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Speed of Expertise Acquisition Depends upon Inherited Factors

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Abstract: This paper challenges the current dominant view of expertise acquisition by reintroducing inherited factors in the learning process. Studies in experimental psychology have consistently shown that expert performance correlates with the amount of domain-specific knowledge that the experts have acquired through practice. This finding has led to the view that nurture dominates nature with respect to expertise acquisition. We review studies in neurobiology that have shown that the biological processes underlying long-term memory storage engage genetic mechanisms. Thereby, we lay out a framework that provides the basis for re-interpreting psychological data in a psychobiological light. We advance a genetic hypothesis which accounts for individual differences in expertise acquisition. We briefly discuss the consequences of our hypothesis on education.

Keywords:

expertise, gene, neural plasticity, long-term memory, talent

Nurture and Expertise

Understanding the nature of expertise and talent is an important scientific question that has occupied the attention of philosophers, psychologists, biologists, and neuroscientists. Two main strands are apparent in this field of research. On the one hand, scientists supporting the idea of innate talent believe that expertise depends not only on the environment but also on biological mechanisms involving heredity (Galton, 1869; Plomin, 1999). In doing so, those scholars emphasize that nature is a necessary condition for performance to emerge. On the other hand, scientists defending the role of practice have underplayed the role of "nature" and emphasized the importance of "nurture" by pointing to the amount of domain-specific knowledge necessary for becoming an expert (Ericsson, Krampe, & Tesch-Römer, 1993). There has been little constructive interaction between these two strands of research: for example, Eysenck's book on genius (1995) hardly mentions research in the expertise tradition, and a recent "Handbook of Expertise" (Ericsson, Charness, Feltovich, & Hoffman, 2006) hardly mentions results obtained in the talent tradition[1]. The two approaches are not necessarily in opposition, and the goal of this article is to propose a hypothesis explaining how genetic mechanisms contribute to explain individual differences in the acquisition of the knowledge necessary to become an expert.

Expertise is the ability of individuals to perform very efficiently in a variety of domain-specific tasks. It will come as no surprise that the high level of performance attained by experts is the result of cooperation among multiple psychological mechanisms (Ericsson et al., 2006). Research may be organized along two categories. The first category refers to the studies that were conducted to unravel the mechanisms that make experts perform better than neophytes. This research is comparative in essence and assumes that the differences in any one cognitive component are the result of training (e.g., Abernethy, Neal, & Koning, 1994). The second category is concerned with how experts achieve a high

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standard of performance. That is, this research aims to understand how the components supporting the progression towards expertise evolve (Newell & Rosenbloom, 1981; Rainer & Miller, 2000; Subrahmanyam & Greenfield, 1994). Since the works of Binet (1894) on chess players and mental calculators more than a century ago, data point to the central role played by knowledge stored in long-term memory (LTM).

Countless theorists have emphasized how much domain-specific knowledge matters (Charness, 1991; Rikers et al., 2002). Given the amount of domain-specific knowledge necessary to reach the top level of performance in a field, and the fact that it takes time to acquire it, researchers interested in expertise have often emphasized the importance of dedicated training (Chase & Simon, 1973; Ericsson et al., 1993; Gobet & Campitelli, 2007). An extreme conclusion was put forward by Ericsson, Prietula, and Cokely (2007) when they stated that "new research shows that outstanding performance is the product of years of deliberate practice and coaching not of any innate talent or skill" (p. 1). Accordingly, all the major theories of expertise developed in the last couple of decades have put experience at the core of their theoretical framework (Chase & Simon, 1973; Ericsson & Kintsch, 1995; Gobet & Simon, 1996).

