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President's Column

Was That an Earthquake, or Just an NIMH Aftershock?

Michael F. Green
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You may or may not have noticed, but a sea change in psychological research recently occurred. It is a paradigm shift of sorts, one that involves the way our government funds psychological research. In October of 2004, the National Institute of Mental Health (NIMH) formally reorganized its divisions to emphasize translational research and de-emphasize basic behavioral research. The reorganization is the surface manifestation of a shift in priorities. Depending on who you talk to, this shift in organization and priorities is either: 1) a beneficial movement toward the disease-focused roots of NIMH, or 2) a damaging movement away from basic behavioral research. Also depending on who you speak to, the changes at NIMH are either: 1) congruent with its mission to reduce the burden of mental illness or 2) a shirking of their mission to enhance basic research.

Wait a minute. How is it possible for there to be disagreement about the mission statement of a national institute? Well, it depends on whether we are talking about the current mission statement, or the future one. The Congressional Charter does indeed indicate that NIMH's mission includes the "study of the psychological, social and legal factors that influence behavior." That sounds good for basic behavioral science. However, the mission statement will be revised to say the role of NIMH is to "reduce the burden of mental illness and behavioral disorders through research on mind, brain, and behavior." Notice that basic psychological research will not be specifically mentioned.

Is NIMH able to do that? They already did. There are different types of institutes at the National Institutes of Health (NIH), some of which are disease-focused and some not. NIMH is one of the disease-focused institutes, and despite its strong support of basic behavioral research in the past, it will now expect to see direct disease connections in the research it sponsors. This decision did not stem from concern about the *quality* of basic behavioral research, but about the *relevance* of this research for mental illness. The impression was that behavioral scientists were making beautiful music together, but the sound was reverberating in an echo chamber and not reaching clinical audiences.

Although the rationale is clear, the new focus essentially confers refugee status on a large cohort of basic behavioral scientists. There are assurances, naturally, that federal government will fund basic behavioral research but just not from NIMH. As we will see in a moment, no one has yet filled the gap.

Of all the psychological scientific organizations, SRP might be the least affected by the changes at NIMH because our research activities, by definition, have a disease focus. But that does not mean we are unaffected, it just means we have one degree of separation. Most of us have strong collaborations with basic behavioral scientists. Indeed, many of

us think that the strength of our research relies on the connections to basic behavioral science. If our basic colleagues have trouble maintaining their labs, our programs will suffer, just at a delayed interval.

How should an experimental psychopathologist react? Frankly we are caught in a dilemma. On the one hand, the new priorities will jeopardize support for our basic behavioral colleagues, and eventually jeopardize our collaborative networks. On the other hand, we cannot truly complain about the new priorities without being hypocritical. After all, the leadership of NIMH has articulated the same disease-focused priorities for their institute that we articulate for our research programs.

What should an experimental psychopathologist do? It is easier to say what one should not do. It is pointless to argue at this stage that NIMH *should* fund basic behavioral research. That approach will be unproductive for two reasons. One reason is that it is too late. The NIMH leadership has already made their decisions. A second reason is that other groups have voices that are louder and more effective than ours. For example, Congress pays the bills and NIMH listens. Tom Insel, Director of NIMH, has pointed out that Congress wants to know what NIMH is doing about conditions like schizophrenia, depression, alcoholism and suicide. Congressional staffers do not ask him about basic research. Another force in bringing about change at NIMH comes from family groups. Over the last several years, the family groups have become better organized and have emerged as a highly effective lobby. When they make presentations to Congress or NIH, they talk about their children, not constructs; they are looking for interventions, not models. Simply put, they get listened to.

The current need is to find an alternative home for basic behavioral research at NIH. For example, the NIH Working Group on Research Opportunities in the Basic Behavioral Sciences recommended that basic behavioral sciences should be supported by the National Institute of General Medical Sciences, which has a clear basic science mission. This is a good and sensible suggestion, but one that has not yet been heeded.

One positive concrete step we can take would be to look around us (perhaps just down the hall) for additional translational opportunities with our basic behavioral colleagues. The new NIMH has its eye on *translation*, a term with several different definitions. One definition of translation is from bench to bedside (from basic biology to treatments). A second definition is from efficacy to effectiveness (clinical trials to services). A third definition that applies to SRP is the translation from basic behavioral science to clinical science. According to its strategic plan (www.nimh.nih.gov/strategic/strategicplanmenu.cfm) the NIMH is building on the Decade of the Brain (1990s) to launch the Decade of Translation. If there is good news in this reorganization, it is that SRP members are likely to become attractive research partners for basic scientists as we enter the Decade of Translation. With a little imagination and flexibility we may successfully ride out the jolts created by this funding tremor.

