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Research article

PREVALENCE OF THE *ABCB1* NT230[DEL4] MUTATION IN DIFFERENT DOG BREEDS IN TÜRKİYE

Meryem TOPRAK TUNCER[1*], Merve YÜKSEL[2], Bilgenur KORKUT[3], Ahmet ATEŞŞAHIN[1]

[1]Department of Pharmacology and Toxicology, Faculty of Veterinary Medicine, Fırat University, Elazig, Türkiye; [2]Department of Genetics, Faculty of Veterinary Medicine, Fırat University, Elazig, Türkiye; [3]Faculty of Veterinary Medicine, Fırat University, Elazig, Türkiye

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In this study, the presence of a 4 base pair deletion mutation (nt230[del4]) in the ATP-binding cassette subfamily B member 1 (*ABCB1*) gene was investigated in different dog breeds in Türkiye. For this aim, blood samples collected from a total of 161 dogs of 5 different breeds, including German Shepherd Dogs (GSD), Golden Retrievers (GR), Pomeranians (POM), Toy Poodles (TP), and Kangal Shepherd Dogs (KSD). Genomic DNA was isolated from blood samples using the phenol-chloroform extraction method. Then, to detect the nt230[del4] mutation in the *ABCB1* gene, genomic DNA was amplified using degenerate primers via Polymerase Chain Reaction (PCR) and analysed using the Polymerase Chain Reaction-Restriction Fragment Length Polymorphism (PCR-RFLP) method. The amplified PCR products were digested with a restriction enzyme, separated by agarose gel electrophoresis, and genotypes were determined based on band patterns. According to the analysis results, all dogs examined were found to possess the homozygous wild-type (+/+) genotype, and no mutant allele was detected. These findings indicate that the *ABCB1* gene region exhibits a monomorphic structure in the dog population included in the study. The results obtained may contribute to the identification of genetic variations that could be significant in terms of drug sensitivity in the relevant dog breeds. These findings suggest that *ABCB1* gene variation may be limited in the dog population in Türkiye, implying that the genetic risk regarding drug sensitivity may be low in the relevant breeds.

Keywords: *ABCB1* mutation, dog breeds, pharmacogenetics, drug sensitivity

INTRODUCTION

Transporter proteins localized in plasma membranes have a significant effect on the pharmacokinetics of drugs. Among these proteins, which transport both endogenous

*Corresponding author: e-mail: m.toprak@firat.edu.tr

compounds and exogenous substrates, are the Multidrug Resistance-Associated Protein (MRP), Multidrug Resistance (MDR), Organic Cation Transporter (OCT), Organic Anion Transporter Polypeptide (OATP), Organic Anion Transporter (OAT) and nucleoside transporters [1]. P-glycoprotein (P-gp), an ATP-driven multidrug efflux transporter, is a membrane protein that transports exogenous and endogenous substrates from the interior of cells to the exterior [2]. P-gp is the product of the *ABCB1* gene, also known as the multidrug resistance 1 (*MDR1*) gene, which belongs to the ATP-binding cassette (ABC) superfamily, and is among the best-characterized drug transport proteins [3]. Since P-gp plays a key role in the distribution of drugs to tissues, it has a significant impact on therapeutic efficacy and toxicity. P-gp, the product of the *ABCB1* gene, is considered a highly significant transporter protein. This is because many therapeutic drugs are P-gp substrates, and various mutations affecting P-gp expression in different cells have been identified [4].

Interest in the potential role of genetic variation in *ABCB1* in drug therapy is growing. Many genetic polymorphisms have been identified in *ABCB1*; some of these have been shown to affect P-gp expression levels and substrate transport, and it has been reported that certain polymorphisms influence the pharmacokinetic and pharmacodynamic profiles of drug substrates and may directly impact the outcome and prognosis of specific diseases [5-7]. In addition, mutations in the *ABCB1* gene can lead to the inactivation of the P-gp transporter, resulting in drug accumulation in the brain and potentially causing severe neurotoxicity [8].

