

“specification-selection” architecture). These results are reminiscent of a number of recent experiments involving ambiguous targets (Basso & Wurtz 1998; Bastian et al. 1998; Munoz & Wurtz 1995; Platt & Glimcher 1997), and can be easily simulated with a movement planning field model like that of Thelen et al., as was explicitly done for the experiment of Bastian et al. (1998).

One can predict that competition between actions in a movement planning field will be evident even in the final movement trajectory, with subtle deviations occurring when the activity bill of an unselected potential movement slightly overlaps that of the selected movement. Indeed, such deviations have been shown for reaching movements in the presence of distractors (Tipper et al. 1998), and simulated with a model (Tipper et al. 2000) which is conceptually very similar to that of Thelen et al.

The authors are of course well aware of the support that such models can gain from neurophysiological evidence. Their discussions in sections 4.1.1 and 7.1.3 make this clear. However, I think that one can go far beyond these preliminary comparisons. In fact, one can go so far as to suggest that most neural activity is not so much concerned with representing the world as with “mediating interactions with the world,” through specifying potential actions and selecting among them. One can use neurophysiological data, traditionally interpreted from the perspective of cognitivism, to support theoretical frameworks such as that of Thelen et al. or that of Figure 1, which stand in opposition to many of the assumptions of cognitivism. In fact, if we are indeed poised to witness a shift away from the disembodied computational assumptions of traditional cognitive psychology to a more embodied science of behavior, such a shift may be primarily driven by the growing literature of neurophysiological data.

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Looking closely at infants’ performance and experimental procedures in the A-not-B task

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Abstract: Thelen et al.’s model of A-not-B performance is based on behavioral observations obtained with a paradigm markedly different from A-not-B. Central components of the model are *not* central to A-not-B performance. All data presented fit a simpler model, which specifies that the key abilities for success on A-not-B are working memory and inhibition. Intention and action can be dissociated in infants and adults.

The target article by Thelen et al. is ambitious, but ultimately disappointing. (1) Central to their model of A-not-B is an attempt to account for findings obtained using a paradigm fundamentally different from A-not-B. By arguing that their procedures closely approximate those used in A-not-B studies, they misrepresent what was done in A-not-B studies. (2) Thelen et al.’s model has fatal flaws. (3) The premise of the target article is to solve a puzzle that is not a puzzle. (4) The role of prefrontal cortex was too easily dismissed. (5) Dissociations between “knowing” and “doing” were caricatured.

1. The behavioral paradigm used by Thelen and colleagues (e.g., Smith et al. 1999b) to study A-not-B performance differs from the A-not-B task in several key respects (see Table 1). For example, whereas in the A-not-B task, a trial at B is only administered after an infant has reached correctly; Thelen et al. administered the B trial after a set number of trials at A, even if the infant had reached incorrectly on the last trial of that set. There are ob-

vious problems in measuring “perseveration” in a participant who has not shown a consistent response that might then be perseverated.

2. Thelen et al.’s model faces a number of major problems: (a) “*The relative ambiguity of the task input is a critical parameter in the model*” (sect. 2.2.1, emphases in original). However, this is not critical to the A-not-B error; indeed the kind of ambiguity Thelen et al. produced by using lids and background of the same color and placing the lids close together is not present in most A-not-B studies (see Table 1). That such ambiguity is not central to the A-not-B error, but is central to Thelen et al.’s model, is a huge problem for their model. (b) They claim that a critical part of why infants err on the A-not-B task is because it is entirely novel. However, that cannot be critical because when I tested infants on the task every 2 weeks for 6 months I still saw the A-not-B error consistently at all ages (Diamond 1985). All that practice and repeated exposure had only a modest effect on performance. Older, practiced infants made the A-not-B error as robustly (albeit at a longer delay) as younger, novice infants and as robustly as older, novice infants. (c) They claim that *the act of reaching to A* is critical to why infants err and the probability of making the A-not-B error is a function of the number of previous reaches to A. However, while the number of reaches to A matters when the number of A trials is as large as 8–15, there is no effect whatsoever on the number of repeated reaches to A within the range of 1–3 or even 2–5 (Butterworth 1977; Diamond 1983; Evans 1973). Thelen et al. assert that repetition is essential; but it is not needed at all: The A-not-B error is as robust after one reach to A as it is after three repetitions to A. An even bigger problem for the authors is that infants do not need to reach to A at all to make the A-not-B error. Both Evans (1973) and I (1983) found, with no pretraining trials to A, as robust an A-not-B error from just observing the experimenter retrieve the toy at A as from the infant reaching and retrieving the toy at A. (d) They attribute the A-not-B error in part to the poor reaching skills of infants between 7–12 months. However, infants of 10–12 months are quite skilled reachers, and they show the A-not-B error as robustly as younger infants. (e) They predict that with multiple reversals infants should reach randomly. That prediction has not been confirmed. In Diamond (1985), Diamond and Doar (1989), and Diamond et al. (1994), we administered multiple reversals and found that (i) infants did not reach randomly, but showed a predictable pattern to their reaches, and (ii) on later trials infants performed no worse and were no more likely to reach randomly than on earlier trials.

