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President's Column

Raymond A. Knight
Brandeis University

Some Historical Tidbits about SRP between 1993 and 2002

I inherited the position of treasurer of SRP from Elaine Walker in 1993 during the annual conference in Chicago. That year Irv Gottesman was president and Paul Meehl received the Joseph Zubin Award. It seemed quite fitting that the same year that my classmate, Paul Blaney, and I first became SRP officers, two of our mentors from our alma mater, the University of Minnesota, were being honored. I served as

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From Normal Brain and Behavior to Schizophrenia

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Technical Report CAS/CNS TR-2003-001

Editor's note. Dr. Grossberg was one of the invited speakers at the 2002 meeting of SRP.

Linking Brain to Behavior in Normals and Schizophrenics

An outstanding problem in psychology and neuroscience concerns how to link discoveries about brain mechanisms to the behaviors that they control. A related problem in psychiatry is to understand how abnormal behaviors arise from breakdowns in the brain mechanisms that govern

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Members' Corner

The Years of Silence are Past: My Father's Life with Bipolar Disorder

Stephen P. Hinshaw,
University of California, Berkeley

Foreword by Kay Redfield Jamison

In a departure from my usual academic writing on developmental psychopathology, I have written a book that focuses on my father's life. It contains, first, a narrative account of the life experiences of my father, a philosopher who

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President's Column

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treasurer for two terms, taking fiscal responsibility for the 1994 through the 1999 conferences. After my term I continued to be involved in the financial decisions for the last three conferences as well. Simply carrying out my duties during this period, I amassed a substantial amount of data about the society and its members.

Our society is neither large enough to warrant hiring an Executive Director, nor sufficiently wealthy to invest capital in nonessential activities. As a consequence, one activity that we have seriously neglected is recording our history and systematically tracking the changes and developments in our society over the years. In 2001 to address this omission the Executive Board appointed Milton Strauss to be society historian, and he began the task of gathering our records and documents into a central location and organizing them. Stuart Steinhaurer agreed to take over this task this year. The purpose of my presidential column is to make a small contribution to this newly initiated historical undertaking and to communicate to you some of the tidbits of information about SRP that I garnered during my activities as treasurer.

All of my information focuses on the last nine years. First, I will present the growth of our membership during this period. Second, I will examine the research interest pattern of our members to determine whether there have been any major shifts in these interests during this period. Finally, I will look at our conference behavior, describing who attends our conferences and how much the conferences have cost us.

Table 1 presents the annual membership of SRP from 1993 to 2001. In 1997 we introduced for the first time the Associate Membership category. The numbers represent the total number of members in good standing (i.e., dues paying) for each year for each membership category. Between 1993 and 2001 the membership increased 52.1%, from 167 to 254 members.

Figure 1 summarizes the research interests listed by members in their profile for the SRP directory and website. For the 50 members who did not fill out this section of their profile, I searched PSCYINFO for a listing of their publications and I categorized their interests by their major publications. As can be seen in the figure, five categories of research interest were coded: (a) schizophrenia (or autism); (b) mood disorders, including simply an interest in depression; (c) anxiety disorders, including interests in somatoform and dissociative disorders; (d) personality disorders, including a general interest in

Table 1
Membership in SRP between 1993 and 2001

	Year								
Membership Category	**1993**	**1994**	**1995**	**1996**	**1997**	**1998**	**1999**	**2000**	**2001**
Full Members	167	155	173	175	182	196	206	209	220
Associate Members	0	0	0	0	11	20	22	26	34
Total	167	155	173	175	193	216	228	235	254

personality and an interest in aggression; and (e) substance abuse. Because members sometimes indicated interests in two of these categories, the total percent of interests in the graph exceeds 100%. Despite considerable change in membership over these years, due both to the addition of new members and the loss of members through death, resignation, or failure to pay dues, the proportion of the membership interested in these five categories has remained amazingly constant. Across the nine years the largest standard deviation and range of percents was for interest in schizophrenia, respectively, 1.45% and 4.3%. Schizophrenia also constituted the largest interest category, M (9 years) = 58.3%, whereas substance abuse attracted the smallest interest, M = 6.5%.

Table 2 presents different registration status breakdowns of those attending our conferences. The total attendance ranges from a low of 111 for the Coral Gables, Florida conference in 1994 to a high of 237 in Cambridge, Massachusetts in 1998. On average more non-members (M = 56% of attendees) than members (M = 44%) have registered for our conferences. Only in the Boulder, Colorado conference in 2000 did members (n = 97) outnumber non-members (n = 85). Students have accounted for a large percent of our conference attendance (M = 44%). In the Madison conference in 2001 they actually outnumbered the professional registrants for the first time (53% of registrants versus 47%, respectively). Table 2 presents valuable data for projecting total conference attendance from those who have pre-registered, a perennial problem of conference coordinators. In general, most people have pre-registered (M = 75% of attendees).

