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The dissociation between the recall of stimulus frequencies and the judgment of contingency allows the placement of the competition effect in the final causal processing stages

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In the predictive learning and causal reasoning literature it has been suggested that the processing of events is under the control of a competitive mechanism. However, little is known about whether the competitive mechanism operates at the encoding or near the response stages. The present work suggests that measures based on the recall of frequencies of the cells in the contingency table could help us in the placement of the competition principle within the processing stages. As the contingency judgment about a constant symptom-illness relation changed according to the validity of a second different symptom, we concluded in favour of a competition mechanism. However, estimated frequencies did not change as a result of such manipulation. This dissociation suggests that the competitive mechanism operates near the response stage rather than at the stimulus encoding period.

In the predictive learning and causal reasoning literature it has been suggested that the processing of events is subject to a competitive mechanism. According to this principle, the causal or predictive relationship attributed to an element is engaged in a relative way to other potential agents. The competition principle was in fact evidenced in a systematic way in the frame of the relative validity paradigm (Wagner, 1969). In this procedure (see Baker, Vallé-Tourangeau and Murphy, 2000) it is usual to present two stimuli (A and B) as potential predictors of an outcome, and two experimental conditions. In both conditions the contingency of stimulus A is kept constant, whereas the contingency of stimulus B is changed from one condition to the other. The competition is computed by analysing the differences of the contingency judgment for stimulus A. Competition effects such as blocking, relative validity or discounting are interpreted as a coding or learning problem, as implicated by the Rewcorla-Wagner model (see Cobos, Caño, López, Luque,

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& Almaraz, 2000; Shanks, Holyoak and Medin, 1996). That is to say, in any such procedure we would learn that the validity of the stimulus A is low, as there is a good predictor (B) of the consequence.

On the contrary, research on conditioning (Matzel, Schachtman, and Miller, 1985) and causality learning (Matute, Arcediano, & Miller, 1996) has suggested that the competition principle takes place at the final stages of processing, those responsible for the decision or choice of response –i.e. the comparator hypothesis, Miller & Matzel (1988) –.

Recent work on causality learning has been aimed at uncovering the level of processing where the competition principle takes place. Matute et als (1996), following the comparator hypothesis, suggested that competition emerges not at the associative (learning) stage, but when subjects have to make inferential, predictive or diagnostic judgements. They compared inferential and contiguity (associative) judgments in high- and low-relative-validity conditions. The important assumption here is that contiguity judgments are an index of the associative process, but inferential ones are of the comparator process. The reliable interaction between type of judgment and relative validity suggests that competition is taking place beyond the learning stage.

On the opposite position, Cobos et als (2000), using a similar design, found a main effect of relative validity, but no reliable interaction when the type of judgement was manipulated either within- or between-subjects. These data are consistent with the Rescorla-Wagner account: competition occurs at the learning stage. Authors argued that an important procedural difference, list (Matute et al) versus trial-by-trial (Cobos et al) presentations, can account for the differences between Matute et al (1996) and Cobos et al (2000). A point we address here asking subjects to estimate the number of trials for each cell of the contingency table (cell frequencies, see Table 1 for an explanation).

From a normative point of view, the competition is asymptotically equivalent to a conditional statistical computation (Reichenbach, 1956; Salmon, 1984; Suppes, 1970). According to this approach, the predictive-causal validity of each cue is computed conditionalising on the other(s) potential cue(s) considered relevant (Spellman, 1996a). As the most frequent is a binary situation, such computations correspond to delta P (dP), which is a basic contingency computation (Allan, 1980, 1993. See Table 1). The dP is estimated from the difference of two conditional probabilities, estimated from the frequencies of all the possible stimulus combinations (see Table 1 for details). Thus, for any contingency situation we might compare two aspects: the frequencies encoding and the final judgment, which would be based on a probabilistic combination of such frequencies.

