



UNIVERSITY OF PADOVA

Department of General Psychology

Master's Degree in Cognitive Neuroscience and Clinical Neuropsychology

Final dissertation

**Drive at the rhythm of your own heart: a study on Heart Rate
Variability, cognitive functioning and driving performance in
Ferrari Driver Academy drivers.**

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0. Abstract

Racing driving requires the development of extraordinary sensorimotor skills to deliver high-level peak performances in complex environments characterised by multiple stressors, draining drivers' physiological and cognitive resources. Although previous research provided evidence in favour of the role played by the Autonomic Nervous System (ANS) and different cognitive and executive functions in supporting the delivery of a high-level driving performance, further research is needed to deepen our understanding of the exact mechanisms linking physiological and psychological resources to the behavioural outcomes of driving. We adopted an evidence-based theoretical model (*i.e.*, the Neurovisceral Integration Perspective; Thayer and Lane, 2000; Thayer et al., 2009; Thayer et al., 2012) and validated techniques and tools, to investigate, in a sample of elite racing drivers mainly scouted for the Ferrari Driver Academy, the relationship between HRV parameters, indexing the individual availability of physiological resources, and a set of measures of cognitive functions thought to be relevant for driving, including non-executive (simple reaction times) and executive (inhibitory control and WM) ones. We also tried to elucidate whether and how these physiological and cognitive variables can be used to predict driving performance, measured using a very ecological task in a realistic driving simulator.

Based on previous research, we hypothesised that: (a) time-domain HRV indices of parasympathetic cardiac control would be positively associated with measures of inhibitory control (*i.e.*, the performance at a Go/NoGo task) and WM (*i.e.*, the performance at an N-Back task), but not with those of general readiness (*i.e.*, the performance at an SRT task); (b) that driving performance (as indexed by the best and average lap times recorded) would be predicted by HRV indices, as well as by measures of inhibitory control.

The results showed a significant negative correlation between cardiorespiratory coherence and the percentage of commissions at the Go/NoGo task, a negative correlation between coherence and the lap times recorded by the drivers, and a positive correlation between the latter and the mean reaction times (RTs) at the Go trials of the Go/NoGo task. Finally, linear models including coherence, the percentage of commissions at Go/NoGo

and the mean RTs at Go trials as independent variables, proved to be able to explain a significant amount of variance in driving performance.

Our results replicated some findings previously reported in psychophysiology, cognitive psychology, neuropsychology and sport psychology, extending them to the field of motorsport, and provided further support to the Neurovisceral Integration Perspective. Finally, the linear models developed proved to be able to explain a significant amount of variability in peak driving performance in elite racing drivers, providing a useful tool for their assessment and scouting, as well as for future studies in the field.

1. Heart Rate Variability

1.1 What is Heart Rate Variability

Heart Rate Variability (HRV) refers to a group of indexes calculated from the oscillations in the interval between consecutive heart beats (*i.e.*, beat-to-beat changes in RR intervals; Fig. 1; Task Force, 1996). Some HRV indexes reflect the amount of HR fluctuations around the mean HR and are mainly influenced by the extrinsic regulation of the heart rate (HR), especially by the interplay among the activity of the sympathetic and parasympathetic branches of the autonomic nervous system (SNS; PNS; Acharya et al., 2006; Cygankiewicz and Zareba, 2013; Task Force, 1996; see *1.2.1 Cardiovascular function* and *1.2.2 The influence of the SNS and PNS on cardiovascular function*). The clinical relevance of this phenomenon has been known for a long time, starting in the 60s with studies on HRV alterations occurring due to foetal distress (Lee and Hon, 1965), whereas the existence of intrinsic physiological rhythms influencing beat-to-beat variations of the HR signal was discovered in the 70s, underlying the role played by the respiratory cycle (Hirsch and Bishop, 1981; Luczak and Laurant, 1973; Saykrs, 1973).

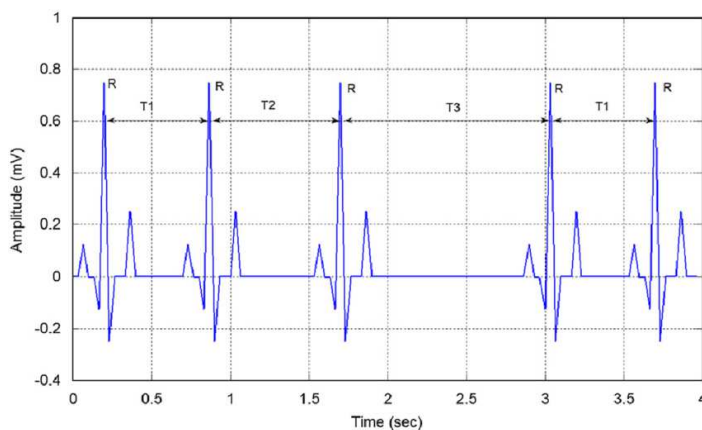


Fig. 1 - Sankari and Adali, 2011. Example of an ECG recording and beat-to-beat changes in RR intervals.

During the last 5 decades, a consistent body of research found reduced HRV to be linked with different diseases and disorders, and to correlate with a higher mortality rate, suggesting the idea that HRV may be used as an index of the state of health and adaptability of the organism (Cygankiewicz and Zareba, 2013; Litscher et al., 2014; Wolf et al., 1977). In particular, reduced HRV has been found in populations affected by

conditions involving the cardiocirculatory system, such as post-infarction patients (Kleiger et al., 1987; Malik et al., 1989), as well as those with cardiomyopathies and congestive heart failures (Boveda et al., 2001; La Rovere et al., 2003; Nolan et al., 1998; Ponikowski et al., 1997). Most importantly, HRV parameters were found to be useful tools in predicting the worsening of the aforementioned pathologies and the total mortality related to them (Cygankiewicz and Zareba, 2013). Reduced HRV was also found to characterise non cardiovascular conditions such as diabetes mellitus (Bernardi et al., 1992; Boulton et al., 2005; Lefrandt et al., 2010; Schonauer et al., 2008; Tesfaye et al., 2010), cerebrovascular diseases (*i.e.*, strokes) (Barron et al., 1994; He et al., 2010; Korpelainen et al., 1997; Korpelainen et al., 1999; Orlandi et al., 2000), and those conditions that do not primarily affect the cardiocirculatory system, such as multiple sclerosis (MS) (Mahovic and Lakusic, 2007; McDougall and McLeod 2003; Merkelbach et al., 2006), muscular dystrophies (*e.g.*, Duchenne and Becker) (Hermans et al., 2010; Schoser et al., 2004; Yotsukura et al., 1995), Parkinson's disease (PD) (Turkka et al., 1987; Van Dijk et al., 1993) and epilepsy (Evrengül et al., 2005), as well as psychiatric conditions such as depression (Kemp et al., 2010) and psychopathology (Beauchaine and Thayer, 2015). These findings, along with the evidence coming from studies showing an increased HRV in trained versus detrained subjects (Hansen et al., 2004) and a beneficial effect of physical exercise on the risk of cardiovascular mortality and sudden cardiac death in patients who underwent myocardial infarction (O'Connor et al., 1989), suggest that HRV is a marker of good health, lower risk of mortality and, above all, of positive adaptability.

1.2 Physiological underpinnings of HRV and the contribution of the sympathetic and parasympathetic autonomic system

1.2.1 Cardiovascular function

The cardiovascular system consists of a closed system connecting the heart to a complex apparatus made of multiple blood vessels, including arteries, veins and capillaries (Gordan, Gwathmey & Xie, 2015). The heart serves through its rhythmic contractions the

specific purpose of maintaining the homeostasis by pumping oxygenated blood to the body, in order to supply nourishment to the cells, and deoxygenated blood to the lungs (Gordan, Gwathmey & Xie, 2015). The heart contains the so-called autorhythmic cardiac cells (or myocytes), mainly located in the sinoatrial (SA) node, which coordinate the electric activity of the heart, giving rise thanks to their unstable membrane potential to its continuous and rhythmic pulsations, that can be measured with the electrocardiogram (ECG; Gordan, Gwathmey & Xie, 2015; Stauss, 2003; Tiwari et al., 2021). A normal ECG recording shows a P wave followed by a QRS complex and a T wave (Fig. 2), reflecting the sequence of electrical and mechanical events involving the heart, starting with the depolarisation of the pacemaker cells in the SA node. The P wave corresponds to the atrial depolarisation followed by atrial contraction, the QRS complex reflects the sequence of ventricular depolarisation and ventricular contraction. Finally, the T wave mirrors the ventricular repolarisation and ventricular relaxation. This sequence of events happens autonomously from the control of the upper centres, thanks to the activity of the intrinsic cardiac nervous system, which gives rise to the heartbeats due to the rhythmic discharge of the pacemakers located in the SA node and the transmission of the action potentials generated there to the other muscular cells constituting the heart's walls (Gordan, Gwathmey & Xie, 2015; Kingma, Simard, Rouleau, 2017). Importantly, in order for the heart to be able to function adaptively and to work effectively, the HR and the cardiac output must be continuously and flexibly tuned according to the body's cells' need for oxygen and nutrients, in a timely manner (Gordan, Gwathmey & Xie, 2015).

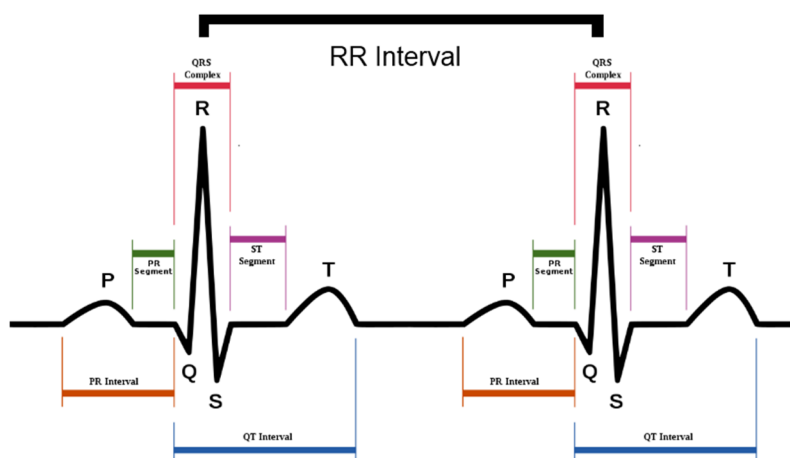


Fig. 2 - Tawakal et al., 2012. A representation of two consecutive QRS complexes, each preceded by a P and followed by T wave, and of the R-R interval among them.

While the ANS allows the rapid adjustment of cardiac function according to the changing requirements of the body, also the central nervous system (CNS) and the endocrine system contribute to regulate the cardiac activity. In absence of any extrinsic influence by the aforementioned systems, the baseline HR set by the activity of the intrinsic cardiac nervous system would be around 100 beats per minute (BPM), and therefore higher than the average resting HR found in healthy subjects (about 60-80 bpm), which is constantly modulated by the ANS and under prevailing parasympathetic activity (Gordan, Gwathmey & Xie, 2015). Moreover, the fundamental role played by the ANS in the modulation of the HR and the genesis of HRV is supported by findings reported by multiple studies on patients who recently underwent heart transplantation, and whose heart is therefore temporarily extrinsically denervated, in which there is a temporary increase of resting HR and the abolishment of beat-to-beat variability (Beckers et al., 2004; Mortara et al., 1994; Toledo et al., 2002).

1.2.2 The influence of the SNS and PNS on cardiovascular function

The ANS is composed of two main branches (Fig. 3) – *i.e.*, the SNS and the PNS – which have antagonist effects on the body and on the heart. The effect generated by the ANS on the heart is, at any time, the net balance between the opposing action of its two components. The SNS is the system responsible for the so-called “fight-or-flight” response, which serves to prepare the body to act under stressful conditions, while the PNS is in charge of controlling the functioning of the internal organs at rest, also lowering the resting HR well below the one set by the pacemaker cells of the SA node, keeping it in the 60-75 bpm range.

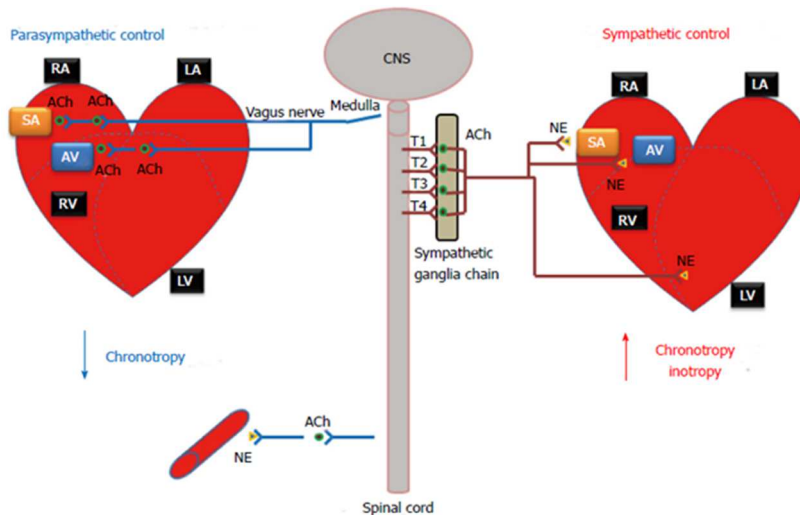


Fig. 3 - Gordan, Gwathmey & Xie, 2015. The ANS structure and the regulatory mechanisms of the heart function, with the pathways used by the ANS to influence the force and rate of heart contractions. CNS: Central nervous system; RA: Right atria; LA: Left atria; RV: Right ventricle; LV: Left ventricle; SA: Sino-atrial node; AV: Atrioventricular node; NE: Norepinephrine; ACh: Acetylcholine.

Concerning the SNS, due to the presence of post-ganglionic noradrenergic fibres, the main neurotransmitter released by its postganglionic neurons is norepinephrine (NE), interacting with β_1 , β_2 , α_1 , and α_2 receptors located in the heart (Tab. 1; Fig. 3), and especially in the atrioventricular (AV) node (Brack, Coote & NG, 2004; Gordan, Gwathmey & Xie, 2015). Overall, the activation of the SNS with the release of NE into the cardiovascular system leads to 4 main effects (Tab. 1): (a) a positive chronotropic effect – *i.e.*, an increase in HR – (b) a positive inotropic effect or an increase of myocardial contractility, (c) a positive dromotropic effect, or an enhancement of conductivity within the heart, and (d) a reduction of HRV (Gordan, Gwathmey & Xie, 2015; Kiyono et al., 2017).

	Heart				Vessels	
	Receptor	Function			Receptor	Function
		Inotropy	Chronotropy	Dromotropy		
Norepinephrine	α_1	+	+	+	α_1	Vasoconstriction
	β_1	+	+	+	β_1	Vasoconstriction
	β_2	+	+	+	β_2	Vasodilation
Acetylcholine	M_2	-	-	-	M_2	Vasodilation

Tab. 1 – Gordan, Gwathmey & Xie, 2015 – Sympathetic and parasympathetic receptors and their effects on the heart and vessels

In regard to the PNS, it exerts its effects on the cardiovascular system mainly via the efferent branch of the vagus nerve and the release of acetylcholine (ACh) by the post-ganglionic neurons, which activates M_2 and M_3 muscarinic receptors (Fig. 3 and Fig. 4) mainly targeting the SA node (Brack, Coote & Ng, 2004; Gordan, Gwathmey & Xie, 2015). Overall, this mechanism triggers (Fig. 3 and Tab. 1) (a) a negative chronotropic

effect, (b) a negative inotropic effect, (c) a negative dromotropic effect, and (d) an increase in HRV, but it has little effect on myocardial contractility (Goldberger et al., 2001; Gordan, Gwathmey & Xie, 2015; Tiwari et al., 2021).

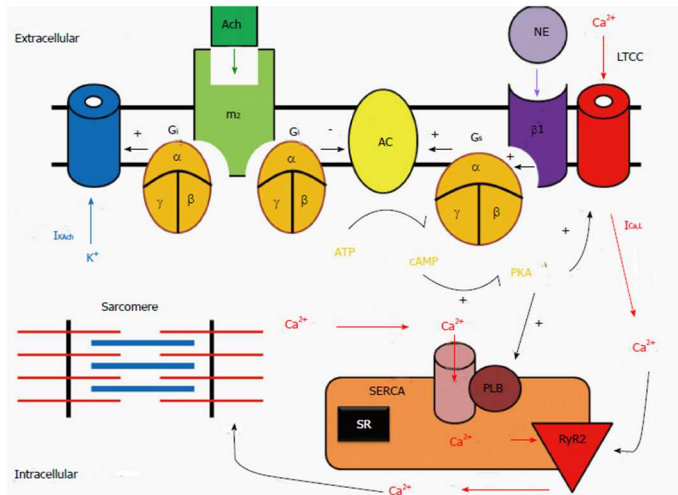


Fig. 4 - Gordan, Gwathmey & Xie, 2015. Signal transduction systems for β -adrenergic and muscarinic receptors stimulations in a cardiac myocyte.

NE: Norepinephrine; β 1: Beta1-adrenergic receptor; Gs: Stimulatory G-protein; ACh: Acetylcholine; m2: Type-2 muscarinic receptors; Gi: Inhibitory G-protein; AC: Adenylate cyclase; PKA: Protein kinase A; ICaL: L-type Ca channel; RyR2: Ryanodine receptor 2; SERCA: Sarcoplasmic reticulum Ca²⁺-ATPase2a; PLB: Phospholamban.

Finally, multiple reflex arcs interact with the ANS in order to regulate cardiovascular functioning. In particular, they comprise baroreflexes (involved in the regulation of blood pressure by modulating cardiac output and the peripheral resistance of the blood vessels), chemoreceptors reflexes (sensitive to the pH of the blood and the concentration of CO₂), and endocrine/paracrine reflexes (which contributes to the regulation of blood pressure through the release of different hormones) (Gordan, Gwathmey & Xie, 2015).

1.2.3 The contribution of the SNS and PNS to HRV

As mentioned before, adjustments in the cardiac activity are necessary to provide nourishment to the cells of the body under constantly changing conditions and to maintain homeostasis. HRV can therefore be conceptualised as the response put in place by the body and particularly the ANS to adjust cardiac functioning, adapting it to the changes occurring to the internal and external environment by dynamically modulating the interplay among the SNS and the PNS (Tiwari et al., 2021). In particular, the research group led by Massimo Pagani developed through the years a model of ANS influence on HRV, that initially became widely accepted by the scientific community, although refuted later on. It comprised 3 core assumptions: (a) cardiac parasympathetic tone can be

indexed by the power of the HF component of the HRV spectrum, (b) cardiac sympathetic outflow can be assessed through the LF component, and (c) the LF-to-HF ratio can be used to measure sympathovagal balance (Malliani et al., 1991; Montano et al., 2009; Pagani et al., 1986). This model is based on the concept of autonomic reciprocity, according to which both branches of the ANS are under reciprocal central nervous control, implying that by increasing the activation of a component, the activation of the other one is decreased (Reyes Del Paso et al., 2013). Although initially widely accepted by the scientific community, this paradigm has been proven to be oversimplistic by multiple modern studies, above all for what concerns the indexes of cardiac sympathetic activity (Kiyono et al., 2017; Reyes Del Paso et al., 2013).

As previously discussed, the effects of parasympathetic activity on the heart functioning are mainly mediated by the efferent branch of the vagus nerve. In particular, efferent vagal activity is considered to be a major contributor to the HF component of the HRV power spectrum (Berntson et al., 1997; Task force, 1996; Malliani et al., 1991). A consistent body of research supports this notion, relying on multiple clinical and experimental findings. For instance, multiple studies performed under controlled respiration at frequencies within the 0.20-0.30 Hz range, known to be associated with enhanced vagal modulation of the cardiac activity (Pagani et al., 1986; Pomeranz et al., 1985), reported an increase in HF measures of HRV and a decrease in the LF-to-HF ratio as compared to baseline (Hayano et al., 1994; Saul et al., 1990; Pomeranz et al., 1985; Shannon, Carley & Benson, 1987). It is also important to note that, at rest, vagal tone prevails and variations in the beat-to-beat intervals are mainly dependent on parasympathetic modulation (Chess, Tam & Calaresu, 1975).

Moreover, multiple systematic reviews reported the existence of a negative relationship between PNS activity and HR and a positive one among the former and HRV (Tiwari et al., 2021). On the one hand, this widely accepted view is supported also by some pharmacological studies, such as the one conducted by Bloomfield and colleagues in 1998. In this research the authors gradually manipulated the level of parasympathetic vagal control on cardiac activity by administering to healthy subjects multiple doses of intravenous phenylephrine, a substance known to increase baroreflex-mediated PNS activity. Findings reported a dose-dependent increase in multiple parameters of HRV –

i.e., RMSSD, pNN50 and HF power – known to be associated with PNS activity, following phenylephrine injection (results shown in Fig. 4, Fig. 5 and Fig. 6).

