More sober research . . .

To the editor — While agreeing with Barbara Culliton's call for ways to foster more daring or innovative efforts in science (*Nature Medicine* **1**, 601), I hasten to add some words of caution. Not all worthy research is the result of spectacular insights or approaches.

The establishment of lithium as an effective treatment for manic-depressive disease took painstaking efforts to establish the basic facts: outcome, clinical use, dose, etc. The result was the introduction of one of the most important interventions in psychiatric illness and one of the great therapeutic advances in medicine, with vast human and economic benefit.

On the other hand, necessary research to confirm or refute a scientist's or laboratory's experimental findings is a critical function of science. While not daring or innovative, it is vital for the development of sufficient supportive evidence in some cases and an appropriate challenge in others. Much of research involves the incremental development of a knowledge base. When one examines an area of science that is fruitful, one might find many pieces of data that would not be considered bold but nonetheless are vital components of the fabric of research that, collectively, may dramatically move a field forward.

So the public should not expect every scientist to produce breakthroughs. To engender creative research is commendable; to suggest that science is lacking in ideas because most or all of science is not of the breakthrough category establishes a criterion that is unrealistic and at variance with the public need for a broad-based program of research. Research, as many other societal functions, is not of one sort. Its various types serve many publicly useful functions.

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. . . And anatomical research

To the editor — Dr. Van Thompson ventured "there is much to be learned from anatomical research" (Anatomical research lives! *Nature Medicine* **1**, 297–298). However, we were puzzled by an apparent omission. The drawing of the deep occipital region from the posterior view shows the rectus capitis posterior minor extending from the posterior tubercle of the atlas (C1) to the occipital bone. On both sides, attached to the posterior arch of the atlas and extending to the occipital bone, is a structure that is unlabelled, but that we take to be the posterior atlanto-occipital membrane. In the drawing of the hemisected head there is no mention of this membrane, but it may be taken to intervene between the rectus capitis posterior minor and the dura; it is difficult to understand how any connective tissue bridge can be directly connected to these structures.

We do, however, agree wholeheartedly with the overall theme of the piece. Two examples illustrate how conclusions derived from simple anatomical studies can cast doubt on accepted notions of pathogenesis.

In leprosy it is claimed that certain peripheral nerves are affected because they are near the skin, and become thickened as a result of invasion by *Mycobacterium leprae*, which multiplies more profusely at superficial sites where the temperature is lower[1]. Yet one of the most superficial nerves in the body must be the cervical branch of the facial nerve, which supplies the platysma muscle whose fibres are situated in the dermis. This nerve and muscle are, however, unaffected in the disease. Enlargement of the great auricular, transverse cervical and lesser occipital nerves does occur and can be identified from photographs of patients[2]. These nerves, although related anatomically to the cervical branch of the facial differ in being superficial sensory nerves. Again the related and relatively superficial accessory nerve, which has no cutaneous distribution, is not enlarged, and the trapezius is not paralysed in leprosy.

The diplegic form of cerebral palsy involving the legs, is associated with prematurity and is considered to be due to periventricular leukomalacia (PVL) interrupting motor fibres serving the legs. However, PVL lesions invariably involve the collateral trigone, which is a considerable distance from the corticospinal fibres to the lower limb, which are in the posterior limb of the internal capsule[3]. Furthermore, these PVL lesions are situated at the site of the optic radiation, yet cortical blindness in diplegia is uncommon unless there is associated centrum semiovale involvement.

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Thompson replies — I appreciate the attention Crawford and Hobbs have given my comment on anatomical research. I considered work by Drs Hack, Robinson and Koritzer describing a hitherto unknown connective tissue bridge between the spinal dura and the rectus capitis posterior minor (RCPM) muscle. The definitive publication (*Spine*, in the press) describes in much greater detail the anatomy of the deep suboccipital region, including the relationship of the posterior atlanto-occipital (PAO) membrane to the muscle–dural connection. The researchers have observed the PAO membrane to be intimately fused to the underlying spinal dura and refer to this 'membranous unit' as the PAO membrane–spinal dura complex. Moreover, although proximity has been reported between the PAO membrane and the RCPM muscle, a review of the literature evidenced no description of a physical connection between these two structures. The previously undescribed 'connective tissue bridge' attaches the RCPM muscle to the PAO membrane–spinal dura complex. Therefore, the connection between muscle and dura is functionally direct, in that the PAO membrane and spinal dura, at the atlanto-occipital junction, are fundamentally a single structure.

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1. Brand, P.W. Temperature variation and leprosy deformity. *Int. J. Lepr.* **27**, 1–7 (1959).
2. Leprosy. in *Manson's Tropical Diseases*, 19th edn (eds Manson-Bahr, P.E.C. & Bell, D.R.) 757–785 (Bailliere Tindall, London, 1987).
3. Crawford, C.L. & Hobbs, M.J. Anatomy of diplegia: An hypothesis. *Devl Med. Child. Neurol.* **36**, 513–517 (1994).