3. The puzzle that Thelen et al. set out to solve is: “While the A-not-B error is entirely robust in the canonical form we described above, even seemingly small alterations in the task conditions can disrupt it” (sect. 1, para. 3). (a) However, many alterations in the task do not affect performance. For example, the A-not-B error is found whether the hiding places differ in left-right or up-down location (Butterworth 1976), and is found whether the toy is hidden under cups (e.g., Neilson 1982), in containers (e.g., Butterworth 1975), behind screens, curtains, or doors (e.g., Harris 1973), on the tabletop under a cloth (e.g., Fox et al. 1979) or inside wells (e.g., Diamond 1985). (b) The A-not-B error is so robust that, despite marked variability in task administration, virtually every lab finds this behavior. Many alterations that make the task easier do not disrupt the A-not-B error altogether, but simply affect the delay at which it occurs and/or how often it is repeated. (c) Alterations in the task that affect how easy it is to remember where the toy was hidden or how strong the pull is to repeat the previously rewarded action (e.g., varying the discriminability of the hiding places, salience of what is hidden, delay between hiding and retrieval, number of reaches before the reversal, or visibility of the “hidden” toy) *should* affect the likelihood of finding the A-not-B error if my theoretical position (that the key abilities required for the A-not-B task are working memory and inhibitory control) is correct, and they do. I see no puzzle here.

There are no data that Thelen et al. present which cannot easily be accounted for by the theory I presented in the early 1980s.

Table 1 (Diamond). *Differences between the procedures used by Thelen, Smith, and their colleagues and those used in studies of A-not-B*

Procedural Element	Procedures used by Thelen et al.	Procedures used by A-not-B Researchers
(a) Discriminability of the covers from the background surface.	Lids designed to blend into the background: Brown lids on a background of the same brown color Thelen et al.: "The notable characteristic of [our] task input was its lack of visual specificity. The two lids . . . blended into the background of the box."	Covers designed to stand out from the background. For example: White covers on black tabletop (Bremner 1978) White covers on green background (Butterworth & Jarrett 1982) Blue or red covers on a brown background (Sophian & Yengo 1985) Light blue covers on dark brown background (Diamond 1985) Most A-not-B researchers go out of their way to make the covers discriminable from the background surface.
(b) Distance between the covers.	Lids placed quite close together (12.5 cm apart, center to center); this is less than half that in most A-not-B studies.	Covers usually placed considerably further apart: 30 cm apart center to center: Acredolo et al. 1985; Appel & Gratch 1984; Benson & Uzgiris 1985; Diamond & Doar 1989; Evans & Gratch 1972 28 cm apart center to center: Bremner 1978; Diamond 1985
(c) Illumination of the room.	Low illumination; dimly lit room in one study. (That would make it harder for infants to see the covers, tell them apart, and distinguish them from the background.)	A-not-B testing is conducted in a brightly lit room. In no study of A-not-B have the lights been dimmed.
(d) Presence of distraction during the delay.	No distraction. Infant permitted to continue to look at, or turn or reach toward, the cued location throughout the delay. No attempt to break infant's fixation on the correct well or to prevent position cueing.	Some studies provided no distraction, but others have: Visual fixation of the correct well prevented (e.g., Diamond 1985; Diamond & Doar 1989; Diamond et al. 1994; Fox et al. 1979; Freeman et al. 1980). Bodily cueing toward the correct well prevented (e.g. Diamond 1985; Diamond & Doar 1989; Diamond et al. 1994; Fox et al. 1979; Harris 1973)
(e) Amount of initial reaching experience at A:		
(e.1.) Number of training trials at the "A" location.	Four training trials administered at A.	No training trials at A (training trials administered at a central location): e.g., Benson & Uzgiris 1985; Bower & Patterson 1972; Diamond 1985; Evans 1973; Fox et al. 1979; Horobin & Acredolo 1986; Schuberth et al. 1978; Sophian & Yengo 1985; Willatts 1979). Where training trials at A have been administered, only 1 or 2 are given, not 4.
(e.2.) Number of initial trials at the "A" location.	Six trials administered at A. This is 50%–500% more initial trials at A than in A-not-B studies.	In <i>all</i> A-not-B studies: Typically only 1 or 2 trials administered at A, and not more than 4, except in studies specifically designed to look at the effect of variation in the number of initial trials at A.
(f) Rule for determining when to switch to the "B" location.	B trial administered after a set number of trials at A, regardless of infant's performance on the A trials. B trial administered even if the infant had reached incorrectly on the preceding A trial.	B trial administered after infant has reached correctly 1 or more times at A, regardless of whether that takes 1, 2, 3, or 4 trials at A. B trial administered <i>only</i> following a correct reach at A.

