Available online at www.sciencedirect.com
ScienceDirect
Behavioural Brain Research xxx (2008) xxx–xxx
BEHAVIOURAL BRAIN RESEARCH
www.elsevier.com/locate/bbr

Research report

The concept of brain plasticity—Paillard's systemic analysis and emphasis on structure and function (followed by the translation of a seminal paper by Paillard on plasticity)

Bruno Will [a,*], John Dalrymple-Alford [b], Mathieu Wolff [b], Jean-Christophe Cassel [a]

[a] *LINC, UMR 7191, ULP-CNRS, IFR des Neurosciences 037, GDR 2905 CNRS, 12 rue Goethe, F-67000 Strasbourg, France*
[b] *Van der Veer Institute for Parkinson's and Brain Research, and Department of Psychology, University of Canterbury, Private Bag 4800, Christchurch 8140, New Zealand*

Received 21 October 2007; accepted 6 November 2007
Available online 5 December 2007

Abstract

Although rejected for the most part of the 20th Century, the idea of brain plasticity began to receive wide acceptance from the 1970s. Yet there has been relatively little theoretical comment on the definition and use of "plasticity" in the field of neurobiology. An early exception to this lack of critical reflection on neural plasticity was provided by Jacques Paillard in a seminal paper that he published in 1976 [Paillard J. Réflexions sur l'usage du concept de plasticité en neurobiology. J Psychol 1976;1:33–47]. As this valuable contribution was published in French, the present authors provide an English adaptation to help convey his ideas to an international audience, together with a contemporary commentary on this paper. Paillard's definition of the term "plasticity" is probably as pertinent today as it was 30 years ago, especially in terms of its relevance to multiple levels of analysis of brain function (molecular, cellular, systemic). Sadly, Jacques Paillard died in 2006; our comments therefore also include a brief biographical tribute to this outstanding neuroscientist.

Keywords: Brain; Concept; Elasticity; Flexibility; Function; Nervous system; Structure

1. Introduction

The principle of brain plasticity is readily acknowledged in contemporary neuroscience, but its general acceptance is relatively recent, beginning in the 1970s. Notions of neuroplasticity had certainly existed previously (e.g., [5]), but the broad concept became current only after the early findings on enriched environments (e.g., [36]) and visual deprivation (e.g., [18]) had been established. Resistance against the principle of brain plasticity was probably mainly due to the influence of the great Spanish neuroanatomist, Santiago Ramon y Cajal, who had firmly postulated that neural connections in the adult brain are fixed and immutable [35]. From this perspective, it is intriguing that Ramon y Cajal himself had speculated that mental exercise, such as learning a musical instrument, might be associated with an increase in the growth of new axon collaterals and new terminal dendrites [34]. This conjecture, which was made prior to the use of the word "synapse" by Sherrington in 1897, was a forerunner to the more recent speculation on "cell assemblies" [17]. Of course, previously neglected concepts of plasticity are universally endorsed in contemporary neuroscience (e.g., for plasticity of spinal neural circuitry [10]; for discussions of the functional properties of neurogenesis, see [2,14,21]. One challenge facing contemporary neuroscience is, however, the almost unbridled proliferation of examples of "brain plasticity". This apparently simple and attractive concept is instead an extraordinary complex and elusive issue, exacerbated by the fact that the idea is conveyed differently by different subdisciplines and often at multiple levels of analysis (from genetic to behavioural). This important issue was encapsulated by an early theoretical paper on this topic [31]. As many of the inherent problems with plasticity remain unanswered today, the current paper provides a contemporary perspective on Paillard's ideas, accompanied by an English translation of his original article. Many of Paillard's comments are perhaps as relevant today as they were at the original time of writing, because they challenge researchers

* Corresponding author. Tel.: +33 390 242 007; fax: +33 390 241 958.
E-mail address: bruno.will@linc.u-strasbg.fr (B. Will).

0166-4328/$ – see front matter
doi:10.1016/j.bbr.2007.11.008

to address the functional properties of any neural change. Sadly, Jacques Paillard died in July 2006. Partly in tribute to this outstanding behavioural neuroscientist, and partly in view of the importance of neural plasticity today, we hope that these contributions will be of value to the ongoing debate on brain plasticity.

