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COMMENTARY ON "THE GENETIC ORIGINS OF LANGUAGE" (TIMOTHY J. CROW)

The perils of theoretical imprecision

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Crow has provided an interesting and provocative paper which summarises and integrates his various ideas, published now over several years, concerning the genetics and evolution of handedness and cerebral dominance, and their intimate connection with psychosis. This paper provides a useful focal point for assessing the hypothesis and trying to disentangle its theoretical core.

I am afraid that as I read the Crow hypothesis, I have an overwhelming sense of *déjà vu*, for it reminds me in several ways of the Geschwind-Behan-Galaburda (GBG) hypothesis. In particular there is a richness and breadth of citation, and a willingness to incorporate a vast mass of seemingly unrelated material under a single theoretical umbrella with almost breathtaking ease. The problem for the reader is trying to understand precisely what is being said and what it means. In the case of the GBG hypothesis, what was intended to be a single summer's work of clarifying the theory ended, only after many a summer, when we decided what the theory actually said (McManus & Bryden, 1991), and then whether it was supported by the evidence (Bryden, McManus, & Bulman-Fleming, 1994). Ultimately the theory crumbled around a

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single empirical issue, one which had stimulated the GBG theory in the first place, and which was sufficiently counter-intuitive to make the world immediately stand and look. If left-handers really do have an excess of immune disorders then that would be the strongest support for the GBG theory. Unfortunately they do not, and the theory then came tumbling down, for its elaborate theoretical superstructure could not be maintained on its foundation of sand.

With the Crow hypothesis we are presented once again with a grand theory about the origins of lateralisation. The problem for other scientists is first of all to try and work out precisely what the theory is saying, because if the theory is not well specified then empirical tests of it are likely to be weak. Secondly, there must be empirical data, preferably well replicated, which provide clear results, which are difficult to explain under any other account, are compatible with the theory, and preferably are also counter-intuitive.

The theory

I do not find it easy to know precisely what the Crow hypothesis is saying. In particular a complex chain of causality is being proposed, and I am not sure which of the links in that chain are strong and which are weak, and I do not know which weak links can be strengthened by other mechanisms, and I do not know whether some parts of the argument are actually entirely superfluous. That makes it difficult to test. However there would seem to be some points on which theoretical leverage can be applied, and for which much clearer explanation is required:

*i. **Sexual selection***. It is proposed (p. 1092) that an X-Y homologous gene could be under sexual selection and provide the effects required. However any autosomal or other gene can also be selected for by sexual selection (and many characteristics which differ between the sexes are determined by autosomes with sex-related expression). How precisely then does sexual selection work on X-Y homologous genes, and what is the characteristic signature which allows it to be differentiated from any other form of sexual selection? At present we have merely a hint that the presence of a sex difference means a) there must be sexual selection and b) there must be X-Y homologous genes. Neither of those assertions is necessarily true, and the theory needs properly unpacking.

ii. The role of age at marriage. Much emphasis is put upon the fact that women in most societies tend to marry earlier than males. While that is undoubtedly of great interest to those trying to understand the different mating strategies of males and females in relation to human evolution, it is not immediately clear that it has a central role to play in the present theory. If it were *not* the case, would the theory come crashing down? If not, then it may be superfluous. Some clear prediction is necessary in order to capture the precise relationship to a theory of lateralisation. Thus, is it the case that men or women who marry earlier should differ in their lateralisation from those who marry later? If so, that is a good and probably counter-intuitive prediction. But the prediction does need to be carefully specified. And it would help also if there were some data to support it.

iii. Rate of neural development. The rate of cortical development is invoked on several occasions as differing both between men and women, and between right and left hemispheres. At what point does brain development cease? In Figure 2, brain growth effectively ceases at a certain point in relation to body size, whereas in Figure 9 brains seem to continue to grow well into adult life (and certainly well past the point at which mate selection occurs). Is it actually true that brains continue to grow throughout the reproductive years (i.e., adulthood)? The sutures of the skull are usually fused by adulthood, and the head usually does not grow (as indicated in everyday life by a constant hat size). Is it then possible that the brain is still continuing to grow? If it is not possible, then surely the plateaux of the various trajectories will have been reached *before* mate choice? Some clarification is urgently needed. Of course, it could be argued that the important process as far as the theory is concerned is mate choice in prehistoric human societies, when puberty and pregnancy may well have coincided. That may be so, but the challenge then is to explain how in modern societies, where marriage is much later, that the proposed effects are still maintained under a very different selection system.

