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Abstract

Huttenlocher, Hedges, and Vevea (2000) (Why do categories affect stimulus judgment? *Journal of Experimental Psychology: General*, 129, 220-241) introduce the category adjustment model (CAM), which posits that participants imperfectly remember stimuli in serial judgment tasks. In order to maximize accuracy, CAM holds that participants use information about the distribution of the stimuli to improve their judgments. CAM predicts that judgments will be a weighted average of imperfect memories of the stimuli and the mean of the distribution of stimuli. Huttenlocher, Hedges, and Vevea (2000) report on three experiments and the authors conclude that CAM is "verified." We attempt to replicate Experiment 3 from Huttenlocher et al. (2000). We analyze judgment-level data rather than averaged data. We find evidence of a bias toward a set of recent stimuli rather than a bias toward the running mean. We also do not find evidence of the joint hypothesis that the participants learned the distribution of stimuli and employed this information in their judgments. The judgments in our dataset are not consistent with CAM. We discuss how the apparent defects in HHV went unnoticed and how such mistakes can be avoided in future research. Finally, we hope that the techniques that we employ will be used to test other datasets that are currently regarded as consistent with CAM or any Bayesian model of judgment.

Keywords: judgment, memory, category adjustment model, central tendency bias, recency effects, Bayesian judgments

It has been known for some time that when participants perform magnitude judgments, there is a bias toward the mean (Hollingworth, 1910; Poulton, 1979). For instance, in the judgment of the length of lines, longer lines tend to be underestimated and shorter lines tend to be overestimated. This effect is sometimes referred to as the *central tendency bias*.[1]

Huttenlocher, Hedges, and Vevea (2000), hereafter referred to as HHV, propose that participants imperfectly remember and perceive the stimuli. Due to these imperfections, HHV posit that participants use information about the distribution of the stimuli to improve their judgments. HHV refer to this as the category adjustment model, hereafter referred to as CAM. CAM predicts that judgments will be a weighted average of imperfect memory of the stimuli and the mean of the distribution (category) of stimuli. In the description of CAM, the authors state, "This process can be likened to a Bayesian statistical procedure designed to maximize the average accuracy of estimation" (p. 220). Since judgments will be an optimal weighted average of the imperfect memory of the stimulus and the mean of the distribution, CAM offers a Bayesian explanation of the central tendency bias.

HHV describe one prediction of CAM as the following, "…the concentration of instances in the category should affect the variability of stimulus estimates. In particular, the variability of estimates of all categorized stimuli should be less when the prior distribution (category) is more tightly clustered; this prediction, which follows from our Bayesian model, is not easily derived from other sets of assumptions" (p. 224).

In order to test the predictions of CAM, HHV perform three experiments. Participants perform serial judgment tasks on the fatness of computer generated images of fish (Experiment

[1] The representativeness heuristic (Kahneman and Frederick, 2002; Kahneman and Tversky, 1973) makes similar predictions.

1), the greyness of squares (Experiment 2), and the lengths of lines (Experiment 3). In each of these experiments, participants perform these judgments under four different distributions of stimuli, which exhibit different means and standard deviations. HHV analyze all three experiments in a similar manner: they examine the pattern of bias, the shape of the bias, and the standard deviations of the bias. These analyses were performed on data that had been averaged across trials and averaged across sets of previous stimuli. HHV conclude by stating, "The experiments verified that people's stimulus estimates are affected by variations in a prior distribution in such a manner as to increase the accuracy of their stimulus reproductions"[2] (p. 220).

CAM has had a large impact on the literature, which includes topics such as the perception of neighborhood disorder (Sampson & Raudenbush, 2004), speech recognition (Norris & McQueen, 2008), overconfidence (Moore & Healy, 2008), categories of sound (Feldman, Griffiths, & Morgan, 2009), spatial categories (Spencer & Hund, 2002), spatial recall (Schutte & Spencer, 2009; Spencer & Hund, 2003; Hund & Spencer, 2003; Crawford & Duffy, 2010; Holden, Curby, Newcombe, & Shipley, 2010), visual illusions (Crawford, Huttenlocher, & Engebretson, 2000), delayed comparison of magnitude (Ashourian & Loewenstein, 2011), judgments of color (Bae, Olkkonen, Allred, & Flombaum, 2015; Olkkonen & Allred, 2014; Olkkonen, McCarthy, & Allred, 2014; Persaud & Hemmer, 2014), judgments of the size of familiar objects (Hemmer & Steyvers, 2009a, 2009b), judgments of the heights of people (Twedt, Crawford, & Proffitt, 2015), judgments of likelihood (Hertwig, Pachur, & Kurzenhäuser, 2005), facial recognition (Corneille, Huart, Becquart, & Brédart, 2004; Roberson, Damjanovic, & Pilling, 2007; Young, Hugenberg, Bernstein, & Sacco, 2009), judgments of facial expressions

[2] We also note that "verified" is a word that appears to be inconsistent with Bayesian inference following an experiment with a limited number of participants performing judgments on a limited set of stimuli.

(McCullough & Emmorey, 2009; Fugate, 2013; Corbin, Crawford, & Vavra, 2017), the perception of drink flavor (Woods, Poliakoff, Lloyd, Dijksterhuis, & Thomas, 2010), and judgments across different domains (Petzschner, Glasauer, & Stephan, 2015). Needless to say, CAM has had a large impact.

However, despite the assertion of HHV to the contrary, one simple alternate hypothesis is that there is a bias toward a set of recent stimuli rather than a bias toward the mean of the distribution.

While we are not able to observe the prior beliefs of the participant, well-known results show that, under mild assumptions, Bayesian learners will have beliefs that converge to the truth (Savage, 1954; Blackwell & Dubins, 1962).[3] Therefore, in the analysis that follows, we use the running mean as a proxy for the participant's perception of the mean of the distribution.

When considering judgments drawn from two different distributions, the sets of recent stimuli are simply noisy versions of the running mean. As such, tests involving averaged data will not be able to distinguish between the hypothesis that there is a bias toward the running mean and the hypothesis that there is a bias toward a set of recent stimuli. Unfortunately, HHV only analyze averaged data and therefore these two hypotheses are not distinguishable. [4] In this paper, we explore the extent to which the data can be explained by this alternate hypothesis.

[3] These references have been in the psychology literature since Edwards, Lindman, and Savage (1963). On page 201, the authors state, "From a practical point of view, then, the untrammeled subjectivity of opinion about a parameter ceases to apply as soon as much data become available. More generally, two people with widely divergent prior opinions but reasonably open minds will be forced into arbitrarily close agreement about future observations by a sufficient amount of data. An advanced mathematical expression of this phenomenon is in Blackwell and Dubins (1962)."

[4] The dangers of analyzing averaged data have been known for some time (Sidman, 1952; Hayes, 1953; Estes, 1956; Siegler, 1987) and such concerns even appear in the recent judgments literature (Cassey, Hawkins, Donkin, & Brown, 2016; Hemmer, Tauber, & Steyvers, 2015).

Further, CAM is a mathematical model, and this allows the researcher to devise non-obvious predictions that are consistent with the model. As Bayesian learners will have beliefs that converge to the true distribution, we should observe learning across trials. In this paper, we subject the data to several tests of learning.

Similar to HHV, Duffy, Huttenlocher, Hedges, and Crawford (2010) claim that the results of their experiments are consistent with CAM. Duffy and Smith (2017) reexamine the data from Duffy et al. (2010) by analyzing judgment-level data rather than analyzing averaged data. Duffy and Smith do not find evidence of CAM in the Duffy et al. data. In particular, Duffy and Smith find that there is a bias toward recent stimuli rather than toward the running mean of the distribution. Duffy and Smith also test whether there is evidence of learning the across trials and they fail to find evidence of learning. Duffy and Smith conclude that the Duffy et al. judgments are not consistent with CAM. In this paper we perform a similar exercise.

Our Replication of Experiment 3 in HHV

Due to changing practices in archiving since the 1990s, the authors of HHV were not able to locate their datasets. Experiment 3 in HHV is perhaps easier to replicate than Experiments 1 and 2. Further, since Duffy and Smith (2017) and Allred, Crawford, Duffy, and Smith (2016) examined judgments of line length, here we focus on HHV Experiment 3. We attempted to reproduce the experimental conditions as well as possible from the description in the paper. We will say more on this below.

Description of Methods

Stimuli were presented electronically using the E-Prime 2.0 software (Psychology Software Tools, Pittsburgh, PA). The sessions were performed on standard 20 inch (51cm) HP monitors. E-Prime imposed a resolution of 1024 pixels by 768 pixels.

There were 4 conditions in which participants viewed and reproduced a series of lines randomly drawn from different frequency distributions. In each condition, participants completed 192 trials. In each trial, participants saw a *target* line on a screen for 2 seconds. The screen then went blank for 1.2 seconds and an initial adjustable line appeared that was 8 pixels (0.35cm) in length. Participants manipulated the length of this line until they judged its length to be that of the target line. This was accomplished by using the “S” key (which made the line decrease in length) or the “L” key (which made the line increase in length). Once satisfied that their response line was equal to the length of the target line, participants pressed ENTER and the next trial commenced. We refer to this response as the *response* line.

In the *short* treatment, participants viewed and reproduced target lines ranging in 16 pixel (0.7 cm) increments from 48 pixels (2.1 cm) to 224 pixels (9.8 cm) in length. Each of the 12 distinct line lengths was estimated once in 16 blocks. In the *long* treatment, participants viewed and reproduced targets ranging in 16 pixel increments from 240 pixels (10.5 cm) to 416 pixels (18.3 cm). Each of the 12 lengths was estimated once in 16 blocks. In the *uniform* treatment, participants viewed targets ranging in 16 pixel increments from 48 to 416 pixels. Each of the 24 lengths were estimated once in 8 blocks. Finally, in the *normal* treatment, participants viewed the 48 and 416 pixel lines once, the 64, 80, 384, and 400 pixel lines twice, the 96, 112, 352, and 368 pixel lines 3 times, the 128, 144, 320, and 336 pixel lines 4 times, the 160, 176, 228, and 304 pixel lines 5 times, the 192, 208, 256, and 272 lines 6 times, and the 224 and 240 lines 7 times.

This constituted a single block. Upon completion, this block was repeated once more. See Figure 1 for a graph summarizing these distributions.

<<Figure 1 about here>>

The thickness of each of these lines was 0.36 cm. In all four conditions, participants estimated a total of 192 lines, and there were no breaks between blocks.

The participants were given partial course credit for their participation. There were 10 participants in the Normal treatment, 9 in the uniform treatment, 11 in the short treatment, and 11 in the long treatment.[5] With 41 participants each offering 192 judgments, we have a total of 7872 observations. We exclude 121 (1.54%) responses that are more than 3 standard deviations from the target. This implies a total of 7751 observations. The study was approved by the Rutgers University Institutional Review Board.

A Discussion of the Design of Our Replication

We discuss the ways in which our replication possibly deviates from Experiment 3 in HHV. One way in which this occurs is because the description of the design is not clear to us. For example, HHV report an inconsistent number of trials in the normal treatment. On page 232 the authors report numbers of trials within a block that sums to 106, which implies a total of 212 trials.[6] This contrasts with the other three treatments in Experiment 3 that have a total of 192 trials. The authors do not offer a justification for having conditions with unequal numbers of trials across treatments. In addition, the caption for Figure 4 in HHV, which provides histograms of the frequency distributions in all three experiments, suggests a number of trials that is

[5] HHV had 10 participants in each of the four treatments.

[6] On page 232, HHV write, “In the normal conditions, the distribution of stimuli within each block was as follows: once at 45 and 390; twice at 60 and 375; three times at 75, 90, 345, and 360; four times at 105, 120, 315, and 330; five times at 135, 150, 285, and 300; six times at 165, 180, 255, and 270; and seven times at 195, 210, 225, and 240.”

different from that reported in the verbal description of Experiment 3. We decided that reporting 212 trials was likely an error and we designed the normal treatment to be consistent with those in Experiments 1 and 2, by having 192 trials.