Paillard's seminal paper was published only shortly after the first demonstration of long-term potentiation (LTP) in the mammalian hippocampus [6]. It sought to provide a meaningful definition of "change" that was sufficient to warrant the label (neuro-) "plasticity". Using systemic analysis as his conceptual framework [44], Paillard briefly covered this "new" concept from an elementary to a holistic level of analysis. Jacques Paillard's 1976 paper on neural plasticity [31] was perhaps one of his most significant, amongst a productivity that spanned 150 French and 143 English peer-reviewed articles. His last paper was published in 2006 [39] (see http://jacquespaillard.apinc.org/). Paillard's work covered a wide range of fundamental, psychological and medical issues. Many were reviews or conceptual papers that dealt with the plasticity in the central nervous system (CNS). The 1976 paper was published in the first issue of the *Journal de Psychologie*, and was entitled *"Réflexions sur l'usage du concept de plasticité en neurobiologie"* (Reflections on the use of the concept of plasticity in neurobiology). Given its relevance to CNS plasticity, the English translation of Paillard's 1976 paper will make it more readily available to a broader audience.

Jacques Paillard was born on 5th March 1920 in Nemours, 90 km south-east of Paris. His primary expertise was the neurophysiology of sensorimotor integration and the perception of body space. In 1947, he was recruited to the then relatively new *Centre National de la Recherche Scientifique* (CNRS) in Paris, which had been created in 1939, starting his research work in Alfred Fessard's[1] laboratory. Ten years later, he moved to the Faculty of Sciences in Marseille where he became a full-professor and one of the leading figures in psychophysiology and motor function in France, achieving an international reputation. Paillard is perhaps best known in France for establishing the CNRS Institute of Neurophysiology and Psychophysiology in Marseille in 1965, which provided an innovative model framework for research in cognitive neuroscience: brain functions were investigated at a variety of integrative levels, from cell function to fully integrated behaviour, using both human data and animal models. With Professor Larry Weiskrantz, Jacques Paillard co-founded the European Brain and Behaviour Society (EBBS), in 1968, and was part of an original council that included figures such as Elisabeth Warrington, Giovani Berlucchi, Konrad Ackert and Hans Kuypers. One year later, Paillard was the local organizer of the very first EBBS meeting. The EBBS still plays a major role in the field of behavioural neuroscience (its last meeting was in Triest, Italy, September 2007).

[1] Until the early 1970s, Alfred Fessard was the head of the *Institut Marey* in Paris. At present, there is an *Institut de Neurobiologie Alfred Fessard* in Gif-sur-Yvette, near Paris.

Paillard's definition of plasticity has the great advantage of clarifying a concept that has been used with different meanings by many people. According to Paillard ([31]; see translation at the end of this article), "The term plasticity is only appropriate in terms of the ability of a system to achieve novel functions, either by transforming its internal connectivity or by changing the elements of which it is made" (p. 43 in the French version; caption of Fig. 2). That is, if there is no new function or no structural change underlying this new function, then plasticity is not the appropriate term.

2. The nature of plastic changes: plasticity vs. flexibility

Using this definition, Paillard cautioned that not every change in the neural system is obligatorily plastic. That is, only those changes that are both structural and functional were defined as plastic changes. Functional adaptations based on preprogrammed or expected environmental changes in hard-wired systems, as in many robots, should not be considered examples of plasticity. As he pointed out in a companion paper, for robots, "each control function is coupled with an aid function to endow the system with flexibility", not plasticity ([32], p. 471). By contrast, the control functions of living organisms can show plasticity through some self-governing reorganization of their inner wiring, the assumption being that this reorganization will be the basic substrate of functional modifications.