Gene polymorphism is defined as the presence of two or more variations (alleles) of a gene across different species or populations. Such genetic variants can change the phenotype through effects on biological processes, including the structure, function or amount of a protein. Gene polymorphisms are most found as a single-base change (single nucleotide polymorphisms, SNPs) [9]. These modifications are involved in numerous biological processes such as the development of certain diseases, drug responses, and effects from exposure to toxic agents [10]. Increasing availability of information on genetic polymorphisms in membrane transporters, especially those involved in drug disposition and response, is possible due to developments in genomic technologies. Polymorphisms in the *ABCB1* gene can affect the function of P-gp. This may lead to significant changes in drug absorption, distribution, metabolism and excretion processes [11]. Many drugs (e.g. chemotherapeutic agents used in cancer treatment or antidepressants) are excreted from cells via P-gp. For this reason, *ABCB1* gene polymorphisms can directly influence drug sensitivity or drug response. For example, some studies showed that the C3435T polymorphism in the *ABCB1* gene, a single nucleotide polymorphism (SNP), could influence the bioavailability of some drugs via affecting P-gp expression levels [12-14]. Therefore, polymorphisms of *ABCB1* are considered as one of the most important research areas in personalized medicine and drug development. In veterinary medicine, gene polymorphisms, especially in dogs, are vitally important to understand the effects of genetic variation on health. Gene polymorphisms can affect the animals' resistance to some diseases,

their response to drugs and their ability to resist environmental stressors. Genetic variation in dogs is an important area of research for the diagnosis, treatment and prevention of disease. In this regard, the *ABCB1* gene and its polymorphisms in dogs are of particular importance. In dogs, as in humans, the *ABCB1* gene encodes for a P-gp, a drug-transporting protein. The *ABCB1* gene mutation is also responsible for the extreme drug sensitivity of some dog breeds [15]. This mutation of 4 base pair deletion in the *ABCB1* gene is named differently in the literature and is commonly shown as nt230[del4], MDR1-1Δ, or c.227_230delATAG (ATAG deletion). In addition to ivermectin and its derivatives, dogs with this mutation are sensitive to commonly used veterinary drugs such as loperamide, acepromazine, butorphanol, vincristine and vinblastine. These drugs may develop toxicity and may even lead to death [16].

In Türkiye, data on *ABCB1* gene polymorphisms in dog breeds are limited, indicating a substantial lack of knowledge, especially regarding genetic variability and drug sensitivity in both indigenous and non-indigenous breeds. Dog breeds such as the German Shepherd Dog (GSD), Golden Retriever (GR), Pomeranian (POM), and Toy Poodle (TP) are among the most common dog breeds owned and seen in clinical settings in the country, although they are not indigenous to Türkiye. In contrast, the Kangal Shepherd Dog (KSD) is an indigenous breed with a large population but has been insufficiently characterized at the genetic level. It is expected that the investigation of *ABCB1* gene mutations in the breeds studied, raised in Türkiye, will raise awareness among veterinarians and breeders. Furthermore, these studies could enhance our knowledge of breed-specific drug sensitivities and toxicity risks, thus aiding the development of personalized therapeutic approaches. Therefore, the purpose of this study was to investigate nt230[del4] in the *ABCB1* gene in the KSD, GSD, GR, POM, and TP breeds obtained from different regions of Türkiye.

MATERIALS AND METHODS

Sample collection

The study was approved by the Local Ethics Committee for Animal Experiments of Fırat University (Approval Number: 2025/12-11). Blood samples were obtained from a total of 161 dogs, including 25 GSD, 20 GR, 18 POM, 28 TP, and 70 indigenous KSD. Samples from GSD, GR, POM and TP were collected from veterinary clinics in İstanbul, Ankara, Antalya, İzmir, and Malatya, whereas KSD were sampled from the principal regional KSD breeding facility in Elazığ, which draws breeding stock from across the wider surrounding area rather than a single closed pedigree line (Figure 1). Breed identity was determined through veterinary clinical/phenotypic assessment combined with owner-reported breed information; no molecular breed-verification testing was performed. Sample size for each breed was defined according to their representation and accessibility through owners, a breeder and veterinary clinics. All dogs were clinically evaluated by responsible veterinarians and were considered healthy,

not showing any clinical signs of disease. Sex and age were not used as selection criteria. The study population consisted of 95 females and 66 males; the age range is from two months to 15 years. Cephalic vein blood samples were collected from each dog using sterile needles and transferred into vacuum ethylenediaminetetraacetic acid (EDTA) tubes. Samples were kept at approximately 4 °C in insulated containers with ice packs and shipped to the laboratory within a few days. They were stored at -20 °C upon arrival before DNA extraction.

