# Does Activation Really Spread?

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In this article we consider three versions of the spreading activation model for retrieval of information from memory. Predictions are derived for activation spreading down a linear network structure as a function of time. To test these predictions, two experiments were performed in which facilitation as a function of time was measured for target words in a linearly structured paragraph. A target word was primed either by a word near to the target in the paragraph structure or by a word far from the target. Results showed that facilitation begins at about the same time for the far and near conditions. This is inconsistent with the predictions derived from the models. Implications of these results are discussed and alternative conceptions of the activation process are described.

In this article we discuss the theoretical and empirical status of the spreading activation process. It is important to investigate this process, because it forms the central mechanism of two current models of memory—a model proposed by Anderson (1976) in the areas of memory, language, and thought, and a model proposed by Collins and Loftus (1975) in the area of semantic memory—and because it is becoming accepted as a mechanism for processes involved in text comprehension (Kieras, 1981; Miller, 1981). Spreading activation has also formed the basis of models in artificial intelligence (Fahlman, 1981; Hinton, 1981; Ouillian, 1967), problem solving (Levin, 1976), language understanding (McDonald & Hayes-Roth, 1978), word recognition (McClelland & Rumelhart, 1981), and word production (Dell. 1980; Dell & Reich, 1977). Although we are concerned in this article with spreading activation as a process of retrieving information from memory, and therefore are most directly concerned with the models of Anderson and Collins and Loftus, the discussion presented here should also have implications for the other areas of research.

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Reprint requests should be sent to Roger Ratcliff, Department of Psychology, Yale University, Box 11A Yale Station, New Haven, Connecticut 06520. In the models of Anderson (1976) and Collins and Loftus (1975), memory is assumed to be an interlinked network of nodes. Each node represents a concept, and links between nodes represent relations between concepts. During the retrieval of information from memory, concepts are activated, and activation spreads through the network. When activation from two concepts intersects, the path between the concepts is retrieved; that is, it becomes available to decision and response processes.

The notion that activation spreads usually carries with it two different and empirically testable assumptions. These assumptions can be found in the Anderson and Collins and Loftus models and in most of the other models that employ the spreading activation mechanism (Dell, 1980; McClelland & Rumelhart, 1981; Quillian, 1967). The first of these assumptions is that the amount of activation arriving at any node is a decreasing function of the number of links (the distance) the activation has traversed. The second assumption is that activation takes some significant amount of time to spread between nodes, for example, on the order of 50-100 msec per link (Anderson, 1976). (It should be noted that the assumption of this rate of spread is not universal; for example, Wickelgren, 1976, has assumed that activation spreads at a rate of 1 msec per link.) The main problem with these assumptions about spreading activation is that they have been difficult to test directly. For example, spreading activation models of semantic

memory (Collins & Loftus, 1975; Quillian, 1967) use the assumptions to derive predictions concerning the relationship between the time to verify a statement and the distance between the concepts of the statement in the semantic network. However, the distance between concepts has been found to be confounded with semantic relatedness (Rips, Shoben, & Smith, 1973). So, support for the assumptions of spreading activation has not been convincing in this area.

In contrast, in the area of research concerning the representation of text in memory, the assumptions of spreading activation can be put to a more convincing test. This is because, first, there is a reasonable amount of agreement that the representation of text is propositional and that propositions are linked together in a network structure (Anderson, 1976; Anderson & Bower, 1973; Fredericksen, 1975; Kintsch, 1974; Rumelhart, Lindsay, & Norman, 1972; van Dijk, 1977) and, second, there is available a method for examining the network structure and the processes that operate on it. This method is the priming technique developed by Ratcliff and McKoon (1978; McKoon & Ratcliff, 1980). In a typical experiment, subjects study paragraphs and then are tested with single words for recognition; they are required to hit one key if the test word is from one of the studied paragraphs and another key if the test word is a new word. It has been found that if a test word (target) is preceded in the test list by another test word (prime) that is close in the meaning structure of the paragraph, then response time to the target is decreased (i.e., a priming effect). The size of the priming effect has been found to be a monotonic function of the distance between the prime and target words in the meaning structure of the paragraph. These effects of distance on priming can be explained by the assumption that activation falls off with distance or by the assumption that activation takes time to spread (or by both assumptions in combination), just as the time to verify statements can be explained in the semantic memory area. However, the distance effects on priming. unlike the distance effects on semantic verification, have been shown not to be confounded by preexperimental semantic relatedness (McKoon & Ratcliff, 1980).

