neural substrates of motivation and to use the models that he has developed to study the effects of manipulations on positive affect. He uses a fairly specific conceptualization of positive affect based on David Watson and Auke Tellegen's work,[129] according to which positive affect is a dominant factor of mood that can be distinguished from happiness.

Zald reviewed research in rats that used a simple T-maze to test motivation and the willingness to expend effort.[130] The animals get a larger reward if they are willing to expend effort to climb over a barrier. Healthy rats will choose the larger reward at least 80 percent of the time. Using this paradigm, the VS and the mPFC have been identified as two critical nodes of an effort-based decision-making circuit. In humans, research on the neurobiology of effort expenditure for rewards has focused on the mesolimbic DA system. Zald reviewed neural projections involving the mPFC, the ventral tegmental area (VTA), and the nucleus accumbens (NAcc), which are all hypothesized to constitute a behavioral activation system that pushes behavior toward rewards.

Decreasing DA concentrations in these regions or blocking DA receptors with haloperidol decreased the willingness for rats to expend effort.[131] Conversely, rats given amphetamine to increase DA levels will seek the larger reward more frequently.[132] These observations are independent of whether or not the animal likes the food, suggesting a role for DA in motivation for rewards rather than short-term hedonic experiences.

In order to use willingness to work as a measure of motivation in humans, alternative approaches to climbing walls had to be developed. One implementation developed by Zald and colleagues is the EEfRT, which involves a simple lever pressing paradigm.[133] Across multiple trials, research subjects must make repeated cost-benefit analyses regarding whether or not a specific monetary reward is worth additional effort relative to a smaller reward that requires less effort. Researchers assess individual differences in the sensitivity to reward magnitude and probability by varying the reward value of the harder task and the probability of reward for

[129] Watson, D., and Tellegen, A. (1985). Toward a consensual structure of mood. Psychol. Bull. *98*, 219–235.
[130] Salamone, J.D., Correa, M., Farrar, A., and Mingote, S.M. (2007). Effort-related functions of nucleus accumbens dopamine and associated forebrain circuits. Psychopharmacology (Berl.) *191*, 461–482.
[131] Salamone, J.D., Cousins, M.S., and Bucher, S. (1994). Anhedonia or anergia? Effects of haloperidol and nucleus accumbens dopamine depletion on instrumental response selection in a T-maze cost/benefit procedure. Behav. Brain Res. *65*, 221–229.
[132] Bardgett, M.E., Depenbrock, M., Downs, N., Points, M., and Green, L. (2009). Dopamine modulates effort-based decision making in rats. Behav. Neurosci. *123*, 242–251.
[133] Treadway, M.T., Buckholtz, J.W., Schwartzman, A.N., Lambert, W.E., and Zald, D.H. (2009). Worth the "EEfRT"? The effort expenditure for rewards task as an objective measure of motivation and anhedonia. PLoS ONE *4*, e6598.

either task across trials. Depressed individuals prefer the easy, less rewarding task and appear less sensitive to reward magnitude relative to healthy controls.[134]

Zald and colleagues recently demonstrated in humans that dopaminergic manipulations indeed alter willingness to exert effort for rewards.[135] In these experiments, administration of amphetamine to research subjects increased effort selectively on low probability trials. This means that increased DA release by amphetamine increases pursuit of unlikely potential rewards. In parallel functional magnetic resonance imaging (fMRI) studies, the mPFC, dACC, and preSMA regions were activated during decisions to expend effort.

Zald then went on to relate EEfRT measures to individual differences in DA functions in humans. He used Fallypride, a DA-D2 receptor binding ligand, to measure DA release by a two-scan protocol. After amphetamine administration, fewer D2 receptors are available, leading to decreased Fallypride binding. With this paradigm, DA release can be used as a trait measure of DA system reactivity. Zald and colleagues found that subjects with high striatal DA release have greater motivation to pursue rewards and novelty. There was a dose–response relationship; the more DA subjects released, the more likely they were to select the high-reward task.