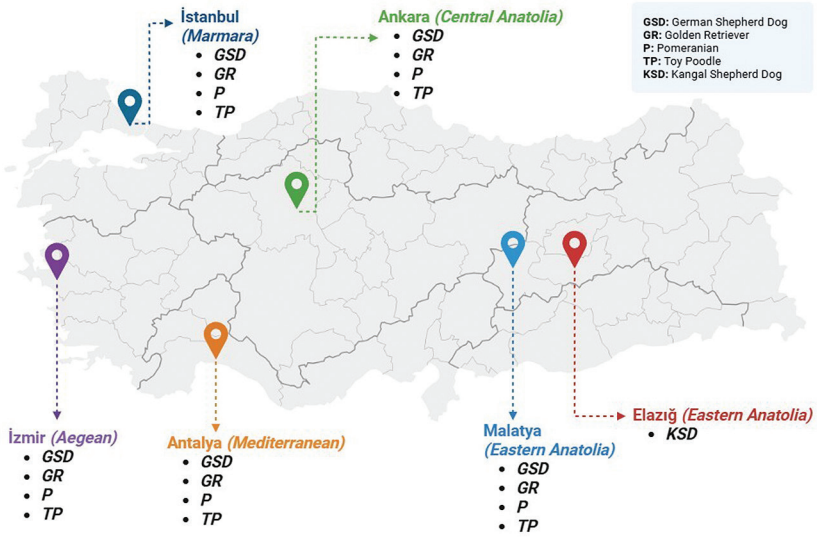

Figure 1. The study population consisted of five dog breeds (n = 161) sampled from multiple provinces across different geographical regions of Türkiye

DNA extraction

Genomic DNA extraction from all blood samples was performed after storage at -20 °C, using a phenol-chloroform extraction method previously described by Sambrook et al. [17]. DNA yield and purity were verified by spectrophotometric analysis (Thermo Fisher Scientific, Waltham, Massachusetts, United States of America) and agarose gel electrophoresis.

PCR-RFLP analysis

According to the method established by Silvestro et al. [18], degenerate primers were used for PCR amplification to identify the associated mutation of multidrug resistance in the *ABCB1* gene, as shown in Table 1.

PCR amplifications were performed in a 20 µL reaction containing 10x PCR buffer, 1.5 mM MgCl□, 10 µM of each primer, and 0.2 mM dNTP mix with 1.5 U Taq DNA polymerase (Thermo Fisher Scientific, Waltham, Massachusetts, United States

of America). For each PCR reaction taken template DNA at a concentration of about 50 ng.

Table 1. Primers used for amplification by PCR of the *ABCB1* gene

Forward	5'-CGCTATTCAAATTGGCTTGATAGG-3'	134/130 bp
Reverse	5'-GAAATTCCTGCATTTGCA**C***AGC-3'	

bp: base pairs; * and underlined base indicates nucleotide degeneracy within the primer sequences.

PCR amplification was performed as described in the modified cycling protocol from Silvestro et al. [18]. The thermal profile created an initial denaturation step at 94 °C for 5 min, then 35 cycles of denaturation at 94 °C for 1 min, annealing at 55 °C for 1 min and extension at 72 °C for 45 s. A final extension step was performed at 72°C for seven min.

The PCR products generated using the degenerate primers designed by Silvestro et al. [18] are expected to yield an amplicon of 134 bp in the reference canine genome. In dogs that carry the nt230[del4] mutation, however, the amplicon is predicted to be 130 bp in length. The difference in size between these two amplicons is too great to be discriminated against by using agarose gel electrophoresis alone. Therefore, confirmation of the presence of the mutation was performed using RFLP analysis of the PCR products. In the following amplification, the PCR fragments were incubated with 5 U of *PvuII* (Thermo Fisher Scientific, Waltham, MA, USA) at 37 °C for 16 h to allow restriction digestion.

RFLP products were visualized by Ultraviolet Transilluminator (ECX20M, Vilber Lourmat, France) after separation on a 2.5% agarose gel. The samples with the wild-type genotype were expected to show a single band of 134 bp (+/+) while heterozygous samples were expected to show three bands of 134, 109, and 21 bp (+/-). The homozygous mutant samples were expected to show two bands of 109 and 21 bp (-/-).

