Habituation to illness: Effects of prior experience with the US on the formation of learned taste aversions in rats

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Experiment 1 investigated the effects of US habituation on the acquisition and extinction of learned taste aversions in rats. Subjects receiving five noncontingent LiCl intubations prior to conditioning failed to develop a conditioned taste aversion, while control subjects experiencing a single saccharin/LiCl pairing displayed a pronounced taste aversion which weakened during subsequent poisonings. Experiment 2 examined whether habituation, defined as a waning of responses to repeated presentation of an illness stimulus, was a possible mechanism for explaining the results of Experiment 1. Subjects showed a decrease in motor activity following an initial LiCl intubation, but less attenuation of activity with successive intubations.

The influence of gustatory novelty upon food intake has been repeatedly demonstrated (Revusky & Bedarf, 1967; Rodgers & Rozin, 1966; Rozin, 1968). Barnett (1958) has shown that wild rats tend to reduce food intake when familiar food is replaced by new substances or when novel objects are placed between the animal and its food. Rats poisoned after the ingestion of both novel and familiar foods not only show a consistent tendency to associate the poisoning with the novel solution (Kalat & Rozin, 1971; Wittlin & Brookshire, 1968), but also tend to increase their neophobia following ingestion of the sublethal dosage (Barnett, 1963). In addition, recent evidence suggests that novelty may be the most important variable affecting the maintenance of an association between taste and its consequences via an attentional or alerting mechanism (Nachman & Jones, 1974).

Familiarity with a taste prior to conditioning has been shown to reduce the magnitude of learned taste aversions (Domjan, 1972; Farley, McLaurin, Scarborough, & Rawlings, 1964; McLaurin, Farley, & Scarborough, 1963; Vogel & Clody, 1972). Similarly, the magnitude of a postconditioning avoidance of saccharin should be mitigated by habituation to the US prior to conditioning, an effect which has been demonstrated by Brookshire and Brackbill (Note 1), using apomorphine hydro-

chloride as the US, and by Elkins (1974), using cyclophosphamide. Brookshire and Brackbill were further able to show that exposure to the US prior to conditioning can alter the effectiveness of the US at the time of the CS-US pairing, but that exposure after conditioning cannot. Braveman (1975) carefully ruled out three possible explanations for these effects of prior US exposure. Neither addition nor tolerance to the illness-producing drug, nor interference or blocking by associations between extraneous stimuli and illness, could account for the decrements in taste aversions that Braveman found. He concluded that the associative capacity of illness can be reduced through preexposure to the US. Gamzu (Note 2) reported that prior exposure to one illness-producing drug followed by conditioning of taste aversion to another drug still resulted in an attenuation of the conditioned aversion. This suggests that the repeated experience of illness itself is the important factor in attenuating the conditioned aversion; exposure to the US *per se* should not produce such an effect. Experiment 1 was designed to examine the effects of US habituation on repeated cycles of acquisition and extinction of subsequent learned aversions.

EXPERIMENT 1

Method
The subjects were 20 250-g female albino rats obtained from a commercial breeder. They were housed in individual wire mesh cages and had ad-lib access to Purina Laboratory Chow. Water was available as described in the schedules below. For 5 days prior to testing, all animals were handled daily by the experimenter. When intubation of the subjects was required, it was done via a rubber stomach catheter attached to a 10-ml syringe.

The rats were randomly assigned to two groups. Ten were assigned to an experimental group for LiCl preconditioning, and 10 were assigned to a saline control group. Following acclima-

tion to the laboratory for 1 week, all animals were placed on a 23-h water deprivation schedule. After 10 days, water was made available from 100-ml graduated Richter tubes only during the specified hour. Following 1 week of familiarization with the Richter tubes, the experimental testing began.

