

Heartworm “vaccines” are not vaccines!

These words came out of my keyboard a couple of days ago. It was one of the same old discussions in which vets end up being blamed for everything that goes wrong. Along the years, I have learnt to ignore them, but sometimes I cannot ignore the sacrifices I had, and I have, to face in order to graduate in veterinary medicine. Summarizing, the story was about an Australian Shepherd, younger than a year old, who died after being given the annual heartworm preventive ([moxidectin](#), commercial name Proheart 6). To be honest, it is still not clear whether the dog died because of this drug, or by accidentally eating some poisonous plants in the garden. But, according to people, he died because of an ignorant vet. A mass revolt with more than 200, very confused, comments, exploded.

People refuse to believe that avermectins (ivermectin, moxidectin, milbemycin selamectin...) used for heartworm prevention, hence at extremely low dosages, are perfectly safe for dogs who are [MDR1- Multi Drugs Resistance Gene](#) (affected). The dosage is too low to intoxicate them: it would be a whole different story if they were given the dosage to kill demodectic or sarcoptic mites. If you do not believe me, instead of listening to “your cousin”, read the scientific paper [“Toxicology of Avermectins and Milbemycins \(Macrocyclic Lactones\) and the Role of P-Glycoprotein in Dogs and Cats”](#). Furthermore, they are all the same: it is plain nonsense to give moxidectin, because [ivermectin](#) is toxic to MDR1 dogs... These molecules belong to the same class. [I am not listing here the products commercial names, as they tend to be changed in different countries, just check your tablets box for the active component].

veterinarians, prefer the long lasting formula, because it is more "convenient".

I personally do not like it, I do not really like the idea of giving to an animal anything that is going to remain in his body for months. Why? It is very simple:

- I do not know how long it will actually last;
- I do not know how and at which speed it will be metabolized;
- I am afraid of adverse effects. Albeit deemed safe, some dogs can experience side effects and, in this case, I will not be able to contrast them, there are no antidotes and these side effects could last for months...

So, what happened with the Australian Shepherd? First of all, as far as I know, he had never been tested for the MDR1 gene so we do not know if he really had a multi drugs resistance. Second, he was given Pro-heart 6, the long lasting moxidectin. I said above that moxidectin tablets are safe for MDR1 dogs. Is it the same for the injection? It should be safe but, for reason number 2 and 3 I would not recommend this product in a breed known for MDR1. Washington State University, on its website, gives this same advice. And neither I would recommend it for a pup/growing dog as you might need to give him a dose for "adult weight" and because younger dogs can be more sensitive to some drugs. When in doubt, err on the safe side!

I hope this can clarify some of the doubts, but please do not go around stating that "vets are ignorant goats" while, at the same time, trying to look smart by defining "vaccine" a [macrocylic lactone](#).

PRA & NCL-D nel setter inglese // PRA & NCL-D in the English Setter

For English Scroll Down

La sigla PRA (rcd 4) sta per Atrofia Progressiva della Retina mentre la sigla NCL-D è acronimo di Lipofuscinosi Neuronale Ceroide. Cosa sono?

Si tratta di due malattie genetiche presenti in diverse razze canine, tra esse il setter inglese. Personalmente, sono a conoscenza della NCL-D da almeno 20 anni ma solo pochi anni fa avevo appreso che fosse stata individuata la mutazione. Mi risultava altresì che l'unico laboratorio che effettuava i test fosse in Repubblica Ceca.

Per quanto riguarda la [PRA](#), invece, la disponibilità del test per il setter inglese e per il setter gordon è relativamente recente ma da diversi anni la malattia è conosciuta e testata nel setter irlandese, ne avevo parlato anche nel mio libro sui setter.

