HPLI #764815, VOL 24, ISS 1

Broadening the Scope of Cultural Neuroscience

Meghan L. Meyer, Baldwin M. Way, and Naomi Eisenberger

QUERY SHEET

This page lists questions we have about your paper. The numbers displayed at left can be found in the text of the paper for reference. In addition, please review your paper as a whole for correctness.

Q1. Au: Fincher (2008) and Gangestad (2011) do not have matching references. Provide references or remove citations.
Q2. Au: Suffredini & O'Grady (1999) does not have a matching reference. Provide reference or remove citation.
Q3. Au: Roberts et al. (2008): Provide reference or remove citation.
Q4. Au: Gusnard (2001): Provide reference or remove citation.
Q5. Au: The Van Boven et al. reference does not list Welch as an author; please make citation and reference consistent.
Q6. Au: Please list the final draft year for Meyer et al. here.
Q7. Au: Fincher et al. does not have a matching text citation; please add citation or delete reference.
Q8. Au: Gangestad, Haselton, & Buss does not have a matching text citation; please add citation or delete reference.
Q9. Au: Gangestad & Thornhill does not have a matching text citation; please add citation or delete reference.
Q10. Au: Gusnard et al. does not have a matching text citation; please add citation or delete reference.
Q11. Au: Meyer et al.: Please list the final draft year for the manuscript.
Q12. Au: Singles does not have a matching text citation; please add citation or delete reference.
Q13. Au: Suffredini et al. (1999) does not have a matching text citation; please add citation or delete reference.
Q14. Au: Van Boven, Loewenstein, & Dunning (2005) does not have a matching text citation; please add citation or delete reference.

TABLE OF CONTENTS LISTING

The table of contents for the journal will list your paper exactly as it appears below:

Broadening the Scope of Cultural Neuroscience
Meghan L. Meyer, Baldwin M. Way, and Naomi Eisenberger

Psychological Inquiry, 24: 1–6, 2013

ISSN: 1047-840X print / 1532-7965 online
DOI: 10.1080/1047840X.2013.764815

Psychology Press
Taylor & Francis Group

Broadening the Scope of Cultural Neuroscience

Meghan L. Meyer
Psychology Department, UCLA, Los Angeles, California

Baldwin M. Way
Psychology Department, The Ohio State University, Columbus, Ohio

Naomi Eisenberger
Psychology Department, UCLA, Los Angeles, California

Chiao, Cheon, Pornpattanangkul, Mrazek, and Blizinsky offer a comprehensive review of cultural neuroscience research. For such a young field, cultural neuroscience has made great strides in the effort to understand the neural and genetic mechanisms underlying cultural differences in psychology. Here, we pose a set of questions that, if addressed in the future, may help develop the field. First, can cultural neuroscientists more deeply probe how environmental factors, such as pathogen threats, may have influenced genetic selection and, in turn, cultural differences in psychology (i.e., the culture–gene coevolutionary theory)? Second, can cultural neuroscientists help unravel whether and how aspects of cultural psychology are susceptible to change? Third, what can a cultural neuroscience perspective give back to other, related disciplines such as social cognitive neuroscience, genetics, and psychology more broadly?

Can Cultural Neuroscience Test the Culture-Gene Coevolutionary Theory?

As Chiao et al. mention, the guiding biological theory of why differences in psychology persist across cultures is that through the course of evolution, different psychological phenotypes may have better ensured survival in different geographic regions. Although provocative, to our knowledge, this theory tends to be used as a framework to understand observed differences in psychology across cultures. The next step is to empirically test models of how cultural differences in psychology may have emerged from genetic selection. To this end, neuroscience methods may allow a closer examination of the ideas behind culture–gene coevolution.

For example, under the umbrella of culture–gene coevolutionary theory is the parasite–stress theory of sociality, which suggests that the threat of infectious and parasitic diseases led to psychological traits that prioritize connection to in-group members and avoidance of out-group members, two characteristics associated with collectivism when in-group/out-group distinctions are based on personal relationships (e.g., kin, friends, community vs. strangers; see Brewer & Yuki, 2007, for a review). The underlying idea here is that preferential association with in-group members and avoidance of out-group members is important for minimizing the spread of infection from novel pathogens. To date, support for this theory and its link to cultural differences in psychology has been correlational. Studies associate infectious disease prevalence, as well as pathogen prevalence, across global regions and nations to greater collectivism (vs. individualism; Fincher, 2008; Gangestad, 2011) and conformity (Murray, Trudeau, & Schaller, 2011). In addition, regions of the world with more pathogens also have more individuals carrying alleles that have been associated with collectivism (Chiao & Blizinsky, 2010; Way & Lieberman, 2010).