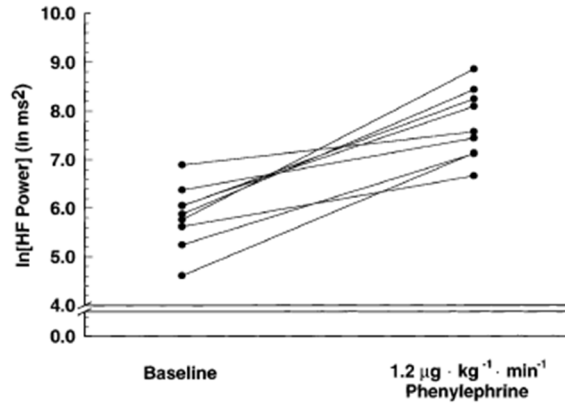
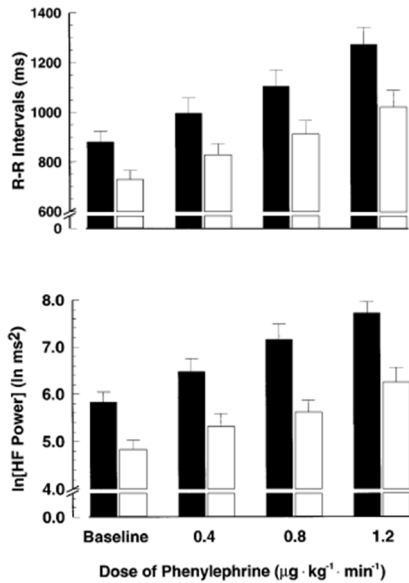


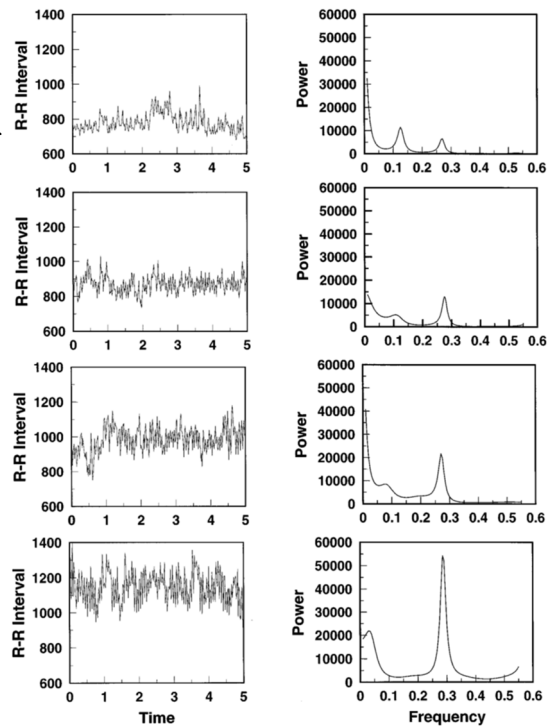
Fig. 5 - Bloomfield et al., 1998. Individual data showing the changes in ln[HF Power] for all 9 subjects from the supine baseline state to supine infusion of highest dose of phenylephrine (1.2 $\mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) condition. All 9 subjects showed an increase in their values for ln[HF power] during phenylephrine infusion.

Fig. 4 - Bloomfield et al., 1998. Changes in R-R intervals (top) and ln[HF Power] (bottom) in the supine (solid bars) and head-up tilt position (open bars) for each dose of phenylephrine compared to baseline. Increasing doses of phenylephrine lead to a progressive increase in R-R intervals and ln[HF Power] in both the supine position and in head-up tilt position. Note that the relationship between supine and head-up tilt values for both R-R intervals and ln[HF Power] is the same for each dose of phenylephrine. <-

Fig. 6 - Bloomfield et al., 1998. R-R intervals (left) and power spectra (right) for baseline state (top panels) and for increasing doses of phenylephrine infusion (remaining 3 sets of panels, top to bottom).

Left: R-R interval tachograms from one subject at baseline (supine) and during supine phenylephrine infusion. Note a dose-dependent increase in mean R-R interval and in the amplitude of R-R interval cyclic fluctuations following increasing doses of phenylephrine.

Right: power spectra for each tachogram of R-R intervals. With increasing doses of phenylephrine, increases in the amplitude of the cyclic fluctuation of R-R interval at respiratory frequency occur, appearing in the power spectra as an increase in the area under curve in HF bandwidth (0.15–0.40 Hz) ->



On the other hand, a new alternative perspective on the relationship between PNS activity and HRV has been proposed by Goldberger and colleagues. Their research group suggested that such a relationship might not be linear, and that it could be instead better described by a concave-down quadratic function, able to account not only for the initial increase in HRV coupling the one in PNS function, but also for the saturation of the HRV response occurring with a very high parasympathetic activation. This model is supported for instance by their study published in 2001, in which they investigated the effects of PNS stimulation and withdrawal on HRV, after an intravenous injection of propranolol, a substance inducing β -adrenergic blockade and the inhibition of the effects of SNS activity on HRV (Goldberger et al., 2001). Results showed that (a) HRV increases following PNS stimulation until it reaches a plateau level and that (b) beyond the plateau level HRV decreases following additional PNS stimulation, fitting the aforementioned quadratic model (shown in Fig. 7). Moreover, they found marked inter-individual differences in the relationship assessed, also influenced by an age-related effect.

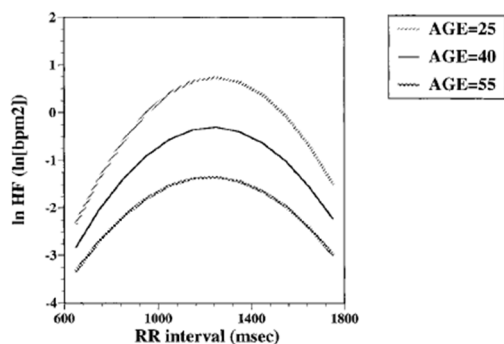


Fig. 7 - Goldberger et al., 2001. Plots of the concave-down quadratic functions describing the HRV-PNS relationship, predicted by regression analysis for ages 25, 40, and 55 years.

As mentioned before, two different views on the relationship between SNS and HRV exist. On the one hand, the older model proposed by Massimo Pagani's group stated that the LF component of HRV can be used as an accurate index of sympathetic modulation on cardiac activity, whereas the LF-to-HF ratio would represent an appropriate measure of sympathovagal balance, which would be under tonic and phasic modulation resulting from the interaction of three factors: (a) reciprocal central neural modulation, (b) peripheral inhibitory reflex mechanisms, and (c) reflex mechanisms of peripheral excitation (Fig. 8; Malliani et al., 1991). First, this view is supported by multiple studies on healthy subjects and clinical populations showing that manoeuvres known for enhancing sympathetic activity (*e.g.*, passive tilt or standing up) lead to an increase in LF and a decrease in HF, resulting in an increased LF-to-HF ratio (Fallen, Kamath & Ghista,

1988; Guzzetti et al., 1988; Lindqvist et al., 1990; Lombardi et al., 1987; Pagani et al., 1986; Pagani et al., 1988; Pomeranz et al., 1985; Vybiral et al., 1989). Other studies based on the execution of manoeuvres enhancing vagal activity (e.g., paced breathing within the resting physiologic range) showed an effect consisting of the increase of HF and a strong decrease in the LF-to-HF ratio (Fallen, Kamath & Ghista, 1988; Pomeranz et al., 1985; Saul et al., 1990; Shannon, Carley and Benson, 1987). Altogether these findings seem to be consistent with the existence of a direct relationship between sympathetic activity and the LF band of the HRV spectrum, and of a reciprocal inverse relationship between SNS and PNS manifesting through the LF-to-HF ratio.

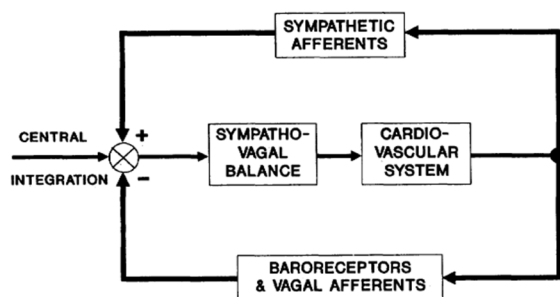


Fig. 8 - Malliani et al., 1991. Schematic representation of different, opposing feedback mechanisms that contribute, along with central integration, to the neural control of the cardiovascular system activity. While negative feedback mechanisms are mediated by baroreceptive and vagal afferent fibres from the cardiopulmonary region, which excite the vagal and inhibit the sympathetic outflow, positive feedback mechanisms are mediated by sympathetic afferent fibres that excite the sympathetic and inhibit the vagal outflow.

On the other hand, though, more recent findings (well summarised by Kiyono, Reyes Del Paso and their respective groups) have strongly challenged the validity of the model proposed by Pagani and colleagues, suggesting a new, innovative paradigm to explain the interrelation among the two autonomic branches and the HRV spectrum. First, this perspective relies on the observation that PNS and SNS are not algebraically additive, and that their dynamic interaction can occur both in terms of reciprocity or coactivation (Berntson, Cacioppo & Quigley, 1993). According to this hypothesis indeed, at least 3 modes of cardiac autonomic control can occur: (a) a coupled reciprocal one, in which the activations of the two autonomic branches are inversely correlated, (b) a coupled non reciprocal one, in which the correlation between the activities of the two branches is positive, and (c) an uncoupled one, where the occurrence of changes in PNS and SNS activity happens in a reciprocally independent fashion (Reyes Del Paso et al., 2013). Second, this view embraces the concept of “accentuated antagonism” (Levy & Zieske, 1969; Uijtdehaage & Thayer, 2000), emphasising parasympathetic predominance on cardiac functioning over the sympathetic one. This aspect is supported by findings of

multiple studies, which fit the idea of HRV being determined mainly by parasympathetic activity.

Research made on the effects of vagal and sympathetic blockade showed that while the former significantly decreased LF power, the latter has no significant effect on it. For instance, Taylor and colleagues (1998) studied the effects of autonomic blockade on the HRV power spectrum in healthy subjects, by intravenously administering atenolol (responsible for β -adrenergic sympathetic blockade), atropine sulphate (responsible for parasympathetic blockade), the combination of the two (resulting in a complete autonomic blockade) and saline (acting as a placebo). Their results displayed: (a) no significant effect of sympathetic inhibition on VLF, LF and HF power as compared to placebo, and (b) a significant suppression of VLF, LF and HF power caused by the abolition of parasympathetic activity (occurring both alone and in the context of complete autonomic blockade). Altogether these findings are consistent with the hypothesis of parasympathetic predominance on cardiac functioning throughout the HRV spectrum.

Studies using psychophysiological manipulations to consistently increase the sympathetic outflow in most cases showed no raise in LF power, but instead a reduction. For instance, Arai and colleagues (1989) studied the effects of physical exercise (known to increase sympathetic tone) in healthy subjects. In particular, they measured the HRV of the participants before, during and after performing physical activity on a cycle ergometer. Results showed a significant decrease of both HF and LF from baseline, which would be inconsistent with the idea of LF power being a marker of sympathetic cardiac control.

Research focused on the pharmacological modulation of sympathetic tone also showed changes in HRV power spectrum that are inconsistent with the first model presented here. A good example is provided by the work published by Ahmed and colleagues in 1994, where they stimulated sympathetic cardiac control in healthy subjects by administering epinephrine, an endogenous catecholamine also released during physical exercise and following myocardial infarction, and isoproterenol, a pure beta-adrenergic agonist (Ahmed et al., 1994). Their findings showed no increase in LF power for any of the two pharmacological sympathetic stimulations, although a significant increase in the LF-to-HF ratio was present following isoproterenol infusion which suggest that LF power (as well as the LF-to-HF ratio) might not be an appropriate measure of sympathetic cardiac

control, operated both via direct sympathetic neural stimulation and the indirect effects of circulating β -adrenergic agonists.

Interestingly, the model proposed by Pagani's group has been disproved also due to its inability to correctly predict HRV-related differences between groups differing according to their basal sympathetic tone. An example is provided by the paper published by van de Borne's team in 1997, where they compared the muscle sympathetic nerve activity (MSNA) and HRV power spectrums of a group of patients with chronic heart failure (CHF) and one of healthy controls (van de Borne et al., 1997). First, results displayed significantly higher values of MSNA for the CHF group, supporting the presence of a raised sympathetic tone. Second, while both HF and LF were present in all the control subjects, only 4 out of 21 CHF patients showed a LF component in the HRV power spectrum (although significantly decreased as compared to healthy controls), with all the other patients displaying a total abolishment of such frequencies. On the contrary, the decrease in HF power detected in the CHF group was not significant. Altogether, these findings are in complete disagreement with the hypotheses hold by the traditional paradigm, according to which we would expect increased LF power in the CHF group, due to raised sympathetic tone. Once again, experimental data support the need of reconsidering the role played by SNS in the genesis of LF, and the validity of the LF band as an index of sympathetic cardiac control.

Furthermore, multiple pieces of research found the LF band not to be correlated with other valid measures of cardiac sympathetic control. For instance, Sloan and colleagues (1996) assessed in healthy subjects, exposed to mental stress, the relationship between LF band and the level of circulating catecholamines [*i.e.*, epinephrine (E) and norepinephrine (NE)], which has been proven to be an accurate index of sympathetic influence on heart (Dimsdale & Ziegler, 1991). In particular, they measured changes in HRV, E and NE plasma levels, as well as in HR and blood pressure, by comparing a resting condition versus the administration of a mental stressor (*i.e.*, an arithmetic test), known to enhance cardiac sympathetic control (Langer et al., 1985; Sherwood et al., 1986). While HR and NE concentration increased significantly, a non-significant increase in E plasma levels and a significant decrease in VLF, LF and HF from baseline to stressor were recorded, with the LF-to-HF ratio remaining stable across conditions. No significant correlation was found between changes in catecholamines plasma levels and any of the HRV power

bands (see Tab. 2). These findings are in contrast with the expectations of the traditional model, which would predict the mental stressor to increase not only sympathetic activity (and therefore the catecholamines plasma levels), but also LF and the LF-to-HF ratio.

	ln LF power	ln (LF/HF)	ln VLF
Baseline			
Epinephrine	0.24	0.31	-0.13
Norepinephrine	0.12	0.33	-0.19
Mental arithmetic			
Epinephrine	0.10	-0.04	-0.02
Norepinephrine	-0.17	0.26	-0.18
Delta Score			
Epinephrine	0.24	0.26	-0.19
Norepinephrine	0.18	0.04	-0.03

Tab. 2 - Sloan et al., 1996. Pearson correlation coefficients among HRV power spectral and neurohumoral indices of cardiac sympathetic activity at baseline, during a mental arithmetic task and among their deltas across conditions.

Therefore, taking into account the findings summarised here above, the traditional model of autonomic modulation of cardiac activity seems to be over-simplistic, being valid only under the assumption of autonomic reciprocity among the SNS and PNS (which holds only in a subset of conditions), and when normalising the data referring to the different power bands. In conclusion, it is reasonable to accept the existence of a role played by both autonomic branches on the genesis and fine tuning of the HRV power spectrum as well as the prognostic utility of the different HRV indices. Nevertheless, is clear the need for new and more accurate parameters to assess and estimate autonomic balance and sympathetic activation, based on the concept of “accentuated antagonism” (and parasympathetic dominance) and accounting for the different modes of cardiac autonomic control explained before.

1.3 Measures of HRV

Overall, measurements of HRV are easy, non-invasive, and show good reproducibility if performed under standardised conditions, such as those indicated by the guidelines published in 1996 by the Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology (Acharya et al., 2006). The gold standard method to measure HRV is based on continuous electrocardiographic (ECG or EKG) recordings, but at rest other techniques such as photoplethysmography (PPG) proved to be reliable as well. According to the aforementioned guidelines, in order to collect reliable data, it is fundamental to choose an appropriate sampling rate, ideally

within the 250-500 Hz range or at least above 100 Hz, if supported by a satisfactory interpolation algorithm, used to provide a good reconstruction of the QRS complex and to reduce errors due to ectopic beats (*i.e.*, abnormal beats that are due to unusual impulses; Nabil and Reguig, 2015), arrhythmic events, noise and missing data (Task force, 1996). Furthermore, in order to compute HRV, it is necessary to keep trace of the RR intervals – *i.e.*, the interval of time between two consecutive R waves – and to clean the recordings of cardiac activity from artifacts and ectopic beats, to obtain the NN intervals – *i.e.*, the intervals between adjacent QRS complexes induced by sinoatrial node depolarisations (see 1.2.1 *Cardiovascular function*). Four different clusters of techniques have been developed in order to measure HRV: (a) time-domain methods, (b) frequency-domain methods, (c) rhythm pattern analysis, and (d) non-linear methodologies (Task force, 1996). In order to standardise the measurements, two types of recordings are typically recommended: (a) 5-minute short-term recordings under stable conditions, and (b) nominal 24-hour (or at least 18) recordings, which should include data acquired overnight, in order to assess the contribution of day-night differences to the long-term HRV values (Task force, 1996). Regardless of the method used, though, it is of fundamental importance to preprocess the data and eliminate artifacts and ectopic beats before running the analyses (Acharya et al., 2006).

1.3.1. Time-domain methods

Time-domain methods (Tab. 3) represent the simplest tools used to assess HRV and rely upon the utilisation of ECG or PPG recordings, in order to detect each QRS complex and determine the normal-to-normal (NN) intervals, calculated after correcting for artifacts and ectopic beats, and/or the instantaneous HR. Particularly, both statistical and geometrical methods can be used in order to assess HRV based on time-domain techniques. Statistical methods refer to two classes of measures, including: (a) those computed using direct measurements of the NN intervals or the instantaneous HR, and (b) those calculated using the differences between NN intervals, with the latter ones allowing for comparisons among HRV recorded during different kinds of activities (e.g., baseline at rest, physical exercise, sleep, etc.; Task force, 1996). Along with the mean HR, variables typically calculated within the time domain are the standard deviation of the NN

interval (SDNN), reflecting the cyclic components underlying the variability during the recording period, the standard deviation of the average NN (SDANN) interval calculated over short periods (usually 5-minute long), used to measure the variability in the HR induced by cycles shorter than 5 minutes, and the SDNN index, representing the mean of the standard deviation of the NN interval, calculated over 24 hours and divided into 5-minute long blocks, used to estimate the variability due to cycles shorter than 5 minutes (Task force, 1996). One of the most commonly used time-domain measures of HRV is the square root of the mean squared differences of successive NN intervals (RMSSD), but other measures can also be found in the literature, such as the NN50, representing the number of interval differences of successive NN intervals greater than 50 ms, and the pNN50, obtained by dividing the NN50 by the total number of NN intervals. Among these, the most widely used ones are the mean HR, SDNN and RMSSD, due to their computational simplicity (Acharya et al., 2006).

Variable	Units	Description
Statistical measures		
SDNN	ms	Standard deviation of all NN intervals.
SDANN	ms	Standard deviation of the averages of NN intervals in all 5 min segments of the entire recording.
RMSSD	ms	The square root of the mean of the sum of the squares of differences between adjacent NN intervals.
SDNN index	ms	Mean of the standard deviations of all NN intervals for all 5 min segments of the entire recording.
SDSD	ms	Standard deviation of differences between adjacent NN intervals.
NN50 count		Number of pairs of adjacent NN intervals differing by more than 50 ms in the entire recording. Three variants are possible counting all such NN intervals pairs or only pairs in which the first or the second interval is longer.
pNN50	%	NN50 count divided by the total number of all NN intervals.
Geometric measures		
HRV triangular index		Total number of all NN intervals divided by the height of the histogram of all NN intervals measured on a discrete scale with bins of 7.8125 ms (1/128 s). (Details in Fig. 2)
TINN	ms	Baseline width of the minimum square difference triangular interpolation of the highest peak of the histogram of all NN intervals (Details in Fig. 2.)
Differential index	ms	Difference between the widths of the histogram of differences between adjacent NN intervals measured at selected heights (e.g. at the levels of 1000 and 10 000 samples) ^[21] .
Logarithmic index		Coefficient φ of the negative exponential curve $k \cdot e^{-\varphi t}$ which is the best approximation of the histogram of absolute differences between adjacent NN intervals ^[22] .

Tab. 3 – Task Force, 1996. Summary and definitions of the most common time-domain measures of HRV.

Geometrical methods convert series of NN intervals into geometric patterns. 3 main general approaches are used to determine HRV based on the geometric/graphic properties of the data, that are: (a) the conversion of basic measurement of the geometric patterns into a measure of HRV, (b) the interpolation of the geometric pattern by a mathematically defined shape (e.g., triangles, curves) and the use of parameters belonging to the mathematical shape to compute HRV, and (c) the classification of the geometric shape

into multiple pattern-based categories that represent different classes of HRV (e.g., the different categories of Lorenz plots) (Task force, 1996). The most widely used HRV geometrical measures are the (a) HRV triangular index, highly insensitive to artifacts and ectopic beats and requiring less data preprocessing, and (b) the triangular interpolation of NN interval histogram (TINN; Acharya et al., 2006; Task force, 1996). Furthermore, another common methodology used is the Poincaré geometry, a technique relying on non-linear dynamics, depicting RR interval fluctuations by using a recurrence plot (Acharya et al., 2006). As a rule of thumb, it has been suggested that the major advantage of geometric methods lies in the relative insensitivity to the analytical quality of the NN interval series, whereas their disadvantage is represented by the need of a relatively high number of NN intervals required to extrapolate a geometric pattern from the data (Task force, 1996). Most importantly, the methodology used to measure HRV should be specifically selected based on the aim of the investigation, as each of the aforementioned tools comes with strengths and weaknesses, and it is considered inappropriate to compare data obtained by measurements of NN intervals/instantaneous HR with those coming from data related to the differences between NN intervals, as well as to compare time-domain measures derived by recordings of different durations (Task force, 1996).

1.3.2 Frequency-domain methods

Frequency-domain methods represent an effective non-invasive tool to obtain an accurate assessment of the autonomic influence on cardiac functioning. These methods use power spectral density (PSD) estimates to describe how power (or variance) of the HRV is distributed as a function of frequency (Fig. 9; Acharya et al., 2006; Pagani et al., 1984; Task force, 1996).

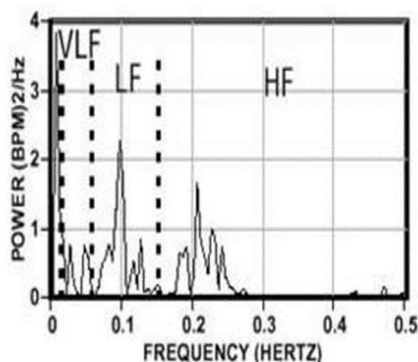


Fig. 9 - McCraty and Shaffer, 2015. Representation of the HRV power spectrum. It shows the power (y-axis) expressed as a function of the frequency (x-axis), also highlighting different frequency bands.

