

*The highlight for May is by Dr. Robert Batsell, Jr., from the Department of Psychology at Kalamazoo College in Michigan. As Dr. Batsell notes in his highlight, his introduction to taste aversion learning began with his association with Mike Best at Southern Methodist University. Working with Dr. Best, Dr. Batsell became interested in taste potentiated odor aversion (TPOA), a phenomenon whereby the pairing of an odor/taste compound with toxicosis produces greater aversions to the odor than that produced by pairing the odor directly with toxicosis. Given that overshadowing might normally be expected under these conditions, these findings had major implications for the uniqueness of conditioned aversions and the manner by which this field differed from traditional learning theory. The early work on this topic in Best's laboratory set the stage for Dr. Batsell's extensive foray into characterizing this topic and exploring the mechanisms mediating its display. As Dr. Batsell describes in his highlight, over the past 15 years he and his colleagues have assessed the relative roles of within-compound conditioning and sensory and-gate channeling in mediating this unique form of learning. Although the initial assessments with TPOA learning appeared to support a role of within-compound conditioning, via a variety of procedures (e.g., inflation, extinction), Dr. Batsell determined that TPOA and within-compound conditioning could be dissociated, i.e., one could demonstrate evidence of within-compound conditioning without the establishment of TPOA, even using cues that have reliably produced TPOA in other assessments. Such findings have led Dr. Batsell to argue for a configural-elemental model to account for potentiation, a model that suggests a multifaceted basis for the learning and how such learning might be impacted by manipulations following conditioning. Dr. Batsell's work on the potentiation of odor aversion learning by tastes illustrates a systematic research effort that has parceled out experimentally the possible mediators of this unique phenomenon. This research (along with other creative work from his laboratory) is nicely summarized in his recent chapter "Mechanisms of overshadowing and potentiation in flavor aversion conditioning" (co-authored with Gayla Paschall) in Steve Reilly and Todd Schachtman's edited collection Conditioned Taste Aversion: Behavioral and Neural Processes (Oxford University Press, 2009).*

### *Chasing Potentiation*

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When I was a student at Southern Methodist University in the early 1980s, I began my studies in biology. During my first semester, the General Biology instructor announced that the Biology Department was hosting a lab tour open to all students who wanted to be involved in research. I attended the tour and marveled at the electron microscopes and endless rows of cell cultures, but ultimately wondered if anybody was working on the entire organism. The prompt reply was "We don't do that here, you should go talk to the

guy who works with rats in the psychology department.” As I crossed campus to meet Dr. Michael Best, I could not have imagined that these steps would set the direction for my professional career.

Even though I had not yet taken a psychology class, Mike Best gave me a chance. In retrospect, this probably had a lot to do with Mike’s own undergraduate experiences at Macalester College where his budding interests in psychology were supported. As noted by other contributors to this site like Mike Domjan and Andy Mickley, Mike Best did his graduate work at the University of Virginia under the direction of Dr. Phil Best. [It was merely coincidental that Mike and Phil shared the same surname, but Mike loved to say that his “real” name was Mike Grabowski, but Phil had such an influence on him, he legally changed his name to Mike Best. Mike told this story with such a straight face that many casual acquaintance of that time thought it to be true.] Mike’s graduate work focused on taste-aversion learning, with notable work especially in demonstrating that contextual cues can modulate taste-aversion learning (P.J. Best, M.R. Best, & Mickley, 1973) and that CTA could be used to study conditioned inhibition (Best, 1975). When I was invited to join the lab team they were eagerly exploring a newly reported phenomenon in taste-aversion learning—taste-potentiated odor aversion [TPOA]. TPOA is the increased aversion to an odor cue that is produced when the odor is presented in compound with a taste prior to illness compared to the odor aversion produced when odor alone precedes illness. In other words, the addition of the taste increases or *potentiates* the learning to the odor. This finding had captured the attention of associative learning theorists because all of the prominent formal models of learning at that time (and still today), like the Rescorla-Wagner model (1972), would predict that the presence of the taste should decrease or *overshadow* the learning to the odor. Thus, the very presence of potentiation was a direct challenge to these theories, and a viable mechanism for potentiation was warranted.