Although the mechanism underlying the relationship between pathogen prevalence and collectivism is not yet clear, some studies suggest that it may be mediated, in part, by inflammatory processes—the immune system's first line of defense against pathogens, which may increase sensitivity to social cues. As evidence for this possibility, exposing human subjects to a low-dose bacterial endotoxin, known to increase inflammatory activity in a safe and time-limited manner (Andreasen et al., 2008; Suffredini & O'Grady, 1999), has been shown to increase sensitivity to negative social cues relevant to in-group status and out-group avoidance, two processes associated with collectivism. For instance, individuals who showed a greater inflammatory response to endotoxin showed greater pain-related neural activity (dorsal anterior cingulate cortex [dACC], anterior insula [AI]) to social exclusion (Eisenberger, Inagaki, Rameson, Mashal, & Irwin, 2009), which may reflect increased sensitivity to threats to in-group status, which is heightened among individuals from collectivistic cultures (Markus & Kitayama, 1991). Similarly, individuals exposed to bacterial endotoxin versus placebo showed increased threat-related neural (amygdala) sensitivity to threatening images of strangers (Inagaki, Muscatell, Irwin, Cole, & Eisenberger, 2011), which may support increased avoidance of out-group members. Finally, some data suggest that not only does direct exposure to a pathogen increase inflammatory activity but that simply viewing diseased-looking people increases inflammatory activity as well (Schaller, Miller, Gervais, Yager, & Chen, 2010). Thus, heightened inflammatory activity in response to the presence or mere possibility of pathogens may increase neural and behavioral sensitivity to threatening social cues and hence may be involved in promoting some aspects of collectivistic cultures, particularly those associated with in-group and out-group biases.

Indeed, cultural neuroscientists are well positioned to unpack some of the causal relationships between the microbial world and cultural differences in psychology. For example, do threats of and/or actual infection momentarily increase psychological states associated with collectivism such as interdependent self-construal, power–distance, and tightness–looseness? And is this relationship bidirectional (e.g., Can priming interdependent self-construal increase sensitivity to cues of infectious threats?)? Building on the studies mentioned earlier, cultural neuroscientists could examine whether low-dose endotoxin (vs. placebo) enhances psychological states associated with collectivism as well as the neural mechanisms underpinning such effects. For example, in addition to showing that inflammatory activity increases threat-related neural sensitivity to threatening images of strangers (Inagaki et al., 2011), it would also be interesting to examine whether inflammatory activity also increases reward-related neural sensitivity to close others, which may compose the neural basis of an increased preference for in-group members. Similarly, building on the findings that viewing diseased others can increase inflammatory activity (Schaller et al., 2010), it would be worth examining whether viewing diseased others also leads to in-group preference and out-group avoidance. Such studies would provide important experimental evidence to further understand the relationships between pathogen prevalence and collectivism.

In fact, if threat of infection gave way to psychological traits that prioritize connection to in-group members through culture–gene coevolution, then it stands to reason that individuals with genes associated with collectivism may also be more protected against infection. Of interest, there is some evidence that alleles associated with collectivism may also help the immune system protect against infectious disease. For example, the short allele of the serotonin transporter gene (5HTTLPR), which Chiao et al. note is associated with collectivism around the world, has also been associated with increased proinflammatory activity, which may help the immune system mount a defensive response to protect against infection (Fredericks et al., 2010). Indeed, given that 98% of human serotonin is located outside of the central nervous system (Cooper, Bloom, & Roth, 2003) and is critically involved in immunity (Mössner & Lesch, 1998), it makes sense that some of the relationships between the serotonin transporter gene and collectivism may be mediated through inflammatory processes. Paradigms that test relationships between infection and collectivism could therefore also test whether, within the same individuals, carrying specific alleles alters the inflammatory response to infection (perhaps in response to endotoxin) as well as psychological states associated with collectivism.

Can Cultural Neuroscientists Help Unravel Whether and How Aspects of Cultural Psychology Are Susceptible to Change?

