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# Aetiological Association between Asperger's Traits, Social Anxiety and Social Competences in Developmental Ages: A Multivariate Twin Study

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**Abstract**

Although studies in literature underlined the association between Asperger's Traits (AT), Social Anxiety (SA) and their relationship with low Social Competences (SC) in developmental ages, little is known about the nature of this co variation. In 398 Italian twin pairs aged (8-17), we used a Behavioral Genetics Approach to investigate whether the phenotypic correlation between these three phenotypes could be better explained by direct causal effects, or by common etiological factors acting simultaneously as elements of risk/protection for Asperger's traits, Social Anxiety and (low) Social Competences. We found a significant negative correlation between AT/SA and SC, and a significant positive correlation between AT and SA, consistently in line with previous studies.

Results from causal analysis indicated no direct causation between variables of interest. In multivariate analysis, the co variation between SC and SA was exclusively explained by genetic factors, whereas both genetic and shared environmental components contributed to the phenotypic correlation between SC and AT. Furthermore, the co variation between SA and AT was due to genetic and unique environmental factors common to the traits.

Our results support the adoption of an empirical view in the clinical intervention of Asperger condition in relation to social difficulties across developmental ages.

**Keywords:** Asperger's traits; Social competences; Social anxiety; Twins; Treatment

## Abbreviations

AT: Asperger Traits; SA: Social Anxiety; SC: Social Competences; AS: Asperger's Syndrome; SAD: Social Anxiety Disorder; HFA: High-Functioning Autism; ASD: autism spectrum disorder; BAP: Broad Autism Phenotype; ITR: Italian Twin Registry; MZ: Monozygotic; DZS: same-sex dizygotic; DZU: unlike-sex dizygotic; DZ: Dizygotic; OCD: Obsessive-Compulsive Disorder; CBCL: Child Behaviour Checklist; SCARED: Screen for Child Anxiety Related Disorders; AIC: Akaike Information Criterion

## Introduction

The co-occurrence between Asperger's Syndrome and Social Anxiety Disorder in developmental ages is fairly documented in literature.

Asperger's Syndrome (AS), subtype of autism spectrum disorder, is characterized by "impairments in reciprocal social development and by markedly restricted repertoire of activities and interests" [1-3], without clinically significant impairment in language and cognitive development. Its reported prevalence rates are around 0.3-0.5% and the female to male ratio is 1:4 [4]. Social Anxiety Disorder (SAD) is described as "the fear of humiliation or embarrassment in social and/or performance situations, which may lead to avoidance of social situations, social withdrawal and isolation" [2]. The prevalence of SAD is estimated around 12% and the female to male ratio is 3:2 [5].

The association between Autism Spectrum Disorders (ASD), including Asperger's Syndrome, and anxiety is well documented. Russell and Sofronoff [6] observed that children with AS showed similar levels of anxiety compared to clinically anxious children; in addition, they obtained scores of anxiety significantly higher than normally developing children. On the other hand, a higher rate of autistic traits was reported by adolescents with anxiety disorders [7]. Furthermore, in his review, Mazzone collected several studies reporting associations between Asperger's Syndromes and internalizing symptoms, including anxiety problems, in developmental ages [8].

Concerning specifically Social Anxiety Disorder, literature reports a significant co-occurrence with ASD. Simonoff and co-workers, investigating anxiety disorders in children with ASD, found that SAD was the most comorbid condition, presented in 29% of the sample [9]. Some other studies explored SAD in children and adolescents with a diagnosis of Asperger's Syndrome and High-Functioning Autism (HFA), finding significantly increased social anxiety symptoms in young patients with AS/HFA compared to their peers [1]. Furthermore, recent studies suggested that SAD was associated specifically with higher IQ in youth with ASD [10,11], as in Asperger's Syndrome.




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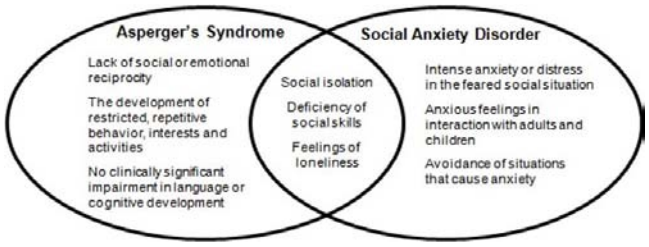


