

1. Knickmeyer et al 2006 (Autism)

The investigators do not actually investigate autistic individuals in this study. What is their rationale for arguing that their results are relevant to their theory of autism, and what role does evolution play in their theory?

The theory: According to their Extreme Male Brain theory of autism, certain characteristics of the brain are sexually dimorphic, and autism is considered an extreme form of the male brain. In particular, according to this theory males are not as good as females at 'mind reading' and social skills generally, and autistic individuals are worse yet.

*The relevance: The investigators argue that high fetal testosterone level can masculinize the male brain beyond the male-typical average (based mostly on animal studies) and that therefore individuals who were exposed to high prenatal levels of testosterone should show evidence of extreme male brain, in particular, a tendency **not** to see social implications in the cartoon stimuli they are shown.*

The evolutionary argument: Because in the EEA females needed social skills because they migrated from their natal social group to their adult social group, and also because they were primary care-givers. Conversely, men stayed in the social group they were born into, and the most important skills for them were hunting skills which presumably required very different skills. Moreover, social skills may have been selected against because of intense male-male competition.

[I think their argument is flawed in a number of ways, but in any case, that's their argument.]

2. Beaulieu & Bungental 2008 (Post-partum depression)

(a) What is their rationale for their prediction that a depressed mother should put less parental investment into a pre-term infant than into a full-term infant, whereas a non-depressed mother should put more parental investment into a pre-term infant than into a full-term infant.

Their assumption is that in general post-partum depression occurs when the new mother is short on resources (either internal, e.g., she's not well, or external, e.g., she's poor, father is absent, that sort of thing). They thus argue that depressed mothers will tend cut their losses with a high-risk infant, i.e., invest relatively less in a high-risk infant than a low-risk infant. Conversely, non-depressed mothers will tend to invest relatively more in low-risk infants because (a) the mothers can, and (b) the infant needs and will benefit from the extra care.

(b) What is wrong (weird/screwy) with their prediction for the contingent model shown in Fig. 1?

*Their predictions are about **relative** investment, yet their graph indicates that on average both kinds of mothers invest **absolutely** the same amount in infants, the only difference being how each responds to high vs. low-risk infants. If you take the graph of their theory on its face, it says that a depressed mom will invest absolutely more in a low-risk child than will a non-depressed mom!*

(c) Despite the weirdness of Fig. 1, their results in Fig. 2 actually agree with the prediction quite well. Can you explain (explain away) how this might have happened given their method for measuring maternal investment?

This probably due to their very poor 'proxy' measure for actual maternal investment: it may be clever, but it is actually not a measure of significant maternal investment. Moreover, in the experiment the mothers were given an equal number of resources, whereas their theory is that depressed moms are beginning with fewer resources. What would have been much better would have been to get some real data on the actual investment of these mothers in their kids, say like the measures of actual investment used in adoption study we read earlier in the course.

3. *Lalumière, et al 2001 (Psychopathy) vs. Shaner et al 2004.*

Each of these studies proposes a theory of the evolutionary basis of the particular mental illness they are considering. If you were a *molecular geneticist* intent on finding *particular genes* for particular mental illnesses, and you *believed* both of these theories, which of the two illnesses would you consider a better bet for your molecular research? Explain why.

Lalumière, et al propose that psychopathy is a balanced polymorphism (or mixed ESS) where the 'cheater' strategy coexists with the 'normal' strategy so long as it is relatively rare (1% looks about right). Thus a gene for the 'cheater' strategy/phenotype would be a good bet.

Shaner et al propose that there is no gene "for" schizophrenia, rather there is a gene (or genes) for sensitivity to the individual's condition, such that the phenotype will be better than normal when the individual's condition is good and poorer than normal when the individual's condition is bad. Condition itself depends on genes and environmental factors, but the genes in question are many. In particular, many different deleterious mutant genes will wind up having the negative effect. Thus though schizophrenia will be heritable, you should not be able to find one or even a few particular genes that 'cause' schizophrenia.

4. *Kanazawa & Hellberg 2010 (IQ and drug abuse)*

Suppose you found a negative correlation between individuals' IQ and number of offspring they have ('smarter' people have fewer kids; perhaps this is true, I don't know). Try to relate this (hypothetical) correlation to Kanazawa's theory about general intelligence. You have to be clear why you think the correlation follows from the theory, is contrary to it, or cannot be related to it.

According to Kanazawa's Savanna-IQ Interaction hypothesis, abstract intelligence was advantageous specifically for solving novel problems. (Think of it as a specialized general-purpose module!) For the negative correlation above to be consistent with this theory, you would have to take one of two approaches. The simplest and best argument is that trying new things in this case results in lower RS, so that this lowered birth rate would be a maladaptive consequence of the higher general IQ caused by differences between the modern world and the EEA (birth control, etc.). Alternatively, you could argue that it is adaptive and that these intelligent people are having fewer kids but investing more in each one, so actually their RS is higher, not lower than their less-intelligent brethren. This is not quite so good an approach because it is 'special pleading', using something not in the question to effectively reverse the information given (though of course it is fairly plausible). A related approach is to pick up on Kanazawa's hypothesis that sexual exclusivity is evolutionarily novel for men and argue that this would lead to lower RS for men due to passing up opportunities for polygyny (although this is a bit of a stretch, it does use information from the article).