WHY DO PEOPLE DIFFER IN THEIR ACHIEVEMENT MOTIVATION? A NUCLEAR TWIN FAMILY STUDY

Although many previous studies have emphasized the role of environmental factors, such as parental home and school environment, on achievement motivation, classical twin studies suggest that both additive genetic influences and non-shared environmental influences explain interindividual differences in achievement motivation. By applying a Nuclear Twin Family Design on the data of the German nationally representative of TwinLife study, we analyzed genetic and environmental influences on achievement motivation in adolescents and young adults. As expected, the results provided evidence for the impact of additive genetic variation, non-additive genetic influences, as well as twin specific shared environmental influences. The largest amount of variance was attributed to non-shared environmental influences, showing the importance of individual experiences in forming differences in achievement motivation. Overall, we suggest a revision of models and theories that explain variation in achievement motivation by differences in familial socialization only.

Key words: achievement motivation, behavioral genetics, Nuclear Twin Family Design

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Introduction

Motivation gives to the people’s behavior direction, intensity and persistence (Spinath, Toussaint, Spengler, & Spinath, 2008). Achievement motivation is an important key qualification in a modern society due to its central role for learning and career success, as well as for lifelong learning in general (Looser, 2011; Röhr-Sendlmeier, & Kröger, 2014). It has been defined as the striving to increase or to keep, as high as possible, one’s own capabilities in all activities in which a standard of excellence is thought to apply, and where the execution of such activities can therefore either succeed or fail (Heckhausen, 1967). Due to its high relevance, personality research has been examining the factors that influence the development of individual differences in achievement motivation (Heckhausen & Heckhausen, 2010).

On the one hand, achievement motivation has been investigated from a behavioral genetic perspective, and it has been found to be genetically influenced (e.g., Spinath, 2001; Spinath et al., 2008). On the other hand, most theories and models that attempt to explain differences in the motivation to perform focus on education and socialization, emphasizing the role of school and parental home for the development of individual differences in achievement motivation (Deci & Ryan, 2004; Heckhausen & Heckhausen, 2010; Wigfield & Eccles, 2000).

Influences of Social Contexts

Previous studies have shown a connection between children’s achievement motivation and home, as well as parental factors (Mansour & Martin, 2006). The intellectual and performance-related stimulating value in the parental home, as well as a connection with the parental performance pressure, were shown to be correlated with the achievement motivation of children (Heckhausen & Heckhausen, 2010). Additionally, cross-sectional and longitudinal correlations between achievement motivation and children’s social integration in their family were found (Looser, 2011; Looser, 2017): Specifically, achievement motivation in adolescence correlated with perceived quality of the parent-child relationship, a consistent parenting style, an authoritative educational style in the parent-child interaction, the perceived well-being at home, and the parent-child intensity of conversation. Negative correlations were found between achievement motivation and frequent conflicts at home, and an inconsistent parenting style.

Furthermore, factors of the school setting correlated with different levels of achievement motivation: Positive correlations were found between achievement motivation and the teacher-student relationship, the feeling of well-being at school, the perception of the teacher’s appreciation, emotional affection and attribution concerning aptitudes, the feeling of competence within the class and the recognition by classmates. Negative correlations were found between achievement motivation and school norm violations (Looser, 2011). Additional
supporting factors in the school context were education aimed at the interests and lifestyles of students, an appreciative teacher-student-relationship, an educational leadership style of schools, and a combination of high performance-related expectations and positive social relationships in general (Looser, 2017; Wigfield, Eccles, Schiefele, Roeser, & Davis-Kean, 2006). An importance of the school setting for the development of achievement motivation was emphasized by studies showing correlations between teachers’ reference orientation and students’ motivation (Heckhausen & Heckhausen, 2010; Schlag, 2013).

