Early repolarization patterns associated with increased arrhythmic risk are common in young non-Caucasian Australian males and not influenced by athletic status

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BACKGROUND Early repolarization (ER) with a horizontal ST segment (ST-h) and high-amplitude J waves in the inferior leads is associated with an increased risk of cardiac arrhythmic death. The effect of ethnicity and athletic status on this increased-risk ER pattern has not been established. Aboriginal Australian/Torres Strait Islander and Pacific Islander/Maori (non-Caucasian [non-C]) subjects are well represented in Australian sport; however, the patterns and prevalence of ER in these populations are unknown.

OBJECTIVE The purpose of this study was to assess the prevalence and effect of athletic activity on ER patterns in young non-C and Caucasian (C) subjects.

METHODS Twelve-lead ECGs of 726 male athletes (23.8% non-C) and 170 male controls (45.9% non-C) aged 16–40 years were analyzed for the presence of ER, defined as J-point elevation (J wave, QRS slur, or discrete ST elevation) $\geq$0.1 mV in $\geq$2 inferior (II, III, aVF) or lateral (I, aVL, V_4–V_6) leads. ST morphology was coded as horizontal (ST-h) or ascending (ST-a). "Increased-risk ER" was defined as inferior ER with ST-h and J waves $>$2 mV.

RESULTS Regardless of athletic status, ER and increased-risk ER were more prevalent in non-C than in C subjects (53.8% vs 32% and 7.6% vs 1.2%, respectively, $P < .0001$). Whereas lower heart rate, larger QRS voltage, and shorter QRS duration were predictors of ER, non-C ethnicity was the only independent predictor of increased-risk ER (odds ratio 17.621, 95% confidence interval 4.98–62.346, $P < .0001$).

CONCLUSION ER patterns associated with increased arrhythmic risk are more common in young non-C than C subjects and were not influenced by athletic status. The long-term clinical significance of ER in these populations is yet to be determined.

KEYWORDS Early repolarization; Athlete; Indigenous Australian; Ethnicity; Ventricular fibrillation

ABBREVIATIONS **AMI** = acute myocardial infarction; **C** = Caucasian; **C-A** = Caucasian athlete; **CI** = confidence interval; **C–NA** = non-Caucasian nonathlete; **ER** = early repolarization; **LVH** = left ventricular hypertrophy; **NA** = nonathletic; **non-C** = subject of Aboriginal Australian/Torres Strait Islander and Pacific Islander/Maori heritage; **non-C–A** = non-Caucasian athlete; **non-C–NA** = non-Caucasian nonathlete; **OR** = odds ratio; **QRSd** = QRS duration; **QTc** = corrected QT interval; **SCD** = sudden cardiac death; **ST-a** = ascending ST segment; **STE** = ST-segment elevation; **ST-h** = horizontal ST segment

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Introduction

Early repolarization (ER) is generally considered a normal finding in young athletic individuals. The most common description of ER in athletes is J-point elevation with an associated ascending ST segment (ST-a) in the anterolateral ECG leads. The benign nature of this pattern has been confirmed in several studies.[1–3] However, in more recent years it has become clear that not all patterns of ER are the same. Since 2008 when Haissaguerre et al[4] described a high prevalence of prominent J waves or QRS slurs in the inferolateral ECG leads in survivors of sudden cardiac arrest

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due to idiopathic ventricular fibrillation, a series of studies have confirmed an association with idiopathic ventricular fibrillation, as well as an increased risk of atrial and ventricular tachyarrhythmias and death in the context of acute myocardial ischemia (AMI).[4–10] The highest arrhythmic risk has been associated with inferior ER with a horizontal ST segment (ST-h) and high-amplitude (>2 mV) J waves.[11–13] The prevalence of this pattern in young athletic populations and the effect of athletic training are unclear, as most studies focusing on athletes have not considered ST-segment morphology and have lacked age-matched, nonathletic (NA) controls (most control groups consist of middle-aged subjects).[3,7,12] Furthermore, descriptions of the patterns and prevalence of ER in young athletic populations of ethnic backgrounds other than African American or Caucasian are lacking.