On the other hand, some differences are due to the constraints imposed by our computer program. HHV has stimuli sizes that have an odd number of pixels, whereas we are constrained to have only an even number of pixels. As a result, HHV have lines that range in 15 pixel (0.5 cm) increments from 45 pixels (1.5 cm) to 390 pixels (13 cm). By contrast, our experiment has lines that range in 16 pixel increments from 48 pixels to 416 pixels.[7]

One additional difference relates to the initial adjustable line. HHV report an initial adjustable line of 2 pixels. However, this is shorter than the minimum line that we could produce on E-Prime. Therefore our initial adjustable lines are 8 pixels.

Finally, we used modern, 20 inch LCD screens whereas HHV used smaller CRT displays. The displays used in HHV are no longer commercially available.

We also concede that there are possibly differences between HHV Experiment 3 and our replication regarding the brightness of the display, the velocity with which the adjustable line increased or decreased in length, the distance between the participant and the display, etc. We also note that there could be differences between the sets of participants. However, it is our opinion that these differences are not significant enough to affect our results.

Results

Summary statistics

In order to compare our data with the data obtained by HHV, we look to their reported summary statistics. We define the *response bias* to be the response minus the target. HHV report

[7] We also note that the HHV lines had a thickness of 0.23 cm

the standard deviations of the response bias[8] in 6 different settings: the central 10 targets in the normal treatment, the central 10 targets in the uniform treatment, the short treatment, the shortest 12 targets in the uniform treatment, the long treatment, and the longest 12 targets in the uniform treatment. We list the standard deviations reported by HHV and the standard deviations in our data in the analogous settings. As does HHV, we also test for the differences between the categories. HHV performs t-tests of the differences of the natural logs of the standard deviations.[9] We perform the identical analysis on our data. We report both the results of HHV and our results. We note that the reported natural logs of the standard deviations are calculated, not as the log of the average across targets, but as the average of the natural logs of the standard deviation within each target. We summarize this in Table 1.

Table 1: Standard deviations of response bias in HHV and our data

	SD_{HHV}	$\ln SD_{HHV}$	SD_{ours}	$\ln SD_{ours}$	Obs_{ours}	t_{HHV}	p_{HHV}	t_{ours}	p_{ours}
Normal, central 10	50.92	3.926	29.82	3.385	1155				
Uniform, central 10	60.27	4.083	34.75	3.544	711				
difference	-9.35	-0.157	-4.93	-0.159		-2.31	.033	-2.75	.013
Uniform, shortest 12	39.22	3.618	31.03	3.432	860				
Short	38.70	3.624	21.04	3.025	2105				
difference	0.52	-0.006	9.99	0.407		0.05	.960	6.18	<.001
Uniform, longest 12	77.66	4.345	40.27	3.692	828				
Long	52.31	3.948	39.88	3.683	2066				
difference	25.35	0.397	0.39	0.009		7.19	<.001	0.29	.78

Notes: We provide the standard deviations reported by HHV in Experiment 3 (Table 3) and the standard deviations in our data within the same setting. We report the average of the natural logs of the standard deviations for HHV and our data. We report the number of our observations within each category. We report the t-statistic of the difference between the natural logs and the p-value of a two tailed test, as reported in HHV and that for our data. The tests involving the normal and uniform distributions have 18 degrees of freedom. The remaining tests have 22

[8] HHV refer to this variable simply as *bias*. However, we also examine biases with different definitions, so we employ the term response bias.

[9] Given their reported degrees of freedom, it seems as if HHV conducted the tests assuming an equal variance between the samples. The reader might be concerned about the appropriateness of this. Our results are not changed when we conduct paired t-tests or unpaired t-tests that do not assume an equal variance.

degrees of freedom. Although we note that HHV apparently incorrectly report 18 degrees of freedom for the short tests in Tables 1, 2, and 3.

Similar to HHV, we find differences in two out of the three tests. The results are similar when we perform the tests on the raw standard deviations rather than the natural log of the standard deviations.[10]

We also note that our participants were not less accurate than the HHV subjects. We are therefore confident that the results that follow are not driven by excessively inattentive or inaccurate participants.

On their decision to restrict attention to the central 10 targets for the test of the difference between the normal and uniform treatments, HHV write, "We should restrict ourselves to a region within the categories where the certainty of membership is equal. Because the certainty that a stimulus is in the category decreases more markedly near the boundaries for a normal distribution than for a uniform distribution, we elected to compare standard deviations over a central region where participants were quite certain of the category for both distributions. Hence, we focused our attention on the 10 most central stimuli."[11]

We do not find this to be a compelling argument to exclusively examine the central 10 targets. We decided to investigate this matter by performing tests on a range of restricted target values. We perform a test on all of the data (24 targets), only the central 22 targets, only the central 20 targets, and so on, until only the central 6 targets. We perform these tests on both the raw data and the logged data. We summarize our analysis in Table 2.

[10] We find a significant difference between the normal and the uniform treatments ($t(18) = 2.75$, $p = .013$) and a significant difference between the short treatment and the short lines in the uniform treatment ($t(22) = 6.73$, $p < .001$). However, we do not find a significant difference between the long treatment and the long lines in the uniform treatment ($t(22) = 0.29$, $p = .77$).

[11] Page 229.

Table 2: Various t-tests for differences in standard deviations by target restrictions

Central	24	22	20	18	16	14	12	10	8	6
Normal	32.88	32.69	32.21	31.68	31.16	30.87	30.57	29.82	29.68	29.90
Uniform	35.65	35.53	35.46	35.34	35.03	34.85	34.64	34.75	34.61	34.88
t-statistic	-1.64	-1.61	-1.91	-2.31	-2.49	-2.34	-2.41	-2.75	-2.56	-2.61
p-value	.107	.114	.064	.027	.018	.027	.025	.013	.023	.026
ln Normal	3.477	3.470	3.457	3.443	3.428	3.418	3.409	3.385	3.382	3.394
ln Uniform	3.562	3.560	3.560	3.558	3.550	3.545	3.540	3.544	3.539	3.547
t-stat	-1.76	-1.75	-2.05	-2.39	-2.51	-2.39	-2.41	-2.75	-2.53	-2.63
p-value	.086	.088	.047	.022	.017	.024	.025	.013	.024	.025

Notes: We restrict attention to various central target lengths. For each, we list the average of the normal and uniform treatments, and the t-statistics and the p-values associated with a two-tailed test. The upper panel shows this for raw standard deviations and the lower panel for the natural log of the standard deviations.

We see that the p-value attains its smallest value at the restriction to only the central 10 values. We further note that our p-values are .013 for both specifications in this restriction, whereas it is .033 for HHV. We do not know if the HHV data exhibit a similar relationship. We admit that 10 is a round number and this could have been the basis for the decision to report the test restricted to only the central 10 targets. However, it is curious that only a single specification[12] is reported by HHV (the central 10 targets) and, in our data, this happens to be restriction with the lowest p-value.

While HHV do not report the mean response bias, ours (*M* = -10.88, *SD* = 38.71) is significantly different from zero (*t*(7750) = -24.74, *p* < .001). As Allred et al. (2016) discovered that the length of the initial adjustable line affects judgments, we conjecture that these underestimates are due to the short initial adjustable line.

Next we examine the mean of the response bias within treatments across targets. Figure 2, offers a summary of this data.

<<Figure 2 about here>>

[12] We use the term *specification* to refer to the complete set of assumptions in the analysis, including the functional form, the choice of explanatory variables, the assumptions regarding the error term, and the set of data under consideration.

CAM predicts that each treatment will have a mean response bias of zero at the means of their distributions. However, this appears to not be the case in our data. However, we find that the mean response bias is negative in the uniform, normal, and long treatments.[13] When we restrict attention to the central two values in every treatment[14] we see similar results.[15] There seems to be a particularly stark difference in the response bias of the short and long treatments. Since the short treatment distribution and the long treatment distribution are identical (same number of targets, same frequencies, etc.) with the exception of the specific target sizes, this is a particularly troubling difference. We find that the mean response bias of judgments in the short treatment is significantly different from that in the long treatment, $t(22)= -3.99$, $p < .001$. This is robust to the specification of the test.[16] These significant relationships are clearly not consistent with CAM.

We do not know if these features exist in the HHV data, as the authors do not report any of these tests. Rather, the authors merely assert, "For all conditions, responses are shrunken toward a central value. There is overestimation of short line lengths and underestimation of long lengths" (p. 232). The first sentence is curiously imprecise. The second sentence is simply a restatement of the central tendency bias. Clearly it would have been preferable for HHV to report any subset of the tests that we perform. Below we will say more about the differences between the response biases in the short and long treatments.

[13] Mean response bias is significantly less than zero in the normal treatment ($M = -10.40$, $SD = 38.09$, $t(1891) = -11.88$, $p < .001$), the uniform treatment ($M = -10.72$, $SD = 45.41$, $t(1687) = -9.69$, $p < .001$), and the long treatment ($M = -21.97$, $SD = 42.67$, $t(2065) = -23.41$, $p < .001$), but not in the short treatment ($M = -0.55$, $SD = 23.40$, $t(2104) = -1.08$, $p = .28$).

[14] Here we only include targets 224 and 240 in the normal and uniform treatments, targets 320 and 336 in the long treatment, and targets 128 and 144 in the short treatment.

[15] Restricted to the central two values, mean response bias is significantly less than zero in the normal treatment ($M = -10.27$, $SD = 27.65$, $t(278) = -6.20$, $p < .001$), the uniform treatment ($M = -9.61$, $SD = 32.89$, $t(144) = -3.51$, $p < .001$), and the long treatment ($M = -20.80$, $SD = 38.17$, $t(342) = -10.10$, $p < .001$), but not in the short treatment ($M = 1.31$, $SD = 21.73$, $t(351) = 1.13$, $p = .26$).

[16] We conduct a paired t-test ($t(11) = -10.85$, $p < .001$), an unpaired t-test that does not assume an equal variance ($t(18)= -3.99$, $p < .001$), and a t-test that does not assume an equal variance over all observations ($t(3191.3) = -20.05$, $p < .001$), and the results are not changed.

Repeated measures regressions for running mean

CAM asserts that there is a bias toward the running mean of the stimulus sizes. Here we explore whether we find evidence of this. We define the *running mean* variable to be the mean of the targets that the participant has viewed in the previous trials. We conduct regressions with target and running mean as independent variables and response as the dependent variable.

In order to account for the lack of independence between two observations associated with the same participant, we employ a standard repeated measures technique. We assume a single correlation between any two observations involving a particular participant. However, we assume that observations involving two different participants are statistically independent. In other words we employ a repeated measures regression with a compound symmetry covariance matrix.[17]

We restrict each of the regressions to a distribution treatment.[18] Since there is not a running mean on the first trial, we analyze data from trials 2 to 192.[19] These regressions are summarized in Table 3.[20]

[17] We include the repeated measures because it is a better model. However, the results without repeated measures are qualitatively similar to those with repeated measures, in this and in subsequent analyses.

[18] The pooled analysis appears in the "None" specification of Table 5.

[19] Regressions that analyze data from trials 2 through 192 have 7713 observations. The total 7751 minus 41 subjects making judgments on the first trial, however 3 first trial judgments are excluded due to their inaccuracy.

[20] Table 3 and the regression tables that follow are not consistent with the APA format for regressions. However, the APA format makes it difficult to display multiple specifications because the coefficient estimates and the standard errors are listed in separate columns. Since we prefer to display multiple specifications in each table, we present the regressions in a format, standard in other fields, with a regression in each column.

Table 3: Random-effects repeated measures regressions of the response variable

	Normal	Uniform	Short	Long
Target	0.766***	0.753***	0.833***	0.730***
	(0.008)	(0.008)	(0.008)	(0.014)
Running mean	0.149*	0.083	-0.009	0.060
	(0.068)	(0.059)	(0.080)	(0.122)
-2 Log L	18290.1	16703.6	18746.8	20433.3
Observations	1882	1680	2095	2056

Notes: We provide the coefficient estimates with the standard errors in parentheses. We examine trials 2 through 192. We do not provide the estimates of the intercepts or the covariance parameters. † indicates significance at $p < .1$, * indicates significance at $p < .05$, and *** indicates significance at $p < .001$. -2 Log L refers to negative two times the log-likelihood.