Lastly, the development of individual differences in achievement motivation is affected by leisure activities and peer relationships (Nelson & DeBacker, 2008; Heckhausen & Heckhausen, 2010) and correlates with socio-cultural backgrounds (Röhr-Sendlmeier, Jöris, & Pache, 2012). All in all, individual differences in achievement motivation seem to be explainable partly by influences of the parental home, school, gender, school type, peer-relationships, and socio-economic background, as shown by previous research.

**Behavioral Genetic Studies**

Next to studies considering only environmental influences on achievement motivation, Röhr-Sendlmeier and Kröger (2014), as well as Bergold, Röhr-Sendlmeier, Heuser, Bieling and Burdorf (2014), have found significant correlations between parents’ and adolescent children’s achievement motivation. These family correlations may point to learning influences and/or genetic transmission from parents to the offspring. Behavioral genetic studies allow distinguishing both pathways of intergenerational similarity.

The most frequently used research design in behavioral genetics is the Classical Twin Design (CTD; see Knopik, Neiderhiser, DeFries, & Plomin, 2017). The CTD compares the covariance of MZ twins who share 100% of their segregating genes with the covariance of DZ twins sharing 50% of their segregating genes. Structural equation modeling of these covariance matrices allows estimation of additive genetic influences, the net effect of both non-additive and shared environmental influences, as well as non-shared environmental effects on individual differences.

Spinath (2001) used the CTD on achievement motivation in a sample of German adult twins reared together. Additive genetic influences were found to explain 41% of the phenotypic variance, while the remaining variance could be explained by non-shared environmental effects. Kovas et al. (2015) used data from over 13,000 twins aged 9 to 16, from six different twin studies in six different countries. Almost identical to the results of Spinath, they found that about 40% of the variance could be attributed to genetic factors and non-shared environmental influences (60%). These findings clearly pointed out a moderate genetic influence on motivational personality traits, such as achievement motivation, and thus they were not in line with current popular motivation theories which explained individual differences in achievement motivation by environmental factors, such
as factors of the parental home and school environment only (Deci & Ryan, 2004; Heckhausen & Heckhausen, 2010; Wigfield & Eccles, 2000).

There is a seeming inconsistency in the results of family studies, which report correlations between characteristics of the family and genetically informative that do not indicate an effect of the environment shared by family members. A possible explanation is that variables such as the parent-child-relationship, parenting behavior and school variables, although usually regarded as examples of shared environments, affect siblings in the same family differently. Thus, the effects of these variables are correctly identified as non-shared (Bleidorn et al., 2018; Knopik et al., 2017).

In addition, the CTD is not optimally suited for investigating these influences, because it is based on strict assumptions which need to be met in order to obtain accurate estimates (Keller, Medland, & Duncan, 2010). The CTD tries to estimate three or four parameters by using MZ twin and DZ twin variance-covariance matrices: Additive genetic influences ($a^2$), non-additive genetic influences ($i^2$), and shared environmental influences ($c^2$), which are mutually confounded in the CTD and only two of these parameters can be estimated. Since parameters of non-additive genetic influences cannot be estimated in the presence of additive genetic effects and shared environmental effects, either $i^2$ or $c^2$ are fixed to 0. If the assumption is violated, parameters for additive genetic effects are overestimated, and parameters for non-additive genetic effects and shared environmental effects are be underestimated (Kandler & Papendick, 2017).

Another assumption of the CTD is that assortative mating does not have an effect on the examined trait (Keller et al., 2010). Assortative mating describes the fact that people choose their partners according to their own genetically influenced characteristics. If this is the case, the parents of twins are more similar to each other than it would be expected under random mating, which would raise the genetic relatedness of DZ twins, but obviously not the perfect genetic correlation of MZ twins as well. Consequently, no considering assortative mating results in overestimating shared environmental influences and underestimating genetic influences (Knopik et al., 2017).

Furthermore, genetic and environmental influences are rarely independent of each other. However, the CTD relies on the assumption that gene-environment correlation and interaction have no influence on the trait under study (Keller et al., 2010). If this assumption is violated though, it would also result in biased parameter estimates. Moreover, the CTD does not provide detailed information about the origin of shared environmental effects (Bleidorn et al., 2018).