(continued)

Table 1 (Diamond). (Continued)

Procedural Element	Procedures used by Thelen et al.	Procedures used by A-not-B Researchers
(g) Criterion for determining whether a reach is correct or not.	Painstaking frame-by-frame analyses of the videotape to see if a slightly faster contact to one lid can be detected. If so, it is scored as a reach to that lid, not both.	Scoring is never done by frame-by-frame analysis. An infant who touches both covers at almost the same instant is not scored as having intended to reach the cover that was contacted a millisecond earlier, but as having reached to both covers.
(h) Infants' rationale for reaching, the reward for reaching correctly, and the penalty for reaching incorrectly. Why reach to one location rather than another?	Reaches were usually for a visible lid, exactly like the visible lid at the other location. No toy was usually hidden and no reward usually provided for a correct reach. When a toy was hidden, infants were allowed to have it on each trial, whether or not they had reached correctly. Thus, there was no difference in outcome of a correct or incorrect reach.	The two covers are identical, but reach is for a hidden toy, located under only one of the covers. The reward for a correct reach is getting to play with the toy. Some studies penalize an incorrect reach by not letting the infant have access to the toy on that trial (e.g., Diamond 1985; Diamond & Doar 1989; Diamond et al. 1994; Horobin & Acredolo 1986).

Thelen et al. suggest that “it is not clear in Diamond’s account, why . . . the number of A reaches or the distinctiveness of the targets should matter so profoundly” (sect. 6.1.1). Yet, it follows straightforwardly that anything that increases the strength of the prepotent tendency that must be inhibited (as would increasing the number of reaches to A) or that makes less distinct the information that must be held in mind (e.g., reducing the distinctiveness of the targets) should make errors more likely. Indeed, the parameters of Thelen et al.’s model – “the target position must be remembered during the reach” (working memory, sect. 3.1.4) and “the motor memory of the just-completed movement is also retained and integrated into the next plan” (thus requiring inhibitory control) – map directly onto the parameters in my account. The onset of locomotion might be related to improved A-not-B performance because locomotion onset provides an index of maturational level or because infants attend more closely to the kind of information they must hold in mind in the A-not-B situation once they are locomoting; these findings pose no problem for my theoretical perspective. Finally, Thelen et al. assert that my theory “cannot account for the looking-reaching decalage.” (sect. 1.1, para. 4) It can and it has (Diamond 1998).

