‘An educated guess’ by Gadgil *et al.*

Every summer monsoon is unusual, each in its own way. The 2019 monsoon seemingly lost track of the calendar and did everything a month late. June rains were well below normal, more like a typical May, but then the monsoon took hold with July a bit above normal, followed by very wet months in August and September and even into October. There was catastrophic flooding at times when the monsoon should be waning or at an end.

In the spring the equatorial Pacific, the most effective single predictor of the monsoon, was in an El Niño, presaging a weak monsoon. It was a weak El Niño, and rainfall was forecast to be only slightly below normal. Towards the end of July, Gadgil *et al.*[1] discussed the progress of the monsoon up to that time and speculated about the rest of the season. They attributed the poor June rains to El Niño, but by July the tropical Pacific was close to a neutral ENSO state, thus providing little guidance for the future. Their speculations relied instead on the state of the atmosphere over the Indian Ocean as measured by the EQUINOO index, the difference between outgoing longwave radiation (OLR) over the western and eastern equatorial Indian Ocean. More convection in the west, a positive EQUINOO, is favourable for the monsoon. A large body of prior work by these authors and their collaborators had established this relationship. EQUINOO in July 2019 could reasonably be taken to explain the better rains in July, but Gadgil *et al.*[1] went much further, making the risky ‘educated guess’ that above-average rainfall would continue through the monsoon season. In this issue of *Current Science*[2] they show that their guess verifies.

This success is evidence for a connection between monsoon and atmospheric circulation in the equatorial Indian Ocean, but like all good research it raises more questions than it answers. Is it evidence that EQUINOO is a cause of monsoon variations, rather than, say, that they are both caused by something else? Are EQUINOO and the monsoon parts of a single mode, or are they truly distinct? Is EQUINOO predictable? If so, then predicting it would be valuable for predicting monsoon rainfall. EQUINOO itself has only weak persistence from month to month, providing limited direct help for prediction. However, Gadgil *et al.*[2] suggest more interesting and potentially useful ideas about the genesis and persistence of favourable (and unfavourable) EQUINOO states. Their hypothesized physical mechanism involves a trigger in cyclones over the Bay of Bengal and persistence arising from ocean–atmosphere interactions over the equatorial Indian Ocean, akin to the Bjerknes feedback in the Pacific that is at the heart of the ENSO phenomenon. These new ideas do us the greatest service, by providing a physical plausible and tractable line of inquiry for further research.

1. Gadgil, Sulochana, Francis, P. A. and Vinayachandran, P. N., *Curr. Sci.*, 2019, **117**(5), 783–793.
2. Gadgil, Sulochana, Francis, P. A. and Vinayachandran, P. N., *Curr. Sci.*, 2019, **117**(11), 1782–1784.

MARK A. CANE

Lamont-Doherty Earth Observatory of Columbia University,
Palisades, NY 10964, USA
e-mail: mcane@ldeo.columbia.edu

Myeloperoxidase-463G/A polymorphism in patients with diabetic nephropathy in Sikkim, India

Diabetic nephropathy (DN) is one of the most common causes of end-stage renal disease (ESRD). Despite major advances made in the area of diagnosis and treatment of DN, death cases related to this ailment are not significantly reduced. Thus it is important to identify the risk factors for this condition.

Hyperglycaemia causes excessive generation of reactive oxygen species (ROS) and oxidative stress, which in turn precedes the development of endothelial cell dysfunction and plays a key part in the pathogenesis of DN[1]. Myeloperoxidase (MPO), a lysosomal enzyme found abundantly in neutrophils, generates ROS. A single-nucleotide polymorphism (SNP), -463G>A at its promoter site leads to loss of SP1 transcription factor binding site[2] and subsequently reduced production of ROS. Thus -463G allele of MPO can be hypothesized to contribute in the development of DN.

Individuals with type-2 diabetes mellitus (DM) visiting Central Referral Hospital, Sikkim Manipal Institute of Medical Sciences, Gangtok, Sikkim, were recruited for the study. DM patients without DN were considered as controls and those with DN were cases. The study was approved by the Institutional Ethics Committee.

DNA was isolated from collected blood samples using QI Amp Blood Mini Kit. Biochemical details were collected from hospital records. Written consents were obtained from all the participants. PCR was carried out with Master Mix (New England Biolab or NEB) using 10 pmol of each forward and reverse primer, 5′-GGTATAGGCACACAATGGTGAG-3′ and 5′-GCAATGGTTCAAGCGATTCTTC-3′ respectively. PCR condition included an initial denaturation at 94°C for 2 min followed by 30 cycles of 30 sec denaturation at 94°C, 30 sec annealing at 58°C, 30 sec extension at 72°C, and final extension at 72°C for 5 min. SNP was detected by digesting 6 µl of PCR product with 5U of *Aci*I restriction enzyme (NEB) at 37°C for 1½ h. Digestion product was visualized in 1.5% agarose gel. Following digestion, three bands at 169, 120 and 61 bp were produced for G/G