P A R T III

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SOCIAL DECISION-MAKING, NEUROECONOMICS, AND EMOTION

p0010

CHAPTER

14

c00014

Neuroscience and the Emergence of Neuroeconomics

Antonio Damasio

OUTLINE

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p0010 It is certainly the case that in the mid-1990s the term *neuroeconomics* was not in use, and that the field of studies that now goes by that name did not yet exist. However, the foundational facts were available, the key ideas were in the air, and all were ready to be focused on a new target. It takes two to dance, and in this case there were indeed two partners: behavioral economics and neuroscience.

p0020 As I see it, the behavioral economics partner was by then well established and had contributed a central idea – namely, that rational choice could not account satisfactorily for a considerable number of economic behaviors. To substantiate this idea, behavioral economics had gathered a remarkable roster of facts (for review, see Kahneman, 2003).

p0030 The neuroscience partner had also contributed a combination of facts and ideas. I will review some of those, from today's perspective, and round up my comments with a reflection on the notion of biological value, an indispensable construct in neuroeconomics.

FROM NEUROSCIENCE TO NEUROECONOMICS

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p0040 A number of neuroscience developments were of special relevance in the emergence of the new field of neuroeconomics, and I will begin by highlighting those that have to do with the neural basis of decision-making. A brief round-up of the critical evidence reveals the following facts which came to light between the mid-1980s and the mid-1990s:

o0010 1. Previously normal individuals who sustained bilateral brain damage centered on the ventral and medial sectors of the prefrontal cortices exhibited, after the onset of damage, marked defects of decision-making. The defects were especially notable for social behaviors.

o0020 2. In two areas of social behavior, the defects were so evident that they practically required no special diagnostic tool; these areas were *interpersonal*

relationships and, notably, decision-making having to do with *financial* issues. (Curiously, these findings already foreshadowed the main bodies of research that were to spring from them: social neuroscience and its exploration of moral aspects of behavior (examples of which can be found in Tania Singer's work and in the work of my research group); and the neuroscience of economic behaviors, a specialization of the pursuit into the neural underpinnings of social behavior, of which Ernst Fehrs' work is a great example.)

o0030 3. The patients with ventromedial prefrontal lesions had remarkably preserved intellect, as measured by conventional neuropsychological instruments, and an equally remarkable defect of emotional behavior. The emotional defect consisted of a rather general diminished emotional resonance, along with specific and notable impairments in social emotions – for example, in compassion and embarrassment.

p0080 In brief, patients, who had had normal social behavior until the onset of their brain dysfunction, and who certainly had not had any comparable difficulties in making sound decisions until lesion onset, were now deciding poorly and generally doing so against their best interests and the interests of those closest to them. This was happening in spite of their intellectual instruments being essentially preserved. The patients had no detectable impairments of logical reasoning, no defects of learning and recall of the kind of knowledge required to make sound decisions, and no defects of language or perception. Yet their decisions were flawed. Upon having the flaw pointed out to them, they did recognize that they could have done better. Once placed in similar future situations, however, they were likely to make comparably defective decisions.

p0090 The contrast between defective emotion on the one hand and preserved intellect on the other led me to propose that, somehow, disturbed emotional signaling could explain the decision defect. This idea formed the basis for the so-called *somatic marker hypothesis*, which was aired in several articles during the 1990s. Easily accessible summaries of findings and theory can be found in *Descartes' Error* (Damasio, 1994), and in an article for the *Transactions of the Royal Society* (Damasio, 1996).

p0100 I never considered the hypothesis as anything but a beginning, the start of an exploration of the role of emotion in decision-making, but I did think that such a possibility was well worth entertaining; namely, that emotion would play an important role in decision-making not just for the worst, as was then the traditional view, but for the *better*. I was persuaded that emotion might well account for some of the decision anomalies brought to light by the work of Kahneman and Tversky. And I did note, from the outset, that the "emotion" concept used in the theory was nothing but the tip of the iceberg. Underneath that iceberg there were the mechanisms of drives and motivations as well as those of reward and punishment, which are the fundamental constituents of the emotion machinery. I ventured that those were the factors most likely to play the main modifying role in the decision process, from a neural perspective, at either conscious or unconscious level.

