

**National Institute on Aging  
Institute for Personality and Social Research**

**Workshop on Advancing Integrative Psychological Research  
on Adaptive and Healthy Aging**

Thursday, May 21, 2009  
Institute of Personality and Social Research  
University of California, Berkeley

This meeting summary was prepared by Sarah Holley, under contract to the National Institute on Aging. The findings and views reported in this document reflect both individual and collective opinions of the workgroup participants and not necessarily those of the National Institute on Aging or the report author.

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## Table of Contents

Executive Summary .....	iv
List of Abbreviations .....	vi
Workshop Summary: Introduction .....	1
Introduction and Framing the Questions .....	1
NIA interests .....	1
NIA vision .....	3
Session 1: Fundamental Social and Affective Processes in Aging.....	4
Why isn't aging depressing? .....	4
Aging and the use of emotional cues to guide social judgments .....	6
What is the function of age-related positive gaze preferences? .....	7
Effective emotion regulation in older adulthood: Converging levels of analysis .....	8
Correlates of social position in brain serotonergic function .....	10
Exploring the effects of attachment relationships on reactions to transitions .....	11
Session 1: Open discussion.....	13
Session 2: Healthy Aging over the Lifecourse .....	15
Stress, social processes, and health over the lifecourse .....	15
Biology of resilience: Oxytocin, positive adaptation and health .....	16
Loneliness: Cause and target .....	18
Relationship disruptions and health: From social epidemiology to social psychophysiology .....	19
The promise of interventions for promoting well-being .....	21
Psychosocial influences on longevity biomarkers.....	22
Session 2: Open discussion.....	23
Session 3: Decision Making in Aging .....	26
Decision making in aging: Emerging insights from affective neuroscience and neuroeconomics .....	26
Neural basis of decision making in aging.....	28
Age and sex differences in the effects of stress on decision making .....	29
No time to waste: Understanding why older adults are less subject to the sunk-cost fallacy.....	30
Wanting and liking for sex by gender and age.....	32
Session 3: Open discussion.....	33
General Discussion .....	36
References .....	38

Appendix 1: Agenda ..... 42

Appendix 2: Contact Information ..... 44

Appendix 3: Suggested Background Readings ..... 49

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## Executive Summary

Human development does not end with the entry into adulthood. Research is increasingly demonstrating that older adults continue to undergo important changes over the entire course of the lifespan. These changes touch every domain, including biological, social, emotional, and cognitive functioning. While the picture for certain processes (e.g., cognitive and physical) remains one of decline, other areas show promise for adaptive development (e.g. social and emotional). Furthermore, none of these changes occur in a vacuum. Changes across various domains of functioning are inextricably linked, and many later-life developments are influenced by processes or events set into motion during earlier stages of life. The great challenge for researchers is to uncover the factors that underlie both positive and negative changes over the lifespan, as well as to determine how the various systems interact. In doing so, advances can be made toward understanding age-related changes in important outcomes such as mental and physical well-being, the ability to maintain healthy relationships or make good decisions, or even morbidity and mortality. Such research will further enable the development of targeted interventions that can promote healthy aging.

In recent years, the National Institute on Aging (NIA) Division of Behavioral and Social Research (BSR) has sponsored a number of meetings to promote the integration of psychology with other disciplines, around topics such as stress and health, decision-making, and social and economic behavior, in order to promote cross-disciplinary fertilization. Several meetings, held in conjunction with the National Academy of Sciences, have assessed the state of field of psychology of aging and yielded major reports (e.g. “When I’m 64” and “Aging Mind” designed to chart a path for future studies and programs. These meetings have brought together researchers from a number of fields, including psychology, neuroscience, economics, behavioral genetics, and public health. The current workshop, held on May 21, 2009, at the University of California, Berkeley, in conjunction with the Annual Meeting of the Association for Psychological Science in San Francisco, focused on integrative psychological research on adaptive and healthy aging. The workshop was chaired by Dr. Robert Levenson (University of California, Berkeley) and Dr. Lisbeth Nielsen (NIA) and included 57 participants representing a variety of approaches to the field of human development and aging research.

The workshop focused on three major themes. The first session explored fundamental social and affective processes in aging, including the causes and consequences of age-related shifts in emotional experiences and emotion regulation. The next series of presentations focused on healthy aging over the lifecourse, concluding with a provocative discussion of how to accurately conceptualize health outcome measures, particularly in the area of biomarkers. The third set of talks focused on decision-making and incorporated neuroscience, social, and cognitive approaches to the investigation of age-related changes in this domain. Invited perspectives were followed by discussions in which meeting participants clarified findings and generated ideas for future research. The agenda was structured to allow the speakers to present ongoing research findings and questions, and to maximize the opportunity for discussion among all participants. Dr. Nielsen identified three areas to address across the discussion topics: 1) encourage consideration of how to advance study of adaptive and healthy aging using integrative approaches bridging psychology and other disciplines; 2) encourage cross-talk between scientists studying social and emotional function, psychological and biological processes in health, and decision-making; and 3) identify key opportunities and needs for advancing the science in each domain.

Across all of the presentations and discussions, several prominent themes were explored and key challenges for future research were identified. These including the following:

- Taking a lifecourse perspective on aging research. Speakers and participants stressed the importance of looking at aging as a process from birth to death. They further highlighted the need to consider the effect of early life influences on later life outcomes. Many age-related outcomes

don't just appear; they are often the result of early-occurring or cumulative factors. For example, influences such as a non-supportive early environment can affect mental and physical health later in life. It is important for research to take such processes into account. Interdisciplinary longitudinal studies may be particularly valuable in this regard.

- Studying individual differences as well as group differences in aging. Research has shown that older adults differ from younger adults in some significant psychological domains (e.g., social functioning, emotional functioning, and decision-making). These group differences can have both positive and negative implications and are important to capture. But in addition to these differences between older and younger adults, there is also a great deal of variability within each age group. Future studies aimed at identifying the factors that may account for such individual differences will be important advancing the field of aging research.
- A focus on healthy/adaptive aging processes and outcomes. There can be a tendency to focus on negative processes and outcomes in health research, but aging research needs to investigate positive processes and outcomes as well. Related to this theme, researchers must be careful in how they conceptualize such positive variables. Negative is often not just the absence of positive, and vice versa. For example, while morbidity and mortality are clear negative outcome variables, what constitutes adaptive or healthy aging is less well-defined. Once positive variables such as well-being and resilience are understood, researchers can further work to identify the causal mechanisms that contribute to an individual's positive adaptation over the lifespan.
- A need for multi-level research and analysis. Given that aging processes cut across so many domains of functioning, researchers must look to form interdisciplinary collaborations that incorporate multiple methodologies and perspectives. For example, motivation and goals have been shown to change over time; these goals affect emotional experiences and decision-making. The differences are manifest in neural and behavioral indices, such as brain imaging data and gaze cues, and can have social consequences. Interdisciplinary research that cuts across all of these levels of analysis is necessary to fully understand how these processes unfold. Related to this area, such collaborative endeavors can help researchers make the best possible decisions about how to best approach their question of interest. Participants spoke of the danger of over-interpreting findings, as well as the challenge in knowing how to apply findings (particularly with regard to genetic or biomarker findings).
- A translational approach that links laboratory science to the real world. While many important findings are being established in the laboratory, researchers must find a way to translate the findings to real life interventions that serve to improve people's lives. Further, a big challenge for work in this area is to not only determine what interventions would work, but when to apply them. Are there critical periods in development? What needs to be applied early vs. can show positive effects when applied later? In keeping these questions in mind, basic science can contribute to understanding and improving people's lives.

Importantly, these themes were not mutually exclusive. Taken together, the presented talks and discussions highlighted the need for interdisciplinary research that incorporates multiple levels of analysis, and which takes a lifecourse perspective and individuals differences into account. Ultimately, the goal of such studies is to pinpoint findings that can be applied to the real world in order to improve functioning across the lifespan. The workshop speakers and participants were encouraged to focus on future-directed research ideas that bridged disciplines and could advance the field in such ways. Representatives from the NIA urged participants to "think large-scale" and suggested ways to create collaborations and secure funding, including RFA's, RO1 applications, and program project grants. A goal for this meeting was not only to present a snapshot of ongoing studies and questions but also to set the stage for future ongoing meetings designed to bring researchers together across disciplines in order to advance the field of aging research.

## List of Abbreviations

5-HT	Serotonin
5-HTT	Serotonin transport protein
AD	Alzheimer's disease
APS	Association for Psychological Science
BSR	Behavioral and Social Research Program
CHASRS	Chicago Health, Aging, and Social Relations Study
CHS	Charleston Heart Study
CRP	C-reactive protein
CSR	Central Nervous System Serotonergic Responsivity
fMRI	Functional magnetic resonance imaging
HPA	Hypothalamic-pituitary-adrenal
IL-6	Interleukin 6
IPSR	Institute for Personality and Social Research
MI	Myocardial infarction
NAS	National Academy of Science
NIA	National Institute on Aging
NIMH	National Institute of Mental Health
NSHAP	National Social Life, Health and Aging Project
PFC	Prefrontal cortex
RAND	Research and Development Corporation
RFA	Request for applications
SES	Socioeconomic status
SST	Socioemotional selectivity theory
TRED	Task-Rated Emotional Difficulty Index
TPR	Total peripheral resistance
VL PFC	Ventrolateral prefrontal cortex

**National Institute on Aging/Institute for Personality and Social Research  
Workshop on Advancing Integrative Psychological Research on Adaptive and Healthy Aging**

**Workshop Summary: Introduction**

This workshop “Advancing Integrative Psychological Research on Adaptive and Healthy Aging” was organized by the University of California at Berkeley, Institute for Personality and Social Research (IPSR) and the Division of Behavioral and Social Research (BSR) of the National Institute on Aging (NIA). It took place at Berkeley one day prior to the 2009 annual convention of the Association for Psychological Science (APS) (held in San Francisco on May 22-25, 2009). The workshop focused on advancing discussion in core emerging areas of psychological science in aging concerning emotional function, social relationships, and social behaviors. The goal was to highlight novel approaches currently being taken in the field, and encourage cross-talk and interaction between researchers in the field of aging and mainstream psychological scientists.

***Introduction and Framing the Questions***

**Robert Levenson, University of California, Berkeley**

The workshop was structured with two goals in mind. First, it features relatively short talks in order to maximize the opportunity for discussion. Second, this workshop is oriented toward encouraging people to talk about the things that they are really interested in and excited about, including the things that they don't understand yet. This is a gathering people of people with strong interests in aging research, and the NIA is very interested in future-oriented research ideas that may come out of this meeting.

***NIA interests***

**Lisbeth Nielsen, National Institute on Aging**

Last year at APS, the NIA held a didactic workshop entitled *Workshop on Entering into Aging Research*, which highlighted the accomplishments of leading researchers in psychology of aging and provided information on NIA grants. Today's workshop is intended to build on last year's, with the goal of providing exposure to ongoing aging research and offering a better chance to see, and tackle, some of the scientific questions in this area.

The NIA has also been holding a series of related meetings which incorporate areas of health psychology, psychology and economics, and social, cognitive and affective neuroscience, including the NIA Workshops on *Neuroeconomics of Aging* and *Social Neuroscience of Aging*, and NIA Workshops on *Allostatic Load* and *Stress, Aging, the Brain and the Body*. In addition, there was a meeting last summer with the Economic and Social Research Council (ESRC) in the U.K., which was designed to bridge laboratory and survey research to address questions about social and economic behavior in aging with a focus on social neuroscience.

The Division of Behavioral and Social Research (BSR) is focused on building research programs that cut across psychology, economics, demography, sociology, and related fields. These areas of emphasis include: health disparities due to racial, ethnic, socioeconomic, or gender differences; changes in aging minds; ways to increase health expectancy; the relationship between health, work, and retirement; interventions and behavior change; the ways that genetics, behavior, and the social environment interact; and understanding the burden of illness and the efficiency of health systems. Psychology has something to bring to bear on all of these questions.

There are several themes that cut across the entire Division. First, BSR takes a life course perspective and looks at aging as a process from birth to death. The NIA not only wants to understand phenomena particularly relevant to adulthood and older age, but also to examine early life influences on later life

outcomes. Second, BSR is interested in biobehavioral linkages and collaboration with other NIA programs in neuroscience, biology, and clinical practice. Third, we are focused on looking at multilevel interactions among the psychological, physiological, social, and cultural levels. Fourth, we'd like to see efforts to develop and improve methodology and measurement for all of the domains which we study. And finally, the NIA urges the translation and application of the findings of the basic sciences to some of the critical questions of aging.

Every four years our division is reviewed by our National Advisory Council on Aging, and last year (2008) was our most recent review. A number of subcommittees convened to discuss current directions and future needs in many areas, including: social neuroscience and neuroeconomics; the psychology of aging; behavioral and population genetics; behavioral economics and community interventions; health disparities; cognitive interventions; and questions related to measuring time use, well-being, and the experience of death.

Several broad recommendations for BSR program development came out of this review, including: 1) emphasize integrative science and multilevel analyses; 2) promote studies that look at issues of aging from a life course perspective; 3) encourage the team science necessary to advance these kinds of agendas; and 4) promote new training opportunities like this workshop today. Finally, the key recommendation was that the NIA should support research that integrates multiple levels of inquiry (e.g. genes, biomarkers, neural systems, behaviors). Such projects are necessary for elucidating the pathways that link social behaviors and social environments to age-related outcomes. Ultimately, these findings will be crucial to form the interventions that will improve the lives of older people.

The review of our psychology portfolio emphasized that most innovative research in psychology crosses traditional subdisciplines. People are focusing on how social, emotional, and cognitive factors interact to influence the ways in which people live their lives (e.g., how they make decisions, respond to stress, maintain important relationships, regulate strong emotions, and how these efforts affect their physical health). The best research is addressing these issues at multiple levels and blurs the traditional boundaries of cognitive, social, and personality research.

Further, studies are needed that help improve adaptive daily functioning and identify causal mechanisms that contribute to an individual's resilience. Thus, a focus of this meeting is to have a dialogue about what constitutes adaptive or healthy aging, and how basic science can contribute to understanding and improving people's lives. The concept of health needs to be broadened from the idea of being free of disease and disability to a conception that includes well-being, thriving, and living well in your environment. The NIA is very encouraged to provide the infrastructure and support for efforts that will support both large scale research and innovative training to advance this goal.

In its social neuroscience and neuroeconomics portfolio, NIA has been focusing on decision-making and economic behavior. Development of this area could benefit from a greater emphasis on the effectiveness and role of social relationships, emotional reasoning, and emotion regulation across the lifespan, as well as the influence of social and emotional factors in social behavior, decision-making, and health. There is also the potential to incorporate genetic approaches in these domains, to examine the ways that gene expression and age-related changes in the central nervous system affect social cognition, emotion, and economic decision making across the lifespan. Finally, BSR is interested in integrating social neuroscience and neuroeconomics with behavioral intervention research to improve understanding of how incentives influence behavior change.

The point of today's meeting is to bring researchers new to aging together with leaders in the field, to encourage a greater focus on issues of relevance to the NIA Mission, and to continue to advance the dialogues begun at other meetings over the past few years. The main hope is to address these three areas over the course of the day:

- 1) Encourage consideration of how to advance study of adaptive and healthy aging using integrative approaches bridging psychology and other disciplines,

- 2) Encourage cross-talk between scientists studying social and emotional function, psychological and biological processes in health, and decision-making,
- 3) Identify key opportunities and needs for advancing the science in each domain, and to begin to address how the research being discussed could really help change and improve people's lives.

### ***NIA vision***

#### **Richard Suzman, National Institute on Aging**

Workshops like these--that give people a broader perspective and promote more integrative research--are a cornerstone of BSR programmatic efforts. About 15 years ago, the NIA set up a Summer Institute at RAND for social and behavioral scientists that included several days of didactic research presentations by some of the best people in the field. When those meetings began, training programs in demography and economics were very fragmented and partial. Bringing graduate students and junior faculty together for a series of master lectures had the benefit of getting the leading experts in multiple areas together for several days, talking and lecturing and engaging in discussions about how to move the field forward. This RAND Summer Institute (RSI) has become an annual event for demographers and social scientists, and has provided the entrée in to aging research for many new investigators. The RSI is paired with a mini-Med School and is designed to introduce behavioral researchers to biology and clinical medicine. Dr. Suzman suggested that participants consider attending both ([www.rand.org/labor/aging/rsi/](http://www.rand.org/labor/aging/rsi/)). This meeting today represents an effort on the part of BSR to stimulate something like this process in psychology and related fields.

BSR is unique in our desire to see vertical integration from basic to applied research in the programs we support. A focus on application can drive basic science in important ways. For psychologists, BSR offers the opportunity to forge links to other disciplines that are customers for your research (e.g., the demographers, the sociologists, the economists, the epidemiologists). The emphasis is on both depth and breadth. The Division is open to high risk research applications, and we are willing to accept the risk of failure. Taking risks is necessary to break down old paradigms and establish new ones.

BSR does not subscribe to a narrow vision of aging research, although this view is not always shared by study sections, and we are trying hard to change that outmoded stereotype. Many constructs that are studied in childhood are highly relevant to aging, and aging processes begin much earlier in the lifespan than age 65. Our largest study starts at age 51, and many of our studies begin at age 30. We are increasingly searching for or helping create birth to old age longitudinal studies. We are interested in both the process of development and aging over the life course, and also older people. So we take a very broad view of what science is aging-relevant, and encourage applications across the spectrum. The critical thing for applications to NIA is to be able to show and have a reasonable story for why the factors under study should be of interest to the larger aging research community.

Perhaps less known to this group, the NIA supports a large number of longitudinal studies. Some were started by psychologists, but they are increasingly interdisciplinary. Many have significant components of cognitive and personality assessment, and they are increasingly collecting DNA and are moving towards looking at gene-behavior association studies in the future. There is a new age of efforts for integrating the genetic information of large, national, longitudinal populations into the behavioral model.

In upcoming RFA's, BSR will be soliciting applications for small networks, for behavioral economics applied to changing health behaviors, and for integration of longitudinal studies (especially twin and family studies, directed toward integration of genetic analysis). We are also trying to bring the field and the lab closer together, and have more interaction between them. This will happen in other ways in larger areas surrounding comparative effectiveness research, for which NIH has received 400 million dollars. This movement is going to continue to increase integration between observational studies and interventions.

The stimulus funds created a wonderful opportunity and generated a lot of work developing new ideas. Moving forward, participants should discuss with NIA staff ways in which their research programs can be brought to bear on important issues relevant to aging and the opportunities our new initiatives represent.

## Session 1: Fundamental Social and Affective Processes in Aging

### *Why isn't aging depressing?*

Laura Carstensen, Stanford University

Emotion does not follow the same downward trajectory as cognitive and physical aging. Everyone initially assumed that the emotion system would deteriorate like everything else, but it turns out that it's not true. It's not that everything is great, but it's wrong to say that aging leads to depression or even unhappiness. There is some evidence that it may be quite the opposite.

Consider the oldest people that ever lived. Jeanne Louise Calment maintained a great sense of humor through her 122 years of life. When asked at age 120 what kind of future she expected, she said, "A short one." When introduced to the oldest man who ever lived (age 115), Dr. Carstensen asked him, "What makes you happy?" He said, "Being around people who are happy." Then she asked, "And what makes you sad these days?" He replied, "What's there to be sad about?"

