Thompson, Lashley, and Spearman: Three Views of the Biological Basis of Intelligence

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An important problem for the empirical psychologists of a century ago was the differentiation of human from non-human intelligence.[1,2] The strategy they adopted to demonstrate interspecific differences involved (1) establishing a plausible definition of "intelligence," (2) developing tests of the construct which could be performed by a number of species, and (3) arraying the performances of the different species according to test scores.[3–6] This approach rarely yielded a clear phylogenetic progression of test performances, although a few notable successes have been reported.[7,8]

For most of the twentieth century, American comparative psychology has been rooted in Thorndike's conclusion of 1898 that there are no qualitative interspecific differences in intellect.[6,8,9] In retrospect, failure to validate this basic tenet of Darwinism appears to have been due to longstanding disagreements over the nature of intelligence in the human animal, much less in infrahuman species. Over time, the search for a consensual operational definition of the construct in animals was largely abandoned and replaced by investigations of specific components of the behavioral repertoire, as represented in paradigms such as classical conditioning, maze learning, passive avoidance and the like. In the process of exploring these areas, one of the experimental methods that emerged was the induction of brain lesions to produce variability in performance, a practice that came to be called "neuropsychology."[10,11] Karl Lashley is generally considered the founder of that subdiscipline of experimental psychology.[12]

INTELLECTUAL RETARDATION

At the turn of the century, empiricism was introduced into the study of mental retardation with the objective investigations of Binet and Simon.[13] Despite the contemporaneous emergence of neuropsychology and the scientific study of mental retardation, few early investigators drew parallels between animals with experimentally induced brain lesions and mentally retarded humans. There were several reasons why these lines of inquiry failed to converge, the two most important being that there had never been an agreed-upon operational definition of intelligence in non-human species, and that the investigation of defective performance in animals was typically confined to a limited set of laboratory tasks, thereby constraining inferences that a general intellectual loss

had been sustained. Adding more tests to a battery was not of much help, since animal strains with elevated performance on one problem would not necessarily be bright on another.[14–17] Thus, investigators seemed to discount the obvious parallels between performance deficit in animals and human mental retardation.

The Animal Model

In 1979, when Robert Thompson began his search for an animal model of mental retardation, his first order of business was to operationally define the elusive construct "intelligence." Analyzing the composition of human intelligence tests, he determined that they could be conceptualized as sets of problem-solving tasks similar to those he used with laboratory rats. Furthermore, the simplest interpretation of the work of Spearman[18] and other intelligence theorists was that "intelligent" individuals performed well on a great number of tasks, while retardates performed poorly on a great number of tasks. He therefore concluded that an acceptable operational definition of a "mentally retarded rat" would be an animal that performed poorly on all problem solving tasks.

This premise was tested in 28 original experiments conducted by Thompson and his colleagues, employing a paradigm in which animals were brain-lesioned in infancy and tested in adulthood.[19–46] As the result of these studies, in which more than 60 brain sites were investigated, Thompson found that a particular set of eight brain structures—(1) substantia nigra, (2) superior colliculus, (3) median raphe, (4) ventral tegmentum, (5) ventrolateral thalamus, (6) pontine reticular formation, (7) caudatoputamen, and (8) globus pallidus—were critical for every problem-solving task, including mazes, sensory discriminations, detours, puzzle boxes and various tests of inhibition. FIGURE 1 is a schematic parasagittal section of the rat brain showing the approximate locations of these structures.

Along the way, Thompson began calling animals lesioned in one or more of these critical brain structures "mentally retarded rats" and the constellation of eight brain structures themselves the "general learning system."[23,24,33,37] Later, he determined that it would be more accurate to call this system the "nonspecific mechanism" *(Nsp)* in view of the fact that lesions to the system disrupted problem-solving performances in a manner clearly unlike the "specific" way that lesions to other brain structures affected the same set of performances.[44] That is, the decrement in performance associated with every other lesion was usually explicable from the requisite test performances and the correlative neuroanatomy of the brain area involved (e.g., occipital lobe and visual discrimination). Moreover, the performance deficits associated with all other lesions were linked to a particular test, or at most a subset of tests, but not all tests.

The Spearman Two-Factor Theory

The parallels between the *Nsp* and Spearman's general intelligence, or *g*, factor were inescapable. Spearman viewed *g* as a trait that was involved in the

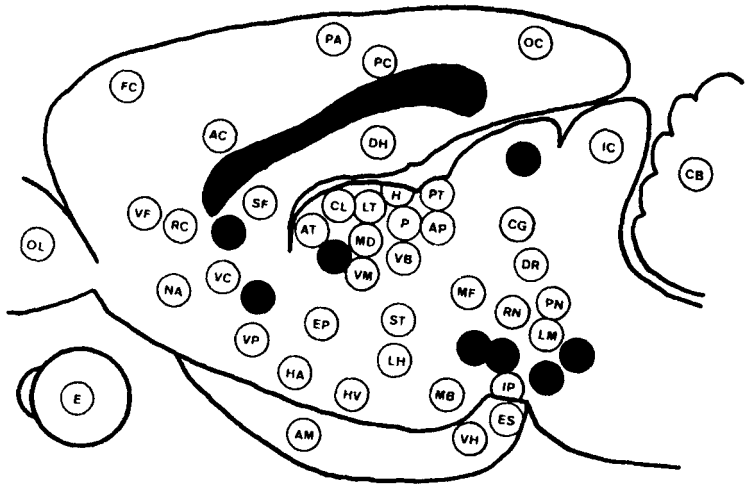

FIGURE 1. Schematic parasagittal section of the rat brain showing the approximate locations of 50 structures lesioned in various experiments conducted from 1979–1989 in the Thompson laboratory. For brain structures signified by abbreviations, see TABLE 2. (From Thompson, Crinella, and Yu.[44] Reprinted by permission.)

performance of all intellectual tasks, a conclusion largely based on the observation that virtually all tests of human performance correlated positively among themselves.[48] There were also specific factors in Spearman's "two-factor" theory—sources of variance peculiar to each test. These bore a theoretical resemblance to specific brain mechanisms Thompson had identified—that is, subsets of brain structure that could be tied to the requirements of some laboratory problem-solving tasks, but not others.

