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Lifetime Exposure to Recreational Swimming Training and its Effects on Autonomic Responses

This is the final peer-reviewed author's accepted manuscript (postprint) of the following publication:

*Published Version:*

Piras, A., Cortesi, M., Di Michele, R., Trofè, A., Raffi, M. (2021). Lifetime Exposure to Recreational Swimming Training and its Effects on Autonomic Responses. *INTERNATIONAL JOURNAL OF SPORTS MEDICINE*, 42(5), 425-431 [10.1055/a-1224-3842].

*Availability:*

This version is available at: <https://hdl.handle.net/11585/776796> since: 2024-12-09

*Published:*

DOI: <http://doi.org/10.1055/a-1224-3842>

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1 **Title: Lifetime Exposure to Recreational Swimming Training and its Effects on**  
2 **Autonomic Responses**

3

4 **Abstract**

5       The aim of the present investigation was to assess the effect of long-term  
6 recreational swimming training on the cardiac autonomic responses in the healthy  
7 population. 70 habitual recreational swimmers ( $48.6 \pm 14.3$  yrs.) and 60 sedentary adults  
8 ( $51.5 \pm 10.4$  yrs.) were recruited. Arterial blood pressure was recorded with participants in  
9 supine position for 10 minutes, and the last 5 minutes were used to assess heart rate  
10 variability, baroreflex sensitivity, and hemodynamic analysis. The analysis of the  
11 questionnaire showed that the swimmers had practiced swimming for a mean of 14 years  
12 and 207 minutes/week. No difference was detected for body mass index between groups.  
13 Heart rate variability showed significant differences between groups both in the time and  
14 frequency domain analysis. We also found significant differences for baroreflex  
15 sensitivity. At rest, cardiac output and stroke volume were higher, whereas, heart rate,  
16 mean arterial pressure and total peripheral resistances were lower in the swimmers than  
17 in the sedentary subjects. Since heart rate variability measures are independent predictors  
18 of mortality, the present findings suggest that habitual recreational swimming may be  
19 protective against sudden cardiovascular events and, more in general, have a positive  
20 impact on cardiovascular health.

21 **Keywords:** exercise; baroreflex sensitivity; heart rate variability; hemodynamic.

22 **Running head:** Physical activity and autonomic nervous system

23

24

25

26 **Introduction**

27           Being inactive increases the risk of developing a variety of diseases, including  
28 diabetes, hypertension, overweight, osteoporosis, and depression [1]. Additionally,  
29 sedentariness has been associated with higher all-cause and cardiovascular mortality, and  
30 cardiovascular disease (CVD) is the first leading cause of mortality in industrialized  
31 countries [1]. Conversely, physical exercise is associated with hemodynamics changes,  
32 increasing volume load at the heart level during endurance training, in contrast to pressure  
33 load during strength exercise. These differences in loading will cause different  
34 cardiovascular responses to exercise, changes that are well described as “athlete’s heart”  
35 [2]. Exercise training conducted for long term influences cardiac rhythmic, inducing sinus  
36 bradycardia during resting condition. The cardiovascular system is generally controlled  
37 by autonomic regulation through the activity of sympathetic and parasympathetic  
38 pathways of the autonomic nervous system (ANS). It is widely presumed that regular  
39 endurance program induces adaptations in the ANS that result in both changes in several  
40 cardiovascular variables at rest and in alterations in the reflex control of the circulation  
41 [3]. Analysis of heart rate variability (HRV) and baroreflex sensitivity (BRS) enable  
42 understanding of this control mechanism. The ANS regulates heart rate during physical  
43 activity. The ANS is composed of a parasympathetic and a sympathetic branch that  
44 operate in a reciprocal and inverse manner: a decrease in heart rate variability is caused  
45 by an increase in sympathetic activity combined with decreased parasympathetic drive,  
46 whereas an increase in HRV is characterized by parasympathetic reactivation and  
47 sympathetic withdrawal. HRV can be evaluated by time and frequency domain indices,  
48 reflecting the activity of the autonomic nervous system. Among the most used indices,  
49 the standard deviation of normal beat-to-beat intervals (SDNN), the root-mean-square of

50 successive R–R (RMSSD) and high frequency (HF) power has been suggested to reflect  
51 indexes of cardiac vagal outflow [4]. Instead, low frequency (LF) power is mediated by  
52 both cardiac vagal and sympathetic nerves [5].

