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Spread of Activation

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Spreading activation serves the function of quickly spreading an associative relevancy measure over declarative memory. The assumptions and evolution of the ACT* theory of spreading activation are discussed with a detailed mathematical formulation. It is assumed that activation is very rapidly spread over a semantic network and that various levels of activation are established throughout the network. Level of activation maps onto processing time by controlling the amount of time that it takes for the condition of a production rule to match. The ACT* spreading-activation theory predicts an exponential decay of activation with distance in network structure and an interaction of activation levels with the complexity of productions that are matched and executed.

In this article we describe the basic assumptions of spreading activation in the ACT* theory (Anderson, 1983a). Like several other recent theories that make use of the spreading-activation construct (e.g., Anderson, 1976; Collins & Loftus, 1975; Collins & Quillian, 1969, 1972; Hayes-Roth, 1977), ACT* assumes that declarative knowledge (i.e., facts that are known) is encoded as a network structure, consisting of nodes representing concepts and links representing associations among concepts. Network information becomes available for processing when it is activated. Spreading activation is the process by which activation spreads from node to node along network links, making knowledge associated with particular sources of activation (i.e., a foci of attention) available for processing. One can think of spreading activation as a process that identifies knowledge relevant to a current focus of attention and that favors the processing of that knowledge. This construct has been used in models addressing data from a variety of paradigms including the retrieval of categorical facts (e.g., *a canary is a bird,* Collins & Quillian, 1969, 1972), the facilitation of the processing of items by the presentation of associated items (e.g., McKoon & Ratcliff, 1980; Meyer & Schvaneveldt, 1971), fact retrieval (e.g., Anderson, 1974, 1976), and text comprehension (Kieras, 1981; Miller, 1981).

The ACT* theory of spreading activation differs in several important respects from its predecessor, the ACTE theory (Anderson, 1976). In the next section we review the ACTE theory and the empirical evidence motivating the evolution of the spreading activation theory in ACT*. We then present the assumptions and the mathematical formulation of spreading activation in ACT* with an emphasis on two basic points. First, to a close approximation, we can think of activation as spreading instantaneously. Second, over a fair range of network structure, activation will decay exponentially with the distance it spreads. Both of these facts offer important simplifications in analyzing the dynamics of spreading activation.

Spreading Activation in ACTE

Both the ACTE and the ACT* theories involve a fundamental division between *declarative* and *procedural* knowledge. Declarative information is encoded in the long-term memory network. The spreading-activation process is defined on this declarative, long-term network. The procedural component consists of productions that are condition–action pairs. The condition of a production

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specifies some pattern that must be *matched* by active information in declarative memory. If that condition is matched, the action will be *executed.* Production actions may specify additions to active declarative memory, new goals, or external responses. It is the sequencing of these condition matches followed by action executions that gives rise to behavior. A typical but simplified production is as follows:

IF asked "Who LVverb LVobject"
and it is known that LVagent LVverb LVobject
THEN say "LVagent"

The terms prefixed by LV are variables. The above production might apply if a person was asked, "Who defeated Carter?" and it is stored in long-term memory that Reagan defeated Carter. The variable bindings would be LVagent = Reagan, LVverb = defeated, and LVobject = Carter. The production would generate the answer, Reagan. This production clearly illustrates that time to generate a memory report will depend on both time to retrieve information from memory and time to match the condition of the relevant production.

Activation in ACTE was all-or-none, that is, declarative information could either be active or not. The active partition of the declarative memory defined the system's *working memory* (an idea similar to Shiffrin, 1975). Productions could only match to active information. In ACTE, the significant factor in determining reaction time (RT) was time for activation to spread to the relevant knowledge structures so that they would be available for pattern matching. The time to actually achieve the pattern matching was not significant. Thus ACTE was, in considerable part, a theory about what determined the rate of activation spread. In this respect, ACTE was like many other theories of spreading activation (e.g., Collins & Loftus, 1975; McClelland, 1979).

There are now good reasons to reject this analysis. It has been shown in priming paradigms that priming effects are very rapidly evident after the onset of the prime (Fischler & Goodman, 1978; Ratcliff & McKoon, 1981; Schustack, 1981) and at nearly full strength. For instance, Ratcliff and McKoon had subjects study paragraphs of the following form:

The scientist nudged the sheriff.
The sheriff stared at the spacecraft.
The spacecraft transported an alien.
The alien drew a weapon.
The weapon vaporized a mountain.