RESULTS

Digest of PCR amplicons (134/130 bp) with the *PvuII* restriction enzyme we determined that all 161 dogs tested were homozygous for the wild-type genotype (+/+) and no mutant alleles (Figure 2).

Genotype distribution, allele frequencies, and information on age and sex for the five dog breeds are shown in Table 2.

All dogs exhibited a homozygous wild-type genotype (+/+), indicating a monomorphic pattern in the studied population. The mutant allele was not detected in any of the five breeds studied. Accordingly, the allele frequencies were calculated as 0.00 for the

mutant allele and 1.00 for the normal/wild-type allele, both for each breed and for the study population as a whole. Due to the absence of polymorphism in the studied population, statistical analyses such as Hardy-Weinberg equilibrium testing were not applicable.

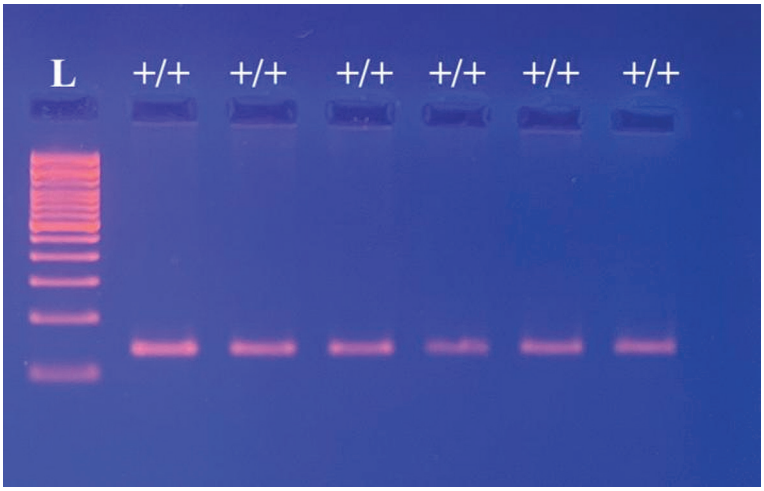

Figure 2. Agarose gel electrophoresis image of the PCR-RFLP pattern for the *ABCB1* nt230[del4] mutation, showing representative homozygous wild-type (+/+) samples from the studied canine population. Lane L represents a 100 bp DNA ladder

Table 2. Genotype distribution and allele frequency of the *ABCB1* nt230[del4] mutation by breed, with age and sex data, in the studied dog population

Breeds	**Age**	**n (dogs, F:M)**	**+/+ (n, %)**	**+/- (n, %)**	**-/- (n, %)**	**Alleles (2×n)**	**Mutant allele frequency**	**Wild-type allele frequency**
Kangal Shepherd Dog	6 months-9 years	70 (F:37, M:33)	70 (100%)	0 (0%)	0 (0%)	140	0/140 = **0.00**	140/140 = **1.00**
German Shepherd Dog	2 months-6 years	25 (F:16, M:9)	25 (100%)	0 (0%)	0 (0%)	50	0/50 = **0.00**	50/50 = **1.00**
Golden Retriever	2 months-15 years	20 (F:14, M:6)	20 (100%)	0 (0%)	0 (0%)	40	0/40 = **0.00**	40/40 = **1.00**
Pomeranian	2 months-10 years	18 (F:12, M:6)	18 (100%)	0 (0%)	0 (0%)	36	0/36 = **0.00**	36/36 = **1.00**
Toy Poodle	1-12 years	28 (F:16, M:12)	28 (100%)	0 (0%)	0 (0%)	56	0/56 = **0.00**	56/56 = **1.00**
All breeds (total)		161 (F:95, M:66)	161 (100%)	0 (0%)	0 (0%)	322	0/322 = **0.00**	322/322 = **1.00**

+/+: Homozygous wild-type genotype; +/-: Heterozygous genotype; -/-: Homozygous mutant genotype; n: number of dogs sampled per breed; values in parentheses indicate the female and male ratio within each breed (F: female, M: male)

DISCUSSION

In this study, nt230[del4] in the *ABCB1* gene, known to be associated with drug sensitivity in dogs, was investigated in five different dog breeds commonly found in Türkiye. The finding of the study provides important epidemiological data for population-based pharmacogenetic research.

