2011

# The Genetic Basis of Creativity and Ideational Fluency The Genetic Basis of Creativity and Ideational Fluency

Mark A. Runco
*University of Georgia*

Ernest P. Noble
*University of California - Los Angeles*

Roni Reiter-Palmon
*University of Nebraska, Omaha*, rreiter-palmon@unomaha.edu

Selcuk Acar
*University of Georgia*

Terry Ritchie
*University of California - Los Angeles*

# The Genetic Basis of Creativity and Ideational Fluency

Mark A. Runco[a] , Ernest P. Noble[b] , Roni Reiter-Palmon[c] , Selcuk Acar[a], Terry Ritchie [d] & Justin M. Yurkovich[c]

a University of Georgia, Athens

b Semel Institute for Neuroscience and Human Behavior, UCLA

c University of Nebraska at Omaha

d Neuropsychiatric Institute, UCLA

Correspondence should be sent to Mark A. Runco, Torrance Creativity Center, University of Georgia Athens, 323 Aderhold Hall, Athens, GA 30602. E-mail: runco@uga.edu

**Abstract:**

Reuter, Roth, Holve, & Hennig (2006) described what they called the *first candidate gene for creativity*. This study replicated and extended their work for a more careful analysis of five candidate genes: Dopamine Transporter (DAT), Catechol-O-Methyltransferase (COMT), Dopamine Receptor D4 (DRD4), D2 Dopamine Receptor (DRD2), and Tryptophane Hydroxylase (TPH1). Participants were 147 college students who received a battery of tests of creative potential. Multivariate analyses of variance indicated that ideational fluency scores were significantly associated with several genes (DAT, COMT, DRD4, and TPH1). This was apparent in both verbal and figural fluency ideation scores, before and after controlling general intelligence. Yet fluency, alone, is not an adequate measure of creativity, and the index that is by far the most important part of creativity (i.e., originality) had a negligible relationship with the genes under investigation. Hence, in contrast to earlier research, the conclusion offered here is that there is a clear genetic basis for ideational fluency, but that fluency, alone, is not sufficient to predict or guarantee creative performance. Hence, at present, the genetic basis of creativity remains uncertain.

Creativity will only be well understood when its genetic basis is identified. Fortunately, great headway in this direction is being made, largely as a result of advanced technologies. For years the genetic basis of creativity could only be inferred from patterns of correlations among monozygotic versus dizygotic twins (Barron, 1972; Reznikoff, Domino, Bridges, & Honeyman, 1973; Runco, in press-b), or from differences in the correlations between children and their biological or foster parents. Now genes themselves can be examined. A much more accurate picture of the biological bases of creativity is emerging.

Reuter et al. (2006) described what they called the *first candidate gene for creativity*. They pointed to DRD2 because of its association with various measures designed to estimate creative potential. Clearly, these findings were merely a first step. In fact, that step may have been slightly askew; the earlier results were limited in several ways. First, Reuter et al. used an uncommon measure of creativity, making it somewhat difficult to interpret the meaning of their findings. Their measure was reliable (.85) but the description of it implies a connection with mathematical talent and convergent thinking. The latter is required whenever solutions are found by generating conventional or single correct ideas, and that is in direct contrast to divergent thinking, which is assessed with open-ended problems and seems to play a role in many creative performances (Guilford, 1968; Runco, 1991). Reuter et al. did describe their work as a pilot study.

One of the six subtests used by Reuter et al. (2006) was described as if it would provide similar results to the very commonly used Alternative Uses test of divergent thinking (DT; Guilford, 1968; Runco, 1991; Torrance, 1995) but the scores from this subtest were merely added into a composite, along with the other five subtests used by Reuter et al., and those other five subtests have dubious connection to creativity. They are much too convergent and preclude the original thinking that is vital for true creativity (Cropley & Cropley, 2009; Runco, 1988). It is unfortunate that Reuter et al. added all six of their subtest scores into one composite. This served to confound convergent and divergent thinking and blur any specific relationship with creative potential.

