Christopher Badcock

Evolutionary origins of autism and psychosis?


You may cite this version as:
Available in LSE Research Online: March 2007

LSE has developed LSE Research Online so that users may access research output of the School. Copyright © and Moral Rights for the papers on this site are retained by the individual authors and/or other copyright owners. Users may download and/or print one copy of any article(s) in LSE Research Online to facilitate their private study or for non-commercial research. You may not engage in further distribution of the material or use it for any profit-making activities or any commercial gain. You may freely distribute the URL (http://eprints.lse.ac.uk) of the LSE Research Online website.
The exact genes concerned have so far defied definition. But if both disorders were linked to the imprinting mechanism, much might be readily explained. This is because imprints are erased each generation, and as the case of Angelman, Prader-Willi, and Bedworth-Wiedemann syndromes show, can have genetic causes which are not necessarily heritable in the classical Mendelian manner. Again, autism can be caused by tuberous sclerosis and environmentally induced by prenatal valproic acid, thalidomide, or oral infection. Such factors could easily account for the expression of imprinted genes, and certainly could directly affect the relative development of the different parts of the brain where they are preferentially expressed. Many symptoms of autism have been interpreted in terms of deficits in what you might call mentalism defined as our way of reasoning, whereas autistics notoriously are not. When you say someone is ‘being paranoid’: you perceive a severe anomaly in any caller who in your evolutionary past would presumably have been the mother and her relatives.

The imprinted brain

The other organ in which imprinted genes are mainly expressed is the brain. Experimental studies on chimeric mice which have larger than normal genetic contributions from one or the other parent show that maternal genes are more expressed in the parts of the brain that correspond to the human neo-cortex, and that paternal genes are predominantly expressed in parts corresponding to the limbic system in human beings. To see what this means in practice, imagine that you are on a diet. The part of your brain that is making you hungry was built by your mother’s genes, appetites, along with other emotional and instinctual drives, originates in parts of the limbic system like the hypothalamus. But the part of your brain that makes you worry about getting fat and want to resist沃德 made from your mother’s genes – especially the prefrontal cortex, which plays a major role in inhibition and control of outputs from the limbic system – for example, countering feelings of hunger.

Evidence that imprinted genes play a conflicting role in behavior is suggested by two other paediatric syndromes: Angelman and Prader-Willi.

In both cases there is abnormal expression of imprinted genes in a region of chromosome 15. In Angelman there is more paternal and less maternal gene expression, but in Prader-Willi the opposite is the case. The underlying logic of this reflects the contrasting costs and benefits of growth to the mother as opposed to the father. Although the mother invests more in her offspring to grow them to exactly the same extent as the father, which invests in his offspring only in their genes.

In the tangible terms of a child’s birth-weight, the mother’s contribution is hundreds of billions of times greater than the father’s, which is only a single sperm! As a result, paternal-activated imprinted genes grow much faster than materianally-activated ones, and are particularly strongly expressed in the placenta – an organ primarily designed to extract resources from the mother. Indeed, an abnormal conceptus with androgynous features and growth anomalies whatsoever from the mother result in a massive proliferation of the placenta without any associated fetal growth.

Autism and psychosis

Autism and schizophrenia are extreme deviations, little or no language, and tend to be diagnosed as autistic. Prader-Willi cases with two copies of chromosome 15 from one’s mother (and none from the father) are always diagnosed psychiatric in adulthood and show less severe retardation. Further hints that imprinted genes may have something to do with autism and psychosis come from the finding that autistic has heavier birth-weight (especially males) while schizophrenics are lighter at birth. There is also the discover fact that paternal genes were more prominent in autism. Again, more paternal and less maternal gene expression was associated with high-risk genotype and subsequent autism, and vice versa with schizophrenia.

As mentioned earlier, autism has been defined as a mental disorder or ‘autistic psychosis’. In the same way, schizophrenia has been defined as a psychosis. But both may be caused by the same imprinted genes and their expression may be triggered by environmental factors.

