A compelling article about the Liver gene, which I thought was of interest when considering the introduction of 'non-recognised colours' (NRC) into the Boston Terrier gene pool. *(Fiona King, Secretary, SBTC)*

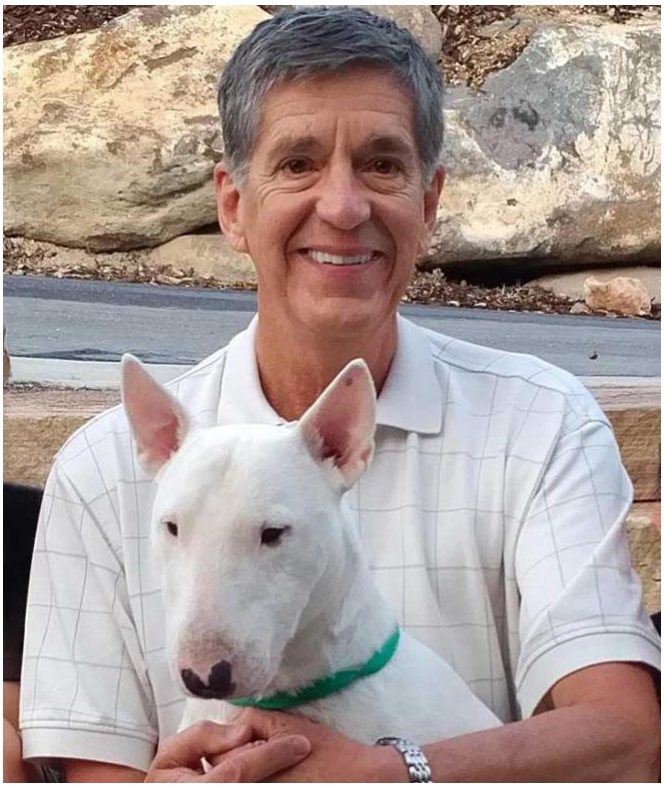
LIVER COLOR IN BULL TERRIERS

Written and reproduced by Carl Pew DVM MRCVS with his kind permission.

Carl's bio: https://southmtnpet.com/veterinarians/Carl-Pew-DVM,-MRCVS

The appearance of a liver tri-color male Bull Terrier advertised at stud on the internet has caused the BT fancy to question the value or risks associated with using this color pattern in a breeding scheme.

In the 40 years I have been breeding, liver color in Bull Terriers has popped up occasionally in reports, discussions, and in personal observations. I have personally seen one liver brindle bitch, one all white bitch, and now in this most recent liver tri-color dog.



All Bull Terriers carry the B locus gene which is a black overlay gene from production of black pigment (melanin). Review of dog coat color inheritance publications, especially the most recent, state that all Bull Terriers are double dominant BB because all Bu ll Terriers have black nose and pads pigmentation. We know of course that some Bull Terriers must be carriers of a recessive b gene since we have knowledge of liver colored Bull Terriers. Therefore, when the B locus gene is present in either BB or Bb the dog will have a black coat overlay with black nose and pads (melanin) but when present in double recessive (bb) the body produces a different type of melanin called Phaecomelanin which is red-brown (liver) in all the areas of normal melanin distribution. So, in this instance (bb), the dog becomes liver coated with light yellowish eyes, a liver nose and liver pads in areas where it should have been black melanin colored.

The medical studies of black pigment mutation centers around the change of melanin pigmentation to Phaecomelanin pigmentation. Melanin pigment is present in many locations throughout the body in humans and dogs. These include the hearing apparatus, eyes, brain, skin and more. Melanin serves as an important antioxidant in these locations to protect against harmful oxygen free radicals which cause a myriad of destructive changes to cellular functions. Phaecomelanin is a poor antioxidant and does not function similarly to melanin in any of these organ systems.

There is documented proof that loss of melanin in the hearing apparatus of the ear (Cochlea) increases the rate of hearing loss from both loud noise exposure and aging loss in humans. Canine hearing research has linked the loss of melanin containing cells in the hearing apparatus directly to congenital deafness in the dog. Other research has shown a protective function for melanin in the light receptor layer of the eye (retina) as well.

The recessive b gene must occur with some frequency in the breed since it persistently but infrequently pops up. Whether this gene expression occurs with higher frequency depends on reporting from breeders, have they seen this?, how often?, what did they do? (cull, place in pet home, etc?) I think that with the current breeding population so heavily slanted toward black tri-color, there is a significant risk for many more liver colored animals to be produced. Thus, we may be leading the breed towards potentially more phaecomelanin complications spread widely through our breeding population. Certainly, if the currently offered stud dog is used, it will introduce the recessive b gene rapidly into our dogs as every offspring will carry at least one copy of the recessive b gene and serve as carriers. Our early breed mentors who wrote the breed standard may not have been familiar with the science, but they certainly understood the importance of black (melanin) pigmentation.

A prominent breeder once said at a BTCA meeting about deafness “We all know deafness is bad, but if you want it, breed to it”. The same applies to liver color in Bull Terriers, in my opinion, it’s bad, but “If you want it….”

END