Sharing some History…

Editors' note: Thanks to our illustrious Society members, we are privy to special exchanges and memories of some very special people in the history of experimental psychopathology. In this issue, we include excerpts from Rue Cromwell's informal letter to a colleague (Herb Spohn) in which he shares his thoughts and memories of Manfred Bleuler.

January 23, 2005

Dear Herb:

I had an insight this morning about the relation between Bleuler and Kraepelin. I should share it before it fades. For the first time this winter I set a fire in the fireplace and watched it burn down. For some reason, my thoughts went to Eugen Bleuler.

Joe Zubin had come to me once after introducing son Manfred Bleuler for a presentation. He had referred to Eugen Bleuler and Emil Kraepelin as friends. Manfred later accosted Joe to assert adamantly that his father and Kraepelin were never friends. Indeed, he said, they were very close colleagues. Although Zubin was brought from Lithuania to America in a dress at age two or so, he was fully aware of the European customs of social station with "Sie" and "du." Nevertheless Joe was puzzled. I had no answer.

I thought then of the visit I had made to Manfred Bleuler in his home in the village of Zølliken near Zürich. The home had once been a small farmhouse and the Bleuler family homestead. Now the farms were gone and the house blended in as part of a beautiful and quiet suburban street. The Herr doctor Professor Manfred Bleuler and I had had many exchanges earlier, yet I felt greatly the honor of visiting a great man of accomplishment in our mental health profession. My colleague and I were warmly greeted. Bleuler's wife was a very beautiful, pleasant, and somewhat quiet but attentive woman. I learned later that she had come from a Romano gypsy tribe and that their marriage was not one typical of the European tradition of social station.

We spoke of many things, but what my fireplace flames this morning (and Zubin's query) brought back to me was Manfred's description of his father's early years. Eugen Bleuler was the son of a farmer. In those days, as Manfred related, only members of the nobility were privileged to enter college. An amount of rebel spirit was in the air with the grandfather and his fellow Swiss farmers. They met to discuss their lot and decided that they would each buy books with the aim to assemble a library. Thereby the fathers would give their own children a college education. It was in this way that Manfred's father Eugen was introduced to academia.

Time passed. The great Burghølzli psychiatric hospital was built in Zürich. Since no psychiatrists were trained in Switzerland, the local authorities launched the

hospital by recruiting a group of psychiatrists from Germany. Soon it was apparent to all that the Germans did not understand the local dialect and subculture. Likely also they did not want to be bothered with ordinary patient needs and concerns. One patient may want a favorite piece of clothing from home; another may be concerned about placement of the baby in her absence; another may want to talk about a relationship with the spouse that was becoming frayed; another may want a bar of soap.

By this time Eugen Bleuler had been accepted into medical school at Bern. However, being identified as a local farm boy he was an ideal person to recruit and deal with the patients' needs and concerns. So it came to be that Eugen Bleuler's introduction to mental illness was not under medical school or psychiatric supervision. It was not to learn and detect the signs and symptoms of psychopathology. It was to minister to the patients through hours of listening and helping.

As we now know, this beginning with the mentally ill led to a distinguished career as the Director of the Burghølzli, mentoring many mental health professionals, coining the term schizophrenia, and advancing theory and research on the disorder. Heralded in his work were the cardinal features ("the four A's") of splitting of personality (a) association, (b) affect, (c) ambivalence, and later, (d) autism.

As my fire grew bright I fell upon an insight, correct or not, that Bleuler's necessarily more intimate relationship with patients, some his neighbors, sprang from a personal conceptual network more closely related to 'du" than "Sie." With the German psychiatrists it was the opposite. With this distinction, I surmized, Bleuler formed descriptor constructs linguistically more a subjective rendering as might occur if the patient were attempting to describe himself. In contrast, with the traditional German constructs, the patient is unlikely to assign an idea of his own as false (delusions) or a perception of his own as false (hallucinations). Other people did that for him. Thus the nosological typecasting had a relatively more external form. More important, however, the surface terms of the two respective approaches lent themselves to conceptual networks relatively more separated in the European linguistic tradition. It is likely that the Bleuler-Kraepelin differences are wider apart in structure for Europeans than Americans, and some Americans (like me) may be only vaguely aware of or fail to sense the network separation.

