excellent' (>0.90). Third, all models were run to predict both ED v. non-ED cases (n = 1402) and AN v. BN cases (n = 588), AN and BN only). Further details specific to each analytic approach are provided in the online Supplementary Material (S1).

Results

Sample descriptives

Demographics for the full sample, the ED samples, including AN and BN and the healthy control group are presented in Table 1. Most of the sample was female, employed and had high education levels.

Predictive performance

Predictive performance (AUC) was greater for distinguishing ED from healthy individuals (LR = 0.88; LASSO = 0.89; PREs = 0.82) than for differentiating individuals with AN ν . BN (LR = 0.71; LASSO = 0.70; PREs = 0.69; Table 2).

Based on AUC scores, predictive performance for distinguishing ED from healthy controls was greatest for the LASSO method, followed by conventional LR and then THE PREs. However, each method for the overall ED analyses was comparable, based on F1 scores (LR = 0.80; LASSO and PREs = 0.81), which considers precision and recall. For differentiating AN from BN cases, predictive performance was comparable based on AUC scores, although F1 scores favoured the PREs (0.74) method, followed by conventional LR (0.71), and then the LASSO (0.69) approach.

Important predictors

Conventional and penalised (LASSO) logistic regression

Coefficient estimates for the conventional and penalised (LASSO) LR models predicting ED (*v.* non-ED) and AN (*v.* BN) are presented in Table 3.

	Full sample $(n = 1402)^{a}$		Controls (<i>n</i> = 760)		ED (<i>n</i> = 588)		AN (<i>n</i> = 333)		BN (<i>n</i> = 255)	
	М	S.D.	М	S.D.	М	S.D.	М	S.D.	М	S.D.
Age (years)	24.90	8.27	23.62	8.20	26.39	8.10	26.19	8.70	26.65	7.26
BMI	20.91	4.50	21.52	2.91	19.76	4.17	17.56	2.73	22.52	4.04
BMI range	10.47-43.08		13.84-38.29		10.47-43.08		10.47-27.99		15.57-43.08	
	п	%	п	%	п	%	п	%	п	%
Sex										
Female	1073	92	586	89	451	96	258	96	193	97
Education										
Primary school	245	19	194	26	50	9	21	7	29	13
Secondary school	384	29	159	21	210	40	119	40	91	40
University degree	592	45	370	49	206	39	128	43	78	35
Professional degree	102	8	34	4	61	12	33	11	28	12
Employment										
Never been employed	411	33	330	47	73	14	51	18	22	10
Unemployed	270	22	109	15	153	30	88	30	65	30
Employed part-time	249	20	135	19	112	22	62	21	50	23
Employed full-time	321	26	135	19	171	34	90	31	81	37

Table 1. Sample demographics

ED, eating disorder; AN, anorexia nervosa; BN, bulimia nervosa. ^a54 cases missing ED status in raw data, but imputed in analyses; M, mean; s.o., standard deviation; *n*, number of cases.

Table 2. Predictive performance of the different statistical models

	AUC	Model fit	F1 score
ED v. Non-ED			
Conventional logistic regression	0.88	Good	0.80
Penalised regression (LASSO)	0.89	Good	0.81
Prediction rule ensemble	0.82	Good	0.81
AN v. BN			
Conventional logistic regression	0.71	Fair	0.71
Penalised regression (LASSO)	0.70	Fair	0.69
Prediction rule ensemble	0.69	Poor	0.74

ED, eating disorder; AN, anorexia nervosa; BN, bulimia nervosa; AUC, area under the curve. AUC and F1 scores range from 0–1, with higher scores reflecting greater accuracy.

For the conventional LR approach, we retained all predictors in the model. The strongest five predictors of ED status were own appearance concerns influenced eating ($\beta = 0.81$), number of times ate in fast-food restaurants ($\beta = 0.80$), unwanted sexual experiences ($\beta = 0.64$), doing schoolwork at school ($\beta = 0.61$) and family relationships influenced eating ($\beta = 0.61$),

The strongest five predictors of AN status were knowing anyone with an AN diagnosis ($\beta = 1.08$), had first meal of day before lessons started ($\beta = 0.99$), played computer games ($\beta = 0.93$), food prepared for respondent ($\beta = 0.79$), and own appearance concerns influenced eating ($\beta = -0.71$).

