

A family history of alcoholism relates to alexithymia in substance use disorder patients

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Abstract

Objectives: Previous research identified alexithymia as a potential risk factor for substance use disorders (SUD). More insight into the relation between alexithymia and SUD is needed in order to treat SUD effectively. Therefore, we investigated whether a familial vulnerability to alcoholism relates to the presence and severity of alexithymia in SUD patients.

Method: Hospitalized, abstinent SUD-patients ($n = 187$), were assessed with the Toronto Alexithymia Scale (TAS-20) and Addiction Severity Index (EuropASI). A maternal, paternal, and total continuous measure of the Family History of Alcohol (FHA) was developed. Kruskal-Wallis tests and Spearman correlations were used to relate the composite scores of FHA to alexithymia as a categorical and continuous measure. Multivariate regression models were performed to control for the effects of confounders on the relation between FHA and alexithymia.

Results: Compared to moderate (33%) and low (17%) alexithymic SUD-patients, high alexithymic (50%) patients were more likely to have fathers with alcohol problems ($P = 0.004$). Such a difference was not found for mothers with alcohol problems. The composite FHA-score was significantly associated with alexithymia ($R_s = .19$, $P = 0.01$). However, only a paternal FHA, independent from disturbed family functioning, related to the degree of alexithymia ($\beta = .13$, $P = 0.06$), especially to the Difficulty Identifying Feelings as measured by the TAS-20 ($\beta = .16$, $P = 0.02$).

Conclusions: The relation between a paternal FHA and a higher degree of alexithymia in SUD-patients suggests that alexithymia could mediate the familiarity of alcoholism or SUD in the paternal line.

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1. Introduction

Sifneos first described the notion of alexithymia in 1973 [1] as the inability to express emotions or feelings. Alexithymia is mostly seen as a personality construct characterized as a deficit in the ability to cognitively process and regulate emotions [2]. Whereas the prevalence of alexithymia in population-based studies varies between 8% and 15% [3], rates of up to 67% have been reported in patients with alcohol use disorders (AUD) [4] and up to 50% in patients with other substance use disorders (SUD) [5,6]. In socio-demographic studies, alexithymia has been associated with older age, low educational level, low socio-economic

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status, poor perceived health, and depression, although not all of these associations have been consistently observed in all studies [7–9]. Additionally, in SUD, alexithymia was related to state-anxiety and depression [10]. As alexithymia has been described as a potential risk factor for SUD, and in some studies, it has been related to negative treatment outcomes, improving the understanding of the relation between alexithymia and SUD could be of importance in the treatment of SUD and the optimization of treatment interventions [4,11,12].

In the context of further research into the relationship between alexithymia and AUD, in particular in view of the potential role of alexithymic traits in the etiology of AUD, three previous studies looked at the effect of a family history of alcoholism (FHA) on alexithymia [13–15]. One study found strong alexithymic features in non-alcoholic sons with an extensive generational paternal history of alcoholism, but not in non-alcoholic sons without any family history or with alcoholic fathers without an extensive family history [13]. In the second study consisting of 100 male patients with alcohol dependence, no relation between a FHA and alexithymia was found [14]. However, both studies assessed alexithymia using the Sifneos-Schalling Personality Scale (SSPS), which lacks sufficient validity and internal reliability [2]. The most recent study conducted with a non-clinical population found an association between the Toronto Alexithymia Scale (TAS-20) and being the offspring of an alcoholic parent, as defined by the Children of Alcoholics Screening Test (CAST) [15]. This population comprised 314 volunteers (54% female and 53% university students) aged 18–45 years, all of whom reported at least occasional alcohol consumption and 6% using an illicit drug more than once per month. Unfortunately, the results were limited in specificity because the CAST does not allow differentiation between mothers or fathers with alcohol problems.

