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Systematic Review of Risk Factors for Spontaneous Atrial Fibrillation in Dogs: Insights from 2.36 Million Cases

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ABSTRACT

While numerous investigations in humans have recognized multiple determinants influencing atrial fibrillation (AF) onset, equivalent data concerning dogs remain scarce. The present systematic review aims to identify the primary factors contributing to AF occurrence in canine populations. In compliance with the PRISMA 2020 framework, an extensive search was carried out across the Web of Science and Scopus databases to locate studies documenting naturally developed AF in dogs. The quality of the gathered evidence was evaluated according to the National Institute of Health's Evidence Grading System. Out of an initial pool of 1,043 papers, twenty met the inclusion criteria, representing a total of 2,359,275 dogs, of which 4,807 displayed spontaneous AF. Major determinants for AF onset among dogs with cardiac disorders—particularly myxomatous mitral valve disease (MMVD) and dilated cardiomyopathy (DCM)—included hereditary factors in Irish Wolfhounds, elevated body weight, and enlargement of the left atrium. Differences between MMVD and DCM were identified, such as the influence of congestive heart failure and echocardiographic evidence showing elevated left atrial pressure or right atrial dilation. Additionally, comparisons with humans revealed that age and male sex, strong predictors in people, are unreliable indicators for dogs.

Keywords: Canine, Supraventricular arrhythmia, Heart disease, Electrocardiogram, Echocardiogram

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Introduction

Atrial fibrillation (AF) is the most frequently observed supraventricular rhythm disorder in dogs, with an estimated rate of 0.15% in the general canine population [1]. This percentage differs notably between breeds, from 0.04% in Miniature Poodles to as high as 8.9% in Irish Wolfhounds [1–3]. AF prevalence also varies according to underlying heart disorders, ranging from 2.7% to about 45% in dogs suffering from myxomatous mitral valve disease (MMVD) and dilated cardiomyopathy (DCM), respectively [4–6]. Some animals develop AF without any apparent heart abnormality—defined as primary or lone AF [3, 7]—although, in most cases, the condition develops secondary to structural heart disease that triggers remodeling of the left atrium [3, 7, 8]. Common underlying causes include MMVD, DCM, and advanced left-sided congenital heart defects (CHD) [3]. Among all species, left atrial enlargement (LAE)—or simply, excessive left atrial mass—is widely recognized as the essential substrate for AF formation [3, 9]. Consequently, both LAE and body weight (BW) are regarded as key factors predisposing dogs to AF [10].

In human medicine, AF onset correlates with multiple demographic and clinical elements, including genetic background, age, sex, ethnicity, and the presence of both cardiac (e.g., hypertension, heart failure, coronary artery disease) and systemic disorders (e.g., diabetes, chronic kidney disease) [11–15]. In contrast, the risk profile for AF in dogs remains less clearly defined. Some dogs experience a transient (paroxysmal) AF pattern [16, 17], terminating spontaneously or within seven days of onset, but most cases present as continuous (lasting over seven days), long-term persistent (beyond twelve months), or permanent [3, 11]. Chronic loss of atrial contractions and the typically accelerated ventricular rate reduce ventricular filling, lower cardiac output, and elevate left ventricular filling pressures. Over time, these changes cause myocardial remodeling (tachycardia-induced cardiomyopathy) [3]. Consequently, AF is linked to a higher probability of cardiac-related death, especially due to congestive heart failure (CHF) or sudden cardiac arrest [3, 18–20].

Given the poor outcomes associated with AF in dogs suffering from heart disease, understanding its contributing elements is essential. This review, therefore, compiles and assesses available data on AF risk factors in dogs and contrasts them with findings from human research.

Materials and Methods

Stage 1—search strategy

This systematic review was conducted under the PRISMA 2020 guidelines [21]. The investigation targeted peer-reviewed publications describing spontaneous AF cases in dogs. Relevant studies were retrieved from Scopus and Web of Science, encompassing all records published through October 2023.

The search utilized the following keyword combination and Boolean operators: “atrial fibrillation” OR “supraventricular arrhythmia” AND “dog” OR “canine” AND “risk factor” OR “predictor” OR “susceptibility” OR “cause” OR “influence” OR “prevalence” OR “incidence.”

All search results were organized in Microsoft Excel, including author names, titles, journal information, publication years, volumes, issues, and page numbers. Duplicates and papers written in languages other than English were excluded.

Stage 2—screening

During the preliminary screening, two reviewers (GA and CP) independently examined all retrieved papers based on their titles and abstracts. The goal of this step was to exclude incomplete submissions (those presenting only abstracts), non-veterinary or non-clinical studies (including experimentally induced AF in dogs), review papers, and works related to species other than dogs. Whenever discrepancies arose between the two reviewers, a third evaluator (CG) was consulted to reach an agreement.

Stage 3—eligibility

Following the initial screening, studies considered potentially relevant were independently reviewed by GA and CP through detailed examination of the title, abstract, and complete text. Moreover, the reference lists of all selected studies were reviewed to locate additional papers not retrieved in the first search. These newly identified sources were also evaluated for inclusion. Both reviewers carefully analyzed each full-text document to verify its compliance with the selection criteria. Disagreements were resolved through discussion, and in case of continued uncertainty, the third reviewer (CG) was involved.

Studies included in the final review met the following criteria:

• Peer-reviewed English-language publications addressing canine AF, regardless of arrhythmia type (paroxysmal, persistent, or permanent).

• Original research papers—such as case series, observational cohort, cross-sectional, case-control, or randomized controlled studies. Review papers and single-case reports were not accepted.

• Reports including dogs diagnosed with AF and/or conditions predisposing to AF development.

Quality assessment

To determine the reliability of the full-text papers, the Evidence Quality Grading System introduced in 2013 by the National Heart, Lung, and Blood Institute (NIH) was used [available at https://www.nhlbi.nih.gov/health-topics/study-quality-assessment-tools, accessed 17 October 2023]. Quality evaluations were performed independently by GA and CP, and any disagreements were clarified by CG.