As fate would have it E. Bleuler's classic and well-written 1911 treatise on "the group of schizophrenias" was not translated into English until 1950. Other things were brewing that caught the public and professional eye. In America William James had just died and the legacy of pragmatist vs. Titchenerian structuralism was still being debated. In 1913 came Watson's behaviorist manifesto. Goddard, having translated Binet's test into English in 1910, set the stage for school psychology and the child guidance movement of the 1920s. In 1916 Stern used Binet's MA (mental age) in ratio with CA to invent IQ. England was introducing social psychology to America via James MacDougall. Even Bleuler's own interests competed with the dissemination of the schizophrenia opus. He mentored Hermann Rorschach who released his cards in 1911. [During our conversation Manfred told me proudly that he was the first person

ever to administer the Rorschach to children. As a youngster he grew up in the Director's living quarters on the campus of the Burghølzli. He had generous contact with his father's colleagues and protégés. One day Hermann Rorschach asked little Manfred if he would take the ink blots to school and record what his fellow pupils said about them. This he did.] Also distracting from the 1911 publication was protégé Carl Jung, who shared with Bleuler an interest in Freud's psychoanalysis. Only through general psychiatry texts did most people learn of Bleuler's creative work and new ideas about schizophrenia.

With my newfound thought of the "du-Sie" distinction and E. Bleuler's humble rural background likely an influence upon his son's thinking, my fire-gazing thoughts turned to Holland and a personal experience I had regarding formality and distance. A psychology colleague of strict Prussian upbringing often visited me in America from Grøningen and then Utrecht. Once he complimented me on my fluency in shifting back and forth from personal matters to formal professional matters practically from sentence to sentence. He said he was unable to do this and felt it was a handicap. I had only a weak grasp of what he was talking about and certainly was unaware of my own behavior. Later my enigma was somewhat unwoven when a postdoctoral fellow from Amsterdam worked with me. He had earlier worked with my Utrecht colleague. Once he had been invited to dinner at my colleague's Utrecht home. Toward the end of an enjoyable and congenial meal the student raised a casual question about the time scheduling of a dissertation problem. My colleague stopped him abruptly and asked him to rise from the table and go with him to his study. This was disquieting. But there the professor proceeded still cordially, although formally, to deal with the student's academic questions. The student, of Dutch country background, felt prompted later to tell me the story.

In our conversation in Zølliken Manfred explained to me that the major contact that his father had with Emil Kraepelin, aside from occasional professional meetings, was during Kraepelin's sojourn to and from his annual vacation in Italy. Manfred was a child and remembered it well. Kraepelin would arrive with an extra suitcase filled with case folders. In the evening following dinner they would lay out the folders and enter into intense discussion that lasted far into the night. The next day he would pack up the folders and continue on his journey. Then upon return from Italy the scenario would be repeated. These intense discussions apparently occurred for a number of years. Manfred described them as never acrimonious but certainly very exhilarating. It was Zubin's knowledge of these regular meetings that had prompted him to assume the "du" and to use the word friend where it did not belong.

I first met Manfred Bleuler in 1976 when I helped organize the Second Rochester International Congress on Schizophrenia. I was his host and experienced his most gentle, polite, and friendly "European" bearing. I could not reciprocate fully; I was in awe of both him and his name.

At that time honorary doctoral degrees were awarded to him, Eric Strømgren of Copenhagen, and Joseph Zubin of the Psychometrics Branch of NYSPI. As the

hooding took place I was a member of the platform party as escort to David Shakow, who had been awarded the honorary doctorate at the previous Rochester Congress. I recall Shakow telling me that his high regard for the three compelled him to come in spite of his age and health. It was probably his last major trip away from Washington. [A few years later at a schizophrenia conference at Clark University Manfred's health prevented his attendance, so he asked Joseph Zubin to read his paper for him. It indeed was the last conference Zubin attended.] Being more adventuresome than safe as a youth Manfred had suffered a fall in the Alps that created a severe deformity in his back. Extra time was needed for dressing. On stage as he was hooded he had the misapprehension that the hood contained sleeves. As the hood was dropped over his head from behind he had a small period of flailing his arms wildly. With lifelong experience with his own disability he recovered immediately with grace. Later, as I lost range of motion in my shoulders, I empathized with Manfred's movements whenever I put on a tight shirt.