LASSO analyses retained 26 predictors of both non-ED v. ED and AN v. BN. The strongest five predictors of ED status were own appearance concerns influenced eating ($\beta = 0.75$), family relationships influenced eating ($\beta = 0.56$), doing schoolwork at school ($\beta = 0.43$), relationships with friends influenced eating ($\beta = 0.41$) and number of meals ate in fast-food restaurants ($\beta = 0.38$),

The strongest five predictors of AN status were played computer games ($\beta = 0.59$), own appearance concerns influenced eating ($\beta = -0.59$), having first meal of day before lessons started ($\beta =$ 0.58), schoolwork at school ($\beta = 0.44$) and knowing someone with an AN diagnosis ($\beta = 0.41$).

Prediction rule ensemble

Analyses identified 33 rules to differentiate individuals with an ED from those without and 13 prediction rules to differentiate individuals with a diagnosis of AN from those with a diagnosis of BN. Twenty distinct predictors were included in the 33 prediction rules for differentiating ED from non-ED participants, while twelve predictors were included in the 13 prediction rules for differentiating individuals with AN *v*. BN. Table 4 provides the ten strongest rules for differentiation of ED *v*. non-ED and AN *v*. BN [full list presented in online Supplementary Table S2 (ED *v*. non-ED) and online Supplementary Table S3 (AN *v*. BN)].

The most important prediction rules for ED v. non-ED cases suggest that individuals were <u>more</u> likely to have an ED if they report: (1) at least some influence from *family relationships on eating* (scores above 0), had to complete schoolwork at school (scores greater than 0), and reported at least some (>once a week) consumption of fatty/sugary snacks (scores greater than 1).

Conversely, participants were <u>less</u> likely to have an ED, if they exhibited the following protective factors in combination: (2) a low score on *own physical appearance concerns influenced eating* (scores of 3 or less), never *eating at fast-food restaurants*, and

less family relationship influence on their eating (scores less than or equal to 2); and (3) no reported influence of family relationships on eating (scores of 0) and low reported mass media influenced eating and relationships with friends influenced eating (scores of less than or equal to 1).

The best prediction rules for AN *v*. BN cases suggest that AN was <u>more</u> likely for individuals who: (1) *played computer* (scores greater than 0) and reported at least some *negative parenting from father* (scores greater than 0.33); and (2) reported usually *having first meal of day before lessons started* (scores greater than 0) and doing *schoolwork at school*.

Participants were <u>more</u> likely to present with BN if: (3) they reported that their *own appearance concerns influenced their eating* (scores greater than 2) and the absence of *strict parental rules about food* (scores of 0).

The most important predictor for both models was *own* appearance concerns influenced eating. For differentiating ED v. non-ED cases, family relationships influenced eating, relationship with friends influenced eating, frequency of eating fatty/sugary snacks, and family weight/shape concerns influenced eating were the next most important predictors across all prediction rules. In contrast, for differentiating AN v. BN cases, the next most important predictors were played computer games, parents had strict rules about food, negative parenting from father, and had first meal of day before lessons started (Fig. 1).

Discussion

We compared the results of the conventional LR analyses to two ML (LASSO and PREs) techniques in predicting ED classification and diagnostic ED subtype based on numerous risk factors. Three main findings emerged. First, we found a higher prediction accuracy for classifying ED v. non-ED cases relative to classifying AN v. BN cases across each approach. Second, the highest accuracy was obtained for the two regression methods (binary LR and LASSO regressions), although the PRE technique relied on fewer predictors with comparable accuracy. Third, a range of individual and familial risk factors emerged across the three statistical approaches, with different risk factors emerging depending on the outcome classification (EDs v. non-EDs and AN v. BN).

Our first main finding indicated that the prediction accuracy was higher for differentiating overall EDs from the healthy controls than AN from BN. This finding is in line with the notion of transdiagnostic risk factors for any ED (e.g. Stice, Marti, & Durant, 2011), and highlights the importance of developing ED prevention programmes targeting all EDs, rather than just one ED subtype. Our finding is also in agreement with the results of Haynos et al.'s (2020) findings, which found that for any ED diagnosis persistence, a baseline BN, and an absence of a partial baseline binge eating disorder (BED) diagnosis were found to be important predictors for the year 1, but not the year 2, follow-up prediction models. However, it should be noted that the models in Haynos et al.'s (2020) study yielded '*poor*' predictive performance, which might partially explain these inconsistent findings.