However, Thompson was at odds with Spearman's view on the biological basis of *g*, since Thompson viewed the construct as a reflection of the operation of a limited number of structures, whereas Spearman saw the presence of the factor as evidence of some property of the entire brain:

> The factor was taken ... to consist in something of the nature of an "energy" or "power" which serves in common the whole cortex (or possibly, even, the whole nervous system).
>
> (Ref. 18, p. 5)

FIGURE 2 schematizes the remarkable conceptual similarities between Spearman's and Thompson's formulations.[39,49]

The Lashley Connection

The Thompson-Lashley connection has already been alluded to in this volume and elsewhere.[50] Briefly, Thompson had been a research assistant at the

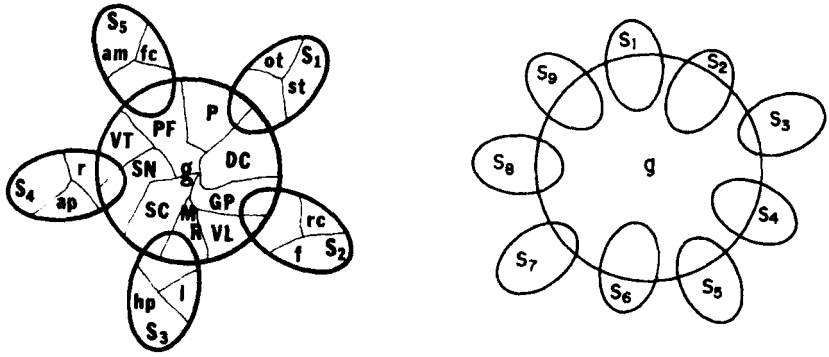

FIGURE 2. *Left:* Thompson's graphic depiction of Spearman's *g* as a biological construct. Large inner circle shows structures of the nonspecific mechanism, which is necessary for performance of five specific tests (indicated by overlapping ovals; adapted from Thompson Crinella, and Yu[44]). g = general factor; s_1–s_5 = specific factors. *Right:* Spearman's graphic depiction of his two-factor theory of intelligence (adapted from Jensen[49]). g = general factor; s_1–s_9 = specific factors. Small ovals indicate tests of abilities, which require both *g* (area of overlap) and specific factors (non-overlapping areas) for their performance. For brain structures signified by abbreviations, see TABLE 2.

Yerkes Laboratory, then under the direction of Lashley, and this had been a nodal experience for Thompson. The fact that Lashley had related his findings to Spearman's theory had not gone unnoticed by Thompson. In fact, Lashley had undertaken his magnum opus in hopes of clarifying two of the preeminent theoretical controversies of that time: (1) the aggregate versus the unitary view of intelligence, and (2) the localizationist versus the mass-action view of neural activity underlying intelligent behavior.[51] In his monograph, Lashley classified intelligence theories into aggregate-unitary viewpoints, with Thorndike and Thurstone being proponents of the former, while he and Spearman represented the latter. Also like Spearman, he saw intelligence as a property of the whole brain.

In undertaking his famous study, Lashley posed the rhetorical question: "Can we find in animals a constellation of activities, having some of the attributes of human intelligence, and capable of modification through experimental control of nervous functioning?" (Ref. 18, p. 13). He assumed that the brain was more or less uniform for all rats and that the only differences would therefore be attributable to the brain lesions he induced.

TABLE 1 outlines one of Lashley's experiments, having to do with the effects of lesions on original learning (he also conducted a retention experiment). A total of 35 animals, 19 operates and 16 controls, were used. In order to determine the effects of lesions, each animal was ranked according to (1) percent destruction of the cerebral cortex and (2) performance on each test. Spearman rank-order correlation coefficients were then calculated between (1) and (2).

Lashley's conclusions are among the best-known in psychological lore: (1)

TABLE 1. Outline of Original Learning Experiment in Lashley's 1929 Study

	Subjects		
	Original *n*	Died	Studied
Lesioned[a]	50	31	19
Controls	22	6	16
			Total *N* = 35

Problems	Incentives
Maze 1	Hunger
Maze 2	Hunger
Maze 3	Hunger
Brightness Discrimination	Shock/hunger
Retention: Maze 1	Hunger
Retention: Maze 2	Hunger
Retention: Brightness	Shock/hunger
Reversal: Maze 1	Hunger
Maze 4	Hunger
Inclined-plane discrimination	Shock/hunger
	Dependent Measures
For Performance	Time
	Errors
	Trials
For Lesions	Percent cortical destruction

[a]Lesions were induced in the frontal, lateral, occipital, and parietal cortices.

the degree of deficit is proportional to the magnitude of the cortical damage; (2) problem-solving deficits are produced by lesions in any cortical area; (3) diverse lesions of equal magnitude produce equal problem-solving deficits; and (4) the amount of overall intellectual retardation is solely dependent on the extent of cortical destruction. These conclusions did not prove impregnable to subsequent findings, including Lashley's later work. However, Lashley's view (in keeping with Spearman) that intelligence was a unitary trait continues to find adherents.[52,53]

THOMPSON'S CONTRIBUTION TO INTELLIGENCE THEORY

Thompson's goal was more modest than Lashley's. He did not envision resolving metatheoretical disputes, but rather only developing an animal model of mental retardation. Nevertheless, in the pursuing his limited aim, he was also able to cast light on the two longstanding questions that had concerned Lashley and others, as illustrated by the study described below.