PSD can be calculated with parametric or non-parametric methods, although in most cases the results are comparable among these two classes of tools. While the formers are characterised by a smaller number of samples required to obtain accurate estimates of PSD, the latter ones employ an easier algorithm to be computed – *i.e.*, the Fast Fourier Transform (FFT) – which comes with the limitations of (a) not being very suitable for the analysis of non-stationary signals, and of (b) being unable to locate the different events along the time scale (which can be solved by using wavelet transform; Acharya et al., 2006). Overall, frequency-domain methods assess multiple spectral components. Particularly, short-term recordings (usually 2- to 5-minute long) allow to detect three main ones, namely: very low frequency (VLF) (the estimation of which is usually not very accurate in this type of recordings), low frequency (LF), and high frequency (HF) components, with LF and, above all, HF mainly reflecting changes in the autonomic modulation of the heart period (Task force, 1996). Measurements of these bands can be expressed in absolute values of power (ms^2), whereas LF and HF can also be assessed in normalised units (n.u.), representing the value of each power component in proportion to the difference between the total power and the VLF component. Long-term recordings, instead, assess the sequence of NN intervals in a 24-hour interval, detecting ultra-low frequency (ULF) components, along with the VLF, LF, HF ones. In general, they face the problem of ‘stationarity’ (Fisher et al., 2014; Task force, 1996). In fact, long-term recordings require that each mechanism responsible for the modulation of the heart period in a specific frequency remains stable throughout the whole recorded period, in order to assess the specific modulations produced by each of these mechanisms. Nevertheless, the mechanisms responsible for the modulation of LF and HF are not stationary over a 24-hour period, therefore the results obtained from long-term recordings (as well as those derived from 5-minute segments averaged over a 24-hour period) may hide useful information regarding the autonomic modulation of these frequencies, which would be instead available with short-term recordings (Task force, 1996). When it comes to spectral analyses with frequency-domain methods, it is important to maintain the mechanisms modulating the HR during the recording period as stable as possible, in order to attribute each spectral component to the underlying physiological mechanisms. Finally, long-term time- and frequency-domain HRV parameters have been found to be strongly correlated

with each other, due to both mathematical and physiological reasons, with the formers being easier to calculate (Task force, 1996).

1.3.3 Frequency bands in the HRV power spectrum

As mentioned above, some of the most widely used methods to assess HRV are those relying upon frequency-domain analysis, subdividing the HRV power spectrum into different power bands (see Tab. 4 for a summary). Although most of the literature on HRV have focused on the role played by LF and HF, ULF, defined as the power range up to 0.003 Hz, and VLF, comprised between 0.003 and 0.04 Hz, account together for 95% of total power in long-term recordings. Interestingly, ULF and VLF play a big role in clinical interpretations and prognostic evaluations in different pathological conditions (Bigger et al., 1992; Fei et al., 1996; Hadase et al., 2004; Tsuji et al., 1996), but their physiological interpretation has still to be fully clarified (Task force, 1996). Nevertheless, some hypotheses concerning their sources have been advanced. For instance, available empirical evidence supports the role played by circadian rhythms (regulated by autonomic and hormonal contributions) (Barrett et al., 2001; Stauss, 2003) and muscle activation (Serrador, Finlayson & Hughson, 1999) in influencing ULF. Concerning VLF, a big role has been found to be played by thermoregulatory processes in response to thermal stimuli, such as core cooling, peripheral vasoconstriction and skin-surface cooling (Fleisher et al., 1996), as well as by humoral activity (Stauss, 2003). LF, comprised within the 0.04-0.15 Hz range, and HF, related the 0.15-0.4 Hz or respiratory range, have been traditionally associated respectively to the influence of sympathetic and parasympathetic cardiac control, leading to the interpretation of the LF-to-HF ratio as an index of autonomic balance (Malliani et al., 1991). Over the last 2 decades a consistent body of research has challenged this view, suggesting different interpretations for the LF band (Kiyono et al., 2017; Reyes del Paso et al., 2013). Indeed, while there is still common agreement on parasympathetic tone being responsible for the HF band, different potential mechanisms underlying LF have been suggested. Among these, the most reported ones are those relying upon baroreflex sensitivity (Goldstein et al., 2011), affecting the LF band through the mediating action played by the PNS, in agreement with

the “accentuated antagonism” model of interaction among the two autonomic branches (Levy & Zieske, 1969; Reyes Del Paso et al., 2013; Uijtdehaage & Thayer, 2000).

Variable	Units	Description	Frequency range
Analysis of short-term recordings (5 min)			
5 min total power	ms ²	The variance of NN intervals over the temporal segment	approximately ≤ 0.4 Hz
VLF	ms ²	Power in very low frequency range	≤ 0.04 Hz
LF	ms ²	Power in low frequency range	0.04–0.15 Hz
LF norm	n.u.	LF power in normalised units LF/(Total Power–VLF) \times 100	
HF	ms ²	Power in high frequency range	0.15–0.4 Hz
HF norm	n.u.	HF power in normalised units HF/(Total Power–VLF) \times 100	
LF/HF		Ratio LF [ms ²]/HF [ms ²]	
Analysis of entire 24 h			
Total power	ms ²	Variance of all NN intervals	approximately ≤ 0.4 Hz
ULF	ms ²	Power in the ultra low frequency range	≤ 0.003 Hz
VLF	ms ²	Power in the very low frequency range	0.003–0.04 Hz
LF	ms ²	Power in the low frequency range	0.04–0.15 Hz
HF	ms ²	Power in the high frequency range	0.15–0.4 Hz
<i>a</i>		Slope of the linear interpolation of the spectrum in a log-log scale	approximately ≤ 0.04 Hz

Tab. 4 – Task Force, 1996. Summary of HRV frequency-domain measures and their frequency ranges in short- and long-term recordings.

1.3.4 Rhythm pattern analysis

Rhythm pattern analysis allows to overcome some limitations characterising time- and frequency-domain methods, occurring due to irregularities of the RR series. Overall, these analyses are based on the attempt to detect the rhythm defining blocks of several RR intervals and to assess the relationship among the different blocks without taking into account the internal variability (Task force, 1996). In particular, two of the most commonly used methods are the (a) interval spectrum and the (b) spectrum of counts methods, which are useful to assess the relationship between HRV and other physiological variables (*e.g.*, blood pressure, respiration, arrhythmia), but their explanation is beyond the purpose of this current manuscript.

1.3.5 Non-linear methods

Non-linear methods are useful to study non-linear processes underlying the genesis of HRV and to understand the role played by many physiological factors (*e.g.*, haemodynamic, electrophysiological, humoral and circadian ones), as well as by those concerning central and autonomic nervous regulatory processes, and to interpret their complex reciprocal interplay. These methodologies are based on the “theory of chaos”, which assesses the patterns and deterministic laws underlying complex dynamic systems, such as the ones contributing to the genesis of HRV (Acharya et al., 2006; Cohen, Hudson & Deedwania, 1996; Kaplan & Cohen, 1990). Typical problems that must be faced when using these methods are represented by the high levels of random noise present in non-linear biological data, the short experimental datasets available due to the low frequencies characterising such signals, and their non-stationarity.

1.4 Additional factors influencing HRV

1.4.1 Sex- and age-related effects

Available empirical evidence supports the existence of a role played by sex and age in influencing HRV indices. A good example is provided by the findings of the study conducted by Jensen-Urstad and colleagues in 1997, where they investigated in healthy subjects (age: 20-69) the effect of the aforementioned factors, by collecting 24-hour long-term ECG recordings. Interestingly, they found strong effects for both factors. Concerning age, they reported that, while mean HR was not significantly affected, within frequency-domain total power (TP), VLF, LF and HF were negatively correlated with it, with a decrease of about 30% when comparing TP among the 20-29 and the 60-69 group. Moreover, results showed age-related differences concerning HRV circadian variations, with younger subjects displaying a stronger increase in HF power at night, as compared to older ones. Concerning sex, women displayed lower TP, VLF, LF and LF-to-HF ratio in the frequency-domain, as well as lower SDNN and SDNN index in the time-domain. Results also showed significant correlations between different HRV indices, with TP, VLF and LF being correlated to the SDNN index, and HF being associated with pNN50.

Another study conducted by Umetani's research group (1998) replicated the aforementioned findings, showing an age-dependent decrease in HRV, characterised by measure-dependent patterns. Moreover, they reported a significant attenuation of sex-related differences in HRV, occurring after menopause (see also Stein et al., 1997). Hormonal influences on the ANS may act as a potential cause for the reduction in sex-related differences occurring later on life (Cygankiewicz and Zareba, 2013).

Finally, a meta-analysis conducted by König and Thayer in 2016 focused on sex-differences in HRV including 172 papers. The authors reported lower mean RR interval and SDNN, lower TP, VLF and LF, as well as increased LF-to-HF ratio and HF in females as compared to males. Moreover, the authors reported that with increasing age sex-related differences on SDNN were diminished, whereas older females showed a significantly greater RMSSD when compared to males. Altogether, the available evidence underlines the importance of taking into account sex and age when interpreting HRV indexes.

1.4.2 Physical training and physical exercise

Physical exercise and training are believed to play a role in modifying HRV and, in turn, HRV has been suggested to be a promising non-invasive tool to monitor the training status in athletes (Plews et al., 2013). Concerning training, most of the available evidence comes from studies on recreational or moderately trained subjects, while data collected in elite athletes are rare and much more limited (Plews et al., 2013). Regarding the former population, there is substantial agreement in claiming that moderate training loads are effective in increasing not only aerobic fitness, but also HRV, whereas higher levels of training seem to reduce HRV (Plews et al., 2013). Manzi and colleagues studied the relationship between training, HRV parameters, and performance in a sample of recreational long-distance runners (Manzi et al., 2009). First, they found that the LF and the LF-to-HF ratio showed a U-shaped curve, showing high power at the beginning of the training programme, followed by a progressive decrease until the middle of it, and a progressive increase until the end. Second, they reported a bell-shaped curve followed by HF and R-R intervals, which increased midway through the programme and then decreased with the progressive increase of the training loads. Interestingly, the n.u.LF assessed at the last training session before the marathon inversely correlated with the time

obtained during the race. On the contrary, n.u.HF showed a significant positive correlation with the race time. Altogether, these findings support the idea that physical training can alter HRV indices, with the latter being useful tools to predict sport performance and, therefore, the effectiveness of the training.

Another study by Iwasaki and colleagues (2003) on previously untrained subjects undergoing 12 months of progressive endurance training reported similar findings. First, their results showed that, within the time-domain, HRV, as indexed by the standard deviation of R-R variability (SDR-R), was correlated to the dose of exercise with a second-order regression, following a bell-shaped curve (Fig. 10). Indeed, SDR-R significantly increased from baseline to 3 and 6 months, but then decreased at 9 and 12 months, reaching baseline levels again. Second, within the frequency-domain, HRV also increased significantly at 3 and 6 months from baseline levels, to then decrease at 9 and 12 months, returning to initial levels. Once again, a bell-shaped curve described changes in HRV within the frequency-domain following increasing training loads, with the relationship between training and HRV being defined by a second-order regression model. Finally, the authors advanced three hypotheses to explain the pattern of changes involving HRV. The first proposed model was based on training-induced enhanced stimulation of the PNS, which would lead to increased HRV, until a saturation of parasympathetic cardiac control is reached. The second hypotheses advanced relied on the claim that initially moderate training loads would increase parasympathetic activation, whereas afterward more prolonged training would decrease sympathetic influence on cardiac functioning. The third hypothesis, also supported by the findings of Lewis and colleagues (1980), suggested instead that increasing training loads would act directly on intrinsic cardiac regulation, independently of autonomic mechanisms, and would also account for the observed reduction in resting HR throughout the training programme. Although the physiological mechanisms underlying the changes in HRV induced by training have not been clarified yet, the relationship between endurance training and cardiac variability seems to be best described by a bell-shaped second order relationship, characterised by an initial rise in HRV following moderate levels of physical activity, and a subsequent progressive decrease following increasing loads of training.

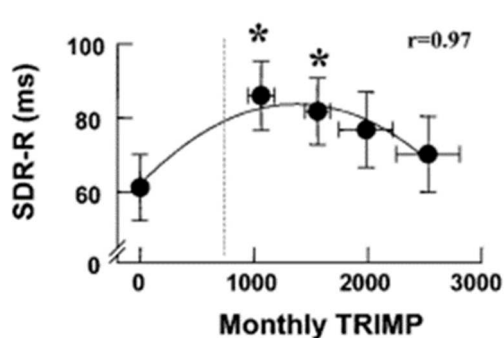


Fig. 10 – Iwasaki et al., 2003. Dose-response relationship among exercise intensity (monthly TRIMP) and a measure of HRV (SDR-R).

Other studies conducted on professional elite athletes replicated the findings obtained in nonprofessional recreational athletes discussed here above (Plews et al., 2013). In particular, Iellamo and colleagues assessed the effects of training on HRV in a sample of athletes of the Italian junior national team of rowing, training for and participating in the World Championship (Iellamo et al., 2002). First, the authors reported that up to 75% of the training load a progressive bradycardia appeared from baseline, along with a significant progressive increase in the HF band and non-significant trends toward the reduction of the LF band and the LF-to-HF ratio. Second, results showed that at 100% training load an opposite pattern of changes occurred, characterised by a relative increase in HR, a significant decrease in HF and an increase in the LF component and the LF-to-HF ratio. The authors measured salivary cortisol concentration to control for stress levels. Since no changes in cortisol concentration were found throughout the training programme, they concluded that the changes in autonomic cardiac control could not be explained by changes in stress levels. In conclusion, the authors interpreted the changes in HRV as the result of an initial predominance of the PNS in modulating cardiac function induced by moderate training levels, and followed by a switch to a sympathetic predominance in the control of cardiac function, induced by maximum training loads and indicating neurovegetative adaptation to increase athletic performance.

As discussed above, moderate levels of training appear to enhance HRV, while higher or maximal training loads are associated with its reduction. Interestingly, HRV indices of vagal control have been found to rebound beyond pretraining levels during lighter training or recovery periods, with such a rebound being associated with improved performance (Plews et al., 2013). The research group led by Hautala evaluated changes in HRV before and after a 75 km cross-country skiing race (Hautala et al., 2001). Their results showed a non-significantly different average HR 1 day after the race as compared to the day before

the race, whereas it was found to be significantly lower on day 2 after the competition. Concerning HRV, on day 1 after the race the n.u.HF band was significantly lower and the n.u.LF band significantly greater than on the pre-race day (Fig. 11). On the second day following the competition the pattern reversed, n.u.HF came back to pre-race levels or exceeded them, while the n.u.LF component was reduced to baseline levels again or even lower (Fig. 11). The LF-to-HF ratio followed the same pattern as n.u.LF. Furthermore, large individual differences were observed in the recovery of n.u.HF, with the time required to return to pre-race baseline levels being inversely correlated with maximal oxygen consumption, measured during a cycling test administered before the skiing competition (Fig. 12). These findings support the presence of a rebound in HRV occurring after exercise, which was interpreted by the authors as a protective mechanism that compensates for altered cardiovascular hemodynamic after exercise, and highlight the presence of a correlation between some HRV indices and cardiorespiratory fitness, suggesting that individual fitness may play a role in influencing the recovery of altered autonomic cardiac control after physical effort.

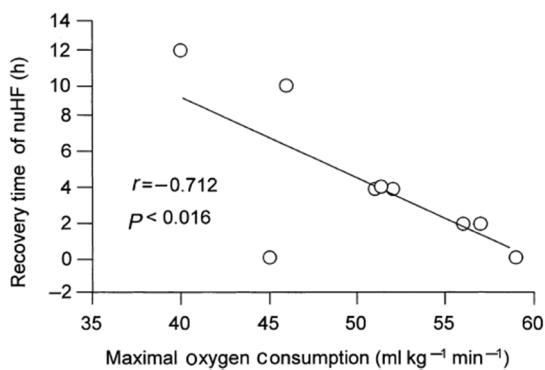


Fig. 11 – Hautala et al., 2001. HRV before and after the race. Representative examples of power spectra (upper panels) and Poincare plots (lower panels) before (left side) and during the 1st (middle) and 2nd night (right side) after the race. While no significant difference concerning the average HR is present when comparing pre- and post-race data, decreased vagally-mediated HF power and increased LF-to-HF ratio are found during the two nights following the race (as measured between 3 and 4 a.m. <-

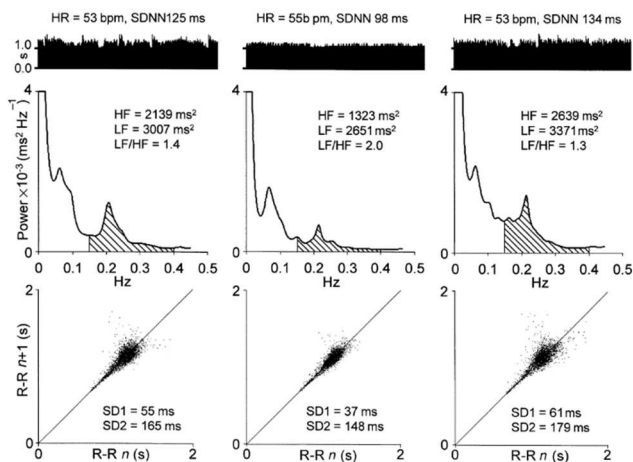


Fig. 12 – Hautala et al., 2001. Correlation between post-race recovery time of n.u.HF (h) and maximal oxygen consumption during a pre-race cycling test. Spearman's correlation coefficient is given. ->

Furthermore, Plews and colleagues suggested in their literature review (2013) that HRV could be used as an index of fatigue and/or overtraining, based on the assumptions of Goldberger's model describing a second-order quadratic relationship between the

influence of the PNS on the heart and some indices of HRV (Goldberger et al., 1994; Goldberger et al., 2001). According to this model, at both low (high HR) and high (low HR) levels of parasympathetic predominance on cardiac function, HRV indices reflecting vagal activity are reduced. Usually, athletes present with low HR and high HRV parameters at rest, but over-training might lead to a saturation of the PNS mediated by the saturation of the cholinergic receptors on the myocytes, consequently reducing the respiratory modulation of HRV. Based on these assumptions, Plews suggested the use of the ratio between the weekly average of ln RMSSD (a measure of HRV) and the R-R interval to detect states of fatigue induced by over-training, as opposed to states of readiness to perform (Fig. 13).

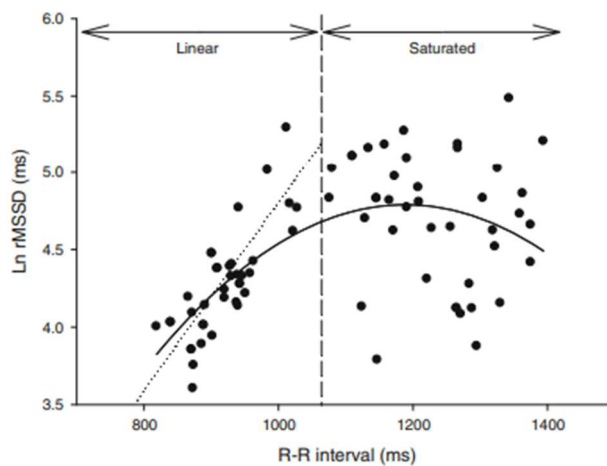


Fig. 13 – Plews et al., 2013. An example of the relationship between the R–R intervals and the ln RMSSD in a subject with increasing bradycardia, used as a measure of over-training induced fatigue. A saturation of HRV occurs with long R–R intervals. Interestingly, at shorter R–R intervals there is a linear relationship between the duration of such intervals and ln RMSSD (dotted line), which disappears with longer interval durations, due to the saturation of HRV.

In conclusion, the available evidence supports the role played by physical training in altering HRV and suggests that HRV assessment may be a useful non-invasive tool to monitor training status, to detect states of fatigue and readiness to perform, and to predict performance in a variety of endurance sports and subjects. By doing so, it is important to adopt longitudinal monitoring to understand the optimal HRV and its correlation with training status, since high inter-individual variability in HRV is present, also depending on fitness levels and training history. Finally, it has been suggested that an initial increase in HRV values may be an index of positive adaptation and effective coping with training loads, whereas a reduction in HRV in the days or weeks before the main competition may indicate that the athlete is fresh and ready to perform, making HRV measures a useful tool for sport psychology.

1.4.3 Mental stress

Mental stress, also characterising motorsport, has been associated with changes in HRV both in naturalistic environments and as induced by various methods in laboratory contexts (Kim et al., 2018). For example, Sloan's research group assessed in healthy subjects the relationship between long-term HRV recordings and a self-reported measure of stress (Sloan et al., 1994). The results showed a significant negative correlation between stress and the average R-R interval as well as a positive correlation between self-reported psychological stress and the LF-to-HF ratio. Overall, the authors interpreted these findings as indicating a stress-induced shift in cardiac autonomic control toward a reduction in parasympathetic influence or relative sympathetic predominance. Other studies conducted in laboratory contexts reported similar findings. For example, Delaney and Brodie (2000) used short-term recordings to assess HRV changes in healthy subjects exposed to mental stress. In particular, the experimental group was administered the Stroop Word Color Conflict Task, a test of inhibitory control, and was told that the best-performing subject would have received a cash prize, in order to increase the stress load. The results showed significant alterations in HR and HRV within both the time- and frequency-domain when exposed to mental stress. In particular, the authors recorded a significant stress-induced increase in HR and the LF-to-HF ratio, as well as significant reductions in SDNN, RMSSD, pNN50 and the HF band. As in the study discussed above, the authors interpreted these results as supporting the occurrence of a shift in cardiac autonomic control toward reduced parasympathetic activity and relative sympathetic predominance.