53 Physical activity and exercise are highly suitable for health promotion as well as  
54 prevention and treatment of risk factors for cardiovascular disease [6]. However, the  
55 optimal dose remains unclear [7]. Constant practice of physical exercise is associated with  
56 a lower risk of cardiovascular events, and elite athletes live longer than the general  
57 population [8]. It can be hypothesized that physical exercise would be effective in  
58 improving the autonomic balance in the general population while also developing  
59 physical fitness. On this point, there is a debate about the dose-response relationship of  
60 exercise necessary to influence the autonomic nervous system responses in order to be an  
61 effective means to positively modify factors that are associated with increased incidence  
62 of cardiac events. Until now, several longitudinal studies have been conducted on the  
63 effect of exercise training on HRV in non-athletes, although no consistent changes have  
64 been observed both in HRV parameters and BRS [9]. The authors blamed the short  
65 duration of the training program, suggesting that to obtain some effect on ANS  
66 parameters, the training program should last at least for a period of one year. Many  
67 features disturb the physiological significance of these studies. Two of them are the age  
68 and sex, which contributes to the discrepant findings in the literature. HRV decreases with  
69 age and changes as a function of sex, so that studies should take into consideration a  
70 sample size of both sexes with a wide range in terms of age. Loimaala et al. [10]  
71 investigated the effect of training on 26 sedentary subjects with age from 35 to 55 years  
72 old, with a training program of 5 months. They found no significant change of HRV time  
73 and frequency domain parameters or BRS in either of the exercise groups. An exercise-

74 training program of only 5 months was not able to modify the cardiac vagal outflow in  
75 sedentary, middle-aged persons.

76 Duration and intensity of training are the most important factors that could affect  
77 cardiac autonomic function. When researchers investigate the effect of exercise on HRV  
78 or BRS in a group of athletes with respect to the sedentary population, they found, in most  
79 of the cases, significant differences. The majority of studies that have compared HRV  
80 between well-trained and sedentary subjects have revealed that athletes have significantly  
81 higher HRV indexes than inactive subjects, even though contradictory findings have also  
82 been conveyed [9]. In all these studies, measures of HRV have been compared between  
83 high-level young athletes and sedentary, age-matched counterparts, but the results do not  
84 provide information as to whether the cardiac autonomic function can be enhanced by  
85 exercise training in the middle-aged sedentary population. A limitation of these studies is  
86 the relatively short duration of the training program, lasting no more than five months  
87 [10].

88 The world health organization (WHO) guidelines for physical activity  
89 recommend, for adults aged 18–64 years, at least 150 minutes of moderate-intensity  
90 aerobic physical activity throughout the week or to do at least 75 minutes of vigorous-  
91 intensity aerobic activity throughout the week, or an equivalent combination of moderate-  
92 and vigorous-intensity activity [1]. Moreover, they recommend that, for health benefits,  
93 adults should increase their moderate-intensity aerobic physical activity to 300 minutes  
94 per week, or engage in 150 minutes of vigorous-intensity aerobic physical activity per  
95 week, or an equivalent combination of moderate- and vigorous-intensity activity [1].  
96 Despite recreational swimming is quite widespread in the general population as an  
97 habitual mode of exercise, little is known on whether and how habitual swimming training

98 affects the cardiac autonomic response and, more in general, the cardiovascular health.  
99 Moreover, swimming has been widely promoted and prescribed without the underpinning  
100 of firm scientific support from clinical studies [6]. Therefore, the aim of our study was to  
101 investigate the effects of long-term swimming on the cardiac autonomic response,  
102 baroreflex sensitivity, and hemodynamic activity in the healthy population. The increased  
103 cardiac vagal activity associated with swimming training could be of clinical significance,  
104 since both are related to increased life expectancy and prevention of cardiovascular  
105 events.

