1300(m), 1200(s)] and 4-methyl-7-isopropyl- [IIId, m p. 189–90°, UV λ_{max}^{EtOH} (log ε) 282(4.23)] benzofuran-2-carboxylic acids.

These acids were decarboxylated to 5,6-dimethyl-benzofuran [IVb, b p. 220–22°, picrate, m p. 63–64°], 4,7-dimethyl benzofuran[7] [IVc, b p. 217–18°, picrate m p. 102–03° UV λ_{max}^{EtOH} (log ε), 253(4.23), 300 (4.24)], 5,7-dimethylbenzofuran[7] [IVa, b p. 222–24°, picrate, m p. 78–79] and 4-methyl-7-isopropyl benzofuran[7] [IVd, b p. 235–37°, picrate m p. 97–98°].

Elemental analyses of all the compounds agree with the expected structural formulae.

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TERREIC ACID—A DIABETOGENIC MYCOTOXIN IN RATS

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ASPERGILLUS TERREUS, a common food contaminant, grows on foods and feeds when storage conditions are poor, and produces a number of secondary metabolites like terreic acid, terrein and terremutin[1]. Among these, terreic acid has been reported to be toxic to man and laboratory animals[2].

Eventhough many mycotoxins appear to disturb the carbohydrate metabolism, the effect of mycotoxins on the serum insulin levels which is a major factor controlling the blood glucose level has not been reported. Hence the effect of terreic acid ingestion on blood glucose, liver glycogen and serum insulin levels in rats has been reported here.

A. terreus was grown on Czapek-Dox liquid culture medium for 15 to 20 days, terreic acid extracted from the culture filtrate by the method of Kaplan *et al*[2] and purified using preparative thin layer chromatography. It was crystallised using absolute alcohol and the purity tested by comparing with an authentic sample. Bread, 400 g, containing calcium propionate as preservative was mixed with water to have 30 % moisture, sterilized, cooled, inoculated with spores of *A. terreus* (10^6 spores/ml) and allowed to get incubated at 27–28°C for 20 days. After this, the contaminated material was sterilised free of the fungus using chloroform and dried well to remove any traces of chloroform. This material was mixed with the normal diet in the ratio of 1:3 and fed as the contaminated diet.

Weanling albino rats of Wistar strain weighing about 35–40 g were divided into three groups. The first group received normal diet and served as control while the second group of animals was injected intraperitoneally with the pure terreic acid at a dosage of 0.2 mg/rat, dissolved in 0.5 ml of sterile water, and the third group of rats was fed with the contaminated diet containing terreic acid in a concentration such that each rat consumed 50 μg of terreic acid daily. Toxicity studies were carried out on rats for a period of 24 weeks, which is a long term experiment on a mycotoxin with a high LD_{50} value. At the end of the experimental period, the rats were starved overnight

Table 1 *Fasting blood glucose level and liver glycogen level in control and experimental rats. The values are expressed as mean ± S.D. for six animals in each group.*

Experimental animals	Tests carried out: Fasting blood glucose level mg/dl of blood	Tests carried out: Liver glycogen mg/gm of wet tissue
Control	68.8 ± 5.1	30.5 ± 2.1
Toxin injected	91.9 ± 3.9[a]	18.8 ± 1.2[a]
Contaminated diet fed	78.5 ± 4.4[b]	25.1 ± 1.8a

[a] $p < 0.001$; [b] $p < 0.01$

Table 2 *Blood glucose levels during the performance of glucose tolerance test in control and experimental rats. The values are given as mean ± S.D for six animals in each group.*

Experimental animals	Blood sugar levels in mg/dl of blood				
	Fasting level	30′	60′	120′	180′
Control	68.8 ± 5.1	140.6 ± 8.3	101.8 ± 5.7	75.1 ± 6.6	66.5 ± 6.8
Toxin injected	91.9 ± 3.9[a]	193.3 ± 7.0[a]	145.2 ± 9.4[a]	105.9 ± 6.4[a]	96.5 ± 5.4[a]
Contaminated diet fed	78.5 ± 4.4[b]	164.0 ± 9.2[a]	112.8 ± 8.4[c]	93.5 ± 9.8[b]	88.1 ± 5.6[a]

[a] $p < 0.001$; [b] $p < 0.01$; [c] $p < 0.05$

Table 3 *Serum insulin levels in control and in experimental rats during the glucose tolerance test. The values are given as mean ± S.D for six animals in each group.*

Experimental animals	Serum insulin level μIU/ml				
	Fasting level	30′	60′	120′	180′
Control	17.6 ± 1.0	24.3 ± 4.7	31.1 ± 5.8	20.8 ± 4.0	18.4 ± 2.9
Toxin injected	9.9 ± 3.0[a]	14.3 ± 1.1[a]	18.9 ± 3.4[a]	10.3 ± 1.0[a]	9.9 ± 0.7[a]
Contaminated diet fed	13.3 ± 1.5[a]	17.3 ± 1.2[a]	24.9 ± 1.9[b]	14.6 ± 1.4[a]	11.1 ± 1.2[a]

[a] $p < 0.001$; [b] $p < 0.02$

and fasting blood glucose level was estimated by the modified method of Sasaki *et al*[3]. The liver glycogen level was estimated by the method of Morales *et al*[4].