Structural modifications comprise changes in the structural connectivity network (i.e., the connections enabling interactions between elements of a given system) and changes concerning the constitutive elements of the system themselves, of which neurons are the fundamental units. It is now well-accepted that a substantial number of new brain neurons are generated daily. Paillard like others in the field were unaware of adult neurogenesis; indeed, the early evidence from Altman [3] had been effectively ignored. The existence of neurogenesis is poignant to Paillard's comments concerning changes in the elements of a system. This phenomenon occurs in at least two regions of the adult mammalian brain, the subventricular zone and the dentate gyrus (e.g., [1]). However, to understand the functional impact of neurogenesis [21] one also has to take into account several epigenetic cellular factors such as adrenal corticosteroids (e.g., [8]), gonadal hormones (e.g., [40]) and trophic factors (e.g., [11]) as well as physiological and environmental factors like housing conditions, physical exercise (e.g., [19]) and learning opportunities [20]. Furthermore, survival of newly generated neurons may depend on what happens during an initial post-proliferative period of sensitivity, when the newborn cells are in the process of being integrated into cerebral networks [16,41]. From a functional standpoint, the involvement of neurogenesis in memory formation, such as the encoding of time, is one possibility [2,21]. These recent data on adult neurogenesis could not of course have influenced Paillard's views, but his definition of plasticity is nonetheless clearly relevant. For neurogenesis to reach his criterion of plasticity, definitive demonstration is required that newly generated neurones actually contribute to changes in the functional properties of existing networks or systems.

Please cite this article in press as: Will B, et al., The concept of brain plasticity—Paillard's systemic analysis and emphasis on structure and function (followed by the translation of a seminal paper by Paillard on plasticity), Behav Brain Res (2008), doi:10.1016/j.bbr.2007.11.008

It has, however, been more traditional to think in terms of changes in the properties of neuronal networks that occur within a fixed neuronal population, without considering the introduction of any new neurones. The obvious example here concerns modifications of the functional characteristics of a neural system via changes in the properties of synaptic contacts, as occurs in long-term potentiation (LTP) and long-term depression (LTD). LTP and LTD are associated with lasting structural modifications of the neural network, both at the level of microstructural changes to existing synapses and in terms of synaptogenesis (e.g., [12,22,25,42]). There seems to be a general agreement on the involvement of LTP/LTD and synaptogenesis in memory formation. An elegant study by McNaughton et al. [27] demonstrated that pre-acquisition saturation of LTP to levels that prevented any further potentiation within the hippocampus resulted in impaired spatial reference memory. More recently, it has been demonstrated that passive avoidance learning is able to induce LTP in some hippocampal CA1 synapses and LTD or no modification in others [45]. As concerns LTP, similar changes have been described after fear conditioning within the amygdala [26]. These findings and other lines of converging evidence provide substantial support for the idea that LTP (or LTD) is a process that produces structural changes that give rise to some aspects of memory formation, which therefore provides an example of plasticity. LTP and LTD can thus be considered as plastic changes as these lasting functional changes involve lasting structural changes such as the integration of new AMPA receptors or the growth of new synaptic contacts (e.g., [47]).

Paillard proposed that the concept of plasticity should not be restricted to a modification that in essence would be only functional or only structural. Therefore, expressions such as "functional plasticity" or "structural plasticity" may appear awkward or inadequate when used to account for modifications that are detected only functionally (with no evidence for structural changes) or structurally (with no evidence for functional changes). Thus, what we are tempted to define as functional plasticity should be nothing else than the result of novel properties gained by a system having reorganized at least part of its structural characteristics as a result of the transient action of internal (e.g., physiological) or external (environmental) constraints. The structural modification must be lasting and must result in a lasting functional change, despite the change-triggering internal or external constraints being only temporary. Some robots exhibit functional changes (adaptations), but without any "spontaneous" modification of their internal structure, and therefore fail to exhibit plasticity. For example, Walter's "turtle" ([43]; see also [33], for a recent comment) was preprogrammed to anticipate constraints that it was going to encounter in a particular and well-defined environment. Gaining the capability to respond to these constraints, by integrating their impact, was in fact an *a priori* faculty of the system that had been conceived and/or programmed in order to deal with probable situations. Similarly, Paillard mentions that functional changes due to illness or fatigue are not examples of plasticity as they are not the expression of lasting structural changes. In fact, illness or fatigue just alter normal operations in a system (e.g., loss of efficacy in some information exchanges) that has its established properties modified within an otherwise unchanged structural setup.

