inert[20,21]. But the soil C inventory below 1 m depth at Paragominas exceeds soil C above 1 m (Fig. 4*a*), as well as exceeding above-ground biomass (18 kg C $m^{-2}$). Contrary to the assumption of inertness, our isotopic data indicate the presence of a significant fraction of modern C in deep soils. First, the $\Delta^{14}CO_2$ (see Fig. 4 legend for definition of this quantity) below 1 m depth remains at or above modern atmospheric levels (Fig. 4*b*), indicating that the high concentrations of $CO_2$ observed at depth (Fig. 4*c*) result from root respiration and from microbial decay of carbon fixed by plants within the past 30 years[22]. Second, the $\Delta^{14}C$ of soil organic matter declines with depth, as expected, because the proportion of very old C increases with depth (Fig. 4*d*), but 10–15% of the soil C at 8 m depth may be modern (if the old C is essentially radiocarbon dead at a $\Delta^{14}C$ value of −1,000‰, the remaining modern fraction at +143‰ to +200‰ would be ~13% of the total C present at 8 m depth: $[(-850+1{,}000)/1{,}000]/[1+(143/1{,}000)] = 0.13$). This result suggests that up to 3 kg C $m^{-2}$ occurring below 1 m depth cycles on annual to decadal timescales, and that this C would be subject to change if root distributions were changed by land-use practices.

In the degraded pastures, woody plants have deep-root systems (Fig. 2) that may maintain the deep-soil C pool if fine-root turnover rates are similar to those in the forest. We have concentrated our studies of soil C dynamics on forests and degraded pastures, as these ecosystems are clearly the most abundant at present in eastern Amazonia. However, disk-harrowing and herd rotation can effectively exclude woody vegetation in more intensively managed pastures, and these practices are becoming more common[16,17]. Based on root distribution (Fig. 2), we would predict that these managed pastures would lose deep-soil C and possibly gain surface-soil C (a hypothesis which we now intend to test). Differences in pasture management that affect distributions of C inputs throughout the soil profile may help explain why some studies have shown increases while others have shown decreases in C inventories of the surface soil following conversion of forest to pasture[23–25].

How common is deep rooting in the Amazon Basin? Roots have been inferred to 5 m depth in a forest of Surinam[27]. Our shaft excavations to 8 m depth at forest sites that are less seasonal (Trombetas, Manaus) and more seasonal (Santana de Araguaia) than Paragominas (Fig. 1) revealed root distributions very similar to that of the Paragominas forest. These initial field data indicate that deep rooting is common in Amazonia.

We estimated the geographical distribution of deeply rooting forests in Brazilian Amazonia by overlaying monthly estimates of canopy greenness from AVHRR satellite imagery with a Geographic Information Systems database of rainfall from 212 weather stations. Where forests are evergreen but seasonal drought is significant (<1.5 mm $d^{-1}$ during the driest three months) we deduce that the forest must rely on water uptake from deep soil. The forests meeting these criteria cover an area of $\sim 1.8 \times 10^6$ $km^2$, which is most of the eastern and southern half of the Amazonian closed-canopy forest (Fig. 1). Hence deep roots play an important role in maintaining dry-season canopy greenness and evapotranspiration in the regions where human activity is concentrated. Deep roots are not limited to seasonally dry regions and may also play a role in nutrient uptake.

Deep roots help explain why Amazonian evergreen forests extend well into a region characterized by a long dry season. Understanding the effect of human land-use practices on regional budgets of water and carbon will require knowledge of the basic processes involving deep roots and deep soils. □

Received 10 February; accepted 16 November 1994.

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ACKNOWLEDGEMENTS. We thank V. Mendes for assistance with data gathering, K. Schwalbe for help with graphics, L. Martinelli and P. de Camargo for providing $\Delta^{13}C$ data and C. Uhl, L. Martinelli, D. Schimel and G. Woodwell for comments on the manuscript. This work was supported by NASA, the US NSF, the US Agency for International Development Global Climate Change Program, the MacArthur Foundation and the Andrew Mellon Foundation.