The testing procedure consisted of a 10-day sequence during which the experimental group received experience with poisoning while the control group received physiological saline. Both groups were then given one exposure to the novel taste of saccharin and subsequently poisoned with lithium chloride. This taste/poison pairing was followed by a series of preference trials on which intake and preference for saccharin and/or water were monitored for both groups. Specifically, Days 1-10 were designated as "experience" days. On Days 1, 3, 5, 7, and 9, the subjects in the experimental group were removed from their cages and intubated with 6 ml of a 0.15-m LiCl solution (Rozin & Kalat, 1971); subjects in the control group went through the same procedure as the experimental group, except that they were intubated with 6 ml of isotonic saline. Subjects in both groups rested 48 h between intubations. On Day 11, designated as the test day, animals in both groups were given a two-bottle test with a choice between 1% saccharin solution and tap water during their 1-h drinking period. Both fluids were presented at room temperature, and the saccharin solution was prepared fresh daily to avoid any saturation of the solution. For this two-bottle test, the animals were kept in their home cages, and the two Richter tubes were inserted through an opening in the wire mesh and anchored onto the cage. A small container was placed under the mouth of the tube to catch any spillage. Following the 1-h two-bottle test, animals in both groups were intubated with the same concentration of a LiCl solution as given to experimental animals during the 10 experience days.

On the following day, during their 1-h drinking period and daily thereafter, the subjects were presented with a two-bottle preference test of a 1% saccharin solution and tap water. Each time the mean amount of saccharin consumed by both groups stabilized at the baseline level (mean amount consumed on Day 11, test day) for 5 days, subjects in both experimental and control groups were repoisoned and placed on extinction until the baseline was again attained. The poisoning and extinction procedure was repeated until mean level of saccharin consumption of subjects in both groups failed to reflect a conditioned aversion to the baseline.

Results

The results show that a conditioned taste aversion failed to develop when subjects received five noncontingent LiCl intubations prior to conditioning. In contrast, a pronounced taste aversion was formed by subjects in the control group following a single pairing of saccharin and LiCl poisoning. This finding is presented in Figure 1. Immediate suppression of saccharin drinking can be noted by subjects in the saline control group following a single intubation with LiCl, while a corresponding increase in saccharin consumption is observable by those subjects in the experimental group preexposed to LiCl. A Mann-Whitney two-tailed test performed on the saccharin consumption data revealed a significant difference between the two groups during the extinction period following the first poisoning (U = 7, p = .002).

The data also indicate that subjects in the LiCl preconditioning group did not form a conditioned taste aversion to their drinking water during preconditioning with the US. Mann-Whitney tests per-
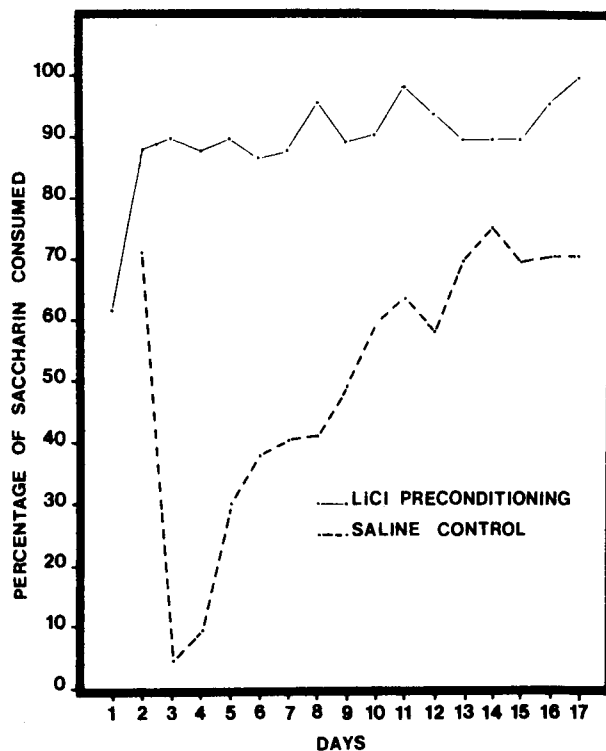

Figure 1. Mean percentage of fluid consumed. Values are shown for both groups following the first LiCl/saccharin pairings.

formed on fluid consumption data from subjects in the control and experimental groups during baseline and preconditioning phases indicate no significant differences between these groups (T = 46.5, p > .05; T = 99, p > .05).