p0110 In retrospect, it is apparent that these early observations and interpretations benefited from and became part of a major revival of interest in the neuroscience of the emotions, which had been much neglected until the last decade of the twentieth century. The new work on the emotions encompassed work in experimental animals – a prime example is Joseph Le Doux's exploration of the fear conditioning paradigm in rodents (Le Doux, 1996) – as well as the human lesion studies conducted by our group. Over a brief period of time a growing number of investigators were able to identify critical stages in the emotional process, and discover the main induction sites for emotions such as fear (the amygdaloid nuclei) and the social emotions (the ventromedial prefrontal cortices). We were also able to establish a principled distinction between emotion and feeling (see below), and to identify the insular cortex as a principal neural substrate for feelings (Damasio, 1994; Damasio *et al*., 2000). Social neuroscience and neuroeconomics were by then ready to exploit functional neuroimaging to its full advantage.

p0120 Adopting today's neuroeconomics perspective, I would summarize the somatic-marker hypothesis as follows:

o0040 1. Emotion plays a role in decision-making, but it should be clear that, under the term *emotion* I include both (a) the neural subprocesses of automated life regulation that are part and parcel of emotion action programs, namely reward and punishment processes and drives and motivations; and (b) the neural substrates of the perceptual read-outs of emotion action programs, namely emotional *feelings*.

o0050 2. In the original somatic-marker hypothesis outline, I suggested that the emotional influence on the decision-making process was exerted neurally, at multiple neural levels, from the high level of feelings substrates to the level of reward and punishment signaling (see Damasio, 1996). Needless to say, I remain convinced of the importance of these points and I wish to emphasize them because

so often, especially in discussions on the notion of biological value, the concept of emotion becomes dangerously amputated. Separating emotion from its reward and punishment components is a major conceptual problem. Another major conceptual problem comes from confusing emotion (which is an action program) with a *feeling* of emotion (which is the conscious, cognitive sequel to the action program). These are different phenomena with different neural substrates.

o0060 3. Emotion plays its role either consciously or non-consciously, depending on the stage of the process and the circumstances. When emotion influences decisions consciously, the deciding subject may be aware of the "marker" and even refer to it – for example, report a "gut feeling." But decisions may also be influenced covertly, and the hypothesis states that non-conscious "biases" can alter the processing networks and drive the process in a particular direction. I conceived of this, and still do, as operated by specific neuromodulators acting on different levels of neural circuitry, all the way to the central cortex.

o0070 4. In the framework of the somatic marker hypothesis, the abnormal decision-making that we described in our patients resulted from a cognitive malfunction that was rooted in an emotional malfunction. In other words, the emotional defect did not explain the anomaly alone; the emotional malfunction altered the cognitive process.

o0080 5. The term "somatic" needs some clarification. It conjured up the *body-relatedness* of the physiological mechanisms I was invoking. I believed, and still do, that the decision-making machinery we make use of in social matters in general, and in economic matters in particular, is a fair use of the mechanisms of decision-making that began as routines of life regulation focused on body physiology. Hence the word *somatic*.

o0090 6. What I meant by *marker* in the somatic-marker hypothesis is sometimes misinterpreted. The marker in the hypothesis is a *memory trace*. The marker was learned in past experiences of the subject, in which certain situations required a decision, evoked certain options of action, prompted a decision, and resulted in specific outcome. The outcome would have been, in the emotional sense, positive or negative, rewarding or punishing. In other words, the marker stands for situations in which certain facts (the premises of a problem; the options of action; the factual outcome) were associated with certain emotional outcomes. The marker signals the conjunction, in past experience, of certain categories of situation or outcome with certain categories of emotional response. The marker as memory trace is recorded in higher-order cortical circuitry, of which the ventro-medial prefrontal cortices are the most notable example.

o0100 7. When situations of a certain category re-present themselves to the decider subject, the marker is reactivated. In other words, processing a situation strongly resembling another situation regarding which decisions were made, prompts recall of related information. The recall may or may not come to consciousness, but in either case it promotes the replication of the emotional state associated with the particular class of situation, option, or outcome. In normal individuals, the marker "weighs in" on the decision process. In cases of ventromedial prefrontal damage, it fails to do so.

p0200 The somatic market hypothesis prompted several experimental tests of its validity, and inspired the development of the Gambling task (Bechara *et al*., 1994). The task provided the first laboratory diagnostic procedure for patients with ventromedial prefrontal damage – a rather useful advance, given that these patients generally passed all other neuropsychologic tests and only exhibited their defects in real life and real time. The task was also instrumental in showing a persuasive correlation between indices of emotional change (skin conductance responses) and the advantageous or disadvantageous playing of the card game (Bechara *et al*., 1997). The poor performance of prefrontal patients was accompanied by largely flat skin conductance responses which failed to discriminate between advantageous and disadvantageous decks.

