middle grade gneisses, respectively, and the eclogite-facies rocks themselves have been retrograded to assemblages that are compatible with those in the enclosing rocks. (2) Admittedly, there is no certainty that equilibrium has been preserved between mineral cores. However, mineral cores have relatively uniform compositions within each sample, and different mineral grains within each sample yield consistent core temperatures[6]. (3) I clearly stated that pressure estimates for the country-rock eclogites at Lien, based on the jadeite content of omphacite, are minimum values only. Although many of Kroghs' pressure estimates for eclogites in the Basal Gneiss terrane are minimum values, as he stipulated, others are true estimates, based on the coexistence of orthopyroxene or phengite with garnet and omphacite.

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GABA mediated circling from substantia nigra

THE dialogue between Waddington[1] and Martin and Haubrich[2] emphasizes the complexity of the interaction between striatal dopamine systems and nigral γ-aminobutyric acid (GABA) mechanisms as recently pointed out by Arnt and Scheel-Kruger[3]. This is particularly true when interpreting behavioural changes in terms of biochemical parameters. The effects of manipulating nigral GABA mechanisms on striatal dopamine release are complex. While nigral application of GABAergic drugs, such as muscimol, increases the firing rate of dopaminergic neurones[4], and the release of dopamine in the ipsilateral caudate nucleus[5–7], GABA itself exerts a biphasic action first stimulating and then inhibiting striatal dopamine release[8]. Furthermore, the nigral application of picrotoxin stimulates dopamine release in the striatum, an effect which is prevented or reversed by diazepam[9], which itself decreases dopamine release in striatum[10] (presumably by facilitation of nigral GABA transmission). Some GABAergic terminals (possibly those projecting to zona compacta) therefore seem to inhibit the activity of nigral dopamine cells, while others (possibly those projecting to zona reticulata) may indirectly activate the same nigral dopamine cells, perhaps via a GABA interneurone. Whether turning behaviour induced by the intranigral administration of muscimol is due indirectly to alteration of dopamine release in the striatum is disputed. In fact, the effect of intranigral injection of GABAergic compounds is critically dependent on the exact site of injection, an issue which has been neglected in the correspondence on this topic.

The initial observation of Tarsy and his colleagues[11] that picrotoxin injected into substantia nigra zona compacta caused contraversive rotation was followed by the discovery of Scheel-Kruger *et al.*[12] that ipsiversive circling resulted from the injection of picrotoxin into a caudal nigral site. Subsequently, James and Starr[13] (and, independently, ourselves[14]) demonstrated that the direction of rotation produced by injection of picrotoxin into rostral nigra is reversed on injection of this compound into caudal nigra, suggesting the existence of separate GABA mechanisms. We obtained similar findings injecting picrotoxin, and also muscimol in caudal and rostral nigra.

The circling produced by the action of muscimol or picrotoxin at each nigral site can be distinguished on the basis of its dependence on intact striatal dopamine mechanisms. Thus, while circling induced by injection of GABA-active compounds into rostral nigra is reduced by 6-hydroxydopamine lesions of the nigro-striatal pathway, or by systemically administered haloperidol, the same treatments are ineffective in inhibiting circling induced by manipulation of GABA mechanisms in caudal nigra[1,13,14]. These findings support Waddington's assertions of non-dopamine-mediated contraversive circling being induced by muscimol injected into substantia nigra zona reticulata[1]. The exact site used by Martin and Haubrich[5] is not clear, but must have involved caudal nigra as muscimol induced contraversive rotation. However, the muscimol perfused into substantia nigra also may have reached GABA receptors in rostral nigra to activate the ascending nigro-striatal pathway so causing increased striatal dopamine release. This would account for the simultaneous occurrence of these phenomena.

The argument as to whether or not the striataldopamine release caused by intranigral muscimol is responsible for contraversive circling is redundant. The circling behaviour initiated by an asymmetry of striatal dopamine function is clearly mediated by strio-nigral fibres and the substantia nigra. Thus, lesioning of the descending strio-nigral pathway prevents contraversive circling behaviour induced by the administration of apomorphine to animals with a unilateral 6-hydroxydopamine lesion of the nigro-striatal pathway[15]. In addition, kainic acid-induced destruction of neurones in substantia nigra zona reticulata, or the injection of picrotoxin or bicuculline into nigra ipsilateral to a 6-hydroxydopamine lesion of the nigro-striatal dopamine pathway, results in reduction, abolition or reversal of the contraversive rotation produced by apomorphine[16]. All these data point to a primary role of a neuronal pathway from caudal nigra independent of dopamine, which is responsible for the mediation of the circling phenomena. The importance of this nigral mechanism is emphasized by the work of Papadopoulos and Huston[17] who showed that spontaneous circling behaviour induced by a unilateral electrolytic lesion of substantia nigra was not influenced by the removal of the telencephalon (including striatum and neocortex). Indeed, we have demonstrated that a hemi-transection rostral to nigra severing all rostral nigral afferents and efferents does not attenuate contraversive circling induced by intranigral administration of muscimol (unpublished observations).

In conclusion, we support Waddington in his assertion that the contraversive circling due to intranigral muscimol administration is not due to striatal events. There can be little doubt as to the importance of a non-dopaminergic nigral output pathway in the mediation of circling behaviour. The nature of this pathway remains unknown as does its target site. It does not seem to involve the known projections of the nigra to the superior colliculus[18] or thalamus[19] and we are now investigating the involvement of nigro-reticular pathways.

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