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Influencing Factors for Maximum Heart Rate Achieved for Cardiac Patients

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COLUMN ARTICLE

Abstract

The report has examined the determinants of the maximum heart rate achieved (MHRA) for 303 heart patients based on probabilistic modeling. It is observed that mean MHRA is lower for older patients ($P < 0.0001$) than younger. It is higher for patients with atypical angina ($P = 0.0139$) and non-anginal pain or asymptomatic ($P = 0.0690$) than patients with typical angina. It increases if the resting blood pressure ($P = 0.0213$) increases. It also increases if the serum cholesterol level ($P = 0.0325$) increases. It is higher for the patients having no exercise induced angina ($P = 0.0014$) than the patients with having it. It is higher for the patients with fixed defect ($P = 0.0053$) and reversal defect ($P = 0.0114$) than normal. Variance of MHRA is higher at older ages ($P = 0.0013$) than younger. It increases if the serum cholesterol level ($P = 0.0058$) decreases. In addition, MHRA variance is higher for the patients with typical angina than patients with atypical angina ($P = 0.0089$) and non-anginal pain or asymptomatic ($P = 0.0707$).

Keyword: Blood Pressure; Chest Pain; Non-Constant Variance; Probabilistic Modeling; Serum Cholesterol Level

Associated factors for maximum heart rate achieved

Generally, it is very difficult to identify the individual heart rate changes causal factors by a heart specialist. In practice, there are many factors which influence heart rate to speed up, or slow down, or vary inexplicably. These factors are known as heart rate determinants which are not completely recognized in the heart disease literature [1,2]. The peak, maximum and basal heart rate measurement values are generally used in clinical medicine and physiology [3-5]. In practice, the percentage of maximum, or peak, or a fixed percentage of heart rate is applied to prescribe the exercise intensity, or medicine in both the disease prevention and the rehabilitation programs [6,7]. It is well known that a slow heart rate extraction during the first and second minute after severe exercise is an independent predictor of overall mortality [8,9]. The peak, or basal, or maximum heart rate is broadly applied as a criterion for acquiring peak exertion in the identification of maximal aerobic capacity [4,10,11].

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The current report seeks the following queries: What are the determinants of maximum heart rate achieved? What are the effects and the associations of the determinants with MHRA? These queries are examined in the report with the help of a real data set containing 303 heart patients with 14 study characters which is given in UCI machine learning repository. Interested readers can observe the data set, and the data collection method in UCI machine learning repository. It is not reported herein. The 14 study characters are as follow: Age (in years), Sex (1 = male; 0 = female), Chest pain (CP) (1 = typical angina; 2 = atypical angina, 3 = non-anginal pain or asymptomatic), Resting blood pressure (Trestbps) (in mm Hg on admission to the hospital), Serum cholesterol (Chol) (in mg/dl), Fasting blood sugar (Fbs) ((Fbs> 120 mg/dl) (1 = true; 0 = false)), Resting electrocardiographic (Restecg) (resting electrocardiographic results -- value 0 = normal; 1 = having ST-T wave abnormality (T wave inversions and/or ST elevation or depression of > 0.05 mV)), Maximum heart rate achieved (Thalach), Exercise induced angina (Exang) (1 = yes; 0 = no), ST depression induced by exercise relative to rest (Oldpeak), Slope (the slope of the peak exercise ST segment -- value 1 = upsloping, 2 = flat, 3 = downsloping), Ca (number of major vessels (0-3) colored by fluoroscopy), Thal (3 = normal; 6 = fixed defect; 7 = reversable defect), Target (num: diagnosis of heart disease (angiographic disease status) value 0: < 50% diameter narrowing; 1:> 50% diameter narrowing (in any major vessel: attributes 59 through 68 are vessels)). Note that most the physiological data sets are heterogeneous and non-normally distributed. They are generally modelled using joint generalized linear models (JGLMs) under Log-normal and Gamma distribution [12-15]. The present report has derived probabilistic model of the maximum heart rate achieved using JGLMs under both the above distributions.

The principal purpose of the current report is to derive the relationship of MHRA (maximum heart rate achieved) (Thalach) with the remaining other 13 study characters. The considered data set is a multivariate data, and the random study variable is MHRA. The association of MHRA is only can be derived through probabilistic modeling. Note that the response MHRA is positive, continuous, heteroscedastic, and non-normally distributed. So, it may be modeled by using suitable transformation when the variance is stabilized under the transformation. When the variance is not stabilized, it can be modeled by JGLMs under Log-normal and Gamma distributions, which are clearly presented in [12-15]. For detailed views about JGLMs, interested readers can visit [12,15].

The MHRA is the focused response random variable which is detected as heteroscedastic, and it is not stabilized by any appropriate transformation. MHRA is considered as the response (or dependent) variable, and the remaining other 13 study characters are considered as the explanatory variables. MHRA has been modeled by applying JGLMs under both the Log-normal and Gamma distributions. The appropriate models have been accepted based on the smallest value of Akaike information criterion (AIC) (within each class) which minimizes both the squared error loss and predicted additive errors [16, p. 203-204]. All the included factors in the mean and variance models are statistically significant.

