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SHORT COMMUNICATION

SQTL and competitive sports: a challenge for medicine in Latin America

SQTL y deporte competitivo: un reto para la medicina en América Latina

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ABSTRACT

The QT interval, measured on the ECG, represented the time of ventricular depolarisation and repolarisation, and its prolongation indicated a risk of serious arrhythmias or sudden death. In athletes, this prolongation was sometimes observed as a physiological adaptation, although in certain cases it responded to pathologies such as Long QT Syndrome (LQTS). As competitive sport became more professional in Latin America, concerns grew about latent cardiac conditions, especially after sudden deaths in elite athletes. The diagnosis of prolonged QT required detailed evaluations, as intense training altered normal heart parameters. The lack of standardised protocols, systematic screening and access to advanced technology made it difficult to detect LQTS, increasing the risk in genetically susceptible populations. Despite some advances in education and partial reforms, inequalities in cardiac care persisted, making prolonged QT a medical and social challenge.

Keywords: QT; Athletes; Sudden Death; Diagnosis; Intense Training.

RESUMEN

El intervalo QT, medido en el ECG, representó el tiempo de despolarización y repolarización ventricular, y su prolongación indicó riesgo de arritmias graves o muerte súbita. En atletas, dicha prolongación se observó a veces como una adaptación fisiológica, aunque en ciertos casos respondió a patologías como el Síndrome de QT Largo (SQTL). A medida que el deporte competitivo se profesionalizó en América Latina, crecieron las preocupaciones sobre condiciones cardiológicas latentes, especialmente tras muertes súbitas en deportistas de élite. El diagnóstico del QT prolongado requirió evaluaciones detalladas, debido a que el entrenamiento intenso alteró parámetros normales del corazón. La falta de protocolos homogéneos, cribados sistemáticos y acceso a tecnología avanzada dificultó la detección del SQTL, incrementando el riesgo en poblaciones genéticamente susceptibles. A pesar de algunos avances en educación y reformas parciales, persistieron desigualdades en la atención cardiológica, lo que convirtió al QT prolongado en un desafío tanto médico como social.

Palabras clave: QT; Deportistas; Muerte Súbita; Diagnóstico; Entrenamiento Intenso.

INTRODUCTION

The QT interval, measured on a standard 12-lead electrocardiogram (ECG), represents the total duration of ventricular electrical activity, encompassing both ventricular depolarization and repolarization. It extends from the beginning of the QRS complex to the end of the T wave and constitutes an important electrocardiographic marker for evaluating ventricular repolarization. Abnormal prolongation of this interval may reflect delayed myocardial repolarization and increased electrical instability, potentially creating a substrate for malignant ventricular arrhythmias. Among the most clinically relevant complications associated with QT prolongation are torsades de pointes, ventricular fibrillation, syncope, cardiac arrest, and sudden cardiac death.(1,2) These consequences make the assessment of ventricular repolarization particularly important in populations exposed to intense physiological stress, including competitive athletes.

In athletes, interpretation of the QT interval is especially complex because regular and intensive physical training produces multiple cardiovascular adaptations. Some trained individuals may present sinus bradycardia and changes in ventricular repolarization as part of the physiological adaptation commonly associated with the athlete's heart. Consequently, a modest increase in the absolute QT interval may occur without necessarily representing an underlying disease. However, QT prolongation may also be the electrocardiographic manifestation of a potentially life-threatening condition such as Long QT Syndrome (LQTS).(3) Differentiating physiological adaptation from pathological QT prolongation is therefore essential because an incorrect interpretation can lead either to unnecessary restriction from competitive sports or, conversely, to failure to identify an athlete at increased risk of a serious cardiovascular event.

