most species are well accepted by livestock, but ingestion of large amounts of foliage can lead to animal mortality. The toxic principles have not been specifically determined, but proteolytic enzymes, coumarins, furo-coumarins and alkaloids are all known to occur in the genus. Three sheep with clinical signs of inappetence and diarrhea were referred to Shiraz Veterinary Teaching Hospital. The owner claimed about the inappetence and depression in other sheep in the herd. Blood and rumen samples were taken and submitted to laboratory. Inspection of the environment and history taking revealed that Ficus leaves were harvested and fed to them. Blood samples were analyzed for CBC and no abnormal findings were observed, except increase in PCV (42%). The collected fresh rumen fluid sample showed a predominance of inactive protozoa. This finding was indicative of adverse effect of Ficus leaves on rumen microflora. The toxicated animals were treated by balanced electrolytes plus 5% dextrose, the rumen tonics and transfaunation. The treated animals improved within 12 h. To the best of authors' knowledge, although there are reports of outbreaks of neurotoxicoses due to Ficus leaves in other *Ficus* spp., this is the first report on a digestive disorder caused by ingestion of Ficus leaves in a sheep herd.

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P1103
Onion (*Allium cepa*) toxicosis in a goat herd

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Ingestion of onion (*Allium cepa*) is known to be toxic to many animals. The present study describes onion poisoning in a goat herd which was admitted to the Shiraz Veterinary School Hospital (Shiraz University). The cases occurred in the first week of October 2009, in a goat herd in Shiraz suburbs with 85 head goats. During the previous four weeks, the herd had received a ration of onion. The first animal affected voided dark brown urine. On day 2, animal 1 was dead, animal 2 was recumbent, and two others were reluctant to rise. On day 3, animal 2 was dead and animal 3 was recumbent. By day 4, animal 3 was dead and a fourth animal was recumbent. The urine color from the remaining animals was changed. The blood samples from the fourth animal plus two other animals and the carcass of animal 3 submitted for necropsy. The carcass had a distinct onion-like odor. The kidneys were dark brown and petechial hemorrhages were present on the cortices. The urinary bladder contained dark red urine. The blood samples were shown large numbers of Heinz bodies and anemia. Onions contain hemolytic agents. The remainder of the affected animals in the group were removed from the onion and given supportive treatment and placed on a ration of corn hay and oats. They showed a marked improvement during the next week. It must be emphasized that, in the seasonal abundance of onions and its cheapness, it should draw attention to this syndrome.

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P1104
Young children with austim spectrum disorders: Can aluminium bodyburden cause metabolism disruption?

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Autism, an abnormality involving language skills, social interaction deficits and stereotypic/repetitive behavior, which triggering or genetic factors are controversial, elusive and remain unknown. Improvement or recovery are also scattered or poorly documented. An ethical pilot study was conducted in 12 children of both sexes, age between 3 and 12 y.o. after evaluation by a multiprofessional team in order to ascertain their level of disability according to a PDD behavior inventory followed by a complete clinical and laboratorial investigation. Simultaneously, the bodyburden of metals was assayed either by AAS or ICP measurements in hair, blood and urine. As many patients had nutritional and immunoallergic impairments, their parents were advised to adjust their food intake, to correct and reduce metabolic and the hypersensitivity gaps. First results showed that most of the patients were naive of treatment, but all ($n = 12$) of them beared high levels of aluminium in the body (in hair, blood and/or urine). Neither copper, lead nor mercury had even elevated rates whereas many of them demonstrated several discrepancies in their lipidic profile, uric acid and high degree of food allergies. The correlation between the severity of signs and symptoms and the behavioral pattern found in many patients seems to be compatible with metabolism disturbances provoked by aluminium overload in healthy children and those chronically exposure to the agent. Psychophysiological dissimilarities were daily described to score behavioral changes. Parents were then advised to avoid cooking with any aluminium pottery and other related food, drinkable and disposable vials to evict Al overload.

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P1105
The cutaneous lesions of dioxin exposure: Analysis of a case of acute TCDD intoxication

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The molecular pathology and molecular toxicology of the so-called "chloracne" syndrome, the most specific sign of dioxin poisoning in humans, has not been updated in the past decade due to the lack of appropriate models. We followed for 5 years a man who had been exposed to an acute dose of TCDD [Lancet 374 (2009) 1179–1185]. A molecular medicine approach of skin and skin lesion analysis, including immunohistochemistry, whole-genome microarrays and TCDD analysis by GC/HRMS, aimed at identifying appropriate therapy, was followed. The skin lesions were found to be functional hamartomatous neoformations that concentrate the poison into the skin, and developed parallel to the loss of sebaceous glands. They accumulated TCDD up to 12 months after exposure. Immunohistochemical analyses showed a focal expression of CYP1A1 in the epithelial walls of the hamartomas; we have also found this very special distribution in other human cases of "chloracne". CYP1A1 was the most induced gene