Methods

Subjects and Surgery

Five hundred weanling (21 to 25 days old) male Sprague-Dawley albino rats were operated on under deep chloral hydrate anesthesia. Cortical lesions were created by aspiration, while subcortical lesions were made stereotaxically by passing a constant anodal current through an electrode. TABLE 2 lists the lesion locations, which numbered 49 in all. Sham-operated control animals (n = 75) underwent the same surgical procedure as the experimental animals, except for the drilling of the skull and the insertion of the suction tip or lesion electrode.

TABLE 2. List of Brain Lesion Locations and Abbreviations

Neocortex
Frontal cortex (ventral) VF
Frontal cortex (dorsal) FC
Occipitotemporal cortex OC
Parietal cortex PA

Other Telencephalic Areas
Frontocingulate cortex AC
Cingulate cortex (posterior region) PC
Entorhinosubicular area ES
Nucleus accumbens septi NA
Septofornix area SF
Hippocampus (dorsal) DH
Hippocampus (ventral) VH
Entopeduncular nucleus EP
Amygdala AM
Caudatoputamen (ventral) VC
Caudatoputamen (rostral) RC
Caudatoputamen (dorsal) DC
Globus pallidus GP
Ventral pallidum VP
Olfactory bulbs OL

Thalamus
Anterior complex AT
Ventrolateral complex VL
Centrolateral region CT
Lateral complex LT
Mediodorsal complex MD
Ventromedial region VM
Ventrobasal region VB
Parafascicular region P
Habenular nuclei H

Hypothalamus
Anterior region HA
Ventromedial region HV
Posterolateral region LH
Mamillary bodies MB
Subthalamus ST

Pretectal area
Medial pretectal area PT
Anterior pretectal nucleus AP

Brainstem reticular formation
Midbrain area (paramedial) MF
Pontine area (paramedial) PF

Other brainstem structures
Interpedunculocentral tegmental area IP
Raphe area (median) MR
Raphe area (dorsal) DR
Lateral midbrain area LM
Pedunculopontine area (dorsal) PN
Cerebellum CB
Substantia nigra (lateral) SN
Ventral tegmental area VT
Central gray (midbrain) CG
Inferior colliculus IC
Superior colliculus SC

Other structures
Eyes E

Procedure

Following a 3-week recovery period, at approximately 42 days of age, the animals began training on the test battery described in TABLE 3. The appetitively motivated detour problems were learned first, followed in sequence by the visual discrimination, maze, and inclined-plane problem.

The detour apparatus is shown in FIGURE 3. It was divided into a start box, choice chamber, and goal box. A guillotine door separated the start box from the choice chamber. Interchangeable partitions fitted with a platform, cylinder, or ladder could be positioned between the choice chamber and goal box to form the three detour problems. During preliminary training, a partition containing a centrally positioned opening at floor level was used. For Problem A, the partition used in preliminary training remained in the apparatus along with a platform that sloped upward into the choice chamber so that the animal had to climb onto it to reach the window to the goal box. Problem B consisted of a partition containing a centrally located plastic cylinder that extended into the choice chamber and was elevated above the floor so that the animal had to climb into the cylinder in order to crawl forward into the goal box. Problem C consisted of a partition containing a window located above the floor that could only be reached by climbing a vertically positioned ladder that extended into the choice chamber which the animal had to climb in order to reach the elevated window to the goal box.

For the *visual discrimination problem,* a two-choice Thompson-Bryant discrimination box was used, utilizing the motive of escape-avoidance of mild foot shock (1.0–1.5 mA), as shown in FIGURE 4. It consisted of a start box, choice chamber, and goal box. The floor of the start box and choice chamber was a metal grid, whereas the goal box was constructed of wood. Two windows, 14.0 × 14.0 cm, at the far end of the choice chamber, provided the only means by which the animal could enter the goal box. A pair of gray cards mounted on wooded blocks was used in preliminary training. The stimuli for the visual discrimination problem consisted of a white card and a black card.

The *three-cul maze* is shown in FIGURE 5. It was designed for the use of the motive of escape-avoidance of mild footshock. The start box and maze proper contained a grid floor, while the goal box floor was made of plywood. The true path, which measured 120.5 cm from the start box exit to the goal box entrance,

TABLE 3. Test Battery Employed by Thompson in Study of Original Learning in 575 Animals

Test	Measure	Motive
Detour A	Errors/latency	Food/water
Detour B	Errors/latency	Food/water
Detour C	Errors/latency	Food/water
Black-white discrimination	Errors/shocks	Escape/avoid shock
Three-cul maze	Errors/shocks	Escape/avoid shock
Inclined-plane	Errors/shocks	Escape/avoid shock

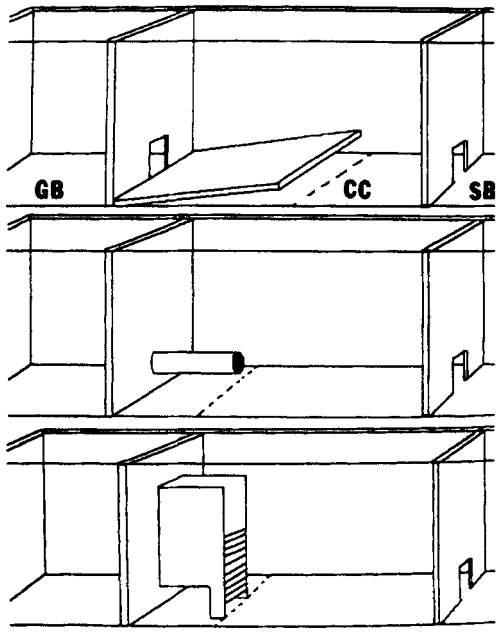

FIGURE 3. Three climbing detour tests. The interrupted lines indicate "blind alleys" (errors). In the problem shown in the *top* panel (Problem A), animal is required to climb from the choice chamber floor up onto the platform in order to reach the window to the goal box; in the problem shown in the *middle* (Problem B), the animal is required to climb into the hollow tube and crawl through it into the goal box; and in the problem in the *bottom* (Problem C) the animal is required to climb the ladder to reach a platform with a window into the goal box. SB = start box; CC = choice chamber; GB = goal box.

consisted of a 90° turn to the left, a 180° turn to the right, followed by a 90° turn to the left. The entire apparatus was painted flat black, except for the grid floor and clear Plexiglas lid.