Zald discussed the utility of this operationalization of motivation for studies of PPWB. Intensive fMRI studies cannot be easily integrated into large epidemiological studies, but they may be realistic when conducted in smaller, targeted investigations. There, they may fulfill an important role in capturing motivational aspects of wellbeing. He emphasized that this work mainly aims at measuring the eudaimonic side of wellbeing. While there are no data yet on health outcomes, Zald provided examples of possible future uses. These include studies of the willingness to expend effort for health. He speculated that willingness to exercise would correlate with conscientiousness, which is linked to a large number of health outcomes. It may be difficult to assess physical effort in some populations using this paradigm, but cognitive effort (e.g., math tasks) is much easier to implement. Furthermore, the exact contributions of reward seeking versus cost valuation should be examined in greater detail in the future. Another possible limitation of the EEfRT paradigm was also noted; the use of fiscal rewards might show large variability in different age groups or individuals with differing socioeconomic status (SES), limiting its usefulness in larger efforts.

Zald concluded his presentation by noting ongoing efforts to connect these studies to the real world by investigating possible correlations between changes in decision making and changes in measured DA function during aging, and by examining the relationship between EEfRT performance and time spent studying and exercising in college students.

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[134] Treadway, M.T., Bossaller, N.A., Shelton, R.C., and Zald, D.H. (2012). Effort-based decision-making in major depressive disorder: a translational model of motivational anhedonia. J. Abnorm. Psychol. *121*, 553–558.

[135] Wardle, M.C., Treadway, M.T., Mayo, L.M., Zald, D.H., and De Wit, H. (2011). Amping up effort: effects of d-amphetamine on human effort-based decision-making. J. Neurosci. *31*, 16597–16602.

ROLE OF MICROBIOME GUT BRAIN INTERACTIONS IN RESILIENCE

Emeran Mayer, M.D., University of California at Los Angeles

In reviewing the bidirectional interactions of the microbiome-gut-brain axis,[136] Emeran Mayer stated that this axis can be viewed as a link between three "supersystems": the brain, the enteric nervous system (ENS) with its closely related endocrine and immune components, and the gut microbiome. As such, it links our largest surface with the environment made up by the gut with our brain and the external environment, and has to be assumed to be an important component of the organism's resilience to internal or external perturbations.

The ENS constitutes the largest accumulation of neurons outside of the CNS. Because of it size and complexity, it also has been called the "little brain" or "second brain." It comprises 200 to 600 million neurons, some of which have similar morphological, electrophysiological and signaling properties as brain cells. The ENS is considered the third branch of the ANS in addition to the sympathetic and parasympathetic branches. If the connections from the brain to the enteric nervous system are interrupted, then the ENS assures the autonomic functioning of the gut, but the ENS is no longer able to provide important feedback to the brain. The enteric nervous system and the gut microbiome receive sympathetic and parasympathetic signals from the CNS and modulatory influences from the hypothalamic pituitary adrenal axis.

Mayer reviewed various "intestinal target cells" or "transducer cells," including enterochromaffin cells, immune cells, smooth muscle, and enteric neurons. Under steady state, these cells signal to visceral afferent fibers to constantly inform the brain about events in the gut. The brain, via the branches of the autonomic nervous system, can adjust their sensitivity acutely and change their properties during chronic stimulation. By this latter mechanism, chronic stress is likely to change our interactions with the gut and the gut microbiome. Similarly, neuroplastic changes, which are likely to occur at all levels of the gut-brain axis in patients with chronic abdominal pain, can lead to increased afferent signaling to the brain and influence mood, affect, and cognition. In summary, the gut-brain axis is a highly plastic system of high relevance for health and chronic disease.

Enteroendocrine (EE) cells in the gut form the largest endocrine organ of the body and use 20 different hormones and signaling molecules. Ten discrete EE cells have been described; those secreting serotonin (i.e.,enterochromaffin cells, ECs) have received the most attention because they produce 95 percent of the body's serotonin. They can sense nutrients, bile salts, short chain fatty acids, bitter and sweet taste, bacterial products, and quorum-sensing molecules via specialized receptors on their luminal surface.

Mayer reviewed research from the past 10 years on gut-brain interactions and neuronal correlates of gut-induced changes in brain signaling. He noted a long-standing interest in common chronic visceral pain conditions, such as irritable bowel syndrome (IBS) and often

[136] Mayer, E.A., and Tillisch, K. (2011). The brain-gut axis in abdominal pain syndromes. Annu. Rev. Med. *62*, 381–396.

comorbid syndromes involving the esophagus and upper GI tract. In spite of the large prevalence and frequent supposed breakthroughs in the understanding of the causes of IBS, no effective therapy has yet been developed.

