IN THE UNITED STATES DISTRICT COURT
FOR THE DISTRICT OF COLORADO

Civil Action No. 07-cv-00722-WDM-MJW

SONYA DIAS,
HILARY ENGEL,
SHERYL WHITE,
Individually and on behalf of all persons similarly situated,

Plaintiffs,

v.

THE CITY AND COUNTY OF DENVER, COLORADO;
JOHN W. HICKENLOOPER, in his official and individual capacity;
NANCY SEVERSON, in her official and individual capacity;
DOUG KELLY, in his official and individual capacity;
JUAN ZALASAR, in his official and individual capacity,

Defendants.

REPORT OF KAREN OVERALL, MA, VMD, PhD

I have been retained by Plaintiffs' counsel to offer my opinion regarding the effectiveness of breed specific bans based on my knowledge of animal behavior, veterinary medicine, and canine genetics. My qualifications for offering this opinion are as follows:

I received my Bachelor's and Master's degrees in Biology concomitantly from the University of Pennsylvania in 1978. My Master's degree concentration was in ethology (the traditional study of behavior and communication systems of animals). My thesis focused on non-vocal signaling behaviors in a group of captive chimpanzees. I was awarded my veterinary degree from the University of Pennsylvania School of Veterinary Medicine in 1983, after a 1-year academic leave, most of which was spent at the Smithsonian Tropical Research Institute in Panama where I worked on foraging behavior of 3-toed sloths. I matriculated as a PhD candidate in the Zoology department, University of Wisconsin – Madison in 1986. During the time I was completing my research for my PhD degree, I undertook a residency in veterinary animal behavior at Penn Vet (1987-1989). I became board certified in veterinary behavioral medicine and a Diplomate of the American College of Veterinary Behaviorists in 1995, the first time that it was possible to sit the exam after presenting your credentials. I successfully defended my dissertation in December 1996 and was awarded my PhD in 2007. My PhD focus was behavioral ecology and evolutionary biology, and my PhD research focus on mating

systems and egg and hatchling survival in a protected lizard. I have been continuously certified as an Applied Animal Behaviorist by the Animal Behavior Society since 1994. After graduating from veterinary school I worked in both small animal private practices and practiced emergency veterinary medicine while taking graduate school courses. Beginning in 1988 I began to run the Behavior Clinic at the Veterinary Hospital of the University of Pennsylvania, which I continued to do for more than a dozen years. In 2001 I was offered an Associate Professor position, with tenure, at the University of Illinois, Champaign-Urbana, under the faculty excellence scholar program, a program designed to attract gifted faculty in areas with unmet needs. Unfortunately, a new dean was appointed after I had accepted the position and I declined it when the promised position for my husband evaporated. I then joined the Center for Neurobiology and Behavior in the Psychiatry Department, School of Medicine, University of Pennsylvania as a Research Associate. I have remained at the CNB since, focusing on canine models of human psychiatric conditions, and the behavioral genetics of canine fears, phobias, and anxieties, including those involved in canine aggression.

During the time I was on the faculty of the Veterinary School at Penn I began to lecture to continuing education groups in veterinary medicine. Over the years I have given hundreds of continuing education and research lectures at meetings nationally and internationally. I continue to lecture and teach courses in veterinary behavioral medicine to veterinarians, although now my primary focus is the behavioral genetics of clinical conditions and normal behaviors in pet dogs.

I am currently co-chair of the joint NIJ, FBI, and DHS / TSA Scientific Working Group on Dogs and Orthogonal Detector Guidelines (SWGDOG). I also serve on the boards of numerous charitable and canine organizations.

I have consulted with lawyers in an expert capacity and periodically testified as an expert witness since 1990. Although I consult on ~6-8 cases a year, fewer than a dozen of these have gone to trial over the years. I have testified in civil and criminal cases for both the defense and the prosecution. I have been qualified / certified as an expert in animal behavior, canine behavior and behavioral genetics, canine aggression, the use of canines in law enforcement, and canine scent detection capabilities.

Scientific Support for Breed Bans

Proponents of breed bans, Denver's included, assert that their basis is rationally rooted in the biology of canine behavior and genetics. The Denver ban, in particular, posits as 'factual' findings, determinations, or assertions about "instinct", specific behaviors involved in fighting, and "a genetic predisposition to aggressiveness". These purported attributes, which are used in the breed ban to support an assertion of dangerousness, are at the core of my research and clinical interests.

