Case report:
This report deals with a new congenital and possibly inherited defect observed in twelve Romagnola calves submitted because of**facial deformities** and enlarged abdomen.
All calves were already dead upon admission: ten were stillborn and two lived only a few hours.
We have decided to call this defect “**Congenital Paunch Calf Syndrome**” because of the main pathological findings (abdominal enlargement) and because this is the name farmers use to describe the affected animals.

Necropsy findings
*Marked subcutaneous oedema*, especially in ventral part of the abdominal wall (all but one calf).
*Ascites fluid in the abdominal cavity* (in some cases up to 10 liters). The liquid ranged from yellow to red, with different grades of turbidity.
*Hepatic cysts on the peritoneal surface of the left hepatic lobe and/or of the hepatic hilus with serous or reddish fluid content* (seven calves).
*Moderate to severe diffuse hepatic fibrosis*. The liver was moderately tough and enlarged with irregular and enhanced lobular pattern. Upon surface section, the parenchima was irregularly separated by slight fibrous bands.
*Cardiac malformations* (all but two calves): characterized by atrial, interventricular septal defect or patent ductus arteriosus.

“Clinical” findings
- **Enlarged and floating abdomen**, denoting a considerable abdominal effusion.
- **Facial deformities**: shortened and flattened face and in some cases enlarged head.
- **Cleft palate** in four cases.
- **Disproportionate shortness of the limbs** in 1 case.

Histopathological findings (*)
*Hepatic fibrosis*. The lobular architecture of the liver appeared altered with widespread fibrosis in periportal areas and around centrolobular veins.
In some lobules, the fibrosis was extended to perisinusoidal spaces. In some cases, capsular fibrotic thickening, telangiectasia (only in one case), cellular degeneration and atrophy were noted. The capsular thickening was also related to chronic localized peritonitis (four cases). Fibrosis was more evident in Masson trichrome and Gomori stained sections.
*No copper-induced fibrogenesis* was detected with the rhodanine method.

(*) Microscopical examination was performed on 5µ sections stained with Haematoxylin and Eosin (HE), Masson trichrome stain, Gomori and rhodanine techniques.

Discussion and conclusion
Congenital fibrosis of liver is a relatively rare disease of infants and young humans adults caused by an autosomic recessive gene. At this moment we suppose a genetic cause also for the “**Congenital paunch calf syndrome**”. Therefore we are looking for some possibly related familial lines.
We have not found any relationship with other potential etiological factors (e.g., infective, toxic, nutritional, carential, …). Further studies supported by immunohistologic and ultrastructural investigations are needed.