The question of memory storage is not only the province of psychology; it is also a field of neurobiology and genetics. While experimental psychologists were unraveling the psychological mechanisms called on to store information, neurobiologists were looking at how events occur at a lower level of analysis. The advances made in the last decades have established a causal relationship between molecular events and behavioral observations. The scientific community concerned with cell and molecular biology is building a new view of memory processing. In striking contrast, few cognitive scientists in the field of expertise have drawn links between neurobiology of memory and expertise acquisition. It seems natural to have a close look at what biology and genetics have to say about memory processing and through it about expertise acquisition.

A Brief Review of the Neurobiology and Genetics of Memory

Nearly half a century ago, Hebb (1949) put forward his cell assembly theory and its related concept of Hebbian learning. Hebb stated his main hypothesis as follows:

> Let us assume that the persistence or repetition of a reverberatory activity (or "trace") tends to induce lasting cellular changes that add to its stability ... When an axon of cell A is near enough to excite a cell B and repeatedly or persistently takes part in firing it, some growth process or metabolic change takes place in one or both cells such that A's efficiency, as one of the cells firing B, is increased. (p. 62).

Decades of research have confirmed Hebb's hypothesis: Neurobiology has identified neural plasticity as the biological mechanisms associating one neuron to another as a result of experience-dependent activity (see Kandel, 2001 for a review). Neural plasticity is underpinned by morphological changes (Bailey & Chen, 1983). The growth of new synapse terminals requires the synthesis of new proteins, which implies *de facto* genetic mechanisms (Bailey, Montarolo, Chen, & Kandel, 1992). The changes underlying memory storage are triggered by experience-dependent stimulations (Montarolo et al., 1986; Nguyen, Abel, & Kandel, 1994). The biological mechanisms in charge of monitoring neural plasticity reorganize the circuit so as to encode the new item into memory. The reorganization of the neural circuit is very accurate and only the neurons and synapses concerned with the ongoing learning are modified (Martin et al., 1997). The process of consolidating new memories goes on not only while we are practicing but also while we are sleeping (Guan, Peng, & Fang, 2004; Stickgold & Walker, 2007). Much research has been conducted on long-term potentiation (LTP), which is defined as a facilitation of transmission that lasts for hours resulting from the simultaneous activity of pre- and post-synaptic elements (Bliss & Lomo, 1973; Cooke & Bliss, 2006). Critical to our discussion, LTP takes place in the hippocampus, the brain structure supposed to serve as a transitional station for storing new memories (Broadbent, Squire, & Clark, 2004; Gupta et al., 2009; Gutbrod et al., 2006; Rolls, Xiang, & Franco, 2005; Wieser & Wieser, 2003).

As Kandel (2001) details, many biochemical compounds and regulatory mechanisms are at work for new memories to be stored durably. Some of them play a crucial role in memory storage and as such have been at the crux of intense research. The compounds of interest include the N-methyl D-aspartate (NMDA) receptors, the cAMP response element binding (CREB) protein (Mayford & Kandel, 1999) and the brain-derived neurotrophic factor (BDNF) (Wibrand et al., 2006). We review hereafter some experiments highlighting the central role that these chemical compounds play in memory storage. The point of the review is not so much to be a comprehensive introduction to the neurobiology of memory as to show that genetic variability at the molecular level affects information storage. Thus, expertise relies both upon the acquisition of domain-specific material and upon genetic, inherited mechanisms. Furthermore, these studies are a crystal-clear illustration that molecular events impact on the behavioral level.

Intriguingly, these data and the one collected since then have not been considered within the field of expertise acquisition. In the next couple of sections, we expose the data necessary to understand the points that lead us to build our hypothesis. First, we report the results of genetic studies conducted to understand how allelic variability of memory-related genes modulate behavioral performance. Then, we report a few studies showing that the regulation of the expression in hundreds of genes is involved in the building up of a new memory record.