**Figure 1:** Asperger's syndrome and social anxiety disorder.

Little is known about etiological mechanisms underlying the co-occurrence among our phenotypes of interest. The few twin studies presented in literature explored the co variation between anxiety and autism more broadly. Hallett and colleagues [12] examined the possible relationship between autistic traits and internalizing features across middle to late childhood (7-8 years at time-point 1; 12 years at time-point 2). They found a significant but modest genetic correlation between phenotypes at both time-point 1 and time-point 2 (ra=.17), while low estimates of non-shared environmental overlap were observed (time-point 1 re=.08; time-point 2 re=.14). The influence of shared environment was much higher at time-point 1 (rc=1), and became nonsignificant at time-point 2. In addition, by using a Cross Lagged Analysis, Hallett found that autistic traits seemed to have a direct, yet modest, impact on the development of internalizing traits; at the same time, the reverse association was also significant, albeit weaker. Hallett and co-workers [11], in a later study, investigated anxious symptoms in twin children with a diagnosis of ASD. Researchers divided the sample in four categories: 1) ASD children 2) BAP (Broad Autism Phenotype) children, who were primarily co-twins of subjects with autistic spectrum conditions, 3) unaffected co-twins, 4) controls. They found that children with ASD or BAP showed significantly higher anxiety symptoms than controls for all parent-rated anxiety subscales (including SAD), with no significant differences between strict autism and sub-clinical autism. In addition, unaffected co-twins reported significantly increased social anxiety, generalized anxiety and panic conditions compared to controls. For this reason, researchers hypothesized aetiological overlap between ASD and anxiety. Scherff and colleagues (2014) used a twin study design to understand why internalizing traits and autistic features co-occur in teens. They focused on five specific sub domains of autistic traits: attention to details/special interests; social unease; poor mentalizing; solitariness; poor imagination. Under a bivariate model, the genetic correlation between the two phenotypes was modest (ra males=.30; ra females=.12) as was the non-shared environmental correlation (re males=.10; re females=.20), while the shared environmental correlation was higher (rc males=.53; rc females=1).

By a clinical view, one of the main features common to ASD and SAD disorders refers to the presence of difficulties in the social skills domain. Impaired social skills were often reported in children with Social Anxiety Disorder [13,14] and in children with autism spectrum disorder [15], specially Asperger's Syndrome [16]. Bierman and Welsh [17] defined social skills as "social, emotional, cognitive skills and behaviours that children (people) need for successful social adaption", including a broad range of competences, verbal and not. Interpersonal skills represent a key behaviour to develop healthy relationships and social adapting [18]. Nomara Santos [19] investigated a possible relationship between AS and SAD regarding social functioning, finding that both disorders differed significantly from the control group in social domain. A graphical representation of features which are specific to each condition and those which are common to both is reported in (Figure 1) [19]. Starting from social skills impairments in both AS and SAD, Scharfstein and colleagues tried to establish similarities and differences. Children who met criteria for SAD appeared less able to maintain a conversation, with inappropriate responses, inappropriate affect, longer speech latencies, and responses with few words. Children with AS seemed to be more inclined to interact appropriately during brief social interactions, but their skills are insufficient for more extensive or more complex interactions [20]. Thus, socialization deficits play a pivotal role in both conditions, also because social skills deficits do not remit with development [21]. Concerning the nature of the co variation between social skills deficits and AS/SAD, only one twin study explored the association between social competences and anxiety problems. In this study, the best fitting bivariate model showed a significant genetic mediation in the negative correlation between variables (ra=-.56) [22].

Thus, although a number of studies globally underlined the co-occurrence between AS, SAD and social skills deficits (i.e. low social competences), no studies in literature investigated the origins of the relationship among these three phenotypes at the same time. Thus, it seems interesting to further investigate the association between AS, SAD and low social competences. Different mechanisms could explain this co variation. It is possible that AS and SAD cause phenotypically a lack of social competences, or that these variables share common etiological influences. First of all, causal analysis can be applied to investigate the possible direct causation between variables. If this hypothesis is rejected, multivariate twin analyses can be applied to decompose the observed correlation between the three traits into proportions due to additive genes, to familiar environment and to unique environmental influences in an aetiological investigative view [23]. Thus, the purpose of this study is to explore the relationship among Asperger's Traits (AT), Social Anxiety (SA) and (low) Social Competences (SC), and the sources of this co variation, using a sample of children and adolescent twins from general population. To our knowledge, this is the first study to analyse these three phenotypes with a multivariate approach.

By taking advantage of the twin method, we sought to address three main questions:

1) Assessing the size and the direction of the co variation among Asperger's traits, Social Anxiety and Social Competences.

2) Investigating whether the phenotypic correlation between these phenotypes could be better accounted for by a direct causal effect of Asperger's traits or Social Anxiety traits upon Social Competences, or by a latent, "third etiological factor" orchestrating the observed co variation.

3) In absence of clear causal effects, exploring the nature of latent, shared etiological factors that can simultaneously act as elements of risk/protection for AT, SA and SC in the developmental years.

