

# **TODAY'S NEUROSCIENCE, TOMORROW'S HISTORY**

A Video Archive Project

**Professor Sir Michael Rutter**

Interviewed by Richard Thomas

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Interview Transcript

## **Early training and influences, the Maudsley Hospital, London**

At the time I came to the Maudsley, all but fifty years ago, most of us had trained in something else before we came into psychiatry and I was advised to do that. And I did, and trained in neurology and general medicine, and as it happened, also a period in paediatrics, although that was sort of almost accidental. As it turned out, that was a very useful experience to have had. So the context was different. The ethos here was definitely of questioning -- that was the order of the day. Aubrey Lewis, who dominated British psychiatry at that time and certainly dominated here, loved intellectual argument; he loved questioning. It had to be done really well but he was not a one for accepting theory or accepting authority. It didn't matter what the prestige was of the person who said it, the question was, 'What was the evidence, and how would you know?'

And that was a model that I adopted, like most of the people in the social psychiatry unit. So I had contact with him during my clinical training but then also in the research unit. In the research unit, Neil O'Connor and Beate Hermelin were the two experimental psychologists concerned with autism but who brought an experimental way of thinking, which made a big impact on me. I never worked directly with them but they certainly were mentors. Jack Tizard was also a mentor -- another psychologist -- and he brought me in on the Isle of Wight study and was very generous in giving me the responsibility for taking it forward; another iconoclast, but also somebody who ... to whom it was very important to use research to deal with practical matters and policy issues. And he did that in a very innovative way, and that made an impact.

Well, Aubrey Lewis had decided that I'd do child psychiatry although I actually wasn't very keen on the idea at the time, but he was right and I was wrong in that he did recognise that it: (a) played to my strengths, and (b) the research I'd already done was relevant to that. And he made two stipulations, one of which surprised me, and one of which I welcomed. The one I welcomed was that I had to learn about child development. The one that surprised me was that I should not be trained in child psychiatry at all because he said that firstly, the training was indifferent quality, which was correct at that time; but secondly, he thought that it was likely to inhibit creative thought, and he wanted me to develop the subject as a somebody who'd bring originality to it.

### **The need to question your own research**

One of the things in science is that you need to question your own findings, your own ideas, ruthlessly, and to put it in a flip way, what you want to do, if you're being ambitious, is to not just change your mind but you're the person who shows that it was necessary rather than your competitors. And you show what can be done to build on that.

So, yes, an example, would be that in my *Maternal Deprivation Reassessed*, I said that the evidence showed that there was, with a few minor exceptions, very little in the way of long-term effects of early experiences that were independent of later experiences. Now factually, that is still correct but what I had overlooked is that that was the wrong way of putting it, because it supposes that early environments are independent of later environments, and the evidence from our own research as well as others, indicates that's often not the case; that early experiences shape later experiences and that you do get important long-term effects, not because of independent effects, although we are aware now that those are more common than we thought, but because of chain effects -- one thing leading to another. So, yes, I did change my mind on that. I'm sure I was right to change my mind on that.

### **Epidemiological Psychiatry defined – the study of Romanian adoptees**

Well, epidemiology is the study of the distribution of either disorders or traits in the population, and it can be used in two sorts of ways, both of which we used on the Isle of Wight. One is giving you information for the planning of services, in other words, a very practical role for epidemiology but the other is using the evidence on associations between risk factors, and in our case, psychopathology, as a way of getting ideas on causal mechanisms. And so I was keen from the outset to be able to use epidemiology in this sort of way, aware of course that observational data, you've got to be questioning of as to whether the inference of causation is justified or not.

All people in behavioural sciences learn early on that you can't infer cause from a correlation or an association because it may be a coincidence, it may be due to selection into the groups that are associated -- a whole variety of reasons. But you can get much closer to a causal inference, provided that you have an experimental approach. Now sometimes you can actually use experiments but of course in mental disorder, many of the risk factors you can't manipulate experimentally. It would not be ethical or practical. So one of the things that over the years, that I've been very much involved with, is finding ways of using natural experiments as a way of testing the possibility that it *is* involved in a causal pathway. Of course with multi-factorial disorders -- disorders due not to a single cause but a lot of things coming together -- there isn't a basic cause, but on the other hand there certainly are factors that make a huge ... that play a huge part in the causal pathway.