As would be expected, target is significantly related to response in every treatment. However, running mean is significant only in the normal treatment. In other words, in these specifications, without any other independent variable, there is only weak evidence of a bias toward the running mean.[21]

Repeated measures regressions for preceding target lines

Here we explore a simple alternate hypothesis to CAM: participants are affected by previous targets rather than the running mean. We note that recency effects and sequential effects have been studied in the literature.[22] We perform an analysis similar to that summarized in Table 3 but we include an additional independent variable: the previous target. These regressions are summarized in Table 4.[23]

[21] This is robust to the specification of the error term. See Table A1 in the Supplemental Online Appendix.

[22] See Jesteadt, Luce, and Green (1977), Staddon, King, and Lockhead (1980), Petzold (1981), Laming (1984), DeCarlo and Cross (1990), Choplin and Hummel (2002), Stewart, Brown, and Chater (2002), Petzold and Haubensak (2004), Wilder, Jones, and Mozer (2009), Yu and Cohen (2009), Jones, Curran, Mozer, and Wilder (2013).

[23] The pooled analysis appears in the “Prec 1” specification of Table 5.

Table 4: Random-effects repeated measures regressions of the response variable

	Normal	Uniform	Short	Long
Target	0.766***	0.753***	0.835***	0.735***
	(0.008)	(0.008)	(0.008)	(0.014)
Running mean	0.104	0.051	-0.097	-0.022
	(0.069)	(0.060)	(0.080)	(0.123)
Previous target	0.030***	0.025**	0.053***	0.058***
	(0.008)	(0.008)	(0.008)	(0.014)
-2 Log L	18284.3	16701.3	18715.3	20423.0
Observations	1882	1680	2095	2056

Notes: We provide the coefficient estimates with the standard errors in parentheses. We examine trials 2 through 192. We do not provide the estimates of the intercepts or the covariance parameters. † indicates significance at *p* < .1, ** indicates significance at *p* < .01, and *** indicates significance at *p* < .001. -2 Log L refers to negative two times the log-likelihood.

We note that running mean is not significant in any of the treatments. By contrast, previous target is significant at .01 in each treatment.[24] Finally, by comparing Tables 3 and 4, we note that the coefficient estimates for target are relatively unaffected by including previous target.

We also explore whether including additional sets of recently viewed stimuli can help predict response. As the analysis above, we include a specification that has an independent variable that is the preceding target line, which we refer to as *Prec 1*. We also calculate the average of the preceding 3, the preceding 5, and the preceding 10 target lines. We refer to these specifications, respectively, as *Prec 3*, *Prec 5*, and *Prec 10*. In order to maximize our data, Prec X is calculated as the mean of as many available previous targets as possible, but constrained to not be more than X. Our analysis below considers each of these 4 specifications for the preceding target line variables. We refer to this set of variables as *preceding targets*. We also include a specification without any information about the previous targets, which we label as *None*. Finally, because the results of Tables 3 and 4 suggest that there are differences among the

[24] This is robust to the specification of the error term. See Table A2 in the Supplemental Online Appendix.

treatments, we estimate a dummy variable for each treatment. These regressions are summarized in Table 5.

Table 5: Random-effects repeated measures regressions of the response variable

	None	Prec 1	Prec 3	Prec 5	Prec 10
Target	0.765***	0.766***	0.766***	0.766***	0.766***
	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)
Running mean	0.0870*	0.0383	0.0274	0.0438	0.0293
	(0.0368)	(0.0372)	(0.0384)	(0.0398)	(0.0435)
Preceding targets	-	0.0343***	0.0444***	0.0331**	0.0478*
		(0.0045)	(0.0084)	(0.0117)	(0.0193)
-2 Log L	74784.3	74734.9	74763.9	74783.3	74784.2

Notes: We provide the coefficient estimates with the standard errors in parentheses. We examine trials 2 through 192. We do not provide the estimates of the intercepts, the treatment dummies, or the covariance parameters. All regressions have 7713 observations. † indicates significance at $p <$ .1, * indicates significance at $p < .05$, ** indicates significance at $p < .01$, and *** indicates significance at $p < .001$. -2 Log L refers to negative two times the log-likelihood.

In the specification without any information about previous lines, running mean is significant. However, in each of the specifications that include information about previous stimuli, running mean is not significant, whereas preceding targets are significant. This suggests that the preceding lines are much better predictors of responses than the running mean.[25]

Analysis of simulated data consistent with a key feature of CAM

Given the stark results above, a researcher might worry that our techniques are not sufficiently sensitive to detect evidence of CAM. In particular, a researcher might note that the standard deviation of running mean decreases across trials and this might prevent a satisfactory inference of the running mean coefficient. In order to investigate this matter, we simulated a simple dataset that is consistent with a key feature CAM and has parameters similar to that found in our data. We took the sequence of targets and added to each a normally distributed noise term, with a zero mean and a standard deviation of 25 pixels. We refer to the sum of target and the noise as the *memory* variable. We then define the *response25* variable to be the weighted average

[25] This is robust to the specification of the error term. See Table A3 in the Supplemental Online Appendix.

of memory and running mean. Although our analysis above suggests that an increase of the running mean by 1 pixel would lead to a larger response by .087 pixels, here we use a weight of .08:

$$\text{Response25} = .92(\text{Memory}) + .08(\text{Running mean}).$$

These simulated judgments are consistent with a key feature of CAM in that response25 is biased toward running mean but not toward recent lines. Additionally, there is a slightly lower weight on running mean than in the original dataset. Therefore, detecting a relationship between running mean and response is slightly more difficult in our simulated data than in the original data. We perform the identical analysis to that performed in Table 5, which we summarize in Table 6.

Table 6: Random-effects repeated measures regressions of the simulated response25 variable

	No Prec	Prec 1	Prec 3	Prec 5	Prec 10
Target	0.921***	0.921***	0.921***	0.921***	0.921***
	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)
Running mean	0.103***	0.102***	0.104***	0.116***	0.107***
	(0.026)	(0.027)	(0.028)	(0.028)	(0.031)
Preceding targets	-	0.0009	-0.0006	-0.0103	-0.0037
		(0.0033)	(0.0062)	(0.0086)	(0.0142)
-2 Log L	71295.1	71304.6	71303.4	71301.4	71301.7

Notes: We provide the coefficient estimates with the standard errors in parentheses. We examine trials 2 through 192. We do not provide the estimates of the intercepts, the treatment dummies, or the covariance parameters. All regressions have 7831 observations. † indicates significance at $p < .1$ and *** indicates significance at $p < .001$. -2 Log L refers to negative two times the log-likelihood.

In every specification, running mean is significant and preceding targets is not significant. In the Supplemental Online Appendix, we include an analysis similar to Table 6, performed on the response35 variable, which has a standard deviation of 35 not 25. The qualitative results hold.[26]

Why are there such stark differences between the results from the simulated and non-simulated data? The mean of a set of recent lines is a noisy version of the running mean. But

[26] See Table A4 in the Supplemental Online Appendix.

unless there is actually a bias toward recent lines, recent lines will be worse predictors than the running mean. Our simulated data does not have a bias toward recent lines. Accordingly, our analysis does not detect such a bias. On the other hand, in our non-simulated data there is a bias toward the running mean and our analysis identifies this to be the case. In summary, we completely reject the claim that our techniques are unable to detect a bias toward the running mean, should such a bias exist.

Responses with zero mass across trials

Although our direct tests of CAM fail to find evidence in support of the model, we now look for evidence of learning across trials. One such test of CAM is that, as participants observe the distribution across trials, Bayesian participants will improve their understanding of the distribution across trials. In fact, under mild assumptions, two Bayesian observers with different initial priors will both have posteriors that converge to the true distribution (Savage, 1954; Blackwell & Dubins, 1962).

Therefore, if the participants make judgments consistent with CAM then they should learn the lower bound of the distribution and the upper bound of the distribution. In other words, the participants should learn where the distribution has zero mass. If the participant is Bayesian then they should have diminishing priors on line lengths that are longer than the maximum in their distribution or shorter than the minimum in their distribution. Accordingly, the participant should offer such a response with a diminishing frequency across trials.

We define the *zero mass dummy* to be 1 if the response is greater than the maximum[27] in the distribution or less than the minimum[28] of the distribution, and a 0 otherwise. In Figure 3 we plot the average of this variable across trials.

[27] For the uniform, normal, and long treatments the maximum is 416. For the short treatment the maximum is 224.

[28] For the uniform, normal, and short treatments the minimum is 48. For the long treatment the minimum is 240.

<<Figure 3 about here>>

Figure 3 suggests that the zero mass dummy is not decreasing across trials. To test this, we perform the following analysis. We offer different measures of the rate of the learning. In one specification, the independent variable is simply the trial number. But perhaps the learning is not linear and follows the square root. Therefore, we offer a second specification where the independent variable is the square root of the trial number, which we refer to as *Sqrt. Trial*. In the remaining four specifications, we use a categorical variable indicating whether the trial is among the first 5, among the first 10, among the first 20, or among the first half of trials.

We conduct the analysis similar to those above but with some differences. First, due to the discrete nature of the zero mass dummy, we conduct a logistic regression. Second, we account for the repeated measures by a fixed-effects regression. In other words, we estimate a dummy variable for every participant. Third, the zero mass dummy might depend on the target size and the treatment, so we control for this possibility by estimating a dummy variable for each target in each distribution treatment. Table 7 summarizes this fixed-effects analysis. We note that CAM predicts negative estimates for Trial and Sqrt. Trial, but positive estimates for the others. There are 455 responses with a zero mass and 7296 without.

Table 7: Fixed-effects logistic regressions of the zero mass dummy variable

	Trial	Sqrt. Trial	First 5	First 10	First 20	First half
Trial	-0.0015	-0.026	0.062	0.199	0.283†	0.251*
	(0.001)	(0.017)	(0.355)	(0.250)	(0.171)	(0.111)
-2 Log L	2247.2	2247.0	2249.3	2248.7	2246.7	2244.2

Notes: We provide the coefficient estimates with the standard errors in parentheses. We examine trials 1 through 192. We do not provide the estimates of the intercepts, the participant dummy variables, or the treatment-target dummy variables. All regressions have 7751 observations. † indicates significance at $p < .1$ and * indicates significance at $p < .05$. -2 Log L refers to negative two times the log-likelihood.

We find evidence of learning but only in the First half specification. The reader might be concerned that participants exhibit exhaustion over the entire 192 trials. Accordingly we analyze only the first half of the trials.[29] There we do not find evidence of learning as measured by the zero mass dummy variable. Tables 7 and A5 produce a total of 11 specifications and we find a significant relationship in only one specification. We also note that in the 11 specifications, three do not even have the correct sign as predicted by CAM.

Bias toward the running mean across trials

We find only mixed and weak evidence that participants learn the distribution: we do not find evidence that zero mass responses become less likely across trials, except in the First half specification. However, this is not the unique indirect test of CAM. Another indirect test relates to the bias toward the running mean across trials.

CAM purports that participants combine their noisy perception and memory of the target with their priors of the distribution of the stimuli. HHV offers (p. 239) the following formalism that response is a weighted average of the mean of the noisy, inexact memory of the target (M) and "the central value of the category" (ρ):

$$\text{Response} = \lambda M + (1\text{-}\lambda)\rho.$$

The inexactness of the memory of the target has a standard deviation of σ_M and the "standard deviation of the prior distribution" is σ_P. The weight between M and ρ is a decreasing function g(.) of the ratio of these two standard deviations:

$$\lambda = g\left(\frac{\sigma_M}{\sigma_P}\right).$$

This implies that the smaller the standard deviation of the prior distribution, the greater the bias toward the mean of the distribution. We note that this decrease in standard deviation is precisely what happens over the course of an experiment. Before the participant has been exposed to any lines, the distribution is unknown and the participant relies on presumably diffuse

[29] See Table A5 in the Supplemental Online Appendix.

priors. However, as the participant repeatedly views target lines of various lengths, the standard deviation of the posteriors decreases. The line lengths that have been observed will have increased posteriors and the line lengths that have not been seen have reduced posteriors. This produces a decreasing standard deviation of the prior distribution across trials. Based on this, CAM predicts that the bias toward the mean will increase over the course of the experiment.[30]

We construct a variable that is designed to capture the extent to which the response is closer to the mean than it is to the target. We define *running mean bias* to be the distance between the target and the running mean minus the distance between the response and the running mean:

Running mean bias = | Target – Running mean | – | Response – Running mean |.