A substantial genetic influence of alexithymia has been demonstrated in a small and extensive twin pair study. This was replicated in a study that controlled for depressive symptoms [16–18]. Results from the first, small study [16] indicated that familial influences contributed to all 3 subscales of the TAS-20, i.e. Difficulty Identifying Feelings (DIF), Difficulty Describing Feelings (DDF) and Externally Oriented Thinking (EOT). The results also suggested that DIF and DDF were primarily influenced by nongenetic shared family environmental factors and EOT by genetic factors. However, because of the very small sample (77 twin pairs) and the way of recruiting, the different results could be due to selection bias [17]. Therefore the authors are not very confident concerning the distribution of the genetic and shared family environmental factors to the subscales of the TAS-20 [16]. The two other larger studies (8,785 [17] and 729 [18] twin pairs) found no differences between the subscales of the TAS-20. The Danish study [17] found nonshared environmental effects of 50–56%, heritabilities of 30–33% and 12–20% of the variance of the alexithymia scores being explained by shared environmental effects. In

the Italian study [18] nonshared environmental factors accounted also for most of the variation in the TAS-20 and its subscales. When corrected for depression and gender a heritability factor of 33% was found for the total TAS-20, with no significant differences between the subscales. No significant contribution of shared environmental influences on alexithymia was found [18].

Based on this genetic and familial influence, a higher percentage of alexithymia is expected in parents and other family members of alexithymic patients. As alexithymia and alcohol use disorders are related, this could be a reason for more alcohol problems in the relatives of alexithymic patients [4,15]. However, in alexithymic patients with SUD or AUD, other genetic, environmental or familial mechanisms could of course have an important role in the alcohol problems of their relatives [19].

As part of an often shared environmental or familial mechanism, problems with alcohol in parents could result in neglecting their child's emotional states, leading to emotional self-regulation deficits, such as alexithymia. The latter has been shown in a recent meta-analysis on parental bonding and alexithymia [20]. A lack of maternal care, but also maternal and paternal overprotection, related to alexithymia [20]. In line with this, a disturbed family functioning has been found to relate to the development of alexithymic characteristics [21]. Similar finding was observed for a history of neglect or sexual abuse, regardless of whether it occurred within the family [22–24].

In this study, our aim was to test the hypothesis that the presence of a FHA would be related to higher levels of alexithymia in SUD-patients while controlling for disturbed family functioning and other variables, representing a combination of shared and unshared environmental issues.

2. Method

2.1. Subjects

Participants were SUD inpatients from three addiction treatment centres in the East and South part of the Netherlands. The study sample participated in a randomized controlled trial investigating a Shared Decision Making Intervention (SDMI) in addiction health care that was carried out between January 2005 and December 2006 and published in 2009 [25]. Overall, 187 of the 212 participants (88%) in the RCT were willing to participate in the alexithymia study and were assessed accordingly. No distinction was made for the kind of substance(s) used. Exclusion criteria were being younger than 18 years of age, insufficient knowledge of the Dutch language, severe psychiatric co-morbidity precluding their participation in the SDM-intervention, or no signed informed consent. All patients have been diagnosed according to *DSM-IV-TR* as having one or more substance related disorders. The Dutch Ethical Assessment Committee for Experimental Investigations on People (No. 4.108) approved the study, and all participants gave written informed consent.

2.2. Instruments

For the assessment of alexithymia, the Dutch version of the TAS-20 was used [26–28]. It comprises three factors, DIF, DDF, and EOT. The TAS-20 demonstrated good internal consistency ($\alpha = .81$) and test-retest reliability over a three-week interval ($r = .77$). The TAS-20 further has a three factor structure congruent with the alexithymia construct [26,27]. Each item was measured on a five-point Likert scale ranging from “completely disagree” to “completely agree”. We used the total score and the 3 factor scores of the TAS-20 as dependent measures. The TAS-20 total scores can be categorized according to the empirically derived cut-off points suggested by Taylor et al. [2]. A total score of 61 and above indicates a high alexithymia score, scores between 52 and 60 represent a moderate degree, and scores of 51 and below indicate a low alexithymia score. The internal consistency of the total Dutch TAS-20, DIF, and DDF in psychiatric outpatients and students varied between $\alpha = .67$ and $\alpha = .85$. The internal consistency of the EOT in general population and psychiatric patient samples was lower ($\alpha = .52$ – $.66$). The test-retest reliability in psychiatric outpatients over a three-month interval was satisfactory for the total TAS-20 ($r = .74$) and DIF-factor ($r = .71$) but was less so for the DDF- ($r = .68$) and EOT-factors ($r = .66$) [28].

Type of substance use disorder was assessed by using the Composite International Diagnostic Interview, Substance Abuse Module (CIDI-SAM) [29]. The CIDI-SAM is an expanded and more detailed version of the substance use sections of the CIDI. The interview questions address the diagnostic criteria of *DSM-IV-TR* and *ICD-10* psychoactive SUD.