This tool evaluates study strength for multiple research types (e.g., cohort, cross-sectional, or case series) through a questionnaire of at least 12 items, each scored as "Yes," "No," or "Other" (meaning not applicable, unclear, or not reported). Studies were categorized as follows:

• High quality (Good): All criteria marked "Yes."
• Moderate quality (Fair): Most criteria marked "Yes."
• Low quality (Poor): Most criteria marked "No" or "Other."

Results and Discussion

Identification and selection of relevant articles

An outline of the search process is provided in **Figure 1**.

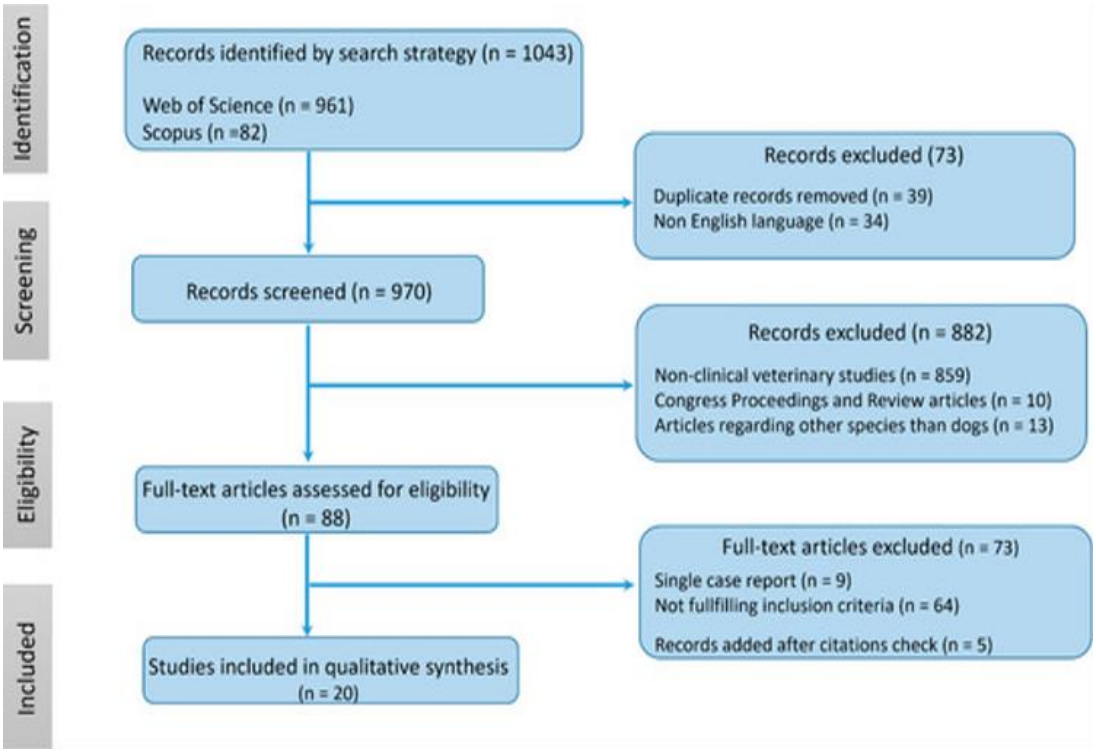

Figure 1. Flow diagram summarizing the literature selection process.

Overall, 1,043 papers were retrieved—82 from Scopus and 961 from Web of Science. Duplicates (n = 39) and non-English publications (n = 34) were eliminated, leaving 970 records. Of these, 882 were removed for the following reasons: non-clinical or non-veterinary content (n = 859), incomplete format or review type (n = 10), and research involving non-canine species (n = 13). After assessing 88 full-text studies for inclusion, 73 were excluded because they consisted of single-case reports or did not describe conditions contributing to AF. An additional five papers were incorporated from citation searches that had not appeared in the initial query. Thus, the final systematic review comprised 20 studies involving 2,359,275 dogs, among which 4,807 presented spontaneous AF.

For each included study, detailed information was systematically recorded in Microsoft Excel, including:

- Publication information: First author and year.
- Study data: Type of study, total number of dogs (including controls, if present), breed composition, underlying cardiac diseases, proportion of AF and CHF cases, and overall quality grade.
- Main outcomes: Reported risk determinants.

Study characteristics and quality assessment

A summary of the included studies and their quality ratings is displayed in **Table 1**. Among all, 11 (55%) were retrospective case-control studies, 1 (5%) was retrospective cross-sectional, 2 (10%) retrospective cohort, 3 (15%) retrospective observational, and 3 (15%) case series. Publication dates ranged from 1971 to 2023, with most

papers (14 of 20; 70%) released after 2016. Fourteen studies (70%) analyzed multiple breeds, while three (15%) were restricted to a single breed—Irish Wolfhound, Dogue de Bordeaux, or Dobermann Pinscher (one each). One paper (5%) did not specify the breeds included.

Table 1. Summary of the 20 studies analyzed in this systematic review addressing AF risk factors in dogs.

First Author (Reference)	Year	Study Design	Total Sample Size	Breed	Heart Condition	AF Prevalence (%)	CHF Prevalence (%)	Identified Risk Factors	Quality Assessment
Bolton GR [16]	1971	Case Series	5	Mixed	Congenital Heart Disease, Myxomatous Mitral Valve Disease	100	80	Sex; Body Weight	Poor
Bohn FK [22]	1971	Retrospective Observational	877	Mixed	Myxomatous Mitral Valve Disease, Congenital Heart Disease, Other Heart Disease	6.3	90.9	Sex; Age; Breed	Poor
Boevé MH [23]	1984	Cohort	59	Mixed	Congenital Heart Disease, Non-Specific Heart Disease	100	100	Breed; Sex	Poor
Bonagura JD [24]	1986	Case Series	81	Mixed	Dilated Cardiomyopathy, Myxomatous Mitral Valve Disease	100	Not Reported	Breed; Sex; Age	Poor
Guglielmini, C [8]	2000	Case-Control	205	Not Reported	Congenital Heart Disease, Myxomatous Mitral Valve Disease, Dilated Cardiomyopathy	24.4	Not Reported	Body Weight; Left Atrial Enlargement (Left Atrial Dimension)	Fair
Westling, J [1]	2008	Retrospective Observational	2,352,633	Mixed	Non-Specific Heart Disease	0.15	Not Reported	Large Breed; Sex	Poor
Vazquez, DMP [17]	2016	Case Series	7	Mixed	Myxomatous Mitral Valve Disease, Dilated Cardiomyop	100	57.1	Neurally Mediated Syncope	Fair

