matters arising

Interarc spreading in the Carpathian area

BOCCALETTI and GUAZZONE (ref. 1) have invoked the interarc spreading hypothesis to explain the genesis of the present structure of the Mediterranean area. We consider it necessary to clear up some fundamental points regarding one of the sectors they discussed.

The area inside the Carpathian Bend (including the Transylvania Basin in Rumania) was presented by Boccaletti and Guazzone as an interarc basin, the front and back inarc parts of which would be the Neogene volcanic zones in the East Carpathians and Apuseni Mountains. Because such a hypothesis has so far not been examined and argued but only stated[2-4] we point out here the main geological facts which must be considered.

First, the basement of the Transylvanian Basin comprises both metamorphic and igneous (pre-Cenomanian) rocks, similar to those in the Carpathians[5]. Second, the sedimentary undeformed cover includes Palaeogene and early Miocene deposits continuous over large areas[5]. Third, there have so far been no observations indicating anomalous heat flow in this area.

The first point precludes any interpretation of the Transylvanian area as an interarc basin. If, however, this evidence were not considered, the second point would preclude a late Miocene age for the spreading process. Nevertheless, if the spreading were of that age, the Transylvanian Basin, being younger than the Pannonian Basin, should have a higher or comparable heat flow to that of the latter.

Finally, we emphasise that Karig[6] developed the idea of interarc spreading for volcanic island arcs; his hypothesis cannot, however, be extended indiscriminately to apply to continental areas such as that inside the Carpathian Bend.

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DRS BOCCALETTI AND GUAZZONE REPLY —The structural model comprising a consuming (or contracting) zone, a folded arc, a magmatic arc, and spreading (or distended) zones, after Karig's formulation[1] and after additional ideas by Dickinson[2] and Dewey *et al.*[3,4] must be considered the best model to date for investigating fossil continental margins, both in wide, contracting palaeo-oceans and in small, contracting palaeomarginal basins. Differences between oceanic and continental arcs are probably only a matter of differences in the ages of the contracting processes and differences in the dimensions of contracting areas. Consequently, different stages of maturity probably also occur in areas beyond the arcs.

In that sense all Mediterranean basins, both present and fossils, are very different from those of the western Pacific. Even the Tyrrhenian Basin, which is generally accepted as a typical marginal basin in the Mediterranean area (with active subduction, typical polarity and migration, and typical folded and magmatic arcs) also shows a crustal substratum that is strongly thinned and very hot. It seems to be complicated, however, by many sialic microfragments and sialic seamounts. These sialic 'lenses' are probably actual examples of a segmentation stage in the pre-Tyrrhenian crust. The original floor of the Tyrrhenian Sea, as well as the substrata of the other Mediterranean basins, was never entirely substituted by typical oceanic crust.

The points raised by Radulescu and Sandulescu[7] can be explained as effects of a low level of maturity of evolution in the Carpathian arc–trench system, where the back-arc crust has been distended, thinned and in places fragmented to a certain degree. In fact, Tortonian sediments directly overlie the basement in many places of the Transylvanian Basin[6]. Some spreading may occur under cover, caused by discontinuous subduction; perhaps that can be correlated with rotated arc migration. Incidentally, the age of the oldest deposits contemporaneous with the spreading in the basin must be related to later surface volcanism.

Stegena[7,8] suggest that the inter-Carpathian area should be considered to be "warm". Comparisons between the Pannonian and Transylvanian basins could not give the same result if the directions of the investigation profiles are changed. In fact, it has been clearly demonstrated that the northern Carpathians are inactive whereas the south-eastern Carpathian Arc is still active[9]. Geothermic correlations between different basins should take into account the different thicknesses of their covers. The only conjecture that we can advance about the comparison between the Pannonian and Transylvanian basins is that the second is probably less 'mature' than the first.

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LSD and dopamine receptors

PIERI *et al.*[1] reported a circling behaviour response to LSD in rats with unilateral chemical lesions of the ascending medial forebrain bundle. They used two rotational models; one was produced by 6-hydroxydopamine injection and the other by 5,6-dihydroxytryptamine injection. The factor common to both models was a deficit of dopamine in the ipsilateral forebrain. The authors demonstrated strong maximal contralateral turning responses to LSD given in a dose range of 0.1–1.5 mg kg^{-1}. Only the time course of turning seemed to be dose dependent.

We have repeated these experiments in similar animal models and have found the effects less convincing. Indeed, LSD

produced apomorphine-like (contraversive) circling behaviour in only 40–50% of animals tested, and then at the very high drug levels of 1 and 1.5 mg kg^{-1}. In the turning mouse model of von Voigtlander and Moore[2], where 1 mg kg^{-1} apomorphine induced a mean rate of turning of 5 min^{-1}, no satisfactory data for LSD could be obtained. In the dose range 0.025–0.2 mg kg^{-1}, LSD induced neither turning behaviour or postural asymmetries nor significantly modified apomorphine or amphetamine-induced circling, as may be predicted were it a potent dopamine agonist. Where rotation was induced following administration of 1.5 mg kg^{-1} LSD the duration was of the order of 35–45 min; not 2 h as Pieri *et al.* suggest. We agree with them that rotation was prevented by previous treatment with haloperidol (0.5 mg kg^{-1}), and not after α-methyl-*p*-tyrosine (250 mg kg^{-1}).