## Methods

### Participants

This study is based on the responses given by parents of twins belonging to the population-based Italian Twin Registry (ITR). Currently, the ITR contains information on approximately 28000 twins, and is involved in both general population- and clinical-based studies on various complex phenotypes, with behavioural and psychiatric genetics as major areas of investigation [24].

Across three different waves, families with twins aged between (8-17), living in the provinces of Milan and Lecco were selected from the ITR database for psychometric studies of various nature and aims [25,26]. The recruitment methods are reported in detail elsewhere [27]. A total of 398 complete twin pairs (mean 13.06±2.59) accepted to take part in this study [25,26] (for differences in socio-demographic characteristics between participants and other families, Supplementary Materials).

Zygosity was assigned by the parent-rated Goldsmith questionnaire [28], which has an accuracy of determination of 99.4% [29]. According to its algorithm, there were 74 Monozygotic (MZ) male, 70 MZ female, 134 same-sex dizygotic (DZS, 53 males, 81 females), and 120 unlike-sex Dizygotic (DZU) twin pairs. Zygosity distribution did not differ from that observed in the larger ITR population, and the MZ/DZS/DZU ratio was 1.1/1.0/0.9. Due to the relatively small sample size, the analyses were not stratified by age and gender. Thus, same- and unlike-sex DZ pairs were grouped into the DZ category and MZ male and female pairs were also combined, for a total of 144 MZ and 254 DZ twin pairs.

The research was accepted by the ethical committee and all the parents signed a consent form for the participants.

### Measures

**Child behavior checklist:** The Child Behaviour Checklist (CBCL) [6-18] [30] is one of the most widely-used research parent instruments to assess child and adolescent skills and behavioural/emotional problems in both epidemiological and clinical samples, in youth ages 6-18. It consists in a standardized questionnaire in which parents rate 118 behavioural and emotional problem items exhibited by their child in the past 6 months. The 118 problem items have been factor-analyzed into eight empirically based syndrome scales. The recent Achenbach System of Empirically Based Assessment also includes six DSM-Oriented Scales (DOS), which aim to cover common childhood mental disorders. To generate the six DOS, 22 clinicians rated the degree of consistency of CBCL items [30] with the corresponding DSM-IV criteria. Furthermore, the questionnaire encompasses three competence scales - Social Competence, Activities Competence and School Competence Scales - and it could be applied to the screening of children with other clinical conditions not included in the DOS, like OCD [31] or suspected ASD [32,33].

Considering the nature of this questionnaire, we chose the autistic scale as a suitable measure to assess Asperger's traits and the Social Competence Scale to measure Social Competences.

CBCL Autistic Scale: Rescorla [32] suggested a subscale, the Autistic Bizarre Scale, as indicative of autism and autism-related conditions in a clinical sample of 204 preschoolers. Specifically, she obtained this sub-scale from factor analysis, which underlined that five CBCL items significantly differentiated the autistic group with mild and severe behavioral and emotional problems from controls. Two successive studies supported the validity of the Autistic Bizarre Scale [34,35] confirming the predictive power of this subscale to discriminate autistic spectrum from other psychiatric conditions. Thus, knowing that AS is characterized by impairments in reciprocal social development, markedly restricted repertoire of activities and interests and absence of cognitive and language delay [2], we decided to choose Rescorla Scale to value our Asperger's traits. The items, in fact, do not refer specifically and distinctly to general significant delay in cognitive development and impairments in language, which are path gnomonic of strict autism or of the severest form of the DSM-5 autistic spectrum condition [36] (for specific items, see Supplementary Materials).

CBCL Social Competence Scale: this scale investigates social skills that refer to social, emotional, cognitive skills and behaviours that children need for successful social adaption (for specific items, see Supplementary Materials).

### Screen for child anxiety related emotional disorders

Screen for Child Anxiety Related Disorders (SCARED) is a child and parent self-report screening instrument for childhood anxiety disorders, based on the DSM-IV [25]. It examines several subtypes of anxiety with 41 items for a target population of 8-18 years. Originally, the SCARED questionnaire was devised to screen anxiety disorders in clinical samples [37], but it is also used as a valuable screening tool in community samples [38].

Children have to rate the frequency with which they experience each symptom. According to the original factorial structure of the questionnaire, the 41 items can be divided into five subscales [37]: 1. Panic Disorder or Significant Somatic Anxiety, 2. Generalized Anxiety Disorder, 3. Separation Anxiety Disorder, 4. Social Anxiety Disorder/Social Phobia, 5. School Phobia. With a cut-off point of 25, the SCARED shows good sensitivity (70%) and accuracy in distinguishing children suffering from anxiety disorders from those who do not (specificity: 67%) [39]. Specific cut-off points are also suggested for the five subscales [37,39] indicating the presence of disorders. We chose the SCARED child self-report/Social Anxiety Disorder subtype as a suitable index to measure Social Anxiety (for specific items, see Supplementary Materials).