MHRA analysis results are placed in table 1, which shows that Gamma fit (AIC = 2602.685) is better than Log-normal fit (AIC = 2611). The final Gamma fitted MHRA models (Table 1) show the following outcomes. It is observed that mean MHRA is lower for older patients (P < 0.0001) than younger. It is higher for patients with atypical angina (P = 0.0139) and non-anginal pain or asymptomatic (P = 0.0690) than patients with typical angina. It increases if the resting blood pressure (P = 0.0213) increases. It also increases if the serum cholesterol level (P = 0.0325) increases. It is higher for the patients having no exercise induced angina (P = 0.0014) than the patients with having it. It is higher for the patients with fixed defect (P = 0.0053) and reversal defect (P = 0.0114) than normal. Variance of MHRA is higher at older ages (P = 0.0013) than younger. It increases if the serum cholesterol level (P = 0.0058) decreases. In addition, MHRA variance is higher for the patients with typical angina than patients with atypical angina (P = 0.0089) and non-anginal pain or asymptomatic (P = 0.0707).

Gamma fitted MHRA mean ($\hat{\mu}$) model (from table 1) is

$\hat{\mu}$ = exp. (5.0286 - 0.0060 Age + 0.0490 Chest pain2 + 0.0330 Chest pain3 + 0.0010 Resting BP + 0.0002 Cholesterol -0.0585 Exercise induced angina + 0.0919 Thal2

+ 0.0852 Thal3 + 0.0628 Target), and the Gamma fitted MHRA variance ($\hat{\sigma}^2$) model is $\hat{\sigma}^2 = \exp.$ (-4.0324 + 0.0320 Age -0.6820 Chest pain2 - 0.3818 Chest pain3 - 0.0054 Cholesterol - 0.6336 Target).

The mean and variance MHRA models are presented above by two equations. It is observed that mean MHRA is explained by Age, Chest pain, Resting BP, Cholesterol, Exercise induced angina, Thal and Target, while its variance is explained by Age, Chest pain, Cholesterol and Target.

In data analysis, the valid interpretations can only be drawn from the data derived probabilistic model which is tested to be true. Thus, the derived model should be verified applying model diagnostic tools. So, model diagnostic graphical tools are applied for the final selected Gamma fitted MHRA models (Table 1), which is revealed in figure 1. The absolute residuals are plotted against the MHRA Gamma fitted values (Table 1) in figure 1a, which is nearly linear, implying that variance is constant with the running means. Figure 1b displays the mean Gamma fitted MHRA normal probability plot (Table 1), which does not reveal any lack of fit. Thus, both the figures establish that the Gamma fitted MHRA models (Table 1) are clearly approximate form of the unknown true models.

Model	Covariate	Gamma model				Log-normal model			
		Estimate	s.e	t-value	P-value	Estimate	s.e	t-value	P-value
Mean model	Constant	5.0286	0.07072	71.103	< 0.0001	5.0071	0.07166	69.872	< 0.0001
	Age	-0.0060	0.00077	-7.784	< 0.0001	-0.0062	0.00078	-7.959	< 0.0001
	Chest pain2	0.0490	0.01980	2.474	0.0139	0.0530	0.02001	2.649	0.0085
	Chest pain3	0.0330	0.01807	1.825	0.0690	0.0358	0.01831	1.952	0.0519
	Resting BP	0.0010	0.00041	2.314	0.0213	0.0010	0.00042	2.412	0.0165
	Cholesterol	0.0002	0.00011	2.149	0.0325	0.0003	0.00011	2.365	0.0187
	Exr. indu. Angina	-0.0585	0.01817	-3.219	0.0014	-0.0576	0.01842	-3.128	0.0019
	Thal2	0.0919	0.03269	2.812	0.0053	0.0980	0.03318	2.953	0.0034
	Thal3	0.0852	0.03346	2.547	0.0114	0.0918	0.03399	2.701	0.0073
	Target	0.0628	0.01960	3.202	0.0015	0.0682	0.01991	3.427	0.0007
Dispersion model	Constant	-4.0324	0.7211	-5.592	< 0.0001	-4.0468	0.7288	-5.553	< 0.0001
	Age	0.0320	0.0099	3.236	0.0013	0.0336	0.0100	3.371	0.0008
	Chest pain2	-0.6820	0.2591	-2.632	0.0089	-0.6962	0.2590	-2.688	0.0076
	Chest pain3	-0.3818	0.2105	-1.814	0.0707	-0.3833	0.2103	-1.823	0.0693
	Cholesterol	-0.0054	0.0019	-2.779	0.0058	-0.0055	0.0019	-2.842	0.0048
	Target	-0.6336	0.1979	-3.201	0.0015	-0.6438	0.1982	-3.249	0.0013
AIC		2602.685				2611			

Table 1: Results for mean and dispersion models for maximum heart rate achieved from Gamma and Log-Normal fit.