Severity of substance use at baseline was measured using the European Addiction Severity Index (EuropASI) [30,31]. The EuropASI is a clinical research interview designed to assess problem severity in 7 areas of functioning: physical health, employment, alcohol and/or drug use, legal, family/social, and psychiatric. Seven severity domains with scores that could range from 0 (no problem) to 9 (extremely serious problem) were derived from this interview. The family/social domain of the EuropASI represents an estimate of family and social problems and includes items assessing patients' history of trauma (physical or sexual). It is a combination of shared and unshared environmental issues.

2.3. Data analysis

Because alexithymia is better conceptualized as a continuous rather than a categorical variable [32], we especially examined associations with alexithymia as a continuous variable. However, to gain more insight on our patient sample from a clinical point of view, we also examined some associations with alexithymia as a categorical variable with low, intermediate, and high levels. Chi-Square tests were used to analyse dichotomous or categorical data and ANOVA or the Kruskal-Wallis test were used to analyse continuous data.

To assess the degree of FHA, we created a maternal, paternal, and total FHA variable by multiplying 1st family degree by a factor 3, 2nd degree by a factor 2, and 3rd degree by a factor 1, and then adding these scores up to a total maternal and paternal FHA-variable. We did use a genealogical classification of the different degrees of family relatedness: 1st family degree was represented by the biological parents (score range: 0–2: 0 = no parent with alcoholism; 1 = one parent with alcoholism etc.), 2nd degree by grandparents (score range: 0–4), brothers and sisters (score range: 0–4) and 3rd degree by aunts and uncles (score range: 0–4). Other (biological) family members were not assessed by the EuropASI and therefore not available. In case of unfamiliarity or doubt about the FHA or absence of specific family members, values were scored as negative for alcoholism. To check the validity of these variables, we related them to a well-known typology of alcohol dependence, namely Early (EOA) (≤ 25 years) or Late Onset Alcoholism (LOA) (> 25 years) with more evidence of familial alcoholism for EOA [33]. EOA or LOA was based on the EuropASI-question that inquired about the age at which patients started to drink 5 or more units of alcohol per occasion.

Multivariate linear regression models were conducted with total TAS-20 and factor scores as the dependent variables. The predictor variables, mostly based on previous research, were total, maternal and paternal FHA, age, gender, and EuropASI “family/social relations” and “psychiatric” domains [7,11,24,25]. Variables with a $P < 0.2$ in univariate analyses were entered in a full multivariate model. Subsequently, non-significant variables were removed, one by one, until R -squared changed by more than 10%.

All statistical tests were 2-sided, considered significant at a P value ≤ 0.05 , and conducted using SPSS for Windows (release 16.0).

3. Results

3.1. Patient characteristics

According to the cut-off score, 37% ($n = 69$) had a high degree of alexithymia, 30% ($n = 56$) had a moderate degree of alexithymia, and 33% ($n = 62$) had low alexithymia. The mean TAS-20 score was 55.7 ($SD = 11.3$). Forty-seven (25%) patients were female. Mean age was 40.7 years ($SD = 10.9$) and 94% was born in the Netherlands. Seventeen percent was married, 39% was divorced or widowed, and 44% had never been married. Forty-four percent was unemployed and mean years of education was 11.4 ($SD = 3.0$). Most, 54%, preferred alcohol as the substance of preference, 29% polydrugs, 11% cocaine or other stimulants, 4 % cannabis, and 2% other substances, with no gender differences. Alexithymia was measured as a continuous variable related to years of education ($r = -.19$, $P = 0.01$) and the EuropASI “psychiatry” domain ($r = .31$, $P < 0.001$), but unrelated to gender, age, country of birth,

kind of relationship, degree of employment, years of alcohol or drug use, type of substance dependence, substance preference, and other EuropASI domains.

3.2. Relation between FHA and early (EOA) or late-onset alcoholism (LOA)

No significant differences were found between the maternal FHA in EOA ($n = 82$) [Mdn = 0.0 (IQR = 0.0–2.2)] and in LOA ($n = 53$) [Mdn = 0.0 (IQR = 0.0–1.0)] ($U = 1845.0$, $z = -1.7$, $P = 0.09$) and between the paternal FHA ($n = 82$) in EOA [Mdn = 0.0 (IQR = 0.0–3.0)] and in LOA ($n = 53$) [Mdn = 0.0 (IQR = 0.0–3.0)] ($U = 1969.5$, $z = -1.0$, $P = 0.31$). However, a difference was found between EOA ($n = 81$) [Mdn = 3.0 (IQR = 0.0–6.0)] and LOA ($n = 53$) [Mdn = 2.0 (IQR = 0.0–4.0)] ($U = 1714.0$, $z = -2.0$, $P = 0.05$) for the total FHA-variable.