Longitudinal data on the trajectory of emotional experience also looks pretty positive. The balance of emotional experience (growth curves of positive minus negative emotion) appears improve until the very end of life (manuscript in preparation). After approximately age 70, there is a slight downturn, but never returning to levels as low as it was in early life. In emotion research, it appears the early years are the hardest years (20's – 30's). This finding has been supported both cross-sectionally and longitudinally.

Many consider this paradoxical: how can it be, given that so much does go wrong, that older people are doing as well as they are? Dr. Carstensen's group has been working from the socioemotional selectivity theory (SST).<sup>1-3</sup> This theory of motivation is grounded in the uniquely human ability to monitor time. People know when they are coming closer to the end of life. Goals are always set in a temporal context. They suggest that goals change in systematic ways, not because of aging per se, but because the time horizon is getting shorter. When time is open-ended, as it is typically perceived in youth, people are in a motivational state to gather information, learn new things, take risks, etc. But as time horizons become constrained, people shift to a more present-focus and pursue goals that are about meaning, well-being, and can be realized in the present. Under those conditions, how one feels at the moment takes priority. Emotion is well-maintained in part because more resources are being afforded to the emotion regulation.

Selection is the key here. People are selecting cognitively, they are selecting socially, and they are selectively remembering things. This may be aimed at regulating emotion states. The literature on social networks shows that they get smaller over time, but these networks are being selectively pruned to eliminating non-meaningful relationships. In some ways, social relationships become more emotionally supportive. It's the best ones that remain.

Emotional experience seems to be better with age. Older people are better at regulating conflict.<sup>4,5</sup> They have fewer conflicts and show more avoidance of conflict in an effort to reset the temperature of the conflict. When overhearing derogatory comments, older people don't get as angry as younger people; instead, they get sad<sup>6</sup>. It also appears that poignancy (the co-occurrence of positive and negative emotions) increases with age.<sup>7</sup> Thus, the emotion system isn't broken, it's just different.

Dr. Carstensen and colleagues have examined the cognitive consequences of these motivational shifts. Goals direct attention, memory, and action. If goals change, you would expect systematic changes in what people attend to and what they remember. Mara Mather led a study wherein subjects were shown two images of the same person making an emotion face (positive or negative) and a neutral face.<sup>8</sup> When the neutral face was paired with the negative face, older people looked more toward neutral. When the neutral face was paired with the positive face, older people looked more toward positive. This suggests that resources are being allocated to well-being and regulation.

Many studies have identified a relative preference among older people for positive information over negative information. This may reflect a developmental shift in motivation across adulthood which we call the "positivity effect."<sup>8-12</sup> This shift is adaptive for one's place in the lifecycle, is context specific, and is

aimed at realizing chronically activated goals. It's not good in all circumstances, however, and it probably wouldn't be good for younger adults. For example, Löckenhoff showed that if you present people with a decision (e.g., about health care plans), older people disproportionately attend to positive information over negative.<sup>13</sup> It seems this may be goal directed, not because of neural degradation. When Löckenhoff told subjects to be as accurate as they can (i.e. changed the goal), the positivity effect disappeared.

Dr. Carstensen and colleagues have investigated differences in the anticipation of positive and negative emotion. In an fMRI study, older people appear to respond to potential gains as younger people do (similar brain activation). In loss trials, however, younger adults show similar levels of activation, but older adults do not show much activation.<sup>14</sup> This could have the practical implication of making older adults more susceptible to fraud. Further, Samanez-Larkin has recently done work showing that older adults may learn just as well as younger adults on positive learning trials (gain learning), but they don't learn well on loss trials (avoidance learning). This has implication for how one should frame information and intervene when you need a message fully understood and remembered by older adults.

In sum, it seems that there is a systematic change in motivation that benefits emotion and emotional well-being. It's a change that directs cognitive resources to particular kinds of stimuli. Now that this pattern has been identified, we can apply it to a lot of applied questions systematically to see if we can use these kinds of findings to improve functioning in different areas.

*Discussion:*

A participant (name unknown) asked what was understood about the variability in the changes Dr. Carstensen had discussed. Dr. Carstensen acknowledged that she hasn't looked a lot at individual differences, but she wouldn't rule them out. Social selection is good for mental health, so if you look at people who don't do the pruning process, they are suffering more than people who are.

David Sbarra then asked if he could just prune right now and acquire the benefit of deepened meaning, or does the life course process require that we broaden and expand first and then select? Further, if you were to reverse-engineer these processes, what are the applications? Dr. Carstensen noted that you don't have to be old to see the same effect. Studies show that people who have HIV (i.e., who are closer to the end of their lives even though they are younger) show the same sorts of changes. The SST is not an age theory, but a theory of shifting time horizons. But can one volitionally say, "I'm going to deepen my experiences?" Dr. Carstensen is currently involved in research where they are training Buddhist meditation in younger adults to see if they can instantiate this kind of positivity effect. It's the paradoxical idea that if you make people think more about the end of life they shift in a positive direction.

Sonja Lyubomirsky asked whether the effect may be due to the way that people are interacting with older adults. For example, people may be less negative toward older adults, and thus it may be a social effect. Dr. Carstensen noted that she is currently looking at younger adult's time horizons with different social partners. For example, if you are interacting with your grandmother, do you also shift your goals for that relationship? This seems to be supported by the data.

Finally, Natalie Denburg asked whether the effects hold for people with mental illness. Dr. Carstensen replied that she doesn't know. They tried to run a study on depression but couldn't find enough older subjects. She hypothesized, however, that because the phenomenon stems from regulatory efforts, one wouldn't expect to see the effect in people with dysfunctional abilities to regulate. Mara Mather conducted a study supporting this motivational explanation. Specifically, if you place high cognitive load on subjects during a task, the positivity task is eliminated and actually becomes a negativity effect. This suggests that people are putting some effortful cognitive energy into positivity. Further, in that study, people with the highest levels of executive functioning show the positivity effect the most. This suggests that you wouldn't expect to see the positivity effect in individuals with dementia, for example.

***Aging and the use of emotional cues to guide social judgments***  
**Louise Phillips, Aberdeen University**

Dr. Phillips's research focuses on age differences within normal aging and abnormal aging, such as dementia, stroke, etc, and seeing how these things affect social cue processing in older age. In particular, she is looking at changes across the lifespan in the abilities and motivations to process emotional information from other people. The research is starting to more clearly show the effects aging has on emotion perceptions, as well as provide an understanding of some of the factors that underlie those differences (e.g., neural, motivational, cognitive, and perceptual issues). She is further interested whether there are social consequences to age-differences in the ability and motivation to interpret emotion cues from other people.

To give some background, Dr. Phillips cited a meta-analysis of studies in which subjects were shown pictures of Ekman faces and asked to identify what kind of emotion was being shown. There was a relatively clear pattern of results: older adults were less accurate in identifying facial expressions of sadness, anger and fear.<sup>15</sup> The age differences between happiness and surprise were much smaller, and older people were actually better than younger people at identifying expressions of disgust.

Thus, it seems that as people get older, they get worse at identifying some expressions of emotions, which represent important social cues. What might the social consequences of these differences in emotion recognition be? Dr. Phillips and colleagues are examining whether there might be a relationship between emotion recognition and aspects of social participation. They are looking at this in stroke and dementia patients, as well as in studies using longitudinal designs to see whether any age effects in emotion perception might have implications for social judgments, or for judgments of social approach.

There is evidence that gaze cues (where eyes are looking) moderates perception of emotion. For example, researchers found that when younger adults are asked which of two faces is more angry (and shown picture of two angry faces, one with gaze straight ahead and one with gaze off to the side), they tend to pick the one with direct gaze over the one with averted gaze.<sup>16</sup> This makes sense—if someone is angry and looking directly at you, they might be angry at you (and this might imply a response is needed).

Past research has shown that older people have more trouble recognizing anger,<sup>17</sup> and that older people process information about eyes differently.<sup>18</sup> For example, older adults are less able to discriminate subtle differences in eye gaze, and they are less susceptible to joint attention (following eye gaze of other people). Because of this, Dr. Phillips and colleagues investigated whether there are age differences in integrating gaze and emotion cues. They hypothesized that older people would make less distinction between direct and averted gaze when responding to angry faces. To test this, they began with the basic task of asking which of two faces was angrier (direct gaze or averted gaze). Then, to assess social approach behavior, they asked which the subject would be most likely to ask for a favor.

Results showed that young people more often selected the direct gaze as being angrier, whereas older people performed exactly at chance. In the social task, they expected that people would choose the face with the averted gaze, because that face would be perceived to be less threatening. This was true for young people—they chose the person with the averted gaze to ask for a favor significantly more often. Older adults, on the other hand, were significantly more likely to choose the person with the direct gaze. This suggests that younger and older adults differ in the effects of emotion on social judgments. Unlike younger adults, that older adult's judgments of emotion appear unaffected by gaze direction.

One interpretation for the findings integrating eye gaze and emotion involves a cognitive load, and older people are not performing as well. Other explanations implicate motivational factors or neural changes. Further, older adult's decisions about approach behavior were influenced by eye contact rather than by the emotion-eye gaze interaction. This suggests that there are different reasons for older and younger adult's social decisions. These age-related changes in emotional-social cue responses could have important influences on social behavior or social consequences. Finally, she noted that a key challenge for this research is reaching outside of the lab and looking in more detail at real-life social behaviors.

*Discussion:*

Steve Manuck asked if the lack of discrimination may reflect a more basic lack of recruitment of attentional resources, which would be consistent with the reduced amygdale activation in response to the angry faces (and is also associated with age-related declines in androgen levels). Dr. Phillips replied that they are considering that explanation, but questioned whether that could explain the kind of opposite choice that they found. It might be able to—subjects think, “This person is looking at me so I’m going to choose them.” Related to that point, Bob Levenson suggested that perhaps in later life, people feel more invisible, so they show a preference for the face that is attending to them.

Laura Carstensen asked if it was possible that some emotions (e.g., happiness, disgust) are just easier to recognize than some of the other emotions (e.g., surprise). Dr. Phillips noted a correction to her slides: their meta-analysis showed very small age differences in identifying happiness and surprise (older people were worse). But those differences so small that they weren’t significant in any of the individual studies. Further, one way to deal with this issue of different signal strengths is to make the expression less intense, therefore making the task more difficult. When they have done that, however, older people still identified disgust better than younger people. Bob Levenson pointed out that more muscles (i.e., perceptual cues) were involved in the expressions that showed no age differences (i.e., happiness, surprise, and disgust) compared to those that showed age differences (i.e., anger, sadness, and fear). Dr. Phillips noted that care needs to be taken to not over-interpret the data as she can’t tell what aspects of the findings are related specifically to the Ekman faces versus more general aspects of emotion.

Finally, a participant (name unknown) asked if the difficulty was in differentiating between different types of emotions, or in identifying negative emotions. Dr. Phillips responded that differentiating between negative emotions seems to be the key issue here.

***What is the function of age-related positive gaze preferences?***

**Derek Isaacowitz, Brandeis University**

Dr. Isaacowitz discussed the degree to which older and younger adults differ in visual processing of emotional images, and to what extent older adults are pursuing emotional goals and goals of emotional regulation in their visual processing of emotional images. In past work, he and his colleagues have found evidence of possible “positivity effects” in older adults’ visual processing—older adults preferentially attend to positive over negative stimuli. Recent research has tried to demonstrate what the function of these visual preferences might be. It’s hard to determine whether older adults are indeed using these positive visual preferences to try to regulate how they feel. These are attempts to illustrate that positive gaze preferences in older adults show a mood regulatory function.

Dr. Isaacowitz and his colleagues used eye tracking methodology. Generally, the subject sits in front of a monitor with the eye tracking device underneath, pointed at the subject’s eye. The subject views sets of visual stimuli, such as pairs of emotional faces, and is asked them to view these faces naturally, as if they were home watching television, in order to get a sense of their naturalistic gaze preferences.

The goals were first to replicate previous findings that older adults look more at positive stimuli, and then to test whether there was a link between older adults looking pattern and their mood. If positive gaze preferences reflect older adult’s attempts to regulate emotions, one would then expect that older subjects who have a mood they need to regulate would demonstrate positive gaze preferences. And that is, in fact, what they found. Subjects participated in an eye-tracking task where they were shown two faces: one neutral and one emotional (either positive or negative). When subjects were in a positive or neutral mood, there was not much effect. Young adults tended to show mood congruent gaze patterns, and older adults didn’t show much preferential looking. But when subjects came to the task in a negative mood, differences emerged. Younger adults in tended to look more at the negative faces, whereas older adults tended to look at happy faces and look away from all three negative emotion faces. Thus, it seems that positive gaze preferences were activated when older adults were motivated to regulate this mood.<sup>19</sup>

This provides some support for the idea that positive gaze preferences show a mood regulatory function. But the further functional question is: does this work? The answer is sometimes. It seems to depend on a number of other factors. It seems that looking at positive things sometimes helps older people to feel better, but it appears to depend on factors both related to the person and to the situation. In particular, age makes a difference (this is not surprising). Trait attentional functioning (i.e. how well the person's attentional abilities are operating) makes a difference as well. The time length of the regulatory task also seems to matter (e.g. is it a long task that requires regulation over a long time period, or is the situation one where regulation can happen quickly?).

To follow this up, Dr. Isaacowitz and colleagues conducted a study where subjects did a task that was very boring. Over this 25-minute boring task, most subjects experienced mood impairment, with mood ratings going down from the beginning to the end of the task. They found that whether the task made people feel worse or whether people were able to stave off the negative mood effect varied by age and the function of the attentional system. Specifically, the older subjects who stayed okay (mood did not decline) during the task looked at more positive stimuli. They were also the ones who had lower conflict scores (meaning their executive functioning was good). Older subjects, looking at happy faces, with good executive functioning, felt the best (i.e. they staved off negative mood). There were very different results for young adults who were able to feel okay—they showed the least positive preference in their gaze, but had the best executive functioning. Other older adults showing the positive gaze preference still suffered a mood decline if they weren't high on executive function. In sum, older adults with high levels of executive control feel best if attending to positive stimuli, whereas young adults with high executive control feel best when looking less at the positive stimuli.

The next question to address is why this is. Are the young adults using the non-positive emotion stimuli as cues for reappraisal or some other regulation strategy? It's clear that younger adults are not benefitting from positive looking strategies like the older adults do in some cases.

In terms of implications, clearly there are age differences in gaze patterns related to emotion or mood regulatory processes. But there are individual differences in whether those regulatory processes lead to adaptive regulatory outcomes (feeling good). What this means is that we need to think about different paths to emotion and mood regulation, for different individuals in different contexts; not all paths lead to successful regulation. Some paths for some people in some contexts are maladaptive. Finally, looking at the function of these looking preferences in real-time is complex. But it's important to figure out specifically what things like positive gaze preferences are doing for older people and when.

*Discussion:*

Jenny Beer asked if perhaps the findings in the study with the negative mood induction might change if the subjects were subjected to cognitive load. Dr. Isaacowitz noted that studies have been done where cognitive load was manipulated in older subjects, and results showed that a big cognitive load disrupts positivity effects, but a small cognitive load is okay. We don't know as much about cognitive load effects on gaze and emotion regulation in younger adults. Dr. Beer suggested that perhaps what he was seeing is that people were moving to default strategy instead of regulating emotion.

***Effective emotion regulation in older adulthood: Converging levels of analysis***  
**Fredda Blanchard-Fields, Georgia Institute of Technology**

What are candidates to explain the paradox of why older adults tend to remain happy throughout most of their adult lives? One explanation lies in emotion, specifically the tendency to avoid negative arousal and manage arousal. This leads to the questions: what constitutes effective emotion regulation, and are older adults really more effective at that?

In past research, Dr. Blanchard-Fields has found that older adults report that they use a variety of context-sensitive regulation strategies, they read the problem situation, they are more concerned about regulating emotions as well as solving the problem, and they strategically employ combinations of strategies to deal with their emotions. In general, they report that they are more effective. But the field

needs converging levels of analysis to deal with this question of effectiveness. To move beyond self-report, Dr. Blanchard-Fields is conducting a study wherein subjects are asked, "In the past two hours, what problems did you experience and how did you regulate them?" Further, more research is needed that integrates emotions and stress (e.g. biomarkers of physiological wear and tear) and that explores the links between behavior and brain activation (e.g. what is being activated when emotions are being regulated). Such studies will allow more objective measures of effective regulation in terms of physiological arousal and health outcomes.

Dr. Blanchard-Fields has recently conducted studies of on-line emotion regulation. One thing that older adults do best is avoid negative emotions and high arousal (i.e. they select it out). But you can't always do that. People are exposed to negative emotion, so how do they regulate when you can't select it out?

Dr. Blanchard-Fields defined emotion regulation efficiency as the degree to which there are cognitive costs (i.e., it's very easy vs. difficult) for older adults to regulate emotion. The decline in cognitive control with age should lead to more effortful emotion regulation in older adults in comparison to young adults. There is neuroscience evidence for increased activation of older adults in areas related to cognitive control, and executive functioning is implicated in emotion regulation (e.g., Oschner). So you would think that would mean that older adults probably aren't so good at emotion regulation. It takes too much cognitive effort. The whole history of cognitive aging research shows that when you increase cognitive load, older adults do more poorly. However, given that growing older is related to increased focus on and better regulation of emotions, it could be less costly for older adults than it is for young adults

The researchers used a dual-task paradigm. Dr. Blanchard-Fields and colleagues induced disgust using film clips, then instructed individuals to either down-regulate that disgust (i.e., make it as positive as you can), gave them no instructions at all, or did not show them the film. As they subjects are either regulating (or not) the emotions, they also have to perform a working memory task known to tax executive functioning. They were interested in the degree to which regulating emotion disrupted learning in this working memory task. They found that instructions to down-regulate emotions disrupted working memory performance in young adults, but not in older adults.<sup>20</sup> Specifically, for young adults, regulating emotion disrupted performance in the working memory task, but for older adults, it actually enhanced their performance. This suggests that something is going on with the executive functioning implicated in emotion regulation which should make it more difficult for older adults. Perhaps for them, it's a more well-practiced skill. Dr. Blanchard-Fields is now collaborating with Erin Senesac to look at another form of emotion regulation: suppression. When they ask older adults to suppress the expression of emotion then perform a Stroop task, there appears to be more of a cognitive cost on their performance.

In general, they are interested in trying to define the possible automaticity of emotion regulation in older adults, and the issue of suppression of the expression of emotion may require greater resources. It's a much more complex picture than just one of older adults being better at regulating emotions than young adults when you start getting at fine-grained analyses.

Anger is emerging as a special case. Older adults seem to avoid anger and are worse at recognizing anger. So she and colleagues did a study looking at angry, sad, and happy faces. Young adults show increased amplitudes of the P1 components at occipito-temporal electrodes for all emotional facial expressions (happy, sad, and angry) as compared to neutral. That is, there is an emotion enhancement effect when looking at an emotion stimulus. Older adults show no effect in response to happy or sad faces, but they actually show a deamplification in response to angry faces. That is, older adults withdraw attention from angry faces. They not only select out, but they deamplified from that negative emotion.