## 106 **Methods**

### 107 *Subjects*

108 The study sample consisted of 70 habitual recreational swimmers ( $48.6 \pm 14.3$  yrs.;  
109 52 males, 18 females) and 60 sedentary healthy adults ( $51.5 \pm 10.4$  yrs.; 23 males, 37  
110 females). Swimmers were recruited from three different swimming pools of the same city.  
111 Inclusion criteria were having practiced swimming for at least 1 year, having an age  
112 greater than 30 years, and no particular cardiovascular pathologies that may affect  
113 physical exercise. On the other hand, for the control group, we recruited healthy people  
114 who have not practiced any specific physical exercise in the last year of their life, and an  
115 age greater than 30 years old.

116 The participants were asked to avoid smoking and drinking alcoholic or  
117 caffeinated beverage before the experimental procedures, and none of them was under  
118 medication. This study was approved by Bioethics Committee of our University, and all  
119 participants were informed of the benefits and risks of the investigation prior to signing  
120 an institutionally approved informed consent document to participate in the study [11].

### 121 *Procedures*

122           The participants compiled a questionnaire with different questions on their  
123 anthropometric, demographic, habits, training, and health data. Thereafter, we recorded  
124 blood pressure with participants placed in a supine position for 10 min in a quiet room  
125 with stable temperature (22°C; 52% of humidity), without speaking or making any  
126 movements, and with a respiratory frequency maintained at 12-15 breaths/min following  
127 the rhythm of a metronome (0.20-0.25 Hz). Participants wore a finger plethysmography  
128 for non-invasive continuous blood pressure monitoring (100 Hz, Portapres device Mod.  
129 2, The Netherlands), necessary to extract time series of beat-to-beat intervals and systolic  
130 as well as diastolic pressures. Time series were filtered to exclude artefacts and analyzed  
131 with Kubios HRV software (v. 2.0, 2008, University of Kuopio, Finland).

### 132 *Measurements*

133           Time domain HRV indices investigated were the square root of the mean squared  
134 differences of successive R-R intervals (RMSSD), and the standard deviation of normal  
135 to normal R-R intervals (SDNN). Spectral analysis provides two main frequency  
136 components: low frequency (LF) ranging from 0.04 to 0.15 Hz, and high frequency (HF)  
137 centered at the breathing frequency (0.15-0.4 Hz). It has been shown that HF is an index  
138 of vagal tone, whereas both sympathetic and vagal activities contribute to the LF of HRV  
139 [4]. In order to provide an index of sympathetic and vagal modulation, we expressed both  
140 LF and HF in normalized units [4]. Such normalized units are obtained by dividing the  
141 power of each component by total variance from which the very-low-frequency  
142 component had been subtracted and multiplying this value by 100 [ $\text{HF}/(\text{total power} -$   
143  $\text{VLF}) \times 100$ ], the same for LF. Although they have a mixed origin, the low and high  
144 frequency components measured in normalized units provide quantitative markers of  
145 cardiac sympathetic and vagal modulation, respectively [12, 13].

146 Baroreflex sensitivity was evaluated using Beatscope version 1.1a (TNO/BMI,  
147 The Netherlands), with a BRS add-on module based on cross-correlation analysis. This  
148 method is based on time domain of spontaneously occurring sequences of 4 or more  
149 consecutive beats characterized by either a progressive rise in systolic blood pressure  
150 (SBP) and R-R interval (+R-R /+SBP sequences) or by a progressive decrease in both  
151 measures (-R-R/-SBP sequences). The slope of the regression line between SBP and R-R  
152 interval changes is taken as an index of BRS modulation of heart rate (HR), same as the  
153 laboratory method based on injection of vasoactive drugs [14].

