1985

Conditioned Reinforcement: Order Out of Chaos

Robert Stephen Harris
University of Rhode Island

Follow this and additional works at: https://digitalcommons.uri.edu/oa_diss

Recommended Citation

Harris, Robert Stephen, "Conditioned Reinforcement: Order Out of Chaos" (1985). *Open Access Dissertations*. Paper 956.
https://digitalcommons.uri.edu/oa_diss/956

This Dissertation is brought to you for free and open access by DigitalCommons@URI. It has been accepted for inclusion in Open Access Dissertations by an authorized administrator of DigitalCommons@URI. For more information, please contact digitalcommons-group@uri.edu.

CONDITIONED REINFORCEMENT:

ORDER OUT OF CHAOS

BY

ROBERT STEPHEN HARRIS

A DISSERTATION SUBMITTED IN PARTIAL FULFILLMENT OF THE
REQUIREMENTS FOR THE DEGREE OF
DOCTOR OF PHILOSOPHY
IN
PSYCHOLOGY

UNIVERSITY OF RHODE ISLAND

1985

Abstract

The empirical research on conditioned reinforcement, after nearly half a century, has failed to yield a coherent and conclusive body of literature to justify its extensive use as a genuine phenomenon and a basic construct within the field of learning. The primary reason for this dilemma is a history of the use of inadequate controls and the use of paradigms which are subject to interpretations other than that of conditioned reinforcement. In order to determine conclusively, the necessary and sufficient conditions for the establishment of a conditioned reinforcement effect, intracranial stimulation of the lateral hypothalamus was used as the primary reinforcer in a study using multiple controls to remove and assess all possible sources of confounding. Following implantation, each subject was first placed in a two lever chamber for three consecutive days to obtain baseline levels of responding for a neutral stimulus. Three groups of 8 subjects pressed for one neutral stimulus on one lever and no stimulus on the second lever. Two groups of 8 subjects pressed for one neutral stimulus on one lever and the second neutral stimulus on the second lever.

In the second or pretraining phase, all subjects were placed in a single lever chamber in which lever pressing resulted in delivery of intracranial stimulation (ICS). After three 30 minute sessions on consecutive days, subjects failing to meet a 200 press per session criterion

were eliminated from the study.

Subjects were next given Pavlovian training with ICS in a chamber free of manipulanda. Of the groups that had received previously only one baseline neutral stimulus, one was presented with that stimulus 0.5 s prior to ICS (forward pairing). A second group received the neutral stimulus randomly relative to ICS. A third group received the neutral stimulus in an explicitly unpaired format relative to ICS. Both of the groups that received two neutral stimuli, received one stimulus 0.5 s prior to ICS. One group received the second stimulus randomly relative to ICS, and the second group received the second stimulus in an explicitly unpaired format relative to ICS.

All groups received 100 randomly spaced presentations of the neutral stimulus or stimuli per day in 100 minute sessions for four consecutive days, with ICS presented 100 times on Day 1 and an average of 50 times thereafter intermittently.

Subjects were tested during one 40 minute session in the original two lever chamber. Results indicated that subjects significantly increased responding for the forwardly paired stimulus from baseline to test. Increases in responding for control stimuli from pretest to test were either non-significant or significant but weaker than increases for the forwardly paired stimulus. Results were interpreted as clearly demonstrating a conditioned reinforcement effect, for which Pavlovian temporal

contiguity is, empirically, the necessary and sufficient condition. The results were discussed in terms of various theories of conditioned reinforcement and a revised model was proposed, emphasizing the arousal of a hypothetical fractional neural excitatory response and its conditioning to a CS as the basis for all conditioned reinforcement effects.

Acknowledgement

This completed document represents the final stage in a long, sometimes very satisfying, sometimes very difficult process of obtaining a doctorate degree. It was my naive belief, as I crossed the major hurdle of comprehensive examinations in the spring of 1981, that this last phase, having been well planned, would follow an orderly path to rapid completion of my graduate career.

After several false starts, abruptly terminated by the demands of my professional career, I began in September of 1983 to undertake the completion of this last step anticipating a May 1984 graduation date with consistent effort, but, otherwise, with relative ease.

As I write this last section of my dissertation in early May of 1985, I am caused to reflect on the last 20 months of daily effort on this project, and to wonder how a person of adequate training and ability could have been so naive as to think "relative ease" was in any way descriptive of this process. The outcome of this reflection is one of profound humility and, at the same time, tremendous excitement--humility that I so badly misjudged the task before me in September of 1983, and excitement that the project worked and worked well and that the long hours, days and months of frustration, doubt, renewal and frustration again are only days away from a final conclusion.

This project has been completed with the assistance

and support of many people to whom I am sincerely and profoundly grateful. In the early stages of this project, three undergraduate assistants, Bonnie McLennan, Wendy Silvestri, and Ursula Roxin, were both helpful and supportive. Wendy and Ursula worked diligently as members of our surgery team while Bonnie assisted me in assembling the apparatus and in equipment construction. Ursula continued to work intermittently during the remainder of the project battling the confusion of the library reference section to obtain many of the more recent publications mentioned in the dissertation.

The psychology department secretaries, and Diane Sipe in particular, were of tremendous assistance in responding to innumerable small (and sometimes not so small) requests. Diane was especially helpful in processing, tracking down, and obtaining the frequent supplies needed to conduct this research. In addition to their assistance, the members of the secretarial staff were always friendly, concerned, and efficient, even under circumstances of a heavy workload and short staffing. This level of responsiveness is not often found and is thoroughly appreciated.

Steve Colucci generously coached me through the early stages of learning the Prime Editor and Runoff word processing systems without which the process of editing successive drafts of this document would have taken months, aside from the cost of professional typing of the final draft. Steve's patience with my repeated questions, the

generosity of his time, and the support he has provided through the more frustrating phases of this project have been important to me and to the success of this project.

Joe Rossi introduced me to the concept of omega-squared as a measure of relative magnitude of effects, a statistical procedure which was critical to several specific explanations of the results. His explanation and instruction was clear and thorough, and left me with the skills necessary to perform the analyses and accurately interpret the outcomes. Joe's assistance was generously provided and sincerely appreciated.

The staff of Rhode Island Youth Guidance Center where I'm employed has been an understanding and supportive cast of characters during the past 20 months. This project, in spite of my most determined efforts to the contrary, has had an impact on the running of the agency which can be at best described as a major inconvenience. Kevin Plummer, Golda Bender, Sarah Dinklage, Stuart Vyze, Robbie Lasser, Merrill Kidman, Dan Hoy, Sue Campbell and the rest of the staff and students have remained remarkably calm and understanding in the wake of repeated delays, cancellations, and a myriad of other annoyances caused by my absence. It has been critical to my ability to concentrate on this research to know that the consultation department has been staffed by a group of hard working, highly skilled, and highly successful professionals. I regret my inability to offer to these folks the level of

leadership and support with which I am satisfied.

Kevin Plummer's consistent, thorough conceptualization and handling of department responsibilities and ongoing problems has been particularly important to my ability to continue this research. I have relied heavily on Kevin's skill, support, and willingness to assume responsibilities above and beyond reasonable expectations. It is difficult to express how important his professional support and his friendship have been in these last several months.

There is a group of people some of whom I had never met and none of whom I knew well before I started this research, for whom my face has probably become all too familiar over the past several months. This group is the lab crew, Sally Hazard, P. J. Vuono, Tom Rieg, Tony Riccitelli, Stuart Vyze, Paige DiBiasio, Debbie Bannon, and Susan Blank. I began in September of 1983 as a stranger out of the past whose invasion into the space of this group was regarded with some reasonable degree of caution. This caution has undergone several transformations over time, from skepticism about this research, to disbelief that I could persist so long through so many failures and still believe it would work, to cautious acceptance of preliminary positive outcomes, to strong encouragement to continue, and finally to a desire by some to participate in what they believe to be a valuable and fruitful area of scientific investigation. These people whom I knew little or not at all have become friends over the course of these

transformations and their personal and professional contributions to this project have exceeded probably their own awareness.

Tom, I think, began asking for his final copy of this document about 340 subjects ago. Tom's diligent maintenance of the colony and my animals this year, and his interest, concern, and participation in the continuing research together with his dry teasing have made the lab area an enjoyable place to be.

Tony's skills in the physiology lab, his willingness to share those skills, his sharing of responsibility for seeking and obtaining research supplies, his assistance in problem solving, his wit and sense of humor, and his support have been invaluable.

P. J.'s judgement at times may seem a little skewed. Anyone who volunteers to double-check the calculations for 160 follow-up tests and type the references for a document of these proportions is either suffering from muddled thinking, or is genuinely one of the friendliest and most generous people one could have the privilege of knowing.

P. J.'s support, assistance, and caring have been immeasurable. And it never seems to end.

Sally's response to this research and to me personally and professionally has undergone perhaps the most dramatic change, from cautious skepticism to exceptional support and encouragement. Her patience as colony caretaker, a job for which she had little fondness, was exceptional as the

promised ceiling of 32 animals more than once exceeded 100. The graphs prepared by Sally, which appear in the Appendix, do not reflect the long hours, and frequent and frustrating changes required to achieve the current product, for which Sally's initial motivation was merely that of getting some practice in graph-making. Her enthusiasm for this research, her interest in it and her willingness to help, her sense of humor, and her ability to tolerate my obnoxiousness have added greatly to my enjoyment of this project.

Without the guidance, encouragement, and support of my committee throughout my graduate career and particularly during this project, I would certainly not have reached this point. Al Silverstein, Nelson Smith, Wayne Velicer, Dom Valentino, Jim Prochaska, and Jim Loy are all faculty for whom I have a great deal of respect. Each has contributed one or more critical elements to my professional growth and training for which I am sincerely grateful.

In completing this dissertation, the involvement of two committee members has been particularly important. Nelson Smith has been instrumental in insuring that the necessary work space, equipment, and supplies were available. Without the availability of his knowledge and skill in apparatus programming and his command of the practical elements of animal research, this project would not have succeeded. His willingness to thoroughly review the unsuccessful outcome of each pilot effort and to

contribute to the next strategy was a significant contribution. His participation, encouragement, and support through the final stages of the project are greatly appreciated.

Al Silverstein's contribution to this dissertation began early in my graduate career with my first directed readings in conditioned reinforcement. His contribution to my knowledge of the field, to the development of my skills in experimental design and critique, and to my development as a scientist-practitioner is profound. Working with Al is a rigorous process, like no other to which I've been exposed. The outcome of that rigor is a graduate career and a piece of research for which confidence comes easily. Al's standards for one's command of the literature and for experimental research are high, and he has applied those standards as we have conceptualized, redesigned, and completed this project. His attention to this project has been thorough and it has been critical to its final success. He has shared the discouragement of early failures and provided strong encouragement as more positive results became apparent. His excitement at the outcomes and his enjoyment in discussing those outcomes and their theoretical implications has made this difficult task exciting for me as well. I'm sure that often over the past few years, Al has doubted that I would reach the point of writing the acknowledgement section of this dissertation. His support, faith, and encouragement have been

instrumental in bringing me to this point. I could not have been more fortunate than to have had Al as my major professor.

Finally, my wife, Pam (Zappardino), deserves tremendous credit for my having successfully reached this point. Only a fellow graduate student could have understood my virtual absence from our personal lives for the past 20 months. The demands on both of us have been many and the strains great. Yet we have survived. And not without incredible effort, patience, and understanding on Pam's part. As chief surgeon for this research, Pam's contribution was more than substantial. It was she who performed the critical phases of electrode implantation for 340 animals, without which this research could not have been done. She has been understanding of my dramatic changes in temperament, of which there have been many in the past few months, and has shared in my excitement as I near the end, even though her own research proceeds at a frustrating and, often times, discouraging pace. I am deeply grateful for her assistance, understanding, love, and support.

My most sincere thanks to all of you.

Table of Contents

	<u>Page</u>
List of Tables	xvii
List of Figures	xxiii
Introduction	1
Method	8
Subjects	8
Apparatus	9
Procedure	10
Results	16
Discussion	38
References	57
 Appendix A	
Conditioned Reinforcement: Reality or Myth?	62
Table A1: Outline of Sequence of Experimental Phases	133
Results: Detailed Presentation	135
 Appendix B	
Analyses and Graphs of Number of Lever Presses for Within Subjects Controls	168
 Appendix C	
Analyses and Graphs of Number of Stimulus Presentations for Within Subjects Controls	192
 Appendix D	
Analyses and Graphs of Lever Depression Duration for Within Subjects Controls	216
 Appendix E	

Analyses and Graphs of Number of Lever Presses for Between Subjects Controls	243
---	-----

Appendix F

Analyses and Graphs of Number of Stimulus Presentations for Between Subjects Controls	266
--	-----

Appendix G

Analyses and Graphs of Lever Depression Duration for Between Subjects Controls	288
---	-----

Appendix H

Analysis of Number of Lever Presses for Pretest Between Group Preferences for Light and Tone	312
--	-----

Appendix I

Analysis of Number of Stimulus Presentations for Pretest Between Group Preferences for Light and Tone	315
---	-----

Appendix J

Analysis of Lever Depression Duration for Pretest Between Group Preferences for Light and Tone	318
--	-----

Appendix K

Analysis of Number of Lever Presses for Pretest Within Group Preferences for Light and Tone	321
---	-----

Appendix L

Analysis of Number of Stimulus Presentations for Pretest Within	
--	--

Group Preferences for Light and Tone	323
Appendix M	
Analysis of Lever Depression	
Duration for Pretest Within	
Group Preferences for Light and Tone	325
Appendix N	
Analysis of Number of Lever Presses for	
Pretest Preferences for Right and Left	327
Appendix O	
Analysis of Number of Stimulus	
Presentations for Pretest	
Preferences for Right and Left	329
Appendix P	
Analysis of Lever Depression	
Duration for Pretest	
Preferences for Right and Left	331
Appendix Q	
Analyses of Between Group	
Differences in Age	333
Appendix R	
Analyses of Between Group	
Differences in Percent of Training Pairings . .	336
Appendix S	
Analyses of Between Group	
Differences in Weight	339
Appendix T	
Analysis of Between Group	

Differences in Number of	
Pretraining Days to Criterion	342

Appendix U

Analysis of Between Group	
Differences in Training Time	344

Appendix V

Analysis of Between Group	
Differences in Pretraining	
Lever Presses to Criterion	346

Appendix W

Analyses of Between Group	
Differences in Days of the	
Experimental Procedure	348

Bibliography	352
------------------------	-----

List of Tables

<u>Table</u>	<u>Page</u>
1. Schematic Design of ANOVA Subsets	18
2. Means and Standard Deviations of Lever Presses for Within Subjects Controls	22
3. Means and Standard Deviations of Lever Presses for Between Subjects Controls	28
4. Mean Changes in Lever Presses from Pretest to Test for Each Lever	43
A1. Outline of Sequence of Experimental Phases	133
A2. Means and Standard Deviations of Stimulus Presentations for Within Subjects Controls	143
A3. Means and Standard Deviations of Lever Depression Duration for Within Subjects Controls	148
A4. Means and Standard Deviations of Stimulus Presentations for Between Subjects Controls	155
A5. Means and Standard Deviations of Lever Depression Duration for Between Subjects Controls	158
B1. Analysis of Variance Summary Table for Lever Presses for Within Subjects Controls	168
B2. BC (Stimulus x Pretest/Test) Summary Table of Lever Presses for Within Subjects Controls	177

B3.	Simple Main Effects Tests of Lever Presses for B (Stimulus) at C (Pretest/Test) for Within Subjects Controls	178
B4.	Simple Main Effects Tests of Lever Presses for C (Pretest/Test) at B (Stimulus) for Within Subjects Controls	179
B5.	AB (Group x Stimulus) Summary Table of Lever Presses for Within Subjects Controls	180
B6.	Simple Main Effects Tests of Lever Presses for A (Group) at B (Stimulus) for Within Subjects Controls	181
B7.	Newman-Keuls Analysis of Lever Presses for Between Group (A) Differences at the Paired (B1) Stimulus Level for Within Subjects Controls	182
B8.	Simple Main Effects Tests of Lever Presses for B (Stimulus) at A (Group) for Within Subjects Controls	183
B9.	AC (Group x Pretest/Test) Summary Table of Lever Presses for Within Subjects Controls	184
B10.	Simple Main Effects Tests of Lever Presses for A (Group) at C (Pretest/Test) for Within Subjects Controls	185
B11.	Newman-Keuls Analysis of Lever Presses for Between Group (A) Differences in the Test (C2)	

Phase for Within Subjects Controls	186
B12. Simple Main Effects Tests of Lever Presses for C (Pretest/Test) at A (Group) for Within Subjects Controls	187
B13. ABC (Group x Stimulus x Pretest/Test) Summary Table of Lever Presses for Within Subjects Controls	188
B14. Simple, Simple Main Effects Tests of Lever Presses for B (Stimulus) at AC for Within Subjects Controls	189
B15. Simple, Simple Main Effects Tests of Lever Presses for C (Pretest/Test) at AB for Within Subjects Controls	190
B16. Simple Interaction Effects Tests of Lever Presses for BC (Stimulus x Pretest/Test) at A (Group) for Within Subjects Controls	191
C1. Analysis of Variance Summary Table for Stimulus Presentations for Within Subjects Controls	192
C2. BC (Stimulus x Pretest/Test) Summary Table of Stimulus Presentations for Within Subjects Controls	201
C3. Simple Main Effects Tests of Stimulus Presentations for B (Stimulus) at C (Pretest/Test) for Within Subjects Controls	202
C4. Simple Main Effects Tests of Stimulus Presentations for C (Pretest/Test) at	

	B (Stimulus) for Within Subjects Controls	203
C5.	AB (Group x Stimulus) Summary Table of Stimulus Presentations for Within Subjects Controls	204
C6.	Simple Main Effects Tests of Stimulus Presentations for A (Group) at B (Stimulus) for Within Subjects Controls	205
C7.	Newman-Keuls Analysis of Stimulus Presentations for Between Group (A) Differences at the Paired (B1) Stimulus Level for Within Subjects Controls	206
C8.	Simple Main Effects Tests of Stimulus Presentations for B (Stimulus) at A (Group) for Within Subjects Controls	207
C9.	AC (Group x Pretest/Test) Summary Table of Stimulus Presentations for Within Subjects Controls	208
C10.	Simple Main Effects Tests of Stimulus Presentations for A (Group) at C (Pretest/Test) for Within Subjects Controls	209
C11.	Newman-Keuls Analysis of Stimulus Presentations for Between Group (A) Differences in the Test (C2) Phase for Within Subjects Controls	210
C12.	Simple Main Effects Tests of Stimulus Presentations for C (Pretest/Test) at A (Group) for Within Subjects Controls	211

C13.	ABC (Group x Stimulus x Pretest/Test) Summary Table of Stimulus Presentations for Within Subjects Controls	212
C14.	Simple, Simple Main Effects Tests for Stimulus Presentations for B (Stimulus) at AC for Within Subjects Controls	213
C15.	Simple, Simple Main Effects Tests for Stimulus Presentations for C (Pretest/Test) at AB for Within Subjects Controls	214
C16.	Simple Interaction Effects Tests of Stimulus Presentations for BC (Stimulus x Pretest/Test) at A (Group) for Within Subjects Controls	215
D1.	Analysis of Variance Summary Table for Lever Depression Duration for Within Subjects Controls	216
D2.	BC (Stimulus x Pretest/Test) Summary Table of Lever Depression Duration for Within Subjects Controls	225
D3.	Simple Main Effects Tests of Lever Depression Duration for B (Stimulus) at C (Pretest/Test) for Within Subjects Controls	226
D4.	Simple Main Effects Tests of Lever Depression Duration for C (Pretest/Test) at B (Stimulus) for Within Subjects Controls	227
D5.	AB (Group x Stimulus) Summary Table of Lever Depression Duration for Within Subjects Controls	228

D6.	Simple Main Effects Tests of Lever Depression Duration for A (Group) at B (Stimulus) for Within Subjects Controls	229
D7.	Newman-Keuls Analysis of Lever Depression Duration for Between Group (A) Differences at the Paired (B ¹) Stimulus Level for Within Subjects Controls	230
D8.	Simple Main Effects Tests of Lever Depression Duration for B (Stimulus) at A (Group) for Within Subjects Controls	231
D9.	AC (Group x Pretest/Test) Summary Table of Lever Depression Duration for Within Subjects Controls	232
D10.	Simple Main Effects Tests of Lever Depression Duration for A (Group) at C (Pretest/Test) for Within Subjects Controls	233
D11.	Newman-Keuls Analysis of Lever Depression Duration for Between Group (A) Differences in the Test (C2) Phase for Within Subjects Controls	234
D12.	Simple Main Effects Tests of Lever Depression Duration for C (Pretest/Test) at A (Group) for Within Subjects Controls	235
D13.	ABC (Group x Stimulus x Pretest/Test) Summary Table of Lever Depression Duration for Within Subjects Controls	236
D14.	Simple, Simple Main Effects Tests of Lever	

	Depression Duration for B (Stimulus) at AC for Within Subjects Controls	237
D15.	Simple, Simple Main Effects Tests of Lever Depression Duration for C (Pretest/Test) at AB for Within Subjects Controls	238
D16.	Simple Interaction Effects Tests of Lever Depression Duration for BC (Stimulus x Pretest/Test) at A (Group) for Within Subjects Controls	239
D17.	Analysis of Variance Summary Table for Natural Log Transformation of Lever Depression Duration for Within Subjects Controls	240
D18.	Means and Standard Deviations of Natural Log Transformation of Lever Depression Duration for Within Subjects Controls	241
E1.	Analysis of Variance Summary Table for Lever Press for Between Subjects Controls	243
E2.	BC (Stimulus x Pretest/Test) Summary Table of Lever Presses for Between Subjects Controls	252
E3.	Simple Main Effects Tests of Lever Presses for B (Stimulus) at C (Pretest/Test) for Between Subjects Controls	253
E4.	Simple Main Effects Tests of Lever Presses for C (Pretest/Test) at B (Stimulus) for Between Subjects Controls	254

E5.	AB (Group x Stimulus)	
	Summary Table of Lever Presses for	
	Between Subjects Controls	255
E6.	Simple Main Effects Tests of Lever Presses for	
	A (Group) at B (Stimulus) for	
	Between Subjects Controls	256
E7.	Newman-Keuls Analysis of Lever Presses for	
	Between Group (A) Differences at the	
	Paired (B ¹) Stimulus Level for	
	Between Subjects Controls	257
E8.	Simple Main Effects Tests of Lever Presses for	
	B (Stimulus) at A (Group) for	
	Between Subjects Controls	258
E9.	AC (Group x Pretest/Test)	
	Summary Table of Lever Presses for	
	Between Subjects Controls	259
E10.	Simple Main Effects Tests of Lever Presses for	
	A (Group) at C (Pretest/Test) for	
	Between Subjects Controls	260
E11.	Simple Main Effects Tests of Lever Presses for	
	C (Pretest/Test) at A (Group) for	
	Between Subjects Controls	261
E12.	ABC (Group x Stimulus x Pretest/Test)	
	Summary Table of Lever Presses for	
	Between Subjects Controls	262
E13.	Simple, Simple Main Effects Tests of	
	Lever Presses for B (Stimulus) at AC for	

	Between Subjects Controls	263
E14.	Simple, Simple Main Effects Tests of Lever Presses for C (Pretest/Test) at AB for Between Subjects Controls	264
E15.	Simple Interaction Effects Tests of Lever Presses for BC (Stimulus x Pretest/Test) at A (Group) for Between Subjects Controls	265
F1.	Analysis of Variance Summary Table for Stimulus Presentations for Between Subjects Controls	266
F2.	BC (Stimulus x Pretest/Test) Summary Table of Stimulus Presentations for Between Subjects Controls	275
F3.	Simple Main Effects Tests of Stimulus Presentations for B (Stimulus) at C (Pretest/Test) for Between Subjects Controls	276
F4.	Simple Main Effects Tests of Stimulus Presentations for C (Pretest/Test) at B (Stimulus) for Between Subjects Controls	277
F5.	AB (Group x Stimulus) Summary Table of Stimulus Presentations for Between Subjects Controls	278
F6.	Simple Main Effects Tests of Stimulus Presentations for A (Group) at B (Stimulus) for Between Subjects Controls	279
F7.	Simple Main Effects Tests of Stimulus Presentations for B (Stimulus) at A (Group) for	

	Between Subjects Controls	280
F8.	AC (Group x Pretest/Test)	
	Summary Table of Stimulus Presentations for	
	Between Subjects Controls	281
F9.	Simple Main Effects Tests of Stimulus	
	Presentations for A (Group) at C (Pretest/Test) for	
	Between Subjects Controls	282
F10.	Simple Main Effects Tests of Stimulus	
	Presentations for C (Pretest/Test) at A (Group) for	
	Between Subjects Controls	283
F11.	ABC (Group x Stimulus x Pretest/Test)	
	Summary Table of Stimulus Presentations for	
	Between Subjects Controls	284
F12.	Simple, Simple Main Effects Tests of	
	Stimulus Presentations for B (Stimulus) at	
	AC for Between Subjects Controls	285
F13.	Simple, Simple Main Effects Tests of	
	Stimulus Presentations for C (Pretest/Test) at	
	AB for Between Subjects Controls	286
F14.	Simple Interaction Effects Tests of	
	Stimulus Presentations for	
	BC (Stimulus x Pretest/Test) at	
	A (Group) for Between Subjects Controls	287
G1.	Analysis of Variance Summary Table for	
	Lever Depression Duration for	
	Between Subjects Controls	288
G2.	BC (Stimulus x Pretest/Test)	

	Summary Table of Lever Depression	
	Duration for Between Subjects Controls	297
G3.	Simple Main Effects Tests of Lever Depression	
	Duration for B (Stimulus) at C (Pretest/Test) for	
	Between Subjects Controls	298
G4.	Simple Main Effects Tests of Lever Depression	
	Duration for C (Pretest/Test) at B (Stimulus) for	
	Between Subjects Controls	299
G5.	AB (Group x Stimulus)	
	Summary Table of Lever Depression	
	Duration for Between Subjects Controls	300
G6.	Simple Main Effects Tests of Lever Depression	
	Duration for A (Group) at B (Stimulus) for	
	Between Subjects Controls	301
G7.	Newman-Keuls Analysis of Lever Depression	
	Duration for Between Group (A)	
	Differences at the Paired Stimulus (B1)	
	Level for Between Subjects Controls	302
G8.	Simple Main Effects Tests of Lever Depression	
	Duration for B (Stimulus) at A (Group) for	
	Between Subjects Controls	303
G9.	AC (Group x Pretest/Test)	
	Summary Table of Lever Depression	
	Duration for Between Subjects Controls	304
G10.	Simple Main Effects Tests of Lever Depression	
	Duration for A (Group) at C (Pretest/Test) for	
	Between Subjects Controls	305

G11.	Newman-Keuls Analysis of Lever Depression Duration for Between Group (A) Differences in the Test (C2) Phase for Between Subjects Controls	306
G12.	Simple Main Effects Tests of Lever Depression Duration for C (Pretest/Test) at A (Group) for Between Subjects Controls	307
G13.	ABC (Group x Stimulus x Pretest/Test) Summary Table of Lever Depression Duration for Between Subjects Controls	308
G14.	Simple, Simple Main Effects Tests of Lever Depression Duration for B (Stimulus) at AC for Between Subjects Controls	309
G15.	Simple, Simple Main Effects Tests of Lever Depression Duration for C (Pretest/Test) at AB for Between Subjects Controls	310
G16.	Simple Interaction Effects Tests of Lever Depression Duration for BC (Stimulus x Pretest/Test) at A (Group) for Between Subjects Controls	311
H1.	Analysis of Variance Summary Table of Lever Presses for Pretest Between Group Preferences for Light and Tone	312
H2.	Means and Standard Deviations of Lever Presses for Pretest Between Group Preferences for Light and Tone	313
I1.	Analysis of Variance Summary Table of	

Stimulus Presentations for Pretest Between Group Preferences for Light and Tone	315
I2. Means and Standard Deviations of Stimulus Presentations for Pretest Between Group Preferences for Light and Tone	316
J1. Analysis of Variance Summary Table of Lever Depression Duration for Pretest Between Group Preferences for Light and Tone	318
J2. Means and Standard Deviations of Lever Depression Duration for Pretest Between Group Preferences for Light and Tone	319
K1. Analysis of Variance Summary Table of Lever Presses for Pretest Within Group Preferences for Light and Tone	321
K2. Means and Standard Deviations of Lever Presses for Pretest Within Group Preferences for Light and Tone	322
L1. Analysis of Variance Summary Table of Stimulus Presentations for Pretest Within Group Preferences for Light and Tone	323
L2. Means and Standard Deviations of Stimulus Presentations for Pretest	

Within Group Preferences for Light and Tone	324
M1. Analysis of Variance Summary Table of Lever Depression Duration for Pretest Within Group Preferences for Light and Tone	325
M2. Means and Standard Deviations of Lever Depression Duration for Pretest Within Group Preferences for Light and Tone	326
N1. Analysis of Variance Summary Table of Lever Presses for Pretest Preferences for Right and Left Lever	327
N2. Means and Standard Deviations of Lever Presses for Pretest Preferences for Right and Left Lever	328
O1. Analysis of Variance Summary Table of Stimulus Presentations for Pretest Preferences for Right and Left Lever	329
O2. Means and Standard Deviations of Stimulus Presentations for Pretest Preferences for Right and Left Lever	330
P1. Analysis of Variance Summary Table of Lever Depression Duration for Pretest Preferences for Right and Left Lever	331
P2. Means and Standard Deviations of Lever Depression Duration for Pretest	

	Preferences for Right and Left Lever	332
Q1.	Analysis of Variance Summary Table of Between Group Differences in Age	333
Q2.	Means and Standard Deviations of Between Group Differences in Age	334
Q3.	Newman-Keuls Analysis of Between Group Differences in Age	335
R1.	Analysis of Variance Summary Table of Between Group Differences in Percent of Training Pairings	336
R2.	Means and Standard Deviations of Between Group Differences in Percent of Training Pairings	337
R3.	Newman-Keuls Analysis of Between Group Differences in Percent of Training Pairings	338
S1.	Analysis of Variance Summary Table of Between Group Differences in Weight	339
S2.	Means and Standard Deviations of Between Group Differences in Weight	340
S3.	Newman-Keuls Analysis of Between Group Differences in Weight	341
T1.	Analysis of Variance Summary Table of Between Group Differences in Number of Pretraining Days to Criterion	342
T2.	Means and Standard Deviations of Between Group Differences in Number of	

Pretraining Days to Criterion	343
U1. Analysis of Variance Summary Table of Between Group Differences in Training Time	344
U2. Means and Standard Deviations of Between Group Differences in Training Time	345
V1. Analysis of Variance Summary Table of Between Group Differences in Number of Pretraining Lever Presses to Criterion	346
V2. Means and Standard Deviations of Between Group Differences in Number of Pretraining Lever Presses to Criterion	347
W1. Analysis of Variance Summary Table of Between Group Differences in Number of Days of the Experimental Procedure	348
W2. Means and Standard Deviations of Between Group Differences in Number of Days of the Experimental Procedure	349
W3. Analysis of Variance Summary Table of Natural Log Transformation of Between Group Differences in Number of Days of the Experimental Procedure	350
W4. Means and Standard Deviations of Natural Log Transformation of Between Group Differences in Number of Days of the Experimental Procedure	351

List of Figures

<u>Figure</u>	<u>Page</u>
B1. Number of lever presses for B (Stimulus) at C (Pretest/Test) for paired/nothing subjects.	169
B2. Number of lever presses for B (Stimulus) at C (Pretest/Test) for paired/random subjects.	171
B3. Number of lever presses for B (Stimulus) at C (Pretest/Test) for paired/explicitly unpaired subjects.	173
B4. Number of lever presses for B (Stimulus) at C (Pretest/Test) for within subjects controls.	175
C1. Number of stimulus presentations for B (Stimulus) at C (Pretest/Test) for paired/nothing subjects.	193
C2. Number of stimulus presentations for B (Stimulus) at C (Pretest/Test) for paired/random subjects.	195
C3. Number of stimulus presentations for B (Stimulus) at C (Pretest/Test) for paired/explicitly unpaired subjects.	197
C4. Number of stimulus	

presentations for B (Stimulus) at C (Pretest/Test) for within subjects controls.	199
D1. Lever depression duration for B (Stimulus) at C (Pretest/Test) for paired/nothing subjects.	217
D2. Lever depression duration for B (Stimulus) at C (Pretest/Test) for paired/random subjects.	219
D3. Lever depression duration for B (Stimulus) at C (Pretest/Test) for paired/explicitly unpaired subjects.	221
D4. Lever depression duration for B (Stimulus) at C (Pretest/Test) for within subjects controls.	223
E1. Number of lever presses for B (Stimulus) at C (Pretest/Test) for paired/nothing subjects.	244
E2. Number of lever presses for B (Stimulus) at C (Pretest/Test) for random/nothing subjects.	246
E3. Number of lever presses for B (Stimulus) at C (Pretest/Test) for explicitly unpaired/nothing subjects.	248
E4. Number of lever presses for B (Stimulus) at C (Pretest/Test) for between subjects controls.	250

F1.	Number of stimulus presentations for B (Stimulus) at C (Pretest/Test) for paired/nothing subjects.	267
F2.	Number of stimulus presentations for B (Stimulus) at C (Pretest/Test) for random/nothing subjects.	269
F3.	Number of stimulus presentations for B (Stimulus) at C (Pretest/Test) for explicitly unpaired/nothing subjects.	271
F4.	Number of stimulus presentations for B (Stimulus) at C (Pretest/Test) for between subjects controls.	273
G1.	Lever depression duration for B (Stimulus) at C (Pretest/Test) for paired/nothing subjects.	289
G2.	Lever depression duration for B (Stimulus) at C (Pretest/Test) for random/nothing subjects.	291
G3.	Lever depression duration for B (Stimulus) at C (Pretest/Test) for explicitly unpaired/nothing subjects.	293
G4.	Lever depression duration for B (Stimulus) at C (Pretest/Test) for	

between subjects controls.

295

Conditioned Reinforcement:

Order out of Chaos

Conditioned reinforcement, as an explanatory mechanism, has maintained a prominent position within the area of applied behavior analysis. It is generally accepted that very little of the complex behavior displayed by adult humans is under the control of primary reinforcers (SRs). Having thus omitted primary reinforcement as a means by which to explain the majority of human behavior, behavior analysts have quite freely invoked the concept of conditioned reinforcement. In this context, it is generally accepted that a conditioned reinforcement effect has occurred when a stimulus which does not possess any inherent drive reduction or incentive properties, that is, is considered "neutral", has increased the probability of a response upon which it is contingent. It is, likewise, generally accepted that the conditioned reinforcer (Sr) has acquired its reinforcement capacity by virtue of its prior association, in some manner, with an SR or other effective Sr.

However, the empirical research on conditioned reinforcement has, after nearly half a century, failed to yield a coherent and conclusive body of literature to justify its extensive use as an explanatory mechanism. A history of problematic experimental paradigms (extinction and chained) (Wike, 1966) and inattention to adequate control conditions has left little in the way of an

understanding of the necessary and sufficient conditions for the establishment of a conditioned reinforcement effect or the theoretical interpretation of how such an effect might operate.

The extinction paradigm of research is subject to confounding by stimulus generalization decrement (Crum, Brown, & Bitterman, 1951; Elam, Tyler, & Bitterman, 1954; Wike, 1966). Chained paradigms, on the other hand, are subject to confounding of discriminative stimulus functions with S_r functions (Wike, 1966). New learning paradigms, while free of such confounding effects, yield the least durable results (Wike, 1966). Critical to the control issue is the use of adequate pseudo- and counterconditioning controls (Rescorla, 1967). The design problems, both paradigm and control, have lead some authors (e.g., Bolles, 1975; Longstreth, 1971), to seriously question the existence of conditioned reinforcement as a phenomenon.