# Impaired recognition of emotion in facial expressions following bilateral damage to the human amygdala

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**STUDIES in animals have shown that the amygdala receives highly processed visual input[1,2], contains neurons that respond selectively to faces[3], and that it participates in emotion[4,5] and social behaviour[6]. Although studies in epileptic patients support its role in emotion[7], determination of the amygdala's function in humans has been hampered by the rarity of patients with selective amygdala lesions[8]. Here, with the help of one such rare patient, we report findings that suggest the human amygdala may be indispensable to: (1) recognize fear in facial expressions; (2) recognize multiple emotions in a single facial expression; but (3) is not required to recognize personal identity from faces. These results suggest that damage restricted to the amygdala causes very specific recognition impairments, and thus constrains the broad notion that the amygdala is involved in emotion.**

We studied subject S.M., a 30-year old woman with normal IQ (low average; Wechsler Adult Intelligence Scale-Revised (WAIS-R) full scale = 86), a high-school education, and a neuropsychological profile remarkable for a history of defective personal and social decision making[9,10]. On all occasions of testing, her mood was stable and cheerful, with no indication of depression on either observation or formal assessment (Beck Depression Inventory, Minnesota Multiphasic Personality Inventory). Her visual-perceptual discrimination assessed using unfamiliar faces was normal[10]. S.M. suffers from Urbach–Wiethe disease[11], a condition that caused a nearly complete bilateral destruction of the amygdala, while sparing hippocampus and all

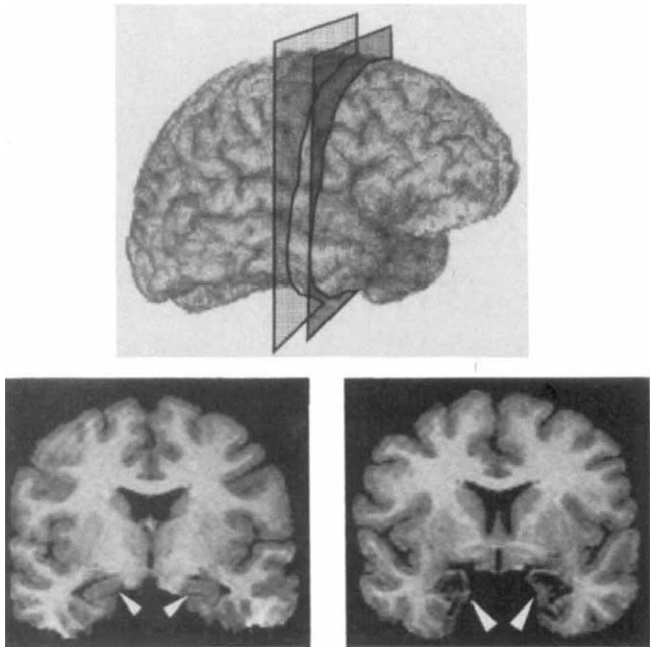


FIG. 1 $t_1$-weighted MR images of S.M.'s brain. Planes of section are shown at the top, on a three-dimensional reconstruction[26] of S.M.'s brain. There is extensive bilateral amygdala damage (lower right image, large arrowheads) with sparing of neocortex and hippocampus (lower left image, small arrowheads). The tissue of the amygdala has been replaced by mineral deposits as a result of Urbach–Wiethe disease[11].

neocortical structures, as revealed by detailed neuroanatomical analyses of her computed tomography (CT)[10] and magnetic resonance imaging (MRI)[9] scans (Fig. 1).