For example, the study of Romanian adoptees -- children who'd suffered profound deprivation in Romania. There are two sorts of ways in which one could test the causal inference. The first is, if the environment changes, does their behaviour and their development change? And the answer was clearly yes. There was a dramatic catch-up -- not complete -- but very substantial. So that made it clear that something about the conditions before they came to this country, i.e. in the depriving institutions, had caused their problems. But then, you've got another way, which is that some of them had deficits; they didn't fully recover. They had problems of various kinds. So then the question is can you show systematic relationships between their pre-adoption experience and their outcomes, and what we have found is, yes, strong effects. And, moreover, effects that are as powerful at age eleven as they were at age six as they were at age four. So in this way, you have a fairly powerful inference that in some way these are involving causal pathways and so the skill, if you like, both the art and the science of using epidemiology in this way, is thinking of circumstances that will test.

### **Autism and the MMR vaccine**

To give a very different example: there was a lot of hoo-hah in 1998 of a paper that claimed that the vaccine MMR caused the epidemic of autism. Well, it occurred to me that one way of testing that was to see if there were countries that had stopped using MMR where others had continued using it, and it turned out Japan had, for reasons that were actually unconnected with the measles part of the vaccine. And so the question is: what happened to the rate of autism in Japan after MMR was withdrawn? And what our findings showed is, that the rate continued going on up -- that the withdrawal of a supposed risk factor had not made any difference and if anything, it had got worse, not better. So here was a way of testing, and in this case, disproving an environmental mechanism. In other cases -- as with the institutional

deprivation -- of supporting an environmental mechanism. So there are lots of examples of that kind, and that has become very much something that has remained an abiding interest and something I've tried to take forward.

### **Maternal Deprivation – relationships and attachments**

Maternal deprivation was initially seen by Bowlby in terms of separation experiences, and one of the things that came out of the review that I did was that although that may be a part of it, actually it isn't the key thing; that one needs to think about deprivation in terms of either a disruption of a relationship or an interference with the functioning of that relationship and not simply with separation. And so one of the things we did early on was to look at whether the effects on mental ... the risk of mental disorder were the same for parental divorce and parental death. And interestingly, the effects are much stronger on divorce than on death. So death is, as it were -- a much more permanent separation obviously -- but did not involve the conflict and discord that seems to be the key driving factor.

I agreed with Bowlby more than I disagreed but I did disagree on several key things. The first was the notion that separation was the key thing, and he came actually round to my view on that, partly as a result of his own study of children admitted to TB sanatoria and who were separated. The other was the focus on there being just one attachment, whereas I think the epidemiological evidence indicates that most children form multiple attachments. Now, it's not to say that the main attachment isn't particularly important but it is to say that the norm is several, and it is probably adaptive to have more than one. There's a wonderful book looking at this called *Kith, Kin and Hired Hands* which is making the point that throughout history and across all societies, it is very unusual not to have people other than the mother involved in the care of the children. Sometimes done this way, and sometimes that way. And I also differed in that it seemed to me psychoanalytic theory was completely hopeless in dealing with this. Bowlby initially gave a lot of credit to psychoanalysis but his theories on attachment were greeted with extreme hostility of a personal kind and later, in his ... I think probably his last book in 1988, he said that psychoanalysis was never more wrong than in its theory of development. So he and I were actually pretty close together. He laid emphasis on the value of psychoanalysis in forcing one to think about feelings and I'd agree with that, but he agreed with me in the end, that the specifics of that were just totally wrong.

## Lost relationships – coping, resilience and genetic factors

People nowadays don't use the terminology 'deprivation' very much but I think the findings have stood the test of time, and that dealing with acute stress responses and with anti-social behaviour reflect probably two rather different mechanisms, so that because we are social animals, we respond with distress to loss. This is true in infancy and it's true in extreme old age, that relationships are very important to us. So that the effects of either rejection or death of a loved one is a *stress*, and we now know that this is a pretty general finding and we have some beginning understanding of some of the biology that's involved in that.

There are a range of strategies that have been used in research to try and look at this, and to take three different examples: one is that one needs to think about it in terms of coping. That's to say, you have physiological coping and you have psychological coping, so that the neuroendocrine response to parachute jumping is quite different in experienced jumpers than in people making their first parachute jump. They have adapted and their bodily systems have adapted so it is no longer the same stress it was first time. and there are plenty of other examples of a similar kind of thing.