The potential use of positive intracranial stimulation (ICS) as a primary reinforcer in the establishment of a conditioned reinforcement effect, offers some inherent benefits for addressing the fundamental question of the necessary and sufficient conditions for S_r establishment. In using more traditional S_R s, the possibility of confounding by the consummatory process poses difficulties from the standpoint of differentiating among various theoretical interpretations of the critical conditions for

the establishment and operation of S-rs: for example, discriminative stimulus (SD) (Keller & Schoenfeld, 1950), response contingent (Bilbrey & Winokur, 1975; Silverstein & Lipsett, 1974), and elicitation (Keehn, 1962; Wike, 1966) theories.

Because ICS requires no consummatory response, and, in specific neural sites, elicits no observable unconditioned response (UCR), to which response facilitation interpretations might be adduced, it possesses the potential, for delineating among competing theoretical perspectives. Further, it offers the advantage of greater experimental control over training parameters (e.g., pairing configuration, magnitude, directness of presentation, timing of delivery) and is highly resistant to satiation effects during training. Finally, it is important to note that the use of ICS, as the primary reinforcer, circumvents the potentially confounding problem of requiring that any particular drive state be present in the test phase, because of the absence of the need for a preexisting drive or deprivation state.

Four previous studies (Knott & Clayton, 1966; Mogensen, 1965; Seward, Uyeda, & Olds, 1959; Stein, 1958) using ICS have resulted in conflicting outcomes. Stein (1958) obtained a free operant baseline in a two-lever chamber, and subsequently trained subjects in a lever free chamber using 400 presentations of a tone paired with ICS in a Pavlovian delayed conditioning format. Upon the the

completion of training subjects were returned to the two lever chamber. Pressing one lever consistently resulted in presentation of the tone while pressing the second lever had no effect. An increase, during testing, of response rate and a preference for the tone lever were interpreted as evidence of conditioned reinforcement. Subjects who, in a post training phase, failed to press a lever for ICS were used as controls. While Stein's results are encouraging, the lack of an appropriate within or between subjects control results in an inability to rule out both pseudoconditioning and sensitization as the factors responsible for positive results. (For an explanation of the relative value of within and between subject designs, see Appendix A, p. 64.) In order to effectively control for differences in responding attributable to pseudoconditioning and sensitization, the presentation of a second stimulus, randomly associated with ICS in training, is necessary (Rescorla, 1967). Alternately, in order to adequately control for counterconditioning effects, the use of a stimulus related to ICS in an explicitly unpaired format, is required.

Using an extinction paradigm, Seward, Uyeda, and Olds (1959) failed to replicate Stein's results. However, their use of a simultaneous pairing procedure in training, a procedure used as a control in Pavlovian conditioning (Hall, 1976), could easily account for the negative outcome of this effort.

Mogensen's (1965) failure to replicate is more difficult to interpret, primarily because of the minimal procedural information available to the reader. However, Mogensen and Cioe (1977), upon reviewing the field, subsequently concluded that the effect had been adequately demonstrated, an interpretation which is open to doubt. These authors accounted for the conflicting results of this set of studies, with reference to the possible importance of pre-experimental deprivation states in establishing an Sr effect. Indicating the use of non-deprived animals in the Seward, et al. (1959) and Mogensen (1965) studies, and the absence of relevant data on this point for Stein's (1958) and Knott and Clayton's (1966) procedures, the authors cited related evidence (DiCara, 1966; DiCara & Deutsch, 1966) suggesting the importance of deprivation states in the investigation of ICS produced Sr effects.

Knott and Clayton (1966) replicated Stein's results and added evidence regarding the issue of Sr durability, finding that partial pairing of the neutral stimulus with ICS increased Sr strength. These authors, however, used, as their control group, one which received a tone but no ICS in training. As with Stein's study, neither the within subjects control (no tone lever) nor the between subjects control adequately ruled out the possibility of pseudoconditioning effects.

The results of Stein's (1958) and Knott and Clayton's (1966) studies, while poorly controlled, suggest the

possibility empirically, that the necessary and sufficient condition for the establishment of a conditioned reinforcement effect is that of Pavlovian temporal contiguity. If confirmed, such results, involving no SD training component, would cast doubt on Keller and Schoenfeld's theoretical interpretation of Sr. Likewise, the absence of a response requirement or a consummatory response raises serious doubts regarding either the response contingent or the elicitation hypothesis.

Given the confusing state of the field of conditioned reinforcement and the questions regarding several of the major theoretical models currently available, a study is required which employs an adequately controlled, new learning design in order to establish, convincingly, the necessary and sufficient condition(s) for the formation of a conditioned reinforcement effect based upon ICS as primary reinforcement.

The present study attempted to accomplish this task. Five groups were employed, using a Pavlovian format similar to that of Stein (1958) and Knott and Clayton (1966). The new learning test required the development of an increased degree of preference for the temporally contiguous (paired) training stimulus over pretest (baseline) levels. Both baseline and test measures were obtained in a two-lever chamber in which delivery of the paired stimulus was contingent upon pressing one of the two levers.

Using within subjects comparisons, several different

groups were examined for preferences either for: (a) the paired stimulus versus no stimulus (paired/nothing group), (b) the paired stimulus versus a randomly presented stimulus (paired/random group), (c) the paired stimulus versus an explicitly unpaired stimulus (paired/explicitly unpaired group), (d) the randomly presented stimulus versus no stimulus (random/nothing group), or (e) the explicitly unpaired stimulus versus no stimulus (explicitly unpaired/nothing group).

Within group comparisons were employed, since some studies (e.g., Saltzman, 1949) suggest that this type of comparison reflects greater sensitivity to the conditioned reinforcement phenomenon. Between group comparisons for the paired/nothing, random/nothing, and explicitly unpaired/nothing groups were examined, as well as for the paired stimulus alone, the paired stimulus in the context of a random stimulus, and the paired stimulus in the context of an explicitly unpaired stimulus.

Critical to the demonstration of a genuine conditioned reinforcement effect would be the establishment of an increase in degree of preference for the lever producing the paired stimulus, in contrast to all increases for the various control comparisons. Having employed adequate sensitization (no stimulus), pseudoconditioning (random), and counterconditioning (explicitly unpaired) controls, an increased preference for the paired stimulus lever would indicate, unequivocally, a conditioned reinforcement

effect. It would also suggest, because of the use of a Pavlovian training procedure and ICS as the primary reinforcer, reexamination and elimination of several competing explanations of the Sr phenomenon.

The finding that one or more of the control conditions resulted in an equivalent or greater increase in lever preference to that for the paired stimulus, would cast serious doubt on the existence of the phenomenon as a reinforcement mechanism and would provide strong support for those authors (e.g., Bolles, 1975; Longstreth, 1971) who suggest that verification of the phenomenon is seriously questionable. But a demonstration of conditioned reinforcement in the present design would also yield information on the relative sensitivity of within and between subject comparisons to the phenomenon as well as whether "pseudoconditioning" effects of such preferences also exist. (For a more detailed theoretical discussion and literature review, see Appendix A, p. 62.)

Method

Subjects

Subjects were 93 experimentally naive adult male rats of the Sprague-Dawley strain, obtained from the Charles River Breeding Laboratory. Of the 93 animals used in the study, 41 failed to reach the pretraining criterion for a positive ICS effect, 4 lost their bipolar implants (see Implantation below) prior to the completion of the experimental procedure, 4 were eliminated as a result of

experimental error, 3 were eliminated as a result of equipment malfunction, and 1 died during a pretesting session, leaving a total of 40 subjects. Animals were housed singly and maintained on ad libitum feeding and drinking schedules throughout the experimental procedure. Subjects weighed an average of 524.9 g (range; 372.0-610.0 g) and averaged 130.5 days in age (range; 84-175 days) at the outset of the experimental procedure.

Apparatus

Subjects were trained and tested in three Scientific Prototype Manufacturing Corp. Model A100 lever boxes (length, 23.50 cm; width, 20.64 cm; height, 19.05 cm) (unaltered unless otherwise specified), housed in a light-proof and sound-deadened environmental chamber (length, 55.25 cm; width, 49.53 cm; height, 49.53 cm), constructed in the University of Rhode Island psychology department. The chamber was lighted continuously by a 6 W incandescent house light, mounted on the lower forward section of the left side wall. Ventilation was provided by an externally mounted fan which directed room air through a downwardly vented opening in the lower rear right wall of the environmental chamber. A similar vent in the lower front portion of the left wall permitted air to leave the closed chamber.

A flashing light stimulus was provided by a 15 W incandescent light mounted on the rear wall of the environmental chamber. The light was centered opposite the

transparent Plexiglas side wall of the lever box. A tone stimulus was provided by a Guam Model 4A05 speaker mounted on the upper center of the right side wall and activated by a Lehigh Valley adjustable tone generator, Model 381-05.

Three lever boxes were used for specific phases of the experimental procedure. Box #1, used for both pretesting and testing, contained two Scientific Prototype rat levers positioned on either side of one wall, approximately equidistant from the ceiling and floor grid. Box #2, used for pretraining, contained one Scientific Prototype rat lever, positioned centrally on the wall opposite the lever wall of Box #1. In Box #2, both the lever wall and the wall opposite the lever were painted black. Box #3, used for training, was similar to Box #1 and #2 with the exception that no manipulanda were present. The apparatus was automated primarily by solid state programming equipment with electro-mechanical equipment being used for relay and counting functions.

Procedure

Gentling. All subjects were handled for 10-15 s daily for three days prior to surgery. Following one day of post-surgery observation, subjects were handled daily for 10-15 s until the start of pretesting.

Implantation. Upon reaching a weight of 200-250 g (approximately 5-6 days after arriving in the colony), subjects were anesthetized, placed in a Scientific Prototype Model F small animal stereotaxic and implanted

with chronic, wound insulated bipolar electrodes (Plastics Products Model MS 303-2). Implants were directed at the lateral hypothalamus (-2.5 mm posterior to bregma, +1.8 mm lateral, and +8.3 mm vertical) (Hart, 1976), due to the minimal amount of forced movement produced by intracranial stimulation of this area. Brain stimulation was provided by a Grass Model SD9 stimulator at a frequency of 60 cps and a pulse-pair duration of 4 ms, at approximately 1.6 V, using a monophasic, normally polarized pulse. Stimulation was delivered through a metal-spring sheathed cable (Plastics Products Model [305-211] 40"-TT2 [CS]) extending from the center of the lid on the side opposite to the lid hinge. Subjects were housed in their home cage for a minimum of 14 days post-surgery recovery before the start of pretesting.

Pretesting. Subjects were randomly divided into five groups of 8 each. Using a free operant procedure, all subjects were pretested in the two-lever chamber, with the chamber lid remaining closed but unfastened. For subjects in Groups A (paired/nothing), B (random/nothing), and C (explicitly unpaired/nothing), responding on one lever produced either a 1.0 s, 1403 kHz, 104 dB tone or a 15 W flashing light, flashing at a rate of four flashes per second. Parameters of the tone and light were set at these levels to insure discriminability without aversiveness. Pressing the second lever produced nothing. The stimuli were counterbalanced across subjects for right-left

position, with equal numbers of subjects per group receiving either the tone or flashing light.

For subjects in Groups D (paired/random) and E (paired/explicitly unpaired), pressing one lever resulted in either the tone or the flashing light; pressing the second lever resulted in the second stimulus. Light and tone were counterbalanced across subjects relative to right and left positions.

Three measures were taken for each subject; (a) total number of presses per lever, (b) total number of stimuli presented (subjects could press more than once during the presentation of the 1.0 s stimulus), and (c) total time each lever was depressed.

All subjects were pretested in 40 min daily sessions for three consecutive days.

Pretraining. On the fourth day of the procedure, all subjects were placed in the single lever chamber, with the lid closed and fastened. Pressing the lever resulted in an immediate 0.5 s presentation of ICS on a continuous schedule of reinforcement. Subjects were required to self-train to a criterion of 200 presses in a 30 min session. Subjects were given access to the pretraining chamber for three consecutive daily sessions. Upon meeting the 200 press criterion in one session, pretraining was discontinued for that subject. For 9 subjects for whom there were equipment failures, or whose responding was appetitive but inefficient (as indicated by occasional

pauses), pretraining was extended to a maximum of seven sessions (4 subjects/four sessions, 3/five sessions, 1/six sessions, and 1/seven sessions).

The introduction of the pretraining phase subsequent to the baseline, in order to determine those animals for whom ICS was a reliable SR, differs from the procedures used by Stein (1958) and Knott and Clayton (1966). Stein used a procedure similar to the one described herein, but introduced it only after all animals has completed the experimental procedure. Stein's study, therefore, pretested, trained, and tested all subjects first, regardless of their final role in the study, a procedure which was avoided in this study for reasons of efficiency. Knott and Clayton, on the other hand, tested all subjects prior to initiating the experimental procedure in a distinctly different "Skinner" box from that used in the subsequent experiment, by reinforcing a corner position preference in the box. The procedure used in the present study was chosen (a) to avoid the possibility of sensitization effects confounding the baseline or pretest phase, as a result of prior exposure to ICS, and (b) to develop a reliable lever press response from which the "new" learning of a lever press preference would be substantially reduced in difficulty, in contrast to what it might have been in the absence of single lever press training.

Training. Following pretraining, all subjects were

trained using a Pavlovian, that is, non response-contingent, procedure in a chamber free of manipulanda, with the lid closed and fastened. Neutral stimuli (a tone and/or flashing light) of 1.0 s duration were delivered at a variable interval, averaging one presentation per minute. Training involved four daily sessions, averaging 100 min per session, with 100 presentations of one neutral stimulus for Groups A, B, and C, and 100 presentations of both stimuli for Groups D and E. On Day 1 of training, all subjects received 100 presentations of 0.5 s of ICS. On Days 2, 3, and 4, subjects received ICS at a rate averaging 50 % of Day 1. Thus, the ratio of ICS presentation to paired stimulus presentation was 1:1 for Day 1 and 1:2 for Days 2, 3, and 4.

For Group A, the neutral stimulus and ICS were paired using a Pavlovian forward conditioning procedure with the onset of ICS occurring 0.5 s after the onset of the neutral stimulus (an interstimulus interval considered within the optimal range for Pavlovian conditioning [Hilgard & Marquis, 1940]) and the offset of both occurring simultaneously. On Day 1, ICS followed the onset of the neutral stimulus on all 100 presentations of the latter. On Days 2, 3, and 4, ICS followed the neutral stimulus on an average of one-half of all presentations of the latter.

For Group B, the neutral stimulus and ICS were presented independently in a "truly random" format

(Rescorla, 1967). On Day 1, both the neutral stimulus and ICS were presented 100 times. On Days 2, 3, and 4, the neutral or random stimulus was presented 100 times. The ICS was presented on an average of 50 times. The number of total overlaps of the neutral stimulus and ICS was measured, as was the number of Pavlovian forward overlaps (the number of overlaps in which the onset of the neutral stimulus preceded the onset of ICS.)

For Group C, the neutral stimulus and ICS were presented independently in an "explicitly unpaired" format. This group was identical to Group B, with the exception that the onset of the neutral stimulus was blocked if the ICS was being presented, or conversely, that the onset of the ICS was blocked if the neutral stimulus was being presented, thereby eliminating all overlaps of the neutral stimulus and ICS.

For Group D, one neutral stimulus was paired with ICS, as in Group A, using a Pavlovian forward pairing procedure with the onset of ICS occurring 0.5 s after the onset of the neutral stimulus and the offset of both occurring simultaneously. A second stimulus was presented in a "truly random" format relative to ICS presentation. Total and forward overlaps were measured as in Group B.

For Group E, one neutral stimulus was paired with ICS, using a Pavlovian forward pairing procedure with the onset of ICS occurring 0.5 s after the onset of the neutral stimulus and the offset of both occurring simultaneously.

A second stimulus was presented in an explicitly unpaired format relative to ICS presentation.

Testing. The testing procedure followed one day after the last training session and was identical to the pretesting procedure, with the exception that one session was used for testing, in contrast to the three sessions used for pretesting. As in the pretesting phase, three dependent measures were taken for each subject; (a) total presses per lever, (b) total stimuli presented, and (c) total time each lever was depressed. (For a more concise outline of experimental phases, see Appendix A, Table A1, p. 133.)

Results

Six $3 \times 2 \times 2$ ANOVAs, with repeated measures across the latter two factors, were used to analyze the data. The six ANOVAs were divided into two subsets, each of which analyzed data from the same three dependent measures, but across different groups of subjects, within each subset. Factor A for three of the analyses compared three different groups (Groups A, D, & E; see Method, p. 14), each of which were presented with a Pavlovian pairing of one stimulus with ICS during training. These groups differed according to the class of comparison control conditions for use with the ICS-paired stimulus (A1, no stimulus, Group A; A2, randomly presented stimulus, Group D; A3, explicitly unpaired stimulus, Group E). Factor A for the remaining three analyses compared three groups (Groups A, B, & C)

each of which received only one stimulus on one lever, varied by training configuration (A1, paired, Group A; A2, random, Group B; A3, explicitly unpaired, Group C), with "no stimulus" on the alternate lever.

For one subset of three analyses, Factor B compared the paired (B1) stimulus with its control (B2) condition (no stimulus, random stimulus, or explicitly unpaired stimulus). For the second subset of analyses, Factor B compared the critical comparison stimulus (paired, random, or explicitly unpaired) (B1) with its "no stimulus" (B2) control. Factor C compared measures taken during pretesting with the same measures taken subsequent to training, specifically assessing average responding across three days of pretesting (C1) with responding on the one day of testing (C2). Table 1 represents the design and conditions for each of the two sets of analyses.

For each set of three analyses, three dependent measures were assessed; (a) frequency of lever presses, (b) frequency of stimulus presentations obtained as a result of lever pressing, and (c) cumulative duration of lever depression, measured in seconds. Results of the stimulus presentation and duration measures were similar to results of the lever press measures for each subset of analyses. For this reason, primary emphasis will be given to a presentation of the results of the lever press analyses. Stimulus presentation and duration measure results will be included only in the limited cases where

Table 1

Schematic Design of ANOVA SubsetsSubset 1

	C1	C2
	Pretest	Test
B1 Paired	A1B1C1	A1B1C2
A1 Group*		
B2 No Stimulus	A1B2C1	A1B2C2
B1 Paired	A2B1C1	A2B1C2
A2 Group		
B2 Random	A2B2C1	A2B2C2
B1 Paired	A3B1C1	A3B1C2
A3 Group		
B2 Explicitly Unpaired	A3B2C1	A3B2C2

Subset 2

	C1	C2
	Pretest	Test
B1 Paired	A1B1C1	A1B1C2
A1 Group		
B2 No Stimulus	A1B2C1	A1B2C2
B1 Random	A2B1C1	A2B1C2

A2 Group

B2 No Stimulus

A2B2C1

A2B2C2

B1 Explicitly Unpaired

A3B1C1

A3B1C2

A3 Group

B2 No Stimulus

A3B2C1

A3B2C2

*n = 8 for each group.

those results differ from the lever press results. (See Appendix A3 for a complete discussion of significant results in analyses of the stimulus presentation and lever depression duration measures, p. 135.)

Because of the large number of significant outcomes, the presentation of the results will begin with a summary of the major outcomes of both lever press analyses. F values relative to these outcomes will be presented in the more detailed discussion of each analysis which follows.

Two major outcomes are apparent from a joint examination of both lever press analyses. First, overall responding increased in both analyses from the pretest to test phase. Secondly, this difference is accounted for primarily by a significant increase in responding for the Pavlovian paired stimulus from pretest to test phases. In those limited cases where increases from pretest to test for control conditions were significant, effects were weak relative to paired comparisons.

In the comparison of groups in the first analysis, each of which received a paired stimulus contrasted against a control condition, responding in the paired/nothing group was the strongest, followed by responding in the paired/random group, with responding for the paired/explicitly unpaired group being the weakest. No parallel effect was found for the second analysis, using a between subjects comparison of paired, random, and

explicitly unpaired conditions.

Accounting for (a) significant interactions in both analyses, (b) the significant between groups difference in the first analysis, and (c) a preference for the paired stimulus as compared to control conditions in the first analysis, was a significant preference for the paired stimulus in the test phase.

Analysis 1

Means and standard deviations from the first analysis (measuring frequency of lever presses across paired/nothing, paired/random, and paired/explicitly unpaired groups) are represented in Table 2. Results of a test of the assumption of homogeneity of variance (F_{max} [$12, 7$] = 16.49, n.s.), fail to reject the null hypothesis. (For all tests of the assumption of homogeneity of variance, a violation was assumed for any F_{max} value exceeding the table value of $p < .01$.)

Overall results indicate a significant difference (a) between groups receiving the paired stimulus in the three different control conditions, (Factor A) ($F[2, 21] = 5.37$, $p < .05$), (b) a significant preference for the the paired stimulus as compared to the control condition (Factor B) ($F[1, 21] = 6.13$, $p < .05$) and (c) a significant increase in responding from pretest to test phases (Factor C) ($F[1, 21] = 24.43$, $p < .05$), as well as a stimulus (B) by pretest/test (C) interaction ($F[1, 21] = 8.66$, $p < .05$).

While no ABC interactions are indicated in the overall

Table 2

Means and Standard Deviations of Lever PressMeasure for Within Subjects Controls

Group	Stimulus		Pretest	Test
A1 (Group A)	Paired	<u>M</u>	30.38	60.75
		<u>SD</u>	23.77	28.79
	No Stimulus	<u>M</u>	28.88	35.38
		<u>SD</u>	15.50	10.70
A2 (Group D)	Paired	<u>M</u>	21.50	42.38
		<u>SD</u>	14.00	23.69
	Random	<u>M</u>	19.50	31.75
		<u>SD</u>	9.27	16.89
A3 (Group E)	Paired	<u>M</u>	11.50	26.63
		<u>SD</u>	7.54	16.79
	Explicitly Unpaired	<u>M</u>	12.75	23.88

SD

7.09

12.84

results for any of the first three analyses, a priori predictions of the locus of change within each group dictated the assessment of follow-up effects for B at each level of AC and C at each level of AB. Specifically, it was predicted that critical comparisons in assessing increased preferences for paired stimuli and controls would include pretest versus test (C1 vs. C2) comparisons for each stimulus condition (B) within each group (A). Of somewhat less importance, are comparisons of preferences of each group for the critical stimulus (B1) as compared to its control (B2) condition separately in the test (C2) phase, as well as the pretest (C1) phase.

Of greatest importance to the purpose of this study is the finding that responding for the paired stimulus (B1) in all three groups significantly increased from the pretest (C1) to the test (C2) phase ($F[1, 63] = 20.57, p < .05$, C at A1B1; $F[1, 63] = 9.72, p < .05$, C at A2B1; $F[1, 63] = 5.10, p < .05$, C at A3B1). In contrast, analyses of the pretest to test increases for the control conditions failed to yield significant results. Comparisons of effect size indicate that increased responding in the paired/nothing group was more than twice that of the paired/random group and almost five times that of the paired/explicitly unpaired group. Additionally, increased responding for the paired/random group was more than twice that of the paired/explicitly unpaired group. Omega squared values for the comparisons

of relative magnitude yield results of 0.067 for C at A1B1, 0.030 for C at A2B1, and 0.014 for C at A3B1, resulting in a relative magnitude for C at A1B1 of 2.23 times that of C at A2B1 and 4.79 times that of C at A3B1 and a relative magnitude for C at A2B1 of 2.14 times that of C at A3B1.

In contrast to the lever press measure, a significant increase from pretest to test, for the stimulus presentation and duration measures, was also found for the random control ($F[1, 63] = 4.53, p < .05$, stimulus presentation; $F[1, 63] = 4.72, p < .05$, lever depression duration). Comparisons of relative effect size indicated, however, that increased responding for the paired stimulus was almost three times that of the random stimulus for the stimulus presentation measure and more than one and one half times that of the duration measure (see detailed discussion in Appendix A, Analysis 2 and Analysis 3, p. 142.)

Results of these analyses indicated that for the paired/nothing (A1) group only the paired (B1) stimulus in the test (C2) phase was preferred significantly more than the "no stimulus" (B2) control ($F[1, 63] = 14.38, p < .05$). No other significant pairwise differences for B at AC were identified.

While no AB interaction was significant for this first analysis, or either of the remaining two within this subset, the assessment of between group differences in responding for one (A1) versus two stimuli (A2 & A3) was of

a priori importance. For this reason, the analysis of simple main effects for A at each level of both B and C was performed. Follow-up tests (B at each level of A) indicated that subjects responded significantly more for the paired (B1) stimulus in the test (C2) phase than in the pretest (C1) phase [$F(2, 21) = 7.13, p < .05$, A at B1; $F(2, 21) = 4.85, p < .05$, A at C2). Newman-Keuls follow-up tests further indicated that subjects in the paired/nothing (A1) group responded significantly more than subjects in the paired/random (A2) and paired/explicitly unpaired (A3) groups and that paired/random subjects responded more than paired/explicitly unpaired subjects. While results for the stimulus presentation and duration measure were similar, results of the Newman-Keuls analysis for the stimulus presentation measure failed to yield significant differences for the paired/nothing versus paired/random comparison.

Follow-up tests further indicated that paired/nothing (A1) subjects responded more on the lever producing the paired (B1) stimulus than on the lever producing "no stimulus" (B2) [$F(1, 21) = 7.90, p < .05$]. The finding that subjects, overall, responded significantly more for the paired (B1) stimulus in the test (C2) than in the pretest (C1) phase is confirmed by a follow-up analysis of the BC interaction [$F(1, 21) = 8.17, p < .05$].

When compared at the individual group level, all groups were found to increase overall responding from

pretest (C1) to test (C2) ($F[1, 21] = 10.76, p < .05, A1$; $F[1, 21] = 8.64, p < .05, A2$; $F[1, 21] = 5.45, p < .05, A3$). While follow-up tests indicate that responding overall was greater in the test (C2) phase than in the pretest (C1) phase for both the paired (B1) stimulus and its control (B2) ($F[1, 21] = 33.06, p < .05, C \text{ at } B1$; $F[1, 21] = 6.70, p < .05, C \text{ at } B2$), comparisons of relative magnitude indicate that the paired stimulus change from pretest to test was more than five times that of the control stimulus. Omega squared values for the latter comparison yield results of 0.108 for C at B1 and 0.019 for C at B2, resulting in a relative magnitude for C at B1 of 5.68 times that of C at B2.

Analysis 2

The second analysis measured frequency of lever presses across paired/nothing, random/nothing, and explicitly unpaired/nothing groups. The means and standard deviations of this analysis are represented in Table 3. Results of a test of the assumption of homogeneity of variance ($F_{\max}[12, 7] = 13.32, n.s.$), failed to reject the null hypothesis.

Results of the overall analysis indicate an increase in responding from pretest to test (Factor C) ($F[1, 21] = 10.46, p < .05$), a significant interaction of the group variable (Factor A) with the stimulus variable (Factor B) ($F[2, 21] = 5.84, p < .05$) and a significant three-way (Groups x Stimulus x Pretest/Test) interaction ($F[2, 21] =$

Table 3

Means and Standard Deviations of Lever PressMeasure for Between Subjects Controls

Group	Stimulus		Pretest	Test
A1 (Group A)	Paired	<u>M</u>	30.38	60.75
		<u>SD</u>	23.77	28.79
	No Stimulus	<u>M</u>	28.88	35.38
		<u>SD</u>	15.50	10.70
A2 (Group B)	Random	<u>M</u>	23.38	28.88
		<u>SD</u>	13.14	22.83
	No Stimulus	<u>M</u>	33.75	36.25
		<u>SD</u>	20.11	31.75
A3 (Group C)	Explicitly Unpaired	<u>M</u>	20.00	26.75
		<u>SD</u>	8.70	10.54
	No Stimulus	<u>M</u>	23.50	37.38

SD

9.50

20.09

4.67, $p < .05$). For the lever depression duration measure, a significant group (A) by pretest/test (C) interaction was found ($F[2, 21] = 4.14$, $p < .05$) and a significant interaction of the stimulus variable (B) with the pretest/test (C) variable ($F[1, 21] = 5.78$, $p < .05$).

Follow-up tests of pretest/test differences for each group indicate that only subjects in the paired/nothing (A1) group, responded significantly more in the test (C2) phase as compared to the pretest (C1) phase ($F[1, 21] = 9.95$, $p < .05$). While this result is supported, for the most part, by the stimulus presentation and duration measures, it was found that overall responding, as measured by lever depression duration, increased from pretest (C1) to test (C2) for the explicitly unpaired/nothing (A3) group, ($F[1, 21] = 8.43$, $p < .05$).

Follow-up tests (of B (Stimulus) at levels of C (Pretest/Test) and C at levels of B) also indicate a significant increase in responding for the critical comparison stimulus (B1; paired, random, and explicitly unpaired) in the test (C2) phase as compared to the pretest (C1) phase [C at B1] ($F[1, 21] = 12.74$, $p < .05$). No similar increase is apparent for the "no stimulus" (B2) control for the lever press measure. While results are identical for the stimulus presentation measure, a significant increase in responding for the "no stimulus" (B2) control from pretest (C1) to test (C2) is indicated for the duration

measure ($F[1, 21] = 6.68, p < .05$). Comparisons of relative effect sizes indicate, however, an effect for the critical comparison stimulus of more than four and one half times that of the "no stimulus" control (see detailed discussion in Appendix A, Analysis 6, p. 157.)

Follow-up analyses of between group differences (follow-up tests of the AB interaction) indicate that differences across groups in levels of responding were found for the critical comparison (B1) stimulus but not the "no stimulus" (B2) lever ($F[2, 21] = 4.14, p < .05$). Newman-Keuls follow-up tests further indicate that subjects in the paired/nothing (A1) group responded more for the paired (B1) stimulus than did subjects in either comparison group for the control (B2) stimulus (random or unpaired). Also indicated is the result that paired/nothing (A1) subjects preferred the paired (B1) stimulus overall as compared to the "no stimulus" (B2) control ($F[1, 21] = 6.86, p < .05$). The ABC interaction indicates, however, that this is more clearly the case in the test (C2) phase than the pretest (C1) phase (see below). While similar effects are not found for the stimulus presentation measure, differences across groups for the paired (B1) stimulus are found for the lever depression duration measure, as well as between group differences for the test (C2) phase ($F[2, 21] = 6.78, p < .05$). Newman-Keuls follow-up tests of the latter indicate significantly greater responding in the test (C2) phase by paired/nothing (A1) subjects as compared to

random/nothing (A2) and explicitly unpaired/nothing (A3) subjects.

Follow-up tests of the ABC interaction indicate a preference, in the paired/nothing (A1) group only, for the paired (B1) stimulus in the test (C2) phase as compared to the pretest (C1) phase [$F(1, 63) = 18.74, p < .05$]. In the test (C2) phase, the paired/nothing group also preferred the paired (B1) stimulus in contrast to the "no stimulus" (B2) control [$F(1, 63) = 13.08, p < .05$]. Of critical importance is the outcome that no other significant effects, pretest to test or otherwise are identifiable at this level of follow-up analysis for random, explicitly unpaired and "no stimulus" controls. In other words, compared to all possible controls responding for the paired stimulus only, increased from pretest to test.

While this latter finding is the case for the lever press measure, follow-up analyses of the stimulus presentation measure indicate a significant increase in responding from pretest (C1) to test (C2) for the "no stimulus" (B2) control for subjects in the explicitly unpaired/nothing (A3) group [$F(1, 63) = 4.40, p < .05$]. The same effect is indicated for the duration measure [$F(1, 63) = 5.20, p < .05$], as well as an increase in responding for the explicitly unpaired stimulus (B1) [$F(1, 63) = 6.66, p < .05$]. Comparisons of effect size indicate that increased responding for the explicitly unpaired stimulus was approximately one and one third times that of the "no

stimulus" control (see detailed discussion in Appendix A, Analysis 6, p. 157.)

Control Analyses: Counterbalancing

In order to rule out possible confounding effects of experimentally counterbalanced factors, a number of additional "control" analyses were performed. Three $3 \times 2 \times 2$ ANOVAs, with repeated measures across the second factor, were performed across the three groups receiving only one stimulus to determine relative preferences for light and tone stimuli. Factor A was the between groups factor (paired/nothing, random/nothing, explicitly unpaired/nothing), Factor B, the modality factor (light/tone) and Factor C, the stimulus factor (stimulus/no stimulus). Analyses were performed for each of the three dependent measures indicated in the main body of this section. An overall preference for the light over the tone (Factor B), $F(1, 18) = 9.58, p < .05$, is consistent across groups for the lever depression duration measure. No other significant effects were obtained. $F_{\max}$ values for the three analyses ($F_{\max} [6, 3] = 11.90, p > .05$, lever press; $F_{\max} [6, 3] = 8.85, p > .05$, stimulus presentations obtained; $F_{\max} [6, 3] = 21.37, p > .05$, lever depression duration) failed to reject the null hypothesis.

Three 2×2 ANOVAs, with repeated measures across the latter factor, were performed for the two groups receiving two stimuli to determine relative preferences for light and tone. Factor A was the between groups factor

(paired/random, paired/explicitly unpaired) and Factor B, the modality factor (light/tone). Again, the three dependent measures were those assessed in the main analyses. Results of the tests of the assumption of homogeneity of variance failed to reject the null hypothesis for two dependent measures ($F_{\max} [4, 7] = 4.40$, $p > .05$, lever press; $F_{\max} [4, 7] = 5.06$, $p > .05$, stimulus presentations obtained), but violated the assumption of homogeneity for one dependent measure ($F_{\max} [4, 7] = 32.03$, $p < .01$), lever press duration. However, because the $F_{\max}$ is considered robust with respect to moderate violations of the assumption of homogeneity (Box, 1953), the analysis of the results for the latter dependent measure proceeded. Again, a stronger preference for the light is indicated for the lever press measure ($F [1, 14] = 9.85$, $p < .05$) and for the stimulus presentation measure ($F [1, 14] = 7.47$, $p < .05$).

Three 5×2 ANOVAs, with repeated measures across the second factor, were performed to determine right/left preferences across groups. Factor A was the between groups factor across the five training conditions. Factor B was the right/left preference factor. Once again, three analyses were performed for each of the three dependent measures used in the main analyses. $F_{\max}$ results failed to reject the null hypothesis for two dependent measures ($F_{\max} [10, 7] = 10.08$, $p > .05$, lever press; $F_{\max} [10, 7] = 8.82$, $p > .05$, stimulus presentations obtained). For the third dependent measure, the ANOVA was considered robust (Box,

1953) relative to a moderate violation of the assumption of homogeneity of variance, $F_{\max}(10, 7) = 30.47, p < .01$. For all three analyses, the right lever was preferred relative to the left lever for all groups ($F[1, 35] = 4.33, p < .05$, lever press; $F[1, 35] = 13.23, p < .05$, stimulus presentations obtained; $F[1, 35] = 19.65, p < .05$, lever depression duration). While the preference for the lever producing the light and the preference for the right lever were consistent across subjects, and, therefore, considered to have had no effect on the major outcomes of the study, the importance of the counterbalancing of stimulus and lever position is underlined by the results of these control analyses.

Control Analyses: Confounding Variables

In order to rule out the influence of additional potentially confounding variables, seven one way ANOVAS were performed across groups to assess between group differences relative to age, percent of training pairings on Days 2, 3, and 4 of training, weight, number of pretraining days to criterion, length of training sessions, number of pretraining lever presses to criterion (in the 30 min session in which criterion was met), and length of the experimental procedure.