Single Gene Variability

NMDA receptors are the recipient of pre-synaptic activity. This critical position as gatekeepers led researchers to examine and demonstrate their key role in neural plasticity (Bear, Kleinschmidt, Gu, & Singer, 1990; Huerta, Sun, Wilson, & Tonegawa, 2000; Larkin et al., 2008; Li, Niu, Jiang, & Hu, 2007; Nakazawa et al., 2002; Roesler et al., 2000; Shimizu, Tang, Rampon, & Tsien, 2000; Tsien, Huerta, & Tonegawa, 1996; Wanisch, Tang, Mederer, & Wotjak, 2005). It is thus natural to assess the extent to which the synthesis of the proteins building these receptors influences subsequent events. Tsien et al. (1996) used adult mice to test how much spatial processing is impaired when NMDA receptors located in the CA1 region of the hippocampus are not synthesized. The results revealed that spatial memory was impaired but that non-spatial memory was not. Another genetic study carried out by Nakazawa et al. (2002) in the CA3 region of the hippocampus showed that NMDA receptors are essential for pattern completion (another visuospatial ability) to take place. Such findings support the view that NMDA receptors should be functional in the hippocampus for spatial memory to work properly. Hence, we could expect that forms of expertise requiring spatial processing, such as chess and mathematics, recruit NMDA receptors to a large extent. This hypothesis is supported by evidence showing that an increase in NMDA receptors leads to a facilitation in memory storage (Stecher, Müller, & Hoyer, 1997). Hence, to some extent, the speed with which new pieces of domain-specific information are stored in the long term depends on the correct synthesis of NMDA receptors.

Many studies have shown that the CREB protein is involved in long-term memory encoding. CREB differs considerably from a receptor such as the NMDA. CREB is a nuclear protein that modulates the transcription of genes with cAMP response binding elements in their promoters. The CREB protein has two forms, one (CREB 1) is an activator and the other (CREB2) is a repressor. Many studies have documented the fact that CREB2 inhibits the effect of CREB1 so that the level of CREB1 has to pass a threshold for the genetic machinery to enter the game (Bartsch et al., 1995; Dash, Hochner, & Kandel, 1990). The experience-dependent protein is CREB1. When stimulations are strong enough, this molecule is activated and binds to promoters triggering the cascade of processes underlying long-term storage. Many studies have been devoted to unravel the role that CREB1 plays in LTM formation (Leutgeb, Frey, & Behnisch, 2005; Mizuno et al., 2002; Pittenger et al., 2002; Pittenger & Kandel, 1998). Bourtchuladze and colleagues (1994)

showed that CREB1 was involved in the formation of long-term memories. Another study of interest to our argument was conducted by Gass et al. (1998). The authors were interested in the impact of differential CREB1 levels on performance in a water maze. In their study, CREB mutant mice were impaired in water maze learning and fear conditioning but devoid of any deficits in the social transmission of food task. This study demonstrates gene dosage dependent learning deficits in mice with hypomorphic CREB alleles. These results are of high relevance to the current discussion as they show that genetic variability in one, crucial component of the chain of events underlying memory storage alters expertise acquisition in a specific task.

Another key stage in the reorganization of neural circuits is the creation of the material used to rewire a network of neurons. The BDNF is a neurotrophin playing various roles in cell functioning, one of which being to modulate plasticity. Two studies illustrate that allelic differences, Valine (Val) vs. Methionine (Meth), may change the speed with which new memories are stored. At the cell level, a study has been conducted to explore how much polymorphism in this protein influences its topographic distribution (location within the neuron), which in turn affects its function. The results revealed that carriers of the Val version of the gene were more likely to cope with memory demands than were carriers of the Met allele (Egan et al., 2003). The study shows that the neurotrophin BDNF has a key role to play in the regulation of experience-dependent synaptic plasticity. In fact, this result seems fairly general, and an fMRI study with humans has demonstrated that allelic variability modulates performance as reflected by the higher level of activity displayed by the carriers of the Val allele (Hariri et al., 2003).