My own relationship with Manfred Bleuler took a course I did not expect. I started to become aware of his rejection of formality and distance with our subsequent correspondence after the 1976 Congress and with his Christmas cards. The card each year had a printed insert giving the news of relatives and family whom I had not met, and I am sure many others received the same. Then on one occasion I sent a holiday greeting card on which was printed a lengthy poem by my brother. It was a poem about the challenges and joys of growing up on an isolated farm with no nearby friends and peers. The poem struck a major chord with Manfred, and he sent many letters discussing it. An exchange of letters even occurred between him and my brother. As Manfred explained, the poem had come at a time when his daughter, apparently also a rebel and a farmer in spirit, married a young farmer. They moved to a farm so high in the Alps that they were snowbound except during the warm summer months. In spite of Manfred's identification with his grandfather and the family farming heritage, he feared that such isolation would be detrimental to the psychological growth of his daughter and grandchildren. My brother's poem had been uniquely reassuring.

But with his repeated personal letters to me, the shoe came onto the other foot. I sometimes wished to withdraw and find the safety of distance. I could not call him Manfred or assume the "du" level of engagement. I was uncomfortably aware of being a poor country boy dealing with a great man.

…As the embers still glowed in my fire another memorable moment came back to me. When Manfred Bleuler gave his words of acknowledgement in the evening after the honorary doctorate awards, he stunned the audience by beginning, gently and sincerely,

"I am greatly honored and in awe that you should give this award to a simple country doctor."

Warmest regards, Rue

Awards 2004

Each year, the Society for Research in Psychopathology presents two awards, namely, the Joseph Zubin Award and the Smadar Levin Award.

George Brown was the 2004 recipient of the Joseph Zubin Award, given for lifetime contributions to the understanding of psychopathology.

Lea Dougherty was the 2004 recipient of the Smadar Levin Award**,** given to the graduate student or other predoctoral individual who makes the most outstanding poster presentation. Her advisor is Daniel Klein. The title of the poster was "A growth curve analysis of the course of dysthymic disorder: The effects of chronic stress and moderation by adverse parent-child relationships and family history".

"Something's Coming, Something Good…"

Psst…the 20th anniversary of our wonderful Society is fast approaching. We would like to mark the occasion by soliciting additional contributions from our membership. Are there any special memories that you would care to share? Do you have any thoughts about the future of our field? What are some of the studies that influenced you most? Please share! Contact either one of the co-editors.

New SRP Members

Our Society is growing!

Full Members: Morris Bell (Yale Univ.), Emily Durbin (Northwestern Univ.), Brandon Gibb (Binghampton University), Elizabeth Hayden (IUSM), William Horan (UCLA), Julia Kim-Cohn (Institute of Psychiatry). Thomas Lynch (Duke Univ.), Joshua Miller (Western Psychiatric Institute), Brian O'Donnell (Indiana Univ.), Diego Pizzagalli (Harvard Univ.), Yuri Rassovsky (UCLA), Jason Schiffman (Univ. of Hawaii), Mark Sergi (California State Univ.), and Jonathan Wynn (UCLA).

Associate Members: Peter Bachman (UCLA), Matt Boden (Univ. of Illinois, Champaign-Urbana), David Cicero (Univ. of Missouri), Maria Daversa (McLean Hospital), Lilian Dindo (Univ. of Iowa), Emily Haigh (Kent State Univ.), Erin Haugen (Univ. of North Dakota), Christopher Harte (McLean Hospital), Douglas Samuel (Univ. of Kentucky), Stewart Shankman (SUNY, Stony Brook), Sarah Tarbox (Univ. of Pittsburgh), and Judy Thompson (Univ. of Pittsburgh).

Welcome to all our new members!

Invited Contribution

Editor's note. Dr. Kiecolt-Glaser was one of the invited speakers at the 2004 meeting of SRP. Her talk was entitled, "Negative Emotions Can Be Deadly: New Perspectives from Psychoneuroimmunology".