We perform a random-effects repeated measures analysis, similar to that summarized in Tables 3-6. However, we employ the independent variables in the analyses summarized in Table 7. Table 8 summarizes this random-effects analysis. CAM would predict positive estimates for Trial and Sqrt. Trial and negative estimates for the others.

Table 8: Random-effects regressions of the running mean bias variable

	Trial	Sqrt. Trial	First 5	First 10	First 20	First half
Trial	0.0251***	0.486***	-7.176**	-5.002***	-5.004***	-2.277***
	(0.00556)	(0.0960)	(2.178)	(1.463)	(1.026)	(0.613)
-2 Log L	72401.9	72145.3	72399.4	72399.4	72388.1	72399.0

Notes: We provide the coefficient estimates with the standard errors in parentheses. We examine trials 2 through 192. We do not provide the estimates of the intercepts, the covariance parameters, or the treatment-target dummy variables. All regressions have 7713 observations. ** indicates significance at $p < .01$, and *** indicates significance at $p < .001$. -2 Log L refers to negative two times the log-likelihood.

Here we find strong evidence of an increase in the bias toward the running mean across trials. We also note that this result is robust to restricting attention to the first half of trials.[31] It is

[30] Below we will say more about the mathematical content of HHV.

[31] See Table A6 in the Supplemental Online Appendix.

further robust to expressing the bias toward the running mean as a ratio, as this measure would be more similar to the weight (λ) between the memory and the distribution in CAM. With running mean bias ratio, we also find strong evidence of an increasing bias toward the running mean across trials.[32,33]

These results seem to be consistent with CAM but a deeper look into these results suggests otherwise. We perform an analysis to learn whether there is an increasing bias toward the previous line across trials. We therefore construct the variable *previous bias*, which is the analogous to running mean bias. We conduct the analysis identical to that in Table 8 but with this new dependent variable. This analysis is summarized in Table 9.

Table 9: Random-effects regressions of the previous bias variable

	Trial	Sqrt. Trial	First 5	First 10	First 20	First half
Trial	0.0220**	0.447***	-9.920***	-7.090***	-5.000***	-1.977**
	(0.0069)	(0.118)	(2.679)	(1.800)	(1.264)	(0.754)
-2 Log L	75551.2	75541.6	75535.9	75534.9	75535.4	75545.2

Notes: We provide the coefficient estimates with the standard errors in parentheses. We examine trials 2 through 192. We do not provide the estimates of the intercepts, the covariance parameters, or the treatment-target dummy variables. All regressions have 7713 observations. ** indicates significance at $p < .01$, and *** indicates significance at $p < .001$. -2 Log L refers to negative two times the log-likelihood.

We see that in every specification, there is a greater bias toward the previous line across trials. Whereas the results of Table 8 are consistent with both learning the distribution and employing this information in judgments, the results in Table 9 are not consistent with this explanation. When we restrict attention to the first half of trials we also see strong evidence of an increase in the bias toward the previous lines across trials.[34,35]

[32] See Table A7 in the Supplemental Online Appendix.

[33] We note that most of the results that we report in this paper are similar to those reported in Duffy and Smith (2017). The exception is the relationship between running mean bias and trials. Duffy and Smith do not find a relationship in the Duffy et al. (2010) data, but here we find a strong relationship.

[34] See Table A8 in the Supplemental Online Appendix.

[35] Interestingly, we note a positive correlation between the response time and previous bias ($r(7711) =.038$, $p = .002$) but no such relationship between response time and running mean bias ($r(7711) = -.015$, $p = .19$).

Error across trials

Another implication of CAM relates to the errors across trials. Figure 3 in HHV reports a monotonic relationship between the variance of the responses and the standard deviation of the prior distribution (σ_P) decreases across trials.[36] CAM therefore predicts that, as the participants learn the distribution, the errors will diminish across trials.

We define the *absolute response bias* variable to be the absolute value of the response bias. Absolute response bias is a measure of the error of the judgment. We perform an analysis similar to Table 9, but with absolute response bias as the dependent variable. Table 10 summarizes this random-effects analysis. CAM would predict negative estimates for Trial and Sqrt. Trial, and positive estimates for the other specifications.

Table 10: Random-effects regressions of the absolute response bias variable

	Trial	Sqrt. Trial	First 5	First 10	First 20	First half
Trial	0.0325***	0.568***	-2.016	-3.777**	-4.542***	-2.328***
	(0.0047)	(0.081)	(1.679)	(1.195)	(0.860)	(0.524)
-2 Log L	70390.9	70383.2	70424.9	70417.0	70399.8	70408.9

Notes: We provide the coefficient estimates with the standard errors in parentheses. We examine trials 1 through 192. We do not provide the estimates of the intercepts, the covariance parameters, or the treatment-target dummy variables. All regressions have 7751 observations. † indicates significance at $p < .1$, ** indicates significance at $p < .01$, and *** indicates significance at $p < .001$. -2 Log L refers to negative two times the log-likelihood.

Rather than see errors diminish across trials, we actually see increasing errors across trials in the Trial, Sqrt. Trial, First 10, First 20, and First half specifications. These results are not consistent with the participants learning the distribution and using this knowledge to improve their judgments. Perhaps exhaustion is driving these results. Therefore, we analyze only the first half of trials are our results are similar to that from Table 10.[37] In conclusion, not only do we not find evidence that judgments improved across the trials, we observe that judgments actually

[36] Below we will say more about the mathematical content of HHV.

[37] See Table A9 in the Supplemental Online Appendix.

become worse across trials. In other words, even though running mean bias increases across trials, this does not appear to be consistent with learning the distribution, which is a central component to CAM.

Difference between the long and short treatments across trials

Earlier we found that there were differences in the response bias between the long and short treatments, and that this is not consistent with CAM. On the other hand, it might not be reasonable to expect that these differences diminish before the participants learned the distribution. According to this view, we should see the response bias in these treatments converge to zero, as the participants learn the distribution. In Figure 4, we plot the average response bias across trials, for the long and short treatments.

<<Figure 4 about here>>

Figure 4 suggests that response bias in neither the long treatment nor short treatment converges to zero. To test this, we take the mean response bias in the short treatment and subtract the mean response bias in the long treatment for every period. We note that this difference (*M* = 25.16; *SD* = 16.52) does not appear to be converging to zero.

We also acknowledge that Figure 3 is rather noisy. Further, from Tables 3-5 we know that response bias varies by the target. Therefore, in the analysis below, we control for the target. We define the *normalized target* to be the target minus the mean of the distribution. In the short treatment, the normalized target is the target minus 136, and in the long treatment, the normalized target is the target minus 328. This variable allows us to compare the shortest target in the short treatment with the shortest target in the long treatment, the target longer than the shortest, and so on. We also employ the variety of independent variables for the trials, as was used in the analyses summarized in Tables 7-10. We define *short* to be a dummy variable that indicates whether the trial is the short treatment. We also include the interactions with the relevant measures of trials. If participants are learning the distribution and employing this information then we would see these differences declining across trials. This analysis is summarized in Table 11.

Table 11: Random-effects regressions of the response bias variable

	Trial	Sqrt. Trial	First 5	First 10	First 20	First half
Intercept	-22.28***	-22.85***	-21.80***	-21.80***	-21.78***	-22.60***
	(4.79)	(5.00)	(4.66)	(4.66)	(4.67)	(4.71)
Norm. target	-0.220***	-0.220***	-0.219***	-0.220***	-0.220***	-0.220***
	(0.008)	(0.008)	(0.008)	(0.008)	(0.008)	(0.008)
Short	23.05**	23.67**	21.18**	21.35**	21.24**	21.32**
	(6.77)	(7.07)	(6.59)	(6.59)	(6.60)	(6.65)
Trial	0.0033	0.096	-6.602	-3.289	-1.755	1.277
	(0.012)	(0.196)	(4.111)	(2.930)	(2.085)	(1.262)
Trial*Short	-0.018	-0.250	7.187	0.145	1.067	0.072
	(0.016)	(0.276)	(5.728)	(4.085)	(2.928)	(1.776)
-2 Log L	39928.6	39918.0	39904.2	39905.7	39908.6	39909.3

Notes: We provide the coefficient estimates with the standard errors in parentheses. We examine trials 1 through 192. We do not provide the estimates of the covariance parameters. All regressions are restricted to the short or the long treatments and have 4171 observations. † indicates significance at $p < .1$, ** indicates significance at $p < .01$, and *** indicates significance at $p < .001$. -2 Log L refers to negative two times the log-likelihood.

We interpret the intercept as the average response bias for the mean target in the long treatment when the independent variables are zero, and trial as describing the trajectory of this value across trials. In no specification do we see that the average response bias in the long treatment increases toward zero. We interpret the short variable as an estimate of the amount that judgments in the short treatment is larger than those in the long treatment at the mean of the targets and when the other independent variables are zero. We also interpret the interaction of short and trial as difference in the trajectory of the short and long treatments. We also do not find evidence of learning here.

To confirm what we suspected from Figure 4, we do not find evidence that the difference in the average response bias is diminishing across trials. There appears to be persistent differences between the treatments: lines in the long treatment are underestimated and lines in the short treatment are overestimated. Again, this is not consistent with CAM.

Since the length of the initial adjustable line does not vary across trials, we cannot distinguish between the hypothesis that it is caused by the very short initial adjustable line or the hypothesis that that there is a bias toward the center of the screen. But regardless of the cause, these results are not consistent with CAM.

A few comments on the mathematical content of HHV

Figure 3 in HHV is justified by the mathematical content on page 241. We turn our attention to the relevant text: Section Vll, Subsection A. There HHV considers the relationship between the variance of the responses to the standard deviation of the distribution. The authors represent the variance as:

$$S(R) = \ g\left(\frac{\sigma_M}{\sigma_P}\right)\sigma_M,$$

and the partial derivative with respect to σ_P as:[38]

$$S'(R) = -\left(\frac{\sigma_M}{{\sigma_P}^2}\right)\ g\left(\frac{\sigma_M}{\sigma_P}\right).$$

HHV define g(0)=1 and g(c)=0 for some “large” c. Apparently g(x) is not defined for x > c. The authors investigate the implications of σ_P=0. But this has the undesirable feature of dividing by zero, once in S(R) and twice in S’(R). Further, the authors cannot be making an argument about the limits as σ_P converges to zero because, for any c there exists a σ_P>0 small enough so that g(.) is not defined.

[38] We use the notation of HHV. However, it is not clear to us why the authors chose to employ such non-standard notation for the partial derivatives. Standard notation would be $\frac{\partial S(R)}{\partial \sigma_P}$.

Despite these serious and fundamental errors, it would not seem to render the relationship between the variance of the responses and standard deviation of the prior distribution (σ_P) to be non-monotonic. However, it is troubling to see an expression that divides by zero and considers values that are not defined. Further, considering that the experiment only considers settings where $\sigma_P > \sigma_M$ and that the text does not provide an indication on how to generate $\sigma_P = 0$ in the laboratory, it is our view that restricting attention to $\sigma_P > \sigma_M$ is reasonable.