Many of these shortcomings can be overcome, if data from additional family members are available. In the current study, the Nuclear Twin Family Design (NTFD) was used. Data of parents and available siblings of the twins were collected in addition to data of MZ and DZ twins reared together. These additional measurements increased statistical power and allowed estimation of more parameters that are less biased (Bleidorn et al., 2018; Keller et al., 2010).
Method

Sample

This study uses the data of the TwinLife study, a longitudinal twin family study that examined more than 4,000 same-sex twin pairs and their family representative for twin families in Germany (Hahn et al., 2016). The first wave of data collection took place between 2014 and 2015 (Brix et al., 2017). The twins and their families were grouped in four age cohorts. This analysis used the data from the two oldest twin cohorts (C17; age 17 and C23; age 23). The data were collected by means of interviews in the participants’ homes. Table 1 provides an overview of sample sizes and age distributions.

Table 1
Age distribution of sample

<table>
<thead>
<tr>
<th></th>
<th>M</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>C17</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MZ</td>
<td>17.01</td>
<td>16-18</td>
</tr>
<tr>
<td>DZ</td>
<td>17.02</td>
<td>16-18</td>
</tr>
<tr>
<td>Siblings</td>
<td>18.65</td>
<td>5-44</td>
</tr>
<tr>
<td>Mothers</td>
<td>47.74</td>
<td>34-63</td>
</tr>
<tr>
<td>Fathers</td>
<td>50.53</td>
<td>34-73</td>
</tr>
<tr>
<td>C23</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MZ</td>
<td>23.06</td>
<td>21-25</td>
</tr>
<tr>
<td>DZ</td>
<td>23.03</td>
<td>21-25</td>
</tr>
<tr>
<td>Siblings</td>
<td>24.82</td>
<td>7-50</td>
</tr>
<tr>
<td>Mothers</td>
<td>52.59</td>
<td>41-69</td>
</tr>
<tr>
<td>Fathers</td>
<td>55.25</td>
<td>42-79</td>
</tr>
</tbody>
</table>

Note. C17 - younger cohort, C23 - older cohort, MZ - monozygotic twins, DZ - dizygotic twins, M - mean.

Measurement

Zygosity. The zygosity of the twins was determined by using a self-report zygosity questionnaire (Oniszczenko, Angleitner, Strelau, & Angert, 1993). This questionnaire consisted of three parts: Items to determine the similarity of the external appearance of the twins, items to determine the frequency with which the twins were confused by others, and items to assess the zygosity of the twins by the parents. The results of the zygotic questionnaire were validated and corrected by using genetic fingerprinting (Hahn et al., 2016).
Achievement Motivation. Achievement motivation was measured for over 16-year olds by two items (Good achievements mean a lot to me and In order to get ahead in life, I am prepared to put in great efforts), and a sum score was built. Table 2 shows the descriptive statistics and reliability estimates (Cronbach’s α) for different sample groups. Scores have been corrected for linear age and gender differences, as these can distort parameter estimates (McGue & Bouchard, 1984).

Table 2
Descriptive statistics for achievement motivation

<table>
<thead>
<tr>
<th></th>
<th>N</th>
<th>M</th>
<th>SD</th>
<th>α</th>
</tr>
</thead>
<tbody>
<tr>
<td>MZ (T1)</td>
<td>1015</td>
<td>8.38</td>
<td>1.32</td>
<td>.59</td>
</tr>
<tr>
<td>MZ (T2)</td>
<td>1020</td>
<td>8.34</td>
<td>1.43</td>
<td>.66</td>
</tr>
<tr>
<td>DZ (T1)</td>
<td>1019</td>
<td>8.30</td>
<td>1.34</td>
<td>.57</td>
</tr>
<tr>
<td>DZ (T2)</td>
<td>1016</td>
<td>8.18</td>
<td>1.48</td>
<td>.65</td>
</tr>
<tr>
<td>Siblings</td>
<td>633</td>
<td>8.18</td>
<td>1.41</td>
<td>.65</td>
</tr>
<tr>
<td>Mothers</td>
<td>1898</td>
<td>8.03</td>
<td>1.37</td>
<td>.64</td>
</tr>
<tr>
<td>Fathers</td>
<td>1159</td>
<td>8.15</td>
<td>1.39</td>
<td>.73</td>
</tr>
</tbody>
</table>

Note. MZ - monozygotic twins, DZ - dizygotic twins, T1 - first-born twin, T2 - second-born twin, M - mean, SD - standard deviation; α - Cronbach’s alpha.