					athy, Congenital Heart Disease				
Jung, SW [18]	2016	Case-Control	64	Mixed	Myxomatous Mitral Valve Disease	51.5	100	Body Weight	Fair
Noszczyk-Nowak, A [25]	2017	Retrospective Observational	1189	Mixed	Cardiological Referrals	13.4	Not Reported	Body Weight; Age; Sex	Fair
McAulay, G [26]	2018	Case-Control	64	Doberman Pinscher	Congenital Heart Disease, Cardiomyopathy, Non-Cardiomyopathy	39	Not Reported	Heart Rate; Left Atrial Enlargement (LA:Ao); Left Ventricular Enlargement; Fractional Shortening; Right Atrial/Right Ventricular Enlargement	Fair
Neves, J [27]	2018	Case-Control	42	Mixed	Congenital Heart Disease, Myxomatous Mitral Valve Disease, Dilated Cardiomyopathy	50	Not Reported	Pulmonary Artery Tissue Doppler Imaging	Fair
Vollmar, C [20]	2019	Case-Control	104	Irish Wolfhound	Asymptomatic Dogs	50	36.5	Left Atrial Enlargement (Left Atrial Dimension)	Fair
Ward, J [28]	2019	Case-Control	220	Mixed	Dilated Cardiomyopathy, Myxomatous Mitral Valve Disease	27.7	100	Heart Rate; Body Weight	Fair
Fousse, LS [29]	2019	Case-Control	463	Irish Wolfhound	Non-Specific Heart Disease	70.6	Not Reported	Genetics	Fair
Friederich, J [30]	2020	Case-Control	48	Doberman Pinscher	Dilated Cardiomyopathy	47.9	100	Right Atrial Enlargement	Fair
Tyrrell, WD [2]	2020	Cohort	618	Irish Wolfhound	Dilated Cardiomyopathy	8.9	Not Reported	Age; Left Atrial	Fair

								Enlargement	
Baron Toaldo, M [31]	2020	Case-Control	44	Mixed	Myxomatous Mitral Valve Disease	50	77.2	Peak Atrial Longitudinal Strain	Fair
Guglielmini, C [4]	2020	Cross-Sectional	2194	Mixed	Myxomatous Mitral Valve Disease	2.7	89.8	Body Weight; Left Atrial Enlargement (Left Atrial Dimension, LA:Ao); Peak E-wave Velocity; Fractional Shortening; Congestive Heart Failure	Fair
Borgeat, K [32]	2021	Case-Control	269	Mixed	Congenital Heart Disease, Myxomatous Mitral Valve Disease, Dilated Cardiomyopathy, Arrhythmogenic Right Ventricular Cardiomyopathy	52.7	66.1	Body Weight; Congestive Heart Failure	Fair
Guglielmini, C [5]	2023	Case-Control	89	Mixed	Dilated Cardiomyopathy	43.8	94.8	Left Atrial Enlargement (Left Atrial Dimension); Right Atrial Enlargement	Fair

Abbreviations: BW = body weight; C = cohort study; CC = case–control study; CHD = congenital heart disease; CHF = congestive heart failure; Cm = cardiac mass; CaS = case series; CrS = cross-sectional study; DbB = Dogue de Bordeaux; DCM = dilated cardiomyopathy; DP = Dobermann Pinscher; E max = mitral E-wave peak velocity; FS = fractional shortening; IW = Irish Wolfhound; LA: Ao = ratio of left atrial to aortic diameter; LAD = left atrial diameter; LAE = left atrial enlargement; LVE = left ventricular enlargement; MMVD = myxomatous mitral valve disease; NCm = non-cardiac mass; NR = not reported; NSHD = non-specified heart disease; OHD = other heart disease; PALS = peak atrial longitudinal strain; PA-TDI = period from P-wave onset on ECG to A' wave peak; RAE = right atrial enlargement; R-OB = retrospective observational study; RVE = right ventricular enlargement.

The predominant cardiac disorders recorded were CHD (8/20 studies, 40%), MMVD (11/20 studies, 55%), DCM (9/20 studies, 45%), and arrhythmogenic right ventricular cardiomyopathy (1/20, 5%). Four publications (20%) did not clearly specify the type of cardiac abnormality involved.

No high-quality research was detected in the final group of studies. Nevertheless, 15 of them (75%) presented a moderate reporting standard, while 5 (25%) were rated as low quality. The typical issues reducing study quality were insufficient sample size rationale, poor methodological description, and incomplete data reporting.

Risk factor outcomes

A summary of all identified AF-related risk factors is shown in **Table 1**. Factors linked to AF onset included both clinical and echocardiographic characteristics. Commonly cited risk determinants across the reviewed works were sex, age, BW, breed, and LAE, with the latter assessed through absolute LAD and the LA: Ao ratio. Other contributing factors included increased heart rate at admission, LVE (noted by larger diastolic and systolic diameters adjusted for BW), RAE, RVE, the presence of CHF, reduced FS, and elevated E max. Advanced echocardiographic modalities such as TDI and speckle-tracking echocardiography (STE) provided additional markers—namely, longer PA-TDI intervals and lower PALS values. Genetic predisposition and neurally mediated syncope episodes were also implicated, highlighting the multifactorial origin of AF.

Risk direction

Among the examined clinical parameters, age was identified as a risk factor in four studies (20%). Male dogs were at increased risk in six papers (30%). Breed and BW were evaluated in 11 reports (55%), showing that large and giant breeds with higher BW have a greater likelihood of developing AF. Elevated heart rate at first presentation and CHF each appeared in two papers (10%). A single study (5%) identified genetic susceptibility—particularly among Irish Wolfhounds—and another (5%) linked neurally mediated syncope. Regarding echocardiographic findings, LAE appeared in six papers (30%), RAE in three (15%), reduced FS in two (10%), while RVE, LVE, increased E max, increased PA-TDI, and decreased PALS were each reported once (5%).