The clinical significance of the *ABCB1* nt230[del4] mutation has been well-defined in previous studies. [19-23]. Although *ABCB1* polymorphisms have also been investigated in other species, including cats [24], goats [25], pigs [26], and cattle [27], these studies have largely examined gene expression, tissue distribution, or variants whose functional significance has not yet been validated, rather than a single, well-characterized, clinically significant deletion comparable to nt230[del4]. Although *ABCB1*-related drug sensitivity is pharmacologically important across various animal species, the situation in dogs is specific in that a single deletion variant has been identified in numerous breeds, is well-characterized, and is directly linked to clinical toxicity [28]. This suggests that long-term selective breeding and intra-breed genetic homogeneity in dogs may play a significant role in the emergence of clinically meaningful pharmacogenetic variants. Thus, dogs can be considered one of the species in which *ABCB1* variations are most clearly reflected in clinical practice.

The distribution of the *ABCB1* nt230[del4] mutation among dog breeds has been detailed in several population-based studies. For example, in a study, a nonsense mutation caused by a 4 base pair deletion in the *ABCB1* gene was investigated in Border Collie dogs, and ivermectin sensitivity was identified in this breed. The study reported that nt230[del4] in the *ABCB1* may be associated with ivermectin and moxidectin sensitivity [29]. It is known that certain Collie breeds, the Australian Shepherd, the Shetland Sheepdog, and some Sighthound breeds are more susceptible to this mutation. For example, in a study conducted in Australia, the same mutation in *ABCB1* was detected in nine dog breeds, with the most frequently affected breeds being the Collie (Rough Collie: 44.5% heterozygous, 28.4% homozygous; Smooth Collie: 25.1% heterozygous, 28.9% homozygous), Australian Shepherds (43.0% heterozygous, 10.5% homozygous), White Swiss Shepherds (25.3% heterozygous, 0% homozygous), and Shetland Shepherds (23.8% heterozygous, 3.88% homozygous). Among the 32 dogs from 18 non-herding breeds tested, the same mutation was detected in one Cocker Spaniel (1 out of 1 tested, i.e., 100% heterozygous, 0% homozygous) and one Labradoodle (1 out of 4 tested, i.e., 25% heterozygous, 0% homozygous) [30]. In a study conducted on the Border Collie breed in Mexico, the presence and allele frequencies of the *ABCB1* nt230[del4] mutation were examined, and the mutant allele frequency was determined to be 3.3%, with the vast majority of genotype frequencies consisting of the wild-type (wild/wild, 95.2%), while heterozygous (2.9%) and homozygous mutant (1.9%) individuals were reported to be present at lower rates [31].

In a study conducted in Italy, the frequencies of mutant alleles associated with the same 4 base deletion in the *ABCB1* gene were examined in 32 different dog populations (31 breeds and mixed breeds). The study found that the mutated alleles were present in: Rough Collie (mutant allele frequency: 66%), Smooth Collie (mutant allele frequency: 75%), Border Collie (mutant allele frequency: 4%), Bearded Collie (mutant allele frequency: 5%), Shetland Sheepdog (mutant allele frequency: 23%), Australian Shepherd (mutant allele frequency: 35%), White Swiss Shepherd (mutant allele frequency: 7%), Old English Sheepdog (mutant allele frequency: 8%), Whippet (mutant allele frequency: 10%), and in some mixed-breed dogs (mutant allele frequency: 15%). Among these 9 breeds, the highest mutant allele frequencies were observed in the Smooth Collie (75%) and Rough Collie (66%) breeds [32]. This indicates that the distribution of the *ABCB1* mutation is distinctly breed-specific. In particular, high mutant allele frequencies have been reported in herding dog-derived breeds such as the Collie, Australian Shepherd, and Shetland Sheepdog [30,32,33]. In contrast, it has been reported that the mutation frequency is quite low or has not been detected at all in breeds not directly related to the herding dog lineage [34,35].