### Statistical analyses

**Phenotypic and twin correlations:** Due to the effect of sex and age on Social Anxiety and Social Competences phenotypes, as testified by the regression results (SA: adjusted R2=0.006, F=3.368, p=0.035; SC: adjusted R2=0.031, F=12.866, p<0.001), standardized residual variables were calculated for all phenotypes and then used for our analyses. Then, we computed phenotypic correlation coefficients between AT, SA and SC in the whole sample, considering twins as single individuals. Cross twin-within trait correlations (between the two twins for the same trait) and cross-twin/cross-trait correlations (between one trait in twin 1 and the other trait in twin 2, and vice versa) were also calculated (for details, see Supplementary Materials).

### Causal analyses

To examine the presence of a direct causal effect of AT/SA upon

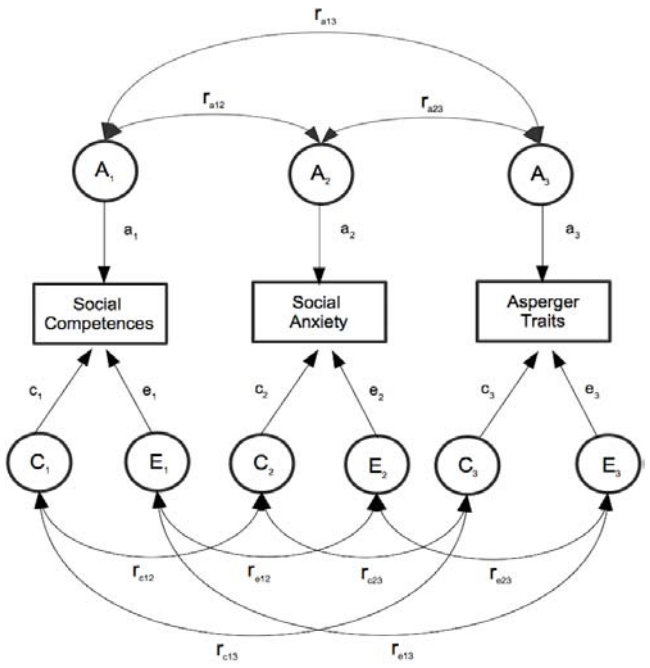


**Figure 2:** Multivariate Model for Social Competences, Social Anxiety and Asperger's traits. Specifically, the figure shows the Correlated Factors Model, the standardized version of the Cholesky Decomposition Model. Observed phenotypes are shown in squares. Latent factors are shown in circles. A1-3, C1-3, E1-3: additive genetic, shared environmental and unique environmental effects on Social Competences, Social Anxiety and Asperger's traits respectively; a1-3, c1-3, e1-3: path coefficients representing the effect of the latent variables on phenotypes; ra, rc, re: genetic, shared environmental, and unique environmental correlations.

SC, we applied the MZ Intrapair Differences Method [40]. By this method, if the two phenotypes were causally linked, the MZ within–pair differences in AT (or in SA) would be significantly associated with the MZ within–pair differences in SC. The presence of a significant regression coefficient would argue in favour of the causal hypothesis, whereas the absence of a significant regression coefficient would deny the hypothesis of a causal effect of AT (or SA) upon SC.

### Model fitting analysis

To test the source of the association between AT, SA and SC we applied a multivariate twin model. Model-fitting analyses were carried out with the Open Mx program, a Structural Equation Modelling package and matrix algebra processor that is flexible to fit a variety of mathematical models [41].

The multivariate design allows for the separation of the total phenotypic variance and covariance of traits into proportions due to additive genetic factors (A), shared environmental factors (C, including socio-economic level, religion, style of parenting, etc.) and finally unique (individual-specific) environmental factors (E, including illness, relationships with peers, etc). The model compares MZ and DZ twin phenotypic resemblances by assuming a correlation between the twins' additive genetic influences of 1.0 for MZ pairs (all genes are shared) and of 0.5 for DZ pairs (DZ twins share half of their segregating genes on average), and a correlation between the twins' shared environmental influences of 1.0 for both MZ and DZ twin pairs (i.e. shared environmental influences are assumed to be

**Table 1:** Phenotypic and within-trait correlations among variables (**p<0.01).

| | Social | | |
|---|---|---|---|
| Competences | Social | | |
| Anxiety | Asperger | | |
| traits | | | |
| Phenotypic Correlations | | | |
| Social Competences | 1 | - | - |
| Social Anxiety | -.17** | 1 | - |
| Asperger traits | -.28** | .18** | 1 |
| Within-Trait Correlations | | | |
| MZ | 0.71 | 0.55 | 0.58 |
| DZ | 0.66 | 0.29 | 0.25 |

of equal magnitude for MZ and DZ twins, as endorsed by the 'Equal Environments Assumption'; [42]).

