3. Sterflinger, K. (2000). Fungi as geologic agents. Geomicrobiol. J. *17*, 97–124.
4. Rhee, Y.J., Hillier, S., and Gadd, G.M. (2012). Lead transformation to pyromorphite by fungi. Curr. Biol. *22*, 237–241.
5. Hawksworth, D.L. (2001). The magnitude of fungal diversity: the 1.5 million species estimate revisited. Mycol. Res. *105*, 1422–1432.
6. Kis-Papo, T., Grishkan, I., Oren, A., Wasser, S.P., and Nevo, E. (2001). Spatiotemporal diversity of filamentous fungi in the hypersaline Dead Sea. Mycol. Res. *105*, 749–756.
7. Gleeson, D.B., Clipson, N., Melville, K., Gadd, G.M., and McDermott, F. (2005). Characterization of fungal community structure on a weathered pegmatitic granite. Microbial Ecol. *50*, 360–368.
8. Gadd, G.M. (2007). Geomycology: biogeochemical transformations of rocks, minerals, metals and radiounuclides by fungi, bioweathering and bioremediation. Mycol. Res. *111*, 3–49.
9. Ruisi, S., Barreca, D., Selbmann, L., Zucconi, L., and Onofri, S. (2007). Fungi in Antarctica. Rev. Environ. Sci. Biotechnol. *6*, 127–141.
10. Rooney, D., Hutchens, E., Clipson, N., Baldini, J., and McDermott, F. (2010). Microbial community diversity of moonmilk deposits at Ballynamintra cave, Co. Waterford, Ireland. Microb. Ecol. *60*, 753–761.
11. Burford, E.P., Fomina, M., and Gadd, G.M. (2003). Fungal involvement in bioweathering and biotransformation of rocks and minerals. Mineralogical Mag. *67*, 1127–1155.
12. van Schöll, L., Kuyper, T.W., Smits, M.M., Landeweert, R., Hoffland, E., and van Breemen, N. (2008). Rock-eating mycorrhizas: their role in plant nutrition and biogeochemical cycles. Plant. Soil *303*, 35–47.
13. Burford, E.P., Kierans, M., and Gadd, G.M. (2003). Geomycology: fungi in mineral substrata. Mycologist *17*, 98–107.
14. Smits, M.M., Herrmann, A.M., Duane, M., Duckworth, O.W., Bonneville, S., Benning, L.G., and Lundstrom, U. (2009). The fungal–mineral interface: challenges and considerations of micro-analytical developments. Fung. Biol. Rev. *23*, 122–131.
15. Fomina, M., Burford, E.P., Hillier, S., Kierans, M., and Gadd, G.M. (2010). Rock-Building Fungi. Geomicrobiol. J. *27*, 624–629.
16. Papanikolaou, N.C., Hatzidaki, E.G., Belivanis, S., Tzanakakis, G.N., and Tsatsakis, M. (2005). Lead toxicity update. A brief review. Med. Sci. Mon. *11*, 329–336.
17. Gadd, G.M. (2010). Metals, minerals and microbes: geomicrobiology and bioremediation. Microbiology *156*, 609–643.

[1]Environmental Microbiology Group, School of Biology and Environmental Science and Earth Institute, University College Dublin, Belfield, Dublin 4, Ireland. [2]School of Earth and Environment, The University of Western Australia, 35 Stirling Highway, Crawley, WA 6009, Australia.
*E-mail: nicholas.clipson@ucd.ie

DOI: 10.1016/j.cub.2011.12.037

Social Neuroscience: More Friends, More Problems…More Gray Matter?

The social brain hypothesis generically posits that increasing social group size relates is associated with an increase in neocortex size. A new study identifies, within a species, the specific neural circuit that may confer the primate ability to manage social relationships as they increase in number.