Now that leads on to the second mechanism or related mechanism, which is that most of the human resilience research has focused on avoiding stress and adversity or diminishing its impact in some ways. But if one thinks about this in a biological sense, that's the wrong way to think about it, so that if you want to protect people against infections, you don't put them in a cocoon and stop them ever having contact with bacteria and viruses -- you expose them. But you expose them in ways that they can cope with. That may happen in terms of natural immunity or, of course, it may happen through vaccination. So the psychological equivalent is to say: what could we do to enable children to cope successfully with hazards? Because challenges, stress – that's part of growing up and you have to learn to cope, and the only way you learn is through exposure.

The third mechanism is where the genetics comes in, where we know that the response to the acute stresses -- and the Dunedin work with Caspi and Moffitt, for example, shows that genetic factors play a role in influencing susceptibility to the environments. So one needs then to be looking at the genetic pathways that are involved in this, either *increasing* risk or increasing protection according to which end you're looking at.

So resilience is a real phenomenon but the ways of looking at it immediately bring you into biological research and they bring you into – well, I've mentioned three different sort of mechanisms but there are of course more. What we know much less about is the mediating

role of neuroendocrine factors; that there *are* neuroendocrine effects of a very important kind is not in doubt but do they account for the behavioural effect? We don't know. They could, but that is research still to be completed.

### **Autism – a brain disorder with important genetic factors**

The prevailing view in the sixties was that autism was an unusually early variety of schizophrenia and that it was due to poor parenting and other psychogenic causes. And the research that I'd been involved with cast doubt on that in indicating that the differences from schizophrenia were immense, and that it seemed very unlikely that it was anything to do with schizophrenia. But also the research had cast doubt on the notion that this was a psychogenic disorder, and our earlier follow-up study had provided really basically the first evidence that it was a neurodevelopmental disorder.

The follow-up study was important, I think, because we had excluded the children who had got a known neurological disorder but in spite of that, about a quarter of them developed epileptic attacks, epileptic seizures, in late adolescence. And that was, in a way, the first clear indication that there was a brain basis for this supposedly psychogenic disorder, and our research also showed that cognitive deficits played an important role, and language functioning similarly played an important role. So that what I was part of at that time was a paradigm shift from autism being a psychogenic disorder to autism being a brain disorder, and that this began very early. It's got nothing to do with schizophrenia, and a little bit after that we also did the first twin study, Susan Folstein and I.

And incidentally, that would be another example where I changed my mind because I wrote a paper in the mid-sixties concluding – along, I have to say, with some very distinguished geneticists, that it was unlikely that genetic factors played any major role in autism. And my reasoning, like theirs, was on the basis that the rate of autism in siblings, in brothers and sisters, was quite low -- below five per cent. I'd no sooner got this wretched paper published than I realised that was a stupid inference. The facts are correct, but the real focus should not be on the absolute low rate but the extremely high relative rate. So yes, five per cent is very low, but compared to what at that time was estimated as the four per ten thousand in the general population, that suggested not only that genetic factors were important but they were *hugely* important. And the twin study, although based on very small numbers, showed that *was* the case. It also involved, what at the time proved quite controversial, was the notion that the genetic liability extended beyond the handicapping disorder, and geneticists were very reluctant to accept that. It is now of course mainstream. So it was a very exciting

time of having findings that, as it were, brought in the biology and brought it in in a way which is testable and could lead on to understanding mechanisms.

### **Autism – tests for diagnosis and measurement reveal a wider view of the disorder**

At the time I entered child psychiatry, one really had no idea what to do with these kids, and indeed, didn't know much about what to ask the parents. So one of the things that my colleagues and I did was develop a set of instruments for interviewing parents on the one hand, and observing the young people on the other, so that with the observation, what we set about doing was to devise a set of social presses i.e. situations in which there was an expectation for social interaction and for communication, in order to see how the young people responded to that. So it was a sort of experimental or quasi-experimental approach to this, and those have now become sort of gold standard instruments used in autism research.

So the interplay between the research and clinical work, I've always had, always enjoyed, always learned from, and that has tied in with longitudinal studies, so one of the real interests is following these kids into adult life. And indeed, I've got somebody who the family want to see me about because I first saw him as a child but in three years time he will be retiring at the age of sixty and they want to consider the implications for – he's actually very independent, but he needs support. So dealing with those sides of the family, I've also enjoyed doing. So this is where the psychosocial and the biology, the clinical and the research all come together.