The present investigation used well-recognized tests of creative potential and scored them for originality (to allow a clear interpretation about creativity) as well as fluency (to replicate the earlier findings). A

more homogeneous measure of creative potential was used, thereby minimizing the possibility of any possible confound with general intelligence or mathematical talent. Flexibility scores, also commonly used in studies of creative potential, were calculated from the divergent thinking tests, in addition to originality and flexibility. This is all common practice when estimating the potential for creative problem solving (Guilford, 1968; Torrance, 1995).

The calculation of separate fluency, originality, and flexibility scores is of critical importance. Fluency only represents productivity. It can be quite meaningful, but it is not nearly as closely tied to creativity as is originality. Indeed, originality is the key to creativity. All creative things must be original. They must also be fitting somehow, aesthetically or functionally, but they must be original or they are not creative. This is one reason why fluency is sometimes unrelated to creativity. A person can be extremely fluent but only generate common and unoriginal (or useless) ideas and solutions. Originality can be defined in terms of the uniqueness or unusualness of ideas (Torrance, 1995). Flexibility represents the variety of ideas given and is also often associated with creative problem solving (Guilford, 1968; Runco, 1991).

In addition to employing common procedures for the assessment of creative potential, this research also extended earlier efforts by expanding the number of genes from 3 to 5: Dopamine Transporter (DAT), Catechol-O-Methyltransferase (COMT), Dopamine Receptor D4 (DRD4), D2 Dopamine Receptor (DRD2), and Tryptophane Hydroxylase (TPH1). The methods used to isolate each are described in the following. These were chosen primarily because of Reuter et al.'s (2002) findings about dopamine, as well as explicit hypotheses from Eysenck (1997) about the bases of creativity.

## Method

### *Participants and Procedure*

The sample consisted of 147 students from a Midwestern university (98 women and 49 men). All participants were White (self-reported). The mean age was 21.6 (*SD* = 3.8).

Participants were recruited through announcements in classrooms and received extra credit for participation. Participants signed up for group sessions. When participants arrived in the lab, they first completed the paper-and-pencil measures. Buccal (cheek) cells for DNA analysis were then taken. The buccal cells were suspended in a 0.9% saline solution. They were sent to UCLA where, after centrifugation, DNA was isolated from the deposited cells using standard procedures. Polymorphisms of the 5 genes were determined using method described in earlier research. These are indicated by the citations for each polymorphism:

- DAT1 (Vanderbergh et al., 1992),
- COMT (Hoda et al., 1966),
- DRD4 (Lichter et al., 1993),
- DRD2 (Grandy, Zhang, & Civelli, 1993), and
- TPH1 (Reuter et al., 2006).

After complete description of the study to the subjects, written informed consent was obtained.

The genes under investigation were analyzed as dichotomous variables, as in previous investigations on these same genes. Thus for the DRD2 it was A1+ vs. A1-; for DAT it was 9R+ vs. 9R-; for COMT it was V+ vs. V-; for DRD4 it was 7R+ vs. 7R-; for TPH1 it was A+ vs. A-. The risk alleles were as follows: for the DRD2 it was the A1+ allele; for DAT it was the 9R+ allele; for COMT it was the V+ allele; for DRD4 it was the 7R+ allele; and for TPNH1 it was the A+ allele. (See Table 1.) The table is formatted to allow easy comparisons with the earlier genetic studies, cited earlier.