Maia ten Brink and Asif A. Ghazanfar

The oil industrialist, John D. Rockefeller, once said that "The ability to deal with people is as purchasable a commodity as sugar or coffee and I will pay more for that ability than for any other under the sun". It is unclear how many people Rockefeller actually bought in his lifetime, but at the very least he recognizes that the ability to interact effectively with them would be more valuable than anything else in the world. Who could argue with that? Consider the many different relationships in your life — your parents, siblings, extended family, friends, colleagues, competitors, and so on. As for all primates, our lives are an intricate web of relationships; every relationship is unique and it seems for each we are performing an energetically costly balancing act. Naturally, adding more individuals to one's web increases the effort required to maintain all. How do we manage this? There is, of course, the banal suggestion that it is our bigger brains that confer this ability. While there is a positive correlation between neocortex size and social group size across primate species [1], this is a rather coarse measure and not particularly illuminating — there are a number of other behavioral variables that also correlate with the size of the neocortex. A recent study by Sallet and colleagues [2] gives us greater insights into what *specific* neural circuits may mediate the cognitive balancing act required for increasing the size of one's social group.

Using magnetic resonance imaging (MRI) scans, Sallet *et al.* [2] investigated whether group size is related to brain differences in rhesus monkeys (*Macaca mulatta*). According to veterinary considerations, 23 monkeys were assigned to groups ranging in size from one to seven individuals. The authors capitalized on this restructuring of their institution's monkey colony by scanning each monkey's brain 15 months after the group assignments and then asking: is there a correlation between an individual's social group size and the neocortex? They compared differences in an individual brain's gray matter density relative to a group-averaged brain by measuring where, and how much, the imaged brain needed to be expanded or compressed in order to match the average brain. The 'determinant' of the resulting matrix is a scalar value that can be used as a dependent variable and regions which showed a >5 mm^3 difference from the average brain were identified as significant. Remarkably, the authors found that *specific* regions of the neocortex varied in the gray matter density according to social group size. The regions with increased gray matter are known to be important for processing social signals (facial expressions, eye gaze, vocalizations) in monkeys [2]: the superior temporal sulcus, the superior and inferior temporal gyri, the amygdala and the rostral prefrontal cortex (and we know that most are directly connected with each other [3]). For every additional member of a social group, density increased by about 5%.

This pattern of results is consistent with three recent human studies relating social network size (as measured by Facebook or questionnaires) to the superior temporal sulcus [4], the amygdala [5] and the prefrontal cortex [6]. Nevertheless, to lend credence to the idea that these are indeed regions involved in the task of balancing social

relationships, Sallet *et al.* [2] measured whether social rank influenced gray matter density independent of group size. They found that monkeys with a higher rank had greater gray matter density in the inferior temporal gyrus and the rostral prefrontal cortex. What they did not find is also intriguing. Despite looking for such an effect, the authors did not find any relationship between the so-called 'mirror neuron system' and social group size. Some hypothesize that the mirror neuron system plays a critical role in social cognition, possibly by inferring the actions of others through simulation [8]. Thus, it is surprising that there were no changes in gray matter density in any of the core regions comprising this system.

Overall, the main finding of Sallet *et al.* [2] is that there is a relationship between specific neocortical circuits, their size and the number of individuals in a group. What the data do not tell us is whether the differences in gray matter density are *caused* by differences in social group size. That is, did the assignment to the bigger groups lead to increases in gray matter density? Or did the monkeys with bigger social circuits end up in the bigger groups because they could handle the complexities better? Although the authors seem certain that monkeys were assigned to groups according to criteria orthogonal to an individual monkey's sociality [7], all veterinary assignments to different groups must take sociality into account as fighting between macaque monkeys can be quite vicious (both in the wild and captivity). Furthermore, the reasons some monkeys get along (or do not get along) with others are not easily related to their personality measures and are often impossible to discern. Thus, the lack of random assignment of monkeys to different group sizes, and the lack of MRI data before the group assignments were made, preclude us from knowing whether increasing group size causes the increase in gray matter density.

That said, it is well-established that the adult brain is quite plastic and that this plasticity is reflected in changes in specific brain regions. For example, a recent combined MRI–histochemical study of mice trained to perform one of two spatial tasks showed task- and region-specific gray matter density changes (using the same measure as Sallet *et al.* [2]) and that these changes were associated with the remodeling of neuronal processes as measured by a stain for a growth-associated protein (GAP-43) [9]. Like task-induced gray matter changes in mice, it should be the case that if the group size in primates is the cause of the gray matter increases in regions processing social signals, then there should be evidence that an individual's ability to interact within the group changes (gets better) with time along with a corresponding increase in gray matter density. Sallet *et al.*'s [2] study provides both the methodological and scientific framework for such a longitudinal study. It also begs the question of whether or not the modification of these circuits requires interactions with only conspecifics or if any social agent would do. Do humans, for instance, get the same gain of gray matter density increases from interacting with other species, such as dogs? Conversely, in dogs, we know that human contact does not fully replace the need for socialization with other dogs, but it does reduce the effects of early deprivation [10]. Does such differential early experience impact this very same 'group size-related' neocortical circuit?