For the record, Thelen et al. make some assertions about experimental design and procedures used in my work that are incorrect (a) “The delay and the number of repetitions are not independently controlled. So it is impossible to tell whether failure is due to delay or to repetition” (sect. 3.2.1). Not true. Diamond (1985) reported the effect of systematically increasing or decreasing the delay within a session. Diamond (1983) reported the effect of systematically varying the number of repetitions at A. (b) “The actual number of reaches to A before the switch is not reported and is unknown.” It is known and reported (Diamond 1983). As reported, (a) in >90% of testing sessions, infants were correct on both initial trials at A and so received only 2 A trials, and (b) infants never made more than one error on the initial A trials and so never received more than 4 A trials. Diamond (1983) and others have shown that varying the number of A trials within that small range has no effect whatsoever on the A-not-B error. (c) “These procedures commonly make 12 or 15 target switches (sect. 6.2.1, last para.)” Not so. These procedures never make more than 5 switches at the delay used for testing and never more than 2 switches at other delays prior to that – hence no more than 7 switches (Diamond 1983; 1985; Diamond et al. 1994; 1997).

4. Thelen et al. question whether A-not-B performance can be assumed to be a marker of dorsolateral prefrontal cortex maturation. The following addresses their reservations in turn:

(a) The evidence for prefrontal involvement comes from work

with rhesus monkeys where only a similar, but not identical, task was used. Not so. The task used with monkeys (Diamond & Goldman-Rakic 1989; Diamond et al. 1989) was as identical as the A-not-B task used in one infancy lab is to that used in another infancy lab. I have argued that results on a similar task (delayed response) are also relevant because A-not-B and delayed response are essentially the same task and the developmental progressions on both tasks are identical in human infants and infant rhesus monkeys (Diamond 1991a; Diamond & Doar 1989). Most compelling, the results with human infants and with monkeys with lesions of dorsolateral prefrontal cortex closely parallel one another. They fail the task in the same ways and under the same conditions; parametric variations in the task have the same effects on the performance of both groups. See Table 2.

(b) “Second, the problem of circularity. The evidence that prefrontal cortex ‘matures’ between 8 and 12 months is performance on A-not-B type tasks” (sect. 6.2.1). Not so. In human infants, studies of surface electrical activity indicate maturational changes in prefrontal cortex during the period that infants are improving on A-not-B and such changes are correlated with A-not-B performance (e.g., Bell & Fox 1992; 1997; Fox & Bell 1990). There is also considerable independent evidence of prefrontal cortex maturation during the period (1–4 months of age) that infant rhesus monkeys are improving on the A-not-B task. Take one aspect of prefrontal maturation (maturational changes in the prefrontal dopamine system): During the period of 1–4 months, the density of prefrontal dopamine receptors is increasing (Lidow & Rakic 1992) and the distribution within prefrontal cortex of axons containing tyrosine hydroxylase (essential for the production of dopamine) changes markedly (Lewis & Harris 1991; Rosenberg & Lewis 1995).

(c) “The progression of prefrontal cortex towards adult-like states is very gradual, extending into adolescence (sect. 6.2.1). That is correct; it extends even into adulthood (Huttenlocher & Dabholcar 1997 and Sowell et al. 1999). It is hardly contradictory for prefrontal cortex to undergo critical maturational changes between 8–12 months of age but not to be fully mature until many years later (see, e.g., Diamond 1996). I have never said, however, that “prefrontal maturity *alone* is the critical element” as Thelen et al. erroneously attribute to me. I have always maintained that prefrontal maturation plays a critical role, but not the only role.

5. Thelen et al. take strong exception to assertions of a division between what children “know” and what they can demonstrate they know. Their characterization that “one foundational assumption behind these dual-process (knowing vs. acting) accounts is that there lives, in the baby’s head, a creature that is smarter than

Table 2 (Diamond). *Close parallels between the performance of prefrontal monkeys and human infants on the A-not-B and delayed response tasks*