TABLE 1
Genotype and Allele Frequencies of COMT, DRD2, Dopamine Transporter, DRD4, and TPH1

| *COMT VAL MET* | *DRD2 A1 A2* | *Dopamine Transporter 9R Non9R* | *DRD4 7R Non7R* | *TPH1 A C* |
|---|---|---|---|---|
| VAL VAL 16 (13.6%) | A1A1 2 (1.6%) | 9R9R 11 (8.3%) | 7R 7R 7 (5.9%) | AA 5 (3.7%) |
| VAL MET 69 (58.5%) | A1A2 54 (44.3%) | 9R Non9 R 56 (42.1%) | 7R Non7R 28 (23.5%) | AC 70 (51.9%) |
| MET MET 33 (28.0%) | A2A2 66 (54.1%) | Non9R Non9R 66 (49.6%) | Non7R Non7R 84 (70.6%) | CC 60 (44.4%) |
| VAL 42.8% | A1 23.7% | 9R 29.3% | 7R 13.3% | A 22.5% |
| MET 57.2% | A2 76.2% | Non9R 70.7% | Non7R 86.7% | C 77.5% |

### *Measures*

#### *DT*

Creative potential was measured using DT tests. Two different types of DT tests were used. Verbal DT was evaluated using three items. Participants were asked to “List as many things as you can that are square,” “List as many things as you can that move on wheels,” and “List as many things as you can that make noise.” A second set of three DT items were figural. The figural DT items showed participants a line drawing and asked participants to name all the things it can be. All DT items have demonstrated good reliability in various studies (e.g., Runco, 1991; Wallach & Kogan, 1965). The responses for each DT item, verbal and figural, were scored in several ways. First, the number of different ideas listed was counted as used a measure of fluency. The originality of each idea was evaluated using the number of individuals who listed the idea relative to the total sample. Only ideas that were given by less the 5% of the sample were scored as original. Finally, flexibility was defined as the ability to break mental set. In the context of DT tests, flexibility was indicated by ideas that were not strongly related to one another and therefore suggest different conceptual categories. In order to evaluate flexibility, items were first assigned into broader categories, and the number of different categories used was the flexibility score.

#### *Realistic creative problem solving*

Creative problem solving was evaluated by providing participants with an open-ended, real-life problem situation (Reiter-Palmon, Illies, Kobe, Buboltz, & Nimps, 2009). Participants were then asked to provide a solution to this problem. Solutions were then rated by trained raters, using the consensual assessment technique (Amabile, 1990) for quality, originality, and complexity. Quality was defined as the degree to which the solution is useful, appropriate, and actually solves multiple facets of the problem. Originality was defined as the degree to which the solution is novel, unique, and not structured by the problem. Complexity

was defined as the number of different ideas or components expressed in the solutions. Interrater reliability was .79 for originality, .81 for quality, and .87 for complexity.

*Wonderlic intelligence test*

General mental ability was measured using the Wonderlic, a paper-and-pencil, 12-minute timed test (Wonderlic, 1992). It includes 50 items measuring both verbal and quantitative ability. The Wonderlic correlates very highly with the WAIS-R (r = .92), and test–retest reliabilities range from .82 to .94.

*Analyses*

All five genes were in Hardy-Weinberg Equilibrium and in linkage equilibrium. The genes under investigation were analyzed as dichotomous variables as in a previous study (Conner, Hellemann, Ritchie, & Noble, 2009). For the COMT, the presence of the Val homozygote (Val/Val) and Val heterozygote (Val/Met) or Val+ was considered a genetic risk marker for hypodopaminergic functioning compared to the Met homozygote (Met/Met) or Val-. For the DRD2, the presence of the A1 homozytote (A1/A1) and A1 heterozygote (A1/A2) or A1+ was considered a genetic risk marker for hypodopaminergic functioning compared to the A2 homozygote (A2A2) or A1−. For the Dopamine Transporter, the presence of the 9 R homozygote (9R/9R) and 9R heterozygote (9R/Non9R) or 9R+ was considered a genetic risk marker for hypodopaminergic functioning compared to the Non9R homozygote (Non9R/Non9 R) or 9R-. For the DRD4, the presence of the 7R homozygote (7R/7R) and 7R heterozygote (7R/Non7R) or 7R+ was considered a genetic risk marker for hypodopaminergic functioning compared to the Non7R homozygote (Non7R/Non7R) or 7R−.

