

INFORMATION TO USERS

This manuscript has been reproduced from the microfilm master. UMI films the text directly from the original or copy submitted. Thus, some thesis and dissertation copies are in typewriter face, while others may be from any type of computer printer.

The quality of this reproduction is dependent upon the quality of the copy submitted. Broken or indistinct print, colored or poor quality illustrations and photographs, print bleedthrough, substandard margins, and improper alignment can adversely affect reproduction.

In the unlikely event that the author did not send UMI a complete manuscript and there are missing pages, these will be noted. Also, if unauthorized copyright material had to be removed, a note will indicate the deletion.

Oversize materials (e.g., maps, drawings, charts) are reproduced by sectioning the original, beginning at the upper left-hand corner and continuing from left to right in equal sections with small overlaps. Each original is also photographed in one exposure and is included in reduced form at the back of the book.

Photographs included in the original manuscript have been reproduced xerographically in this copy. Higher quality 6" x 9" black and white photographic prints are available for any photographs or illustrations appearing in this copy for an additional charge. Contact UMI directly to order.

UMI

**A Bell & Howell Information Company
300 North Zeeb Road, Ann Arbor MI 48106-1346 USA
313/761-4700 800/521-0600**

PREVIEW

**DISSOCIATION: THE ROLE OF COGNITIVE INHIBITION
IN A NEW EMPIRICAL FRAMEWORK**

by

Paul Freeman

A DISSERTATION

Presented to the Faculty of

The Graduate College at the University of Nebraska

In Partial Fulfillment of Requirements

For the Degree of Doctor of Philosophy

Major: Psychology

Under the Supervision of Professor William D. Spaulding

Lincoln, Nebraska

December, 1997

UMI Number: 9815886

UMI Microform 9815886
Copyright 1998, by UMI Company. All rights reserved.

**This microform edition is protected against unauthorized
copying under Title 17, United States Code.**

UMI
300 North Zeeb Road
Ann Arbor, MI 48103

DISSERTATION TITLE

Dissociation: The Role of Cognitive Inhibition in a New Empirical Framework

BY

Paul Freeman

SUPERVISORY COMMITTEE:

APPROVED

DATE

Signature

William Spaulding
William Spaulding, Ph.D.

Typed Name

12/9/97

Signature

Cal Garbin
Cal Garbin, Ph.D.

Typed Name

12-9-97

Signature

Debra Hope
Debra Hope, Ph.D.

Typed Name

12/9-97

Signature

John Flowers
John Flowers, Ph.D.

Typed Name

12/9/97

Signature

Gargi Roysircar Sodowsky
Gargi Roysircar Sodowsky, Ph.D.

Typed Name

12/9/97

Signature

Typed Name



GRADUATE COLLEGE
UNIVERSITY OF NEBRASKA

DISSOCIATION: THE ROLE OF COGNITIVE INHIBITION IN A NEW EMPIRICAL FRAMEWORK

Paul Freeman, Ph.D.

University of Nebraska, 1997

Adviser: William D. Spaulding

Forty-nine clinically diagnosed subjects were administered the *Dissociative Experiences Scale* (a self-report measure of disturbances in memory, identity, awareness, and thought) and a computerized negative priming task (a reaction-time measure of the extent to which distracting visual information is inhibited from access to verbal report). Both between-group and correlational comparisons demonstrated significant relationships between a “high” frequency of reported dissociative experiences and a reduced ability to inhibit the emotional identification of threat words. “High” dissociators also demonstrated a superior ability to inhibit the location of threat words and to inhibit the identification of neutral words. These effects confirmed three of four hypotheses derived from a theoretical model of dissociation emphasizing the role of *cognitive inhibition* as “the mechanism which controls and limits the contents of consciousness” (Frith, 1979, p. 225). The finding of superior inhibition for the identification of neutral words was not explicitly predicted, but can be assimilated within the proposed model. Results are interpreted as providing supportive evidence for cognitive inhibition as a mechanism underlying both the “intrusive” (described as abrupt

shifts in thought, behavior, and perceived self-identity) and “avoidant” (described by the ability to ignore painful experiences) aspects of dissociation. These findings are seen as providing an empirical framework for the investigation of dissociative processes, while having important implications for the assessment and treatment of dissociative symptoms in psychiatric disorders.