3.3. Relation between FHA and alexithymia

High alexithymic patients were more likely to have alcoholic fathers but not mothers compared to moderate and low alexithymic patients (Table 1). When comparing patients with none, one, or two alcoholic parents, we found that high alexithymic patients were more likely to have two alcoholic parents compared to moderate and low alexithymic patients. Looking at the paternal, maternal, and total FHA, high alexithymic patients obtained higher scores on the total, paternal, and maternal FHA compared to low alexithymic patients.

Mean (SD) of the total TAS-20 for patients with non-alcoholic mothers did not differ from the mean obtained by patients with alcoholic mothers [$M = 55.3$ (11.4); $M = 58.1$ (11.4); t (181) = 1.2, $P = 0.23$]. These patients did not differ in the DIF [$M = 18.8$ (6.4); $M = 20.9$ (6.7); t (181) = 1.6, $P = 0.11$], DDF [$M = 16.3$ (4.3); $M = 16.4$ (3.8); t (181) = 0.7, $P = 0.91$] and EOT [$M = 20.3$ (4.2); $M = 20.9$ (4.1); t (181) = 0.7, $P = 0.47$]. However,

differences were found for patients with non-alcoholic versus alcoholic fathers on the TAS-20 [$M = 53.9$ (11.7); $M = 59.1$ (9.8); t (177) = 3.0, $P = 0.003$], the DIF-factor [$M = 17.9$ (6.5); $M = 21.2$ (5.5); t (177) = 3.3, $P = 0.001$] and the DDF-factor [$M = 15.8$ (4.4); $M = 17.1$ (3.9); t (177) = 2.0, $P = 0.05$], but not on the EOT-factor [$M = 20.2$ (4.1); $M = 20.8$ (4.2); t (177) = 1.0, $P = 0.32$].

When comparing patients with none, one, or two alcoholic parents, the total TAS-20 [$M = 53.9$ (12.0); $M = 57.9$ (9.3); $M = 60.1$ (11.3); F (2, 173) = 3.5, $P = 0.03$] and the DIF-factor [$M = 18.0$ (6.6); $M = 20.1$ (5.5); $M = 22.3$ (6.3); F (2, 173) = 4.5, $P = 0.01$] differed, but not the DDF and EOT-factors. Post hoc tests (Tukey HSD) showed a significant difference between patients with none and two alcoholic parents in the DIF-factor only.

The results indicated significant correlations (Spearman) between maternal FHA and both the total TAS-20 ($R_s = .15$, $P = 0.04$; $n = 187$) and the DIF-factor ($R_s = .20$, $P = 0.006$; $n = 187$), between paternal FHA and both the total TAS-20 ($R_s = .19$, $P = 0.01$; $n = 187$) and the DIF-factor ($R_s = .24$, $P = 0.001$; $n = 187$), and between total FHA and both the TAS-20 total ($R_s = .19$, $P = 0.01$; $n = 186$) and the DIF-factor ($R_s = .28$, $P < 0.001$; $n = 186$).

3.4. Estimating the relation between the FHA and alexithymia, controlling for gender, age, EuropASI “family/social relations” and “psychiatry” domains

When performing multivariate regression models with the total TAS-20 or TAS-20 factors as dependent variables, total FHA contributed a small part ($\beta(r) = .16$, $P = 0.02$) to the variance in the DIF-factor and although not significantly ($\beta(r) = .13$, $P = 0.11$) to the variance in the total TAS-20, next to the “psychiatry” domain of the EuropASI and the years of education (Table 2). Regression models conducted separately for the maternal and paternal line indicated that only paternal FHA contributed a small part to

Table 1

Family history of alcoholism: characteristics of low, moderate and high alexithymic patients ($n = 187$).