Due to the retrospective nature of all included research, statistical strength was frequently limited; many relied solely on comparisons between AF and non-AF cases or lacked formal analysis, especially in case series. Only seven studies (35%) used statistical methods like univariate or multivariate logistic regression to determine independent predictors of AF. Four of those (20%) specifically focused on evaluating both clinical and echocardiographic variables to estimate odds ratios for AF development in dogs affected by MMVD or DCM. Additionally, one study assessed AF heritability in Irish Wolfhounds to clarify genetic influence in that breed.

Table 2 compares the clinical risk factors for AF between humans and dogs with MMVD or DCM. Unlike in people, age and sex were not determining factors for canine AF, whereas elevated BW and the existence of CHF were shared risk indicators for both species in MMVD cases, but not in DCM.

Table 2. Comparison of AF risk factors between humans [11] and dogs affected by myxomatous mitral valve disease (MMVD) [4] or dilated cardiomyopathy (DCM) [5].

Risk Factor	Humans	Dogs (Overall)	MMVD	DCM
Genetics	Yes	NA	Yes (Irish Wolfhound)	Yes (Irish Wolfhound)
Age	Yes	No	No	No
Male sex	Yes	No	No	No
Lifestyle[1]	Yes	NA	NA	NA
Obesity	Yes	NA	NA	NA
Body weight	Yes	Yes	Yes	No
Concurrent non-cardiac diseases[2]	Yes	NA	NA	NA
Congestive heart failure	Yes	Yes	Yes	No

1 Physical activity, alcohol intake, smoking; 2 Diabetes, chronic kidney disease, inflammatory conditions, COPD, and obstructive sleep apnea; NA = not assessed/applicable; IWV = Irish Wolfhound.

Table 3 lists the echocardiographic parameters linked to atrial fibrillation (AF) in dogs with MMVD and DCM, along with the corresponding odds ratios (ORs). A higher absolute value of LAD was identified as a notable indicator of AF in both MMVD (OR 5.28) and DCM (OR 3.58). Meanwhile, a relative enlargement of the left atrium—represented by the LA: Ao ratio—was associated with AF risk solely in dogs with MMVD (OR 14). Elevated E max and reduced FS (OR 2.2 and 0.91, respectively), together with right atrial enlargement (RAE) (OR 4.02), were also found to increase AF likelihood, though their influence differed depending on the cardiac condition.

Table 3. Echocardiographic indicators of atrial fibrillation and related odds ratios (OR) in dogs diagnosed with myxomatous mitral valve disease (MMVD) [4] or dilated cardiomyopathy (DCM) [5, 30].

Risk Factor	MMVD	OR (MMVD)	DCM	OR (DCM)
Left atrial diameter	Yes	5.28	Yes	3.58
Left atrial diameter to aortic diameter ratio	Yes	14	No	-
Right atrial enlargement	Not Evaluated	-	Yes	4.02
Peak velocity of mitral E wave	Yes	2.2	No	-
Fractional shortening	Yes	0.91	No	-

NE: not examined.

Table 4 details additional echocardiographic factors connected with AF occurrence, reporting cut-off thresholds, sensitivity, specificity, and diagnostic reliability based on the area under the receiver operating characteristic curve (AUC). Among dogs suffering from CHD, MMVD, or DCM, a PA-TDI value of 81.2 ms served as a strong predictor for AF onset (AUC = 0.896). In dogs with MMVD, the most accurate discriminators were LAD (AUC = 0.979), LA: Ao ratio (AUC = 0.931), and E max (AUC = 0.900), using cut-offs of >3.45 cm, >1.8, and >102 cm/s, respectively. For dogs with DCM, LAD values exceeding 4.66 cm produced the greatest predictive ability (AUC = 0.816). Given the distinct rates of AF between MMVD and DCM populations, LAD > 3.45 cm and >4.66 cm corresponded to positive predictive values of 20.7% and 67.5%, respectively.

Table 4. Diagnostic reliability, sensitivity (Se), and specificity (Sp) of echocardiographic and clinical indicators predicting AF development in dogs with myxomatous mitral valve disease (MMVD) or dilated cardiomyopathy (DCM).

Cardiac Disease	Cut-Off	AUC	Sensitivity (%)	Specificity (%)	Reference
Body weight (kg)					
MMVD	7.6	0.735	96.6	44.4	[4]
DCM	>36	0.740	79	58	[5]
LAD (cm)					
MMVD	>3.45	0.979	98.3	89.8	[4]
DCM	>4.66	0.816	90	66	[5]
LA:Ao					
MMVD	>1.8	0.931	98.3	78.5	[4]
DCM	>1.73	0.637	95	38	[5]
Ao					
DCM	>2.3	0.686	85	50	[5]
LVDDn					
MMVD	>1.82	0.854	81.4	82.2	[4]
LVSDn					
MMVD	>1.08	0.875	79.7	86.7	[4]
FS (%)					
MMVD	≤40.1	0.682	70.7	60.1	[4]
E max (cm/s)					
MMVD	>102	0.900	91.1	79.0	[4]
DCM	>79	0.647	89	42	[5]
PA-TDI (ms)					
CHD, MMVD, DCM	81.2	0.896	81	90.5	[27]

PALS (%)					
MMVD	≤28	0.721	80	65	[31]

Abbreviations: Ao = aortic diameter; AUC = area under the curve; E max = peak mitral E-wave velocity; FS = fractional shortening; LA: Ao = left atrial-to-aortic diameter ratio; LAD = left atrial diameter; LVDDn = left ventricular diastolic diameter normalized by body weight; LVSDn = left ventricular systolic diameter normalized by body weight; PALS = peak atrial longitudinal strain; PA-TDI = interval from P-wave onset on ECG to A' wave peak.

This review assessed 20 publications covering 2,359,275 dogs, of which 4807 were diagnosed with AF. None of the studies achieved a "high" quality designation. The majority—mainly those issued after 2016—were rated as "moderate," while earlier works, especially from the 1970s and 1980s, were classed as "low" due to weaker methodological standards.