Our second result was that all three statistical approaches performed well when taking into consideration the indices (AUC and FI) and outcomes (ED ν . non-ED and AN ν . BN). This finding is consistent with a recent systematic review which found no performance benefit of ML over conventional LR (Christodoulou et al., 2019). Our result is also in line with the ED-specific study by Espel-Huynh et al. (2021), which found all SVM models

Table 3. Parameter estimates for conventional logistic regression and penalised (LASSO) regression for ED v. Non-ED and AN v. BN

		Non-ED v. ED		AN v. BN		
Full variable name	Abbreviation	Conventional logistic regression	LASSO	Conventional logistic regression	LASSO	
Domain 1						
Own appearance influenced eating	OwnAppInf	0.81	0.75	-0.71	-0.59	
Family weight/shape concerns influenced eating	FamAppInf	0.11	0.07	0.04	0.01	
Family Relationships influenced eating	FamRelInf	0.61	0.56	0.04	0.04	
Relationships with friend influenced eating	FriendRelInf	0.44	0.41	-0.01		
Joint dieting with family member(s) influenced eating	FamDietInf	-0.34	-0.30	-0.16	-0.15	
Teasing about eating habits by family/friends influenced eating	TeaseEatInf	-0.02	-0.01	-0.26	-0.07	
Teasing about weight/shape by family/friends influenced eating	TeaseAppInf	-0.16	-0.12	0.20	0.06	
Joint dieting with friends influenced eating	FriendDietInf	-0.01		-0.11	-0.05	
Mass media influenced eating	MediaInf	0.13	0.11	-0.01	-0.02	
Knew someone with low weight/AN	KnewAN	-0.19		1.08	0.42	
Knew someone overweight/overeating/BN	KnewBN	-0.06		-0.40		
Domain 2						
Number of relatives living at home	NumRels	0.25	0.13	0.01		
Number of family members present at most meals	NumFammeals	-0.07	-0.05	0.10	0.0	
No. of times ate meals together	NumTogether	-0.07	-0.06	0.18	0.0	
Value placed on food	FoodVal	0.00		-0.69	-0.3	
Inclusion in social events/meals	SocialFood	0.07	0.07	-0.12	-0.0	
Frequency with which ate fatty/sugary snacks	SnackFreq	0.39	0.37	0.05	0.02	
Ate meals at regular/set times of the day	AteReg	0.31	0.08	-0.70		
Had first meal of day before lessons started	MealBefore	0.29	0.15	0.99	0.58	
Number of times ate in fast-food restaurants	NumмealsRes	0.80	0.38	0.04		
Food prepared for family	FoodprepFam	-0.35	-0.14	0.25	0.12	
Food prepared for respondent	FoodprepRes	0.15		0.79	0.18	
Attention paid to healthy eating in the family	HltheatFam	0.08		-0.13		
Attention paid to healthy eating for the respondent	HltheatRes	-0.11		0.14		
Parents had strict rules about food	StrictRules	-0.24		0.66	0.20	
Access to salty or sugary snacks was more restricted than friends	SaltSugarRest	-0.25	-0.18	-0.33		
Frequency food was used as a reward	FoodReward	0.01		0.40	0.08	
Frequency access to food was restricted as a form of punishment	FoodRestPun	-0.24		-0.30		
Shortage of basic foods in family	BasicShortage	0.11		-0.34		
Shortage of luxury foods in family	LuxuryShortage	-0.01		-0.13		
Domain 3						
Positive parenting from mother	PosMoth	-0.30	-0.24	0.05	0.04	
Negative parenting from mother	NegMoth	0.21	0.16	0.16	0.03	
Abusive relationship from mother	AbuMoth	-0.07		-0.21	-0.10	
Positive parenting from father	PosFath	-0.02		0.04		
Negative parenting from father	NegFath	0.35	0.29	-0.12		

Table 3. (Continued.)

