Psychological Review
1994, Vol. 101, No. 1, 185-187

0033-295X/94/$3.00

Priming and Theories of Memory: A Reply to Ratcliff and McKoon

Timothy P. McNamara

Ratcliff and McKoon (1994) attempt to buttress the case for non-spreading-activation models of associative priming by showing that first-response free association probabilities do not predict priming effects, and sequential effects in lexical decisions are predicted by at least one non-spreading-activation model. The author argues that their attempt to predict priming from free association is not informative because they did not propose a model of how association in memory is manifested in free association, these predictions depended on assumptions that are not consistent with the model tested, the compound-cue model is a poor model of sequential effects, and non-spreading-activation models still cannot explain the absence of inhibition following nonword primes when responses to the primes are not required.

Ratcliff and McKoon (1994) present two major arguments in their article: First, they show that first-response free-association probabilities do not predict priming effects. This finding leads them to conclude that free association is an invalid index of association in memory, which implies that mediated priming may be direct priming between weakly associated concepts. Second, they attempt to show that the sequential effects that I have documented in lexical decisions (McNamara, 1992a, 1992b) can be explained by a non-spreading-activation model of priming. In this article, I respond to these arguments and discuss a number of theoretical issues that may be getting lost in the fray.[1]

"Spreading-Activation Theories Versus Compound-Cue Theories" Is a False Dichotomy

The compound-cue model is a hypothesis about the content of retrieval cues and, by itself, does not predict anything about performance in tests of memory. Ratcliff and McKoon (1988) combined the compound-cue model with models of memory that do not rely on spreading-activation mechanisms (e.g., Gillund & Shiffrin, 1984; Hintzman, 1986; Murdock, 1982), producing what I have called, for lack of a more imaginative term, *non-spreading-activation* models of priming. Of course, a compound-cue model could be combined with a spreading-activation model of memory. Indeed, in ACT (Adaptive Control of Thought; Anderson, 1983), priming occurs because the prime and the target are both sources of activation; in other words, the prime and the target are both "cues" to memory. The construction-integration model of text comprehension (Kintsch, 1988) also combines spreading-activation mechanisms with a cue-based knowledge retrieval process.

This point is not a pedantic clarification of terminology: It implies that investigators are not forced to choose between the advantages of an activation-based memory system and the appeal of integrative combination of cues at the time of retrieval. The point that I tried to make in my article (McNamara, 1992a) is that the joint combination of the compound-cue model (as described by Ratcliff & McKoon, 1988; cf. Dosher & Rosedale, 1989) with certain models of memory does not provide a compelling account of associative priming in lexical decisions, recognition, and naming.

Spreading Activation Is Not Wedded to Localist Theories of Memory

I have observed in conversations and articles a reflexive association between spreading-activation and localist theories of memory. It is true that spreading-activation mechanisms were originally proposed as part of localist theories of memory (e.g., Quillian, 1967). However, they also can be combined with distributed models. Dell's (1986) model of speech production, for example, uses distributed representations and spreading activation as a retrieval mechanism.

Spreading activation can be thought of as a type of pattern completion: The presentation of a concept partially instantiates concepts with which it shares associative relations, semantic features, or both. Framed in this way, similarities between spreading-activation processes and many distributed models are more apparent. The difference between spreading-activation and many other pattern-completion algorithms is that spread of activation causes partial instantiation of those concepts that share features with partially instantiated concepts. The decay rate of activation is a passive constraint on the number of concepts activated.

Quantitative Fits of Models to Priming and Free-Association Probabilities Are Not Informative in the Absence of Explicit Models of Free Association

If someone had told me 6 years ago that the status of mediated priming would depend on whether free associations should be

Preparation of this article was supported by National Science Foundation Grant DBS 9222002.

Correspondence concerning this article should be addressed to Timothy P. McNamara, Department of Psychology, 301 Wilson Hall, Vanderbilt University, Nashville, Tennessee 37240.

[1] Several of the arguments presented by Ratcliff and McKoon (1994) are similar to arguments published elsewhere (McKoon & Ratcliff, 1992) and have been discussed in McNamara (in press).

collected using a *first-response* or a *continued-association* method, I would have considered abandoning the entire enterprise. Happily, I can make my point without wading too deeply.

Ratcliff and McKoon (1994) show that if associative strength is measured using first-response probabilities, ACT predicts that virtually no priming should occur for nonmediated stimuli (MR pairs, named for *M*cKoon & *R*atcliff, 1992). It should be noted, however, that if free-association probabilities are measured using the continued-association procedure, ACT predicts a priming effect of between 8 and 13 ms for nonmediated pairs, depending on details of the implementation (McNamara, 1993; the observed value was 13 ± 6 ms). How is one to know which test of the model is correct?