In the previous section we suggested ways in which cultural neuroscientists may begin to empirically test the biological mechanisms through which ancient environmental factors may have shaped cultural differences in psychology observable today. However, a crucial aspect of culture that is often overlooked in cultural neuroscience is that cultural values and the psychology of cultural participants can *change*. By focusing on the predictors of cultural differences (rather than just the consequences), might we be able to understand shifts in cultural psychology?

For example, as cultures become able, through modern medicine, to reduce the risk of pathogens, do cultural differences in psychology change? This idea can be illustrated by research on attraction, which finds that heterosexual women show different biases in attraction to men across their menstrual cycle (e.g., increased preferences for indicators of male genetic quality during ovulation; e.g., Roberts et al., 2008); however, **Q3** women taking contraceptive pills that prevent ovulation show reductions in these biases (see Alvergne & Lummaa, 2010, for a review). Hence, modern manipulations of biological systems may have downstream consequences for psychology. In the context of reduced pathogens, as regions of the world, which have historically experienced pathogen threats, reduce these threats through modern medicine and technology (e.g., water purification, antibiotic use, etc.), does the populations' sociality change in lockstep?

Along these lines, pharmacological advances may also lead to perhaps surprising changes in psychological variables that typically vary across cultures. Some of the genes that vary cross-culturally and are associated with collectivistic ideology are associated with neural systems often pharmaceutically manipulated to treat mental health conditions. For example, selective serotonin re-uptake inhibitors, which are commonly used to treat depression, anxiety disorders, and personality disorders, are thought to increase extracellular levels of the serotonin neurotransmitter (Wong, Perry, & Bymaster, 2005). Of interest, one social behavior known to vary cross-culturally—costly punishment

(Henrich et al., 2006), or the willingness to incur personal costs to ensure fairness—can be increased and decreased with pharmacological interventions working on the serotonin system (Crockett, Clark, Lieberman, Tabibnia, & Robbins, 2010; Crockett, Clark, Tabibnia, Lieberman, & Robbins, 2008). Although unanticipated, results like these seem to suggest that the use of selective serotonin re-uptake inhibitors could affect psychological states associated with collectivism, and as medication use spreads through a population, there may be corresponding changes in cross-cultural differences in psychology.

Another potential source of change in psychology among cultural participants may be shifts from rural to urban living, which commonly accompanies economic growth. Indeed, some of the shifts from rural to urban living are under way in many cultures associated with collectivism (e.g., China; Fan, 2008; Li, 2006). Of interest, some evidence suggests that individuals who either grew up or currently live in a city show distinct neural responses to social stressors relative to those living in rural settings (Lederbogen et al., 2011). By extension, it seems reasonable to predict that as populations within a culture shift from rural to urban environments, so may cultural differences in psychology. If this is the case, fMRI methods used in cultural neuroscience may be able to track these shifts, which may not always be detectable via self-report (Falk, Berkman, Whalen, & Lieberman, 2011), at the level of regional neural activation.

Can Cultural Neuroscience Inform Related Disciplines?

Research questions in cultural neuroscience often start from a psychological difference (e.g., individualism vs. collectivism) to answer questions about how these differences are reflected in the brain and/or are associated with certain genes. As a result, a potential criticism of cultural neuroscience research is that mapping, and redescribing, culture in biological terms is limited in scope. To avoid redundancy and redescription, cultural neuroscience may benefit by casting research questions that simultaneously inform other related disciplines. In fact, approaching certain questions from a cultural neuroscience perspective may shed light on research findings that may have been overlooked were it not for this approach.

Neural Computations

For example, can a cultural neuroscience approach help determine the computational properties of specific brain regions? In the past few decades, social cognitive neuroscientists have focused heavily on the neural substrates that underpin how people think about mental states, traits, and beliefs (i.e., "mentalizing"; see Lieberman, 2010, for a review) and consistently find that a neurocognitive network engages when people mentalize (consisting of medial prefrontal cortex [MPFC], dorsomedial prefrontal cortex, posterior cingulate/precuneus; temporoparietal junction [TPJ]; posterior superior temporal sulcus; and temporal poles). However, the computational roles of these regions involved in mentalizing are unclear. Specifically, there is some debate surrounding whether it is the TPJ or MPFC that plays a specialized role (necessary and sufficient) in representing mental states (Mitchell, 2005; Saxe & Wexler, 2005). Supporters of the MPFC account suggest that we understand others' minds by simulating their mind from our own perspective (e.g., "Watching this scary movie makes me very nervous, so my friend watching it beside me must also be very nervous"), and given the role of MPFC in self-processing (Gusnard, 2001; Kelley et al., 2002), MPFC activation is necessary for mentalizing, via simulation from the self-perspective (Mitchell, Macrae, & Banaji, 2006). On the other hand, some argue that simulation from the self-perspective is not necessary for mentalizing, and instead suggest that rule-based social cognition about how minds work (e.g., "People choose things that they desire, so if my friend chose to watch a scary movie, she must like them"), supported by TPJ, is necessary for mentalizing (Saxe & Wexler, 2005).