		Non-ED v. ED		AN v. BN	
Full variable name	Abbreviation	Conventional logistic regression	LASSO	Conventional logistic regression	LASSO
Abusive relationship with adult other than parents	Abuse	-0.02		-0.19	
Unwanted sexual experience	UnwantedSex	0.64	0.36	0.08	
Abusive relationship from father	AbuFath	-0.40	-0.32	0.06	
Schoolwork at school	Schoolwork	0.61	0.43	0.67	0.44
Schoolwork at home	SchoolworkHome	-0.02		-0.18	
Watched TV/videos	TV	-0.15		-0.52	
Played computer games	CompGames	-0.04		0.93	0.59
Read books	Books	-0.58	-0.31	0.43	0.11
Played with friends/siblings	Play	0.01		0.66	
Played sport	PlaySport	-0.06		-0.18	
Domain 4					
Body satisfaction as a child	BodSatChild	0.29	0.25	-0.15	-0.08

ED, eating disorder; AN, anorexia nervosa; BN, bulimia nervosa.

performed similarly well compared to the conventional LR analyses with. The best performing SVM in Espel-Huynh et al.'s (2021) was the radial-kernel SVM (AUC = 0.94), which was almost identical to the performance of the LR (AUC = 0.93). However, the current finding contradicts the findings by Haynos et al. (2020), which suggested clearer improvements in the predictive performance of ML models in comparison to conventional LR. It is worth noting that our study had an overall better prediction accuracy for the overall ED models for all statistical models (AUC average = 0.86) than Haynos et al.'s (2020) study (AUC average = 0.72]. It is possible that this might have resulted from the fact that our study focused on a more comprehensive range of risk factor predictors for EDs, whereas Haynos et al.'s (2020) models were based on a mixture of demographic, clinical and treatment predictors. Larger sample size in the present study may have also facilitated stronger predictive performance in the current test sample.

Given the similarly adequate predictive performance in our study across all methods, consideration of the feature selection capabilities helps provide context for how each model achieved this level of accuracy. Whereas the conventional LR required all 46 predictions, the LASSO approach required only 26 predictors. Similarly, given that predictive performance of the PREs was comparable to the other two methods, their greatest value is in: (1) identifying interaction terms not anticipated *a priori*, and (2) developing a more parsimonious set of predictors through the predictive value of incorporating these interaction terms. In the current study, the PREs identified 20 risk factors for the ED ν . non-ED model and 12 risk factors for the AN ν . BN model (*cf.* 46 predictors in the LR model), highlighting the gain in parsimony relative to decrement in predictive performance.

Interaction terms, as they are included in the PREs are seldom featured in ED risk models. We articulated a range of interactions in our decision rules, many of which were anticipated given the current ED risk factor literature (Stice & Desjardins, 2018; Stice et al., 2011). However, there was one noteworthy exception, in that for BN, we found one PRE to be based on *own appearance*

concerns influenced eating and the <u>absence</u> of *strict parental rules about food*. Contradicting this finding, previous research has shown strict parenting practices around food to lead to BN (e.g. MacBrayer, Smith, McCarthy, Demos, & Simmons, 2001). Future research is needed to verify these unexpected prediction rules identified in the current study.

Finally, we found some common ED risk factors for the three statistical approaches. The strongest risk factor for the overall ED models was the extent to which one's own appearance concerns influenced eating. This finding is in accordance with the findings of recent prospective studies (Stice, Gau, Rohde, & Shaw, 2017; Stice et al., 2011), which also revealed body dissatisfaction and existing disordered eating pathology to be the most salient proximal risk factors for EDs. A few other top-ranked risk factors [e.g., family relationships influenced eating (Krug et al., 2009), doing schoolwork at school, eating in fast-food restaurants (e.g. Mitchell et al., 2015) and eating sugary snacks (e.g. Easter et al., 2013) were also shared amongst all three statistical approaches for the overall ED models. All these factors have commonly been revealed in previous ED risk factor studies, except for schoolwork undertaken at school. It is possible that undertaking schoolwork at school might interfere with the establishment of a nurturing stable home environment by spending more time away from parents and siblings, though this variable requires future consideration in large-scale prospective studies.

Implications

The findings of our current study provide important insights into the utility of the novel computational approaches of ML approaches such as LASSO and PREs for advancing research on ED risk factors, with the final aim of developing data-derived personalised prevention and early intervention efforts. Findings of complex interactions among proposed risk factors highlight that the role of a specific risk factor may depend on a range of other contextual influences in one's environment. As such, prediction rules may enable a more nuanced understanding at the