Autism is often triggered by overgrowth of the placenta. Evidence that imprinted genes play a conflicting role in behavior is suggested by two other paediatric syndromes: Angelman and Prader-Willi. In both cases there is abnormal expression of imprinted genes in a region of chromosome 15. In Angelman there is more paternal and less maternal gene expression, but in Prader-Willi the opposite is the case. The underlying logic of this reflects the contrasting costs and benefits of growth to the mother as opposed to the father. Although the mother invests more in her offspring to grow them to exactly the same extent as the father, which invests in his offspring only in their genes.

In the tangible terms of a child’s birth-weight, the mother’s contribution is hundreds of billions of times greater than the father’s, which is only a single sperm! As a result, paternal-activated imprinted genes grow much faster than materianally-activated ones, and are particularly strongly expressed in the placenta – an organ primarily designed to extract resources from the mother. Indeed, an abnormal conceptus with androgynous features and growth anomalies whatsoever from the mother result in a massive proliferation of the placenta without any associated fetal growth.

Autism and psychosis

Autism and schizophrenia are extreme deviations, little or no language, and tend to be diagnosed as autistic. Prader-Willi cases with two copies of chromosome 15 from one’s mother (and none from the father) are always diagnosed psychiatric in adulthood and show less severe retardation. Further hints that imprinted genes may have something to do with autism and psychosis come from the finding that autistic has heavier birth-weight (especially males) while schizophrenics are lighter at birth. There is also the discover fact that paternal genes were more prominent in autism. Again, more paternal and less maternal gene expression was associated with high-risk genotype and subsequent autism, and vice versa with schizophrenia.

As mentioned earlier, autism has been defined as a mental disorder or ‘autistic psychosis’. In the same way, schizophrenia has been defined as a psychosis. But both may be caused by the same imprinted genes and their expression may be triggered by environmental factors.

Autism is often triggered by overgrowth of the placenta. Evidence that imprinted genes play a conflicting role in behavior is suggested by two other paediatric syndromes: Angelman and Prader-Willi. In both cases there is abnormal expression of imprinted genes in a region of chromosome 15. In Angelman there is more paternal and less maternal gene expression, but in Prader-Willi the opposite is the case. The underlying logic of this reflects the contrasting costs and benefits of growth to the mother as opposed to the father. Although the mother invests more in her offspring to grow them to exactly the same extent as the father, which invests in his offspring only in their genes.

In the tangible terms of a child’s birth-weight, the mother’s contribution is hundreds of billions of times greater than the father’s, which is only a single sperm! As a result, paternal-activated imprinted genes grow much faster than materianally-activated ones, and are particularly strongly expressed in the placenta – an organ primarily designed to extract resources from the mother. Indeed, an abnormal conceptus with androgynous features and growth anomalies whatsoever from the mother result in a massive proliferation of the placenta without any associated fetal growth.

Autism and psychosis

Autism and schizophrenia are extreme deviations, little or no language, and tend to be diagnosed as autistic. Prader-Willi cases with two copies of chromosome 15 from one’s mother (and none from the father) are always diagnosed psychiatric in adulthood and show less severe retardation. Further hints that imprinted genes may have something to do with autism and psychosis come from the finding that autistic has heavier birth-weight (especially males) while schizophrenics are lighter at birth. There is also the discover fact that paternal genes were more prominent in autism. Again, more paternal and less maternal gene expression was associated with high-risk genotype and subsequent autism, and vice versa with schizophrenia.

As mentioned earlier, autism has been defined as a mental disorder or ‘autistic psychosis’. In the same way, schizophrenia has been defined as a psychosis. But both may be caused by the same imprinted genes and their expression may be triggered by environmental factors.

Autism is often triggered by overgrowth of the placenta. Evidence that imprinted genes play a conflicting role in behavior is suggested by two other paediatric syndromes: Angelman and Prader-Willi. In both cases there is abnormal expression of imprinted genes in a region of chromosome 15. In Angelman there is more paternal and less maternal gene expression, but in Prader-Willi the opposite is the case. The underlying logic of this reflects the contrasting costs and benefits of growth to the mother as opposed to the father. Although the mother invests more in her offspring to grow them to exactly the same extent as the father, which invests in his offspring only in their genes.