As the validity computation is based on frequencies of stimuli combinations, we could use a typical contingency judgment situation and ask for an estimation of the frequencies of the four types of information at the end of the experimental session. Thus, we obtain two relative validity conditions with the same contingency between A and the outcome, but with different contingency between B and the outcome. Several non-associative models of causality learning (Catena, Maldonado, & Cándido, 1998; Cheng, 1997,

Cheng & Novick, 1992; White, 2002, see Perales, Catena, Ramos, & Maldonado, 1999, for a review) assume that subjects count the number of trials in each cell of the contingency table, and that cell frequencies are used to derive causality (or covariation/contingency) judgments. Hence, frequencies computation precedes causality estimation. However, there is no role for cell frequencies in associative-like (Comparator hypothesis, Rescorla-Wagner) models. And there is no way to determine how frequencies can be derived from the processing of events assumed by these models, or what location in the hierarchy of processes involved in causality computation does cell frequencies estimates have.

Table 1. Computation of contingencies in a one-predictor/one outcome situation.

	Y	~Y	Probability
A	a	b	P_S= a/a+b
~A	c	d	P_N= c/c+d
			$dP = P_S - P_N$

Note: dP was estimated from the difference of two conditional probabilities, P_S and P_N. These probabilities were estimated from the cells frequencies (a, b, c, and d). The two predictor values were symbolized as A and no A (~A), and the two values of the outcome were symbolized as Y and no Y (~Y).

Our departure point is, therefore, completely different from those of Matute et al (1996) and Cobos et al (2000). We assumed, as most non-associative causal models do, that cell frequencies computation is one step before covariation/contingency/causality estimation. The rationale then would be straightforward. If the competition effect took place in the initial stages of predictive-causal processing, encoding of trial types, then the cells frequencies estimation, in the same way as the contingency judgment, would differ in the two experimental conditions.

In other words, the validity of stimulus B would affect encoding of stimulus A both in frequency and in perceived contingency. However, if the competition effect took place in the final processing stages, nearer the response, then the contingency judgment for A should be different but at the same time the recall of frequencies involving A would remain the same. That is, the contingency of the "shadowing" stimulus B would not affect the frequencies' recall but it would affect the final contingency judgment. Therefore, in addition to this "direct" contingency judgment, we would compute a subjective judgment, from the cells estimates, following the standard dP. Hence, we assume that if competition takes place near the response, direct contingency, but not subjective, judgment will be sensible to the validity manipulation.

METHOD

Participants. Thirty-six University undergraduates volunteered for course credits. Eighteen participants were randomly assigned to each validity group, High and Low, with a very similar distribution of sex and age.

Design and Procedure. A medical diagnostic task was used (i.e. Chapman and Robbins, 1990; Price and Yates, 1993, 1995). Subjects were told that they should imagine being members of a medical committee that had to evaluate the relationship between certain symptoms and a fictitious illness. On each trial there appears the photograph of a face representing a fictitious patient. On the photo, information was presented about symptoms (presence or absence) and, immediately afterwards, about the disease (presence or absence). Symptoms were coloured squares placed on different locations of the patient's face and the illness was represented as a square with the name of the syndrome -Luchy- written inside it.

The names of the symptoms ("Prurosis" and "Regmentacion") and the disease were invented words. Besides, the two symptoms were counterbalanced, so that for half the participants the type A relative validity symptom was labelled "Prurosis" and B symptom was called "Regmentacion"; for the remaining participants, it was carried out in an inverse way. Thus, from now on we will refer to the cues as A and B instead of using the symptoms' labels.

Half of the subjects received a high validity (0.43/0.80) and the other half a low validity (0.43/0.41) condition. B-outcome contingency was 0.80 and 0.41, respectively for high and low validity conditions. A-outcome contingency was 0.43 for both conditions. So, we have a between-subject factor, validity conditions: high vs low, and three dependent variables, direct judgment, subjective judgment and cells estimates. Details on the number of trials for each cell are displayed in Table 2.

