

Neurosciences

The neurobiology of aggressive behaviour

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Abstract

Neurobiological research on aggressive behaviour comes up against particular difficulties that stem from the multifactorial origin of any social behaviour and from the fact that it evolves over time under the shaping influence of experience. From a historical point of view, the conceptual framework progressively switched from a deterministic causality based on the spatial distribution of a specifically-related ‘neural substrate’ to a probabilistic causality taking into account all the multiple contextual and developmental determinants with their underlying brain processes and mechanisms. With regard to ethical issues, the role and the weight ascribed to biological determinants in the generation of aggressive behaviour greatly influence the way in which one plans to fight against such behaviour. *To cite this article: P. Karli, C. R. Biologies 329 (2006).*

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Résumé

La neurobiologie des comportements d’agression. Les recherches d’ordre neurobiologique sur les comportements d’agression se heurtent à des difficultés particulières, qui sont liées à l’origine multifactorielle de tout comportement social et au fait que ce dernier évolue dans le temps sous l’influence structurante de l’expérience. D’un point de vue historique, le cadre conceptuel a progressivement changé, pour passer d’une causalité déterministe basée sur la répartition spatiale d’un « substrat nerveux » censé être spécifiquement lié à l’agression à une causalité probabiliste prenant en compte les multiples déterminants contextuels et développementaux, avec les processus et les mécanismes cérébraux qui les sous-tendent. Quant aux enjeux d’ordre éthique, le rôle et le poids attribués aux déterminants biologiques des comportements d’agression influent grandement sur la façon dont on envisage de lutter contre ces comportements. *Pour citer cet article : P. Karli, C. R. Biologies 329 (2006).*

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Mots-clés : Comportement d’agression ; Comportement social ; Déterminants biologiques ; Neurobiologie comportementale ; Causalité probabiliste ; Problèmes conceptuels ; Enjeux éthiques

1. Introduction

The study of brain–behaviour relationships underlying aggressive behaviour comes up against a number of difficulties. Some of them concern the entire field of brain–behaviour relationships, while some others more

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specifically relate to brain functioning underlying aggression. A major difficulty lies in the fact that even though experimental research in the field of behavioural neurobiology is carried out – for obvious ethical reasons – on the brain of some animal species, it basically aims at a deeper understanding of the biological determinants of our own personality and behaviour. Now, it is clear that data concerning brain–behaviour relationships can hardly be extrapolated from any animal species to man in the way one would rightfully extrapolate data concerning heart or liver physiology, or even elementary brain mechanisms, because the human being can by no means be reduced to his biological identity and functioning. A related difficulty stems from the fact that most experimental manipulations are strictly localized in time and in space, with results obtained out of history and out of context, even though developmental and contextual factors play an important role in the elaboration of behaviour.

As regards more specifically aggressive behaviour, there obviously arise two additional difficulties. On the one hand, unlike feeding, drinking, sexual or maternal behaviour, aggressive behaviour is characterized – in most instances of human aggression – by a multiplicity of origins and aims. On the other hand, in research on aggression much more than in research on any other behaviour, ideological prejudices may interfere in the choice of the working hypotheses and even more so in the interpretation of the obtained results. Even if we do not readily admit it, it is undeniable that the personal vision we have of ourselves, of the supposed ‘nature’ of man, of the origins of our being and becoming, influences – unconsciously, or more deliberately – the way in which we construct the conceptual framework within which we elaborate our working hypotheses and interpret the results obtained when verifying them.

2. The initial conceptual framework

A brief outline of the history of research on the ‘neural substrate’ of aggression will somehow reflect the above-mentioned problems. Half-a-century ago, Hess and Akert showed that an aggressive behaviour together with a marked emotional response could be elicited by applying an electrical stimulation to the cat’s hypothalamus [1]. It is particularly from medial hypothalamic stimulation sites that such behaviour could be induced, in the cat against a rat [2] as well as in the rat against a mouse [3], while attack behaviour without marked emotional signs was induced by stimulating lateral hypothalamic sites, in the cat [2] as well as in the rat [4]. Conversely, lateral hypothalamic lesions proved

to abolish the rat’s spontaneous mouse-killing behaviour [5]. Taken together, these experimental data led to distinguish two major ‘forms’ of aggression, namely an offensive or predatory aggression and a defensive or affective aggression. Much research was then carried out in order to specify the neural structures and pathways from and through which ‘facilitatory’ or ‘inhibitory’ influences were supposed to be exerted on the hypothalamic ‘effector centre’ of either form of aggressive behaviour. The general aim was to put together the ‘neuroanatomy’ of a given form of aggression, i.e. the distribution in space of the ‘neural substrate’ thought to be specifically related to the manifestation of an aggression of that particular kind.