154 The pulse contour method of Wesseling (Modelflow method) was used to evaluate  
155 stroke volume (SV), cardiac output (CO), and total peripheral vascular resistance (TPR)  
156 from the blood pressure waveform [15].

### 157 ***Statistical Analysis***

158 For the analysis of autonomic function, the last 5 min of recordings was used for  
159 calculations, as recommended by guidelines for HRV analysis during short-term  
160 recording [4].

161 The normal distribution of data was verified with Shapiro-Wilk test. Paired sample  
162 t-tests were used to verify, for each parameter, the differences between groups. Effect size  
163 (ES) estimates were calculated by subtracting the means and dividing the result by the  
164 pooled standard deviation. Effect size values of <0.20, >0.20–0.50, >0.50–0.80, >0.80  
165 were considered to represent trivial, small, medium and large differences, respectively  
166 [16]. Mean differences and 95% confidence interval are also presented. The data were  
167 analyzed with SPSS v22.0 (SPSS, Chicago, USA).

168



169 **Results**

170 *Lifetime and current sport experience*

171 The total study sample consisted of 130 participants, divided in 70 swimmers and  
172 60 sedentary subjects. Table 1 displays the population characteristics, with differences  
173 and effect size between groups. The analysis showed a significant difference between  
174 groups for weight, height, number of cigarettes smoked in a day, and we found differences  
175 on how they perceived physical fitness, answering with “I am in a good shape” by 73%  
176 of swimming group with respect to 57% of sedentary participants. On average, the  
177 swimmers trained 3 times per week of 1 hour each, and they had been swimming without  
178 interruption for 14 years. Most of them (71%) practiced unsupervised swimming and 86%  
179 of them did sport since they were young. The control group was sedentary for an average  
180 13 years, with a high variability (standard deviation:17 years).

181 \*\*\*\*\* Table 1 near here \*\*\*\*\*

182 *Cardiac autonomic control indexes*

183 We found a significant difference between groups for blood pressure (mean  
184 arterial pressure,  $t(128)=-3.86$ ,  $p<0.001$ ,  $d=0.68$  (medium), mean diff.= -11.25, 95% CI  
185 [-17.02, -5.49]) and heart rate ( $t(128)=-5.16$ ,  $p<0.001$ ,  $d=0.91$  (large), mean diff.= -10.14,  
186 95% CI [-14.04, -6.25]) (Table 1). Conversely, we did not find significant difference  
187 between groups for BMI ( $t(128)=-0.86$ ,  $p=0.39$ , mean diff.= -0.51, 95% CI [-1.68, 0.66]).

188 In the time domain analysis of HRV, significant differences between groups were  
189 detected for SDNN ( $t(128)=2.14$ ,  $p=0.034$ ,  $d=0.38$  (small), mean diff.= 6.18, 95% CI  
190 [0.47, 11.89]) and RMSSD ( $t(128)=3.47$ ,  $p<0.001$ ,  $d=0.62$  (medium), mean diff.= 11.66,  
191 95% CI [5.01, 18.31]) (Figure 1A). In the frequency domain analysis, swimmers had  
192 higher HF ( $t(128)=2.65$ ,  $p=0.009$ ,  $d=0.47$  (small), mean diff.= 8.36, 95% CI [2.12, 14.59])

193 and lower LF power ( $t(128)=-2.65$ ,  $p=0.009$ ,  $d=-0.47$  (small), mean diff.= -8.37, 95% CI  
194 [-14.62, -2.13]) than sedentary subjects (Figure 1B).