The *inclined-plane problem* involved the use of the single unit T maze (FIG. 6), adapted for the use of the motive of escape-avoidance of mild foot shock. The stem of the T served as the start box and the left and right arms constituted the choice chamber. At the end of each arm was a window through which the rat could enter the end box by pushing aside a black card mounted on a wooden block. The stem and arms of the T had grid floors; each end box floor was made of wood. The entire apparatus was secured to a platform mounted on a dowel, so that one arm could be tilted 11° vertically in relation to the other. The animal's task was to avoid foot shock by seeking the more elevated end box.

At the conclusion of postoperative training, each brain-damaged animal was killed with an overdose of chloral hydrate, its vascular system perfused with normal saline followed by 10% formalin, and the brain removed and stored in 10% formalin for 2–4 days. Each lesioned brain was blocked, frozen, sectioned

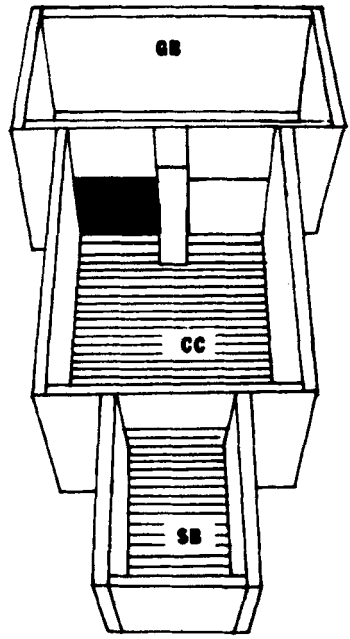

FIGURE 4. Two-choice Thompson-Bryant discrimination box, consisting of start box (SB), choice chamber (CC), and goal box (GB). Two windows, at the far end of the choice chamber (one covered by a black card, the other by a white card), provide the only means by which the animal can enter the goal box.

frontally, and photographed using the section as a negative film in an enlarger. For cortical lesions, the percentage of tissue loss was determined by Lashley's procedure.[51]

Results and Analysis

Brain Lesion Syndromes

On each test, the performance of each lesion group was contrasted with that of the sham-operated controls. If the error score for the lesion group was significantly larger than that of the control group ($p < 0.05$, Mann-Whitney U test, two-tailed), then that specific learning deficit was included within the lesion syndrome. Replicating previous studies, Thompson found that only eight structures were characterized by significant losses on all problems: (1) substantia nigra, (2) superior colliculus, (3) median raphe, (4) ventral tegmentum, (5) ventrolateral thalamus, (6) pontine reticular formation, (7) caudatoputamen, and (8) globus pallidus—the *Nsp.* By a subtractive process, sets of brain structures

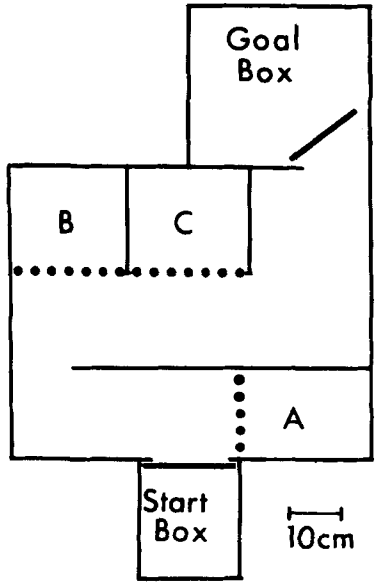

FIGURE 5. Three-cul maze designed for motive of escape-avoidance of mild foot shock. The start box and maze proper contained a grid floor, which could be electrified, while the goal box floor was made of wood. Segments A, B, and C, beyond heavy dotted lines, are blind alleys. Heavy solid line at goal box entrance indicates goal box door.

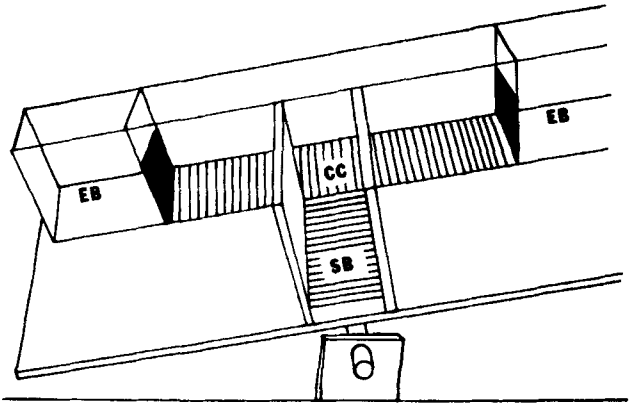

FIGURE 6. Single-unit T maze adapted for motive of escape-avoidance of mild foot shock. The stem of the T serves as the start box (SB), and the left and right arms constitute the choice chamber (CC). At the end of each chamber is a window leading to the end box (EB), reached by pushing aside a card mounted on a wooden block. The stem and arms have a grid floor, while the end box has a wooden floor. The entire apparatus is secured to a platform mounted on a dowel, so that one arm can be tilted 11° above the other (figure shows right arm tilted upwards).

critical for performance of one problem-solving test, but not others, were also identified (i.e., specific mechanisms). All identified mechanisms, including *Nsp,* are listed in TABLE 4. For purposes of the later discussion, it should be noted that the simpler discrimination tests relied on fewer of these mechanisms, while the more complex detour and maze tests appeared to rely on several mechanisms, as shown in TABLE 5.