All measurement instruments, of course, are driven by the concepts of the day. How could they be otherwise? But you want to develop measures that are also sensitive to the unexpected, and with the Autism Diagnostic Interview we tried to do that. It was developed in relation to a somewhat narrower view of autism than would now prevail but our own findings with the Autism Diagnostic Interview, and the research that used it, made it clear that the boundaries went much wider. And our genetic research similarly. I was mentioning with the first twin study, one of the things that people resisted was the notion that the genetic liability went beyond the traditional diagnosis of autism. It was either much broader or it was dimensional, and we still don't know quite which is which, but it's certainly a lot broader than the traditional handicapping disorder. So the genetic evidence *and* the epidemiological evidence clearly pointed to the need for a broader concept, and that's no longer controversial. What is tricky though is knowing where the boundary lies, so clearly autism does not account for all social problems. I mean, we are social animals and that means that it's likely that almost any mental disorder will impinge on social functioning to some degree or other. So, the challenge now, which we're trying to engage with, is how can you tell which



ones are associated with autism and which one's aren't? We don't have firm answers on that although we certainly have some leads.

### Autism – degrees of severity

Well, the notion of a two-hit mechanism so far as autism is concerned, arises from the fact that we know that in the families there's an increased rate of what has come to be called the broader phenotype, meaning autistic-like abnormalities of a much milder kind. Now, they are very like autism in all sorts of ways but they differ in two ways: one is that the broader phenotype is *not* associated with epilepsy and it is *not* associated with mental retardation. And so one of the questions is: given the evidence that the broader phenotype *is* due to the same genetic liability as autism, why do some people have the full picture and some only these milder problems? Could there be... well, let's take two possibilities: one is, it's simply the level of genetic liability. If you have more of it you get the full thing; if you have less of it, you get the milder thing, or is it a two-hit mechanism in which the liability extends very broadly but something carries you over that threshold to develop full autism? Now, this is not a notion that is by any means restricted to autism because there's exactly the same debate in schizophrenia. So that we know that the so-called prodromata of schizophrenia are much commoner than actual schizophrenia -- involving delusions, hallucinations, thought disorder and so on. And so the question is, we know that the liability to both is genetic in large part and that it seems to be the same, but what carries people over into the full syndrome? And is that some new stimulus of a kind that hasn't been recognised? There are various possibilities that are being looked at and we don't know what the answer is as yet.

It's proved really frustrating not to have an answer on the genes as such. We have loads of leads but so far nobody has got good evidence that the actual -- well, it won't be *the* actual -- but one of the genes has been identified. It's puzzling because it is a strongly genetically influenced disorder. We have good measures; we have good strategies, so why on earth has it proved so difficult? And there are a variety of reasons that could be put forward. One is that it is the rule in medicine that there is a genetic heterogeneity; that's to say the particular pattern of genes that is responsible in one person isn't the same as in another. So that makes it much more difficult to tie it down, and it may also be that you are reliant on an interaction with some environmental risk factor. Because we haven't got a good handle on that, that's made it more difficult.

Will it deliver? Yes, I think it will deliver. There are some very good groups doing research in this area. I'd be really surprised if, in the next five to ten years, there aren't several susceptibility genes found, but it *has* proved very difficult. Is that special to autism? No, not at



all. If one looks at disorders like diabetes or coronary artery disease or asthma, the same sort of problem. Now there they have a much better physiological hold on the diagnosis, it has still proved very difficult. So, once you've moved away from single gene disorders, it's tough but it's solvable tough, and we've just got to get on and solve it.

### **Genes and behaviour**

In 2006, I wrote, published a book called *Genes and Behaviour: Nature-Nurture Interplay Explained*, and why did I do it? Well, it seemed to me that a lot of strong, misleading statements were being made both by geneticists on the one side, and anti-geneticists on the other. And there was a need for a book by somebody who was knowledgeable in both camps but, as it were, wasn't a card carrying member, as it were, of either group, and so that's what I set out to do.

One of the tasks, challenges, if you like, was to try to make clear some of the jargon terms that go throughout the literature. 'Susceptibility genes' would be one of those. The term's important because it is dealing with genes as they affect the liability -- susceptibility for something to happen. They're not genes that cause things in a deterministic fashion and so it's a descriptive term simply saying: 'These are genes that have been shown to have a robust association with some kind of outcome that you're interested in but don't cause it in a direct fashion.' Now, that also means that one has to talk about risk, and that virtually all the risks associated with multifactorial disorders -- in other words, disorders involving a mixture of genes and environment -- involve an increase in risk of a probabilistic kind. So that if you've got a particular gene or you've had some horrendous experience, you aren't predestined to develop schizophrenia, depression or whatever it may be, but you'll have an increased risk for it.