DEVELOPMENT

The increasing professionalization and intensity of competitive sports in Latin America have made cardiovascular safety an increasingly relevant component of sports medicine.(4) Athletes currently participate in demanding training programs, frequent competitions, international tournaments, and prolonged seasons that expose the cardiovascular system to considerable physiological stress. In this context, previously silent or latent cardiac abnormalities may become clinically apparent during exercise. Reports of sudden cardiac events among young and elite athletes have increased concern among sports federations, medical teams, coaches, athletes, and specialists in sports cardiology. These events have reinforced the importance of identifying electrical abnormalities that may not produce symptoms during routine daily activities but can become clinically significant during high-intensity exercise.(4,5)

Understanding the relationship between competitive exercise and QT abnormalities is particularly relevant because physical activity produces substantial changes in heart rate, autonomic tone, circulating catecholamines, electrolyte balance, and myocardial oxygen demand. In a healthy athlete, these changes are normally well tolerated. In an individual with an underlying channelopathy or another abnormality of ventricular repolarization, however, the same physiological responses may facilitate the development of ventricular arrhythmias.(3,5) Therefore, the QT interval cannot be interpreted as an isolated numerical value. Its evaluation should take into consideration the athlete's heart rate, symptoms, family history, type of sport, training intensity, medication exposure, electrolyte status, and other ECG findings.

The QT interval is conventionally measured from the beginning of the QRS complex to the end of the T wave. Because its duration is strongly influenced by heart rate, interpretation of the uncorrected QT interval can be misleading. For this reason, mathematical formulas are used to calculate the corrected QT interval (QTc). Among the most widely used methods are the Bazett and Fridericia formulas, although their performance may differ according to heart rate.(5,6) This issue is particularly important in athletes because resting sinus bradycardia is common in highly trained individuals. Depending on the correction method employed, the QTc may therefore be overestimated or underestimated, complicating clinical interpretation.

Traditionally, QTc values above approximately 450 ms in men and 460 ms in women have been regarded as prolonged in clinical evaluation, although interpretation should not depend exclusively on a single threshold.(5-8) Borderline values require particular caution because the distribution of QTc intervals overlaps between healthy individuals and patients with LQTS. Furthermore, a single ECG may not adequately characterize ventricular repolarization because QT duration can vary with autonomic conditions, physical activity, electrolyte concentrations, medications, and measurement technique. Repeated ECG assessment and correlation with the clinical context may

therefore be necessary when an abnormal or borderline QTc is identified.(6-8)

Intensive physical training produces structural, functional, and electrical adaptations of the cardiovascular system. These adaptations may include increased ventricular dimensions, physiological ventricular hypertrophy, increased stroke volume, enhanced vagal tone, and resting sinus bradycardia. Collectively, these findings constitute components of the athlete's heart and are generally considered benign responses to sustained training.(9,10) However, these physiological changes may influence ECG parameters, including ventricular repolarization and the measured QT interval. The challenge for clinicians is to distinguish expected training-related adaptations from findings that indicate an underlying electrical disorder.

This distinction becomes especially relevant when an athlete presents a prolonged or borderline QTc together with other warning signs. A history of unexplained syncope, presyncope, palpitations, seizures of uncertain origin, cardiac arrest, or sudden death among young relatives should increase suspicion of an inherited arrhythmogenic disorder.(3,9,10) Symptoms occurring during exercise or immediately after intense exertion deserve particular attention because they may represent manifestations of ventricular arrhythmia rather than benign consequences of physical fatigue. Thus, the interpretation of QT abnormalities should always be integrated with a comprehensive clinical and family history.

Long QT Syndrome may be congenital or acquired. Congenital LQTS is an inherited cardiac channelopathy associated with abnormalities in ion channels involved in myocardial repolarization. Acquired QT prolongation, in contrast, may result from medications, electrolyte abnormalities, metabolic disturbances, or other clinical conditions.(3,11) Although these mechanisms are different, both can increase susceptibility to ventricular arrhythmias. In competitive athletes, the distinction between congenital and acquired QT prolongation is clinically important because potentially reversible factors may coexist with an underlying genetic predisposition.

Exercise represents a particularly relevant trigger in some forms of congenital LQTS. High-intensity sports such as soccer, athletics, swimming, and cycling involve marked sympathetic activation and rapid fluctuations in heart rate.(3) These physiological conditions may increase arrhythmic risk in susceptible individuals. Nevertheless, the relationship between exercise and arrhythmia is heterogeneous and depends partly on the underlying molecular subtype, individual clinical characteristics, previous symptoms, treatment, and environmental factors.