PREVIEW

CONTENTS

A Theoretical Model

Introduction	1
An overview	2
Cognitive Inhibition of Consciousness	6
Negative Priming	6
Failed Inhibition: A Disorder of Consciousness?	10
Individual Differences in Cognitive Inhibition	13
Dissociation and Reduced Cognitive Inhibition	16
Perceptual Distortions	18
Intrusion of Suppressed Thoughts	19
Distractibility in Response to Stress	21
Focused Absorption	24
The Dissociation of Conscious Perception	25
Relevant and Irrelevant Information Processing	26
Distraction as a “Switch Process”	28
Absorption as Focused Distraction	31
Dissociation as Automatized Distraction	31
Reduced Inhibition: A Vulnerability Factor?	34
Reduced Inhibition & Stress: A Lack of Response Factor	35
A neurobiological hypothesis	38
A response “uncoupling” hypothesis	39
Schema: The Dissociative Role of Inhibition	40
Schema and Dissociative Symptoms	41
Dissociative responses as trauma-induced schema	41
Intrusions of schema due to reduced inhibition	43
Schematic intrusions: Irrelevant dissociative responses	45
Dissociative Symptomatology: Enhanced or Reduced Inhibition?	46
Paradoxical Effects of Reduced Inhibition	47
Patterns of Inhibition Across Functional Systems	48
The Dissociative Disorders: Differential Patterns of Inhibition?	50
Implications for Assessment & Treatment of Dissociative Symptoms	51
Relevance to Issues of Assessment	51
Assessment of precipitating factors	53
Relevance to Treatment Issues	54
Summary and Testable Hypotheses	55
Operationalized Hypotheses	56
Hypothesis 1	56
Hypotheses 2 & 3	57
Hypothesis 4	59
Appended Supplemental Analyses	60

Method

Subjects	62
Measures	62
WAIS-R Vocabulary Subtest	62
Dissociative Experiences Scale (DES-II)	63
DES: Reliability and validity studies	65
Negative Priming Task	67
Apparatus	69
Stimuli: Trial displays	69
Stimuli: Neutral and threat words	70
Stimulus sequencing: Prime-probe paired trials	72
Negative Priming Task Design	74
(1) Neutral control condition	74
(2) Neutral identity condition	74
(3) Neutral location condition	75
(4) Neutral identity-and-location combined condition	75
(5) Threat control condition	75
(6) Threat identity condition	75
(7) Threat location condition	75
(8) Threat identity-and-location combined condition	76
(9) Mixed context: Neutral identity	76
(10) Mixed context: Threat identity effect	76
Calculation of Negative Priming Effects	76
Calculation of priming effects for neutral conditions	76
Alternative control conditions for threat effects	77
Calculation of priming effects for threat conditions	78
General Procedure	79
Negative Priming Task Procedure	79

Results

Data Exclusion	81
Error Rates	82
Outliers & Assumptions of Normality	83
Demographics	85
Preliminary Analyses	87
Mean Reaction Times for Task Conditions	87
Negative Priming Effects	89
Between-Group Comparisons	92
Assessing Group Characteristics	93
Between-Group Comparisons	97
Assessing for a potential confound	103

Discussion

Hypothesis Findings	105
Hypothesis 1	105
Hypothesis 2	107
Hypothesis 3	108
Hypothesis 4	109
Implications of Findings for the Proposed Theoretical Model	110
Findings as Applicable for Emotional Responses	110
Threat identity effects as modeling “emotional triggers”	111
Unexpected Findings: Differential Patterns of Inhibition	113
Enhanced Inhibition as a Mechanism of Dissociative Avoidance	114
Dissociative Avoidance: Differential Aspects of Inhibition	116
Conclusions	118

References

References and Bibliography	122
--	-----

Appendices

Appendix A: The Dissociative Experiences Scale	186
Appendix B: Negative Priming Task Conditions	192
Appendix C: Word Stimuli and Frequencies	197
Appendix D: Supplemental Analyses	198
Correlational analyses	199
Multiple regression analyses	201
Discriminant analyses	204
Evaluating for within-category facilitation	208
Individual differences in task performance	210
Factor analysis of the <i>DES-II</i>	212

Illustrations

Figure 1	Trial stimulus display	70
Figure 2	Prime-probe paired trials for the identity condition	73
Figure 3	Reaction Time Across Task Conditions	88
Figure 4	Negative Priming Effects across Task Conditions	90
Figure 5	“Low” & “High” Dissociators: Neutral Level	98
Figure 6	“Low” & “High” Dissociators: Threat Level	98