Finally, Table 3 gives a gross breakdown of the costs of the last nine conferences. The site expenses include the costs of hotel space, the food, and the poster boards. The meeting expenses comprise things like the printing of the program, postage for conference announcements, secretary help, etc. The speaker, Zubin award, and Levin award expenses include the travel, food, board, and honoraria associated with each of these categories. In general, the heftiest increase has occurred in our site expenses. Between 1994 and 2002 the overall cost of a conference has risen 291%. The halcyon days of the early nineties, when we put together a conference for a pittance, are long gone, and the society

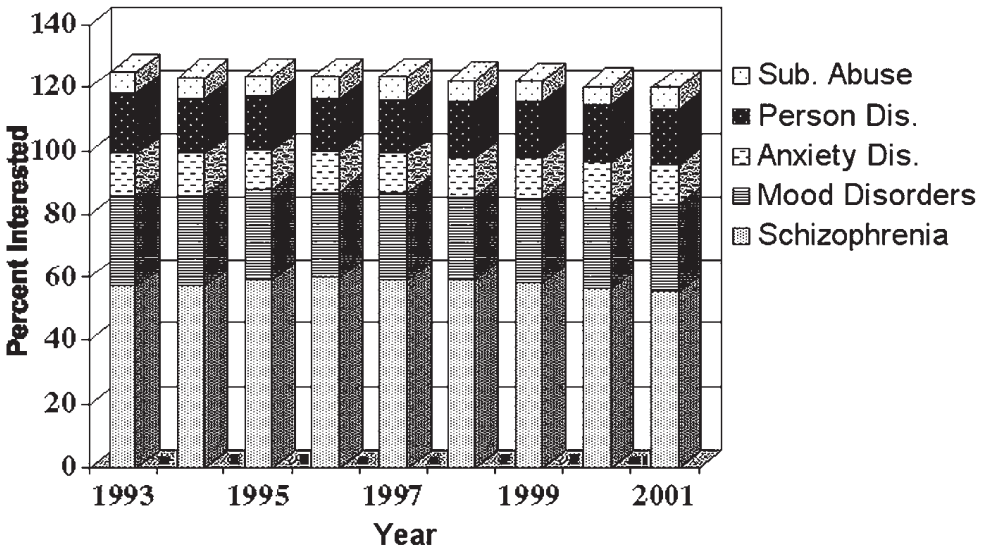

Figure 1.

Members' self-reported research interests between 1993 and 2001

Table 2

Attendance at SRP Annual Conferences between 1994 and 2001

	Year							
Registration Status	1994	1995	1996	1997	1998	1999	2000	2001
Member	54	57	69	74	94	74	97	84
Non-member	57	94	78	131	143	106	85	93
Professional	68	77	92	99	148	105	103	79
Student	43	74	55	106	89	75	79	98
Pre-registered	85	117	116	166	143	140	141	122
Registered Onsite	26	34	31	39	94	40	41	55
Total Attendance	111	151	147	205	237	180	182	177

Table 3

SRP's Annual Conference Expenses between 1994 and 2002

	Year								
Expense	1994	1995	1996	1997	1998	1999	2000	2001	2002
Site	$8,289.42	$9,222.65	$7,052.74	$14,189.95	$19,696.05	$16,913.00	$15,423.90	$25,463.00	$33,852.00
Meeting	$558.08	$481.25	$786.92	$1,720.70	$1,446.95	$701.05	$2,068.41	$1,471.30	$2,554.00
Speakers	$714.94	$772.06	$701.08	$595.86	$1,008.50	$1,313.24	$1,918.00	$1,250.00	$2,051.00
Levin	$0.00	$0.00	$0.00	$0.00	$0.00	$423.73	$230.38	$0.00	$0.00
Zubin	$494.30	$603.31	$1,034.67	$1,573.37	$0.00	$1,533.00	$0.00	$527.00	$832.00
Total	$10,056.74	$11,079.27	$9,575.41	$18,079.88	$22,151.50	$20,884.02	$19,640.69	$28,711.30	$39,289.00

has to adjust its income to match the increased costs we will continue to incur in the future.

Henry Steele Commager (1966) noted that to be ignorant of one's history is to be without a memory and both "to forfeit the rich pleasures of recollection" and to be "condemned forever to make the same discoveries that have been made in the past, invent the same techniques, wrestle with the same problems, commit the same errors." Even the tidbits presented here help us to know who we are, to recollect our past, to reflect on how we are changing, and to project where we are going.

Stephen Grossberg, Ph.D.