After every five trials, participants had to answer the following question: "What is the strength of the relationship between the symptom (A or B) and the illness?". They had to answer using a scale that appeared below the question. The scale went from –100 to +100 with 5-point intervals. Point +100 was labelled as "maximum positive relationship", 0 as "no relationship", and –100 as "maximum negative relationship". A note reminded the participants that they should respond bearing in mind all the information they had seen from the beginning of the session.

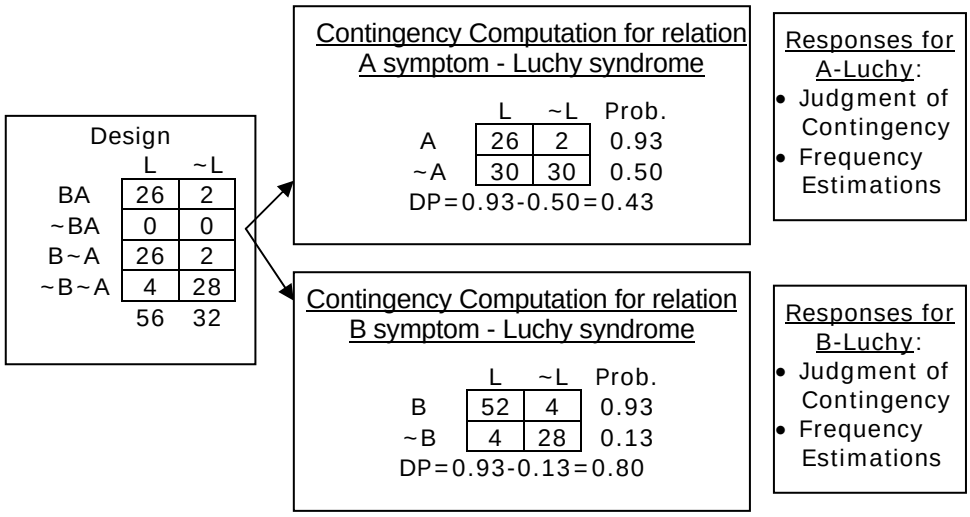
These two symptoms were presented according to a relative validity design with two conditions. We made the validity for the symptom A equal in the two conditions (dP of 0.43), and different regarding the symptom B: dP of 0.80 in the High group and 0.41 in the Low group.

We also asked, at the end of the experiment, an estimation of the cells frequencies for both symptom-illness pairs. Since the competition mechanism is asymptotically evaluated, we will only analyze and present the final judgment (global) and the frequency estimations at the end of the

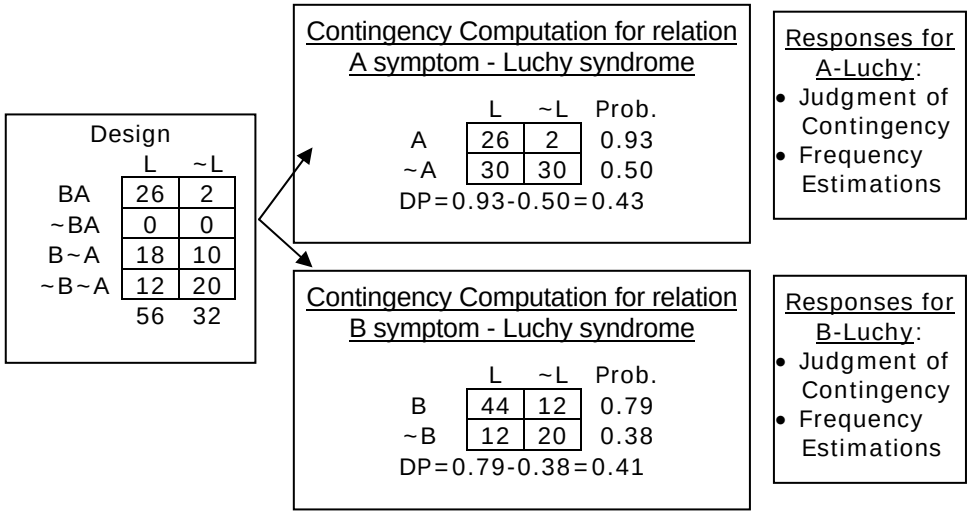
experimental session. Subjective judgment was computed, following dP, [a'/(a'+b')-c'/(c'+d')], being a', b', c', and d', the cells frequency estimates. All the statistical decisions were adopted fixing a significance level of 0.05.