The human face conveys information about a person's identity, expresses emotion, and often signals blends of different emotions at the same time, all of which are elements critical to social behaviour. We therefore used ecologically relevant stimuli (human facial expressions of emotion[12]) to test the hypotheses that bilateral destruction of the amygdala impairs (1) recognition of some basic emotional facial expressions, and (2) recognition of the combination of several emotions shown in a single expression. We compared S.M. to 12 brain-damaged controls of similar IQ (mean WAIS-R full-scale score = 95 ± 9; see Fig. 2 Methods for details). Subjects were shown facial expressions of six basic emotions (happiness, surprise, fear, anger, disgust, sadness), as well as neutral faces, and asked to rate each face according to several emotional adjectives. S.M. differed dramatically from controls in this task. She rated expressions of fear, anger and surprise as less intense than did any of the brain-damaged controls (Fig. 2*a*; mean ratings for S.M. were >3 s.d. below control mean for these emotions). We next assessed S.M.'s recognition by correlating her ratings of facial expressions with the mean ratings given by seven normal controls with no history of brain damage. S.M. showed a severe recognition impairment specific to fear: her ratings of fearful faces correlated less with normal ratings than did those of any brain-damaged control (Fig. 2*b*), and fell 2–5 s.d. below the control mean when the data were converted to a normal distribution. By contrast, S.M.'s recognition of the unique identity of faces was fully preserved: she correctly identified without difficulty 19/19 photographs of familiar faces, some of whom she had not seen for many years, and she easily learned to recognize new faces. These data provide evidence for a double dissociation between processing of facial identity and of facial affect, suggesting that the two are subserved by anatomically separable neural systems[13–16]. Insofar as expressions of a single, basic emotion are concerned, the human amygdala's role appears to be quite specific to recognition of fear. This finding may parallel results from animal studies, which show that amygdalectomy results in abnormal behaviour to frightening stimuli[17–19].

To explore in greater detail S.M.'s abnormal processing of facial expressions, we examined her ability to recognize similarities between different expressions. We analysed S.M.'s ratings

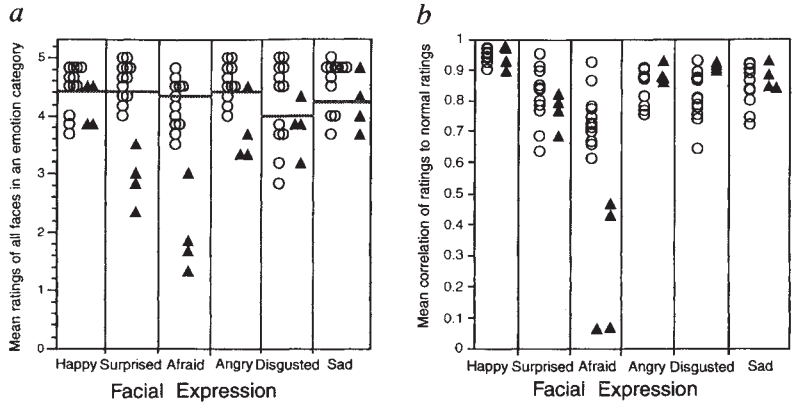



FIG. 2 Ratings of emotional facial expressions on emotional adjectives. *a*, Ratings of the intensity of an emotion expressed by typical expressions of that emotion. Rating scores on the emotional word for which the face was a typical example are shown as the mean of all faces within an emotion category. Data are from 12 brain-damaged controls (○), and from 4 experiments with S.M. (▲). Mean ratings given by 7 normal controls (with no history of brain damage) are denoted by the dotted lines. *b*, Correlations of ratings of facial expressions with the ratings given by normal controls. Mean Pearson correlations are shown of mean normal control ratings ($n = 7$) correlated with the ratings given by brain-damaged controls (○; $n = 12$) and with S.M.'s ratings (▲; 4 experiments). All normal controls' ratings correlated well with one another ($r > 0.7$ for every emotion category).

METHODS. Thirty-nine expressions of facial affect from Ekman[12] were presented nine times in two blocks separated by several hours. Six faces (both male and female) each of anger, fear, happiness, surprise, sadness, disgust, and three neutral faces, were projected on a screen, one at a time. Subjects judged each face on a scale of 0–5 (0 = not at all; 5 = very much) with respect to 9 adjectives: happy, sad, disgusted, angry, afraid, surprised, awake, sleepy, interested (1 adjective per trial). All data were used in Fig. 3 to obtain good MDS solutions; ratings on only the 6 most consistently rated adjectives (excluding the non-emotional words 'awake', 'sleepy', 'interested') were used in all other figures. Independent tasks showed that all subjects understood the adjectives. Correlations of a subject's ratings with normal controls' ratings were calculated for each facial expression, *Z*-transformed to normalize their distribution, averaged over faces that expressed the same emotion, and inverse *Z*-transformed to give the mean correlation for that emotion category. S.M. repeated the experiment 4 times, twice on 2 days separated by 6 months, with 2 experimenters who were blind to the nature of the experiment. Control subjects: all brain-damaged control subjects were selected from our patient registry and had been fully characterized neuroanatomically and neuropsychologically[27]. MRI or CT scans of each subject revealed no sign of damage to the amygdala. We excluded subjects who had emotional dysfunction (such as depression). To reduce a possible effect of task difficulty, our 12 controls included subjects with IQ lower than that of the target subject (S.M.). We took into account visual complexity of the stimuli and the possibly abstract nature of the task by including control subjects with impairments of vision (occipital lesions; $n = 6$) and executive functions (frontal lesions; $n = 3$). Five of the controls had bilateral lesions. All subjects used in our study had given informed consent to participate in research.