In the tangible terms of a child’s birth-weight, the mother’s contribution is hundreds of billions of times greater than the father’s, which is only a single sperm! As a result, paternal-activated imprinted genes grow much faster than materianally-activated ones, and are particularly strongly expressed in the placenta – an organ primarily designed to extract resources from the mother. Indeed, an abnormal conceptus with androgynous features and growth anomalies whatsoever from the mother result in a massive proliferation of the placenta without any associated fetal growth.

Autism and psychosis

Autism and schizophrenia are extreme deviations, little or no language, and tend to be diagnosed as autistic. Prader-Willi cases with two copies of chromosome 15 from one’s mother (and none from the father) are always diagnosed psychiatric in adulthood and show less severe retardation. Further hints that imprinted genes may have something to do with autism and psychosis come from the finding that autistic has heavier birth-weight (especially males) while schizophrenics are lighter at birth. There is also the discover fact that paternal genes were more prominent in autism. Again, more paternal and less maternal gene expression was associated with high-risk genotype and subsequent autism, and vice versa with schizophrenia.

As mentioned earlier, autism has been defined as a mental disorder or ‘autistic psychosis’. In the same way, schizophrenia has been defined as a psychosis. But both may be caused by the same imprinted genes and their expression may be triggered by environmental factors.

Autism is often triggered by overgrowth of the placenta. Evidence that imprinted genes play a conflicting role in behavior is suggested by two other paediatric syndromes: Angelman and Prader-Willi. In both cases there is abnormal expression of imprinted genes in a region of chromosome 15. In Angelman there is more paternal and less maternal gene expression, but in Prader-Willi the opposite is the case. The underlying logic of this reflects the contrasting costs and benefits of growth to the mother as opposed to the father. Although the mother invests more in her offspring to grow them to exactly the same extent as the father, which invests in his offspring only in their genes.

In the tangible terms of a child’s birth-weight, the mother’s contribution is hundreds of billions of times greater than the father’s, which is only a single sperm! As a result, paternal-activated imprinted genes grow much faster than materianally-activated ones, and are particularly strongly expressed in the placenta – an organ primarily designed to extract resources from the mother. Indeed, an abnormal conceptus with androgynous features and growth anomalies whatsoever from the mother result in a massive proliferation of the placenta without any associated fetal growth.

Autism and psychosis

Autism and schizophrenia are extreme deviations, little or no language, and tend to be diagnosed as autistic. Prader-Willi cases with two copies of chromosome 15 from one’s mother (and none from the father) are always diagnosed psychiatric in adulthood and show less severe retardation. Further hints that imprinted genes may have something to do with autism and psychosis come from the finding that autistic has heavier birth-weight (especially males) while schizophrenics are lighter at birth. There is also the discover fact that paternal genes were more prominent in autism. Again, more paternal and less maternal gene expression was associated with high-risk genotype and subsequent autism, and vice versa with schizophrenia.

As mentioned earlier, autism has been defined as a mental disorder or ‘autistic psychosis’. In the same way, schizophrenia has been defined as a psychosis. But both may be caused by the same imprinted genes and their expression may be triggered by environmental factors.

Autism is often triggered by overgrowth of the placenta. Evidence that imprinted genes play a conflicting role in behavior is suggested by two other paediatric syndromes: Angelman and Prader-Willi. In both cases there is abnormal expression of imprinted genes in a region of chromosome 15. In Angelman there is more paternal and less maternal gene expression, but in Prader-Willi the opposite is the case. The underlying logic of this reflects the contrasting costs and benefits of growth to the mother as opposed to the father. Although the mother invests more in her offspring to grow them to exactly the same extent as the father, which invests in his offspring only in their genes.

In the tangible terms of a child’s birth-weight, the mother’s contribution is hundreds of billions of times greater than the father’s, which is only a single sperm! As a result, paternal-activated imprinted genes grow much faster than materianally-activated ones, and are particularly strongly expressed in the placenta – an organ primarily designed to extract resources from the mother. Indeed, an abnormal conceptus with androgynous features and growth anomalies whatsoever from the mother result in a massive proliferation of the placenta without any associated fetal growth.