## Results

A MANOVA was conducted using DAT, COMT, DRD4, DRD2, and TPH1 as the predictors and the three fluency scores from the verbal DT tasks as the criteria. Each of the genes was coded dichotomously (0 = *at risk*, 1 = *nonrisk*). Results indicated that the two DAT groups and the DRD4 groups differed significantly; $F(3, 53) = 18.15$, $p < .001$; $\xi^2_{adj}$ (adjusted Xi squared) = .488 and $F(3, 53) = 14.70$, $p < .001$, and $\xi^2_{adj}$ = .432, respectively. COMT showed these same group differences; $F$s(3, 54) = 3.61, $p = .019$; $\xi^2_{adj}$ = .010. DRD2 approached statistical significance; $F(3, 53) = 2.62$, $p = .060$; $\xi^2_{adj}$ = .083. The effect sizes given take into account Serlin's (1982) observation that sample statistics may overestimate the strength of relationship between dependent and independent variable, depending on the number of levels of a grouping variable, the number of dependent variables, and the sample size. With that in mind, the effect size values (chi-squared) were adjusted.

A second MANOVA used the fluency scores from the figural divergent thinking tests as criteria. Here, there were significant group differences for COMT, TPH1, and DRD4; $F(1, 53) = 4.53$, $p = .007$, $\xi^2_{adj}$ = .164; $F$(1,

53) = 4.71, $p$ = .006, $\xi^2_{adj}$ = .171; and $F$(1, 53) = 3.16, $p$ = .032, $\xi^2_{adj}$ = .107, respectively. DAT showed a marginal difference between the groups; $F$(1, 53) = 2.2, $p$ = .098, $\xi^2_{adj}$ = .063.

***Originality and Flexibility***

The next analyses used the originality scores as the criteria. Ideas generated by less than 5% of the sample were identified, with originality then defined as the number of these unusual ideas produced by any one participant. This method is very frequently used in studies of creativity (for a review see Runco, 1991; Runco, in press). Results for the verbal divergent thinking tests uncovered significant group differences were observed for DAT, DRD4, and DRD2; $F$(1, 53) = 12.72, $p$ < .001, $\xi^2_{adj}$ = .394; $F$(1,53) = 9.10, $p$ < .001, $\xi^2_{adj}$ = .310; and $F$(1, 53) = 3.70, $p$ = .017, $\xi^2_{adj}$ = .130 respectively. In addition, marginally significant results were obtained for COMT; $F$(1, 53) = 2.47, $p$ = .072, $\xi^2_{adj}$ = .076.

Results from MANOVAs that used originality from the figural divergent thinking test scores showed significant results for DAT and DRD4; $F$(1, 53) = 3.91, $p$ = .014, $\xi^2_{adj}$ = .139; and $F$(1, 53) = 3.10, $p$ = .034 $\xi^2_{adj}$ = .104, respectively.

Because originality scores can be contaminated by fluency (Hocevar, 1980; Runco & Albert, 1985), a second originality index was calculated and used in additional analyses. It represented the ratio of original ideas to total ideas (fluency). This kind of ratio has also been used successfully in the past (Runco, 1991) and has the advantage of indicating relationships with originality when fluency has been controlled.

There were no significant effects in the MANOVAs when this index of originality was used—none for the verbal scores, none for the figural scores. This is an extremely important finding. This will be explored in the Discussion section.

MANOVAs using the flexibility score (the number of different conceptual categories used in a person's set of ideas) from the divergent thinking tests as the criteria showed significant results only for DAT; $F$(3, 56) = 4.10, $p$ = .011, $\xi^2_{adj}$ = .140.