Patient characteristics	Low alexithymic (TAS < 52) n = 62 (33.2%)	Moderate alexithymic (51 < TAS < 61) n = 56 (29.4%)	High alexithymic (TAS > 60) n = 69 (36.9%)	χ^2	H	P
Mother %(n)				1.9		0.38
Alcoholic(–)	35.1(54)	29.2(45)	35.7(55)			
Alcoholic(+)	24.1(7)	27.6(8)	48.3(14)			
Father %(n)				11.1		0.004
Alcoholic(–)	41.3(50)	28.1(34)	30.6(37)			
Alcoholic(+)	17.2(10)	32.8(19)	50.0(29)			
Parents %(n)				12.2		0.02
Alcoholic(–)	41.8(46)	26.4(29)	31.8(35)			
Alcoholic(+)	20.0(10)	38.0(19)	42.0(21)			
Alcoholic(++)	18.8(3)	18.8(3)	62.5(10)			
Maternal FHA Mdn(IQR)	0.0(0.0–0.0)	0.0(0.0–1.8)	0.0(0.0–3.0)		7.6	0.02
Paternal FHA Mdn(IQR)	0.0(0.0–1.0)	0.0(0.0–3.0)	0.0(0.0–3.0)		9.3	0.01
Total FHA Mdn(IQR)	1.0(0.0–4.0)	3.0(0.0–5.0)	3.0(0.5–6.0)		9.5	0.009

Notes: Mdn = Median, IQR = Interquartile Range: 25th–75th percentile; FHA = Family History for Alcoholism; H = Kruskal-Wallis test.

Table 2

Multivariate linear regression analyses predicting TAS-20 total and factors from gender, age, the “family/social” and “psychiatry” domains and total family history of alcoholism (FHA).

	<i>B(r)</i>	<i>P</i>	<i>R</i> ²	<i>F</i> change	<i>P</i>
TAS-20 total (n = 185)			.14	9.39	<0.001
“Psychiatry” domain	.28	<0.001			
Years of education	-.15	0.02			
Total FHA	.13	0.11			
DIF-factor (n = 185)			.22	24.93	<0.001
“Psychiatry” domain	.40	<0.001			
Total FHA	.16	0.02			
DDF-factor (n = 186)			.05	9.22	0.003
“Psychiatry” domain	.22	0.003			
EOT-factor (n = 187)			.09	8.70	<0.001
Gender	-.15	0.03			
Years of education	-.26	<0.001			

Non-significant variables were removed until R-squared changed by more than 10%.

the variance in the total TAS-20 ($\beta(r) = .13$, $P = 0.06$) and the DIF-factor ($\beta(r) = .16$, $P = 0.02$) (Table 3).

4. Discussion

In response to our research question, we found that high alexithymic SUD-patients were more likely to have fathers or both fathers and mothers, but not mothers only, with alcohol problems compared to low alexithymic SUD-patients. Next, we found that especially paternal FHA relates to the degree of alexithymia, independent of disturbed family functioning.

The high degree of alexithymia in our abstinent SUD sample is consistent with previous reports [4,10,34]. Higher scores on the “psychiatry” severity EuropASI domain reflect symptoms of anxiety and depression [31]. The relationship between alexithymia and these symptoms

suggests that the high baseline alexithymia score can at least partially be interpreted as a state phenomenon [4,34]. That relationship is also the reason why we controlled for “psychiatry” severity while analysing the association between alexithymia and FHA. Alexithymia was related to years of education, as was found in previous studies of the general population [3,8], but not in all [35]. Salminen et al. [3] argued that alexithymic persons are less likely to seek higher education reflecting the social status, values and emotional atmosphere in the family of origin and therefore yielding information about the individual’s developmental background. In our study, years of education were related to the EOT-factor and not to the DIF- and DDF-factor. The aforementioned studies [3,8] did not consider the relations between years of education and the TAS-20 factors. Further developmental and longitudinal studies of alexithymic persons are therefore needed to address the relation between years of education and alexithymia, in particular on factor level. However, we do agree with Salminen et al. [3] that a higher education often requires more psychological introspection and therefore could be less attractive for alexithymic individuals.

The results indicated no significant differences between patients with and without alcoholic mothers in total alexithymia as a dimensional variable or factor scores. We did find a relation between the maternal FHA and alexithymia as a categorical and dimensional variable (total TAS and DIF-factor). However, the effect sizes for alexithymia as a continuous variable were small and the relation disappeared when controlling for psychiatric problems, including anxiety, depression, and the paternal FHA. Therefore, no relation was found between mothers with an alcohol problem themselves or their families and alexithymia in their offspring.