Altogether, the findings of these studies are consistent with those reported in a recent meta-analysis, supporting the occurrence of parasympathetic withdrawal from cardiac control when exposed to mental stressors in both laboratory-based and ecologically valid environments (Kim et al., 2018). To explain the underlying mechanisms, the authors of the meta-analysis referred to Thayer's Neurovisceral Integration Model (see 2.4 *The Neurovisceral Integration Model*), according to which the prefrontal cortex (PFC), and in particular the medial region (mPFC), and other cerebral areas (*i.e.*, the amygdala) may be involved in both the appraisal of threats, a fundamental process involved in the genesis of mental stress, and the modulation of the nuclei responsible for the tuning of HRV (Thayer et al., 2012). Overall, these findings are relevant to the purpose of this

manuscripts, since they highlight the influence of mental stress on cardiac autonomic control and HRV parameters, and they could be helpful for the assessment and understanding of the psychophysiological changes occurring in racing drivers, who are exposed not only to considerable physiological stressors, but also to mental ones, especially straight before and during racing events.

1.4.4 Medical, somatic and psychiatric disorders

Although the discussion of this topic is beyond the scope of this manuscript, it is important to mention that many disorders affecting the cardiovascular, cerebrovascular, and nervous systems, as well as some psychiatric disorders, present with peculiar patterns of HRV, since they are often characterised by alterations in the activity of the ANS, leading to an imbalance between PNS and SNS activity. For this reason, HRV is an important tool to make clinical interpretations and prognoses. Regarding cardiological conditions, previous studies reported alterations in HRV in patients with acute and sub-acute myocardial infarction (Bigger et al., 1991; Casolo et al., 1992; Lombardi et al., 1996; Task Force, 1996), myocardial dysfunction without infarction (Casolo et al., 1989; Gordon et al., 1988; Kienzle et al., 1992; Mortara et al., 1994; Nolan et al., 1992; Task force, 1996), congestive heart failure (Jiang et al., 1997), recent cardiac transplantation (Fallen et al., 1988; Sands et al., 1989), and diabetes mellitus-related cardiac autonomic neuropathy (Cygankiewicz and Zareba, 2013; Task force, 1996; Pfeifer et al., 1984; Schönauer et al., 2008; Wawryk, Bates & Couper, 1997). Cerebrovascular disorders, including ischaemic cerebral infarction (Barron, Rogovski & Hemli, 1994), have also been related to changes in HRV (Cygankiewicz and Zareba, 2013). Overall, different diseases affecting the cardiovascular and cerebrovascular systems play an important role in generating an impairment of cardiac autonomic control, and such an impairment can be detected in a non-invasive fashion by assessing HRV, making it a valuable clinical tool, useful for clinical evaluations and risk stratification.

Moreover, evidence have been previously found, supporting the presence of a link between HRV alterations and other non-inherently cardiac diseases and neurologic conditions, including multiple sclerosis (Tombul et al., 2011; Brezinova, Goldenberg and Kucera, 2004; Frontoni et al., 1996; Mahovic & Lakusic, 2007) Parkinson's disease

(Turkka et al., 1987; Van Dijk et al., 1993), muscular dystrophies (Yotsukura et al., 1995; Yotsukura et al., 1998; Lanza et al., 2001; Ammendola et al., 2006) and epilepsy (Lotufo et al., 2012).

Interestingly, psychiatric disorders have been related not only to impairments in cognition and ANS dysregulation, but also to changes in HRV. For instance, although the evidence concerning the pattern of HRV changes in depression is still controversial (Yeragani et al., 1991; 2000; Roose, 2001; Gehi et al., 2006; Koch et al., 2019) previous studies showed a link between altered HRV and anxiety disorders and post-traumatic stress disorder (Chalmers et al., 2014), schizophrenia (Okada, Toichi & Sakihama, 2003; Toichi et al., 1999; Böttger et al., 2006), and certain personality disorders, including bipolar disorder (Cohen et al., 2003) and attention deficit/hyperactivity disorder (ADHD) (Börger et al., 1999; Tonhajzerova et al., 2009). Finally, alterations in HRV have been reported also in dementia (Kim et al., 2006) and cognitive impairment (Yang et al., 2010).

Overall, the evidence summarised here shows some of the many disorders involving changes in cardiac autonomic control and supports the idea that HRV analysis may be a useful clinical tool for diagnosis, prognosis and risk stratification.

1.4.5 Drugs and pharmacological interventions

Although beyond the scope of this manuscript, in conclusion to this first introductory chapter, it is important to mention that previous research showed that different classes of drugs interact differently with cardiac autonomic control. In particular, some types of drugs displayed positive effects on HRV, whereas some others were reported to have a negative impact on it. Among the drugs that were found to enhance HRV, some types of antihypertensive and antiarrhythmic (Lakusic, Mahovic & Babic, 2005), including angiotensin-converting enzyme inhibitors (Kontopoulos et al., 1997), have been reported. Concerning those having detrimental effects on HRV, instead, previous studies included antidepressants (Licht et al., 2008) and antipsychotics (*e.g.*, clozapine; Cohen et al., 2001; Rechlin, Claus & Weis, 1994). Therefore, when assessing HRV for clinical and clinical-research purposes, it is important to control for the medications taken by the participants.

2 *Heart Rate Variability, cognitive functioning and the Neurovisceral Integration Perspective*

2.1 *Cognitive Functions*

Human cognition is traditionally subdivided into multiple domains of functioning (Harvey, 2019). A very common view on the topic is the one postulating a hierarchical structure, where more basic sensory and perceptual processes constitute the ground for more complex executive functions in charge of cognitive, affective, and behavioural control (Harvey, 2019). According to this perspective, domains are not independent, but reciprocally interconnected, and can be conceptualised using either a top-down (with the higher-order functions controlling lower-order ones) or a bottom-up (with the higher-order functions stemming from lower-order ones) approach (Tab. 4). Along with the hierarchical perspective, across the history of psychology other alternative approaches have been developed in the attempt to classify the different cognitive domains. For instance, very common strategies are the ones focused on the definition of the general cognitive processes involved in cognition (*e.g.*, memory, language), or the one using lesion studies in order to define the exact function performed by each brain area. Finally, regardless of the approach used and relevantly for the study presented in the following chapters of this manuscript, cognitive domains have traditionally been subdivided into non-executive and executive functions, with the former consisting of lower-order and mainly automatic mechanisms, and the latter referring to higher-order, effortful, goal-directed processes.

Tab. 4 – Harvey, 2019 - Domains of cognitive functioning: presented as a bottom-up conceptualization

Sensation	
	Multisensory
Perception	
	Object recognition
	Organizational strategies
Motor skills and construction	
	Copying
	Drawing
	Other praxic skills
Attention and concentration	
	Selective attention
	Sustained attention/vigilance
Memory	
	Working memory
	Verbal
	Spatial
	Object
	Location
	Working memory components
	Central executive
	Maintenance
	Manipulation
	Episodic/declarative memory
	Verbal
	Nonverbal
	Encoding
	Storage
	Retrieval
	Free recall
	Cued recall
	Forced-choice recognition
	Procedural memory
	Semantic memory
	Prospective memory
	Time-based
	Event-based
Executive functioning	
	Reasoning
	Problem solving
	Component skills management
Processing speed	
	Semantically relevant (fluency)
	Coding and tracking
Language/verbal skills	
	Naming
	Fluency
	Reading and comprehension

2.2 Non-executive functions

The umbrella term of non-executive functions includes a multitude of processes, usually requiring minimal higher-level processing, mainly consisting of automatic effortless mechanisms (Harvey et al., 2019). According to the most widely held view, sensation, perception, motor skills, memory, and some aspects of language fall under this area of cognition. In particular, at the bottom of the hierarchy sensation and perception are placed. Specifically, sensation refers to the ability to detect a stimulus that occurs in one of the five sensory modalities, whereas perception is related to the mechanisms of processing and integration of sensory information into a unitary percept (Harvey, 2019). Usually, sensation is assessed by presenting stimuli within a specific sensory domain, to verify whether the subject is able to acknowledge their presence. In case of failure, then sensory deficits are present. To assess perception, instead, subjects usually undergo structured recognition tests, such as the Visual and Object and Space Perception Battery (Warrington and James, 1991).

Within the hierarchy, just above sensation and perception, cognitive psychologists placed motor skills, including motor speed and accuracy, and construction skills, referred to the ability to either copy or produce drawing of common objects (Harvey, 2019). Common tasks used to assess basic motor abilities from a cognitive standpoint include the Finger Tapping Test (FTT), comprised in the Halstead-Reitan battery (Halstead, 1947; Reitan and Wolfson, 2009), where subjects are required to depress a keyboard key as many times as possible within a given period as a measure of psychomotor speed, the Purdue Pegboard Test, assessing manual dexterity and bimanual coordination (Tiffin and Asher, 1948), and the evaluation of grip strength. Widely used tests to assess visuo-constructive abilities are instead the copy component of the Rey-Osterrieth Complex Figure (ROCF) (Rey, 1941; Osterrieth, 1944), where the individual is required to copy a complex pattern of lines and circles, or other tasks requiring to copy objects and figures, such as the Copy of the Cube subtest of the Montreal Cognitive Assessment scale (MoCA) or the copy of the Intercepting Pentagons subtest of the Mini-Mental State Examination (MMSE) (Folstein et al., 1975). To note, these tasks are not purely visuo-constructive in nature, since they all involve, to different extents, executive functioning and planning.

Moving upward in the hierarchy, memory is probably the most multifaceted of cognitive domains. It comprises multiple subdomains, each of which subserves a specific function and reciprocally interacts with the other ones (Harvey, 2019). Among the subdomains, short-term and long-term stores are included, and declarative and non-declarative processes are differentiated (Milner, Squire and Kandel, 1998; Squire and Zola-Morgan, 2011; Harvey, 2019).

On the one hand, declarative/explicit memory is the component in charge of encoding, maintaining and retrieving representations about episodes, facts (episodic memory), and meanings (semantic memory), and is propositional (its contents are either true or false; Milner, Squire and Kandel, 1998; Squire and Zola-Morgan, 2011). To test the encoding process of declarative memory different tasks are often administered: the Hopkins Verbal Learning Test (HVLT; Brandt, 1991), the California Verbal Learning Test (CVLT; Delis, Kramer, Kaplan, and Ober, 2000), and the Rey Auditory Verbal Learning Test (RAVLT; Rey, 1941; Rey, 1964). To test the storage component, words recall at varying post-encoding time periods is used, whereas to assess retrieval unprompted free recall, cued recall or recognition tasks can be administered (Harvey, 2019).

On the other hand, non-declarative memory is related to the encoding, storage and retrieval of implicit contents, that usually cannot be fully expressed linguistically. It includes procedural memory, related to motor-skills learning, habituation, sensitisation, emotional learning, and Pavlovian or classical conditioning (Milner Squire and Kandel, 1998). Examples of tools used to assess non-declarative memory are the Pattern Sequence Learning Test (Nissen and Bullemer, 1987) the priming component of the Word-stem Priming Test (Perry et al., 1999), and word fragment completion tasks. Finally, prospective memory is related to the ability to remember to perform tasks in the future. In particular, two formats of this subdomain of memory exist: the event-based system allows to remember to perform an action whenever an event happens (*e.g.*, turn off the oven when the timer sounds), while the time-based one associates the action to perform to a specific moment in time (*e.g.*, take the pill in the morning) (Harvey, 2019). Prospective memory can be assessed using tasks requiring to perform a specific action when a target event occurs (*e.g.*, see Einstein and McDaniel, 1990), or more standardised tests such as the Cambridge Prospective Memory Test (CAMPROMPT; Wilson et al., 2005).

Language can be divided into receptive and productive abilities, and into the phonology, morphology, syntax, semantics, and pragmatic subdomains (Paradis, 1998; Harvey, 2019). Verbal expressive language can be studied using a multitude of tools, such as the Cookie Theft picture description task from the Boston Diagnostic Aphasia Examination (Goodglass and Kaplan, 1983) or by assessing free speech, whereas auditory comprehension by providing instructions such as those included in the Token Test (De Renzi and Vignolo, 1962). Phonology can be assessed by administering letter fluency tests. Semantics can be studied by using semantic fluency or object naming tasks, including the Boston Naming Test (Kaplan, Goodglass and Weintraub, 1983) and the Graded Naming Test (McKenna and Warrington 1983), as well as by administering tools like the Pyramids and Palm-trees Test (Howard and Patterson, 1992) or the Hodges' Semantic Battery (Hodges et al., 1992).

Finally, multiple measures of processing speed exist, organised on three levels (Deary, Johnson and Starr, 2010). At a higher level, psychometric tests like the Digit-Symbol Coding and Symbol search subtests of the Wechsler (1997) have been developed. At a lower level, choice reaction time tasks, requiring the subject to respond (usually by pressing a keyboard key) to one of a number of stimuli, and simple reaction time (SRT) tasks (Donders, 1969), requiring responding as quick as possible to the presentation of a single predetermined stimulus (*e.g.*, a light), have been used in literature. Finally, at the lowest level, inspection time tasks are administered, such as the pi-stimulus IT procedure (Nettelbeck, 1987). Many of these higher-level measures, though, include to different extents executive components, like planning, set-switching, strategy setting or inhibitory control. Other lower-level tools, like SRT and inspection time tasks, are instead relatively pure measures of non-executive processing speed, which is considered to be an index of the fundamental capacity of the CNS, constituting the ground for all the higher-level cognitive functions required to be successful in complex environments, such as those constituting everyday life and car racing (Deary, 2000; Deary, Johnson and Starr, 2010). In particular, the relevance of processing speed and other cognitive functions for driving will be further discussed in the third chapter of this manuscript, since a computerised SRT task and other cognitive tasks have been used in the study on racing drivers that will be presented in the last section of this essay.

2.3 Executive functions

Executive functions (EFs), also known as executive control or cognitive control, refer to an ensemble of top-down non-automatic mental processes, related to the ability to concentrate, to pay attention, to withdraw from automatic actions, and to adjust the behaviour to the environmental demands (Diamond, 2013). In particular, three core EFs – *i.e.*, inhibitory control, working memory (WM), and cognitive flexibility – have been proposed in the literature (Lehto et al., 2003; Miyake et al. 2000). These three core EFs are thought to underlie all the other executive processes and abilities, such as reasoning, problem solving, and planning (Collins & Koechlin, 2012; Lunt et al., 2012). Interestingly, reduced individual executive functioning has been associated (Tab. 5) with different health- and quality-of-life-related variables, such as the presence of physical and mental disorders (Baler & Volkow, 2006; Barch, 2005; Diamond, 2005; Fairchild et al., 2009; Lui & Tannock, 2007; Penades et al., 2007; Crescioni et al., 2011; Miller et al., 2011; Riggs et al. 2010), job unsuccess (Bailey, 2007), marital issues (Eakin et al., 2004) and social problems (Broidy et al., 2003; Denson et al., 2011). Furthermore, positive correlations between EFs and quality of life (Brown and Landgraf, 2010; Davis et al., 2010), school readiness (Blair and Razza 2007; Morrison et al., 2010), and school success (Borella et al., 2010, Duncan et al. 2007; Gathercole et al., 2004) were reported (see Tab. 5). Overall, these findings highlight the importance of executive functions in everyday life, in order for the individual to be adaptive, efficient and successful.

Aspects of life	The ways in which EFs are relevant to that aspect of life	References
Mental health	EFs are impaired in many mental disorders, including:	
	- Addictions	Baler & Volkow 2006
	- Attention deficit hyperactivity (ADHD)	Diamond 2005, Lui & Tannock 2007
	- Conduct disorder	Fairchild et al. 2009
	- Depression	Taylor-Tavares et al. 2007
	- Obsessive compulsive disorder (OCD)	Penadés et al. 2007
Physical health	- Schizophrenia	Barch 2005
	Poorer EFs are associated with obesity, overeating, substance abuse, and poor treatment adherence	Crescioni et al. 2011, Miller et al. 2011, Riggs et al. 2010
Quality of life	People with better EFs enjoy a better quality of life	Brown & Landgraf 2010, Davis et al. 2010
School readiness	EFs are more important for school readiness than are IQ or entry-level reading or math	Blair & Razza 2007, Morrison et al. 2010
School success	EFs predict both math and reading competence throughout the school years	Borella et al. 2010, Duncan et al. 2007, Gathercole et al. 2004
Job success	Poor EFs lead to poor productivity and difficulty finding and keeping a job	Bailey 2007
Marital harmony	A partner with poor EFs can be more difficult to get along with, less dependable, and/or more likely to act on impulse	Eakin et al. 2004
Public safety	Poor EFs lead to social problems (including crime, reckless behavior, violence, and emotional outbursts)	Broidy et al. 2003, Denson et al. 2011

Tab. 5 – Diamond, 2013 - Executive functions (EFs) and their importance to just about every aspect of life

2.3.1 *Inhibitory control*

Inhibitory control is thought to consist of the ability to control one's own attention, thoughts, emotions, memories, and behaviours, to allow the individual to work in an adaptive way to the given context, by inhibiting prepotent, irrelevant or interfering stimuli or impulses, and redistributing resources to the most relevant processes and actions (Diamond, 2013; Thayer et al., 2009). Moreover, inhibitory control allows the subject to be flexible and adaptable, being able to disengage from their pre-acquired "auto-pilot" functioning, and to change and choose accordingly to the modifications in the environment. Different types of inhibitory control have been postulated by cognitive psychologists over the last decades. First, attentional inhibition has been proposed to be the mechanism of endogenous regulation of attention, which allows the subject to disengage the attentional focus from exogenous control by salient stimuli, and to redirect it toward the goal-relevant piece of information within the internal or external environment (Posner & DiGirolamo, 1998; Theeuwes, 2010). Second, cognitive inhibition has been suggested to be the ability to suppress prepotent thoughts, memories, and mental representations, and allows to resist to intrusive thoughts as well as to perform intentional forgetting, while also supporting WM (Anderson and Levy, 2009; Diamond, 2013). Third, self-control has been defined as the ability to control one's own actions and emotions, to support a behavioural output that is appropriate to the context and the individual goal, and also includes aspects of delayed gratification (Diamond, 2013; Mischel et al., 1989). Different psychological tasks have been developed to measure inhibitory control, including the Stroop Color and Word Test (SCWT) (Stroop, 1935), the Simon task (Simon, 1990), the Flanker Task (Eriksen and Eriksen, 1974), Go/NoGo paradigms (Donders, 1969), and stop-signal paradigms (Verbruggen and Logan, 2008).

Concerning the SCWT, it is a neuropsychological test used to assess cognitive and self-control abilities (Scarpina and Tagini, 2017). Its most common version consists of 3 different tables that the subject is required to complete as quick possible (Fig. 14). In particular, the first two tables, or "congruous conditions", respectively consist of a list of names of colours printed in black ink (set A), that the subject is required to simply read aloud, and a sequence of colour names printed in the corresponding colour ink (set B). The third or "incongruous condition" (set C), instead, consist of a list of colour-words printed in an inconsistent colour ink, and the participant is asked to identify the sequence

of colour inks, neglecting the actual colour written in letters. This test taps onto inhibitory control since it requires to perform a non-automated task (say the colour of the ink aloud), inhibiting the automatic response of reading the written word. The difficulty of inhibiting the automated response is known as Stroop effect and results in a longer response latency and, often, a higher number of errors in the third condition as compared to the first two.

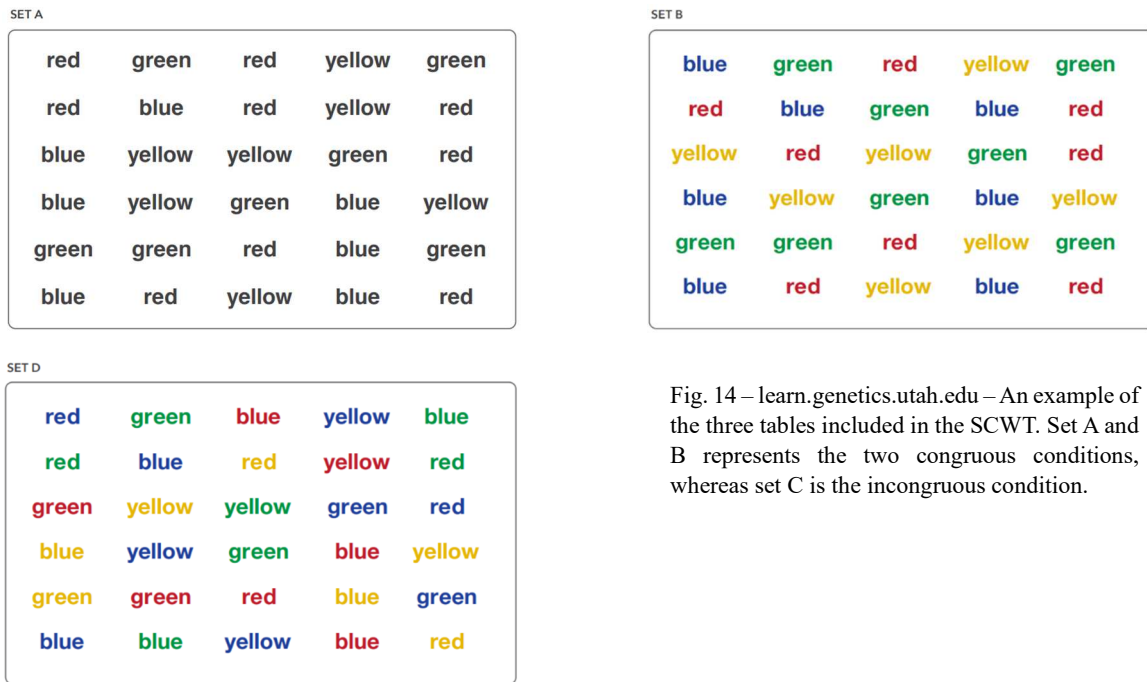


Fig. 14 – learn.genetics.utah.edu – An example of the three tables included in the SCWT. Set A and B represents the two congruous conditions, whereas set C is the incongruous condition.