Tables

Table 1(a)	Conditions of Presentation: Distribution Statistics	84
Table 1(b)	Calculated Priming Effects: Distribution Statistics	85
Table 1(c)	DES-II Scores: Distribution Statistics	85
Table 2(a)	Demographic Descriptive Statistics	85
Table 2(b)	Gender Frequencies	86
Table 2(c)	Ethnicity Frequencies	86
Table 2(d)	Axis I Diagnosis (Frequencies)	86
Table 2(e)	Current Psychiatric Medications (Frequencies)	86
Table 3	Mean Reaction Times for Task Conditions	88
Table 4	Negative Priming Effects for Task Conditions	89
Table 5	DES-II Scores: Descriptive Statistics	93
Table 6	“Low” & “High” Dissociators: Demographic Differences	94
Table 7	“Low” & “High” Dissociators: Medication Frequencies	95
Table 8	“Low” & “High” Dissociators: Axis I Frequencies	96
Table 9	“Low” & “High” Dissociators: Comparisons by Condition	97
Table 10	Overall 2 x 2 x 3 Mixed ANOVA	99
Table 11	Pearson Correlations	199
Table 12	Multiple Regression Model: 6 Priming Effects Entered ...	201
Table 13	Multiple Regression Model: Subject Characteristics	202
Table 14	Multiple Regression Model: Characteristics & Effects	203
Table 15	Discriminant Analysis Model	205
Table 16	Multiple Regression Model: Predictive Discriminators ..	207
Table 17	Individual Differences: “Inhibitors” & “Facilitators”	210
Table 18	DES-II Factorial Analysis: Total Variance Explained ...	212
Table 19	DES-II Factorial Analysis: Rotated Component Matrix ...	213
Table 20	DES-II Forced 4-Factor Model: Total Variance Explained	215
Table 21	DES-II Forced 4-Factor Model: Component Matrix	216
Table 22	Factor 1 (in 7-factor model); Behavioral Shifts	217
Table 23	Factor 4 (in 4-factor model); Behavioral Shifts	217
Table 24	Factor 7 (in 7-factor model); Ability to Ignore Pain	218
Table 25	Factor 3 (in 4-factor model); Distractive Absorption	218

Dissociation:
The Role of Cognitive Inhibition
in a New Empirical Framework

To the surprise of many, several representative surveys have found that at least ten per cent of the clinical population in typical treatment settings suffer from a diagnosable dissociative disorder (Graves, 1989; Ross, 1991). Although the dissociative disorders have been diagnostically described by the American Psychiatric Association (1994) as “a disruption in the usually integrated functions of consciousness, memory, or perception of the environment” (p. 477), a precise and useful definition of dissociation remains elusive. At a recent conference on dissociation, leading experts in the field could not agree in their usage of the term (Singer, 1990). Cardena (1994), in reviewing the literature, concluded that dissociation lacks “a single, coherent referent or conceptualization that all investigators in the field embrace” (p. 15).

Despite a lack of scientific clarity, clinicians continue to find themselves faced with a need to assess and treat clients with symptoms which present as dissociative in nature (Graves, 1989). Unfortunately, many clinicians have not been properly educated and trained in the treatment of these disorders, increasing the likelihood of misdiagnosis and ineffectual treatment outcomes (Putnam, Gurof, Silberman, Barban, & Post, 1986). Clinicians presuming to exceed the boundaries of their professional competence may be susceptible to inducing false memory syndrome and even false “personalities” in a population particularly vulnerable to distortions of reality (Bliss, 1986; Hyman & Billings, 1995; Mersky, 1992; Ofshe & Waters, 1994; Spanos & Burgess, 1994;

Yapko, 1995). Debate over the legitimacy of some dissociative disorders has been heated and divisive, with professionals taking opposing viewpoints in courtroom testimony and other public forums (e.g., Bass & Davis, 1994; Dell, 1988; Horevitz, 1994; Loftus & Ketcham, 1994; Reviere, 1996; Terr, 1994). Such a predicament has important clinical, ethical, and legal implications (Horn, 1993).

The present confusion surrounding the dissociative disorders underscores the need for clinical treatment to be closely tied to experimental research. Treatment of the dissociative disorders is likely to be effective to the extent that it is guided by a coherent theoretical model. Such a model of dissociation must not only incorporate current research findings, but must also be capable of generating hypotheses which can be tested and empirically supported.