For patients with alcoholic fathers, we found a higher degree of total alexithymia, DIF-, and DDF-factors compared with patients of non-alcoholic fathers, as supported by differences among high, moderate, and low alexithymic patients in percentage of alcoholic fathers. Although the relation between the paternal FHA and offspring alexithymia as a dimensional variable (total TAS and DIF-factor) had a small effect size, it remained significant after controlling for psychiatric problems and other possibly confounding factors like gender, age, and problems in the family or social relations. This indicates that a paternal FHA relates to alexithymia in SUD-patients and is in line with the study of Finn et al. [13], that demonstrated a relation between a paternal FHA and alexithymia, but unlike the study of Rybakowski et al. [14], in which no relation between FHA and alexithymia was found. In both studies, the FHA was defined as a categorical variable, unlike our continuous score. Next, these studies used an insufficiently valid and unreliable instrument, the SSPS, for assessing alexithymia and did not account for possible confounders [2,13,14]. Because the SSPS has another factor structure that has not been validated [2], unlike the TAS-20 [26,27], we could not

Table 3

Multivariate linear regression analyses predicting TAS-20 total and factors from gender, age, the “family/social” and “psychiatry” domains and family history of alcoholism (HFA) of the maternal and paternal lines.

	<i>B(r)</i>	<i>P</i>	<i>R</i> ²	<i>F</i> change	<i>P</i>
TAS-20 total (n = 186)			.14	10.04	<0.001
“Psychiatry” domain	.28	<0.001			
Years of education	-.16	0.06			
Paternal FHA	.13	0.06			
DIF-factor (n = 186)			.21	23.68	<0.001
“Psychiatry” domain	.40	<0.001			
Paternal FHA	.16	0.02			
DDF-factor (n = 186)			.05	9.22	0.003
“Psychiatry” domain	.22	0.003			
EOT-factor (n = 187)			.09	8.70	<0.001
Gender	-.15	0.03			
Years of education	-.26	<0.001			

Non-significant variables were removed until R-squared changed by more than 10%.

make a comparison on alexithymia factor levels between our study and that of Finn et al. [13].

High alexithymic patients had a higher percentage of both parents with alcohol problems compared to moderate and low alexithymic patients. Further, patients who had both parents with alcohol problems scored higher on the DIF-factor compared to patients without alcohol abusing parents. However, without controlling for unshared environmental or psychological confounders, this is not a strong indication for a familial component in the relation between alcohol problems of the parents and a higher alexithymia score in their offspring. For the total FHA, we controlled for some shared (family domain of the EuropASI) and unshared (social relation domain of the EuropASI, age and gender) environmental and psychological confounders. We found that total FHA explained a small part of the variance in the total alexithymia and the DIF-factor. Because this part of the explained variance was just as large as for the paternal FHA, only the latter is of interest in explaining a part of the high prevalence of alexithymia in SUD-patients. We have no good explanation for only a paternal familial transmission. In familial transmission and inheritance studies of alexithymia, no gender differences were found [16–18]. Although some evidence exist concerning sex differences in the inheritance of alcoholism [36] most research suggests an equivalent genetic load for alcohol dependence in both genders [37] and the absence of gender difference in familial transference [38].

Our measure of FHA reflects a combination of genetic and shared environmental aspects and it is impossible to discern the genetic (including gene-environment interaction) and the shared environmental part that contributed to the variance of the DIF-factor and the total TAS-20. In previous twin studies on alexithymia [16–18] no agreement was found in the contribution of the genetic and shared environmental parts on the subscales of the TAS-20. Without one or more replications of our finding it therefore makes little sense to look for explanations that only the DIF-factor and not the DDF- and EOT-factor were related to the FHA.

Our results should be interpreted in the context of some limitations. Our measures of depression, anxiety, and family functioning, especially the relation between parents and patients, were rather global and not specific enough to distinguish unshared and shared environmental issues. We constructed a new measure of total FHA, which was related to an EOA. However, our measure of paternal FHA was not, and familial alcoholism in EOA is especially seen in the paternal line [33]. Finally, we used the family history approach and collected information on family members from our patients rather than from these family members themselves.