iv. The necessity of the Annett hypothesis. The balanced polymorphism hypothesis of Annett is invoked on several occasions. However that is a far from uncontroversial theory, which has major theoretical problems (McManus, Shergill, & Bryden, 1993; McManus, 1995), as well as empirical problems. In particular, there have now been at least four independent attempts to replicate its key claim, that intermediate

degrees of handedness are associated with greater intellectual ability, and all have failed (Klicpera & Gasteiger-Klicpera, 1994; Corballis & Palmer, 1996; Natsopoulos et al., 1997; Resch et al., 1997). If the Annett hypothesis fails, does the Crow hypothesis fail also? If so, then it is perhaps simplest to say that probably the Crow hypothesis has already failed unless substantial and dramatic new evidence is found in favour of the Annett hypothesis. If the Crow hypothesis does not depend on the Annett hypothesis, then what precisely is its relation to it? Is it entirely superfluous and the Crow theory sails on alone and unaffected? If so, why invoke the Annett hypothesis in the first place? Finally, on a point of pedantic detail about the Annett and McManus models of handedness, it should perhaps be emphasised that although Crow suggests that the McManus model is very similar to the Annett model (and was said on p. 1086 to be published later), that is not actually correct. The Annett (1978) model was not an additive model but had RS+ as dominant to RS-. In contrast, my own model has always been additive (McManus, 1979; McManus & Mascie-Taylor, 1979), whereas the Annett model only became additive in 1983 (Annett & Kilshaw, 1983); see McManus and Bryden (1992) for a detailed account. It is clear also, I think, that only additive models fit the data adequately.

v. The genetics of X-Y homologous genes. The X-Y homologous gene is a rare and exotic beast — so rare, indeed, that it seems at times as if the Crow hypothesis is dependent upon finding a unicorn. However a unicorn has an important identifying feature which makes it distinguishable from all other beasts, its single central horn. Population genetics is an exact and precise science, and clear mathematical predictions can be made about the behaviour of any genetic system which is distinguishable from any other. At present, the Crow hypothesis has much arm-waving about the separate evolution of X-Y homologous genes, but that is too imprecise to identify such genes. How, in terms of the conventional methods of population genetics, can an X-Y homologous gene system be identified? What precisely is the effect of selecting separately for the X and Y components, and how does that inter-relate to sexual selection? What in particular happens to the Y part of the system? Does it evolve entirely separately? Does it eventually act just as any other Y gene (and if so, why doesn't it disappear as most Y-linked genes seem to do)? Some precise predictions and modelling are needed here.

The empirical evidence

The theory as presented by Crow seems to have only two specific sets of empirical data relating to lateralisation, and these must be looked at carefully, as they are the ultimate foundation on which the theory of lateralisation is built.

*i. **Lateralisation in the sex chromosome aneuploidies.*** Undoubtedly one of the most important empirical observations in the present study concerns the nature of lateralisation in the sex chromosome aneuploidies. Crow summarises the various findings on pp. 1090 and 1091, although it is not terribly easy to visualise the various results. Opposite, I present the key findings in a simple table, which incorporates the findings in normal males and females (XX, XY), the data from Figure 7 of Crow, and the claim on p. 1090 that XYY individuals are similar to those with Klinefelter's syndrome (XXY).

The problem is to find a way to explain the pattern of cells in the table in which Verbal IQ is greater than Performance IQ (these cells are shaded, for clarity). The verbal-performance difference does not relate solely to the number of Y chromosomes (XXX ≠ XX), to the number of X chromosomes (XO ≠ XY; XO ≠ XYY; XX ≠ XXY), to the total number of sex chromosomes (XX ≠ XY), to the relative excess of X or Y chromosome numbers (XO ≠ XXY), or to body type (XO ≠ XXX; XX ≠ XXX). If there is an obvious pattern in these results I am not sure that I can see it. Of course it is possible to have a complex hypothesis (*if* A and *if* B), but with only six combinations, two body types, and several sets of chromosome number it is straightforward to come up with *some* explanatory hypothesis. The problem is to make it biologically convincing. At present there is no published data on lateralisation in the rarer sex chromosome aneuploidies, although they do exist, and it should in principle be possible to study XXXX, XXXY and XXYY karyotypes. Perhaps Crow could make a reasoned prediction in advance as to what should be found? My suspicion is that XXXX, XXXY and XXYY will all show higher performance IQ than verbal IQ, but that is based on nothing more profound than the only cases where verbal IQ is higher being found in the top left hand corner of the table. The crucial challenge for Crow is to explain clearly how all of this relates to the proposed X-Y homologous genes.