Subjects were then presented with a prime and a target noun and had to decide if the target was one that occurred in a studied paragraph. Ratcliff and McKoon varied both the interval between presentation of the prime and the target (stimulus onset asynchrony or SOA) and the distance between the prime and the target in the network structure encoding the study paragraphs. For example, the network distance between the prime *weapon* and the target *mountain* is less than the network distance between the prime *spacecraft* and the target *mountain.* The assumption that activation takes some significant time (i.e., on the order of 50–100 ms) to spread from one node to the next predicts that short SOAs should produce facilitative effects on RTs for primes close in network structure to targets but not for primes that are distant from targets. At longer SOAs, activation would have time to spread from primes to distant targets, and hence, facilitative priming effects should be evident for primes either close or distant in network structure from targets. Thus, the assumption that activation takes significant time to spread predicts an interaction between SOA and prime–target network distance.

Ratcliff and McKoon (1981) found that the facilitative effect of a prime on target recognition increased as the network distance between the prime and the target decreased. However, they also found that this distance effect did not vary with SOA ranging from 50 ms to 350 ms. This is at odds with the notion that activation takes time to spread from node to node. Thus, it has been argued that activation spreads too rapidly for its spread time to be a significant factor in controlling RT latency.

There is another anomaly in many fact-retrieval experiments that points to a problem with ACTE: There is an interaction between the apparent rate of spread and the complexity of the decision that subjects had to make about the information once activated. Anderson (1976, p. 534) noted that the estimate

of the rate of spread of activation varied with task complexity. This makes no sense in the ACTE model. According to the ACTE theory, first the information is activated; then in a separate stage, the information is processed. Thus, variables affecting spread time and variables affecting complexity should be additive. Although there are many possible interpretations of this problem in the ACTE model, the argument here is that the problem was in assuming that the level of activation had no effect on the rate at which the information was processed.

Spreading Activation in ACT*

In ACT*, activation rapidly spreads through the declarative network, setting up various levels of activation throughout the network. In contrast to the all-or-none concept of activation in ACTE, declarative structures in ACT* have continuous levels of activation. The rate at which productions are matched to declarative structures is determined by the level of activation of those structures. In ACT*, it is the time to complete this matching process that is the rate-limiting factor in information processing and not the time to spread activation through the declarative network.

The spreading-activation process assumes a network like the one illustrated in Figure 1. Here we have a network encoding of a set of propositions. As in typical propositional network representations, we have nodes corresponding to the propositions connected to nodes representing the arguments and the relations. Declarative memory is partitioned into a temporary memory that encodes new information from the environment and a long-term memory. There are two propositional nodes in temporary active memory encoding the fact that *The doctor hates the lawyer who is in the bank,* which we will

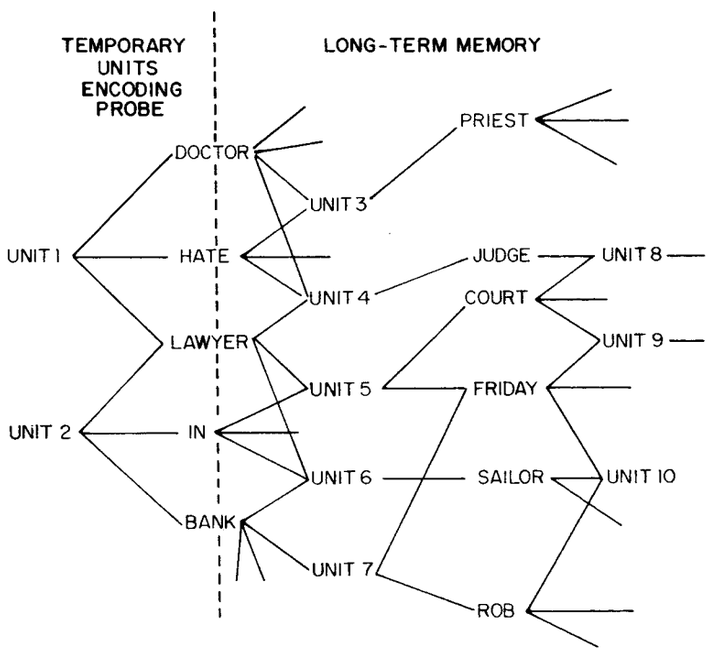

Figure 1. A hypothetical network structure for purposes of illustration. (In temporary, active memory, this is encoded as, *The doctor hates the lawyer who is in the bank.* In long-term memory, the facts are illustrated as follows: *The doctor hates the priest, The lawyer hates the judge, The lawyer was in the court on Friday, The sailor is in the bank, The bank was robbed on Friday,* and *The sailor was robbed on Friday.*)