Identifying the neural circuits related to social group size also lays the foundation for studying a deeper issue: the content of social brain networks. The typical interpretation of the social brain hypothesis presents a view of primates as biologically prepared for social signals such as faces and voices as well as for forms of social engagements that require mental representations of abstract concepts like family relations and alliances in order to negotiate the social landscape. An alternative hypothesis suggests that individuals do not need to hold abstract concepts of family relations and alliances 'in mind' because they can assess circumstances by directly monitoring what is happening around them [11]. According to this view, the active perception of on-going spatial and temporal structure — the statistics — of interacting primates within a social group obviates the need for high level processing involving mental representations. Individuals can use this on-going structure as an accurate and always up-to-date model (for example [12]), allowing for more efficient action selection and execution. With the findings of Sallet *et al.* [2] we are now poised to directly investigate this and other pressing issues regarding the mechanisms of social cognition.

References

1. Dunbar, R.I.M., and Schultz, S. (2007). Evolution in the social brain. Science *317*, 1344–1347.
2. Sallet, J., Mars, R.B., Noonan, M.P., Andersson, J.L., O'Reilly, J.X., Jbabdi, S., Croxson, P.L., Jenkinson, M., Miller, K.L., and Rushworth, M.F.S. (2011). Social network size affects neural circuits in macaques. Science *334*, 697–700.
3. Ghazanfar, A.A., and Santos, L.R. (2004). Primate brains in the wild: The sensory bases for social interactions. Nat. Rev. Neurosci. *5*, 603–616.
4. Petrides, M., and Pandya, D.N. (2007). Efferent association pathways from the rostral prefrontal cortex in the macaque monkey. Neurosci. *27*, 11573–11586.
5. Kanai, R., Bahrami, B., Roylance, R., and Rees, G. (2011). Online social network size is reflected in human brain structure. Proc. R. Soc. B, in press.
6. Bickart, K., Wright, C.I., Dautoff, R.J., Dickerson, B.C., and Barrett, L.F. (2011). Amygdala volume and social network size in humans. Nat. Neurosci. *14*, 163–164.
7. Lewis, P.A., Rezaie, R., Brown, R., Roberts, N., and Dunbar, R.I.M. (2011). Ventromedial prefrontal volume predicts understanding of others and social network size. Neuroimage *57*, 1624–1629.
8. Gallese, V., Keyser, C., and Rizzolatti, G. (2004). A unifying view of the basis of social cognition. Trends in Cognitive Sci. *8*, 396–403.
9. Lerch, J.P., Yiu, A.P., Martinez-Canabal, A., Pekar, T., Bohbot, V.D., Frankland, P.W., Henkelman, R.M., Josselyn, S.A., and Sled, J.G. (2011). Maze training in mice induces MRI-detectable brain shape changes specific to the type of learning. Neuroimage *54*, 2086–2095.
10. Fox, M.W., and Stelzner, D. (1967). The effects of early experience on the development of inter and intraspecies social relationships in the dog. Anim. Behav. *15*, 377–386.
11. Barrett, L., Henzi, P., and Rendall, D. (2007). Social brains, simple minds: does social complexity really require cognitive complexity? Phil. Trans. R. Soc. B *362*, 561–575.
12. Turesson, H.K., and Ghazanfar, A.A. (2011). Statistical learning of social signals and its implications for the social brain hypothesis. Interaction Studies *12*, 397–417.

Neuroscience Institute, Departments of Psychology and Ecology & Evolutionary Biology, Princeton University, Princeton, NJ 08540, USA.
E-mail: asifg@princeton.edu

DOI: 10.1016/j.cub.2011.12.042