My position is that breed bans of any kind are not based in science and that the illusion that they make the public safer is a dangerous one. Banning a breed ignores the following essential aspects of canine biology and risk assessment:

a. Dog behavior is not deterministic. This means that in the absence of any environmental interference, dogs from the same breed will vary considerably in looks and in behavior, as will dogs from the same line and the same litter. In fact, the earliest studies of the behavior of puppies (Scott, Fuller, and colleagues, 1950s and 1960s) noted that the between-individual variation swamped the between-breed variation. This is a finding that has been born out by all subsequent studies on litters of dogs, and all published genetic assays of variation in breeds.

b. Within any breed, dogs bred for looks will vary, relatively speaking, less in looks than will dogs not bred for looks, and dogs bred for work or behavior will vary, respectively, less in how they accomplish the tasks for which they were bred than will dogs not bred with such selection in mind. An example will help. Conformation [show] golden retrievers vary less for looks within the group of show golden retrievers than does the entire population of golden retrievers, which will include show, hunting, and pet / backyard dogs. Border collies selected to work sheep will vary less in behavioral attributes within the group of dogs so selected than do border collies across all populations. This is simple population genetics and it pertains to all aspects of the issues that are always raised when breed bans are considered. By looking at only 1 dog in the group, or any sample from an unknown distribution in the group, no assumptions about the breed distribution with respect to the traits of interest can be made.

c. That said, the patterns discussed in b, above, will themselves vary depending on local culture, country, specific working needs, preferences of breeders, et cetera. Within the USA, show beagles and working beagles are so different that they do not even cluster together genetically when molecular markers are used to compare breeds (Parker et al., 2004). Across the globe, show golden retrievers vary so much in size, shape and color that a novice may not recognize them as the same breed. Implicit in this pattern is that you cannot assume behavior based on some loose standard of looks, as do breed bans, the Denver breed ban included.

d. Dog behaviors, like dog looks, are the result of human tampering with ancestral dogs. This tampering is called artificial selection and it is an essential genetic process upon which all modern agriculture is based. All corn grown in the USA is the result of relentless and varying artificial selection, milk yield in cattle world-wide is the result of artificial selection, and the quality and characteristics of meat from cattle in every feed lot in the USA is the result of ongoing and aggressive artificial selection.

   Ancestral dogs have been genetically separate from wolves for as much as 135,000 years. Dogs have been an integral part of human evolution since modern man developed around 60,000 years ago. Groups of dogs were

developed on the basis of work, and selected to perform certain classes of work at least 15,000 years ago. Different breed classes of dogs are represented in the earliest known artwork, including that in Egyptian monuments. We know that contests testing the skills of different breeds were held at least 1,500 years ago.

The point here is that all modern dog behavior and looks is the result of human selection for certain traits – whether the trait is size, strength, shape, coat color or some behavior. This selection process is continuously ongoing whether people are conscious of it or not, and accidental or deliberate selection for certain behavioral or physical characteristics can alter the conformation or behavior of the selected subset of the breed in as little as a few generations.

The extent to which change in a few generations is possible depends on the heritability of the trait, primarily that part of heritability due to the additive genetic variance. The heritability of any trait can be measured in any known breeding population and is a measure of the variability available on which artificial selection can act. This is also true for "pit bulls" and other breeds targeted in breed bans, but no one collects the data on such populations even though some patterns that are the result of selection are clear for even casual observers to see. For example, there has been an explosion of very tiny / shrunken chihuahuas in the USA after celebrities have been shown with extremely tiny members of the breed. Another example is that we see continuing problems with hips in German shepherds as they become continually larger.