Experimental finding	Rhesus macaques with lesions of dorsolateral prefrontal cortex	7½–9 month old human infants
Succeed when there is no delay.	Harlow et al. 1952; Bättig et al. 1960; Goldman & Rostvold 1970	Harris 1973; Gratch et al. 1974
Succeed when allowed to continue to orient toward the correct well.	Bättig et al. 1960; Miles & Blomquist 1960; Pinsker & French 1967	Cornell 1979; Fox et al. 1979
If their attention is directed back to the A well after the hiding at B, they perform worse. Conversely, if B is covered after A, they perform better on the B trials.	Bartus & Levere 1977	Diamond et al. 1994; Harris 1973
Succeed if a landmark reliably indicates the reward's location.	Pohl 1973	Butterworth et al. 1982
Fail on reversal trials and on the trials immediately following reversals at delays of 2–5 sec.	Harlow et al. 1952; Bättig et al. 1960; Goldman & Rostvold 1970; Fuster & Alexander 1971; Diamond & Goldman-Rakic 1989	Evans 1973; Gratch et al. 1974; Diamond 1985
Succeed on the initial trials at A.	Diamond & Goldman-Rakic 1989	Diamond 1985
Show “deteriorated” performance at delays of 10 sec.	Diamond & Goldman-Rakic 1989	Diamond 1985
Try to self-correct after making an error.	Diamond & Goldman-Rakic 1989	Diamond 1985
Show the A-not-B error if the hiding locations differ in left-right location.	Harlow et al. 1952; Goldman & Rostvold 1970; Diamond & Goldman-Rakic 1989	Gratch & Landers 1971; Diamond 1985
Show the A-not-B error if the hiding places differ in up-down location.	Fuster 1980	Butterworth 1976

the body it inhabits unfortunate caricature. Sometimes infants know the right answer, and are attempting to demonstrate it, but the experimenter has set up a situation where the infant's immature motor abilities get in the way of the infant being able to complete the intended action. I have shown that although infants of 5–7 months understand the concept of contiguity, psychologists have mistakenly assumed they did not, because psychologists “asked” infants to demonstrate this knowledge in situations where infants' inability to precisely aim their reaches and their inability to inhibit the grasp reflex got in the way of demonstrating their cognitive competence (Diamond & Gilbert 1989; Diamond & Lee, in press). All of us have been in situations where we knew more than we could show at the moment. For example, suppose a person you have called very often changes her phone number; suppose even that the first few digits remain the same. You will often dial the old number, or at least begin dialing it. Sometimes that will be because you forgot that the number had changed (i.e., at that moment you didn't “know” the correct number). Sometimes, if you are like me, that will be because even though you go to the phone reminding yourself of the new number, you dial the old number anyway (i.e., your behavior was “captured” by a prepotent action tendency and did not accurately reflect what you “knew”). The more your working memory is taxed (by holding other things in mind or distractions), and/or the harder the prepotent tendency is to inhibit (the more often you called the old number recently, the more similar the beginnings of the two numbers), the more likely you are to make this error. When normal adults are distracted, stressed, tired, or not paying close attention they often make errors characteristic of frontally-damaged adults or frontally-immature children.

In quoting me concerning this (“Infants really know where the [object] is even when they reach back to where they last found it”). Thelen et al. omitted the critical modifier (“sometimes”). We have long known that caching and looking are coupled in infants; I doc-

umented that (Diamond 1983; 1988; 1991b), as have others (e.g., Bruner 1973). I have said and written repeatedly that it is rare to see a stark dissociation between where an infant is looking and reaching, just as it is rare to see a clear, full-blown surprise reaction in an infant to finding A empty when the toy was hidden at B. However, both of these behaviors, when they occur, provide a glimpse into the two abilities required by the A-not-B task – the ability to inhibit the prepotent tendency to reach back to A and the ability to hold in mind where the reward was last hidden.

Movement planning and movement execution: What is in between?

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Abstract: Although the model proposed by Thelen and co-authors provides a detailed explanation for the processes underlying reaching, many aspects of it are highly speculative. One of the reasons for this is our lack of knowledge about transformation of a hand movement plan into joint movements. The leading joint hypothesis (LJH) allows us to partially fill in this gap. The LJH offers a possible explanation for the formation of movement and how it may be represented in memory. Our explanation converges with the dynamic model described in the target article.

Thelen et al.'s model provides a logical scheme of the complicated processes involved in reaching in general, and in particular, in infants. However, many aspects of this model are hypothetical because many mechanisms underlying reaching are still largely unknown. In this commentary, we focus on the gap between what is