		X chromosomes			
		O	X	XX	XXX
Y chromosomes	0	†	V>P NSC = 1 XYD = 1 BT: Female Turner's (XO)	V>P NSC = 2 XYD = 2 BT: Female Normal female	P>V NSC = 3 XYD = 3 BT: Female 'Superfemale' XXX
	Y	†	P>V NSC = 2 XYD = 0 BT: Male Normal male	P>V NSC = 3 XYD = 1 BT: Male Klinefelter's (XXY)	?
	YY	†	P>V NSC = 3 XYD = -1 BT: Male 'Supermale' (XYY)	?	?
	YYY	†	?	?	?

Key: V>P (shaded): verbal better than performance; P>V: performance better than verbal; NSC: number of sex chromosomes; XYD: difference in number of X and Y chromosomes (+ve X>Y, -ve Y>X); † = lethal; ? = not known; BT = body type.

*ii. **Handedness in the NCDS.*** Crow provides two figures (5 and 6) and several pages of text (pp. 1087-1090) re-analysing data from the National Child Development Study on the relationship between hand differences and intellectual ability. Readers with a long memory may well find Figure 5 familiar since it is indistinguishable in substance from Figure 3 in McManus (1985), where it formed a corner stone in the argument that Annett's phenotypic description of handedness was incorrect; it is however, along with other previous analyses of handedness in the NCDS, uncited here. The analysis of the NCDS data is problematic,

and Figure 6 in particular is potentially very misleading for several reasons:

a) RELHAND is described in terms of 20 percentile bins. The result is that the entire group of left handers is dropped in to bins 1 and 2 at the extreme left of the figure, making any study of them impossible. With a sample size into five digits, that limitation is clearly not necessary. Moreover, Annett's theory is not couched in terms of percentiles, but in relation to a normal distribution along a linear scale of R-L. It is therefore very difficult to visualise the relationship between Figure 6 and Annett's theory.

b) The only important effects in Figure 6 are the dips at the third bin, equivalent to equality of the two hands, and at the 20th bin, consisting of the very extreme right-handers. However both of these are difficult to interpret. More especially, even if they are correct, they do not in any serious way correspond to the effects that Annett would predict, which are spread across the entire distribution, and should predict a peak of verbal ability at around the 50th percentile, i.e., bin 10-11, an effect which is clearly not present in Figure 6.

c) RELHAND is a deceptive measure, being calculated as (R-L)/(R+L). It depends therefore on the interplay between R-L and R+L. That is, RELHAND cannot be interpreted without information on the equivalence of R+L across groups. That information is not presented, and there is every reason to believe it is not equivalent across groups. Details of the actual numbers of squares marked are given elsewhere (McManus, 1985), but to a first approximation a typical individual marks about 90 squares with their right hand and 70 with their left, giving R-L = 20, R+L = 160, and RELHAND = 12.5%.

d) The dip at bin 3, where RELHAND is approximately zero, is particularly problematic in terms of the magnitude of R+L. In interpreting the NCDS data it should be remembered it is an entire population sample, and data are included for every person whom it is possible to include. Consider a severely mentally or physically handicapped child (and all children are included in the study), then that child is likely to make relatively few marks in the boxes with either their right or their left hand. That means both R and L will be very small. For arguments sake, imagine they are on average 9 and 7, i.e., one tenth of the mean population values. Although the difference is still proportionately equivalent, in practice sampling variation will mean that such individuals will in many cases produce values of, say, 8 and 8 (i.e., RELHAND =

0%). Bin 3, where RELHAND is zero, is therefore much more likely to contain individuals who are severely handicapped. It is therefore not surprising that the IQ of the group in Bin 3 is relatively lower than other groups.

e) Some of the children in the study will be hemiplegic or hemiparetic, due to spasticity or other neurological abnormalities. Their good arm will perform relatively normally on the test, whereas their bad arm will perform very poorly indeed. (See Bishop [1984] for a detailed description of the NCDS data in these terms). R-L will therefore be either large negative or large positive, and RELHAND will be very large negative or very large positive, putting the children into bins 1 or 20. Bin 1 cannot be studied properly in Crow's Figure 6, but bin 20 is precisely where it is proposed, following the Annett theory, that a drop occurs in verbal ability. More likely is that bin 20 has a disproportionate excess of hemiplegic or hemiparetic children, who will also tend to have lower IQs.

f) The Annett hypothesis does not refer only to verbal ability but also to non-verbal abilities, and according to the Crow and the Annett hypotheses it would seem that these should complement one another. In other words an additional figure equivalent to Figure 6, but plotting non-verbal ability should show the reverse pattern if Crow's interpretation is correct. If my interpretation in the points above is correct then bins 3 and 20 will also be impaired on non-verbal ability, since intellectual deficit will occur across the board. Predictions could also be made for the mathematics and reading scores which were also collected in the NCDS at age 11 (and we have analysed extensively elsewhere, McManus & Mascie-Taylor, 1983).