Mayer's research group focuses on readouts in the brain of abnormal signals from the gut. In a recent study of cortical thickness,[137] they compared 83 individuals with IBS to 194 controls. They saw increased cortical thickness in somatosensory cortex areas and decreased thickness in regions of emotion regulation circuits. Further studies revealed corresponding changes in white matter connectivity. Overall, the IBS brain is characterized by extensive structural remodeling and altered function, including enhanced sensory perception, compromised prefrontal inhibition (including altered prefrontal-limbic-pontine input to endogenous pain modulation systems), and enhanced emotional arousal.

In the future, Mayer's group plans to extend this approach to study gut brain interactions in health and various disease states. They will explore the relationship between brain signatures (i.e., structural and functional brain changes) and other –omics datasets to create distinct signatures or biomarkers of chronic disease and wellness.

Mayer also briefly reviewed the human gut microbiome.[138] By cell count, humans are 90 percent bacterial. Individuals differ in the composition of their microbial enterotypes.[139] Emerging findings suggest a significant role of the bacteria in gut-brain communication; furthermore, microbiome diversity has been suggested to predict resilience and protect against obesity. Bacteria produce metabolites homologous to catecholamines, gamma-aminobutyric acid (GABA), tryptophan, histamine and serotonin, and many other substances. To understand whether and how these signals act as communication signals between microbial communities and signals to the brain, affecting its structure and function, remains a future challenge.

Mayer concluded his talk by emphasizing the emerging role of bidirectional microbiome-gut-brain interactions in wellness and disease.[140] Distinct signatures of these closely connected systems may become as relevant as biomarkers.

[137] Jiang et al., under review.
[138] Berg, R.D. (1996). The indigenous gastrointestinal microflora. Trends Microbiol. *4*, 430–435.
[139] Arumugam, M., Raes, J., Pelletier, E., Le Paslier, D., Yamada, T., Mende, D.R., Fernandes, G.R., Tap, J., Bruls, T., Batto, J.-M., et al. (2011). Enterotypes of the human gut microbiome. Nature *473*, 174–180.
[140] Mayer, E.A. (2011). Gut feelings: the emerging biology of gut-brain communication. Nat. Rev. Neurosci. *12*, 453–466; Rhee, S.H., Pothoulakis, C., and Mayer, E.A. (2009). Principles and clinical implications of the brain-gut-enteric microbiota axis. Nat. Rev. Gastroenterol. Hepatol. *6*, 306–314.

GENOMIC APPROACHES

Steve Cole, Ph.D., School of Medicine, University of California at Los Angeles

Gene expression studies have become powerful tools to study the molecular changes leading to increased risk of morbidity and death. An expression study of social isolation, one of the most robust environmental risk factors for disease and mortality in humans, revealed a non-random signature of up- and down-regulated genes that were associated with isolation.[141] Pro-inflammatory genes that are known to be involved in disease processes of CVD, cancer, and neurodegeneration were particularly relevant. The same general pattern of increased inflammatory gene expression and reduced activity of interferon and antibody genes has been observed across a wide variety of adverse life circumstances (e.g., low SES in childhood and adulthood, social loss, post-traumatic stress, cancer diagnosis, social threat, loneliness, social instability, chronic stress, low social rank, caregiving for seriously ill people, and depression). Experiments in macaques have provided converging evidence for these profiles.[142] Because of its consistent emergence, this pattern has been called conserved transcriptional response to adversity (CTRA). According to Steve Cole, the challenge for the field now lies in the identification of higher-order correlates in this pattern.

Cole noted that details of current endeavors to understand the origin of these patterns were beyond the scope of the current presentation, but that white blood cells of the monocyte type appear to account for most of the transcriptional reprogramming.[143] Recent research has also started to identify some of the key biological pathways (e.g., HPA- and SNS-regulated gene transcription) mediating these cellular effects, answering questions about which particular hormonal pathway is connected to which particular gene profile.[144] This knowledge will enable researchers to then search for upstream causes of these events.

Cole summarized that the results presented so far can tell us about how not to live. But can genetic expression profiles also be used to answer more positive questions about true happiness? Does the genome prioritize one type of wellbeing over another (e.g., hedonic wellbeing over eudaimonic wellbeing)?