In their review, Chiao et al. note that there is a cultural difference in recruiting these regions during mentalizing. Koreans, who endorse a preference for social hierarchy, recruit TPJ, whereas Westerners, who endorse a preference for egalitarianism, recruit MPFC (Mathur, Harada, Lipke, & Chiao, 2010). Although not framed to address the debate, these results suggest that pitting the MPFC and TPJ as mutually exclusive accounts of specialized regions for mentalizing may overlook the possibility that the regions support different mentalizing strategies that become more or less specialized depending on the cultural environment. This seems possible given that if one generally believes that everyone is equal, recruiting MPFC to simulate others' minds from your own perspective may be an efficient strategy, as one's self should serve as a good proxy to equivalent others. In cultures endorsing a strong sense of hierarchy, there may be a general emphasis on the fact that individuals and their mental states are separate from one's own mental state, and hence a rule-based mentalizing strategy may be more effective than simulating other minds from the self-perspective. Cross-cultural mentalizing studies aimed to test which brain regions are necessary for mentalizing may therefore offer novel input on the debate between the simulation account supported by MPFC and the rule-based accounts supported by TPJ in mentalizing—perhaps finding not that one region is *the* region but rather that one region may be *the* region depending on the optimal mentalizing strategy tailored to cultural ideologies.

Similarly, can the question of whether there are cultural differences in neural responses to empathy discussed by Chiao et al. be turned on its head and posed as “What can culturally mediated neural responses during empathy tell us about empathic processes more broadly?” For example, social psychologists have consistently shown what are known as “empathy gaps,” which refer to the failure to empathize or experience limited empathy for others when their experience is different from our own psychological state (Bryce et al., 2004; Van Boven, Lowenstein, Welch, & Dunning, 2005). Q5 Because empathy gaps can have negative consequences (doctors underestimate patients’ pain, Lowenstein, 2005; peers underestimate another’s hurt feelings, Nordgren, Banas, & MacDonald, 2011), researchers are interested in how to close empathy gaps, and there is some evidence that incorporating victims into self-representations (e.g., “self-other overlap”) may reduce empathy gaps (Cialdini, Brown, Lewis, Luce, & Neuberg, 1997). However, experimental manipulations of incorporating a victim into the self-concept arbitrarily (e.g., telling participants they share a similar group membership with a target) is likely to be a weak manipulation to ensure cognitive representations of the target are tied to self-representations. Instead, it would be ideal to examine whether externally valid forms of self–other overlap do indeed reduce empathy gaps. Moreover, if we want to understand the brain mechanisms through which self–other overlap reduces empathy gaps, then real-world examples of self–other overlap would be preferable, as again, self–other overlap may need to be meaningfully developed over time in order for neural signals to adequately reflect integrated self–other representations.

As it turns out, a cultural neuroscience approach works well to test whether and how self–other overlap reduces empathy gaps, given that in collectivistic cultures, individuals show a greater proclivity to incorporate close others into the self-concept, or “interdependent self-construal” (Markus & Kitayama, 1991). Indeed, we recently used a cultural neuroscience approach to examine if self–other overlap in individuals with interdependent self-construal reduced empathy gaps, focusing on the neural underpinnings of the effect. We found that during fMRI scanning, Chinese individuals with strong interdependent self-construal reported sharing more negative emotions with a close-other victim compared to a stranger victim during an empathy paradigm previously shown to induce empathy gaps (Meyer et al., 2012). Moreover, the neural data suggest that enhanced empathy for the close-other was partially due to communication between the MPFC, which supports overlapping conceptual representations between the self and close-others in individuals with interdependent self-construal (Chiao et al., 2009) and limbic regions previously associated with empathy for physical pain (dACC and AI). In addition, individuals who endorsed the most self–other overlap with their close-other, as well as stronger levels of interdependent self-construal (Meyer et al.), showed the Q6 most neural activation in these regions. Thus, a cultural neuroscience approach not only sheds light on culturally mediated neural responses during empathy but also offers empirical support for the general claim that self–other overlap may reduce empathy gaps and further suggests that the mechanism underlying the effect may be communication between MPFC and limbic regions.