Depression: A Central Pathway to Morbidity and Mortality

Janice Kiecolt-Glaser
Ohio State University

Both major depression and subthreshold depressive symptoms are associated with substantial health risks [1]. Depression can impact health through many pathways; these influences may occur through health behaviors or compliance with medical regimens, as well as through alterations in the functioning of the CNS, immune, endocrine, and cardiovascular systems. Our lab's focus is on the ways that depression contributes to morbidity and mortality through immune dysregulation. We highlight a central immunological mechanism that serves as a gateway for a range of age-associated diseases, the dysregulation of proinflammatory cytokine production, particularly interleukin 6 (IL-6).

Depression enhances the production of proinflammatory cytokines, including IL-6 [2-4]. Importantly, both depressive symptoms and syndromal depression are associated with heightened plasma IL-6 levels [2]. Following successful pharmacologic treatment, elevated IL-6 levels decline in patients with a major depression diagnosis [5]. Moreover, both physical and psychological stressors can provoke transient increases in proinflammatory cytokines [6, 7]. Thus, production of IL-6 and other proinflammatory cytokines can be directly stimulated by negative emotions and stressful experiences, providing one direct pathway.

In addition to their association with enhanced secretion of proinflammatory cytokines, depression and distress can also have direct adverse effects on a variety of other immunological mechanisms, including the down-regulation of cellular and humoral responses, and these changes are large enough to be clinically significant. For example, vaccine responses demonstrate clinically relevant alterations in immune responses to challenge under well-controlled conditions; accordingly, they serve as a proxy for response to an infectious agent [8-10]. More distressed and more anxious individuals produce immune responses to vaccines that are delayed, substantially weaker, and/or shorter-lived [8-10]; as a consequence, it is reasonable to assume these same individuals would also be slower to develop immune responses to pathogens; thus, they could be at greater risk for more severe illness. Consistent with this argument, adults who show poorer responses to vaccines also experience higher rates of clinical illness. In addition, other researchers have shown that distress can alter susceptibility to cold viruses [11]. Furthermore, distress also provokes substantial delays in wound healing [12].

Increased susceptibility to infectious disease and poorer recovery from infection are substantial and important problems; in addition, however, they carry additional risks. Repeated, chronic, or slow-resolving infections or wounds enhance secretion of proinflammatory cytokines. Thus, depression can directly affect the cells of the immune system and modulate the secretion of proinflammatory cytokines; in addition, depression

may also contribute to prolonged or chronic infections or delayed wound healing, processes that indirectly fuel proinflammatory cytokine production.

Moreover, both syndromal depression and subsyndromal depressive symptoms may sensitize the inflammatory response system such that there is heightened responsiveness to stressful events as well as pathogens. In recent work from our laboratory, participants with modest levels of depressive symptoms had higher levels of IL-6. Following influenza vaccination, individuals reporting more depressive symptoms also showed enhancement of IL-6 two weeks after receiving the vaccine, while there was little change in IL-6 among those reporting little or no symptomatology, despite the fact that the levels of depressive symptoms among the sample were quite low[4]. Our IL-6 data are consistent with other evidence of cross-sensitization between cytokines and stressors in human and animal studies[3, 7].

If there is greater cross-sensitization between cytokines and stressors in caregivers compared to controls, then we would expect greater longitudinal change in the former. To evaluate this hypothesis, our longitudinal community study assessed IL-6 production over 6 years in 119 men and women who were caregiving for a spouse with dementia and 106 noncaregivers, with a mean age at study entry of 71 for the full sample[13]. On entry into this portion of our larger longitudinal study, 28 of the caregivers' spouses had already died, and an additional 50 of the 119 spouses died during the 6 years of this study.

Caregivers' average rate of increase in IL-6 was about four times as large as that of noncaregivers. Moreover, the mean annual changes in IL-6 among former caregivers did not differ from that of current caregivers even several years after the death of the impaired spouse. There were no systematic group differences in chronic health problems, medications, or health-relevant behaviors that might have accounted for caregivers' steeper IL-6 slope.

What might be the consequences of these differences over time? Epidemiological studies of individuals 65 or older have found that the highest quartile had serum IL-6 values greater than 3.19 pg/ml[14]. As one illustration of risk, participants in the upper quartile had a 2 fold greater risk of death compared to the lowest quartile[14]. Applying this value to the data from our study[13] suggests that caregivers would on average cross that line around age 75, while controls would cross sometime after age 90. We next consider evidence which suggests that the etiology and course of a very broad range of diseases may be altered by dysregulated inflammatory responses.