The test animals suffered hyperglycemia accompanied with a drastic decrease in liver glycogen (table 1). Glucose tolerance test was carried out by the method used by Shanmugasundaram *et al*[5]. After overnight fasting, blood samples of the animals were collected. The glucose load administered was 3.5 g/kg body weight as a 10% solution. Blood samples were collected from tail vein at 30, 60, 120 and 180 minutes after glucose ingestion and divided into two sets. One set of the blood was collected using sodium fluoride as anticoagulant and this was used for glucose estimation. In the other sample, the serum was separated and the insulin assayed by the method of Herbert *et al*[6], using the principle of radioimmuno assay. For the assay, ^{125}I-insulin, antisera, standard insulin and other reagents were obtained from Bhabha Atomic Research Centre, Bombay.

The results given in table 1 reveal that during *A. terreus* toxicosis there is a drastic decrease in liver glycogen accompanied by an increase in blood glucose. Suzuki *et al*[7] have reported a similar observation of depletion of liver glycogen with an increase in blood glucose in rats during ochratoxin administration.

In normal rats, the maximum blood glucose level is reached within 30 min of glucose load (table 2) and the fasting level is reached at the end of 180 min. However, in the experimental rats, the glucose tolerance test results are characteristic of the maturity onset diabetes.

In the case of serum insulin levels, the experimental animals show a highly significant decrease, at fasting levels of glucose and even after the glucose administration (table 3) which indicates that the toxin might have lessened the insulin secretion by affecting the β-cells of pancreas.

It can be concluded from our studies that mycotoxins with high LD_{50} value should not be discarded as not so deleterious. Foods and feeds containing such toxins, if fed over a long period can affect β-cells of pancreas as in 'maturity onset diabetes'.

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OCCURRENCE OF A NEW BASE METAL MINERALISED ZONE IN THE GNEISSOSE ROCKS OF THE ASKOTE CRYSTALLINES, PITHORAGARH DISTRICT, KUMAUN HIMALAYA

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THE Precambrian rocks of Askote Crystallines constitute a detached northern outlier of the overturned Almora-Dudhatoli thrust sheet of the Kumaun Lesser Himalaya[1]. The Crystallines comprise a doubly plunging synform, thrust over the sedimentary terrain of the younger Garhwal Group of Lesser Himalaya. The area under investigation is located on the slightly overturned northern limb of the synform, near its south-eastern closure. The axis of the synform trends WNW-ESE. The rock types include coarse to very coarse grained biotite bearing augen- and porphyroblastic gneisses underlain by chlorite-sericite-muscovite-biotite-quartz schists. The gneisses occupy the core of the synform (figure 1).

Megascopically, the gneisses are leucocratic, compact, and coarse grained, exhibiting surreitic (dilation) and ophthalmitic (augen) structures. Parallel arrangement of muscovite and biotite define the gneissosity. Microscopic study reveals that the gneisses consist of subangular to angular, elongated, interlocking grains of quartz with muscovite, biotite, and large elongated porphyroblasts of potash felspars and sodic plagioclase. Felspar poikiloblasts with inclusions of quartz, biotite, and muscovite, are also not uncommon. The felspars almost always exhibit perthitic intergrowths, vein and patch perthite being the most common. Myrmekitic texture is noticed at the contact of plagioclase and K-felspars, and within plagioclase porphyroblasts. Mineralogical and microtextural studies of the rocks indicate that they have been subjected to the katazonal grade of metamorphism.

Yellow limonitic stains, indicative of a mineralised zone trending NNW-SSE, are traceable over a strike length of about 700 m along the Gurji Gad near Askote. The width of surface indications rarely exceeds 20 m. In places the mineralised zone is exposed on the surface. Nowhere else in the Himalaya has the sulphide mineralisation been reported in high grade metamorphics. Mineralogical and textural studies indicate that the sulphide minerals in the gneisses along the Gurji Gad occur as epigenetic replacements and are emplaced by the agency of hydrothermal fluids invading the country rocks. Preliminary examination reveals the presence of copper-lead-zinc minerals. The main sulphide minerals, in the order of decreasing abundance are galena, chalcopyrite, pyrite, and sphalerite. The mineralising solutions invaded the country rocks along foliation planes and joints, and replaced the rock minerals along grain boundaries, cleavages, and fractures. Large porphyroblasts of felspars have been replaced by the sulphides along fractures normal to their axes of elongation. In most cases, the replacement is readily identified because much of the original phase still remains in the form of 'islands' in the replacing sulphides.

The occurrence of copper-lead-zinc ores in sheared sericite-chlorite-muscovite-biotite-quartz schists at Barigaon, about 500 m up the slope of the Gurji valley, has been known for quite some time. The nature of the base metal mineralisation at Barigaon, its mineral assemblage, paragenesis, and associated alteration has already been described[2–6]. Detailed studies of the wall-rock alteration, gangue mineral assemblages, and textural studies of sulphides of the Barigaon mineralised zone, indicate that the mineralisation is of hypothermal grade. The discovery of a mineralised zone along the Gurji Gad attains significance by virtue of its nearness to the known occurrence of the sulphide lode at Barigaon. Sulphide and gangue mineral assemblages of the Barigaon and Gurji mineralisation are essentially identical. The emplacement of both orebodies is along the foliation, and the sulphide zones are elongated in the direction of the major fold axes, roughly trending NW-SE, suggesting thereby an identical structural control.

Similarity between the Gurji and Barigaon mineralised zones may be considered to be indicative of a common parentage. Under these circumstances, pros-