p0210 The task attracted an intriguing controversy regarding how conscious the normal individuals who played the card game were of the winning strategy. When critics Maia and McClelland (2004) administered the gambling task using our procedures they replicated our results, as has been the case with all other authors who have done so. However, when Maia and McClelland used a different set of instructions for the task, one that probed ongoing knowledge in a deeper manner, the results predictably revealed that the subjects knew about the winning strategy earlier than in our version. The deeper probing was responsible for a greater scrutiny of the task by the subject, and injected into the process a degree of knowledge that our version of the procedures did not prompt. In no way do the results of the modified task contradict our original task results, or the idea that deciders, in the Gambling task or in other situations, may be influenced by non-conscious factors, emotional or otherwise (a recent study by Persaud *et al*., 2007, bears

out this point nicely). And in no way does the modified task compromise the somatic marker hypothesis, since the hypothesis specifies that the emotional role in decisions can be played out either consciously or non-consciously.

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DECISION-MAKING, EMOTION, AND BIOLOGICAL VALUE

p0220 Let me conclude by turning to the issue of biological value. Neuroscience has identified several chemical molecules that are, in one way or another, associated with value – dopamine, cortisol, oxytocin, and prolactin. Neuroscience has also identified a number of subcortical neuron nuclei, located in the brainstem and hypothalamus, which manufacture those molecules and deliver them to selected parts of the brain and of the body. The complicated neural mechanics of those molecules is an important topic of neuroscience that many committed researchers wish to unravel. What prompts the release of those molecules? Where do they go exactly? What do they accomplish? But somehow, discussions about all the new facts come up short when turning to the most central questions:

o0110 1. Where is the engine for the value systems
o0120 2. What is the biological primitive value?

p0250 We need to know why things came to be this way.

p0260 The gist of my answers is as follows. Value is indelibly tied to need, and need is tied to life. The valuations we establish in everyday social and cultural activities have a direct or indirect connection with human biology and, in particular, with the processes of life regulation known by the term *homeostasis*. Value relates, directly or indirectly, to survival.

p0270 Because survival means different things in the perspectives of genes, cells, systems, whole organisms, and cultures, the origins of value will appear to be different depending on the target of the observation. Let me begin by considering the whole-organism level.

p0280 The machinery of homeostasis has been designed (obviously, by "designed" I mean achieved by selectional processes over evolutionary time) to protect the integrity of living organisms and, to state it crudely, as far as organisms go, the paramount value for these organisms consists of healthy survival to an age compatible with procreation. Accordingly, I regard the physiological state of tissues within a living organism, specifically, *the state of living tissue within a homeostatic range*, as the deepest origin of biological value and valuations. Toward one extreme of the homeostatic range the viability of living tissue declines and the risk of disease and death increases; toward the other extreme of the range, living tissue flourishes and its function becomes more efficient and economic. States closer to the former extreme are less valuable than states in the middle range and states closer to the latter extreme. The primitive of organism value is inscribed in the physiological parameters of the state itself. It is plausible that other processes and objects acquire their assigned value by reference to this primitive of organism value.

p0290 The values attributed to objects and activities will bear some relation, no matter how indirect or remote, to the maintenance of living tissue within a homeostatic range. As noted, the neurobiology literature tends to be vague regarding the issue of value. Some accounts mention the machinery of punishment and reward as the basis of value, and some remind us of the chemical molecules related to such machinery, but I do not believe that those facts constitute a good answer.

p0300 The origins of value as outlined for a whole organism apply quite well to an individual cell. Value is still defined in terms of a physiological state. However, it is reasonable to wonder how the conditions described for cells and organisms come to be. To approach such an issue, we must consider events that took place in a long ago evolutionary past – a reverse form of engineering that is never easy. We have spent most of our scientific history observing whole organisms and their major components while obfuscating, down below, the gene level where each organism began. And that is the level we must go to in order to discover where the power of homeostasis originates.

p0310 We can begin by considering that, in order to continue their existence over generations, gene networks needed to construct perishable, complex, and yet successful organisms that served as vehicles for their advancement; and that in order for organisms to behave in that successful manner genes must have guided the design of those organisms with some critical instructions.

p0320 My hypothesis is that a good part of those fundamental instructions ended up constructing devices capable of conducting general life regulation (homeostasis), distributing rewards, applying punishments, and helping predict the next situation of an organism – in brief, devices capable of executing what we have come to call emotions, in the broad sense of the term. The early sketch of these devices was first present in organisms without mind or consciousness, in fact without a brain, but the regulating devices attained the greatest complexity in organisms that do have all three: brain, mind, and consciousness. I suspect the empowering instructions were an important engine early in evolution, and everything suggests that they are still in use today, from the level of operations that

regulates our metabolism to the level of human behaviors present in sociopolitical activities and, of course, in economics in the narrow sense of the term.

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