An Overview

The purpose of this study is threefold: (1) to develop a coherent theoretical model of dissociation which can generate experimental inquiry; (2) to delineate an experimental methodology for investigating dissociative phenomena, and; (3) to provide construct validation for some limited, but conceptually critical aspects of the proposed theoretical model. Supportive findings for an articulated model of dissociation would have relevant implications for the clinical assessment and treatment of dissociative disorders.

Adopting a definition from *The American Heritage Dictionary* (Morris, 1982), dissociation is conceptualized here as “the separation of a group of related psychological activities into autonomously functioning units” (p. 382). This definition dovetails nicely with current models of brain functioning which assume “that information processing

takes place through the interactions of a large number of simple processing elements called units, each sending excitatory and inhibitory signals to other units" (McClelland et al., 1986, p. 10). These models describe how excitatory signals can activate multiple units into "associatively" integrated functions, while inhibitory signals can separate, or "dissociate", these units into autonomously operating functions (Grigsby & Schneiders, 1991; Grigsby, Schneiders, & Kaye, 1991).

If this neuro-cognitive description can be applied to the level of psychological functions, then a relationship is posed between dissociative and inhibitive processes (Li & Spiegel, 1992; Nasby & Yates, 1996). In particular, the recent emphasis on a process of cognitive inhibition in human perception and thought (for reviews see Dagenbach & Carr, 1994; Dempster & Brainerd, 1995) suggests itself as a means of describing dissociative processes.

The first section of this study, entitled *The Cognitive Inhibition of Consciousness*, will provide the groundwork for an empirically based model of dissociation by reviewing experimental findings concerning *cognitive inhibition*. Defined as "the mechanism that controls and limits the contents of consciousness" (Frith, 1979, p. 225), *cognitive inhibition* may be seen as dissociative in the sense that it limits conscious awareness.

It should be noted that while the term "consciousness" is not rigorously defined in the diagnostic nosology, it can be experimentally defined in terms of a subject's access to cognitive information for verbal report. In this context, unreportable information can be shown to influence behavior in a manner which parallels psychiatric concepts associated with "unconscious motivation"; *implicit learning* tasks serving as a prime example (e.g.,

Bartis & Zamansky, 1986; Cohen & Squire, 1980; Eredelyi, 1985; Greenwald, 1992; Jacoby, Lindsay, & Toth, 1992; Kihlstrom, 1992, 1993; Kunst-Wilson & Zajonc, 1979; Shimamura, 1986). Hereafter, “consciousness” and “awareness” will be used as terms compatible with such operational and experimental definitions, with the tentative assumption that processes so defined are more or less parallel to the phenomenological and experiential processes being described.

Also presented in the first section are findings supportive of Frith’s (1979, 1981) hypothesis that deficits in cognitive inhibition will result in the intrusion of irrelevant distractions into the flow of conscious experience. As one of the diagnostic criteria for the dissociative disorders is “a disruption in the usually integrated functions of consciousness”, the second section (entitled *Dissociation and Reduced Inhibition*) explores potential relationships between reduced cognitive inhibition and dissociative experiences characterized by the intrusion of distorted perceptions, distractions, and traumatic memories. The third section, entitled *The Dissociation of Conscious Perception*, suggests that reduced inhibition may, over time, become automatically triggered as a learned response to perceptions of stressful situations. Reduced inhibition will increase one’s ability to focus on perceptual distractions as a means of avoiding (or *dissociating from*) an awareness of psychologically painful experiences.

The fourth section, entitled *Schema: The Dissociative Role of Inhibition*, proposes that schema, defined as “relatively stable cognitive patterns [which] form the basis for the regularity of interpretations of a particular set of situations” (Beck, Rush, Shaw, & Emery, 1979, p. 12), also operate as “functional units”. Schema, as functional

units, may be dissociated from each other through active inhibition, or may symptomatically intrude into awareness when inhibition is reduced. Schema developed in reaction to traumatic situations may also incorporate learned responses which reduce inhibition to facilitate defensive dissociative strategies such as distraction.