Mutations affecting genes encoding cardiac ion channels constitute the molecular basis of many cases of congenital LQTS. Among the genes most frequently associated with the syndrome are KCNQ1, KCNH2, and SCN5A.(12-14) Alterations in these genes can disrupt potassium or sodium currents involved in ventricular action potential formation and repolarization. The resulting electrical instability may prolong ventricular repolarization and increase susceptibility to early afterdepolarizations and malignant ventricular arrhythmias.(12,13)

Genotype may also influence the circumstances under which arrhythmic events occur. LQT1, commonly associated with pathogenic variants in KCNQ1, has a particularly important relationship with physical exercise and sympathetic stimulation. Other genetic subtypes may show different trigger profiles, including emotional stress, sudden auditory stimuli, sleep, or resting conditions.(12-14) This genotype-phenotype relationship illustrates why evaluation of an athlete with suspected LQTS should extend beyond the resting ECG whenever clinical circumstances justify additional investigation.

Acquired QT prolongation represents another important concern in sports medicine. Numerous pharmacological agents can affect ventricular repolarization, including certain antiarrhythmics, antibiotics, antipsychotics, antidepressants, and antihistamines.(15) Athletes may use prescription medications for unrelated medical conditions without being aware of their potential effect on QT duration. In addition, combinations of several QT-prolonging medications may further increase risk, particularly when additional predisposing factors are present.

Electrolyte abnormalities constitute another potentially modifiable mechanism. Prolonged exercise, high environmental temperatures, excessive sweating, dehydration, gastrointestinal losses, and inadequate replacement of electrolytes can alter potassium, magnesium, and other electrolyte concentrations. In susceptible athletes, these changes may contribute to electrical instability, especially when combined with QT-prolonging medications or an underlying channelopathy.(15) Consequently, evaluation of prolonged QT should include consideration of reversible causes rather than automatically assuming a congenital disorder.

The use of performance-enhancing or doping substances represents an additional concern. Competitive pressure may expose some athletes to substances capable of producing direct or indirect cardiovascular effects, including changes in autonomic tone, blood pressure, heart rate, myocardial structure, or electrolyte balance.(15) From a preventive perspective, sports medicine

programs should therefore incorporate a detailed review of prescribed drugs, over-the-counter medications, supplements, and other substances used by athletes when investigating unexplained QT prolongation.

Despite the potential clinical importance of this condition, epidemiological information concerning QT abnormalities among Latin American athletes remains limited. Most of the evidence guiding contemporary interpretation and management has been generated outside the region, while systematic studies involving Latin American athletic populations remain comparatively scarce.(16) This evidence gap complicates estimation of the true prevalence of prolonged QT, congenital LQTS, and associated arrhythmic events among athletes in the region.

Cases of sudden cardiac death and potentially arrhythmogenic abnormalities have nevertheless been reported in Latin American sports settings, including countries such as Brazil, Argentina, Colombia, Mexico, and other nations with large populations participating in organized competitive sports.(16) These events highlight the potential consequences of clinically silent cardiovascular disorders and reinforce the importance of pre-participation cardiovascular evaluation. However, isolated cases cannot by themselves establish the prevalence of LQTS or determine the proportion of sports-related sudden deaths directly attributable to QT abnormalities. More systematic regional research is required to clarify this relationship.

Latin America also represents a particularly heterogeneous population from a genetic and demographic perspective. The region includes populations with varying proportions of Indigenous American, European, African, and Asian ancestry, producing substantial genetic diversity.(17,18) This diversity may influence the distribution of genetic variants related to cardiac electrophysiology. Consequently, extrapolating genetic frequencies and reference data obtained exclusively from other populations may not fully represent the genetic architecture of Latin American athletes.

Population ancestry is therefore a potentially relevant factor when studying inherited arrhythmia syndromes. Certain ion-channel variants and genetic polymorphisms may differ in frequency among populations, and the clinical significance of a genetic finding must be interpreted carefully.(17,18) Genetic diversity should not be considered a deterministic predictor of cardiovascular events, but it reinforces the need for representative regional studies capable of defining the prevalence and clinical significance of variants in diverse Latin American populations.

Another major challenge is the absence of uniform cardiovascular screening practices throughout the region. Pre-participation medical evaluations differ considerably between countries, sports disciplines, federations, clubs, and levels of competition.(19) Professional athletes may have access to specialized cardiological assessment, whereas amateur, school, university, or community athletes may undergo only basic medical examinations or no formal cardiovascular evaluation. These differences create important inequalities in opportunities for early detection.