Even after three saccharin/LiCl pairings and extinction trials, subjects in the LiCl preconditioning group failed to show a decrease in saccharin consumption. The amount of water and saccharin consumed by those subjects is presented in Figure 2, and, as can be seen from the figure, water consumption remained at a low stable level while saccharin consumption was maintained at a much higher level. For all three extinction periods which followed poisoning trials, water consumption was found to be significantly lower than saccharin consumption (U = 0, p = .002, Mann-Whitney, two-tailed). However, by examining the three corresponding extinction periods of the control subjects that received no LiCl prior to conditioning, it can be seen in Figure 3 that these subjects developed a dramatic aversion following the first poisoning, an aversion roughly one-half as strong following the second poisoning, and very little aversion following the third. A Friedman two-way ANOVA performed on these data showed a significant difference in saccharin consumption over the initial 10 days of the first and second extinction periods [$\chi^2r(9) = 24.55$, p = .05; $\chi^2r(9) = 21.22$, p = .05].
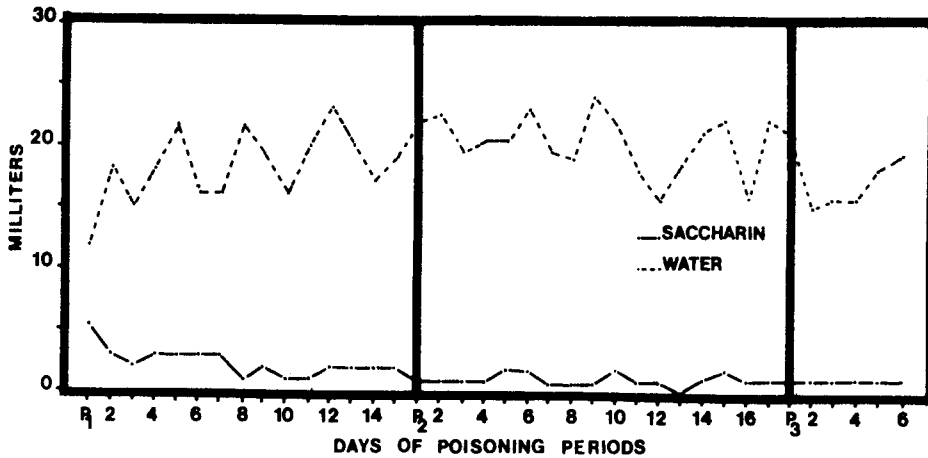
Figure 2. Mean amounts of saccharin and water consumed in milliliters over three poisonings (P₁, P₂, and P₃) for subjects in the LiCl preconditioning group.

EXPERIMENT 2

Experiment 1 demonstrated that subjects receiving five exposures to the US prior to conditioning failed to learn a saccharin taste aversion and that repeated experiences of acquisition and extinction resulted in a waning of the effects of taste aversion conditioning in a group not given the preexposure experience. The purpose of Experiment 2 was to examine whether habituation, defined as a waning of responses to repeated presentation of the illness stimulus, was a possible mechanism by which these phenomena occur. The toxic effects of LiCl have been demonstrated to result in a decrease in drinking (Nachman, 1963), a decrease in eating (Best & Zuckerman, 1971), gastrointestinal disturbance (Shou, 1957), and a period of observable inactivity (Nachman & Ashe, 1973). Braveman (1975) showed that tolerance to the illness-producing agent was insufficient as an explanation, but he did not measure tolerance by activity level. Operationally, it is difficult, at the behavioral level, to distinguish between tolerance and habituation. In the present research, the activity level of the subjects during each illness episode resulting from a series of three US exposure trials was monitored. It was hypothesized that an inverse relationship between attenuation of activity and the number of exposures to LiCl would be found, and that this decrease in attenuation of activity would represent the effects of habituation to repeated LiCl presentations.

Method

The subjects were eight female albino rats weighing 250-275 g. They were obtained from a commercial breeder and housed in individual cages. The subjects were given free access to Purina Laboratory Chow and water.

The rats were tested for locomotor activity in an open-field apparatus constructed with ¼-in. (1.9-cm) plywood to form a box 10 in. high and 18 × 18 in. wide (25.4 × 45.7 × 45.7 cm). The floor of the box was marked off into a grid composed of nine 6 × 6 n. (15.24 × 15.24 cm) squares. Intubation was done in the same manner as in Experiment 1.