Genetics

As previously mentioned, cultural neuroscientists are well positioned to begin testing the culture–gene coevolutionary theory. Genetic results from such research may extend beyond this question to more generally inform the psychological literature. Indeed, a cross-cultural approach has already begun to shape how researchers think about the functions of genes that affect social processes. For example, early gene studies among primarily Caucasian samples suggested that the short allele of the serotonin transporter gene (5HTTLPR) was related to negative mental health outcomes (e.g., depression; Caspi et al., 2003). However, in collectivistic countries it appears to be protective (Chiao & Blizinsky, 2010). In response to these results, geneticists have reframed their thinking about the short allele (as well as other alleles; see Way & Lieberman, 2010) as an allele that heightens sensitivity to all aspects of the social environment—both positive and negative—which may be helpful in a collectivistic culture and perhaps more harmful in an individualistic culture.

New genetic data are being acquired at an ever-increasing rate that when coupled with cultural data should yield new insights for answering complex questions such as which psychological processes are affected by genes. For example, population geneticists have noted that genetic selection is not the only mechanism that could explain such differences in allele distribution as seen with the 5-HTTLPR. Events such as genetic drift (fluctuations in allele frequency due to chance) or allele surfing (increased genetic drift occurring at the edge of a wave of population expansion) could cause differences in allele frequency between populations that are similar in magnitude as to that seen with the 5-HTTLPR (Hofer, Ray, Wegmann, & Excoffier, 2009; Keinan, Mullikin, Patterson, & Reich, 2007). By definition, these mechanisms do not have any affect on survival or reproductive success. If future genetic data can help to delineate between selective or neutral genetic processes, it will serve to focus experimental psychological research. If selective pressures explain population differences in 5-HTTLPR allele frequency, then psychological explanations such as outgroup avoidance (e.g., Chiao et al. target article) would be likely. Whereas if neutral processes can account for differences in 5-HTTLPR distribution, then other psychological mechanisms might be involved, such as gene–cultural "fit" (Way & Lieberman, 2010). Thus, population genetic data could inform 5-HTTLPR psychological research beyond the cross-cultural domain.

Conclusion

In sum, Chiao et al. provide a thorough review of the cultural neuroscience literature, which has made great progress in a short time. Moving forward, we hope researchers address questions like the ones posed here to help broaden the scientific scope of the field. We urge cultural neuroscientists to go beyond mapping known cultural differences in psychology to the brain and begin to more deeply examine the environmental precursors that lead to these differences in the first place. Experimental social psychological methods are likely to be most fruitful on this front. Such findings may in turn help us understand how and why certain aspects of culturally mediated psychology are susceptible to change, as well as lead to otherwise unanticipated insights into related disciplines also working toward understanding the biology of human psychology.

Note

Address correspondence to Naomi Eisenberger, Department of Psychology, 1285 Franz Hall, University of California, Los Angeles, Los Angeles, CA 90095-1563. E-mail: neisenbe@ucla.edu

References

Alvergne, A., & Lummaa, V. (2010). Does the contraceptive pill alter mate choice in humans? *Trends in Ecology & Evolution*, *25*, 171–179.

Andreasen, A. S., Krabbe, K. S., Krogh-Madsen, R., Taudorf, S., Pedersen, B. K., & Møller, K. (2008). Human endotoxemia as a model of systemic inflammation. *Current Medicinal Chemistry*, *15*, 1697–1705.

Brewer, M. B., & Yuki, M. Y. (2007). Culture and social identity. In S. Kitayama & D. Coven (Eds.), *Handbook of cultural psychology* (pp. 307–322). New York, NY: Guilford.

Bryce, C. L., Loewenstein, G., Arnold, R. M., Schooler, J., Wax, R. S., & Angus, D. C. (2004). Quality of death: Assessing the importance placed on end-of-life treatment in the intensive-care unit. *Medical Care*, *42*, 423–431.