The Cholesky Decomposition model assumes three distinct sets of genetic and environmental (shared and unique) influences on each variable. For n variables, a Cholesky decomposition includes n independent genetic and environmental set factors; the first factor loads on all traits, the second one loads on all traits except the first, the third factor loads on all traits except the first two and so on. This model provides the fullest explanation of data because it does not impose any restrictions on the genetic and environmental contributions to covariation. The Correlated Factors model is the standardized version of the Cholesky Decomposition [43]. It assumes that genetic, shared environmental and unique environmental factors related to a phenotype are correlated to the factors related to the other phenotypes. The genetic (ra), shared environmental (rc) and unique environmental (re) factors correlations are indicators of the extent to which genetic or environmental influences on two traits in the model overlap [43]. Bivariate heritability represents the percentage of the phenotypic correlation between two traits that can be explained by shared genetic effects. The same reasoning and computing applies to environmental influences.

In this study, a Cholesky Decomposition Model (and its corresponding Correlated Factors Solution) was used to examine the genetic and environmental relationship between AT, SA and SC, with the SC scores entered as the first variable (Figure 2). Our analyses began with a full ACE model and, by stepwise deletion of variance and covariance components, preceded with more parsimonious models to search for the best-fitting solution. The chi-square ($\chi^2$) difference test and Akaike information Criterion (AIC=$\chi^2$-2df; [44]) were used to inform model fit when exploring more parsimonious models, where the lowest AIC value reflects a balance between goodness of fit and parsimony.

## Results

### Phenotypic and twin correlations

Table 1 shows phenotypic correlations between AT/SC, SA/SC and AT/SA. Specifically, we found significant negative correlations between AT and SC (r=-.28, p<0.01) and SA and SC (r=-.17, p<0.01), while we observed a significant positive correlation between AT and SA (r=.18, p<0.01). Table 1 also shows the cross-twin/within-trait correlations for all three phenotypes; the correlations were higher in

**Table 2:** Cross-twin cross-trait correlations among variables.

| | MZ | DZ |
|---|---|---|
| Cross-Twin Cross-Trait Correlations | | |
| Social Competences/Social Anxiety | -0.15 | -0.08 |
| Social Competences/Asperger traits | -0.28 | -0.24 |
| Asperger traits/Social Anxiety | 0.14 | 0.11 |

**Table 3:** MZ Twin intrapair differences model.

| Dependent Variable | Independent Variable | β | t | p | CIs |
|---|---|---|---|---|---|
| Social Competences | Social Anxiety | 0.103 | 1.136 | 0.258 | -.03 to .11 |
| Social Competences | Asperger traits | 0.022 | 0.235 | 0.814 | -.31 to .39 |

MZ than in DZ pairs, suggesting that AT, SA and SC are influenced by genetic factors to different extents. Table 2 shows the cross-twin/cross-trait correlations, which included negative correlations between SC/SA and SC/AT, and positive correlation among AT and SA. Results suggest that genes seem to play a role in explaining the covariance between SC and SA, while both genetic and environmental influences participate to the covariance between SC/AT and SA/AT.

## Causal analyses

Table 3 shows the results of MZ intrapair differences analysis. Regression coefficients of the intrapair difference scores in AT/SA on the intrapair difference scores in SC were non-significant, and this was not compatible with a direct causal effect.

## Model fitting analyses

First of all, we implemented a saturated model suggesting the absence of a significant effect of twin order and Zygosity on means and variances.

Table 4 shows the results of the model fitting analyses, which started with a full ACE model. The Cholesky model (and its corresponding Correlated Factors Solution) was used as the base model, without testing more sophisticated models (i.e. Independent Pathways Model or Common Pathways Model) because of the limited power conveyed by our sample.

Three more parsimonious models were then implemented: 1. an AE model (Model 2), meaning that all phenotypic variance and covariance among variables could be explained by additive genetic and unique environmental factors; 2. A CE model (Model 3), which assumes only shared and unique environmental factors; 3. An E model (Model 4), which assumes that idiosyncratic environmental effects are sufficient to explain variance and covariance of variables. The deterioration of fit indices clearly showed the inadequacy of AE, CE and E models, while the ACE model provided the best fit indices. Thus, successive modelling was based upon the ACE model (Model 1).