Analyses

The NTFD model was fitted to the data with AMOS Version 24 (Arbuckle, 2014) by using the full information maximum likelihood algorithm. Since the NTFD included the data of the twins, full siblings, and biological parents, the design allowed the decomposition of the variance in achievement motivation into various genetic and environmental components. The model is depicted in Figure 1.
Figure 1. Nuclear Twin Family Model for monozygotic twins (the upper figure) and dizygotic twins (the lower figure). $a$ - additive genetic effects; $e$ - non-shared environmental effects incl. measurement error, $i$ – epistasis-effects, $f$ - environmental transmission from fathers to offspring, $m$ - environmental transmission from mothers to offspring, $s$ - shared environmental effects between siblings, $t$ - shared environmental effects between siblings, $\mu$ - phenotypic correlation of parents.

The NTFD model specified additive genetic effects ($a^2$), non-additive genetic effects ($i^2$; epistasis), non-shared environmental effects confounded with measure-
ment error ($e^2$), and shared environmental effects that were further partitioned:
The inclusion of a non-twin sibling allowed to separate environmental influences
shared among all children in a family ($cs^2$) from environmental influences that
were exclusively shared by the twins ($ct^2$). By including data of the mothers and
fathers of the twins, parameters for parental environmental transmission from
a mother to offspring ($m^2$), parental environmental transmission from a father
to offspring ($f^2$), and from both parents to offspring ($m^2 + f^2 + 2mf\mu$) could be
estimated, while considering the influence of the correlation between the parents
(i.e. assortative mating, $\mu$). In addition, the model we applied allowed us to esti-
mate the influence of passive gene-environment-correlation ($a^2m[1+\mu]+a^2f[1 +
\mu]$; Bleidorn et al., 2018). Passive gene-environment-interaction occurred when
parents created environmental conditions that matched the child’s genetic pre-
disposition due to genetic correspondence with the child (Knopik et al., 2017).

In this NTFD model, non-additive genetic effects and environmental effects
shared by all children of the families could not be estimated in the presence of
each other (Kandler, Gottschling, & Spinath, 2016). We chose the model $cs = 0$ as a
baseline model. This model allowed the estimation of non-additive genetic effects
instead of sibling-specific environmental effects, and it was chosen because the
correlations provided indication for non-additive genetic influences (see Table 4,
a model with $i = 0$, yielded a poorer fit and resulted in a parameter estimate of $cs =
0$). We reduced the baseline model by testing whether a model fixing $m = 0$ and
$f = 0$ parameters, and an even more parsimonious model ($m=f=ct=0$), resulted in
significantly poorer model fit without any effects of the environment shared by
family members. For nested model comparisons, we used the $\chi^2$-difference test.
Further goodness-of-fit indices which were considered, were the comparative fit
index (CFI; Bentler, 1990), where values close to 1 indicated a good fit, the root
mean square of approximation (RMSEA; Browne & Cudeck, 1992), where values
close to 0 indicated a good fit and the Akaike information criterion (AIC; Akaike,
1969, 1970), where smaller values indicated a better fit.

Results

Family Correlations

Correlations of different family-dyads are shown in Table 3. The correlation
for the MZ twins was more than twice as high as the correlation for the DZ twins.
Moreover, the correlation of the MZ twins was substantially higher than in all the
other family dyads. This indicated that both additive and non-additive genetic in-
fluences might play a role in explaining individual differences in achievement mo-
tivation. The average parent-child and twin-sibling correlations were lower than
the correlation of the MZ twins, which indicated relevant environmental influ-
ences on differences in achievement motivation, which were shared only by the
twins, and not with siblings or parents. The correlations between a mother and a father were not significant, and therefore they provided no evidence for assortative mating.