assume is presented to the subject. Nodes corresponding to the main concepts (doctor, hate, lawyer, in, bank) are sources of activation. They would receive activation from the words in the presented sentence. It is from these sources that activation spreads throughout the network. For purposes of understanding the spread of activation, we can think of long-term memory as a network of nodes, where we do not distinguish between proposition nodes and their arguments.

Activation spreads from one node to another node throughout the network. There is *reverberation,* that is, activation can spread from node i to node j and then back to node i. There are three properties of this spreading process. First, the total activation emitted by a node is less than its own activation, that is, if a_i is the activation of node i, it sends pa_i units of activation to all connected nodes where $p < 1$. Because of this factor, the amount of activation decreases exponentially with distance from source. Only a fraction p^n of the activation is left after it has spread n links. The second property is that the activation emanating from node i is divided among the nodes connected to i according to their relative strength. If s_j is the strength of node j, which is connected to i, S is the strength of all nodes associated with i, and a_i is the activation level of i, then pa_is_j/S is the activation sent to j. The third property is that activations converging on a node from multiple sources will sum. This means that a knowledge structure will be processed more rapidly if additional sources can spread activation to it.

These factors will interact to set up a stable pattern of activation in the network in response to a probe. The probe enters activation at nodes corresponding to the terms in the probe, and the pattern of activation builds around these nodes. The term *pattern of activation* refers to the different levels of activation achieved in the surrounding network. The rate of matching productions to declarative network structures is proportional to the level of activation of the declarative structures and is inversely proportional to the complexity of the production condition being matched. Thus, the time to match a production with a condition pattern of complexity C to a declarative structure of activation A will be rC/A (see Anderson, 1983a, chap. 4 for details). This expression predicts the interaction of pattern complexity and level of activation that ACTE failed to address.

Mathematical Formulation of Spreading Activation

There are two potentially important factors that could determine processing time. One is the time for activation to spread through the network. The other is the time for the production conditions to match. The claim in ACT* is that to an approximation, the critical factor is the time for conditions to match and that we can regard the spreading process as instantaneous. Many people find this counterintuitive. The mathematical development that follows shows how this could be true.

We can conceive of the network over which activation spreads as an $n \times n$ matrix $\mathbf{R}$ with entries r_{ij}. The value $r_{ij} = 0$ if there is no connection between nodes i and j. Otherwise, it has a value that reflects the relative strength of connection from node i to node j. A constraint in the ACT* theory is $\Sigma_i\, r_{ij} = 1$. This guarantees that the activation from node i is divided among the attached nodes according to their strength. The level of activation of a node will increase or decrease as the activation coming into it from various sources increases or decreases.

It is most natural to conceive of the activation of a node as continuously varying with increases or decreases in the amount of input. A bound is placed on the total activation of a node because of a decay process. This conception can be captured by the model of a leaky capacitor, and the change in activation of node i is described by the following equation:

$$\frac{da_i(t)}{dt} = Bn_i(t) - p^*a_i(t), \qquad (1)$$

where $n_i(t)$ is the input, and the negative factor reflects decay in activation proportional to the level of activation.

The input to a node depends on whether it is a source node. If it is not, the input is just the activation received from nodes connected to it. If it is a source node, it receives an additional amount of activation, c_i. Thus, we can express this as follows:

$$n_j(t) = c_j(t) + \sum_i r_{ij} a_i(t - \delta t), \qquad (2)$$

where $c_j(t) = 0$ if j is not a source node at time t, and $c_j(t) = c_j$ if it is. The term in the sum indicates that the activation from node i is divided among the attached nodes according to the relative strengths r_{ij}. The value δt appears in Equation 2 to reflect the delay in transmission between node i and node j.