Then we proceeded with further refinements by two criteria. First, we tried to drop estimates which included a zero value in their confidence intervals (rc12 and rc23). Such correlations were dropped without a significant fit deterioration (Model 5 and 6). Secondly, we tried to drop very low correlations (re12=.08 and re13=-.04) that, moreover, explained a tiny portion of the total phenotypic correlation. Again, we dropped both correlations without a significant worsening of the model fit (Model 7 and 8). Finally, we tried to drop the remaining correlations (i.e. ra12, rc13, re23) and in all cases, we observed a significant deterioration of the model fit (Model 9).

Figure 3 is a graphical representation of the best final model explaining variance and covariance among variables by Correlated Factors Solution. Firstly, the model shows the univariate estimates of genetic, shared environmental and unique environmental contributions for each phenotype. Furthermore, the model assumes genetic factors as the only source of co variation between SC and SA, genetic and unique environmental factors as sources of co variation between SA and AT, and genetic and common environmental factors as sources of co variation between SC and AT. Specifically, the nature of the co variation between SC and SA seems to be exclusively genetic, with a 53% genetic overlap, explaining 100% of the covariance. Regarding the co variation between SC and AT, genetic and common environmental factors seem to play an important role. In fact, the traits share 33% of their genes, accounting for 48% of the covariance, and 89% of the common environmental factors, explaining the remaining 52% of the covariance. Finally, concerning the co variation between SA and AT, it seems to be accounted by genetic and unique environmental shared factors. The genetic and unique environmental overlaps are respectively 23% (corresponding to 69% of the co variation) and 13% (corresponding to 31% of the co variation). All

**Table 4:** Model-fitting statistics.

| Model | compared to model | -2LL | df | AIC | diff.LL | diff.df | p |
|---|---|---|---|---|---|---|---|
| 1. Full ACE model | - | 6066.5 | 2273 | 1520.5 | - | - | - |
| 2. AE model | 1 | 6095.78 | 2279 | 1537.78 | 29.27 | 6 | 0 |
| 3. CE model | 1 | 6109.06 | 2279 | 1551.6 | 43.09 | 6 | 0 |
| 4. E model | 2 | 6407.27 | 2285 | 1837.27 | 311.5 | 6 | 0 |
| 5. Model 1 + drop rc12 | 1 | 6066.56 | 2274 | 1518.56 | 0.06 | 1 | 0.81 |
| 6. Model 5 + drop rc23 | 5 | 6067.1 | 2275 | 1517.1 | 0.54 | 1 | 0.46 |
| 7. Model 6 + drop re13 | 6 | 6067.24 | 2276 | 1515.24 | 0.14 | 1 | 0.71 |
| 8. Model 7 + drop re12 | 7 | 6067.93 | 2277 | 1513.93 | 0.69 | 1 | 0.41 |
| 9. Model 8 + drop ra12 | 8 | 6090.19 | 2278 | 1534.19 | 22.26 | 1 | 0 |

A: Additive genetic factors; C: shared environmental factors; E: unique environmental factors; AIC: Akaike Information Criterion; df: degrees of freedom; diff.df: difference in degrees of freedom; diff.LL: difference in log likelihood statistic; -2LL: -2 log likelihood statistic; ra, rc, re: genetic, shared environmental, and unique environmental correlations. Boldface type indicates the best fitting model.

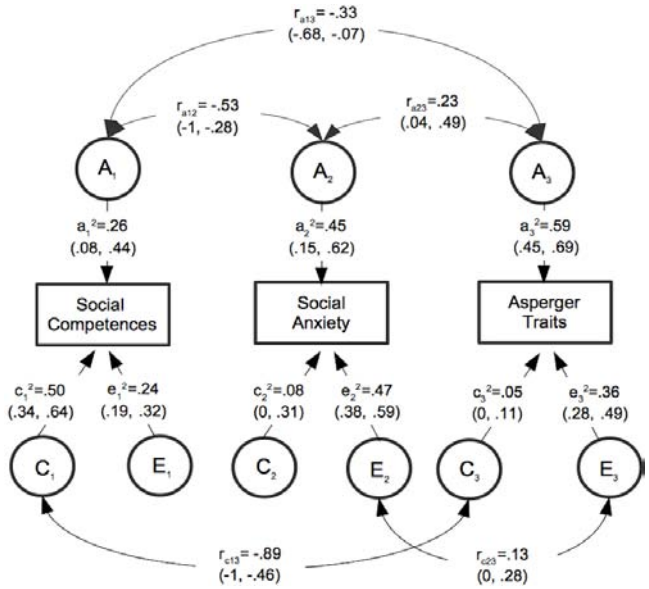


**Figure 3:** Best-fitting Correlated Factors solution on Social Competences, Social Anxiety and Asperger's traits.
A, C, E: additive genetic, shared environmental and unique environmental influences on phenotype; a2, c2, e2 standardized variance estimates; ra, rc, re: genetic, shared environmental, and unique environmental correlations. 95% Confidence Intervals in parentheses.

results are summarized in Table 5.