Following acclimation to the laboratory and prehandling by the experimenter, the subjects were tested to obtain a baseline for activity. The subjects were transported from the animal housing facility to a quiet isolated room for testing. Each subject was
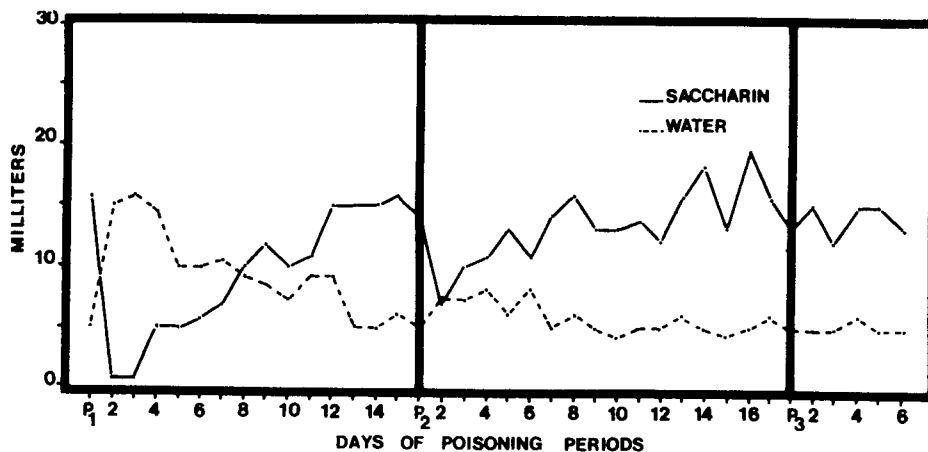

Figure 3. Mean amount of saccharin and water consumed in milliliters over three poisonings (P₁, P₂, and P₃) for subjects in the saline control group.

individually tested by removing it from its home cage and placing it in the open-field apparatus. Following a 3-min acclimation period, the experimenter silently recorded activity. Cumulative entries were made every 60 sec for a 30-min period, recording the number of times that at least one-half of the rat's body crossed a line on the grid floor. Instances of grooming, rearing, sniffing, shuddering, urination, and defecation were also noted by employing a modified frequency checklist. Three baseline activity measures were obtained for each subject. Following baseline activity measures, the rats were randomly divided into two groups. Four rats were assigned to an experimental group (LiCl experimental) and poisoned on LiCl, and four were assigned to a control group (saline control) which received physiological saline on the same schedule. Subjects in the experimental group were individually placed in the open-field apparatus and given 3 min for acclimation. The same measures obtained for baseline activity were recorded each minute for a 10-min period. At the end of the 10-min period, the subject was removed from the apparatus and intubated with 6 ml of a 0.15-m LiCl solution (Rozin & Kalat, 1971). The subject was then returned to the apparatus and given another 3-min acclimation period to minimize any alterations in activity due to the intubation procedure and handling. Two consecutive 10-min periods of observation were then made to make a total of 30 min of observation. The subject was immediately returned to its home cage following testing. Subjects in the control group received the identical procedure except that they were intubated with 6 ml of isotonic saline. Three such poison testing trials were carried out for all subjects in each group with a 48-h intertrial interval.

A reversal was carried out 48 h after completion of the three trials. For one trial, all four subjects in the experimental group were intubated with 6 ml of physiological saline, while those in the control group were intubated with 6 ml of a 0.15-ml LiCl solution. The same observational procedures that were used during the three previous trials were also employed for the reversal.

Results

The results of Experiment 2 showed a decrease in activity following intubation with LiCl. However, there appeared to be less attenuation of activity with subsequent intubations. Table 1 shows the mean open-field activity scores (in number of lines crossed/60-sec period) of subjects in both groups during the 10 min preceding intubation and the two 10-min observation periods following intubation for each of the three poisoning trials. As can be seen in Table 1, the activity level of both groups following intubation was depressed relative to their baselines, particularly in the case of the LiCl subjects. In addition to a possible decrease in activity due to intubation and handling, depression of activity in the LiCl group appears to be due to the debilitating effects of LiCl poisoning. Mann-Whitney two-tailed tests per-

formed on the periods following intubation revealed a marginally significant difference between control and experimental subjects for the first poisoning trial (U = 0, p = .014; U = 2, p = .057).

Data from the second poisoning trial indicate a reduced effect of LiCl poisoning, although the mean LiCl group activity was somewhat below baseline level. No significant difference was found between the activity levels of both groups during the first 10-min observation period (U = 5, p = .243, Mann-Whitney, two-tailed), but a marginally significant difference was found during the second observational period (U = 2, p = .057).