*Discussion:*

Louise Phillips noted that when she and colleagues have done suppression tasks with older adults, they were actually better at suppressing. She wondered whether it was a trade-off, that older adults were actually suppressing more and asked if Dr. Blanchard-Fields measured the extent to which they were actually suppressing. Dr. Blanchard-Fields responded that they were collecting data on facial expressions and planned to code behaviors (i.e., suppression). Older adults don't tend suppress emotions that much, so it might require more cognitive effort. The cognitive depletion literature tends not

to distinguish between downregulating experience and suppression of emotion, and there might be really different costs. Someone then asked if there were age differences in the downregulation success, and Dr. Blanchard-Fields confirmed that there were no differences in this area or in emotional reactions.

Lani Shiota asked if Dr. Blanchard-Fields might be able to zero in on what strategies people were using to downregulate, and wondered what specific downregulation instructions were being used. Dr. Blanchard-Fields could not remember the precise language, but thought that subjects in that condition were instructed to downregulate your emotions so that they feel positive. The key point is that it is still an open question about what they were doing to downregulate the experience of emotion. In a new study, they are differentiating between different regulation strategies. They have found that younger adults begin to look like older adults when they were asked to avoid, not reappraise, negative stimuli when watching a negative film. She noted that further work is needed to pinpoint emotion regulation strategies.

James Gross asked Dr. Blanchard-Fields to discuss why she had to drop some subjects. Were there differences in this group as a function of age? Dr. Blanchard-Fields explained that they were using Fear Factor clips as stimuli, and several young adults found the clips to be funny. Those were the primary subjects who were “thrown out,” though she noted that they are looking at those subjects as well.

### ***Correlates of social position in brain serotonergic function***

**Steve Manuck, University of Pittsburgh**

Epidemiology has identified a number of risk factors for atherosclerotic cardiovascular disease, including psychosocial (e.g., mood/ affective traits, disorders, hopelessness, anger/hostility, social isolation), lifestyle/habit (e.g. smoking, physical inactivity, diet, immoderate alcohol use), and biological risk factors (e.g., atherosclerosis, dyslipidemia, obesity, blood pressure, insulin resistance, inflammation, autonomic dysregulation, cardiovascular reactivity). Many of these risk factors tend to covary in populations and aggregate in individuals. Therefore, even though these risk factors might superficially appear to have distinct etiologies, they are often strongly associated (e.g. high levels of anger/hostility and IL-6). This has prompted Dr. Manuck and colleagues to consider whether there are common mechanisms that might join many risk factors in populations and in individuals. To start with, they chose central neuromodulator serotonin (5-HT). Among other reasons, they chose 5-HT because it has been found to modulate activity in neural circuitry of emotional processing and motivation, as well as those governing circulatory function, metabolism, neuroendocrine and autonomic function.

5-HT function is studied in their research through genetic variation, but more commonly through a neuropharmacologic challenge by which net central nervous system serotonergic responsivity (CSR) is indexed as the relative rise in concentration of the pituitary hormone prolactin following acute stimulation by administration of a serotonin agonist. They looked at all of the aforementioned risk factors (psychosocial, lifestyle, biological) and found that they are all associated with low 5-HT functioning, as reflected by the CSR index. For example, socioeconomic indicators (low education, income) at both the individual and community levels are associated with all of the same risk factors for heart disease that are associated with attenuated CSR. What does that mean? Is there an association between 5-HT function and social positioning as well? There is some precedent for thinking so from experimental primate studies. They found that chronically perturbing the status relationship of male cynomolgus monkey by repeatedly reorganizing social group memberships lowered 5-HT levels in the prefrontal cortex of these animals in relationship to those of unperturbed males (i.e., monkeys housed in stable social groups).<sup>21</sup> Second, peer rearing, which is a model of maternal deprivation causes lifelong suppression of 5-HT turnover in rhesus monkeys, both as juveniles and adults, relative to mother-reared adults. Finally, in humans, individuals of lower SES, lower in income and educational attainment, in a community sample, exhibit the least CSR.<sup>22</sup>

Going back to the sample of peer-reared rhesus monkeys, the effect was only seen in monkeys that carried a particular allele for a regulatory polymorphism in the 5-HTT gene that confers reduced transcriptional efficiency on that gene.<sup>23</sup> There is an orthologous polymorphism in humans, located in chromosome 17. We find that the covariation in socioeconomic indicators with CSR varies by genotype

such that SES accounts for a quarter of the variance in 5-HT function among individuals who are homozygous for the deletion allele (s/s) and none among individuals who are homozygous for the l/l allele, with intermediate effects for those with one long and one short allele.

Finally, another way of indexing social disparities is via census data characterizing neighborhoods. Dr. Manuck and colleagues asked whether socioeconomic disadvantage may be related to reduced CSR in their community sample in areas of lower income, and found that area-level socioeconomic indicators did covary with CSR.<sup>24</sup> This association was independent of personal SES indicators, and also independent of enduring personal characteristics that are related to neighborhoods of residence such as personality, IQ, and diet. There were some age differences in this association—area level predictors were associated with reduced CSR functioning for older (but not younger) men, and younger (but not older) women.

Community SES in younger women is associated with a number of cardiovascular risk factors. Adjusting for CSR, those associations dissipate. While meditational conclusions can't be made from cross-sectional data, it seems that the influences of SES indicators, which cannot be explained away by other person-level variables, may influence risk through its effect on the brain's biotonegic system.

*Discussion:*

Robert Levenson asked about the relationship between age and frontal 5-HT. Dr. Manuck noted that there is a decline—the aforementioned studies show an age main effect, and it probably accounts for about 14-15% of the variance, which is appreciable.

Laura Kubzansky asked whether there was one type of allele where peer-raising was not harmful. Peer-raising was not associated with variation in 5-HT turnover in case of homozygosity for the insertion allele of the transporter (note: rhesus macaques are rarely homozygous for short alleles). Dr. Kubzansky then wondered how this might fit with the idea that some of these alleles create hypersensitivity to the environment. Dr. Manuck indicated that this is relatively consistent—his data (and other evidence for gene-environment interactions) shows that certain genotypes are associated with a heightened sensitivity to environment, not necessarily pointing you in one direction or another, but rather the diathesis-stress models which suggests that environmental adversity conjoined with a particular genetic variant would produce the negative outcome. It seems that much of the data is consistent with the notion of genotype-dependent plasticity, and at the other end, genotype-dependent constraints on plasticity to environment.

Lis Nielsen noted that what's really interesting about this, from the aging perspective, is the extent to which the accumulation of adversity over the life course is leading to a typical aging disease outcome (e.g., cardiovascular disease) and the extent to which it might shed light on who's likely to develop adaptive patterns of regulation, and who's at risk. The studies she knows of typically control for SES, but the sample sizes aren't as large as what you are working with. It may be that the incorporation of specific lab measures with larger-scale studies could begin to shed light on some of those questions.

***Exploring the effects of attachment relationships on reactions to transitions***  
**Michael Lamb, University of Cambridge**

Dr. Lamb focused on children's reactions to life transitions, and he began by talking about the transition to childcare. This is the point at which children go from being cared for pretty much exclusively by parents to spending a considerable amount of time in childcare settings. This transition has profound effects on children's social and emotional well-being and behavior.

Some of that is evidenced by looking at children's attachments to their mothers. If you look at children's attachment to their parents twice, once pre-childcare then again after half of those children begin childcare and half do not, children who start childcare are twice as likely to change their security of attachment to their mother as those who do not. There is a considerable amount of transition taking place, even though the proportion of children who are secure is the same for both groups, so the transition is taking place in both directions.

To explore this transition, Dr. Lamb and colleagues undertook a study to examine children over the course of the transition to childcare. Multiple assessments occurred at home before the transition (baseline security and physiological reactivity), followed by intensive observations both during the transition to childcare and after. This study was conducted at German childcare centers, where, unlike in the U.S., parents are expected to stay with their children for several days around this transition time, so we have assessments of attachment during this time (Adaptation Phase) and after (Separation Phase).

Data showed that once children started the process of adaptation to childcare, there were very high increases in the expression of negative emotion, but over time, those levels started to decrease. That is, after they had been going for a while, the children adjusted. They next wanted to see whether there were physiological correlates to these changes which would allow exploration of the individual differences in these processes. One measure involved salivary cortisol. Data from a study of American childcare centers shows that the levels of cortisol over the first hour of childcare increase quite dramatically in the early days, but by later in the process you do not see dramatic increases over that time. The question is: what happens after the first hour for the children who are spending a long time in childcare?

They found that the younger children don't show the usual diurnal decline in cortisol. The very youngest children showed dramatically higher levels of cortisol later in the day.<sup>25</sup> This has been interpreted by many researchers to be a risk factor. In recent studies in German centers, we don't find the same pattern—kids showed the usual diurnal variations. The difference is quality of care. Thus, it appears that high quality care does make a dramatic difference for children's experiences (e.g. reduces the stress).

What else accounts for individual differences? We looked at differences in cardiac reactivity and related it to attachment security. The measure was RSA (higher levels are better). During adaptation period, securely attached children had higher levels of RSA. This effect carries over into the first day, and then the differences disappear. Is this because these children are forming meaningful relations to caregivers? This is not supported. On the other hand, it's clear that the mother's sensitivity and involvement in the children's lives did have an effect. Attachment to mother either stayed secure, or became secure, the longer the children stayed making the transition to childcare.

Current studies are looking at other transitions, such as the transition to school and the transition from secondary school to upper level school. The hope is to see comparable processes of emotional factors and particularly attachment relationships helping to explain how children make these transitions.

*Discussion:*

Mara Mather asked if, in the study examining cortisol patterns on different days, the levels of waking cortisol were different for the different days. Dr. Lamb confirmed that there is a significant difference between waking levels of cortisol on Friday and Sunday. The next question was whether, then, that meant the children were on a weekly rhythm. Dr. Lamb suggested that yes, they are. At the end of the week, they have a cumulative exposure to moderately stressful experiences, so levels are higher at that point going into the day. But by mid-morning, the differences are lost.

Brian Knutson asked if anyone has looked at kids who are homeschooled or stay home, and the reliability of attachment styles in those children. Dr. Lamb pointed out the control group portion of his dataset where 75 of the kids were not being sent to childcare. These children showed a fair amount of transition in attachment security (though this was about half the rate of those who changed in the childcare group).

Natalie Denburg asked him about his feelings of retrospective report of attachment, and whether she could feasibly do this with elderly people—how good would their retrospective accounts be? Dr. Lamb suggested that adult attachment researchers may be better equipped to address this question. Shelley Taylor responded—she has been looking at constructions and reconstructions that people make of their family environment based on their age now, it appears that age does not moderate the reconstruction of the affective tone or frequency of people who report negative experiences. There does not appear to be systematic distortion on the reconstruction of childhood events.

## **Session 1: Open discussion**

**Moderators: Lisbeth Nielsen and Robert Levenson**

Bob Levenson highlighted four common topics from the first panel: emotions, attachment, environment, and genes. He then asked the group to consider the following questions: What's next? What are the implications of this for how people live their lives successfully as they age? What are the most important things that we need to know, and are there obstacles to us knowing them?

Laura Carstensen's response was that in terms of adaptation: ultimately, we are looking to improve functioning across the life course. Some of what we learn about aging can also help improve younger people's lives. People want to create lives where they are not overly stressed in some chronic way, can achieve goals, and have successful relationships. In some cases, older people seem to be on to some strategies that are useful for emotional well-being. We're beginning to understand under what conditions these effects are present or certain strategies work. We need to put these pieces together to understand adaptation. The big win would be being able to change people who aren't doing well.

The discussion turned to individual differences. Dr. Levenson pointed out that people seem to be doing pretty well at the group level, but there is a great deal of variability. We can say that everything's okay because the group isn't doing as bad emotionally as everyone thought, or we can look at this variability and try to see who's doing well. Dr. Carstensen responded that we've identified a way of aging that is very good, and generally people are on that trajectory. But data shows that a high level of executive functioning predicts that trajectory, so perhaps that is the target. If people have cognitive/social control to achieve goals, they do well. If they don't have that control, the goals and outcomes are different.

Howie Rosen posed the question of what represents a deficiency that is common with aging versus an adaptation that is healthy and volitional. Dr. Carstensen suggested again that if cognitive abilities are relatively well-preserved, then older adults seem to do well. We know that it's not a serendipitous consequence of something going wrong (e.g., people focus on the positive because they aren't complex), so that's already a step forward.

Elisa Epel reminded the group that we need to keep a focus on what predicts good physical health. By the time you get older and have that shift in time perspective/emotion regulation, you are already on a trajectory of when you are going to die. So what sets people up on good health behaviors early in life? One major factor is time perspective, a sense of imminent vulnerability and mortality. So thinking about what mediates healthy adaptation when you are older, is the time shift the crucial element? Are there other mediators we can induce in younger people? And what are the moderators of this adaptation? Further, Dr. Epel noted a parallel in the stress field and the SST literature. People show growth and adaptation through severe trauma and stress—perhaps it is the threat to life. Dr. Carstensen noted that time perspectives shift gradually, and goals also follow that pattern. People show those same kinds of shifts during major transitions (e.g., graduating from college). It's not about death, it's about endings.

The workgroup then discussed how we deal with our knowledge of genetic risk. Dr. Levenson asked how close the field is to taking certain genotypes (e.g., long variant vs. short variant of SERT) and telling people they need to plan their lives differently. That is, how to play the hand that was dealt genetically in a way that minimizes problems and maximizes happiness? Steve Manuck indicated that the judgment would be right now that no genetic variation, even in areas with a lot of studies, is considered sufficiently predictive to pass on information to plan one's life around. It may be more interesting to know people's genotype in order to know who to target for more behavioral interventions. For example, regulatory variation MAO-A interacts with early adversity to predict violence. This is only true, however, for those who have certain attitudes, which aren't heritable themselves. This offers a better point of intervention than the underlying neurobiology. Functional neurobiology is very far from being understood.

Dr. Levenson asked if we, as a field, are afraid to pull trigger on using genetic data (i.e., to move forward and apply it in these ways that might have some benefits)? If so, why? Shelley Taylor responded that perhaps we don't need to be trigger happy. We know what good environments are, and we know that they benefit everybody. We don't need to take genetic background into account for many of the

recommendations we can make (e.g., try to get stress out of your life, have good friends). Dr. Levenson posed the question: if we have limited resources, should we target those who are at greatest risk (e.g., short alleles, insecure attachment)? Lisbeth Nielsen suggested that the best use of resources is to establish abilities early, versus trying to fix something that is broken later. That is why a life course perspective is critical—we need to target stages where interventions are most effective.

Dr. Carstensen asked if we know enough to say that if you have a particular profile in a particular environment (e.g., low SES), you will actually benefit from things like increased exercise? One participant (name unknown) reported recent findings showing that the relationship between early financial stress and later insulin resistance can be buffered by exercise at the earlier age. Laura Kubzansky suggested approaching this issue in terms of using the data to have insight into when to pull the trigger. For example, when are sensitive periods? Are the effects reversible? If a sensitive window was missed, who is the most vulnerable? We shouldn't discard the value of thinking from a social perspective and figuring out and directing policy at environments that are really most toxic. For example, do the cortisol variations that Michael Lamb presented predict adaptational/behavioral/health outcomes down the road? This would be a way to get at those sensitive, critical periods. On a related note, some early childhood research indicates that some stress is good so the child learns coping skills—perhaps the higher cortisol levels in those children predicts positive adaptation later. Following out over time would give insight into those adaptational processes, as well as what points of intervention would be most helpful. Dr. Lamb noted that the chronically high cortisol kids are the ones who are acting out. For challenges to be helpful, it needs to be kind of challenges you can master.

Dr. Nielsen applied this discussion to aging, asking what experimental studies show how adaptational processes in older age are affected when an individual faces challenges. Or what kind of studies would we need to answer that question? Louise Phillips responded that we need to understand social consequences much more. Some things that look like advantages might have negative consequences as well. Further, people who are having difficulties with things like emotion perception might be impacted in different ways (e.g., in the case of dementia). There may be specific interventions that may be useful, or there may be a use in educating people (e.g., dementia caregivers) on these changes and processes.

Derek Isaacowitz pointed out that we don't know that much about older adults who are poor regulators. Is it that they have always been this way, or are previously adaptive regulatory strategies just not working for a novel stressful challenge? We need to know more about this group—those who aren't doing so well. Dr. Phillips noted that this is a group which is very hard to recruit for studies. Fredda Blanchard-Fields agreed—it's not that all older adults are doing well, and we need innovative techniques to look at different levels of analysis (e.g., brain activation) in order to people doing well vs. not doing well, and why.

## Session 2: Healthy Aging over the Lifecourse

### ***Stress, social processes, and health over the lifecourse***

Shelley Taylor, University of California, Los Angeles

Dr. Taylor started by listing a few examples of research in the domain of stress, social processes, and health over the life course. This included: 1) animal models looking at early experiences and their lifelong effects on both behavioral and health outcomes (e.g., Meaney, Coplan and Rosenbloom, Suomi); clinical research looking at long term physical and mental health effects of things like maltreatment, abuse, and PTSD (e.g., Yahuda, Pollak); 3) developmental research on risky families (e.g. Repetti, Taylor), which has also involved relatively normal families that are marked by conflict or cold, unnurturing behaviors and show surprisingly similar lifelong effects like health indicators; and 4) childhood SES research, which shows health implications into adulthood (e.g., Chen, Miller).

The focal question that these lines of research raise is: how is it that the things that happen early in life effect mental and physical health late in life? Often you don't see the effects at all during adolescence, but then they reemerge in 40's and 50's. Somehow the processes are being set into effect, and the damage is being stored, in ways that achieve these long-term effects on health outcomes.

There are a number of metatheoretical perspectives which all have support. A big challenge will be trying to fit them together because they make very different assumptions and come from very different research traditions. The first is a developmental focus on socioemotional skills and deficits (e.g. poor emotion regulation, low social support). The notion is that early life experiences set into effect patterns of emotion regulation and the social/relational capabilities which have long term effects. Companion models include the idea that there are accumulating physiological costs to the ineffective regulation of stress (e.g. McEwen, allostatic load model). Implicitly, this model suggests that much of the damage is done during stressful times, though this is still an empirical question that should be investigated. A third set of models addresses early programming/critical periods (e.g., Barker hypothesis on prenatal nutrition; Meaney's HPA axis work). Finally, there are more "tonic" models that assume that if you have coping skills and regulatory skills in place, you can keep the organism on an even keel (e.g., Lyubomirsky, Pressman). To integrate these research perspectives, approaches are needed that cut across multiple levels of analysis. Dr. Taylor's current research is an example of such work, focusing on genes, neural processes in the brain, and neuroendocrine and immune functioning.

Miller and Chen have been looking at early life SES and how it affects glucocortical signaling with potential health implications across the lifespan, controlling for changes in SES.<sup>26</sup> Their findings parallel Meaney's work on glucocorticoid receptors in animal studies. Dr. Taylor current work has focused on the 5-HTTLPR gene. Building on Caspi's research showing that people homozygous for the short allele are more vulnerable to depression across the lifespan, she and her colleagues have found that this gene seems to be responsive to the environment, rather than a straightforward risk for depression. That is, people homozygous for the short allele had greater depressive symptomatology if they had experienced early adversity (conflict, neglect, cold/unnurturing environment) but significantly less depressive symptomatology if the early environment was supportive.<sup>27</sup>

They also looked at the current environment, and showed that this effect is somewhat malleable. That is, people homozygous for the short allele had greater depressive symptomatology if they were currently experiencing adversity but less depressive symptomatology if current environment was relatively stress-free.<sup>27</sup> This might undermine the disadvantage that might be conferred by an early risky environment.