In the absence of data showing the distribution of behaviors within any breed - and their heritability - bans on breed and, or reproduction within that breed are scientifically and logically flawed. In the absence of such data, decisions are not made on the basis of science, but on the basis of the politics, celebrity or as a result of the popular case of the moment. Because there is no data demonstrating the distribution of behaviors within any breed and their heritability, the "factual determination" specifics about behavioral propensities listed in the Denver breed ban are invalid.

e. Once a trait has been selected it will stay in the population unless there is a cost to having it. There is a presumed, apparent cost to most of the configurations of breeds since feral dogs populations usually revert to the look of the ancestral dogs in relatively few generations. This occurs everywhere in the world, and in all areas that it occurs the dogs are of similar, moderate size and of one of a few regionally typical forms (e.g., Asian and SE Asian feral dogs often have curled tails, North American stray and feral dogs often have shepherd-like tails). The Denver breed ban fails to acknowledge that within any breed, a tightly constrained and selected population of fighting dogs that has been selected to exhibit pathological behavior can exist. It is essential to

understand that no normal dog seeks to fight another dog to the death because to do so would adversely affect the dog's own ability to survive. Such behaviors and their sequelae occur in groups of dogs *only* when humans deliberately select for pathological and dangerous behaviors for some reason other than the welfare of the dog. The Denver breed ban reveals an appalling misunderstanding of some easily controlled processes that would identify one population of dogs that potentially pose a risk for other dogs.

f. When the traits are behavioral, they may not be expressed except in certain environmental situations. This is also true for physical traits although the environmental effects on expression may not be as apparent as are behavioral traits to the casual observer. One of the best examples of the effect of environment on the expression of a trait influenced by genetics occurs in the hip dysplasia issue: dogs with lax hips seldom develop clinical disease if they are kept very thin; those with tight hips seldom develop the disease regardless of how heavy they are, although osteoarthrides are more common the more weight any animal carries. Dogs selected to herd sheep may never exhibit the selected behaviors if they never see sheep. These dogs may chase other moving targets (e.g., balls, running dogs and running children), but the overall pattern of the behavior is not identical in cases where it has been examined. This is to be expected since dogs were not – until recently and the advent of flyball competitions – selected to chase balls.

g. Under no circumstances, regardless of selection for specific behaviors, is the dog insensitive to input from his or her external physical, behavioral and resource environment.

    i. Dogs that may never have shown any propensity to hunt may do so when starved.
    ii. Dogs who are in an uncertain social or physical environment (e.g., humane shelters, veterinary offices) will behave completely differently than they would when not in that environment.
    iii. Dogs that may have been aggressive or obnoxious in one home may exhibit completely different behaviors when in another home.
    iv. Dogs that have been in homes where nipping or biting was tolerated stop nipping and biting both when the behavior is no longer tolerated in the same home (e.g., when the human behavior changes) or when adopted into a home where such behaviors are not considered acceptable.
    v. Dogs that have pathological aggression can recover from it and cease to bite once their people understand and implement the steps needed to help the dog recover.

h. Finally, 'risk' is a statistical quality that can be assessed in numerous ways.

i. *Breed-specific bite rates* indicate the percentage of dogs of a specific breed involved in biting incidents. For this calculation to be made we would need to know the populations of all biting *and* non-biting individuals. Only when we have these data can we establish breed-specific bite rates. Of course, we also have to define "bite" and ensure everyone is assessing the dogs in a consistent manner. Currently, the definition of a "bite" varies considerably and this assessment is not independent of prejudice or politics: "family" dogs are often said to "nip", whereas select breeds are said to "bite", even when the family dog has done more physical damage. Accordingly, to truly and accurately calculate breed-specific bite rates we would have to identify the circumstances of the bite to determine whether the population as a whole was at risk. For example, if Labrador Retrievers are found to "bite disproportionately" – once the criteria for assessing a "bite" have been agreed on, and we looked further and learn that most Labradors live in families with children and that almost all the bites were to children who were teasing the dog, the "risk" to the general population of humans and dogs is negligible. Not only are such data unknown for the Denver population, in general, but no attempt has been made to assess the complete 'biting' population – including those dogs whose bites did not make the press, or to assess the 'non-biting' population of any breed. Such an approach is particularly injurious and prejudicial to dogs of breeds that are targeted to be banned. The data available to calculate breed-specific bite rates are not available in Denver, nor is their collection mandated by the breed ban. This means that no scientific assessment of the rationale or effectiveness of the ban is possible.

ii. *Relative risk* indicates how much more or less likely a specific breed is to be involved in a bite incident, compared with other breeds. Relative risk requires a good estimate of populations of all breeds. These data are not available in the Denver population of dogs, nor is the collection and maintenance of such data mandated by the breed ban.