The studies reported above illustrate the fact that an imperfection in the synthesis of a molecule might impair behavioral efficacy. Many other molecules involved in the process of synaptic plasticity are currently the focus of intense research, such as PKA (Huang, Kandel, Varshavsky, Brandon, & Qi, 1995; Yamamoto, Urakubo, Tominaga-Yoshino, & Ogura, 2005); PKC (Watterson, Watson, Meyer, & Lenox, 2002; Zhang et al., 2005); CaMK II (Colbran & Brown, 2004; Mayford, Wang, Kandel, & O'Dell, 1995); and cell adhesion molecules (Benson & Tanaka, 1998; Biederer et al., 2002; Pascual, Pozas, Barallobre, Tessier-Lavigne, & Soriano, 2004). Similar to the picture drawn by the results described above, these studies have yielded data favouring the view that changes at the molecular level have an impact on the organism's ability to store information. Hence, the studies reviewed establish a link between the molecular level and the behavioural level. Inherited factors constrain the ability to learn and specific alleles may help or impair learning. The next section addresses the question of how complex the neural machinery is to be for memory to store information efficiently.

Involvement of Numerous Genes

Gene expression profiling enables scientists to screen the activity of the whole genome at once. Indeed, microarray analysis has provided the scientific community with a unique quantification of the number of genes involved in simple behaviours (Azami et al., 2006; Cavallaro, D'Agata, & Alkon, 2002; Cirelli & Tononi, 2000; Irwin, 2001; Luo et al., 2001; Park, Gong, Stuart, & Tang, 2006; Robles et al., 2003; Tian et al., 2007; Wibrand et al., 2006). Experience-dependent gene transcription in the hippocampus provides a clue about the high number of genes involved in the encoding of new memories.

To have a rough estimate of the number of genes involved in a simple form of learning, such as spatial orientation in a water maze. Haberman, Lee, Colantuoni, Koh and Gallagher (2008) have probed the activity of 15,923 genes. When the rats were completing a spatial learning task 554 genes proved to be differently activated. It is worth noting that microarray analyses were restricted to the CA3 region of the hippocampus. Hence, for the purpose of learning, the cellular machinery can mobilize more than 500 genes. Another estimate, taking into account the duration of memory consolidation has

been provided by Park et al. (2006). The authors have stimulated hippocampus slices with four trains of 100 Hz spaced by 30 s. Gene activity was screened after 30, 60, 90, and 120 minutes following LTP induction. Microarray analyses revealed that a total of 1,664 genes were involved in synaptic plasticity. Another study of interest regarding our hypothesis is the one carried out by Robles et al. (2003). The authors used rats to profile how many genes have changed their level of expression during spatial learning. The results have shown that up to 19 genes, out of the 120 whose activity was screened, have been the targets of either an up or down regulation. More crucially, gene activity profile has been demonstrated to be location dependent. That is, the level of expression of a given gene is differently affected according to the location of the hippocampus wherein activity takes place.

Hence, the genetic profile depends not only on the type of learning but also on the stage and the location where memory processing takes place. The studies mentioned above are part of a growing body of evidence, conducted with the microarray technique (Chowers et al., 2003; Cirelli & Tononi, 2000; Crocker, Costain, & Robertson, 2006; Irwin, 2001), which contributes to demonstrate that cognitive processes depend upon the regulation of the expressions of numerous genes.

Expertise Has a Genetic Basis

It is now well-established that memory records are encoded in neural networks (Alvarez & Squire, 1994; Fuster, 1997; Kelley, 2004; Platel, Baron, Desgranges, Bernard, & Eustache, 2003). It is the reorganization of such networks that enables the brain to store new memories. The studies reviewed above are a sample of the numerous experiments conducted to link learning and genetics; they bring to light two key facts regarding our discussion. First, long-term memory storage requires the regulation in expression of numerous genes. Second, the quantity of activity-regulated genes involved in the process of memory storage varies during the course of events, showing that biological mechanisms are in charge of selecting which genes are to be activated and at which time step. In addition to studies screening the genome, we reported key results from studies in the field of memory storage that bridge the gap between molecular and behavioral levels. By manipulating the alleles coding for the molecules involved in memory storage, scientists have shown that the efficiency with which a new memory record is stored depends upon the correct synthesis and appropriate levels of expression of experience-activated genes. In summary, allelic variation in gene expression have an effect on behavioral performance.