Table 4. Prediction rules for predicting ED status

Rule	Imp	Coeff	S.D.
ED v. Non-ED			
OwnAppInf <=3 & NummealsRes <=0 & FamRelInf <=2	0.29	-0.59	0.49
FamRelInf <=0 & MediaInf <=1 & FriendRelInf <=1	0.23	-0.46	0.49
FamRelInf > 0 & SnackFreq > 1 & SchoolWork > 0	0.20	0.44	0.46
FriendRelInf <=1 & NegMoth <=2	0.19	-0.39	0.49
FriendRelInf <=1 & FamAppInf <=2 & AbuseFath > 0	0.19	-0.45	0.41
OwnAppInf > 2 & FamDietInf <= 0	0.16	0.36	0.45
OwnAppInf <= 3 & SnackFreq <= 2 & FamAppInf <= 2	0.14	-0.29	0.46
PosMoth > 2.5 & NegFath <=2	0.12	-0.24	0.50
OwnAppInf <= 2.5 & FamRelInf <= 2 & MediaInf <= 1	0.10	-0.20	0.50
SnackFreq <= 2 & UnwantedSex <= 0 & StrictRules <= 0	0.10	-0.22	0.44
AN v. BN			
OwnAppInf>2 & StrictRules <=0	0.30	-0.61	0.49
CompGames > 0 & NegFath > 0.33	0.23	0.57	0.41
MealBefore > 0 & SchoolWork > 0	0.19	0.42	0.47
OwnAppInf <= 2.5 & SnackFreq > 2	0.18	0.43	0.42
OwnAppInf <= 3.5 & SaltSugarRest <= 0	0.12	0.23	0.50
CompGames <= 0 & OwnAppInf > 1.5	0.07	-0.15	0.49
OwnAppInf <= 2 & SaltSugarRest <= 0	0.06	0.14	0.40
FoodVal <=0	0.05	0.10	0.50
MediaInf <=1	0.04	0.08	0.50
0 < = FamDietInf < = 4	0.02	-0.02	1.00

ED, eating disorder; AN, anorexia nervosa; BN, bulimia nervosa; Imp, variable importance; Coeff, coefficient; s.b., standard deviation.

Variable names: AbuseFath = 'Abusive relationship from father', CompGames = 'Played computer games', FamAppInf = 'Family weight/shape concerns influenced eating', FamDietInf = 'Joint dieting with family member(s) influenced eating', FamRelInf = 'Family Relationships influenced eating', FoodPrepRes = 'Food prepared for respondent', FriendRelInf = 'Relationships with friend influenced eating', MealBefore = 'Had first meal of day before lessons started', MediaInf = 'Mass media influenced eating', NegFath = 'Negative parenting from father', NegMoth = 'Negative parenting from mother', NumwealsRes = 'Number of times ate in fast-food restaurants', OwnAppInf = 'Own appearance influenced eating', PosMoth = 'Positive parenting from mother', SaltSugarRest = 'Access to salty or sugary snacks was more restricted than friends', SchoolWork = 'Schoolwork at school', SnackFreq = 'Frequency with which ate fatty/sugary snacks', StrictRules = 'Parents had strict rules about food', UnwantedSex = 'Unwanted Sex'.

individual level of both the risks to an individual and the mechanisms that place this individual at risk.

In the current study, all statistical approaches yielded favourable results for our risk predictions. However, the LASSO and PREs have several desirable features over conventional LR analyses, which makes them attractive approaches for future ED risk prediction studies. First, they tend to use less variables to achieve accuracy. This can be useful for screening purposes since researchers would need less measures to assess one's risk status. Second, the PREs approach utilises an automated search for useful interactions among a list of variables and may thus identify interactions not considered in previous research. It is therefore described as an exploratory, hypothesis-generating approach (Stice & Desjardins, 2018).

Limitations

The results of this study must be interpreted within the context of some methodological limitations. First, the retrospective, selfreport data collection procedures may have limited the validity and the reliability of our findings. It is for instance possible that some of the risk factors may have been heavily biased by the person's active ED status at the time of assessment (e.g. appearance concerns were identified as an important predating risk factor for an ED in hindsight simply because it was a salient factor during the illness). Second, all presumed risk factors were collected post ED diagnosis, which does not allow us to translate the findings into prediction before ED onset. All inferences about prediction are therefore primarily based on the prediction accuracy of our statistical models. Third, the current ED sample was assessed using DSM-IV-R criteria (APA, 2000). Since information on symptom level was not available, we could not convert these diagnoses into DSM-5 (APA, 2013) diagnoses. Fourth, the sample sizes for AN-R and AN-BP were not large enough to allow for separate analyses. Future studies would benefit from comprising larger ED samples to assess prediction accuracy in a range of ED diagnoses [also including BED and otherwise specified feeding and eating disorder (OSFED)]. Fifth, control participants were not explicitly screened for psychiatric diagnosis; therefore, it is not clear that the current model differentiated individuals with EDs from psychiatrically healthy individuals. Sixth, data on race/ethnicity were not collected on this sample (therefore it is unclear the extent to which the result generalise across this key demographic). Finally, the current study was not able to assess