The lack of systematic ECG screening may contribute to underdiagnosis of QT abnormalities because LQTS can remain clinically silent until the occurrence of syncope, ventricular arrhythmia, or sudden cardiac arrest.(19) An athlete without symptoms may therefore continue participating in high-intensity competition despite having a potentially relevant repolarization abnormality. Conversely, indiscriminate interpretation of athletic ECGs without appropriate expertise may generate false-positive findings and unnecessary restrictions. Screening strategies must therefore balance sensitivity with appropriate interpretation.

Although some national federations and professional organizations require cardiovascular examinations before participation, protocols vary substantially.(20) The content of these assessments may range from medical history and physical examination to resting ECG, echocardiography, or exercise testing. In many settings, standardized evaluation of ventricular repolarization is not systematically incorporated, and access to specialized sports cardiology remains concentrated in major cities or high-level professional institutions.

Exercise testing may provide clinically useful information when resting findings are inconclusive. QT behavior during increasing heart rates and particularly during recovery can reveal abnormal repolarization patterns that may not be evident on a single resting ECG.(20,21) Nevertheless, exercise-based interpretation of QT dynamics requires appropriate methodology and clinical expertise. The objective is not merely to identify an isolated prolonged measurement but to understand the behavior of ventricular repolarization under physiological stress.

Episodes of sudden death among young athletes in Latin American countries have stimulated discussion regarding the adequacy of existing cardiovascular screening systems.(20,21) Postmortem investigations of sudden cardiac death are essential for identifying structural or electrical disorders and for protecting relatives who may share inherited susceptibility. However, access to specialized cardiovascular pathology and postmortem genetic analysis remains heterogeneous, limiting the ability to determine the precise mechanism of death in some cases.

This limitation has broader epidemiological consequences. When sudden deaths are not investigated using standardized protocols, deaths caused by primary electrical disorders may remain unexplained or be attributed to nonspecific causes. As a result, the contribution of inherited channelopathies such as LQTS to sports-related mortality may be underestimated.(20,21) Strengthening registries of sudden cardiac death in athletes and improving postmortem investigation could therefore provide valuable information for future prevention strategies.

The resting 12-lead ECG remains a fundamental tool for detecting electrical abnormalities associated with increased cardiovascular risk.(22) It is relatively accessible, non-invasive, rapid, and considerably less expensive than advanced cardiac imaging or genetic testing. However, ECG interpretation in athletes differs from interpretation in the general population because regular training produces characteristic electrical adaptations. Expertise in athlete-specific ECG interpretation is therefore necessary to avoid both overdiagnosis and underdiagnosis.

When prolonged QT is identified, clinicians should verify measurement accuracy before making clinical decisions. Determining the end of the T wave can be difficult when T-wave morphology is abnormal or when U waves are present. Heart-rate correction introduces another source of variability, especially in athletes with marked bradycardia.(22) For this reason, manual verification of automated ECG measurements and repeated recordings may be appropriate when a clinically significant abnormality is suspected.

An abnormal QTc should also be interpreted in conjunction with other clinical information. The presence of symptoms, family history of premature sudden death, documented ventricular arrhythmias, characteristic T-wave abnormalities, or persistent QT prolongation increases the probability of an underlying channelopathy.(22,23) In contrast, a transient abnormality associated with a reversible medication or electrolyte disturbance may have a different clinical implication. Comprehensive assessment is therefore preferable to decisions based on an isolated QTc threshold.

Genetic testing can contribute to the confirmation of congenital LQTS in appropriately selected patients and may permit identification of a specific molecular subtype.(23) When a pathogenic variant is detected, genetic evaluation can also have implications for relatives because congenital LQTS may affect multiple members of the same family. Cascade screening may identify individuals who have not yet developed symptoms but who could benefit from preventive evaluation and management.

However, access to cardiovascular genetic testing remains unequal throughout Latin America.(23) High costs, limited availability of specialized laboratories, insufficient numbers of genetic counselors, and geographic concentration of advanced diagnostic services may restrict its routine use. In addition, genetic testing does not always provide a definitive answer. Variants of uncertain significance can complicate interpretation, particularly in populations that remain underrepresented in international genomic databases. These limitations reinforce the importance of integrating genetic results with clinical and electrocardiographic findings.

