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Population genetics

Variant at serotonin transporter gene predicts increased imitation in toddlers: relevance to the human capacity for cumulative culture

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Cumulative culture ostensibly arises from a set of sociocognitive processes which includes high-fidelity production imitation, prosociality and group identification. The latter processes are facilitated by unconscious imitation or social mimicry. The proximate mechanisms of individual variation in imitation may thus shed light on the evolutionary history of the human capacity for cumulative culture. In humans, a genetic component to variation in the propensity for imitation is likely. A functional length polymorphism in the serotonin transporter gene, the short allele at 5HTTLPR, is associated with heightened responsiveness to the social environment as well as anatomical and activational differences in the brain's imitation circuity. Here, we evaluate whether this polymorphism contributes to variation in production imitation and social mimicry. Toddlers with the short allele at 5HTTLPR exhibit increased social mimicry and increased fidelity of demonstrated novel object manipulations. Thus, the short allele is associated with two forms of imitation that may underlie the human capacity for cumulative culture. The short allele spread relatively recently, possibly due to selection, and its frequency varies dramatically on a global scale. Diverse observations can be unified via conceptualization of 5HTTLPR as influencing the propensity to experience others' emotions, actions and sensations, potentially through the mirror mechanism.

1. Introduction

Socially learned cumulative culture has enabled humans to colonize diverse niches of the world [1]. While high-fidelity 'production' imitation is seen as one key to cumulative culture [2], social processes, including prosociality, group identification and teaching, have also been implicated [3,4]. Thus, another form of imitation, social mimicry, may facilitate cumulative culture. Social mimicry increases affiliation and interdependent self-construal, and being mimicked can induce prosociality [5], potentially motivating teaching behaviour.

Understanding the proximate origins of individual variation in imitative behaviour may provide insight into the evolutionary history of our psychological capacity for cumulative culture. A genetic component to variation in imitation is likely; twin studies show that imitation is heritable [6]. Functional variation at *SLC6A4*, the serotonin transporter gene, is a good candidate.

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Table 1. Model-averaged fixed effects parameter estimates. Relative variable importance (RVI) is the sum of Akaike weights for models that include the relevant variable. Unconditional standard errors are shown in parentheses.

	dependent variable: EIS				dependent variable: SIR			
	estimate	2.5%	97.5%	RVI	estimate	2.5%	97.5%	RVI
short allele	0.08 (0.04)	0.00	0.17	0.67	0.12 (0.05)	0.03	0.21	0.93
male	−0.05 (0.04)	−0.13	0.03	0.42	0.12 (0.05)	0.04	0.21	0.94
MDI	0.36 (0.04)	0.28	0.45	0.99	0.40 (0.04)	0.31	0.48	1.00
EIS					0.03 (0.04)	−0.04	0.11	0.35
SIR	0.05 (0.04)	−0.03	0.13	0.42				

A length polymorphism at this gene, 5HTTLPR (serotonin transporter linked polymorphic region), influences *SLC6A4* expression [7].

The short allele at 5HTTLPR was originally implicated in susceptibility to anxiety and depression [8]; there is now strong evidence that 5HTTLPR plays a role in gene–environment interactions and social cognition and behaviour in general [9]. The observation of poorer outcomes in adverse environments—and better outcomes in nurturing environments [10]—may arise from an association between the short allele and heightened sensitivity to environmental stimuli [11,12]. Physiological [13], socio-cognitive [14] and affective [15] evidence suggests that this heightened sensitivity extends to social stimuli. Homberg & Lesch [11], as well as Falk *et al*. [16], propose that the short allele may therefore result in greater social conformity. Further evidence that 5HTTLPR influences social learning or imitation *per se* is provided by an association between the short allele and increased observational fear conditioning [17], as well as anatomical differences in the mirror neuron system [18] and activational differences upon viewing emotional expressions [18] and performing a joint action task [19].

Using previously collected data, we evaluate whether variation at 5HTTLPR contributes to behavioural variation in production imitation and social mimicry, both of which are supported by the mirror neuron system but in interaction with different neural systems [20]. Toddlers in the US, genotyped for 5HTTLPR [21], were given the opportunity to imitate an adult following presentation of object manipulation tasks [6]. They were also assessed for spontaneous imitation of adult vocalizations and gestures.

2. Material and methods

Elicited and spontaneous imitation were assessed for each twin of 311 same-sex twin pairs, age 24 months [6]. An adult experimenter modelled three novel multistep object manipulations (puppet and rattle, each three-step, and birdhouse, seven-step) and then gave the object to the subject to play with for a set time (see [6] for protocol). Subjects received one point for each imitated step and the correct order; thus, the composite Elicited Imitation Score (EIS) ranges from zero to 16. Spontaneous Imitation Rate (SIR) is the per-minute mean number of repetitions of the experimenter's vocalizations or motor behaviours that were not explicitly modelled for the child. SIR was scored, by multiple coders, from 20 min of video taken during administration of the Bayley Mental Development Index (MDI) [22]; inter-rater reliability ($r = 0.96$, $p < 0.001$). Genotyping of 5HTTLPR was conducted as part of a panel chosen to evaluate genetic influences on ADHD; protocols, including quality control measures, are described in [21]. A final sample of 577 genotyped subjects was available for the current investigation.

We assessed relationships between EIS/SIR and 5HTTLPR with Gaussian mixed models. The distribution of SIR + 0.1 was log-transformed; EIS, SIR and MDI were centred at the mean and divided by two standard deviations. We addressed potential correlations due to sampling twins by including varying intercepts; twin pairs were assigned to cluster j, and individuals (monozygotic) or twin pairs (dizygotic) to cluster k [23]. All subsets of the model with fixed effects short allele + male + MDI + EIS/SIR were assessed with Akaike information criterion [24]. To predict EIS/SIR based on the models and data, we drew samples, from the joint posterior distribution across models, in proportion to each model's Akaike weight [25].