3. The origin of plastic changes: plasticity vs. maturation

Maturation is a process by which an organism gains in structural and functional complexity, partly on the basis of genetically determined factors, partly on the basis of its interaction with environmental factors, and which requires constant and changing interactions between genetic and environmental factors. Lasting changes not due to environmental pressure, i.e., those resulting from normal maturation of developing organisms, were excluded by Paillard from his concept of plasticity. Paillard's criterion also excluded a concept such as predeterminism. But what about predisposition (or preparedness; [37])? The extent to which the latter concept can be dissociated from that of plasticity is not clear. In their elegant series of experiments in the Californian white-crowned sparrows showing different region-dependent dialects, Marler and Tamura [23] demonstrated that, within the first 100 days of life, these birds learned the song dialect from the males of their population of origin, when exposed to it. When exposed instead during the same period to males from another population, living in a non-overlapping region and characterized by another dialect, they learned the song dialect of this other population and kept its phonological characteristics for the remainder of their lives. Furthermore, when immature white-crowned sparrows from different populations with different dialects were reared together with no contact to any conspecific adult, they all developed the same song, i.e., a new "subsong" lacking dialect characteristics, yet still recognizable as species-specific. The same song was developed when they were reared in the absence of contact to adults with immature birds from their own population. In this example, developing a predetermined song in the absence of appropriate environmental constraints has probably much less to do with plasticity than learning the dialect of the conspecific population to which these birds are normally exposed; the latter is a "predisposed" (prepared) faculty that probably relies upon plasticity-related changes within the birds' brains. Clearly, the faculty of developing a song, but not that of learning a dialect, is predetermined and does not appear to be a manifestation of plasticity.

Other developmental examples concern the functional consequences of brain damage and the subsequent potential for structural repair and recovery of function. In some instances, factors associated with normal brain maturation may explain differences in recovery of function after the same brain injury in immature vs. mature animals. Oliverio [29] showed that lesions of the septal nucleus in two strains of mice (C57 and SEC), whose brains "mature" at different rates, had different outcomes depending on the age of the animal at the time of surgery. When surgery was performed in adult animals, removal of the septum produced the same problems in emotional reactivity and learning in both strains. Conversely, when surgery was done on the second day of life, only the strain that was neurologically more developed at that age showed a lesion effect. It seems likely that at least part of the difference between these strains was due to

Please cite this article in press as: Will B, et al., The concept of brain plasticity—Paillard's systemic analysis and emphasis on structure and function (followed by the translation of a seminal paper by Paillard on plasticity), Behav Brain Res (2008), doi:10.1016/j.bbr.2007.11.008

the degree of neurological maturation at the time of injury, not a potential difference in plasticity. More generally, specific cerebral regions are known to mature at different rates, irrespective of strain differences. In monkeys, Goldman [15] showed that the magnitude of the behavioural deficits induced by the lesions in the prefrontal cortex can either decrease or increase between 1 and 2 years after surgery as a function of the site of the lesion. When the lesions destroyed the dorsolateral part of the prefrontal cortex, the monkeys literally "grew into" deficits between 1 and 2 years post-surgery, whereas they "grew out" of deficits when the lesion destroyed the orbital part of the prefrontal cortex. It is assumed that deficits will emerge only if the damaged brain region is already at least partly mature – i.e., it has some degree of functional specificity – at the time of surgery. If maturation is incomplete at the time of injury, a deficit may be detected in brain injured animals by comparison to controls only when the damaged structure reaches a more complete degree of functional specificity in the normal (intact) animals. In other words, when the deficits increase over time (i.e., after dorsolateral prefrontal lesions) it is likely, as suggested by Goldman, that this increase is due to the maturation of the dorsolateral prefrontal region in control animals and its absence in animals with lesions; by contrast, when the deficits decrease over time (i.e., after orbital prefrontal lesions), it is more likely that this decrease may reflect neuronal plasticity. Therefore, structural and functional changes are not obligatorily plastic changes, when they are simply associated with normal maturational stages of a given system.