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normal behaviors. During the past few decades, neural models have been developed on how normal cognitive and emotional processes learn from the environment, focus attention and act upon motivationally important events, and cope with unexpected events. When arousal or volitional signals in these models are suitably altered, they give rise to symptoms that strikingly resemble negative and positive symptoms of schizophrenia, including flat affect, impoverishment of will, attentional problems, loss of a theory of mind, thought derailment, hallucinations, and delusions. These models thus suggest how an imbalance that is created in otherwise normal brain mechanisms can ramify throughout the brain to create the clinical symptoms that are observed.

Another key theme in these models is that constraints on brain development and learning greatly constrain the kinds of information processing that govern both normal and abnormal behaviors. For example, one of these models is called a CogEM model, because it joins together Cognitive, Emotional, and Motor processes (Grossberg, 1982, 1984b). The CogEM model tries to explain how emotional centers of the brain, such as the amygdala, interact with sensory and prefrontal cortices (notably ventral, or orbital, prefrontal cortex) to generate affective states, attend to motivationally salient sensory events, and elicit motivated behaviors. Closing the feedback loop between cognitive and emotional centers is predicted to generate a cognitive-emotional resonance that can support conscious awareness. When such emotional centers become depressed, negative symptoms of schizophrenia emerge in the model (Grossberg, 1984a, 2000b), as summarized below. Such emotional centers are modeled as opponent affective processes, such as fear and relief, whose response amplitude and sensitivity are calibrated by an arousal level and chemical transmitters that slowly inactivate, or habituate, in an activity-dependent way. These opponent processes exhibit an Inverted-U whereby behavior become depressed if the arousal level is chosen too large or too small. Underaroused and overaroused depression can be distinguished clinically by their parametric properties. Negative symptoms are proposed to be due to the way in which depressed affective opponent processes interact with other circuits, notably cognitive and motor circuits, throughout the brain.

A related model suggests how brain mechanisms of cognitive learning, attention, and volition work, and may give rise to positive symptoms like hallucinations during schizophrenia and other mental disorders. This Adaptive Resonance theory, or ART, model (Grossberg, 1980, 1999b) proposes an answer to the "stability-plasticity dilemma;" namely, how the brain can learn quickly throughout life without being forced to forget previously learned memories just as quickly. ART proposes how normal learning and memory may be stabilized through the use of learned top-down expectations. In other words, we are "intentional" beings so that we can learn quickly without suffering catastrophic forgetting. These expectations learn prototypes that are capable of focusing attention upon the combinations of features that comprise conscious perceptual experiences. When top-down expectations are active in a priming situation in the absence of bottom-up information, they can modulate or sensitize their target cells to respond more effectively to

future bottom-up information that matches the prototype. Such expectations cannot, however, fully activate these target cells under most circumstances. When bottom-up inputs do occur, an active top-down expectation selects the cells whose input features are consistent with the active prototype, and suppresses those that are not. This matching process can synchronize and amplify the activities of selected cells. Such a matching process has been mathematically proved to be necessary to stabilize the memory of learned representations in response to a complex input environment (e.g., Carpenter and Grossberg, 1991). In order to realize these matching properties, top-down expectations and attention were predicted to be controlled by top-down on-center off-surround networks. A balance between top-down excitation and inhibition in the on-center of this network leads to the modulatory effect in the on-center on its target cells, even while cells that are in the off-surround may be strongly inhibited. Recent psychophysical and neurophysiological data have supported this prediction; see Raizada and Grossberg (2003) for a review.

The ART model proposes how the brain has exploited the modulatory property of expectations and attention to enable fantasy, imagery, and planning activities to occur. In particular, phasic volitional signals can shift the balance between excitation and inhibition to favor net excitatory activation when a top-down expectation is active. Such a volitionally-mediated shift enables top-down expectations, in the absence of supportive bottom-up inputs, to cause conscious experiences of imagery and inner speech, and thereby to enable fantasy and planning activities to occur. If, however, these volitional signals become tonically hyperactive during a mental disorder, the top-down expectations can give rise to conscious experiences in the absence of bottom-up inputs and volition. Many data about schizophrenic hallucinations can be clarified by these model properties (Grossberg, 2000a). Related work has predicted the detailed laminar circuits within the visual cortex wherein these top-down expectations and volitional signals may act, and by extension in other sensory and cognitive neocortical areas (Grossberg, 1999a, Grossberg and Raizada, 2000; Raizada and Grossberg, 2003). ART also predicts that the contents and level of abstractness of learned prototypes may determine the contents and abstractness of hallucinations. A similar breakdown of volition may lead to delusions of control in the motor system.

Attention, Affect, and Volition in Schizophrenia

These CogEM and ART models bring a new perspective to thinking about the well-known fact that schizophrenia involves a loss of attentional control, motivational defects, and disorganized behavior. Kraepelin (1913/1919) early noted that "This behavior is without doubt clearly related to the disorder of attention which we very frequently find conspicuously developed in our patients. It is quite common for them to lose both inclination and ability on their own initiative to keep their attention fixed for any length of time" (pp. 5-6). Attentional deficits in schizophrenia have also been emphasized by a number of other workers; e.g., Bleuler (1911/1950), Braff (1985) and Mirsky (1969).