Caspi, A., Sugden, K., Moffitt, T. E., Taylor, A., Craig, I. W., Harrington, H., et al. (2003). Influence of life stress on depression: Moderation by a polymorphism in the 5-HTT gene. *Science*, *301*, 386–389.

Chiao, J. Y., & Blizinsky, K. D. (2010). Culture–gene coevolution of individualism-collectivism and the serotonin transporter gene (5-HTTLPR). *Proceedings of the Royal Society B: Biological Sciences*, *277*, 529–537.

Chiao, J. Y., Harada, T., Komeda, H., Li, Z., Mano, Y., Saito, D. N., et al. (2009). Neural basis of individualistic and collectivistic views of self. *Human Brain Mapping*, *30*, 2813–2820.

Cialdini, R. B., Brown, S. L., Lewis, B. P., Luce, C., & Neuberg, S. L. (1997). Reinterpreting the empathy–altruism relationship: When one into one equals oneness. *Interpersonal Relations and Group Processes*, *73*, 481–494.

Cooper, J., Bloom, F., & Roth, R. (2003). *The biochemical basis of neuropharmacology.* New York, NY: Oxford University Press.

Crockett, M. J., Clark, L., Lieberman, M. D., Tabibnia, G., & Robbins, T. W. (2010). Impulsive choice and altruistic punishment are correlated and increase in tandem with serotonin depletion. *Emotion*, *10*, 855–862.

Crockett, M. J., Clark, L., Tabibnia, G., Lieberman, M. D., & Robbins, T. W. (2008). Serotonin modulates behavioral reactions to unfairness. *Science*, *320*, 1739.

Eisenberger, N. I., Inagaki, T. K., Rameson, L. T., Mashal, N. M., & Irwin, M. R. (2009). An fMRI study of cytokine-induced depressed mood and social pain: The role of sex differences. *NeuroImage*, *47*, 881–890.

Falk, E. B., Berkman, E. T., Whalen, D., & Lieberman, M. D. (2011). Neural activity during health messaging predicts reductions in smoking above and beyond self-report. *Health Psychology*, *30*, 177–185.

Fan, C. (2008). *China on the move: Migration, the state, and the household*. London: Routledge.

Fincher, C. L., Thornhill, R., Murray, D. R., & Schaller, M. (2008). Pathogen prevalence predicts human cross-cultural variability in individualism/collectivism. *Proceedings of the Royal Society*, *275*, 1279–1285. Q7

Fredericks, C. A., Drabant, E. M., Edge, M. D., Tillie, J. M., Hallmayer, J., Ramel, W., et al. (2010). Healthy young women with serotonin transporter SS polymorphism show a pro-inflammatory bias under resting and stress conditions. *Brain, Behavior, and Immunity*, *24*, 350–357.

Gangestad, S. W., Haselton, M. G., & Buss, D. M. (2006). Toward an integrative understanding of evoked and transmitted culture: The importance of specialized psychological design. *Psychological Inquiry*, *17*, 138–151. Q8

Gangestad, S. W., & Thornhill, R. (2008). Human Oestrus. *Proceedings of the Royal Society B.*, *275*, 991–1000. Q9

Gusnard, D. A., Akbudak, E., Shulman, G. L., & Raichle, M. E. (2001). Medial prefrontal cortex and self-referential mental activity: Relation to a default mode of brain function. *Proceedings of the National Academy of Sciences USA*, *98*, 4259–4264. Q10

Henrich, J., McElreath, R., Barr, A., Ensminger, J., Barrett, C., Bolyanatz, A., et al. (2006). Costly punishment across human societies. *Science*, *312*, 1767–1770.

Hofer, T., Ray, N., Wegmann, D., & Excoffier, L. (2009). Large allele frequency differences between human continental groups are more likely to have occurred by drift during range expansions than by selection. *Annals of Human Genetics*, *73*, 95–108.

Inagaki, T. K., Muscatell, K. A., Irwin, M. R., Cole, S. W., & Eisenberger, N. I. (2011). Inflammation selectively enhances amygdala activity to socially threatening images. *NeuroImage*, *59*, 3222–3226.

Keinan, A., Mullikin, J., Patterson, N., & Reich, D. (2007). Measurement of the human allele frequency spectrum demonstrates greater genetic drift in East Asians than in Europeans. *Nature Genetics*, *39*, 1251–1255.

Kelley, W. M., Macrae, C. N., Wyland, C. L., Caglar, S., Inati, S., & Heatherton, T. F. (2002). Finding the self? An event-related fMRI study. *Journal of Cognitive Neuroscience*, *14*, 785–794.