4. The persistence of plastic changes: plasticity vs. elasticity

Paillard used also the term "lasting", although not in the sense of "final", "definitive" or "permanent", to specify the kind of alteration that he considered as plastic [31]. Many researchers have been unwilling to define the term plasticity or to limit its use so "that it may now be used to refer to virtually any long-lasting change in the nervous system which can somehow be attributed to the internal or external environment" ([7], p. 242). Yet, if it does not refer to permanence, the term "lasting" itself is extremely difficult to define. One possibility is to frame plasticity in terms of a balance between the duration of a plasticity-triggering event, which should be short but can be repetitive, and the duration of the resulting changes, which should be relatively long by comparison. It is, however, impossible to propose a minimal duration of changes over which a modification can be considered plastic, as this will depend on the nature of the phenomenon of interest (e.g., LTP, genesis of new dendritic spines, memory formation), that is, the level of organization of the modification and the specific level of the experimental focus (e.g., synapses, neuronal networks, behaviour of an organism). For instance, if one considers the potential substrates of a process such as memory consolidation, and particularly the view that this process relies on reorganization of neuronal networks [13,28], the plasticity-triggering events may last a few seconds to several minutes, but the trace of these events can be retrieved over years and even decades (e.g., in episodic memory). At the synaptic level, sustained activation of a presynaptic fiber over a few milliseconds can modify the functional properties of the related synapse for several hours, days, and perhaps weeks. Yet, plasticity at the synaptic level can (must?) be linked, perhaps even causally, to the plasticity of networks or systems (e.g., [4,9,13,24,28]). It is generally accepted that consolidation of a memory requires activation of intracellular signalling cascades leading eventually to synaptic remodelling (a structural change). It is also rather well accepted that, at the cellular and synaptic levels, one possible contemporary model of memory formation is LTP. Thus, if one considers a memory as a final "product", it appears that, before it is fixed in the long-term, it goes through a series of molecular, cellular and system modifications/reorganizations, which may follow different time courses at different levels of analysis, and could intervene in a timely scaled manner. These changes can all be regarded as manifestations of plasticity, whatever the level.

Thus, a functional modification most likely reflects plasticity when an event-triggered change survives long after the triggering event. But what if it does not, especially if the system recovers its initial properties soon after the triggering event ends? In this latter case, other concepts, such as "elasticity", seem more appropriate, elasticity being defined as "the property of distortable bodies to restore their original shape and volume when the force that was exerted on them has ceased" ([31]; see translation at the end of the present article). The concept of elasticity is easy to understand when the return to the initial functional state occurs immediately or soon after the cessation of the trigger event. However, when this return is protracted or somewhat delayed, the boundary between plasticity and elasticity becomes less certain. For instance, when the behavioural effects of a few weeks of differential housing conditions in rodents (e.g., enriched vs. standard or impoverished housing) last for several weeks after such housing (e.g., [30,46]), are they an example of plasticity, irrespective of whether they may not be more permanent? However, elasticity is exactly what can be characterized by (i) a fast reversibility of the new functional outputs that appear in response to internal or external constraints, or (ii) a fast reversibility of the related structural changes if these modified outputs actually rely upon structural changes, or the absence of modifications at a structural level.

5. Conclusions

With these semantic caveats in mind, one can then accept as plastic any other lasting changes that are both structural and functional, and in which the functional modifications are a direct consequence of the structural reorganizations. Such changes can be observed in young, adult and aged intact living organisms. Furthermore, there are examples of lasting functional recovery after brain damage which also reflect a structural reorganization of the underlying network (e.g., [38]). As these changes in brain-damaged subjects are relatively long-lasting and implement both functional and structural modifications, they can also be considered as plastic.