Since the time of Kraepelin, many efforts have been made to classify schizophrenic symptoms across distinct patient populations, including the basic classifications into negative and positive symptoms, or deficit and

nondeficit symptoms (Buchanan et al., 1997; Bustillo et al., 1997). Liddle (1994) has segregated schizophrenic symptoms into "three distinguishable syndromes: (1) psychomotor poverty (poverty of speech, flat affect, decreased spontaneous movement); (2) disorganisation (disorders of the form of thought, inappropriate affect); and (3) reality distortion (delusions and hallucinations)" (p. 43), which have been supported by several studies (Arndt et al., 1991; Pantelis et al., 1991; Sauer et al., 1991). Liddle suggested that two of these syndromes "reflect volitional disorders: psychomotor poverty reflects a difficulty initiating activity and disorganisation reflects a difficulty in the selection of appropriate activity" (p, 43). Both of these problems are, moreover, associated with impairment in neuropsychological tests of frontal lobe function.

In a different direction, Frith (1992, 1994) has interpreted schizophrenic symptoms as impairments in the processes that underlie a "theory of mind", including the ability to represent beliefs and intentions. For example, when asked to describe photographs of people, schizophrenics described their physical appearance, rather than their mental states (Pilowsky and Bassett, 1980). Frith noted, however, that the theory of mind approach "does not explain the other major feature of negative schizophrenia: their impoverishment of will." (Frith, 1994, p. 150). He also wrote that "mental states include not only affects and emotions, but also goals and intentions. A person who was unaware of their goals could, on the one hand, be a slave to every environmental influence or, on the other hand, be prone to perseverative or stereotyped behaviour, because they would not have the insight to recognize that certain goals were unobtainable or inappropriate" (Frith, 1994, p. 151).

All of these properties have explanations using CogEM and ART. In particular, these models analyze how attention is regulated during normal cognitive and cognitive-emotional interactions, and how it breaks down when these normal processes experience some sort of imbalance. Such models point to processes that have not been as actively considered as they might be towards explaining schizophrenic behavioral symptoms.

Gated Dipole Opponent Processing

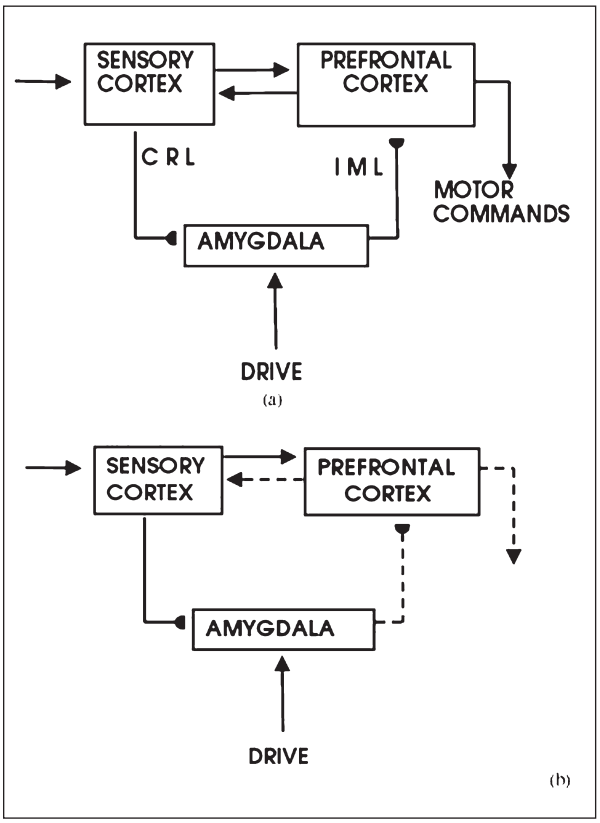
One such process is opponent processing, whether of opponent emotions, like fear and relief, or of opponent perceptual features, like red and green. Opponent processing plays a key role in controlling the dynamical reset and rebalancing of sensory, cognitive, emotional, and motoric representations in response to rapidly changing environmental inputs. Such opponent processing circuits exhibit a Golden Mean of optimal behavior at an intermediate arousal level (Grossberg, 1972, 1980, 1984a, 1984b). For larger or smaller levels of arousal, behavior deteriorates in different ways, thereby giving rise to an Inverted-U as a function of arousal level. In particular, when arousal is too small, such an opponent process causes an elevated behavioral threshold, since there is not enough arousal to support a more normal threshold. Paradoxically, it also gives rise to behavioral hyperexcitability when this elevated threshold is exceeded. When arousal is too large, the opponent process causes a low behavioral threshold. Paradoxically, it also gives rise to behavioral hypoexcitability when this reduced threshold is exceeded. Due to these properties, an increase in arousal can decrease the sensitivity of an