### **How genes might play a part in schizophrenia and autism**

Nowadays, differentiations are made between polygenic disorders and oligogenic disorders. 'Oligo' -- simply meaning more than one gene but not a huge number. It's not a sharp cut-off, but disorders like schizophrenia or autism probably are not polygenic in the sense of hundreds of different genes. They probably involve maybe a dozen different genes, but not vast numbers. And that what one has to understand is the different ways in which they play a part in the causal process, so that, for example, one of the key issues is, let's suppose for the sake of discussion, that there are four genes concerned. One question is: do they all impinge on the same causal pathway, or do they bring about the disease or disorder through different routes? And we don't know that, but it does mean that the research has got to look at both sorts of possibilities.

The traditional notion is that each gene has only one protein product but actually it turns out to be more complicated than that. To begin with, the whole process by which genes -- meaning the DNA -- affect the RNA, which then affects gene expression, affects proteins, affects the folding, involves many DNA elements. So there's not just one gene. There is, if you like, a whole family of genes, all of which are playing a part *and* in which environmental factors are also playing a part in that. So it's a dynamic concept now in a way that it didn't used to be.

And that means we must take on board in our research, in molecular genetics, the possibility that it ... we shouldn't be focusing on just genes for schizophrenia or autism but for sub-varieties. So that one might be looking at genes that are concerned with the social aspects of autism, different genes concerned with repetitive behaviours, and different genes concerned with communication. Suggestions have been made that that is happening. The evidence, to my mind, is still a bit contradictory but there's no doubt it could work like that, and the research must take on board that as a possibility. Going back to the point made earlier, genes don't code for diseases; they code for proteins, for chemicals, which play a part in the development of the disease, but they aren't the disease as such.

### **Genes and environments – anti-social mothers, adoptive parents and child behaviour**

Environments don't come randomly. They come through behaviour so that, for example, you and I will have had an upbringing that has been shaped by our parents' behaviour. Our parents will have provided us with genes but they will also have provided us with environment, and that is technically talked about as a passive gene-environment correlation. But then there is also research, some of which I have been involved with, looking at active and evocative gene-environment correlations, in which the individual's behaviour influences how other people respond.

The effect is shown in an adoption study -- The Colorado Adoption Study -- which was looking at children who are either born to anti-social mothers or to mothers who, as far as one knows, had not had those sort of problems, and were then adopted. And the question that was being investigated was whether the fact that the child had a biological parent who was anti-social influenced the behaviour of the rearing parent, the adoptive parent, and the answer was: yes it did, but it came about through the child's behaviour. And again we come back to this notion: some children are easy and fun to deal with; others are very difficult, and so it's not a direct effect of the gene -- it's the effect of the child's behaviour on the parent. But it is influenced, of course, by the child's genes as well as the child's environment.

### **Anti-social behaviour – why some people develop it**

The research undertaken by my colleagues Caspi and Moffitt, using data from the Dunedin study, hit the headlines in 2002 with their evidence that a particular genetic variant made a big difference in whether people developed anti-social behaviour in response to maltreatment. If you had one variety of the gene, maltreatment -- although obviously not a good thing -- carried with it a very small risk. On the other hand, if you had a different variety, it carried a big risk, and this was the first concrete indication in humans of gene-environment interaction in relation to psychopathology. And what it meant is that people needed to think about the fact that genes actually don't code for behaviours. You don't inherit a gene for anti-social behaviour or for any other behaviour for that matter. What genes code for are proteins, and the proteins have actions and the actions may be relevant to people's way of responding to stresses -- chronic stresses like maltreatment or acute stresses like unpleasant life events in adolescence. So this interplay between genes and environment has become a big topic.