Table 3
Achievement motivation correlations between dyads of twin family members (for z-standardized residuals/corrected for age and sex differences)

<table>
<thead>
<tr>
<th>Dyads</th>
<th>N</th>
<th>r</th>
<th>95% C.I.</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>MZ T1 and MZ T2</td>
<td>1013</td>
<td>.37</td>
<td>[.32 - .43]</td>
<td>.00</td>
</tr>
<tr>
<td>DZ T1 and DZ T2</td>
<td>1015</td>
<td>.18</td>
<td>[.11 - .25]</td>
<td>.00</td>
</tr>
<tr>
<td>Sibling and T1</td>
<td>632</td>
<td>.14</td>
<td>[.06 - .22]</td>
<td>.00</td>
</tr>
<tr>
<td>Sibling and T2</td>
<td>631</td>
<td>.11</td>
<td>[.04 - .18]</td>
<td>.00</td>
</tr>
<tr>
<td>Mother and T1</td>
<td>1893</td>
<td>.08</td>
<td>[.03 - .12]</td>
<td>.00</td>
</tr>
<tr>
<td>Mother and T2</td>
<td>1893</td>
<td>.10</td>
<td>[.05 - .15]</td>
<td>.00</td>
</tr>
<tr>
<td>Mother and sibling</td>
<td>598</td>
<td>.01</td>
<td>[.06 - .09]</td>
<td>.79</td>
</tr>
<tr>
<td>Father and T1</td>
<td>1152</td>
<td>.04</td>
<td>[.02 - .10]</td>
<td>.16</td>
</tr>
<tr>
<td>Father and T2</td>
<td>1151</td>
<td>.05</td>
<td>[.00 - .10]</td>
<td>.08</td>
</tr>
<tr>
<td>Father and sibling</td>
<td>387</td>
<td>.14</td>
<td>[.03 - .24]</td>
<td>.00</td>
</tr>
<tr>
<td>Father and mother</td>
<td>1025</td>
<td>.03</td>
<td>[.03 - .09]</td>
<td>.32</td>
</tr>
</tbody>
</table>


Results of the Nuclear Twin Family Model

Fit indices for the NTFD are given in Table 4, and results of the model comparisons can be found in Table 5.

Table 4
Nuclear Twin Family Design: Model comparison tests and fit-statistics

<table>
<thead>
<tr>
<th>Model</th>
<th>$\chi^2$</th>
<th>df</th>
<th>p</th>
<th>CFI</th>
<th>RMSEA</th>
<th>AIC</th>
</tr>
</thead>
<tbody>
<tr>
<td>$cs = 0$</td>
<td>28.98</td>
<td>23</td>
<td>.18</td>
<td>.97</td>
<td>.011</td>
<td>62.98</td>
</tr>
<tr>
<td>$cs = 0 \ m = 0 \ f = 0$</td>
<td>32.12</td>
<td>24</td>
<td>.15</td>
<td>.97</td>
<td>.012</td>
<td>62.12</td>
</tr>
<tr>
<td>$cs = 0 \ m = 0 \ f = 0 \ ct = 0$</td>
<td>38.05</td>
<td>26</td>
<td>.06</td>
<td>.94</td>
<td>.015</td>
<td>66.05</td>
</tr>
</tbody>
</table>

Note. $cs$ - environmental effects shared by siblings, $m$ - environmental transmission from a mother to offspring, $f$ - environmental transmission from a father to offspring, $ct$ - environmental effects shared by twins, $p$ - two-sided significance, CFI - Comparative Fit Index, RMSEA - Root Mean Square of Approximation, AIC - Akaike Information Criterion.
The model dropping effects of the environment shared by all siblings ($cs$) of a family and the parental paths ($m$ and $f$) represented the best compromise between model fit and parsimony. In addition, we tested for cohort differences in the etiology of achievement motivation, by estimating a four-group model allowing for cohort (age) specific parameter estimates. This model did not significantly improve the fit indicating that parameter estimates could be generalized across the age range studied here.