underaroused opponent process of this kind, and can bring it into the normal behavioral range. The model proposes that, in this way, a pharmacological "up" like amphetamine can reduce the hypersensitivity of attention deficit disorder children. These properties emerge through interactions across the entire opponent processing circuit. They cannot be understood just by looking at the pharmacology or neurophysiology of individual cells within the circuit. How such opponent processes work during normal behavior and schizophrenia is described in Grossberg (1984a, 1984b, 2000b). When their output signals become depressed, such opponent processes are predicted to lead to various symptoms of flat affect. When their effects ramify throughout the sensory and prefrontal cortices with which they interact, they can lead to all the negative symptoms that are summarized above.

Negative Symptoms as Emergent Properties of System-Wide Interactions

The most immediate effect of a depressed response in the outputs of emotion-representing areas is flat affect, although how this is understood must be carefully evaluated; see below. This defect, in turn, causes an inability to represent others' beliefs and intentions, in the sense that all mental states that depend upon interpreting one's own emotional state, or the emotional states of others, will be diminished. This happens in the CogEM model because emotionally charged sensory inputs, such as the emotional expressions on other people's faces, will activate the appropriate part of inferotemporal cortex but will not elicit an appropriate emotional response from the amygdala and related emotion-representing circuits; see Figure 1. As a result, photos of people would necessarily be described physically, rather than in terms of emotionally relevant mental states (Pilowsky and Bassett, 1980).

Figure 1



A problem with impoverishment of will, as well as with the setting of goals and intentions, will then indirectly arise. This happens in the model because the depressed response of the emotional representations depresses the incentive motivational signals that would normally activate the prefrontal cortex in response to motivationally salient events (Figure 1). As a result, the prefrontal cortex will not be adequately activated, and a hypofrontal condition will emerge (Weinberger, 1988). Due to this hypofrontality, the working memory representations and plans that are ordinarily formed within the prefrontal cortex will be degraded, so goals will not form in a normal fashion.

Given a hypofrontal response, top-down signals from the prefrontal cortex to the sensory cortices will also be reduced or eliminated (Figure 1). As a result, the sensory representations will not be able to use these top-down signals to organize information-processing according to its emotional meaning or motivational goals. Said in another way, motivationally irrelevant information will not be blocked from attention, so it will be able to continually intrude, leading to distractability. Or, in Kraepelin's words, schizophrenics "lose both inclination and ability on their own initiative to keep their attention fixed for any length of time."

Neurobiological Correlates

The CogEM model also clarifies the following types of anatomical, neurophysiological, and biochemical data. The amygdala, and related structures, has been identified in both animals and humans to be a brain region that is involved in learning and eliciting memories of experiences with strong emotional significance (Aggleton, 1993; Davis, 1994; Gloor et al., 1982; Halgren et al., 1978; LeDoux, 1993). The orbitofrontal cortex is known to be a major projection area of the ventral, or object-processing, cortical visual stream (Barbas, 1995; Fulton, 1950; Fuster, 1989; Rolls, 1998; Wilson et al., 1993), and cells in the orbitofrontal cortex are sensitive to the reward associations of sensory cues, as well as to how satiated the corresponding drive is at any time (e.g., Mishkin and Aggleton, 1981; Rolls, 1998). Ubiquitous positive feedback occurs between cortical regions (Felleman and Van Essen, 1991; Macchi and Rinvik, 1976; Sillito et al., 1994; Tsumoto, Creutzfeldt, and Legéndy, 1978), including prefrontal and sensory cortices. In addition, the ventral prefrontal cortex and the amygdala are involved in the process by which responses are selected on the basis of their emotional valence and success in achieving rewards (Damasio et al., 1991; Passingham, 1997). In particular, Fuster (1989) has concluded from studies of monkeys that the orbital prefrontal cortex helps to suppress inappropriate responses. These monkey data are consistent with clinical evidence that patients with injury to orbital prefrontal cortex tend to behave in an inappropriate manner (Blumer and Benson, 1975; Liddle, 1994). Other research has suggested that schizophrenia may involve a chronic deficiency in striatal glutamate transmission due to decreased activity in those regions of the prefrontal cortex that project to the striatum (Andreasen, 1990; Carlsson, 1988; Grace, 1991; Lynch, 1992). The CogEM model suggests that one possible cause of decreased prefrontal activity may be a reduction in incentive motivational signals from depressed amygdala circuits that project to the prefrontal cortex.