## Discussion

To our knowledge, this represents the first study to examine the nature of the association between AT, SA and SC, in a sample of twin children and adolescents from general population, aiming to: (1) assess the size and the direction of the co variation between AT, SA and SC; (2) evaluate possible causality; (3) explore, by a multivariate twin approach, the nature of latent, shared etiological factors that could simultaneously act as elements of risk/protection for these phenotypes.

Firstly, our data showed a moderate negative correlation between AT/SA and SC, suggesting that as these psychopathological traits increased Social Competences decreased, in line with previous studies on the deficiency of social skills in Asperger condition [16] and in Social Anxiety [13] in developmental ages. Instead, a significant positive correlation was found between AT and SA, demonstrating the direct relationship between these variables as described in literature [1,18]. Regarding causal analyses, data showed that direct causation seemed not to be the main explanation for the correlation among variables. In fact, using the MZ twin Intrapair differences method [40], neither Asperger's traits nor Social Anxiety had an effect on (low) Social Competences. Thus, the impairment of social skills seems to be not directly caused by these two conditions.

With respect to multivariate twin modelling, we observed three main results. Firstly, regarding the co variation between SC and SA, shared genes seemed to play a distinct role, as more than half of involved genes were suggested to be common to the two phenotypes. The genetic correlation was negative, meaning that the same genes led to higher Social Anxiety and lower Social Competences (and vice versa). Furthermore, these shared genes seemed to fully explain the covariance between the traits. These findings appeared in line with a previous study conducted by Pesenti-Gritti et al. [22], where shared genetic factors which influenced simultaneously anxiety problems and social competences in developmental age could be considered the best explanation for the phenotypic covariation of these phenotypes. However, our results do not exclude a role of environmental influences in SC as single phenotype, and thus SA may be a secondary effect of the impairment of social skills due to negative environmental experiences (such as parenting style, negative experiences with peers, traumatic social events, victimization, etc).

Secondly, concerning the co variation between SC and AT, genetic and shared environmental factors seemed to play an important role. Both influences were negative, meaning that genetic and shared

**Table 5:** Genetic and environmental variance-covariance components and correlations standardized components.

| | A | C | E |
|---|---|---|---|
| Vp (Social Competences) | 0.26 (0.08;0.44) | 0.50 (0.34;0.64) | 0.24 (0.19;0.32) |
| Vp (Social Anxiety) | 0.45 (0.15;0.62) | 0.08 (0;0.31) | 0.47 (0.38;0.59) |
| Vp (Asperger traits) | 0.59 (0.45;0.69) | 0.05 (0;0.11) | 0.36 (0.28;0.49) |
| Cov (Social Competences - Social Anxiety) | 1 | - | - |
| Cov (Social Competences - Asperger traits) | 0.48 | 0.52 | - |
| Cov (Social Anxiety - Asperger traits) | 0.69 | - | 0.31 |
| | Correlations | | |
| | ra | rc | re |
| Social Competences - Social Anxiety | -0.53 (-1;-0.28) | - | - |
| Social Competences - Asperger traits | -0.33 (-0.68;-0.07) | -0.89 (-1;-0.46) | - |
| Social Anxiety - Asperger traits | 0.23 (0.04;0.49) | - | 0.13 (0;0.28) |

A, C, E: additive genetic, shared environmental and unique environmental influences on phenotype; Vp (Social Competences): phenotypic variance of Social Competences; Vp (Social Anxiety): phenotypic variance of Social Anxiety; Vp (Asperger traits): phenotypic variance of Asperger's traits; ra, rc, re, genetic, shared environmental, and unique environmental correlations. By dividing the bivariate heritability by the phenotypic correlation, it is possible to obtain the proportion of genetic covariance between two traits, and the same procedure can be applied to environmental covariance: Cov (Social Competences - Social Anxiety): covariance between Social Competences and Social Anxiety; Cov (Social Competences – Asperger traits): covariance between Social Competences and Asperger's traits; Cov (Social Anxiety - Asperger traits): covariance between Social Anxiety and Asperger's traits.