Paillard's definition of plasticity has the advantage of providing a theoretical frame to the use of the term, in which different forms of plasticity can be distinguished: developmental

Please cite this article in press as: Will B, et al., The concept of brain plasticity—Paillard's systemic analysis and emphasis on structure and function (followed by the translation of a seminal paper by Paillard on plasticity), Behav Brain Res (2008), doi:10.1016/j.bbr.2007.11.008

plasticity (observed pre- and/or post-natally in normal animals under environmental pressure and which cannot be explained by preprogrammed maturation), adaptive plasticity (observed in intact animals, whether young, adult or aged, who are engaged in a learning process), and restorative plasticity (observed in brain-damaged individuals, whether young, adult or aged, who show modifications that the nature of the lesion alone does not explain or/and that the vicariance potential within a system cannot account for). Our general assumption here is that when an experience, whatever its nature, results in enduring functional modifications, and a lasting change in the structural characteristics of a system, whatever the level of analysis to account for it, then the term "plasticity" is an appropriate word to characterize the phenomenon.

Shortly before his death, Jacques Paillard and Bruno Will agreed that the seminal 1976 paper would be more readily available to a wider audience if it was published in English. By offering the Neuroscience community our adaptation of his paper we would like to pay tribute to this influential neuroscientist and provide some insight to his thoughts on "brain plasticity." Given the tendency in contemporary neuroscience to use "plasticity" as a common but ill-defined catch-all expression, his views on how we should limit the use of "plasticity" remain as valid today as they were back in the mid-seventies.

Acknowledgements

The authors are grateful to the *Presses Universitaires de France* for allowing us to include a translation of their material in our article. They also wish to express their gratitude to Dr. Henry Buchtel for the rapid transmission of a pdf version of his 1978 article [7].