In this note we separate the two spreading activation assumptions and examine directly their different predictions. To do this we use the priming technique; specifically, we trace out the time course of the priming effect. The procedure is the same as that described above except that the subject is not required to respond to the prime, only to the target word. To trace the time course, the time between the onset of the prime and the onset of the target (the stimulus onset asynchrony, SOA) is varied. To observe the effect of distance between concepts, the prime and target are either near to each other in the structure of the paragraph (the near priming condition) or far apart (the far priming condition). With these two manipulations, SOA and distance, we can determine whether activation takes time to spread and whether the amount of activation that arrives at a concept is a function of the distance traveled.

## Modeling the Activation Process

In this section, we derive predictions for activation as a function of time and distance from three versions of the spreading activation model. The predictions are derived with respect to the structures of the paragraphs used as materials in the experiments presented below. An example of the paragraphs used in the experiments follows:

The scientist nudged the sheriff. (N1 V N2)
The sheriff stared at the spacecraft. (N2 V N3)

The spacecraft transported an alien. (N3 V N4)

The alien drew a weapon. (N4 V N5)
The weapon vaporized a mountain. (N5 V N6)

In this example, N1 stands for Noun 1, V for verb, and so forth. The representation of this example in ACT notation (Anderson, 1976) is shown in Figure 1. The ACT notation is used because we test predictions of the ACT model, but other models would assume an equivalent structure.

In the priming procedure, a prime is presented and then a target word follows. For

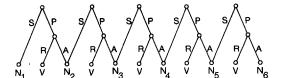


Figure 1. The structure in ACT notation of the sample paragraph displayed in text. (Note that instance-to-concept and word links have been omitted. N = noun, V = verb, S = subject, P = predicate, R = relation, A = argument.)

this procedure, the retrieval assumption made by spreading activation models is that activation spreads from the prime, through the paragraph structure, to the target (e.g., Anderson, 1976). As the target becomes activated, response time to the target is shortened. There are two different conceptions of the spreading activation process: discrete (all-or-none), where it is assumed that a node is either active or not active; and continuous, where it is assumed that a node can have a continuously varying amount of activation. To model the discrete case (e.g., Anderson, 1976), we need to know the probability that activation has spread down a link to activate a neighboring node as a function of time. From this probability function, predictions about activation functions can be made. In the continuous case (Collins & Loftus, 1975; Dell, 1980; Levin, 1976; McClelland, 1979; McClelland & Rumelhart, 1981), the rate at which activation spreads down a link and the function that describes that spread are required. In the following derivations, two probability distributions for the discrete case are considered: the exponential (used by Anderson, 1976) and the normal. These two distributions are at two extremes: The exponential is a highly skewed distribution that allows some processes to be completed in negligible time, whereas the symmetric normal (with small variance) does not allow very rapid completions. Other unimodel distributions that would be reasonable representations of temporal processes would fall between the exponential and the normal. Thus, results derived from the exponential and normal cases can be generalized over a range of possible distributions. In the continuous case, the usual assumption is made: that the rate of change of activation at a node is proportional to the difference in activation levels between that node and adjacent nodes.

### Discrete Model: Exponential Distribution

In a model in which the amount of activation at a node is discrete, a node is not activated until, first, an adjacent node is activated and, second, the activation spreads across the connecting link. The exponential distribution has been used by Anderson (1976; mainly for mathematical tractability) as the distribution of link traverse times. In the experiment presented below, there are two conditions: The prime and target are near each other, separated by only 3 links (e.g., N5 to N6), or the prime and target are far from each other, separated by 9 links (e.g., N2 to N6). If instance-to-concept links (Anderson, 1976, p. 444) were added, then the difference would be even larger: 7 links versus 22 links. To calculate the amount of activation at the target as a function of time, it is necessary to convolve the exponential distributions for each of the links between the prime and the target (3 links for the near condition, 9 for the far condition). To simplify modeling, we assume that the time constant of each exponential distribution is the same (i.e., the rate of spread of activation down each link is the same). The distribution function for the convolution of n exponential distributions, each with time constant  $\tau$ , is given by:

$$F(t) = 1 - \sum_{i=0}^{n-1} \frac{(t/\tau)^i e^{-(t/\tau)}}{i!}.$$
 (1)