Exercise testing represents another useful component of the diagnostic evaluation. Dynamic QT assessment during exercise and recovery can reveal repolarization abnormalities that are absent or borderline at rest.(24) This approach may be particularly relevant in athletes because their symptoms, when present, often occur during physical exertion. Exercise testing can simultaneously provide information regarding symptoms, rhythm disturbances, heart-rate response, functional capacity, and ventricular repolarization.

Ambulatory ECG monitoring may also be useful in selected athletes when intermittent arrhythmias, unexplained palpitations, or episodic symptoms are suspected. Nevertheless, no single diagnostic test should be considered sufficient for all cases. The evaluation of suspected LQTS generally requires integration of clinical history, family history, resting ECG findings, exercise response, medication exposure, electrolyte status, and, where appropriate, genetic information.(22-24)

Management of LQTS depends on its cause, severity, genotype, symptoms, and individual risk profile. Beta-blocker therapy constitutes an important treatment strategy for many patients with congenital LQTS, particularly when adrenergic stimulation is an important trigger.(25) Management of acquired QT prolongation requires identification and correction of reversible causes, including discontinuation or replacement of QT-prolonging medications when clinically appropriate and correction of relevant electrolyte abnormalities.(15,25)

Patients at higher arrhythmic risk may require more intensive preventive strategies. In selected cases, an implantable cardioverter-defibrillator (ICD) may be considered to provide protection against life-threatening ventricular arrhythmias.(25) Decisions regarding device implantation require specialist assessment because ICD therapy has potential complications and significant implications for young and physically active individuals. Treatment should therefore be individualized rather than based exclusively on the presence of a prolonged QT interval.

The question of sports participation is particularly complex. Historically, athletes with inherited arrhythmia syndromes were frequently advised to avoid competitive sports. Contemporary clinical decision-making increasingly requires individualized assessment based on diagnosis, genotype, symptoms, previous arrhythmic events, treatment response, sport characteristics, and the availability of emergency resources.(25) The risk associated with sports participation is not identical for every patient with LQTS, and decisions should therefore involve clinicians with expertise in inherited arrhythmias and sports cardiology.

For young athletes, restriction from competition can have consequences extending beyond cardiovascular health. Competitive sport may represent a professional career, educational opportunity, source of income, social identity, or central component of personal development. Consequently, recommendations concerning sports participation involve medical, ethical, psychological, and social dimensions. Appropriate counseling should communicate risk clearly while respecting the athlete's values and ensuring that decisions are based on the best available clinical evidence.(25)

Emergency preparedness is another essential component of cardiovascular safety in sports. Even comprehensive screening cannot identify every athlete at risk of sudden cardiac arrest. Sports facilities, clubs, schools, and competition venues therefore require effective emergency response systems, personnel trained in cardiopulmonary resuscitation, and rapid access to automated external defibrillators. These measures complement rather than replace cardiovascular screening and clinical management.

The quality and availability of sports medicine services vary markedly throughout Latin America. Differences exist not only between countries but also within countries and according to the athlete's level of competition. Professional clubs may have cardiologists, physiotherapists, imaging facilities, and structured medical protocols, whereas amateur athletes may have limited access even to basic pre-participation assessment. Such disparities can delay identification of prolonged QT and other potentially important cardiovascular abnormalities.

Geographic inequality further compounds this problem. Advanced cardiovascular evaluation and genetic services are frequently concentrated in large urban centers, while athletes in rural or economically disadvantaged areas may face substantial barriers to specialized assessment. These differences raise questions of health equity because the ability to participate safely in competitive sport should not depend exclusively on socioeconomic status, geographic location, or affiliation with a wealthy professional organization.

A related challenge is the absence of a unified regional framework establishing minimum cardiovascular assessment standards for competitive athletes. Requirements are often determined independently by national federations, individual sports organizations, clubs, universities, or local medical teams. This fragmented approach can result in significant differences in the type and quality of cardiovascular screening received by athletes participating in similar levels of physical activity.

The effectiveness of screening also depends on the professionals responsible for its implementation. Coaches and sports administrators play an important role in recognizing warning symptoms and ensuring access to medical evaluation, but they cannot substitute for appropriately trained healthcare professionals. Unexplained syncope during exercise, exertional chest symptoms, sustained palpitations, a family history of premature sudden death, or abnormal ECG findings require qualified medical assessment rather than informal interpretation.