References

[1] Abrous DN, Koehl M, Le Moal M. Adult neurogenesis: from precursors to network and physiology. Physiol Rev 2005;85:523–69.
[2] Aimone JB, Wiles J, Gage FH. Potential role for adult neurogenesis in the encoding of time in new memories. Nat Neurosci 2006;9:723–7.
[3] Altman J. Are new neurons formed in the brains of adult mammals? Science 1962;135:1127–8.
[4] Arshavsky YI. The seven sins" of the Hebbian synapse: can the hypothesis of synaptic plasticity explain long-term memory consolidation? Prog Neurobiol 2006;80:99–113.
[5] Bethe A. Plastizität und Zentrenlehre. Handb Norm Path Physiol 1930;15:1175.
[6] Bliss TV, Lømo T. Long-lasting potentiation of synaptic transmission in the dentate area of the anesthetized rabbit following stimulation of the perforant path. J Physiol (London) 1973;232:331–56.
[7] Buchtel HA. On defining neural plasticity. Arch Ital Biol 1978;116(3–4):241–7.
[8] Cameron HA, Gould E. Adult neurogenesis is regulated by adrenal steroids in the dentate gyrus. Neuroscience 1994;61:203–9.
[9] Dudai Y. The neurobiology of consolidations, or, how stable is the engram? Ann Rev Psychol 2004;55:51–86.
[10] Edgerton VR, Tillkaratne NJK, Bigbee AJ, De Leon RD, Roy RR. Plasticity of the spinal neural circuitry after injury. Ann Rev Neurosci 2004;27:145–67.
[11] Emsley JG, Hagg T. Endogenous and exogenous ciliary neurotrophic factor enhances forebrain neurogenesis in adult mice. Exp Neurol 2003;183:298–310.
[12] Engert F, Bonhoeffer T. Dendritic spine changes associated with hippocampal long-term synaptic plasticity. Nature 1999;399(6731):66–70.
[13] Frankland PW, Bontempi B. The organization of recent and remote memories. Nat Rev Neurosci 2005;6:119–30.
[14] Gage FH. Neurogenesis in the adult brain. J Neurosci 2002;22(3):612–3.
[15] Goldman PS. An alternative to developmental plasticity; heterology of CNS structures in infants and adults. In: Stein DG, Rosen JJ, Butters N, editors. Plasticity and recovery of function in the central nervous system. London: Academic Press; 1974. p. 149–74.
[16] Greenough WT, Cohen NJ, Juraska JM. New neurons in old brains: learning to survive? Nat Neurosci 1999;2:203–5.
[17] Hebb D. Organization of behavior. New York: John Wiley; 1949. p. 1–335.
[18] Hubel DH, Wiesel TN, LeVay S. Plasticity of ocular dominance in monkey striate cortex. Philos Trans R Soc Lond Ser B: Biol Sci 1977;278:377–409.
[19] Kempermann G, Kuhn HG, Gage FL. More hippocampal neurons in adult mice living in an enriched environment. Nature 1997;386:493–5.
[20] Leuner B, Mendolia-Loffredo S, Kozorovitskiy Y, Samburg D, Gould E, Shors TJ. Learning enhances the survival of new neurons beyond the time when the hippocampus is required for memory. J Neurosci 2004;24:7477–81.
[21] Leuner B, Gould E, Shors TJ. Is there a link between adult neurogenesis and learning? Hippocampus 2006;16:216–24.
[22] Malenka RC, Bear MF. LTP and LTD: an embarrassment of riches. Neuron 2004;44:5–21.
[23] Marler P, Tamura M. Culturally transmitted patterns of vocal behaviour in sparrows. Science 1964;146(3650):1483–6.
[24] Martin SJ, Clark RE. The rodent hippocampus and spatial memory: from synapses to systems. Cell Mol Life Sci 2007;64:401–31.
[25] Matsuzaki M, Honkura N, Ellis-Davies QC, Kasai H. Structural basis of long-term potentiation in single dendritic spines. Nature 2004;429:761–6.
[26] McKernan MG, Shinnick-Gallagher P. Fear conditioning induces a lasting potentiation of synaptic currents in vitro. Nature 1997;390(6660):607–11.
[27] McNaughton BL, Barnes CA, Rao G, Baldwin J, Rasmussen M. Long-term enhancement of hippocampal synaptic transmission and the acquisition of spatial information. J Neurosci 1986;6(2):563–71.
[28] Moscovitch M, Nadel L, Winocur G, Gilboa A, Rosenbaum RS. The cognitive neuroscience of remote episodic, semantic and spatial memory. Curr Opin Neurobiol 2006;16(2):179–90.
[29] Oliverio A. Functional consequences of infant septal lesions in two strains of mice. Brain Res 1979;175:131–4.
[30] Pacteau C, Einon D, Sinden J. Early environment and dorsal hippocampal ibotenic acid lesions: long term influences on spatial learning and alternation in the rat. Behav Brain Res 1989;34:79–96.
[31] Paillard J. Réflexions sur l'usage du concept de plasticité en neurobiologie. J Psychol 1976;1:33–47.
[32] Paillard J. Le codage nerveux des commandes motrices. Rev E E G Neurophysiol 1976;6(4):453–72.
[33] Pfeifer R. On the role of embodiment in the emergence of cognition—Grey Walter's turtles and beyond. In: Proc. Of the Workshop "The Legacy of Grey Walter 2002". 2002. http://www. ifi.unizh.ch.
[34] Ramon y Cajal S. The Croonian lecture—"La fine Structure des Centres Nerveux". Proc R Soc Lond 1894;55:444–68.
[35] Ramon y Cajal S. Degeneration and regeneration of the nervous system (R.M. May, Trans.). Oxford University Press: London; 1928.
[36] Rosenzweig MR, Bennett EL. Psychobiology of plasticity: effects of training and experience on brain and behavior. Behav Brain Res 1996;78:57–65.
[37] Seligman EP, Hager JL. Biological boundaries of learning. New York: Appleton Century Crofts; 1972. p. 1–480.
[38] Stein DG, Brailowsky S, Will B. Brain repair. Oxford: Oxford University Press; 1995. p. 1–156.
[39] Stenneken P, Prinz W, Cole J, Paillard J, Aschersleben G. The effect of sensory feedback on the timing of movements. Evidence from deafferented patients. Brain Res 2006;1084(1):123–31.
[40] Tanapat P, Hastings NB, Reeves AJ, Gould E. Estrogen stimulates a transient increase in the number of new neurons in the dentate gyrus of the adult female rat. J Neurosci 1999;19:5792–801.

