Umbilical hernias occur due to weakened supportive muscles around the umbilical stump or navel area of the pig. This causes the umbilical opening not to close properly and intestines protrude through the intestinal wall to form the “ball-like” structure often seen on the pig. The frequency ranges from 0.4 to 1.2% (Searcy et al., 1994). Hernias are classified as direct or indirect depending on whether intestinal loops outside the abdomen are covered by peritoneum or vaginal tunic (indirect), or whether intestines directly contact skin (direct) (Grindflek et al., 2006). Intestines in direct contact with skin stimulate formation of adhesions. Umbilical hernias, of any size, are usually direct and therefore complicated by adhesions that can interfere with normal digestion. A moderate amount of adhesion should only somewhat reduce the pigs’ performance and their carcasses should be of similar value to pigs that do not have this condition. However, problems arise if the intestines are ruptured during the slaughter process and the intestinal contents contaminate the carcass. Often these pigs are sent to specialty harvest facilities that can accommodate them and slaughter them with minimal risk of carcass condemnation. This re-sorting in the market chain causes the reduction in value.

The genetic control of umbilical hernias is not entirely clear. A “familial” cause has been suggested and a few specific genes have been recently shown to associate with this condition (Zhao et al., 2008). However, in general this condition is not due to simple inheritance of a few genes. Environmental conditions definitely play a role in the incidence of this defect. It is thought that environmental compromises such as navel infections early in life may be linked to the incidence of this condition. Proper sanitation and hygiene may have a greater chance of reducing the incidence of this condition than trying to eliminate certain boars or dams.

If there is a genetic influence to this condition, it may be related to poor environmental conditions. This indicates there may be genetic variability controlling the musculature of the navel and those with a propensity with weaker navel muscles in a poor environment could trigger this belly rupture condition.

Environmental factors such as abnormal stretching of the umbilical cord (during farrowing or placing naval clips too close to the skin) or infection of the umbilical stump could contribute to failure of the umbilical cord opening to close. Investigation of a hereditary component is complicated by the nature of the defect. While there may be a range in the ability of the umbilical stump to close, the characteristic is not easily measured except in the extreme (categorical measure – affected vs not affected).