Although Ratcliff and McKoon (1994) and I could argue back and forth about the relative merits of these methods of collecting free-association responses, one inescapable fact remains: The choice of any method is an implicit theoretical claim. Different models of free association will lead to different choices of measures. Ratcliff and McKoon do not propose a model of free association in the context of a spreading-activation model or a non–spreading-activation model, so there is no way to know what technique of assessing associative strength is appropriate.

I add that Ratcliff and McKoon's (1994) major criticism of the continued-association procedure, that early responses may influence latter ones, may be a virtue. In a priming paradigm, the functional associations are those activated or created by the appearance of the target in the context of one of its associates, the prime. These associations must be more similar to those activated by the prime and its associates or the target and its associates than to those activated by the prime or the target alone.

Ratcliff and McKoon's Attempt to Predict Priming From Free-Association Probabilities Is Not Consistent With the Assumptions of ACT

Ratcliff and McKoon (1994) set the strength of association from node i to node j, r_{ij}, to be equal to the probability of recalling j given i as a cue, $P(j \mid i)$. To my knowledge, free association has not been implemented in a spreading-activation model. A natural implementation, however, is for $P(j \mid i)$ to be a function of the activation level of j when i is a source of activation (as in cued recall; see Anderson, 1983). In ACT, activation level is a function of node strength and the structure of the network. It is easy to construct ACT networks in which the associative strength from a cue word to each of two other words is identical, but the activation levels differ when the cue is a source of activation. Indeed, this will happen any time the two associates differ in the number of associates ("fan") they have. It is even possible for $r_{ij} > r_{ik}$ but for the activation of k to exceed that of j when i is a source of activation. In sum, Ratcliff and McKoon's demonstration that free association does not predict priming is based on a faulty implementation of ACT.[2]

Free Association Isn't Perfect, But It Isn't Necessarily Useless Either

These considerations make it sound as though free association is totally worthless as a measure of association in memory, which seems to be the conclusion that Ratcliff and McKoon (1994) want us to accept. A less radical conclusion, however, is also justified. These problems arise because of the way Ratcliff and McKoon use free association. Other uses make weaker assumptions. For example, the use of distance effects to test non–spreading-activation models assumes that directly associated items will appear as mutual associates in free association; equivalently, if neither the prime nor the target appears as an associate of the other, then they are not directly associated. Because these models require direct association (or in the case of SAM [search of associative memory], one indirect associate) to predict priming, the presence of priming without association is a problem (modulo concerns about power and sensitivity; see McNamara, 1992a).

I want to add here that "pure" semantic priming (e.g., Seidenberg, Waters, Sanders, & Langer, 1984) is not embarrassing to spreading-activation models or to the use of free association. The Collins and Loftus (1975) model, for example, contained lexical and semantic networks. Associative distance can still be defined as well. The task is to construct chains of associates based on free association such that primes and targets are not semantically related. Many of the three-step primes and targets used by McNamara (1992b) may meet this criterion (although a diligent critic may find semantic relations that I missed).

Compound-Cue Mechanisms Are Poor Models of Sequential Effects

The fits of SAM to the data on sequential effects (Ratcliff & McKoon, 1994, Table 4) look good because the model appears to explain with a single mechanism the facilitative effects of related primes and the inhibitory effects of nonwords. This analysis assumes, however, that nonwords are included in compound cues and that sequential effects are adequately explained by the compound-cue model. I have shown in another article that the compound-cue model is a poor model of sequential effects (McNamara, in press). In particular, a review of the literature shows that nonwords produce inhibition on subsequently appearing word targets when subjects respond to the nonwords (e.g., McNamara, 1992b) but not when they do not respond to the nonwords (e.g., Borowsky & Besner, 1993;

[2] On a more technical note, the predicted activation levels in Ratcliff and McKoon's (1994) Table 1 result from two assumptions that cannot both hold in an ACT network: (a) that the strengths of primes and targets are equal and (b) that the associative strengths have the values given in Ratcliff and McKoon's Figure 1. In ACT, $r_{jk}/r_{ji} = s_k/s_i$. For example, in Figure 1, .114/.154 = .74, which means that the target must be .74 times the strength of the prime for MA pairs (named for *M*cNamara & *A*ltarriba, 1988); the same ratio is .048/.115 = .42 for MR pairs. Because Ratcliff and McKoon's networks are internally inconsistent, the absolute levels of activation in Table 1 are not interpretable, and the predicted priming effects within networks (differences in activation levels) correspond to letting target strengths vary across networks (McNamara, 1993). Ratcliff and McKoon's claim in the Appendix that in a dense network it is impossible to obtain node strengths to satisfy all link strengths is true but irrelevant. Node strength is the fundamental construct in ACT; associative strength is derived from node strength. Moreover, as I point out in this article, it is inappropriate to set associative strengths in the manner used by Ratcliff and McKoon.

McNamara, in press). Exactly the same pattern of results holds in item recognition tasks. In short, sequential effects of this kind are a response or decision effect, not a memory retrieval effect. At this point, one may be tempted to suggest that non–spreading-activation models could account for sequential effects in the same way as spreading-activation models. They can: The problem is that non–spreading-activation models predict effects of nonwords after the sequential effects have been removed, but none is observed (McNamara, 1992b).

