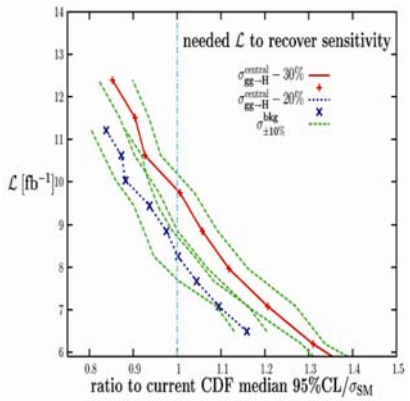
# In this issue

## Higgs boson at LHC

Hunting for the Higgs boson has begun at the Large Hadron Collider now in all earnest. While the particle physicists had been quite at home with hunting for truth about the laws of physics on the 'femto' scale using their complex detectors and the gigantic accelerators, it is only with the Large Hadron Collider that the rest of the world became aware, with a bang, of the scale of things in this particle adventure. The moment of reckoning for the particle theorists and their beloved Standard Model (SM) is arriving ever closer with the wonderful performance of the biggest man-made accelerator: the LHC. In this issue, Rohini Godbole (**page 1155**) reports the current status of physics searches for the ever elusive Higgs boson as well as for the footprints of physics beyond the Standard Model (BSM) of particle physics.

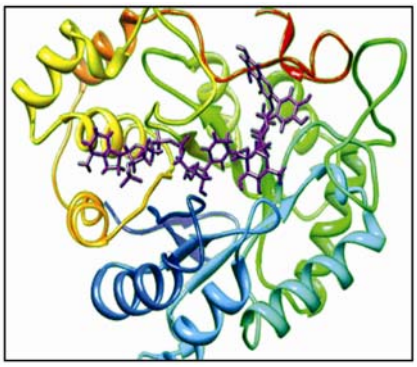
In fact, before the LHC came on line the last word about the 'direct' searches for the Higgs bosons was said by the experiments at the proton–antiproton collider: the Tevatron, which had claimed an exclusion of a Higgs in the mass range around 165 GeV/$c^2$. It was pointed out that due to the rather small number of possible Higgs bosons expected to be produced at the Tevatron, one would need to study about twice the number of collisions, for this conclusion to be unambiguous. This is indicated by the figure shown below. Since the Tevatron was winding down its operations, there was no possibility of getting then an unambiguous answer to the question from that machine. The much higher energy of the proton beams at the LHC collider increases the Higgs production rate in this mass range substantially. Hence with the data collected the LHC experiments confirmed the Tevatron exclusion to a much higher level of significance. This is but one example of the harvest that the physicists have begun reaping at the LHC, made possible due to the excellent performance of the machine, the detectors as well as theoretical/analytical tools set in place, in a collaborative exercise by experimentalists and theorists together.




Additional luminosity required for the Tevatron exclusion to be unambiguous (Baglio, J. *et al.*, 2011).

## ALR2 inhibition by dietary molecules

Currently there are approximately 200 million diabetic people all over the world and this number is expected to double by 2030. Long-term secondary complications are the main cause of morbidity and mortality in diabetic patients. Aldose reductase (ALR2) catalysed accumulation of osmotically active sorbitol has been implicated in the development of diabetic complications, like cataract, retinopathy, neuropathy and nephropathy. Although a number of ALR2 inhibitors (ARI), both synthetic and natural, have been tested for diabetic complications, most of them have met with limited success in clinical trials because of efficacy or undesirable side effects. Thus, there is a need for developing and evaluating new and more efficacious ARI with no or minimum safety concerns. Reddy *et al.* (**page 1191**) describe the inhibition of ALR2 by rutin, a flavonol glycoside of quercetin, present in many dietary sources such as fruits, vegetables and black tea. In this study they have shown inhibition of human recombinant ALR2 by rutin and provided insights into the nature of inhibition. Inhibition appeared to be relatively specific to ALR2 over other closely related members of the aldo–keto reductase superfamily to which ALR2 belongs. Molecular docking studies were also conducted to substantiate the binding pattern and selective inhibition of ALR2 by rutin. The data indicate that rutin seems to interact with ALR2 at active site residues Val-47, Gln-49, Trp-111, Leu-300, Leu-301, Val-297, Ala-299 and exhibit hydrophobic interactions with Trp-219 and Phe-122. It appears that rutin might bind to ALR2 in an open type of conformation because of the formation of a hydrogen bond with Leu-300. Compared to the well-known synthetic inhibitors which



occupy active site with limited contacts, rutin interactions are extended into the hydrophobic cleft called specificity pocket, suggesting effective inhibition of ALR2. Further, the study demonstrates the ability of rutin to suppress intracellular sorbitol accumulation in red blood cells under high glucose conditions, which is suggestive of translating its impact to *in vivo* conditions. Although, there have been major advances in the control of diabetes through dietary changes, hypoglycemic agents, insulin and islet transplantation, the management of long-term complications of diabetes, such as blindness, renal and neurological problems, remain serious problems to be dealt with. The results of the present study indicate the ARI potential of a dietary molecule rich in many food sources and a scope for controlling secondary complications of diabetes by rutin as a pharmacological agent or through food sources that are rich in EA.