The findings are exciting because they actually carry quite major implications. To begin with, the relevant genes are not genes for anti-social behaviour or in the other studies, genes for depression or genes for schizophrenia. Nor indeed are they genes for reactions to particular environments because to deal with the depression example, maltreatment showed the same interaction as acute life events did in adolescence. So it's responsivity but not to a specific environmental hazard, but that is not enough. There have been some very interesting studies, particularly by Daniel Weinberger's group at National Institute of Health, in which he has taken, together with his colleagues, these same genetic variants, taken samples of volunteers who do *not* have psychopathology -- they are chosen deliberately that they are not anti-social, they are not depressed, they are not schizophrenic -- and then looked to see whether the effects in terms of brain functioning, for example in the amygdala, is different according to which genetic variant you have. And it's been found that it is. It's been replicated by other people so it's a solid finding, but it immediately tells you we've got to get away from thinking that this is a gene for a disorder. It's a gene for a brain response to a particular kind of range of stimuli that is involved in the pathways to disorder, but it probably works dimensionally and it works in normal individuals as well as in those who develop disorder. So, this has really taken us a long way from the search for *the* gene for schizophrenia and no leading geneticist would talk like that anymore, but they did a decade ago.

### **Anti-social behaviour – why it matters**

There are two ways of looking at this. The first is: is there evidence that it matters? In other words, is it simply naughty behaviour to which you should take a punitive approach? Well, the evidence is clear-cut that it's associated with a raft of medical problems with a much

increased mortality. And, so, it matters, and therefore is properly considered a disorder. But then the second issue is: okay, but where do you draw the line? And here I think that the big change -- in the whole of medicine actually, not just in psychiatry -- is a recognition that many of these concepts in essence are dimensional rather than categorical, so that you don't, as it were, have a watershed between somebody who's emotional entirely normal and here is somebody with a depressive illness, any more than you do between somebody whose behaviour is entirely normal and this person has an anti-social personality disorder.

The risk factor for which there is the strongest evidence of a major effect, would be child abuse and neglect. I mean, that has a strong association with later anti-social behaviour plus associations with other forms of psychopathology as well. It's not just anti-social behaviour. But there is good evidence that not only is that a risk factor, but that it is an environmentally mediated risk. In other words, it's not just genetics.

### **Anti-social behaviour – the role played by genes that most of us have**

We know a good deal about some of the neurotransmitters that are involved -- the chemicals in the brain that play a role. We also know something about the physiology, so that having a very low pulse rate, a low response to stress, is associated with increased risk. Some debate as to what the mechanisms are but it's a well-substantiated finding. And there are, of course, the range of things that go with impulsivity and risk-taking, which are associated, and those also have biochemical correlates. I mean, one needs to talk about as correlates in that there was the sort of old-fashioned notion that, as it were, there was the biology and the biology 'caused', as it were, the behaviour. But it is much more complicated than that. It's a two-way interplay and it makes more sense to think about these as two facets of the same thing.

What we have is a common variant, so we're not talking about a gene that is pathological in itself. These are common genes and the other findings from the Dunedin study are of a similar kind. So they've looked at it in relation to anti-social behaviour, in relation to depression, in relation to schizophrenia, and the three key genes are found -- one or other of those three gene variants -- are found in something like eighty per cent of the population. So, we all carry these normal variants and they don't lead to disease in the way that you get with cystic fibrosis or haemophilia (the bleeding disease), which are directly genetic diseases. These are genes of a normal kind where one variant is having an effect on neurochemical pathways that indirectly carry a risk, so one needs to think about it not as *the* gene for anti-social behaviour. It is not the gene for anti-social behaviour, but rather a gene that has neurochemical effects, or influences neurochemical functions, in ways that carry risk. A probabilistic risk -- not deterministic.

### **Anti-social behaviour – the difference between corporal punishment and maltreatment**

It's difficult to define exactly what is meant by maltreatment but what is *very* striking, particularly from the research undertaken by my colleague Sarah Jaffee, that physical punishment – corporal punishment – and maltreatment are two very different things. They work differently in terms of genetic and environmental influences and they work differently in terms of risks, so that maltreatment is not just, as it were, a severe variety of corporal punishment. It involves using punishment as a way of getting rid of your own anger rather than as a considered disciplinary act. It is a very different phenomenon. Now, for a whole variety of reasons, I think it is undesirable to be hitting children. It's not a good way of disciplining them but it *is* different from maltreatment. The problem is that that same study by Sarah Jaffee, showed that those who regularly used corporal punishment were more likely to escalate into maltreatment, and one can sort of see why. If you're constantly dealing with tensions and crises by hitting out, then of course, it's likely to get out of hand at some point.