Some Open Questions and the Need For Quantitative Brain/Behavior Models

A brief verbal summary such as I have just attempted leaves out so many details that it is subject to misinterpretation. In the case of the CogEM and ART models, whatever be their shortcomings, they offer a precise mechanistic explanation of how interactions among model brain mechanisms give rise to normal and abnormal behavioral properties. The same is not true of intuitive and heuristic attempts to explain schizophrenic symptoms which, albeit necessary to advance our understanding, are inherently too weak to unambiguously bridge the gap between brain and behavior. The discussion above raises a number of questions when it is confronted by

various recent data. For example, it has been proposed that some schizophrenics who exhibit symptoms of flat affect may experience more intense emotions than ordinarily supposed, particularly negative emotions, and that flat affect is due to the fact that their observable responses are reduced (e.g., Alpert et al. 2000). The essential property for explaining the effects of flat affect in the CogEM model is that a late stage in emotional processing, one that feeds incentive motivational signals to the prefrontal cortex, is depressed and thereby negatively impacts sensory, cognitive, and motoric processing. Earlier stages of emotional processing may be intact without disrupting model predictions. This being said, questions remain about how some of these patients were tested — in particular, using verbal stories about emotional situations is not necessarily a reliable way to assess experienced emotion.

It has been suggested that flat affect may not be a primary symptom of schizophrenia because flat affect and hallucinations do not always covary (Serper et al., 1996). The CogEM and ART models clarify, however, that these two types of symptoms may be due to distinct brain mechanisms. It has also been suggested that the early appearance of flat affect, before schizophrenic symptoms occur, and the fact that it is sometimes not followed by such symptoms (Alpert, 1985), may suggest that it is only a "risk factor" for schizophrenia (Alpert and Angrist, 2003). Given that there are many reasons why an early symptom may not immediately lead to a fully blown syndrome, one might just as well wonder if the early onset does not provide some evidence that it can be a cause. For example, autoreceptors can, in various brain systems, maintain a robust system response until such a large loss is experienced that they can no longer compensate for it. Such compensatory effects may be occurring in, say, the amygdala-to-prefrontal pathways. Experiments to study this and related pathways more closely would be most valuable towards clarifying this issue.

More generally this article points to how well-known psychological processes such as affective opponent processing, top-down expectations, incentive motivation, volitional gating, and attentional blocking may break down in schizophrenics. Neural models like CogEM and ART, by describing these processes clearly and quantitatively, may make it easier to think about and test their implications when they are subjected to one or another kind of imbalance.

GRANTS AND ACKNOWLEDGMENTS

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MEMBERS' CORNER

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suffered from lifelong (and for the most part misdiagnosed) bipolar disorder. Second, it is a portrayal of my own reactions as a son growing up in silence about his episodes—chiefly because the doctors warned my father never to discuss mental disorder with his children—only to have them revealed once I reached young adulthood and my father began to disclose his life's story to me. Third,

the book contains commentary about key issues related to bipolar disorder in particular and serious mental disorder in general: diagnosis and misdiagnosis, causes and treatments, resilience and courage, family silence and family discussion regarding mental disorder, and the relationship between the inner symptoms of mental disorder and the outer social and political world. I believe that the book provides a unique perspective on the field, joining a growing of number personal and family accounts of serious mental illness but adding the perspectives of a son who is a clinical scientist.

Briefly: My father, Virgil Hinshaw Jr., was born in 1919 just outside Chicago, the fourth of four boys. The extended family contained many individuals with high achievement and others with serious mental conditions. He lost his mother at age 3 to cancer, and his father (who was Chairman of the Prohibition National Committee) moved the family out West. His father remarried, and Virgil soon gained two half-brothers. His step-mother, however, began a series of harsh punishments of Virgil, which gave him a sense that he was deserving of cruel treatments in hospitals in the years to come. The Great Depression began when he was 10 years of age.

At age 16, in 1936, Virgil began to become agitated in the late summer, preoccupied with thoughts of Hitler and the potential for worldwide fascism. Increasingly irrational, he jumped from the porch roof of the family home, believing both that he could fly and that he could stop Hitler through his act. Thus began his first, psychotic-proportion, manic episode, which lasted 6 months and took him to near death in a county facility (he believed that the food was poisoned). His diagnosis was schizophrenia, as was typical for any American patients with psychotic features during most of the 20th Century.

Upon his release, he completed 11th grade with a straight A average, subsequently attending Stanford and Princeton, studying with such luminaries as Russell and Einstein and earning a doctorate in philosophy. Yet other episodes ensued, the most severe of which occurred during the 1950's, when Virgil was a young assistant professor at Ohio State and a father of two young children—my sister and me. He received numerous ECT's and was on maintenance neuroleptics for over 20 years, still carrying a diagnosis of schizophrenia despite his rapid recoveries and prolonged periods of euthymia between episodes. His life thus alternated between high achievement and crushing periods of illness.