Table 2. Design of the experiment.

A. HIGH VALIDITY CONDITION (0.43 / 0.80)



B. LOW VALIDITY CONDITION (0.43 / 0.41)



Note: Two symptoms - B and A- were the probable predictors of the outcome -the Luchy's syndrome (L)- for the two groups: High (top) and Low validity (bottom). The design (right) leads us to computations according to the dP contingency approach (second column). The probabilities (Prob.) and dP coefficients for these calculations are computed as in Table 1. The dP are given for B (upper part of each square) and A symptoms (lower part). ~ stands for the absence of the stimulus.

RESULTS

The most interesting results are summarised in Figure 1 and Figure 2. On the left we can see the mean estimations for each of the four cells frequencies. On the right we show the participants' direct (Observed) contingency judgment, and also the 'Subjective' judgment as a function of the frequencies estimations. The Figure 1 represents the results for symptom A and the Figure 2 those for symptom B.

Mean contingency judgments on cue A appear to be higher in the low than in the high validity group. A t-test on these judgments indicated that this difference was reliable $t(34)= -2.45$; $p<0.05$. However, there were no significant differences regarding the subjective judgment ($t<1$). Mean judgment for cue B was higher for the high than for the low validity group, $t(34)= 3.16$, $p<0.05$. Subjective judgments for B were higher for the high than for the low validity condition, $t(34)= 3.27$, $p<0.05$. Therefore, validity manipulation affected, as expected, the judgment on B both in subjective and direct contingency judgments, but affected, as expected, only to direct judgments on A.

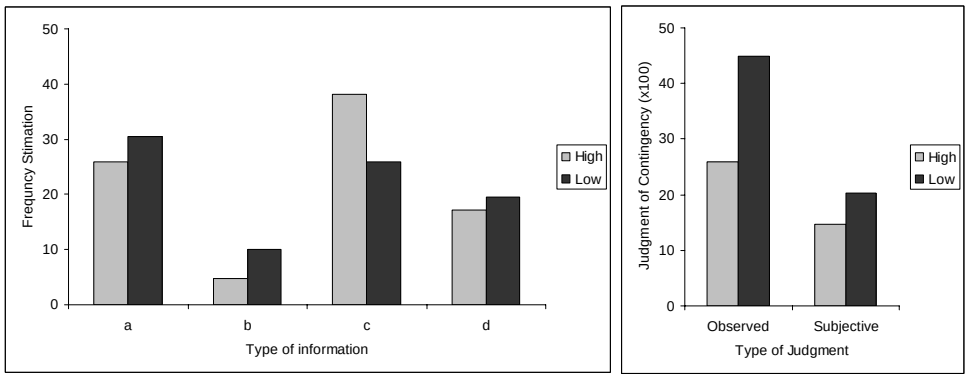


Figure 1. Cells estimates, subjective dP, and Judgment of Contingency for symptom A.

Mean frequency estimations of cells a, b, c, and d were very similar in the two validity conditions for A. A 4 (Cell: a, b, c or d) x 2 (Validity Condition: High vs. Low) ANOVA was very near to significance, $F(3,102)= 2.44$; $MCe= 250.81$; $p=0.068$. Even though this is not relevant for our working hypothesis, we will focus on a detailed analysis of simple effects, assuming the interaction effect, thus gaining statistical power. Furthermore, any differences between validity groups in any cell estimate would work against our main hypothesis, as can be interpreted as an index of validity effects.