Next we turn our attention to Subsection B. There HHV considers the relationship between the response bias and the standard deviation of the prior distribution. The authors represent the response bias as:

$$B(R) = \left(g\left(\frac{\sigma_M}{\sigma_P}\right) - 1\right)(\mu - \rho),$$

where μ is the mean of the noisy memory of the line length and ρ is the "central value of the distribution." The authors report that partial derivative with respect to σ_P is "difficult to compute," because "σ_P depends on μ." First, it is not at all clear to us why the standard deviation of the prior distribution should depend on the mean of the noisy memory of a particular line length under consideration. Accordingly, we calculate the partial derivative of the response bias with respect to σ_P as:[39]

$$B'(R) = -\left(\frac{\sigma_M}{{\sigma_P}^2}\right) g'\left(\frac{\sigma_M}{\sigma_P}\right)(\mu - \rho).$$

Again the authors investigate the response bias at σ_P=0. Again, this constitutes dividing by zero. And further, arguments on the limit as σ_P converges to zero do not make sense because there are values of $\sigma_P > 0$ small enough so that g(.) is not defined. However, in contrast to the discussion of the variance, where the results would seem to be unchanged, these non-monotonicity

[39] Again, we use the notation of HHV. Standard notation would be $\frac{\partial B(R)}{\partial \sigma_P}$.

arguments seem to crucially depend on dividing by zero. HHV write “B(R) is never monotonic as σ_P is varied because B(R) always has a maximum.” We do not understand why the authors assert this as true and it seems to rely on arguments where g(.) is not defined. If we restrict attention to the setting of the experiment ($\sigma_P > \sigma_M$) then it would seem that we are left with a monotonic relationship between response bias and $\sigma_{P.}$

Conclusions

Since the authors of HHV were not able to provide their data to us, we replicated their Experiment 3. Our data and their data are similar in some respects. Both HHV and our results indicate that 2 out of 3 comparisons reported by HHV have significantly different standard deviations. We also note that our data does not have more noise than the HHV data. We further note that our data shares some qualitative features with HHV, for instance in each of the four treatments, there is a negative relationship between the response bias and the target length. However, in our data, we find that judgments in the normal, uniform, and long treatments have a mean response bias that is negative. This persists when we restrict the analysis to the targets adjacent to the means in the distributions. We also find that the 12 targets lengths in the short treatment are overestimated relative to the 12 targets in the long treatment. This is not consistent with CAM. We do not know if these results exist in the HHV data because they do not report a test of these features.

Further, HHV analyzed data averaged across previous stimuli. This renders the hypothesis that there is a bias toward the running mean and the hypothesis that there is a bias toward recent targets to be indistinguishable. By contrast, we conduct an analysis of the judgment-level data in order to determine if there is a bias toward the running mean or a bias toward recent targets. Our analysis shows that there is not a bias toward the running mean but rather a bias toward recent targets. Because some might be worried that our techniques would not be able to detect a bias toward the running mean, should such a bias exist, we simulate data that has a bias toward the running mean but not toward recent lines. Our techniques correctly identify this relationship. We therefore reject the criticism that our techniques would not identify a bias toward the running mean, should such a bias exist in the data.

HHV analyzed data averaged across trials, and therefore learning properties are not able to be examined. We test some implications of CAM related to the participants learning the distribution of targets and employing this information in their judgments. We do not find evidence that responses that have a zero mass are declining across trials. While we find evidence of an increase of the running mean bias across trials we also find evidence of an increase in the previous bias across trials. Additionally, we find that the errors in the judgments increase, rather than decrease, across trials. Finally, we find that the difference in the response bias between the long and short treatments does not diminish across trials. In summary we do not find evidence that the participants are learning the distribution and employing this information to improve their judgments. These results are not consistent with CAM.

Taken together we simply do not find evidence that the judgements in our data are consistent with CAM. In addition to Duffy and Smith (2017), this is now the second paper that performs a careful judgment-level analysis of the data and does not find support for CAM.

More generally, CAM is a *Bayesian model of judgment*. Specifically, Bayesian models of judgment make the joint hypothesis that participants learn the distribution of stimuli and they use this information in their judgments in accordance with Bayes' rule. There is a spirited discussion of the merits of these Bayesian models.[40] We contribute to this literature by demonstrating that a

[40] See Barth, Lesser, Taggart, and Slusser (2015), Bowers and Davis (2012a, 2012b), Cassey et al. (2016), Chater, Tenenbaum, and Yuille (2006), Chater et al. (2011), Duffy and Smith (2017), Elqayam and Evans (2011), Goodman et al. (2015), Griffiths, Chater, Norris, and Pouget (2012), Griffiths and Tenenbaum (2006), Hahn (2014), Jones and Love (2011a, 2011b), Hemmer and Steyvers (2009a, 2009b), Lewandowsky, Griffiths, and Kalish (2009), Marcus

judgment-level analysis shows that our data are inconsistent with CAM. We encourage researchers to employ our techniques in different settings in order to learn the extent to which the predictions of CAM, or any Bayesian model of judgment, are accurate.

Further, aside from Duffy and Smith (2017), we are the first to apply to Bayesian models of judgment the well-known results that Bayesians with very different initial priors will have posteriors that converge to the true distribution (Savage, 1954; Blackwell & Dubins, 1962). In our analysis we do not see evidence of learning, either because there was no learning or because the learning did not manifest itself in the judgments. Regardless, it does not seem that our results could be consistent with any Bayesian model of judgment.

One question is, "How could the shortcomings of HHV go unnoticed?" For instance, it is not clear to us how the analysis of HHV could *verify* that the bias in judgments does not stem from recent stimuli and that the judgments are Bayesian. It is our view that the mathematical content of HHV contributed to its lack of scrutiny. The inclusion of mathematical formalism, even if it is unrelated to the topic, enhances the perception of the quality of the research (Eriksson, 2012). It is possible that the mathematical content of HHV dissuaded readers and reviewers from carefully judging the paper. Paradoxically, this includes noticing the errors in the mathematics itself.[41] Everybody knows that one should not divide by zero. Yet, there on page 241 we find multiple instances of dividing by zero. It is rather surprising that we are possibly the first researchers to notice this since HHV was first submitted in 1998.

We also add that the formalism was not used to derive non-obvious testable predictions. Indeed, the implication that the absolute response bias is not monotonic in σ_P is not considered in

and Davis (2013, 2015), Mozer, Pashler, and Homaei (2008), Perfors, Tenenbaum, Griffiths, and Xu (2011), Petzschner, Glasauer, and Stephan (2015), Sailor and Antoine (2005), Tauber et al. (2017), and Tenenbaum, Griffiths, and Kemp (2006).

[41] For instance, we note two violations of the chain rule on page 239.

the comparisons with σ_P and the variance in the responses. We hope that our work contributes to the introspection required to improve the use of mathematical models in psychology.

Further, consider the term “fine-grained memory” that is used to refer to the memory of the length of the particular stimulus. This term appears throughout HHV. It is not clear to us how this is an improvement over “stimulus memory” or simply “memory.” This is particularly true since there are not comparisons of memories with more or less granularity. The use of this term is an example of, what in our view, is the needlessly opaque language employed by HHV. A consequence of this opaque language is that the reader can suffer from the “Guru effect” (Sperber, 2010) whereby the reader confers more authority and plausibility to a paper when it contains opaque language. It is our view that the opaque writing of HHV also contributed to its lack of scrutiny.

Additionally, HHV always offered analyses with a single specification, which we admit is standard in the psychology literature. In other words, HHV uses only a single type of test, a single set of explanatory variables, a single functional form, a single set of assumptions for the error term, and a single set of data under consideration. This becomes all the more serious given the curious choice of examining only the central 10 values for the standard deviations of the normal and uniform treatments. Reporting more than one specification, as we make a point of doing, can help diminish the chances of arriving at incorrect conclusions and give the reader a greater confidence in the results (Simmons, Nelson, & Simonsohn, 2011; Steegen, Tuerlinckx, Gelman, & Vanpaemel, 2016). We hope that our efforts here contribute to the ongoing discussion of improving the methods and conventions of psychological science (Wagenmakers, Wetzels, Borsboom, & Maas, 2011; Wicherts et al., 2016).

Finally, our efforts highlight the importance of maintaining and sharing datasets so that researchers can scrutinize results, as this becomes more problematic in the more distant past (Vines et al., 2014).

References

Allred, S., Crawford, L.E., Duffy, S., & Smith, J. (2016). Working memory and spatial judgments: Cognitive load increases the central tendency bias. *Psychonomic Bulletin & Review, 23*(6), 1825-1831.

Ashourian, P., & Loewenstein, Y. (2011). Bayesian inference underlies the contraction bias in delayed comparison tasks. *PloS ONE, 6*(5), e19551.

Bae, G. Y., Olkkonen, M., Allred, S. R., & Flombaum, J. I. (2015). Why some colors appear more memorable than others: A model combining categories and particulars in color working memory. *Journal of Experimental Psychology: General, 144*(4), 744-763.

Barth, H., Lesser, E., Taggart, J., & Slusser, E. (2015). Spatial estimation: A non-Bayesian alternative. *Developmental Science, 18*(5), 853-862.

Blackwell, D., & Dubins, L. (1962). Merging of opinions with increasing information. *Annals of Mathematical Statistics, 33*, 882-886.

Bowers, J. S., & Davis, C. J. (2012a). Bayesian just-so stories in psychology and neuroscience. *Psychological Bulletin, 138*(3), 389-414.

Bowers, J. S., & Davis, C. J. (2012b). Is that what Bayesians believe? Reply to Griffiths, Chater, Norris, and Pouget (2012). *Psychological Bulletin, 138*(3), 423-426.

Cassey, P., Hawkins, G. E., Donkin, C., & Brown, S. D. (2016). Using alien coins to test whether simple inference is Bayesian. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 42*(3), 497-503.

Chater, N., Goodman, N., Griffiths, T. L., Kemp, C., Oaksford, M., & Tenenbaum, J. B. (2011). The imaginary fundamentalists: The unshocking truth about Bayesian cognitive science. *Behavioral and Brain Sciences, 34*(4), 194-196.

Chater, N., Tenenbaum, J. B., & Yuille, A. (2006). Probabilistic models of cognition: Conceptual foundations. *Trends in Cognitive Sciences, 10*(7), 287-291.

Choplin, J. M., & Hummel, J. E. (2002). Magnitude comparisons distort mental representations of magnitude. *Journal of Experimental Psychology: General, 131*(2), 270-286.

Corbin, J. C., Crawford, L. E., & Vavra, D. T. (2017). Misremembering emotion: Inductive category effects for complex emotional stimuli. *Memory & Cognition, 45*(5), 691-698.

Corneille, O., Huart, J., Becquart, E., & Brédart, S. (2004). When memory shifts toward more typical category exemplars: Accentuation effects in the recollection of ethnically ambiguous faces. *Journal of Personality and Social Psychology, 86*(2), 236-250.

Crawford, L. E., & Duffy, S. (2010). Sequence effects in estimating spatial location. *Psychonomic Bulletin & Review, 17*(5), 725-730.

Crawford, L. E., Huttenlocher, J., & Engebretson, P. H. (2000). Category effects on estimates of stimuli: Perception or reconstruction? *Psychological Science, 11*(4), 280-284.

DeCarlo, L. T., & Cross, D. V. (1990). Sequential effects in magnitude scaling: Models and theory. *Journal of Experimental Psychology: General, 119*(4), 375-396.

Duffy, S., Huttenlocher, J., Hedges, L. V., & Crawford, L. E. (2010). Category effects on stimulus estimation: Shifting and skewed frequency distributions. *Psychonomic Bulletin & Review, 17*, 224-230.

Duffy, S., & Smith, J. (2017). Category effects on stimulus estimation: Shifting and skewed frequency distributions-A reexamination. *Psychonomic Bulletin & Review*, forthcoming

Edwards, W., Lindman, H., & Savage, L. J. (1963). Bayesian statistical inference for psychological research. *Psychological Review, 70*(3), 193-242.

Elqayam, S., & Evans, J. S. B. (2011). Subtracting "ought" from "is": Descriptivism versus normativism in the study of human thinking. *Behavioral and Brain Sciences, 34*(5), 233-248.

Eriksson, K. (2012). The nonsense math effect. *Judgment and Decision Making, 7*(6), 746-749.