195 \*\*\*\*\* Figure 1 near here \*\*\*\*\*

196 Swimmers showed higher cardiac output ( $t(128)=2.88$ ,  $p=0.005$ ,  $d=0.51$   
197 (medium), mean diff.= 0.71, 95% CI [0.22, 1.19]) and stroke volume ( $t(128)=5.08$ ,  
198  $p<0.001$ ,  $d=0.90$  (large), mean diff.= 20.61, 95% CI [12.58, 28.63]), meanwhile total  
199 peripheral resistances was lower ( $t(128)=-3.16$ ,  $p=0.002$ ,  $d=-0.55$  (medium), mean diff.=  
200 -0.37, 95% CI [-0.60, -0.14]) with respect to the sedentary group. We also found  
201 significant differences between the trained and sedentary subjects on BRS ( $t(128)=2.01$ ,  
202  $p=0.046$ ,  $d=0.35$  (small), mean diff.= 3.34, 95% CI [0.06, 6.63]) (Figure 2).

203 \*\*\*\*\* Figure 2 near here \*\*\*\*\*

## 204 **Discussion**

205 The results of this investigation suggest that the constant practice of recreational  
206 swimming for long time, 14 years on average in our subjects, have a strong effect on  
207 cardiac autonomic function evaluated by HRV and BRS. Indexes of cardiac vagal  
208 outflow, that is SDNN, RMSSD, and HF power, were clearly different in swimmers as  
209 compared to sedentary counterparts. Furthermore, differences were observed in BRS,  
210 which is a measure of reflex cardiac vagal responsiveness [17–20]. Loimaala et al., [10]  
211 suggest that, to obtain a clinically significant increase in HRV and BRS, exercise training  
212 should be practiced consistently for many years. For this reason, in this investigation, we  
213 have assessed swimmers exercising for long time to determine if that type of exercise  
214 elicited a change in the cardiac autonomic control in the healthy population.

215 In a recent study, Bessem et al. [2] showed that > 3 hours/week is the best  
216 minimum (current) level of exercise exposure to induce a significant increase in training-  
217 related ECG changes. Moreover, when a subject is exposed to over 3000 exercise hours  
218 (lifetime), the prevalence of training related ECG changes significantly increased  
219 compared to an exposure of <3000 hours. A limitation of that study was that, as in our  
220 study, data were collected retrospectively through a questionnaire. It is indeed very  
221 difficult to collect a big cohort of lifetime exposure to exercise in any other way.  
222 According to the current literature, the autonomic cardiovascular adaptations with aerobic  
223 endurance exercise training are associated with decreased cardiovascular risk factors, and  
224 reduced risk factors are associated with elevated autonomic vagal activity [21].

225 Previous studies have demonstrated that the practice of swimming decreased  
226 systolic blood pressure in adults >50 years of age. These decreases are obtained within a  
227 relatively short period, using a frequency and intensity of exercise that most healthy older  
228 adults are able to perform [22]. In addition, the hypotensive effects of swimming are  
229 accompanied by significant improvements in arterial compliance, endothelium-  
230 dependent vasodilation, and cardiovagal BRS, important factors of vascular functions that  
231 are closely related with the pathogenesis of cardiovascular diseases [23]. However,  
232 research on the effects of swimming on cardiovascular health profile is extremely limited,  
233 and in the most of cases are controversial [6]. We can hypothesize that these different  
234 cardiac autonomic responses after swimming training could be related to this training  
235 mode, which differs from other aerobic activity (i.e. running training) due to the body  
236 position in the water, breathing pattern, temperature regulation, the water (hydrostatic)  
237 pressure, which leads to a decrease in systemic vascular resistance [24–27]. To our  
238 knowledge, our study is the first showing the beneficial role of swimming in modulating

239 cardiac autonomic control by improving HRV, BRS, and hemodynamic variables.  
240 Prospective longitudinal cohort studies have shown that impaired cardiac autonomic  
241 control is a strong predictor of all-cause and cardiovascular disease mortality [28] and  
242 can be identified by evaluating indices of heart rate variability, baroreflex sensitivity, and  
243 hemodynamic parameters.