Conclusions

The Crow hypothesis is an interesting speculation, but it can be no more than that until a) it makes some more specific theoretical predictions, which if they are to be of any use to population geneticists, must be quantitative rather than merely qualitative; and, b) its clear predictions are testable against specific data and the theory shows an explanatory power over and above other possible explanations. At present, the only data presented which are properly related to lateralisation, on the sex chromosome aneuploidies, and on verbal ability in relation to handedness, are far from clear in their support of the Crow hypothesis over

any other. Of course all these problems could readily be finessed if the X-Y homologous genes were actually found and sequenced....

REFERENCES

Annett, M., & Kilshaw, D. (1983). Right and left-hand skill: II. Estimating the parameters of the distribution of L-R differences in males and females. *British Journal of Psychology, 74,* 269-283.

Bishop, D. V. M. (1984). Using non-preferred hand skill to investigate pathological left-handedness in an unselected population. *Developmental Medicine and Child Neurology, 26,* 214-226.

Bryden, M. P., McManus, I. C., & Bulman-Fleming, M. B. (1994). Evaluating the empirical support for the Geschwind-Behan-Galaburda model of cerebral lateralization. *Brain and Cognition, 26,* 103-167.

Corballis, M. C., & Palmer, R. E. (1996). Predicting reading ability from handedness measures. *British Journal of Psychology, 87,* 609-620.

Klicpera, C., & Gasteiger-Klicpera, B. (1994). Linkshaendigkeit und Legasthenie: Kein Beleg fuer die Rechsverschienungstheorie bei Wiener Kindern, aber Hinweise auf Verzerrungen bei der Auswahl von Kindern fuer Foerdermassnahmen. *Paediatrie und Paedologie, 29,* 11-15.

McManus, I. C. (1979). *Determinants of laterality in man.* University of Cambridge: Unpublished PhD thesis.

McManus, I. C. (1985). Right- and left-hand skill: failure of the right shift model. *British Journal of Psychology, 76,* 1-16.

McManus, I. C. (1995). Achilles' right heel: the vulnerabilities of the right shift theory. *Cahiers de Psychologie Cognitive/Current Psychology of Cognition, 14,* 565-574.

McManus, I. C., & Bryden, M. P. (1991). Geschwind's theory of cerebral lateralization: Developing a formal causal model. *Psychological Bulletin, 110,* 237-253.

McManus, I. C., & Bryden, M. P. (1992). The genetics of handedness, cerebral dominance and lateralization. In I. Rapin & S. J. Segalowitz (Eds.), *Handbook of Neuropsychology,* Volume 6, Section 10: *Child neuropsychology* (Part 1) (pp. 115-144). Amsterdam: Elsevier.

McManus, I. C., & Mascie-Taylor, C. G. N. (1979). Hand-clasping and arm-folding: a review and a genetic model. *Annals of Human Biology, 6,* 527-558.

McManus, I. C., & Mascie-Taylor, C. G. N. (1983). Biosocial correlates of cognitive abilities. *Journal of Biosocial Science, 15,* 289-306.

McManus, I. C., Shergill, S., & Bryden, M. P. (1993). Annett's theory that individuals heterozygous for the right shift gene are intellectually advantaged: Theoretical and empirical problems. *British Journal of Psychology, 84,* 517-537.

Natsopoulos, D., Kiosseoglou, G., Xeromeritou, A., & Alevriadou, A. (1997). Do the hands talk on mind's behalf? Differences in language ability between left- and right-handed children. *Brain and Language*, in press.

Resch, F., Haffner, J., Parzer, P., Pfueller, U., Strehlow, U., & Zerarn-Hartung, C. (1997). Testing the hypothesis of the relationships between laterality and ability according to Annett's right shift theory: findings in an epidemiological sample of young adults. *British Journal of Psychology, 88,* 621-635.