Table 5

<table>
<thead>
<tr>
<th>Model</th>
<th>$\chi^2$</th>
<th>df</th>
<th>$\Delta \chi^2$</th>
<th>$\Delta df$</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td>$cs = 0$</td>
<td>28.98</td>
<td>23</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$cs = 0 \ m = 0 \ f = 0$</td>
<td>32.12</td>
<td>25</td>
<td>3.15</td>
<td>2</td>
<td>.20</td>
</tr>
<tr>
<td>$cs = 0 \ m = 0 \ f = 0$</td>
<td>32.12</td>
<td>25</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$cs = 0 \ m = 0 \ f = 0 \ ct = 0$</td>
<td>38.05</td>
<td>26</td>
<td>5.93</td>
<td>1</td>
<td>.01</td>
</tr>
<tr>
<td>$cs = 0 \ m = 0 \ f = 0$</td>
<td>32.12</td>
<td>25</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$cs = 0 \ m = 0 \ f = 0 \ with \ C. D.$</td>
<td>68.45</td>
<td>51</td>
<td>36.33</td>
<td>26</td>
<td>.08</td>
</tr>
</tbody>
</table>

*Note. $cs = 0$ - no environmental effects shared by siblings, $m = 0$ - no environmental transmission from a mother to offspring, $f = 0$ - no environmental transmission from a father to offspring, $ct = 0$ - no environmental effects shared by twins, C. D. - with cohort-differentiation between cohort 3 and 4; for the model $cs = 0 \ m = 0 \ f = 0$ with C.D. each parameter (except μ = phenotypic correlation of the parents) was estimated, $p$ - two-sided significance.*

The selected model provided evidence for additive genetic influences ($a^2$), non-additive genetic influences ($i^2$), twin-specific shared environmental influences ($ct^2$) and non-shared environmental influences ($e^2$). Standardized path coefficients and standardized variance components are shown in Table 6.
Table 6

NTFD: Standardizes path coefficients and variance components of the best-fitting model

<table>
<thead>
<tr>
<th>Model</th>
<th>A</th>
<th>i</th>
<th>μ</th>
<th>m</th>
<th>f</th>
<th>cs</th>
<th>ct</th>
<th>e</th>
</tr>
</thead>
<tbody>
<tr>
<td>cs = 0 m = 0 f = 0</td>
<td>.41</td>
<td>.37</td>
<td>.03</td>
<td>.00</td>
<td>.00</td>
<td>.28</td>
<td>.79</td>
<td></td>
</tr>
</tbody>
</table>

Standardized variance components

<table>
<thead>
<tr>
<th>Model</th>
<th>a²</th>
<th>i²</th>
<th>COV</th>
<th>PAR</th>
<th>ct²</th>
<th>e²</th>
</tr>
</thead>
<tbody>
<tr>
<td>cs = 0 m = 0 f = 0</td>
<td>.17</td>
<td>.13</td>
<td>.00</td>
<td>.00</td>
<td>.08</td>
<td>.62</td>
</tr>
</tbody>
</table>

Note. a - additive genetic effects, i - epistasis-effects, μ - phenotypic correlation between the parents, m - environmental transmission from a mother to offspring, f - environmental transmission from a father to offspring, cs - shared environmental effects between siblings, ct - shared environmental effects between twins, e - non-shared environmental effects (incl. measurement error), COV - passive gene-environment-correlation, PAR - environmental transmission from both parents to offspring.