Please cite this article in press as: Will B, et al., The concept of brain plasticity—Paillard's systemic analysis and emphasis on structure and function (followed by the translation of a seminal paper by Paillard on plasticity), Behav Brain Res (2008), doi:10.1016/j.bbr.2007.11.008

[41] Tashiro A, Makino H, Gage FH. Experience-specific functional modification of the dentate gyrus through adult neurogenesis: a critical period during an immature stage. J Neurosci 2007;27:3252–9.
[42] Van Harreveld A, Fifkova E. Swelling of dendritic spines in the fascia dentate after stimulation of the perforant fibers as a mechanism of post-tetanic potentiation. Exp Neurol 1975;49:736–49.
[43] Walter WG. A machine that learns. Sci Am 1951;185:60–3.
[44] Weiss P. Self-differentiation of the basic patterns of coordination. Comp Psychol Monogr 1941;17(4):1–9.
[45] Whitlock JR, Heynen AJ, Shuler MG, Bear MF. Learning induces long-term potentiation in the hippocampus. Science 2006;313(5790):1093–7.
[46] Will B, Galani R, Kelche C, Rosenzweig MR. Recovery from brain injury in animals: relative efficacy of environmental enrichment, physical exercise or formal training (1990–2002). Prog Neurobiol 2004;72(3):167–82.
[47] Yuste R, Bonhoeffer T. Morphological changes in dentritic spines associated with long-term synaptic plasticity. Annu Rev Neurosci 2001;24:1071–89.

doi:10.1016/j.bbr.2007.11.030

Research report

Reflections on the use of the concept of plasticity in neurobiology Translation and adaptation by Bruno Will, John Dalrymple-Alford, Mathieu Wolff and Jean-Christophe Cassel from J. Paillard, J Psychol 1976;1:33–47

Bruno Will [a,*], John Dalrymple-Alford [b], Mathieu Wolff [b], Jean-Christophe Cassel [a]

[a] *LINC UMR 7191, Université Louis Pasteur-CNRS, IFR de Neurosciences 037, GDR CNRS 2905, 12 rue Goethe, F-67000 Strasbourg, France*
[b] *Van der Veer Institute for Parkinson's and Brain Research, and Department of Psychology, University of Canterbury, Private Bag 4800, Christchurch 8140, New Zealand*

Received 21 October 2007; accepted 6 November 2007
Available online 5 December 2007

Abstract

After having underlined the ambiguities of the concept of plasticity and the dangers of its purely metaphoric use in neurobiology, it is suggested that we return to a more precise definition of the structure, the operating principles and the function of the "systemic" unit or "integron" relevant to the particular level of analysis in question. Any change can then be described as a modification of function, a change in the operation principles, or an alteration of the material structure of the system.

It is suggested that the term plastic should be restricted to describing, among the possible variations in the operating principles or the function of a given system, any lasting alteration of the connectivity network of the system under the influence of an external force or environmental constraint. Therefore, systematic or random variations of performance, functional flexibility or the vicarious[1] processes or strategies that can be found in a rigidly wired system are not justified examples of plasticity.

Keywords: Plasticity; Neurobiology; Linguistic

Progress in neurobiology requires an interdisciplinary dialogue. Both the importance and abundance of various facts collected at a variety of levels of analysis of nervous system function suggest that a vertical approach to some questions in neurobiology is now possible. Molecular phenomena at the cellular level can be directly linked to processes that give rise to both basic function and behavioural phenomena that express the products of such integrated function at the level of the whole organism. It is well-known that interplay across different fields of knowledge is often restricted by different semantic limitations. From this perspective, one may question whether the concept of plasticity has been useful. The term is in fashion. A variety of expressions are used such as phenotypic plasticity, synaptic plasticity, morphological plasticity, functional plasticity, plasticity of sensory-motor coordination, behavioural plasticity, etc. Is such a generalisation of the concept justified? Are these different types of plasticity sufficiently precise to be of heuristic value in generating novel hypotheses and experiments so that the concept is useful in neurobiology? This issue merits attention because it immedi-

* Corresponding author.
[1] Vicarious process: "taking over the functions" of damaged tissue (note added by the translators).

Please cite this article in press as: Will B, et al., Reflections on the use of the concept of plasticity in neurobiology, Behav Brain Res (2008), doi:10.1016/j.bbr.2007.11.008