Morbidity, Mortality, and Inflammatory Immune Response

Inflammation has been linked to a spectrum of conditions associated with aging, including cardiovascular disease. The association between cardiovascular disease and IL-6 is related in part to the central role that this cytokine plays in promoting the production of C-reactive protein (CRP), an important risk factor for myocardial infarction. Cardiovascular disease is the leading cause of death, and individuals with high levels of both IL-6 and CRP were 2.6 times more likely to die over a 4.6 year period than those who had low levels of both [14].

In addition to cardiovascular disease, inflammation has been linked to a spectrum of conditions associated with aging, including osteoporosis, arthritis, type 2 diabetes, certain lymphoproliferative diseases or cancers (including multiple myeloma, non-Hodgkin's lymphoma, and chronic lymphocytic leukemia), Alzheimer's disease, and

periodontal disease [15]. In fact, more globally, chronic inflammation has been suggested as one key biological mechanism that may fuel declines in physical function leading to frailty, disability, and, ultimately, death [16].

In other work elevated serum IL-6 levels predicted future disability in older adults, a finding that may reflect the effects of the cytokine on muscle atrophy, and/or to the pathophysiologic role played by the cytokine in particular diseases [17]. IL-6 and CRP also play a pathogenic role in a range of diseases associated with disability among the elderly (osteoporosis, arthritis, and congestive heart failure, among others) [17]. Thus, the clinical importance of immunological dysregulation for older adults is highlighted by increased risks across diverse conditions and diseases.

Health Behaviors

In addition to the direct influences of psychological states on physiological function, distressed individuals are more likely to have health habits that put them at greater risk, including poorer sleep, a greater propensity for alcohol and drug abuse, poorer nutrition, and less exercise, and these health behaviors have cardiovascular, immunological, and endocrinological consequences [18]. Higher plasma IL-6 and CRP levels are associated with adverse health habits: values for both are higher in smokers than nonsmokers, in individuals who report less physical activity, and in those with a higher body mass index [16, 17]. However, IL-6 has robust relationships with morbidity and mortality, even after controlling for health behaviors [16, 17].

Pharmacologic treatments hold promise. A prospective trial of statins produced reductions in CRP, providing evidence that these drugs have anti-inflammatory effects in addition to their ability to lower lipids [19]. Additionally, the use of antidepressants can normalize activation of the inflammatory response system in patients with a major depression diagnosis [5]. The question of whether cognitive treatments for depression have similar positive consequences is an important arena for future research.

Conclusions

Many lines of evidence now indicate that IL-6 may function as a "...global marker of impending deterioration in health status in older adults" (p. 645) [17]. Indeed, even after the point at which risk factors such as cholesterol, hypertension, and obesity predict health deterioration less successfully among the very old, chronic inflammation continues to be an important marker [17]. We argue that depression (both syndromal and subsyndromal) directly prompts immune dysregulation, and these processes may lead to subsequent maladaptive immune and endocrine changes [6, 7, 20]. Production of IL-6 and other proinflammatory cytokines can be directly stimulated by depression, providing one direct pathway. In addition, depression and stress may also contribute to prolonged infection or delayed wound healing, processes that fuel sustained proinflammatory cytokine production. Thus, research which addresses the dysregulation of the immune and endocrine systems associated with depression could substantially enhance our understanding of psychological influences on health, particularly among the elderly.

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Members' Corner

Remembering Phil Holzman (May 2, 1922 – June 1, 2004)

Sohee Park
Vanderbilt University

Last summer, we lost one of the founding members of the SRP. For me, it is almost impossible to dissociate the experience of attending SRP meetings from seeing Phil at all the talks and the poster sessions. I can picture him now, sitting in the front row, concentrating, sometimes with the furrowed brow, often nodding and always ready with a good question. His vast intellectual breadth and enthusiasm for science always inspired me to read more, think better, work harder and love life.

Phil (Dr. Holzman or Dr. H as we used to call him) was my teacher and advisor; the best mentor one could possibly wish for. I believe this feeling is shared by all of his students.

Recently, I was trying to understand what made somebody a truly great mentor and this is what I concluded. The signs of an outstanding, inspirational, great advisor become apparent when one examines the outcome of the weakest students rather than the best students. I came to that conclusion after having advised graduate students for a decade. It is not difficult to be an excellent advisor when the students arrive, already brilliant, focused, motivated and productive so that they just sail through graduate school. There were many such students when I was at Harvard and their advisors suffered no headaches.