In conclusion, we did find a relation between a paternal FHA and a higher degree of alexithymia in SUD-patients, with an indication that this relation is partly based on a familial component. The clinical importance of this finding is that alexithymia could mediate the familiarity of alcoholism or SUD in the paternal line, however, to a

small extent, given the correlations of the paternal FHA with total TAS-20 ($\beta(r) = .13$) and DIF-factor ($\beta(r) = .16$). In order to look for more causal models to explain this relationship, structural equation modeling should be performed on larger datasets of twins and extended family that would provide both data on substance use and alexithymia of all family members. Further, more directly, candidate genes may be considered to test for associations between genetic polymorphisms shown to be related to alexithymia and alcoholism [39,40]. With more indications for alexithymia as an endophenotype that mediates familial risk for alcoholism, it would be interesting to develop evidence-based treatments for alexithymia, which to our knowledge do not exist.

References

- [1] Sifneos PE. The prevalence of 'alexithymic' characteristics in psychosomatic patients. *Psychother Psychosom* 1973;22:255–62.
- [2] Taylor GJ, Bagby RM, Parker JDA. Disorders of affect regulation. Cambridge: Cambridge university press; 1997.
- [3] Salminen JK, Saarijarvi S, Aarela E, Toikka T, Kauhanen J. Prevalence of alexithymia and its association with sociodemographic variables in the general population of Finland. *J Psychosom Res* 1999;46:75–82.
- [4] Thorberg FA, Young RM, Sullivan KA, Lyvers M. Alexithymia and alcohol use disorders: a critical review. *Addict Behav* 2009;34:237–45.
- [5] Haviland MG, Shaw DG, MacMurray JP, Cummings MA. Validation of the Toronto alexithymia scale with substance abusers. *Psychother Psychosom* 1988;5:81–7.
- [6] Oyefeso A, Brown S, Chiang Y, Clancy C. Self-injurious behaviour, traumatic life events and alexithymia among treatment-seeking opiate addicts: Prevalence, pattern and correlates. *Drug Alcohol Depend* 2008;98(3):227–34.
- [7] Mattila AK, Salminen JK, Nummi T, Joukamaa M. Age is strongly associated with alexithymia in the general population. *J Psychosom Res* 2006;61:629–35.
- [8] Franz M, Popp K, Schaefer R, Sitte W, Schneider C, Hardt J, et al. Alexithymia in the German general population. *Soc Psychiatry Psychiatr Epidemiol* 2008;43:54–62.
- [9] Saarijarvi S, Salminen JK, Tamminen T, Aarela E. Alexithymia in psychiatric consultation-liaison patients. *Gen Hosp Psychiatry* 1993; 15:330–3.
- [10] Haviland MG, Hendry MS, Shaw DG, Henry JP. Alexithymia in women and men hospitalized for psychoactive substance dependence. *Compr Psychiatry* 1994;35:124–8.
- [11] Loas G, Fremaux D, Otmani O, Lecercle C, Delahousse J. Is alexithymia a negative factor for maintaining abstinence? A follow-up study. *Compr Psychiatry* 1997;38:296–9.
- [12] Ziolkowski M, Gruss T, Rybakowski JK. Does alexithymia in male alcoholics constitute a negative factor for maintaining abstinence? *Psychother Psychosom* 1995;63:169–73.
- [13] Finn PR, Martin J, Pihl RO. Alexithymia in males at high genetic risk for alcoholism. *Psychother Psychosom* 1987;47:18–21.
- [14] Rybakowski J, Ziolkowski M. Clinical and biochemical heterogeneity of alcoholism: the role of family history and alexithymia. *Drug Alcohol Depend* 1991;27:73–7.
- [15] Lyvers M, Onuoha R, Thorberg FA, Samios C. Alexithymia in relation to parental alcoholism, everyday frontal lobe functioning and alcohol consumption in a non-clinical sample. *Addict Behav* 2012;37:205–10.
- [16] Valera EM, Berenbaum H. A twin study of alexithymia. *Psychother Psychosom* 2001;70:239–46.
- [17] Jorgensen MM, Zachariae R, Skytthe A, Kyvik K. Genetic and environmental factors in alexithymia: a population-based study of 8,785 Danish twin pairs. *Psychother Psychosom* 2007;76:369–75.