Additive genetic influences explained 17% of the variance in achievement motivation, whereas epistasis-effects explained 13%. Moreover, the results provided evidence for significant twin-specific shared environmental influences, which accounted for about 8% of individual differences in achievement motivation. These environmental influences were specific to twins (ct²), and not shared with non-twin siblings or parents. The largest portion of the variance (62%) could be attributed to non-shared environmental influences (e²). There was no evidence for sibling-specific and parent-specific shared environmental effects, effects of assortative mating, and effects of passive gene-environment correlation.

Discussion

This study used data from a large representative twin family sample to examine genetic and environmental contributions to the development of individual differences in achievement motivation using an NTFD model. To the best of our knowledge, this was the first study of achievement motivation that included data of full siblings and biological parents in addition to using the data of twins only, and thus could test additional parameters that were less biased. The results of the NTFD analyses suggested a significant influence of genetic effects (a² + i² = 30%), which was, however, smaller than that found in previous studies by using the CTD (Kovas et al., 2015; Spinath, 2001; Spinath et al., 2008). Furthermore, the current study showed a significant influence of environmental experiences shared by twins (ct² = 8%). The largest part of variance could be attributed to non-shared
environmental effects ($e^2 = 62\%$), just like it was shown in the previous studies using the CTD (Kovas et al., 2015; Spinath, 2001; Spinath et al., 2008).

Consistent with the previous research, this study did not provide any evidence for shared environmental influences shared by parents and offspring, as well as by twins and their non-twin-siblings. Thus, at first glance, the results contradicted studies that could demonstrate a significant relation between achievement motivation and socio-cultural background, family climate and performance expectations, role model effects, and parenting style (Heckhausen & Heckhausen, 2010; Looser, 2011; Röhr-Sendlmeier et al., 2012; Röhr-Sendlmeier & Kröger, 2014).

Two points are important to note. First, studies correlating (even over time) parental characteristics, or characteristics of the home environment, with offspring’s characteristics are not informative of the connecting path, which may be environmental or genetic. Our study emphasizes the importance of a genetic path. Second, as outlined above, our results do not imply that parental or family influences are irrelevant to achievement motivation. However, these characteristics might differentially affect children reared in the same family. For example, the parent-child-relationship, which was shown to be correlated with achievement motivation (Looser, 2011), might differ between the children in one family.

The small, but significant effect of the twin-specific environment implies influences of shared demographics, age-specific experiences, peer-groups, and social experiences. Previous studies have confirmed that leisure activities and peer-relationships supply social contacts and opportunities of interaction that can influence a person’s achievement motivation (Heckhausen & Heckhausen, 2010). Moreover, compared to siblings of different ages, twins are more likely to attend the same school and classes, and thus make similar experiences at the same age that might affect achievement motivation. Those experiences in the school context might be, for example, the reference orientation, the classroom management of the teachers, and the educational leadership style of the school, which demonstrably influence the achievement motivation of students (Heckhausen & Heckhausen, 2010; Looser, 2017; Wigfield et al., 2006). Finally, twins share the timing of events and changing environmental conditions. For example, an economic situation of the family may improve over the years to the effect that an older sibling grows up in tight economic conditions, whereas family finances are more relaxed for younger siblings.

As mentioned above, the largest amount of variance (62\%) could be attributed to non-shared environmental effects (including a measurement error). Individual experiences, such as different peer-relationships, parent-child and teacher-student relationships, experiences in the family and in the school context, as well as individual life events might therefore be of great importance for the emergence of individual differences in achievement motivation (Bakadorova & Raufelder, 2014; Mansour & Martin, 2006, Martin, Marsh, McInerney, Green, & Dowson, 2007; Nelson & DeBacker, 2008).
Limitations

Though the NTFD requires less stringent assumptions than the CTD, allowing a more precise and detailed analysis of genetic and environmental influences on individual differences, it also has its limitations. Firstly, although the NTFD allows to determine the impact of passive gene-environment correlation, it does not obtain enough information to investigate other types of gene-environment interplay, such as active or reactive gene-environment correlations or interaction (Bleidorn et al., 2018; Keller et al., 2010). Active and reactive gene-environment correlations are confounded with the genetic variance component, and can therefore lead to an overestimation of heritability coefficients, when not taken into account (Bleidorn et al., 2018). Likewise, ignoring gene-environment-interactions could also lead to biased estimates (Kandler & Papendick, 2017).