Lederbogen, F., Kirsch, P., Haddad, L., Streit, F., Tost, H., & Schuch, P. (2011). City living and urban upbringing affect neural social stress processing in humans. *Nature*, *474*, 498–501.

Li, S. (2006). Population migration and urbanization in China: A comparative analysis of the 1990 population census and the 1995 National One Percent Sample Population Survey. *International Migration Review*, *38*, 655–685.

Lieberman, M. D. (2010). Social cognitive neuroscience. In S. T. Fiske, D. T. Gilbert, & G. Lindzey (Eds.), *Handbook of social psychology* (5th ed., pp. 143–193). New York, NY: McGraw-Hill.

Lowenstein, G. (2005). Hot–cold empathy gaps and medical decision making. *Health Psychology*, *24*, S49–S56.

Markus, H. R., & Kitayama, S. (1991). Culture and the self: Implications for cognition, emotion, and motivation. *Psychological Review*, *98*, 224–253.

Mathur, V. A., Harada, T., Lipke, T., & Chiao, J. Y. (2010). Neural basis of extraordinary empathy and altruistic motivation. *Neuroimage*, *51*, 1468–1475.

Meyer, M. L., Masten, C. L., Ma, Y., Wang, C., Shi, Z., Eisenberger, N. I., et al. (2012). Empathy for the social suffering of friends and strangers recruits distinct patterns of brain activation. *Social Cognitive and Affective Neuroscience*. doi:10.1093/scan/nss019

Meyer, M. L., Masten, C. L., Ma, Y., Wang, C., Shi, Z., Eisenberger, N. I., et al. (XXXX). *An interdependent self is an empathizing self: Interdependent self-construal correlates with empathic neural responses*. Manuscript in preparation. **Q11**

Mitchell, J. P. (2005). The false dichotomy between simulation and theory-theory: The argument's error. *Trends in Cognitive Sciences*, *9*, 363–364.

Mitchell, J. P., Macrae, C. N., & Banaji, M. R. (2006). Dissociable medial prefrontal contributions to judgments of similar and dissimilar others. *Neuron*, *50*, 655–663.

Mössner, R., & Lesch, K-P. (1998). Role of serotonin in the immune system and in neuroimmune interactions. *Brain, Behavior, and Immunity*, *12*, 249–271.

Murray, D. R., Trudeau, R., & Schaller, M. (2011). On the origins of cultural differences in conformity: Four tests of the pathogen prevalence hypothesis. *Personality and Social Psychology Bulletin*, *37*, 318–329.

Nordgren, L. F., Banas, K., & MacDonald, G. (2011). Empathy gaps for social pain: Why people underestimate the pain of social suffering. *Journal of Personality and Social Psychology*, *101*, 120–128.

Saxe, R., & Wexler, A. (2005). Making sense of another mind: The role of the right temporo-parietal junction. *Neuropsychologia*, *43*, 1391–1399.

Schaller, M., Miller, G. E., Gervais, W. M., Yager, S., & Chen, E. (2010). Mere visual perception of other people's disease symptoms facilitates a more aggressive immune response. *Psychological Science*, *21*, 649–652.

Singles, T. (1994). The measurement of independent and interdependent self-construals. *Personality and Social Psychology Bulletin*, *20*, 580–591. **Q12**

Suffredini, A. F., Hochstein, H. D., & McMahon, F. G. (1999). Dose-related inflammatory effects of intravenous endotoxin in humans: Evaluation of a new clinical lot of Escherichia coli O:113 endotoxin. *The Journal of Infectious Disease*, *179*, 1278–1282. **Q13**

Van Boven, L., Loewenstein, G., & Dunning, D. (2005). The illusion of courage in social predictions: Underestimating the impact of fear of embarrassment on other people. *Organizational Behavior and Human Decision Processes*, *96*, 130–141. **Q14**

Way, B., & Lieberman, M. D. (2010). Is there a genetic contribution to cultural differences? Collectivism, individualism, and genetic markers of social sensitivity. *Social Cognitive and Affective Neuroscience*, *5*, 203–211.

Wong, D. T., Perry, K. W., & Bymaster, F. P. (2005). Case history: The discovery of fluoxetine hydrochloride (Prozac). *Nature Reviews. Drug Discovery*, *4*, 764–774.