Cole recently had a unique chance to study this question by collaborating with Barbara Fredrickson and her group at the University of North Carolina, Chapel Hill. They recruited 80

[141] Cole, S.W., Hawkley, L.C., Arevalo, J.M., Sung, C.Y., Rose, R.M., and Cacioppo, J.T. (2007). Social regulation of gene expression in human leukocytes. Genome Biol. *8*, R189.
[142] Cole, S.W., Conti, G., Arevalo, J.M.G., Ruggiero, A.M., Heckman, J.J., and Suomi, S.J. (2012). Transcriptional modulation of the developing immune system by early life social adversity. Proc. Natl. Acad. Sci. U.S.A. *109*, 20578–20583.
[143] Cole, S.W., Hawkley, L.C., Arevalo, J.M.G., and Cacioppo, J.T. (2011). Transcript origin analysis identifies antigen-presenting cells as primary targets of socially regulated gene expression in leukocytes. Proc. Natl. Acad. Sci. U.S.A. *108*, 3080–3085.
[144] Irwin, M.R., and Cole, S.W. (2011). Reciprocal regulation of the neural and innate immune systems. Nat. Rev. Immunol. *11*, 625–632.

healthy adults, brought them into the laboratory, assessed hedonic and eudaimonic wellbeing[145] and depressive symptoms,[146] and extracted DNA from their white blood cells. They then simultaneously assessed expression of all 21,000 human genes.

They found that eudaimonic and hedonic affects had very similar inverse relationships with depression, which means that individuals with greater levels of hedonic and eudaimonic wellbeing had lower levels of depression. They then looked at the biology and the clusters of antibody-related genes, antiviral interferon genes, and inflammation. Eudaimonic wellbeing was associated with a "healthy" profile of high levels of interferon- and antibody-related gene transcripts and reduction of pro-inflammatory gene transcripts. Hedonic wellbeing, although fairly highly correlated with eudaimonic wellbeing, produced an adverse expression vector containing greater expression of inflammation-related genes and lesser expression of genes involved in interferon antiviral responses and antibodies. The study controlled for many behavioral confounders, such as substance abuse and other unhealthy behaviors. Cole noted that the human genome has had a long time to evolve, and the apparent preference of eudaimonic wellbeing may teach us something about what it has historically meant to "live well" over the course of human evolution.

Cole concluded his presentation with a brief review of additional studies showing similar changes in gene expression profiles resulting from experimental interventions such as cognitive-behavioral stress management, mindfulness-based stress reduction, and yogic meditation.[147]

DISCUSSION

Participants noted that the resolution with which tools such as the Mental Health Continuum Short Form (MHC-SF) or Center for Epidemiologic Studies Depression (CES-D) Scale measure hedonic or eudaimonic wellbeing and depression may be insufficient to draw detailed conclusions about these concepts. These tools also have limited power to differentiate between affective and somatic components, each of which contains a large number of individual concepts. The field must pay more attention to the adequacy of the measurement tools used; much information can be lost when these constructs are not sufficiently detailed. Hedonic and

[145] Keyes, C.L.M. (2002). The mental health continuum: from languishing to flourishing in life. J. Health Soc. Behav. *43*, 207–222.
[146] Radloff, L.S. (1977). The CES-D Scale. A Self-Report Depression Scale for Research in the General Population. Appl. Psychol. Meas., *1(3),* 385–401.
[147] Black, D.S., Cole, S.W., Irwin, M.R., Breen, E., St Cyr, N.M., Nazarian, N., Khalsa, D.S., and Lavretsky, H. (2013). Yogic meditation reverses NF-κB and IRF-related transcriptome dynamics in leukocytes of family dementia caregivers in a randomized controlled trial. Psychoneuroendocrinology *38*, 348–355.

eudaimonic wellbeing can be highly correlated (Pearson's r has ranged from 0.25 to 0.73)[148] and separating their effects will require sophisticated methods in large samples.

Mayer noted that the gut research field has not yet discovered a poor-health associated transcriptional response profile similar to the CTRA pattern seen in lymphocytes. Roughly three different enterotypes are seen around the world, and each can produce several different profiles. Decreased diversity in gut gene expression was found to be associated with reduced resilience to several diseases. These expression patterns may correlate with altered networks at brain levels. Mayer further believes that microbiome shotgun sequencing might become a routine clinical test in the near future at a cost of as little as $500 per test.

When asked what the best biomarkers for anabolic and restorative processes might be, participants generally agreed that there was no magic bullet. Glycosylated hemoglobin, blood pressure, sleep, lipids, BDNF, OT, connectivity, and networks may all prove useful in carefully defined contexts. A "barcode of health" is, however, unlikely to be found. Rather, concepts such as assortivity may provide guidance on how to look at multiple systems at once and see which systems are most robust and which are likely to break down. Mayer noted a recent trend of the field to move toward system biology, which suggests that changes in individual hormones taken out of context have essentially no predictive power. Carter replied that there might, however, be important hierarchies where some components may be more critical than others.

