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Language Acquisition: Do as You Hear

Dispatch

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It is not uncommon to recognize a specific action by the sound it creates. Neurons have been discovered in monkey premotor cortex that may contribute to this ability; they respond to both performing an action and hearing its action-related sound, and may be critical for communicating with others, learning gestures and even acquiring language.

The study of how we understand the meaning of actions performed by others has seen a resurgence in the last few years, triggered in part by the discovery of a population of neurons that becomes active both when a monkey executes a specific action and when it sees another individual make a similar movement [1-3]. The existence of these so-called 'mirror' neurons has given credence to the direct-matching hypothesis, which stipulates that actions are understood because observation of an action activates the same neural circuitry required to perform that action. For example, a single cell that discharges when a monkey grasps an object with its fingers would also discharge when it sees another monkey picking up a small fruit. The monkey would then recognize that specific movement because it mapped the observed action onto its own neural motor representation. The existence of mirror neurons in humans was recently suggested by transcranial magnetic stimulation (TMS) and brain imaging studies, which showed that the observation of complex actions induces changes in motor cortex excitability [4-6] and activates brain areas involved in the generation of observed movements [7].

It has been proposed that imitation abilities in humans evolved out of the mirror system [8,9]. A recent study [10] has now provided support for an involvement of mirror neurons in the acquisition of language, even without the requirement of visual input. Kohler, Rizzolatti and colleagues [10] recorded single units in the monkey premotor cortex (area F5) and found a population of neurons that discharge when the monkeys perform, see or hear the same action. These 'audiovisual' mirror neurons can show exquisite selectivity. For example, the authors describe a cell that discharged when a monkey broke a peanut, saw an experimenter break a peanut or heard a peanut being broken out of view. However, observation of similar actions, or exposure to their associated sounds, did not modulate the firing rate of this neuron. These observations within individual cells were confirmed in a population analysis, where it was shown that neurons could discriminate between different action sounds.

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Furthermore, the specific action-related sounds associated with a given neuron elicited the strongest responses during the observation and execution of that preferred action.

These data have important implications, not only for understanding actions, but also because of the insights they provide into how language may develop in humans. Area F5, where audiovisual mirror neurons are located, is the monkey homologue of human motor speech area BA44 (Broca's area) [11], and imaging data in humans have revealed the presence of an observation–execution matching system within Broca's area [7]. So, in addition to strongly suggesting the involvement of mirror cells in imitation and action understanding, the new data of Kohler *et al.* [10] indicate that the human premotor mirror neuron system may very well be involved in the imitation and acquisition of speech [12].

Indeed, it was recently reported that passive speech listening in human subjects leads to increased excitability of the primary motor cortex representation of tongue muscles [13]. In that study, TMS-induced motor-evoked potentials from the tongue were significantly increased when listeners heard words containing a double 'r' consonant, suggesting a resonance mechanism similar to that reported during action observation. Although imaging studies have revealed no activation of primary motor cortex when subjects passively view actions being performed [14], it has been shown that premotor activation can affect the excitability of the primary motor cortex [15]. The results of Kohler et al. [10] add further support to the notion that speech acquisition may involve a mechanism whereby speech listening activates speech motor centers, in line with the motor theory of speech perception [16]. Indeed, the discovery of audiovisual mirror neurons [10] shows that auditory input can be integrated by the cortical areas involved in speech production.

A number of interesting inquiries and experiments come to mind to follow-up on the concept of the mirror cell system being multi-modal and critical for language acquisition. Most humans are left-hemi-sphere dominant for vocalizations and language. This might be the consequence of an asymmetry in the mirror cell system, with a left-hemispheric predominance. If so, inter-hemispheric differences in the mirror cell system should be demonstrable in humans, even though none has been noticed in monkeys. Anatomical studies demonstrating interhemispheric differences in the size of Broca's area in humans might index such asymmetries in the mirror cell system. Furthermore, most humans are righthanded, but preferred use of the right hand generally does not develop in humans until the second year of life and clear asymmetry in the use of one hand during the first year of life is generally considered a neurological sign of congenital or perinatal brain damage. The human predominance of right-handedness may develop as a secondary consequence of the preferential activation of the left-hemispheric premotor mirror cell system during gesture and vocalization - language acquisition.

Speech production or exposure to speech sounds can facilitate the cortico-spinal projection of the dominant, but not of the non-dominant hemisphere in humans [17]. Longitudinal developmental studies to assess when such lateralization develops would be most interesting.

If the mirror cell system plays a critical role in language acquisition, delays in language acquisition in the blind ought to be expected. Indeed, language development in blind children has been extensively studied and shown to be abnormally delayed [18]. In addition, blind children tend to use many formulae and imitations in their speech. One possible explanation would be to consider such verbal routines and stereotypic speech the equivalent of copied gestures. Rather than imitating in gestures, blind children reproduce chunks of maternal speech associated with particular contexts, possibly because of the activation of mirror cells by the sounds associated with actions now demonstrated by Kohler *et al.* [10].