Unfortunately, I was not one of these superstars. Instead, I was one of the weaker students. Phil's brilliance as a mentor lay in finding the potential in students like myself, however hypothetical and minute that potential may have been. He then fostered and nurtured our tenuous talents until they resonated, grew and turned into reality. I was intellectually unfocused with interests in so many different areas without being passionate about any of them.

Phil was able to turn this around and shape me so that I could define completely absorbing questions to tackle, develop skills to carry out the types of studies that I was imagining in my head, become independent, and eventually train and support my own students in time. He did all this by drawing out the best and the most positive qualities in

his students, and by gently pushing us with optimism, by listening to our ideas with 200% attention and interest no matter how busy or tired he was and by giving us unconditional, generous support. He wanted us to keep asking impossibly difficult questions, to be persistent and to be eternally optimistic about the power of science and the human mind.

After I graduated and moved away, I continued to ask him for advice on all aspects of my life and although he is no longer physically here, I still talk to him. When real or perceived difficulties arise or when I feel overwhelmed, I ask myself what he might have said, what he might suggest. And it helps me. So I continue my imaginary conversations with him. In this way, he is very much alive in my life.

His influence on my research obviously continues to be a big factor. In fact, a few years ago, I became very interested in the idea that self-processing may be a core problem in schizophrenia. During a casual chat with a schizophrenic patient, we learned that she did not find her face familiar. In contrast, she had no problems recognizing us or her family members or famous people like Elvis. So we began to examine at self-face recognition in schizophrenic subjects and found that many patients do not find their faces familiar although they would grudgingly recognize their faces if they are confronted explicitly. We then turned out attention to self-voice recognition. When I started to search literature, to my amazement, the last person who looked at self-voice recognition was Phil in the 1960s! I was so excited that I called him right away. We talked about his earlier voice recognition studies, which involved those old tape machines with big reels. He was delighted that someone was taking it up again and that it was one of his own students. I was very happy that I somehow stumbled upon this fascinating area and that there was this personal connection across time. He sent me all of his reprints the next day. My students and I have been working on self-processing studies ever since and the project has now branched out into questions about the nature of mental representations. Phil asked me about this self-recognition study when we met for the last time in May 2004.

In early May of 2004, Phil and I had organized a symposium dedicated to the legacy of Pat Goldman-Rakic at the Biological Psychiatry annual meeting. Pat's sudden death in the previous summer from a traffic accident had stunned us. After the symposium, we chatted a little before he headed for Boston. He encouraged me to finish the self-face and self-voice experiments and asked me about my plans for the future, about my son. Then, a quick kiss on the cheek, a friendly wave and his voice trailing behind him' "See you at SRP in October." It was not to be.

I don't understand death at all. It seems both too abstract and concrete at the same time. How does one ever come to grips with the permanent absence of someone? How does one make it tangible? It seems to me that Phil has gone on a long journey and that I continue to talk to him, the way one might write to someone far away. With my students we continue the wonderful work that started originally in his lab and it is good to see how my students branch out and find new questions and better answers. They will in turn train their own students, who will remember that Phil was at the epicenter of all this and without him, one part of the intellectual universe would have remained undiscovered, unexplored and unloved. I cherish this idea of continuity over time.

Finally, I thank Phil for the "family" that he has given us; those of us who went through his lab are forever connected to one another. But he also made sure we are integrated and embedded into the academic world. While I was still a graduate student, he introduced me to almost everybody I know now at SRP. Such a feeling of community is palpable at SRP meetings, which is, I believe, one of the reasons why SRP continues to thrive and flourish. More than a decade ago, Phil established the Smadar Levin Award in memory of his brilliant student, and with this award we celebrate young scientists every year. I hope we will be able to establish a Philip Holzman Memorial Award one day.

Kudos!!!

Congratulations to….

•Robert Krueger, University of Minnesota, who recently received the American Psychological Association Early Career Award for Achievement in Individual Differences.

•David Watson, F. Wendell Miller Professor of Psychology, University of Iowa, who is the incoming Editor of the *Journal of Abnormal Psychology*.

Newsletter Update

Beginning in Fall 2005, the Society newsletter will be published in electronic form. The plan is to send a PDF file to each member via e-mail. We will also have a copy placed on the website so that it can be downloaded.

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