Although none of the presentations focused specifically on endorphins, they certainly play a role in the pain suppression paradigm discussed by Eisenberger. Participants further noted that the role of sex hormones in PPF was not discussed during this meeting.

BRAINSTORMING

The workshop covered broad research areas, provided an informative tour of novel biological systems, and opened new windows for the understanding of how the human body functions. The diverse background has been very inspiring for further discussions. Nielsen encouraged participants to think about the different constructs and their utility, and to think about the most important questions that must be answered to move the field forward.

To illustrate the kinds of questions that may be relevant, Nielsen offered a number of examples that were developed by members of the organizing committee at the end of the first day:

1) **There is a significant need for rich and well-defined measures of positive psychological functioning and related biological processes.** Are there measures that can be obtained

[148] Ryff, C.D. (1989). Happiness is everything, or is it? Explorations on the meaning of psychological wellbeing. J. Persona. Soc. Psychol., *57(6),* 1069–1081.

with relatively little effort? Which measures are necessary to address questions of causality?

2) **Investigators must consider the possible bidirectionality of associations.** For example, the weight of the current evidence suggests that exercise causes positive emotions, but could there be significant effects in the opposite direction?
3) **The field must get better clarity on the role of positive psychological states in health.** Individual research programs need to clarify the extent to which positive states are considered ends in themselves, i.e., as target outcomes for health promotion, or the extent to which they are important as means to other ends, for example as potential motivators of behavior change, or as aids in self-management of disease.
4) **Knowledge gained in this field has great implications for intervention design.** What are the best targets for interventions? When are systems malleable? Are there sensitive periods and windows of plasticity? Are there individual differences in responsiveness to interventions? If biological embedding is too strong for reversal of trajectories, then can compensatory systems provide improvements?
5) **The nature of the psychological phenomena under study must be better understood.** When talking about experience, motivation, dispositions, etc., are we looking to increase or decrease, or do we seek an optimal adaptive state?

Participants then discussed the most important ingredients and designs of future efforts. Results from these discussions are summarized in the "Critical Issues" section of the Executive Summary of this report.

APPENDIX 1 – WORKSHOP AGENDA

Tuesday, March 12

11:00 am	**Registration**	**Symphony IV Foyer**
12:30 pm	**Lunch**	**Symphony III**
1:00 pm	**Welcome** Lis Nielsen, National Institute on Aging Arthur Stone, Stony Brook University and Princeton University	**Symphony IV**
1:20 pm	**Session 1:** **Psychological factors and health** *This session will review what is already known about positive psychological functioning in relation to health and underlying biology. Three areas of positive psychological functioning will be discussed: psychological wellbeing, social connectedness, and self-regulation. Speakers will present the strongest evidence available regarding associations between positive psychological functioning and health and disease outcomes such as cardiovascular disease. Drawing from this evidence, the speakers also will address the possible pathways underlying these associations, with a particular emphasis on biological mechanisms.* *Primary questions for the speakers to address:* • *What health and disease outcomes are associated with your area of positive psychological functioning?* • *What are the possible pathways and mechanisms underlying the association between positive psychological functioning and disease?*	
1:20 pm	Introduction to psychological factors and health: Julia Boehm	
1:30 pm	Positive psychological wellbeing: Laura Kubzansky	
1:50 pm	Salubrious social connections: Tara Gruenewald	
2:10 pm	Self-regulation: Suzanne Segerstrom	
2:30 pm	Question and discussion period	
2:45 pm	**Coffee break**	**Symphony IV Foyer**

3:00 pm **Session 2:** **Symphony IV**

Known biological factors

This session will describe key biological processes that have been identified as being relevant to or indicative of positive physiological functioning. The first speaker will define the characteristics of restorative biological processes and indicate how they differ from deteriorative biological processes such as atherosclerosis. Subsequent speakers will present brief overviews of each known restorative biological process, indicate how or why the biological process can be considered restorative, and demonstrate links between the biological process and positive psychological functioning.