The concept of "lone" or primary AF, which is increasingly debated in human cardiology [11], continues to appear in veterinary literature to describe AF occurring without any identifiable heart condition [7]. This form appears infrequently and mainly in large or giant dog breeds, particularly the Irish Wolfhound, which shows a hereditary tendency toward the disorder [29]. Some dogs may experience paroxysmal AF presumed to be neurally mediated, often following syncopal episodes [17]. Nonetheless, the majority of canine AF cases are chronic or permanent and arise secondary to structural cardiac abnormalities such as DCM, MMVD, or CHD—collectively defined as secondary AF [7].

In humans, numerous investigations have examined the diverse contributors to AF [11–15], encompassing demographic (age, sex, ethnicity), lifestyle (smoking, alcohol intake, exercise), systemic (body height, hypertension, obesity, diabetes, kidney impairment), cardiovascular (heart failure, valvular disorders, coronary or congenital heart disease), and genetic aspects [11–15]. The current review supports that certain determinants established in humans—especially structural cardiac issues like valvular disease—are also relevant to dogs. Conversely, other human-related risk factors such as behavioral or systemic characteristics (e.g., tobacco use, alcohol consumption, or body size) are either non-applicable or insufficiently studied within canine populations [4, 5].

Demographic factors

When comparing demographic influences on atrial fibrillation (AF) between people and dogs, both shared trends and unique distinctions become evident. In humans, AF onset is strongly associated with hereditary background, increasing age, male gender, and Caucasian origin [11]. In contrast, canine studies repeatedly note that AF occurs more frequently among large-sized breeds [1, 5, 6, 9–11, 16, 18–24]. A notable example is the Irish Wolfhound, where AF shows high heritability and likely follows a dominant inheritance pattern [29]. However, comparable genetic investigations in other frequently affected breeds have not yet been carried out. The higher AF incidence observed in larger dogs is thought to arise mainly from their predisposition to dilated cardiomyopathy (DCM), rather than from an isolated genetic vulnerability to AF [3, 5].

With respect to sex, early reports—mostly without proper statistical testing—proposed that male dogs might have an increased likelihood of AF [1, 8, 16, 17, 24, 25]. Yet, modern analyses using multivariable logistic regression contradict this idea, indicating that, unlike humans, male dogs do not show a sex-based risk for AF [4, 5, 30, 31]. The higher male incidence reported in older literature is now considered a reflection of their greater risk for acquired cardiac problems such as myxomatous mitral valve disease (MMVD) and DCM [6, 33]. Similarly, the role of age remains debatable. While older studies observed a connection between advancing age and AF [2, 22, 24, 25], newer multivariate models found no significant link between age and AF in dogs with either MMVD or DCM [4, 5, 31]. In people, aging and associated disorders often lead to atrial remodeling [34]; however, such age-dependent remodeling has not yet been clearly demonstrated in dogs. Evidence definitively linking older age to AF development in canines is still lacking.

Body weight and atrial size

Increased body weight (BW) has emerged as a notable risk determinant for AF across both species, pointing to a shared pathogenic mechanism [4, 8, 11, 16, 18, 22–25, 28]. This relationship is particularly strong in MMVD-affected dogs [4], even though MMVD primarily affects small breeds [33]. Among these smaller dogs, individuals weighing more than 20 kg show a 5.8-fold higher probability of developing AF [35]. On the other hand, within populations affected by DCM—a disease typical of large or giant dogs—recent research indicates that BW loses

its independent predictive role once confounding factors are adjusted for via multivariate analysis [5]. Therefore, although BW serves as a general indicator of AF susceptibility in dogs with cardiac disorders overall [28], its specific predictive value becomes negligible when examining only DCM cases, where most dogs already have substantial body mass.

Closely tied to BW, left atrial size is another strong factor influencing AF development in both humans [11] and animals [3, 8, 9]. The left atrium and pulmonary veins act as major sites for the initiation and persistence of AF in human patients [34]. Enlargement of the left atrium, a clear sign of atrial remodeling due to cardiac stress, has been reported in five of the studies analyzed [2, 4, 5, 8, 26]. Using two-dimensional echocardiography, clinicians can evaluate this through absolute left atrial diameter (LAD) or the left atrium-to-aorta (LA: Ao) ratio [36]. When predicting AF in dogs with MMVD or DCM, LAD proves to be the most effective single measurement, with established thresholds of >3.45 cm for MMVD and >4.66 cm for DCM [4, 5]. These measurements outperform LA: Ao in both sensitivity and specificity, as reflected by higher diagnostic accuracy (AUC 0.979 and 0.816 for MMVD and DCM, respectively) compared with LA: Ao ratios (AUC 0.931 and 0.637) [4, 5]. Hence, absolute LAD measurement offers a more practical predictive parameter than LA: Ao, which many echocardiographers still prefer [36]. However, this impressive diagnostic performance must be interpreted considering the far greater AF prevalence in dogs with DCM [5, 6] than in those with MMVD [4]. For example, around one in five dogs with MMVD and LAD > 3.45 cm will develop AF, compared to roughly two in three DCM dogs with LAD > 4.66 cm [4, 5]. Similarly, in human cases of embolic stroke of undetermined source, patients with LAD above 4.0 cm are twice as likely to experience paroxysmal AF compared to those with LAD ≤ 4.0 cm [37].