What she is pointing out is that this is a particular risk group and that it is likely that if one could change the behaviour of the parent and the style of interaction with the child, it would have beneficial effects. Now there are a whole series of behavioural treatments developed by people such as Caroline Webster-Stratton, Jerry Patterson and others, which do work. So her point is not that you should use *this* approach or *that* approach, but rather in deciding when to intervene. This is a group that have a particularly high risk and this is one where you should place your main attention.

### **The Camberwell Interview: assessing families' influence on risks for children's behaviour**

We've talked about a number of topics in some detail but perhaps it would be useful just to say a word or two about some of the other things that I've been involved with over the years. One was the development of the Camberwell interview, which is a collaborative enterprise with George Brown. We came at it from a different background but we were both aware that if we were interested in the way families influenced risks for children's behaviour or other relatives' behaviour, we had to have ways of measuring it and of quantifying it. And so we developed ways of doing that. The negative expressed emotion was based on careful ratings of how people talked about some other person in answer to open questions only, not about symptoms but: 'What's Joe like?' And we also developed measures of marital relationships.

### **Do schools influence behavioural and scholastic problems?**

A second example would be the study we did of secondary school children in London back in the seventies, in which other people had noted differences among schools in rates of

behavioural difficulties and also scholastic problems. The question was, was that because of what the children were like when they came to the school or was it an effect of the school on the functioning of the children? And we recognized that in order to study that we had to have a longitudinal study following the children through from primary school to secondary school, and actually we followed them up into work. And we had to have good measures of the school. So we had to have, if you like, the equivalent of what we did years before on families, in terms of what went on in the school, both in terms of teaching, discipline, the whole range of things. The bottom line was we found quite strong school effects. The academic establishment greeted this with profound scepticism and said it wouldn't hold up, but it did hold up, and subsequent research showed that we rather underestimated school effects rather than reverse. The interest, of course, in not just whether there's an effect but being able to tie it down, as it were -- which aspects of schooling mattered and which didn't.

Throughout, I've been interested in trying to explain to the lay public something about what we're doing, and so, in that era, I wrote a book called *Helping Troubled Children*, which aimed to explain what disorders in children were about, how we thought about them and what are the things we could do to help. And I think it's still actually in print, some -- whatever it is -- thirty-two years later.

### **Depression in childhood and what happens in adult life**

Another topic I became concerned with was depression, because back in the sixties the given wisdom was that depression was excessively rare in childhood, and that if it occurred it had got nothing much to do with adult depression. So with the late Dick Harrington, we did a follow-up study of children who had shown depressive problems to see what happened to them. The results were quite dramatic in showing there was a hugely increased risk of depression in adult life and it was entirely diagnosis-specific, so that that really altered the way people began to think about depression. And subsequently there have been a lot of other studies that have taken the matter further. Nowadays, nobody doubts that depression occurs in childhood. It is of interest that the ways in which it shows itself in children are somewhat different and their response to medication is somewhat different. So that's a puzzle still to be resolved.

### **Policy and punishment: the case of young people**

I had a lot to do with ministers during those early years. A lot of our research was funded by the Department of Education as it then was, or the Department of Health, a little bit by the Home Office. But all of that changed during the eighties, and the situation in the nineties was

that politicians didn't want researchers asking questions. They wanted simply evidence that their policies were correct, which is not an appropriate role for a scientist.

Let me give a specific example where there has been much debate over the British tendency to lock up more and more and more young people. And the evidence is that it makes things worse. That's pretty consistent and is not really in the least bit controversial among scientists. Now, you can't recommend that that should change but what one can say is, if you're wanting revenge on the criminal, fine, lock them up if that's what you want to do. If, however, you're wanting them to behave in acceptable ways in the future, then this is actually not a good way to go. You have to decide that.

### **What is science?**

I think that people often misunderstand what scientists have to offer so that back in, I guess, the sixties, I was involved in a tennis match with the House of Commons. I was playing competitive tennis a bit in those days, and one of the people said, 'What a pity there aren't more scientists in the House of Commons.' And there were of course hardly any at all, and so I said, 'Yes, that is a pity, but what would you want from the scientists?' And they said, 'Facts.' And I said, 'Well, facts are the epiphenomena of science. Science is actually not about facts. Science is about how to solve problems, finding a way of understanding what is involved. And yes, of course, that will give rise to factual evidence but facts are what emerge from it. Science is the act of finding out; discovering what needs to be done through either observational studies or experiments, or whatever it may be, to find out how the world works.'

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