Results of tests of the assumption of homogeneity of variance failed to reject the null hypothesis for five of the ANOVAS (age, $F_{\max}[5, 7] = 6.53, n.s.$; weight, $F_{\max}[5, 7] = 5.83, n.s.$; number of pretraining days to

criterion, $F_{\max} [5, 7] = 4.04$, n.s.; length of training sessions, $F_{\max} [5, 7] = 8.87$, n.s.; number of pretraining lever presses to criterion, $F_{\max} [5, 7] = 4.70$, n.s.). The $F_{\max}$ result for the analysis of the percent of training pairings ($F_{\max} [5, 7] = 43.16$, $p < .01$) reflects a moderate violation of the assumption of homogeneity of variance for which the overall analysis should be relatively robust (Box, 1953). The $F_{\max}$ result for the analysis of length of experimental procedure ($F_{\max} [5, 7] = 184.49$, $p < .01$) represents a serious violation of the assumption of homogeneity of variance. For this reason, data from the last analysis were transformed using a natural log transformation to reduce the level of error variance. The resulting $F_{\max}$ value ($F_{\max} [5, 7] = 16.77$, $p < .01$) reflects a negligible violation of the assumption of homogeneity of variance.

Of the seven analyses, three indicate significant differences. Results of the analysis of differences in age ($F[4, 35] = 4.23$, $p < .05$) indicate that the paired/nothing group was older at the outset of the experiment than the paired/explicitly unpaired, random/nothing, and explicitly unpaired/nothing groups (mean difference = +32.53). (Means and standard deviations of between group differences in age are represented in Appendix Q, Table Q2, p. 337.) Results of the analysis of percent of training pairings ($F[4, 35] = 3.58$, $p < .05$) indicate that paired/random subjects received significantly more pairings of the paired stimulus with ICS

on Days 2, 3, and 4 of training, as compared to paired/explicitly unpaired subjects. (Means and standard deviations of between group differences in percent of training pairings are represented in Appendix R, Table R2, p. 340.) While the results of the analysis of between group differences in weight are significant ($F[4, 35] = 2.68, p < .05$), Newman-Keuls follow-up tests failed to yield any pairwise differences. It is likely that the significant difference is accounted for by the combination of the paired/nothing (A1) and paired/random (A2) groups (mean of A = 569.69; mean of B = 568.81) as compared to the combination of the paired/explicitly unpaired (A3) and explicitly unpaired/nothing (A5) groups (mean of C = 495.25; mean of E = 484.25).

Because of the possibility that overlaps of the random stimulus with ICS might lead to sufficient pairing for learning to occur in the paired/random and the random/nothing groups, the number of total (forward, backward, and simultaneous) overlaps and the number of forward overlaps were measured for each subject. For the paired/random group, the mean number of total overlaps was 10.75 and the mean number of forward overlaps was 4.5.

Of the 400 random stimuli presented to paired/random subjects, therefore, 2.7% overlapped with ICS, with only 1.1% of the 400 occurring in the optimal forward pairing configuration. As a result of continuous pairing of the paired stimulus and ICS on Day 1 of training, and only

partial pairing (50%) on Days 2, 3, and 4, the paired stimulus overlapped with ICS on 64% of the occasions (400), that is, (i.e., the paired stimulus presentations), was presented. While the paired stimulus preceded and overlapped with ICS 100% of the times that it was presented, the random stimulus overlapped for 4.2% of the ICS presentations and preceded and overlapped with ICS for 1.8% of the presentations of the latter.

For the random/nothing group, the mean number of total overlaps was 11.25 and the mean number of forward overlaps was 4.75. Of the 400 random stimuli presented, 2.8% overlapped with ICS, with only 1.2% occurring in the optimal forward pairing configuration. The random stimulus overlapped for 4.6% of the ICS presentations and preceded and overlapped with ICS for 1.9% of the presentations of the latter.

Discussion

Results of the three analyses (lever press, stimulus presentation, lever depression duration) comparing the Pavlovian conditioned groups to their respective controls (no stimulus, random stimulus, explicitly unpaired stimulus) offer unquestionable evidence of the establishment of a conditioned reinforcement effect. This finding is further bolstered by results of the second set of three analyses comparing the critical comparison stimulus (paired, random, explicitly unpaired) with its "no stimulus" control.

Subjects in each of the three Pavlovian pairing groups significantly increased responding on the lever delivering the paired stimulus, from pretest to test phases, for all three dependent measures. No significant differences from pretest to test were noted for the lever producing either no stimulus or for the lever producing the explicitly unpaired stimulus for all three dependent measures. While for the lever press measure, no difference was found from pretest to test for the random control, a significant increase was found for the stimulus presentation measure and the lever depression duration measure. As indicated in the results section, however, the magnitude of the effect for the conditioned stimulus (CS) is approximately three times that of the randomly paired stimulus for the stimulus presentations measure and approximately one and one-half times that of the random effect for the lever depression duration measure.

In the second set of analyses (in which the original data from the paired/nothing group were again included), a significant increase from pretest to test was noted for the lever delivering the paired stimulus, for all three dependent measures. For the stimulus presentation and duration measures, the lever delivering no stimulus in the explicitly unpaired/nothing group showed a significant increase from pretest to test. For the lever press measure, the results approach but do not reach significance. For the duration measure, a significant

increase from pretest to test was noted for the explicitly unpaired stimulus as well.

A potential problem resulting from the finding that the relative magnitude for the unpaired stimulus was approximately one and one-third times that of the no stimulus lever, is explicable in two ways. First, the lever depression duration result is opposite to that of both the stimulus presentation outcome and the lever press measure. Secondly, a subject by subject review of the data revealed two subjects whose responding for the unpaired stimulus declined for the lever press and stimulus presentation measure, while increasing for the duration measure. In other words, while these two subjects pressed fewer times and consequently received fewer explicitly unpaired stimulus presentations, they held the lever depressed for longer periods of time with each press. It is, therefore, likely that these two subjects account for this anomaly in the data. While it is possible that the performance of these subjects indicates a true variation in the learned effect, it should be noted that, regardless of experimental phase, subjects were observed occasionally resting in a position which held the lever depressed. Because the duration of the stimulus was consistently 1.0 s, longer durations of lever depression would decrease access to the Sr, and it is probable that the results of the two subjects in question reflect the latter explanation. The absence of a systematic pattern of

similar results among the remaining subjects further supports such an explanation.

Three additional patterns of outcome warrant comment. First, in all six analyses, absolute level of responding increased from pretest to test. However, the overall statistically significant differences from pretest to test in the six analyses are accounted for by increases in responding only for the forwardly paired stimulus only from pretest to test. No significant differences from pretest to test were found for the random, explicitly unpaired, and no stimulus conditions. However, while statistical analyses fail to offer the necessary support, the data nevertheless suggest the existence of both pseudoconditioning and sensitization effects for the control stimuli and "no stimulus" controls.

Secondly, responding was significantly greater for both paired/nothing and paired/random subjects than for the paired/explicitly unpaired group on all three measures, with the paired/nothing group being superior to the paired/random group on the lever press and duration measures. On the other hand, paired/nothing subjects, in comparison to random/nothing subjects and explicitly unpaired/nothing subjects, responded at a significantly higher level only for the duration measure. No other significant between groups differences were obtained among the comparisons of these groups.

An examination of potential confounding variables

which could affect between groups differences, such as age, weight, pretraining lever presses to criterion, etc., offers no explanation for this pattern of between group differences in overall level of responding in contrast to the absence of a similar effect for paired/nothing, random/nothing, and explicitly unpaired/nothing comparison groups. While differences in absolute numbers across groups appear to exist in the pretest phase for the paired stimulus groups, only in the test phase were their differences statistically significant. Measures of relative magnitude (see Results, p. 24) reflect these between group differences. It is possible that subjects in the two-stimulus groups were low responders, that is, they would have yielded low response frequencies regardless of the stimulus conditions. Mixed support for this position is offered from a review of mean levels of baseline responding for all five groups (see Tables 2, p. 22, & 3, p. 28) indicating that paired/nothing and random/nothing groups are relatively equivalent (in absolute numbers), with explicitly unpaired/nothing and paired/random groups being somewhat lower, and relatively equal, while the paired/explicitly unpaired group, for whom the paired stimulus change from pretest to test was weakest, is substantially lower than any of the other four. However, an examination of the changes in lever press responding from pretest to test for each lever, as represented in Table 4, reveals that increases for the two-stimulus groups

Table 4

Mean Changes in Lever Presses fromPretest to Test for Each Lever

Group	Critical	Control
	Comparison Lever	Lever
Paired/Nothing	+30.37	+6.50
Paired/Random	+21.88	+12.25
Paired/Explicitly Unpaired	+15.13	+11.13
Random/Nothing	+5.50	+2.50
Explicitly Unpaired/Nothing	+6.75	+13.88

were moderate to large in comparison to other groups. An argument favoring low initial response frequency as an explanation would predict relatively low increases from pretest to test for groups whose initial response levels were low. Given these mixed to contradictory results, a low response frequency explanation finds little overall support.

The results, therefore, suggest that the differences are largely attributable, to a context effect, that is, to the fact that the paired/nothing group, as well as the random/nothing and explicitly unpaired/nothing groups, were exposed to one stimulus while the paired/random and paired/explicitly unpaired groups were exposed to two stimuli. It is possible that the presence of a control stimulus in pretesting and testing, in contrast to a "no stimulus" control, resulted in a somewhat weaker conditioned reinforcement effect for the paired stimulus, perhaps as a result of a stimulus competition or overshadowing factor for the two-stimulus groups. Pretest to test increases in responding for each lever, as represented in Table 4, suggest a context effect for the paired stimulus in the one- versus two-stimulus groups, based on the observation that, in absolute numbers, the changes for the two-stimulus groups are approximately two-thirds to one-half that of the paired/nothing (one-stimulus) group. That is, responding for the paired

stimulus increased more from pretest to test when only one stimulus was present than when there were two. Further support is offered for the existence of influence from a context stimulus, specifically, that the lever producing no stimulus in the explicitly unpaired/nothing group reflects a greater increase than for the no-stimulus lever in the paired/nothing context. This latter negative context effect suggests that a non-predictive (or explicitly unpaired) stimulus may inhibit sensitization effects substantially less than a predictive (or paired) stimulus.

The third and last outcome of importance is the finding that for the random lever in the paired/random group, the increase in responding from pretest to test was significant for both the stimulus presentation and duration measures, and approached significance for lever press measure. However, for the random lever in the random/nothing group, on the other hand, no comparable effect was found. Further, no effect was obtained for the explicitly unpaired lever in the paired/explicitly unpaired group.

Again, the results are suggestive of a context effect for the two stimulus condition (paired/random) as compared to the one stimulus condition (random/nothing. That is, the between groups difference between paired versus random lever increases in pretest to test levels of responding was substantially greater than the within groups paired versus random difference, again reflecting a possible

overshadowing or stimulus competition factor. Further support for a context variable is provided by the observation that the differences between levels of responding for the paired/random group, and the random/nothing are negligible. The presence of a significant difference from pretest to test for the random stimulus in the paired/random group suggests either a pseudoconditioning or stimulus generalization effect. Before firm conclusions may be drawn, further investigation of the interaction between magnitude of Sr effect and initial level of responding is necessary.

Empirically, the results establish, from both within groups and between groups comparisons, that the necessary and sufficient condition for the formation of a conditioned reinforcement effect is that of traditional Pavlovian temporal contiguity. Both between and within groups comparisons employed control conditions identical to the Pavlovian conditioned stimulus except for their temporal relationship (random or explicitly unpaired) to the primary reinforcer. Further, the use of a new learning criterion (the development of an increased preference for the paired lever) required the Sr to be employed in a manner basic to the operational definition of a reinforcer.

It is important to note that this effect was achieved in the absence of a drive state resulting from experimentally induced deprivation (e.g., hunger resulting from food deprivation). In contrast to Mogensen and Cioe's

(1977) suggestion to the contrary, it would appear that a specific drive state is unnecessary for the establishment of an Sr effect. From a theoretical perspective, this outcome poses a problem for models of Sr operation which adhere to drive reduction as a fundamental explanatory mechanism.

From a theoretical perspective, the results of this study lay to rest the contention that Sr effects are always explicable by some other, more parsimonious mechanism (Bolles, 1975). The most obvious alternative explanation rendered inapplicable in the present study, is the discrimination hypothesis (Crum, Brown, & Bitterman, 1951; Elam, Tyler, & Bitterman, 1954; Wike, 1966). This theoretical approach suggests that greater stimulus generalization decrement for control conditions, using an extinction paradigm, accounts for the differential effect which is traditionally and unnecessarily attributed to conditioned reinforcement, that is, that subjects discriminate the cessation of primary reinforcement more efficiently in control conditions than experimental conditions due to the greater contrast between training and testing for controls. While the results of this study do not cast doubt on the discrimination hypothesis as it may apply to the positive results of some studies employing extinction paradigms, they do establish the legitimacy of conditioned reinforcement as a separate empirical phenomenon.

The present results also offer a direct challenge to two additional and distinctly different theoretical perspectives regarding the establishment and operation of conditioned reinforcers, that is, the discriminative stimulus hypothesis (Keller & Schoenfeld, 1950) and the response contingent hypothesis (Bilbrey & Winokur, 1975; Silverstein & Lipsett, 1974). Keller and Schoenfeld, (1950) proposed that a neutral stimulus must function as a discriminative stimulus in order for it to become a conditioned reinforcer, that is, that the establishment of a stimulus as an SD is a necessary condition for the establishment of an Sr. While a number of studies (Dinsmoor, 1950; Klein, 1959; Saltzman, 1949; Schoenfeld, Antonitis, & Bersh, 1950) have offered support for the hypothesis that a discriminative stimulus, by virtue of its SD properties, is sufficient to function as a conditioned reinforcer, these studies do not demonstrate that SD properties are necessary for the establishment of a conditioned reinforcement effect. The Pavlovian training component of the present study required no discriminative stimulus function for the neutral stimulus. It is suggested, therefore, that while the establishment of a neutral stimulus as a discriminative stimulus, may produce a conditioned reinforcer, it is the contiguous temporal relationship with the primary reinforcer which is the necessary feature of that procedure for establishing the effect.

Silverstein and Lipsett (1974), and Bilbrey and Winokur (1975) suggest that paired delivery of the neutral stimulus and primary reinforcer in training must be response contingent in order to establish a conditioned reinforcement effect, that is, that the subject must perform some response to obtain the neutral stimulus/primary reinforcer pair in order to establish a conditioned reinforcement effect. The results of the present study suggest, however, that a response contingency is not a necessary condition for the establishment of an SR, as no operant was necessary with the use of a Pavlovian training procedure and ICS as the SR.

While the present results eliminate the need to consider the traditional experimentally defined operant as a necessary component of SR learning, the possibility that a consummatory response might be a necessary precursor for an SR effect requires further consideration. The theoretical model which specifically addresses consummatory responding is that of the elicitation hypothesis (Wike, 1966) (or S-R hypothesis [Hull, 1951]). Before discussing the elicitation hypothesis at greater length, however, it is necessary to consider three components of consummatory behavior (Denny & Ratner, 1970): appetitive behavior, elicited consummatory behavior, and post-consummatory behavior. Varying across these three categories are degrees of stereotypy and uninterruptability, with fixed responses at their peak during and toward the later portion

of the elicited consummatory segment. The first category, appetitive behavior, is instrumental and in terms of S_r models is most similar to experimentally defined operants or magazine components of a training sequence as discussed in the response contingent hypothesis above.

Post-consummatory behaviors include those which follow a consummatory behavior with decreasing stereotypy as elapsed time after the consummatory behavior increases.

Post-consummatory behaviors follow relatively reliable patterns and serve to disengage the animal from one consummatory process and direct it to the appetitive phase of the next consummatory process, as from eating to sleeping. It is, therefore, the elicited consummatory behaviors per se to which this discussion will be confined. These may be defined as overt fixed action patterns (Lorenz, 1950) characterized by their stereotypic nature and their uninteruptability.

Hull's elicitation hypothesis does not find adequate support within the results of the present study. It was Hull's position that a neutral stimulus must acquire the capacity to elicit a fractional anticipatory goal response (r_g), that is, some fraction of the consummatory goal response (R_g) associated with the S_R . For reasons discussed in the introduction, the use of ICS as a primary reinforcer possesses inherent advantages relative to more traditional reinforcers. This is particularly the case with respect to testing the elicitation hypothesis. It

might be reasonably argued, with the use of a reinforcer such as food, that while the Pavlovian training procedure involves no experimentally designed SD component, the neutral stimulus in training functions as a CS for the elicitation of the consummatory response. Keehn's (1962) results suggested that this is an important variable. Keehn found that only those subjects who were permitted to engage in some portion of the original consummatory response following an Sr showed a conditioned reinforcement effect. Because of the absence, in any traditional way, of a consummatory process in this study, the results clearly contradict that necessity.

It is important to note that Pliskoff, Hawkins, and Wright (1964) have suggested that a stereotyped posturing response can occur in the 0.5 s delay between the onset of the neutral stimulus and the onset of the ICS. For this reason, an implant site was selected for this study in which motor effects were found to be infrequent. Further, two types of observations of the subjects during the experimental procedure are noteworthy. The 16 subjects, observed to have sustained elicited motor effects and for whom responding for ICS was appetitive, failed to meet the pretraining lever press criterion for inclusion in the study. Second, observations of subjects included in the study indicated no observable conditioned response (CR) or UCR during pretraining or training phases. Because no overt CRs were observed, it can not be necessarily

concluded that they did not exist; however, this observation is compatible with those of Crowder, Smith, Davis, Noel, and Coussens (1972), Knott and Clayton (1966), and Stein (1958).

One final hypothesis regarding the necessary conditions for Sr must be considered, the information hypothesis. The current study neither contradicts nor supports this hypothesis. As defined by Egger and Miller (1962), the information hypothesis suggests that a stimulus must be informative and reliable regarding the subsequent occurrence of the SR in order to function as an Sr. While the paired stimuli in the present study, by Egger and Miller's definition, could be considered both informative and reliable relative to their respective controls, the present study was not designed to test those variables against a competing theoretical viewpoint.

The present data, for the most part, do not require consideration of information and reliability issues. An exception is that of performance on the random lever when used as a within subjects control (paired/random group) in contrast to a between subjects control (random/nothing group). As previously indicated, responding on the within subjects random lever increased significantly from pretest to test (mean difference = +12.25 responses), while responding on the between subjects lever did not (mean difference = +5.50 responses). One interpretation of this finding suggests that significance was obtained for the

within subjects control, because informational variables were activated for the paired stimulus as a result of training, and, therefore, to a lesser extent for the random stimulus, as well. In contrast, significance was not obtained for the between subjects controls because information variables were inadequate to predict SR in the random/nothing group during training and were, therefore, not activated. While a pseudoconditioning explanation is better supported by the mean number of random stimulus/ICS forward overlaps (totalling 4.5 of a possible 250), the information hypothesis is also a credible interpretation. Support for the information and stimulus generalization hypotheses is strengthened by the absence of what might be considered a pseudoconditioning effect for the random stimulus in the random/nothing group, where such an effect would be predicted to occur from a pure pseudoconditioning hypothesis.

A variant of the elicitation hypothesis, the contiguity hypothesis (Harris, 1981) suggests that the neutral stimulus must acquire through contiguous pairing, the capacity to elicit, not a fractional consummatory response, but a fractional excitatory response (re), that is, some fraction of the UCR associated with the SR (or goal stimulus [SG]). The concept of an re endeavors to deviate from the concept of rg by eliminating the anticipatory or expectancy arousal component for the SR in the establishment of a new learning Sr effect.

Essentially, the use of rg relies on the incentive value of SR as the functional motivator in the operation of Sr on a new response. The concept of re , on the other hand, relies on the excitatory value attached to the neutral stimulus, that is, conditioned arousal, to provide the necessary incentive value for the establishment of a new learning Sr effect. Before discussing theoretically the manner in which re s operate, once established, it is important to consider critical differences in the way rg s and re s are established and the distinctions between the two. First, the re is conceptualized as the initial excitatory CR to be established in the conditioning process, and is in closest proximity, in time, to the UCR. Secondly, it clearly has no instrumental or consummatory properties as they are traditionally defined. Finally, during the establishment phase, re has no discriminative directional or guiding functions relative to the SR (or SG), a property which is common to rg . In the Hullian model rg s give rise to sg s which have a directive function. For example, in a T-maze, right and left turns (rg s) will be differentially associated to different sg s which will function to guide the subsequent chain of behavior leading to SG . It is partly by virtue of this process that a neutral stimulus acquires Sr properties. Because re is conceptualized as an internal excitatory response and as the CR in closest proximity to the UCR, that is, the terminal response in a chain leading to SR , directional properties are not needed.

While the reader might argue that re is, by virtue of these parameters, merely a subclass of rg and, therefore, not worthy of separate consideration, this author believes that the elements listed above are sufficiently important to identify re as a distinctly separate construct. From a theoretical perspective, two requirements, therefore, can be delineated for the establishment of a conditioned reinforcement effect; that of arousal of an excitatory potential by SG which is conditionable (e), and contiguity of that potential with some neutral stimulus. With respect to the effect of Sr s , once established, upon the learning of a new response, the contiguity (or re) hypothesis differs from hypotheses relying on rg in the specification of three points. First, the hypothesis is non-specific, in the sense that no particular class of stereotyped consummatory response is postulated for the Sr effect. Second, it is non-specific in that no specific expectancy or desire for the original UCS (or SR) is required. Finally, it is hypothesized that the Sr (or se) is specific in that it arouses behavior explicitly for itself as an incentive rather than increasing performance as a general heightener of excitability, arousal, or alertness.

The results of the present study are compatible with an excitatory model of Sr establishment and operation as are the results of Stein (1958) and Knott and Clayton (1966). Denny and Ratner's (1970) discussion of the consummatory process associated with ICS further elucidates

the excitatory model. As these authors point out, while it is possible to sustain appetitive behavior, for example, lever pressing, using ICS as the reinforcer, no overt consummatory response is measurable. Referring to the work of Spies (1965) and others (Roberts, Steinberg, & Means, 1967; Hutchinson & Renfrew, 1966), Denny and Ratner suggest the possibility that because of the ability of ICS, with proper placement, to elicit overt consummatory responses, those responses are always accompanied by a subcortical neural impulse. It is this neural impulse, it is hypothesized, which serves, in the present study, as the UCR for the establishment of a fractional excitatory response or CR to the conditioned reinforcer or CS.

In summary, it is apparent that Pavlovian temporal contiguity is necessary and sufficient, empirically, for the establishment of a conditioned reinforcement effect. Further investigation is necessary to distinguish between theoretical approaches suggesting the relative contributions of informational, expectancy, arousal, or contiguity factors as necessary elements for the establishment and operation of the effect. The results of the present study indicate the value of designs which employ both pretest and test comparisons as well as attention to magnitude of effect issues in the use of between versus within subject designs.

References

- Bilbrey, J., & Winokur, S. (1975). Conditioned reinforcement: Effects of temporal contingency and percentage of pairing. Journal of General Psychology, 93, 247-260.
- Bolles, R. C. (1975). Theory of Motivation (2nd ed.). New York: Harper & Row.
- Box, G. E. P. (1953). Non-normality and tests of variances. Biometrika, 40, 318-335.
- Crowder, W. F., Smith, S. C., Davis, W. M., Noel, J. T., & Coussens, W. R. (1972). Effect of morphine dose size on the conditioned reinforcing potency of stimuli paired with morphine. Psychological Record, 22, 441-448.
- Crum, J., Brown, W. L., & Bitterman, M. E. (1951). The effect of partial and delayed reinforcement on resistance to extinction. American Journal of Psychology, 64, 228-237.
- Denny, M. R., & Ratner, S. C. (1970). Comparative Psychology: Research in Animal Behavior (revised ed.). Homewood, Ill.: The Dorsey Press.
- DiCara, L. V. (1966). Brain stimulation and secondary reinforcement. Dissertation Abstracts, 27, (2157-B).
- DiCara, L. V., & Deutsch, J. A. (1966). Secondary reinforcement as a function of drive in intracranial self-stimulation. Proceedings of the 74th Annual

Convention of the American Psychological Association,
105-106.

Dinsmoor, J. A. (1950). A quantitative comparison of the discriminative and reinforcing functions of a stimulus. Journal of Experimental Psychology, 40, 458-472.

Egger, M. D., & Miller, N. E. (1962). Secondary reinforcement in rats as a function of information value and reliability of the stimulus. Journal of Experimental Psychology, 64, 97-104.

Elam, C. B., Tyler, D. W., & Bitterman, M. E. (1954). A further study of secondary reinforcement and the discrimination hypothesis. Journal of Comparative and Physiological Psychology, 47, 381-384.

Hall, J. F. (1976). Classical Conditioning and Instrumental Learning: A Contemporary Approach. Philadelphia: Lippincott Company.

Harris, R. S. (1981). Conditioned reinforcement: reality or myth? Unpublished manuscript, University of Rhode Island, Psychology Department, Kingston, Rhode Island.

Hart, B. L. (1976). Experimental Psychobiology: A Laboratory Manual. San Francisco: W. H. Freeman & Company, (p. 76).

Hilgard, E. R., & Marquis, D. G. (1940). Conditioning and Learning. New York: Appleton-Century.

Hull, C. L. (1943). Principles of Behavior.

New York: Appleton-Century-Crofts.

Hutchinson, R. R., & Renfrew, J. W. (1966). Stalking, attack, and eating behaviors elicited from the same sites in the hypothalamus. Journal of Comparative and Physiological Psychology, 61, 360-367.

Keehn, J. D. (1962). The effect of post-stimulus conditions on the secondary reinforcing power of a stimulus. Journal of Comparative and Physiological Psychology, 55, 22-26.

Keller, F. S., & Schoenfeld, W. N. (1950). Principles of Psychology. New York: Appleton-Century-Crofts.

Klein, R. M. (1959). Intermittent primary reinforcement as a parameter of secondary reinforcement. Journal of Experimental Psychology, 58, 423-427.

Knott, P. D., & Clayton, K. N. (1966). Durable secondary reinforcement using brain stimulation as the primary reinforcer. Journal of Comparative and Physiological Psychology, 61, 151-153.

Longstreth, L. E. (1971). A cognitive interpretation of secondary reinforcement. Nebraska Symposium on Motivation, 33-80.

Lorenz, K. (1950). The comparative method in studying innate behavior patterns. In Symposium of the Society for Experimental Biology, Vol. IV, Physiological Mechanisms in Animal Behavior, Cambridge, England: Cambridge University Press, 221-268.

Mogensen, G. J. (1965). An attempt to establish

secondary reinforcement with rewarding brain stimulation. Psychological Reports, 16, 163-167.

Mogenson, G., & Cioe, J. (1977). Central reinforcement: A bridge between brain function and behavior. In W. K. Honig & J. E. R. Staddon (Eds.), Handbook of Operant Behavior (p. 570-595). Englewood Cliffs, New Jersey: Prentice-Hall, Inc.

Pliskoff, S. S., Hawkins, T. D., & Wright, J. E. (1964). Some observations of the discriminative stimulus hypothesis and rewarding electrical stimulation of the brain. Psychological Record, 14, 179-184.

Rescorla, R. A. (1967). Pavlovian conditioning and its proper control procedures. Psychological Review, 74, 71-80.

Roberts, W. W., Steinberg, M. L., & Means, L. (1967). Hypothalamic mechanisms for sexual, aggressive, and other motivational behaviors in the opossum. Journal of Comparative and Physiological Psychology, 64, 1-15.

Saltzman, I. J. (1949). Maze learning in the absence of primary reinforcement: A study of secondary reinforcement. Journal of Comparative and Physiological Psychology, 42, 161-173.

Schoenfeld, W. N. Antonitis, J. J., Bersh, P. J. (1950). A preliminary study of training conditions necessary for secondary reinforcement. Journal of Experimental Psychology, 40, 40-45.

Seward, J. P., Uyeda, A. A., & Olds, J. (1959).

Resistance to extinction following cranial self-stimulation. Journal of Comparative and Physiological Psychology, 52, 294-299.

Silverstein, A., & Lipsett, L. P. (1974). The role of instrumental responding and contiguity in the development of infant secondary reinforcement. Journal of Experimental Child Psychology, 17, 322-331.

Spies, G. (1965). Food versus intracranial self-stimulation reinforcement in food-deprived rats. Journal of Comparative and Physiological Psychology, 60, 153-157.

Stein, L. (1958). Secondary reinforcement established with subcortical stimulation. Science, 127, 466-467.

Wike, E. L. (1966). Secondary Reinforcement. New York: Harper and Row.

Appendix A

Conditioned Reinforcement:

Reality or Myth?

Few concepts have been used as extensively as explanatory mechanisms within the field of learning as has that of conditioned or secondary reinforcement. Some stimuli appear to be, by their inherent nature, reinforcing and will in the absence of any prior experience increase the subsequent frequency of responses which they follow. Other stimuli seem to have little, if any, effect on behavior upon their initial occurrence. Yet, after a sufficient period of certain classes of experience with them, these stimuli seem to acquire the ability to affect behavior in ways similar to those stimuli requiring no special prior experience. The latter stimuli are referred to as conditioned or secondary reinforcers (Sr s) while the former are termed primary reinforcers (SRs).

Very little human behavior is under the direct control of primary reinforcers such as food or water. On the contrary, human behavior seems most frequently greatly affected by both such tangibles as money and material possessions and intangibles in the form of praise, attention, and success. These stimuli are often conceptualized by learning theorists to be under the heading of conditioned reinforcers. It is not surprising, therefore, that a large body of experimental and applied literature has been devoted to attempts to understand and

explain the necessary and sufficient conditions by which conditioned reinforcers are established and maintained.

Three decades ago, Keller and Schoenfeld (1950), writing in the Skinnerian tradition, predicted:

In reaching our goal, the principle of secondary reinforcement will be of great analytical assistance. When added to the other functions of stimuli, it gives us a powerful and indispensable tool for the solution of many vexing and absorbing problems of human action. (p. 260)

It is not surprising, given such an optimistic introduction, that behavior analysts, over the past several years, have attempted to take advantage of the construct in order to parsimoniously explain a wide range of complex human behavior.

Hull's (1951) (revised) definition of conditioned reinforcement has served as the basic model upon which all other definitions have expanded. Hull states:

A neutral receptor impulse which occurs repeatedly and consistently in close conjunction with a reinforcing state of affairs, whether primary or secondary, will itself acquire the power of acting as a reinforcing agent. (p. 28)

Basic Questions

On the basis of this formulation as well as other more complex formulations, extensive experimentation has been conducted in an attempt to answer three basic questions about the nature of conditioned reinforcement. They are:

- (1) What are the necessary and sufficient conditions for the establishment of an Sr?
- (2) Once established, how does the Sr operate on

or affect behavior?

- (3) By what means can conditioned reinforcement strength be maintained?

However, in spite of the plethora of investigations of this widely accepted phenomenon a careful examination of the literature reveals that little is yet known conclusively with regard to any of these questions. There is, in fact, only minimal evidence for the sheer existence of conditioned reinforcement as a construct. Experimental outcomes suggesting reasonable answers to the more complex issues cited above are even less available.

Conditioned reinforcement studies have employed both absolute experimental designs and differential (Lawson, 1957) or relative designs. In the former, a subject in one group receives exposure to one experimental treatment condition, for example, a specific magnitude of reward, while subjects in other groups are exposed to other related treatment conditions, for example, additional levels of reward magnitude. When the differential, within-subjects design is used, on the other hand, subjects are exposed to contrasting experimental conditions either sequentially or simultaneously.

While differential designs are generally thought to be more sensitive to variations in treatment effects, some question has been raised as to whether this increased sensitivity is an artifact of failure of the experimenter to adequately differentiate the treatment variables at the

outset of an experimental procedure which employs an absolute design (Wike, 1966). Butter and Thomas (1958), for example, found that subjects displayed significantly shorter response latencies and increased lever pressing for a high concentration of a sucrose solution (24%) as compared to a low concentration (8%) in an absolute design, while Lawson (1957) varying magnitude of reward in an absolute design found no difference. Butter and Thomas suggest that the problem with Lawson's procedure was one of using two magnitudes of SR for which there was no differential reinforcement effect, thereby minimizing the likelihood of a differential conditioned reinforcement effect. Relative or differential designs, however, are critical to the investigation of some of the more complex theoretical mechanisms invoked to account for the establishment of Sr effects, such as, informational variables (Egger & Miller, 1962). Specifically, the activation of the information variables, in an absolute design, is relatively impossible to separate from more basic Sr variables, such as, contiguity. Further, the differential procedure employs the use of a preference criterion in testing which permits the assessment of relative differences in the incentive value of previously neutral stimuli. Finally, from the perspective of error variance, the use of a differential procedure attenuates within subject error and thereby enhances sensitivity to treatment effects.

Research Paradigms

Historically, one of the major problems contributing to the current state of affairs in the study of conditioned reinforcement has been the use of experimental paradigms, whose results lend themselves to alternative and, in some cases, more parsimonious explanations. Three major paradigms have been employed to examine conditioned reinforcement effects. The extinction paradigm was the first to be used historically and has been used extensively since. While considerable research has been undertaken using this paradigm, the results of this research have been seriously questioned, from the standpoint of the more parsimonious explanation of stimulus generalization decrement (see below). A second major paradigm, that of chained schedules, has been employed extensively as well, and has been reviewed in depth by Kelleher and Gollub (1962). While chained schedules have offered promise for investigating the bases for durability and maintenance of Sr effects, problems in separating conditioned reinforcer functions from discriminative stimulus functions of the neutral stimulus in the chain, as well as the confounding influence of the continued presence of the primary reinforcer, result in limited promise for specific answers to basic questions about Sr establishment and operations (see below). The third major paradigm, the new learning paradigm, while offering greater clarity in addressing questions of Sr establishment and operation, has a major

drawback resulting from the rapid extinction of the Sr value of the neutral stimulus in the new learning situation, thereby resulting in weak and transitory effects.

The extinction paradigm presents several excellent example of the problems encountered in the interpretation of results in this area of research. This paradigm, can employ either a free operant or discrete trial procedure. Typically, experimental subjects are trained to respond to obtain a presentation of the neutral stimulus followed contiguously by the SR. Control subjects, on the other hand, receive only the SR (the neutral stimulus is either randomly presented or absent altogether) contingent upon performing the response of interest. Subjects are then tested by being placed in the original training situation and measured for level of responding during extinction. A conditioned reinforcement effect is said to occur if responding by experimental subjects, who are continuing to receive the previously neutral stimulus during testing, is slower to extinguish than is that of control subjects.

Bugelski (1938), whose study was one of the first in the literature to report conditioned reinforcement effects (Wolfe [1936] and Cowles [1937] were earlier), used a between subjects extinction design, in which one group trained to lever press for a click and SR continued to receive the click as an Sr in the test phase contingent upon lever pressing, while for the control group lever

pressing produced no stimulus in testing. Bugelski found that subjects receiving the lever press contingent click during testing were significantly slower to extinguish than the control subjects. Delayed extinction for the experimental group was interpreted as indicating a conditioned reinforcement effect.

The major difficulty with the extinction paradigm, as Wike (1966) points out, is that conditioned reinforcement effects, to the extent that they occur, are inseparable from the effects of stimulus generalization. This explanation, designated by some authors (Crum, Brown, & Bitterman, 1951; Elam, Tyler, & Bitterman, 1954; Wike, 1966) as the discrimination hypothesis of S_r effects, points out that experimental conditions have changed more radically for control subjects whose responding is no longer followed by any consequence, than for experimental subjects who continue to receive the previously neutral stimulus as they did during training. It is argued therefore that control subjects experience greater stimulus generalization decrement from training to testing than is the case for experimental subjects, and that their ability to discriminate extinction from training alone can sufficiently account for differential effects in the test phase. It is therefore unnecessary to invoke conditioned reinforcement as an explanatory mechanism. This is a particularly serious problem in any situation where response occurrences are massed through time. A stimulus

generalization decrement interpretation of data from studies relying on the extinction paradigm, therefore, throws into question a large body of literature previously offered in support of the existence of conditioned reinforcement.

