pendent indicators will clarify the stratigraphy below 110 kyr.

More generally, higher-resolution records of both $\delta^{18}O$ of O_2 and methane trapped in ice cores will shed more light on the correlation between Greenland and Antarctica documented in ref. 1. In this context, the existence of rapid changes in the methane concentration[19,20] should be extremely useful, in particular for the late glacial. In addition, changes in the methane concentration, an indicator of large-scale variations in the continental biosphere, and small changes in the Dole effect both appear to be affected by the precessional insolation cycle[3,19]. These indicators may thus provide an independent way to check absolute chronologies and possibly link ice core and continental records. One inherent limitation to the approach is the uncertainty in the difference between the age of the ice and that of the trapped air which may be more than 1,000 years in a low-accumulation site such as Vostok. This uncertainty is lower in higher-accumulation sites such as those of the completed West Antarctic Byrd core and planned drilling sites in both West and East Antarctica.

Thanks to air bubbles trapped in ice cores, we can now hope to know more accurately the leads and lags between Northern and Southern Hemisphere climates and more generally between climate forcings and the various parts of the climate system over the past 100 kyr. This is important not only for understanding past climate changes but also because this period contains information on climate sensitivity and variability which could be the key to our future climate. □

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NEUROPSYCHOLOGY

Faces, fear and the amygdala

John Allman and Leslie Brothers

Two reports — one on page 669 of this issue[1], the other to appear in *Brain*[2] next February — provide rare and remarkable evidence that in humans a brain structure called the amygdala participates in the perception of social signals. Both groups find that damage to the amygdala produces selective deficits in the perception of facial expression. The amygdala is roughly the size and shape of an almond nut, and lies deeply buried in the temporal lobe.

Adolphs and colleagues[1] studied a patient (S.M.) who had suffered destruction of the amygdala as a consequence of Urbach–Wiethe disease. In this condition, the amygdala in both hemispheres is nearly completely destroyed by the deposition of calcium while the hippocampus and neocortex are spared. S.M. is unable to discern fear in facial expression, and is unable to discriminate between fine differences in other facial expressions that are readily perceived by normal subjects. Young *et al.*[2] studied patient D.R., who has partial lesions in the amygdala on both sides as a consequence of treatment for epilepsy. D.R. is very poor at matching photographs depicting facial expressions to either the name of the emotion portrayed or to other photographs illustrating the same emotion; this patient also has great difficulty in telling whether individuals are looking at her or away, which normal subjects do with ease.

These two papers add substantial support to the growing evidence from animals that the amygdala has a central role in social communication. In the 1950s it was discovered that light touch to the skin could be an extremely potent stimulus for driving amygdala neurons in the cat[3]; flank-rubbing is an important means of affiliative signalling in cats. So this finding lends itself to the retrospective interpretation that the investigators had stumbled upon a social function in the cat amygdala. Indeed, subsequent single-neuron recording in the amygdala of freely moving cats showed that "miaowing was the most effective of all sensory stimulation tested"[4].

The idea that there is a specific neural substrate for social cognition in primates was first proposed by Kling and Steklis[5]. From their studies of the effects of brain lesions on social behaviour in monkeys, they proposed that a system including the amygdala, temporal pole and orbital frontal cortex underpins social ties in monkey groups. In monkeys, neurons in the superior temporal sulcus, an area connected to the amygdala, are sensitive to images of faces as well as to the direction of gaze and orientation of the faces[6,7]; and lesions in the superior temporal sulcus produce a selective deficit in the capacity to discriminate the direction of gaze in face images[8]. Neurons of the amygdala are similarly sensitive to the direction of gaze[9].

Mary Evans

Fearful features, from nineteenth-century France.

In humans, it could be that certain conditions stem from defects associated with the amygdala. For example, autistic children are inattentive to facial expression, and it seems that they fail to interpret gaze direction normally[10,11]. It is possible that the interactional problems typical of autism arise from dysfunction in this neural system that converges in the amygdala. It is also possible that abnormal activity of the system could yield the distorted perception of social signals that is typical of the paranoid delusional state.

The direction of one's gaze signals the object of one's attention (that is, what one 'has in mind') while facial expression indicates how one is disposed to behave. When mutual eye contact is established,

both participants know that the communication loop between them has been closed, and for primates of all species this is the most potent of social situations. The discovery that the human amygdala is involved in detecting both gaze direction and facial expression shows that it is indeed part of a brain mechanism for representing the intentions and dispositions of others. But we have a long way to go before we have a complete picture of the human amygdala's responsiveness. Could the amygdala also be essential for the interpretation of social touch and voice intonation — or even the sounds of footsteps? Studies on the monkey amygdala which used a wide range of social stimuli found single neurons that responded to dimensions of the social environment ranging from body movements to interactions taking place between other monkeys[9,12].