The LSD post hoc indicated that all the mean frequencies of these estimations were equivalent between the two conditions. For **type a** estimation, $t(34)= -0.75$; Standard Error of Difference (SE)= 6.09; $p>0.05$; **for type b** estimation $t(34)= -1.91$; SE= 2.79; $p>0.05$; **for type c** estimation

t(34)= 1.97; SE= 6.20; p>0.05; and **for type d** estimation t(34)= -0.67; SE= 3.47; p>0.05. Therefore, even relaxing the criterion for statistical significance, no effect of validity manipulation was observed on A cells estimates.

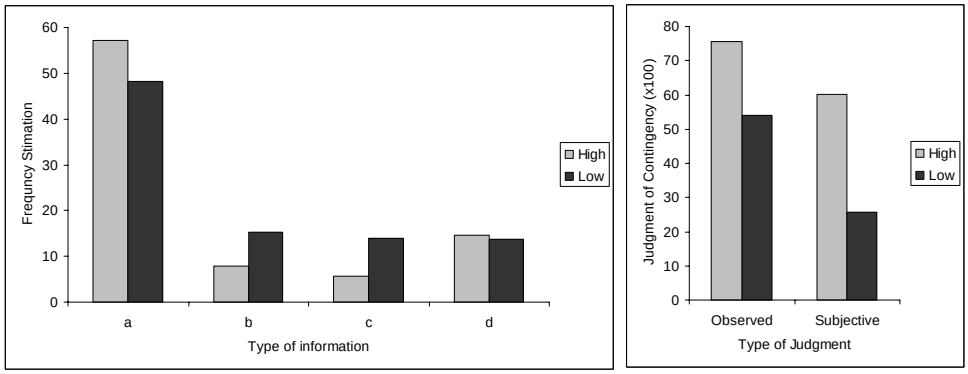


Figure 2. Cells estimates, subjective dP, and Judgment of Contingency for symptom B.

In general, there were differences on mean frequency estimations in some of the cells between the two validity conditions for B symptom. A 4 (Cell: a, b, c or d) x 2 (Validity Condition: High vs. Low) ANOVA was significant, F(3,102)= 6.13; MCe= 93.51; p<0.05. Thus, we will focus on a detailed analysis of simple effects, assuming the interaction effect.

The LSD post hoc indicated that the mean frequencies of these estimations were equivalent between the two conditions of validity for type "b" and "c" but not for type "a" or "d". For **type a** estimation, t(34)= 1.94; Standard Error of Difference (SE)= 4.53; p>0.05; **for type b** estimation, t(34)= -3.60; SE= 2.01; p<0.05; **for type c** estimation, t(34)= -4.10; SE= 2.03; p<0.05; and **for type d** estimation, t(34)= 0.35; SE= 2.67; p>0.05. Therefore, an effect of validity manipulation was observed on B symptom-illness cells estimates.

DISCUSSION

Contingency judgment on a stimulus "A" depended on the relation between a second stimulus B and the outcome. However, the estimates of frequencies of each type of trial (a, b, c, and d) did not depend on the contingency of B. It appears that subjects were able to compute accurately the information necessary to do a correct estimate of A-illness relationship, but they were not able to use it in their contingency judgments. The direct comparison between the two types of judgments, subjective judgment derived from cells estimates and direct judgment displayed in Figure 1 supports our conclusion.

Moreover, it was confirmed that the judgment task was adequately understood, as results about the shadowing symptom –B– evidence. The direct comparison between the two types of judgments, subjective judgment

derived from cells estimates and direct judgment, displayed in Figure 2, supports our conclusion. Differently from what A results evidenced, the validity manipulation caused differences in the direct contingency judgment, in the subjective one, and also in the frequencies estimations. This can be explained considering that the programmed contingencies for this stimulus involved different frequencies for the two validity conditions.