Despite prolonged absences for hospitalization, Virgil maintained his teaching job and was a sensitive and loving father. His eventual rediagnosis and treatment with lithium, however, was a case of "too little, too late," given that he had begun a slow decline in his final years, losing much of his cognitive prowess.

To portray my father's life, I use material gained from the discussions he had with his father as well as his journals, letters from family members, and discussions with my mother, who stayed by Virgil despite an almost complete lack of support from doctors and society at large. My hope, overall, is to humanize serious psychopathology.

Near the time of my father's death in 1995, I began to write drafts of various portions of his life, obtaining his permission to do so during the last talks I ever had with him. Yet, it took a number of years of work to put

the many portions of his story into place and to find a voice for conveying my own reactions to learning of my father's condition. These reactions include a tendency to internalize silence, a strong need for control, and a series of worries about my own mental stability and about whether to have children. Yet through the writing, I have opened myself to an appreciation of the dual need for (a) first-rate science and (b) cogent personal accounts and disclosures, if a better means of helping those with psychopathology is to emerge. The book is intended for both a clinical/scientific and a general audience. It may be particularly beneficial for students and trainees in the field. Currently available in hardcover, the paperback edition is scheduled for release in early 2004. The work could serve as a valuable supplement for courses in clinical psychology, abnormal psychology, or psychopathology.

*From the foreward by Kay Jamison: "Hinshaw has written a powerful account of what it is to be the son of a man who lost not only his way, but his mind as well...he conveys his father's struggles with clear-eyed compassion and describes vividly the complexities of their relationship...he has written a compelling book about fathers and sons, madness, and the intolerance of society and the academic and medical communities."

*From Dante Cicchetti: "...an astounding work...an important contribution to breaking down the barriers caused by stigmatizing mental disorder. It is very rare to find a scientist-clinician capable of producing a work that reflects this breadth...The book has the makings of a classic in the field."

*From Sir Micheal Rutter: "...This splendid book is positive and definite in what it says, but is appropriately cautious and well-balanced...thought-provoking."

*From Norman Endler: "...of significant value to sufferers of bipolar disorder and their families as it offers a new understanding of the illness."

Cambridge University Press, 2002; ISBN 0-521-81780-3; $25.00

KUDOS!!!

Hearty congratulations to:

- Dr. Philip S. Holzman, for receiving the American Psychological Foundation's Alexander Gralnick Research Investigator Award. See the nice article written about him in the January 2003 issue of the APA's Monitor on Psychology.

- Drs. Lyn Abramson and Lauren Alloy, for being named joint recipients of APA Division 12 Distinguished Scientific Contribution Award.

- Drs. Donald R. Lynam and Deanna M. Barch for being awarded the Distinguished Scientific Early Career Contributions to Psychology in psychopathology.

SRP2002 in Review

Observations and Reflections on the 2002 Meeting of the Society for Research in Psychopathology

Steven M. Silverstein
(2002 meeting Program Chair)
Weill Medical College of Cornell University

The 17th annual meeting of the Society for Research in Psychopathology was held in San Francisco from September 26th-29th, 2002, at the Hyatt, Fisherman's Wharf. As with all past SRP meetings, this one continued a number of longstanding SRP traditions (in terms of format, quality of presentations, etc.), and also had several highlights. It is the latter I will focus on in this column. First though, I would like to thank my fellow Program Committee members (Dara Manoach, David Miklowitz, and Dan Klein) for their time and hard work in helping to prepare this year's program. A very special thank you also goes to Ann Kring for coordinating all of the local arrangements.

One of the goals of the Program Committee for this meeting was to choose paper presentations that went beyond descriptions of specific studies. Specifically, a goal was to have papers that explored, clarified, or challenged the ways we think about and conduct psychopathology research. For example, one of the biggest challenges facing SRP researchers today involves how to bridge the gap between the signs and symptoms of psychopathology and their underlying biological mechanisms. This challenging issue was addressed by the two invited speakers. Barbara Knowlton described the functions of the basal ganglia, and how impairments in these structures can lead to a variety of psychological and behavioral deficits. This research provides a bridge between biological mechanisms and overt psychopathology that is critical for the development of more realistic, and therefore powerful, theories of psychopathology. Steven Grossberg addressed the biology-psychopathology relationship in a different, but equally fascinating manner. Beginning with concepts from classical conditioning, and moving through neural network theory and known aspects of cortical functioning, he demonstrated how a comprehensive model of schizophrenic psychopathology can be developed, and what such a model should address. Taken together, these two talks highlighted the importance of conceptualizing psychopathology on multiple levels, and of moving beyond correlational or isomorphic approaches towards the development of models that are mutually constrained by both biology and behavior and that can generate predictions at multiple levels of analysis.