During the 1980s, two prominent models of potentiation were advanced. On one hand, John Garcia and his associates proposed the sensory and-gate channeling model (Garcia, Lasiter, Bermudez-Rattoni, & Deems, 1985). The *sensory and-gate channeling model* explains potentiation through the activation of two defense systems. First, the internal or gut defense system processes threats with ingestive consequences, and taste cues are selectively processed within this system. Second, the external defense system processes threats to the periphery of the organism (i.e., visual cues and auditory cues would be processed via the external defense system). Odor cues are unique in that they can be processed by either the internal or the external defense system. If the odor occurs alone it will be processed within the external defense system, but if the odor is presented in conjunction with a taste, it will be *gated* into the internal defense system. Once the odor has been admitted into the internal defense system, it will be processed like a taste, resulting in the significantly stronger odor-illness association.

Durlach and Rescorla (1980) proposed a different account, the *within-compound association model*. In this model, three associations that form during conditioning mediate potentiation: 1) a taste-illness association, 2) an odor-illness association, and 3) a taste-odor within-compound association. During subsequent odor testing, the odor can

activate the unconditioned stimulus representation both through the direct odor-illness association, and via the indirect odor-taste-illness association. In contrast, the significantly weaker odor aversion observed in the odor-alone control group occurs because this group only has the odor-illness association.

Throughout the 1980s, Mike Best's lab and many others conducted variations of these basic potentiation experiments to elucidate the mechanism of this phenomenon. Some studies yielded support for the sensory and-gate channeling model, whereas others favored the within-compound association model. Specifically, during my undergraduate years, Mike and his collaborators, like John Batson and Cindy Meachum, were exploring taste-mediated potentiation of contextual cues (Best, Batson, Meachum, Brown, & Ringer, 1985; Best, Brown, & Sowell, 1984; Best & Meachum, 1986). Based on my early experiences in the lab, I decided to go to graduate school and begin my own pursuit of potentiation.

#### *Naturally-Produced Odorous Emissions.*

During my graduate work at Texas Christian University, I worked with Dr. H. Wayne Ludvigson; Ludvigson had completed his Ph.D. with Dr. Kenneth Spence at the University of Iowa. Early in Wayne's research career he studied rats' approach speed on reinforced trials and nonreinforced trials. Not surprisingly, when the color of the runway signaled the goal event, rats would run fast in the presence of the S+ color and slowly in the presence of the S- color. Surprisingly, Wayne observed that the control rats, that always ran in the gray runway, also began to show the characteristic pattern of fast running on rewarded trials and slow running on nonreward trials. Notably, Wayne saw that the initial rats in the squad did not show this differential patterning, but only the later members in the squad showed knowledge of the goal events. After eliminating other possibilities, Wayne determined that rats emitted naturally produced odors when they found food (reward odor) or an empty food cup (nonreward odor), and that subsequent rats were using these odors as discriminative stimuli of the goal events.

When I joined the lab team, Wayne wanted to capitalize on my knowledge of flavor-aversion conditioning, and our initial work was a combination of our expertise as we explored aversive conditioning of these naturally produced odors (Batsell & Ludvigson, 1989). As my experience grew, Wayne gave me more latitude in designing the experiments, and I wondered whether a rat might emit a distinctive odor if it encountered an aversive flavor at the end of the runway. I trained rats to traverse a runway in anticipation of water reinforcement. After they had learned this task, I replaced the water with a flavored liquid that had been paired with illness. I still remember the initial test trial when the aversive flavor was in the goal. The first rats ran quickly down the runway, but the third rat stopped halfway down the runway, turned around, and started back to the start box. I knew at that very moment that I had gotten it right. We determined that the odor emitted in the presence of a signal of illness was different from the Reward Odor and the Nonreward Odor, so we termed it Aversion Odor (Batsell, Ludvigson, & Kunko, 1990). In some follow-up experiments as part of my dissertation, we tried to pair this naturally produced odor with taste to determine if this powerful

Aversion Odor might be able to potentiate the taste, but these stimuli would not combine in a synergistic manner. Thus, I recognized that I would need to go in another direction to understand potentiation effects.