***Controlling Intelligence and ANOVA of Realistic Problems***

Importantly, MANOVAs were also conducted using intelligence (the Wonderlic) test scores as a covariate. Results confirmed the relationships reported above. The same creativity criteria were related to the same

genes. Intelligence was only a significant covariate in two cases (flexibility from the figural divergent thinking tests and quality scores from the realistic problems), but even there, the relationships between creativity test scores and genes were unchanged. (Flexibility actually showed a stronger relationship with DRD4 after controlling intelligence, but was still only $p = .074$.) Very clearly, intelligence did not contribute to the relationships found between creativity test scores and the genes examined in this research.

Scores from the realistic, open-ended problem were then analyzed. As only one problem was used, an ANOVA was used to determine the effects of genes on solution quality, originality, and complexity. No significant effects were found.

## Discussion

These results support earlier findings from Reuter et al. (2006) that fluency is related to certain genes. This replication is useful because a different set of tasks was used in this research. These tasks have a much clearer connection to creativity than the tasks in the earlier research. Note also that this research found connections between fluency and several dopamine genes rather than just DRD2.

It is quite important that the relationships with fluency were not biased by general intelligence. In other words, the associations uncovered here are not merely the result of a genetic basis of general ability. In fact, as noted, the relationship between divergent thinking and the genes was sharper (and remained significant) when intelligence test scores were statistically controlled.

At least as important is that the genes investigated were not related to originality or flexibility from the various DT tasks, nor with scores on the realistic tests of creative problem solving. Given that (a) fluency is not, by itself, indicative of creativity; and (b) the measures most directly related to actual creative potential (i.e., originality indices) were not associated with genetic group differences, the obvious conclusion is that fluency has a genetic basis, where as originality, and therefore creativity, does not. At least originality and creativity are not related to the genes examined in this research, and these genes were, of course, chosen because there was a theoretical justification for them. Other genes may be more strongly related to originality and creativity than those examined here. That can be determined by future research. Still, the genes investigated here were chosen in part because theories have described creativity as something that might depend on certain processes and, therefore, certain genes (Eysenck, 1997).

The key to interpreting these findings is remembering that originality is vital for true creativity, but fluency is not. All definitions of creativity include originality and label it a prerequisite, or at least note that it is necessary but not sufficient for creativity (Cropley & Cropley, 2009; Runco, 1988, 2006). Fluency is not sufficient because original things are sometimes useless, and fluent individuals are sometimes unoriginal. High fluency has been found to characterize some samples that are low in actual creativity. Psychotics, for example, may be highly fluent, even though they are not truly creative (Eysenck, 1997). Still, it is interesting that ideational fluency may have a genetic basis.

Perhaps what is being expressed in fluency is merely a kind of cognitive energy that leads to a large number of ideas being produced. For some individuals, these can be elaborated, evaluated, filtered, and adapted such that some ideas become original and creativity is eventually expressed. For other people, the ideas are worthless or never developed, and creativity is not expressed. In this light, the present research is consistent

with the idea that there is a significant difference between creative potential and actual creative performance. Certainly, a larger sample should be employed in future research, but these results suggest that ideational fluency, alone, has a genetic basis. Given the likely interplay between nature and nurture in the fulfillment and expression of creative potentials (Runco, in press-a), these findings should be interpreted along with previous studies of the impact of experience on creativity (e.g., Runco & Acars, in press; Runco & Albert, 2005).

## References


Amabile, T. M. (1990). Within you, without you: The social psychology of creativity, and beyond. In M. A. Runco & R. S. Albert (Eds.), *Theories of creativity* (pp. 61–91). Newbury Park, CA: Sage.

Barron, F. (1972). *Artists in the making*. New York: Seminar Press.

Conner, B. T., Hellemann, G. S., Ritchie, T. L., & Noble, E. P. (2009). Genetic, personality, and environmental predictors of drug use in adolescents. *Journal of Substance Abuse Treatment*, 38, 178–190.

Cropley, D., & Cropley, A. (2009). *Fostering creativity: A diagnostic approach for higher education and organizations.* Creskill, NJ: Hampton Press.