iii. The *population attributable fraction percent (PAF%)* is a measure of a breed's effect on overall population. PAF% is used in the context of comparing numbers of biting dogs from different breeds. The data needed to calculate PAF% are almost never known and are certainly not known in the Denver population of dogs. Estimations of PAF% are often calculated from licensed dogs. This is a fatally flawed approach. The population of licensed dogs already varies greatly with human populations and the individual human's sense of responsibility in complying with law (the dogs cannot register themselves for a license). With any breed ban any estimate of PAF% will become less accurate and even more unreliable because compliance with licensing laws for any banned breed will be non-existent.

As a scientist, a veterinarian, and a dog owner, I understand that the vast majority of any breed of dogs does not attack people or other dogs. My data and those of others in the field, show that the vast majority of bites to humans occur within family pet situations, and that if a child is involved, more often than not the dog is provoked (De Keuster et al., 2005; Overall and Love, 2001). The vast majority of bites in the USA are to male children between the ages of 5 and 9 years. This is a human pattern, not a breed pattern(Overall and Love, 2001; Love and Overall, 2001). Bites to family members number in the millions of bites per year, but these are not the data that get the attention of legislators.

Logic and science demand that we understand that if the millions of bites to humans a year in an owned population of pet dogs depends on human behavior, that human behavior is also an essential factor in the bites of breeds that are media magnets and that are the focus of breed bans. If this is so, especially given the history of artificial genetic selection discussed above, banning breeds will do nothing, but regulating human behaviors with dogs could be extremely powerful.

Laws based on pseudoscience, media magnets, and a woeful ignorance of the real risks inherent in interactions with dogs and the contexts in which those interactions occur are worse than wrong. Such laws, like the Denver breed ban, are dangerous. The impression that these laws will make us safer is a false one. Worse yet, these bans perpetuate risk to the public by failing to address the tough issues involving situations which put people and dogs at risk including but not restricted to:

1. dogs bred for illegal dog fighting that overflow into the stray, feral, or shelter populations,
2. the cause of canine overpopulation which feeds the stray, feral, continually reproducing and recycled group of ill-treated street dogs that is the *sole* result of irresponsible human relationships with dogs, not breed;
3. irresponsible dog ownership where abusive training practices that enhance a dog's risk for aggression are tolerated;
4. irresponsible and uneducated dog ownership where inappropriate aggression is tolerated and, or encouraged, often because the owner mistakenly thinks an aggressive dog will protect the family;
5. irresponsible and neglectful dog ownership where dogs and children are taught that violence is a solution to conflict.

In the absence of addressing the 5 issues above, all of which are issues of human responsibility, banning one breed or one cluster of breeds will not alter the risk of any of the problems that the ban apparently seeks to redress. All that will happen is that the shape, style, and breed of the dogs exhibiting the behaviors that attract media and legislative attention will change.

Canine Genetics

Bans by any category are always suspect on scientific grounds because of the variation within populations. This finding is important because of the undiscussed consequences of Denver's pit bull ban.

People who have behaved in illegal or unethical ways will not change their behaviors because there is a specific ban on one aspect of their non-compliant behaviors – they will merely take these behaviors underground. Those who comply with breed bans and who care about whether their property is insured will forfeit perfectly lovely dogs because they are law abiding citizens who do not wish to contribute to risk or be thought by an insurance company to contribute to it. When you combine these 2 factors you end up with a breeding population of the 'banned breed' that is now comprised almost wholly of potentially problematic dogs. If there is a genetic component to any of the behaviors that have encouraged people to take their dogs underground, as is likely in the case of fighting dogs, the Denver legislation and the legislators who argued for it will have increased the proportion of inappropriately behaved dogs within the breed. This outcome is due to basic population genetics.

The arguments supporting breed bans are also flawed with respect to findings in molecular genetics. Impressions about what molecular genetics can and cannot do are often misunderstood by both those who believe that this technique will identify specific "aggressive" dogs and by those who fear that such tests will implicate innocent dogs. It is essential that everyone with a stake in this issue understands the current state of canine molecular genetics.