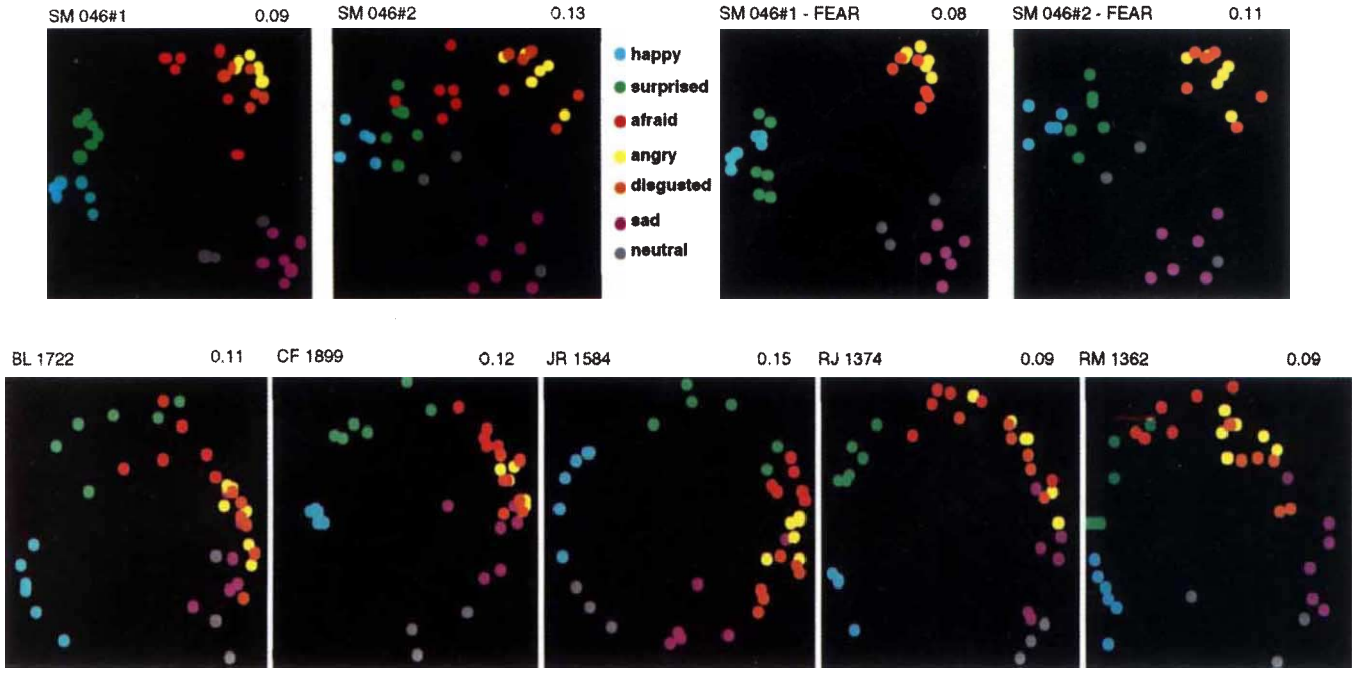
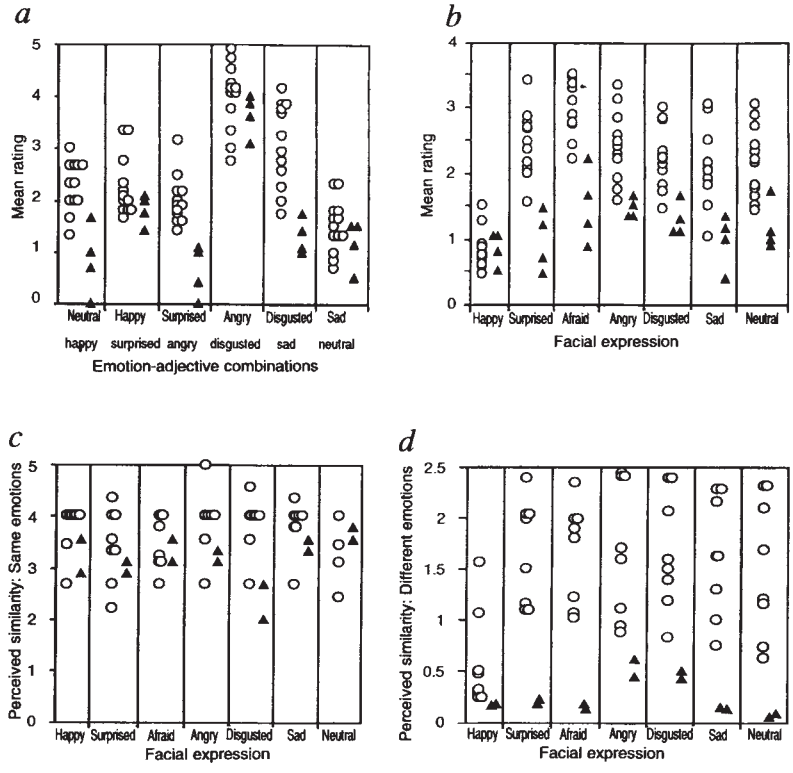