As indicated above, a second major paradigm is that of chained schedules (Kelleher & Gollub, 1962). Originally regarded as the paradigmatic answer to the durability problem, chained schedules, upon closer examination, have been subject to a number of interpretive difficulties due to confounding possibilities. Typically, chained schedules involve two or more response components in which different schedules of reinforcement are used in each component. In the prototype procedure, a discriminative stimulus (SD) (S1) sets the condition for the initiation of responding on the first schedule. At a fixed point, the discriminative stimulus is terminated and a second stimulus (S2) is presented signaling that the second schedule is in effect. At the end of this second component (or the last component if the chain includes more than two components), the SR is delivered thereby terminating the instrumental chain as it is defined. Presumably, consummatory behavior continues to occur. It is the second stimulus in the chain which is considered to be the SR because it maintains the first response in the chain.

It is undeniably the case that the second stimulus in the chain is capable of maintaining high probabilities of

responding for R₁ and also of functioning as a stimulus cue in the presence of which an R₂ has a high probability of emission. Empirically, these functions are difficult to separate. From a theoretical perspective, however, the confounding is even more complex than attempting to discriminate S_r from cue functions. At first glance, the chained schedule S_r effect would seem to present little problem for a discriminative stimulus hypothesis (Keller & Schoenfeld, 1950), which claims that once it is established as an S_D, S₂ then functions as an S_r for responding in the initial segment of the chain. Upon closer examination, however, it is clear that either the offset of S₁ (an S₁ is usually used as a part of the design), the onset of S₂, the initiation of R₂, or some combination of the three can function as the S_r. Further, once identified as the critical event separating the two response chains, the extent to which that event functions as an S_r for prior responding or merely an S_D for subsequent responding is impossible to pull apart. From this perspective, the discriminative stimulus hypothesis of S_r acquisition is thrown into question, along with any consideration of other hypotheses. Adding to the confusion, is the possible delayed influence of the primary reinforcer on responding in the the first segment of the chain. The major difficulty with this paradigm, therefore, is that the identification of a specific event as the S_r is not possible. The final criticism of chained schedules results

from the identification of an S_r effect by the change in rate of R_1 when S_2 is withdrawn. Aside from the arbitrary designation of S_2 as the S_r , its removal results in a stimulus generalization decrement phenomenon much like that of extinction tests, where conditions differ on the basis of presence or absence of a stimulus in the comparison conditions, as distinct from presence or absence of a contingency in the critical comparison with the stimulus always present. The difference can therefore be attributed to a change in stimulus conditions as opposed to removal of a reinforcer. Hence, because of the inadequacy of this model in permitting further resolution of these questions of establishment, operation, and durability, another large body of data must be set aside.

The new learning paradigm remains as the only procedure among the three which, when adequately controlled, does not fall prey to serious methodological criticisms. Subjects in this paradigm can be trained in one of three basic ways. In a Pavlovian, or non response-contingent training format, subjects are placed in the training situation and presented with forward pairings of the neutral stimulus followed by primary reinforcer, independent of any response requirement other than S_R approach. In an operant or instrumental format, subjects are required to perform some experimentally defined operant in the training phase in order to obtain the neutral stimulus/primary reinforcer pair. In the discriminative

stimulus procedure, subjects are required to perform an experimentally defined operant in the presence of a neutral stimulus (or SD) the result of which is the delivery of the SR. Subsequent to training in each of the three formats, animals are placed in the test situation where the previously neutral stimulus is delivered contingent upon the performance of a new operant with no presentation of the SR. Hence the designation "new learning" paradigm. In order to meet the requirements of a new learning paradigm, any training response must differ in some critical aspect from the test response. When established as a part of a properly controlled design, the ability of the Sr to increase the probability of the new response can be taken as a clear indication of the existence of a conditioned reinforcement effect.

Unfortunately, this procedure invariably provides the least robust effects in testing. This must be the case because the strength of the Sr is extinguishing as the new response is being learned. Sr strength very rapidly drops below threshold and responding among experimental subjects quickly becomes indistinguishable from that of controls. While the potential superiority of the procedure in permitting an uncontaminated demonstration of the phenomenon is unquestionable, it may often be the case in any particular study, that extinction of the conditioned reinforcement effect occurs before the threshold for a significant difference between experimental and control

subjects is reached. Moreover, the establishment of a durable effect is particularly difficult. The result is that there are only a limited number of studies which most would agree qualify as having demonstrated a conditioned reinforcement effect (Crowder, Smith, Davis, Noel, & Coussens, 1972; Doerries, 1974; Hyde, 1976; Knott & Clayton, 1966; Saltzman, 1949; Silverstein & Lipsett, 1974; Zimmerman, 1959), and not all of even this small number indicate an ideally controlled design, in all cases.

Control Issues

In addition to the problems presented by the use of methodologically inadequate or weak paradigms, studies of SR have been open to frequent criticisms regarding the issue of adequate controls (Wike, 1966). Critical to any design is the need for a control group in which all training conditions have been replicated to an equal degree with the exception of the temporally contiguous presentation of the neutral stimulus and SR. Rescorla (1967) argues that the neutral stimulus in the control group must have a "truly random" relationship to the SR, in which some pairings might occur by chance with the SR, in order to adequately control for pseudoconditioning effects. If the neutral stimulus is explicitly unpaired, Rescorla points out, it is possible for the neutral stimulus to acquire conditioned aversive properties, or counterconditioning, by signalling the absence of the SR. It might be reasonably argued, on the other hand, that

fortuitous pairings of the random stimulus with the SR could be sufficient to establish a weak conditioned reinforcement effect in some cases (Beninger & Phillips, 1980). This possibility could be adequately addressed by a comparison of the random control condition to that of an explicitly unpaired control. From a theoretical perspective, the use of these controls has additional relevance. A seldom mentioned explanation of how Sr s affect behavior makes use of the primary reinforcing properties of stimuli thought to be neutral as a result of the absence of their exposure to prior training. The primary reinforcement hypothesis (Kling & Schrier, 1971) emphasizes manipulatory, exploratory, and novelty aspects of stimulus events as means by which "neutral" stimuli function as primary reinforcers. Without the use of control stimuli equated for all variables with the exception of Pavlovian pairing with the primary reinforcer, an SR hypothesis cannot be ruled out. While it would seem prudent, under the circumstances, to consider both random and explicitly unpaired controls, few experiments have adequately addressed the issue of control to this extent.

Alternative Explanations of Sr: The Myth?

The aforementioned control difficulties have led some authors (e.g., Longstreth, 1971; Bolles, 1975) to argue that conditioned reinforcement as set forth by Skinner (1938) and Hull (1951) is a mythical phenomenon. Longstreth (1971) for example, having examined both lower

animal and human evidence, concludes that there are no studies which convincingly demonstrate a true Sr effect. Instead, Longstreth argues that those effects, attributed to conditioned reinforcement, are more reasonably interpreted as (a) the result of inducing a frustration drive by withdrawing reinforcement or (b) the result of a cognitive or informational process. Longstreth also suggests that what are often identified as Sr s in adults (e.g., money, praise, grades, etc.) are in reality, sources of information regarding the probable behavior of those individuals who control access to those incentives. Further, he suggests that the presentation of stimuli (Sr s) alone (in the absence of any information relative to possible incentives), which have been previously associated with SR, results initially in heightened responsiveness due to the increase in frustration drive, a phenomenon particularly observable in extinction paradigms, but also present in new learning procedures where response generalization occurs. Subsequently, subjects learn to avoid the previously neutral (now aversive) stimulus, thereby reducing the frustration. It is merely the heightened responsiveness resulting from frustration which, Longstreth suggests, is attributed spuriously to conditioned reinforcement. It is Longstreth's position that, while Sr s may function in an associative manner with lower animals and human subjects below the age of four or five (a position for which he insists there is inadequate

evidence), for humans beyond that age, their function takes on the character of a cognitive process wherein sources of S_rs (e.g., parents, employers, friends) are evaluated on the basis of their power to predict subsequent delivery of primary reinforcement.

Bolles (1975) offers a second alternative for what has traditionally been conceived of as conditioned reinforcement, the sign-post hypothesis. Using an example of Longstreth's (1971), Bolles argues that a youngster who behaves ideally from the moment he is called to dinner through the completion of the meal is doing so in order to maximize the possibility that his parents, having been pleased with his prior behavior, will grant his subsequent request to remain awake later to watch TV. According to Bolles, the social approval for appropriate mealtime behavior, which has been labeled traditionally as a conditioned reinforcer, serves for this youngster as a sign-post or a means to an end, that is, a means to achieving his desired goal of a delayed bedtime.

While Longstreth's skepticism regarding conditioned reinforcement is warranted based on the paucity of data supporting an S_r effect, the presence of a core of well-controlled studies (Hyde, 1976; Saltzman, 1949; Silverstein & Lipsett, 1974) suggesting an increase in responding for an S_r rather than a control stimulus in a distinctly new situation as a result of an S_r presents difficulties for a frustration hypothesis.

A number of problems exist for the sign-post explanation of Sr effects. Aside from the fact that this theory offers an explanation only for the operational function of Sr and not for its establishment, it also often fails to discuss why goals serve as goals. TV watching and/or a delayed bedtime are certainly not primary reinforcers, in any traditional sense of the term. If not, why are they valued and how did they come to be so? Are they a means to a further end or an end in themselves? An answer to either of the latter alternatives raises obvious follow-up questions, including why the experimental Sr cannot be treated in the same manner. Finally, while it may be the case that the youngster is manipulating for a later bedtime, in many cases it may be that bedtime is firmly set and independent of mealtime behavior. While Bolles would be likely to respond with a second, third, and fourth equally plausible alternative goal, what he fails to provide is an justification for the necessity of using cognitively based sign-post elements in all examples where more parsimonious conditioned reinforcement explanations might apply. In other words, the youngster may well have firmly established eating habits, developed and maintained by use of positive intermittent feedback, from his parents. A more Tolmanian interpretation of the sign-post theory (1932) would suggest that Sr functions as an expectancy for SR and, hence, as an incentive. Rather than denying the existence of Sr as a phenomenon, as Bolles does, the latter

merely specifies how an Sr operates, that is, as an incentive specific to an expectancy for SR.

The actual motivation of the youngster, notwithstanding, it is readily apparent from the foregoing discussion that conditioned reinforcement as an explanatory mechanism in applied situations has enjoyed a substantially more prominent position than its empirical support merits. It is not surprising, given the current state of affairs, that conditioned reinforcement as a construct is under serious question.

In response to this dilemma, the remainder of the present discussion will be devoted to an attempt to resurrect the construct of conditioned reinforcement from this rather dismal state of affairs. The discussion will be based on the fundamental position that the phenomenon of conditioned reinforcement does exist as indicated by a limited number of studies and that these studies, while neither ideally designed nor controlled, permit the preliminary formulation of a model of the establishment, operation and maintenance of Sr s. Relevant data will be presented in support of the model together with suggestions for further research. Because of the general lack of effective controls in the Sr literature, the reader should assume that data presented suffer from one or more control problems mentioned above, unless otherwise indicated.

Theories of Conditioned Reinforcement

A number of theories have been set forth to detail the

necessary and sufficient conditions for the establishment and operation of conditioned reinforcement. Explanatory mechanisms for these two questions have not been separated by most authors and, hence, will be considered together occasionally. One of the first hypotheses to attempt to explain Sr establishment required that a stimulus function as a discriminative stimulus first in order for it to acquire secondary reinforcing properties. Dinsmoor's (1950) study employing an extinction paradigm is typically offered as support for this approach. Briefly, animals trained with a discriminative stimulus for lever pressing were divided into three groups for testing; one group receiving the discriminative stimulus as an Sr (in the absence of an SR) contingent upon lever pressing in the extinction test, while the second continued to receive it as a discriminative stimulus in the absence of the Sr as well. A third group received no stimulus in the test phase. While responding was lower for the control group, no differences were found between the Sr and SD groups, thereby providing support for the discriminative stimulus hypothesis.

In addition to the hypothesis that the use of a previously neutral stimulus as an Sr for a new response requires its previous establishment as an SD, variations of the SD hypothesis have been employed to explain the manner in which Sr s operate. These include (a) the functioning of a neutral stimulus as an SD for the test response, (b)

its functioning as an SD for appetitive behavior associated with the SR, and (c) its functioning as an SD for any operant, regardless of its role in the experimental procedure.

While Dinsmoor's study and others (Klein, 1959; Saltzman, 1949) have offered support for the fact that a discriminative stimulus can function as an Sr, the evidence to date has not established the conclusion that it is necessary that a stimulus become a discriminative stimulus in order for it to function as an Sr. Furthermore, it is important to note that Dinsmoor's study might be more accurately viewed as an investigation of the manner in which Sr s, once established, operate on responding, rather than as an examination of establishment variables. Specifically, all groups in the study were trained identically, that is, given the same experience in the establishment of the Sr, but were tested differentially; in other words, tested in a manner which addressed the issue of Sr operation.

The information hypothesis is another of the theoretical approaches which has received critical attention in the literature. Egger and Miller (1962, 1963) conducted a series of experiments in which a pair of neutral, sequentially-overlapping stimuli (S1-S2) were presented in a Pavlovian training paradigm. In one condition (A) experimental subjects received both S1 and S2 throughout training, while in the other condition (B), S2

was presented continuously with S1 being presented only intermittently. Egger and Miller found that S1, the informational stimulus, was superior in Condition A, and that S2, the reliable (although occasionally redundant) stimulus was superior in Condition B. They interpreted their results as indicating that a stimulus will function as an Sr to the extent that it is informative; i.e., signals the forthcoming delivery of SR, is non-redundant and reliable. These functions must be acquired for Sr establishment and are major mechanisms for the development of a "new learning" Sr effect.

While Egger and Miller's (1963) interpretation seems quite reasonable, more recent investigations in this area (Borgealt, Donahue & Weinstein, 1972; Hancock, 1982; Thomas, Berman, Serednesky, & Lyons, 1968) have argued that Egger and Miller omitted a critical component in the test phase. It is pointed out that while S1 occurred alone in training, this was never the case for S2. As such, the more appropriate test would involve a comparison of S1 and the S1-S2 compound, rather than S2 alone. Thus the superiority of S1 in contrast to S2 can be easily attributed either to less stimulus generalization decrement, as its presentation in testing is more similar to training than is the case when presenting S2, to masking of S2, or to some combination of the preceding. In support of this point of view, comparisons employing the S1-S2 compound during testing (Borgealt, et al., 1972; Thomas,

et al., 1968) have found it to be superior to S1 alone and S2 alone.

Hendry (1969) expanded the information hypothesis to include two major hypotheses, which stem from the basic premise that stimuli which reduce uncertainty are reinforcing. The first major hypothesis, the clue hypothesis, is based on Egger and Miller's (1962, 1963) studies and proposes that previously neutral stimuli function as "clues" signifying what to expect (e.g., the subsequent occurrence of the SR). The second hypothesis, the cue hypothesis, relies heavily on the importance of differential discriminative stimulus properties of previously neutral stimuli in experiments investigating the reinforcement of observing responses (Wyckoff, 1952; Wyckoff, 1969). Based on the observation in these studies that stimuli function as S_rs to the extent that they reduce uncertainty, Hendry (1969) hypothesizes that conditioned reinforcers are "cues" signifying to the subject what to do in order to obtain the SR. In support of the cue hypothesis, Hendry cites two predictions. First, stimuli associated with multiple schedules will function as S_rs so long as the multiple schedule remains in effect. This prediction would be made by both SD and cue hypotheses. Secondly, stimuli associated with identical multiple schedule components will not function as S_rs. This prediction would not evolve from an SD hypothesis as both are functioning as SDs for subsequent

responding. The cue hypothesis, operating on the assumption that SDs must be informative with respect to differential responding in order to function as S_rs, would make the latter prediction.

Evidence suggesting problems for the cue hypothesis has already been discussed within the context of criticisms of Egger and Miller's results. With respect to the cue hypothesis, Bower, McLean, and Meacham (1966) used a concurrent schedule design in which identical mixed versus multiple schedules were available on right or left keys. FI 10 and FI 40 schedules were programmed to occur an equal number of times in random sequence. Subjects indicated a preference for the multiple schedule key. This finding is congruent with the hypothesis that a reduction in uncertainty increases S_r effects.

To further test this assumption, Bower, et al. reduced uncertainty in both schedules by programming the FI 10 and FI 40 components in a 20%/80% balance, respectively. Based on this reduction in uncertainty, the preference for the key associated with the multiple schedule should have had less informative value. The predicted result was that preference for the multiple key should be reduced. Such was not the case. A reduction in the uncertainty of the schedules did not reduce S_r effects as a cue hypothesis would predict. To the extent that uncertainty reduction is a verifiable phenomenon, as the results of Bower, et al.'s first experiment would suggest, it may be the case that it

is most relevant as an issue in situations involving complex schedules where the SR is continually available, and wherein the Sr is acting as a cue for responding to obtain the SR.

A fourth approach, the arousal hypothesis, proposes that the function of Sr training is to enhance the subjects arousal to the neutral stimulus in a non-specific manner (Kling & Schrier, 1971). Hence in the confined test environment, responding is increased as a result of increased arousal resulting from the repeated presentation of the neutral stimuli. Temporal contiguity is not required for training, nor is an explicit response contingency necessary in testing. The use of a random control in a new learning design would adequately address the legitimacy of the arousal model.

An extension of the arousal model (Kling & Schrier, 1971) suggests that an increase in a specific response in testing, results from proximity to the response manipulanda at the time of Sr presentation. It is suggested that specific responding (e.g., lever pressing) increases as a result of increased arousal before the subject has had an opportunity to move any distance from the response manipulanda. A cyclical pattern results in which increased arousal results in increased responding which results in presentation of the Sr, which results in a further increase in arousal. While this approach is plausible for the learning of a new response in a free operant paradigm, it

is difficult to understand in terms of a discrete trial paradigm in which the subject is removed from the runway or T-maze immediately subsequent to Sr presentation. Also, because this hypothesis relies on sensitization of the response by the SR for the "Sr" effect, the use of a randomly presented control stimulus would adequately resolve the relative importance of arousal aspects of Sr training for subsequent Sr operation.

The elicitation hypothesis represents a fifth model of Sr establishment, as well as of its operation. It was Hull's position in 1951 that a potential Sr must acquire its reinforcing capacity through contiguous pairing with a stimulus which elicits a fractional anticipatory goal response (rg); that is, some fraction of the consummatory or goal response (RG) associated with the SR. In partial support of this model, Keehn (1962) found that only those subjects permitted access to the majority of the approach/consummatory response, in testing, that is, subjects for which large portions of the sg-rg chain remained in place, demonstrated a conditioned reinforcement effect. (For a more detailed discussion of the Keehn study and problems associated with it, see p. 109.)

While Keehn did not compare the relative importance of approach (operant) behaviors and elicited (respondent) behaviors in his study, it is likely that for those subjects blocked from SR approach, the elicitation of early phases of the consummatory chain did not occur, while the

opposite was true for those subjects who were not blocked. Partial support for the elicitation hypothesis is offered, therefore, despite the ability to separate the two response components (operant and respondent).

In a further specification of the elicitation hypothesis to include an account of how S_rs operate, Bugelski (1956) reinterpreted his earlier data (1938) and proposed that the lever press contingent click did not reinforce the preceding operant in the traditional sense but rather elicited subsequent responding. While Bugelski's statement does not explicitly espouse a respondent model, subsequent references to his work (Kelleher & Gollub, 1962; Wike, 1966) have interpreted it as such. Bugelski proposed that the click elicited a magazine approach, consummatory responding and, subsequently, lever press responding. Failure to find food at the usual site would fail therefore to elicit subsequent lever pressing, thus resulting in extinction of the lever press response.

Wyckoff, Sidowski, and Chambliss (1958) expanded Bugelski's original hypothesis to include two processes. According to these authors, an S_r functions as a cue (a) to bring the subject into the vicinity of the magazine (i.e., as an S_D) and (b) to increase the frequency of the response of interest by energizing the subject (i.e., as an arouser). Wyckoff et al. specifically distinguished their proposal from a strict elicitation approach and moved

toward the arousal/SD model of Sr operation. Cited in their introduction are references to the Keller and Schoenfeld (1951) hypothesis of Sr development and related research. Specific reference is made to the discriminative stimulus function of the cue. Their proposal is more similar to what has been called by Bolles (1967) and Kelleher and Gollub (1962) a facilitation (or discriminative stimulus) hypothesis, that is, the maintenance of behavior by virtue of the discriminative stimulus properties of an Sr.

Wyckoff et al. (1958) in an attempt to establish a cue function comparable to what is normally considered an Sr effect--a condition they refer to as "cue error"--trained subjects to approach a dipper and lick in response to a buzzer in two separate experiments. During the test phase of one experiment, experimental subjects pressed a lever for the sound of the buzzer. A control group received the buzzer at 1.0 min intervals contingent upon not responding during the previous 10 s. In a second study, operant control of the licking response was developed by training subjects on a variable interval schedule in the presence of the buzzer. Subjects were likewise tested with click presentations contingent upon lever pressing. Controls in Experiment II were yoked as a control for the cue functions of the buzzer. That is, each control subject received click presentations independent of any specific response, simultaneously with contingent click

presentations to the experimental subject with whom the control was paired. Failure of experimental subjects to differ significantly from controls was interpreted as a failure to demonstrate any Sr effects beyond that of a cue function.

Kelleher and Gollub (1962) cited three problem areas in the Wyckoff et al. (1958) study. First, the reinforcement procedure was somewhat out of the ordinary. The licking response served two functions, that is, that of an operant which was reinforced as well as a part of the consummatory response subsequent to reinforcement (Wyckoff et al., 1958). The separate functions of this response and the specific impact of the reinforcer on these separate functions is not clear. Secondly, the ISI was varied around a mean of 16 s. Berish's (1951) results indicate an optimal ISI for the establishment of Sr strength at 1.0 s with Sr strength diminishing toward Berish's upper limit of 10.0 s, where only weak Sr effects were obtained. As others have noted (Crowder, Morris, & McDaniel, 1959), Wyckoff et al.'s ISI was less than optimal. It is conceivable that this interval alone precluded the establishment of Sr effects.

Thirdly, Kelleher and Gollub (1962) noted that there is some evidence to suggest that the more effective reinforcer may have been buzzer termination rather than buzzer onset. It is therefore possible that the most effective Sr was not that made contingent upon lever

pressing.

A number of authors (Bolles, 1967; Hall, 1966; Kelleher & Gollub, 1962; Wike, 1966) refer to the numerous studies by Crowder and his associates (Crowder, Gay, Bright, & Lee, 1959; Crowder, Gill, Hodge, & Nash, 1959; Crowder, Morris, & McDaniel, 1959) as evidence of studies using techniques similar to Wyckoff et al.'s (1958) in which positive Sr effects were demonstrated. One technique common to those studies was the use of the yoked control. As Church (1964) pointed out, however, the use of a yoked control procedure renders the results of studies such as these open to question.

A model proposed by Bolles (1967) prior to his sign post hypothesis emphasizes the ability of a previously neutral stimulus to sustain a response chain leading to the SR. This "association" hypothesis suggests that eliciting stimuli set the occasion for subsequent responding which results in acquisition of the primary reinforcer. The original eliciting stimuli reinforce new responses, thereby extending the chain. This model may be considered a variant of the elicitation hypothesis as employed by Keehn, and extended to the issue of Sr operation. Referring to Cowles (1937), Bolles points out that when tokens could no longer be exchanged, that is, the response chain was broken, Sr s lost their effectiveness. But Bolles neglects to consider the issue of Sr durability and the possibility that Sr effects were extinguishing upon removal of the SR.

As Bolles (1967) concedes, the use of ICS as a primary reinforcer in a Pavlovian training procedure (e.g., Knott & Clayton, 1966; Stein, 1958) poses problems for an associative explanation. That is, how are Sr s established in ICS studies in the absence of a response elicited by the neutral stimulus upon which the new learning chain in testing could be developed?

Employing Hull's (1943) and Spence's (1956) concept of fractional anticipatory goal stimuli (sg s) and fractional anticipatory goal responses (rg s), Wike (1969) proposed an incentive hypothesis for Sr effects. According to this hypothesis, Sr s act as conditioned motivators. Behavior is subsequently strengthened through the resultant increase in the motivation to respond. Marx and Murphy (1961), for example, demonstrated that a buzzer previously paired with food would result in faster runway responding when sounded in the start box.

That a previously neutral stimulus in an Sr experiment could function as a conditioned motivator is not surprising. To suggest that all Sr s function in that manner is to make a sizable leap from the data. For example, subjects in the Marx and Murphy study were given response contingent training using a nose poke upon which the buzzer paired with food was contingent. After training subjects to run an alley for food, and extinguishing that response, the buzzer was sounded in the alley start box. It is not surprising that the buzzer, which had previously

signalled the delivery of food (and the appetitive/consummatory responses associated with it), would increase running speed in an alley in which running had been previously food reinforced, when the buzzer was presented in the start box. That it would not increase running would be surprising. However, the extent to which the buzzer would also function as an *Sr* for a new learning response was not examined, leaving the reader to conclude that one cannot rule out a conditioned reinforcement effect that could occur in lieu of a conditioned motivator effect.

Bolles (1967) explained the incentive effect of *Sr* s as being due to their capacity to elicit behavior which typically precedes the *SR*. This approach adds response specificity to the arousal model previously discussed. As previously mentioned, from a Tolmanian (1932) perspective, the incentive value of *Sr* s would derive from their ability to arouse an expectancy for *SR*. While these approaches are similar in some respects (e.g., their reliance on the importance of the *SR*), the former emphasizes response elicitation, while the latter emphasizes cognitive expectancy mediation.

Proposed Model

It is this author's view, that a variant of the elicitation model, provides the most accurate characterization of existing critical data. Specifications of the *rg* response class in the case of food reinforcers typically include a variety of operants, such as locomotor

approach, chewing, salivating, anticipatory swallowing, smelling, etc.. The position outlined here differs from such a traditional elicitation-approach theory in that the response class is limited exclusively to excitatory neural responses (re s) which typically are associated with the goal response. Thus, it is hypothesized that a neutral stimulus must acquire the capacity, through contiguous pairing with a goal stimulus, to elicit a fractional excitatory response, re; that is, some central neural component of the unconditioned arousal response (UCR) associated with the SR.

A typically implied characteristic of rg is that of the anticipatory function it plays in the chain of fractional anticipatory goal stimuli (sg s), signaling the forthcoming goal stimulus (SG). A large measure of its reinforcement capacity in the development of a new learning response may therefore be directly attributable to its ability to steer the subject in the direction of SG. Thus, in establishing an Sr, little incentive value need be ascribed to the sg itself since it elicits or signals the subsequent rg s in the chain. That is, in the establishment and operation of Sr s, the sg is merely an elicitor of a goal oriented response directed toward SG. The role of Sr s (and, hence, of sg s) is to function as mediational elicitors of the next response in the chain for the subject engaged in some aspect of the appetitive/consummatory process.

The concept of rg , therefore, by incorporating such an associative, anticipatory component, relies on the incentive value of SG as the functional motivator during the learning of some new response with Sr as the reinforcer. This concept assumes that all behavior is, in some manner, directed toward the goal of obtaining primary reinforcement. The concept of re , on the other hand, relies more heavily on the excitatory value newly attached to the neutral stimulus itself; that is the conditioned arousal elicited by Sr is hypothesized to provide the necessary incentive value for the establishment of a new learning Sr effect. The use of the concept, re , thereby, moves away from the necessity for exclusive reliance on the incentive value of SG . The distinction may be summarized as that of the discriminative function of rg versus the less directed arousal or incentive function of re .

From a theoretical perspective, many of the same parameters critical to the development of rg are critical to the development of re . For example, it may be assumed that for any given neutral stimulus, the extent to which either rg or re attach their incentive value to it is dependent upon (a) the vigor of rg and/or re , and (b) the strength of conditioning of the neutral stimulus to rg and/or re . Before proceeding to discuss the manner in which re s theoretically operate, once established, it is important to consider critical differences in the way re s and rg s are established. First, the re is conceptualized

as the first excitatory conditioned response (CR) to be established in the conditioning process, and is in closest proximity to the UCR. Other detachable responses elicited by the SR may be conditioned to Sr later in the sequence. Secondly, re very clearly has no instrumental or consummatory properties, as these are traditionally defined. Finally, during the establishment phase, re has no discriminative or guiding functions relative to the SR (or SG), a property which is common to rg. While the reader might argue that re is, by these parameters, merely a subclass of rg and, therefore, not worthy of separate consideration, this author believes the attributes listed above are sufficiently important to identify re as a distinctly separate construct. Operationally, the re hypothesis in contrast to an rg hypothesis would predict that an Sr effect could be established in the absence of any observable or measurable consummatory responding. A well-controlled investigation of Sr effects using procedures similar to those of Stein (1958), Knott and Clayton (1966), and Crowder, et al. (1972), if similar in outcome would support such an operational distinction. Other operational distinctions may also emerge.

Once conditioned to elicit re, the neutral stimulus acquires by virtue of the neural excitatory response elicited by it, the ability to function nonspecifically as a reinforcer, and as a motivator for any appetitive behavior which leads to it. Three specific points further

delineate the re hypothesis of Sr operation. First, the hypothesis is non-specific, in the sense that no particular class of stereotyped consummatory response is required for the Sr effect. Second, it is non-specific in that no specific expectancy or desire for the original UCS (or SR) is required. Finally, it is hypothesized that the Sr (or se) operates specifically as an incentive. Thus, only performance which produces the Sr is increased, as opposed to a general heightening of excitability, arousal, or alertness. Studies (Knott & Clayton, 1966; Stein, 1958) eliminating the consummatory component in the training process by means of the use of intracranial stimulation (ICS) as the primary reinforcer suggest that there are distinctively separate consummatory and neural excitatory aspects of reinforcers. This distinction is critical to the non-specificity and non-expectancy hypotheses of re. An expectancy model would predict that an Sr, once established for a particular SR, would be less easily developed as an Sr for a second, somewhat different SR than would a new neutral stimulus. This prediction is based on the assumption that an expectancy for the first SR would require "unlearning" in order to be replaced by an expectancy for the new SR. Such would not be the case for a neutral stimulus with no prior exposure to an SR. An re hypothesis on the other hand would suggest the opposite. That is, it would be predicted that pairing of a new SR with a previously established Sr would further enhance its

Sr strength relative to a newly paired Sr, as a result of the separate re s functioning additively, independent of the consummatory process and independent of an expectancy for a specific SR. Thus, from an re hypothesis, theoretical issues of anticipatory goal responding and expectancy for the SR are no longer required to the explanation of Sr operation.

Theoretically, therefore, from the standpoint of the contiguity model, two requirements are necessary and sufficient, in combination, for the development of a conditioned reinforcement effect. The first is a conditionable excitatory potential, (e), and the second is Pavlovian temporal contiguity. While the latter is easily defined empirically and easily measured, the basis for the latter is more theoretical, and thereby requires verification indirectly as a result of examining a network of experimental results which suggests a conditioned excitatory response as the most plausible and best supported of the theoretical alternatives. As a point of clarification, the definition of the concept of Sr, as herein stated, is not intended to exclude "elicited" responses, for example, a particular pattern of respiratory response or a change in heart rate, but merely to indicate the lack of necessity for their inclusion.

Stein's (1958) work using ICS as the primary reinforcer was perhaps the first to shed light on this somewhat limited set of necessary and sufficient

conditions. Before discussing Stein's study in detail, some discussion is warranted regarding the use of ICS (a) as a primary reinforcer, and (b) as the primary reinforcer in the establishment of a conditioned reinforcement effect. As Mogensen and Cioe (1977) pointed out, since the initial discovery of ICS as a reinforcer and its designation as the neural substrate for conventional reinforcers, and as identical, therefore, in nature to conventional reinforcers (Olds, 1956), evidence has mounted regarding dissimilarities between the two (ICS and conventional reinforcers). These authors examined ICS variables in the context of acquisition, extinction, secondary reinforcement, priming, and partial reinforcement, among others, and concluded that apparent dissimilarities are attributable to procedural differences, which, when adequately equated, result in negligible differences between conventional reinforcers and ICS.

The use of ICS as a primary reinforcer in the establishment of a conditioned reinforcement effect, offers some inherent benefits for addressing the fundamental question of the necessary and sufficient conditions for Sr establishment. In using more traditional SRs, as implied above, the possibility of confounding by the consummatory process poses difficulties from the standpoint of differentiating among various theoretical interpretations of the critical conditions for the establishment and operation of Sr s: for example, discriminative stimulus

(Keller & Schoenfeld, 1950), response contingent (Silverstein & Lipsett, 1974; Bilbrey & Winokur, 1975), and elicitation (Keehn, 1962; Wike, 1966) theories.

Because ICS requires no consummatory response, and, in specific neural sites, elicits no observable unconditioned response (UCR), to which response facilitation interpretations might be adduced, it possesses the potential, for delineating among competing theoretical perspectives. Further, it offers the advantage of greater experimental control over training parameters (e.g., pairing configuration, magnitude, directness of presentation, timing of delivery) and is highly resistant to satiation effects during training. Finally, it is important to note that the use of ICS as the primary reinforcer circumvents, because of the absence of the need for a preexisting drive or deprivation state, the problem of requiring that any particular drive state be present in the test phase, which could confound interpretations of the outcomes.

Returning to Stein's (1958) study, subjects were placed in a two lever chamber and followed presses on one lever consistently with a tone (Phase I). No preferences were indicated for either lever. During the second phase of the experiment (Phase II), subjects were delivered paired presentations of the tone and ICS in the absence of the levers. Tone onset preceded ICS by 0.5 s and terminated simultaneously with ICS offset after 1.0 s of

tone presentation. Phase I conditions were then reinstated as a test of the newly acquired Sr effects of the tone (Phase III). Finally in the last phase of the experiment (Phase IV), ICS was made contingent upon the performance of the lever press response. The latter was a test of the SR effects of ICS sites. Comparisons were made between Phase I and Phase III for those subjects who demonstrated SR effects in Phase IV. Preferences for the tone-contingent lever as well as increases in response rates were evident.

Some control problems are evident in Stein's procedure. In order to firmly establish the necessity for stimulus contiguity, it would have been necessary to include a randomly presented neutral stimulus during Phase II of training. Without the addition of this control, conclusions necessarily must be considered tentative.

However, the suggestion is that Stein was able to develop a conditioned reinforcement effect in the absence of observable elicitation effects. Stein also pointed out that the absence of a response requirement associated with the presentation of ICS, either approach or consummatory, rules out the necessity for the development of a discriminative stimulus function for the Sr. He further argued against the likelihood of a superstitious effect, that is, the reinforcement of a specific operant by chance, because of the brevity of the 0.5 s interval between tone and ICS onset.

Observations of subjects during this 0.5 s interval

during the training phase, failed to yield any evidence of response effects. While a description of a rigorous controlled observational procedure would be necessary in order to provide more conclusive evidence of the absence of systematic response effects in the conditioned stimulus (CS)-unconditioned stimulus (UCS) interval, it is hypothesized here that the effect of the SR in testing was due to the transfer of some fraction of the excitatory response (RE or UCR) associated with the delivery of ICS. While Pliskoff, Hawkins, and Wright (1964) did observe "superstitious" behavior during the interstimulus interval (ISI), it remains the fact that Stein was able to demonstrate an SR effect in the absence of observing any such behavior, and that defines the lower limit of the parameters of SR establishment using ICS as the SR.

