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Chaotic mice metabolism

METABOLISM, in simple words, can be defined as the set of chemical reactions – occurring within a cell – that sustain life. It includes both, the biochemical processes that break down food to release energy; and those biochemical processes which use this energy to power various life processes – locomotion, reproduction, breathing – in effect anything and everything that makes life *alive*.

Verily, metabolism can be thought of as the base line of paper cards atop which the pyramidal heap of 'life' rests. And even if but a single card at the base is disturbed, the entire structure collapses.

Metabolic syndrome (MetS), as the name suggests, is a health disorder that disturbs this precarious balance of the 'base cards.' And once the base is disturbed, the tremors are felt across several organ-systems resting on the base. Indeed, MetS is characterized by the co-occurrence of several cardiovascular and lipid abnormalities which 'run' (*syn*) 'together' (*drome*). Not surprisingly, therefore, the onset of MetS leads to CHAOS, another universally accepted acronym for the metabolic syndrome: **C**oronary artery disease, **H**ypertension, **A**dult onset diabetes, **O**besity, and **S**troke.

MetS today has reached epidemic proportions. An alarming 20% of the world's population – 1.4 billion people – are affected by this disease. Furthermore, besides having a deleterious impact on human health, such a prevalence of MetS in a developing country like India, which only spends 5% of its GDP on healthcare, also has several negative socio-economic ramifications as well.

So what causes MetS? Several etiological factors seem to significantly increase the chances of one's falling prey to MetS. Such factors include obesity and disorders of adipose tissue; insulin resistance; and other independent physiological factors, such as abnormally functioning liver molecules. Furthermore, recent studies report that ageing, and hormonal changes also contribute to the escalation of this syndrome.

Considering the aforesaid factors, one realizes why MetS is a particularly difficult disorder to treat or even prevent: There are simply too many variables involved. Although several studies have endeavoured to understand the molecular and genetic changes which result in MetS, most of these studies are based on data extracted only from clinical trials. Intensive studies based on animal models to definitively establish which genes and tissues have a causal role in the development of MetS are largely absent, however.

A Research Communication, **page 1157**, addresses this lacuna in knowledge by using the male Wistar rat as an animal model to probe into the nuances of MetS escalation. In this study, researchers attempt to induce MetS in the rats by feeding them a specially formulated high-fat-simple-carbohydrate diet for five months.

So, do the rats become obese? Is MetS induced? Is the glucose tolerance of the rat impaired? Does the diet lead to...CHAOS?

Let us just say that one would be none the wiser if one continued to indulge in anything too sweet and too fatty.

Breaking DNA backbone

ANCHOR one end of a rope ladder, and twist it from the other end. Twist it again. And again. Twist it as many times as necessary to get exactly four rungs between each helix. From afar, what does this structure look like: The twists, the two helices of the side rope, the tiny knots in the side rope stacked one atop the other from one end to the other.

Epiphany. Why it reminds one of the B-DNA double helix of course! The B-DNA double helix comprises two anti-parallel strands which are bonded together by nitrogenous base pairs. Each strand comprises (like the knots in the side-rope) sugar–phosphate groups stacked one atop the other. Given such a precarious structure of DNA, composed of so many different molecules twisted and turned, how is its structural integrity maintained? What stabilizes the DNA structure?

Three molecular interactions stabilize the structure. First, hydrogen bonds – the rungs of the ladder – between the base pairs of the strands ensure that the strands are joined: Two bonds between AT; three, between GC. Second, stacking interactions between the sugar–phosphate groups, which make the backbone of each strand, 'weld' the sugar–phosphate groups to one another. And third, covalent bonds link the AT/GC base pairs to the sugar–phosphate units, thus reinforcing the structure further.

Several studies have computed the strength of these molecular interactions and have established that the contribution of the stacking interaction of the sugar–phosphate to the overall stability of DNA is comparable to that of the hydrogen bonds. However, there have been only a handful of studies that have gone a step further and tried to find out as to how stable the DNA would be *without* the sugar–phosphate group. In other words, given that the sugar–phosphate groups most assuredly constrain the geometry, and hence the stability of the DNA, how crippling an impediment are they? And if indeed they constrain DNA geometry significantly, what are the differences in DNA stability with the sugar–phosphate backbone, and without it?

A Research Communication, **page 1126**, computationally 'breaks' the DNA backbone, and endeavours to answer each of these questions.

Seeding solar fertile roofs

THE dense and crowded rooftops of Indian cities are 'solar fertile.' Solar panels can be mounted atop the roofs of medium and high rise buildings, and connected to one another, thus creating an extensive solar grid to meet the anthropogenic demands for electricity. Indeed, a General Article, **page 1080**, reveals that solar panel grids installed atop medium and high rise building clusters generate enough electricity not only for the building dwellers, but also a significant surplus.

This technology, known as the roof-top photovoltaic module (RTPV), has been around for many years and is being used extensively world over. The European Photovoltaic Industry Association, for instance, estimates that 40% of the EU's demand would be met by RTPV by 2020.

Given such positives of RTPV, the question begs to be asked: India, being a tropical country, is endowed with a preponderance of solar radiation, then why isn't RTPV being adopted on a large scale? Yes, installation of an RTPV costs upwards a lakh, but annual savings works out to be around ten thousand rupees. Further, in some states, Tamil Nadu and Gujarat for example, there are government schemes which subsidize the RTPV costs and also pay the owner for producing electricity; yet why aren't our roofs crackling with electricity? Isn't it high time we seeded them with RTPV?

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