The functions for 3 links and 9 links, calculated assuming  $\tau=50$  msec, are shown in Figure 2. The 50-msec value is at the low end of the range of values obtained by Anderson (1976) in fits of ACT to data. It was chosen here because it comes closer to approximating the data obtained in the experiment presented below than higher values. The plots in Figure 2 show that for activation spreading down 3 links, the curve begins to rise above zero activation at about 50 msec and rises quickly, reaching half the asymptotic value a little before 150 msec. For activation spreading down 9 links, the curve

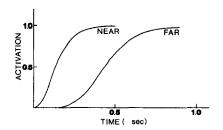


Figure 2. The amount of activation as a function of time for near and far priming conditions predicted by the discrete exponential and continuous spreading activation models. (The time constant for a single link is assumed to be 50 msec in both cases.)

begins to rise above zero activation at about 250 msec and rises rather more slowly than the 3-link curve, reaching half the asymptotic value a little before 450 msec. If instance-to-concept and word links were included, then the near curve would rise to half asymptotic value in 350 msec and the far curve in 1100 msec.

The distribution in Equation 1 is a cumulative distribution function (i.e., is normalized to 1). To produce the function relating amount of priming and SOA, the distribution would be convolved with a distribution representing encoding, decision, and response processes and multiplied by a constant representing the asymptotic amount of activation (perhaps different constants for the near and far conditions). Addition of a distribution representing encoding, decision, and response processes would slow the rate at which the curves rose to their maximum values but would not alter the relative positions or shapes of the curves.

#### Discrete Model: Normal Distribution

If it is assumed that the distribution of the time for activation to spread down each link is normal and that this distribution has relatively small variance relative to the distribution for encoding, decision, and response processes, then it is easy to derive the amount of activation at the target as a function of time. The activation functions for the near and far priming conditions rise to asymptote at the same rate but are shifted relative to each other. The far priming curve is shifted by the difference in number of links (9-3)

multiplied by the mean of the normal distribution. If we assume (as before) that the mean is 50 msec, then the curves are shifted by 300 msec  $[50 \times (9-3)]$ . If the variance of the component activation processes is not negligible, then the far activation curve is still shifted relative to the near curve, but the rate of approach to the asymptote for the far curve will be slowed relative to the rate for the near curve (i.e., the functions will be similar to the exponential case above).

#### Continuous Flow Models

In these models, the amount of activation at a node is assumed to be a continuous variable driven by activation from surrounding nodes in a continuous manner (Dell, 1980; Levin, 1976; McClelland, 1979; McClelland & Rumelhart, 1981). It is often assumed that the rate of change of activation at a node is proportional to the sum of the differences between the activation levels at that node and the surrounding nodes. If the exponential assumption is made (the assumption that the rate of change is directly proportional to the difference), then the amount of activation at a node as a function of time can be derived. For paragraphs like the example displayed above, it can be assumed that a node is activated only by its immediate neighbor; that is, activation passes down the linear structure. Thus.

$$\frac{da_i(t)}{dt} = \frac{1}{\tau} (a_{i-1}(t) - a_i(t)), \qquad (2)$$

where  $\tau$  is the time constant and  $a_i(t)$  is the activation level of node i at time t. (For a more complete treatment of this kind of model, see McClelland, 1979.) To solve Equation 2, we take Laplace transforms of both sides of the equation:

$$sa_i^*(s) - a_i(0) = \frac{1}{\tau} (a_{i-1}^*(s) - a_i^*(s)).$$
 (3)

If the system has no initial activation,  $a_i(0) = 0$ , and Equation 3 can be rewritten as

$$a_i^*(s) = a_{i-1}^*(s) \left(\frac{1}{1+\tau s}\right).$$
 (4)

Rewriting Equation 4 in terms of the trans-

formed activation of the first node in the linear structure gives Equation 5:

$$a_i^*(s) = a_0^*(s) \left(\frac{1}{1+\tau s}\right)^i.$$
 (5)

If we assume that the activation of the first node in the linear structure is a step function beginning at t = 0, then  $a_0^*(s) = 1/s$ . Using a partial fraction expansion, we find that

$$a_i^*(s) = \frac{1}{s} - \tau \sum_{i=1}^n \frac{1}{(1+\tau s)^n}.$$
 (6)