However, the differences of frequencies estimations were only significant for the disconfirming cells of relation –b and c– but not for the confirming ones –a and d–. The direction of the differences in b and c corresponds to normative positions (lower frequencies in the high validity condition than in the low validity one). This could be explained considering that even though the programmed difference was the same for the four cells in absolute terms, the normative impact of the differences in those cells that involved differences , -4 vs 12 for "b" and 4 vs 12 for "c"- was proportionally higher and more prominent with regards to those cells in which there were no differences -52 vs 44 for "a" and 28 vs 20 fpr "d"- (see the lower section of the two quadrants in Table 2 for normative details).

The differences observed in contingency judgment to A add to the ones found in many studies using other procedures (see, for example, Matute et al, 1996). In fact most of the studies on competition using the relative validity paradigm have systematically replicated this competitive effect (Spellman, 1996b. See Baker et als, 2000 for an empirical review). Usually the competition has been interpreted suggesting that learning about A is affected by the validity of B (see Shanks et als, 1996), that is to say, that subjects do not correctly encode the relationship between A and the outcome because B shadows this relation.

However, the fact that the recall of cell frequencies on the A-outcome relationship was not affected by the validity of B cannot be easily accommodated by this encoding deficit explanation. If encoding was inadequate we would have observed the same differences in cells frequencies count than in contingency estimates. Therefore the present results do not follow the predictive learning literature, or causal reasoning studies, although they are consistent with those of Price and Yates (1993, 1995). According to these authors, there was a predictive competition effect on the different types of subjective judgment connected with the comprehension of statistical relationships. This effect was observed for a global judgment and for the estimation of the conditional probabilities (1995, Experiment 1). However, the competition effect was absent for the combinations of stimulus estimations (1995, Experiment 2)[1].

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[1] In our terminology, the global judgment is comparable to a dP subjective estimation, the estimation of the conditional probabilities (or relative proportions) is comparable to P_S-P_N subjective estimations, and the frequency estimations of raw data are similar to the frequency estimation of the 4 types of essays relevant to the combinations of binary events (see Tables 1 and 2).

Thus, in general terms, our results agree with others obtained in causal learning literature. Nevertheless, the Price and Yates (1995) experiments did not measure the two types of answers in the same experiment, and in fact the competition effect magnitude clearly varied throughout the different experiments introduced. Our results show, as a whole, the possibility that the competition effect is not a matter of encoding but rather, of decision-making. Consequently, these results tend to suggest that the effect takes place in later stages of processing, either at an inferential one, as suggested by Matute et al (1966) and DeHouwer (2002), or at evaluative/decision ones, although the recall of events is not altered following the competition principle. In fact, recent judgment literature seems to point in this direction (for a review, see Shanks et als, 1996). For example, Jones, Wills and McLaren (1998) explain what could be attributed to the learning mechanism and to the final stages of decision-making, by thoroughly characterising the decision-making mechanism. Following these authors, the differences observed in contingency judgments cannot be attributed to the operation of a learning mechanism, but rather, to the operation of a decision-making one, that turns what has been learnt into responses.

RESUMEN

La disociación entre el recuerdo de las frecuencias estimulares y el juicio de contingencia permite ubicar del efecto de competición en las etapas finales del procesamiento causal. En el ámbito del aprendizaje predictivo y el razonamiento causal, se supone que el procesamiento de los eventos está sujeto a un mecanismo de carácter competitivo. Normalmente se asume que dicha competición tiene lugar en las etapas iniciales de codificación. Sin embargo, algunas investigaciones recientes desafían esta concepción. Nuestro trabajo sugiere que las medidas basadas en el recuerdo de las frecuencias de las casillas de la tabla de contingencia ayudan a localizar el nivel de procesamiento en el que se produce la competición. En la medida en la que el Juicio de Contingencia sobre un síntoma constante cambió según la validez de un segundo síntoma, podríamos concluir a favor del principio de competición. No obstante, la estimación de las frecuencias no varió en función de dicha manipulación. En consecuencia, podríamos más bien situar la acción competitiva en las etapas finales de procesamiento.

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