First, the goal and outcome of most molecular genetic studies is to identify a region on a chromosome that is statistically associated with some pattern of disease, morphology or behavior. In other words, when you look for 'markers' you are looking at regions that vary only between the population of individuals that have the trait of interest when compared with those from populations not having the trait. When you identify this region, the expectation is that the gene of interest is within the region. This is the same rule that allows us to genetically determine that dogs and wolves are different species, that Australian shepherds and whippets are different breeds, and that you have 4 samples of the same dog's blood rather than 1 sample from each of 4 dogs. That said, unless a diagnostic test for products of the gene or a specific region of the genetic code is available, you seldom actually identify the specific 'gene' of interest.

Second, the above statistical pattern allows you to assign possible risk, but usually neither cause nor a mechanism for the occurrence of the trait or pattern in which you are interested. Part of this is because the argument is a probabilistic one. If you have the pattern in the region of note you are more at risk for the problem; if you do not have it your risk is lower. Seldom have such regions been shown to be deterministic. Effects of other genes and environments are always factors to consider.

Third, in the vast majority of cases, one gene or gene region variation (called a genetic polymorphism) is not sufficient to generate a specific outcome or phenotype (what the outcome looks like to those assessing it) and no where is this more true than for behavior.

The field of molecular behavioral genetics is still in its relative infancy except for organisms like mice and *Drosophila* (fruit flies) which lend themselves to genetic manipulation in the lab. Dogs are incredibly complex organisms and no one should expect to identify a region of DNA that – regardless of environment or other gene effects – produces one, specific, invariant behavior.

To use an example that demonstrates the complexity of canine genetics we can look at a study that has been done to explain physical size. Using the genetics of size as an example is helpful because (1) we can measure this in a way that everyone who measures it can get the same answer (e.g., the dog is 47 cm at the shoulder) and (2) everyone has a gut feeling about what it means to be relatively smaller or taller. Researchers at the University of Utah have looked at size in Portuguese Water dogs (Chase et al., 2005). These dogs are well known to have females that are much smaller than males. While this pattern is true for many dog breeds, the pattern is extreme in this breed and it is thought that modern Portuguese water dogs are all descended from a relatively small number of ancestors ('founders'), meaning that you will have less overall variation. Having less overall variation is useful for these types of studies because – as already stated – they are probabilistic. This means that if you have a population that doesn't have a lot of genetic variation and you find variation within it that corresponds well to the trait in which you are interested, you can be reasonably comfortable that the variation you detect and the trait are linked.

Using more than 500 microsatellite markers within complete genomes scans of more than 500 dogs Chase et al. learned canine chromosome 15 (CFA15) had a strong association with body size, and that the most common place the markers varied was close to insulin-like growth hormone-factor-1 gene (IGF1). Further work revealed that CFA15 interacts with another locus on the X chromosome that does something remarkable: it escapes 'inactivation'. In species that use XY sex determination – including dogs and humans - everyone needs one X chromosome to live, but everyone needs only 1. Females have 2 (XX) and males have 1 (XY). To escape the dosage effect that would result if 2 X chromosomes were active, females 'inactivate' one X chromosome. In the case of Portuguese Water Dogs it is the activation of this region of the X chromosome that matters. Females that have 2 copies of the same X-chromosome pattern (homozygotes) and who also have 2 copies of the large-size CFA15 pattern will be as big as males. All females who have only 1 copy of the X-chromosome pattern (heterozygotes) are small regardless of CFA15 status.

This story is complex and it focuses on easily measured traits. We should expect the genetics of specific behaviors to be even more complex. There are early data to support this contention.

1. Data from Japan indicate that overall measures of heterozygosity (how much variation there is within a breed across a number of individuals) vary considerably with breed. However, for many loci (regions that contain the suspect genes) involved in neurotransmitter function, in some cases no variation across members of the breed has been reported, despite the fact that the dogs involved all behave