environmental factors common to the traits led to increase a trait and to decrease the other (and vice versa). To our knowledge, this is the first investigation of the aetiological nature of the co variation between SC and AT, although a social skills deficit in Asperger's Syndrome is well known from literature [16]. From a clinical point of view, our results may indicate that, even though shared environmental influences (i.e. parenting style or common family experiences) do not have a major role in explaining the aetiology of AT (as shown by our univariate results and previous literature, e.g. [45]), they do in the co variation of AT with SC. In fact, our results show a sizable degree of shared environmental mediation in the co variation of these phenotypes, and so we may hypothesize that a lack of a social parenting style may increase impairments of Social Competences in children or adolescents with Asperger's traits. On the other hand, a better parenting style favoring the development of social skills may protect from more severe deficits in Social Competences in children with Asperger's traits. Therefore, in addition to the SET-C (Social Effectiveness Treatment – Children; [46]), a precise and intense type of Social Skills Training tailored to Asperger's Syndrome [47], a parent training could be suitable for the treatment of social skills deficits in AS, improving social competences.

Thirdly, regarding the positive co variation between SA and AT, it seemed to be accounted by genetic and unique environmental factors common to the traits. Both aetiological correlations were positive, meaning that the same genes and unique environmental influences led at the same time to higher Social Anxiety and Asperger's traits. Our study is in line with Hallett et al. [12], who considered the association between autistic-like and internalizing traits during developmental ages, finding a strong association between shared environmental influences on these two traits at ages [7-8], which decreased drastically at age 12. Also in our sample (mean age 13), we found a negligible shared environmental influence. This result is in contrast with Scherff et al. [48], where shared environmental correlations between autistic features (especially "social unease sub domain") and internalizing traits were significant at ages [12-14]. Actually, it should be specified that both these studies considered ASDs and internalizing traits, not specifically Asperger's traits and Social Anxiety, but in samples without cognitive impairments. Thus, the lack of cognitive delay in ASDs' children could be considered like Asperger condition by definition, as in Hallett and co-workers' study, where internalizing-like behaviour was associated with higher IQ and increased social and communication deficits in ASD children. In addition, to measure internalizing traits, Hallett [12] chose the emotional problems subscale of the Strengths and Difficulties Questionnaire (SDQ), whose sensitivity to predict social phobia was estimated around 72.7% [49]. So, we could suppose that SA could be included in the definition of internalizing traits by Hallett. Anyway, our data are in line with both Hallett et al. [12] and Scherff et al. [48] who also found genetic and non-shared environmental correlations between the traits. In the light of these results, we may suppose different aetiological influences at different developmental stages; for instance, shared environmental factors seem to be specific to younger age, while genetic and unique environmental factors could play a significant role later in life. The observation that shared environmental influences decrease with age agrees with literature data in Behavioural Genetics [50].

Therefore, given 1) the large etiological overlap between Social Anxiety and Social Competences, 2) the role of shared environmental influences in mediating the association between Asperger's traits and low Social Competences, and 3) the different shared environmental role in mediating the association between AT and SA at different developmental ages (significant in childhood, negligible during adolescence, [12]), it seems possible to turn these empirical results into a clinical strategy for the treatment of social domain difficulties in Asperger condition.

Specifically, in a first phase of development, when also shared environmental factors seem to play an important role in the co variation between SC and AT, the treatment of Asperger condition should include a familiar environment intervention. In this context, a parent training, including an improvement of social parenting style could have a central role in influencing children's social and communication styles. With growth, when genetic and unique environmental factors seem to be the main determinants of the relationship between Social Anxiety and Asperger's traits, the SET-C could be a suitable and efficient treatment [47].

Our findings must be interpreted in the context of some potential limitations; however, the same limitations could be useful guidelines for future studies. Firstly, the sample size is relatively small, yielding limited power to detect certain sources of variance and covariance, particularly shared environment (a typical weakness of the classical twin study). Furthermore, the sample size did not allow us to properly address important issues such as age and sex differences in the genetic and environmental effects, which have been previously detected for various behavioral problems [51]. Secondly, our study did not explore the possible causal effect of Asperger's Syndrome on Social Anxiety Disorder. Children with Asperger's Syndrome were found to be socially motivated and seemed to desire interpersonal relationships, but the awareness of their own social deficits, combined with negative feedbacks or rejections from others, could make them socially anxious [52]. Further studies would be needed to evaluate this hypothesis. A possible third limitation is that our study focused on a general population sample, and it could be argued that the present results may not necessarily apply to individuals with extreme or clinical symptoms scores. Finally, this is a cross-sectional design, and it should be acknowledged that a longitudinal study would be more adequate to investigate the causal relationship between competences and psychopathological symptoms, and their association over time.

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## Conclusion

Results indicated that direct causation is not the main explanation for the co variation between AT/SA and low SC. The significant phenotypic correlations among variables could be driven by etiological overlaps, which could lead to new clinical suggestions.

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