Eysenck, H. J. (1997). Creativity and Personality. In M. Runco (Ed.), *The creativity research handbook* (Vol. 2). Cresskill, NJ: Hampton Press.

Grandy, D. K., Zhang, Y., & Civelli, O. (1993). PCR detection of the TaqA RFLP at the DRD2 locus. *Human Molecular Genetics*, 2, 2197.

Guilford, J. P. (1968). *Intelligence, creativity and their educational implications*. San Diego, CA: RR Knapp.

Hocevar, D. (1980). Intelligence, divergent thinking, and creativity. *Intelligence*, 4, 25–40.

Hoda, F., Nicholl, D., Bennett, P., Arranz, M., Aitchison, K. J., & Al-Chalabi, A., et al (1966). No association between Parkinson's disease and low-activity alleles of catechol o-Methyltransferase. *Biochemical and Biophysical Research Communications*, 228, 780–784.

Lichter, J. B., Barr, C. L., Kennedy, J. L., Van Tol, H. H. M., Kidd, K. K., & Livak, K. J. (1993). A hypervariable segment in the human dopamine receptor D4 (DRD4) gene. *Human Molecular Genetics*, 2, 767–773.

Reiter-Palmon, R., Illies, M. Y., Kobe, L., Buboltz, C., & Nimps, T. (2009). Creativity and domain specificity: the effect of task type on multiple indexes of creative problem-solving. *Psychology of Aesthetics, Creativity, and the Arts*, 3, 73–80.

Reuter, M., Roth, S., Holve, K., & Hennig, J. (2006). Identification of first candidate genes for creativity: A pilot study. *Brain Research*, 1069, 190–197.

Reznikoff, M., Domino, G., Bridges, C., & Honeyman, M. (1973). Creative abilities in identical and fraternal twins. *Behavioral Genetics*, 3, 365–377.

Runco, M. A. (1988). Creativity research: originality, utility, and integration. *Creativity Research Journal*, 1, 1–7.

Runco, M. A. (1991). *Divergent thinking*. Norwood, NJ: Ablex.

Runco, M. A. (2006). *Creativity: Theories and themes: research, development, and practice*. San Diego: Academic Press.

Runco, M. A. (in press-a). *Divergent and creative thinking.* Cresskill, NJ: Hampton Press.

Runco, M. A. (in press-b). Behavioral genetics of intelligence. In V. S. Ramachandran (Ed.), *Encyclopedia of human behavior* (2nd ed.). Oxford, England: Elsevier.

Runco, M. A., & Acar, S. (2010). Do tests of divergent thinking have an experiential bias? P*sychology of Art, Creativity, and Aesthetics*, 4, 144–148.

Runco, M. A., & Albert, R. S. (1985). The reliability and validity of ideational originality in the divergent thinking of academically gifted and nongifted children. *Educational and Psychological Measurement*, 45, 483–501.

Runco, M. A., & Albert, R. S. (2005). Parents' personality and the creative potential of exceptionally gifted boys. *Creativity Research Journal*, 17, 355–368.

Serlin, R. C. (1982). A multivariate measure of association based on the Pillai-Bartlett procedure. *Psychological Bulletin*, 91, 413–417.

Torrance, E. P. (1995). *Why fly? A philosophy of creativity*. Norwood, NJ: Ablex.

Vandenbergh, D., Persico, A.,Hawkins, A., Griffin, C., Li, X., & Jabs, E., et al. (1992). Human dopamine transporter gene (DAT1) maps to chromosome 5pl5. 3 and displays aVNTR. *Genomics*, 14, 1104–1106.

Wallach, M. A., & Kogan, N. (1965). *Modes of thinking in young children*. New York: Holt, Rinehart & Wilson.

Wonderlic, E. F. (1992). *Wonderlic personnel test*. Libertyville, IL: Wonderlic Personnel Test.