differently. This means that even with virtually no variation in genes that control neurochemicals dogs still behave differently from each other, and that patterns of neurochemical polymorphisms do not map directly on to specific behaviors in the general population. When mtDNA – DNA inherited only from the mother – is used the analysis is less complex, but for somatic illnesses (eg, mammary neoplasia, hip displasia, mitral valve abnormalities) that appear to run in family lines, there is still no evidence that any one gene is causal or that one gene causes the same conditions in all breeds or that disease directly maps onto any identified haplotypes (sequence inherited as a block) (Takahasi et al., 2002). That said, microsatellites may be useful to distinguish family lines within mixed-breed populations (Oishi et al., 2005). Identifying potential family lines in mixed-breed populations means that we can group dogs together that are more genetically alike than others, building a potential family tree. Obviously the story is more complex than variation in one or a few genes (Ito et al., 2004; Takeuchi et al., 2005). Some markers may ultimately identify functional polymorphisms in the regulatory region of the gene involved in the functioning of neurochemical genes, but the relationship to specific behavioral suites remains unknown (van den Berg et al., 2005).

2. When survey questionnaire data about general behaviors are used to compare high versus low heterozygosity dogs with respect to a few neurotransmitters, there appears to be a hint of a broad trend, but no predictions about individual dog behavior could be made based on pattern of genetic markers studied (Masuda et al., 2004; Ogata et al., 2006).
3. The most discrete study to look at specific polymorphisms in genes thought to be associated with "temperament" did so with respect to 4 genes and dogs being reared as guidedogs. When suites of behaviors that make good guide dogs were examined, only one polymorphism of one gene (SLC1A2) was shown to have an effect (Arata et al., 2007). Obviously, no one gene or set of genes will be deterministic for success as a guide dog. These types of data support the contention that simple blood tests for complex behaviors are not likely to be a realistic expectation.
4. Studies that have approached the effects of the genetics of breed on behaviors associated with aggression found that >95% of dogs of all breeds – including those on breed lists (bull terriers, American Staffordshire terriers, pit bull terriers, et cetera) behaved appropriately and in context, and that most of the out-of-context behaviors were responses to behaviors owners exhibited when they misunderstood the dog (Schalke et al., 2007). Additionally, when the behavior of golden retrievers was compared, under varied and controlled circumstances, with that of members of breeds of dogs on banned lists, no differences were detectable (Ott et al., 2007)

Denver's Ban

In summary, there are no behavioral phenotypic data, no population genetics data, and no molecular data to support banning a dog simply because it is a member of a certain breed.

Specific applications of all of these scientific findings to the Denver breed ban clearly show its flaws.

1. The breed ban will not make citizens safer because certain breeds are relinquished or hidden because bites from these breeds do not comprise any significant proportion of dog bites. If any government wishes to protect people from dog bites the greatest impact is will be made by educating owners of dogs about normal and problematic canine behavior, and educating children about how to react to dogs in their own homes, in the homes of their friends, and on the street. This fact has been recognized by the AVMA and they are now supporting *The Blue Dog* bite prevention program for children. It should also be implicit and apparent that any time a breed or individuals of a breed are hidden, they are not 'safer' for the public. In fact, these dogs will experience fewer of the circumstances that could make them safer, and their owners will not have available to them information that could prevent tragedies.

2. One of the reason pit bulls have been targeted in this ban involves the assumption that they do more damage than other breeds when they bite. This is not completely true (Sacks et al., 1989, 2000). There are 3 factors involved in the amount of damage done when a dog bites: force with which the dog hits the human (or dog), mass of muscles used in biting, and behavior characterizing the bite.

    a. Force equals mass times acceleration, so any 3 breeds of the same weight – no matter how it is distributed – and moving at the same speed will hit a human with the same force. Pit bulls often weigh less than the average exuberant Labrador retriever, a fact that needs to be considered here.
    b. However, the power or force with which jaws act depends both on muscle mass and the area of attachment for the muscles involved. The larger the area of attachment, given the same muscle mass and the same tooth size, the more force will be delivered. Pit bulls and all, heavily jawed breeds – most, but not all of which are mastiff breeds - will accordingly deliver more force when they bite, all other things being equal. Heavily muscled jaws can deliver bites of 600-800 pounds of pressure a square inch – which is about what a tapir exerts to crack some tropical nuts.
    c. However, the behavioral characterization of a bite is perhaps the most essential aspect of any dog bite when determining how much damage is done. Unfortunately, this aspect of dog bites has received little attention. A dog that inhibits his bite and barely touches the skin does little physical damage. Biting and releasing is one key behavior used to evaluate how dangerous dogs are. Dogs that "repeat bite" are more dangerous than dogs that bite once and release. Dogs that hold on are more dangerous than others. Dogs that hold on and will not relinquish their victim can cause serious deep tissue injury due to crushing, and subsequent tearing. In most other types of dogs bites, much of the damage is done by tissue tearing which occurs primarily when the victim

tries to escape, but can also occur if the dog shakes the victim. None of these patterns of biting is breed-specific.