Estes, W. K. (1956). The problem of inference from curves based on group data. *Psychological Bulletin, 53*(2), 134-140.

Feldman, N. H., Griffiths, T. L., & Morgan, J. L. (2009). The influence of categories on perception: Explaining the perceptual magnet effect as optimal statistical inference. *Psychological Review, 116*(4), 752-782.

Fugate, J. M. (2013). Categorical perception for emotional faces. *Emotion Review,* 5(1), 84-89.

Goodman, N. D., Frank, M. C., Griffiths, T. L., Tenenbaum, J. B., Battaglia, P. W., & Hamrick, J. B. (2015). Relevant and robust: A response to Marcus and Davis (2013). *Psychological Science, 26*(4), 539-541.

Griffiths, T. L., Chater, N., Norris, D., & Pouget, A. (2012). How the Bayesians got their beliefs (and what those beliefs actually are): Comment on Bowers and Davis (2012). *Psychological Bulletin, 138*(3), 415-422.

Griffiths, T. L., & Tenenbaum, J. B. (2006). Optimal predictions in everyday cognition. *Psychological Science, 17*(9), 767-773.

Hahn, U. (2014). The Bayesian boom: Good thing or bad? *Frontiers in Psychology, 5*, 765.

Hayes, K. J. (1953). The backward curve: A method for the study of learning. *Psychological Review, 60*(4), 269-275.

Hemmer, P., & Steyvers, M. (2009a). Integrating episodic memories and prior knowledge at multiple levels of abstraction. *Psychonomic Bulletin & Review, 16*(1), 80-87.

Hemmer, P., & Steyvers, M. (2009b). A Bayesian account of reconstructive memory. *Topics in Cognitive Science, 1*, 189-202.

Hemmer, P., Tauber, S., & Steyvers, M. (2015). Moving beyond qualitative evaluations of Bayesian models of cognition. *Psychonomic Bulletin & Review, 22*(3), 614-628.

Hertwig, R., Pachur, T., & Kurzenhäuser, S. (2005). Judgments of risk frequencies: Tests of possible cognitive mechanisms. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 31*(4), 621-642.

Holden, M. P., Curby, K. M., Newcombe, N. S., & Shipley, T. F. (2010). A category adjustment approach to memory for spatial location in natural scenes. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 36*(3), 590-604.

Hollingworth, H. L. (1910). The central tendency of judgment. *The Journal of Philosophy, Psychology and Scientific Methods, 7*(17), 461-469.

Hund, A. M., & Spencer, J. P. (2003). Developmental changes in the relative weighting of geometric and experience-dependent location cues. *Journal of Cognition and Development, 4*(1), 3-38.

Huttenlocher, J., Hedges, L. V., & Vevea, J. L. (2000). Why do categories affect stimulus judgment? *Journal of Experimental Psychology: General, 129*, 220-241.

Jesteadt, W., Luce, R. D., & Green, D. M. (1977). Sequential effects in judgments of loudness. *Journal of Experimental Psychology: Human Perception and Performance, 3*(1), 92-104.

Jones, M., Curran, T., Mozer, M. C., & Wilder, M. H. (2013). Sequential effects in response time reveal learning mechanisms and event representations. *Psychological Review, 120*(3), 628-666.

Jones, M., & Love, B. C. (2011a). Bayesian fundamentalism or enlightenment? On the explanatory status and theoretical contributions of Bayesian models of cognition. *Behavioral and Brain Sciences, 34*(4), 169-188.

Jones, M., & Love, B. C. (2011b). Pinning down the theoretical commitments of Bayesian cognitive models. *Behavioral and Brain Sciences, 34*(4), 215-231.

Kahneman, D., & Frederick, S. (2002). Representativeness revisited: Attribute substitution in intuitive judgment. In Gilovich, T., Griffin, D., & Kahneman, D. (Eds.), *Heuristics and biases: The psychology of intuitive judgment*, Cambridge University Press, 49-81.

Kahneman, D., & Tversky, A., (1973). On the psychology of prediction. *Psychological Review, 80*(4), 237-251.

Laming, D. (1984). The relativity of 'absolute'judgements. *British Journal of Mathematical and Statistical Psychology, 37*(2), 152-183.

Lewandowsky, S., Griffiths, T. L., & Kalish, M. L. (2009). The wisdom of individuals: Exploring people's knowledge about everyday events using iterated learning. *Cognitive Science, 33*(6), 969-998.

Marcus, G. F., & Davis, E. (2013). How robust are probabilistic models of higher-level cognition? *Psychological Science, 24*(12), 2351-2360.

Marcus, G. F., & Davis, E. (2015). Still searching for principles: A response to Goodman et al. (2015). *Psychological Science, 26*(4), 542-544.

McCullough, S., & Emmorey, K. (2009). Categorical perception of affective and linguistic facial expressions. *Cognition, 110*(2), 208-221.

Moore, D. A., & Healy, P. J. (2008). The trouble with overconfidence. *Psychological Review, 115*(2), 502–517.

Mozer, M. C., Pashler, H., & Homaei, H. (2008). Optimal predictions in everyday cognition: The wisdom of individuals or crowds? *Cognitive Science, 32*(7), 1133-1147.

Norris, D., & McQueen, J. M. (2008). Shortlist B: A Bayesian model of continuous speech recognition. *Psychological Review, 115*(2), 357-395.

Olkkonen, M., & Allred, S. R. (2014). Short-term memory affects color perception in context. *PloS ONE, 9*(1), e86488.

Olkkonen, M., McCarthy, P. F., & Allred, S. R. (2014). The central tendency bias in color perception: Effects of internal and external noise. *Journal of Vision, 14*(11), 1-15.

Perfors, A., Tenenbaum, J. B., Griffiths, T. L., & Xu, F. (2011). A tutorial introduction to Bayesian models of cognitive development. *Cognition, 120*(3), 302-321.

Persaud, K., & Hemmer, P. (2014). The influence of knowledge and expectations for color on episodic memory. *Proceedings of the Cognitive Science Society, 36*, 1162-1167.

Petzold, P. (1981). Distance effects on sequential dependencies in categorical judgments. *Journal of Experimental Psychology: Human Perception and Performance, 7*(6), 1371-1385.

Petzold, P., & Haubensak, G. (2004). The influence of category membership of stimuli on sequential effects in magnitude judgment. *Perception & Psychophysics, 66*(4), 665-678.

Petzschner, F. H., Glasauer, S., & Stephan, K. E. (2015). A Bayesian perspective on magnitude estimation. *Trends in Cognitive Sciences, 19*(5), 285-293.

Poulton, E. C. (1979). Models for biases in judging sensory magnitude. *Psychological Bulletin, 86*(4), 777-803.

Psychology Software Tools, Inc. [E-Prime 2.0]. (2012). Retrieved from http://www.pstnet.com.

Roberson, D., Damjanovic, L., & Pilling, M. (2007). Categorical perception of facial expressions: Evidence for a “category adjustment” model. *Memory & Cognition, 35*(7), 1814-1829.

Sailor, K. M., & Antoine, M. (2005). Is memory for stimulus magnitude Bayesian? *Memory & Cognition, 33*, 840-851.

Sampson, R. J., & Raudenbush, S. W. (2004). Seeing disorder: Neighborhood stigma and the social construction of “broken windows.” *Social Psychology Quarterly, 67*(4), 319-342.

Savage, L.J. (1954). *The Foundations of Statistics*. Wiley, New York. Reprinted in 1972 by Dover, New York.

Schutte, A. R., & Spencer, J. P. (2009). Tests of the dynamic field theory and the spatial precision hypothesis: Capturing a qualitative developmental transition in spatial working memory. *Journal of Experimental Psychology: Human Perception and Performance, 35*(6), 1698-1725.

Sidman, M. (1952). A note on functional relations obtained from group data. *Psychological Bulletin, 49*(3), 263-269.

Siegler, R. S. (1987). The perils of averaging data over strategies: An example from children's addition. *Journal of Experimental Psychology: General, 116*(3), 250-264.

Simmons, J. P., Nelson, L. D., & Simonsohn, U. (2011). False-positive psychology: Undisclosed flexibility in data collection and analysis allows presenting anything as significant. *Psychological Science, 22*(11), 1359-1366.

Spencer, J. P., & Hund, A. M. (2002). Prototypes and particulars: Geometric and experience-dependent spatial categories. *Journal of Experimental Psychology: General, 131*(1), 16-37.

Spencer, J. P., & Hund, A. M. (2003). Developmental continuity in the processes that underlie spatial recall. *Cognitive Psychology, 47*(4), 432-480.

Sperber, D. (2010). The guru effect. *Review of Philosophy and Psychology, 1*(4), 583-592.

Staddon, J. E., King, M., & Lockhead, G. R. (1980). On sequential effects in absolute judgment experiments. *Journal of Experimental Psychology: Human Perception and Performance, 6*(2), 290-301.

Steegen, S., Tuerlinckx, F., Gelman, A., & Vanpaemel, W. (2016). Increasing transparency through a multiverse analysis. *Perspectives on Psychological Science, 11*(5), 702-712.

Stewart, N., Brown, G. D., & Chater, N. (2002). Sequence effects in categorization of simple perceptual stimuli. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 28*(1), 3-11.

Tauber, S., Navarro, D. J., Perfors, A., & Steyvers, M. (2017). Bayesian models of cognition revisited: Setting optimality aside and letting data drive psychological theory. *Psychological Review, 124*(4), 410-441.

Tenenbaum, J. B., Griffiths, T. L., & Kemp, C. (2006). Theory-based Bayesian models of inductive learning and reasoning. *Trends in Cognitive Sciences, 10*(7), 309-318.

Twedt, E., Crawford, L. E., & Proffitt, D. R. (2015). Judgments of others' heights are biased toward the height of the perceiver. *Psychonomic Bulletin & Review, 22*(2), 566-571.

Vines, T. H., Albert, A. Y., Andrew, R. L., Débarre, F., Bock, D. G., Franklin, M. T., Gilbert, K. J., Moore, J. S., Renaut, S., & Rennison, D. J. (2014). The availability of research data declines rapidly with article age. *Current Biology, 24*(1), 94-97.

Wagenmakers, E. J., Wetzels, R., Borsboom, D., & Maas, H. L. J. (2011). Why psychologists must change the way they analyze their data: The case of psi. *Journal of Personality and Social Psychology, 100*(3), 426-432.

Wicherts, J. M., Veldkamp, C. L., Augusteijn, H. E., Bakker, M., van Aert, R. C., & Van Assen, M. A. (2016). Degrees of freedom in planning, running, analyzing, and reporting psychological studies: A checklist to avoid p-hacking. *Frontiers in Psychology, 7*, 1832.

Wilder, M., Jones, M., & Mozer, M. C. (2009). Sequential effects reflect parallel learning of multiple environmental regularities. *Advances in Neural Information Processing Systems, 22*, 2053-2061.

Woods, A. T., Poliakoff, E., Lloyd, D. M., Dijksterhuis, G. B., & Thomas, A. (2010). Flavor expectation: The effect of assuming homogeneity on drink perception. *Chemosensory Perception, 3*(3-4), 174-181.

Young, S. G., Hugenberg, K., Bernstein, M. J., & Sacco, D. F. (2009). Interracial contexts debilitate same-race face recognition. *Journal of Experimental Social Psychology, 45*(5), 1123-1126.

Yu, A. J., & Cohen, J. D. (2009). Sequential effects: Superstition or rational behavior? *Advances in Neural Information Processing Systems, 21*, 1873-1880.