Stein's results have been somewhat controversial primarily from the standpoint of unsuccessful attempts to replicate his findings. Seward, Uyeda, and Olds, (1959) used an extinction paradigm in order to assess the ability of ICS to impart sufficient reinforcing strength to the neutral stimulus to prolong extinction. In addition to the use of a paradigm with which there are serious methodological problems, Seward, et al., employed a simultaneous pairing of the neutral stimulus and the SR. The latter is often used as a control procedure in studies of classical conditioning on the grounds that the neutral stimulus acquires no CS properties in this way (Hall,

1976). In light of the paradigm and pairing configuration problems outlined, Seward, et al.'s conclusion regarding the inability to establish an Sr using ICS as the SR is not warranted.

Mogensen's (1965) replication effort also failed to find significant Sr effects. It is difficult, however, to critically evaluate Mogensen's study, because of the paucity of information offered in the report of the procedure and results. Without substantially more detailed information, it is impossible to determine if Mogensen's findings offered a credible challenge to those of Stein. It is important to note, however, that this same author (Mogensen and Cioe, 1973), in a subsequent review of the literature, concluded that a conditioned reinforcement effect can be established, using ICS as the primary reinforcer. These authors accounted for the conflicting results of this set of studies by referring to the possible importance of pre-experimental deprivation states in establishing an Sr effect. Indicating the use of non-deprived animals in the Seward, et al. (1959) and Mogensen (1964) studies, and the absence of relevant data on this point for Stein's (1958) and Knott and Clayton's (1966) procedures, the authors cited related evidence (DiCara, 1966; DiCara & Deutsch, 1966) suggesting the importance of deprivation states in the investigation of ICS produced Sr effects.

To the extent that deprivation state is critical, the

importance of this explanation cannot be taken lightly in terms of the theoretical explanations of Sr. The requirement of a deprivation state for Sr learning has implications for drive reduction, arousal, elicitation, and expectancy hypotheses (among others) of Sr establishment and operation. Given its theoretical impact, it seems unlikely that Stein and Knott and Clayton would have failed to mention the use of a deprivation procedure in their presentation, had it been a factor in their studies.

Knott and Clayton (1966), employing a delayed pairing configuration with a 0.5 s ISI in a Pavlovian training procedure, found that experimental subjects in a new learning test demonstrated a significant Sr effect. Knott and Clayton employed three groups; a 100% pairing group, a partial (50%) pairing group, and a group which received the tone but no ICS. While Knott and Clayton's controls were only slightly more adequate than Stein's, the confirmation of Stein's effect subsequent to two contradictory outcomes, supports the possibility that Stein's results are more than artifact. Further, Knott and Clayton obtained a significantly stronger effect for the partial pairing group relative to the continuous group, which suggests a differential learning effect rather than a sensitization effect for which the opposite result would be likely.

Using morphine as an SR, Crowder, Smith, Davis, Noel, and Coussens (1972) conducted a study from which additional conclusions supportive of the present hypothesis can be

drawn. Using a model similar to Stein's, Crowder, et al. placed animals in a single lever operant chamber for a 5 hour pretraining period, for the purpose of establishing a baseline. Each response was followed by the presentation of a buzzer together with an infusion of 0.018 ml of saline. At the end of the 5 hour baseline period the lever was removed and subjects were presented with 100 Pavlovian buzzer-morphine pairings.

Testing followed the next day, beginning at the same time as the original operant period. With the lever once again in the chamber, subjects were delivered buzzer-saline pairs contingent upon lever pressing. Animals were then given a second 5 hour test period in which buzzer-morphine pairings were delivered contingent upon lever pressing. The latter session was used to delete subjects not responding to the morphine as an SR. Three groups of subjects, each receiving different doses of morphine, were used.

A significant increase above baseline operant levels was found for responding in the test phase, as well as a significant positive effect of magnitude of morphine. Like Stein's study, no random CS presentation controls were used, therefore rendering the results somewhat inconclusive. However, a within-subjects effect (baseline to test) comparable to that obtained by Stein was identified. As with the Knott and Clayton finding of a partial reinforcement effect, the finding by Crowder, et

al. of differential dosage effects is somewhat more convincing of an Sr effect. In addition, attending to the criticism of Stein's work by Pliskoff, et al. (1964), subjects in the Crowder, et al. study were closely observed for the development of superstitious behavior in response to the buzzer in both the Pavlovian training phase and the lever training phase. No stereotyped behavior was observed.

To the extent that the results of Crowder, et al., together with those of Stein and Knott and Clayton, can be interpreted as indicative of a conditioned reinforcement effect, they appear to eliminate the need for an overt response on the part of the subject in order to attach Sr effects to the neutral stimulus, and are at odds with the results of some studies (e.g., Silverstein & Lipsett, 1974) which appear to indicate that a training-phase response of some kind is required for the establishment of such effects.

Silverstein and Lipsett (1974) required human infants in one group to perform an operant in order to receive paired presentations of a tone and food. Another group received the tone and food non-response-contingently during training, while a third group received the tone as a discriminative stimulus for responding in order to receive the SR. It is noteworthy that this study is one of the few to have used an explicitly unpaired within subjects control stimulus. These authors found that only the group who had

received the stimulus pair contingent upon responding demonstrated any Sr effects. Results were interpreted as indicating that a neutral stimulus must not only be contiguous with the SR but also must be contingent upon some response during training in order to establish an Sr effect.

The results of this study are not easily accounted for in terms of the contiguity hypothesis proposed herein. However, it is noteworthy that the Sr effect in this procedure was the result of only 20 pairings. The critical distinction between the response contingent and Pavlovian groups involved the provision that, for the former, an R-S sequence be established whereby the subject produced a specific identifiable response, which was then followed immediately by the neutral stimulus paired with the SR. It is possible that, because of the response requirement, subjects in this group were assured of discriminating the critical pair of stimuli from competing environmental cues, and thereby received the maximum exposure to the pairing procedure. Because no particular pattern was required of subjects in the Pavlovian group, it cannot be guaranteed that they discriminated the contiguous relationship between the neutral stimulus and the SR, particularly in light of the limited number of pairings used. Data on the latency between SR delivery and the eating response were not presented, but might, if available, shed some light on this hypothesis. Of note, however, is the fact that the

combined means for number of responses to both the "Sr" and the neutral stimulus in the non-response-contingent group were roughly equivalent to the Sr in the contingent group. This perhaps indicates that some excitatory effect had accrued to both stimuli in the non-response contingent group at this early point in training as a result of the subject's inability to discriminate any contiguous relationship between the neutral stimulus and the primary reinforcer. Regardless of the interpretation of the results, however, the use of adequate controls indicates the establishment of a genuine Sr effect.

A study by Doerries (1974) does not specifically address the issue of the necessity for response contingent training but does, nevertheless, shed some light on the issue. Doerries employed a training procedure whereby one group of subjects was required to perform only one training task while a comparison group was required to perform four training tasks. No differential effects were found for this variable. Two possible interpretations were offered. The first was based on the fact that all four tasks were distinctly different from the test response thereby limiting the predicted generalizability of the multiple task procedure. The second alternative was based on the similarity of the last response in the operant chain for all four tasks, namely that of the food cup orientation and the consummatory response. It was suggested that the similarity of this latter factor outweighed any differences

established in the earlier portions of the response chain, thereby functionally eliminating the intended multiple task procedure.

While these explanations are plausible, the results obtained also would be predicted by the contiguity hypothesis. According to this hypothesis, the critical factor is that the subjects in different groups discriminated both stimuli in succession regardless of the conditions upon which they are delivered. Following this line of reasoning, it seems entirely possible that equivalent results were obtained due to the fact that both the single task procedure and the multiple task procedure were equally effective in insuring that the subject discriminated the contiguous pairing.

Powell and Cole (1973), investigating the effects of interspersing re-pairing of Sr with SR in testing, reported evidence suggesting that not only is contiguity insufficient for the establishment of an Sr effect, but that a response contingency is inadequate as well. These authors used a discrete trial procedure in which animals were reinforced in a distinctive goal box for running down a straight alley. After continuous reinforcement for the first nine trials, subjects were reinforced on a VR 2 schedule across the remaining 41 trials with a distinctly different goal box present at the end of the alley on non-reinforced trials. During testing, subjects were presented with the T-maze discrimination task with the food

paired goal box as the Sr. For one half of the subjects, however, additional pairing was provided prior to each test trial by allowing the subject to consume the food after being placed directly in the appropriate goal box detached from the T-maze.

Powell and Cole found that, while re-pairing resulted in a preference for the food-paired goal box in the test phase, no preference was indicated for the non-re-pairing group. Aside from obvious control issues such as the need for equivalent pairing between testing groups, the study's results are questionable. While the authors argued that the superiority of the re-pairing group and the failure to find significant within subject differences for the response contingent contiguity group indicates the inadequacy of a contiguity approach (whether response contingent or not), it is more likely the case that the number of pairings (30) provided to the latter group was insufficient to sustain a T-maze preference. While each presentation of the Sr during the test phase of the contingent contiguity group began to induce extinction, such effects were eliminated in the re-pairing group. It is interesting to note that re-pairing over 18 test trials could establish a significant preference subsequent to the elimination of the response requirement imposed during testing. While appetitive and consummatory behaviors continued to be present in the test phase, a major portion of the response chain was eliminated. Although this piece

of evidence would not stand alone in support for the Pavlovian contiguity model, it does indicate a compatible outcome.

A final study which must be examined in this context is that of Keehn (1962). Unlike Silverstein and Lipsett (1974), who focused on response requirements prior to the presentation of the Sr in training, Keehn focused on responding subsequent to the presentation of the Sr. Simplifying the explanation of Keehn's procedure considerably, water deprived subjects were required to remain in one compartment for 30.0 s at the end of which a 2.0 s tone (Sr) was presented which was then followed by the opening of a partition permitting subjects to enter an adjacent compartment by running under or climbing over a barrier and consume water from a drinking tube.

In the test phase, one group of subjects was required to lever press for the tone and then was permitted to follow the same chain of stimuli and responses with the exception that water was no longer available from the inoperative drinking tube. Another group of subjects upon receiving the lever-press contingent tone was required to perform a new operant (the opposite of the barrier climbing or running response used in training) in order to gain access to the drinking tube. In a second test phase of the experiment, following retraining, a third group was kept in the original compartment by virtue of keeping the partition closed.

Results indicated that only those subjects permitted in testing to engage in the same response following the tone as that which was required during training demonstrated any conditioned reinforcement effects. Keehn concluded from these data that merely pairing a neutral stimulus with an SR was insufficient to establish a conditioned reinforcement effect. Rather, he argued, after the delivery of the Sr, it is necessary for the subject to complete a large portion of the appetitive and consummatory response chain previously associated with the primary reinforcer in order for Sr effects to be apparent. However, a closer examination of this conclusion is required.

First, a review of Keehn's procedure indicates that the design in the test phase resembles more closely extinction in an explicit chaining procedure than the typical new learning procedure. Subjects were allowed to press a lever (or touch a rod) (R1), exit from the first compartment (R2), cross the second compartment (R3), and engage in the consummatory response (R4). The designated Sr was delivered subsequent to the performance of R1 and terminated prior to R2 occurring (the partition opened immediately subsequent to the offset of the tone), thereby prohibiting the occurrence of R2 prior to the offset of the tone. The Sr in this procedure is similar to the discriminative stimulus used by Silverstein and Lipsett (1974) in that the pairing was established using a trace

conditioning design. The more traditional, and effective, procedure, for Sr pairing, is the delayed procedure in which the delivery of the Sr continues to the start of, or even overlaps with presentation of the SR.

It is generally accepted that trace conditioning is a far more difficult procedure than delayed conditioning with which to get significant effects (Hall, 1976). Given that this trace conditioning procedure employed only 68 paired presentations of the Sr and SR, it is not surprising that the tone by itself was unable to strengthen a new operant and that feedback from an approach chain was needed to mediate the time interval.

Another problem involving those subjects required to learn a new response in order to gain access to the drinking apparatus is that they were required to learn not one, but two new operants with the tone serving as the Sr for one and discriminative stimulus for the other. This rather stringent requirement is uncommon in new learning procedures, especially because of the weak effects generally found using this paradigm.

Thus it can be seen that the designation of the tone as the Sr seems to be a rather arbitrary one. From the perspective of a chained procedure, subjects in the first group were allowed to engage in each sequential aspect of the chain up to the point of fluid intake, subsequent to the delivery of the identified Sr. For the second group, a new response was required subsequent to the delivery of the

Sr, resulting in a substantial disruption of the chain, while for the third group, the portions of the chain subsequent to the presentation of the Sr were blocked. In addition, it must be added that for Group 1, training and testing chambers were identical, while for the remaining groups, substantial environmental differences between training and testing were needed in order to achieve the differences in response requirements. It is in fact probable that the Sr in this procedure involved a stimulus complex incorporating minimally, the opening of the partition, the distinctive cues of the second compartment, and the empty drinking tube, among which the designated Sr (the tone) was relatively inconsequential. The fact that this complex was unchanged only for the group of subjects permitted in testing to engage in the same operant required in training can easily account adequately for their striking superiority. This latter interpretation of the findings could more succinctly be identified as a substantial difference in stimulus generalization decrement from training to testing. At most, the Keehn results indicate that an unchanged response is required when a long-term trace conditioning procedure is used during Sr training.

Before proceeding to discuss more specific issues with regard to the Pavlovian contiguity hypothesis, it is important to note that the results of studies using a discriminative stimulus model for the establishment of Sr

(e.g., Dinsmoor, 1950; Schoenfeld, Antonitis, & Bersh, 1950) are compatible with the contiguity hypothesis. Provided that the requirements of contiguity are met, it is assumed that in order for a stimulus to function as a discriminative stimulus, it must have initially been established as an *Sr*, that is, it must have accrued some excitatory component, *re*, of the consummatory response. Having been thus enhanced, it is thereafter capable of signaling the appropriateness of an approach response to the *SR*. The continued contiguity of the discriminative stimulus with the *SR* would in fact enhance the *re* and thereby enhance the discriminative stimulus's *Sr* strength. It is important to note, however, that differences in contiguity exist between the traditional contiguity procedure (Pavlovian or response contingent) for *Sr* establishment and the discriminative stimulus approach to *Sr* establishment. The latter approach, by virtue of requiring an operant between the onset of the neutral stimulus and the delivery of the *SR*, involves a substantially a longer *ISI*. This point is important, as it would suggest differential effectiveness in the use of traditional contiguity and *SD* approaches, favoring the former.

In contrast, the results obtained by Egger and Miller (1962) and their subsequent interpretation are not compatible with the strictly Pavlovian model. However, as pointed out earlier, more recent studies have indicated

that the most potent Sr effect is obtained by using the training stimulus compound in testing. In light of such findings, Egger and Miller's results were interpreted as indicative of a stimulus generalization decrement phenomenon. These latter findings render the results of information hypothesis studies to date compatible with the contiguity hypothesis.

In order to explicate the details of the Pavlovian contiguity hypothesis as stated, several specific predictions regarding magnitude of the SR, number of pairings, and pairing configuration will be evaluated. In order to better understand the parameters of e , variables affecting the excitatory response, re , must be systematically discussed with specifications of predicted outcome and supportive evidence. The differential effect of magnitude of the primary reinforcer would be expected to strike a balance between the extent to which increasing magnitude of the SR can be employed without reaching satiation. Satiation, in this context, is relevant in terms of the continuation or cessation of consummatory responding, the result of which involves the elicitation of the excitatory response (RE) (and consequently re), for more conventional SRs. Given that re may vary in strength, variations in magnitude of SR would be predicted to result in differential re strength and consequently differential Sr strength. Magnitudes of the SR beyond satiation would terminate consummatory responding and therefore the

critical associative process. As long as subjects remained below threshold for satiation of the SR, however, variations in the magnitude of Sr would result in a differential strength of re for each neutral stimulus. Parameters such as the amount of primary reinforcer delivered in contrast to the time allowed for consumption are critical as well; for example, if more water is available than can be consumed in the water access period, differences in the strength of Sr should not be as apparent.

The previous study mentioned by Crowder, et al. (1972) provides support for the aforementioned prediction. These authors varied the concentration of the morphine solution used as the SR from 0.0032 mg/kg to 0.032 mg/kg to 0.32 mg/kg across groups. Results indicated that the magnitude of the conditioned reinforcement effect increased linearly with the quantity of morphine used. It would be predicted that increasing dosages would result in an extended linear trend up to the point at which the dosage of morphine was capable of transferring no greater re s. At this point, the curve would be expected to reach an asymptote. Using a differential procedure, Catania (1963) found that rate of pecking on two independent keys varied in proportion to variation in the duration of SR (food) availability (3.0 s vs. 6.0 s). Using an absolute training procedure, Butter and Thomas (1958) were able to demonstrate differential effects of Sr using varying

110
concentrations of a sucrose solution. The same pattern would be predicted for ICS. However, caution would be required in increasing the magnitude of the ICS (and other SRs, for example, morphine) beyond the asymptote in order to avoid the development of avoidance effects, from a stimulus intensity which was aversive.

As Lawson (1957) pointed out, magnitude of reward can be defined in terms of the amount of reward available to the subject, the amount of consummatory activity permitted and the "taste-stimulation" or level of excitatory activation per presentation in the more general sense. On the assumption that these distinctions make little difference in the specification of principles of SR magnitude effect, Lawson points out, that his study, as most others, avoids distinguishing among these operations. However, while Lawson, downplays the importance of the distinctions among these subcategories, the differences in terms of the present hypothesis are critical. The most critical subcategory for producing a differential effect of magnitude is the last, that is, the level of excitatory activation. It is this category which is varied in the studies of Crowder, et al. (1972), and Butter and Thomas (1958) .

Negligible effects would be expected from the remaining subcategories of SR magnitude with the restriction of a lower limit permitting the full activation of the consummatory response before access to the SR was

terminated. This latter restriction is critical given that inadequate activation of the consummatory response could be assumed to result in a diminished intensity of RE, therefore, resulting in diminished activation of re and association of that diminished re to the neutral stimulus. Given the activation of the full consummatory response, that is, subjects being permitted full access to some SR, variations, in an absolute design, in the quantity (number of units) of that SR (assuming that incentive value per unit is held constant) or the length of time permitted for access would be predicted to yield negligible results. Placed in an absolute test situation, those subjects would be expected to perform to a single maximum level, whether rewarded with the SR associated with the greater quantity of SR or the lesser quantity. This prediction is based upon the hypothesis that for any given magnitude of a unit of SR, regardless of the number of units available, only one maximum level of RE is possible.

Results of Catania (1963), D'Amato (1955), Hopkins (1955), Kelleher (1966) and Lawson (1957) are consistent with the hypothesis that, for any given magnitude of a unit of SR, only one maximum level of RE is possible. Briefly these authors found that varying quantity, quality, and duration of an SR in an absolute test revealed no between group differences (Catania, 1963; Hopkins, 1955; Lawson, 1957). On the other hand, with the use of a differential procedure, within group differences were observed (Catania,

1963; D'Amato, 1955; Kelleher, 1966; Lawson, 1957), a result which indicates preferences for more vigorous re s.

With respect to the parameter of number of pairings, the Pavlovian contiguity hypothesis would predict that as the number of pairings of the Sr with the SR increases, up to an empirically identifiable point, the strength of the Sr would increase accordingly. Beyond that point increases in responding would be expected to level off. This finding would be predicted on the basis of the number of pairings being critical to the maximization of re for the Sr in question, as in any Pavlovian conditioning process. It is assumed that for any SR, there is a given RE and that optimal contiguity will permit transfer of a maximum fraction of that response. Theoretically, a set number of repeated pairings would be necessary in order for that maximum to be reached. Fewer pairings would result in weaker Sr effects as a result of the elicitation of a weaker association of re to the Sr. An increased number of pairings, on the other hand, would not be expected to yield any effects beyond the asymptotic maximum. Studies by Bersh (1951), Hull (1951), and Miles (1956) confirm these predictions.

Klein's (1959) examination of intermittent pairing with the SR during training would at first glance seem to contradict this prediction, in that Klein found an inverse relationship between intermittency and Sr strength. Saltzman's (1949) examination of the relative strength of

continuous and intermittent training when facilitated by re-pairing during testing suggests that Klein's results are indicative of an extinction phenomenon rather than an Sr effect. (For more detailed discussion of Saltzman's study, see p. 128.) Saltzman's results indicated greater reinforcement strength, but more rapid extinction, for the continuously reinforced group as compared to the alternately reinforced group. These results would suggest that intermittent pairing in the Klein study delayed extinction of an established Sr to varying degrees rather than having established varying degrees of Sr strength. Together, these results can be interpreted as empirically supportive of a Pavlovian conditioning of excitatory response hypothesis regarding the parameter of number of pairings.

Because of the importance of temporal contiguity in this model, the pairing configuration is also a critical variable. While this issue was discussed at an earlier point in the context of Keehn's (1962) and Silverstein and Lipsett's (1974) studies, it bears further consideration as a separate issue. Predictions along this line favor the simultaneous forward conditioning or delayed procedure wherein the neutral stimulus onsets prior to and overlaps with the onset of the SR. This condition permits optimal contiguity between the neutral stimulus and the SR. The trace conditioning procedure wherein the neutral stimulus offsets prior to the onset of the SR would be predicted to

be somewhat less effective due to less optimal contiguity and consequently the possibility that some discriminable stimulus between the offset of the neutral stimulus and the onset of the SR would interfere with conditioning. The issue of discriminability of the neutral stimulus is likewise a problem in the context of "simultaneous" conditioning (not to be confused with simultaneous forward conditioning) wherein both the neutral stimulus and the SR are delivered simultaneously. Because of the activity generated via the consummatory response and its associated stimuli, it is minimally likely that the neutral stimulus will be discriminated in the simultaneous paradigm. This line of reasoning also applies to the case of backward conditioning as supported by Doerries (1974), wherein the neutral stimulus onsets subsequent to the onset of the SR. While this author knows of no comparative studies in the area of conditioned reinforcement which have directly addressed the issue of the pairing configuration, these predictions are in line with results of classical conditioning studies (see, e.g., Terrace, 1973) in which these procedures have been employed.

Along this line, it is important to consider briefly the issue of variations of the ISI when using the delayed (or trace) procedure. It would be predicted from the Pavlovian contiguity hypothesis that the same problems raised with respect to the trace conditioning procedure and the simultaneous and backward procedures, exist here. That

is, it would appear that the discriminability of the relationship between the neutral stimulus and the SR in training would be hampered either by long delays (approximating problems of the trace procedure) or extremely short delays (approximating problems of the simultaneous procedure). The resultant performance curve according to these predictions, should approximate an inverted U with the optimal ISI for the attachment of re to the neutral stimulus occurring at some intermediate though reasonably close point relative to the SR. These predictions are supported by the results obtained by Bersh (1951), though he used an extinction paradigm to obtain them. As is the case in virtually all areas of the field, more stringent tests are necessary, but preliminary indications are compatible with a Pavlovian contiguity approach.

To the extent that there are different internal loci for SRs, as would be suggested by the variety of neural sites identified both as appetitive for ICS and as correlated with various forms of consummatory responding, it is conceivable that a cumulative effect would accrue when the neutral stimulus is paired across different SRs. Denny and Ratner (1970) point out is that it is possible to differentially elicit consummatory responding with subcortical stimulation of appropriately placed electrodes (Roberts, W. W., Steinberg, M. L., & Means, L., 1967), suggesting the occurrence of electrical impulses in the

subcortex in correlation with consummatory behavior.

Research involving the pairing of ICS in multiple sites with a single neutral stimulus is necessary in order to investigate the hypothesis that it is possible to develop a cumulative Sr effect across those sites. This hypothesis follows from the non-specific (re: SR) nature of re previously postulated.

Once established, according to the Pavlovian contiguity model, the Sr functions independently. As previously indicated (p. 92), the presentation of Sr contingent upon a response elicits re. As a result of this property, the Sr is thereafter capable of functioning in a manner which strengthens performance of the response upon which it (the Sr) was originally contingent or in a manner which conditions a new response which it (the Sr) is subsequently made contingent. While the Sr effect weakens or extinguishes when presented without SR, and must therefore be strengthened through re-pairing with the original SR or with a new SR, it operates for an identifiable period independent of a preexisting drive state or the incentive value of the original SR.

It must be pointed out that, whereas re elicitation effects may have accrued to the neutral stimulus, it does not necessarily follow that a demonstrable Sr effect will be obtainable in a new learning situation. As indicated earlier, the failure of the training procedure to adequately attach re s of sufficient strength would result

in a Sr whose strength is below the threshold necessary for the establishment of an Sr effect in the testing situation in question. Variations in learning difficulty of the test response would impact upon the demonstration of Sr effects as well. Given, as Denny and Ratner (1970) speculate, that consummatory responses (UCRs) associated with conventional SRs can be elicited by specifically located ICS (or the unconditioned stimulus [UCS]), it should be possible to elicit similar, though less potent, consummatory responses subsequent to the presentation of a neutral stimulus paired with selectively located ICS. Using an ICS-paired CS (or Sr) to elicit a consummatory response, while using the same CS in an experimental procedure to vary either training parameters or test difficulty in order to demonstrate a differential Sr effect, would support the existence of a threshold construct. That is, by increasing the potency of the training procedure or reducing test difficulty, it should be possible to impart to an ICS-paired CS, which elicits only fractional consummatory responses (but is not of sufficient strength to demonstrate an Sr effect), the ability to function as an Sr in a new learning test.

An additional consideration is also important to note. One of the most critical properties of the Sr is that it is learned. Therefore, with each successive presentation of the Sr in the absence of the SR, its effects extinguish. This phenomenon presents a problem wherein, as a new response is being reinforced by the Sr, its Sr strength is

diminishing. If the latter occurs more rapidly than the former, only a careful analysis of the early stages of testing will yield any results if any are, in fact, to be found. These two factors, that is, the failure of Sr strength initially to reach threshold, and the rapidity with which an Sr effect, once learned, extinguishes, account for the failure of many studies to obtain reliable and durable effects.

In spite of these difficulties, some predictions are possible within the context of the Pavlovian contiguity hypothesis regarding the operation of Sr s in learning situations. It would be predicted from the Pavlovian model that once excitatory effects had accrued to the Sr, the Sr could function somewhat autonomously, that is, independent of the conditions used to establish the Sr. Some support is offered for this position by D'Amato's (1955a) study investigating the ability of Sr effects to transfer across drive states (hunger and thirst) from training to testing. D'Amato found that Sr effects could be reliably transferred from hunger to thirst (and vice versa) and therefore concluded that when motivational states are roughly equivalent from training to testing (a point not empirically verified), Sr effects will transfer across those motivational states. It is important to note, however, that hunger and thirst deprivation states involve a number of stimuli which overlap across the two conditions, thereby attenuating somewhat D'Amato's

conclusions. conclusion is in accord with the Pavlovian contiguity hypothesis. However, a strictly Pavlovian theoretical point of view would further predict that the incentive value of the Sr would in fact function autonomously, that is, in the absence of any experimentally prearranged drive state. Nevin (1966) trained deprived subjects to respond for food or water as the primary reinforcer using a buzzer as an SD. Subjects were then satiated and tested. Results indicated a significant preference for the lever producing the buzzer in a two-lever test chamber. Although some authors have failed to obtain an Sr effect with satiated subjects (Schlosberg & Pratt, 1956; Platt & Wike, 1962), several others (Gilbert & Sturdivant, 1958; Platt & Wike, 1964; Seward & Levy, 1953; Wike & Casey, 1954) have observed positive effects similar to Nevin. Wike and Casey (1954), for example, found that satiated subjects would traverse a runway toward a goal box containing food at a faster rate than control subjects running toward an empty box. These authors concluded that sight of food would function as an effective conditioned reinforcer for satiated subjects. The results of these studies support the latter prediction of Sr effects in the absence of a drive state in testing. In lieu of the development of the optimal Sr effect, a preexisting motivational state could interact with the Sr to enhance the conditioned reinforcement effect by increasing overall responding and thereby increasing the

likelihood that the response of interest will be emitted in testing with a frequency sufficient to permit the Sr to establish a selective effect. Studies in which variations in levels of deprivation, that is, corresponding variations in arousal, are contrasted against supra- and subthreshold parameters of Sr establishment, for example, number of pairings with differential outcomes resulting, would support a prediction of interaction of drive state and Sr strength. Further research is necessary to verify the parameters of the such an effect.

The final major issue which requires some attention is that of durability. Because of the fact that conditioned reinforcement is invoked to account for such robust phenomena as the reinforcing power of money, praise and interpersonal attention, among others, the failure of studies of conditioned reinforcement to find fairly durable effects has drawn sharp criticism (Bolles, 1975; Longstreth, 1971).

Before responding to this issue in terms of empirical predictions and data which support those predictions, an important point must be made about the requirement that the laboratory must duplicate real life. While the criticisms raised in the literature on the durability issue are not without merit, it must be remembered that in real life, humans are exposed to innumerable pairings and re-pairings of neutral stimuli with already developed Sr s and SRs. To attempt to duplicate the pairing procedure typical of most

real life conditioned reinforcers would be an overwhelming and ill-advised longitudinal task for the average experimental laboratory.

Still, despite the substantial contrasts between the laboratory and real life, it remains important to obtain an SR effect with some reasonable measure of durability. From the context of a Pavlovian model, it would be predicted that in order to bolster the diminishing SR strength due to the extinction of re, occasional re-pairings of the SR and SR or reconditioning would be necessary. (This prediction begins to replicate what is assumed to be the "natural" pairing procedure which occurs in the everyday maintenance of conditioned reinforcers.)

Within the area of classical conditioning, intermittent re-pairing has been found to restore measurable CRs to their original strength. Saltzman (1949) and Powell and Cole (1977) have obtained durable effects using interpolated re-pairings for every test trial. Notably, for the subjects in Powell and Cole's study, no response beyond that of the eating the food pellet in the appropriate, detached goal box was required during the re-pairing procedure.

To the extent that this prediction is valid, the procedure used in both of these studies was liberal, that is, a re-pairing trial was matched with each test trial. Strong support requires the use of a considerably more conservative re-pairing procedure. Zimmerman's (1959)

moderately well-controlled procedure indicates promising results along this line. During a discrete trial training procedure, subjects were first trained on a continuous schedule and then transferred to an increasingly intermittent VR schedule. During testing, subjects were required to press a lever which was followed by a 2.0 s buzzer or ready signal and the opening of the start box door. Subjects received the Sr complex of the buzzer, start box door opening and runway access on a continuous schedule at the outset of testing and thereafter received an increasingly thin FR schedule of Sr presentation which ultimately reached a value of 1:20. Not only did experimental subjects indicate significant Sr effects over the testing period but responding was characterized by the traditional cumulative response curve obtained on an FR schedule of reinforcement (Ferster and Skinner, 1957). These results offer strong support for the prediction that durable effects can be maintained with intermittent re-pairing during testing. Saltzman (1949) found that subjects continuously reinforced during training made more correct choices prior to the onset of extinction of the Sr during the testing phase of a discrete trial paradigm than did those subjects who were intermittently reinforced. While the continuously reinforced subjects showed less of an effect initially, relative to the alternately reinforced group, Saltzman was able to eliminate this difference by employing interpolated reinforced trials during testing.

Continuous reinforcement during training was therefore interpreted as the more effective procedure for the establishment of Sr effects, but subject to more rapid extinction during testing and therefore enhanced in durability with the addition of re-pairing trials. The extent to which intermittent reinforcement during training interacts with the reconditioning effect requires further investigation.

Effects of distributed versus massed practice during testing would be expected to follow the Pavlovian model in terms of a spontaneous recovery effect. That is, to the extent that small block trials of massed practice are spaced across time within a limited period of time, Sr effects would be predicted to be enhanced by a spontaneous recovery phenomenon. This appears to be the case, as indicated by the results of Doerries (1974) in which distributed practice during testing was found to be superior to massed practice, due to a spontaneous recovery effect.

The concept of a "generalized conditioned reinforcer" (GCR) has been proposed by Skinner (1953) to account for the substantial reinforcement strength and durability, of learned rewards, such as money, praise, attention, etc. It is suggested that these stimuli acquire and maintain their exceptional potency by virtue of their frequent association with a variety of primary reinforcers (incentives) and/or drive states. Several studies (e.g., Kanfer, 1960; Nevin,

1966; Wike & McNamara, 1955) investigating the GCR concept have yielded positive outcomes, while some outcomes have shown the contrary (Porter & Miller, 1957; Seward, 1963). As Wike (1966) and Kelleher and Gollub (1962) suggested, the former must be considered cautiously due to control problems, small, transient between group effects, and confounded deprivation states, among others. A Pavlovian contiguity model is compatible with the concept of a generalized conditioned reinforcer. The previous discussion of re in terms of the cumulative effects of pairing with several different SRs pertains here. That is, it has been suggested that is possible to elicit consummatory responding from several specific loci in the brain using ICS. It is assumed, therefore, that re s may have multiple neural loci, each associated with one or more primary reinforcers. While the magnitude of re for any one site is restricted by a maximum upper limit, the association of a previously neutral stimulus with more than one primary reinforcer would result in the attachment of multiple re s to the Sr. The effect would be cumulative therefore and would require a distinct extinction process for each re in order to lose the effect.

Given the paucity of data, the discussion of conditioned reinforcement herein could be easily interpreted as an elaborate effort to explain what is basically a null hypothesis. However, as indicated, a small number of studies with fairly well-controlled designs

have found reliable effects which are best understood in terms of some type of conditioned reinforcement phenomenon.

It is clear, however, that the studies to date provide no clearcut answer to the question of the necessary and sufficient conditions for the establishment of a conditioned reinforcement effect, to say nothing of the issues of operation and durability. Of paramount importance, therefore, is the establishment of a reliable Sr effect using a well-controlled new learning procedure.

Consistent with the existing data, it is hypothesized that the minimum necessary condition empirically for a conditioned reinforcement effect is Pavlovian temporal contiguity. Further, specific predictions are possible regarding the effects of number of pairings, magnitude of SR, configuration of the training pair, variations of the ISI, and the use of multiple SRs (see above) on the establishment of Sr effects. The question of the effect of training across drive states and under conditions of intermittent pairing during training also requires investigation.

In terms of both establishment and operation of Sr s, clarification is needed regarding the relative efficacy of contiguity variables and informational variables. Investigations of the interaction of drive states and Sr on new learning tasks are relevant to the issue of Sr autonomy. Relative to questions of operation, research regarding interaction of drive states and Sr threshold are

important, as well, in specifying the nature of the Sr effect. Studies confirming the ability of the Sr to function as a CS for overt consummatory responses, simultaneously with positive and null Sr effects resulting from procedural variations would lend further support to the understanding of Sr functioning. Perhaps of greatest importance to the issue of Sr operation, however, is the use of Sr s to establish a variety new learning effects.

In addition, further verification of the effects of distributed testing and re-pairing of Sr and SR during testing, upon the durability of the Sr is necessary, as well as the interaction of this latter factor with intermittent pairing during training. Further investigation of GSRs is needed, as well, with careful consideration of previous procedural difficulties.

It is unfortunate that substantial research efforts have focused for so long on reasonable questions with ineffective paradigms and inadequate controls. However, in spite of this vast body of literature which offers limited insight, the existence of the relatively small kernel of reliable results offers some hope that this widely cited phenomenon might be verified and understood.

Appendix A

Table A1

Outline of Sequence ofExperimental Phases

Phase I. Pretesting (two lever chamber)

A. Groups A, B, and C

1. one lever--neutral stimulus
2. one lever--no stimulus

B. Groups D and E

1. one lever--neutral stimulus (tone)
2. one lever--neutral stimulus (light)

Phase II. Pretraining (one lever chamber): all groups trained identically to press lever for continuous reinforcement schedule of ICS delivery

Phase III. Training (Pavlovian; no lever in chamber)

- A. Group A; neutral stimulus forwardly paired with ICS
- B. Group B; neutral stimulus randomly presented relative to ICS
- C. Group C; neutral stimulus presented in explicitly unpaired format relative to ICS
- D. Group D
 1. one neutral stimulus forwardly paired

with ICS

2. one neutral stimulus randomly
presented relative to ICS

E. Group E

1. one neutral stimulus forwardly paired
with ICS
2. one neutral stimulus presented in
explicitly unpaired format relative
to ICS

Phase IV. Testing

- A. Groups A, B, and C (identical to I. A.)
- B. Groups D and E (identical to I. B.)