Structure of Performance

At this point, Thompson's analysis turned to the relationship between the *Nsp* and intelligence, as exemplified by Spearman's *g.*

Analysis of the Control Group. As a first step, the data from 75 sham-operated animals were considered. Error scores on the four tests were intercorrelated, and the resulting correlation matrix shown in TABLE 6. It was apparent that there was negligible intraindividual consistency in performance from one task to another. Thus, one of the prerequisites for assuming the existence of a general or *g* factor (i.e., significant positive correlations among all tests) was absent. This result was expected, as consistent individual differences among albino rats do not emerge without the imposition of brain lesions.

Analysis of Brain-Damaged and Control Rats. The data from the 75 sham-operated animals were combined with data from 349 lesioned animals (151 of the original 500 lesioned animals either died before testing could be completed [n = 38] or had lesions which proved to be asymmetrical on histological examination [n = 113]). TABLE 6 is a correlation matrix for the four variables.

Inspection of TABLE 6 reveals a different degree of intraindividual consistency from that seen in TABLE 5. First, the correlations were all positive. Second, half of them were statistically significant. Finally, a sizeable portion of the variance in maze performance could be accounted for by detour performance. These findings warranted the follow-up using the technique initially developed by Spearman, factor analysis. The procedure used, key cluster analysis, was selected because the underlying principle, domain sampling, made it unnecessary to rely on many of the restrictive assumptions of conventional factor analysis.[54,55]

The analysis resulted in the extraction of two factors, together accounting for 100% of the total communality (i.e., common factor variance). It was apparent that the first factor, which accounted for 95% of the variance, was a general factor on which all tests had significant positive loadings, as seen in TABLE 7. Thus, there was a robust first factor which accounted for most of the variability in an assorted set of problem solving tests, presumably the *g* factor that Spearman and others had identified.[18,52,53]

Construct Validity. In addition to psychometric evidence supporting this position, the factor-loading matrix provided evidence that the first factor was conceptually similar to Spearman's *g.* The strongest evidence for this conclusion stemmed from the fact that the test with the highest factor-loading (.76) was the maze problem. Such tests are generally reported to have higher *g* loadings than simpler sensory discrimination or reaction-time tasks,[49,56] and further re-

TABLE 4. Brain Structures Comprising the Specific Problem-solving Mechanisms and the Nonspecific Mechanisms *(Nsp)*, as Indicated by Lesion Syndromes

Vestibular-kinesthetic-proprioceptive	Visual discrimination	Place-Learning	Motor learning[a]	Visuospatial-attentional	Inhibition	Nonspecific
Anterior cing.	Eyes	Olfactory bulbs	Dorsal frontal c.	Anterior pretectal n.	Amygdala	Substantia nigra
Posterior cing.	Occipital cortex	Amygdala	Anterior cing.	Parietal c.	Dorsal hippo.	Ventrolateral th.
Cerebellum	Entorhinosubicular n.	Ventral frontal c.	Centrolateral th.	Central gray		Globus pallidus
Septofornix	Entopedunuclar n.	Ventral caudate	Lateral th.			Superior colliculus
Ventral hippo.	Anterior hypoth.	Inferior coll.	Mediodorsal th.			Dorsal caudatoputamen
Anterior th.	Subthalamus	Anterior hypoth.	Ventromedial th.			Median raphe n.
Centrolateral th.	Posterolateral hypoth.	Rostral caudate	Parafascicular th.			Pontine reticular form.
Parafascicular th.	Dorsal raphe n.	Ventromedial th.				Ventral tegmentum
Lateral th.	Ventrobasal th.					
Mediodorsal th.						
Ventromedial th.						
Median pretectal n.						

ABBREVIATIONS: cing. = cingulate gyrus; hippo. = hippocampus; th. = thalamus; n. = nucleus; hypoth. = hypothalamus; c. = cortex; coll. = colliculus; form. = formation.

[a]Structures in the motor learning mechanism identified in Thompson, Gallardo, and Yu.[30,31]

TABLE 5. Participation of Specific and Nonspecific Mechanisms in Five Problem-Solving Tests

	Mechanism						
Test	Nonspecific	Vestibular	Visual Discrimination	Place-Learning	Visual attentional	Inhibition	Motor learning
Detours	++	++	++	++	++	++	
Mazes	++	++	++	++	++	++	
Visual discrimination	++		++		++		
Inclined-plane	++	++			++		
Latch boxes[a]	++	++					++

[a]Latch box problem information from Thompson, Gallardo and Yu.[30,31]

view of the correlation matrix indicates that the two sensory discrimination tests had considerably lower factor-loadings.

Competing Factor Interpretations. At this point, Thompson began to consider competing theories which had been advanced to account for the fact that some tests, such as mazes, tend to have high *g* loadings, while others, like sensory discriminations, do not. The most parsimonious explanation was that maze tests simultaneously sample more basic mechanisms than do the simpler discrimination problems. This view had been advanced some years ago by Sir Godfrey Thomson, and was more recently reintroduced in an elegant systems theory analysis by Detterman.[57,58] For Detterman, there would be nothing special about maze problems other than their *wholeness,* operationally defined as the first unrotated principal component (factor)—what others would call *g.* Detterman, unlike Spearman, attributed the inordinate strength, or wholeness, of

TABLE 6. Correlation Matrices for 75 Sham-operated Animals (Below Diagonal) And for 75 Sham-operated Animals Combined With 349 Lesioned Animals (Total N = 424; Above Diagonal) on Four Performance Measures[a]