Primary questions for the speakers to address:

- *What characterizes a restorative process?*
- *What restorative characteristics do each of the known biological factors have?*
- *Are the biological processes associated with positive factors from which we could extrapolate (e.g., depression)?*

3:00 pm	Restorative biological processes and health: A useful concept?: Ted Robles
3:20 pm	The healing power of love: An oxytocin hypothesis: Sue Carter
3:40 pm	Heart rate variability as an integrative index of resilience: Julian Thayer
4:00 pm	Immunity and positive psychobiology: Marian Kohut
4:20 pm	Exploring the neural correlates of receiving and giving social support: Naomi Eisenberger
4:40 pm	Question and discussion period
5:00 pm	**Adjourn**

Wednesday, March 13

8:00 am	**Continental breakfast**	**Symphony III**
8:30 am	**Session 3:** **Research areas that may suggest novel biological factors** *This session will explore research areas that may suggest novel biological processes with positive or restorative features, but that have not yet been formally investigated as such. Speakers will present brief overviews of each research area, describe known associations between psychosocial factors and relevant biological processes, and assess whether the biological process could be considered restorative or could be linked with positive psychological functioning.* *Primary questions for the speakers to address:* • *Why might this research area be a good candidate for identifying novel and restorative biological processes?* • *What biological processes might indicate restoration or something other than deterioration?* • *Have biological processes been linked with other psychosocial factors or even positive psychological functioning?*	**Symphony IV**
8:30 am	Setting the stage for novel biological factors: Suzanne Segerstrom	
8:50 am	Sleep as a source of resilience and restoration: Orfeu Buxton	
9:10 am	Physical activity: Implications for psychosocial function, cognition, and brain: Art Kramer	
9:30 am	Dopamine, motivation, and decision to expend effort: David Zald	
9:50 am	**Coffee Break**	**Symphony IV Foyer**
10:05 am	**Session 3 (Continued)**	**Symphony IV**
10:05 am	Role of brain-gut interactions in resilience: Emeran Mayer	
10:25 am	Genomic approaches: Steve Cole	
10:45 am	Question and discussion period	
11:00 am	**Coffee Break**	
11:15 am	**Session 4: Brainstorming** Introduction: Lis Nielsen	**Symphony 4**
12:15 pm	**Closing Remarks**	
12:30 pm	**Adjourn**	

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APPENDIX 3 - LIST OF SUGGESTED READINGS

Julia K. Boehm and **Laura D. Kubzansky**. 2012.
The Heart's Content: the Association Between Positive Psychological Wellbeing and Cardiovascular Health*, Psychological Bulletin* 138(4): 655-691 (July).

Orfeu M. Buxton, Karen Hopcia, Grace Sembajwe, James H. Porter, Jack T. Dennerlein, Christopher Kenwood, Anne M. Stoddard, Dean Hashimoto, and Glorian Sorensen. 2012.
Relationship of Sleep Deficiency to Perceived Pain and Functional Limitations in Hospital Patient Care Workers, *Journal of Occupational and Environmental Medicine* 54(7): 851- 858 (July).

C. Sue Carter and Stephen W. Porges. 2013.
The Biochemistry of Love: an Oxytocin Hypothesis, *European Molecular Biology Organization Reports* 14(1): 12-16 (January).

Michael H. Antoni, Susan K. Lutgendorf, Bonnie Blomberg, Charles S. Carver, Suzanne Lechner, Alain Diaz, Jamie Stagl, Jesusa M.G. Arevalo, and **Steven W. Cole**. 2012.
Cognitive-Behavioral Stress Management Reverses Anxiety-Related Leukocyte Transcriptional Dynamics, *Biological Psychiatry* 71: 366-372 (February).

Naomi I. Eisenberger and **Steve W. Cole.** 2012.
Social Neuroscience and Health: Neurophysiological Mechanisms Linking Social Ties with Physical Health, *Nature Neuroscience* 15(5): 669-674 (May).

Tara L. Gruenewald, Diana H. Liao, and Teresa E. Seeman. 2012.
Contributing to Others, Contributing to Oneself: Perceptions of Generativity and Health in Later Life*, The Journals of Gerontology, Series B: Psychological Sciences and Social Sciences* 67(6): 660-665 (November).

Marian L. Kohut, Megan M. Cooper, Michael S. Nickolaus, Dan R. Russell, and Joan E. Cunnick. 2002.
Exercise and Psychosocial Factors Modulate Immunity to Influenza Vaccine in Elderly Individuals*, Journal of Gerontology: Medical Sciences* 57A(9): M557-M562 (September).

Marian L. Kohut, D.A. McCann, D.W. Russell, D.N. Konopka, J.E. Cunnick, W.D. Franke, M.C. Castillo, A.E. Reighard, and E. Vanderah. 2006.
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