Cosa comportano queste malattie nello specifico? L'atrofia progressiva della retina causa cecità nei soggetti affetti. Il test a disposizione indaga su una delle forme di PRA presenti nel setter inglese. E' possibile, purtroppo, che ce ne siano anche altre. Non esistono terapie per la PRA. La patologia è caratterizzata da insorgenza tardiva, si sviluppa cioè in soggetti adulti che potrebbero già essersi riprodotti.

Secondo il laboratorio Antagene, la mutazione responsabile della patologia è presente nel 7% della popolazione dei setter francesi (moltissimi dei quali, mi preme ricordarlo, hanno antenati italiani). Sono stati altresì riscontrati casi di PRA (rcd4) in setter inglesi norvegesi, di sangue italiano e non.

Sulla [lipofuscinosi](#) non ho dati numerici da trasmettere ma mi preme sottolineare che è una patologia neurodegenerativa GRAVE che porta a morte del soggetto. Un cane affetto da lipofuscinosi difficilmente raggiunge l'anno di età e trascorre i suoi pochi mesi di vita miseramente, causando sofferenza anche ai proprietari destinati a vederlo spegnere. E' pertanto dovere degli allevatori e degli appassionati impedire che questo accada. Non esistono terapie per la NCL-D.

Cosa hanno in comune queste due patologie? Si tratta di patologie autosomiche recessive, causate da un unico gene che è recessivo. Questo significa che noi possiamo testare il DNA per individuare il gene con un semplice prelievo di saliva o di sangue. Ogni soggetto possiede due copie dello stesso gene. Attraverso l'esame del DNA possiamo scoprire se entrambe le copie sono "sane", in quel caso si parla di cane "**esente**" e omozigote; se è "portatore" (una copia è mutata), quindi il soggetto è "**portatore**" e eterozigote oppure "**affetto**" (due copie mutate). Trattandosi di geni che si comportano in maniera recessiva solo i soggetti "affetti" (due copie mutate), manifesteranno la malattia. I soggetti portatori NON manifesteranno la malattia ma, se si intende usarli in allevamento, vanno accoppiati SOLO con soggetti esenti e i cuccioli vanno poi ricontrollati in quanto il 25% di loro (un cucciolo su quattro) sarà composto da portatori. Un soggetto portatore può trasmettere il gene mutato alla prole. Un soggetto affetto trasmette sicuramente il gene mutato alla prole pertanto NON va messo in riproduzione.

Il costo dei test sul DNA dipende dal laboratorio a cui vi rivolgete ma, ultimamente, ci sono buone offerte. Da [Antagene](#) ho pagato 98 euro per entrambi i test. Si tratta di una cifra da leggersi all'interno di un programma di selezione, ogni allevatore e ogni appassionato, prima di pensare a fare cucciolate, dovrebbe fare tutto il possibile per mettere a mondo soggetti prima ancora di essere "bravi" e "tipici" siano "sani".