- [18] Picardi A, Fagnani C, Gigantesco A, Toccaceli V, Lega I, Stazi MA. Genetic influences on alexithymia and their relationship with depressive symptoms. *J Psychosom Res* 2011;71:256–63.
- [19] Wang JC, Kapoor M, Goate AM. The genetics of substance dependence. *Annu Rev Genomics Hum Genet* 2012;13:241–61.
- [20] Thorberg FA, Young RM, Sullivan KA, Lyvers M. Parental bonding and alexithymia: a meta-analysis. *Eur Psychiatry* 2011;26:187–93.
- [21] Lumley MA, Mader C, Gramzow J, Papineau K. Family factors related to alexithymia characteristics. *Psychosom Med* 1996;58:211–6.
- [22] Berenbaum H. Childhood abuse, alexithymia and personality disorder. *J Psychosom Res* 1996;41:585–95.
- [23] Bermond B, Moormann PP, Albach F, van Dijke A. Impact of severe childhood sexual abuse on the development of alexithymia in adulthood. *Psychother Psychosom* 2008;77:260–2.
- [24] Evren C, Evren B, Dalbudak E, Ozelik B, Oncu F. Childhood abuse and neglect as a risk factor for alexithymia in adult male substance dependent inpatients. *J Psychoactive Drugs* 2009;41:85–92.
- [25] Joosten EAG, de Jong CAJ, de Weert-van Oene GH, Sensky T, van der Staak CPF. Shared decision-making reduces drug use and psychiatric severity in substance-dependent patients. *Psychother Psychosom* 2009;78:245–53.
- [26] Bagby RM, Parker JDA, Taylor GJ. The Twenty-Item Toronto Alexithymia Scale: I. Item selection and cross-validation of the factor structure. *J Psychosom Res* 1994;38:23–32.
- [27] Bagby RM, Taylor GJ, Parker JDA. The Twenty-Item Toronto Alexithymia Scale: II. Convergent, discriminant, and concurrent validity. *J Psychosom Res* 1994;38:33–40.
- [28] Kooiman CG, Spinhoven P, Trijsburg RW. The assessment of alexithymia – a critical review of the literature and a psychometric study of the Toronto Alexithymia Scale-20. *J Psychosom Res* 2002;53:1083–90.
- [29] Compton WM, Cottler LB, Dorsey KB, Spitznagel EL, Mager DE. Comparing assessments of *DSM-IV* substance dependence disorders using CIDI-SAM and SCAN. *Drug Alcohol Depend* 1996;42:217–9.
- [30] McLellan AT, Luborsky L, Woody GE, O'Brien CP. Improved diagnostic evaluation instrument for substance abuse patients – Addiction Severity Index. *J Nerv Ment Dis* 1980;168:26–33.
- [31] Hendriks VM, Kaplan CD, Vanlimbeek J, Geerlings P. The Addiction Severity Index – reliability and validity in a Dutch addict population. *J Subst Abuse Treat* 1989;6:133–41.
- [32] Parker JDA, Keefer KV, Taylor GJ, Bagby RM. Latent structure of the alexithymia construct: a taxometric investigation. *Psychol Assess* 2008;20:385–96.
- [33] Leggio L, Kenna GA, Fenton M, Bonenfant E, Swift RM. Typologies of alcohol dependence. From Jellinek to genetics and beyond. *Neuropsychol Rev* 2009;19:115–29.
- [34] de Timary P, Luts A, Hers D, Luminet O. Absolute and relative stability of alexithymia in alcoholic inpatients undergoing alcohol withdrawal: relationship to depression and anxiety. *Psychiatry Res* 2008;157:105–13.
- [35] Parker JDA, Taylor GJ, Bagby RM. The alexithymia construct: relationship with sociodemographic variables and intelligence. *Compr Psychiatry* 1989;1989(30):434–41.
- [36] Schuckit MA, et al. Sex differences in how a low sensitivity to alcohol relates to later heavy drinking. *Drug Alcohol Rev* 2012;31(7):871–80.
- [37] Walters GD. The heritability of alcohol abuse and dependence: a meta analysis of behavior genetic research. *Am J Drug Alcohol Abuse* 2002;28:557–84.
- [38] Campbell JM, Oei TP. A cognitive model for the intergenerational transference of alcohol use behaviour. *Addict Behav* 2010;35:73–83.
- [39] Ham BJ, Lee MS, Lee YM, Kim MK, Choi MJ, Oh KS, et al. Association between the catechol *O*-methyltransferase Val108/158-Met polymorphism and alexithymia. *Neuropsychobiology* 2005;52:151–4.
- [40] Walter NT, Montag C, Markett SA, Reuter M. Interaction effect of functional variants of the BDNF and DRD2/ANKK1 gene is associated with alexithymia in healthy human subjects. *Psychosom Med* 2011;73:23–8.