By the third poisoning trial, only a slight decrease in activity can be detected, and no significant differences were found between the groups for either period following intubation (U = 8, p = .557). Again, in the experimental group, however, there appears to be some reduction in activity relative to the high baseline level, indicating that LiCl poisoning may still be slightly aversive.

Results obtained from the behavioral observations supplement the locomotor activity data obtained with the open-field apparatus by demonstrating less attenuation of activity following successive poisonings. By the third poisoning trial, no observable differences in locomotor activity and movement-related behaviors could be detected between the poisoned and nonpoisoned subjects.

In addition, data obtained from the reversal manipulation, in which subjects in the LiCl experimental group were intubated with physiological saline and subjects in the saline control group received LiCl, indicate little difference between the effects of the procedure on both groups. The activity level of the LiCl experimental group during the reversal was found to be quite similar to that of the control subjects on the first day of the intubation series for all observational periods, even though the LiCl subjects had received three prior poisoning trials.

GENERAL DISCUSSION

As "baitshyness" appears to involve a case of Pavlovian conditioning in which the internal aversive events resulting from poisoning act as the US and the taste of the poisoned foodstuff as the CS, it was predicted that preconditioning habituation to the US should attenuate the association. Further, habituation may not only reduce unconditioned suppression to a stimulus, but may also block conditioning to it in a manner similar to preexposure to the CS (Lubow, 1965). Results of Experiment 1, in which a conditioned aversion to saccharin was blocked by three preconditioning LiCl intubations, support similar results by Brookshire and Brackbill (Note 1) and Elkins (1974). In both of these studies, an inverse relation was found between the frequency

Table 1
Mean Activity Scores for LiCl and Saline Control Groups for Each 10-Min Period of Three Successive Poisonings

	Poisoning 1			Poisoning 2			Poisoning 3		
	1	2	3	1	2	3	1	2	3
LiCl	10.7	2.5**	1.5*	9.4	3.7	2.5*	11.2	6.5	4.1
Control	7.9	4.1	4.6	6.4	4.0	5.3	8.2	5.3	5.6

*p = .057
**p = .014

of the preconditioning exposure to toxic illness and the initial magnitude of the aversion. Brookshire and Brackbill proposed that habituation to the US renders it less novel at the time of the CS-US pairing, suggesting that it is the subject's perception of the US as novel at the time of pairing which is the critical factor. In explaining his data, Elkins emphasizes habituation to illness and also stresses the reduction of the informational value of a single CS-US pairing due to repeated instances of illness related to specific consummatory responses.

Although Elkins (1974) and Brookshire and Brackbill (Note 1) attempted to account for their data, their results do not clarify the mechanism by which preconditioning US exposure weakens subsequent aversions. Results of Experiment 2 indicate that habituation to illness may be a contributing factor, since less attenuation of activity was found on successive poisonings, just as the magnitude of the conditioned aversion to saccharin varied over successive poisonings in Experiment 2. The failure to condition after prior US exposure may well be due in part to habituation to the debilitating effects of LiCl poisoning. However, an increase in activity level over subsequent poisonings cannot be interpreted solely as habituation to illness, since Vogel and Clody (1972) have obtained results with methylatropine that suggest that drug-induced nausea is not required for the formation of a conditioned taste aversion. Nachman and Ashe (1973) have also determined that the threshold dose needed to produce an aversion is approximately 1.0 mg/kg of a 0.15-m LiCl solution, a dosage at which the rat does not manifest the usual symptoms associated with lithium poisoning. However, the results of the present study suggest that subjects did habituate to LiCl over successive poisonings. Symptoms typically associated with LiCl poisoning, such as inactivity (Nachman & Ashe, 1973) and gastrointestinal disturbance (Shou, 1957) were not observed by the last poisoning trial. In addition, the activity level in the present experiment was suppressed following the first poisoning and partially suppressed during the second, indicating that LiCl was observably decreasing activity.