FIG. 3 Multidimensional scaling (MDS) of perceived similarity judgements of emotions expressed by faces. Each coloured point represents one of the 39 facial expressions which subjects had to rate. Euclidean distance between points corresponds to perceived dissimilarity between stimuli. Small numbers in upper left are the subject's code; to the right is the MDS stress, a measure of 'good-ness-of-fit' (smaller numbers are better fits). Top, MDS plots from 4 experiments with S.M. Each plot uses non-overlapping data from 2 different experiments. The left 2 plots show MDS on all the faces; the right 2 plots show MDS on all faces except fear. The similar structure of the left and right plots suggests that impaired recognition of fear alone does not account for the clustering seen. Bottom, Representative plots from 5 of the brain-damaged controls. We examined the data from all 12 controls, and also scaled all data with fearful faces subtracted; in every case we obtained circular arrangements. No control showed clusters like S.M., even when MDS stress was high.

METHODS. For each subject, ratings from 2 experiments were summed. Rating profiles for each of the 39 expressions were correlated with one another. This correlation matrix was used to compute non-metric MDS of dissimilarities in Euclidean 2-space with two algorithms[28,29] that yielded identical results. Similarity varied with Euclidean distance as a smooth, nearly linear, monotonically decreasing function. We show MDS solutions only for a stress $\leqslant 0.15$, corresponding to an RSQ $> 0.9$ (RSQ is a measure of the proportion of the variance in the data accounted for by the MDS solution).

FIG. 4 Recognition of multiple emotions in facial expressions. *a*, Mean ratings of facial expressions on adjectives denoting emotions adjacent in the MDS plots of Fig. 3. The emotion-adjective combinations are shown on the *x*-axis; data were averaged over 6 faces within each emotion category except neutral (3 faces), and over the 2 cases where emotional expression and adjective are interchanged. S.M.'s (▲, 4 experiments) low ratings compared to the controls' ratings (○, $n = 12$) correspond to the gaps in her MDS plots. *b*, Mean ratings of all the emotions other than the prototypical emotion recognized in an expression. S.M. tends not to endorse any emotions other than the prototypical ones. *c*, Direct similarity ratings of faces expressing the same emotion. *d*, Direct similarity ratings between faces expressing different emotions. Each category shows average similarity ratings of all 54 possible pairings of expressions (3 expressions of the given emotion ×6 other emotions ×3 expressions). S.M. fails to recognize similarity between expressions of different emotions (number of s.d. different from control mean: happy = 0.8, surprised = 2.8, afraid = 3, angry = 1.6, disgusted = 2.1, sad = 1.9, neutral = 2.5).