It is quite understandable that scholars engaged in the study of human aggression could hardly see what they might learn from such neurobiological research and from the emerging theoretical constructs. And for someone carrying out a rather broad and long-term research on the rat’s mouse-killing behaviour (mostly called ‘muricidal’ behaviour), it was not especially agreeable to read and to hear that this kind of research was of no interest whatsoever for a better understanding of human aggression. It is also revealing that at that time the biennial meetings of the International Society for Research on Aggression (ISRA) consisted in fact of two separate meetings run in parallel, one dealing with ‘human aggression’ and one dealing with ‘animal aggression’. Only progressively was it decided – in the early 1980s – to have some modest ‘joint sessions’ in addition.

3. A fundamental change in scope

Just as progressively, a major change in scope had already occurred as regards the conceptual framework underlying most animal studies. This change derived from the twofold insight that the emotional components did not accompany and characterize some specific form of aggression but rather precede the overt behaviour, and that the individual’s past experience took an important part in the elaboration of any social behaviour [6,7]. The emphasis was no longer put on the ‘form’ of any aggression, but on the ‘function’ of this particular means of action used as a ‘behavioural strategy’. Basically, an aggression can be – and in many instances proves to be – an effective behavioural strategy that allows us to get at something, to get hold of it, or to get away from something, to get rid of it (‘appetitively motivated’ or ‘aversively motivated’ aggression). From this viewpoint, the lateral and the medial hypothalamus are no longer to be seen as the ‘generator’ or ‘effector’ sites of two distinct forms (two separate ‘models’) of overt aggressive be-

haviour. Since these two regions of the hypothalamus are major components of the neuronal systems of appetite and aversion, respectively, their activation greatly contributes to the elaboration of the ‘motivational state’ (involving both the notion of ‘motive of action’ and that of ‘motor’) that possibly induces an aggression.

The brain as a whole takes on the global function of a two-way mediation in the development of social interactions across the life course. The mediation thus maintained in the individual’s interactions with the social environment largely involves affective processes as well as reference to past experience. In fact, in man and animal alike (with obvious differences in complexity), social behaviour has marked affective and historical dimensions. On the one hand, brain processes are brought into play that allow the individual to experience positive – and avoid negative – social emotions in the manifold interactions with a particular and changing environment. On the other hand, it is precisely in and through these interactions that the social behaviour acquires its historical dimension and that it in turn reflects and dynamically shapes an individual life history. It is not surprising, therefore, that brain structures that are deeply involved in the constitution of – and reference to – affective memory, namely the amygdala (in the depth of the temporal lobe) and the prefrontal cortex (in particular, its orbito-frontal and anterior cingulate regions), were found to be similarly involved in the shaping, elicitation, and evolution of social behaviour [8,9].

This fundamental change in scope actually comes down to a clear switch – in the search of the biological determinants of aggressive behaviour – from a deterministic to a probabilistic causality. As a consequence, research will aim at specifying all those factors and processes that contribute to determine the probability that an aggression be elicited in the face of a given event or situation. Quite obviously, such a biological perspective can more easily be reconciled with the psychosocial perspectives that are elaborated in the study of human aggression [10,11]. Many relevant individual features can be fruitfully subjected to neurobiological investigation, such as: the level of overall emotional responsiveness, as well as the more specific sensitivity to the aversive character of threat, provocation, or frustration; the proneness to impulsive responding; the generation, individual degree, and behavioural repercussions of anxiety; the individual ‘behaviour style’ shaped early in life; the affective state of social comfort and the affiliative behaviours that both generate and reflect it; the way of recording – and referring to – the individual life history, especially the history of specific and relevant reinforcement; the changes in both cognitive and affective

significance that may result from processes or events such as familiarization, success or defeat, and punishment.

Data obtained with the modern techniques of molecular neurobiology and functional brain imaging clearly show that, in many instances, similar processes and mechanisms are brought into play in both animal and human aggression. A few examples will suffice to illustrate the latter statement. In the rat, the amygdala was shown to play a major role in the process of familiarization together with its aggression-preventing effect [12]. Similarly, the differential strength of amygdala activation in human subjects facing unfamiliar Black and White individuals was no longer obtained when the stimulus faces belonged to familiar and positively regarded Black and White individuals [13]. In the human patient as well as in the monkey, lesions of the prefrontal cortex provoke an inadapted and rather compulsive social behaviour, and a reduced prefrontal cortex functioning was observed in brain imaging performed in a number of ‘affective’, impulsive, murderers [14]. Research carried out on animals has repeatedly shown that serotonergic neurotransmissions take an important part in moderating both the generation of aversive emotions and the occurrence of aggressive behaviour. Likewise, impulsive violence has been linked to serotonergic abnormalities in humans [15], while an activation of serotonergic neurotransmissions through the use of selective serotonin reuptake inhibitors was found to reduce anger and aggression both towards oneself and others [16].