The Simon task is based (Fig. 15) on a double stimulus-response (S-R) association, requiring pressing a button on the left of the keyboard whenever a pre-determined stimulus “A” appears (*e.g.*, a triangle), and to do the same on the right, whenever the stimulus “B” is presented (*e.g.*, a circle). Although the location of the stimulus presentation is irrelevant for the task, empirical data showed that people are on average slower to respond whenever the target stimulus is displayed on the opposite side to the associated response. This phenomenon, also known as Spatial Compatibility Effect, arises from the necessity of inhibiting the automatic action of responding on the same side as the stimulus, in order to perform the target one (Hommel, 2011; Lu and Proctor, 1995). Thus, the Simon task can be used as a measure of self-control.

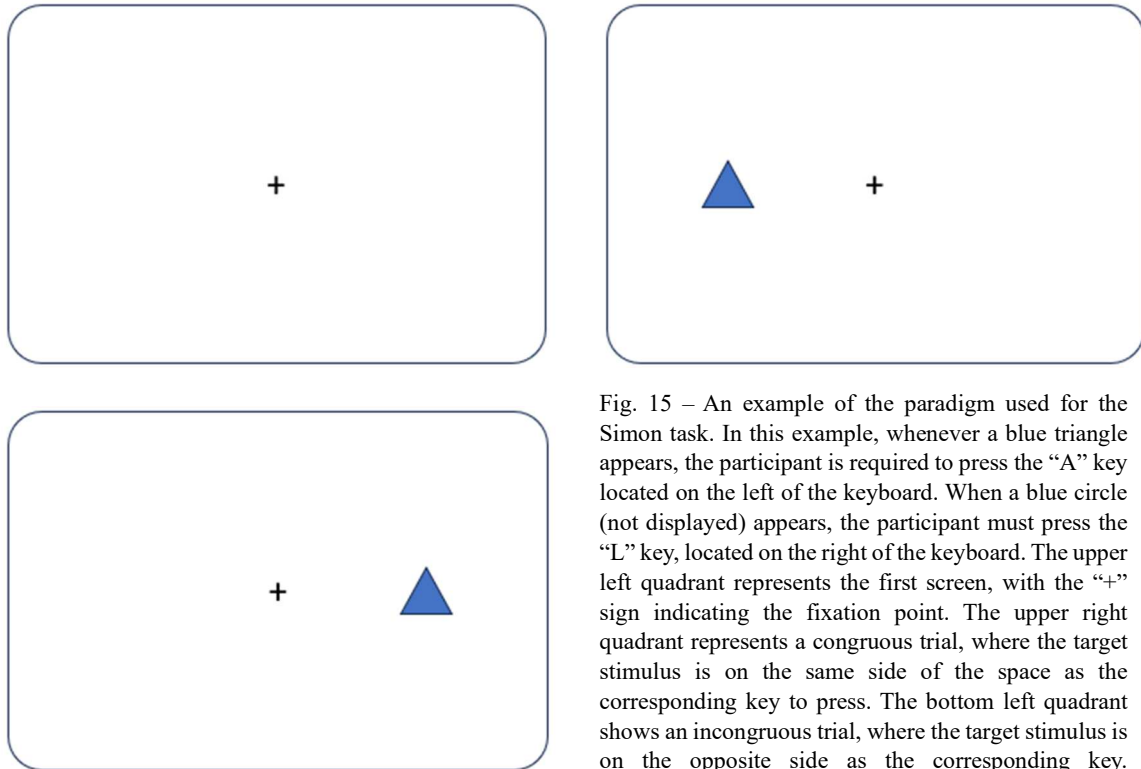


Fig. 15 – An example of the paradigm used for the Simon task. In this example, whenever a blue triangle appears, the participant is required to press the “A” key located on the left of the keyboard. When a blue circle (not displayed) appears, the participant must press the “L” key, located on the right of the keyboard. The upper left quadrant represents the first screen, with the “+” sign indicating the fixation point. The upper right quadrant represents a congruous trial, where the target stimulus is on the same side of the space as the corresponding key to press. The bottom left quadrant shows an incongruous trial, where the target stimulus is on the opposite side as the corresponding key. According to the Simon effect, people are on average faster in responding to the congruous rather than the incongruous trials.

The Flanker task (Fig. 16) assesses attentional inhibition, since it requires the use of selective attention and the endogenous direction of it, to inhibit the salience of non-relevant exogenous stimuli. In particular, it requires to focus on the stimulus presented centrally, which is an arrow indicating the side of the keyboard where the subject is required to press a button as a response. Along with the central or target stimulus, a line of flanking stimuli (other arrows) is presented around it. Evidence has shown that when the flanking stimuli are compatible with the target one (*i.e.*, they point to the same direction) subjects are on average faster to respond than when the flanking arrows are incompatible with the target one (*i.e.*, they point to the opposite direction), since in the latter case top-down inhibitory control is required (Eriksen and Eriksen, 1974).

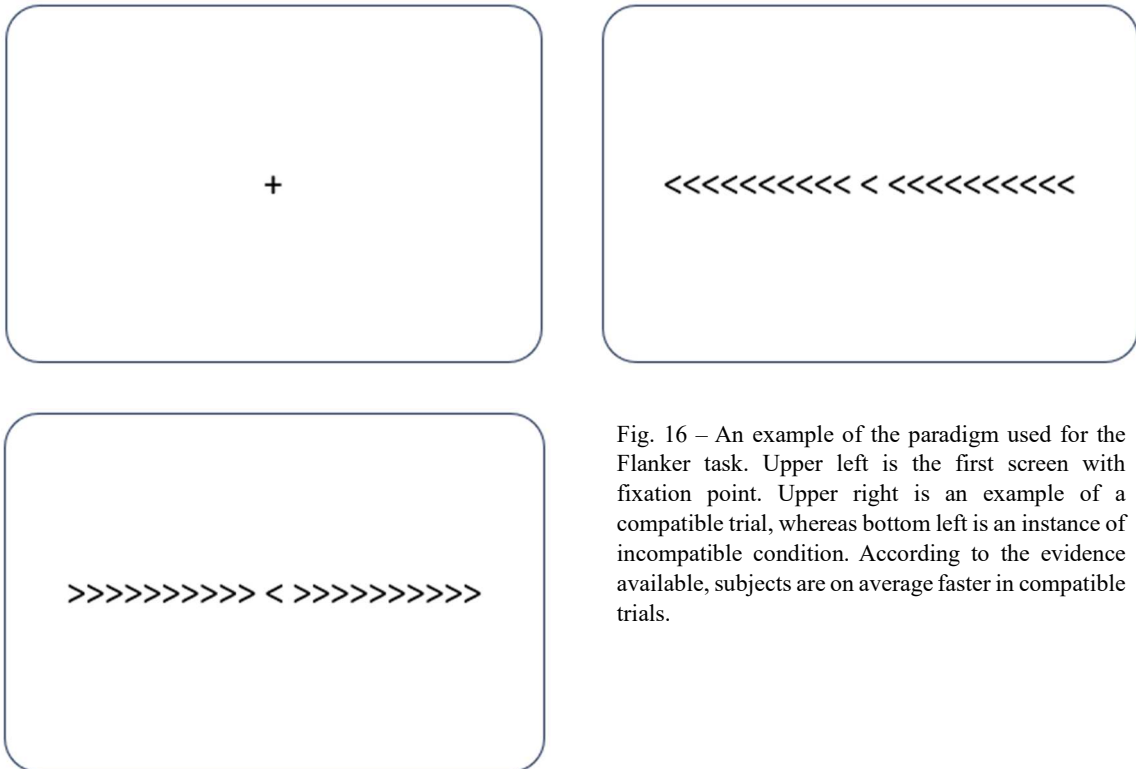


Fig. 16 – An example of the paradigm used for the Flanker task. Upper left is the first screen with fixation point. Upper right is an example of a compatible trial, whereas bottom left is an instance of incompatible condition. According to the evidence available, subjects are on average faster in compatible trials.

Go/NoGo paradigms are of particular interest since a computerised Go/NoGo task has been administered during the study involving racing drivers that will be discussed in the next chapters of the current manuscript. Go/NoGo tasks are a type of neuropsychological test designed for the measurement of self-control, sustained attention, which stems from inhibitory control, and its flipside, known as impulsivity (Mestanikova et al., 2015). In the traditional version of this test participants are required to selectively respond as rapidly as possible, generally with a button-press, to the presentation of the Go stimuli, and to inhibit such a response whenever the NoGo stimuli are presented. In the most common variant of this paradigm (Fig. 17) the requirement is to press a computer key only after an “X” is presented, and to inhibit the response whenever a “Y” is shown. Nevertheless, other variants have been reported in the literature and validated, such as those using faces with emotions (Yu et al., 2014), coloured circles and geometric shapes (Thomalla et al., 2014), airplanes (Go) and bombs (NoGo; Nelson et al., 1998), as well as auditory modalities (Shucard et al., 2008).

Furthermore, more complex designs have been developed, such as those where the subject is initially required to respond to “X” and then to “Y” in an alternated fashion (Garavan et al., 1999). Usually, Go trials are presented more frequently than NoGo ones, in order to make the corresponding response the automatic action to perform (Gacsi and Bunford, 2021). Performance efficiency is usually assessed in terms of correct detections (accuracy), number of responses performed by mistake to No/Go trials (commissions), number of omitted responses to Go stimuli (omissions) and reaction times (RTs; Tab. 6; Mestanikova et al., 2015). In particular, from a cognitive standpoint, errors of commission are interpreted as a sign of impulsivity, while those of omission are supposed to reflect attentive failures (Barkley, 1991; Halperin et al., 1991). RTs can instead be taken as an index of processing speed, whereas reaction time variability (RTV) may be a proxy of the individual’s nervous system stability (Karalunas et al., 2014).

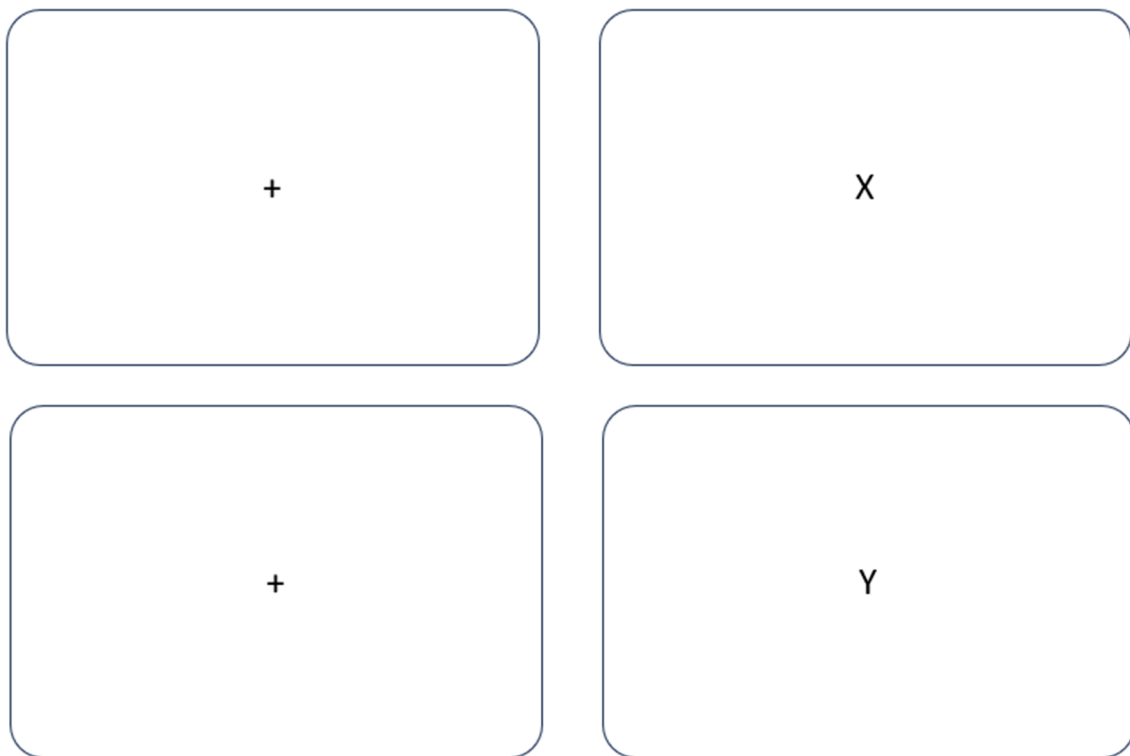


Fig. 17 – An example of a traditional Go/No paradigm. Upper left, the initial slide with the fixation point. Upper right, a Go trial, with the target stimulus “X” requiring to press a key (*e.g.*, the spacebar). Bottom left, another fixation slide, followed by a NoGo trial, with the stimulus “Y” requiring to inhibit the response.

Correct detection	indicate the number of times the client responded to the target stimulus
Omission errors	indicate the number of times the target was presented, but the client did not respond/click the button
Commission errors	indicate the number of times the client responded but no target was presented
Reaction times (RT)	this measure the amount of time between the presentation of the stimulus and the client's response

Tab. 6 – Mestanikova et al., 2015 – Standard parameters assessed in Go/NoGo CPTs

Finally, two different versions of the stop-signal paradigm exist. In the one reported by Aron, Robbins and Poldrack (2004) subjects are required to perform speeded responses on Go trials (*e.g.*, when a “X” is presented), as well as to withdraw from responding in NoGo trials (*e.g.*, when a “Y” is shown) and in Stop trials (Fig. 18) – *i.e.*, when the target Go stimulus is presented and immediately followed by another stimulus (usually a beep) indicating to inhibit the automated response. In another version, reported by Diamond (2013), the Go signal is presented on all trials and on a minority of them is followed by the Stop signal, indicating not to perform the automated response. In both versions of this paradigm, the stop-signal reaction time (ssRT) – *i.e.*, the duration of the stopping process – is taken as an index of inhibitory control.

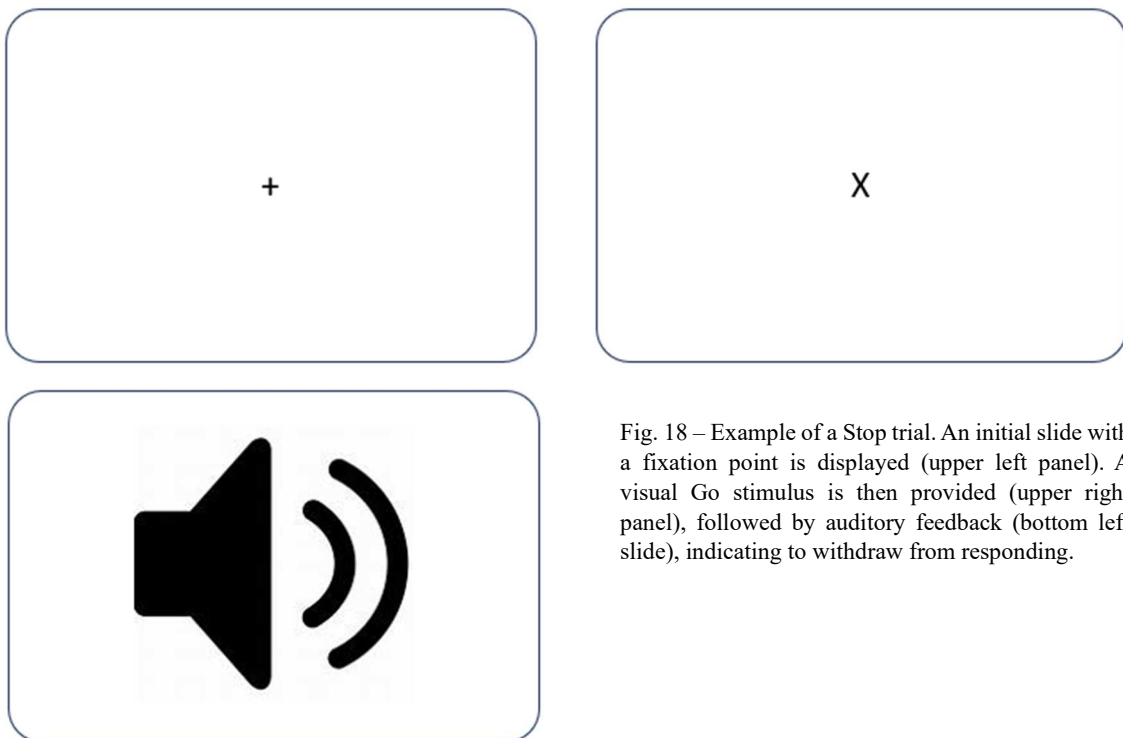


Fig. 18 – Example of a Stop trial. An initial slide with a fixation point is displayed (upper left panel). A visual Go stimulus is then provided (upper right panel), followed by auditory feedback (bottom left slide), indicating to withdraw from responding.

2.3.2 Working memory

According to the most widely held view in cognitive psychology, working memory has been defined as the ability to hold non perceptually present information in mind while mentally working with it, for instance to create relationships between different items and concepts or to solve a problem and complete a task, allowing to reach a cognitive or behavioural goal (Baddeley, 2000; Diamond 2013). The construct of WM was introduced for the first time by Baddeley and Hitch (1974), who proposed a 3-component model (then revised; Fig. 19) including a central executive, acting as an attentional control hub, and two slave systems, known as the phonological loop, critical for manipulating and storing speech-based information, and the visuo-spatial sketchpad, performing a similar function for visual and spatial information (Baddeley and Hitch, 1974; Baddeley and Hitch, 1994). Subsequently, Baddeley (2000) reformulated the multi-componential model of WM by adding a third slave system, named episodic buffer (Fig. 20). Furthermore, previous research reported that WM is the product of the interaction between functionally specialised brain regions, subserving different cognitive subprocesses integrated within a large-scale fronto-parietal network (Owen et al., 2005)

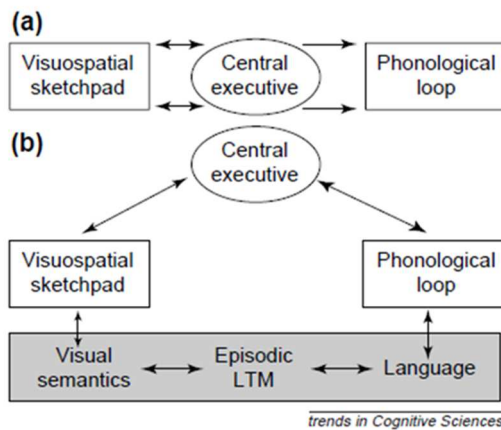


Fig. 19 – Baddeley, 2000. (a) The initial three-component model of working memory proposed by Baddeley and Hitch, postulating an attentional controller (central executive), and two slave systems – *i.e.*, the phonological loop and the visuospatial sketchpad.

(b) A revision of the WM model, highlighting the interactions between WM components (white) and crystallised cognitive systems, in charge of long-term knowledge, and in particular, between: (1) phonological loop and long-term phonological learning mechanisms in children (mother tongue) and adult (foreign languages), (2) visuospatial sketchpad and visual semantic storages.

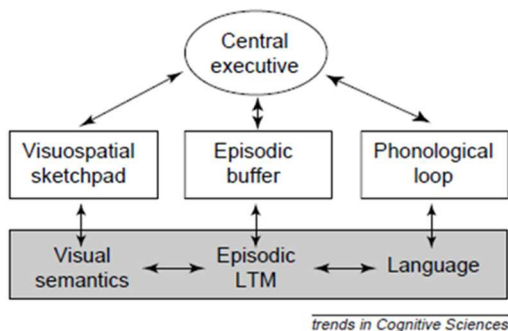


Fig. 20 – Baddeley, 2000. A further revision of the multi-component WM model. The Episodic buffer, here included, is assumed to store information in a multi-dimensional code, and to provide a temporary interface between the other slave systems and LTM. Under the control of the Central executive, the Episodic buffer is thought to bind information from different sources into coherent episodic representations that can be accessed and retrieved consciously, contributing to long-term episodic learning.

More specifically, although the mechanisms underlying it are by far the least known, the central executive is considered the main component of WM. It is thought to redirect attentional resources to the slave-systems depending on the current goal and environmental demands, as well as to get access to their contents by directing conscious awareness (Baddeley and Hitch 1994, Baddeley, 2000). Moreover, the empirical evidence available suggests that the central executive plays an important role in several executive functions, including dual task performance, attentional focusing, attention switching and in the interface between WM and Long-Term Memory (LTM), but further research concerning the cognitive and biological processes involved is desperately needed (Baddeley et al., 1986; Baddeley, 1996; Baddeley, 1998).

The phonological loop has been characterised as “a temporary phonological store in which auditory memories decay over a period of few seconds, unless revived by articulatory rehearsal”, and the verbal WM stemming from it is thought to support the retention of sequential information (Baddeley, 2000). From a phylogenetic standpoint, the phonological store component of the loop is assumed to be evolved to support speech perception, whereas the articulatory rehearsal one to support speech production. Overall, this model of phonological loop provide an effective account of different phenomena, such as: (a) the phonological similarity effect, according to which sequences of similarly sounding letters or words are harder to be remembered; (b) the word length effect, showing that it is on average easier to recall a sequence of short words rather than of long ones; (c) the effect of articulatory suppression, displaying that it is more complicated to remember a verbal item, when one is not allowed to rehearse it; (d) the transfer of information between codes, consisting of the translation of visual information (*e.g.*, a written word) into the auditory code.

The visuo-spatial sketchpad is instead assumed to hold visuospatial information and to underlie visuospatial WM, as well as to be fractionable into separate and dissociated visual, spatial and kinaesthetic components, mainly relying upon the right prefrontal structures (Baddeley and Hitch, 1994; Baddeley, 2000; Logie, 1986; Mishkin, Ungerleider, and Macko, 1983). In particular, the sketchpad has been proposed to be involved in different functions fundamental also for racing driving, such as the planning and execution of spatial tasks, (Hatano and Osawa, 1983), the creation and updating of visuospatial models to keep track of changes occurring in the visual perceptual world over

time (Kahneman, Treisman and Gibbs, 1992), as well as the maintenance of orientation in space and the direction of spatial movements (Thomson, 1983).