Taking the inverse Laplace transform of Equation 6 gives

$$a_i(t) = 1 - \sum_{i=0}^{n-1} \frac{(t/\tau)^i e^{-t/\tau}}{i!},$$
 (7)

which is identical to Equation 1. Thus the special case of the continuous flow model, in which activation flows down the linear structure of the paragraph, yields the same activation function as the discrete model (Anderson, 1976), in which the spread of activation is exponentially distributed (see Figure 2). Note that this derivation does not take into account any inhibitory processes, which would serve to reduce the asymptotic levels of activation (and in the long run produce a steady-state constant level of activation). There are several ways in which inhibition can be modeled: limited-capacity strength (Anderson, 1976), inhibitory connections (Levin, 1976; McClelland & Rumelhart, 1981), and decay in or dampening of the network (Anderson, 1976; Levin, 1976; McClelland & Rumelhart, 1981). However, with any of these mechanisms, the form of the activation functions would not be affected.

## Summary

The three cases of the spreading activation model—the discrete models with exponential and normal distributions and the continuous flow model—all make the same prediction about the times at which the near and far activation functions rise above zero. Specifically, they predict that the onset of activation in the far condition will be several hundred milliseconds later than the onset of activation in the near condition (see Figure

2). In addition, the discrete model with the exponential assumption and the continuous flow model predict that the far function will have a considerably slower rise time than the near function. In the discrete case, most other distributions that could be assumed to represent the process of spreading activation will yield predictions that lie somewhere between the predictions for the normal and exponential distributions: There will be a shift in the time intercept in the far condition, and there will be a slowing in the rate of approach to asymptote, depending on the skew and variance. If the distribution is extremely skewed, more than the exponential, then there will be a smaller intercept difference but a larger slope difference. These predictions are tested in the following experiments.

# Experiment 1

A study-test procedure was used in this experiment. On each trial, a subject read two paragraphs. Then test pairs were presented. The first word of each pair was the prime, presented for a variable amount of time (the SOA). The subject made no response to the prime. Then the target word was presented, and the subject was required to respond yes or no according to whether the target had appeared in the studied paragraphs.

The two independent variables in the experiment were the type of priming and the SOA. The prime was near the target or far from the target in the structure of the paragraph, or the prime was a string of random letters, a neutral priming condition. (Random letters were used instead of an unrelated word in order to avoid strategic inhibition effects; see Ratcliff & McKoon, 1981.) The SOA was 100, 150, 250, or 350 msec.

### Method

Subjects. The subjects were 24 Dartmouth undergraduates, each of whom participated in one 50-min. session and was paid \$3.

Materials. Sixty paragraphs like that shown in the previous example were assembled. Each paragraph was five sentences long, and each had a linear structure like that shown in Figure 1. Near priming pairs were N3-N2 (i.e., Noun 3 prime and Noun 2 target) and N5-N6; far priming pairs were N5-N2 and N3-N6. N3 and N5 were chosen so as not to be preexperimentally semantically related to N2 or N6. Negative test words came from a pool of words not used in any paragraph.

*Procedure.* On each trial of the experiment, there were two paragraphs to study and eight test pairs. Each subject was tested with 30 trials preceded by 2 practice trials.

The 2 paragraphs for the study phase of each trial were selected randomly without replacement from the total set of 60 paragraphs. The eight test pairs were selected as follows: First, there were two priming pairs for each of the studied paragraphs. One of these pairs was N2 as target preceded by a prime (N3, N5, or random letters) and the other was N6 as target preceded by a prime (N3, N5, or random letters). For each studied paragraph, there was also a pair made up of a word from the paragraph (a randomly chosen noun or verb) and a negative target. The other two test pairs were a pair made up of a word from one of the studied paragraphs and a target from the other studied paragraph (randomly chosen nouns or verbs) and a pair with random letters as the prime and a negative word as the target. The eight pairs were placed in random order in the test list with the constraints that (a) a priming pair (a pair with N2 or N6 as the target) could not appear in Position 1, (b) a word from one of the studied paragraphs could not appear in the test pair immediately preceding a priming pair of that paragraph, and (c) no word could appear in the test list more than once.

Presentation of materials and collection of data were controlled by a microcomputer interfaced with Dartmouth's time-sharing system. Materials were displayed on a cathode-ray tube (CRT) screen and responses collected on the CRT keyboard.