As if to leave no doubt about the importance of bridging the laboratory-clinic divide, Robert Heinssen of NIMH gave a lunchtime presentation on NIMH's Translational Research Initiative. In his talk, he highlighted the benefits of a research approach that incorporates laboratory and clinical findings. To highlight the importance of this area further, he mentioned that NIMH will be increasingly looking for, and expecting, translational efforts in grant applications in years to come. This news was digested well by most audience members.

Just as viewing psychopathology from multiple levels can help us develop more realistic views of our subject matter, the use of more sophisticated statistical methods can provide us with a more veridical assessment of the

effects of our independent variables. The issues of the limitations imposed by traditional statistical methods, and the advantages of alternative ones were discussed in two presentations. One, by Jim Neufeld, demonstrated how formal stochastic modeling methods can be used to develop individual performance profiles in cognitive psychopathology research. This is an important topic because cognitive psychopathology research typically looks at group effects only, whereas it is increasingly being recognized, in both laboratory and treatment research, that modeling at the level of individual patient performance first, and then using the resulting parameters as independent variables, can lead to the identification of more homogeneous, and clinically meaningful, subgroups. The other paper tackling methodological issues was delivered by Eric Turkheimer. He convincingly demonstrated that non-linear and non-parametric classification and regression methods can enhance our understanding of real-world behavioral data. This was a stimulating and challenging talk that posed a challenge to the audience as to whether to adopt these new methods, as opposed to continuing to rely on the widely accepted, but severely limited, and outdated methods involving null hypothesis significance testing.

Two special highlights of the conference included the Presidential and Zubin Award addresses. In both cases, the audience was treated to career-long reviews of research challenges and triumphs by leading thinkers in psychopathology research. For his presidential address, Ray Knight led the audience through a series of elegant attempts to model heterogeneity among sexual offenders. This talk itself was a model of how to approach science from the Popperian perspective. That is, in addition to demonstrating the positive results that can be gained from elegant research designs and the use of advanced statistical methods, Ray demonstrated how the search for, and openness to, evidence that disconfirms one's hypotheses, can be just as, if not more influential in the development of a research program than finding evidence that supports one's theories. In his Zubin Award address, Rue Cromwell reviewed a number of challenges in schizophrenia research faced by earlier workers in the field, how he and others addressed those challenges, and how their findings have influenced current thinking about schizophrenia. In a sincere and informative set of introductory remarks to the Zubin Award address, Will Spaulding (a former Cromwell graduate student), made it clear how many current concepts in schizophrenia research were anticipated by Rue's earlier work.

In addition to the presentations mentioned above, there were a number of other excellent single papers and symposia covering topics such as cognitive deficits in schizophrenia and other developmental disorders, potential risk markers for mood disorders, psychopathy, life stress, intervention research, and childhood mood disorders. This wide variety of important topics speaks to the current depth, as well as the breadth, of SRP membership, the latter being a goal towards which the organization has been working for several years.

No discussion of an SRP meeting would be complete without mention of the poster sessions. This year's high poster total (N=106) reflected well on new membership and member and student attendance. As with the paper presentations, the posters covered a wide range of topics from methodology to

cognitive and biological mechanisms underlying personality, anxiety, mood, and psychotic disorders. All of this increased the difficulty of the work of the Smadar Levin Committee [thanks go to Stuart Steinhauer (chair), Sheri Johnson and Chris Patrick], who had to choose the outstanding poster by a student. Due to the consistent excellence of the posters, two runners up (Patricia DiParsia and Junghee Lee) were chosen in addition to the winner, Brian Hicks.

In short, this year's meeting continued the tradition of high quality research presentations that has become the hallmark of SRP, and presented some challenges to the future of psychopathology research. It will be interesting to see, in the years to come, how SRP members respond to the conceptual, methodological, and NIMH imposed challenges presented at this year's meeting.

New SRP Members

Full Members: John Forsyth (SUNY-Albany), David Fresco (Kent State Univ.), Pamela Keel (Harvard Univ.), Bjorn Meyer (Louisiana State University), Tonmoy Sharma (Maudsley Hospital, London), Scott Sponheim (Minneapolis VA Medical Center), Edelyn Verona (Kent State Univ.), Amy Weisman (Univ. of Miami), and Amy Wenzel (Univ. of North Dakota).

Associate Members: Eva Levine (New York), and Kevin Wu (Univ. of Iowa).

Welcome!

A Note from the Editor.

Thank you to all of you who sent me feedback on the previous issue. Please join me in welcoming Craig Neuman as the co-editor of the newsletter. Please feel free to submit ideas for commentaries, articles, and feedback regarding the issue. Also, we are considering publishing the newsletter in electronic format only. Another option is to continue to mail out the newsletter and to post past newsletters on the Internet; this may serve to attract additional members. We'd welcome your feedback about these options.

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