Figure 1: Target distribution for the normal (A), uniform (B), short (C), and long (D) treatments

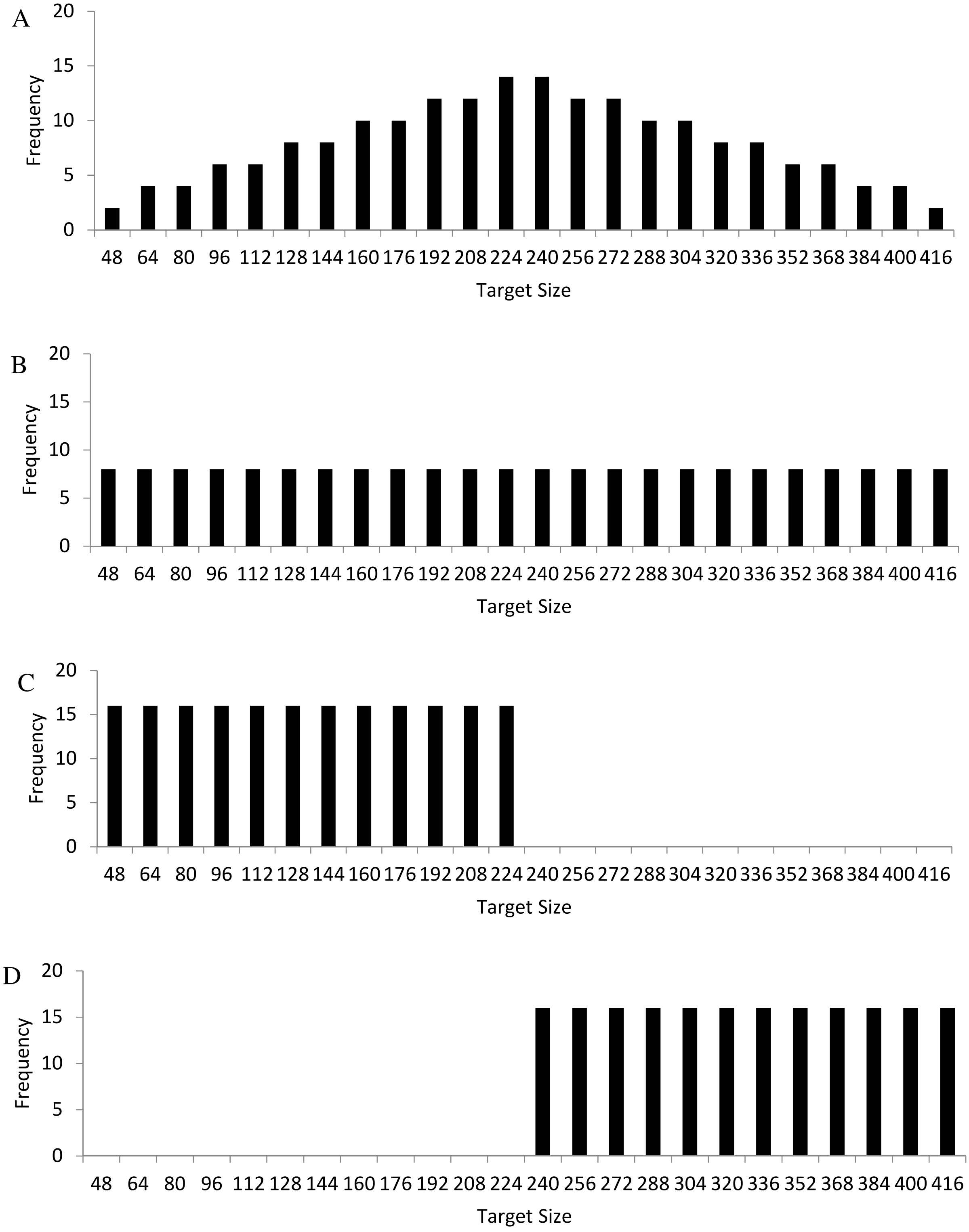


Figure 2: Response bias across targets for the normal (A), uniform (B), and short and long (C) treatments

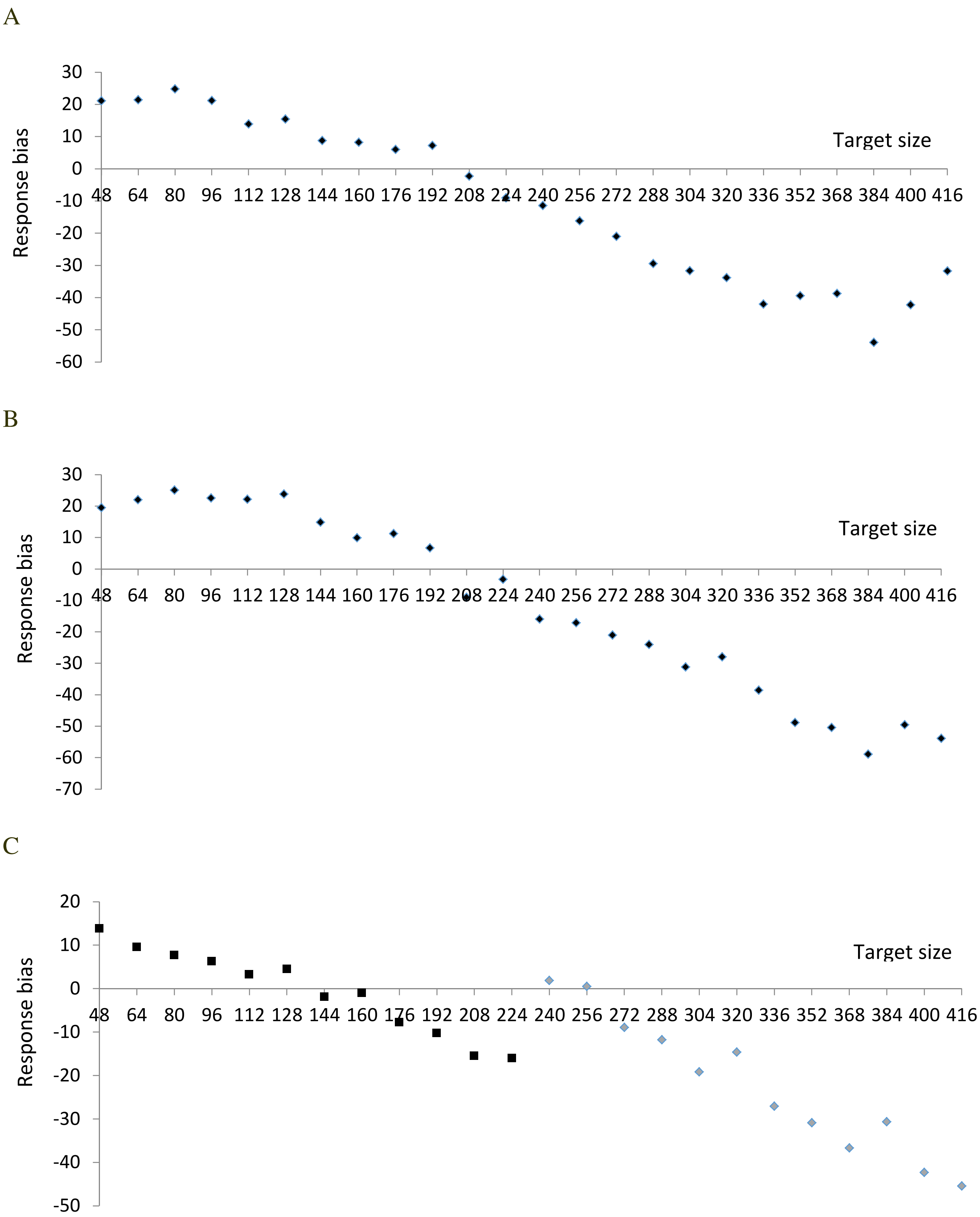


Figure 3: Mean zero mass dummy across trials

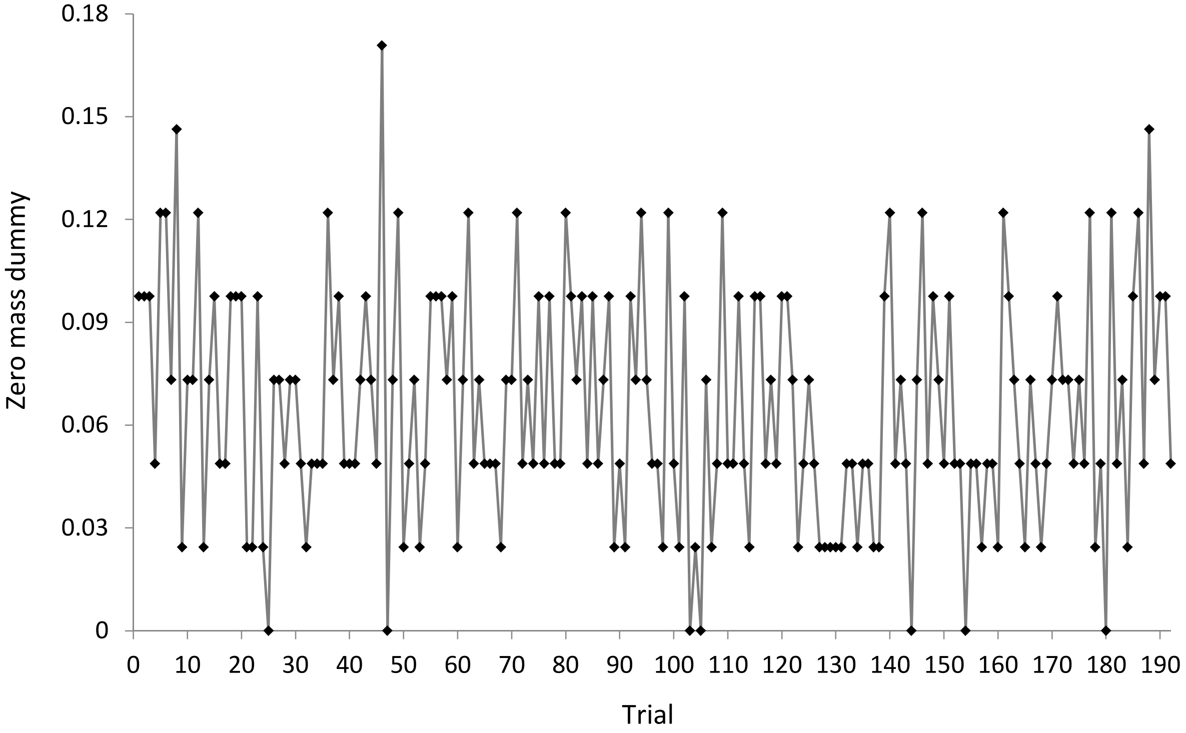
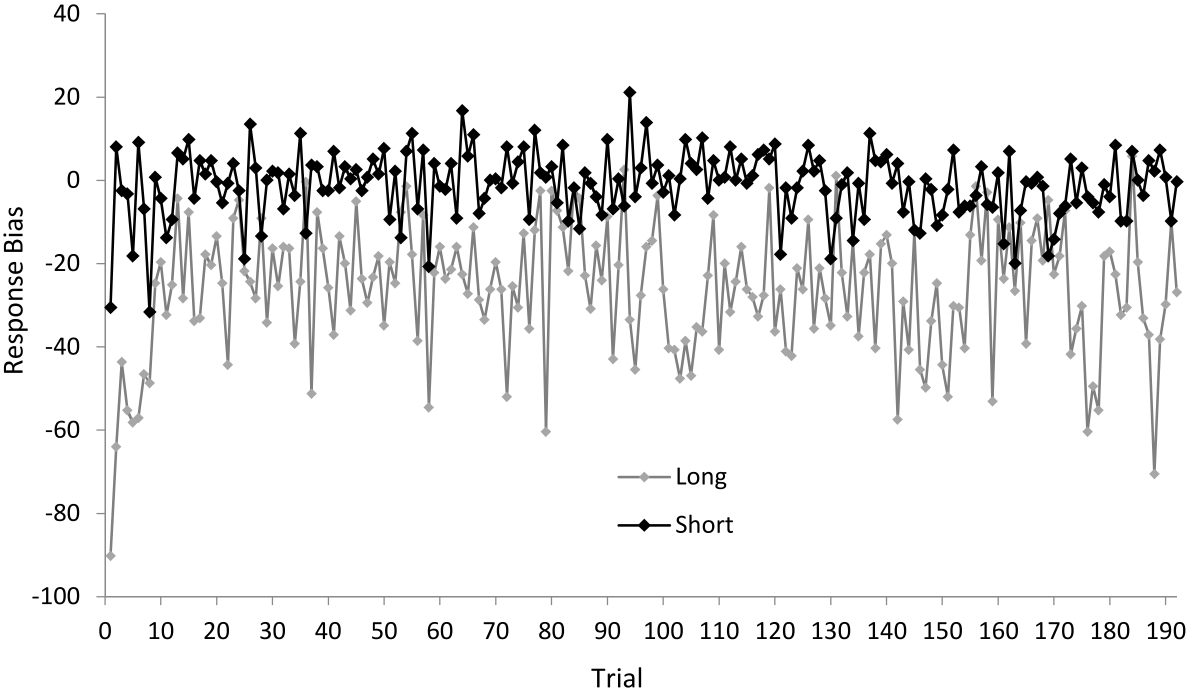


Figure 4: Mean response bias for long and short treatments across trials



Supplemental Online Appendix

Running mean regressions, fixed-effects analysis

The analysis summarized in Table 3 finds only weak evidence that the running mean is related to response. However, the reader might be concerned that the results are not robust to the specification of the repeated nature of the data. Here we perform an analysis with the same independent variables but we offer a different repeated measures specification. We do not assume a correlation between judgments by the same participant, but rather we account for the heterogeneity by estimating a unique intercept for each participant. In other words, rather than running random-effects regressions, here we run fixed-effects regressions. These regressions are summarized in Table A1.