In another experimental animal model, namely, audiogenic seizures in inbred strains of mice, dopamine agonists have been shown to diminish the severity of the seizure response[3,4]. In our experiments, apomorphine was at least 10 times more potent than LSD in blocking the clonic phase of the seizure in 50% of animals.

The pharmacology of LSD is very confused, but has previously been associated with central 5-HT neurones[5–7]. Pieri *et al.*, however, have claimed a direct and potent action of LSD on the dopaminergic receptor *in vivo*. Our data do not confirm LSD as being a potent dopamine agonist, although it seems to be capable of stimulating the dopamine receptor in very high doses.

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PIERI ET AL. REPLY—The results reported[1] correspond to a well defined model, namely rats unilaterally lesioned in the medial forebrain bundle with 5,6-HT or 6-OHDA. These two types of lesion have been extensively investigated with biochemical[2,3] and histofluorescence methods (H. P. Lorez, unpublished), and result in a marked and long-lasting depletion of dopamine (DA), possibly leading to the subsequent development of denervation supersensitivity, (1 mg kg^{-1} apomorphine being able to induce more than 15 turns min^{-1} for 30 min). Lesioned animals were challenged with apomorphine and only those responding with a clear circling (about 50–60%) were subsequently used for the study of the effect of other agents, including LSD (80% of responses). The data obtained with several hundreds of rats have been described in part previously[4] and a more extensive paper is in the press[5]. The fact that Pycock and Anlezark[6] seem to have used a different animal species and a different lesion, clearly yielding less denervation supersensitivity (1 mg kg^{-1} apomorphine inducing a mean rate of turning of 5 min^{-1}), precludes any reasonable comparison.

Concerning the relative potencies of apomorphine and LSD as striatal DA receptor agonists, it seems from our data that LSD is slightly more potent than apomorphine. By no means, however, do we claim that this applies to completely different models such as that reported by Pycock and Anlezark[6] (namely, audiogenic seizures in mice).

It should also be stressed that low doses of LSD elicit a significant slowing of DA turnover in rat striatum (0.2 mg kg^{-1} intraperitoneally) and retina (0.5 mg kg^{-1} intraperitoneally). In addition, the striatal adenylate cyclase is activated by concentrations of LSD as low as 10^{-6} M (ref. 7). Thus, there is also biochemical evidence for DA receptor stimulating properties of LSD.

We did not intend to suggest that the DA receptor stimulant effect of LSD observed in our experimental conditions should be the final answer to the confused pharmacology of the compound. The DA-like component of LSD action in the central nervous system is, in our opinion, additive and not alternative to the stimulant action of the compound on 5-HT receptors. In circling behaviour, however, DA receptor stimulation seems to be important, at least in our model[5].

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Mimetic talking by parrots

THE paper by Gregory and Hopkins[1] has prompted me to speculate on the question; What is the biological significance (or survival value) of 'talking' by parrots? Of course, parrots do not actually talk, they only imitate speech. In fact, they imitate, not only speech, but many sorts of discernible sound patterns that are likely to be produced repeatedly in their environment. Indeed, the articulated sounds, I suggest, signify a kind of 'functional' mimicry, whereby an animal is camouflaged by becoming one more source of its natural environmental noise. Yet, the animal does not presumably utter the mimetic sound patterns at random times and spaces, but rather it may utter them as responses to specific outer signals. This behaviour implies the existence of integrated neuronal circuits; and as a dominant role of the visual system is known in birds[2], the pupil constriction observed, suggests that certain (learned) visual patterns may act as signals (stimuli) that trigger talking behaviour. Thus, pupil constriction may serve to obtain clearer, optimal images of such visual patterns. Guzmán-Flores (personal communication) has found that parrots fail to learn recurrent, tape-recorded utterances, in which the correlative significant visual stimuli are missing.

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Hereditary persistence of foetal haemoglobin

ALTHOUGH the interpretation of Martinez and Colombo[1] may be correct, their conclusions are based on uncertain evidence and an alternative explanation that fits within the classical scheme is equally plausible. Their argument rests entirely on the statement that "foetal haemoglobin (HbF) levels show intrafamilial segregation in β^{thal}". From this statement, they conclude that III_2 cannot simply be a β^{thal} heterozygote like I_2. Our experience shows that this is not invariably so: parents, offspring, and siblings who are β^{thal} heterozygotes may differ in level of HbF. Consequently, we believe that III_2 is only a β^{thal} heterozygote and that his slightly elevated HbF is the result of his particular expression of the condition. The inheritance pattern then follows normally. I_1, II_1, and III_1 all have a chromosome with S and hereditary persistence of foetal Hb (HPFH); II_1 also has β^{thal}; III_1 and III_2 have a normal chromosome from II_2.