Test	1	2	3	4
1. Detours	—	**.12**	**.49**	**.11**
2. Black-white discrimination	−.04	—	**.25**	**.16**
3. Maze	.08	−.09	—	**.25**
4. Inclined-plane	.01	.04	.08	—

[a]For N = 75, with four variables, the following significance levels apply:
$p < .05$; $r = .304$
$p < .01$; $r = .362$
For N = 424, with four variables, the following significance levels apply:
$p < .05$; $r = .139$
$p < .01$; $r = .167$

TABLE 7. Loadings for Four Performance Tests on Two Factors Derived from Key Cluster Analysis

Test	Factor I	Factor II	Communality
Maze	.76	−.49	.52
Detours	.63	−.72	.35
Inclined-plane	.33	−.04	.14
Black-white discrimination	.33	−.04	.14

g to the degree of interrelationship between parts of the entire system (here, the sum total of behavioral variation present in four problem-solving tests). Detterman took the position that if tests were selected so that each sampled only a single element of the subject's response repertoire (here, a single specific mechanism), essentially zero intercorrelations would be found, and *g* would not emerge. As seen in TABLE 5, the maze problem did, in fact, sample mechanisms which were not necessary for the visual discrimination task (response inhibition, place learning, and vestibular proprioceptive discrimination) or the inclined-plane problem (response inhibition, place learning, and visual discrimination).

Centrality, as opposed to wholeness, was defined by Detterman as the extent to which a single variable determines system operation. The critical question, it then seemed, was whether the maze problem had high loadings on the first factor because it measured something special (centrality) or simply sampled a number of interrelated performance elements (wholeness). A definitive answer had been eluding factor analysts for half a century.

Limitations of Factorial Evidence. While a number of specific mechanisms appeared to mediate performance on tests with high *g* loadings, there was also one mechanism, response inhibition, which was unique to maze (and detour) performance. Hence, Detterman's theory (that is, *g* as an amalgam of elementary processes) could not be discounted, but neither could the notion that one specific mechanism was *central* to tests with high *g* loadings. There existed the possibility that the response inhibition mechanism might dramatically drive up the *g* loadings of tests where it was a required element. Perhaps response inhibition could be considered a metacomponent, having a superordinate role in the guidance of lesser systems.[59]

Lesion Evidence

In order for psychometric *g* to emerge, variability in performance was experimentally induced with lesions. This fact is amply illustrated by FIGURE 7, showing separate frequency distributions for lesioned and unlesioned animals on the *g* factor. Since no more than a few (usually 4 to 6) of the 349 lesioned animals had experienced lesions in any one of the 49 different lesions, but half

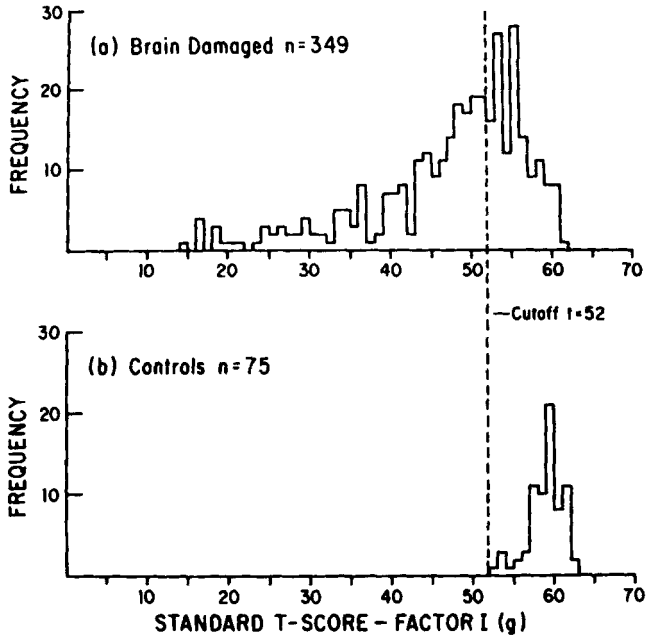

FIGURE 7. *Top:* Frequency distribution of standard T scores (mean = 50; SD = 10) on the *g* factor for 349 rats lesioned at approximately 21 days of age in one of 50 brain sites. *Bottom:* Frequency distribution for 75 sham-operated animals.

the animals showed poorer performance than any control, it is obvious that any number of lesion types resulted in lowered scores on the *g* factor. The critical question for Thompson was whether or not the most destructive of the lesions which lowered scores on psychometric *g* would be those to the structures of the *Nsp.*

Using a form of "extension analysis," in which external variables are defined in terms of factorial space, Thompson conducted another analysis.[60] Employing a "sleeper" program so that the 49 brain lesions, now treated as dependent variables, did not influence the factor structure, a factor loading for each lesion was determined. Inspection of TABLE 8 reveals several noteworthy findings. First, the majority of lesions had no significant loadings on the *g* factor. Second, many loadings tended to emphasize non-traditional roles for certain brain structures. For example, lesions of the superior colliculus and olfactory bulbs seemed to have a significant effect on *g* factor scores. Third, as Thompson had hypothesized, only a few structures seemed implicated in the highest forms of problem-solving behavior in the rodent, as represented by the *g* factor.

The Nsp *and* g. It would have made for a tidy story had the structures composing the *Nsp* been identical to those with high loadings on psychometric *g*. To Thompson's disappointment, only two of the eight *Nsp* structures, the

TABLE 8. Loadings for Lesion Areas on the Psychometric *g* Factor, Showing All Structures with Loadings Greater than +.10

Area of Lesion	Loading
Posterolateral hypothalamus	.31
Parietal cortex	.26
Superior colliculus	.22
Dorsal hippocampus	.21
Occipitotemporal cortex	.21
Posterior cingulate cortex	.21
Olfactory bulbs	.18
Mammilary bodies	.18
Anterior thalamus	.18
Ventromedial thalamus	.11
Median raphe nuclei	.11
Anterior cingulate cortex	.10
Septofornix	.10

superior colliculus and the median raphe nuclei, had a significant loading on psychometric *g*. The remaining six structures of the *Nsp* (i.e., ventral tegmentum, dorsal caudatoputamen, globus pallidus, substantia nigra, ventrolateral thalamus and pontine reticular formation) did not emerge as significant external correlates of *g*. On the contrary, these structures were characterized by rather minimal *g* loadings (< +.05; see TABLE 9).