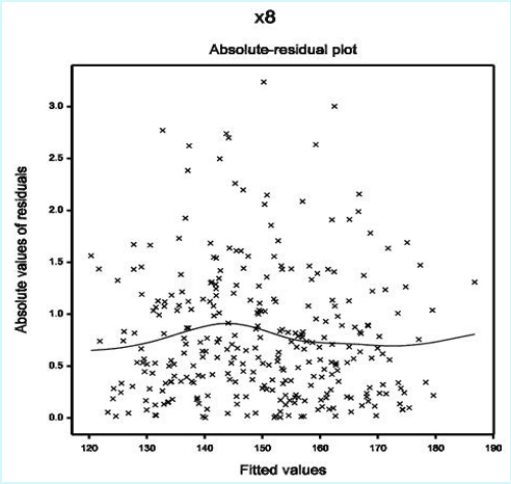

Figure 1a

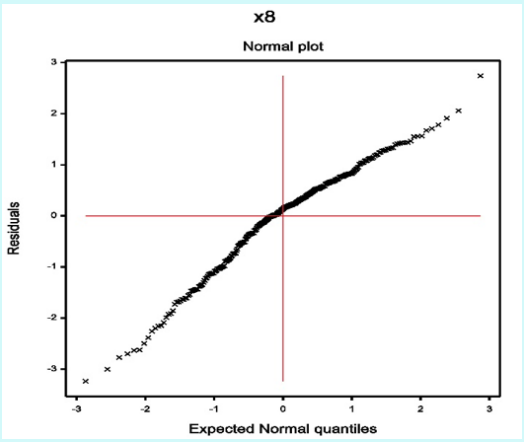

Figure 1b

Figure 1: For the joint Gamma fitted models of maximum heart rate (Table 1), the (a) absolute residuals plot with respect to the fitted values, and (b) the normal probability plot for the mean model.

Final Gamma fitted MHRA models are given above (Table 1) which interpret the following:

1. The mean MHRA is negatively associated with Age (P < 0.0001), indicating that MHRA is higher at younger ages than older which is observed in practice.
2. Mean MHRA is positively associated with Chest pain (1 = typical angina; 2 = atypical angina, 3 = non-anginal pain or asymptomatic) at level 2 (P = 0.0139) and level 3 (P = 0.0690), concluding that MHRA is higher for the patients with atypical angina, or non-anginal pain, or asymptomatic than patients with typical angina.
3. Mean MHRA is positively associated with Resting BP (P = 0.0213), concluding that MHRA increases as resting BP increases.
4. Mean MHRA is positively associated with Cholesterol (P = 0.0325), indicating that MHRA increases as cholesterol increases.
5. MHRA is negatively associated with Exercise induced angina (1 = yes; 0 = no) (P = 0.0014), interpreting that MHRA is higher for the patients having no exercise induced angina than the patients with having it.
6. MHRA is positively associated with Thal (3 = normal; 6 = fixed defect; 7 = reversable defect) at level 6 (P = 0.0053) and level 7 (P = 0.0114), implying that MHRA is higher for the patients with fixed defect and reversal defect than normal.
7. MHRA is positively associated with Target (num: diagnosis of heart disease (angiographic disease status) value 0: < 50% diameter narrowing; 1: > 50% diameter narrowing (in any major vessel: attributes 59 through 68 are vessels)) (P = 0.0015), concluding that MHRA is higher for the patients with angiographic disease status with > 50% diameter narrowing than others.
8. Variance of MHRA is positively associated with Age (P = 0.0013), concluding that MHRA variance is higher at older patients than younger.
9. MHRA variance is negatively associated with Chest pain, concluding that MHRA variance is higher for the patients with typical angina than patients with atypical angina (P = 0.0089) and non-anginal pain or asymptomatic (P = 0.0707).
10. MHRA variance is negatively associated with Cholesterol (P = 0.0058), concluding that it increases as cholesterol level decreases.
11. MHRA variance is negatively associated with Target (P = 0.0015), indicating that it is higher for the patients with angiographic disease status with < 50% diameter narrowing than others.

The above conclusions are derived based on both JGLMs under Log-normal and Gamma distributions. Note that both the models have the identical interpretations. In addition, the standard error of the estimates are very small, concluding that estimates are stable. AIC and graphical diagnostic tools indicate the appropriate models which present the above interpretations. It is observed that some of the above conclusions are observed in practice. Cardiac patients and heart specialists will be benefitted from the report. Maximum heart rate achieved is to be cared at older ages along with resting blood pressure, cholesterol level, chest pain status.

CONFLICT OF INTEREST

The authors confirm that this article content has no conflict of interest.

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