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Father's Genes: not so selfish after all?

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At the time, it seemed a brilliant idea. If you wanted to rebuild psychoanalysis on bedrock biology, what better basis than genetic conflict? Psychoanalysis was conflict psychology, and genetic conflict was the quintessence of evolution: genes that got themselves copied into the future had been selected, those that hadn't, weren't. Evolution was as simple as that, and psychoanalysis could be that simple too if psychological conflict was rooted in genetic conflict!

Furthermore, the nature of the conflict was clear. By the 1990s, we knew that some genes were only expressed from one parent's copy and that conflict between the parents over investment in the offspring was the likely basis. Mammalian fathers would want more investment during gestation and lactation than the mammalian mother because the father got all the benefit without any of the cost: selfish genes indeed! Mothers, on the other hand, would be paying the cost, so their genes would moderate investment. *IGF2* was the paradigm: a growth hormone gene expressed only from the father's copy, and silenced by the mother.

Now came my great idea: what Freud called the *id* must be the psychological agent of the paternal genome: hence its enslavement to the pleasure principle and its demanding, instinctive, and never-satisfied quality. The *ego* would then be the psychological agent of the maternal genome: hence its commitment to the reality principle and its ability to inhibit, postpone, and repress. Id-ego conflict would indeed be rooted in the genome and written into DNA if this were true.

But then came the clincher: what about the *superego*? Answer: genetic conflict is minimized if offspring share the same paternal genes. We already knew that gestational hypertension (caused by paternal genes because it increases food supply to the placenta) is reduced if the father is present during the pregnancy. The mechanism remains a mystery, but the rationale is that, if the father is still present he is likely to be the father of the next baby, and so his selfish genes should modify their demands on the mother's cardio-vascular system: they're going to need it again!

With this kind of thing in mind, I proposed that if a child shared the same father with its

siblings and had reached later childhood, the superego would emerge as an agent re-enforcing the ego and adding the father's sanction to the mother's interests, which were now much the same as his. (Later childhood would be the critical period, because almost always it is older siblings who are asked to make sacrifices for younger ones rather than vice versa.)

Furthermore, I didn't need any soft, psychological environmentalism to propose how this could come about. My suggestion was that the smell centers of the lower brain, known to be built by paternal rather than maternal genes, would enable the child to detect common paternity in siblings via smell—an effect already well documented in other mammals. Immediately this would explain the well-known (if often glossed-over) fact that children without fathers—and therefore much more likely to lack siblings with common paternity—are also more likely to show anti-social behavior, impulsiveness, and disruptiveness than those with a resident father.

But of course, it was wrong! As I have pointed out before, if this was true, autistic children would have revealed the Freudian id, but in fact have cognitive profiles much more compatible with the diametric model: invariably hypo-mentalistic (and sometimes hyper-mechanistic in the case of autistic savants).

Nevertheless, it was the Freudian psychology that was wrong, not necessarily the rest. A different role for paternal gene expression later in childhood remains a strong theoretical possibility as an intriguing mathematical model proposed by Francisco Úbeda de Torres and Andy Gardner makes clear. Most intriguingly of all, they suggest that after weaning and in adulthood altruism will be promoted by paternally-active genes and egoism by maternally-active ones—the exact opposite of what happens prior to weaning.

This might certainly explain the remarkable change seen in Prader-Willi syndrome (PSW). Prader-Willi is caused by imbalanced gene expression in the mother's direction, and the version involving duplication of the mother's chromosome 15 (without one from the father) invariably results in psychosis in adulthood—just as the imprinted brain theory predicts. PWS children are sleepy, undemanding and poor sucklers in infancy—just as you would expect if maternal, resource-limiting genes are in control. But PWS cases become rampant food-foragers in later childhood and obese as a result. (I heard of a case which resulted in institutionalization because the child, denied access to food in his own home by securely-locked storage, burgled surrounding houses in his quest for forage!)

This certainly sounds more like the Freudian id, but according to Úbeda's model results from maternal genes switching to the opposite strategy—resource-demand—after weaning. Indeed, Úbeda and Gardner point out that their “model indicates that psychotic-spectrum disorders can be explained by a hyper-egoistic brain”—along with the

Machiavellianism that often goes with it. Autistic spectrum disorders, on the other hand, would go with what they term a “hyper-altruistic brain” and explain the notable emotional empathy often seen in autism.

The New York Times remarked a couple of years ago that the imprinted brain theory “provides psychiatry with perhaps its grandest working theory since Freud, and one that is founded in work at the forefront of science.” Thanks to Úbeda and Gardner, that is even more true today.

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