It is perhaps paradoxical that dissociation, rather than being descriptive of a simple process of inhibition, can also be described by failures in that inhibitive process. This may parallel the dissociative states in post-traumatic stress disorder characterized, paradoxically, by both the avoidance and the intrusive recollection of trauma-related experiences. The fifth section, entitled *Dissociative Symptomatology: Enhanced or Reduced Inhibition?*, seeks to resolve this paradox by describing how neurobiological processes of excitation and inhibition actually occur together as patterns across functional systems. In any behavioral response, some functions will be integrated into higher functions while others will be dissociated into independent functions. At the same time, both enhanced and reduced levels of inhibition may result in dissociative outcomes. Enhanced inhibition may be related to dissociative symptoms characterized by avoidance while reduced inhibition may be related to symptoms which are intrusive in nature.

The final theoretical section considers some of the implications of the proposed model for the clinical assessment and treatment of dissociative symptoms.

The premise that inhibitive processes may underlie dissociative functions provides a theoretical framework for a new understanding of dissociation. By applying the experimental literature concerning inhibition to descriptions of dissociation in the clinical literature, a new and comprehensive theory of dissociation can be formulated.

In contrast to previous theories, this framework will have the potential advantage of better lending itself to experimental inquiry.

The Cognitive Inhibition of Consciousness

Converging evidence depicts the brain as composed of vast neuronal networks of distributed, parallel systems which function to process a massive amount of information both prior to and outside the realm of conscious awareness (e.g., Baars, 1993; Ballard, 1986; Cohen, Dunbar, & McClelland, 1990; Kihlstrom, 1987, 1993; Kinsbourne, 1993; McClelland, 1979; Rumelhart, Hinton, & McClelland, 1986; Tulving & Shacter, 1990). In contrast, the limited stream of conscious awareness is seen as containing only that information which is most relevant to an individual's current goals (Posner & Boies, 1971). This situation implies the existence of some kind of mechanism in order to screen out information which distracts from (or is irrelevant to) the current goals of consciousness. Such a selective mechanism was posed by early “filter” models of selective attention (Broadbent, 1958). More recently, however, the process of selection has been theoretically identified with *cognitive inhibition* as “the mechanism that controls and limits the contents of consciousness” (Frith, 1979, p. 225).

Negative Priming

Although an active process of inhibition has long been implicated in selective attention (James, 1890; Pillsbury, 1908), most traditional models of attention have exclusively emphasized excitatory processes (e.g., Anderson, 1976; Broadbent, 1958; Van der Heijden, 1981). Such models propose that relevant information is activated into awareness, while irrelevant information passively decays; inhibitive processes are

deemed unnecessary in this scheme.

The notion of *cognitive inhibition* was derived as an explanatory concept for an interesting finding on the Stroop (1935) color-word task. In the original version of the task, subjects are asked to identify the color of the ink which is used to print a color name. When the color of the ink is incongruent with the color word (for instance, identifying the green ink used to print the word "red") there is a pronounced interference effect, in terms of a longer reaction time, as compared to the control condition of naming the ink color of words which are not color names.

Dalrymple-Alford and Budayr (1966) noticed that reaction times were even slower when the ink color to be named was identical to the distracting color word on the preceding trial. Neill (1977) extended this finding from a blocked condition to random presentations of the condition throughout the Stroop task. He concluded that "the preceding distractor response was selectively inhibited, making it relatively less available as an appropriate response during the next trial" (p. 444).

The experimental paradigm for cognitive inhibition can be generalized as follows: when a distractor on one trial becomes the target response on the successive trial, there is an increased latency of response due to inhibition persisting from the previous trial. Also known as the "distractor-suppression effect" (Neill & Westberry, 1987) or as "backward priming" (Hasher & Zacks, 1988), this experimental effect is generally referred to as "negative priming" (Tipper, 1985; e.g., as used in the majority of articles cited below for demonstrating negative priming effects).

Negative priming is a robust effect which has been demonstrated with a wide

variety of stimuli, including:

1. Between different letters (Fox, 1994b; Hasher, Stoltzfus, Zacks, & Ryma, 1991; McDowd & Oseas-Kreger, 1991; Neumann, 1989; Neumann & DeSchepper, 1992; Tipper & Cranston, 1985)
2. Between local and global aspects of letters (Briand, 1994)
3. Between different words (Beech, McManus, Baylis, Tipper & Agar, 1991; Bullen & Hemsley, 1987; Fuentes & Tudela, 1992; Hasher & Zacks, 1988; Hoffman & MacMillan, 1985; Ratcliff & McKoon, 1981; Tipper & Driver, 1988; Yee, 1991)
4. Between color names and word names (Stroop color words; Beech, Agar, & Baylis, 1989; Beech, Baylis, Smithson, & Claridge, 1989; Beech & Claridge, 1987; Beech, Powell, McWilliams, & Claridge, 1989, 1990; Benoit et al., 1992; Dalrymple-Alford & Budayr, 1966; Enright & Beech, 1990, 1993; Fox, 1994a; Laplante, Everett, & Thomas, 1992; Lowe, 1979, 1985; Neill, 1977; Neill & Westberry, 1987; Tipper, Bourque, Anderson, & Brehaut, 1989)
5. Between different color patches (cited in Houghton & Tipper, 1994)
6. Between different pictures (Gernsbacher & Faust, 1991; Tipper, 1985; Tipper & Driver, 1988; Tipper, Weaver, Cameron, Brehaut, & Bastedo, 1991)
7. Between different random shapes (cited in Houghton & Tipper, 1994)
8. Between different spatial locations (Milliken, Tipper, & Weaver, 1994; Tipper, Brehaut, & Driver, 1990; Tipper, Weaver, & Houghton, 1994; Tipper,

Weaver, Kirkpatrick, & Lewis, 1991)

9. Between static and moving objects (Tipper, Brehaut, & Driver, 1990)
10. Between different perceptual modalities (Driver & Baylis, 1993; Greenwald, 1972)
11. Between different locations in the selective reaching for objects (Tipper, Lortie, & Baylis, 1992)

The generality of the negative priming effect suggests an integral role of inhibition in the selection of conscious thought and behavior (e.g., see Tipper, Weaver, & Houghton, 1994). *Cognitive inhibition*, however, may actually be composed of multiple independent inhibitive processes. Separate inhibitive effects have been found for responding to a target's identity (e.g., naming tasks) as compared to responding to that same target's location; when the response is dependent upon both a target's identity and location combined, the separate inhibitive effects for identity and location have also been found to be combined in an additive fashion (Sullivan & Faust, 1993; Tipper, Weaver, Cameron, et al., 1991).

Although separate and independent, these inhibitive processes are hypothesized to work in concert to limit the contents of consciousness. Which function is inhibited is dependent upon the current goals of selective attention (Tipper, et al., 1994). By choosing to focus on an object's identity or its location, or some combination of both, we choose our functional level of awareness (Marcel, 1983a, 1983b). *Cognitive inhibition* is, in this sense, a reflection of the information which is inhibited at whichever functional level our attention is directed.

The negative priming effect has been found to diminish as the number of distractors are increased (Neumann, 1989; Neumann & DeSchepper, 1992). This spreading fan-like effect of inhibition can be interpreted as a counterpart of *spreading activation* (Van der Heijden, 1981; Broadbent, 1982). If spreading activation increases the probability of associated information reaching conscious awareness (Broadbent; Libet, 1993; Underwood, 1982; Van der Heijden) then spreading inhibition may be assumed to decrease such a probability (e.g., Neill & Westberry, 1987).

Cognitive inhibition thus *dissociates* nonconscious from conscious functional systems in terms of the processing of information. Within this context, the dissociative role of cognitive inhibition can be seen as a necessary feature of normal brain functioning.

Failed Inhibition: A Disorder of Consciousness?

Evidence for the role of cognitive inhibition in limiting conscious awareness comes from a body of work supportive of Frith's (1979, 1981) depiction of schizophrenia as a "disorder of consciousness". Noting that individuals diagnosed with schizophrenia have long been observed to have attentional deficits and difficulties inhibiting distracting information (e.g., Bleuler 1911/1950; Broadbent, 1958; Cromwell, 1968; Maher, 1983; McGhie & Chapman, 1961; Venables, 1963, 1978), Frith proposed that the symptoms of schizophrenia could be explained in terms of a breakdown in cognitive inhibition.

In brief, Frith suggested that without cognitive inhibition there would not be a mechanism to filter out unnecessary and irrelevant information from conscious awareness. While information would be processed in a normal manner prior to

consciousness, irrelevant information would fail to be inhibited and would directly intrude into the normal flow of conscious perception, language, thought, and behavior. The consequences of these intrusions are observed as symptoms of schizophrenia.