d. Mastiff-type breeds, including pit bulls possess muscle mass that makes an attack by an animal that is not inhibited potentially more damaging, but because of the physics of force delivery, a larger non-mastiff dog can do as much injury as a slightly smaller mastiff-type dog. The aspects affecting severity of bites discussed here have not been shown to vary across breeds in any consistent way except that dogs with bigger mouths can grab more tissue in 1 bite. Having viewed autopsy and injury photos of damage done from dog bites I can say with certainty that while it is possible to identify injury that is the results of tearing v. injury that is the result of a crush, it is impossible to discern the breed of dog that inflicted the bite from the actual damage. In some rare cases it is possible to identify dental structure of the dog that has caused the injury.

e. The argument has been made that pit bills – because of the nature of the breed – are inherently more dangerous because they are genetically predisposed to attack. There are absolutely no data to support this myth, and anyone seeking to advance such reasoning is cautioned that none of the genetic data cited above, including those on the development of behaviors (Ott et al., 2007; Schalke et al., 2007), support this contention of behavioral determinism. The small subset of dogs bred **by** pathological humans to be pathological **to** humans is not restricted to any breed, even as characterized by an enthusiastic press. These dogs are also subject to the same patterns of genetic selection as discussed above, and should be very rare, indeed, because breeding for such acute pathology is not simple. Even when scientists select for specific pathologies in laboratory situations, they do not get just that pathology and they do not get only one level of that pathology. To say that a breed ban will eliminate these dogs is to say that genetic determinism is the cause of all dog behavior. This is simply not accurate.

Public Safety

If the Denver government is serious about decreasing the number of bites to dogs it needs to focus on the populations of dogs and humans posing a risk.

1. Illegal behavior by humans needs to be stopped. This means enforcing bans on animal fighting and incarcerating or otherwise preventing those who engage in animal fighting from doing so with a newly acquired batch of dogs.
2. Abuse and mistreatment of animals of any kind – including abandonment of animals - needs to be stopped and penalized and adults and children must be taught about the link between animal abuse and child and spousal abuse. Children, especially, must have as part of their school curriculum training in the appropriate care and handling of pet dogs.
3. Schools and community groups need to incorporate *The Blue Dog* program into all pre-school and early-school training, and they need to reinforce this is later years with hands on training. This program will be most successfully

introduced and used if primary care practitioners, pediatricians, and veterinarians work together in the educational effort.

4. Accurate reporting of dog bites needs to done in a manner that is scientifically useful. This means that all dogs should be licensed and microchipped so that they can be followed through the population, and all bites should be reported in a manner that encourages – not discourages – people to do so. When dogs are determined to be problematic, humane care, assessment and treatment must be mandated or the dog must be relinquished. However, assessment and treatment must be the initial step, in part, because it is the only way we will obtain accurate data on dog bites.
5. Veterinarians and human physicians need to join together to understand risky behaviors on the parts of humans when interacting with dogs, and to understand the type of bite inflicted by the dog in a manner that accounts for the choice of canine behaviors, given the circumstances.
6. The community must support humane care and training of all pets, including detailed training for families with dogs. Schools and scouting groups can help by promoting dog training.
7. The CSU veterinary school has no specialist in veterinary behavioral medicine. This single omission does more to deprive the community of education about these issues than does any other single factor. If the legislature is serious about implementing any of these options, a tenure rank faculty position is mandatory.

If the safety of the citizens and decreasing the probability of dog bites is the goal, these are the only measures that have been shown to be effective.

References

Arata S, Kaneko F, Momazawa Y, Ogata N, Yoshizawa S, Fukui R, Tawada S, Nakamura T, Kikusui T, Takeuchi Y, Mori Y. Search for temperament-associated genes in guide dogs (abstract). J Vet Behav: Clin Appl Res 2007;2:97-98.