It may be argued that genes are useful only as long as they are activated, an argument supporting the nurture standpoint. For example, a neuron may not use part of its genetic material if this material has not been called on by stimulation: If training does not take place, memory storage does not either. This argument emphasizes the role played by nurture over the one played by nature. Our point arises as a result of the opposite reasoning. For a neuron to act appropriately, it needs to have a functional genetic code. If neurons are unable to synthesize NMDA receptors, they have no possibility to store new spatial memories. Learning emerges from the interaction between genes and experience. An interaction needs *both* parties to be present. A fully functional genetic material is therefore necessary. Allelic variability implements various degrees of match between the individual and the environment.

As memory storage depends upon the cooperation between multiple genes, memory performance is determined by the set of alleles coding for the whole process of memory storage and regulating mechanisms. As a consequence, when considering the behavioral level, it is not so much the virtues of one specific allele that are important but rather how the set of alleles and intracellular mechanisms cooperate as a whole so as to respond appropriately to environmental stimulation (i.e., the activation from a connected neuron).

An advantageous pattern of alleles would enable rapid encoding of selected material, and may allow some individuals to progress faster. Should the set of alleles generate suboptimal performance, then the individual would be slower to acquire the meaningful structures allowing the understanding of, and thus the adaptation to, the environment.

Accounting for Variance in Expertise Acquisition

The core of our hypothesis is that some individuals have a pattern of alleles that allows the best cooperation between the various mechanisms entering the equation of memory storage. We term this pattern the "expertise specific optimal pattern" (ESOP). The ESOP involves different genes from one domain of expertise to another. For example, chess and piano playing require the chunking of different types of memory records. Music reading is the pairing of a visual pattern (a chord such as C-E-G) with a motor action (the three fingers that have to strike the keyboard simultaneously). For music reading to be improved, the chunks have to integrate visual and motor information of increasing complexity. In chess, motor programs are not of interest. Rather, visual symbols (chess pieces) create a net of attack-defense relations within a spatially finite world. It is likely that the exact mechanisms underpinning high performance are different in the two domains of expertise.

The second point is that within one domain of expertise, allelic variability accounts for the differences in speed with which expertise is acquired. This hypothesis provides explanations as to why studies have so far failed to identify a clear list of inherited factors (Ericsson et al., 1993). First, as noted earlier, the level at which experts' performance is measured (i.e., behavioral level) is much higher than the level at which processes are operating (i.e., molecular level). Second, one should not look for a single determining factor but rather for a set of intertwined mechanisms. Behavioral performance is only but the tip of the iceberg.

By using a very large sample of players, Roring and Charness (2007) have found that experts' performance peaks in average at 43.8 years. The more appropriate age to start chess is before 12 years (Gobet & Campitelli, 2007). Players have thus about twenty years to train so as to reach their zenith in performance. How much should a player practice during this period to reach expert level? Gobet and Campitelli (2007) analyzed data from chess players and put forward an estimate of about 11,000 hours. The point of interest is not so much the mean as the variance: Gobet and Campitelli noted that while one player attained master level with 3,016 hours of practice, another needed as much as 23,608 hours. Finally, the authors mentioned the fact that some players had spent more than 25,000 hours of practice without attaining the master level. The ESOP hypothesis suggests a simple explanation to account for this amazing individual variability. Everything else being equal, the maximal performance reached by a player with an advantageous pattern of genes is beyond what the less gifted player can attain, because the latter cannot capture as much domain-specific information. In consequence, considering a definite period of training, some players accumulate enough domain specific knowledge to reach expertise; while others, less gifted, do not reach the threshold.