Echocardiographic predictors

Several additional echocardiographic markers independently predict AF onset in dogs. Increased mitral E max and decreased fractional shortening (FS) are strong indicators in MMVD [4], while right atrial enlargement serves as a predictive feature in DCM [5, 30]. Elevated mitral E max reflects higher left atrial pressure [38], corresponding with progressive atrial dilation and structural remodeling that promote AF persistence [39]. Increased mitral E max also signifies poorer prognosis in dogs suffering from MMVD [40–44]. Similarly, right atrial enlargement in DCM-affected dogs correlates with AF occurrence [5, 30], paralleling trends observed in human patients with heart failure and preserved ejection fraction [45]. Canines with heart disease and AF are more likely to show signs of right-sided congestive heart failure compared with those without AF [28]. Despite intense focus on left atrial morphology and function, right atrial structural changes and dysfunction remain insufficiently studied in both humans and dogs [28, 46]. Further research on this topic is essential. Other potentially meaningful predictors include increased left ventricular diameters and reduced peak atrial longitudinal strain (PALS) in MMVD, and elevated PA-TDI values in dogs with left-sided heart disease [4, 27, 31]. Notably, a PA-TDI threshold of 81.2 ms demonstrated reliable predictive accuracy for AF occurrence [27]. In human medicine, tissue Doppler imaging (TDI) and speckle-tracking echocardiography (STE) are already used to assess atrial performance [47, 48]. In dogs, reduced PALS values have similarly been linked to higher cardiac mortality in MMVD cases [31].

Heart rate and congestive heart failure (CHF)

The influence of heart rate and CHF on secondary atrial fibrillation (AF) in dogs remains a matter of ongoing debate. Elevated heart rate is a frequent observation among canines with AF and concurrent heart disease, largely reflecting enhanced sympathetic activation — a physiological result of progressive cardiac dysfunction [4, 5, 26, 28, 31]. Nonetheless, sustained tachycardia associated with AF may itself precipitate tachycardia-induced cardiomyopathy [2, 49]. Clarifying the bidirectional link between AF and heart rate poses difficulties in both veterinary and human medicine, as each can act as either a cause or a consequence of the other. Increased heart rate is commonly documented as a clinical feature in subjects with AF [3, 11]. Within the reviewed literature, two studies [26, 28] identified elevated heart rate upon presentation as a risk indicator for AF occurrence, one reporting an odds ratio (OR) of 1.123 [28], underscoring the variable's predictive value.

High heart rate also carries prognostic significance in dogs already diagnosed with AF, serving as an unfavorable prognostic element. Canines exhibiting rates below 125 bpm on 24-hour Holter monitoring display extended survival compared to those exceeding this threshold [19, 50]. Similarly, an elevated heart rate determined during echocardiographic assessment independently predicts adverse outcomes in AF cases associated with MMVD or DCM [51]. In addition, CHF has been recognized as an independent determinant of AF onset in dogs affected by

MMVD [4], echoing trends seen in human patients with cardiac disease [11]; however, such a relationship was not demonstrated in DCM cases [5]. Further, a multivariable logistic regression model incorporating LAD and FS revealed that dogs with current or previous decompensated MMVD possessed approximately a fivefold higher probability of developing AF than those with compensated MMVD [4].

The concurrent occurrence of AF and CHF frequently appears in both canine and human cardiac disorders [4, 5, 28, 52, 53]. Yet, determining the precise causal direction remains complex, given their intertwined pathophysiology and overlapping progression, especially in dogs.

Study limitations

Despite offering important insights, this systematic review is subject to certain constraints. First, comparing heterogeneous study populations is inherently challenging—particularly when contrasting breed-specific cohorts with mixed-breed samples. Such diversity introduces genetic and physiological variability that complicates interpretation. Second, the broad publication range (1971–2023) brings potential inconsistencies due to evolving diagnostic tools and research approaches. Earlier works, notably from older case series lacking rigorous statistical assessment, further emphasize the need for careful contextual evaluation.

Because all included studies employed retrospective designs, they could only highlight associations rather than prove causation. Moreover, none achieved a "high-quality" rating, which necessitates cautious consideration when applying these findings clinically. While the risk factors identified remain informative, their strength should be interpreted in light of study quality. To clarify cause-and-effect relationships, future prospective longitudinal studies are strongly encouraged.

Additionally, although Boolean operators "AND" and "OR" were systematically used across all searchable fields (titles, abstracts, and full texts) to ensure comprehensive retrieval, some relevant studies may still have been missed. Possible reasons include the absence of certain papers in the databases examined. Recognizing these gaps reinforces the need for continuous refinement of search strategies and further research efforts.

Conclusion

Atrial fibrillation is a common complication among dogs with left-sided heart disease, especially those suffering from DCM. The results of this review have practical importance for veterinary clinicians. Identifying and understanding specific risk factors provides a framework for anticipating AF onset in affected dogs. The main determinants include high body weight (BW) and left atrial enlargement (LAE). Importantly, the pattern of risk differs between MMVD and DCM, demonstrating disease-specific AF mechanisms. The presence of CHF notably increases risk in MMVD, paralleling human findings. Conversely, factors commonly predictive in humans—such as male sex or advancing age—do not reliably forecast AF in dogs.

Absolute LAD measurement remains the most precise echocardiographic indicator for AF prediction. Moreover, applying advanced imaging modalities, including tissue Doppler imaging (TDI) and speckle-tracking echocardiography (STE), could enhance early identification of at-risk dogs. Because most current research is retrospective, well-controlled prospective studies are essential. Future investigations should incorporate additional parameters, such as electrocardiographic indices, to refine predictive accuracy and deepen our comprehension of AF pathogenesis in the canine population.