Appendix A

Results:

Detailed Presentation

Six 3 x 2 x 2 ANOVAs (in two subsets of three each), with repeated measures across the latter two factors, were used to analyze the data. Factor A for three of the analyses compared three different groups, each of which were presented with a paired stimulus during training. Groups differed according to class of comparison control conditions for use with the ICS-paired stimulus (A1, no stimulus; A2, random stimulus; A3, explicitly unpaired stimulus). Factor A for the remaining three analyses compared three groups, each of which received only one stimulus, varied by training configuration on one lever (A1, paired; A2, random; A3, explicitly unpaired) with "no stimulus" on the alternate lever.

For one subset of three analyses, Factor B compared the paired (B1) stimulus with its control (B2) condition (no stimulus, random stimulus, or explicitly unpaired stimulus). For the second subset of analyses, Factor B compared the critical comparison (B1) stimulus (paired, random, or explicitly unpaired) with its "no stimulus" (B2) control. Factor C compared measures taken prior to pretraining with the same measures taken subsequent to training, specifically assessing average responding across three days of pretesting (C1) with responding on the day of

testing (C2). Table 1 represents the design and conditions for each of the two sets of analyses.

For each set of three analyses, three dependent measures were assessed; (a) frequency of lever presses, (b) frequency of stimulus presentations obtained as a result of lever pressing, and (c) cumulative duration of lever depression, measured in seconds.

Analysis 1

Means and standard deviations from the first analysis (measuring frequency of lever presses across paired/nothing, paired/random, and paired/explicitly unpaired groups) are represented in Table 2 (see Results, p. 22). Results of a test of the assumption of homogeneity of variance ($F_{\max} [12, 7] = 16.49$, n.s.), fail to reject the null hypothesis. (For all tests of the assumption of homogeneity of variance, a violation will be assumed for any $F_{\max}$ value exceeding the table value of $p < .01$.)

Results of the overall analysis indicate significant main effects for Factor A (Paired/Control Conditions), $F(2, 21) = 5.37$, $p < .05$, Factor B (Stimulus), $F(1, 21) = 6.13$, $p < .05$, and Factor C (Pretest/Test), $F(1, 21) = 24.43$, $p < .05$, and a significant interaction effect for the BC interaction, $F(1, 21) = 8.66$, $p < .05$. In other words, a significant difference is indicated (a) across paired/nothing, paired/random, and paired/explicitly unpaired groups (Factor A), (b) between the paired stimulus

Table 1

Schematic Design of ANOVA SubsetsSubset 1

	C1	C2
	Pre test	Test
B1 Paired	A1B1C1	A1B1C2
A1 Group*		
B2 No Stimulus	A1B2C1	A1B2C2
B1 Paired	A2B1C1	A2B1C2
A2 Group		
B2 Random	A2B2C1	A2B2C2
B1 Paired	A3B1C1	A3B1C2
A3 Group		
B2 Explicitly Unpaired	A3B2C1	A3B2C2

Subset 2

	C1	C2
	Pretest	Test
B1 Paired	A1B1C1	A1B1C2
A1 Group		
B2 No Stimulus	A1B2C1	A1B2C2
B1 Random	A2B1C1	A2B1C2

A2 Group

B2 No Stimulus

A2B2C1

A 2B 2C 2

B1 Explicitly Unpaired

A3B1C1

A 3B 1C 2

A3 Group

B2 No Stimulus

A3B2C1

A3B2C2

*n = 8 for each group.

and the control condition (Factor B), and (c) between pretest and test conditions (Factor C), as well as a stimulus (B) by pretest/test (C) interaction.

While no AB interaction was significant for the first analysis, or either of the remaining two, of a priori importance was the assessment of between group differences in responding for one (A1) or two stimuli (A2 & A3). For this reason, the analysis of simple main effects for A at each level of both B and C was performed.

Simple main effects tests of A at each level of B and A at each level of C indicate significant differences in levels of responding across A at B1, $F(2, 21) = 7.13$, $p < .05$, and across A at C2, $F(2, 21) = 4.85$, $p < .05$. In other words, subjects responded significantly more for the paired (B1) stimulus in the test (C2) phase than in the pretest (C1) phase. Newman-Keuls follow-up tests of both significant simple main effects tests indicate that subjects in the paired/nothing (A1) group responded significantly more than subjects in the paired/random (A2) and paired/explicitly unpaired (A3) groups and that paired/random subjects responded more than paired/explicitly unpaired subjects.

Simple main effects tests of B at each level of A indicate significance at A1 only, $F(1, 21) = 7.90$, $p < .05$, that is, paired/nothing (A1) subjects responded more on the lever producing the paired (B1) stimulus than on the lever

producing "no stimulus" (B2).

Simple main effects tests of B at each level of C indicate significance for B at C2, $F(1, 21) = 8.17$, $p < .05$, confirming the finding that subjects responded significantly more for the paired (B1) stimulus in the test (C2) than in the pretest (C1) phase.

Simple main effects tests of C at each level of A indicate significantly greater responding in the test (C2) phase for A1 subjects, $F(1, 21) = 10.76$, $p < .05$, for A2 subjects, $F(1, 21) = 8.64$, $p < .05$, and for A3 subjects, $F(1, 21) = 5.45$, $p < .05$. Simple main effects tests of C at each level of B indicate significantly greater responding for the paired (B1) stimulus in the test (C2) phase, $F(1, 21) = 33.06$, $p < .05$, and for the control (B2) stimulus (or "no stimulus" control) in the test (C2) phase, $F(1, 21) = 6.70$, $p < .05$. Omega squared values for the latter comparison yield results of 0.108 for C at B1 and 0.019 for C at B2, resulting in a relative magnitude for C at B1 of 5.68 times that of C at B2.

While no ABC interactions are indicated in the overall results for any of the first three analyses, a priori predictions of the locus of change within each group dictated the analysis of simple, simple main effects for B at each level of AC and C at each level of AB. Specifically, it was predicted that critical comparisons in assessing increased preferences for paired stimuli and controls would include specifically pretest versus test (C1

vs. C2) comparisons for each stimulus condition (B) within each group (A). Of somewhat less importance, are comparisons of preferences for the critical stimulus (B1) in the test phase (C2) as compared to its within subjects control (B2), as well as the pretest (C1) phase for each group.

Simple, simple main effects tests for B at each level of AC indicate greater responding for the paired (B1) stimulus as compared to its "no stimulus" (B2) control in the test (C2) phase for the paired/nothing (A1) group (B at A1C2), $F(1, 63) = 14.38, p < .05$. In other words for the paired/nothing (A1) group only, the paired (B1) stimulus in the test (C2) phase was preferred significantly more than the "no stimulus" (B2) control. No other significant pairwise differences for B at AC were identified.

Simple, simple main effects tests for C at each level of AB indicate greater responding for the paired (B1) stimulus in the test (C2) phase, as compared to the pretest (C1) phase in the paired/nothing (A1) group (C at A1B1), $F(1, 63) = 20.57, p < .05$, in the paired/random (A2) group (C at A2B1), $F(1, 63) = 9.72, p < .05$, and in the paired/explicitly unpaired (A3) group (C at A3B1), $F(1, 63) = 5.10, p < .05$. In other words, responding for the paired stimulus (B1) in all three groups significantly increased from the pretest (C1) to the test (C2) phase. Omega squared values for the comparisons of relative magnitude yield results of 0.067 for C at A1B1, 0.030 for C at A2B1,

and 0.014 for C at A3B1, resulting in a relative magnitude for C at A1B1 of 2.23 times that of C at A2B1 and 4.79 times that of C at A3B1 and a relative magnitude for C at A2B1 of 2.14 times that of C at A3B1.

Analysis 2

Means and standard deviations for the second analysis (measuring number of stimulus presentations obtained across paired/nothing, paired/random, and paired/explicitly unpaired groups) are represented in Table A2. Results of a test of the assumption of homogeneity of variance ($F_{\max}$ [12, 7] = 8.01, n.s.) failed to reject the null hypothesis.

Results of the overall analysis indicate significant main effects for Factor A (Paired/Control Conditions), $F(2, 21) = 5.61$, $p < .05$, Factor B (Stimulus), $F(1, 21) = 8.90$, $p < .05$, and Factor C (Pretest/Test), $F(1, 21) = 21.22$, $p < .05$, and a significant interaction effect for the BC (Stimulus x Pretest/Test) interaction, $F(1, 21) = 9.36$, $p < .05$.

Simple main effects tests of A at each level of B and A at each level of C indicate significant differences in responding across A at B1, $F(2, 21) = C2$, $F(2, 21) = 4.64$, $p < .05$, indicating that subjects obtained paired (B1) stimulus presentations significantly more in the test (C2) phase. Newman-Keuls follow-up tests indicate that for both A at B1 and A at C2, A1 (paired/nothing) and A2 (paired/random) were significantly greater than A3

Table A2

Means and Standard Deviations of Stimulus PresentationMeasure for Within Subjects Controls

Group	Stimulus		Pretest	Test
A1 (Group A)	Paired	<u>M</u>	25.75	46.50
		<u>SD</u>	17.44	17.20
	No Stimulus	<u>M</u>	25.00	31.00
		<u>SD</u>	14.14	8.38
A2 (Group D)	Paired	<u>M</u>	20.50	37.63
		<u>SD</u>	13.34	18.76
	Random	<u>M</u>	17.38	28.25
		<u>SD</u>	7.69	14.01
A3 (Group E)	Paired	<u>M</u>	10.50	24.00
		<u>SD</u>	6.63	14.83
	Explicitly Unpaired	<u>M</u>	11.63	18.63

SD

7.01

8.28

(paired/explicitly unpaired), that is, both paired/nothing and paired/random subjects received a greater number of paired stimulus presentations in the test (C2) phase than did paired/explicitly unpaired subjects. No differences were found between the paired/nothing and paired/random subjects.

Simple main effects tests of B at each level of A indicate a significant effect of B at A1 only, $F(1, 21) = 6.47$, $p < .05$, indicating that paired/nothing (A1) subjects obtained paired (B1) stimulus presentations significantly more than their "no stimulus" (B2) equivalent. Simple main effects tests of B at each level of C indicate significance at C2, $F(1, 21) = 18.01$, $p < .05$, confirming that subjects obtained paired (B1) stimulus presentations significantly more in the test (C2) phase, than in the pretest (C1) phase.

Simple main effects tests of C at each level of A indicate that the stimulus presentations were obtained at a significantly higher rate in the test (C2) phase, as compared to the pretest (C1) phase, for A1 subjects, $F(1, 21) = 8.05$, $p < .05$, for A2 subjects, $F(1, 21) = 8.82$, $p < .05$, and for A3 subjects, $F(1, 21) = 4.73$, $p < .05$. Simple main effects tests for C at each level of B indicate that both paired (B1) stimulus presentations, $F(1, 21) = 30.37$, $p < .05$, and control (B2) stimulus presentations (or the "no stimulus" equivalent), $F(1, 21) = 6.56$, $p < .05$, were

obtained at a significantly higher level in the test (C2) phase as compared to the pretest (C1) phase. Omega squared values calculated for the latter comparisons yield results of 0.109 for C at B1 and 0.021 for C at B2, indicating a relative magnitude for C at B1 of 5.19 times that of C at B2.

Simple, simple main effects tests for B at each level of AC indicate that paired (B1) stimulus presentations were obtained more frequently than the "no stimulus" (B2) control in the test (C2) phase for the paired/nothing (A1) group (B at A1C2), $F(1, 63) = 9.20, p < .05$. Simple, simple main effects tests for C at each level of AB indicate that paired (B1) stimulus presentations were obtained more frequently in the test (C2) phase than in the pretest (C1) phase in the paired/nothing (A1) group (C at A1B1), $F(1, 63) = 16.49, p < .05$, in the paired/random (A2) group (C at A2B1), $F(1, 63) = 11.23, p < .05$, and in the paired/explicitly unpaired (A3) group (C at A3B1), $F(1, 63) = 6.98, p < .05$. Results also indicate that random (B2) stimulus presentations were obtained more frequently in the test (C2) phase than the pretest (C1) phase (C at A2B2), $F(1, 63) = 4.53, p < .05$. Omega squared values of 0.052 for C at A1B1, 0.034 for C at A2B1, and 0.020 for C at A3B1 indicate a relative magnitude for C at A1B1 of 1.53 times that of C at A2B1 and 2.60 times that of C at A3B1, and a relative magnitude for C at A2B1 of 1.70 times that of A3B1. Using an omega squared value of 0.012 for C at A2B2,

results indicate a relative magnitude for C at A2B1 of 2.83 times that of C at A2B2.

Analysis 3

Means and standard deviations for the third analysis (measuring cumulative duration in seconds of lever depression across paired/ nothing, paired/random, and paired/explicitly unpaired groups) are represented in Table A3. Results of a test of the assumption of homogeneity of variance indicate a significant violation, $F_{\max}(12, 7) = 51.76, p < .05$. However, because the analysis of variance has been found to be relatively robust with respect to moderate violations of the assumption of homogeneity (Box, 1953), further analysis of the data was deemed to be permissible. (The results of an ANOVA using a natural log transformation indicate a failure to reject the assumption of homogeneity of variance [$F_{\max}[12, 7] = 15.46, n.s.$] and replicate the findings of the original analysis with the exception of the absence of a B main effect.)

Results of the overall analysis indicate significant main effects for Factor A (Paired/Control Conditions), $F(1, 21) = 6.27, p < .05$, Factor B (Stimulus), $F(1, 21) = 9.45, p < .05$, and Factor C (Pretest/Test), $F(1, 21) = 25.78, p < .05$, and a significant interaction effect for the BC interaction, $F(1, 21) = 10.88, p < .05$.

Simple main effects tests of A at each level of B and A at each level of C indicate significant differences in durations of lever pressing across A at B1, $F(2, 21) =$

Table A3

Means and Standard Deviations of Lever Depression Duration
Measure for Within Subjects Controls

Group	Stimulus		Pretest	Test
A1 (Group A)	Paired	<u>M</u>	44.23	127.52
		<u>SD</u>	31.00	55.18
	No Stimulus	<u>M</u>	41.35	63.72
		<u>SD</u>	20.55	26.20
A2 (Group D)	Paired	<u>M</u>	39.57	80.06
		<u>SD</u>	40.73	41.45
	Random	<u>M</u>	28.73	62.38
		<u>SD</u>	12.54	35.21
A3 (Group E)	Paired	<u>M</u>	13.34	59.17
		<u>SD</u>	7.67	51.35
	Explicitly Unpaired	<u>M</u>	21.79	40.46

SD

21.67

19.96

8.25, $p < .05$, and across A at C2, $F(2, 21) = 5.34$, $p < .05$, indicating that subjects depressed the lever delivering the paired (B1) stimulus for a significantly longer duration in the test (C2) phase. Newman-Keuls follow-up tests indicate that for both A at B1 and A at C2, A1 (paired/nothing) was significantly greater than A2 (paired/random) and A3 (paired/explicitly unpaired) and A2 was significantly greater than A3, that is, paired/nothing subjects depressed the paired (B1) stimulus in the test (C2) phase for significantly greater durations than the paired/random and paired/explicitly unpaired groups and that paired/random subjects, in turn, depressed the lever for significantly longer durations than paired/explicitly unpaired subjects.

Simple main effects tests for B at each level of A yield a significant effect at A1 only, $F(1, 21) = 11.35$, $p < .05$, indicating that paired/nothing (A1) subjects depressed the lever delivering the paired (B1) stimulus for a significantly longer period of time than the "no stimulus" (B2) lever. Simple main effects tests of B at each level of C indicate significance at C2, $F(1, 21) = 19.96$, $p < .05$, confirming that subjects depressed the lever delivering the paired (B1) stimulus significantly more during the test (C2) phase than in the pretest (C1) phase.

Simple main effects tests of C at each level of A1 indicate that the levers remained depressed for

significantly longer durations in the test (C2) phase as compared to the pretest (C1) phase for A1 (paired/nothing) subjects, $F(1, 21) = 14.31$, $p < .05$, for A2 (paired/random) subjects, $F(1, 21) = 7.11$, $p < .05$, and for A3 (paired/explicitly unpaired) subjects, $F(1, 21) = 5.36$, $p < .05$. Simple main effects tests for C at each level of B indicate that both the paired (B1) stimulus lever, $F(1, 21) = 36.51$, $p < .05$, and the control (B2) lever, $F(1, 21) = 7.06$, $p < .05$, were depressed for significantly longer periods of time in the test (C2) phase as compared to the pretest (C1) phase. Omega squared values calculated for the latter comparisons yield results of 0.158 for C at B1, and 0.027 for C at B2, indicating a relative magnitude for C at B1 of 4.57 times that of C at B2.

Simple, simple main effects tests for B at each level of AC indicate that the lever delivering the paired (B1) stimulus was depressed significantly longer than the control (B2) lever for the paired/nothing (A1) group only in the test (C2) phase (B at A1C2), $F(1, 63) = 16.95$, $p < .05$. Simple, simple main effects tests for C at each level of AB indicate that the lever delivering the paired (B1) stimulus was depressed significantly longer in the test (C2) phase than in the pretest (C1) phase in the paired/nothing (A1) group (C at A1B1), $F(1, 63) = 28.64$, $p < .05$, in the paired/random (A2) group (C at A2B1), $F(1, 63) = 6.80$, $p < .05$, and in the paired/explicitly unpaired (A3) group (C at A3B1), $F(1, 63) = 8.73$, $p < .05$. Results

also indicate that the lever delivering the random (B2) stimulus was depressed significantly longer in the test (C2) phase than in the pretest (C1) phase, (C at A2B2), $F(1, 63) = 4.72, p < .05$. Omega squared values of 0.112 for C at A1B1, 0.024 for C at A2B1, and 0.031 for C at A3B1 indicate a relative magnitude for C at A1B1 of 4.67 times that of C at A2B1 and 3.61 times that of C at A3B1, and a relative magnitude for C at A3B1 of 1.29 times that of C at A2B1. Using an omega squared value of 0.015 for C at A2B2, results indicate a relative magnitude for C at A2B1 of 1.60 times that of C at A2B2.

Analysis 4

Means and standard deviations from the fourth analysis (measuring frequency of lever presses across paired/nothing, random/nothing, and explicitly unpaired/nothing groups) are represented in Table 3 (see Results, p. 28). Results of a test of the assumption of homogeneity of variance [$F_{\max} [12, 7] = 13.32, n.s.$], failed to reject the null hypothesis.

Results of the overall analysis indicate a significant main effect for Factor C (Pretest/Test), $F(1, 21) = 10.46, p < .05$, and a significant interaction effect for the AB (Groups x Stimulus) interaction, $F(2, 21) = 5.84, p < .05$, and the ABC (Groups x Stimulus x Pretest/Test) interaction, $F(2, 21) = 4.67, p < .05$.

Simple main effects tests of C at each level of A indicate a significant difference in the level of

responding for C at A1, $F(1, 21) = 9.95, p < .05$. In other words, subjects in the paired/nothing (A1) group responded significantly more in the test (C2) phase as compared to the pretest (C1) phase. Simple main effects tests of C at each level of B indicate a significant increase in responding for the critical comparison stimulus (B1; paired, random, and explicitly unpaired) in the test (C2) phase as compared to the pretest (C1) phase (C at B1), $F(1, 21) = 12.74, p < .05$.

Follow-up analyses of between group differences (follow-up tests of the AB interaction) indicate that differences across groups in levels of responding are found for the critical comparison (B1) stimulus but not the "no stimulus" (B2) lever [$F(2, 21) = 4.14, p < .05$].

Newman-Keuls follow-up tests further indicate that subjects in the paired/nothing (A1) group responded more for the paired (B1) stimulus than did subjects in either comparison group for the control (B2) stimulus (random or unpaired).

Simple main effects tests of B at each level of A indicate a significant difference for B at A1 only, $F(1, 21) = 6.86, p < .05$, that is, subjects in the paired/nothing (A1) group preferred the lever delivering the paired (B1) stimulus as compared to the control (B2) lever.

Simple, simple main effects tests of B at AC and C at AB indicate significant effects for B at A1C2, $F(1, 63) = 13.08, p < .05$, and C at A1B1, $F(1, 63) = 18.74, p < .05$, indicating a preference in the paired/nothing (A1) group

for the paired (B1) stimulus in the test (C2) phase as compared to the pretest (C1) phase and as compared to the "no stimulus" (B2) control in the test (C2) phase.

Analysis 5

Means and standard deviations for the fifth analysis (measuring number of stimulus presentations obtained across paired/nothing, random/nothing, and explicitly unpaired/nothing groups) are represented in Table A4. Results of a test of the assumption of homogeneity of variance [$F_{\max}(12, 7) = 10.62$, n.s.] failed to reject the null hypothesis.

Results of the overall analysis indicate a significant main effect for Factor C (Pretest/Test), $F(1, 21) = 7.37$, $p < .05$ and significant interaction effects for the AB (Groups x Stimulus) interaction, $F(2, 21) = 5.43$, $p < .05$, and the ABC (Groups x Stimulus x Pretest/Test) interaction, $F(2, 21) = 4.24$, $p < .05$.

Simple main effects tests of C at each level of A indicate a significant increase in number of stimulus presentations obtained in the test (C2) phase for paired/nothing (A1) subjects (C at A1), $F(1, 21) = 6.62$, $p < .05$, as compared to the pretest (C1) phase.

Simple main effects tests of C at each level of B indicate an increase in number of presentations of the critical comparison stimulus (B1) obtained in the test (C2) phase (C at B1), $F(1, 21) = 8.85$, $p < .05$, as compared to the pretest (C1) phase.

Table A4

Means and Standard Deviations of Stimulus Presentation

Measure for Between Subjects Controls

Group	Stimulus		Pretest	Test
A1 (Group A)	Paired	<u>M</u>	25.75	46.50
		<u>SD</u>	17.44	17.20
	No Stimulus	<u>M</u>	25.00	31.00
		<u>SD</u>	14.14	8.38
A2 (Group B)	Random	<u>M</u>	21.25	24.25
		<u>SD</u>	12.37	20.13
	No Stimulus	<u>M</u>	28.63	30.13
		<u>SD</u>	15.69	24.67
A3 (Group C)	Explicitly Unpaired	<u>M</u>	17.75	23.75
		<u>SD</u>	7.57	8.68
	No Stimulus	<u>M</u>	21.13	32.75

SD

8.29

17.47

Simple main effects tests of B at each level of A indicate a significant difference for B at A1 only, $F(1, 21) = 5.20$, $p < .05$, that is, subjects in the paired/nothing (A1) group obtained presentations of the paired (B1) stimulus significantly more frequently than its equivalent control (B2).

Simple, simple main effects tests of B at each level of AC and C at each level of AB indicate significant effects for B at A1C2, $F(1, 63) = 7.83$, $p < .05$, and for C at A1B1, $F(1, 63) = 14.03$, $p < .05$ and C at A3B2, $F(1, 63) = 4.40$, $p < .05$. In other words, subjects in the paired/nothing (A1) group obtained presentations of the paired (B1) stimulus significantly more in the test (C2) phase than in the pretest (C1) phase, and in the test phase, significantly more than the "no stimulus" (B2) control. On the other hand, subjects in the explicitly unpaired/nothing (A3) group increased responding for the "no stimulus" (B2) control condition in the test (C2) phase as compared to the pretest (C1) phase.

Analysis 6

Means and standard deviations for the final analysis (measuring cumulative duration in seconds of lever depression across paired/nothing, random/nothing, and explicitly unpaired/nothing groups) are represented in Table A5. Results of a test of the assumption of homogeneity of variance ($F_{\max}(12, 7) = 19.03$, n.s.)

Table A5

Means and Standard Deviations of Lever Depression Duration
Measure for Between Subjects Controls

Group	Stimulus	Pretest	Test
A1 (Group A)	Paired	<u>M</u> 44.23	127.52
		<u>SD</u> 31.00	55.18
	No Stimulus	<u>M</u> 41.35	63.72
		<u>SD</u> 20.55	26.20
A2 (Group B)	Random	<u>M</u> 36.18	41.23
		<u>SD</u> 19.45	36.18
	No Stimulus	<u>M</u> 43.28	50.97
		<u>SD</u> 12.65	46.44
A3 (Group C)	Explicitly Unpaired	<u>M</u> 31.20	66.68
		<u>SD</u> 27.04	35.55
	No Stimulus	<u>M</u> 33.32	64.57

SD

21.64

28.75

failed to reject the null hypothesis.

Results of the overall analysis indicate a significant main effect for Factor C (Pretest/Test), $F(1, 21) = 21.70$, $p < .05$, and significant interaction effects for the AB (Groups x Stimulus) interaction, $F(2, 21) = 5.19$, $p < .05$, the AC (Groups x Pretest/Test) interaction, $F(2, 21) = 4.14$, $p < .05$, the BC (Stimulus x Pretest/Test) interaction, $F(1, 21) = 5.75$, $p < .05$, and the ABC interaction, $F(2, 21) = 5.40$, $p < .05$.

Simple main effects tests for C at each level of A indicate significant differences for C at A1, $F(1, 21) = 20.95$, $p < .05$, and C at A3, $F(1, 21) = 8.43$, $p < .05$, that is, subjects in both the paired/nothing (A1) group and the explicitly unpaired/nothing (A3) group depressed the lever for significantly longer cumulative durations in the test (C2) phase as compared to the pretest (C1) phase.

Simple main effects tests of C at each level of B indicate significant differences for C at B1, $F(1, 21) = 27.16$, $p < .05$, and C at B2, $F(1, 21) = 6.68$, $p < .05$, that is, both levers remained depressed for significantly longer cumulative durations in the test (C2) phase as compared to the pretest (C1) phase. Omega squared values of 0.105 for C at B1 and 0.023 for C at B2 indicate a relative magnitude for C at B1 of 4.57 times that of C at B2.

Simple main effects tests of B at each level of C indicate a significant difference for B at C2 only, $F(1,$

21) = 6.95, $p < .05$, indicating that subjects in the test (C2) phase depressed the lever delivering the critical comparison stimulus (B1) for significantly longer durations than the control (B2) lever.

Simple main effects tests of B at each level of A indicate a significant effect for B at A1 only, $F(1, 21) = 11.80$, $p < .05$, indicating that subjects in the paired/nothing (A1) group depressed the lever delivering the paired (B1) stimulus for significantly longer cumulative durations than the control (B2) lever.

Simple main effects tests of A at each level of B and A at each level of C resulted in significant effects for A at B1, $F(2, 21) = 7.49$, $p < .05$, and A at C2, $F(2, 21) = 6.78$, $p < .05$, confirming that subjects produced longer durations for the critical comparison stimulus (B1) in the test (C2) phase. Newman-Keuls follow-up tests indicate that A1 (paired/nothing) durations were significantly longer than A2 (random/nothing) durations and A3 (explicitly unpaired/nothing) durations.

Simple, simple main effects tests of B at AC indicate a significant effect of B at A1C2 only, $F(1, 63) = 21.92$, $p < .05$, that is, subjects in the paired/nothing (A1) group depressed the lever in the test (C2) phase delivering the paired (B1) stimulus for significantly longer cumulative durations than the control (B2) lever.

Simple, simple main effects tests of C at each level of AB indicate significant effects for C at A1B1, $F(1, 63)$

= 36.54, $p < .05$, for C at A3B1, $F(1, 63) = 6.66$, $p < .05$, and for C at A3B2, $F(1, 63) = 5.20$, $p < .05$. In other words, subjects in the paired/nothing (A1) group and the explicitly unpaired/nothing (A3) group depressed the stimulus (B1) lever for significantly longer cumulative durations in the test (C2) phase than in the pretest (C1) phase. Additionally, subjects in the explicitly unpaired/nothing (A3) group depressed the control (B2) lever for significantly longer cumulative durations in the test (C2) phase than in the pretest (C1) phase. Omega squared values of 0.143 for C at A1B1 and 0.023 for C at A3B1 indicate a relative magnitude for C at A1B1 of 6.22 times that of C at A3B1. An omega squared value of 0.017 for C at A3B2 indicates a relative magnitude for C at A3B1 of 1.35 times that of C at A3B2.

Control Analyses: Counterbalancing

In order to rule out the possible confounding effects of experimentally counterbalanced factors, a number of additional control analyses were performed. Three $3 \times 2 \times 2$ ANOVAs, with repeated measures across the latter factor, were performed across the three groups receiving only one stimulus to determine relative preferences for light and tone stimuli. Factor A was the between groups factor (paired/nothing, random/nothing, explicitly unpaired/nothing), Factor B, the modality factor (light/tone) and Factor C, the stimulus factor (stimulus/no stimulus). Analyses were performed across the three

dependent measures discussed in the main body of this section. An overall preference for the light over the tone (Factor B), $F(1, 18) = 9.58, p < .05$, is consistent across groups for the lever depression duration measure. No other significant effects were obtained. $F_{\max}$ values for the three analyses ($F_{\max} [6, 3] = 11.90, p > .05$, lever press; $F_{\max} [6, 3] = 8.85, p > .05$, stimulus presentations obtained; $F_{\max} [6, 3] = 21.37, p > .05$, lever depression duration) failed to reject the null hypothesis.

Three 2×2 ANOVAs, with repeated measures across the latter factor, were performed for the two groups receiving two stimuli to determine relative preferences for light and tone. Factor A was the between groups factor (paired/random, paired/explicitly unpaired) and Factor B, the modality factor (light/tone). Again, the three dependent measures were those assessed in the main analyses. Results of the tests of the assumption of homogeneity of variance failed to reject the null hypothesis for two dependent measures ($F_{\max} [4, 7] = 4.40, p > .05$, lever press; $F_{\max} [4, 7] = 5.06, p > .05$, stimulus presentations obtained) but violated the assumption of homogeneity for one dependent measure ($F_{\max} [4, 7] = 32.03, p < .01$, lever press duration). However, because the $F_{\max}$ is considered robust with respect to moderate violations of the assumption of homogeneity (Box, 1953), the analysis of the results for the latter dependent measure proceeded. A significant main effect for Factor B (light/tone) for both

the lever press measure, $F(1, 14) = 9.85, p < .05$, and the measure of stimulus presentations obtained, $F(1, 14) = 7.47, p < .05$, again indicates a stronger preference for the light.

Three 5×2 ANOVAs, with repeated measures across the second factor, were performed to determine right/left preferences across groups. Factor A was the between groups factor across the five training conditions. Factor B was the right/left preference factor. Once again, three analyses were performed across the three dependent measures used in the main analyses. $F_{\max}$ results indicate a failure to reject the null hypothesis for two dependent measures ($F_{\max}[10, 7] = 10.08, p > .05$, lever press; $F_{\max}[10, 7] = 8.82, p > .05$, stimulus presentations obtained). For the third dependent measure, the ANOVA was considered robust (Box, 1953) relative to a moderate violation of the assumption of homogeneity of variance, $F_{\max}(10, 7) = 30.47, p < .01$. For all three analyses, a significant main effect of Factor B was found, ($F[1, 35] = 4.33, p < .05$, lever press; $F[1, 35] = 13.23, p < .05$, stimulus presentations obtained; $F[1, 35] = 19.65, p < .05$, lever depression duration) indicating a significant right preference across groups.

Control Analyses: Confounding Variables

In order to rule out the influence of additional potentially confounding variables, seven one way ANOVAs were performed across groups to assess between group

185

differences relative to age, percent of training pairings on Days 2, 3, and 4 of training, weight, number of pretraining days to criterion, length of training sessions, number of pretraining lever presses to criterion (in the 30 min session in which criterion was met), and length of the experimental procedure.

Results of tests of the assumption of homogeneity of variance failed to reject the null hypothesis for five of the ANOVAs (age, $F_{\max} [5, 7] = 6.53$, n.s.; weight, $F_{\max} [5, 7] = 5.83$, n.s.; number of pretraining days to criterion, $F_{\max} [5, 7] = 4.04$, n.s.; length of training sessions, $F_{\max} [5, 7] = 8.87$, n.s.; number of pretraining lever presses to criterion, $F_{\max} [5, 7] = 4.70$, n.s.). The $F_{\max}$ result for the analysis of the percent of training pairings ($F_{\max} [5, 7] = 43.16$, $p < .01$) reflects a moderate violation of the assumption of homogeneity of variance for which the overall analysis should be relatively robust (Box, 1953). The $F_{\max}$ result for the analysis of length of experimental procedure ($F_{\max} [5, 7] = 184.49$, $p < .01$) represents a serious violation of the assumption of homogeneity of variance. For this reason, data from the last analysis were transformed using a natural log transformation to reduce the level of error variance. The resulting $F_{\max}$ value ($F_{\max} [5, 7] = 16.77$, $p < .01$) reflects a negligible violation of the assumption of homogeneity of variance.

Of the seven analyses, three indicate significant

differences. Results of the analysis of differences in age ($F[4, 35] = 4.23, p < .05$) indicate that the paired/nothing group was older at the outset of the experiment than the paired/explicitly unpaired, random/nothing, and explicitly unpaired/nothing groups. Results of the analysis of percent of training pairings ($F[4, 35] = 3.58, p < .05$) indicate that paired/random subjects received significantly more pairings of the paired stimulus with ICS on Days 2, 3, and 4 of training, as compared to paired/explicitly unpaired subjects. While the results of the analysis of between group differences in weight are significant ($F[4, 35] = 2.68, p < .05$), Newman-Keuls follow-up tests fail to yield any pairwise differences. It is likely that the significant difference is accounted for by the combination of the paired/nothing (A1) and paired/random (A2) groups (mean of A = 569.69; mean of B = 568.81) as compared to the combination of the paired/explicitly unpaired (A3) and explicitly unpaired/nothing (A5) groups (mean of C = 495.25; mean of E = 484.25).

Because of the possibility that overlaps of the random stimulus with ICS might lead to sufficient pairing for learning to occur in the paired/random and the random/nothing groups, the number of total (forward, backward, and simultaneous) overlaps and the number of forward overlaps were measured for each subject. For the paired/random group, the mean number of total overlaps was 10.75 and the mean number of forward overlaps was 4.5.

187

Of the 400 random stimuli presented, therefore, 2.7% overlapped with ICS, with only 1.1% occurring in the optimal forward pairing configuration, as compared to 64% of the paired stimuli occurring in the optimal configuration. While the paired stimulus preceded and overlapped with ICS 100% of the times that it was presented, the random stimulus overlapped for 4.2% of the ICS presentations and preceded and overlapped with ICS for 1.8% of the presentations of the latter.

For the random/nothing group, the mean number of total overlaps was 11.25 and the mean number of forward overlaps was 4.75. Of the 400 random stimuli presented, 2.8% overlapped with ICS, with only 1.2% occurring in the optimal forward pairing configuration. The random stimulus overlapped for 4.6% of the ICS presentations and preceded and overlapped with ICS for 1.9% of the presentations of the latter.

Appendix B

Table B1

Analysis of Variance Summary Table for
Lever Presses for Within Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
<u>Between Ss</u>				
Group (A)	6500.396	2	3250.198	5.367*
Error(s w grps)	12718.062	21	650.622	
<u>Within Ss</u>				
Stimulus (B)	1120.667	1	1120.667	6.129*
A x B	647.146	2	323.573	1.770
Error(B x s w grps)	3839.687	21	182.842	
Pretest/Test (C)	6176.042	1	6176.042	24.433****
A x C	116.146	2	58.073	0.230
Error(C x s w grps)	5308.313	21	252.777	
B x C	888.167	1	888.167	8.660**
A x B x C	432.646	2	216.323	2.109
Error(BC x s w grps)	2153.687	21	102.557	

* $p < .05$ ** $p < .01$ **** $p < .001$

Appendix B

Figure Caption

Figure B1. Number of lever presses for
B (Stimulus) at C (Pretest/Test) for
paired/nothing subjects.