DISCUSSION

Thompson's disappointment was short-lived. He was fond of Mencken's dictum: "For every complex problem there is a simple answer. And it is wrong." A clear correspondence between the *Nsp* and psychometric *g* would have

TABLE 9. Major Factor by Lesion Findings for Structures Comprising the Nonspecific Mechanism

Area of Lesion	Loading
Superior colliculus	.22
Median raphe nuclei	.11
Ventrolateral thalamus	.03
Substantia nigra	.02
Pontine reticular formation	.01
Dorsal caudatoputamen	−.01
Ventral tegmental area	−.09
Globus pallidus	−.10

constituted such a simple answer, and he probably would have intuitively distrusted it in a short while. He was satisfied with the more complicated state of affairs demonstrated by this experiment, which to him seemed more trustworthy. He felt convinced that these results would probably, in the long run, contribute more to the understanding of the biological basis of intelligence than his original supposition that psychometric *g* and the *Nsp* were the same. But how?

Psychometric g

In considering this complex outcome, Thompson was confident of at least one result—a factor psychometrically equivalent to the *g* found in human performance was also present in rat performance (rendered heterogeneous by lesions). As a corollary, scores on the *g* factor were depressed by lesions to several, but not all, brain sites. This finding supported a "functional system" view of *g,* rather than a *g* that reflected some property of the whole brain, as suggested by Spearman.

In the absence of whole-brain involvement, the essence of Spearman's *g*—a biologically based common core—could only be preserved if the brain structures critical for psychometric *g* were important for *all tasks.* But this was decidedly not the case. For example, lesions to the posterolateral hypothalamus had no significant effect on the inclined-plane problem; lesions to the dorsal hippocampus did not appear to affect black-white or inclined-plane discrimination; and, lesions to the occipitotemporal cortex failed to produce defects on two of the three detour problems. Thus, while certain brain structures had noteworthy loadings on the *g* factor, none of them had proven to be critical for all performances.

What, then, of the response inhibition mechanism, which seemed so *central* to tests with high *g* loadings but not to tests with low *g* loadings? It turned out that response inhibition was not as important in determining the *g* loadings of the detour and maze tests as was the absolute number of problem-solving mechanisms. That is, while inhibition was a *necessary* element in maze performance, it proved to be *insufficient* in itself to account for the high *g* loadings of the test. To support this conclusion, Thompson compared the 13 neural structures that might conceivably be thought of as having significant *g* loadings (i.e., > +.10; refer again to TABLE 8) with the neural structures comprising the various problem solving mechanisms (TABLE 4). He found that structures critical for each of the five specific mechanisms, as well as the *Nsp,* were represented: (1) visuospatial attentional mechanism (parietal cortex); (2) visual discrimination mechanism (occipitotemporal cortex and posterolateral hypothalamus); (3) vestibular-proprioceptive-kinesthetic discrimination mechanism (posterior cingulate cortex, anterior cingulate cortex, and anterior thalamus); (4) place-learning mechanism (olfactory bulbs and ventromedial thalamus); and (5) response inhibition (dorsal hippocampus); and (6) *Nsp*—superior colliculus and median raphe. That is, high *g* loading tests cannot be easily learned without the participation of brain structures that mediate at least six mechanisms, and

perhaps more. (It should be noted that the structures listed in TABLE 8 are known to participate in other specific mechanisms not mentioned in this particular study, e.g., motor learning mechanism—anterior cingulate cortex; motivational system—posterolateral hypothalamus and anterior thalamus; olfactory discrimination—olfactory bulbs; etc.) Of course, this is precisely what one would predict from Detterman's theory of psychometric *g:* a high *g* loading means that more systems are sampled by the test. FIGURE 8 is a graphic representation of this particular view of psychometric *g,* adapted to the results of Thompson's experiment.

Centrality within Neural Mechanisms. Next, Thompson wondered why, if the simultaneous sampling of six neural problem-solving mechanisms had been responsible for the high *g* loadings of the maze test, do not all brain structures within each mechanism have high *g* loadings? The answer was simple. The 13 brain structures with high *g* loadings were simply more critical for the preservation of the mechanism than were other structures within the mechanism (i.e., more *central* to that system). Under this interpretation, for example, the most critical structure for the *Nsp* would be the superior colliculus, for the place-learning mechanism, the dorsal hippocampus, and so forth.

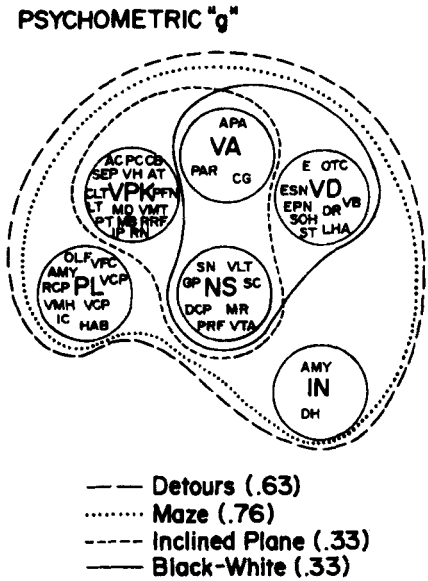

FIGURE 8. Domain sampling characteristics of four tests (represented by various broken lines) that lead to differential loadings on the *g* factor. Legend: IN = inhibition; NS = nonspecific mechanism; PL = place learning mechanism; VA = visuospatial attentional mechanism; VD = visual discrimination mechanism; VPK = vestibular-proprioceptive-kinesthetic discrimination mechanism. For brain structures signified by abbreviations, see TABLE 2.