3. Results

The short allele is present in 70.4% of subjects (21.7% homozygous). All models garnering more than 10% Akaike weight include the short allele. Model-averaged parameter estimates and predictions suggest a small positive effect of the short allele on both EIS and SIR (table 1 and figure 1). Holding the other variables constant, the short allele is associated with an expected approximately 23% increase in SIR and a mean increase of half a point for EIS. Consistent with research that demonstrates shared genetic and environmental influences on mental ability and imitation [26], there is a positive relationship between MDI and imitation.

4. Discussion

Our results suggest that a functional polymorphism at *SLC6A4* contributes to variation in both production imitation and social mimicry in humans. Our study was constrained to a single population and a specific developmental stage; however, our results are corroborated by associations between the short allele and heightened sensitivity to social stimuli [13–16], as well as altered anatomy and functionality in the brain's imitation circuitry [18,19]. Beyond social sensitivity, a vast literature on 5HTTLPR indicates that the short allele is related to diverse phenomena, including: empathy [27], cooperation [28] and dancing [29]. Following Canli & Lesch's [9] suggestion that 5HTTLPR, in interaction with the environment, may influence social behaviour by modulating neural activation in brain regions containing mirror and Von Economo neurons, we note that many seemingly unrelated observations can be reconciled by conceptualization of

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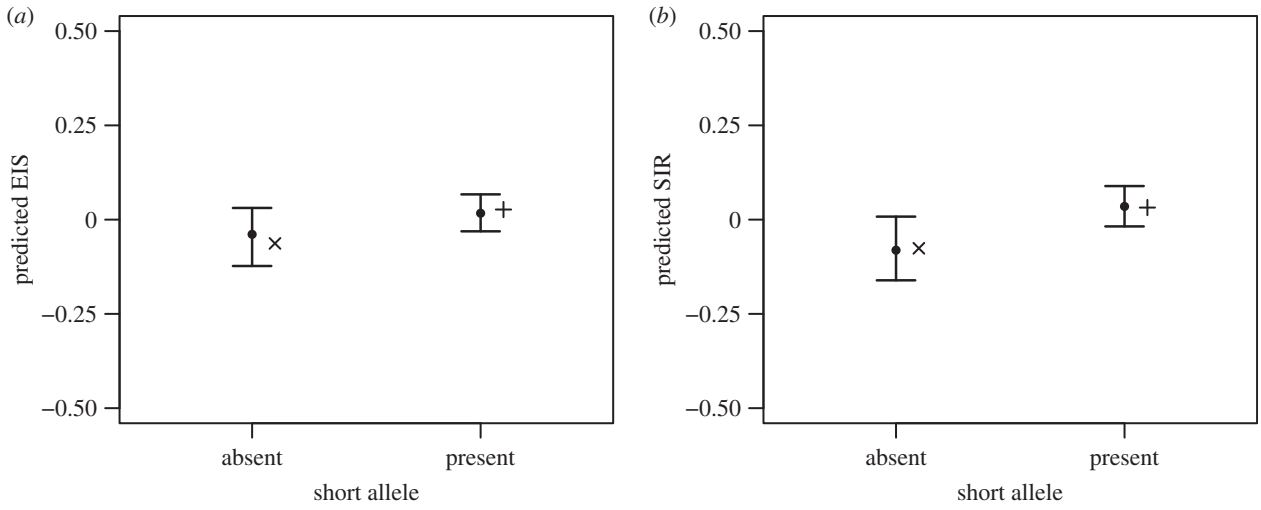

Figure 1. Model-averaged predicted (*a*) EIS and (*b*) SIR; 95% CIs. Mean standardized EIS, SIR indicated by the presence (cross) or the absence (×) of short allele. *Y*-axis depicts two standard deviations.

the short allele as influencing one's ability to experience others' actions, emotions and sensations.

Both production imitation and social mimicry may contribute to cumulative culture, which arises from high-fidelity imitation and social processes including prosociality, group identification and teaching [3,4]. Thus, the evolutionary history of 5HTTLPR may shed light on the development of our psychological capacity for cumulative culture. The short allele varies in frequency on a global scale, spread relatively recently in hominid history, and may have been subject to selection [30]. Signatures of selection, and the geographical distribution of the short allele, should be investigated alongside data on social learning to investigate population-level differences in the propensity for imitation. This is also suggested by Toelch *et al*. [31], who found that individuals who were more likely to use social information self-identified as more collectivist. Indeed, Chiao & Blizinsky [32] report higher frequencies of the short allele in collectivist cultures, and Mesoudi *et al*. [33] observed more social learning in China than in the UK (though not in Chinese immigrants in the UK). Consistent variation in social learning could have profound effects on the distribution of technologies and behaviours within and between groups [34]. Caution is warranted in assuming that our observations will translate to other populations, as the effect of the short allele on *SLC6A4* expression may be epigenetically modulated and depend upon another length polymorphism, in Intron 2 of *SLC6A4*, which varies in frequency around the globe [7,30].

Ethics. The study was approved by the Boston University IRB, Protocol no. 1852E. Informed consent was received from a parent of each of the participants.

Data accessibility. Data deposited in Dryad: http://dx.doi.org/10.5061/dryad.c2pt0.

Authors' contributions. P.A., S.K.F. and K.J.S. conceived of and designed the study; K.B.S. and P.R.B. conceived the present study; K.B.S. analysed the data and wrote the manuscript. All authors critically revised the manuscript, gave approval for final publication and agree to be accountable for all aspects of the work.

Competing interests. We have no competing interests.

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