### *Short-term and Long-term Retention Interval Effects.*

The next step of my professional journey took me back to SMU in 1989 when Mike Best recruited me to return in a faculty position. Mike recruited me to continue our research collaboration, and in particular, he was interested in some recent studies that had come out of Skip Spear's lab that were conducted by Phil Kraemer and the late Jim Miller (Kraemer, Lariviere, & Spear, 1988; Miller, Jagielo, & Spear, 1990). These studies showed recovery of responding to an overshadowed taste across a retention interval, but no recovery of an overshadowed odor. Mike and I thought it was curious that no potentiation was observed in these scenarios, and that the pattern of differences might be quite different if such a combination was used.

Our initial studies using the retention interval technique yielded surprising results. Coincidentally, Mike and I had gone to Fort Worth to watch Mauricio Papini deliver his job talk at TCU. Afterwards, Wayne treated the three of us to Mexican food in Fort Worth's hospital district. We had just collected our first batch of retention interval data, and as we passed graphs around the table, it was clear that we had replicated some of the 21-day effects reported by Spear's lab, but we found surprising differences at the 1-day test. Specifically, the single-element taste aversion was significantly weaker at 1 day than at 21 days.

Over the next few years, Mike and I, along with a number of students, showed that taste aversions are paradoxically weak if tested 1 day after conditioning relative to tests conducted 3, 7, 14 or 21 days after conditioning (Batsell & Best, 1992a, 1992b, 1993). This *short-term retention interval effect* is also found following nonpharmacological, rotationally-induced illness (Batsell & Pritchett, 1995). It appears that this phenomenon occurs because of the surprisingness of the initial illness experience (Batsell & Best, 1994; Batsell & George, 1996). In addition to short-term retention interval effects, our work also confirmed the *long-term retention interval effects*. Yet, in our studies, we found that both overshadowing and potentiation appear to increase at long-retention interval. Moreover, these long-term retention interval effects only occur following simultaneous compound conditioning—they are absent if sequential compound conditioning occurs. Although we did not fully understand these results in the late 1990s, they are very consistent with our current thinking of configural representations in potentiation (Batsell & Paschall, 2009).

### *Augmentation*

In 1996, Mike Best died unexpectedly, and John Batson while visiting Mike's family spent a week at my house. During his stay, John and I discussed a potential collaboration that involved using the blocking design to explore compound conditioning and the short-

term retention interval effects. John contacted me after he completed the first study to let me know that there was no evidence of blocking. In fact, he was seeing the exact opposite of blocking. Excited by the possibility of having a new means of studying synergistic conditioning, over the next few years, both of our labs put all of our resources towards understanding this effect. If a taste (A) is paired with illness in Phase 1, and then taste + odor (AX) are paired with illness in Phase 2, enhanced odor (X) conditioning is recorded. Similarly, if the odor cue is the preconditioned cue, compound conditioning of that odor with a novel taste cue will augment learning of the taste aversion.

For the longest time, we debated something as trivial as the name for this effect. Personally, I had taken to calling the phenomenon “Big Ass Conditioning” and I had a number of files named B.A.C. on both my virtual and literal desktops. Once we were confident in the reliability of the phenomenon, and that it was not simply a variation of potentiation, John eventually convinced me that we should choose a moniker that would be suitable for publication. We spent the better part of one afternoon at my house in Dallas debating possibilities. Finally, discounting the term’s connotations with elective cosmetic surgery, we settled on augmentation. Subsequently, John and I had great success in examining taste + odor interactions using this design (Batsell & Batson, 1999; Batsell, Paschall, Gleason & Batson, 2001; Batson & Batsell, 2000), and the insights produced by this other form of synergistic conditioning has come to shed new light on the old problem of potentiation.