244 Maintenance of positive mental health and the prevention of mental disorders  
245 through regular physical activity has been a focus of considerable research [21]. It has  
246 been widely documented that aerobic exercise significantly alleviates the depressive state  
247 and reduces the level of cortisol and epinephrine excretions, as well as improving  
248 physiological fitness conditions. Regular physical exercise can promote a variety of  
249 psychological and physiological conditions and may be beneficial in the primary care of  
250 adolescent with depressive symptoms [29]. In our investigation, we found differences on  
251 subjective feeling as the 73% of swimming group answered “I am in a good shape” with  
252 respect to 57% of sedentary participants [30]. The effect of physical activity on depressive  
253 symptoms and anxiety is well-established, although dose–response issues still need  
254 experimental research with a prevention-based focus. Moderate to vigorous physical  
255 activity was associated with better self-reported health-related quality of life in healthy  
256 adults [7]. Much of the general population accepts the importance of being physically  
257 active for health and well-being. Moreover, it is widely believed that participation in sport  
258 and physical activity during youth can be a significant factor in lifelong engagement  
259 therein [31]. In fact, most of our participants (86%) have been practiced sport since they  
260 were young (see Table 1). It is widely believed that active participation in sports during  
261 youth is an important prerequisite for adult involvement in physical activity. For this  
262 reason, school physical education is, in turn, often showed as a potential intermediary for

263 enhancing young people's engagement with physically active recreation in their leisure  
264 and, in the longer run, over the lifetime [32].

265 Furthermore, sedentary adolescents have altered body size/composition, and this  
266 has also been associated with subsequent adult health outcomes. In general, unfit subjects  
267 within a fatness category (low or high BMI) had a higher cardiovascular disease risk  
268 profile than their fit counterparts. In our investigation, we did not find significant  
269 differences between groups for BMI. This means that normal BMI with low-level of  
270 cardiovascular fitness has higher probability to develop cardiovascular disease as unfit  
271 subjects with high BMI. In a recent follow-up study, Cornwell et al., [33] found that the  
272 risk of heart failure in elderly was significantly lower among overweight fit and obese-fit  
273 compared with lean and low fit individuals. Taken together, these findings suggest that  
274 higher levels of cardiorespiratory fitness and physical activity are inversely associated  
275 with long-term risk of heart failure across all BMI categories.

## 276 **Conclusions**

277 In conclusion, this retrospective investigation provides convincing evidence of the  
278 significant protective role of moderate-physical activity against the risk of premature  
279 mortality, cardiovascular disease, stroke, hypertension. In many instances, the dose-  
280 response relationship is linear with further health benefits with increasing levels of  
281 activity. As a practical recommendation, we would suggest the use of  $\geq 200$  minutes/week  
282 practicing swimming as a minimum value. We also showed that a lifetime exposure of  
283  $>1$  year of swimming is necessary to elicit cardiac autonomic response, changes in the  
284 baroreflex sensitivity, and hemodynamic activity in healthy population.

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378 **Figure and table legends**

379 **Figure 1.** Histograms represent mean ( $\pm$ SD) of the time (A) and frequency (B) domain  
380 of HRV parameters between swimming (black) and control (grey) group.

381 *Abbreviations:* SDNN - standard deviation of normal to normal R-R intervals; RMSSD -  
382 square root of the mean squared differences of successive R-R intervals; HF – high  
383 frequency; LF – low frequency. Asterisks represent significant differences ( $p<0.05$ ).

384 **Figure 2.** Histograms show the mean ( $\pm$ SD) of hemodynamic variables between  
385 swimming (black) and control (grey) group. Asterisks represent significant differences  
386 ( $p<0.05$ ).

387 **Table 1.** Population characteristics. Data are showed as mean  $\pm$  standard deviation and  
388 number with percentage value between brackets. Bolded text denotes a significant main  
389 effect ( $p<0.05$ ) with effect size values (Cohen's d).