Equations 1 and 2 may be represented more generally if we consider an n-element vector $A(t)$ to represent the activation of each node, another n-element vector $\mathbf{I}(t)$ to represent the inputs, and a third n-element vector $\mathbf{C}^*(t)$ to reflect the activation from the source nodes. Then, Equations 1 and 2 become the following:

$$\frac{d\mathbf{A}(t)}{dt} = B\mathbf{I}(t) - p^*\mathbf{A}(t) \qquad (1')$$

and

$$\mathbf{I}(t) = \mathbf{C}^*(t) + \mathbf{A}(t - \delta t)\mathbf{R}, \qquad (2')$$

where $\mathbf{R}$ is the matrix of connection strengths. The general solution for Equation 1′ is given as follows:

$$\mathbf{A}(t) = \int_{-\infty}^{t} e^{-(t-x)p^*} B\mathbf{I}(x)dx. \qquad (3)$$

There is no general, closed-form solution to this equation. McClelland (1979) dealt with the situation where $\delta t = 0$, and there was no reverberation (i.e., activation did not feed back from a node to itself either directly or through intermediate nodes). The first assumption may be approximately true, but the second assumption is invalid in the current context.

When all $r_{ij} \geq 0$ and $p < 1$, the activation pattern will eventually reach asymptote. When at asymptote, $d\mathbf{A}(t)/dt = 0$, and we can derive the following from Equations 1′ and 2′:

$$\mathbf{A} = \mathbf{C} + p\mathbf{AR}, \qquad (4)$$

where $\mathbf{C} = B\mathbf{C}^*/p^*$ and $p = B/p^*$. If $p < 1$, Equation 4 describes a set of simultaneous linear equations with finite, positive solutions for all of the a_i. Thus, we can determine the activation pattern in the network by solving this set of equations.

In this system, each source of activation supports a particular subset of activation over the network. Let $\mathbf{A}_i(t)$ be a vector describing the subset of activation supported by source i. Then, the total activation is just the sum of the subsets supported by all sources i, that is,

$$\mathbf{A}(t) = \sum_i \mathbf{A}_i(t). \qquad (5)$$

We assume that the input c_i from source c stays constant over the period it is a source. Let $S_i(t)$ denote the sum of activation over all the nodes supported by source i, t seconds after i became a source node. To calculate $S_i(t)$, we need to represent t in terms of the number, n, of δt time units that have passed, that is, $t = n\delta t$. The activation will have had a chance to spread n units or levels deep by the time t. Then,

$$S_i(n\delta t) = c_i \sum_{i=0}^{n-1} p^i \left[1 - \sum_{j=0}^{i} \frac{p(i, n)^j e^{-p(i,n)}}{j!}\right], \qquad (6)$$

where $p(i, n) = p^*(n - i)\delta t$.[1]

In the limit this has the following value:

$$\lim_{t \to \infty} S_i(t) = c_i/(1 - p). \qquad (7)$$

Because the total activation in the network reaches asymptote in the limit, we can also show that the pattern of activation in the network reaches a stable equilibrium in the limit. That is, given a fixed set of sources of activation, all nodes in the network will have stable values of activation after some amount of time. Because the total activation supported by a fixed set of activation sources asymptotes at a fixed value, $\Sigma_i S_i(t)$, the only way the activation at a particular node can increase in value is if there is a corresponding decrease in activation elsewhere in the network. However, the change in activation (Equation 1′)

[1] The outer summation in Equation 6 is over the n levels that activation has spread. The factor p^i is the asymptotic amount of activation that will spread to level i. It is multiplied in the equation by the fraction of asymptotic activation that will have built up on $(n - i)\delta t$ units of time since activation reached the node (see McClelland, 1979). Note that it may be the case that there are loops in the network, and a node at level i will also be counted at level $i + j$. Because activation is additive, we can separately count the activation due to the length i path and the length $i + j$ path, as is done in this equation.

for all nodes supported by the activation sources is a monotonically increasing function of t. Thus, as total network activation reaches asymptote, the activation level at each node reaches a stable value.

If we assume that δt reflects something like neural transmission time, it is reasonable to assign to it a value like 5 ms. Because p^* reflects the decay rate of neural activation, it seems reasonable to assign to it a value such as 1. That is, it has a half-life of about 1 ms. Because $B < p^*$, a plausible value for B, which represents the accumulation of activation, is 0.8, a value we used in all of our simulations. Because $p = B/p^*$, it will also have the value 0.8. Given these values for the basic parameters, it becomes of interest to inquire how rapidly $S_i(t)$ reaches its asymptote value in Equation 7. The rate at which it approaches asymptote is illustrated in Figure 2. As shown, it assumes a value of 50% of asymptote in 20 ms, 80% in 40 ms, 90% in 60 ms, 98% in 100 ms, and greater than 99% in 150 ms.