Such auditory activation of the mirror cells in blind children might drive language acquisition, but be less effective than the combined visual and auditory activation in the sighted, hence the delay in language acquisition in the blind. Interestingly, congenitally blind subjects often show no definite preferred handedness, possibly because of the missing association between gestures and language acquisition. What, then, might happen in blind-deaf children? How did a deaf-blind individual like Laura Bridgeman acquire language? She did so with substantial delay and using tactile exploration as a major part of her learning process. Are mirror cells responsive not only to sound and sight, but also to touch and other sensory stimuli possibly associated with a given performed or observed action?

Finally, pathological, congenital dysfunction of the mirror cell system in humans would be expected to dramatically affect social interactions, as a result of the disruption of gesture interpretation and acquisition. Furthermore, abnormal mirror cells would lead to poor language development. Autism might be the clinical manifestation of the congenital dysfunction of the mirror cell system [19]. Congenitally blind children that have normal mirror cells but lack the visual input to drive them show autism-like behaviors in addition to language acquisition delays (for review see [18]). Clinical differences in autism spectrum disorders might be the consequence of differential dysfunction of various mirror cell subpopulations, for example those that respond to visual versus those that respond to auditory or other sensory stimuli. Studies on the physiology of the mirror cell system in autism might provide critical insight into the pathophysiology and possible therapeutic strategies for this disorder. Furthermore, genetic studies on the development and differentiation of mirror neurons may provide novel insights into the cause of these disorders and into the fundamental basis of the development of human language.

References

- di Pellegrino, G., Fadiga, L., Fogassi, L., Gallese, V. and Rizzolatti, G. (1992). Understanding motor events: a neurophysiological study. Exp. Brain Res. 91, 176–180.
- Gallese, V., Fadiga, L., Fogassi, L. and Rizzolatti, G. (1996). Action recognition in the premotor cortex. Brain 119, 593–609.

- Rizzolatti, G., Fadiga, L., Gallese, V. and Fogassi, L. (1996). Premotor cortex and the recognition of motor actions. Cognit. Brain Res. 3, 131–141.
- Fadiga, L., Fogassi, L., Pavesi, G. and Rizzolatti, G. (1995). Motor facilitation during action observation: a magnetic stimulation study. J. Neurophysiol. 73, 2608–2611.
- Strafella, A.P. and Paus, T. (2000). Modulation of cortical excitability during action observation: a transcranial magnetic stimulation study. Neuroreport 11, 2289–2292.
- Gangitano, M., Mottaghy, F.M. and Pascual-Leone, A. (2001). Phase-specific modulation of cortical motor output during movement observation. Neuroreport 12, 1489–1492.
- Iacoboni, M., Woods, R.P., Brass, M., Bekkering, H., Mazziota, J.C. and Rizzolatti, G. (1999). Cortical mechanisms of human imitation. Science 286, 2526–2528.
- Rizzolatti, G., Fogassi, L. and Gallese, V. (2001). Neurophysiological mechanisms underlying the understanding and imitation of action. Nat. Rev. Neurosci. 2, 661–670.
- Koski, L., Wohlschläger, A., Bekkering, H., Woods, R.P., Dubeau, M.C., Mazziota, J.C. and Iacoboni, M. (2002). Modulation of motor and premotor activity during imitation of target-directed actions. Cereb. Cortex 12, 847–855.
- Kohler, E., Keysers, C., Umiltà, M.A., Fogassi, L., Gallese, V. and Rizzolatti, G. (2002). Hearing sounds, understanding actions: action representation in mirror neurons. Science 297, 846–848.
- Petrides, M. and Pandya, D.N. (1994). Comparative architectonic analysis of the human and the macaque frontal cortex. In: Handbook of Neuropsychology, F. Boller and G. Grafman, eds. (Elsevier, Amsterdam), pp. 17–58.
- 12. Fitch, W.T. (2000). The evolution of speech: a comparative review. Trends Cogn. Sci. 4, 258–267.
- Fadiga, L., Craighera, L., Buccino, G. and Rizzolatti, R. (2002). Speech listening specifically modulates the excitability of tongue muscles: a TMS study. Eur. J. Neurosci. 15, 399–402.
- Grezes, J. and Decety, J. (2001). Functional anatomy of execution, mental simulation, observation and verb generation of actions: a meta-analysis. Hum. Brain Mapp. 12, 1–19.
- Munchau, A., Bloem, B.R., Irlbacher, K., Trimble, M.R. and Rothwell, J.C. (2002). Functional connectivity of human premotor and motor cortex explored with repetitive transcranial magnetic stimulation. J. Neurosci. 22, 554–561.
- 16. Liberman, A.M. and Whalen, D.H. (2002). On the relation of speech to language. Trends Cognit. Sci. 4, 187–196.
- Tokimura, H., Tokimura, Y., Oliviero, A., Asakura, T. and Rothwell, J.C. (1996). Speech-induced changes in corticospinal excitability. Ann. Neurol. 40, 628–634.
- Perez-Pereira, M. and Conti-Ramsden, G. (1999). Language development and social interaction in blind children. (East Sussex: Psychology Press).
- Williams, J.H., Whiten, A., Suddendorf, T. and Perrett, D.I. (2001). Imitation, mirror neurons and autism. Neurosci. Biobehav. Rev. 25, 287–295.