An interesting difference between the results of the present study and that of Elkins (1974) concerns the degree of attenuation of the learned association following preconditioning habituation to the US. Elkins reported that, following six preconditioning cyclophosphamide injections, a learned aversion to saccharin was formed which lasted at least 10 days before extinguishing, while the present study revealed no aversion to saccharin following only five preconditioning LiCl intubations. It is probable that these conflicting results are due to differences in the toxins used. Elkins also suggests that repeated episodes of illness following ingestion of a familiar taste should result in the development

an aversion even in subjects which have been exposed to many instances of prior illness. The results of Brookshire and Brackbill (Note 1) and those of the present study do not support this contention, since no attenuation of activity was noted following three subsequent conditioning trials. It may be argued that the 10-day extinction periods in the present study allowed extensive experience with the familiar taste which additionally blocked any learned aversion.

Results of the present study fail to support a notion of conditioned taste aversion as a "special case" of learning. Results of Experiment 2 indicated an increase in poison tolerance with habituation and present a situation which could readily be interpreted as equivalent to reduction in US intensity, a condition which is known to affect conditioning adversely.

In contrast, Braveman (1975) has concluded that an increase in poison tolerance cannot explain the attenuation of conditioning following preexposure to sickness. His position follows from data which show that pretraining experiences with either LiCl, methylscopolamine, or d-amphetamine sulfate were sufficient to block the formation of a rotation-induced aversion to saccharin. Braveman's work, however, does not rule out an interpretation involving habituation as a reduction in US intensity. It is unlikely that gastrointestinal disturbances induced by either toxins or rotations are so distinct that they do not produce overlapping effects. Gastrointestinal illness, irrespective of the manner in which it is produced, may be lessened over repeated occurrences.

Braveman (1975) has also suggested that animals may associate sickness with eating- or drinking-related cues during US pretraining. When animals are subsequently trained to avoid the distinctive taste of a novel substance, the preexisting associations may interfere with the establishment of a conditioned taste aversion. Although LiCl intubation followed the daily 1-h drinking period by an additional hour in the present study, fluid consumption data do not indicate that the animals developed any aversions to their drinking water. It seems an unlikely possibility that the attenuation of the saccharin aversion in Experiment 1 could have been due to any association between drinking water and illness, which resulted in a greater saccharin intake. Fluid consumption data from rats during baseline measurements of water consumption prior to conditioning showed no difference between control and experimental animals, even though the latter were receiving illness-inducing drugs. The failure to find any decrease in saccharin drinking by the LiCl pretraining group, then, cannot be explained by a learned aversion to water which might have produced strong saccharin preference. The greater degree of attenuation reported by Braveman (1975) compared to that presented here may be a result of the large LiCl dosage and the manner in

which it was administered. Braveman used a LiCl dosage of 0.3 m, twice that used in the present study, and administered the toxin by IP injection rather than oral intubation.

Another interpretation of the effects of pretraining with the US on the formation of a conditioned taste aversion might be made from a contingency viewpoint of conditioning. Although it is unlikely that visual, auditory and tactile cues are associated with poisoning under normal circumstances (Garcia & Koelling, 1966), it is possible that oral intubation may involve gustatory associations. Pretraining may have produced a near asymptotic level of conditioning between the potential gustatory cues arising from the intubation procedure and the effects of the toxin. The saccharin, therefore, may have had little or no capacity to become associated with the US, resulting in failure to form an aversion to saccharin.

Learned safety has been proposed as a mechanism to account for the CS-US delay gradient in learned taste aversions (Kalat & Rozin, 1973). Proponents of this view contend that during the delay the central representation of taste is not lost but, rather is, gradually reclassified from "possibly dangerous" associated with poison to "probably safe" unrelated to poison. The extinction periods following the three successive poison conditioning trials in Experiment 1 present a similar situation in which the subject is not able to forget the taste. Several days after the initial poisoning, the taste becomes associated with the absence of sickness. According to the learned safety view, if, at a later time, the rat is poisoned after drinking the same solution, previous learning that the solution is safe should interfere in some way with its learning that the solution is toxic. In Experiment 1, the magnitude of the aversion was roughly one-half as strong following the second conditioning trial and completely extinguished by the third. These data suggest that safe exposures do appear to reduce the general associability or salience of a solution (Best, 1975).

Our data in conjunction with the findings of others can be explained most satisfactorily, it seems to us, if one adopts the view that subjects may not only habituate to the US, but may also habituate (or show an increased tolerance) to the illness produced by the US, with the result that the US is rendered less intense and conditioning of the taste aversion is adversely affected.

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