Table A1: Fixed-effects repeated measures regressions of the response variable

	Normal	Uniform	Short	Long
Target	0.765***	0.753***	0.833***	0.730***
	(0.008)	(0.008)	(0.008)	(0.014)
Running mean	0.147*	0.082	-0.008	0.063
	(0.069)	(0.059)	(0.080)	(0.122)
-2 Log L	18221.7	16639.9	18684.8	20341.1
Observations	1882	1680	2095	2056

Notes: We provide the coefficient estimates with the standard errors in parentheses. We examine trials 2 through 192. We do not provide the estimates of the intercepts or the participant dummies. † indicates significance at *p* < .1, * indicates significance at *p* < .05, and *** indicates significance at *p* < .001. -2 Log L refers to negative two times the log-likelihood.

Similar to Table 3, here target is significantly related to response in every specification. Also similar to Table 3, running mean is only significant in the normal treatment specification.

Preceding targets, fixed-effects analysis

Table 4 reports that, in every treatment, previous target is significantly related to response whereas running mean is not related to response. This analysis was conducted with a random-effects analysis. Here we perform the analysis with fixed-effects regressions. These regressions are summarized in Table A2.

Table A2: Fixed-effects repeated measures regressions of the response variable

	Normal	Uniform	Short	Long
Target	0.766***	0.753***	0.835***	0.735***
	(0.008)	(0.008)	(0.008)	(0.014)
Running mean	0.102	0.049	-0.095	-0.019
	(0.070)	(0.060)	(0.080)	(0.123)
Previous target	0.030***	0.025***	0.053***	0.058***
	(0.008)	(0.008)	(0.008)	(0.014)
-2 Log L	18215.9	16637.6	18653.4	20330.9
Observations	1882	1680	2095	2056

Notes: We provide the coefficient estimates with the standard errors in parentheses. We examine trials 2 through 192. We do not provide the estimates of the intercepts or the participant dummies. † indicates significance at $p < .1$ and *** indicates significance at $p < .001$. -2 Log L refers to negative two times the log-likelihood.

We note that the results in Table A2 are nearly identical to that in Table 4.

The analysis summarized in Table 5 finds that the preceding targets variable offers a better prediction of response variable than running mean. In other words, rather than running random-effects regressions, here we run fixed-effects regressions. Table A3 summarizes this fixed-effects analysis.

Table A3: Fixed-effects repeated measures regressions of the response variable.

	None	Prec 1	Prec 3	Prec 5	Prec 10
Target	0.765***	0.766**	0.766***	0.766***	0.766***
	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)
Running mean	0.0869*	0.0381	0.0272	0.0436	0.0291
	(0.0368)	(0.0372)	(0.0384)	(0.0398)	(0.0436)
Preceding targets	-	0.0343***	0.0445***	0.0331**	0.0479*
		(0.0045)	(0.0084)	(0.0117)	(0.0193)
-2 Log L	74477.8	74428.4	74457.4	74476.8	74477.7

Notes: We provide the coefficient estimates with the standard errors in parentheses. We examine trials 2 through 192. We do not provide the estimates of the intercepts, the treatment dummies, or the participant dummies. All regressions have 7713 observations. † indicates significance at $p < .1$, * indicates significance at $p < .05$, ** indicates significance at $p < .01$, and *** indicates significance at $p < .001$. -2 Log L refers to negative two times the log-likelihood.

Just as in Table 5, preceding targets is significant in every specification, and running mean is not significant in any specification that accounts for the previous lines.

Simulated response35 variable

In Table 6 we analyzed the simulated Respone25 variable. Here we perform the identical analysis with the simulated response35 variable, which contains noise with a standard deviation of 35 pixels, rather than 25 pixels. Table A4 summarizes this analysis.

Table A4: Random-effects repeated measures regressions of the simulated response35 variable.

	No Prec	Prec 1	Prec 3	Prec 5	Prec 10
Target	0.921***	0.921***	0.921***	0.921***	0.921***
	(0.005)	(0.005)	(0.005)	(0.005)	(0.005)
Running mean	0.112**	0.111**	0.113**	0.130**	0.118**
	(0.037)	(0.038)	(0.039)	(0.040)	(0.043)
Preceding targets	-	0.0012	-0.0008	-0.0144	-0.0052
		(0.0047)	(0.0087)	(0.0121)	(0.0199)
-2 Log L	76560.9	76569.7	76568.6	76566.5	76566.8

Notes: We provide the coefficient estimates with the standard errors in parentheses. We examine trials 2 through 192. We do not provide the estimates of the intercepts, the treatment dummies or the covariance parameters. All regressions have 7831 observations. † indicates significance at *p* < .1, ** indicates significance at *p* < .01, and *** indicates significance at *p* < .001. -2 Log L refers to negative two times the log-likelihood.

In every specification, running mean is significant at .01 and in none of the specifications is preceding targets significant. We also note that a fixed-effects analysis, rather than a random-effects analysis, does not change these results.

We note that the noise in the analysis of Table A4 exceeds that in our original analysis in Table 4, as can be seen by comparing the -2 Log L values. We also note that the noise in the analysis of Table 6 is less than that in the analysis of Table 5, as can be seen by comparing the -2 Log L values. Given the results of Tables 6 and A4, we reject the criticism that the declining standard deviation of running mean prevents satisfactory estimates of the coefficient of the running mean variable. Further, whereas Table 6 and Table A4 perform a random-effects analysis, we also perform fixed-effects versions of these analyses and the results are not changed.

Responses with zero mass across trials

We conduct an analysis similar to Table 7, but Table A5 summarizes this on only the first half of trials. There are 250 responses with a zero mass and 3639 without. As it would not be identified, we do not include the First half specification.

Table A5: Fixed-effects logistic regressions of the zero mass dummy variable.

	Trial	Sqrt. Trial	First 5	First 10	First 20
Trial	0.0005	0.0025	-0.072	0.063	0.178
	(0.0028)	(0.0339)	(0.365)	(0.258)	(0.183)
-2 Log L	1163.5	1163.5	1163.5	1163.4	1162.6

Notes: We provide the coefficient estimates with the standard errors in parentheses. We examine trials 1 through 96. We do not provide the estimates of the intercepts, the participant dummy variables, or the treatment-target dummy variables. All regressions have 3889 observations. † indicates significance at *p* < .1. -2 Log L refers to negative two times the log-likelihood.

Similar to the results in Table 7, here we do not find evidence that zero mass responses became less likely across trials. This suggests that the participants either did not learn this aspect of the distribution or they did not use this to inform their judgments.

Bias towards the mean across trials

Table A6 was performed as Table 8, with the running mean bias as the dependent variable, but on only the first half of trials.

Table A6: Random-effects regressions of the running mean bias variable.

	Trial	Sqrt. Trial	First 5	First 10	First 20
Trial	0.061***	0.803***	-6.404**	-4.150**	-4.065***
	(0.016)	(0.196)	(2.205)	(1.501)	(1.089)
-2 Log L	35919.9	35913.2	35916.6	35918.1	35912.5

Notes: We provide the coefficient estimates with the standard errors in parentheses. We do not provide the estimates of the intercepts, the covariance parameters, or the treatment-target dummy variables. We examine trials 2 through 96. All regressions have 3851 observations. ** indicates significance at *p* < .01 and *** indicates significance at *p* < .001. -2 Log L refers to negative two times the log-likelihood.

Just as in Table 8, here we find strong evidence that the bias toward the running mean increases across trials.

The reader is possibly concerned that the running mean bias variable is not sufficiently close to the weight between the running mean and the noisy memory (λ). Therefore, we define the running mean bias ratio to be the distance between the target and the running mean divided by the sum of the distance between the target and the running mean and the distance between the response and the running mean:

Running mean bias ratio =

| Target – Running mean | / [| Target – Running mean | + | Response – Running mean |].

Table A7 was performed as Table 8, but with the running mean bias ratio on all trials.[42]

Table A7: Random-effects regressions of the running mean bias ratio variable.

	Trial	Sqrt. Trial	First 5	First 10	First 20
Trial	0.00014*** (0.00003)	0.00267** (0.00056)	-0.033** (0.013)	-0.026** (0.009)	-0.026*** (0.006)
-2 Log L	6214.5	6224.9	6215.3	6216.7	6225.5

Notes: We provide the coefficient estimates with the standard errors in parentheses. We examine trials 2 through 192. We do not provide the estimates of the intercepts, the covariance parameters, or the treatment-target dummy variables. All regressions have 7712 observations. ** indicates significance at $p < .01$ and *** indicates significance at $p < .001$. -2 Log L refers to negative two times the log-likelihood.

Similar to that in Table 8, here we find strong evidence that the bias toward the running mean increases across trials.

Table A8 was performed as Table 9, with previous bias as independent variable, but on only the first half of trials.

Table A8: Random-effects regressions of the previous bias variable.

	Trial	Sqrt. Trial	First 5	First 10	First 20
Trial	0.064*** (0.019)	0.875*** (0.238)	-9.253*** (2.669)	-6.610*** (1.817)	-4.389*** (1.319)
-2 Log L	37353.8	37346.2	37342.8	37342.4	37345.2

Notes: We provide the coefficient estimates with the standard errors in parentheses. We do not provide the estimates of the intercepts, the covariance parameters, or the treatment-target dummy

[42] One observation was such that the running mean was equal to both the target and the response, thus implying an undefined running mean bias ratio. Therefore we have one fewer observation in Table A7 than in Table 7.

variables. We examine trials 2 through 96. All regressions have 3851 observations. *** indicates significance at $p < .001$. -2 Log L refers to negative two times the log-likelihood.

As with Table 9, we see that there is an increase in the bias toward the previous line across trials. We also note that a fixed-effects specification does not change these results.

We now perform an analysis, identical to that in Table 10, but restricted to the first half of trials. Table A9 summarizes this analysis. We note that CAM would predict a negative estimate for Trial and positive estimates for the others.

Table A9: Random-effects logistic regressions of the absolute response bias variable.

	Trial	Sqrt. Trial	First 5	First 10	First 20
Trial	0.070***	0.797***	-0.569	-2.467*	-3.494***
	(0.013)	(0.158)	(1.653)	(1.192)	(0.886)
-2 Log L	34881.9	34881.0	34901.6	34898.1	34887.4

Notes: We provide the coefficient estimates with the standard errors in parentheses. We examine trials 1 through 96. We do not provide the estimates of the intercepts, the covariance parameters, or the treatment-target dummy variables. All regressions have 3889 observations. † indicates significance at $p < .1$, * indicates significance at $p < .05$, and *** indicates significance at $p < .001$. -2 Log L refers to negative two times the log-likelihood.

Similar to Table 10, here we find that absolute response bias is increasing in the Trial, First 10, and First 20 specifications.