When interpreting the results of this study, it should be also noted that achievement motivation was only surveyed with two items, and had a moderate internal consistency. Influences of a measurement error could lead to an underestimation of heritability coefficients, since they increased the dissimilarity of monozygotic and DZ twins, and are thus reflected in the variance component of non-shared environmental influences.

Implications and Future Directions

Despite the aforementioned limitations, our study contributes to the research on achievement motivation. The results show that 30% of the individual differences in achievement motivation are influenced by genetic (additive and non-additive) factors, to a small degree by environmental factors which the twins share, and to the biggest part by aspects that are specific for each individual and not shared among family members. This could imply that the family environment of adolescents and young adults plays only a minor role in establishing individual differences in the motivation to perform, and thus contradicts classical educational theories and models. Those non-shared environmental components might well derive from true individual experiences, such as friends and partners. However, it might also reflect experiences that are objectively shared between the children of a family, but perceived differently, such as parenting style. Nevertheless, though we do not challenge the importance of the familial home, our results underpin the necessity to focus on individual aspects of young people in order to understand why they differ in their achievement motivation.

There are obviously several questions left open by our results: What are the environmental influences that contribute to individual differences in achievement motivation? Which of these influences contribute to the similarity of twins, but not non-twin siblings? Detailed measurement of characteristics of the environment in longitudinal genetically informative studies is an obvious way to answer these questions. From a developmental perspective, it is further important to
study both stability and change in the relative contribution of genetic and environmental influences over the lifespan, as well as the contributions of genes and (measured) environments to the stability and change of achievement motivation (Kandler et al. 2010). To answer these questions, it would be helpful to conduct twin studies by using a broader range of age and a longitudinal design to investigate stability and change in variance components over the lifespan (Bleidorn et al., 2018; Kandler & Papendick, 2017). Lastly, we are convinced that future research will profit from epigenetic analyses that provide a novel tool to track environmental influences.

Conclusion

In this study we used the NTFD to derive a detailed picture of the etiology of individual differences in achievement motivation. Like previous studies relying on the classical twin design, we found that variance in achievement motivation was primarily explained by (additive and non-additive) genetic and non-shared environmental influences. In addition, variation could also be explained by environmental factors shared among the twins, albeit to a small degree. Thus, we suggest a revision of models and theories that answer the question of why people differ in their achievement motivation by differences in socialization only.

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ZAŠTO SE LJUDI RAZLIKUJU U MOTIVACIJI KA POSTIGNUĆU?
BLIZANAČKA PORODIČNA STUDIJA

Iako mnoge prethodne studije naglašavaju doprinose sredinskih činilaca, poput roditeljskog doma i školskog okruženja, motivaciji ka postignuću, klasične blizanačke studije sugerišu da i aditivni genetski i nedeljeni sredinski uticaji mogu da objasne individualne razlike u oblikovanju ovog fenotipa. Primenom nuklearnog porodičnog dizajna na podatke nemačke nacionalne TwinLife studije, analizirani su genski i sredinski doprinosi ispoljavanju motivacije ka postignuću kod adolescenata i mladih odraslih osoba. Kao što se očekivalo, rezultati su ukazali na značajne uticaje aditivne i neaditivne genetske komponente, kao i na značajne uticaje deljene sredine. Najveći procenat varijanze objašnjen je nedeljenim sredinskim uticajima, ukazujući tako na važnost individualnih iskustava u formiranju razlika u motivaciji ka postignuću. Rezultati ovog istraživanja ukazuju na potrebnu reviziju modela i teorija koje objašnjavaju varijacije u motivaciji ka postignuću isključivo kroz razlike u porodičnoj socijalizaciji.

**Ključne reči:** bihejvioralna genetika, motivacija ka postignuću, nuklearni porodični dizajn