Nonspecific Mechanism and Spearman's g

Returning to the relationship of the *Nsp* to Spearman's *g*, two facts were apparent: (1) The *Nsp* was necessary for all problem-solving tasks; and (2) a lesion to any one of eight structures within the *Nsp* was sufficient to significantly impair performance. Thus, whether a combination of three, four, or five mechanisms was required for a particular test performance, the *Nsp* would always be one of the ensemble. The simple logic of this position is illustrated in FIGURE 9. In the segment of the Venn diagram common to all tests, the *Nsp* is found.

External Correlates of g *and* Nsp. In order to better compare and contrast psychometric *g* and the *Nsp*, which by now had revealed themselves as distinctive constructs, Thompson referred to their external correlates. What predictions might be made from knowing the status of a subject with regard to either of them? First and foremost, psychometric *g* has long been known to correlate significantly with academic success. The accumulation of items such as analogies, matrices and other complex reasoning tasks with high *g* loadings in tests such as the Stanford Binet Intelligence Scale was primarily due to the fact that tests comprising such items tended to predict success in school. Perhaps then, if one wanted to predict the equivalent of "future academic performance" in the white rat, it would be important to have a battery composed of tests (such as detours and mazes) that had high *g* loadings.

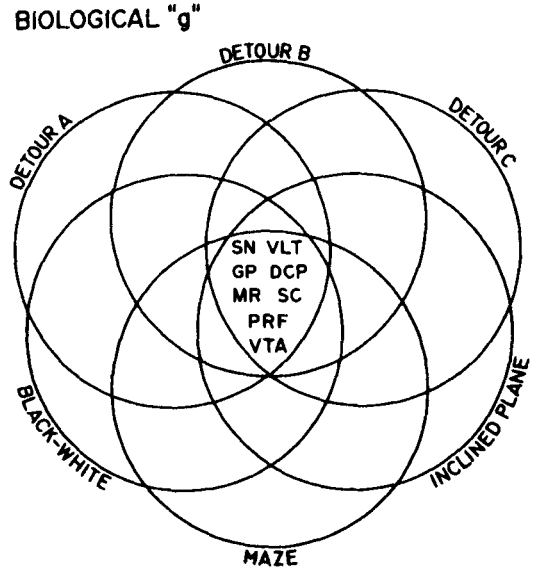

FIGURE 9. Schematic conceptualization of the nonspecific mechanism. Each circle represents a performance test; letters at the center of the diagram are abbreviations for brain structures in the nonspecific mechanism, contained in the area common to all six tests. For brain structures signified by abbreviations, see TABLE 2.

However, Thompson reasoned that such complex performances may not typify the day-to-day world of the white rat, which arguably requires more in the way of continual adaptations to simple environmental demands. In that respect, it had been shown that learning a representative sample of these adaptations—Thompson's battery of animal performance tests—all relied on the *Nsp.* Thus, while the psychometric *g*-deficient animal may be impaired in the rat equivalent of an IQ test (e.g., a maze problem), its survival might be more threatened by loss of the *Nsp.* This would seem to make for a compelling argument in favor of the designation of the *Nsp* as "biological *g.*"

CONCLUSIONS

In developing his animal model of mental retardation, Robert Thompson reencountered two of the oldest and most enduring controversies in psychology: (1) the unity versus the particularity of intelligence and (2) the mass-action versus the localizationist view of brain function. With respect to the first issue, he found evidence that Spearman was correct in positing a special trait underlying all cognitive performance, but incorrect in assuming that the behavioral manifestations of such a trait could be isolated via factor analysis. No sooner was a psychometric *g* factor identified than was its componential, rather than unitary, basis established. Spearman, in the company of Lashley and others, was also incorrect in assuming that *g,* in a biological sense, was a function of the whole brain. The *Nsp,* arguably *biological g,* was not an expression of whole-brain activity, but rather the integrated operation of eight circumscribed structures. Thompson's findings thus pose a problem for those who would subscribe to the position that the *g* derived from factorization of psychometric tests will ultimately converge on a pure biologic counterpart.

With respect to the second issue, mass-action views of brain function had gone out of vogue well before Lashley's death in 1958, and Thompson, who spent his entire career cataloging the effects of brain lesions, would have been the last to contend that biological *g,* that is, the *Nsp,* was a whole-brain function. But the story was somewhat different for psychometric *g,* since tests with high *g* loadings necessitated the operation of many parts of the brain. Thompson viewed the *Nsp,* which consisted of basal ganglia and closely related structures, as exerting an organizing influence on the various specific brain modules that might be tapped in high *g*-loaded tasks. Under such conditions, Thompson was reluctant to discount totally the possibility that the entire brain was also secondarily involved, perhaps in providing a general background or tonus—one version of the mass action position. But he consistently countered any strong mass action argument with evidence that lesions to many brain areas, including rather large ones, produced no significant performance decrements whatsoever.

The analogy Thompson often used when referring to the *Nsp* was that it was like the conductor of a symphony orchestra, who played no instrument (specific function) but ensured that the performance of members of the orchestra were properly integrated. Depending on the composition, some instruments might be missed more than others, and for particularly complex scores (like high *g*-loaded

tasks), the performance could be devastated by the absence (or ill-timed entry) of any number of the instruments. On the other hand, if the conductor, who played no instrument (like the *Nsp*) were not present, the presence of all the individual players would be insufficient to ensure that the composition would be recognized.

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