Each trial was initiated by the subject pressing the space bar on the keyboard. The two paragraphs to study were shown one at a time for 10 sec each. Then the test list began immediately. For each pair in the test list, a plus sign was presented for 150 msec; then the prime was presented for the appropriate SOA (100, 150, 250, or 350 msec); and then the prime disappeared and the target was displayed (one line below where the prime had been). The target stayed in view until the subject made a response. If the response was correct, then, after 200 msec the plus sign for the next test pair was presented. If the response was incorrect, the word ERROR was presented for 2 sec before presentation of the next test pair. Subjects were instructed to respond quickly and accurately.

Design. The six priming conditions (N2 primed by N3, N5, or random letters and N6 primed by N3, N5, or random letters) were combined into three pairs: N3-N2 and N5-N6; random letters-N2 and N3-N6; and N5-N2 and random letters-N6. For a given subject, each paragraph was tested with one of these three pairs. The three pairs were crossed with the four SOAs (100, 150, 250, and 350 msec) to make 12 experimental conditions. The 12 conditions were combined with 12 sets of paragraphs (5 per set) and 12 groups of subjects (2 per group) in a Latin square design. Order of presentation of materials was rerandomized after every second subject.

#### Results

Response times longer than 1,200 msec (about 3% of the response times) were eliminated from the analyses and the reported

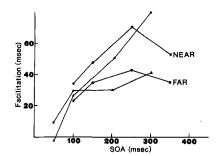


Figure 3. Amount of facilitation as a function of stimulus onset asynchrony (SOA) and priming condition. (The circles refer to Experiment 1, the triangles to Experiment 2.)

statistics, which were based on means from each subject or target word in each condition. Results are shown in Table 1 and Figure 3.

Figure 3 displays the amount of priming for the near and far conditions at each SOA, where the amount of priming was obtained by subtraction from the random letter priming condition. There is a larger priming effect for the near curve at every SOA, and it appears (by extrapolation) that the curves rise above zero at about the same point. It also appears that activation begins to decay at the longest SOA.

The random letter condition was not reliably affected by SOA (F < 1 for analysis of variance with subjects as a random factor, and F < 1 with materials as a random factor). However, response times in the near and far priming conditions were affected by SOA, F(3, 69) = 7.9, p < .001 (subjects analysis), and F(3, 177) = 4.1, p < .01 (materials analysis). Responses in the near prim-

Table 1
Mean Response Time and Percent Errors in
Each Condition of Experiment 1

SOA (in msec)	Priming condition								
	Near		Far		Random letters				
	Msec	%	Msec	%	Msec	%			
100	641	8	652	7	675	9			
150	615	9	628	7	663	11			
250	592	4	620	7	663	11			
350	600	4	617	6	652	11			

Note. SOA = stimulus onset asynchrony.

ing condition were faster than responses in the far condition, F(1, 23) = 8.0, p < .01 (subjects analysis), and F(1, 59) = 5.9, p < .02 (materials analysis). SOA did not interact with near versus far priming. Average standard error of the means was 7.5 msec.

## Experiment 2

The results of Experiment 1 suggest that the onset time of facilitation of responses in the far condition is about the same as the onset time for facilitation in the near condition. However, the onset time could not be measured directly, because there was already facilitation at the fastest SOA (100 msec). In Experiment 2, the fastest SOA was changed to 50 msec. The other SOAs were 100, 200, and 300 msec, and there were 36 subjects. In all other respects, the experiment was identical to Experiment 1.

#### Results

Response times longer than 1,200 msec (about 5% of the response times) were eliminated from the analyses and the reported statistics, which were based on means from each subject or target word in each condition. Results are shown in Table 2 and Figure 3.

Figure 3 shows that, just as in Experiment 1, the priming effect is larger for the near curve than for the far curve. Figure 3 also shows that the two curves rise above zero at about the same point.

The random letter condition was not re-

Table 2
Mean Response Time and Percent Errors in
Each Condition of Experiment 2

SOA (in msec)	Priming condition								
	Near		Far		Random letters				
	Msec	%	Msec	%	Msec	%			
50	659	10	648	10	656	8			
100	639	5	636	7	665	9			
200	604	5	626	7	656	10			
300	598	5	637	7	678	8			

Note. SOA = stimulus onset asynchrony.

liably affected by SOA, F(3, 105) = 1.3(subjects analysis), and F(3, 177) = 1.9(materials analysis). Response times in the near and far priming conditions were affected by SOA, F(3, 105) = 6.1, p < .01(subjects analysis), and F(3, 177) = 8.6, p <.01 (materials analysis). Responses in the near priming condition were faster than responses in the far priming condition according to the subjects analysis, F(1, 35) = 4.9, p < .05, but not according to the materials analysis (F < 1). SOA interacted with near versus far priming, F(3, 105) = 4.1, p < .01(subjects analysis), and F(3, 177) = 2.9, p <.05 (materials analysis). Average standard error of the means was 6.5 msec.