Dr. Taylor has also begun to look at the importance of social stress for moderating these kinds of effects (manuscript in preparation). They looked at cortisol response to the Trier social stress test in relation to allele configuration. Results indicated that people with short alleles show a much sharper cortisol response to the stress task, but there were no differences between the allele groups in the control condition. These findings suggest that stress is needed to potentiate this effect. Such results speak to the question of whether the damage is being done during stressful times.

Recently, Dr. Taylor (in collaboration with Baldwin Way and Naomi Eisenberger) has looked at the opioid system, in particular the  $\mu$ -opioid receptor gene. They are finding that G carriers, who are more prone to social distress than A carriers on this polymorphism, show similar effects. That is, there is an elevated cortisol effect from the G-carriers, but only under conditions of social stress.

Greg Miller (UBC) is currently doing work looking at IL-6 (manuscript in preparation). He found that those who come from riskier families have higher levels of IL-6. Further, people in risky families are only showing this kind of increase in inflammatory response when they have been experiencing stressful events. Again, one sees this moderation of gene effects by stress.

Dr. Taylor went on to highlight the importance of integrating brain imaging techniques. She has been looking at the way in which people from risky families vs. more supportive families regulate their response to stress or threat. The behavioral literature has shown that children from risky families show high levels of avoidant coping, overly aggressive responses to stressors perceived by others to be only moderately challenging, and demonstrate ineffective coping (i.e., coping that does not reduce experienced stress). The question is: is there any neural evidence for these processes?

She has focused on the amygdala and the RVL PFC. Subjects went through three tasks: one where they observed threatening faces, one where they labeled the emotions, and one where they simply indicate gender (control condition). In the observe-only task, offspring from harsh environments showed significantly lower amygdala activity, suggesting they are tuning out the stimuli. But when they had to label the emotion, they showed higher amygdala reactivity. Further, the relationship between amygdala and RVL PFC activity was positively correlated, suggesting that frontal efforts to regulate emotional distress were not serving them well. These findings suggest that growing up in these risky early family environments actually has effects that can be discerned at the neural level.<sup>28</sup>

Dr. Taylor concluded by summarizing her two goals. The first was to raise these multiple, but not mutually exclusive, meta-theoretical models that can provide guidance for understanding the impact of stress and socioemotional functioning on health across the lifespan. Second, she wanted to suggest that these multi-level approaches have already paid off, and that research that integrates psychological and biological processes has the greatest potential to elucidate the underlying mechanisms that will help us to understand how early experiences effect mental and physical health across the lifespan.

*Discussion:*

Steve Manuck noted that each gene seems to have a targeted outcome and specific environmental moderators. But if you look at neural circuitry, genetic variations across the 5-HT spectrum have similar effects. Is there specificity in the association of environmental moderators and particular sources of genetic variation? Dr. Taylor responded that the answer was unknown. Factors like abuse seem to interact with most risky alleles or genes, so that seems like a common pathway. She is currently looking at specific subtypes of risky families to evaluate possible differences in the biological profiles of children who come from families high in conflict vs. neglect vs. cold/unnurturing behavior.

Another participant (name unknown) asked if her findings were specific to social stress or more general? Dr. Taylor responded that she had previously thought it would be general, but in the Trier task they found an effect of having an audience. Moreover, it didn't matter if the audience was supportive or critical. Therefore, it looks like social stress is an important factor.

***Biology of resilience: Oxytocin, positive adaptation and health***  
**Laura Kubzansky, Harvard School of Public Health**

Dr. Kubzansky's work has focused on understanding whether and how emotion or stress influences health or development of disease. There is increasing methodological sophistication in this area. She showed findings from the Interheart Study using data from 52 countries that compared conventional risk factors for acute heart attack (e.g. smoking, obesity, diabetes).<sup>29</sup> Individuals with high levels of psychosocial distress were at over 2.5 times the increased risk for acute MI, after controlling for all other

factors. This is not very different in magnitude from the risk associated with smoking. The attributable risk (incidence of disease in the population that would be eliminated if the risk factor was removed) was substantial at 32.5%.

Much of this work is predicated on the notion that cumulative damage is occurring. We have evidence that dysregulated emotions are toxic but we don't yet fully understand yet the neurobiological mechanisms by which these effects occur. Would we gain more insight by looking at more positive psychological factors? There's limited work in this area. Dr. Kubzansky and colleagues have done some work on this. One example is a study looking at whether emotional vitality protects against heart disease using nationally representative data.<sup>30</sup> Emotional vitality was defined by a sense of positive well-being, interest and engagement in life, and capacity to regulate emotions effectively. Emotional vitality was assessed when study participants were disease free, and participants were then followed for 20 years. After controlling for known cardiovascular risk factors as well as distress and psychological problems, individuals with higher levels of emotional vitality had reduced risk of developing heart disease.

Moving to the larger picture, the research to date suggests that positive factors are more than the absence of negative factors. Further, it's not just about the health behaviors. Findings for positive and negative emotion point to the importance of regulating emotion. Little work has been done on emotion regulation and disease processes in adulthood, but taken together the work thus far points to an interesting new direction - considering a life course perspective. How do people get to a point in midlife where they are chronically distressed, or chronically low in positive affect? There are a few things we know about emotion: 1) it is biologically basic and emerges early in early developing parts of brain; 2) learning to regulate emotion is a major developmental task with consequences for later adaptation/development; 3) patterns of response are shaped by social processes, which implies that you could alter the patterns of response if you could alter the social processes around them; and 4) there is a significant neurobiological component. So the question is: if emotional response patterns start early, are shaped by social processes, and have cumulative health effects over the life course, then what is the underlying neurobiology?

Oxytocin has been recently viewed as one of the underlying systems linking socioemotional processes and health. Dr. Kubzansky and colleagues were interested investigating whether it could inform the research on emotions and disease, and whether perhaps oxytocin is part of the neurobiological groundwork that links early social environment to later emotional processes and disease outcomes.

Animal models have shown that oxytocin inhibits HPA stress responsivity and is a central regulator of attachment and prosocial behaviors. Further, in order for kids to learn to regulate emotions effectively, they have to have a strong social bonding. So can oxytocin help us to understand the relationship between anxiety and stress and positive social relationships? They've been testing the following model: positive social interactions are going to enhance oxytocin, which will then help with effective emotion regulation, which will ultimately down the road lead to health. Oxytocin release is conditioned and conditionable, and therefore they wanted to see if findings of the animal literature translated to humans. Of the limited work in humans so far, most has been done in men only.

Dr. Kubzansky and colleagues are using a placebo-controlled double blind design (research in progress). They manipulate oxytocin and social support (friend vs. no friend), put the subjects under stress, and measure cardiovascular, neuroendocrine, affective responses in men and women. The main questions are: do the effects of oxytocin look similar to social support, do they potentiate the effect of social support, and do they vary across gender or across age? Preliminary findings suggest that oxytocin and social support do dampen stress response. The folks who have oxytocin have more positive affect under stress conditions; the same happens with social support. There were similar (but inverse) effects for negative affect. Change (rise) in systolic blood pressure was also attenuated from baseline to stress tasks under conditions of oxytocin or social support.

They plan to look at many other parameters: stress related cardiovascular phenotypes, autonomic reactivity, heart rate variability, etc. to try to understand in depth what is happening. This idea is to be able to link molecular/cellular information with higher level adaptational processes to better understand

the molecular mechanisms underlying the effects of emotions. How do these get laid down over time? Do they set up trajectories for future outcomes? She then identified the need to translate questions derived from population-based research into experimental studies and back again. This will help to elucidate mechanisms and address concerns of reverse causality or spuriousness. She reiterated the primary questions of the research: when do these things start, are there critical or sensitive periods, what are we going to do about reversibility, or if not reversible, how do we think about targeted interventions? This is highly interdisciplinary work and interdisciplinary teams add a lot of richness to the work.

*Discussion:*

A participant (name unknown) asked how she thinks the damages or benefits will ultimately be stored. Dr. Kubzansky noted that McEwen lays out the idea that biological stress regulatory systems are one way to pick up the damage. The participant then asked if she thought these positive mechanisms also worked via such regulatory systems by keeping the cumulative damage down? Or was there something distinctive about oxytocin or social support? Dr. Kubzansky replied that it might just be stress buffering; oxytocin and social support provide the necessary resources to essentially mitigate the effects of the classic stress cascade. Or it might be that there is something better that comes along. A metaphor is physical activity—you add something new by increasing your muscle mass. That is not just the absence of something bad, but you're actually providing something good. Her hunch is that it's more than just stress buffering, but she notes that we'll need to figure out the specific mechanisms and developing technologies will help us get at that.

***Loneliness: Cause and target***

**Louise Hawley, University of Chicago**

Why study loneliness? Dr. Hawley had recently spoken to a group of practitioners (e.g., psychiatrists, psychologists, social workers, chaplains, hospice workers, counselors) about loneliness and what it meant. They almost immediately grasped the relevance of this for their clientele, and that it hadn't been recognized as such because it doesn't fit into any particular DSM criteria. She then shared several quotes from caregivers and patients reflecting the importance of thinking about the role of loneliness and noted that this is part of the human condition, a universal phenomenon.

Old age is often assumed to be invariably linked with higher loneliness. Cross-sectional and longitudinal evidence indicates that this is not necessarily the case. Loneliness levels are higher in young adults (18-30) than they are in 40-80 year-olds, and not until oldest old age (80+) do loneliness levels surpass levels in young adulthood. Increased loneliness in oldest old age is important to think about in the context of the findings of Dr. Carstensen as it stands in contrast to her findings. Because social relationships are so important for our happiness, what might compensate for the lack of satisfaction with social relationships that still allows adults in oldest old age to maintain relatively high levels of emotional positivity?

In the 50-68 year-old population-based sample in the Chicago Health, Aging, and Social Relations Study (CHASRS), Dr. Hawley and colleagues found a number of unique risk factors for loneliness: marital status was protective (but only if spouse was a confidant), as were number of voluntary associations, physical health symptoms and disability, chronic work and/or social stress, small social network, and poor quality social relationships.<sup>31-33</sup>

What do we mean by loneliness? Loneliness is not about isolation, objectively speaking. Loneliness is a subjective experience of feeling disconnected or left out. People with many friends and social contacts can feel lonely, and people who live rather solitary, isolated lives can feel socially contented. Loneliness is correlated but not synonymous with depression. Depression is more global, loneliness is more specific to the social domain. Factor analyses of self-report depression and loneliness measures show virtually no overlap between loneliness and depressive symptoms on any factor. The only overlap is interpersonal and involves a CESD item that probes about loneliness ("I felt lonely") that co-loads with a loneliness item, "I can find companionship when I want it." Similar results have been shown for the BDI.<sup>34</sup>

Physiological resilience declines with age, we know this. But there are individual differences in the rate of decline, and loneliness is one of those factors that can accelerate the rate of physical decline. Potential mechanisms include poorer health behaviors, greater stress exposure, greater stress perceptions and/or less helpful coping strategies, and less restorative recuperative processes.

One new finding in the CHASRS sample highlights the slowly accumulating cost of loneliness on physiology. Previous studies of young adult undergraduates showed that lonely adults had higher levels of TPR (a determinant of blood pressure) across all experimental laboratory conditions and in the field. If lonely people maintain this pattern of blood pressure regulation, when they reach middle-age, lonely people should start showing this effect in blood pressure (which is not different for lonely vs. not lonely young adults). And that is what they found. In cross-sectional studies, the lonely middle-aged and older group had higher blood pressure than their non-lonely counterparts (Hawkley et al., 2006). That begs the question: is loneliness causal? Looking at longitudinal data using a cross-lag panel approach, they have found that loneliness seems to have a cumulative effect. Loneliness does not predict increases in blood pressure over a one-year period, but its effects accumulate and predict increased blood pressure 2, 3, and 4 years later (manuscript under review)

What do we do about loneliness? Dr. Hawkley and colleagues conducted a meta-analysis (manuscript under review) to examine the effectiveness of loneliness interventions published since 1970. They expected that if you give people a group intervention (provide them with social opportunities in a group setting), they will get less lonely. Surprisingly, the best designed interventions (randomized control studies) showed that there was no effect. Thus, merely putting people with people will not fix the problem. Loneliness is something different than mere access to social opportunities, and that's where we have to focus our efforts.

*Discussion:*

JoNell Strough asked if loneliness might be a marker for something else (e.g., social anxiety), and that is why it doesn't go away when you put a lonely person with others. Dr. Hawkley noted that there is anxiety involved, but for chronic sufferers, the reason loneliness doesn't go away is because it alters social perceptions in ways that set up confirmatory biases and self-defeating behaviors that reinforce the sense of loneliness. In addition, loneliness has a heritable component. It's believed that what's heritable is the degree of sensitivity to the pain of social deprivation. So while some people in a big group won't feel any pain, others in the same context will feel the pain. Construal determines the degree of loneliness experienced, and heritability may influence construal.

David Sbarra noted that a graduate student of his looking at the effect of existential therapy on loneliness. The idea is that it's not social isolation; it's the sense of feeling empty even when one is around people. The goal is to accept that it is part of the human condition. There may be a unique angle for opening people up to the fact that we are all alone.

Jim Coan highlighted the finding that marriage is only positively affects loneliness if the partner is a confidant. Therefore, how do make people friends, or bring people together in a way that is beneficial? Dr. Hawkley responded that understanding what loneliness *is* is the first step in knowing how to intervene. Clearly just putting people with people isn't going to do the job. We've got to change the way the individual thinks about the social environment and relationships, so cognitive behavioral therapy may be well applied here.

***Relationship disruptions and health: From social epidemiology to social psychophysiology***

**Dave Sbarra, University of Arizona**

There is plenty of literature showing that social disruptions are related to broad-based health outcomes. Dr. Sbarra followed up on some of these ideas by focusing on three studies with different *measurement resolutions*. That is, moving from 40-year epidemiological studies where effects accumulate over time to

lab-based study were the effects unfold over a matter of minutes. He suggested that this kind of multilevel science is a great way to elaborate and integrate unique approaches of both paradigms.

In terms of relevance to aging research, he highlighted two points. First, adaptation is a developmental process that unfolds over time. Recovery is a developmental process, and to understand that we need to understand developmental norms in mid-life. Our child development colleagues are very good at this. They think about maladaptation as a consequence of aberrant development (e.g. Sroufe branching tree metaphor). Development is constrained over time by earlier branches on the tree. Second, the accumulation of small effects over time really matters. The ultimate goal is a deeper understanding of time-based mechanisms of action. Related to these ideas, Dr. Sbarra focused his talk on three studies.

Study 1: The social epidemiological literature tends to classify people on their current marital status. But this is just not the way the world works. In mid-life, marital status is quite fluid. Therefore, Dr. Sbarra investigated the effects of divorce in midlife by examining whether changes in marital status alter the risk of early mortality. They used data from the Charleston Heart Study (CHS; N = 1,376), which began in 1960 and had mortality data through 2000.<sup>35</sup> They focused on two groups: always-divorced people (divorced and never remarried), and ever-divorced people (divorced then remarried). Results showed a substantial difference in hazard ratios predicting mortality for the two groups. People in the always-divorced group were 66% times more likely to die than everyone else at each successive measurement period. The ever-divorced group did not have elevated risk hazards. This shows that how we conceptually think about marital separation has a big difference in how we understand its effect.

Study 2: They next looked at whether marital disruptions were associated with clinically meaningful elevations in C-reactive protein (CRP; manuscript under review). CRP is a good indicator of systemic inflammation. Minor elevations in CRP increase risk for many adverse health outcomes, and CRP covaries with psychological stress and depression. They used CRP data from the NSHAP dataset (N = 1,715). Marital disruptions were not associated with elevations in CRP, but there was a protective effect for married men. That is, married men had the lowest levels of CRP relative to women and unmarried men. Interpreted in terms of absolute risk reduction, the net effect of being a married man conferred about the same protective effects as having a normal BMI, being a non-smoker, and being normal-tensive. Note: many people who were smokers were already dead, so data must be interpreted carefully.

Study 3: Among separated adults, how are divorce-related thoughts associated with blood pressure (BP) reactivity? Seventy recently separated adults completed multiple lab visits over a 9-month period.<sup>36</sup> They were evaluated in the lab during a baseline period (thinking about mundane things) and during a time when they are engaged in a divorce-related activation task. During the tasks, they completed appraisal items about emotion regulation and how taxing they found the task. For SBP, they found independent effects for self-reported emotional intrusion-hyperarousal at entry into the study. That is, people who come in with high on this measure come in with systematically higher BP. There was a three-way interaction for regulatory effort during the divorce-specific challenge task: Time X Sex X Regulatory Effort (TRED). That is, men who have difficulty on this task have significant difficulty on the TRED measure.

In sum, there were three exciting findings: (1) the relationship between marital status and mortality, which leads to large questions about social selection or social causation; (2) married men are uniquely protected from clinically meaningful elevations in CRP; and, (3) laboratory-analogue studies can point to potential mechanisms of action. The study of social connectedness and health is ripe with aging-related questions. Dr. Sbarra suggested that the next frontiers included looking at social relationships and cognitive decline (e.g., Franzen), and examining if we can improve health by altering relationships (e.g., Janicki-Devverts)? Everything we know is correlational, so experimental designs are needed.

*Discussion:*

Mara Mather asked if, in addition to always-divorced subjects, he looked at always-single people. Dr. Sbarra confirmed that he had also included this group, and the effect is for always-divorced over always-single or always-widowed. So something about “always being divorced” produces a differential effect.

***The promise of interventions for promoting well-being***  
**Sonja Lyubomirsky, University of California, Riverside**

Dr. Lyubomirsky's research is on happiness, and whether or not it's possible to increase well-being over time, and to sustain it. Happiness is important for optimal functioning, and she is interested in applying this to aging and development over the lifespan.

She began by looking at why are some people are happier than others. The approach was to compare very happy people to unhappier people in terms of cognitive, motivational, judgmental processes and behaviors. There were many interesting findings. For example, happy people are less likely to compare themselves to others. This has led to the important question of whether we increase well-being, and what are the mechanisms by which this can be done? To this end, Dr. Lyubomirsky began doing happiness interventions. She was interested less in what activities or strategies make people happy, but more in the moderators and mediators underlying the effectiveness of various activities in making people happy.

She developed a conceptual model with Sheldon and Schkade.<sup>37</sup> They had a lot of scientific pessimism about whether people could become happier. The behavioral science research indicated that happiness has high heritability coefficient (~50%), so it's clearly partly genetically determined. Then there is a portion that is attributable to life circumstances (~10-20%). Then that leaves "uncharted territory" (~40%), which includes the factors that account for individuals differences in happiness (and undoubtedly includes measurement error). She and her colleagues argue that a lot of what determines happiness is intentional activity. That is, what we can do, the ways that we can think and behavior, where we have control over our happiness (both in positive and negative directions).