These times to approach asymptote are an order of magnitude or more less than typical RTs and are smaller than typical differences between conditions. Thus it seems clear, under these assumptions, that time for activation to spread cannot be what is determining RTs in a typical experiment. Thus, these results are consistent with findings such as those by Ratcliff and McKoon (1981) that failed to show an effect of distance of spread on time. Also, it is in keeping with the adaptive function of spreading activation. Activation is supposed to be a relevancy heuristic for determining the importance of various pieces of information. It serves the function of guiding and making more efficient subsequent processing. It would not make much adaptive sense to compute a spreading-activation process if it took that computation a long time to identify the relevant structures of memory.

Effect of Distance

Another complexity in developing predictions from a spreading-activation model is that one has to worry about potential effects of reverberation, where activation spreads away from the network of concern and spreads back to the network. The point of the analyses that follow is to show that to a good approximation, one can ignore the effects of distant network structure. We start these analyses with some simple cases and work to more complex cases.

Linear chains. Represented in Figure 3 (top) is a simple linear chain that starts with node 0 and extends to nodes 1, 2, 3, and so forth, where node n is connected to both nodes $n - 1$ and $n + 1$ except for node 0, which is connected just to node 1. We will assume that all links in the chain are of equal strength. Assuming that node 0 becomes a source and 1 unit of activation is input at node 0, the asymptotic pattern of activation can be derived. Letting a_i refer to the activation of the i^{th} node in the chain, we have $a_i = kr^i$, where $r = [1 - (1 - p^2)^{1/2}]/p$ and $k = 2/(1 - rp)$. The exception to this rule is the activation for node 0, which has activation level $a_0 = 1/(1 - rp)$. Because r is a fraction, activation level decays away exponentially with distance from source. Assuming $p = 0.8$, which is a typical value in our simulations, we get $r = 0.5$, $k = 3.33$, and $a_0 = 1.67$. Note that although a_0 is only given 1 unit of activation as a source, reverberation increases its level by an additional 0.67 units.

Another kind of linear chain is illustrated in the bottom half of Figure 3. This one is centered at node 0 and extends to $-\infty$ and to $+\infty$ in the two directions. Again, we can solve for asymptotic patterns of activation by assuming 1 unit of input at node 0. Again, activation decays exponentially with distance $a_i = kr^i$, and again, $r = [1 - (1 - p^2)^{1/2}]/p$ and $a_0 = 1/(1 - rp)$, but now $k = 1/(1 - rp)$.

Thus, we see from these examples that activation tends to decay away exponentially with distance from source. Thus, we can safely ignore effects of distant structure. This is one of the reasons why a network will reach a near-asymptotic pattern of activation rather quickly, that is, the distant structure that takes the activation longer to reach has little effect on final pattern of activation. The data of Ratcliff and McKoon (1981) on linear chains in paragraphs are relevant here. They were able to show that priming decays exponentially as a function of distance, as predicted by this analysis.

More complex structure. An interesting question concerns what happens to this pre-

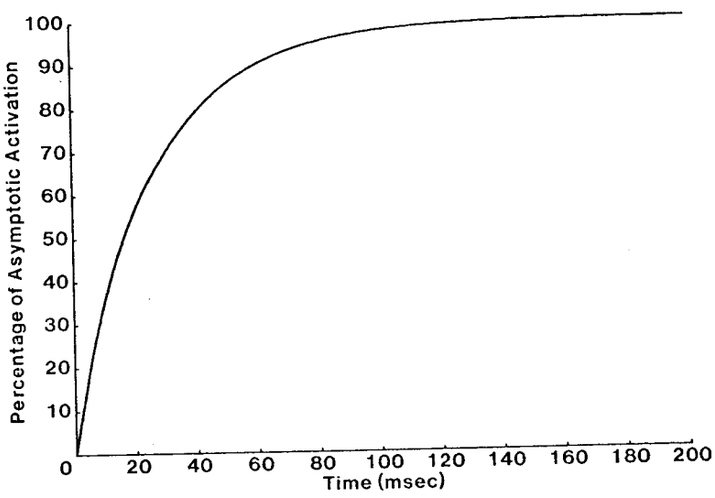

Figure 2. Network activation as a function of time.