Finally, the episodic buffer has been assumed to be a limited-capacity temporary storage system, capable of integrating pieces of information coming from different sources, such as multimodal perceptual representations from the loop or the sketchpad, and episodic information from LTM (Baddeley, 2000). Like the other two slave systems, the episodic buffer is under the control of the central executive, which provides attentional and computational resources and has access to the buffer's contents via conscious awareness. Overall, the ability of this slave system to integrate information across space and time using a multidimensional and multimodal code allows to create and manipulate temporary representations of the physical external environment (*e.g.*, a section of the racing track) as well as of the internal cognitive one. This gives the possibility to create new flexible, goal-directed cognitive representations, and represents a crucial interface between memories and consciousness (Baddeley, 2000).

In the literature, different measures of WM have been used, all having as a common element the requirement of storing and processing the given information (Waters and Caplan, 2003). For instance, a first group of WM paradigms requires the subject to perform an operation on each item of a list or on a list as a whole before repeating a particular item or the list itself. Examples of these tasks include the alphabet span (Craik, Klix, and Hagendorf, 1986), where the individual is asked to repeat back a list of words after arranging them in alphabetic order, the backward digit span (Botwinik and Storandt, 1974), requiring to repeat back a list of digits in reverse order, and the subtract 2 span task (Salthouse, 1988), where the participant has to repeat a series of digits subtracting two from each. A second group of measures requires the subjects to process and store a list of items and, at a certain point of the list itself, to retrieve a previously presented item related to the one currently presented. One of the main paradigms belonging to this group is the N-Back (Kirchner, 1958), which has also been used in the experiment on racing drivers that will be discussed in the next chapters. In particular, two main variants of N-Back exist. In the first one, known as "attentional 0-back task", the subject is required to respond every time the presented stimulus (*e.g.*, a letter) matches a pre-specified target (*e.g.*, the letter "c") (Miller et al., 2009). This condition is thought to predominantly test attentional processes (Nikolin et al., 2015). In the second and most common variant,

instead, the individual is asked to respond, usually by pressing a computer key, whenever the displayed stimulus matches the one presented “n” positions before, and it is thought to be a sensitive measure of WM (Nikolin et al., 2013). In this version, the cognitive load, defined by the chosen “n”, is taken as an independent variable, whereas measures of accuracy and response latencies are considered as dependent variables and indexes of the ability to maintain and update information in the WM (Nikolin et al., 2015). As a general rule, when increasing the cognitive load, WM performance is expected to decrease. A third group of WM measures, instead, is the one requiring reporting the missing item from a sample presented a second time. An example of these measures is the missing digit task (Talland and Quarton, 1965). Finally, other paradigms require to perform an operation while simultaneously storing items unrelated to it, such as in the case of the operation word span task (Turner and Engle, 1989), where the participant is asked to complete a word or digit span task, while also carrying out an arithmetic task.

2.3.3 *Cognitive flexibility*

As previously mentioned, the third core EF is known as cognitive flexibility (the opposite of rigidity) and builds on inhibitory control and WM, appearing later in development (Davidson et al., 2006; Diamond, 2013; Garon et al., 2008). This function is thought to be critical for many skills important for everyday life, since it allows the subject to change perspective spatially and interpersonally, giving the possibility to “wear someone else’s shoes” and to use the Theory of Mind (Bock, Gallaway and Hund, 2015; Diamond, 2013). Moreover, it is considered to be the fundament not only of creativity, but also of other secondary executive functions, including task switching and set shifting (Diamond, 2013). Finally, cognitive flexibility underlies the ability to flexibly adapt to changed goals, priorities, and environmental demands, as well as to take advantage of sudden unexpected opportunities, being therefore fundamental also in the context of racing driving, where the surroundings are constantly and quickly changing (Diamond, 2013).

As in the case of the two other core EFs discussed in the previous paragraphs, a set of multiple tasks has been developed in order to study and assess also cognitive flexibility. Very common ones are, for example, design fluency tasks, where the individual is required to report all the uses they can think of an object, as well as phonologic or

semantic fluency one, where the subject is respectively asked to report all the words they can think of, that begin with a given letter or belong to a specific semantic category. Moreover, this EF is often investigated also by means of task-switching and set-shifting paradigms (Diamond, 2013). A very common one is the Wisconsin Card Sorting Test (WCST; Milner, 1963; Stuss et al., 2000), a test measuring set-shifting ability and tapping onto prefrontal functioning, that requires the participant to deduce the initial sorting criterion based on the feedback given and to flexibly switch to the new one, once it has changed (Diamond, 2013). Common task-switching paradigms are instead the Trail Making Test-B (tapping onto all the three core EFs), where the subject is required to constantly change from the alphabetic dimension to the numeric one (Sanchez-Cubillo et al., 2009), and other ones involving bivalent stimuli, characterised by features relevant to each of the two tasks. Examples of these paradigms are those including pairs of letters and digits (*e.g.*, A2) and requiring the subject to first indicate whether the letter is a vowel or a consonant and then whether the digit is even or odd. Overall, the available evidence shows that even cognitively intact healthy adults are slower to respond on trials where the relevant dimension switches (*i.e.*, they display an inertial tendency), indicating that these tasks are effective in taxing cognitive flexibility (Allport and Wylie, 2000; Meiran, 1996; Rogers and Monsell, 1995).

To wrap up, in these paragraphs the most widely held theoretical frameworks concerning human executive functions and the most common cognitive tests used to assess them have been discussed. Altogether, theoretical constructs and evidence-based data highlight the extreme importance of EFs in everyday life, and their role in making an individual flexible and able to adapt to challenging and quickly changing environments, increasing their chance of survival and success in life. Finally, it seems reasonable to assume, and will be further discussed in the next chapters, a critical role played by inhibitory control, WM, cognitive flexibility and the higher functions stemming from them (Fig. 21) not only when it comes to pure cognitive performance, but also in the context of racing driving and peak sport performance.

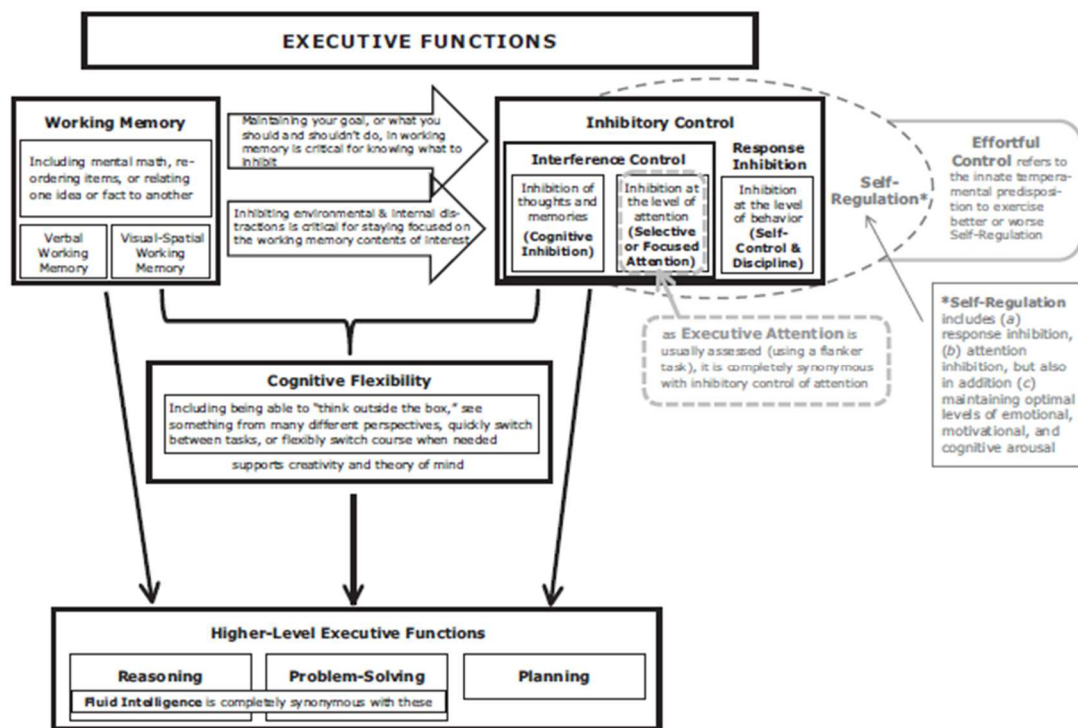


Fig. 21 – Diamond, 2013 – Executive functions and related terms

2.4 Neurovisceral Integration Perspective

Thayer and colleagues proposed a model, known as the Neurovisceral Integration Perspective. This model merges evidence coming from different branches of neurophysiology, neuropsychology and cognitive psychology, supporting the existence of a neural network including structures involved in cognitive, affective and autonomic regulation, underlying the relationship among cardiac autonomic control and executive functioning (Thayer et al., 2009). According to this model, vagally mediated HRV may be a peripheral index reflecting the activity and functional capacity of a set of neural structures involved not only in emotional regulation, but also in the performance of cognitive and especially executive tasks, such as the ones involving inhibitory control and working memory (Thayer et al., 2009; Thayer and Lane 2000). For this reason, the Neurovisceral Integration model claims that subjects with greater resting HRV perform better on tasks relying on executive functions, but not on those relying on non-executive ones. This claim is supported by different studies comparing the performance at different

types of cognitive tasks, as well as by others manipulating resting HRV, both in lab and in ecologically valid contexts.

Previous research reported a continuous activation of prefrontal cortices necessary for the performance of executive functions, allowing for a flexible selection and execution of behaviours that are appropriate to the given context, and the inhibition of other ones that are instead inappropriate (Angius et al., 2019; Compte et al., 2000; Goldman-Rakic, 1996; Miller 2000). Moreover, multiple systems belonging to the CNS (including the PFCs) and involved in cognitive functioning and emotional regulation have been reported (*e.g.*, the rostral limbic system [RLS] and Damasio's emotion circuit), allowing for a flexible adaptation to the given contexts (Damasio, 1998; Devinsky, Morrell and Vogt, 1995).

According to the Neurovisceral Integration model, direct and indirect pathways exist, through which PFCs influence parasympathetic activity by modulating the activation of subcortical structures (Fig. 22; Benarroch, 1993; Ter Horst & Postema, 1997; Thayer and Lane, 2000; Spyer, 1989). The central autonomic network (CAN) has a central role in the Neurovisceral Integration model. It is a network of functional units belonging to the CNS, involved in the visceromotor and neuroendocrine control underlying goal-directed behaviours and adaptability (Benarroch, 1993; Benarroch, 1997). Specifically, the CAN includes the anterior cingulate and the insular cortex, the orbitofrontal and ventromedial PFCs, the central nucleus of the amygdala (CeA), the paraventricular and related nuclei of the hypothalamus, the periaqueductal grey matter, the parabrachial nucleus, the nucleus of the solitary tract (NTS), the nucleus ambiguus (NA), the ventrolateral medulla, the ventromedial medulla, and the medullary tegmental field (Thayer et al., 2009). The CAN is thought to allow for bidirectional flows of information between lower and higher centres of the CNS. On the one hand it allows for the bottom-up integration of peripheral information. On the other hand, the CAN makes it possible to control autonomic functioning, through top-down influences that are mediated by the action of preganglionic neurons belonging to both the PNS and SNS, directly influencing HRV (Saul, 1990). Therefore, it has been suggested that HRV can be considered as an indicator of CNS-ANS integration and central-peripheral neural feedback. According to the Neurovisceral Integration Perspective CAN, RLS, the emotion circuit and other related networks belong to a common functional network responsible for response organization and selection, as well as for the management of physiological resources sustaining cognition, attention and

emotion (Friedman and Thayer, 1998; Thayer and Friedman, 1997; Thayer et al., 2009). Thus, this network is fundamental for the organism to integrate central and peripheral information, in order to be flexible and able to cognitively and physiologically adapt to quickly changing environmental demands.

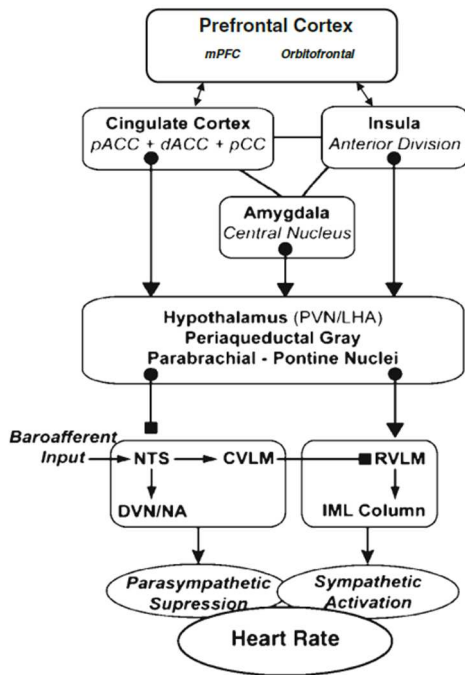


Fig. 22 - Thayer et al., 2009. A diagram showing the pathways theorised to be used by the PFC to influence the control of HR and cardiac activity. The interconnected network including PFC, anterior cingulate cortex and the insula communicates bidirectionally with the amygdala, which is under tonic inhibitory control, exerted by prefrontal vagal pathways

The activation of the CeA inhibits the NTS, which in turn inhibits the caudal ventrolateral medullary (CVLM) inhibitory action on the rostral ventrolateral medullary (RVLM) sympatho-excitatory neurons, simultaneously inhibiting vagal motor neurons in the nucleus ambiguus (NA) and the dorsal vagal motor nucleus (DVN). Furthermore, the CeA can directly trigger the sympatho-excitatory action of the neurons in the RVLM.

Overall, the effect of a blockade of PFC activity would lead to the disinhibition of the CeA, inducing disinhibition of medullary cardioacceleratory circuits and an increase in HR. Figure adapted from Gianaros.

2.4.1 CNS and cortical influence on cardiac activity control

As mentioned above, findings on animal model studies and human studies support the presence of cortical modulation of cardiovascular function. In particular, the CAN modulates the activity of the pacemaker cells in the SA node through the stellate ganglia and the vagus nerve, and its own output is under tonic inhibition by GABAergic neurons located in the nucleus of the solitary tract. According to the Neurovisceral Integration model summarised in Fig. 22, PFCs exert tonic inhibition of the amygdala through the action of GABAergic neurons. Decreased PFCs inhibitory activity induces the activation (or disinhibition) of the central nucleus of the amygdala (CeA), leading to increased HR and reduced HRV, following three different routes:

1. disinhibition of tonically active sympathetic excitatory neurons in the rostral ventrolateral medulla (RVLM) leading to increased sympathetic activation;

2. inhibition of neurons in the NTS, inhibiting neurons belonging to the dorsal vagal motor nucleus, decreasing parasympathetic activity;
3. direct activation sympathetic neurons in the RVLM, underlying an increase in sympathetic activation (Thayer et al., 2012; Saha, 2005).

Therefore, decreased PFCs activity would lead to the disinhibition of the CeA, inducing an increase in sympathetic activity and an inhibition of the parasympathetic one, leading to increased HR and decreased HRV. Conversely, according to this model PFCs activation tonically inhibits cardio-acceleratory networks, increasing HRV and decreasing HR (Thayer et al., 2009). This is supported by a consistent body of research, including both pharmacological and neuroimaging studies. Concerning the formers, Ahern and colleagues (2001) studied the effects of intracarotid administration of sodium amobarbital (ISA), a drug used to inhibit prefrontal activity, on HR and HRV in epileptic patients. Their findings showed a significant increase in HR both for right and left hemisphere inhibition, with the increase being bigger in the right hemisphere inhibition condition (Fig. 23). Concerning HRV, the results displayed a significant increase in the LF-to-HF ratio following right hemisphere inhibition, whereas when the left hemisphere was inhibited only a non-significant trend was found (Fig. 24). Overall, these findings are consistent with the model discussed above, since prefrontal inhibition led to an increase in HR and a decrease in vagally mediated parasympathetic cardiac control. Moreover, this suggests not only an involvement of the prefrontal cortices in the inhibition of subcortical sympatho-excitatory cardio-acceleratory circuits, but also a predominance of the right hemisphere in cardiac chronotropic control.

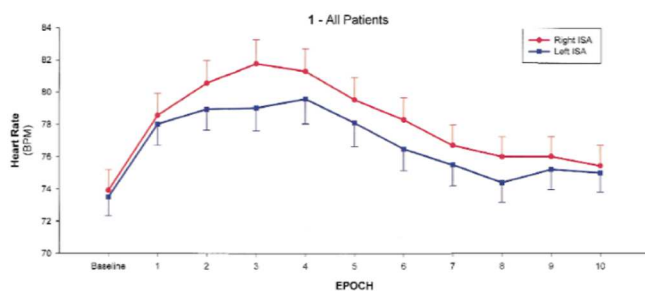


Fig. 23 – Ahern et al., 2001. Raw HR values (bpm) recorded during left and right ISA administration for the group as a whole (N = 73). The data displayed refer to the averages of the 5–10 min before injection (Baseline) as well as to 10 sequential 1-min Epochs after injection. <-

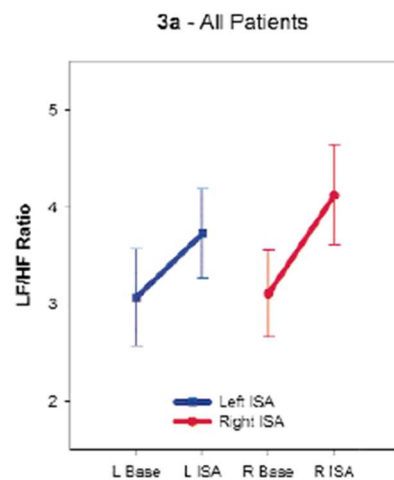


Fig. 24 – Ahern et al., 2001. Changes in the LF/HF ratio occurring during left and right ISA administration for all 73 patients. ->

As mentioned before, also neuroimaging studies support this model, by providing evidence for the role played by prefrontal cortices and other cerebral structures in modulating cardio-acceleratory circuits by enhancing vagal function, leading to increased vagally mediated HRV. For instance, Lane's research group (2008) assessed the neural correlates of HF-HRV during emotional activation, by measuring regional blood flow (rCBF) with positron emission tomography (PET) while displaying neutral or emotional videos. First, their results showed a non-significant reduction of HF-HRV during the emotional condition as compared to the neutral one. Second, across the emotional and neutral conditions a broad bilateral network of cerebral areas positively correlated with HF-HRV, including the medial PFC (mPFC), the ventral striatum and the periaqueductal grey, as well as the left mid-insula, whereas the cuneus showed a negative correlation. Third, independent of the stimulation being emotional or neutral, a significant correlation was found between HF-HRV and the right dorsolateral PFC (dlPFC), pregenual medial PFC, anterior cingulate cortex (ACC) and bilateral parietal cortices. Altogether, the data presented here support the involvement of central nervous structures in the modulation of cardiac vagal control during emotional and non-emotional contexts, with cognitive (neutral images) and emotional (emotional images) control respectively recruiting partially non-overlapping networks. Other pieces of research provided additional evidence supporting this view. For example, a study conducted by Lane and colleagues (2013) assessed the correlation between the activation of the subgenual anterior cingulate cortex (sgACC), of other anterior medial visceromotor regions, and the HF-HRV power band, in healthy and depressed subjects, using a variant of the emotional counting Stroop task. In healthy participants, their results showed a significant correlation between changes in the activation of the sgACC and other related structures (as index by the blood oxygen level dependent [BOLD] signal) and those in HF-HRV power when shifting between background emotional states, whereas in depressed individuals no relationship was found. These findings support the involvement of the CNS and, in particular, anterior cortical structures in the regulation of cardiac activity, with increased cortical activation being related to increased vagally mediated HRV.

2.4.2 *CNS and cortical influence on executive functions*

PFCs are well known for being involved in the performance of executive functions, such as cognitive, emotional and behavioural inhibition, working memory, cognitive flexibility, attentional regulation, set-shifting, extinction and sustained attention, allowing for mental and behavioural flexibility and adaptability to constantly changing environmental demands (Thayer et al., 2009). A consistent body of research supports the view seeing PFCs as the neural substrate responsible for executive functioning and, in particular, inhibition. For instance, Aron and colleagues reviewed the available evidence in order to assess the involvement of the right inferior frontal cortex (riFC) in the network responsible for inhibition, also postulating the mechanisms through which PFCs influence subcortical and posterior-cortical regions to implement cognitive control (Aron, Robbins & Poldrack, 2004). Although initial evidence supporting PFC involvement in inhibitory control was gathered using WCST, due to its complex nature involving different cognitive processes, more recent research on the topic focused on the administration of more purely inhibitory tasks, such as response inhibition and task/set switching paradigms. Overall, a wealth of studies provided support to the claim of riFC being crucial for the inhibitory component of these tasks.