One of the main reasons for the pronounced genetic differences between dog breeds is the presence of small founder populations, closed pedigrees, selective breeding, and the resulting linkage disequilibrium during the modern breed formation process. Neff et al. [36] reported that the MDR1-1Δ allele was transmitted in an identical by descent manner (inherited from a common ancestor without recombination) from a single common ancestor in the Collie lineage and may have emerged before the genetic isolation of purebred breeds. This explains why the variant is clustered in certain purebred breeds. In contrast, in mixed-breed populations, the allele frequency is expected to dilute due to genetic contribution; however, the risk is not completely eliminated. This is because Marelli et al. [32] and Dekel et al. [34] demonstrated that the mutant allele was also reported in mixed-breed dogs. A study by Donner et al. [37] using extensive screening data also revealed that disease variants can be found in both purebred and mixed-breed dogs, though certain variants are much more concentrated in specific purebred breeds. Reports of this mutation in the breeds studied here are scarce outside Türkiye. GSD has occasionally tested positive at low allelic frequency other countries, 0.6% in Brazil [38] and 2.4% in Israel [34], first documented in a North American cohort [39], whereas GR were negative in the largest available North American screening cohort despite being listed in some reviews [40]. No primary prevalence data were identified for POM, and although TP itself has not been directly studied, the mutant allele has been reported in Poodle-cross dogs in Australia [30]. In this context, the absence of mutant allele in our study, particularly in breeds not directly related to the herding dog lineage such as POM, TP, GR and in the KSD population examined, appears biologically consistent in terms of both breed history and possible hybridization dynamics; however, broader samples with well-defined pedigree information are required to rule out rare alleles.

Studies on *ABCB1* gene mutations in dogs in Türkiye are limited. Most existing studies have been conducted in human populations and generally focus on single nucleotide polymorphisms. This issue indicates a significant lack of information regarding the distribution of the clinically important *ABCB1* nt230[del4] mutation dog population in Türkiye. One of the few studies in this field by Baydan et al. [41], investigated the presence of the *ABCB1* mutation in Kangal Shepherd Dogs and Akbaş Shepherd Dogs and they are found no mutant allele in the examined individuals. Meantime the current study supports these findings, it offers a more comprehensive, population-based assessment due to its relatively larger sample size (n = 161) and inclusion of both indigenous breeds and globally recognized breeds commonly kept in Türkiye. The consistency of the conclusion with previous findings suggests that the *ABCB1* nt230[del4] mutation may have a low prevalence of dog populations in Türkiye.

PCR-RFLP employed in this research constitutes a valuable technique for confirming the accuracy of the findings and reducing expenses. Silvestro et al. [18] evaluated the PCR-RFLP and the PCR-High Resolution Melting (HRM) techniques to genotype a 4 base pair deletion on *ABCB1* gene and proved the PCR-RFLP technique as a dependable, specific and practicable technique. The reliable performance of this technique when applied to a considerably larger sample in this research further demonstrates the practicality of employing PCR-RFLP in field and mass screening situations.

Population-based research is indispensable for the determination of the incidence of the *ABCB1* mutation. According to Beckers et al. [42] the mutation frequency seems to be high in populations that have been selected for genetic testing, but the in the clinical population it is generally quite low. Analogous to the research conducted by Donner et al. [37], the incidence of the *ABCB1* mutation in extensive canine populations is quite low. Therefore, the non-detection of the mutation in our sample is a statistically and epidemiologically foreseeable result.

However, the non-detection of a mutation does not necessarily imply complete extinction of the mutation in the sampled dog populations. Specifically, for certain breeds, the small number of studied dogs could result in the non-detection of rare alleles. Moreover, genetic polymorphisms are also subject to environmental (geographic) variation, different breeding schemes and genetic drift [34,36]. Hence research with more participants and in various areas is required. Several additional limitations of this study should be acknowledged. First, breed identity was established through veterinary clinical/phenotypic assessment combined with owner-reported breed information rather than molecular breed-verification testing, which is consistent with standard practice in routine clinical sampling but cannot formally exclude admixture. Second, pedigree records were not systematically available, and although the KSD samples were drawn from the principal regional KSD breeding facility in Elazığ, which draws breeding stock from across the wider surrounding area rather than a single closed line, the relatedness among individual sampled dogs could not be formally assessed; broader sampling across multiple independent breeding centers

and provinces would further strengthen confidence in the generalizability of these findings. Third, as discussed above, our PCR-RFLP assay was validated against the previously established protocol of Silvestro et al. [18] but was not run alongside a mutant or heterozygous positive control, since no such samples were available within our screened population; this is a methodological limitation common to studies of very-low-prevalence variants and one we recommend addressing in future work.