Variable importances

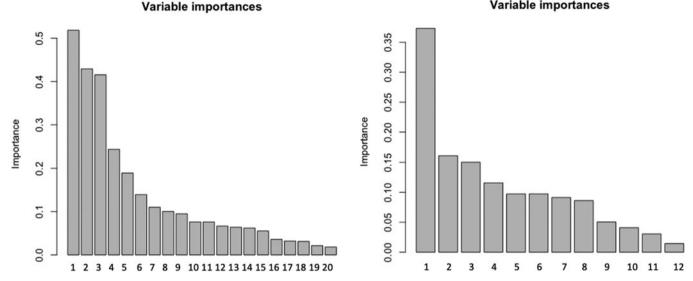


Fig. 1. Overall predictor importance for ED v. Non-ED (left) and AN v. BN (right). Notes. For ED v. Non-ED: 1 = OwnAppInf: 'Own appearance influenced eating'; 2 = FamRelInf: 'Family Relationships influenced eating', 3 = FriendRelInf: 'Relationships with friend influenced eating'; 4 = SnackFreq: 'Frequency with which ate fatty/ sugary snacks'; 5 = FamAppInf: 'Family weight/shape concerns influenced eating'; 6 = MediaInf: 'Mass media influenced eating'; 7 = NumMealsRes: 'Number of times ate in fast-food restaurants', 8 = FamDietInf: 'Joint dieting with family member(s) influenced eating'; 9 = NegMoth: 'Negative parenting from mother', 10 = PosMoth: 'Positive parenting from mother'; 11 = NegFath: 'Negative parenting from father'; 12 = Schoolwork: 'Schoolwork at school'; 13 = BodSatChild: 'Body Sattisfaction as a Child'; 14 = AbuFath: 'Abusive relationship from father'; 15 = SocialFood: 'Inclusion in social events/meals'; 16 = UnwantedSex: 'Unwanted Sex'; 17 = StrictRules: 'Parents had strict rules about food'; 18 = TeaseAppInf: 'Teasing about weight/shape by family/friends influenced eating'; 19 = NumFamMeals: 'Number of family members present at most meals'; 20 = TeaseEatInf: 'Teasing about eating habits by family/friends influenced eating'. For AN v. BN: 1 = OwnAppInf: 'Own appearance influenced eating'; 2 = CompGames: 'Played computer games'; 3 = StrictRules: 'Parents had strict rules about food'; 4 = NegFath: 'Negative parenting from father'; 5= MealBefore: 'Had first meal of day before lessons started'; 6= Schoolwork: 'Schoolwork at school'; 7= SnackFreq: 'Frequency with which ate fatty/sugary snacks'; 8 = SaltSugarRest: ' Access to salty or sugary snacks was more restricted than friends'; 9 = FoodVal: 'Value placed on food'; 10 = MediaInf: 'Mass media influenced eating'; 11 = FamDietInf: 'Joint dieting with family member(s) influenced eating'; 12 = FoodPrepRes: 'Food prepared for family'.

for biological and/ or genetic factors that could have interacted with the current environmental risk factors. Upcoming research should try to replicate the current findings in a longitudinal at-risk sample, including a range of biological, psychological, and social risk factors, before we can draw strong conclusions from the current findings.

Conclusion

This study applied ML methods to predict key ED outcomes from several risk factors. Our findings revealed that all three statistical methods yielded similar and appropriate statistical prediction accuracy. The risk factors covered for the different prediction models were mainly consistent with the literature and covered a range of previously established individual, familial and social ED risk factors, but also identified new risk factors. Even so, the overall performance of these models suggests a need for consideration of additional risk factors if we are to achieve strong predictive accuracy that limits false positives and ensures treatment is prioritised to those in greatest need. Present findings will ideally help generate more complex aetiological models that highlight distinct risk and protective pathways.

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Conflicts of interest. None.

Ethical standards. The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975, as revised in 2008.

The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional guides on the care and use of laboratory animals.