They have been testing various positive activities to see how and why they work. For example, in one study, subjects were instructed to count their blessings. The experimental groups did this either once per week or three times per week. It was a six week intervention with students. People got happier when they counted their blessings once/week (the other groups showed decreases in happiness, likely due to increasing demands of the semester). This shows the importance of timing as a moderator. In another study, people went out and did acts of kindness. This was a 10-week intervention, and they did either high variant or low variant acts. People doing a variety of acts of kindness showed increases in happiness, and this effect was maintained one month after the study ended. People doing a low variant of acts of kindness actually became less happy over the course of the study.

In another study, Dr. Lyubomirsky and colleagues found cultural differences (manuscript under review). Subjects practiced either optimism or gratitude. For optimism, they recorded their life dreams for 10 years down the road in a journal, while the gratitude group wrote gratitude letters. A control group wrote down what they did that week. Overall, people who practiced gratitude or optimism got happier over the course of six weeks relative to controls. Subjects were then divided by Anglo-Americans by Asian communities; Anglo-American subjects showed much larger benefits. They found that Asian-Americans were putting less effort into the intervention, so that might be the reason. Interestingly, when looking at changes in their relationships over time, the Asian-Americans benefited more from both optimism and gratitude tasks. So perhaps it's a cultural difference in how happiness is defined and reported.

They additionally conducted an eight week intervention looking at effort. For subjects in the gratitude or optimism group, the amount of effort put forth was highly correlated with the benefit on well-being. In another study, they found three potential mediators for the effect of practicing optimism/gratitude on well-being: people experience more positive events, more sense of relatedness, and more sense of control/autonomy over their lives.

People's emotional lives differ as they age, and Dr. Lyubomirsky is interested in the implications of this for her work. She suggested that perhaps older people are already doing some of the things that help to increase happiness (e.g., more appreciative, more optimistic or positive).

*Discussion:*

Alex Zautra asked Dr. Lyubomirsky to comment on things that allow people to sustain happiness. Dr. Lyubomirsky noted that she thinks of this as upward spirals. These activities lead people to experience positive emotions, which might affect their self-concept. Acts of kindness makes people feel like a better person. Seeing them work gives people hope that they can become happier. For example, if you do an act of kindness, it makes you feel happy, which might make you feel creative, which might make you connect with your partner, which might make other people approach you, which might improve your relationships. She and her colleagues are working on ways to capture this process.

***Psychosocial influences on longevity biomarkers***

**Elissa Epel, University of California, San Francisco**

Dr. Epel started with a quote from one of the founders of stress research, Hans Selye: *“Every stress leaves an indelible scar, and the organism pays for its survival after a stressful situation by becoming a little older.”* So where are these scars? Where is the memory in the body of stresses? And how can we measure biological aging? As psychologists interested in adaptation, it would be very helpful to have a criteria or an easy mark of biological age so that we could measure adaptive forms of coping.

Biomarkers are a huge deal. Almost every parameter of biological regulation changes with age. So the job of looking at meaningful biomarkers is not easy. Dr. Epel focused on a biomarker of cell aging that involves the ability to replenish tissue and keep dividing as we age. This marker is not only a marker of age but a part of the aging process. Telomeres are the DNA that caps the ends of chromosomes, protects them from fusions, mutations.<sup>38</sup> As people age, mitotic cells divide to replenish tissue, and the telomere shortens. When it's too short, the cell has aged. It goes into arrest or senescence (terminal differentiation stage) where it can no longer do its job. Telomerase is an enzyme that prevents telomere shortening, and promotes cell resilience. One of its main jobs is to protect and actually lengthen telomeres. Telomere length appears to be an interesting ‘psychobiomarker,’ meaning that it's a marker that is influenced by the social, psychological environment, moves with aging, and is predictive of mortality. It looks like an ideal one because it is to social status, perceived stress, objective exposure to chronic stress, depression, and it's predictive of mortality.<sup>39</sup>

The dogma in the field was that telomeres shorten over time, unidirectionally. This is well supported, but it is based entirely on cross sectional studies. Recently, Dr. Epel and colleagues have begun longitudinal studies. In the first, they looked at the MacArthur Healthy Aging sample over two years and found that about 30% of people showed shortened telomere length during this time. But interestingly, about 25% of people lengthened.<sup>40</sup> They replicated this finding several times. So is this phenomenon related to malleable factors in our lives? They found that people over one year who decrease in stress show the lengthening, and people who increase in stress show the shortening. It looks like it's malleable.

She then posed the question: What psychological responses to stress lead to the greatest wear and tear, stress arousal, and earlier mortality? In prior studies, people were exposed to lab stressors from many angles: reappraisal, coping, reactivity. In some new data, it appears that rumination (trait brooding) appears to have an effect (unpublished manuscript). Specifically, high ruminators show elevated arousal to lab tasks and shortening of telomere length one year later, whereas low ruminators show telomere lengthening. Dr. Epel is thinking through which psychological processes she should focus on next.

In sum, means of telomere length can be deceiving. Longitudinal studies are an important way to go. Telomeres are dynamic and lengthening is common, and the telomere/telomerase system is much more complex than we thought (telomerase). More population studies with nested intensive substudies are needed. What this mechanism means for the immune system is not known, so smaller, intensive studies are needed to actually look at this relationship.

*Discussion:*

Lani Shiota asked if Dr. Epel whether she thought the critical part of the stress was subjective stress vs. objective stress. Further, Dr. Epel used the terms “increasing stress” or decreasing stress”—did it appear

to be the level of stress overall that was important or changes in stress? Dr. Epel responded that in general, it was regulation, what people do in response to stress. So perceived stress is a really helpful measure—it appears to pick up threat stress (e.g., feeling out of control) which seems to stimulate the adrenal pretty well. We could also tie this to objective changes in stress.

Steve Manuck asked a technical question about how Dr. Epel sorts leukocytes. Dr. Epel noted that they always do flow cytometry to look descriptively at whether there are differences in numbers of cell types. Is it that stress is causing more memory cells than naïve cells and aging the immune system? They thought they'd see those shifts, but they haven't yet in the two ongoing caregiver studies. Dr. Epel and colleagues are now sorting cells and looking at the type of cells that increase with aging, and that we think stress is causing an increase in as well, so we can see where the damage is. Dr. Manuck then asked whether telomere length covaries across tissues. Dr. Epel responded that it does, weakly. She believes it is immune senescence that is responsible for morbidity and mortality, but they are looking at follicular hair cells and buccal cells to see if they work as nonobtrusive markers. She thinks, however, that the action is in the immune system.

Suzanne Segerstrom noted that telomerase is one of the major mechanisms of cancer. She asked Dr. Epel if she sees any risk in having telomere lengthening. Dr. Epel noted that once there is a cancer cell, telomerase is up-regulated to huge, unnatural levels. We rule out any of those cells in our analyses. But the telomerase field is trying to come up with a drug to increase telomerase, but they never will because of the risk of cancer. That's why Dr. Epel and her group are focused on psychological and behavioral intervention for modulating cell aging.

What about interventions? Dr. Epel brought up a slide from an ongoing collaborative project call the Shamatha Project. Subjects either participated in a three-month, intensive meditation retreat or were in the control group. They found that, post-intervention, the telomerase in meditation group was 30% greater than in the control group. Well-being also went up in the meditation group and was correlated with telomerase. This shows correlations between psychological measures of well-being and telomerase, and it was the meditation that was causing the high end of both. They also looked at a whole panel of biomarkers (e.g., cortisol, BDNF, inflammation) and nothing came out. Telomerase appears to be sensitive to psychological factors and is a great candidate for use in laboratory studies.

## ***Session 2: Open discussion***

**Moderators: Lisbeth Nielsen and Robert Levenson**

Dr. Nielsen presented questions for the discussion sessions: 1) How can this research help to advance the study of adaptive and healthy aging, specifically to improve the adaptive functioning of individuals in their daily environment and identify the causal mechanisms than contribute to their resilience?, 2) What are the most immediately viable transition opportunities across domains represented here: studies of social and emotional function, psychological and biological processes in health, and decision making and behavior change?, and 3) What are the next critical challenges your field needs to tackle?

Sonja Lyubomirsky highlighted the possible utility of biomarkers in intervention studies. She cited Davidson study on meditation and frontal-cortical activation as a good example of this kind of work. Self-report is easy so it's often used, but it's important to combine that data with biological measures.

Dr. Nielsen pointed out that while many people will eventually succumb to a disease, there are other outcomes of interest like happiness and well-being. A different set of questions, then, is what are the biomarkers and the mechanisms, and what are the outcomes we want to see from these interventions? For example, in an intervention on loneliness, how would you know if it was successful? Brian Knutson noted that it seemed the three major themes over these sessions are happiness, health, and wealth. Each is worth considering as an outcome.

A spirited discussion about biomarkers ensued. Suzanne Segerstrom highlighted the need to be careful in the way they are understood and interpreted. For example, cortisol is "the great Rorschach blot of

psychophysiology,” and data may be interpreted by different researchers to mean different things. We are tempted to look at biomarkers and infer some kind of psychological state. For most biomarkers, there are so many potential antecedents that you can’t reverse engineer psychological state that way. A promising direction for this research is to get more epidemiological data about these variables. That would allow researchers to identify risk factors to target at the population level. For example, there is no such data for cortisol reactivity. David Sbarra raised the related issue of calibrated, non-arbitrary outcomes. Calibration is a very important index. On the health outcome side, it’s the difference between practical versus statistical significance. We see differences in statistical models all the time that might not make any practical difference, cortisol being an example of this.

Bob Levenson asked the panel whether they felt allostatic load suffered from that issue. Steve Manuck replied that it suffers a good deal from “circularity.” Also, diseases have very specific pathophysiologies, and there is still little evidence that psychosocially modulated variation in cortisol (as a general mechanism) is responsible for prominent disorders outside the central nervous system. He gave the example of animal models showing sex differences in the relationship between coronary disease, social behaviors, and neuroendocrine factors. It makes more sense to default to the pathophysiology of the disease we’re interested in, rather than think we have some marker that has pan-disease implications. Dr. Nielsen wondered, if cortisol is linked to changes in brain systems for stress reactivity, is it something worth looking at as a biomarker if your focus is on psychological factors like adaptive coping and well-being? Dr. Manuck seemed skeptical; he used memory as an example and pointed out that there were many other factors exerting an effect. Dr. Segerstrom asked about the effects of chronically elevated levels of cortisol. Both Dr. Epel and Dr. Manuck agreed that there really isn’t evidence for the long-term implications of elevated cortisol—animal models and/or epidemiological studies are needed. Dr. Epel noted it is difficult to measure it reliably. If you want to look at what matters for metabolic health over time, insulin resistance is a better target.

Shelley Taylor noted that elevated cortisol by itself is clearly not the mechanism that causes disease. What typically happens is that earlier on, the system would lose its resiliency and you would start to see the flat elevations. One would not necessarily see changes in cumulative cortisol. But if the HPA axis is losing its resiliency, there is nothing good about that. Thus, high cortisol is only meaningful when looking at younger subjects. When looking at older groups, you need to look at different patterns of cortisol for meaningful predictors. She suggested that we have to recognize the inherent qualities of the HPA axis that limit volume of cortisol as something that is clinically meaningful. Dr. Rosen agreed that medically, they haven’t found a use for cortisol. There is so much unexplained variance that it’s not clinically useful. The flip side of the coin is that the bar has to be set very high for clinicians to use a biomarker. Certain biomarkers are very predictive of outcome, but we don’t know how to intervene yet. Until the science advances, clinicians will be reticent to use them until they know what to say as the next step.

Laura Kubzansky suggested that this we have to separate out what we are using the biomarker for. Different biomarkers may be indicative of different pathologies or outcomes. Importantly, we tend to think linearly about these markers, but with biology dysregulation can often happen in both directions (e.g., as with telomeres). One of the features of aging is that physiological states get less and less flexible. She’s thought about psychological adaptation as being more problematic when there’s less variability. This limits the ability to recruit resources, problem solve, etc. The reduction in flexibility that goes along with the biological systems may be a feature of poor psychological adaptation. But all of this requires us to figure out what “normal” is for a lot of these systems. A next major frontier is to think more complexly about variability and flexibility in the various systems.

Returning to the HPA axis, Margaret Kemeny reiterated that we don’t know, when that system is perturbed, if it leads to disease. Rather, it’s a way of capturing perturbation. Importantly, however, we do know that the inflammatory system is important. The dysregulation of inflammation can lead to mortality and all sorts of diseases. Glucocorticoids play an important role in constraining inflammation. Thus, we can look at glucocorticoid regulation of inflammation, as well as at the expression of those receptors on the cells that produce the inflammatory molecules. This is an important outcome that can be measured. In general, HPA activity is important biologically, but we need to look at it in a more sophisticated way. Dr. Kemeny further suggested that we need the same kind of specificity and sophistication at the

psychological level that we're talking about at the biological level. We tend to throw self-report measures at people, and we may have all this overlap because there are individual difference factors that influence how we fill out questionnaires. She noted that she was happy to hear early talks using emotion regulation tasks. These kinds of tasks might be well-applied to the studies of stress its effects on the immune system because they are really capturing people's emotional reactivity in different contexts. It will be important to bridge the two areas.

Louise Hawkley made a plug for society: there is a role for society in permitting the kinds of changes that we want to see. Loneliness is contagious, but lonely people are pushed to the periphery of society. This may be adaptive—others don't want to "catch" what they've got. But how are we going to allow for those people to connect if we don't offer some way of pulling them in. She linked that to Sonja Lyubomirsky's work—individual-level interventions may be useful, but it's nice to also think about it in terms of getting out of your head and benefitting someone else through volunteering (e.g., programs like ExperienceCorp). This is mutually beneficial and enables a safe entry for connections with other people.

### Session 3: Decision Making in Aging

#### ***Decision making in aging: Emerging insights from affective neuroscience and neuroeconomics***

**Brian Knutson, Stanford University**

Dr. Knutson began with a quote from an editorial in *Nature Neuroscience*<sup>41</sup>: “Cognitive science is not yet close to explaining or predicting human decision-making in the real world...” If nothing else, Dr. Knutson wanted to convince the group that something has happened since that time. He began with simple definitions of the fields. First, affective neuroscience: how does the brain value things? New tools can help us gain insight into this question. Second, neuro-economics: how does the brain decide/choose to do something? If you put these things together, value plus decision, you should be able to predict choice. And if we’re talking about aging, we should be able to predict choice as it changes over the lifespan.

We know that as people age, cognitive decline occurs in fluid intelligence and executive functioning. Affective neuroscience offers a way to look at emotion as it happens in the brain dynamically, under choice scenarios. This allows researchers to think not just about diminutions in executive function, but about how emotions operate in interaction with executive function. There is some evidence suggesting that while there are some changes that occur in emotion over the lifespan,<sup>3</sup> there’s also preservation. Just as with preservation of crystallized knowledge, with affect or emotion there might be an interesting asymmetry in the preservation of certain positive vs. negative emotional capacities.

Past research has shown that you can put an electrode in the brain of an animal, and allow it to stimulate the electrode, and the animal will work very hard to do that at the exclusion of other attractive activities. Many of the areas that support this are subcortical. There are other circuits that support aversive behavior—the rat will work very hard to turn off the stimulation. This allows us some points of departure. If we want to look in the brains of humans while feeling and making decisions, where do we need to look? We probably want to look at the cortex and the subcortex, and we need a method that will get us there. We also need to look fast in time because we know that these circuits are modulated by dopamine and other chemicals, and this modulation occurs not just in response to rewards but in anticipation of rewards and punishments. So we need a method with decent spatial resolution and temporal resolution of these circuits. Lucky for us, along came fMRI that allows us to see what’s changing subcortically. And it gives second to second resolution, which is the time scale upon which many of us make decisions and consider what’s going on in the world. So in theory, this temporal resolution should get us not only into the realm of making a choice or responding to an outcome, but also to anticipating what you are going to do.

To summarize some previous work: the emotions that you feel before or during the course of decision-making may influence what you do. This is distinct from reactions you have to something that has already happened. If that’s true, then we need to be able to capture what’s happening in the brain at those time points. And if that’s true, it may be that cues activate these areas, and this activation may be correlated to some degree with self-report. And that should have the capacity to influence behavior, especially if we have measures for valence or values. So that means if we have an index of neural activity that anticipates gains vs. loss, we should be able to make a directional prediction about whether that animal will approach or avoid the outcome that it’s considering.<sup>42</sup>

Dr. Knutson and colleagues developed a task based on animal models to elicit anticipation of gains and losses. They aren’t trying to alter choice or behavioral responses, they were simply trying to manipulate what people expect will happen. They show people cues that indicate gain, then subjects see a rapidly presented target and have to present a target before it disappears. Depending on whether they were successful, subjects can either gain money or avoid losing money. If they are too slow, they either don’t gain money or lose money. The researchers were interested in anticipatory affective activation during the interval before the person has received an outcome. Over 20 studies have been done with this paradigm. The collective results indicate that you tend to see anticipating gain rather than losses of the same magnitude produces greater activation in subcortical regions (e.g. nucleus accumbens and striatum). You also see some interesting things that seem to more associated with losses (parts of anterior insula).<sup>42</sup>

They also looked at anticipation to gains and losses, controlling for performance, of people at different ages.<sup>14</sup> They further asked people how they felt when they saw the cues. Not surprisingly, people reported more positive arousal (e.g. excitement, energy) in response to seeing the gain cue. Young and old people self-report the same level of positive arousal. There are different patterns of self-report for loss cues. Young people report more negative arousal, but there is a blunting of this response for older people. What about in the brain? When they contrasted activation in the anterior insula (which we think is associated with anticipation of losing money), they saw a symmetrical pattern in this region for younger people, but not for older people. Older people had a flatter profile.

What does this mean? First, Dr. Knutson and colleagues thought that maybe older people were just feeling less arousal. But if thinking about this from the perspective of SST, it suggests that older adults may be more responsive to positive stimuli than to negative stimuli. This may be happening not only in response to outcomes but also during anticipation. If anticipatory affect drives learning, drives choice, and so forth, then one should see biases in older adults in how well they learn about losses and how they take risks and approach and avoid potential losses. To test this, they conducted another study where subjects learned to either approach a cue that indicates a gain or avoid a cue that indicates a loss.<sup>43</sup> This was a large community sample. For gain learning, there was no relationship with age. For loss learning, on the other hand, there was a negative association with age. Translating these outcomes to life outside the lab, there was a strong association between performance on this task and their level of debt.

With these new tools, researchers can start to deconstruct choice, such as gain anticipation, loss aversion, and how to integrate those things in the brain. Once these metrics are developed, they can be extended to subjects across the life span. Studies suggest that older subjects show reduced loss anticipation (self-report and brain) and they seem to learn about losses more slowly. Further, other data that suggests they may also have integrative issues across losses and gains.

The implications are that we can try to reconstruct choice. Researchers can specify what an optimal choice path is and try to account for behavior that matches that path and goes beyond that path. We can reintegrate emotion and cognition into decision making. We can strive for prediction, not just correlation. We can try to predict choice in the lab and in the real world. And what if we see biases in older people, or in anybody? If we understand these mechanisms, can we engineer decision prostheses for these people? We want to be sensitive to people and their goals, but once we know that, if they are deviating, could we engineer these more optimal techniques?