Appendix B

Figure B1

Appendix B

Figure Caption

Figure B2. Number of lever presses for
B (Stimulus) at C (Pretest/Test) for
paired/random subjects.

Appendix B

Figure B2

Appendix B

Figure Caption

Figure B3. Number of lever presses for
B (Stimulus) at C (Pretest/Test) for
paired/explicitly unpaired subjects.

Appendix B

Figure B3

Appendix B

Figure B4. Number of lever presses for
B (Stimulus) at C (Pretest/Test) for
within subjects controls.

Appendix B

Figure B4

Appendix B

Table B2

BC (Stimulus x Pretest/Test)

Summary Table of

Lever Presses for

Within Subjects Controls

	C1	C2	Total
B1	507	1038	1545
B2	489	728	1217
Total	996	1766	2762

Appendix B

Table B3

Simple Main Effects Tests of
Lever Presses for
B (Stimulus) at C (Pretest/Test) for
Within Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
B at C1	6.75	1	6.75	0.03
B at C2	2002.09	1	2002.09	8.10**
Error (pooled)		21	244.79	

** $p < .01$

Appendix B

Table B4

Simple Main Effects Tests ofLever Presses forC (Pretest/Test) at B (Stimulus) forWithin Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
C at B1	5874.19	1	5874.19	33.06****
C at B2	1190.02	1	1190.02	6.70*
Error (pooled)		21	177.68	

* $p < .05$ **** $p < .001$

Appendix B

Table B5

AB (Group x Stimulus)Summary Table ofLever Presses forWithin Subjects Controls

	B1	B2	Total
A1	729	514	1243
A2	511	410	921
A3	305	293	598
Total	1545	1217	2762

Appendix B

Table B6

Simple Main Effects Tests of
Lever Presses for
A (Group) at B (Stimulus) for
Within Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
A at B1	5619.50	2	2809.75	7.13***
A at B2	1528.04	2	764.02	1.94
Error (pooled)		21	394.23	

*** $p < .005$

Appendix B

Table B7

Newman-Keuls Analysis of
Lever Presses for
Between Group (A) Differences at the
Paired (B1) Stimulus Level for
Within Subjects Controls

	A3	A2	A1
	Paired/ Nothing	Paired/ Random	Paired/ Expl. Unp.
	305	511	729
A3		206*	424**
A2			218*

* $p < .05$

** $p < .01$

Appendix B

Table B8

Simple Main Effects Tests of
Lever Presses for
B (Stimulus) at A (Group) for
Within Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
B at A1	1444.53	1	1444.53	7.90 *
B at A2	318.78	1	318.78	1.74
B at A3	4.50	1	4.50	0.02
Error (B x s w grps)	3839.69	21	182.84	

* $p < .05$

Appendix B

Table B9

AC (Group x Pretest/Test)Summary Table ofLever Presses forWithin Subjects Controls

	C1	C2	Total
A1	474	769	1243
A2	328	593	921
A3	194	404	598
Total	996	1766	2762

Appendix B

Table B10

Simple Main Effects Tests of
Lever Presses for
A (Group) at C (Pretest/Test) for
Within Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
A at C1	2451.50	2	1225.75	2.86
A at C2	4165.04	2	2082.52	4.85*
Error (pooled)		21	429.20	

* $p < .05$

Appendix B

Table B11

Newman-Keuls Analysis of
Lever Presses for
Between Group (A) Differences in the
Test (C2) Phase for Within Subjects Controls

	A3	A2	A1
	Paired/ Nothing 404	Paired/ Random 593	Paired/ Expl. Unp. 769
A3		189*	365**
A2			176*

* $p < .05$

** $p < .01$

Appendix B

Table B12

Simple Main Effects Tests of
Lever Presses for
C (Pretest/Test) at A (Group) for
Within Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
C at A1	2719.53	1	2719.53	10.76***
C at A2	2183.28	1	2183.28	8.64**
C at A3	1378.13	1	1378.13	5.45*
Error (C x s w grps)	5308.31	21	252.78	

* $p < .05$

** $p < .01$

*** $p < .005$

Appendix B

Table B13

ABC (Group x Stimulus x Pretest/Test)Summary Table ofLever Presses forWithin Subjects Controls

		C1	C2	Total
A1	B1	243	486	729
	B2	231	283	514
A2	B1	172	339	511
	B2	156	254	410
A3	B1	92	213	305
	B2	102	191	293
Total		996	1766	2762

Appendix B

Table B14

Simple, Simple Main Effects Tests of
Lever Presses for
B (Stimulus) at AC for
Within Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
B at A1C1	9.00	1	9.00	0.05
B at A1C2	2575.56	1	2575.56	14.38***
B at A2C1	16.00	1	16.00	0.09
B at A2C2	451.56	1	451.56	2.51
B at A3C1	6.00	1	6.00	0.03
B at A3C2	30.25	1	30.25	0.17
Error (pooled)		63	179.39	

*** $p < .005$

Appendix B

Table B15

Simple, Simple Main Effects Tests of
Lever Presses for
C (Pretest/Test) at AB for
Within Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
C at A1B1	3690.56	1	3690.56	20.57****
C at A1B2	169.00	1	169.00	0.94
C at A2B1	1743.06	1	1743.06	9.72**
C at A2B2	600.25	1	600.25	3.35
C at A3B1	915.06	1	915.06	5.10*
C at A3B2	495.06	1	495.06	2.76
Error (pooled)		63	179.39	

* $p < .05$

** $p < .01$

**** $p < .001$

Appendix B

Table B16

Simple Interaction Effects Tests ofLever Presses forBC (Stimulus x Pretest/Test) at A (Group) forWithin Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
<hr/>				
BC at A1	1140.03	1	1140.03	11.12***
BC at A2	148.78	1	148.78	1.45
BC at A3	32.00	1	32.00	0.31
Error (BC x s w grps)	2153.69	21	102.56	

*** $p < .005$

Appendix C

Table C1

Analysis of Variance Summary Table for
Stimulus Presentations for Within Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
<u>Between Ss</u>				
Group (A)	4102.333	2	2051.167	5.608*
Error(s w grps)	7680.625	21	365.744	
<u>Within Ss</u>				
Stimulus (B)	726.000	1	726.000	8.899**
A x B	150.750	2	75.375	0.924
Error(B x s w grps)	1713.250	21	81.583	
Pretest/Test (C)	3775.042	1	3775.042	21.223****
A x C	64.583	2	32.292	0.182
Error(C x s w grps)	3735.375	21	177.875	
B x C	504.167	1	504.167	9.359**
A x B x C	93.583	2	46.792	0.869
Error(BC x s w grps)	1131.250	21	53.869	

* $p < .05$ ** $p < .01$ **** $p < .001$

Appendix C

Figure Caption

Figure C1. Number of stimulus presentations for
B (Stimulus) at C (Pretest/Test) for
paired/nothing subjects.

Appendix C

Figure C1

Appendix C

Figure Caption

Figure C2. Number of stimulus presentations for
B (Stimulus) at C (Pretest/Test) for
paired/random subjects.

Appendix C

Figure C2

Appendix C

Figure Caption

Figure C3. Number of stimulus presentations for
B (Stimulus) at C (Pretest/Test) for
paired/explicitly unpaired subjects.

Appendix C

Figure C3

Appendix C

Figure Caption

Figure C4. Number of stimulus presentations for
B (Stimulus) at C (Pretest/Test) for
within subjects controls.

Appendix C

Figure C4

Appendix C

Table C2

BC (Stimulus x Pretest/Test)Summary Table ofStimulus Presentations forWithin Subjects Controls

	C1	C2	Total
B1	454	865	1319
B2	432	623	1055
Total	886	1488	2374

Appendix C

Table C3

Simple Main Effects Tests of
Stimulus Presentations for
B (Stimulus) at C (Pretest/Test) for
Within Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
B at C1	10.09	1	10.09	0.15
B at C2	1220.08	1	1220.08	18.01****
Error (pooled)		21	67.73	

**** $p < .001$

Appendix C

Table C4

Simple Main Effects Tests of
Stimulus Presentations for
C (Pretest/Test) at B (Stimulus) for
Within Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
C at B1	3519.19	1	3519.19	30.37****
C at B2	760.02	1	760.02	6.56*
Error (pooled)		21	115.87	

* $p < .05$

**** $p < .001$

Appendix C

Table C5

AB (Group x Stimulus)Summary Table ofStimulus Presentations forWithin Subjects Controls

	B1	B2	Total
A1	578	448	1026
A2	465	365	830
A3	276	242	518
Total	1319	1055	2374

Appendix C

Table C6

Simple Main Effects Tests of
Stimulus Presentations for
A (Group) at B (Stimulus) for
Within Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
A at B1	2910.29	2	1455.15	6.51**
A at B2	1342.79	2	671.40	3.00
Error (pooled)		21	223.66	

** $p < .01$

Appendix C

Table C7

Newman-Keuls Analysis of
Stimulus Presentations for
Between Group (A) Differences at the
Paired (B1) Stimulus Level for
Within Subjects Controls

	A3	A2	A1
	Paired/ Nothing	Paired/ Random	Paired/ Expl. Unp.
	276	465	578
A3		189**	302**
A2			113

** $p < .01$

Appendix C

Table C8

Simple Main Effects Tests of
Stimulus Presentations for
B (Stimulus) at A (Group) for
Within Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
B at A1	528.13	1	528.13	6.47*
B at A2	312.50	1	312.50	3.83
B at A3	36.13	1	36.13	0.44
Error(B x s w grps)	1713.25	21	81.58	

* $p < .05$

Appendix C

Table C9

AC (Group x Pretest/Test)Summary Table ofStimulus Presentations forWithin Subjects Controls

	C1	C2	Total
A1	406	620	1026
A2	303	527	830
A3	177	341	518
Total	886	1488	2374

Appendix C

Table C10

Simple Main Effects Tests of
Stimulus Presentations for
A (Group) at C (Pretest/Test) for
Within Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
A at C ¹	1644.29	2	822.15	3.02
A at C2	2522.63	2	1261.31	4.64*
Error (pooled)		21	271.81	

* $p < .05$

Appendix C

Table C11

Newman-Keuls Analysis of
Stimulus Presentations for
Between Group (A) Differences in the
Test (C2) Phase for Within Subjects Controls

	A3	A2	A1
	Paired/ Nothing	Paired/ Random	Paired/ Expl. Unp.
	341	527	620
A3		186*	279**
A2			93

* $p < .05$

** $p < .01$

Appendix C

Table C12

Simple Main Effects Tests of
Stimulus Presentations for
C (Pretest/Test) at A (Group) for
Within Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
C at A1	1431.13	1	1431.13	8.05*
C at A2	1568.00	1	1568.00	8.82**
C at A3	840.50	1	840.50	4.73*
Error(C x s w grps)	3735.38	21	177.88	

* $p < .05$

** $p < .01$

Appendix C

Table C13

ABC (Group x Stimulus x Pretest/Test)Summary Table ofStimulus Presentations forWithin Subjects Controls

		C1	C2	Total
A1	B1	206	372	578
	B2	200	248	448
A2	B1	164	301	465
	B2	139	226	365
A3	B1	84	192	276
	B2	93	149	242
Total		886	1488	2374

Appendix C

Table C14

Simple, Simple Main Effects Tests of
Stimulus Presentations for
B (Stimulus) at AC for
Within Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
B at A1C1	2.25	1	2.25	0.02
B at A1C2	961.00	1	961.00	9.20**
B at A2C1	39.06	1	39.06	0.37
B at A2C2	351.56	1	351.56	3.37
B at A3C1	5.06	1	5.06	0.05
B at A3C2	115.56	1	115.56	1.11
Error (pooled)		63	104.44	

** $p < .01$

Appendix C

Table C15

Simple, Simple Main Effects Tests of
Stimulus Presentations for
C (Pretest/Test) at AB for
Within Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
C at A1B1	1722.25	1	1722.25	16.49****
C at A1B2	144.00	1	144.00	1.42
C at A2B1	1173.06	1	1173.06	11.23***
C at A2B2	473.06	1	473.06	4.53*
C at A3B1	729.00	1	729.00	6.98*
C at A3B2	196.00	1	196.00	1.88
Error (pooled)		63	104.44	

* $p < .05$

*** $p < .005$

**** $p < .001$

Appendix C

Table C16

Simple Interaction Effects Tests of
Stimulus Presentations for
BC (Stimulus x Pretest/Test) at A (Group) for
Within Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
BC at A1	435.13	1	435.13	8.08*
BC at A2	78.13	1	78.13	1.45
BC at A3	84.50	1	84.50	1.57
Error(BC x s w grps)	1131.25	21	53.87	

* $p < .05$

Appendix D

Table D1

Analysis of Variance Summary Table for
Lever Depression Duration for
Within Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
<u>Between Ss</u>				
Group (A)	20160.122	2	10080.061	6.271**
Error(s w grps)	33752.900	21	1607.281	
<u>Within Ss</u>				
Stimulus (B)	7389.779	1	7389.779	9.449***
A x B	3332.534	2	1666.267	2.131
Error(B x s w grps)	16423.242	21	782.059	
Pretest/Test (C)	39732.564	1	39732.564	25.784****
A x C	1865.292	2	932.646	0.605
Error(C x s w grps)	32359.980	21	1540.951	
B x C	6026.962	1	6026.962	10.875***
A x B x C	2980.016	2	1490.008	2.689
Error(BC x s w grps)	11637.900	21	554.186	

** $p < .01$ *** $p < .005$ **** $p < .001$

Appendix D

Figure Caption

Figure D1. Lever depression duration for
B (Stimulus) at C (Pretest/Test) for
paired/nothing subjects.

Appendix D

Figure D1

Appendix D

Figure Caption

Figure D2. Lever depression duration for
B (Stimulus) at C (Pretest/Test) for
paired/random subjects.

Appendix D

Figure D2

Appendix D

Figure Caption

Figure D3. Lever depression duration for
B (Stimulus) at C (Pretest/Test) for
paired/explicitly unpaired subjects.

Appendix D

Figure D3

Appendix D

Figure Caption

Figure D4. Lever depression duration for
B (Stimulus) at C (Pretest/Test) for
within subjects controls.

Appendix D

Figure D4

Appendix D

Table D2

BC (Stimulus x Pretest/Test)Summary Table ofLever Depression Duration forWithin Subjects Controls

	C1	C2	Total
B1	778	2133	2911
B2	737	1333	2070
Total	1515	3466	4981

Appendix D

Table D3

Simple Main Effects Tests of
Lever Depression Duration for
B (Stimulus) at C (Pretest/Test) for
Within Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
B at C1	35.02	1	35.02	0.05
B at C2	13333.34	1	13333.34	19.96****
Error (pooled)		21	668.12	

**** $p < .001$

Appendix D

Table D4

Simple Main Effects Tests of
Lever Depression Duration for
C (Pretest/Test) at B (Stimulus) for
Within Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
C at B1	38250.52	1	38250.52	36.51****
C at B2	7400.33	1	7400.33	7.06*
Error (pooled)		21	1047.59	

* $p < .05$

**** $p < .001$

Appendix D

Table D5

AB (Group x Stimulus)Summary Table ofLever Depression Duration forWithin Subjects Controls

	B1	B2	Total
A 1	1374	841	2215
A2	957	729	1686
A3	580	500	1080
Total	2911	2070	4981

Appendix D

Table D6

Simple Main Effects Tests of
Lever Depression Duration for
A (Group) at B (Stimulus) for
Within Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
A at B1	19717.79	2	9858.90	8.25***
A at B2	3776.34	2	1888.19	1.58
Error (pooled)		21	1194.67	

*** $p < .005$

Appendix D

Table D7

Newman-Keuls Analysis of
Lever Depression Duration for
Between Group (A) Differences at the
Paired (B1) Stimulus Level for
Within Subjects Controls

	A3	A2	A1
	Paired/ Nothing	Paired/ Random	Paired/ Expl. Unp.
	580	957	1374
A3		377*	794**
A2			417**

* $p < .05$

** $p < .01$

Appendix D

Table D8

Simple Main Effects Tests of
Lever Depression Duration for
B (Stimulus) at A (Group) for
Within Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
B at A1	8877.78	1	8877.78	11.35***
B at A2	1624.50	1	1624.50	2.08
B at A3	200.00	1	200.00	0.26
Error (B x s w grps)	16423.24	21	782.06	

*** $p < .005$

Appendix D

Table D9

AC (Group x Pretest/Test)

Summary Table of

Lever Depression Duration for

Within Subjects Controls

	C1	C2	Total
A1	685	1530	2215
A2	547	1139	1686
A3	283	797	1080
Total	1515	3466	4981

Appendix D

Table D10

Simple Main Effects Tests of
Lever Depression Duration for
A (Group) at C (Pretest/Test) for
Within Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
A at C1	5215.50	2	2607.75	1.66
A at C2	16815.30	2	8407.65	5.34*
Error (pooled)		21	1574.12	

* $p < .05$

Appendix D

Table D11

Newman-Keuls Analysis of
Lever Depression Duration for
Between Group (A) Differences in the
Test (C2) Phase for Within Subjects Controls

	A3	A2	A1
	Paired/ Nothing	Paired/ Random	Paired/ Expl. Unp.
	797	1139	1530
A3		342*	733**
A2			391*

* $p < .05$

** $p < .01$

Appendix D

Table D12

Simple Main Effects Tests of
Lever Depression Duration for
C (Pretest/Test) at A (Group) for
Within Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
C at A1	22056.97	1	22056.97	14.31***
C at A2	10952.00	1	10952.00	7.11*
C at A3	8256.13	1	8256.13	5.36*
Error(C x s w grps)	32359.98	21	1540.95	

* $p < .05$

*** $p < .005$

Appendix D

Table D13

ABC (Group x Stimulus x Pretest/Test)

Summary Table of

Lever Depression Duration for

Within Subjects Controls

		C1	C2	Total
A1	B1	354	1020	1374
	B2	331	510	841
A2	B1	317	640	957
	B2	230	499	729
A3	B1	107	473	580
	B2	176	324	500
Total		1515	3466	4981

Appendix D

Table D14

Simple, Simple Main Effects Tests of
Lever Depression Duration for
B (Stimulus) at AC for
Within Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
B at A1B1	33.06	1	33.06	0.03
B at A1B2	16256.25	1	16256.25	16.95****
B at A2B1	473.06	1	473.06	0.49
B at A2B2	1242.56	1	1242.56	1.30
B at A3B1	297.56	1	297.56	0.31
B at A3B2	1387.56	1	1387.56	1.45
Error (pooled)		63	959.07	

**** $p < .001$

Appendix D

Table D15

Simple, Simple Main Effects Tests of
Lever Depression Duration for
C (Pretest/Test) at AB for
Within Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
C at A1B1	27472.25	1	27472.25	28.64****
C at A1B2	2002.56	1	2002.56	2.09
C at A2B1	6520.56	1	6520.56	6.80*
C at A2B2	4522.56	1	4522.56	4.72*
C at A3B1	8372.25	1	8372.25	8.73**
C at A3B2	1369.00	1	1369.00	1.43
Error (pooled)		63	959.07	

* $p < .05$

** $p < .01$

**** $p < .001$

Appendix D

Table D16

Simple Interaction Effects Tests of
Lever Depression Duration for
BC (Stimulus x Pretest/Test) at A (Group) for
Within Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
BC at A1	7419.45	1	7419.45	13.39***
BC at A2	93.67	1	93.67	0.17
BC at A2	1493.86	1	1493.86	2.69
Error(BC x s w grps)	11637.90	21	554.19	

*** $p < .005$

Appendix D

Table D17

Analysis of Variance Summary Table for
Natural Log Transformation of
Lever Depression Duration for
Within Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
<u>Between Ss</u>				
Group (A)	14.096	2	7.048	4.724*
Error(s w grps)	31.342	21	1.492	
<u>Within Ss</u>				
Stimulus (B)	0.003	1	0.003	0.005
A x B	1.084	2	0.542	0.880
Error(B x s w grps)	12.928	21	0.616	
Pretest/Test (C)	29.004	1	29.004	26.201****
A x C	0.642	2	0.321	0.290
Error(C x s w grps)	23.246	21	1.107	
B x C	3.163	1	3.163	7.305*
A x B x C	0.046	2	0.023	0.053
Error(BC x s w grps)	9.091	21	0.433	

* $p < .05$

**** $p < .001$

Appendix D

Table D18

Means and Standard Deviations of
Natural Log Transformation of
Lever Depression Duration for
Within Subjects Controls

Group	Stimulus	Pretest	Test
A1 (Group A)	<u>M</u>	3.45	4.75
	Paired		
	<u>SD</u>	1.01	0.51
	No Stimulus		
A2 (Group D)	<u>M</u>	3.55	4.07
	<u>SD</u>	0.73	0.44
	Paired		
	<u>M</u>	2.89	4.26
	<u>SD</u>	1.70	0.53
	Random		
	<u>M</u>	3.21	3.98
	<u>SD</u>	0.68	0.60
Paired	<u>M</u>	2.05	3.76
	<u>SD</u>	1.73	0.85

A3 (Group E)

	<u>M</u>	2.68	3.59
Explicitly Unpaired			
	<u>SD</u>	0.98	0.49

Appendix E

Table E1

Analysis of Variance Summary Table for Lever Presses for Between Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
<u>Between Ss</u>				
Group (A)	2394.146	2	1197.073	1.300
Error(s w grps)	19337.812	21	920.848	
<u>Within Ss</u>				
Stimulus (B)	16.667	1	16.667	0.079
A x B	2457.021	2	1228.510	5.836**
Error(B x s w grps)	4420.313	21	210.491	
Pretest/Test (C)	2860.167	1	2860.167	10.463***
A x C	838.146	2	419.073	1.533
Error(C x s w grps)	5740.688	21	273.366	
B x C	260.042	1	260.042	2.429
A x B x C	999.521	2	499.760	4.668*
Error(BC x s w grps)	2248.437	21	107.068	

* $p < .05$

** $p < .01$

*** $p < .005$

Appendix E*

Figure Caption

Figure E1. Number of lever presses for
B (Stimulus) at C (Pretest/Test) for
paired/nothing subjects.

*same as B1

Appendix E

Figure E1

Appendix E

Figure Caption

Figure E2. Number of lever presses for
B (Stimulus) at C (Pretest/Test) for
random/nothing subjects.

Appendix E

Figure E2

Appendix E

Figure Caption

Figure E3. Number of lever presses for
B (Stimulus) at C (Pretest/Test) for
explicitly unpaired/nothing subjects.

Appendix E

Figure E3

Appendix E

Figure Caption

Figure E4. Number of lever presses for
B (Stimulus) at C (Pretest/Test) for
between subjects controls.

Appendix E

Figure E4

Appendix E

Table E2

BC (Stimulus x Pretest/Test)

Summary Table of

Lever Presses for

Between Subjects Controls

	C1	C2	Total
B1	590	931	1521
B2	689	872	1561
Total	1279	1803	3082

Appendix E

Table E3

Simple Main Effects Tests of

Lever Presses for

B (Stimulus) at C (Pretest/Test) for

Between Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
B at C1	204.19	1	204.19	1.29
B at C2	72.52	1	72.52	0.46
Error (pooled)		21	158.79	

Appendix E

Table E4

Simple Main Effects Tests of
Lever Presses for
C (Pretest/Test) at B (Stimulus) for
Between Subjects Controls

<u>Source</u>	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
C at B1	2422.52	1	2422.52	12.74***
C at B2	679.69	1	679.69	3.67
Error (pooled)		21	190.22	

*** $p < .005$

Appendix E

Table E5

AB (Group x Stimulus)

Summary Table of

Lever Presses for

Between Subjects Controls

	B1	B2	Total
A1	729	514	1243
A2	418	560	978
A3	374	487	861
Total	1521	1561	3082

Appendix E

Table E6

Simple Main Effects Tests ofLever Presses forA (Group) at B (Stimulus) forBetween Subjects Controls

<u>Source</u>	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
A at B1	4680.87	2	2340.44	4.14*
A at B2	170.29	2	85.15	0.15
Error (pooled)		21	565.67	

*** $p < .005$

Appendix E

Table E7

Newman-Keuls Analysis of
Lever Presses for
Between Group (A) Differences at the
Paired (B1) Stimulus Level for
Between Subjects Controls

	A3	A2	A1
	Expl. Unp./	Random/	Paired/
	Nothing	Nothing	Nothing
	374	418	729
A3		44	399**
A2			311**

** $p < .01$

Appendix E

Table E8

Simple Main Effects Tests of
Lever Presses for
B (Stimulus) at A (Group) for
Between Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
B at A1	1444.53	1	1444.53	6.86*
B at A2	630.13	1	630.13	2.99
B at A3	399.03	1	399.03	1.90
Error (B x s w grps)	4420.31	21	210.49	

* $p < .05$

Appendix E

Table E9

AC (Group x Pretest/Test)Summary Table ofLever Presses forBetween Subjects Controls

	C1	C2	Total
A1	474	769	1243
A2	457	521	978
A3	348	513	861
Total	1279	1803	3082

Appendix E

Table E10

Simple Main Effects Tests of
Lever Presses for
A (Group) at C (Pretest/Test) for
Between Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
A at C1	584.29	2	292.15	0.49
A at C2	2648.00	2	1324.00	2.22
Error (pooled)		21	597.11	

Appendix E

Table E11

Simple Main Effects Tests of
Lever Presses for
C (Pretest/Test) at A (Group) for
Between Subjects Controls

<u>Source</u>	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
C at A1	2719.53	1	2719.53	9.95***
C at A2	128.00	1	128.00	0.47
C at A3	850.78	1	850.78	3.11
Error(C x s w grps)	5740.69	21	273.37	

*** $p < .005$

Appendix E

Table E12

ABC (Group x Stimulus x Pretest/Test)Summary Table ofLever Presses forBetween Subjects Controls

		C1	C2	Total
A1	B1	243	486	729
	B2	231	283	514
A2	B1	187	231	418
	B2	270	290	560
A3	B1	160	214	374
	B2	188	299	487
Total		1279	1803	3082

Appendix E

Table E13

Simple, Simple Main Effects Tests of
Lever Presses for
B (Stimulus) at AC for
Between Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
B at A1C1	9.00	1	9.00	0.05
B at A1C2	2575.56	1	2575.56	13.08***
B at A2C1	430.56	1	430.56	2.19
B at A2C2	217.56	1	217.56	1.10
B at A3C1	49.00	1	49.00	0.25
B at A3C2	451.56	1	451.56	2.29
Error (pooled)		63	196.98	

*** $p < .005$

Appendix E

Table E14

Simple, Simple Main Effects Tests of
Lever Presses for
C (Pretest/Test) at AB for
Between Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
C at A1B1	3690.56	1	3690.56	18.74****
C at A1B2	169.00	1	169.00	0.10
C at A2B1	121.00	1	121.00	0.61
C at A2B2	25.00	1	25.00	0.13
C at A3B1	182.25	1	182.25	0.93
C at A3B2	770.06	1	770.06	3.91
Error (pooled)		63	196.98	

**** $p < .001$

Appendix E

Table E15

Simple Interaction Effects Tests ofLever Presses forBC (Stimulus x Pretest/Test) at A (Group) forBetween Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
BC at A1	1140.03	1	1140.03	10.65***
BC at A2	18.00	1	18.00	0.17
BC at A3	101.53	1	101.53	0.95
Error (BC x s w grps)	2248.44	21	107.07	

*** $p < .005$

Appendix F

Table F1

Analysis of Variance Summary Table for
Stimulus Presentations for Between Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
<u>Between Ss</u>				
Group (A)	1157.021	2	578.510	1.030
Error(s w grps)	11794.219	21	561.629	
<u>Within Ss</u>				
Stimulus (B)	58.594	1	58.594	0.576
A x B	1126.938	2	563.469	5.543*
Error(B x s w grps)	2134.719	21	101.653	
Pretest/Test (C)	1592.510	1	1592.510	7.366*
A x C	500.396	2	250.198	1.157
Error(C x s w grps)	4540.344	21	216.207	
B x C	75.260	1	75.260	1.492
A x B x C	427.646	2	213.823	4.239*
Error(BC x s w grps)	1059.344	21	50.445	

* $p < .05$

Appendix F*

Figure Caption

Figure F1. Number of stimulus presentations for
B (Stimulus) at C (Pretest/Test) for
paired/nothing subjects.

*same as C1

Appendix F

Figure F'

Appendix F

Figure Caption

Figure F2. Number of stimulus presentations for
B (Stimulus) at C (Pretest/Test) for
random/nothing subjects.

Appendix F

Figure F2

Appendix F

Figure Caption

Figure F3. Number of stimulus presentations for
B (Stimulus) at C (Pretest/Test) for
explicitly unpaired/nothing subjects.

Appendix F

Figure F3

Appendix F

Figure Caption

Figure F4. Number of stimulus presentations for
B (Stimulus) at C (Pretest/Test) for
between subjects controls.

Appendix F

Figure F4

Appendix F

Table F2

BC (Stimulus x Pretest/Test)Summary Table ofStimulus Presentations forBetween Subjects Controls

	C1	C2	Total
B1	518	756	1274
B2	598	751	1349
Total	1116	1507	2623

Appendix F

Table F3

Simple Main Effects Tests of
Stimulus Presentations for
B (Stimulus) at C (Pretest/Test) for
Between Subjects Controls

<u>Source</u>	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
B at C1	133.33	1	133.33	1.75
B at C2	0.52	1	0.52	0.01
Error (pooled)		21	76.05	

Appendix F

Table F4

Simple Main Effects Tests of
Stimulus Presentations for
C (Pretest/Test) at E (Stimulus) for
Between Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
C at B1	1180.09	1	1180.09	8.85**
C at B2	487.69	1	487.69	3.66
Error (pooled)		21	133.33	

** $p < .01$

Appendix F

Table F5

AB (Group x Stimulus)Summary Table ofStimulus Presentations forBetween Subjects Controls

	B1	B2	Total
A1	578	448	1026
A2	364	470	834
A3	332	431	763
Total	1274	1349	2623

Appendix F

Table F6

Simple Main Effects Tests of
Stimulus Presentations for
A (Group) at B (Stimulus) for
Between Subjects Controls

<u>Source</u>	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
A at B1	2236.17	2	1118.08	3.37
A at B2	47.79	2	23.90	0.07
Error (pooled)		21	331.64	

Appendix F

Table F7

Simple Main Effects Tests of
Stimulus Presentations for
B (Stimulus) at A (Group) for
Between Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
B at A1	528.13	1	528.13	5.20*
B at A2	351.13	1	351.13	3.45
B at A3	306.28	1	306.28	3.01
Error (B x s w grps)	2134.72	21	101.65	

* $p < .05$

Appendix F

Table F8

AC (Group x Pretest/Test)

Summary Table of

Stimulus Presentations for

Between Subjects Controls

	C1	C2	Total
A1	406	620	1026
A2	399	435	834
A3	311	452	763
Total	1116	1507	2623

Appendix F

Table F9

Simple Main Effects Tests of
Stimulus Presentations for
A (Group) at C (Pretest/Test) for
Between Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
A at C1	350.38	2	175.19	0.45
A at C2	1307.04	2	653.52	1.68
Error (pooled)		21	388.92	

Appendix F

Table F10

Simple Main Effects Tests of
Stimulus Presentations for
C (Pretest/Test) at A (Group) for
Between Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
C at A1	1431.13	1	1431.13	6.62*
C at A2	40.50	1	40.50	0.19
C at A3	621.28	1	621.28	2.87
Error (C x s w grps)	4540.34	21	216.21	

* $p < .05$

Appendix F

Table F11

ABC (Group x Stimulus x Pretest/Test)Summary Table ofStimulus Presentations forBetween Subjects Controls

		C1	C2	Total
A1	B1	206	372	578
	B2	200	248	448
A2	B1	170	194	364
	B2	229	241	470
A3	B1	142	190	332
	B2	169	262	431
Total		1116	1507	2623

Appendix F

Table F12

Simple, Simple Main Effects Tests of
Stimulus Presentations for
B (Stimulus) at AC for
Between Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
B at A1C1	2.25	1	2.25	0.02
B at A1C2	961.00	1	961.00	7.83**
B at A2C1	217.56	1	217.56	1.77
B at A2C2	138.06	1	138.06	1.12
B at A3C1	45.56	1	45.56	0.37
B at A3C2	324.00	1	324.00	2.64
Error (pooled)		63	122.77	

** $p < .01$

Appendix F

Table F13

Simple, Simple Main Effects Tests of
Stimulus Presentations for
C (Pretest/Test) at AB for
Between Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
C at A1B1	1722.25	1	1722.25	14.03***
C at A1B2	144.00	1	144.00	1.17
C at A2B1	36.00	1	36.00	0.29
C at A2B2	9.00	1	9.00	0.07
C at A3B1	144.00	1	144.00	1.17
C at A3B2	540.56	1	540.56	4.40*
Error (pooled)		63	122.77	

* $p < .05$

**** $p < .001$

Appendix F

Table F'4

Simple Interaction Effects Tests of
Stimulus Presentations for
BC (Stimulus x Pretest/Test) at A (Group) for
Between Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
BC at A1	435.13	1	435.13	8.63**
BC at A2	4.50	1	4.50	0.09
BC at A3	63.28	1	63.28	1.25
Error (BC x s w grps)	1059.34	21	50.45	

** $p < .01$

Appendix G

Table G¹

Analysis of Variance Summary Table for
Lever Depression Duration for
Between Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
<u>Between Ss</u>				
Group (A)	12139.200	2	6069.600	3.227
Error(s w grps)	39494.226	21	1880.677	
<u>Within Ss</u>				
Stimulus (B)	1656.266	1	1656.266	2.202
A x B	7802.676	2	3901.338	5.186*
Error(B x s w grps)	15797.097	21	752.243	
Pretest/Test (C)	22846.584	1	22846.584	21.695****
A x C	8709.703	2	4354.851	4.135*
Error(C x s w grps)	22114.507	21	1053.072	
B x C	2604.271	1	2604.271	5.783*
A x B x C	4864.828	2	2432.414	5.402*
Error(BC x s w grps)	9456.284	21	450.299	

* $p < .05$ **** $p < .001$

Appendix G*

Figure Caption

Figure G1. Lever depression duration for
B (Stimulus) at C (Pretest/Test) for
paired/nothing subjects.

*same as D1

Appendix G

Figure G1

Appendix G

Figure Caption

Figure G2. Lever depression duration for
B (Stimulus) at C (Pretest/Test) for
random/nothing subjects.

Appendix G

Figure G2

Appendix G

Figure Caption

Figure G3. Lever depression duration for
B (Stimulus) at C (Pretest/Test) for
explicitly unpaired/nothing subjects.

Appendix G

Figure G3

Appendix G

Figure Caption

Figure G4. Lever depression duration for
B (Stimulus) at C (Pretest/Test) for
between subjects controls.