Strengthening education in sports cardiology is therefore essential. Universities and medical training programs in Latin America have progressively incorporated topics related to exercise physiology, athlete ECG interpretation, sudden cardiac death, inherited arrhythmia syndromes, and cardiovascular screening. Nevertheless, additional training opportunities and wider dissemination of standardized interpretation criteria remain necessary, particularly outside major referral centers.

Technological development may also improve detection and follow-up. Digital ECG systems, telecardiology, ambulatory monitoring, and remote specialist consultation could potentially extend access to expert interpretation in regions where sports cardiologists are unavailable. However, technology should be accompanied by standardized protocols, quality control, appropriate clinical oversight, and mechanisms for referral when potentially pathological findings are detected.

Research constitutes another fundamental priority. Prospective multicenter studies involving athletes from different Latin American countries could provide more reliable estimates of the prevalence of QT prolongation and other repolarization abnormalities. Such research should consider age, sex, ancestry, sport type, training intensity, competitive level, medication exposure, and relevant clinical outcomes. Regional registries of sudden cardiac arrest and sudden cardiac death in athletes would also improve understanding of the burden and causes of these events.

Greater representation of Latin American populations in genetic research is similarly necessary. Improved genomic characterization could facilitate interpretation of variants detected

in patients with suspected inherited channelopathies and reduce uncertainty associated with underrepresentation in existing databases. Nevertheless, genetic research must be accompanied by appropriate ethical safeguards, informed consent, confidentiality, and access to professional interpretation.

From a public health perspective, prevention requires interventions at several levels. At the individual level, athletes should be educated about warning symptoms, medication-related risks, hydration, electrolyte disturbances, and the importance of reporting relevant family history. At the institutional level, clubs and federations should establish clear cardiovascular assessment and emergency response protocols. At the healthcare-system level, referral pathways should facilitate access to cardiology, electrophysiology, and genetic evaluation for athletes with suspicious findings.

QT prolongation therefore represents a clinically important but frequently silent cardiovascular risk in competitive sports. Its significance ranges from benign physiological variation to the manifestation of an inherited or acquired disorder capable of causing malignant ventricular arrhythmias.(1,3,25) The central clinical challenge is not simply to identify a prolonged QT interval but to determine which findings represent normal athletic adaptation, which reflect reversible acquired factors, and which indicate an underlying arrhythmogenic syndrome requiring specific management.

In Latin America, this challenge is intensified by heterogeneous screening practices, unequal access to specialized cardiovascular care, limited regional epidemiological data, and substantial socioeconomic and geographic disparities. These factors may contribute to underdiagnosis, particularly among young and amateur athletes who do not have access to comprehensive sports medicine programs. At the same time, inappropriate interpretation of athletic ECGs may produce unnecessary diagnostic procedures or restrictions, highlighting the need for specialized clinical expertise.

Systematic cardiovascular evaluation, appropriate ECG interpretation, recognition of warning symptoms, assessment of medication and electrolyte-related risks, access to complementary diagnostic testing, and individualized management constitute fundamental components of prevention. The development of standardized regional strategies could improve early identification of athletes at increased risk while avoiding indiscriminate exclusion from sports participation.

Ultimately, QT prolongation in competitive athletes should not be viewed exclusively as an electrocardiographic abnormality or an isolated clinical problem. It also represents a challenge involving preventive medicine, public health, health equity, sports governance, professional education, and access to diagnostic technology. The ability to identify an athlete at risk before the occurrence of a catastrophic event depends on coordination among athletes, families, physicians, clubs, universities, federations, and health authorities.

CONCLUSIONS

Latin America has the opportunity to strengthen a model of sports medicine based on prevention, scientific evidence, equitable access, and individualized risk assessment. Expanding cardiovascular screening capacity, improving professional training, promoting regional research, establishing appropriate referral pathways, and strengthening emergency preparedness could substantially improve the safety of competitive sports. Such measures would not seek to restrict athletic participation unnecessarily, but rather to create conditions in which athletes can train and compete with an appropriate understanding and management of cardiovascular risk. In this context, the identification and adequate management of QT abnormalities constitute an important component of safeguarding both the health and the future of athletes throughout the region.

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CONFLICT OF INTEREST

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AUTHORSHIP CONTRIBUTIONS

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