*Discussion:*

Jocelyn Sze asked how specific Dr. Knutson thought his findings were to material loss (vs. relational or social loss). Dr. Knutson acknowledged that he doesn't know the answer, but he does know there is overlap in the circuits between primary and secondary rewards. Others have done research showing that the circuits involved in cooperation for rewards are related. It's not clear how these things scale against one another, but they involve the same machinery.

A participant (name unknown) voiced surprise how many people showed no gain learning, and wondered what the implications of this might be. Dr. Knutson suggested that these subjects may have just chosen a cue and stuck with it—perhaps they were not as invested in gain. Or perhaps some subjects were satisfied with the low probability gain and never switched to the high probability gain cue. Those with a perfect score got lucky—the randomly selected the high-probability gain cue first, then stuck with it.

Heather Urry asked how Dr. Knutson thought his findings squared with the positivity effect. He replied that the data demonstrated a lack of response to negative things, vs. a hyper-response to positive things. Mara Mather noted that the research seemed to reflect an age by valence interaction. Looking at just older adults, you can't say you saw a positivity effect, because it may be that your stimulus was more memorable to them for other reasons. Dr. Knutson suggested that was important to consider the specific effects of positivity vs. negativity. The only way to do that is to split out things accordingly and have neutral options. The fact that there may be different learning systems for losses and gains is deeply important for decision-making and would influence if and how we intervene.

***Neural basis of decision making in aging***  
**Natalie Denburg, University of Iowa**

Dr. Denburg began with an overarching hypothesis: there are some seemingly “normal” older adults (i.e. without overt neurological or psychiatric disease) who have or who manifest reasoning and decision making deficits. She posited that this is due to dysfunction in a neural system that includes the VMPFC.

She has approached her research questions using the Iowa Gambling Task, which has been shown to be a sensitive and specific measure of damage in the region of the VMPFC. Patients who have sustained damage to this region end up having social deficits in their everyday lives (e.g. modern day Phineas Gages).<sup>44</sup> She and her colleagues studied these individuals, and the only deficits we could find in these individuals were in their performance on the Iowa Gambling Task.

Therefore, she and her colleagues brought younger and older adults into the lab, and had them perform the Iowa Gambling Task.<sup>45</sup> Looking at the number of cards chosen across three groups (younger, older unimpaired, and older impaired), 93% of younger adults play this task “advantageously,” that is, above chance using a binomial theorem. One third of the older adults performed this task “disadvantageously” (i.e., impaired performance). They chose more from the bad decks than the good decks. Then the older unimpaired adults also perform this task above chance (similar to younger adults).

They also found differences between the impaired and unimpaired individuals in their autonomic responses. In anticipation, before people make a decision, they have bodily responses that shape behaviors, hopefully in advantageous ways. Patients with damage to the VMPFC don’t have those types of advantageous bodily responses. And this is essentially Damasio’s somatic marker hypothesis. What’s interesting here is that unimpaired older adults can anticipatorily discriminate between the good and bad decks. That is, prior to selecting cards, they have differential skin conductance responses. By contrast, older impaired don’t show that discrimination. What’s also interesting is that, for older unimpaired subjects, the response is larger when selecting the advantageous decks. This isn’t supposed to happen. Younger adults typically show the greatest anticipatory response to the bad decks, and a relatively lower anticipation to the good decks. Older adults are more reactive to the good decks than to the bad decks. Perhaps there is a positivity effect going on here. But what’s most important is the difference between the unimpaired and impaired older adults, which may lead them to perform poorly behaviorally.

Dr. Denburg and colleagues wanted to link these lab findings up to the real world. They looked at “consumer decision making.” They took advertisements that the Federal Trade Commission deemed deceptive and created non-deceptive counterparts and administered these to younger and older adults. They then assessed their comprehension of the advertisement’s claims and their purchasing intentions. For the full-disclosure add, everyone (younger, older unimpaired, and older impaired adults) performed comparably. But for the limited disclosure add, for the impaired older adults, comprehension of claims drop significantly, and purchase intentions rise significantly.

They have followed these adults and have now brought them into the lab to explore the neuroanatomical basis for these differences. There are several different possibilities. It may be related to physiological dysfunction in the VMPFC. It may be that these impaired adults are in a sub-syndromal stage of Alzheimer’s disease. It may be the frontal aging hypothesis (e.g. West), which is a theory about dorsolateral aging. They just finished collecting the imaging using structural neuroimaging and FDG-PET scans. A battery of cognitive tasks showed that they are fully intact. They found a lot of differences in the medial prefrontal cortex (DMPFC). There were also significant findings in the right insula and in the caudate. This is in line with previous findings showing that right insula is associated with somatic markers, and that lower caudate volume is associated with poorer prediction ability. In the sMRI findings where they looked at cortical thickness, there were findings in the anterior cingulate.

In summary, impairment in older adult’s decision-making shows involvement of neural circuitry that has been implicated in advantageous decision making (i.e., VMPFC, DMPFC, right insula). There was no evidence for dorsolateral, parietal and/or medial temporal alterations, as in AD. Finally, in the future, Dr.

Denburg will be looking at genetic data from these subjects (e.g., APOE-e4) and is conducting a follow-up fMRI study.

*Discussion:*

A participant (name unknown) asked if Dr. Denburg thought something happened in aging to cause the structural difference, or was it that the difference had always been there but materialized with aging. Dr. Denburg thought that this is an age-related phenomenon, not a premorbid developmental issue. She believes that these people have accrued this deficit over time, so it's a dysfunction of normal aging.

Brian Knutson asked whether Dr. Denburg was using DTI. She responded that they were using this methodology, but data is still being analyzed. Dr. Knutson noted that the good decision-makers were essentially showing response to reward, and so perhaps the people who have problems in certain areas of the brain aren't anticipating reward very well, which fits with the research literature.

***Age and sex differences in the effects of stress on decision making***

**Mara Mather, University of Southern California**

Dr. Mather got interested in the question of what stress is doing to decision making by looking at different literatures across the two disciplines. In the stress literature, there are studies looking at stress effects in humans during fMRI tasks, allowing a look at what regions are most affected. This research matches up with the animal literature (and the findings from previous speakers), which implicates the VMPFC, cingulate, MPF, and the insula. This seems to show striking overlap with decision-making circuitry.

The decision-making literature (e.g. Lowenstein, Damasio) shows that emotion plays a major role in decision-making, and the somatic marker hypothesis posits that bodily state influences decisions. These things point to the idea that stress should influence the way we make decisions. If you think about everyday life, when you make decisions (especially the important decisions), it is often in stressful situations. The decisions themselves may elicit stress, and sometimes you are just in a stressful situation needing to make an important decision. So it seems that stress is going to have important effects on decision-making. Yet there is very little research on the effect of stress on how people make decisions.

Dr. Mather and colleagues were interested in both age differences and sex differences in this area. With age differences, if there are differences in how brain regions associated with both stress and decision-making are faring as people age, there might be differences in how stress influences decision making across the life span. In addition, there is a large literature (e.g. Taylor and others) pointing out how stress affects behavior differently for males and females.

Dr. Mather outlined three studies. She noted that there are many ways you can think about stress. In her studies, they decided to use the cold pressor task (physical stressor where people hold hand in ice water for three minutes). In the control condition, they put their hand in warm water. The ice water condition reliably elicits cortisol elevations about 20 minutes after the stress induction, whereas the control group does not show this cortisol increase. They then timed the decision-making task to occur during that time of elevated cortisol.

Study 1: Subjects played a video driving game.<sup>46</sup> They got points for how far they drove a car while a light was yellow. But at any random point between 2-7 seconds, the light would turn red. If they were still "driving," a siren would sound and they lost all their points. If they had stopped, they heard "yippee" and got to keep their points. In the younger adult group, there was no effect of stress on how much time they spent driving during the yellow light. In the older adult group, those in the control condition drove almost as much as the younger group. But for stressed older adults, there was a dramatic reduction in how much time they spent driving during the yellow lights. Further, older adults in the control condition stopped and restarted less frequently than younger subjects, but stressed older adults stopped and restarted more frequently. It seems like stressed older adults might be indecisive, without a very organized decision-making strategy. With regard to gender, they had equal numbers of male and female

subjects. For younger adults, stress has opposite effects on male and female decision strategies. Males stop and start less frequently if they have been stressed, whereas females stop and start more frequently.

Study 2: This study examined risk-taking tendencies using the balloon analog task. Here, a balloon on a computer screen inflated one button press at a time, and subjects received points for how big it got. Every time it inflated, however, there was a risk that it may pop. Subjects could either choose to cash out or keep inflating. After a stress induction, they found that males increased risk taking and the females decreased risk taking in terms of how big they blew up their balloons.

Study 3: The previous studies suggest that males tend to get more risky under stress, whereas females get more conservative. To investigate what the associated brain mechanisms might be, they adapted the study for the fMRI scanner. And they again found that in the control condition, males and females look pretty similar, but in the stress condition, the males go through balloons than do the females. What was going on in terms of brain activation during the task? One main area showed a stress x sex interaction: the putamen, which is part of the striatum. Males in the stress condition showed greater activation in that region than in the control condition, whereas women in the stress condition showed less activation.

They were interested in whether this was part of the whole decision network, and whether it's interacting differently with other regions for males and females. Therefore, they conducted an independent components analysis of brain activity during the fMRI task. They found that similar brain networks were affected during the decision task across the four groups (male control, males stress, female control, female stress): insula, cingulate, MPFC lateral, inferior parietal, VMPFC, and hippocampus. But in the putamen, there is a stress x sex interaction where both the control females and stress males show the putamen as part of their decision network, but the control males and the stress females do not. Finally, the stressed males seemed to have other regions involved in their network, thalamus, precuneus, and caudate, that were not involved for anyone else (manuscript in preparation).

In conclusion, their findings showed that acute stress affects decision making, but differently depending upon both age and sex. In Study 1, stress reduced older adults' risk taking and increased stops/starts in the driving game. In all three studies, stress increased sex differences in decision making strategies among younger adults. Dr. Mather noted a caveat: she believed that they could come up with tasks in which females would perform better under stress than males. Finally, they found that stress was associated with increased activation in the putamen for males but decreased activation for females.

*No discussion due to time constraints*

### ***No time to waste: Understanding why older adults are less subject to the sunk-cost fallacy***

**JoNell Strough, West Virginia University**

The sunk-cost fallacy is an irrational decision-making bias that occurs when a person makes the decision to invest more as a function of having made a prior investment (as compared to when an investment has not been made). It is a fallacy because the normatively correct decision is to invest the same amount because the cost that has been invested is "sunk" and cannot be recovered.<sup>47</sup>

To look at this effect, Dr. Strough and colleagues gave people hypothetical situations adapted from prior research (e.g., Frisch). For example: "You are staying in a hotel room on vacation. You paid \$10.95 to see a movie on pay TV. After five minutes, you are bored and the movie seems pretty bad. How much longer would you watch?" They also gave people an analogous scenario with the same information, except the information about the payment of \$10.95 was removed. Subjects were then asked how long they would watch, and the two times were compared. If subjects said they would invest longer in the situation where money was paid, they committed the sunk-cost fallacy.<sup>48</sup>

Initial research by Dr. Strough and her colleagues showed that older adults were less likely to commit the fallacy, and they were more likely to make the normatively correct response.<sup>49</sup> Why? Judgment and

decision-making researchers point to loss aversion as the mechanism that underlies the sunk-cost fallacy. Specifically, people are motivated to avoid loss. In a misguided attempt to avoid loss and recover the investment, they invest more. When considering how concern about loss aversion might vary by age, Dr. Strough was familiar with the SST and positivity bias,<sup>50</sup> as well as research showing that older adults have a more balanced view of gains/losses.<sup>51</sup> The age-related positivity bias is in contrast to the finding that younger adults have a negativity bias in information processing.<sup>52</sup> Thus, in her initial research, Dr. Strough hypothesized that older adults would do better on their task, and that is what she found.

In terms of SST, the positivity bias is thought to reflect age-related differences in future time perspective. In later life, as future time perspective becomes more limited, people prioritize emotionally meaningful goals. Thus, there is age-related maintenance or improvement in emotion regulation. If the positivity effect contributes to adult age differences in the sunk-cost fallacy, and the positivity effect is a consequence of age-related differences in future time perspective, then the sunk-cost fallacy should be less likely to occur when future time perspective is limited. Thus, to follow up their initial research, Dr. Strough and her colleagues examined the relation between sunk-cost fallacy and future time perspective.

In the follow-up study, Dr. Strough hypothesized that older adults would be less likely to commit the sunk-cost fallacy and have a less expansive future time perspective; this less expansive future time perspective would be associated with a lesser tendency to commit sunk-cost fallacy. Replicating their initial research, Dr. Strough and colleagues found older adults were less likely to commit the sunk-cost fallacy. Older adults also had a less expansive future time perspective (as expected based on SST). Further and most importantly, the correlation between those two factors suggested that the expansive future time perspective was associated with committing the sunk-cost fallacy.<sup>53</sup>

Dr. Strough suggested that the relationship between sunk-cost fallacy and time perspective reflects that a limited future time perspective induces the positivity effect. She plans to conduct experimental studies which manipulate future time perspective. Dr. Strough and her colleagues are also looking at other irrational decision-making biases that have negative affect as the hypothesized mechanism, like counterfactual reasoning (if-only bias), which is thought to involve regret. Results show the same pattern as the sunk-cost fallacy: older adults are less likely to commit that bias. In another study, Dr. Strough is looking at people's goals when making decisions about sunk costs. Subjects were asked about their decision-making goals. Younger adults seem to have more investment concerns and say things like, "I want to get my money's worth," or, "If I paid for the movie, I am going to see it through." Younger adults seem to be attending more to the amount of the investment, at least when you ask them about their goals. Older adults seem to be mentioning things like as personal characteristics, such as, "This is just the way I am."

#### *Discussion:*

In Dr. Strough's scenario, they lose money to save time. George Loewenstein asked if perhaps the opposite result would occur if time was invested in exchange for money—older people wouldn't want to lose that time, and younger people would show a greater sunk-cost effect. Dr. Strough had used such vignettes and found the same pattern of results in terms of age differences. Her findings were collapsed across four vignettes (two dealing with a time investment, two dealing with a money investment). Laura Carstensen pointed out that it's future time that's scarce, not time invested. Dr. Strough noted there is an investment of time in the movie scenario (i.e., you invested time watching it), though it is important to consider domain specificity.

Louise Phillips asked if it was particular types of situations that Dr. Strough was asking about counterfactual thinking, or were they related to everyday life? Dr. Strough replied that they had adapted classic tasks in decision-making research. They were situations where the person thought about not doing a course of action, but then they did it anyway. Thus, the situations are easy to mentally reverse, and that reversal is what the regret is. Dr. Phillips then asked if there was any evidence for the use of counterfactual thinking in everyday life. Dr. Strough was not sure and noted that counterfactual thinking is an interesting strategy.

Fredda Blanchard Fields asked for clarification as to whether older adults used less counter-factual thinking across all scenarios and domains. The participant had also used similar tasks and had found a

lot of variability in whether they engaged in counterfactual thinking, and personal relevance was an important factor. Older subjects showed a qualitatively different set of criteria for making the decisions than the abstract criteria of counterfactual thinking. Dr. Strough noted that she was very interested in the domain specificity of counterfactual thinking and could reanalyze the data to investigate this issue.

***Wanting and liking for sex by gender and age***  
**George Loewenstein, Carnegie Mellon University**

Sex is important from a physical and emotional health perspective. Sexual frequency is correlated with longevity.<sup>54</sup> It's associated with decreased risk of heart attack, stroke, and prostate cancer.<sup>55</sup> It's the activity that produced highest happiness among a sample of 1,000 women.<sup>56</sup> Those with no sex partners in the past year had highest levels of unhappiness in a national survey of Americans.<sup>57</sup> And increasing sexual activity from once/month to once/week is equivalent in "happiness" to a pay raise of \$50,000.<sup>58</sup> Dr. Loewenstein focused on the relationship between the wanting and liking of sex. From a decision-making perspective, these might seem equivalent—the more you like something, the more you want it. But in fact, there is a lot of research that shows that there are deviations between wanting and liking.<sup>59</sup> Most of the research has focused on people wanting something too much—overwanting, for example, drugs, and then not enjoying the drugs so much. You also have examples of the opposite—people liking sex and not wanting it. To begin their investigation, he and a colleague developed a scale for wanting and liking for sex with an intimate partner—they only looked at people in long term relationships. They also developed a scale general sex drive as well.

One study they conducted included 2,593 U.S. adults involved in intimate relationships. The age range was 18-88 (mean = 47.9 years) and the majority were married (79.4%), with the rest cohabitating or dating. They first looked at frequency of sex by duration of relationship. If the couple has been together less than one year, they had sex an average of more than once per week. By the time they had been together 6-10 years, it dropped off fairly dramatically. In the beginning of the relationship, males and females initiate sex equally. But over time, both males and females agree that males tend to initiate sex more. Males have a more extreme impression of this—the longer the relationship, the more males think they are the ones to initiate sex. Who controls whether they do or do not have sex? In the beginning, it's about equal, but over time both agree that the female controls when they have sex.

Moving to wanting and liking, they found different patterns by age. Wanting dropped as a function of age; this occurred more dramatically for females than for males. Liking also dropped as a function of age; this occurred for males and females at relatively comparable rates. If you compare these two, the big difference between males and females is the wanting of sex, not in how much they actually like sex if they have it. Looking at these two variables by duration of relationship, the female drop-off in wanting is much more dramatic as a function of relationship duration than it is for the males. The patterns of liking are more similar, though females begin higher and end lower. If we look at the relationship between wanting and liking, it's very unusual to want sex if you don't like it, but it's very common to like sex but not want it.

To further assess the relationship between wanting and liking, they normalized the variables across the entire sample and looked at the differences between them. Findings indicate that as people get older, the ratio between wanting and liking initially increases (people want it more relative to liking it), but then it drops off for males (~age 26-35). For females, it appears to drop off steadily from the beginning. Looking at this as a function of relationship duration, the pattern is similar. Regression analyses suggested that marital duration seems to be more important than age, but it was complicated.

They have also evaluated the relationship between mental health, physical health, subjective happiness and satisfaction with life as a function of demographics and sex variables (frequency and liking). Sexual liking is highly associated with mental health, subjective happiness, and satisfaction with life. Sexual frequency is highly associated with physical health, as well as with subjective happiness and satisfaction with life. The causality in these relationships is unknown. Thus, they are currently running first experimental study to examine the causal impact of sexual frequency on health and happiness in married couples. Couples are instructed to either have normal amounts of sex or to double their usual amount.

Generally, there seems to be a lot of benefits to sex. But unfortunately, even though males and females seem to retain their liking for sex, the wanting seems to drop off pretty seriously. This raises the question of how can you improve health and improve quality of life, and how is it possible to maintain wanting? They have started to explore this issue by looking at what variables predict what couples are going to maintain their wanting over time. Partners who “keep discovering new things about their partner” seem to maintain wanting better, and trying new things sexually seems to make a difference.

*Discussion:*

A participant (name unknown) noted that people might like sex to the extent that his or her partner likes sex. Therefore, if the women aren't liking it, is that driving the findings for the men? Dr. Loewenstein noted that they asked subjects how excited are you by your partner and how excited is your partner by you? He could not remember the exact findings, but recalled both wanting and liking for men and women was influenced by how excited they thought their partner was by them and vice versa.