It clearly appears that the neurobiologist should refrain from considering that the data obtained in studying brain mechanisms do ‘explain’, by themselves, the functioning of the human mind and the production of human attitudes and behaviours. To build valid and promising bridges between behavioural neurobiology and human psychology, it is much more pertinent and fruitful to start from well-defined mental or behavioural processes that are likely to – and actually prove to – operate in similar ways in both animal and human beings, and then to carefully study the underlying brain mechanisms.

4. Concluding remarks

Since the brain functions as a mediating organ in the social being’s dialogue with a particular and changing environment, it follows that there are not just one-way relations between brain functioning and ongoing behaviour, but that the brain undergoes – in return – the shaping influence of the multifaceted experience that derives from that behaviour. And this shaping influence of experience does not just concern the content

of the conditioned associations and of the more complex representations carried by the brain, but also the brain mechanisms themselves that are involved in the constitution, the storage and the remodelling of that content. It became more and more apparent in recent years that the brain – and the human brain very especially – is characterized by a high degree of plasticity. In the early stages of life, but to a lesser extent also later on in the life course, the functional development of the neuronal networks as well as that of the systems of neurotransmission and of neuromodulation is greatly influenced by the prevailing environmental conditions and the individual's interactions with those conditions. This shaping influence of experience affects in particular the prefrontal cortex which – in close interaction with the amygdala and other temporal lobe structures – controls the complex interplay of cognitive and affective determinants of social behaviour. It is worth adding that various stressful conditions subjected to early in life may entail functional disruptions in both the amygdala and the prefrontal cortex (especially if there pre-exists a genetically determined vulnerability), which may lead, in turn, to violent behaviour and other behaviour disorders in adolescence [17].

The nature and impact of biological determinants are too often ignored in studies of human aggression. But the opposite position is just as wrong and harmful. As it is on improper biological grounds that assertions were and still are expressed which promote – unconsciously, or more deliberately – the evil myth of ‘the beast within’. Again and again, in a long history of misconceived ‘explanations’ of human aggression and violence, the latter were closely tied up with some supposedly typical and inherited biological feature [10,18]. Two quite concomitant events that occurred in the middle of the last century are worth mentioning in this respect. In 1963, Konrad Lorenz publishes his well-known book *Das sogenannte Böse. Zur Naturgeschichte der Aggression* (English version: *On Aggression*), in which he pre-emptorily stresses “the destructive intensity of the aggression drive, still a hereditary evil of mankind”, and sees “man as he is today, in his hand the atom bomb, the product of his intelligence, in his heart the aggression drive inherited from his anthropoid ancestors, which this same intelligence cannot control” [19]. Two years later, in 1965, Jacobs and collaborators report that a supernumerary Y chromosome was found in a population of subjects treated in institutions (who displayed both a degree of mental deficiency and a propensity to violence or crime) in a higher proportion than in the general population [20]. This publication gave then rise to a number of studies on what quickly became,

for some, the “crime chromosome” or the “supermales genetically programmed for violence”, before more serious research led to the conclusion that it was abusive to consider that the supernumerary Y chromosome ‘predisposes’, or even ‘urges’ a subject towards crime.

The promotion of such wrong ideas steadily persists since, a quarter of a century later, Daniel Koshland, Editor-in-Chief of *Science*, begins a leading article by referring to a recent hostage-taking with murder, which he relates to an “irrational output of a faulty brain”; he then goes on to express his conviction that molecular neurobiology will provide us with new tools that will prove more efficient in the struggle against violence than a number of social measures which – in his eyes – can hardly be more than “band-aid remedies” [21]. In reality, it must be stressed that “the idea that a gene determines a specific component of a behavioral phenotype is losing scientific credibility” [22], and that a highly complex gene-environment interplay is at work, with genetically influenced individual differences in the sensitivity to specific (in particular, stressful and adverse) environmental features [23].

With regard to human aggression, the available scientific facts do by no means impose a vision of necessity and fate; they certainly allow us to adhere to one of freedom, responsibility, and hope. More concretely, it is ill-founded to count on genetic engineering methods to eradicate from the human genome any ‘aggressiveness gene(s)’. And rather than to rely on some direct brain manipulation, we must fight against ‘ordinary’ aggression and violence (not to be confounded with a rare ‘pathological’ aggressiveness, which is a clinical sign – among others – of some brain lesion) by means of an education that leads to cognitive, affective, and moral maturity, the promotion of social change and the development of measures of social defence.

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