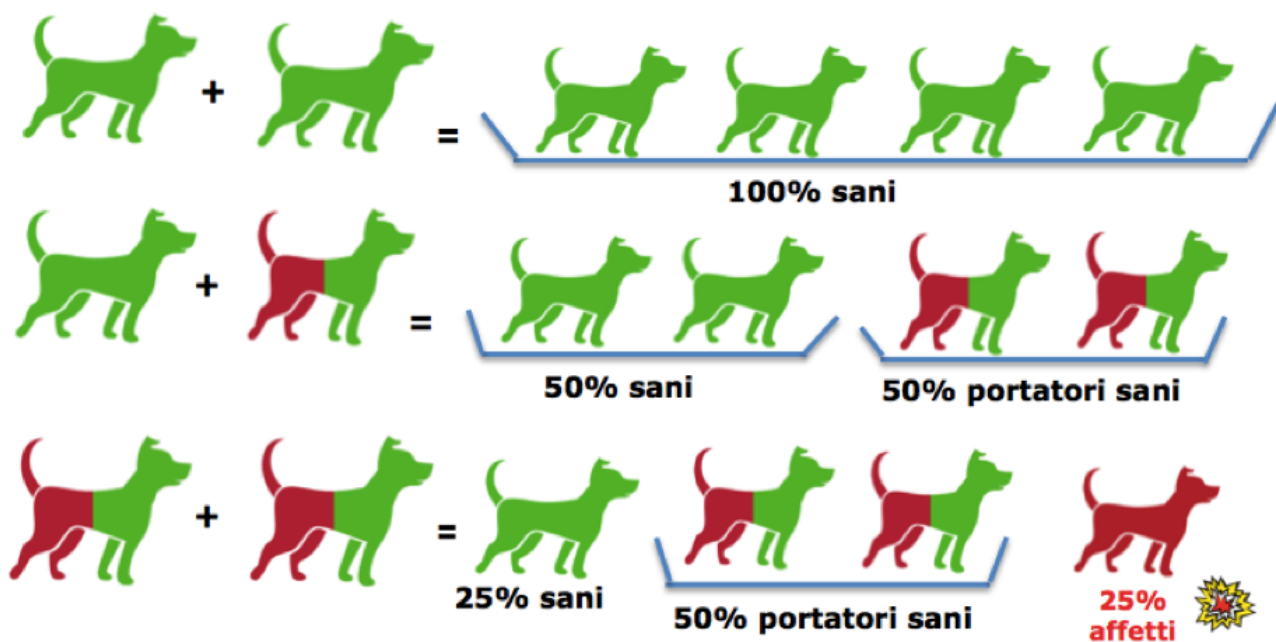


Immagine Antagene

PRA (rcd 4) means Progressive Retinal Atrophy while NCL-D stands for Neuronal Ceroid Lipofuscinosis, two genetic diseases that can be found in some canine breeds, including the English Setter. NCL-D had been known for at least 20 years but, as far as I know, the gene responsible for it had been found only a couple of years ago. I also remember that, at the time, there was only a lab testing for it in Czech Rep.

As for the [PRA](#), the availability of a test for the ES and GS is quite recent as well, while the disease is well known among IS breeders. I wrote about it in my Setter book which came out in 2004.

Which are the symptoms caused by these diseases? PRA causes progressive loss of vision (at night and then in daylight) culminating in blindness. The DNA test identifies only one of the mutations causing PRA (there are more "types" of PRA which seems to affect the ES) and Antagene Lab estimates the mutation to be present in the 7% of the French ES population (most of which has Italian ancestry). There are also cases of PRA (rcd 4) in Norway and carrier dogs who are both of

Norwegian ancestry and of Italian ancestry. There are no therapies for PRA and this is a late onset disease which means the dog might start showing symptoms of the disease after having already been used as a stud/bitch.

I have no numerical data on [lipofuscinosis](#) which is a neurodegenerative disease leading to loss of motor function and vision and to behavioural disorders. The age of onset can vary between 12 and 18 months and the animal will eventually die. It is a serious and painful disease that would devastate owners too, it is therefore very important that breed lovers and breeders work to eradicate it.

Both these diseases are autosomal recessive, hence an animal might have three possible statuses:

Clear (normal homozygous) – Both the copies of the genes are correct, he or she will not develop the disease nor pass the mutation to the prole.

Carrier (heterozygous) – One of the gene copies is mutated, he or she will not develop the disease but will pass the mutation to 50% of the prole. If you intend to breed a carrier, his or her partner must be a **Clear**. In this case, about 25% of the puppies could be carriers as well.

Affected (mutated homozygous) – Both the copies of the genes are mutated. He or she will develop the disease and pass it to all the prole. These dogs must not be used for breeding.

DNA test costs vary according to the laboratory you choose but you can find good deals online. I paid 98 euros (two tests) choosing [Antagene](#), not a huge amount of money if you are a reputable breeder caring about the breed. A reputable breeder must consider health priority, conformation and working ability are very important traits to select for but health should always come first.