From animal experiments there is considerable evidence that the amygdala plays a crucial role in fear conditioning, and indeed the amygdala projects to the structures in the hypothalamus and brain stem that regulate the autonomic expressions of fear such as increased heart rate and sweating[13]. Damasio[14] (one of the authors on the *Nature* study[1]) has proposed that the amygdala and orbital frontal cortex participate in decision-making by correlating somatic states, such as heart rate, with behavioural situations; that is, individuals choose to avoid situations that are associated with a negative outcome marked by 'gut feelings'. It would be extremely interesting to know whether patients with damage to the amygdala are defective in fear conditioning and decision-making abilities. □

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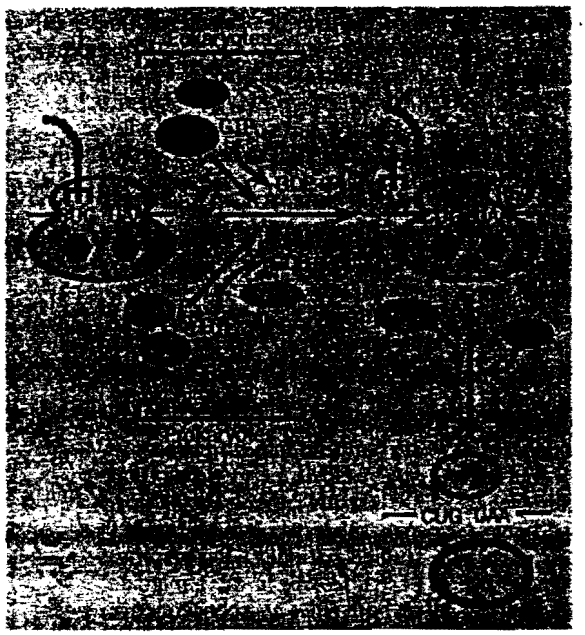
TRANSLATION

Knowing when to stop

Mick F. Tuite and Ian Stansfield

TRIPLET signals built into a messenger RNA's coding sequence tell the ribosome when to stop translating that mRNA. These are the so-called stop codons UAA, UAG and UGA, and in bacteria they signal the termination of mRNA translation by facilitating the binding of polypeptide release factor to the ribosome, thereby stimulating hydrolysis of the final peptidyl–transfer RNA linkage and releasing the completed polypeptide chain from the ribosome (see figure). The same three stop codons are used in eukaryotic mRNAs, but exactly how the codon signals are transmitted to the eukaryotic ribosome has remained something of a mystery.



Polypeptide chain release from the ribosome in prokaryotes and eukaryotes requires one or more release factors (RF) and GTP. Following polypeptide release, the ribosome dissociates from the mRNA with the help of a ribosome release factor (RRF), the identity of which is unknown in eukaryotic cells. Release-factor activity for a given protein is assayed *in vitro* by determining the ability of the protein to release a radioactively labelled peptidyl–tRNA from the ribosomal P site in response to a stop codon occupying the ribosomal A site.

Until now, that is, for on page 701 of this issue[1] Frolova *et al.* report the identification of a family of eukaryotic polypeptides with the biochemical properties expected of a eukaryotic release factor (eRF). The existence of a protein with release factor activity in eukaryotes became apparent following its biochemical demonstration, some 20 years ago, in rabbit reticulocytes[2]. So the subsequent identification of the eRF, and of the gene encoding it, has taken a surprisingly long time.

The assay employed by Frolova *et al.* to purify their eRF, termed eRF1, was originally developed to identify the soluble components of the translation termination machinery of *Escherichia coli*[3]. Three such components have now been found in *E. coli*: RF1 and RF2, which catalyse polypeptide-chain release at UAA/UAG and UAA/UGA codons respectively[3,4], and RF3, which stimulates RF1 and RF2 activity in a GTP-dependent and codon-independent manner[5]. The original studies on the eRF, using the same biochemical assay, showed that a single rabbit eRF will catalyse polypeptide release in response to all three stop codons also in a GTP-dependent manner[2]. The omnipotence of the termination activity of the eRF, together with the almost complete absence of sequence similarity between the newly identified eRF1 family and the bacterial release factors, may indicate a fundamental mechanistic difference between the eukaryotic and bacterial processes.

There has been one false start in the race to identify the eRF by molecular cloning. This was in 1990, with the report[6] of the cloning of a complementary DNA encoding the putative rabbit eRF which showed a significant (more than 90%) amino-acid identity with tryptophanyl-tRNA synthetase (TrpRS), the enzyme used to catalyse the addition of tryptophan onto the mammalian tryptophanyl-tRNA (Trp-tRNA) in an ATP-dependent reaction. But there was a problem — how could this specific, ATP-dependent, tRNA-charging enzyme be required to terminate translation when its usual substrate, Trp-tRNA, can only decode the UGG (Trp) codon? The dilemma was finally resolved last year when it was demonstrated unequivocally that purified mammalian TrpRS does not itself have release-factor activity, but rather co-purifies with a protein that does[7]. That protein, we now know, is the eRF1 polypeptide.

Frolova *et al.* exploited their ability to separate release-factor activity from the TrpRS activity, and used protein sequencing to show that the polypeptide with release-factor activity had significant sequence similarity with a ribosome-