As previously discussed, response inhibition is a cognitive process required to cancel an intended movement/process, and it is tested using Go/NoGo and stop-signal tasks (Aron, Robbins & Poldrack, 2004). Neuroimaging studies displayed that response inhibition is consistently linked with the selective activation of right inferior frontal areas. For instance, Garavan, Ross and Stein (1999), used functional magnetic resonance imaging (fMRI) and a modified version of the classic Go/NoGo task to assess the neural structures underlying inhibitory control. Overall, they reported that in healthy subjects inhibitory control was associated with a predominantly right-lateralised network, primarily involving the right middle and inferior frontal gyri and, secondarily, other right parietal and limbic areas. These findings have been replicated by another study conducted by Garavan's research group, using event-related fMRI and a Go/NoGo task. The findings showed that effective inhibitory control relied on a timely activation of a complex right-lateralised neural network, primarily depending on the right inferior and dorsolateral prefrontal areas (Garavan et al., 2002). Similar findings have been reported also by neuropsychological studies involving subjects with brain lesions. In particular, Aron and

colleagues (2003) assessed inhibitory control in a sample of patients with different right prefrontal lobe lesions, one with left prefrontal lesions, and one of matched controls, using Go/NoGo and stop-signal tasks (Fig. 25), known to recruit the right inferior frontal gyrus (IFG) in healthy subjects. In particular, the participants had to respond to a left- or right-pointing arrow presented on a screen, by pressing respectively a left or right key as quick as possible (Go task), unless they heard a beep (NoGo task), in order to provide a sensitive estimate of inhibitory control. Results showed that the stop signal reaction time (ssRT) was significantly slower in patients with right prefrontal damage compared to patients with left prefrontal lesions and controls. Second, and most importantly, the only lesion site correlated with the ssRT was the right IFG, meaning that the right, but not left, IFG plays a critical role in inhibitory control. Overall, the results are consistent with the crucial involvement of a discrete region of the right PFC, known as IFG, in a specific executive function crucial for adaptive and flexible behavioural outputs (and thought to be important also for racing driving) – *i.e.*, inhibitory control.

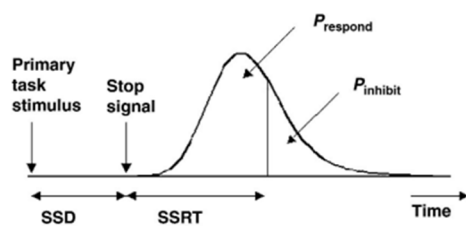


Fig. 25 – Aron et al., 2003. The model used for the estimation of ssRT. No-signal RTs (go trials) are distributed under the curve. On stop trials, a tone occurs after the primary go stimulus at a given stop-signal delay (SSD). The stop signal divides the no-signal RT distribution into two probabilities: a part consisting of responses that are fast enough to escape inhibition (P_{respond} ; left) and a part corresponding to P_{inhibit} (right). Provided SSD is varied to yield 50% P_{inhibit} (the point of median no-signal RT), ssRT is estimable by subtracting average SSD from median no-signal RT. SSD was adjusted based on the subject's performance, in order to keep the P_{inhibit} around 50%.

Convergent evidence suggesting right PFC involvement in inhibitory control was provided also by studies using task-switching paradigms, in which subjects are required to change from performing one task to another, using executive control. Dreher and Berman (2002) used this paradigm along with event-related fMRI, in order to assess the neural processes underlying the switching from a currently performed task, that has to be inhibited, to a previously performed one. The right PFC was more activated when subjects were required to switch to a recently performed task. Similar findings have been reported also by neuropsychological studies (Aron et al., 2004b). They examined task-switching performance in patients with left- and right-PFC lesions, as well as in healthy controls. Results displayed that both patients' groups showed significantly larger switching costs

than controls. In particular, left-lesioned patients showed an impaired top-down control of task sets, required to impose the new mental sets and indexed by a longer time required to perform the switch. Right-lesioned patients, instead, were impaired in the ability to inhibit inappropriate responses and task-sets, committing more errors. Such an impairment was correlated with focal lesions to the pars opercularis of the right IFG. Overall, these findings are consistent with the idea that right PFC and, in particular, the right IFG, are involved in inhibitory control.

Based on the findings discussed in this paragraph, it seems reasonable to claim that cognitive and motor inhibition are related to the activation of the right PFC and, in particular, of the right IFG. Nevertheless, right IFG is not the only structure involved in inhibitory processes, since they are likely to be the result of a more complex and dynamic inhibition occurring at a system-level, involving different regions and structures of the CNS. Based on the evidence coming from different neurophysiological studies, Aron and colleagues suggested in their review (2004) that cognitive inhibitory control, intended as the ‘suppression of inappropriate responses, S-R mappings or task-sets when the context changes, and suppression of interfering memories during retrieval’, might depend on the complex interaction between right PFC, memory-related structures of the Medial Temporal Lobe (MTL), and basal ganglia. Indeed, an fMRI study by Anderson and colleagues (2004), showed that a form of cognitive inhibition – *i.e.*, the suppression of unwanted memory – was associated with greater bilateral dlPFC, vmPFC and ACC activation, as well as reduced right hippocampal activity during suppression as compared to a memory retrieval condition. This suggests an interaction between prefrontal regions and medial temporal structures, aimed at performing an inhibitory control, by suppressing unwanted memories. Moreover, other studies have also reported the involvement of the subthalamic nucleus (STN) in inhibitory processes. Van den Wildenberg reported that patients with Deep Brain Stimulation (DBS) of the STN showed better response inhibition than those with DBS of the thalamus (Aron et al., 2004). Moreover, Hershey and colleagues (2003) assessed cerebral blood flow (CBF) responses to STN stimulation using PET in patients with Parkinson Disease. The results showed that STN stimulation induced excitation of the output neurons in the STN, leading to excitation of the globus pallidus and consequently to the inhibition of thalamic nuclei, resulting in a reduction of excitatory output toward the cortices and enhanced cortical inhibition. Altogether, the

findings reported by the studies discussed seem to be consistent with the model of cognitive inhibition proposed by Aron's research group (2004), hypothesising the existence of an inhibitory system starting from the right IFG, also involving structures of the basal ganglia and the MTL. Overall, the information reported above highlight the contribution of the PFC to cognitive inhibitory control, which is of fundamental importance not only for the performance of all the other executive functions, but also for the execution and maintenance of flexible behaviours, which are adaptive to complex, dynamic and demanding environments, such as the one characterising racing driving.

Another executive function traditionally associated with the activation of the PFC and critical for the performance of context-dependent goal-directed behaviours (such as those involved in racing driving) is working memory (WM). The most widely held model within the scientific community is the one seeing WM not as stemming out from a discrete limited neural network, but at a system level, as a property of the whole brain that supports the successful attainment of goal-directed behaviours carried out by any of several neural systems, including sensory and motor ones, as well as semantic and episodic memory (D'Esposito & Postle, 2015). According to this model, a central role is played by prefrontal cortices, which maintain task-relevant information online during the period of time between the presentation of the information and the actual performance of the required action. This view is supported by a consistent body of research reporting enhanced PFC neural activation during such a period of time, in both animals and humans. A seminal study in this field is the one published by Fuster & Alexander (1971), where monkeys were required to actively maintain information previously shown and no more present, in order to perform a delayed-response task. The results showed a persistent tonic activation of neurons within the PFC during the delay period, critical for the subsequent successful completion of the task. Such findings were then replicated in animals by Kubota & Niki (1971), and in humans by Courtney's (1997) and Zarahn's (1997) research groups. Although it is not clear yet whether this persistent prefrontal activation underlies the activation of cortico-cortical, thalamo-cortical, or local cortical loops, overall, the evidence presented supports the crucial involvement of PFCs in WM, as well as the ability of prefrontal neurons to maintain task-relevant information in absence of the original external stimulus, in order to support goal-directed behaviours. According to the system-level model of WM (D'Esposito & Postle, 2015) such a working memory storage would

depend on transient prefrontal synaptic weights reorganisation rather than on sustained elevated neuronal activity, allowing for the flexible representation of the relevant pieces of information. PFC seems to follow a hierarchical organisation, according to which the rostral regions represent abstract rules and goals and the caudal ones abstract representations of stimulus categories, while cerebral regions located posteriorly to the central sulcus would contain feature- and stimulus-specific representations. The contents represented by the different cortical regions, would then be integrated by cortico-cortical long-range connections, allowing to synchronise the activity of PFC to that of other brain regions, also thanks to the action of cortical dopaminergic projections originating from brainstem nuclei. In particular, some researchers proposed the idea that midbrain striatal neurons would be responsible for two types of gating processes, one regulating the update of the information held by PFC (input gating), and another one influencing the top-down signals sent by the PFC to other lower-level areas of the brain (output gating) (Badre & Frank, 2012, Chatham & Badre, 2013; Frank & Badre 2012). These mechanisms would be consistent with the critical role played by the PFC in WM, since the rostral PFC could use abstract representations of goals and rules to regulate the activity of striatal gating toward the caudal PFC, influencing the contextual representation maintained there. Altogether, the system-level model of WM memory presented here highlights not only the role played by PFCs in the performance of the executive function known as working memory, but also the importance of WM itself in the successful performance of goal-directed behaviours in dynamically changing contexts, such as the one characterising racing driving.

In conclusion, the empirical evidence and theoretical models discussed in this paragraph show well the fundamental role of the CNS and, in particular, of PFCs in regulating executive functioning, and how this is critical for the adaptive, successful and effective individual functioning in highly demanding, constantly changing, dynamic contexts. This is particularly relevant for the experiment that will be discussed in the next chapters of this manuscript, since it will be focused on the performance of racing drivers, who have to constantly compete in environments characterised by high-speed flows of information, requiring a detailed and updated representation of the surroundings, as well as the fine regulation of relevant and the inhibition of irrelevant pieces of information, in order to

perform highly challenging goal-directed behaviours, such as controlling the car at high speed, correctly interpreting other drivers' intentions and overtaking the opponents.

2.4.3 Prefrontal activity, HRV, executive functions

According to the Neurovisceral Integration Perspective (Thayer et al., 2009; Thayer and Lane 2000), a network involving cortical (especially the PFCs) and subcortical areas is responsible for cognitive, executive and emotional modulation, and is also involved in the modulation of physiological ANS activity, supporting homeostasis and goal-directed behaviours (Thayer and Brosschot, 2005; Thayer and Lane, 2000; Thayer et al., 2009). HRV has been suggested as a reliable peripheral index of prefrontal functioning, reflecting the inhibition of sympatho-excitatory circuits, and the performance of executive functions (Thayer and Brosschot, 2005). This view has been strongly supported by a wide body of research, reporting the presence of a positive correlation between measures of HRV and different executive functions, including studies assessing how individual differences in vagally-mediated HRV relate to the performance at executive tasks, as well as other ones investigating the effect of manipulating resting HRV levels on executive functioning, both in lab and ecologically valid contexts.

In particular, a wealth of studies assessed the relationship between HRV and general executive functioning. For instance, Hansen, Johnsen, and Thayer (2003) assessed the differences in cognitive functioning between a high- and a low-HRV group. In this study, 49 healthy subjects were administered a continuous performance test (CPT), including two non-executive tasks (simple reaction time and response latencies to specific stimuli) as well as two executive ones (detection of identical stimuli and simple addition task). The authors reported that high-HRV subjects displayed a significantly better performance on the two executive function tasks than the ones with low-HRV, whereas no difference was reported regarding the results at the non-executive tasks. Moreover, to test whether HRV may be an appropriate index of adaptability of the organism, the same group of researchers tested the changes in performance at the CPT under stressful conditions (2009). In order to do so, they said to the participants that any performance under a certain threshold would have resulted in the delivery of an electrical shock to the fingers. 4 groups of healthy participants were created, (threat of shock vs no threat of shock, high HRV vs

low HRV). First, the results replicated those of the aforementioned study, with the high-HRV group displaying a significantly better performance than the low-HRV one only in executive tasks, in both no threat and threat conditions. Second, during the threat condition, low-HRV individuals showed a general improvement of their performance, particularly reducing their mean reaction time at non-executive tasks. Overall, these findings are consistent not only with the idea that higher resting HRV may be a marker of better executive functioning, but also that individuals with enhanced resting HRV are more tolerant and resilient to changes in the environment, since changes in the contextual stressor had no impact on their cognitive performance. Moreover, samples of salivary cortisol collected after the completion of the aforementioned cognitive tasks were analysed. The results showed that baseline HRV was negatively correlated with cortisol concentration after the tasks completion, supporting the idea that high-HRV subjects are more resilient to environmental stressors, being able to maintain stable their cognitive performance. Furthermore, behavioural approaches used to manipulate resting HRV have been used to strengthen the evidence supporting the relationship between HRV and general executive functioning. Hansen and colleagues (2004) assessed the relationship between physical fitness, HRV and cognitive functioning. They included 37 sailors of the Royal Norwegian Army, who underwent an 8-week aerobic training programme. Afterward, they were randomly assigned either to a trained group (TG), undergoing 4 weeks of further training, or a detrained one (DG), who did not go further with the programme. Cognitive testing, using a CPT with the structure described above, as well as a physical fitness assessment, measured as maximum oxygen consumption (VO_{2max}), were administered at the end of the initial period of training (pre-test) and after the 4 weeks of experimental manipulation (post-test). At pre-test, results showed no between-group difference in terms of VO_{2max} , HRV and CPT. At post-test, instead, the DG group displayed a significant decrease in VO_{2max} and resting HRV. Moreover, a significant improvement in mean reaction times at executive tasks as compared to the pre-test performance was reported for the TG, but not for the DG. Altogether, these findings are consistent with the hypothesis that vagally mediated cardiac control, indexed by HRV, interacts with prefrontal cortices and the executive functions depending on their activity. Moreover, the evidence showing that manipulation of HRV leads to corresponding changes in executive (but not in non-executive) functioning strengthens the hypothesis of

a causal relationship between neural processes indexed by HRV and executive functions (Thayer 2009).

A branch of research has instead focused on studying the relationship between HRV and WM. For instance, Hansen, Johnsen and Thayer (2003) assessed the correlation between resting HRV and the performance at a 2-back task. The results showed that the high-HRV group performed better than the low-HRV one, showing a better accuracy and being able to detect more true-positive responses. Moreover, these findings were replicated in a study conducted by the same research group (2009) and presented above, where high-HRV subjects showed a better performance at the 2-back task both in the threat and no-threat conditions, as compared to low-HRV ones. According to the authors, within the Neurovisceral Integration Perspective, the good and stable performance shown by the high-HRV group regardless of the level of stress might be explained by a heightened vagal tone associated with a better ability to self-regulate resulting in increased adaptability and flexibility. Moreover, evidence in support of the relationship between HRV and WM was also provided by studies using behavioural interventions to manipulate HRV. The study discussed above conducted by Hansen and colleagues (2004) also focused on the relationship between physical fitness, HRV and WM. They administered at pre- and post-test, along with the CPT, a 2-back task. The results showed a significant increase in true-positive responses at the 2-back for the TG only, at the post-test assessment. Altogether, these findings show that it is possible to behaviourally manipulate HRV, and that such manipulation can affect executive and working memory functioning. Once again, these results support the model according to which vagally mediated processes influencing HRV interacts with PFCs, influencing WM performance.

Concerning inhibitory control, research was conducted using Go/NoGo paradigms in order to assess its relationship with HRV. A paper published by Thayer and colleagues (2009) reported the results of an unpublished study conducted by Hansen, Eid, Sollers, Hugdahl, and Thayer, where they administered a Go/NoGo task to 46 healthy subjects, divided in a low-HRV and a high-HRV group. The participants were required to respond or inhibit their response to culturally relevant or culturally neutral stimuli. The first condition (GreenGo/RedNoGo) mirrored the culturally learned aspect of responding to a green signal and withdrawing from responding in presence of a red one. The second condition (RedGo/GreenNoGo) required to override culturally learned behaviours. The

third subtask (BlueGo/PinkNoGo) and the fourth one (PinkGo/BlueNoGo) were instead neutral conditions, unbiased by cultural learned rules. Results reported a better performance for the high-HRV group in all the conditions, except for the GreenGo/RedNoGo condition, as compared to the low-HRV one. The authors interpreted these findings as evidence supporting the hypothesis of the high-HRV group having an increased ability to adapt to environmental stimuli and changing environmental demand, even when this requires overriding deeply learned responses and culturally acquired rules. Cortese and colleagues (2022) investigated the two-way brain-heart interaction during two Go/NoGo tasks. After a 5-minute baseline recording period, 14 healthy subjects were required to perform two versions of a variant of the classic Go/NoGo task, known as the Rule Shift Cards. In the first task, they were required to press the keyboard spacebar when a white square appeared on the display (Fig. 26). During the second one they were asked to press the spacebar only when two equal visual stimuli were presented consecutively (Fig. 26). Based on the median of the total errors, participants were then divided into a Good Performance and a Poor Performance.

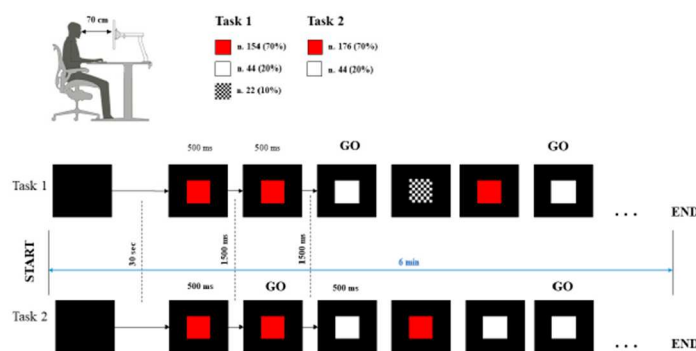


Fig. 26 – Cortese et al., 2022. The participant sits 70 cm away from the monitor with a hand near the spacebar of a keyboard. In the first task the subject is required to hit the spacebar whenever a white square appears on the screen and to withdraw from responding when red or checkered squares are presented. In the second task, the participant is asked to press the spacebar whenever the colour of the square displayed corresponds to the colour of the previous one. Stimulus duration = 500 ms. Interval between the stimuli = 1500 ms.

Results reported a trend toward significance showing a higher baseline lnHF and lnLF HRV in the Good Performance group, as compared to the Poor Performance one. According to the authors, the lack of significance was probably an effect of the small sample size, but the overall results can still be interpreted as a piece of evidence supporting an increased inhibitory control in subjects with higher baseline HRV, consistent with the assumptions of the Neurovisceral Integration Perspective.

Altogether, the findings reported here provide evidence of a specific correlation between baseline HRV and inhibitory capacity, supporting the presence of a relationship between

the neural processes underlying cardiac autonomic control and those giving rise to executive functioning.

Overall, the evidence presented in this paragraph supports the Neurovisceral Integration Perspective, according to which the resting levels of vagally mediated HRV are associated with individual differences in executive functions, including inhibitory control and working memory. In light of this association and of the neuroimaging findings reported before, it seems reasonable to claim that there may be common neural bases for all these processes, and that HRV may be a useful, easy-to-obtain, non-invasive, peripheral index of the integrity of the CNS networks that support goal-directed behaviours and, particularly, of the prefrontal cortices. Most importantly, the evidence summarised here is consistent in supporting the specificity of the relationship between HRV and executive functions, since multiple studies failed to report an association between the former and other non-executive cognitive functions. In conclusion, the theoretical framework and the empirical results discussed support the existence of a reciprocal interconnection between circuits involved in autonomic and cognitive, emotional and behavioural self-regulation, all of which are of extreme importance in the context of racing driving, an activity requiring to constantly and quickly adjust physical, mental and behavioural functioning.

3 *Driving performance and its relationship with cognitive functions and autonomic control*

3.1 *Driving performance and cognitive functioning*

To date, there is a lack of research systematically investigating the direct relationship between individual cognitive functioning and driving performance in professional racing drivers in an ecologically valid context. Studies assessing the importance of cognitive functions for driving performance have been mainly conducted in non-professional populations, sometimes including clinical samples (*e.g.*, neurological patients and patients with cardiovascular diseases). Only a few studies investigated the relationship between cognitive and driving performance in car racing drivers. Also, the sparse studies were conducted using a wide variety of tools and paradigms and focusing on very different populations. Therefore, some initial concepts regarding the relationship between cognitive functions and driving performance will be discussed in the next few paragraphs, but further and more systematic research is needed, to further clarify it.

Racing driving is an activity that requires a high degree of sensorimotor learning and skills acquisition accumulated over years of sustained effort, training, and deliberate practice (Eckardt et al., 2020; Lappi, 2015). From a cognitive viewpoint, it involves multiple high-level cognitive and executive processes, including multisensory integration, decision-making, inhibitory control, as well as planning and execution of flexible goal-directed behaviours, and it requires to combine multiple motor and cognitive components into a coherent global action (Eckardt et al., 2020). Multiple studies reported a strong interrelation between sensorimotor learning and executive functioning, both being correlated with actual driving performance. Such a relationship has been investigated by Eckardt, Roden, Grube, and Schorer (2020), who assessed how cognitive and executive functions related to individual sensorimotor adaptability and driving performance in a Formula 1 (F1) driving simulation. First, they administered to 23 male non-professional racing drivers/e-gamers a set of cognitive tasks to assess response inhibition (SCWT; Stroop, 1935; Simon task; Simon and Wolf, 1963), visuospatial WM (Corsi Block Test; Corsi, 1972; Kessels et al., 2000), mental rotation skills, and WM span (Forward and Backward Digit Memory Test from the Wechsler Adult Intelligence Scale

IV, 2009). Second, participants took part into two driving simulation sessions without opponents. In the first session, they were required to drive as fast as possible while keeping their car on the road (no corner cut was allowed). This first session aimed at matching participants' skills and measuring their adaptive ability, defined as the time required to gain the basic sensorimotor skills needed to reach the threshold performance. The second session aimed at assessing driving performance, operationalised as the mean lap time calculated over ten valid laps, after the driver undercut a pre-set lap time. The results showed that driving experience, measured as the number of years after the acquisition of the driving licence, was not correlated with adaptability nor driving performance. Moreover, inhibitory control and selective attention, as indexed by the performance at the SCWT were positively correlated with both adaptability and driving performance (Fig. 26). Despite the limited sample size, these results support the importance of inhibitory control and executive functioning in sensorimotor adaptability and racing driving performance, as opposed to global non-executive functions, which seems not to be determinant in discriminating a good from a bad driving performance.