Non–Spreading-Activation Models Do Not Have a Compelling Explanation of the Absence of Inhibition Following Nonword Primes

One line of evidence that elicits no comment from Ratcliff and McKoon (except for Footnote 4; 1994) is the absence of inhibition on word targets following nonword primes when a response to the prime is not required. Inhibition of this kind is a straightforward prediction of non–spreading-activation models, and yet five studies in two independent laboratories have failed to find any evidence of it (Borowsky & Besner, 1993; McNamara, 1992a, in press).

Summary

Given the complexities of the models and of the debate, it may help to end by summarizing points on which Ratcliff and McKoon (1994) and I agree and disagree.

We agree that (a) automatic priming is informative about basic retrieval mechanisms, (b) priming is caused by associative relations in memory, (c) multiple cues may be effective at the time of retrieval, (d) McKoon and Ratcliff's (1992) nonmediated stimuli are connected by associative pathways, and (e) non–spreading-activation models predict facilitation on post-target words and inhibition on words following nonwords or foils.

However, we disagree on (a) the mechanisms by which association in memory produces priming, (b) whether it is appropriate to use free-association probabilities to set associative strengths in ACT networks, (c) whether the assumptions required by non–spreading-activation models to explain sequential effects and the benign effects of nonword primes are reasonable and consistent with extant data, and (d) whether data on naming should be included in the debate.

As I stated at the end of my article (McNamara, 1992a), progress in modeling cognitive phenomena depends on the broad view of data and theory that is forced by the use of converging operations (Garner, Hake, & Eriksen, 1956). As the models become more sophisticated and the database more extensive, we should begin to see closure on this debate. Until then, I see much to gain by including compound-cue-like mechanisms in models of the functional stimulus but much to lose by excluding spreading-activation processes from theories of memory and retrieval.

References

Anderson, J. R. (1983). *The architecture of cognition.* Cambridge, MA: Harvard University Press.

Borowsky, R., & Besner, D. (1993). Visual word recognition: A multistage activation model. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 19,* 813–840.

Collins, A. M., & Loftus, E. F. (1975). A spreading-activation theory of semantic processing. *Psychological Review, 82,* 407–428.

Dell, G. S. (1986). A spreading-activation theory of retrieval in sentence production. *Psychological Review, 93,* 283–321.

Dosher, B. A., & Rosedale, G. (1989). Integrated retrieval cues as a mechanism for priming in retrieval from memory. *Journal of Experimental Psychology: General, 118,* 191–211.

Garner, W. R., Hake, H. W., & Eriksen, C. W. (1956). Operationism and the concept of perception. *Psychological Review, 63,* 149–159.

Gillund, G., & Shiffrin, R. M. (1984). A retrieval model for both recognition and recall. *Psychological Review, 91,* 1–67.

Hintzman, D. L. (1986). "Schema abstraction" in a multiple-trace memory model. *Psychological Review, 93,* 411–428.

Kintsch, W. (1988). The role of knowledge in discourse comprehension: A construction–integration model. *Psychological Review, 95,* 163–182.

McKoon, G., & Ratcliff, R. (1992). Spreading activation versus compound cue accounts of priming: Mediated priming revisited. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 18,* 1155–1172.

McNamara, T. P. (1992a). Priming and constraints it places on theories of memory and retrieval. *Psychological Review, 99,* 650–662.

McNamara, T. P. (1992b). Theories of priming: I. Associative distance and lag. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 18,* 1173–1190.

McNamara, T. P. (1993). *Fits of ACT* to priming and to free association probabilities.* Unpublished manuscript.

McNamara, T. P. (in press). Theories of priming: II. Types of primes. *Journal of Experimental Psychology: Learning, Memory, and Cognition.*

McNamara, T. P., & Altarriba, J. (1988). Depth of spreading activation revisited: Semantic mediated priming occurs in lexical decisions. *Journal of Memory and Language, 27,* 545–559.

Murdock, B. B. (1982). A theory for the storage and retrieval of item and associative information. *Psychological Review, 89,* 609–626.

Quillian, M. R. (1967). Word concepts: A theory and simulation of some basic semantic capabilities. *Behavioral Science, 12,* 410–430.

Ratcliff, R., & McKoon, G. (1988). A retrieval theory of priming in memory. *Psychological Review, 95,* 385–408.

Ratcliff, R., & McKoon, G. (1994). Retrieving information from memory: Spreading-activation theories versus compound-cue theories. *Psychological Review, 101,* 177–184.

Seidenberg, M. S., Waters, G. S., Sanders, M., & Langer, P. (1984). Pre- and postlexical loci of contextual effects on word recognition. *Memory & Cognition, 12,* 315–328.

Received September 1, 1993
Accepted September 13, 1993 ■