Bob Levenson noted findings indicating distinct neural circuits for wanting and liking (related to addiction) and asked Dr. Loewenstein if he could frame his findings in those terms. Dr. Loewenstein responded that Berridge has been arguing that the neural circuitry is overlapping but distinct, and so it raises the question as to whether this circuitry for sex is the same as for other things. There must be an overlap. Dr. Levenson suggested looking at people who are old but who are in new couples to get to the question about what effects are due to age. Dr. Loewenstein said he was getting at that question using regression. Duration seems to be more important than age, though there are some gender differences in that effect.

A participant (name unknown) asked why Dr. Loewenstein thought that women, despite liking sex almost as much as men when they have it, wanted it less. Is it an affective forecasting problem, or a recall bias? Dr. Loewenstein believed that for long-married couples, they may still like sex, but they are no longer aroused (a “cold state”). The prescription is to have sex whether you are in the mood or not, because they will probably enjoy it.

### ***Session 3: Open discussion***

**Moderators: Lisbeth Nielsen and Robert Levenson**

Lis Nielsen began the discussion by reiterating the three main questions: 1) How can the work being done in decision-making and aging and related areas be applied to advancing research toward promoting adaptive and healthy aging?, 2) How can each of the three domains represented by the panel sections can talk to one another?, and 3) What is the next big question or direction for the research on decision-making? Bob Levenson added that we've heard a strong case that decision-making is influenced by age, as a both a social phenomenon and as a set of changes in the brain. The question, then, is how do you parse the variance between biology and the social experiences that come with living a long life?

Brian Knutson responded that his work with brain imaging and emotion has made him think more about time, which has made him think more causally. He noted that we tend to think of emotions as driving behavior, memory, etc., but we may be wrong. We need to make testable hypotheses and examine how activation related to actual behaviors or choices. This will help to link brain-based/emotion activation to real choice and social outcomes. If he had unlimited funds, he would focus on linking levels and test temporally-specific predictions across several levels (e.g., brain-based, genetic, psychometric, social, financial outcomes) to look at various types of risk-taking behaviors with incentive-compatible designs.

Sam McClure reiterated Dr. Levenson's framing question, asking what is the relative dependence of age-related changes in the brain and strategic (i.e., reversible) adaptations in terms of understanding successful decision-making or cognition through age? Dr. Nielsen referenced Natalie Denburg's findings, which showed that even with two relatively intact groups of older adults, you seen differences that affect important areas of decision-making. Further, as Laura Carstensen has shown, motivations can be manipulated, suggesting external influences (and not just changes in the brain). Dr. Carstensen followed up, noting that the brain maps behavior. If you can eliminate the positivity effect behaviorally (with simple

instructions), then it seems that age-related brain changes led to that behavioral difference. It's not to say there aren't age-related brain changes, but in this case, it does not seem that the brain is the causal factor in that chain of events. Dr. Levenson noted that his question wasn't chicken vs. egg, but rather how do we design research that can help us understand the relative contribution of these two factors. Dr. Carstensen suggested that if what's changing is desire, and what one finds valuable, then interventions could be aimed at directing people to do what they don't want to do (i.e., focus on both negative as well). Dr. Nielsen followed up, noting that the brain changes seen in aging accrue over a long time, so it is also important to look at what was going on at earlier stages.

Richard Suzman brought up an important difference between aging and experience and pointed out that there is important work to be done in behavior genetics. JoNell Strough noted that even if someone has the genotype, it depends on the environment for the expression. That is a concern with getting to heavily invested in genetic markers, because so much depends on the environment. Natalie Denburg voiced her concern about getting into genetic research: now that we're mapping genome, anything she does feels trivial or is a guessing game. Dr. Suzman replied that candidate gene research does get accepted for funding, but acknowledged that it's an area that is changing rapidly. Shelley Taylor suggested that researchers need to get into that area with some hypotheses about what systems are likely to be involved. You look at these systems and try to make some intelligent assumptions about what systems may be implicated. Then if you can get a big enough sample, you can test it out.

Dr. Taylor then asked Mara Mather about her findings regarding sex differences in decision-making, with women becoming more conservative under stress. Dr. Taylor linked that to recent findings in the behavioral finance literature which indicate that women are better at making investments than men. She wondered if this effect might be due to stress-related investment making (i.e., men are getting more risky, women are getting more protective). Dr. Mather noted that context is an important factor (i.e., risky behaviors can be adaptive when the stock market is going up, bad when it's going down).

Alex Zautra suggested that it might be interesting to look at these decisions under conditions of stress. Dr. Knutson had found differences in incentive-based versus disincentive-based decision-making. In his research, he had found that under stress, self-reports between positive and negative emotion become more unitary and inversely related. Some way of unifying these areas (stress, emotion, and decision-making) would be a useful endeavor. Dr. Nielsen noted that there is a link in terms of the paradigms that stress researchers and decision-making researchers are using, but the outcomes they consider (health outcomes vs. decision-making in daily life) are different. There is a real interface there: health behavior decisions in daily life that lead to later health outcomes.

George Loewenstein described some of his recent related work. They have been conducting a study trying to incentivize older people to do mental activities on Internet, and they have been comparing incentive structures (egoistic, cooperative, competitive). They have had surprisingly high rates of participation (ceiling effects). In general, different aging effects have a lot of ramifications for incentive-change programs. For example, the status-quo effect can be used to get people to do healthy things. If older people are less subject to status-quo effects, then those interventions might not be as effective. Thus, a lot of this research has implications for the effective design of behavior-change programs. Dr. Strough noted that Dr. Loewenstein's idea (in a paper) to structure the environment so that the healthy choice is the default is a great suggestion.

A participant (name unknown) noted findings showing that higher stakes seemed to improve performance for younger adults, but impaired performance for older adults (perhaps because the higher stakes added more stress). He noted that when changing incentives for older subjects, it would be important to make sure it did not cause a negative impact by adding stressors. Dr. Nielsen noted the converse issue—older adults may not have committed the sunk-cost fallacy for the movie because they were not as concerned about losing \$10.95. Dr. Knutson suggested that you need to know what the adults are feeling to understand where the effects are coming from. Another participant (name unknown) noted that this underlines the importance of understanding the causal mechanisms if we want to make practical improvements in people's lives. For example, what is the effect of the incentive—does it motivate or

cause stress? Paradoxically, in our rush to be relevant, we have to think more about causal processes, about underlying mechanisms, about what we are really measuring, and what we are really manipulating.

Dr. Nielsen concluded by highlighting that NIH is in effort to advance the science on behavior change to be more focused on mechanistic aspects from the basic science that can inform the ways that interventions are designed and conducted across all the domains discussed today. One of the goals of all of this is to bring basic science across the fields (e.g., psychology, affective and cognitive neuroscience, neuroeconomics) to the table with the people who are doing the intervention work. Because intervention work is often dispersed across the NIH institutes, there has not been a concerted effort to bring scientists from multiple disciplines together to understand the common psychological, social, behavioral and physiological mechanisms and processes that support and sustain behavior change. She hopes to see some initiatives come out of these discussions and to develop dialogues across the disciplines represented here today.

## General Discussion

Bob Levenson thanked all the workgroup participants for coming. He then asked for feedback. Last year, the workgroup meeting was didactic and focused on the question of what would you need to know how to do in order to translate basic science to the study of aging. The goal this time was to take a snapshot of what was going on in labs of the major players in these three major areas of aging research. We may do something similar in the next year (or over the next several years). What should we do next? What would be useful for either new or established researchers in this field?

Jen Lerner referenced Eric Erickson and asked the group what they thought he might say about the field. What about life stage? We tend to compare old versus young, but is there an interest in starting to look at specific kinds of aging processes, akin to different life goals? It seems like we need to make finer gradations rather than old/young. Can we draw on other work about the challenges unique to different life stages *within* older populations? Dr. Nielsen noted that the research is typically approached via chronological life stages across the life course, but this would imply approaching it based on goal-relevant stages. Dr. Blanchard-Fields indicated that there was a lot of research taking that perspective. Many of the research talks today took an extreme age groups design (old vs. young) to establish a phenomenon. She approaches it from a full developmental model with data over the life course. In the aging field, you have both experimental and developmental approaches.

Richard Suzman noted that the study of the full life course is ready for a renaissance in terms of putting together multiple longitudinal studies. New methodologies and technologies such as genetics, and other disciplines like economics that have different and in some cases better ways of analyzing data, are going to change the whole field. Related to the stage discussion, Laura Carstensen suggested that the classical stage models don't really hold up to empirical tests (even Ericson suggested the stage model might need to be more flexible). Individual variability seems to be the story. Susan Fiske pointed out that status and age confounded, so if the phenomenon that people are interested in covaries with status, researchers might be shooting themselves in the foot by comparing young versus older people (neither of whom tend to be high status).

Margaret Kemeny noted that one nice thing about the workgroup today was the juxtaposition of different tools and different frameworks. One strategy for future meetings is to take this group and put them together in a way that builds research. Take some ideas and connections and make them happen. It starts to happen at a meeting like this, but what do you do next? Related to this point, Lani Shiota noted that certain studies had potential conflicts between findings. It might also be useful to discuss the potential moderators that might account for such divergent findings.

Dr. Nielsen reminded the group of upcoming NIA initiatives. One RFA that has been approved is to develop networks for interactions among researchers. BSR can, to some extent, support meetings on its own. But they can also work with [workgroup participants] to support meeting grants and other activities designed to bring people together. They are happy to consider applications from people who are interested in pushing dialogues forward through seminars, summer institutes, etc., and will try to figure out how to work together to make these things happen. Optimal activities are those that can be sustained over several years.

Dr. Suzman noted that part of the problem is a structural problem. In psychology, NIA does not have a coordinating center. Lacking that infrastructure, it's necessary to structure activities in some other way so that there is some degree continuity, but also self-direction. He also noted that there were not many disciplines represented at the meeting. It would be useful to get together with surrounding disciplines. You could have two meetings, one narrowly focus and one broad. In addition, there are program projects that involve at least three sub R01-like projects with a number of cores; there could be a core designed to bring people together. Finally, he suggested that people think large-scale.

Dr. Nielsen brought up the interface between lab work and survey work. People don't always know about the resources that are publically available. She cited the Health and Retirement Study and several

foreign datasets, such as the English Longitudinal Study on Aging. Dr. Nielsen pointed out that NIA can support work from researchers going to those sources (via regular grant mechanisms) to do secondary analysis on data from these projects. Dr. Sbarra noted that he's been on study sections when this has come up, and the message does need to come out that this is a legitimate way to get work done. Unfortunately, and they all agreed, this kind of work is rare for psychologists, they don't always know how to work with the data, and the culture of reviewers needs to be changed to accept this work. Dr. Suzman further cited the challenge for psychologists of getting word out about their findings.

With regard to cross-disciplinary grants, Laura Kubzansky noted that given a limited amount of space and multiple people contributing, the reviewers inevitable don't think you've spent enough time on the piece that is theirs. There is a call for this type of work, but then there are these built-in barriers to getting cross-disciplinary grants funded. Dr. Suzman said potential solutions are RFA's and program projects.

## References

1. Carstensen, L. L. (1993). Motivation for social contact across the life span: A theory of socioemotional selectivity. In J. E. Jacobs (Ed.), *Nebraska symposium on motivation: 1992, Developmental Perspectives on Motivation* (Vol. 40, pp. 209-254). Lincoln: University of Nebraska Press.
2. Carstensen, L. L., Isaacowitz, D. M., & Charles, S. T. (1999). Taking time seriously. A theory of socioemotional selectivity. *American Psychologist*, *54*(3), 165-181.
3. Carstensen, L. L. (2006). The influence of a sense of time on human development. *Science*, *312*(5782), 1913-1915.
4. Levenson, R. W., Carstensen, L. L., & Gottman, J. M. (1993). Long-term marriage: age, gender, and satisfaction. *Psychology and Aging*, *8*(2), 301-313.
5. Levenson, R. W., Carstensen, L. L., & Gottman, J. M. (1994). The influence of age and gender on affect, physiology, and their interrelations: a study of long-term marriages. *Journal of Personality and Social Psychology*, *67*(1), 56-68.
6. Charles, S. T., & Carstensen, L. L. (2008). Unpleasant situations elicit different emotional responses in younger and older adults. *Psychology and Aging*, *23*(3), 495-504.
7. Ersner-Hershey, H., Mikels, J. A., Sullivan, S. J., & Carstensen, L. L. (2008). Poignancy: mixed emotional experience in the face of meaningful endings. *Journal of Personality and Social Psychology*, *94*(1), 158-167.
8. Mather, M., & Carstensen, L. L. (2003). Aging and attentional biases for emotional faces. *Psychological Science*, *14*(5), 409-415.
9. Charles, S. T., Mather, M., & Carstensen, L. L. (2003). Aging and emotional memory: the forgettable nature of negative images for older adults. *Journal of Experimental Psychology: General*, *132*(2), 310-324.
10. Mather, M., & Carstensen, L. L. (2005). Aging and motivated cognition: the positivity effect in attention and memory. *Trends in Cognitive Sciences*, *9*(10), 496-502.
11. Carstensen, L. L., Mikels, J. A., & Mather, M. (2006). Aging and the intersection of cognition, motivation and emotion. In J. E. Birren & K. W. Schaie (Eds.), *Handbook of the Psychology of Aging* (pp. 343-362): Academic Press.
12. Lockenhoff, C. E., & Carstensen, L. L. (2008). Decision strategies in health care choices for self and others: Older but not younger adults make adjustments for the age of the decision target. *Journals of Gerontology: Series B: Psychological Sciences and Social Sciences*, *63B*(2), P106-P109.
13. Lockenhoff, C. E., & Carstensen, L. L. (2007). Aging, emotion, and health-related decision strategies: motivational manipulations can reduce age differences. *Psychology and Aging*, *22*(1), 134-146.
14. Samanez-Larkin, G. R., Gibbs, S. E. B., Khanna, K., Nielsen, L., Carstensen, L. L., & Knutson, B. (2007). Anticipation of monetary gain but not loss in healthy older adults. *Nature Neuroscience*, *10*(6), 787-791.

15. Ruffman, T., Henry, J. D., Livingstone, V., & Phillips, L. H. (2008). A meta-analytic review of emotion recognition and aging: implications for neuropsychological models of aging. *Neuroscience and Biobehavioral Reviews*, *32*(4), 863-881.
16. Adams, R. B., Jr., & Kleck, R. E. (2005). Effects of Direct and Averted Gaze on the Perception of Facially Communicated Emotion. *Emotion*, *5*(1), 3-11.
17. Phillips, L. H., MacLean, R. D., & Allen, R. (2002). Age and the understanding of emotions: neuropsychological and sociocognitive perspectives. *Journals of Gerontology. Series B, Psychological Sciences and Social Sciences*, *57*(6), P526-530.
18. Slessor, G., Phillips, L. H., & Bull, R. (2008). Age-related declines in basic social perception: evidence from tasks assessing eye-gaze processing. *Psychology and Aging*, *23*(4), 812-822.
19. Isaacowitz, D. M., Toner, K., Goren, D., & Wilson, H. R. (2008). Looking while unhappy: mood-congruent gaze in young adults, positive gaze in older adults. *Psychological Science*, *19*(9), 848-853.
20. Scheibe, S., & Blanchard-Fields, F. (2009). Effects of regulating emotions on cognitive performance: what is costly for young adults is not so costly for older adults. *Psychology and Aging*, *24*(1), 217-223.
21. Fontenot, M. B., Kaplan, J. R., Manuck, S. B., Arango, V., & Mann, J. J. (1995). Long-term effects of chronic social stress on serotonergic indices in the prefrontal cortex of adult male cynomolgus macaques. *Brain Research*, *705*(1-2), 105-108.
22. Manuck, S. B., Flory, J. D., Ferrell, R. E., & Muldoon, M. F. (2004). Socio-economic status covaries with central nervous system serotonergic responsivity as a function of allelic variation in the serotonin transporter gene-linked polymorphic region. *Psychoneuroendocrinology*, *29*(5), 651-668.
23. Bennett, A. J., Lesch, K. P., Heils, A., Long, J. C., Lorenz, J. G., Shoaf, S. E., et al. (2002). Early experience and serotonin transporter gene variation interact to influence primate CNS function. *Molecular Psychiatry*, *7*(1), 118-122.
24. Manuck, S. B., Bleil, M. E., Peterson, K. L., Flory, J. D., Mann, J. J., Ferrell, R. E., et al. (2005). The socio-economic status of communities predicts variation in brain serotonergic responsivity. *Psychological Medicine*, *35*(4), 519-528.
25. Gunnar, M. R., & Donzella, B. (2002). Social regulation of the cortisol levels in early human development. *Psychoneuroendocrinology. Special Issue: Stress and drug abuse*, *27*(1-2), 199-220.
26. Marin, T. J., Chen, E., & Miller, G. E. (2008). What do trajectories of childhood socioeconomic status tell us about markers of cardiovascular health in adolescence? *Psychosomatic Medicine*, *70*(2), 152-159.
27. Taylor, S. E., Way, B. M., Welch, W. T., Hilmert, C. J., Lehman, B. J., & Eisenberger, N. I. (2006). Early family environment, current adversity, the serotonin transporter promoter polymorphism, and depressive symptomatology. *Biological Psychiatry*, *60*(7), 671-676.
28. Taylor, S. E., Eisenberger, N. I., Saxbe, D., Lehman, B. J., & Lieberman, M. D. (2006). Neural responses to emotional stimuli are associated with childhood family stress. *Biological Psychiatry*, *60*(3), 296-301.