References

- Ali, A., Ali, S., Khan, S. A., Khan, D. M., Abbas, K., Khalil, A., ... Khalil, U. (2019). Sample size issues in multilevel logistic regression models. PLoS One, 14(11), e0225427. doi: 10.1371/journal.pone.0225427
- American Psychiatric Association (2000). Diagnostic and statistical manual of mental disorders (4th ed., Text Revision). Washington, DC: American Psychiatric Association.
- American Psychiatric Association (2013). Diagnostic and statistical manual of mental disorders (DSM-5*). American Psychiatric Publication.
- Bardone-Cone, A. M., Abramson, L. Y., Vohs, K. D., Heatherton, T. F., & Joiner, T. E. (2006). Predicting bulimic symptoms: An interactive model of self-efficacy, perfectionism, and perceived weight status. Behavior Research and Therapy, 44(1), 27-42. doi: 10.1016/j.brat.2004.09.009
- Bzdok, D., & Meyer-Lindenberg, A. (2018). Machine learning for precision psychiatry: Opportunities and challenges. Biological Psychiatry: Cognitive Neuroscience and Neuroimaging, 3(3), 223-230. doi: 10.1016/j.bpsc.2017.11.007
- Chekroud, A. M., Zotti, R. J., Shehzad, Z., Gueorguieva, R., Johnson, M. K., Trivedi, M. H., ... Corlett, P. R. (2016). Cross-trial prediction of treatment outcome in depression: A machine learning approach. The Lancet Psychiatry, 3(3), 243-250. doi: 10.1016/S2215-0366(15)00471-X
- Christodoulou, E., Ma, J., Collins, G. S., Steyerberg, E. W., Verbakel, J. Y., & Van Calster, B. (2019). A systematic review shows no performance benefit of machine learning over logistic regression for clinical prediction models. Journal of Clinical Epidemiology, 110, 12-22. doi: 10.1016/j.jclinepi.2019.02.004

- Dwyer, D. B., Falkai, P., & Koutsouleris, N. (2018). Machine learning approaches for clinical psychology and psychiatry. *Annual Review of Clinical Psychology*, 14, 91–118. doi: 10.1146/annurev-clinpsy-032816-045037
- Easter, A., Naumann, U., Northstone, K., Schmidt, U., Treasure, J., & Micali, N. (2013). A longitudinal investigation of nutrition and dietary patterns in children of mothers with eating disorders. *The Journal of Pediatrics*, 163(1), 173–178.e171. doi: 10.1016/j.jpeds.2012.11.092
- Espel-Huynh, H., Zhang, F., Thomas, J. G., Boswell, J. F., Thompson-Brenner, H., Juarascio, A. S., & Lowe, M. R. (2021). Prediction of eating disorder treatment response trajectories via machine learning does not improve performance versus a simpler regression approach. *International Journal of Eating Disorders*, 54(7), 1250–1259. doi: 10.1002/eat.23510
- Fokkema, M., & Strobl, C. (2020). Fitting prediction rule ensembles to psychological research data: An introduction and tutorial. *Psychological Methods*, 25(5), 636–652. doi: 10.1037/met0000256
- Friedman, J., Hastie, T., & Tibshirani, R. (2010). Regularization paths for generalized linear models via coordinate descent. *Journal of Statistical Software*, 33(1), 1–22. doi: 10.18637/jss.v033.i01
- Gelman, A. (2008). Scaling regression inputs by dividing by two standard deviations. Statisics in Medicine, 27(15), 2865–2873. doi: 10.1002/sim.3107
- Goldberg, D. P. (1981). Manual of the General Health Questionnaire (GHQ-28). Swindon, Wiltshire, IK: NFER Nelson Publishing.
- Haynos, A. F., Wang, S. B., Lipson, S., Peterson, C. B., Mitchell, J. E., Halmi, K. A., ... Crow, S. J. (2020). Machine learning enhances prediction of illness course: A longitudinal study in eating disorders. *Psychological Medicine*, 51(8), 1392–1402. doi: 10.1017/S0033291720000227
- Jacobi, C., Hayward, C., de Zwaan, M., Kraemer, H. C., & Agras, W. S. (2004). Coming to terms with risk factors for eating disorders: Application of risk terminology and suggestions for a general taxonomy. *Psychological Bulletin*, 130(1), 19–65. doi: 10.1037/0033-2909.130.1.19
- Klump, K. L., Bulik, C. M., Kaye, W. H., Treasure, J., & Tyson, E. (2009). Academy for eating disorders position paper: Eating disorders are serious mental illnesses. *International Journal of Eating Disorders*, 42(2), 97–103. doi: 10.1002/eat.20589
- Krug, I., Penelo, E., Fernandez-Aranda, F., Anderluh, M., Bellodi, L., Cellini, E., ... Treasure, J. (2013). Low social interactions in eating disorder patients in childhood and adulthood: A multi-centre European case-control study. *Journal of Health Psychology*, 18(1), 26–37. doi: 10.1177/1359105311435946
- Krug, I., Treasure, J., Anderluh, M., Bellodi, L., Cellini, E., Collier, D., ... Fernández-Aranda, F. (2009). Associations of individual and family eating patterns during childhood and early adolescence: A multicentre European study of associated eating disorder factors. *British Journal of Nutrition*, 101(6), 909–918. doi: 10.1017/s0007114508047752
- MacBrayer, E. K., Smith, G. T., McCarthy, D. M., Demos, S., & Simmons, J. (2001). The role of family of origin food-related experiences in bulimic