For example, hallucinatory and delusional symptoms could be attributable to incorrect interpretations of normal experiences. Normally there is only one interpretation of experience which can be conscious at any one time (Marcel, 1983a, 1983b). Without inhibition, however, there would be no narrowing down of alternative explanations (see Houghton & Tipper, 1994; Tipper, Weaver, & Houghton, 1994). Instead, multiple interpretations of an experience could be available at the same time, resulting in abnormal and irrational meanings being attached to what one sees and hears. The subjective experience, being divorced from the reality of others, would be hallucinatory. It is likewise argued that delusions are actually elaborate attempts, based on normal principles of reasoning, to explain irrational interpretations of perceptual experience. This follows from the finding that the very arrival of a perception in experiential awareness is subjectively interpreted as a sign of its importance and of the need to be explained (Posner & Boies, 1971).

In support of Frith's theory, reduced levels of cognitive inhibition have been found in subjects diagnosed with schizophrenia as compared to psychiatric and normal controls (Beech, Powell, McWilliam, & Claridge, 1989; Bullen & Hemsley, 1987; Laplante, Everett, & Thomas, 1992). These results have been extended to normal subjects who score high on measures of schizophrenic-like characteristics (Beech & Claridge, 1987; Beech, Baylis, Smithson, & Claridge, 1989; Beech, et al., 1991).

There is also evidence that auditory hallucinations have their basis in real sounds which activate multiple meanings prior to consciousness (Morton, 1979). This is consistent with findings which suggest that the early, pre-attentional stages of perceptual processing in individuals diagnosed with schizophrenia are unimpaired (Knight, Yourd, & Wooles, 1985; Spaulding, et al., 1980), as is the initial, inherent nature of their thought processes (Maher, 1972).

Schizophrenic subjects, in contrast to normal controls, have been found to evidence difficulties in inhibiting the contextually inappropriate, alternative meanings of ambiguous words (Bullen & Hemsley, 1987). Schizophrenic subjects also evidence illogical perceptual mergings and relationships, as found in their responses to Rorschach ink blots (Exner, 1986; Exner & Weiner, 1982). Difficulties with backward masking tasks (Saccuzzo, Hirt, & Spencer, 1974; Saccuzzo & Miller, 1977; Saccuzzo & Shubert, 1981) could be indicative of the inappropriate mergings and structurings of perceptual experience. The distortion of perceptions due to a failure of inhibition would be an outcome consistent with Marcel's (1980, 1983a, 1983b) conclusions concerning the nature of selection in conscious perception.

While it is not the purpose here to provide an exhaustive account of the consequences which may arise from failures of cognitive inhibition, it may be relevant to point out that this perspective can also accommodate the data normally used to support reduced capacity models of cognitive functioning (see Hasher & Zacks, 1988). Some current theories of schizophrenia assume that many of the deficits evidenced on cognitive tasks are attributable to abnormal limitations in the brain's capacity to process

information (e.g., see Knight, 1984). This reduction in capacity is sometimes associated with a functional imbalance between the brain's hemispheres (e.g., Venables, 1984).

By means of illustration, the findings of general decrements in the processing time of schizophrenics (Knight, et al., 1985; Saccuzzo & Braff, 1981; Yates, 1960) can be explained both as a reduction in capacity and as a failure of inhibition. When the nonconscious parallel processing of information fails to be cognitively inhibited, it intrudes into conscious thought and subsequently becomes relegated to the slow, sequential processing of attentive awareness (see Baars, 1993). Such a failure of inhibition would explain why schizophrenic subjects often appear to be processing in sequence that information which is normally processed in parallel (McGhie & Chapman, 1961; Place & Gilmore, 1980). This conclusion is consistent with Frith's (1979) prediction that, due to reduced cognitive inhibition, "schizophrenics should have particular problems with any situation which involves the use of complex processing systems which normally function below the level of awareness" (p. 232).

Individual Differences in Cognitive Inhibition

There is accumulating evidence that the effects of reduced cognitive inhibition are not limited to individuals diagnosed with schizophrenia. Rather, cognitive inhibition appears to be a dimension along which all individuals may manifest differences. Initial findings of reduced cognitive inhibition in subjects diagnosed with schizophrenia were readily extended to normal subjects scoring high on a measure of schizophrenic-like characteristics (Beech & Claridge, 1987; Beech, et al., 1989; Beech, et al., 1991). These results should not be surprising, given that schizophrenia itself is a term only broadly