Chase K, Carrier DF, Adler FR, Ostrander EA, Lark KG. Interaction between the X chromosome and an autosome regulates size sexual dimorphism in Portuguese Water Dogs. Genome Res 2005;15:1820-1824.

De Keuster T, lamoureux J, Kahn A. Epidemiology of dog bites: a Belgian experience of canine behaviour and public health concerns. Vet J 2005;172:482-487.

Ito H, Nara H, Inoue-Murayama M, Shimade MK, Koshimura A, Ueda Y, Kitagawa H, Takeuchi Y, Mori Y, Murayama Y, Moritat M, Iwasaki T, Ota K, Tanabe Y, Ito S. Allele frequency distribution of the canine dopamine receptor D4 gene exon III and I in 23 breeds. J. Vet Med Sci 2004;66:815-820.

Love M, Overall KL. Dogs and children: how anticipating relationships can help avoid disasters. J Am Vet Med Assoc 2001;219:446-453.

Masuda K, Hashizume C, Ogata N, Kikusio T, Takeuchi Y, Mori Y. Sequencing of canine 5-hydroxytriptamine receptor (5-HTR) 1B, 2A, 2C genes and identification of polymorphis in the 5-HTR1B gene. J Vet Med Sci 2004:66:965-972.

Ogata N, Hashizume C, Momozawa Y, Masuda K, Kikusui T, Takeuchi Y, Mori Y. Polymorphisms in the canine gluatamate transporter-1 gene: identification and variation among five dog breeds. J Vet Med Sci 2006;68:157-159.

Oishi N, Maeda M, Makimura K, Sawaguchi T, Hayashiya M, Kubo T, Kano R, Hasegawa A, Kasahara M. Microsatellite polymorphism in Japanese mongrel dogs. H Vet Med Sci;67:1055-1057.

Ott SA, Schalke E, van Gertner AM, Hackbarth H. Is there a difference? Comparison of golden retrievers and dogs affected by breed specific legislation regarding aggressive behavior (abstract). J Vet Behav: Clin Appl Res 2007;2:92.

Overall KL, Love M. Dog bites to humans: demography, epidemiology, and risk. J Am Vet Med Assoc, 2001;218:1-12.

Parker HG, Kim LV, Sutter NB, Carlson S, Lorenytzen TD, Malek TB, Johnson GS, DeFrance HB, Ostrander EA, Kruglyak L. Genetic structure of the purebred domestic dog. Science 2004;304:1160-1164.

Sacks JL, Sattin RW, Bonzo SE. Dog bite-related fatal bites from 1979 trhough 1988. JAMA 1989;262:1489-1492.

Sacks JL, Sinclair L, Gilchrist J, Golab GC, Lockwood R. Breeds of dogs involved in fatal human attacks in the United States between 1979 and 1998. J Am Vet Med Assoc 2000; 217:836-840.

Schalke E, Ott SA, van Gertner AM, Mittmann A, Hackbarth H. Is breed specific legislation justified? Evaluation of the temperament test of Lower Saxony (abstract). J Vet Behav: Clin Appl Res 2007;2:91-92.

Scott JP, Fuller JL. 1965. Genetics and the Social Behavior of the Dog: The Classic Study. University of Chicago Press, Chicago, IL.

Takahasi S, Miyahara K, Ishikawa H, Ishiguro N, Suzuki M. Lineage classification of canine inheritable disorders using mitochondrial DNA haplotypes. J Vet Med Sci 2002;64:255-259.

Takeuchi Y, Hashizume C, Chon EMH, Momozawa Y, Masuda K, Kiksui T, Mori Y. Canine tyrosine hydroxylase (TH) gene and dopamine B-hydroxylase (DBH) gene: their sequences, genetic polymorphism, and diversities among five different dog breeds. J Vet Med Sci 2005;67:861-867.

van den Berg L, Kwant L, Hestand MS, van Oost BA, Leegwater PAJ. Structure and variation of three canine genets involved in serotonin binding and transport: the serotonin receptor 1A gene (htr1A), serotonin receptor 2A gene (htr2A), and serotonin transporter gene (slc6A4). J Heredity 2005;96:786-796.

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