### Discussion

The experiments were designed to test the predictions made from two assumptions about the spreading activation process. The first assumption is that the amount of activation that arrives at a node is a decreasing function of the number of nodes the activation has traversed. By this assumption, a node in the representation of a paragraph should receive more activation from another node near to it in the representation than from a node farther away. Thus, it would be predicted that response time to a target word would be shorter when the target was primed by a near word than when it was primed by a far word. This prediction was confirmed by the data. The second assumption is that activation takes time to spread between nodes. Three variations of this assumption were examined: two models in which spreading activation was assumed to be a discrete process and one model in which it was assumed to be a continuous process. All three models predict that the amount of activation at the target word should begin to rise above zero much later for the far priming condition than for the near priming condition. This prediction was not confirmed; the onset time for facilitation of responses in the far condition was about the same as the onset time for facilitation in the near condition. Thus the results of our experiment are not consistent with current models of spreading activation. The models could accommodate our data by dropping the assumption that activation takes time to spread and instead assuming that activation spreads at a negligible rate (faster than about 5 msec per link, the limit on the ability of our experiments to measure differences). However, then the spreading activation process could not be used for its designed purpose, that is, to account for temporal variability in data.

The question presented by these results is how to conceptualize distance in a network representation of text. From these results. we can infer that the amount of activation that arrives at a node falls off as a function of distance, but that the time required for activation to arrive at the node is not a function of distance. In other words, activation must be transmitted very quickly through the network structure. However, when activation arrives at a node, response is not instantaneous; in the curves shown in Figure 3, it takes about 300 msec for facilitation to reach asymptote. Thus, the limiting factor in the activation process appears to be the time required to build up activation at a node. A suitable metaphor might come from the physics of light: The intensity of light decreases as a function of distance from the source, but the speed of transmission is very fast. However, even though transmission is very fast, intensity at the source can take time to build up, and therefore, intensity at any distance takes time to build up. For example, a light bulb may take 100 msec to reach maximum brightness and a bonfire may take minutes to build up in intensity both rates that are very much slower than the speed of light.

This kind of activation model has been proposed by Wickelgren (1976) to explain the mechanisms involved in processing semantic information. In this model, information is stored in a semantic network. In retrieval, activation spreads through the system at a very rapid rate (e.g., 1 msec per link). The activation of a node does not influence the activation levels of adjacent nodes. Rather, the activation pulse serves as a signal to begin retrieval, and the limiting factor is the rate of retrieval of strength at each node. This model predicts some aspects of the results of our experiment, namely that activation begins to build up at about the same time and the same rate for both the far and near priming conditions. However, the model has no mechanism to account for the lower asymptotic level of activation in the far condition, because the spreading activation pulse serves only to initiate retrieval and does not affect the amount of retrieved strength. (An asymptotic effect might be obtained by assuming that on a proportion of trials, the activation pulse is interrupted before reaching the target and that the farther away the target, the greater the probability of interruption.)

In contrast to network models of memory, there are feature models. For example, the feature model of Smith, Shoben, and Rips (1974) was designed as a competitor to the network model of Collins and Quillian (1969). Hollan (1975) pointed out that network and feature models are structurally isomorphic; but in a rejoinder, Rips, Smith, and Shoben (1975) argued that the different models suggest different processing mechanisms. As discussed above, the spreading activation processing mechanism cannot account for the data obtained in our experiment. However, the kind of feature model proposed by Smith et al. (1974) or Tversky (1977) would be capable of producing predictions consistent with the data. Relations between concepts would be represented by feature overlap, and priming would be produced by shared features becoming active. Thus, far and near priming would be expected to have the same onset time and rate of rise to asymptote. Asymptotic differences in near and far priming would represent different amounts of feature overlap. The main problem with a feature model however is that it has been applied only to semantic memory; no one has worked out a feature model for the representation of textual materials (e.g., paragraphs).

To summarize, we have examined the notion of spreading activation and derived predictions from several models that incorporate the spreading activation process and assume that the rate of spread of activation is slower than a few milliseconds a link. Data collected in a recognition paradigm were found to be inconsistent with these predictions. Other models were considered that could potentially provide predictions consistent with the data.

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