Approximately 11% of the professional male players in the Australian Football League are of Aboriginal Australian/Torres Strait Islander heritage and up to 30% of professional male players in rugby football codes are of Pacific Islander or Maori heritage, yet we have no data on the patterns and prevalence of ECG patterns in these populations, in whom cardiac mortality related to premature coronary disease far exceeds that of their nonindigenous counterparts.[14,15] Thus, the aim of this study was to determine if the patterns and prevalence of ER in young subjects of Aboriginal Australian/Torres Strait Islander and Pacific Islander or Maori heritage (non-Caucasian [non-C]) may differ from Caucasians (C), and whether athletic training may affect these patterns.

Methods

Study population

De-identified ECGs of 1306 consecutive elite athletes aged 16–35 years who underwent preparticipation cardiac screening inclusive of an ECG between June 2011 and December 2013 were analyzed. Our study methods have been described in detail elsewhere.[16,17] All subjects provided written informed consent, and ethics approval was obtained from the Human Research and Ethics Committee at St. Vincent's Hospital, Melbourne, and the Australian Institute of Sport, Canberra. The gender differences in ER have been well described, so only male athletes were selected for this analysis because the non-C group contained a much smaller proportion of females than the Caucasian athlete (C-A) group. All of the non-Caucasian athletes (non–C-A) were participating in football codes; thus, only C-A footballers (who could be expected to be performing very similar training volume and intensity) were selected as a comparative group. There were 553 C and 173 non-C elite male athletes included.

NA control population

NA subjects were recruited prospectively, predominantly through advertisement for voluntary cardiac screening at a local university. Subjects were excluded if they were participating in 3 or more hours per week of intense exercise, if they were known to have cardiac disease, or if they were older than 40 years. Only males were included in this analysis. As for athletes, ethnicity was determined by a self-reported questionnaire, which contained options including Caucasian, Asian, African, Aboriginal Australian, Torres Strait Islander, Pacific Islander, Maori, and "other." ECGs of a proportion of non-C–NA control subjects were obtained retrospectively, having been collected as part of the Heart of the Heart Study between May 2008 and November 2009.[18] A total of 78 non-C–NA and 92 C–NA males met inclusion criteria and were included in this analysis.

ECG analysis

All ECGs were recorded at rest at 25 mm/s and 10 mm/mV. To blind the interpreting cardiologists as to subject grouping, all ECGs were scanned electronically and coded. Analysis was performed with ECGs in electronic format, magnified to 200%. Reviewers categorized the presence of ER in each lead (except aVR) separately and then by territory (inferior, II, III, aVF or lateral V_4–V_6, I, aVL). Although the anterior leads were analyzed, they were not included in the definition of an ER-positive ECG. Measures of ER, described as follows, are outlined in Figure 1. Morphology of the J point was categorized as J wave (sharp, well-defined hump or notch immediately following a positive QRS complex at the onset of the ST segment), QRS slur (R-wave gradually becomes ST segment with upright concavity), or discrete ST-segment elevation (STE) without a notch or slur. Amplitude of the J wave, QRS slur or discrete STE and the subsequent ST segment (measured at the end of the QRS complex, following the notch or slur, when present) were measured using digital calipers, using the preceding TP segment as baseline. STE 100 ms after merging of the J point and ST segment was measured and coded as ascending (>0.1 mV STE, ascending gradually until the T wave) or horizontal/descending (≤ 0.1 mV STE, continuing as a flat/descending segment until onset of the T wave). The ECG was considered to show ER if there was elevation of the J point (seen as J wave, QRS slur, or discrete ST elevation) of at least 0.1 mV in at least 2 leads within the inferior (II, III, aVF) or lateral (V_4–V_6, I, aVL) territories. To be consistent with previous studies,[3,12] if a J wave was present in 1 lead and a slur in the other, the territory was coded as J wave, and if the ST segment was ascending in some leads and horizontal in others, the territory was categorized as ST-h. "Increased-risk ER" was defined as inferior ER with ST-h and J waves >2 mV.

Other measurements included heart rate, PR interval, QRS duration (QRSd), correct QT interval (QTc) with Bazett correction, and Sokolow Lyon score for left ventricular hypertrophy (LVH; SV1+RV5, mm).

Interobserver and intraobserver variability

In order to assess interobserver reliability, a randomly selected subset of 100 ECGs was analyzed independently