29. Yusuf, S., Hawken, S., Åunpuu, S., Dans, T., Avezum, A., Lanas, F., et al. (2004). Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): Case-control study. *Lancet*, *364*(9438), 937-952.
30. Kubzansky, L. D., & Thurston, R. C. (2007). Emotional vitality and incident coronary heart disease: benefits of healthy psychological functioning. *Archives of General Psychiatry*, *64*(12), 1393-1401.
31. Hawthorne, G. (2008). Perceived social isolation in a community sample: Its prevalence and correlates with aspects of peoples' lives. *Social Psychiatry and Psychiatric Epidemiology*, *43*(2), 140-150.
32. Pinquart, M., & Sorensen, S. (2003). Risk factors for loneliness in adulthood and old age--a meta-analysis. *Shohov, Serge P*, *19*(pp. 111-143).
33. Hawkey, L. C., Hughes, M. E., Waite, L. J., Masi, C. M., Thisted, R. A., & Cacioppo, J. T. (2008). From social structural factors to perceptions of relationship quality and loneliness: the Chicago health, aging, and social relations study. *Journals of Gerontology. Series B, Psychological Sciences and Social Sciences*, *63*(6), S375-384.
34. Booth, R. (2000). Loneliness as a component of psychiatric disorders: The relationship between loneliness and depression, *Medscape General Medicine*, *2*(2).
35. Sbarra, D. A., & Nietert, P. J. (2009). Divorce and death: forty years of the Charleston Heart Study. *Psychological Science*, *20*(1), 107-113.
36. Sbarra, D. A., Law, R. W., Lee, L. A., & Mason, A. E. (2009). Marital Dissolution and Blood Pressure Reactivity: Evidence for the Specificity of Emotional Intrusion-Hyperarousal and Task-Rated Emotional Difficulty. *Psychosomatic Medicine*.
37. Lyubomirsky, S., Sheldon, K. M., & Schkade, D. (2005). Pursuing Happiness: The Architecture of Sustainable Change. *Review of General Psychology. Special Issue: Positive Psychology*, *9*(2), 111-131.
38. Blackburn, E. H., & Gall, J. G. (1978). A tandemly repeated sequence at the termini of the extrachromosomal ribosomal RNA genes in Tetrahymena. *Journal of Molecular Biology*, *120*(1), 33-53.
39. Epel, E. S. (2009). Telomeres in a life-span perspective: A new "psychobiomarker"? *Current Directions in Psychological Science*, *18*(1), 6-10.
40. Epel, E., Stein Merkin, S., Cawthon, R., Blackburn, E. H., Adler, N. E., Pletcher, M. J., et al. (2009). The rate of leukocyte telomere shortening predicts mortality from cardiovascular disease in elderly men *Aging*, *1*(1), 81-88.
41. Brammer, M. (2004). Brain scam? *Nature Neuroscience*, *7*(10), 1015.
42. Knutson, B., & Greer, S. M. (2008). Anticipatory affect: neural correlates and consequences for choice. *Philosophical Transactions of the Royal Society of London. Series B: Biological Sciences*, *363*(1511), 3771-3786.
43. Samanez-Larkin, G. R., Hollon, N. G., Carstensen, L. L., & Knutson, B. (2008). Individual differences in insular sensitivity during loss anticipation predict avoidance learning. *Psychological Science*, *19*(4), 320-323.

44. Denburg, N. L., Tranel, D., & Bechara, A. (2005). The ability to decide advantageously declines prematurely in some normal older persons. *Neuropsychologia*, *43*(7), 1099-1106.
45. Denburg, N. L., Cole, C. A., Hernandez, M., Yamada, T. H., Tranel, D., Bechara, A., et al. (2007). The orbitofrontal cortex, real-world decision making, and normal aging. *Annals of the New York Academy of Sciences*, *1121*, 480-498.
46. Mather, M., Gorlick, M. A., & Lighthall, N. R. (2009). To brake or accelerate when the light turns yellow? Stress reduces older adults' risk taking in a driving game. *Psychological Science*, *20*(2), 174-176.
47. Arkes, H. R., & Ayton, P. (1999). The sunk cost and Concorde effects: Are humans less rational than lower animals? *Psychological Bulletin*, *125*(5), 591-600.
48. Frisch, D. (1993). Reasons for framing effects. *Organizational Behavior and Human Decision Processes*, *54*(3), 399-429.
49. Strough, J., Mehta, C. M., McFall, J. P., & Schuller, K. L. (2008). Are older adults less subject to the sunk-cost fallacy than younger adults? *Psychological Science*, *19*(7), 650-652.
50. Carstensen, L. L., & Mikels, J. A. (2005). At the Intersection of Emotion and Cognition: Aging and the Positivity Effect. *Current Directions in Psychological Science*, *14*(3), 117-121.
51. Wood, S., Busemeyer, J., Koling, A., Cox, C. R., & Davis, H. (2005). Older Adults as Adaptive Decision Makers: Evidence From the Iowa Gambling Task. *Psychology and Aging*, *20*(2), 220-225.
52. Baumeister, R. F., Bratslavsky, E., Finkenauer, C., & Vohs, K. D. (2001). Bad is stronger than good. *Review of General Psychology*, *5*(4), 323-370.
53. Strough, J., DiDonato, L., Schlosnagle, L., Keener, E., McFall, J., & Mehta, C. (2009). The sunk-cost fallacy is associated with older and younger adults future time perspective, *Poster presentation at the annual meeting of the Association for Psychological Science*. San Francisco, CA.
54. Davey-Smith, G., Frankel, S., & Yarnell, J. (1997). Sex and death: are they related? Findings from the Caerphilly cohort study. *British Medical Journal*, *315*, 1641-1645.
55. Leitzmann, M. F., Platz, E. A., Stampfer, M. J., Willett, W. C., & Giovannucci, E. (2004). Ejaculation frequency and subsequent risk of prostate cancer. *JAMA*, *291*(13), 1578-1586.
56. Kahneman, D., Krueger, A. B., Schkade, D. A., Schwarz, N., & Stone, A. A. (2004). A survey method for characterizing daily life experience: the day reconstruction method. *Science*, *306*(5702), 1776-1780.
57. Laumann, E. O., Michael, R. T., & Gagnon, J. H. (1994). A political history of the national sex survey of adults. *Family Planning Perspectives*, *26*(1), 34-38.
58. Branchflower, D. G., & Oswald, A. J. (2004). Money, sex, and happiness: An empirical study. *Scandinavian Journal of Economics*, *106*(3), 393-415.
59. Smith, K. S., & Berridge, K. C. (2007). Opioid limbic circuit for reward: interaction between hedonic hotspots of nucleus accumbens and ventral pallidum. *Journal of Neuroscience*, *27*(7), 1594-1605.

## Appendix 1: Agenda

**7:30 a.m. Coffee and pastries will be available**

**8:00 a.m. Introduction and Welcoming Remarks**

- Robert Levenson, University of California, Berkeley: Introduction and Framing the Questions
- Lisbeth Nielsen, National Institute on Aging: NIA interests
- Richard Suzman, National Institute on Aging: NIA vision

### **Session 1: Fundamental Social and Affective Processes in Aging**

**8:30 a.m. Framing Talk**

Laura Carstensen, Stanford University: Why isn't aging depressing?

**8:50 a.m. Exciting Findings**

- Louise Phillips, University of Aberdeen: Aging and the use of emotional cues to guide social judgments
- Derek Isaacowitz, Brandeis University: What is the function of age-related positive gaze preferences?
- Fredda Blanchard-Fields, Georgia Institute of Technology: Effective emotion regulation in older adulthood: Converging levels of analysis
- Steve Manuck, University of Pittsburgh: Correlates of social position in brain serotonergic function
- Michael Lamb, University of Cambridge: Exploring the effects of attachment relationships on reactions to transitions

**10:00 a.m. Open discussion (Moderators: Bob Levenson and Lis Nielsen)**

**10:30 a.m. Break**

### **Session 2: Healthy Aging Over the Lifecourse**

**11:00 a.m. Framing Talk**

Shelley Taylor, University of California, Los Angeles: Stress, social processes, and health over the lifecourse

**11:20 a.m. Exciting Findings**

- Laura Kubzansky, Harvard School of Public Health: Biology of resilience: Oxytocin, positive adaptation and health
- Louise Hawkley, University of Chicago: Loneliness: Cause and target
- David Sbarra, University of Arizona: Relationship disruptions and health: From social epidemiology to social psychophysiology
- Sonja Lyubomirsky, University of California, Riverside: The promise of interventions for promoting well-being
- Elissa Epel, University of California, San Francisco: Psychosocial influences on longevity biomarkers

**12:30 p.m. Open discussion (Moderators: Lis Nielsen and Bob Levenson)**

**1:00 p.m. LUNCH**

**Session 3: Decision Making in Aging**

- 2:30 p.m. Framing Talk**  
Brian Knutson, Stanford University: Decision Making in Aging: Emerging Insights from Affective Neuroscience and Neuroeconomics
- 2:50 p.m. Exciting Findings**
- Natalie Denburg, University of Iowa: Neural basis of decision making in aging
  - Mara Mather, University of Southern California: Age and sex differences in the effects of stress on decision making
  - JoNell Strough, West Virginia University: No time to waste: Understanding why older adults are less subject to the sunk-cost fallacy
  - George Loewenstein, Carnegie Mellon University: Wanting and liking for sex by gender and age
- 3:50 p.m. Open Discussion (Moderators: Bob Levenson and Lis Nielsen)**
- 4:20 p.m. BREAK**
- 4:50 p.m. General Discussion**

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- Coats, A. H., & Blanchard-Fields, F. (2008). Emotion regulation in interpersonal problems: the role of cognitive-emotional complexity, emotion regulation goals, and expressivity. *Psychology and Aging, 23*(1), 39-51.
- Scheibe, S., & Blanchard-Fields, F. (2009). Effects of regulating emotions on cognitive performance: what is costly for young adults is not so costly for older adults. *Psychology and Aging, 24*(1), 217-223.
- Blanchard-Fields, F. (2007). Everyday problem solving and emotion: An adult developmental perspective. *Current Directions in Psychological Science, 16*(1), 26-31.

#### **Laura Carstensen**

- Carstensen, L. L. (2006). The influence of a sense of time on human development. *Science, 312*(5782), 1913-1915.
- Charles, S. T., & Carstensen, L. L. (2008). Unpleasant situations elicit different emotional responses in younger and older adults. *Psychology and Aging, 23*(3), 495-504.
- Ersner-Hershfield, H., Mikels, J. A., Sullivan, S. J., & Carstensen, L. L. (2008). Poignancy: mixed emotional experience in the face of meaningful endings. *Journal of Personality and Social Psychology, 94*(1), 158-167.

#### **Natalie Denburg**

- Denburg, N. L., Cole, C. A., Hernandez, M., Yamada, T. H., Tranel, D., Bechara, A., et al. (2007). The orbitofrontal cortex, real-world decision making, and normal aging. *Annals of the New York Academy of Sciences, 1121*, 480-498.
- Denburg, N. L., Tranel, D., & Bechara, A. (2005). The ability to decide advantageously declines prematurely in some normal older persons. *Neuropsychologia, 43*(7), 1099-1106.
- Denburg, N. L., Weller, J. A., Yamada, T. H., Shivapour, D. M., Kaup, A. R., Laloggia, A., et al. (2009). Poor Decision Making Among Older Adults Is Related to Elevated Levels of Neuroticism. *Annals of Behavioral Medicine.*

#### **Elissa Epel**

- Epel, E.S., Merkin, S.S., Cawthon, R., Blackburn, E.H., Adler, N.E., Pletcher, M.J., Seeman, T.E. (2009). The rate of leukocyte telomere shortening predicts mortality from cardiovascular disease in elderly men. *Aging, 1*(1), 81-88.
- Epel, E. S., Blackburn, E. H., Lin, J., Dhabhar, F. S., Adler, N. E., Morrow, J. D., et al. (2004). Accelerated telomere shortening in response to life stress. *Proceedings of the National Academy of Sciences of the United States of America, 101*(49), 17312-17315.
- Epel, E.S. (2009). Telomeres in a life-span perspective: A new "psychobiomarker"? *Current Directions in Psychological Science, 18*(1), 6-10.

#### **Louise Hawkley**

- Hawkley, L.C., & Cacioppo, J.T. (2007). Aging and loneliness: Downhill quickly? *Current Directions in Psychological Science, 16*(4), 187-191.
- Cacioppo, J. T., Hughes, M. E., Waite, L. J., Hawkley, L. C., & Thisted, R. A. (2006). Loneliness as a specific risk factor for depressive symptoms: cross-sectional and longitudinal analyses. *Psychology and Aging, 21*(1), 140-151.
- Hawkley, L. C., Masi, C. M., Berry, J. D., & Cacioppo, J. T. (2006). Loneliness is a unique predictor of age-related differences in systolic blood pressure. *Psychology and Aging, 21*(1), 152-164.

**Derek Isaacowitz**

- Isaacowitz, D. M., Toner, K., Goren, D., & Wilson, H. R. (2008). Looking while unhappy: mood-congruent gaze in young adults, positive gaze in older adults. *Psychological Science*, *19*(9), 848-853.
- Isaacowitz, D. M., Wadlinger, H. A., Goren, D., & Wilson, H. R. (2006). Selective preference in visual fixation away from negative images in old age? An eye-tracking study. *Psychology and Aging*, *21*(1), 40-48.
- Isaacowitz, D. M., & Fung, H. H. (Manuscript). Motivation across time and place: What gaze can tell us about aging and culture. In E. Balcetis & D. Lassiter (Eds.), *Social Psychology of Vision*.

**Brian Knutson**

- Ersner-Hershey, H., Wimmer, G. E., & Knutson, B. (2009). Saving for the future self: neural measures of future self-continuity predict temporal discounting. *Soc Cogn Affect Neurosci*, *4*(1), 85-92.
- Knutson, B., & Greer, S. M. (2008). Anticipatory affect: neural correlates and consequences for choice. *Philosophical Transactions of the Royal Society of London. Series B: Biological Sciences*, *363*(1511), 3771-3786.
- Samanez-Larkin, G. R., Gibbs, S. E., Khanna, K., Nielsen, L., Carstensen, L. L., & Knutson, B. (2007). Anticipation of monetary gain but not loss in healthy older adults. *Nature Neuroscience*, *10*(6), 787-791.
- Samanez-Larkin, G. R., Gibbs, S. E., Khanna, K., Nielsen, L., Carstensen, L. L., & Knutson, B. (2007). Anticipation of monetary gain but not loss in healthy older adults. *Nature Neuroscience, Supplementary Results*.

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- Kubzansky, L. D. (2007). Sick at heart: the pathophysiology of negative emotions. *Cleveland Clinic Journal of Medicine*, *74* Suppl 1, S67-72.
- Heinrichs, M., & Domes, G. (2008). Neuropeptides and social behaviour: effects of oxytocin and vasopressin in humans. In I.D. Neumann & R. Landgraf (Eds.), *Progress in Brain Research (Vol. 170)*. Amsterdam: Elsevier, 337-350.
- Kubzansky, L. D., Martin, L. T., & Buka, S. L. (Proof). Early manifestations of personality and adult health: A life course perspective. *Health Psychology*.

**Michael Lamb**

- Ahnert, L., Gunnar, M. R., Lamb, M. E., & Barthel, M. (2004). Transition to child care: associations with infant-mother attachment, infant negative emotion, and cortisol elevations. *Child Development*, *75*(3), 639-650.
- Lamb, M.E. (2009). Child care and new family forms. Background paper for European Science Foundation conference on "Changing childhood in a changing Europe" Nicosia, Cyprus, February 26-27, 2009.

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- Berns, G. S., Laibson, D., & Loewenstein, G. (2007). Intertemporal choice--toward an integrative framework. *Trends Cogn Sci*, *11*(11), 482-488.
- Loewenstein, G., Brennan, T., & Volpp, K. G. (2007). Asymmetric paternalism to improve health behaviors. *JAMA*, *298*(20), 2415-2417.
- Volpp, K. G., John, L. K., Troxel, A. B., Norton, L., Fassbender, J., & Loewenstein, G. (2008). Financial incentive-based approaches for weight loss: a randomized trial. *JAMA*, *300*(22), 2631-2637.

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- Lyubomirsky, S., King, L., & Diener, E. (2005). The benefits of frequent positive affect: does happiness lead to success? *Psychological Bulletin*, *131*(6), 803-855.

Lyubomirsky, S., Sheldon, K. M., & Schkade, D. (2005). Pursuing happiness: The architecture of sustainable change. *Review of General Psychology. Special Issue: Positive Psychology, 9*(2), 111-131.

Lyubomirsky, S., Dickerhoof R., Boehm, J.K., & Sheldon, K.M. (under review). Becoming happier takes both a will and a proper way: Two experimental longitudinal interventions to boost well-being.

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Manuck, S. B., Bleil, M. E., Peterson, K. L., Flory, J. D., Mann, J. J., Ferrell, R. E., et al. (2005). The socio-economic status of communities predicts variation in brain serotonergic responsivity. *Psychological Medicine, 35*(4), 519-528.

Manuck, S. B., Flory, J. D., Ferrell, R. E., & Muldoon, M. F. (2004). Socio-economic status covaries with central nervous system serotonergic responsivity as a function of allelic variation in the serotonin transporter gene-linked polymorphic region. *Psychoneuroendocrinology, 29*(5), 651-668.

Gallo, L. C., & Matthews, K. A. (2003). Understanding the association between socioeconomic status and physical health: Do negative emotions play a role? *Psychological Bulletin, 129*(1), 10-51.

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Novak, D. L., & Mather, M. (2007). Aging and variety seeking. *Psychology and Aging, 22*(4), 728-737.

Mather, M. (2006). A review of decision making processes: Weighing the risks and benefits of aging. In L. L. Carstensen and C. R. Hartel (Eds.), *When I'm 64. Committee on Aging Frontiers in Social Psychology, Personality, and Adult Developmental Psychology*. Washington, DC: The National Academies Press, 145-173.

Mather, M., Gorlick, M. A., & Lighthall, Nichole R. (2009). To brake or accelerate when the light turns yellow? Stress reduces older adults' risk taking in a driving game. *Psychological Science, 20*(2), 174-176.

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Phillips, L. H., MacLean, R. D., & Allen, R. (2002). Age and the understanding of emotions: neuropsychological and sociocognitive perspectives. *Journals of Gerontology. Series B, Psychological Sciences and Social Sciences, 57*(6), 526-530.

Ruffman, T., Henry, J. D., Livingstone, V., & Phillips, L. H. (2008). A meta-analytic review of emotion recognition and aging: implications for neuropsychological models of aging. *Neuroscience and Biobehavioral Reviews, 32*(4), 863-881.

Henry, J. D., Ruffman, T., McDonald, S., O'Leary, M. P., Phillips, L. H., Brodaty, H., et al. (2008). Recognition of disgust is selectively preserved in Alzheimer's disease. *Neuropsychologia, 46*(5), 1363-1370.

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Sbarra, D. A., & Hazan, C. (2008). Coregulation, dysregulation, self-regulation: an integrative analysis and empirical agenda for understanding adult attachment, separation, loss, and recovery. *Pers Social Psychology Review, 12*(2), 141-167.

Sbarra, D. A., Law, R. W., Lee, L. A., & Mason, A. E. (2009). Marital Dissolution and Blood Pressure Reactivity: Evidence for the Specificity of Emotional Intrusion-Hyperarousal and Task-Rated Emotional Difficulty. *Psychosomatic Medicine*.

Sbarra, D. A., & Nietert, P. J. (2009). Divorce and death: forty years of the Charleston Heart Study. *Psychological Science, 20*(1), 107-113.

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Peters, E., Hess, T. M., Vastfjall, D., & Auman, C. (2007). Adult age differences in dual information processes: Implications for the role of affective and deliberative processes in older adults' decision making. *Perspectives on Psychological Science*, 2(1), 1-23.

Strough, J., Mehta, C. M., McFall, J. P., & Schuller, K. L. (2008). Are older adults less subject to the sunk-cost fallacy than younger adults? *Psychological Science*, 19(7), 650-652.

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Taylor, S. E., Eisenberger, N. I., Saxbe, D., Lehman, B. J., & Lieberman, M. D. (2006). Neural responses to emotional stimuli are associated with childhood family stress. *Biological Psychiatry*, 60(3), 296-301.

Taylor, S. E., Way, B. M., Welch, W. T., Hilmert, C. J., Lehman, B. J., & Eisenberger, N. I. (2006). Early family environment, current adversity, the serotonin transporter promoter polymorphism, and depressive symptomatology. *Biological Psychiatry*, 60(7), 671-676.

Taylor, S. E. (in press). Pathways Linking Early Life Stress to Adult Health. In J. Decety and J. Cacioppo (Eds.), *The Handbook of Social Neuroscience*. New York: Oxford University Press.