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References

1. Westling J, Westling W, Pyle RL. Epidemiology of Atrial Fibrillation in the Dog. J Appl Res Vet Med. 2008;6(3):151–4.

2. Tyrrell WD Jr, Abbott JA, Rosenthal SL, Dentino M, Abrams F. Echocardiographic and electrocardiographic evaluation of North American Irish Wolfhounds. J Vet Intern Med. 2020;34(2):581–90.
3. Pedro B, Fontes-Sousa AP, Gelzer AR. Canine atrial fibrillation: Pathophysiology, epidemiology and classification. Vet J. 2020;265(6):105548.
4. Guglielmini C, Goncalves Sousa M, Baron Toaldo M, Valente C, Bentivoglio V, Mazzoldi C, et al. Prevalence and risk factors for atrial fibrillation in dogs with myxomatous mitral valve disease. J Vet Intern Med. 2020;34(6):2223–31.
5. Guglielmini C, Valente C, Romito G, Mazzoldi C, Baron Toaldo M, Goncalves Sousa M, et al. Risk factors for atrial fibrillation in dogs with dilated cardiomyopathy. Front Vet Sci. 2023;10: 1183689.
6. Martin MWS, Stafford Johnson MJ, Celona B. Canine dilated cardiomyopathy: A retrospective study of signalment, presentation and clinical findings in 369 cases. J Small Anim Pract. 2009;50(1):23–9.
7. Menaut P, Bélanger MC, Beauchamp G, Ponzio NM, Moïse NS. Atrial fibrillation in dogs with and without structural or functional cardiac disease: A retrospective study of 109 cases. J Vet Cardiol. 2005;7(2):75–83.
8. Guglielmini C, Chetboul V, Pietra M, Pouchelon JL, Capucci A, Cipone M. Influence of Left Atrial Enlargement and Body Weight on the Development of Atrial Fibrillation: Retrospective Study on 205 Dogs. Vet J. 2000;160(2):235–41.
9. Decloedt A, Van Steenkiste G, Vera L, Buhl R, Van Loon G. Atrial fibrillation in horses part 1: Pathophysiology. Vet J. 2020;263:105521.
10. Bizhanov KA, Abzaliyev KB, Baimbetov AK, Sarsenbayeva AB, Lyan E. Atrial fibrillation: Epidemiology, pathophysiology, and clinical complications (literature review). J Cardiovasc Electrophysiol. 2023;34(1):153–65.
11. Hindricks G, Potpara T, Dagres N, Arbelo E, Bax JJ, Blomström-Lundqvist C, et al. 2020 ESC Guidelines for the diagnosis and management of atrial fibrillation developed in collaboration with the European Association for Cardio-Thoracic Surgery (EACTS). Eur Heart J. 2021;42(5):373–498.
12. Benjamin EJ, Muntner P, Alonso A, Bittencourt MS, Callaway CW, Carson A P, et al. Heart Disease and Stroke Statistics-2019 Update: A Report From the American Heart Association. Circulation. 2019;139(10):e56–e528.
13. Allan V, Honarbakhsh S, Casas JP, Wallace J, Hunter R, Schilling R, et al. Are cardiovascular risk factors also associated with the incidence of atrial fibrillation? A systematic review and field synopsis of 23 factors in 32 population-based cohorts of 20 million participants. Thromb Haemost. 2017;117(5):837–50.
14. Kirchhof P, Lip GYH, Van Gelder IC, Bax J, Hylek E, Kaab S, et al. Comprehensive risk reduction in patients with atrial fibrillation: Emerging diagnostic and therapeutic options—A report from the 3rd Atrial Fibrillation Competence NETwork/European Heart Rhythm Association consensus conference. Eur Pacing Arrhythm Electrophysiol. 2012;14(1):8–27.
15. Joglar JA, Chung MK, Armbruster AL, Benjamin EJ, Chyou JY, Cronin EM, et al. 2023 ACC/AHA/ACCP/HRS Guideline for the Diagnosis and Management of Atrial Fibrillation: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. J Am Coll Cardiol. 2024;83(1):109–279.
16. Bolton GR. Paroxysmal atrial fibrillation in the dog. J Am Vet Med Assoc. 1971;158(1):64–76.
17. Porteiro Vázquez DM, Perego M, Santos L, Gerou-Ferriani M, Martin MWS, Santilli RA. Paroxysmal atrial fibrillation in seven dogs with presumed neurally-mediated syncope. J Vet Cardiol. 2016;18(1):1–9.
18. Jung SW, Sun W, Griffiths LG, Kittleson MD. Atrial Fibrillation as a Prognostic Indicator in Medium to Large-Sized Dogs with Myxomatous Mitral Valvular Degeneration and Congestive Heart Failure. J Vet Intern Med. 2016;30(1):51–7.
19. Pedro B, Dukes-McEwan J, Oyama MA, Kraus MS, Gelzer AR. Retrospective Evaluation of the Effect of Heart Rate on Survival in Dogs with Atrial Fibrillation. J Vet Intern Med. 2018;32(1):86–92.
20. Vollmar C, Vollmar A, Keene B, Fox PR, Reese S, Kohn B. Irish Wolfhounds with subclinical atrial fibrillation: Progression of disease and causes of death. J Vet Cardiol. 2019;24(1):48–57.
21. Page MJ, Moher D, Bossuyt PM, Boutron I, Hoffmann TC, Mulrow C D, et al. PRISMA 2020 explanation and elaboration: Updated guidance and exemplars for reporting systematic reviews. BMJ. 2021;372:n160.
22. Bohn FK, Patterson DF, Pyle RL. Atrial fibrillation in dogs. Br Vet J. 1971;127(6):485–96.
23. Boevè MH, Stokhof AA, Van Den Brom WE. Prognostic significance of the electrocardiogram in dogs with atrial fibrillation: A retrospective study of 59 cases. Res Vet Sci. 1984;36(1):32–6.