symptomatology. International Journal of Eating Disorders, 30(2), 149-160. doi: 10.1002/eat.1067

- Mitchell, J. E., King, W. C., Courcoulas, A., Dakin, G., Elder, K., Engel, S., ... Wolfe, B. (2015). Eating behavior and eating disorders in adults before bariatric surgery. *International Journal of Eating Disorders*, 48(2), 215–222. DOI: 10.1002/eat.22275
- Penelo, E., Granero, R., Krug, I., Treasure, J., Karwautz, A., Anderluh, M., ... Fernández-Aranda, F. (2011). Factors of risk and maintenance for eating disorders: Psychometric exploration of the cross-cultural questionnaire (CCQ) across five European countries. *Clinical Psychology & Psychotherapy*, 18(6), 535–552. doi: 10.1002/cpp.728
- Ranganathan, P., Pramesh, C. S., & Aggarwal, R. (2017). Common pitfalls in statistical analysis: Logistic regression. *Perspectives in Clinical Research*, 8 (3), 148–151. doi: 10.4103/picr.PICR_87_17
- Stice, E. (2002). Risk and maintenance factors for eating pathology: A meta-analytic review. *Psychological Bulletin*, 128(5), 825–848. doi: 10.1037/0033-2909.128.5.825
- Stice, E. (2016). Interactive and mediational etiologic models of eating disorder onset: Evidence from prospective studies. *Annual Review of Clinical Psychology*, 12, 359–381. doi: 10.1146/annurev-clinpsy-021815-093317
- Stice, E., & Desjardins, C. D. (2018). Interactions between risk factors in the prediction of onset of eating disorders: Exploratory hypothesis-generating analyses. *Behavior Research and Therapy*, 105, 52–62. doi: 10.1016/ j.brat.2018.03.005
- Stice, E., Gau, J. M., Rohde, P., & Shaw, H. (2017). Risk factors that predict future onset of each DSM–5 eating disorder: Predictive specificity in highrisk adolescent females. *Journal of Abnormal Psychology*, 126(1), 38. doi: 10.1037/abn0000219
- Stice, E., Marti, C. N., & Durant, S. (2011). Risk factors for onset of eating disorders: Evidence of multiple risk pathways from an 8-year prospective study. *Behavior Research and Therapy*, 49(10), 622–627. doi: 10.1016/ j.brat.2011.06.009
- Stice, E., Marti, C. N., Shaw, H., & Rohde, P. (2019). Meta-analytic review of dissonance-based eating disorder prevention programs: Intervention, participant, and facilitator features that predict larger effects. *Clinical Psychology Review*, 70, 91–107. doi: 10.1016/j.cpr.2019.04.004
- Striegel-Moore, R. H, & Bulik, C. M. (2007). Risk factors for eating disorders. *The American Psychologist*, 62(3), 181–98. doi: 10.1037/0003-066X.62.3.181.
- Tibshirani, R. (1996). Regression shrinkage and selection via the Lasso. *Journal of the Royal Statistical Society: Series B (Methodological, 58*(1), 267–288. doi: 10.1111/j.2517-6161.1996.tb02080.x
- Van Buuren, S., & Groothuis-Oudshoorn, K. (2011). Multivariate imputation by chained equations in R. *Journal of Statistical Software*, 45(3), 1–67. doi: 10.18637/jss.v045.i03