Consequences and Testing of the Hypothesis

Gathering data that test our hypothesis may seem to be a long way away. However, the experiments reported in this article show that such data have begun to become available. The results collected so far in experimental psychology and in neurobiology highlight different but nevertheless complementary viewpoints of the same picture: learning is sensitive to allelic variability. Beyond bridging a gap between many disciplines concerned with memory processes, our hypothesis offers a possible source to study multilevel processes.

Early theoretical attempts to link molecular events and memory storage trace back to more than a couple of decades (Hawkins & Kandel, 1984). Yet, in spite of many clever

insights, the data and techniques available at the time were not accurate enough to enable scientists to draw a clear picture of the molecular-behavioral link. The key issue is to conceptualize the dynamics between levels of organizations: how much of behavior can be understood by analyzing its molecular components and chemical reactions? We believe that the numerous levels of analysis between molecules and behavior have hidden the extent to which the organization of the lower levels affects what psychologists observe at the higher level (e.g., response times and accuracy). This may be the reason why many psychologists have failed to identify the source of talent by analyzing cohorts of behavioral data. The hypothesis not only accounts for the sometimes discrepant results regarding talent but also provides a way to investigate how different levels of activity (e.g., molecular, cell, neural networks) collaborate to generate a discrete, identifiable behavior.

The second major impact of the ESOP hypothesis concerns education. It has been argued that the notion of talent deters people from entering deliberate practice if the first results are not positive. The outcome of a talent policy is often a disaster (Sloboda, 2000). We agree with Sloboda about the fact that talent is not the central factor in deciding for a training strategy. Even though we emphasize the role of inherited factors in attaining the highest levels of expertise, we do not downplay the role of practice. Thus, our view of talent, in domains such as chess and music, does not entail that one reaches expertise without hard and difficult training. However, we believe that denying the existence of a biologically implemented form of talent is denying individual differences and as such making teaching policies far too rigid. While the role of individual differences is widely acknowledged, most educational regimens impose the same content, structure, and lectures to all individuals. In our view, the ability of the individuals to learn is proportional to the biological ability to encode new information, which may vary across domains of learning. Current developments in instructional computer-based technology offer good prospects of developing curricula that are tailored to the abilities and interests of different individuals, and thus help maximize their chances of learning (Gobet & Wood, 1999).

Conclusion

In this article, we put forward the hypothesis that much of the variance in speed of expertise acquisition is accounted for by variations in memory-related allelic profiles. A favorable pattern of alleles enables the carrier to encode domain-specific practice with much more ease and speed. Our hypothesis does not preclude the influence of other factors, such as IQ, on the acquisition of expertise (Grabner, Neubauer, & Stern, 2006; Plomin & Spinath, 2004); nor are we reducing expertise to memory storage. Our point is that a phenomenon typically considered to be part of nurture is actually partly underpinned by inherited mechanisms. Consequently, the place of nature in expertise acquisition is not to be denied anymore, even when simple forms of learning are involved. As such, our hypothesis introduces genetics and inherited factors at the core of expertise acquisition and puts a cloud over the dominant view that "new research shows that outstanding performance is the product of years of deliberate practice and coaching not of any innate talent or skill" (Ericsson et al., 2007, p. 1).

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Notes

[1] However, such interactions occasionally occur. See for example the BBS target article by Howe, Davidson, and Sloboda (1998) titled "Innate talents: Reality or myth?" and the following commentaries, or the exchanges in a special issue on "Nature, Nurture, and Sport Performance" in the International Journal of Sport Psychology, Vol. 38, Issue 1, 2007.

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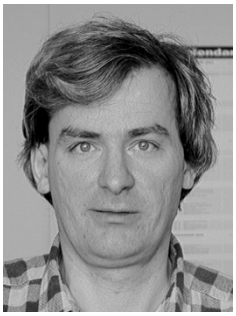

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