METHODS. *c*, *d*, Subjects directly judged similarity of emotion expressed by 21 of the faces used in all other experiments (3 of each of the 7 emotion categories) on a 5-point scale. All possible pairwise combinations (231) of facial expressions were presented as adjacent photographs, a pair at a time. Care was taken to ensure the subject understood both the rating scale and rated similarity of emotion. Data are from 8 of the brain-damaged controls, and from 2 experiments with S.M. All controls whose MDS is shown in Fig. 3 are shown here.



of emotional faces with a multidimensional scaling (MDS) technique, in which the perceived similarity among expressions corresponds to their proximity in the scaled representation (Fig. 3). For example, surprised and happy faces are rated similarly, and hence are adjacent in MDS plots, but happy and sad faces are very different, and hence far apart. The arrangement of emotions in the MDS plots of our controls (Fig. 3) is similar to what has been published for normal subjects, for judgements of emotional facial expressions[20] and emotional words[21] obtained in several different cultures[22]. This arrangement suggests that facial expressions have graded membership in categories of emotion, and that an expression can be a member of more than one emotion category. By contrast, S.M. perceived emotions more discretely, and did not generate the nearly continuous circular ordering that the controls showed. To ensure that S.M.'s abnormal MDS arrangement was not simply a consequence of her impaired recognition of fear, we also present the MDS solutions obtained without fearful faces (Fig. 3). The results are very similar. We also obtained similar results when we scaled stimuli on all the adjectives excluding 'afraid'. S.M.'s inability to perceive similarity between expressions of different emotions thus appears to be independent of her impaired recognition of fear, although the two may nonetheless be consequences of a single processing dysfunction.

Does S.M. fail to recognize similarities between expressions of different emotions because she cannot recognize the blend of multiple emotions conveyed by a single face? A quantitative analysis of S.M.'s inability to recognize multiple emotions in a facial expression is given in Fig. 4. The gaps in her MDS plots (Fig. 3) correspond to the endorsement of very low ratings when judging expressions of different emotions on each other's adjectives (for example, how much disgust is there in sad expressions, and vice versa?) (Fig. 4*a*). S.M. showed a tendency to recognize only the prototypical emotion in a facial expression, with the exception of some expressions that subjects all judge to be very similar, such as anger and disgust (Fig. 4*a*, *b*). This finding was replicated with a task that relies less on language. We asked subjects to rate each of 231 possible pairings of 21 photographs of facial expressions (3 of each emotion) in terms of similarity of the emotion expressed. S.M. appropriately judged all the expressions of the same emotion to be similar to one another, but she failed to recognize similarity among expressions of different emotions (Fig. 4*c*, *d*).

Facial expressions can convey both basic emotions whose expression and recognition may be partly innate[23,24], as well as subtler emotions whose meaning is partially determined by culture[25]. From our results, the amygdala appears necessary both to recognize the basic emotion of fear in facial expressions, and to recognize many of the blends of multiple emotions that the human face can signal. The amygdala may be an important component of the neural systems subserving social cognition in part because fine-grained recognition of the emotions signalled by faces is essential for successful behaviour in a complex social environment. □

Received 29 September; accepted 2 November 1994.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ACKNOWLEDGEMENTS. We thank N. Tranel, K. Brown and J. Marquis for technical assistance and G. W. Van Hoesen for comments. We thank all subjects, especially S.M., for their participation. This study was supported by a grant from the National Institute for Neurological Diseases and Stroke. R.A. is a Burroughs-Wellcome Fund Fellow of the Life Sciences Research Foundation.

# Mutations in the *DAX-1* gene give rise to both X-linked adrenal hypoplasia congenita and hypogonadotropic hypogonadism

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**ADRENAL hypoplasia congenita (AHC) is an X-linked disorder characterized by primary adrenal insufficiency[1,2]. Hypogonadotropic hypogonadism (HHG) is frequently associated with this disorder but is thought not to be caused by the low adrenal androgen levels due to adrenal hypoplasia[3,4]. It is uncertain whether there are two distinct yet physically linked genes responsible for AHC and HHG or a single gene responsible for both diseases. AHC can occur as a part of a contiguous deletion syndrome together with Duchenne muscular dystrophy (DMD) and/or glycerol kinase deficiency (GKD). From the analysis of deletions, the following gene order has been deduced: Xpter-AHC-GKD-DMD-cen[5,6]. An**

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