#### *Return to Potentiation*

In 1999, I took the department chair position at Kalamazoo College in Kalamazoo, Michigan—quite a climate change from my Texas roots! As SMU had decided to shut their animal labs, I was allowed to take most of the taste-aversion equipment and cultural regalia to Michigan, so much of the SMU taste aversion tradition continues today. Since 2001, my research has primarily focused on identifying the mechanism of potentiation; some of this work has been in collaboration with John Batson and all of it has involved Kalamazoo College undergraduates. During 2001, I was invited to write a “State of the Science” review of the synergistic conditioning literature for the journal *Brain & Mind*. This provided the impetus for me to return and re-read the potentiation literature. As I worked on this manuscript along with Kalamazoo undergrad Aaron Blankenship, I was able to see old data from a new perspective, particularly the support for the within-compound association model. This led my lab to initiate a series of experiments to pursue untested or rarely tested predictions of the within-compound association model. In the 2003 report of Batsell, Trost, Cochran, Blankenship, and Batson, we added a second conditioning phase (inflation) to the standard potentiation procedure. The results of these four experiments provided strong evidence against the sensory and-gate channeling hypothesis, and initially gave the impression of support for the within-compound association theory. Yet, subsequent reflection on these results, and a few curious anomalies in the data, revealed the results could also be used to *disconfirm* the within-compound association model.

These inflation data have since led to a series of research reports in which we present data that demonstrate that within-compound associations are not sufficient to produce taste-mediated odor potentiation (Batsell & Paschall, 2009; Batson et al., 2008; Schnelker & Batsell, 2005; Trost & Batsell, 2004). The data that most conclusively show that within-compound associations are not sufficient to produce potentiation come from a 2008 report that again includes data from both Furman University and Kalamazoo College. A few years ago, as we were in the midst of conducting various augmentation studies (A+/AX+) and inflation (AX+/A+) experiments focused on the X cue, John decided to use these similar designs and test the A cue. The summer that John started these studies, a visiting Baylor University undergraduate student, Joaquin Lugo, assisted him. Interestingly, the data revealed that the odor aversion following AX+/A+ conditioning was consistently stronger than the odor aversion following A+/AX+ conditioning. As it remains a real tongue twister to state these conditioning procedures, and to distinguish this effect from similar experiments we had conducted, we took to calling this “the Joaquin effect” (it should be clear that we have issues naming things!). Shortly thereafter, we presented the initial data at Psychonomics, and as he has so many times, Mike Domjan made the keen observation that the A+ trial prevents the formation of the within-compound association during the subsequent AX+ trial, but the within-compound association is able to form with AX+/A+ conditioning. We subsequently performed the experiment to test this prediction, and contrary to this prediction, we found evidence of a within-compound association in the A+/AX+ group that did not show potentiation (see Experiment 4, Batson et al., 2008). For that reason, we were confident in stating that even with taste and odor cues that have been shown to yield potentiation, the within-compound association is not sufficient to produce potentiation.

Instead, our data have pointed to a configuration process as being the mechanism of TPOA, and I have proposed the configural-elemental model to account for TPOA (for more information, see Batsell & Paschall, 2009). In short, during the initial presentation of the taste + odor compound, the organism does not represent these cues as separate elements [T and O], but instead they are represented as a single, salient stimulus [TO]. Because of the enhanced salience of this compound, conditioning to its representation will be stronger than to either element presented by itself. During testing with the odor, the enhanced conditioning to the compound will counteract any generalization decrement due to the absence of the taste, and a strong odor aversion will be recorded. If manipulations of the taste or odor are performed after compound conditioning, this will activate the elemental (or within-compound) association between the taste and odor, and responding will reflect these experiences. The configural-elemental model reflects the latest step in my pursuit of potentiation. It remains to be seen where future research will next lead the journey....

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