diction of exponential decay with distance when we look at more complex network structures. Consider the following situation: A source node has 1 unit of activation, and it is in the middle of a network structure. It is possible to classify all nodes in that network by their minimum link distance from the source node. By definition, a node in level *i*

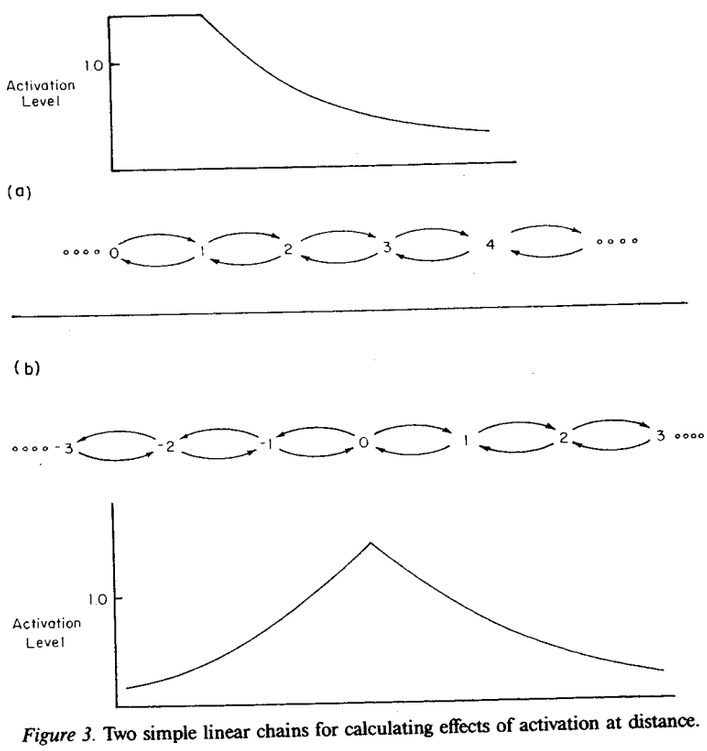

Figure 3. Two simple linear chains for calculating effects of activation at distance.

can be connected to other nodes in level i, to nodes in level $i - 1$, and to nodes in level $i + 1$. Assume that these connections are random except that for all levels i, the average summed strength of connections of a node at i to nodes at $i - 1$ is s_1; to other nodes at i, s_2; and to nodes at $i + 1$, s_3. By definition, $s_1 + s_2 + s_3 = 1$. Let a_i denote the total activation at level i, that is, the sum of activation of all nodes that i links from the source. Let a_0 be the level of activation of the source node.

For all levels except 0 and 1, the following equation describes the pattern of activation:

$$a_i = ps_3a_{i-1} + ps_2a_i + ps_1a_{i+1}. \quad (8)$$

The following equation holds for $i = 1$:

$$a_1 = pa_0 + ps_2a_1 + ps_1a_2. \quad (9)$$

The next equation holds for $i = 0$:

$$a_0 = 1 + ps_1a_1, \quad (10)$$

where 1 is the amount of activation input to the source node. It can be shown that once again level of activation decays exponentially with distance such that $a_i = kr^i$, where r, k, and a_0 equal the following:

$$r = \frac{1 - ps_2 - [(1 - ps_2)^2 - 4s_1s_3p^2]^{1/2}}{2ps_1}, \quad (11)$$

$$k = \frac{p}{r(1 - ps_1r - ps_2 - p^2s_1)}, \quad (12)$$

and

$$a_0 = \frac{(1 - ps_1r - ps_2)}{(1 - ps_1r - ps_2 - p^2s_1)}. \quad (13)$$

Thus, the prediction of exponential decay with distance is a rather general expectation. It is possible to imagine configurations in which nodes at a particular level i are more active than nodes at $i - 1$. However, because the activation decays by a factor p each time it spreads, the long-range trend in activation levels has to be exponential decay.

Conclusion

We presented the motivations and assumptions underlying the ACT* theory of spreading activation. Elsewhere, this theory has been used to address and to integrate a variety of phenomena from the memory literature (Anderson, 1983b), the priming paradigm, and the fact-retrieval literature (Anderson, 1983a, chap. 3).

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