Appendix G

Figure G4

Appendix G

Table G2

BC (Stimulus x Pretest/Test)Summary Table ofLever Depression Duration forBetween Subjects Controls

	C1	C2	Total
B1	893	1883	2776
B2	944	1435	2379
Total	1837	3318	5155

Appendix G

Table G3

Simple Main Effects Tests of
Lever Depression Duration for
B (Stimulus) at C (Pretest/Test) for
Between Subjects Controls

<u>Source</u>	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
B at C1	54.19	1	54.19	0.09
B at C2	4181.33	1	4181.33	6.95*
Error (pooled)		21	601.27	

* $p < .05$

Appendix G

Table G4

Simple Main Effects Tests of
Lever Depression Duration for
C (Pretest/Test) at B (Stimulus) for
Between Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
C at B1	20418.75	1	20418.75	27.16****
C at B2	5022.52	1	5022.52	6.68*
Error (pooled)		21	751.69	

* $p < .05$

**** $p < .001$

Appendix G

Table G5

AB (Group x Stimulus)Summary Table ofLever Depression Duration forBetween Subjects Controls

	B1	B2	Total
A1	1374	841	2215
A2	619	754	1373
A3	783	784	1567
Total	2776	2379	5155

Appendix G

Table G6

Simple Main Effects Tests of
Lever Depression Duration for
A (Group) at B (Stimulus) for
Between Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
A at B1	19712.54	2	19712.54	7.49***
A at B2	244.13	2	122.06	0.09
Error (pooled)		21	1316.46	

*** $p < .005$

Appendix G

Table G7

Newman-Keuls Analysis of
Lever Depression Duration for
Between Group (A) Differences at the
Paired Stimulus (B1) Level for
Between Subjects Controls

	A2	A3	A1
	Random/ Nothing	Expl. Unp./ Nothing	Paired/ Nothing
	619	783	1530
A2		164	755**
A3			591**

** $p < .01$

Appendix G

Table G8

Simple Main Effects Tests of
Lever Depression Duration for
B (Stimulus) at A (Group) for
Between Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
B at A1	8877.78	1	8877.78	11.80***
B at A2	569.53	1	569.53	0.76
B at A3	0.03	1	0.03	0.00
Error(B x s w grps)	15797.10	21	752.24	

*** $p < .005$

Appendix G

Table G9

AC (Group x Pretest/Test)Summary Table ofLever Depression Duration forBetween Subjects Controls

	C1	C2	Total
A1	685	1530	2215
A2	635	738	1373
A3	517	1050	1567
Total	1837	3318	5155

Appendix G

Table 10

Simple Main Effects Tests of
Lever Depression Duration for
A (Group) at C (Pretest/Test) for
Between Subjects Controls

<u>Source</u>	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
A at C1	930.17	2	465.08	0.32
A at C2	19896.00	2	9948.00	6.78**
Error (pooled)		21	1466.87	

** $p < .01$

Appendix G

Table G11

Newman-Keuls Analysis of
Lever Depression Duration for
Between Group (A) Differences in the
Test (C2) Phase for Between Subjects Controls

	A2	A3	A1
	Random/ Nothing	Expl. Unp./ Nothing	Paired/ Nothing
	738	1050	1530
A2		312	792**
A3			480**

** $p < .01$

Appendix G

Table G12

Simple Main Effects Tests of
Lever Depression Duration for
C (Pretest/Test) at A (Group) for
Between Subjects Controls

<u>Source</u>	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
C at A1	22056.97	1	22056.97	20.95****
C at A2	331.53	1	331.53	0.31
C at A3	8877.78	1	8877.78	8.43**
Error (C x s w grps)	22114.51	21	1053.07	

**** $p < .001$

Appendix G

Table G13

ABC (Group x Stimulus x Pretest/Test)

Summary Table of

Lever Depression Duration for

Between Subjects Controls

		C1	C2	Total
A1	B1	354	1020	1374
	B2	331	510	841
A2	B1	289	330	619
	B2	346	408	754
A3	B1	250	533	783
	B2	267	517	784
Total		1837	3318	5155

Appendix G

Table G14

Simple, Simple Main Effects Tests of
Lever Depression Duration for
B (Stimulus) at AC for
Between Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
B at A1C1	33.06	1	33.06	0.04
B at A1C2	16256.25	1	16256.25	21.62****
B at A2C1	203.06	1	203.06	0.27
B at A2C2	380.25	1	380.25	0.51
B at A3C1	18.06	1	18.06	0.02
B at A3A2	16.00	1	16.00	0.02
Error (pooled)		63	751.87	

**** $p < .001$

Appendix G

Table G15

Simple, Simple Main Effects Tests of
Lever Depression Duration for
C (Pretest/Test) at AB for
Between Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
C at A1B1	27472.25	1	27472.25	36.54****
C at A1B2	2002.56	1	2002.56	2.66
C at A2B1	105.06	1	105.06	0.14
C at A2B2	240.25	1	240.25	0.32
C at A3B1	5005.56	1	5005.56	6.66*
C at A3B2	3906.25	1	3906.25	5.20*
Error (pooled)		63	751.87	

* $p < .05$

**** $p < .001$

Appendix G

Table G16

Simple Interaction Effects Tests of
Lever Depression Duration for
BC (Stimulus x Pretest/Test) at A (Group) for
Between Subjects Controls

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
BC at A1	7419.45	1	7419.45	16.48****
BC at A2	13.89	1	13.89	0.03
BC at A3	35.77	1	35.77	0.08
Error (BC x s w grps)	9456.28	21	450.30	

**** $p < .001$

Appendix H

Table H1

Analysis of Variance Summary Table ofLever Presses forPretest Between Group Preferences for Light and Tone

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
<u>Between Ss</u>				
Group (A)	596.374	2	298.187	0.680
Light/Tone (B)	1740.020	1	1740.020	3.969
A x B	55.291	2	27.645	0.063
Error (s w grps)	7890.124	18	438.340	
<u>Within Ss</u>				
Stimulus (C)	172.520	1	172.520	2.684
A x C	332.041	2	166.020	2.583
B x C	31.687	1	31.687	0.493
A x B x C	73.624	2	36.812	0.572
Error (C x s w grps)	1156.625	18	64.256	

Appendix H

Table H2

Means and Standard Deviations of
Lever Presses for
Pretest Between Group Preferences for Light and Tone

Group	Stimulus	Light	Tone
A1 (Group A)	Paired	<u>M</u> 38.50	23.50
		<u>SD</u> 24.28	22.98
	No Stimulus	<u>M</u> 35.75	21.25
		<u>SD</u> 18.66	12.60
A2 (Group B)	Random	<u>M</u> 26.75	20.00
		<u>SD</u> 15.64	11.28
	No stimulus	<u>M</u> 42.25	25.25
		<u>SD</u> 23.59	13.93
A3 (Group C)	Explicitly Unpaired	<u>M</u> 24.75	15.25
		<u>SD</u> 7.04	8.19

	<u>M</u>	28.25	18.75
Nc Stimulus			
	<u>SD</u>	9.06	8.26

Appendix I

Table I1

Analysis of Variance Summary Table of
Stimulus Presentations for
Pretest Between Group Preferences for Light and Tone

<u>Source</u>	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
<u>Between Ss</u>				
Group (A)	350.374	2	175.187	0.583
Light/Tone (B)	1121.333	1	1121.333	3.737
A x B	38.541	2	19.270	0.064
Error(s w grps)	5399.749	18	299.986	
<u>Within Ss</u>				
Stimulus (C)	133.333	1	133.333	4.172
A x C	132.041	2	66.020	2.065
B x C	5.333	1	5.333	0.166
A x B x C	67.041	2	33.520	1.048
Error(C x s w grps)	575.249	18	31.958	

Appendix I

Table I2

Means and Standard Deviations of
Stimulus Presentations for
Pretest Between Group Preferences for Light and Tone

Group	Stimulus	Light	Tone
A1 (Group A)	Paired	<u>M</u> 32.50	19.00
		<u>SD</u> 18.21	16.02
	No Stimulus	<u>M</u> 30.00	20.00
		<u>SD</u> 15.38	12.78
A2 (Group B)	Random	<u>M</u> 24.25	18.25
		<u>SD</u> 15.28	9.97
	No Stimulus	<u>M</u> 35.50	21.75
		<u>SD</u> 19.19	8.95
A3 (Group C)	Explicitly Unpaired	<u>M</u> 21.50	14.00
		<u>SD</u> 6.45	7.39

	<u>M</u>	24.75	17.50
No Stimulus			
	<u>SD</u>	8.05	7.76

Appendix J

Table J1

Analysis of Variance Summary Table of
Lever Depression Duration for
Pretest Between Group Preferences for Light and Tone

<u>Source</u>	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
<u>Between Ss</u>				
Group (A)	939.058	2	469.529	0.826
Light/Tone (B)	5441.085	1	5441.085	9.576**
A x B	424.541	2	212.270	0.373
Error(s w grps)	10226.818	18	568.156	
<u>Within Ss</u>				
Stimulus (C)	53.404	1	53.404	0.170
A x C	199.350	2	99.675	0.318
B x C	8.308	1	8.308	0.026
A x B x C	108.668	2	54.334	0.173
Error(C x s w grps)	5638.193	18	313.232	

** $p < .01$

Appendix J

Table J2

Means and Standard Deviations of
Lever Depression Duration for
Pretest Between Group Preferences for Light and Tone

Group	Stimulus	Light	Tone
A1 (Group A)	Paired	<u>M</u> 59.81	28.65
		<u>SD</u> 20.83	34.07
	No Stimulus	<u>M</u> 52.11	30.58
		<u>SD</u> 13.26	22.37
A2 (Group B)	Random	<u>M</u> 41.42	30.93
		<u>SD</u> 22.11	17.88
	No Stimulus	<u>M</u> 50.98	35.57
		<u>SD</u> 12.68	7.37
A3 (Group C)	Explicitly Unpaired	<u>M</u> 43.57	18.82
		<u>SD</u> 31.89	16.75

	<u>M</u>	45.54	21.09
Nc Stimulus			
	<u>SD</u>	24.94	8.46

Appendix K

Table K1

Analysis of Variance Summary Table ofLever Presses forPretest Within Group Preferences for Light and Tone

<u>Source</u>	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
<u>Between Ss</u>				
Group (A)	561.125	1	561.125	3.619
Error(s w grps)	2170.750	14	155.054	
<u>Within Ss</u>				
Light/Tone (B)	210.125	1	210.125	9.847**
A x B	66.125	1	66.125	3.099
Error(B x s w grps)	298.750	14	21.339	

** $p < .01$

Appendix K

Table K2

Means and Standard Deviations ofLever Presses forPretest Within Group Preferences for Light and Tone

Group		Light	Tone
Paired/Random	<u>M</u>	24.50	16.50
	<u>SD</u>	12.98	8.90
Paired/Explicitly Unpaired	<u>M</u>	13.25	11.00
	<u>SD</u>	8.17	6.19

Appendix L

Table 11

Analysis of Variance Summary Table ofStimulus Presentations forPretest Within Group Preferences for Light and Tone

<u>Source</u>	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
<u>Between Ss</u>				
Group (A)	496.125	1	496.125	3.927
Error (s w grps)	1768.875	14	126.348	
<u>Within Ss</u>				
Light/Tone (B)	190.125	1	190.125	7.469*
A x B	40.500	1	40.500	1.591
Error (B x s w grps)	356.375	14	25.455	

* $p < .05$

Appendix L

Table L2

Means and Standard Deviations ofStimulus Presentations forPretest Within Group Preferences for Light and Tone

Group		Light	Tone
Paired/Random	<u>M</u>	22.50	15.38
	<u>SD</u>	11.95	8.42
Paired/Explicitly Unpaired	<u>M</u>	12.38	9.75
	<u>SD</u>	7.85	5.31

Appendix M

Table M'

Analysis of Variance Summary Table ofLever Depression Duration forPretest Within Group Preferences for Light and Tone

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
<u>Between Ss</u>				
Group (A)	2184.936	1	2184.936	2.695
Error(s w grps)	11351.725	14	810.837	
<u>Within Ss</u>				
Light/Tone (B)	1271.340	1	1271.340	3.948
A x B	66.355	1	66.355	0.206
Error (B x s w grps)	4507.926	14	321.995	

Appendix M

Table M2

Means and Standard Deviations of
Lever Depression Duration for
Pretest Within Group Preferences for Light and Tone

Group		Light	Tone
Paired/Random	<u>M</u>	41.92	26.44
	<u>SD</u>	36.73	20.00
Paired/Explicitly Unpaired	<u>M</u>	22.52	12.79
	<u>SD</u>	21.79	6.49

Appendix N

Table N1

Analysis of Variance Summary Table of
Lever Presses for
Pretest Preferences for Right and Left Lever

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
<u>Between Ss</u>				
Group (A)	3185.575	4	796.394	2.424
Error(s w grps)	11497.375	35	328.496	
<u>Within Ss</u>				
Right/Left (B)	259.200	1	259.200	4.331*
A x B	321.175	4	80.294	1.342
Error(B x s w grps)	2094.625	35	59.846	

* $p < .05$

Appendix N

Table N2

Means and Standard Deviations of
Lever Presses for
Pretest Preferences for Right and Left Lever

Group		Right	Left
Paired/Nothing	<u>M</u>	34.75	24.50
	<u>SD</u>	21.72	16.57
Paired/Random	<u>M</u>	23.13	17.88
	<u>SD</u>	13.10	9.85
Paired/Explicitly Unpaired	<u>M</u>	12.75	11.50
	<u>SD</u>	7.25	7.39
Random/Nothing	<u>M</u>	27.75	29.38
	<u>SD</u>	10.35	23.02
Explicitly Unpaired/Nothing	<u>M</u>	24.50	21.63
	<u>SD</u>	9.78	9.32

Appendix O

Table O1

Analysis of Variance Summary Table of
Stimulus Presentations for
Pretest Preferences for Right and Left Lever

<u>Source</u>	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
<u>Between Ss</u>				
Group (A)	2153.300	4	538.325	2.262
Error(s w grps)	8328.500	35	237.957	
<u>Within Ss</u>				
Right/Left (B)	387.200	1	387.200	13.234****
A x B	88.800	4	22.200	0.759
Error(B x s w grps)	1024.000	35	29.257	

**** $p < .001$

Appendix O

Table O2

Means and Standard Deviations of
Stimulus Presentations for
Pretest Preferences for Right and Left Lever

Group		Right	Left
Paired/Nothing	<u>M</u>	28.75	22.50
	<u>SD</u>	16.10	15.05
Paired/Random	<u>M</u>	21.88	16.00
	<u>SD</u>	12.32	8.43
Paired/Explicitly Unpaired	<u>M</u>	12.25	9.88
	<u>SD</u>	6.80	6.66
Random/Nothing	<u>M</u>	25.63	24.25
	<u>SD</u>	9.68	18.32
Explicitly Unpaired/Nothing	<u>M</u>	22.75	16.13
	<u>SD</u>	8.33	6.17

Appendix P

Table P1

Analysis of Variance Summary Table of
Lever Depression Duration for
Pretest Preferences for Right and Left Lever

<u>Source</u>	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
<u>Between Ss</u>				
Group (A)	6047.728	4	1511.932	1.929
Error(s w grps)	27437.659	35	783.933	
<u>Within Ss</u>				
Right/Left (B)	3976.623	1	3976.623	19.648****
A x B	784.237	4	196.059	0.969
Error(B x s w grps)	7083.659	35	202.390	

**** $p < .001$

Appendix P

Table P2

Means and Standard Deviations of
Lever Depression Duration for
Pretest Preferences for Right and Left Lever

Group		Right	Left
Paired/Nothing	<u>M</u>	44.36	41.22
	<u>SD</u>	25.26	27.28
Paired/Random	<u>M</u>	44.34	23.96
	<u>SD</u>	37.26	16.08
Paired/Explicitly Unpaired	<u>M</u>	24.59	10.72
	<u>SD</u>	20.38	6.75
Random/Nothing	<u>M</u>	46.26	33.21
	<u>SD</u>	11.37	18.45
Explicitly Unpaired/Nothing	<u>M</u>	42.29	22.23
	<u>SD</u>	28.99	11.47

Appendix Q

Table Q1

Analysis of Variance Summary Table of
Between Group Differences in Age

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
Age	11651.849	4	2912.962	4.233**
Error	24086.124	35	688.175	

** $p < .01$

Appendix Q

Table Q2

Means and Standard Deviations of
Between Group Differences in Age

Group	<u>M</u>	<u>SD</u>
Paired/Nothing	156.50	14.64
Paired/Random	143.63	29.85
Paired/Explicitly Unpaired	124.13	37.42
Random/Nothing	117.75	24.66
Explicitly Unpaired/Nothing	110.38	18.09

Appendix Q

Table Q3

Newman-Keuls Analysis of
Between Group Differences in Age

A5	A4	A3	A2	A1
E U/N	R/N	P/E U	E/R	P/N
883	942	993	1149	1252
<hr/>				
A5	59	110	266	369**
A4		51	207	310*
A3			156	259*
A2				103
<hr/>				

* $p < .05$ ** $p < .01$

Appendix B

Table R1

Analysis of Variance Summary Table of
Between Group Differences in
Percent of Training Pairings

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
Percent of Pairings	777.849	4	194.462	3.575*
Error	1903.749	35	54.393	

* $p < .05$

Appendix R

Table R2

Means and Standard Deviations of
Between Group Differences in
Percent of Training Pairings

Group	<u>M</u>	<u>SD</u>
Paired/Nothing	151.88	3.80
Paired/Random	154.63	5.83
Paired/Explicitly Unpaired	144.00	1.85
Random/Nothing	144.25	12.15
Explicitly Unpaired/Nothing	152.25	8.51

Appendix B

Table B3

Newman-Keuls Analysis of
Between Group Differences in
Percent of Training Pairings

	A3	A4	A1	A5	A2
	P/E U	R/N	P/N	E U/N	P/R
	144.00	144.25	151.88	152.25	154.63
A3		0.25	7.88	8.25	10.63*
A4			7.63	8.00	10.38*
A1				0.37	2.75
A5					2.38

* $p < .05$

Appendix S

Table S1

Analysis of Variance Summary Table of
Between Group Differences in Weight

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
Weight	51788.664	4	12947.166	2.679*
Error	169122.430	35	4832.069	

* $p < .05$

Appendix S

Table S2

Means and Standard Deviations of
Between Group Differences in Weight

Group	<u>M</u>	<u>SD</u>
Paired/Nothing	569.69	59.72
Paired/Random	568.81	43.24
Paired/Explicitly Unpaired	495.25	104.42
Random/Nothing	518.75	55.64
Explicitly Unpaired/Nothing	484.25	68.74

Appendix S

Table S3

Newman-Keuls Analysis of
Between Group Differences in Weight

	A5	A3	A4	A2	A1
	E U/N	P/E U	R/N	P/R	P/N
	484.25	495.25	518.75	568.81	569.69
A5		11.00	34.50	84.56	85.44
A3			23.50	73.56	74.44
A4				50.06	50.94
A2					0.88

Appendix T

Table T1

Analysis of Variance Summary Table of
Between Group Differences in Number of
Pretraining Days to Criterion

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
Pretraining Days	3.650	4	0.912	0.740
Error	43.125	35	1.232	

Appendix T

Table T2

Means and Standard Deviations of
Between Group Differences in Number of
Pretraining Days to Criterion

<u>Group</u>	<u>M</u>	<u>SD</u>
Paired/Nothing	1.63	0.92
Paired/Random	1.50	1.07
Paired/Explicitly Unpaired	1.38	0.74
Random/Nothing	2.25	1.49
Explicitly Unpaired/Nothing	1.63	1.19

Appendix U

Table U1

Analysis of Variance Summary Table of
Between Group Differences in Training Time

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
Training Time	2440.857	4	610.214	1.332
Error	16032.740	35	458.078	

Appendix U

Table U2

Means and Standard Deviations of
Between Group Differences in Training Time

<u>Group</u>	<u>M</u>	<u>SD</u>
Paired/Nothing	411.88	29.63
Paired/Random	424.13	17.84
Paired/Explicitly Unpaired	422.50	22.53
Random/Nothing	403.25	9.95
Explicitly Unpaired/Nothing	420.25	22.08

Appendix V

Table V1

Analysis of Variance Summary Table of
Between Group Differences in Number of
Pretraining Lever Presses to Criterion

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
Lever Press	94904.348	4	23726.087	0.376
Error	2207559.620	35	63073.132	

Appendix V

Table V2

Means and Standard Deviations of
Between Group Differences in Number of
Pretraining Lever Presses to Criterion

Group	<u>M</u>	<u>SD</u>
Paired/Nothing	535.36	322.63
Paired/Random	460.00	271.67
Paired/Explicitly Unpaired	479.13	254.81
Random/Nothing	407.88	224.47
Explicitly Unpaired/Nothing	404.00	148.87

Appendix W

Table W1

Analysis of Variance Summary Table of
Between Group Differences in Number of
Days of the Experimental Procedure

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
Days	4262.000	4	1065.500	1.593
Error	23414.376	35	668.982	

Appendix W

Table W2

Means and Standard Deviations of
Between Group Differences in Number of
Days of the Experimental Procedure

<u>Group</u>	<u>M</u>	<u>SD</u>
Paired/Nothing	20.38	14.25
Paired/Random	16.68	6.12
Paired/Explicitly Unpaired	13.13	2.90
Random/Nothing	41.88	39.39
Explicitly Unpaired/Nothing	28.88	39.30

Appendix W

Table W3

Analysis of Variance Summary Table of
Natural Log Transformation of
Between Group Differences in Number of
Days of the Experimental Procedure

Source	<u>SS</u>	<u>df</u>	<u>MS</u>	<u>F</u>
Days	3.370	4	0.843	2.108
Error	14.003	35	0.400	

Appendix W

Table W4

Means and Standard Deviations of
Natural Log Transformation of
Between Group Differences in Number of
Days of the Experimental Procedure

Group	<u>M</u>	<u>SD</u>
Paired/Nothing	2.82	0.64
Paired/Random	2.73	0.39
Paired/Explicitly Unpaired	2.55	0.21
Random/Nothing	3.42	0.80
Explicitly Unpaired/Nothing	2.91	0.86

Bibliography

- Beninger, R. J., & Phillips, A. G. (1980). The effect of pimozide on the establishment of conditioned reinforcement. Psychopharmacology, 68, 147-153.
- Bersh, P. J. (1951). The influence of two variables upon the establishment of a secondary reinforcer for operant responses. Journal of Experimental Psychology, 41, 62-73.
- Bilbrey, J., & Winokur, S. (1975). Conditioned reinforcement: Effects of temporal contingency and percentage of pairing. Journal of General Psychology, 93, 247-260.
- Bolles, R. C. (1967). Theory of Motivation. New York: Harper & Row.
- Bolles, R. C. (1975). Theory of Motivation (2nd ed.). New York: Harper & Row.
- Borgealt, A. J., Donahue, J. W., & Weinstein, A. (1972). Effects of delayed and trace components of a compound CS on conditioned suppression and heart rate. Psychonomic Science, 26, 13-15.
- Bower, G., McLean, J., & Meacham, J. (1966). Value of knowing when reinforcement is due. Journal of Comparative and Physiological Psychology, 62, 184-192.
- Box, G. E. P. (1953). Non-normality and tests of variances. Biometrika, 40, 318-335.
- Bugelski, R. (1938). Extinction with and without sub-goal

reinforcement. Journal of Comparative Psychology,
26, 121-133.

Bugelski, R. (1956). The Psychology of Learning.
 New York: Holt.

Butter, C. M., & Thomas, D. R. (1958). Secondary
 reinforcement as a function of the amount of primary
 reinforcement. Journal of Comparative and
Physiological Psychology, 51, 346-348.

Catania, A. C. (1963). Concurrent performances: A
 baseline for the study of reinforcement magnitude.
Journal of Experimental Analysis of Behavior, 6,
 299-300.

Church, R. M. (1964). Systematic effect of random error
 in the yoked control design. Psychological Bulletin,
62, 122-131.

Cowles, J. T. (1937). Food-tokens as incentives for
 learning by chimpanzees. Comparative Psychology
Monographs, 14, (5).

Crowder, W. F., Gay, B. R., Bright, M. G., & Lee, M. F.
 (1959). Secondary reinforcement or response
 facilitation? III. Reconditioning. Journal of
Psychology, 48, 307-310.

Crowder, W. F., Gill, K., Hodge, C. C., & Nash, F. A.
 (1959). Secondary reinforcement or response
 facilitation? II. Response acquisition. Journal
of Psychology, 48, 303-306.

Crowder, W. F., Morris, J. B., & McDaniel, M. H.

(1959). Secondary reinforcement or response facilitation? I. Resistance to extinction. Journal of Psychology, 48, 299-302.

Crowder, W. F., Smith, S. C., Davis, W. M., Noel, J. T., & Coussens, W. R. (1972). Effect of morphine dose size on the conditioned reinforcing potency of stimuli paired with morphine. Psychological Record, 22, 441-448.

Crum, J., Brown, W. L., & Bitterman, M. E. (1951). The effect of partial and delayed reinforcement on resistance to extinction. American Journal of Psychology, 64, 228-237.

D'Amato, M. R. (1955). Secondary reinforcement and magnitude of primary reinforcement. Journal of Comparative and Physiological Psychology, 48, 378-380.

D'Amato, M. R. (1955a). Transfer of secondary reinforcement across the hunger and thirst drives. Journal of Experimental Psychology, 49, 352-355.

Denny, M. R., & Ratner, S. C. (1970). Comparative Psychology: Research in Animal Behavior (revised ed.). Homewood, Ill.: The Dorsey Press.

DiCara, L. V. (1966). Brain stimulation and secondary reinforcement. Dissertation Abstracts, 27, (2157-E).

DiCara, L. V., & Deutsch, J. A. (1966). Secondary reinforcement as a function of drive in intracranial self-stimulation. Proceedings of the 74th Annual

Convention of the American Psychological Association,
105-106.

Dinsmoor, J. A. (1950). A quantitative comparison of the discriminative and reinforcing functions of a stimulus. Journal of Experimental Psychology, 40, 458-472.

Doerries, L. E. (1974). Durability of secondary reinforcement as a function of manipulation in the training and testing situations. Dissertation Abstracts, 30, (936).

Egger, M. D., & Miller, N. E. (1962). Secondary reinforcement in rats as a function of information value and reliability of the stimulus. Journal of Experimental Psychology, 64, 97-104.

Egger, M. D., & Miller, N. E. (1963). When is reward reinforcing? An experimental study of the information hypothesis. Journal of Comparative Physiological Psychology, 56, 132-137.

Elam, C. B., Tyler, D. W., & Bitterman, M. E. (1954). A further study of secondary reinforcement and the discrimination hypothesis. Journal of Comparative and Physiological Psychology, 47, 381-384.

Ferster, C. B., & Skinner, B. F. (1957). Schedules of Reinforcement. New York: Appleton-Century-Crofts.
441-448.

Gilbert, T. T., & Sturdivant, E. R. (1958) The effect of a food-associated stimulus on operant-level

locomotor behavior. Journal of Comparative and Physiological Psychology, 51, 255-257.

Hall, J. F. (1966). The Psychology of Learning. Philadelphia: J. B. Lippincott Co.

Hall, J. F. (1976). Classical Conditioning and Instrumental Learning: A Contemporary Approach. Philadelphia: Lippincott Company.

Hancock, R. A. (1982). Tests of conditioned reinforcement value of sequential stimuli in pigeons. Animal Learning and Behavior, 10, 46-54.

Harris, R. S. (1981). Conditioned reinforcement: reality or myth? Unpublished manuscript, University of Rhode Island, Psychology Department, Kingston, Rhode Island.

Hart, B. L. (1976). Experimental Psychobiology: A Laboratory Manual. San Francisco: W. H. Freeman & Company, (p. 76).

Hendry, D. P. (1969). Conditioned Reinforcement. Homewood, Ill: Dorsey Press.

Hilgard, E. R., & Marquis, D. G. (1940). Conditioning and Learning. New York: Appleton-Century.

Hopkins, C. O. (1955). Effectiveness of secondary reinforcing stimuli as a function of the quantity and quality of food reinforcement. Journal of Experimental Psychology, 50, 339-342.

Hull, C. L. (1943). Principles of Behavior. New York: Appleton-Century-Crofts.

- Hull, C. L. (1951). Essentials of Behavior. New York: Yale University Press.
- Hutchinson, R. R., & Renfrew, J. W. (1966). Stalking, attack, and eating behaviors elicited from the same sites in the hypothalamus. Journal of Comparative and Physiological Psychology, 61, 360-367.
- Hyde, T. S. (1976). The effect of Pavlovian stimuli on the aquisition of a new response. Learning and Motivation, 7, 223-239.
- Kanfer, F. H. (1960). Incentive value of generalized reinforcers. Psychological Reports, 7, 531-538.
- Keehn, J. D. (1962). The effect of post-stimulus conditions on the secondary reinforcing power of a stimulus. Journal of Comparative and Physiological Psychology, 55, 22-26.
- Kelleher, R. T. (1966). Conditioned reinforcement in second-order schedules. Journal of Experimental Analysis of Behavior, 9, 475-485.
- Kelleher, R. T., & Gollub, L. R. (1962). A review of positive conditioned reinforcement. Journal of Experimental Analysis of Behavior, 5, 543-597.
- Keller, F. S., & Schoenfeld, W. N. (1950). Principles of Psychology. New York: Appleton-Century-Crofts.
- Klein, R. M. (1959). Intermittent primary reinforcement as a parameter of secondary reinforcement. Journal of Experimental Psychology, 58, 423-427.
- Kling, J. W., & Schrier, A. M. (1971). Positive

reinforcement. In J. W. Kling & I. Figgs (Eds.),
Experimental Psychology (3rd ed.) (p. 615-702).

New York: Holt, Rinehart, & Winston.

Knott, P. D., & Clayton, K. N. (1966). Durable
 secondary reinforcement using brain stimulation as the
 primary reinforcer. Journal of Comparative and
 Physiological Psychology, 61, 151-153.

Lawson, R. (1957). Brightness discrimination performance
 and secondary reward strength as a function of primary
 reward amount. Journal of Comparative and
 Physiological Psychology, 50, 35-39.

Longstreth, L. E. (1971). A cognitive interpretation of
 secondary reinforcement. Nebraska Symposium on
 Motivation, 33-80.

Lorenz, K. (1950). The comparative method in studying
 innate behavior patterns. In Symposium of the Society
 for Experimental Biology, Vol. IV, Physiological
 Mechanisms in Animal Behavior, Cambridge, England:
 Cambridge University Press, 221-268.

Marx, M. H., & Murphy, W. W. (1961). Resistance to
 extinction as a function of the presentation of a
 motivating cue in the start box. Journal of
 Comparative and Physiological Psychology, 54,
 207-210.

Miles, R. C. (1956). The relative effectiveness of
 secondary reinforcers throughout deprivation and
 habit-strength parameters. Journal of Comparative

and Physiological Psychology, 49, 126-130.

- Mogensen, G. J. (1965). An attempt to establish secondary reinforcement with rewarding brain stimulation. Psychological Reports, 16, 163-167.
- Mogenson, G., & Cioe, J. (1977). Central reinforcement: A bridge between brain function and behavior. In W. K. Honig & J. E. R. Staddon (Eds.), Handbook of Operant Behavior (p. 570-595). Englewood Cliffs, New Jersey: Prentice-Hall, Inc.
- Nevin, J. A. (1966). Generalized conditioned reinforcement in satiated rats. Psychonomic Science, 5, 191.
- Olds, J. (1956). Runway and maze behavior controlled by baso-medial forebrain stimulation in the rat. Journal of Comparative and Physiological Psychology, 49, 507-512.
- Platt, J. R., & Wike, E. L. (1962). Inaccessible food as a secondary reinforcer for deprived and satiated rats. Psychological Reports, 11, 837-840.
- Platt, J. R., & Wike, E. L. (1964). Food reinforcement for non-hungry-non-thirsty rats: conditioned reinforcement or manipulative behavior? Psychological Record, 14, 311-317.
- Pliskoff, S. S., Hawkins, T. D., & Wright, J. E. (1964). Some observations of the discriminative stimulus hypothesis and rewarding electrical stimulation of the brain. Psychological Record, 14,

179-184.

- Porter, L. W., & Miller, N. E. (1957). Training under two drives, alternately present, vs. training under a single drive. Journal of Experimental Psychology, 54, 1-7.
- Powell, A., & Cole, S. R. (1973). Secondary reinforcement and the reconditioning hypothesis. Psychological Record, 32, 719-722.
- Rescorla, R. A. (1967). Pavlovian conditioning and its proper control procedures. Psychological Review, 74, 71-80.
- Roberts, W. W., Steinberg, M. L., & Means, I. (1967). Hypothalamic mechanisms for sexual, aggressive, and other motivational behaviors in the opossum. Journal of Comparative and Physiological Psychology, 64, 1-15.
- Saltzman, I. J. (1949). Maze learning in the absence of primary reinforcement: A study of secondary reinforcement. Journal of Comparative and Physiological Psychology, 42, 161-173.
- Schlosberg, H., & Pratt, C. H. (1956). The secondary reward value of inaccessible food for hungry and satiated rats. Journal of Comparative and Physiological Psychology, 49, 149-152.
- Schoenfeld, W. N., Antonitis, J. J., Bersh, P. J. (1950). A preliminary study of training conditions necessary for secondary reinforcement. Journal of Experimental Psychology, 40, 40-45.

- Seward, J. P. (1963). The structure of functional autonomy. American Psychologist, 18, 703-710.
- Seward, J. P., & Levy, N. (1953). Choice-point behavior as a function of secondary reinforcement with relevant drives satiated. Journal of Comparative and Physiological Psychology, 46, 334-338.
- Seward, J. P., Uyeda, A. A., & Clds, J. (1959). Resistance to extinction following cranial self-stimulation. Journal of Comparative and Physiological Psychology, 52, 294-299.
- Silverstein, A., & Lipsett, L. P. (1974). The role of instrumental responding and contiguity in the development of infant secondary reinforcement. Journal of Experimental Child Psychology, 17, 322-331.
- Skinner, B. F. (1938). The Behavior of Organisms. New York: Appleton-Century-Crofts.
- Skinner, B. F. (1953). Science and Human Behavior. New York: Macmillan.
- Spence, K. W. (1956). Behavior Theory and Conditioning. New Haven, Conn: Yale University Press.
- Spies, G. (1965). Food versus intracranial self-stimulation reinforcement in food-deprived rats. Journal of Comparative and Physiological Psychology, 60, 153-157.
- Stein, L. (1958). Secondary reinforcement established with subcortical stimulation. Science, 127,

466-467.

- Terrace, H. S. (1973). Classical conditioning. In Nevin, J. A. & Reynolds, G. S. (Eds.), The Study of Behavior: Learning, Motivation, and Instinct. Glenview, Illinois: Scott, Foresman, and Company.
- Thomas, D. R., Berman, D. L., Serednesky, G. E., & Lyons, J. (1968). Information value and stimulus configuring as factors in conditioned reinforcement. Journal of Experimental Psychology, 76, 181-189.
- Tolman, E. C. (1932) Purposive Behavior in Animals and Men. New York: Century.
- Wike, E. L. (1966). Secondary Reinforcement. New York: Harper and Row.
- Wike, E. L. (1969). Secondary reinforcement: some research and theoretical issues. Nebraska Symposium on Motivation, 39-84.
- Wike, E. L., & Casey, A. (1954) The secondary reward value of food for satiated animals. Journal of Comparative and Physiological Psychology, 47, 441-443.
- Wike, E. L., & McNamara, H. J. (1955). A quest for the generalized conditioned reinforcer. Psychological Reports, 1, 83-91.
- Wolfe, J. B. (1936). Effectiveness of token rewards for chimpanzees. Comparative Psychology Monographs, 12 (60).
- Wyckoff, L. B. (1952). The role of observing responses

in discrimination learning. Psychological Review,
59, 431-442.

Wyckoff, L. B. (1969). The role of observing responses
in discrimination learning. In D. P. Hendry (Ed.),
Conditioned Reinforcement (p. 237-260). Homewood,
Ill: The Dorsey Press.

Wyckoff, L. B., Sidowski, J., & Chambliss, E. J. (1958).
An experimental study of the relationship between
secondary reinforcing and cue effects of a stimulus.
Journal of Comparative and Physiological Psychology,
51, 103-109.

Zimmerman, D. W. (1959). Sustained performance in rats
based on secondary reinforcement. Journal of
Comparative and Physiological Psychology, 52, 353-358.