Clinically, the results indicate that in the studied dog breeds there was a low risk of drug toxicity associated with the *ABCB1* nt230[del4] mutation and might be well used as a safety marker for veterinarians especially when P-gp substrate drugs are considered. However, this mutation on its own does not imply the drug has lost its toxicity. Alterations in the *ABCB1* gene, interactions between drugs, or other mechanisms of drug action may also be involved [43,44].

In conclusion, this study demonstrates that the *ABCB1* nt230[del4] mutation was not detected in dog breeds commonly found in Türkiye, thereby providing significant data for this population. The results support the possibility of a low risk of *ABCB1* related drug sensitivity in these breeds and contribute to the literature in the field of veterinary pharmacogenetics. Further comprehensive studies in the future will help to shed more light on the distribution of this genetic variation.

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Authors' contributions

MTT and AA conceived and drafted the study. MTT, MY and BK performed the experiments. MTT, MY and AA analyzed the data. MTT, MY, BK and AA performed the conceptualization, data curation, formal analysis, and wrote the original draft of the manuscript. All authors have approved the final draft of the manuscript.

Declaration of conflicting interests

The authors declare that they have no conflicts of interest.

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Ethical approval

The protocol of the study was approved by the Fırat University Animal Experiments Local Ethics Committee (Approval Number: 2025/12-11, Date: July 8, 2025). Owners of the dogs provided informed consent prior to participation in the study.

Statement of informed consent

This study involved client-owned and farm-owned dogs collected from veterinary clinics and livestock farms in Türkiye. Informed consent was obtained from the owners or authorized representatives of all animals included in the study prior to sample collection. No individual animal suffered unnecessarily at any stage of the investigation.

ORCID iDs

Meryem Toprak Tuncer https://orcid.org/0000-0002-1978-8227
Merve Yüksel https://orcid.org/0000-0001-5176-3137
Bilgenur Korkut https://orcid.org/0009-0000-7772-7484
Ahmet Ateşşahin https://orcid.org/0000-0002-1004-2146

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RASPROSTRANJENOST MUTACIJE *ABCB1* NT230[DEL4] KOD RAZLIČITIH RASA PASA U TURSKOJ

Meryem TOPRAK TUNCER, Merve YÜKSEL, Bilgenur KORKUT,
Ahmet ATEŞŞAHIN

U ovoj studiji, ispitivano je prisustvo mutacije delecije 4 bazna para (nt230[del4]) u genu *ABCB1* (podfamilija B član 1) ATP-vezujuće kasete kod različitih rasa pasa u Turskoj. U tu svrhu, uzorci krvi prikupljeni su od ukupno 161 psa 5 različitih rasa, uključujući nemačke ovčare (GSD), zlatne retrivere (GR), pomerance (POM), toj pudle (TP) i kangalske ovčare (KSD). Genomska DNK je izolovana iz uzoraka krvi korišćenjem metode ekstrakcije fenol-hloroformom. Zatim, da bi se detektovala mutacija nt230[del4] u genu *ABCB1*, genomska DNK je amplifikovana korišćenjem degenerisanih prajmera putem lančane reakcije polimeraze (PCR) i analizirana korišćenjem metode lančane reakcije polimeraze - polimorfizma dužine restrikcionih fragmenata (PCR-RFLP). Amplifikovani PCR proizvodi su digestirani restrikcionim enzimom, razdvojeni elektroforezom na agaroznom gelu, a genotipovi su određeni na osnovu obrazaca traka. Prema rezultatima analize, utvrđeno je da svi ispitani psi poseduju homozigotni genotip divljeg tipa (+/+) i nije otkriven nijedan mutantni alel. Ovi nalazi ukazuju na to da region gena *ABCB1* pokazuje monomorfnu strukturu u populaciji pasa uključenih u studiju. Dobijeni rezultati mogu doprineti identifikaciji genetskih varijacija koje bi mogle biti značajne u smislu osetljivosti na lekove kod relevantnih rasa pasa. Ovi nalazi sugerišu da varijacije gena *ABCB1* mogu biti ograničene u populaciji pasa u Turskoj, što implicira da genetski rizik u vezi sa osetljivošću na lekove može biti nizak kod relevantnih rasa.