24. Bonagura JD, Ware WA. Atrial fibrillation in the dog: Clinical findings in 81 cases. J Am Anim Hosp Assoc. 1986;22(1):111–20.
25. Noszczyk-Nowak A, Michałek M, Kałuża E, Cepiel A, Pasławska U. Prevalence of Arrhythmias in Dogs Examined between 2008 and 2014. J Vet Res. 2017;61(1):103–10.
26. McAulay G, Borgeat K, Sargent J, Mõtsküla P, Neves J, Dukes-McEwan J, et al. Phenotypic description of cardiac findings in a population of Dogue de Bordeaux with an emphasis on atrial fibrillation. Vet J. 2018;234(February):111–8.
27. Neves J, Pedro B, Christley R, Dukes-McEwan J. Usefulness of pulsed-wave tissue Doppler imaging at the mitral annulus for prediction of new-onset atrial fibrillation in dogs. J Vet Cardiol. 2018;20(4):425–37.
28. Ward J, Ware W, Viall A. Association between atrial fibrillation and right-sided manifestations of congestive heart failure in dogs with degenerative mitral valve disease or dilated cardiomyopathy. J Vet Cardiol. 2019;21(1):18–27.
29. Fousse SL, Tyrrell WD, Dentino ME, Abrams FL, Rosenthal SL, Stern JA. Pedigree analysis of atrial fibrillation in Irish wolfhounds supports a high heritability with a dominant mode of inheritance. Canine Genet Epidemiol. 2019;6(1):11.
30. Friederich J, Seuß AC, Wess G. The role of atrial fibrillation as a prognostic factor in doberman pinschers with dilated cardiomyopathy and congestive heart failure. Vet J. 2020;264(1):105535.
31. Baron Toaldo M, Mazzoldi C, Romito G, Poser H, Contiero B, Cipone M, et al. Echocardiographic predictors of first onset of atrial fibrillation in dogs with myxomatous mitral valve disease. J Vet Intern Med. 2020;34(4):1787–93.
32. Borgeat K, Pack M, Harris J, Laver A, Seo J, Belachsen O, et al. Prevalence of sudden cardiac death in dogs with atrial fibrillation. J Vet Intern Med. 2021;35(5):2588–95.
33. Keene BW, Atkins CE, Bonagura JD, Fox PR, Häggström J, Fuentes VL, et al. ACVIM consensus guidelines for the diagnosis and treatment of myxomatous mitral valve disease in dogs. J Vet Intern Med. 2019;33(3):1127–40.
34. Lin YK, Chen YA, Lee TI, Chen YC, Chen SA, Chen YJ. Aging modulates the substrate and triggers remodeling in atrial fibrillation. Circ J. 2018;82(5):1237–44.
35. Borgarelli M, Zini E, D'Agnolo G, Tarducci A, Santilli RA, Chiavegato D, et al. Comparison of primary mitral valve disease in German Shepherd dogs and in small breeds. J Vet Cardiol. 2004;6(1):27–34.
36. Kuo MYW, Häggström J, Gordon SG, Höglund K, Côté E, Lu TL, et al. Veterinary echocardiographers' preferences for left atrial size assessment in dogs: The BENEFIT project. J Vet Cardiol. 2024;51(1):157–71.
37. Perlepe K, Sirimarco G, Strambo D, Eskandari A, Karagkiozi E, Vemmou A, et al. Left atrial diameter thresholds and new incident atrial fibrillation in embolic stroke of undetermined source. Eur J Intern Med. 2020;75(1):30–4.
38. Oyama MA, Sisson DD, Bulmer BJ, Constable PD. Echocardiographic estimation of mean left atrial pressure in a canine model of acute mitral valve insufficiency. J Vet Intern Med. 2004;18(5):667–72.
39. Wu N, Li J, Xu X, Yuan Z, Yang L, Chen Y, et al. Prediction model of new onset atrial fibrillation in patients with acute coronary syndrome. Int J Clin Pract. 2023;2023(1):3473603.
40. Baron Toaldo M, Romito G, Guglielmini C, Diana A, Pelle NG, Contiero B, et al. Prognostic value of echocardiographic indices of left atrial morphology and function in dogs with myxomatous mitral valve disease. J Vet Intern Med. 2018;32(3):914–21.
41. Morgan KRS, Monteith G, Raheb S, Colpitts M, Fonfara S. Echocardiographic parameters for the assessment of congestive heart failure in dogs with myxomatous mitral valve disease and moderate to severe mitral regurgitation. Vet J. 2020;263(1):105518.
42. Borgarelli M, Savarino P, Crosara S, Santilli RA, Chiavegato D, Poggi M, et al. Survival characteristics and prognostic variables of dogs with mitral regurgitation attributable to myxomatous valve disease. J Vet Intern Med. 2008;22(1):120–8.
43. Sargent J, Muzzi R, Mukherjee R, Somarathne S, Schranz K, Stephenson H, et al. Echocardiographic predictors of survival in dogs with myxomatous mitral valve disease. J Vet Cardiol. 2015;17(1):1–12.
44. Tidholm A, Häggström J. Prognostic value of selected one-, two- and three-dimensional and Doppler echocardiographic methods to assess severity in dogs with myxomatous mitral valve disease. J Vet Cardiol. 2022;39(1):89–101.

45. Ikoma T, Obokata M, Okada K, Harada T, Sorimachi H, Yoshida K, et al. Impact of right atrial remodeling in heart failure with preserved ejection fraction. J Card Fail. 2021;27(6):577–84.
46. Gorter TM, Van Melle JP, Rienstra M, Borlaug BA, Hummel YM, Van Gelder IC, et al. Right heart dysfunction in heart failure with preserved ejection fraction: The impact of atrial fibrillation. J Card Fail. 2018;24(3):177–85.
47. De Vos CB, Weijs B, Crijns HJGM, Cheriex EC, Palmans A, Habets J, et al. Atrial tissue Doppler imaging for prediction of new-onset atrial fibrillation. Heart. 2009;95(10):835–40.
48. Pavasini R, Fabbri G, Fiorio A, Campana R, Passarini G, Verardi FM, et al. Peak atrial longitudinal strain is predictive of atrial fibrillation in patients with chronic obstructive pulmonary disease and coronary artery disease. Echocardiography. 2021;38(6):909–15.
49. Lishmanov A, Chockalingam P, Senthilkumar A, Chockalingam A. Tachycardia-induced cardiomyopathy: Evaluation and therapeutic options. Congest Heart Fail. 2010;16(3):122–6.
50. Pedro B, Mavropoulou A, Oyama MA, Linney C, Neves J, Dukes-McEwan J, et al. Optimal rate control in dogs with atrial fibrillation—ORCA study. J Vet Intern Med. 2023;37(3):887–99.
51. Romito G, Darida S, Valente C, Poser H, Contiero B, Cipone M, et al. Prevalence and prognostic role of L wave and selected clinical and echocardiographic variables in dogs with atrial fibrillation. J Vet Intern Med. 2023;37(1):47–57.
52. Prabhu S, Voskoboinik A, Kaye DM, Kistler PM. Atrial fibrillation and heart failure—cause or effect? Heart Lung Circ. 2017;26(10):967–74.
53. Maisel WH, Stevenson LW. Atrial fibrillation in heart failure: Epidemiology, pathophysiology, and rationale for therapy. Am J Cardiol. 2003;91(6 Suppl):2D–8D.