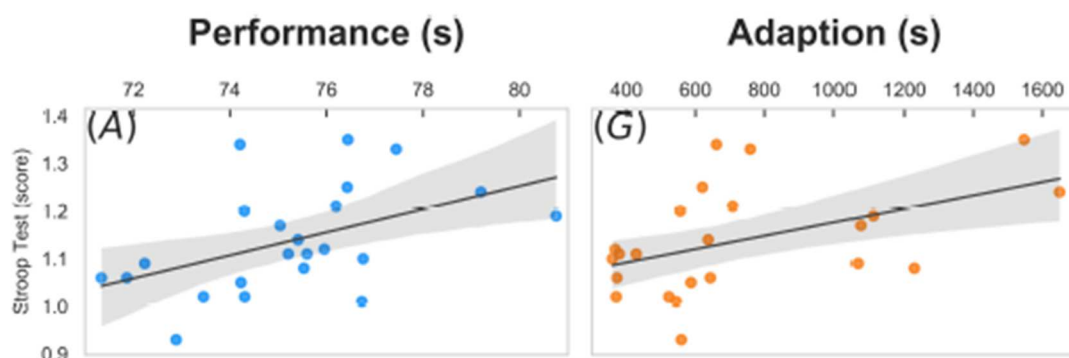


Fig. 26 – Eckardt et al., 2020. Significant correlations between the score at the SCWT and (A) peak driving performance, indexed by the mean lap time over 10 valid laps during the second session and (B) sensorimotor adaptation skills, as indexed by the time required to reach the threshold performance during the first session.

Although the criticality of EFs in driving performance is widely accepted, there is still disagreement concerning the role played by the single executive processes, probably due to the wide variety of cognitive tests and measures of driving performance used. Walshe and colleagues (2017) reviewed the relationship between EFs, and different measures of driving performance in adolescents and young adults in non-racing contexts. Overall, the reviewed evidence suggested that EFs play an important role in driving performance, and that inefficient executive functioning is related to more driving errors, dangerous driving behaviours and higher risks of crashing. The aggregated results showed that low self-

report WM was associated with more crashes and traffic citations, whereas low performance-based WM was related to self-report inattentive driving and poor lane maintenance. Low WM indicates poorer ability to update online information as well as to develop situational awareness and is more likely to be saturated whenever the environmental demands increase, with detrimental effects on driving performance. Concerning inhibitory control, a lack of inhibitory capacity has been mainly related to unsafe and risky driving, speeding over the limit, and poor lane maintenance in simulated driving. It has been suggested that the relationship between low inhibitory control and reduced driving performance might be associated to the inability to inhibit irrelevant information draining attentional resources. Overall, the findings reported by this review are consistent with the idea that WM and inhibitory control, depending on the frontal lobes, are fundamental functions required to maintain a good driving performance, especially in complex and highly demanding environments, such as those characterising real-world roads and circuits.

The importance of EFs for driving performance is supported also by evidence coming from studies on neuropsychological patients. A study from Cizman Staba and colleagues (2022) assessed the correlations between different measures of cognitive function and the performance at a professional driving simulator in a sample of survivors of traumatic brain injuries, ischaemic strokes, multiple sclerosis, and brain tumours. They administered the Test of Attentional Performance (TAP; Zimmermann and Fimm, 2002), including subtests of alertness, distractibility, selective and divided attention. In particular, alertness was assessed using an SRT task (Donders, 1969), distractibility, defined as the ability to maintain a central attentive focusing ignoring the distractors, was investigated using the vigilance subtest of the TAP, selective attention was investigated by mean of a Go/NoGo paradigm (Donders, 1969), and divided attention, focused on planning and problem-solving abilities, by administering the Tower of London test (TOL; Culbertson and Zillmer, 2005). Then, after a 5-minute adaptation period, participants were required to drive for 30 minutes in a professional simulator, in three different scenarios (rural area, freeway, city), each of which had pre-programmed traffic-related challenges. The authors selected multiple indices of driving performance (Tab. 7).

Driving performance outputs	Performance parameters recorded
Average reaction time [ms]	Reaction time is defined as the time from a predefined trigger in the simulation (ex. 10 m before a stop sign) until the driver presses the brake pedal with at least 75 N. As valid reaction times were taken into consideration only values between 0.5 seconds and 4 seconds; reaction times shorter than 0.5 seconds were considered as a "cheat", whereas over 4 seconds as a "miss".
Total driving time [ms]	The total duration of the drive, recorded in milliseconds.
Average jerk [km/h ³]	The average value of jerk throughout the whole driver, where jerk is defined as km/h ³ (sudden accelerations and breaking).
Percentage of time that jerk was excessive [%]	The average amount of time the jerk value was higher than 1.07 km/h ³ (when accelerating) and below -1.47 km/h ³ (when breaking).
Percentage of driving time at unadjusted speed in fog [%]	The percentage of time the drivers spent driving over the speed limit while driving in the foggy part of the simulation. The speed limit at this part was 70 km/h and was indicated with a speed limit sign.
Percentage of time the irregular use of rearview mirror [%]	The percentage of time the driver did not regularly check the rearview mirror, where checking the rearview regularly was defined as checking the rearview mirror at least once every 10 seconds.
Percentage of travel time with too short safety distance [%]	The percentage of time the drivers spent driving with too short safety distance, where the appropriate headway distance was calculated based on the 1.8 second-rule (in some countries also used 2-second rule).
Off-road driving [%]	The percentage of time the driver spent driving off-road, where off-road is defined as the vehicle being off the road lane with at least 50% of the vehicle.
Stop sign violation	The number of times the driver did not stop the vehicle in front of a Stop sign.

Tab. 7 – Cizman Staba et al., 2022. Driving performance outputs assessed in the study.

Overall, subjects with higher levels of alertness, executive functioning, selective and divided attention, and lower distractibility also showed shorter RTs in the driving simulator, being more ready to react to stimuli occurring in the surroundings. In particular, inhibitory control, as indexed by distractibility, was the variable showing the strongest association with the quality of driving, being positively correlated with less off-road driving in the rural scenario and more time regularly using the rearview mirror in the highway scenario, and negatively correlated with jerky ride and speeding over the limit in the highway and urban scenario. In conclusion, the findings reported by Staba's group (2020) supported the critical role played by selective attention, executive planning skills, processing speed and inhibitory control in underlying efficient driving. Moreover, they showed in a neurologic population how individual with better cognitive and executive functions may be more able to deliver a good and safe driving performance, being better at acting within complex and highly demanding environments.

Another study focused on the role played by WM, sustained attention, and behavioural inhibition in explaining negative driving outcomes, including aberrant driving behaviour, driving errors, driving violations and crashes (Tabibi et al., 2015). The authors administered the Wechsler Digit Span Backward task (to assess WM), Continuous Performance Task (CPT; a measure of sustained attention), Go/No-go task (to measure inhibitory control), and the Driving Behaviour Questionnaire (DBQ; assessing aggressive violations, ordinary violations, errors, and lapses). The authors reported a positive correlation between inhibitory control, as indexed by the performance at the Go/NoGo task, and aberrant driving behaviour, self-reported driving errors, violations and the

number of individual crashes. Furthermore, after controlling for age and gender, results showed that, among cognitive functions, inhibitory control was the best and only significant predictor for aberrant driving behaviours when performing a linear regression. Finally, while driving errors correlated with all the executive domains assessed, only inhibitory control was a significant predictor when computing a linear regression, supporting the hypothesis that driving behaviours are mainly explained by inhibitory control.

Support to the involvement of EFs in the processes underlying effective driving has been provided also by the study of Gaudet and colleagues (2013). This study included a group of patients with a diagnosis of cardiovascular disease (CVD) who recently suffered a cardiac event and a healthy control group. Participants' driving was evaluated at a driving simulator using the Manitoba Road Test Form, assessing infractions in 5 areas: (a) starting/stopping/backing, (b) signal violations/right of way/inattention, (c) moving on roadway, (d) passing/speed, and (e) turning. Cognitive functions were assessed using the Attention Network Test (ANT), providing a measure of alerting, orientating and general EFs (Fan et al., 2002). Results showed that driving performance was significantly worse in the CVD group and that, after adjusting the model for age, sex, education, diabetes and hypertension, the CVD group displayed significantly worse EFs compared to the control group. Also, a significant positive correlation between driving performance and EFs emerged. Once again, these findings clearly linked executive functioning to driving performance, across healthy and clinical populations.

Although the previous studies involved different samples of non-professional drivers, the importance of cognitive functions for driving has been studied also on the field, on professional racing drivers. For instance, Turner and Richards (2015) assessed changes in selective attention demand throughout a World Rally Championship motorsport event. In this study, 5 drivers and 5 codrivers were administered a performance test (including visual and auditory selective attention task) consisting of a map-search task which required to find 80 target symbols distributed on a coloured map in 120 seconds maximum. Also, they were administered an elevator counting with distraction task, which required to count target tones while ignoring the distractors. Equivalent versions of the test were administered at the beginning and at the end of the reconnaissance day, and at the beginning, in the middle and at the end of the actual race day. Participants showed the

lowest selective attention levels at pre-reconnaissance and pre-rally, and the highest performance at mid- and post-rally. Overall, these results (Fig. 27) support the additional recruitment of cognitive resources during racing driving events, due to their significant attentional demand. The authors noted that physical exercise, hydration levels and circadian rhythms might have influenced the attentional performance.

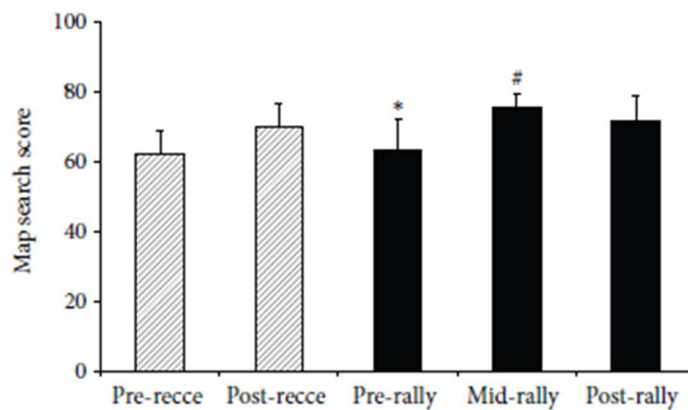


Fig. 27 – Turner and Richards, 2015. Scores (mean and SD) of drivers and codrivers at the map search test (selective attention) at the different time-points during the racing event. *Significantly lower than post-reconnaissance ($P = 0.007$), mid- ($P = 0.002$) and post-rally ($P = 0.01$); # Significantly higher than pre- ($P = 0.002$) and post-reconnaissance ($P = 0.01$).

Contrarily to the findings discussed here above, some other studies failed to find significant relationships between attentional/executive functioning and driving performance, underlining once again the need for further research investigating such a relationship. For instance, Liebherr and colleagues (2019) assessed the link between driving performance, different measures of attention and EFs. Driving performance was assessed using a static driving simulator close to production vehicles of the compact class, while the single participant was allowed to drive freely in a realistic scenario interacting with other fictional vehicles on the map. Driving performance was indexed by the so-called Index Of Performance (IOP), a composite index taking into account multiple parameters, including steer behaviour, activity on the pedals, and lane drifts. Moreover, visual selective, divided and switching attention (Switching Attentional Demands Task), auditory selective attention (Oddball-task), set-shifting (Modified Card Sorting Test), task-switching (TMT-B), vigilance (D2 test), inhibition (Go/NoGo), and WM (visual digit-span task) were assessed. Surprisingly, the results displayed no significant correlation between the IOP and any of the specific attentional domains, inhibition, and WM.

Nevertheless, the available evidence reported in the literature is consistent in supporting the existence of an association between higher executive functions and better driving performance, although no agreement has been reached yet, concerning the exact role

played by each single function. This can be due to the fact that different research groups included in their studies different populations (*e.g.*, non-professional and professional drivers, healthy and neuropsychologic subjects), using different approaches (*e.g.*, laboratory or field studies), and different psychological tests, to assess the relationship between cognition and driving. Therefore, future research on the topic will have to embrace a more systematic approach, better defining the constructs investigated, and being more consistent in selecting the tools and populations included, allowing to reach a broader consensus regarding the role played by cognitive functions in driving and to build a more coherent conceptual model of the abilities underlying the driving performance.

3.2 Driving performance, physiological functioning and autonomic cardiac control

Car racing is associated with a unique set of physiologic challenges (Reid and Lightfoot, 2019). In particular, the magnitude of physiologic stressors in racing may vary depending on the racing series, the type of car used, the duration of the event and the specific technical regulations. Nevertheless, some common stressors have been identified across the series and include environmental and metabolic heat, elevated g-force (up to 5-g), vibrations, carbon monoxide exposure, elevated and prolonged noise, and notable cardiovascular and mental stress (Fig. 28; Watkins, 2006; Reid and Lightfoot, 2019). The exposure to these highly challenging conditions requires a fine and constant physiological regulation, including cardiac autonomic control, in order to maintain the homeostasis and support an effective performance (Reid and Lightfoot, 2019; Yamakoshi et al., 2010).

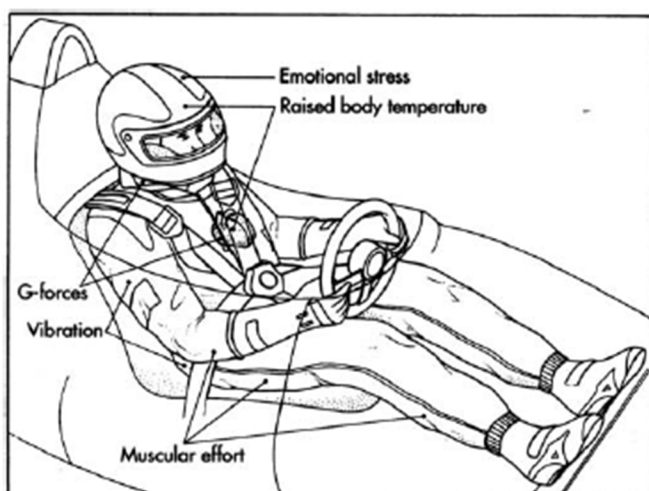


Fig. 28 – Watkins, 2006. Examples of emotional and physical stressors experienced by a Formula One driver (from Bertrand C, Keromes A, Lemeunier BF, Meistelmann C, Prieur C, Richalet JP. *Physiologie des Sports Mecaniques*. 1st International Congress of Sport Automobile, Marseilles, 1983).

Over the decades, multiple studies investigated the changes in cardiovascular regulation during racing. The first ECG recordings from racing drivers during a competition were published by Taggart, Gibbons, and Somerville in 1967 (Fig. 29). The authors reported increases in HR before the start of the competition (150-180 bpm) and an ulterior increase right before the start (200-205 bpm), maintained during the actual race. These results highlight the level of cardiovascular stress the drivers are exposed to during racing and underline the importance of well-functioning cardiovascular regulatory mechanisms. Other studies replicated these findings. Yanagida and colleagues (2016) reported significantly higher average HR during racing as compared to resting values and a positive correlation between average driving speed and HR. This correlation has been reported also by other papers (Jacobs et al., 2002; Watkins, 2006). Interestingly, a positive correlation has been reported only between HR and average speed, but not between the former and instantaneous speed, since HR tends to increase during corners, due to enhanced muscular/neural activity and g-forces, where speed tends to decrease (Jacobs et al., 2002). This is clearly visible in the data published by Watkins (2006) and collected on Ferrari F1 driver Didier Pironi (Fig. 30).

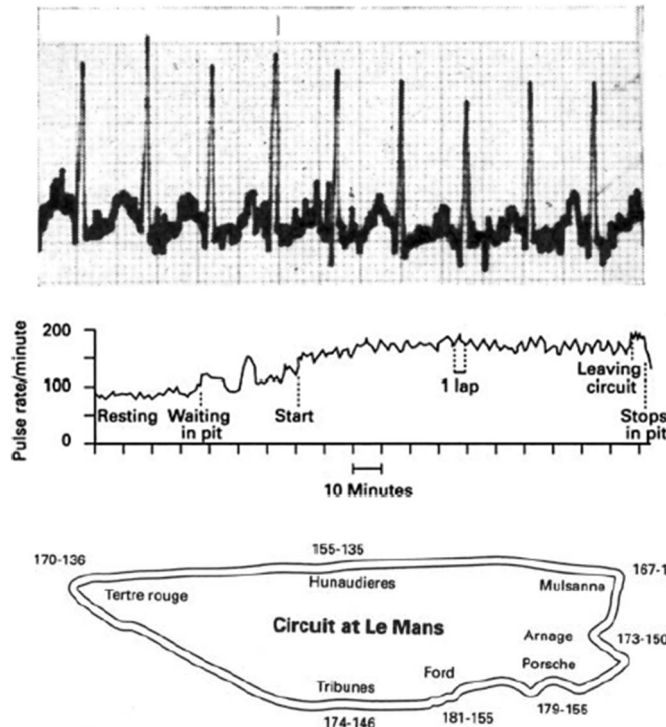


Fig. 29 – Taggart, Gibbons and Somerville, 1967; Reid and Lightfoot, 2019. The data illustrate heart rate elevation during racing. Upper panel: The first physiologic data obtained from a driver athlete during competition; tracing of electrocardiogram (ECG) was obtained 1 min after start of the race; driver is approaching a sharp bend at approximately 115 mph (185 kph). Lower panel: sustained heart rate elevation during a 2-h stint; labels depict driver activities at various times.

Fig. 30 – Watkins, 2006. Variations in Pironi's HR, recorded over eight laps. Notably, HR is at its lowest on the Mulsanne Straight, despite the speed being at its highest.

Furthermore, available evidence suggests that significant individual differences in racing-related cardiac adaptation exist (Watkins, 2006; Bedini et al., 1995). This is clearly visible in the data recorded on Ferrari F1 drivers Gilles Villeneuve and Didier Pironi during a practice session at the Fiorano racetrack (Fig. 31). These data replicated the correlation between average lap speed and mean HR, and also showed consistent differences in HR among the two subjects attributable, according to the author, to differences in the personality-dependent ability to cope with mental stress, and physical fitness. The role played by individual aerobic fitness in modulating race-related changes in HR was highlighted by Schwaberg and colleagues (1987), since they reported that elite Formula 1 drivers who are more aerobically fit (as indexed by the VO₂ max) show smaller racing-related increases in average HR. This might be explained by the fact that aerobically fitter subjects have more resources to invest in cardiac regulation and in the maintenance of homeostasis, while delivering a peak-performance. Finally, along with physical stressors, mental ones also play a role in inducing a rise in HR, since such a rise appears before the race start, when physical stressors are not present yet (Taggart, Gibbons, and Somerville, 1969; Watkins, 2006; Baroody, Thomason and O'Brian, 1973), and it partially decreases shortly after the start along with nervous tension levels (Bedini et al., 1995). Altogether, the findings discussed until here are consistent in providing support to the presence of high levels of physiological stress during car racing, challenging drivers' physiology and requiring effective and efficient autonomic and somatic regulatory mechanisms, in order to maintain homeostasis and support the delivery of the best peak-performance.

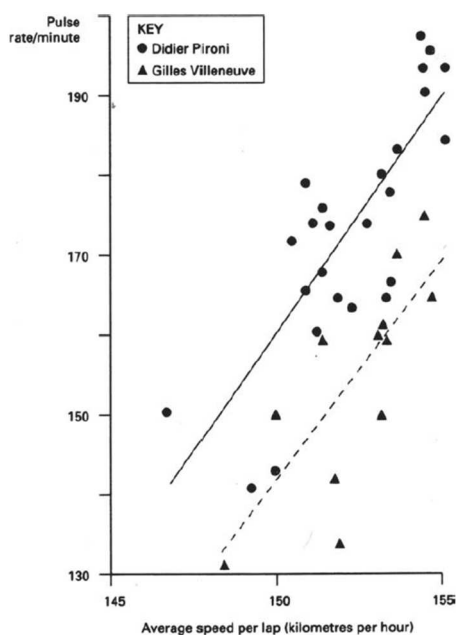


Fig. 31 – Watkins, 2006. Data collected during Formula 1 practice sessions at Fiorano. Each point represents a lap. Clear differences can be seen in Villeneuve's and Pironi's HR, with the former barely reaching 175 bpm and the latter exceeding 200 bpm.

Within the last 3 decades, some studies not only replicated the findings discussed above, but also tried to assess HRV online during racing activities, by appositely modifying ECG recorders. Bedini and colleagues (1995) recorded ECG signals as well as latero-lateral and antero-posterior accelerations, instantaneous velocity, steer angle and lap times in the 2 Formula 1 drivers racing for the F1 Minardi team during 6 races of the 1995 F1 World Championship. The aggregated data showed a significant initial increase in HR followed by a small but significant decrease about 10 minutes after the session start. An exploratory analysis of RR variability during driving displayed a reduction in HRV indexes (*i.e.*, SDNN and PN50) compared to baseline levels, suggesting a reduction in total HRV explained by a reduction in parasympathetic activity. These findings can be interpreted as a decrease in vagally-mediated cardiac autonomic control during racing driving. Another study published by Yamakoshi and colleagues in 2010 investigated the influence of g-force on cardiovascular activity in amateur kart drivers. They recorded ECG signals to measure instantaneous HR and RR intervals while participants were asked to drive as to record their best lap. Moreover, lap times as well as blood pressure and eardrum temperature just before and straight after the driving session were recorded. In line with previous studies, an increase in HR at the start of the driving period, followed by a partial decrease and stabilisation around 150 bpm was reported. Moreover, a significant correlation between HR, g-force and lap time was found. HRV was significantly reduced throughout the driving session as compared to baseline, consistently with the findings reported by Bedini et al. (1995). In particular, changes in HRV were related to a significant increase in the LF-to-HF ratio, interpreted by the authors as the consequence of the suppression of vagally-mediated parasympathetic cardiac control, coupled with an increase in sympathetic activity. A significant increase in eardrum temperature and a decrease in systolic and diastolic blood pressure was recorded over the 5 minutes following the end of the driving session, suggesting a reduction of the influence of the sympathetic ANS branch over the vasomotor system from before to after the driving session. Overall, the data underline the presence of a shift in the sympathovagal balance leading to an increase in HR and a reduction in vagally-mediated HRV during the race, but the authors suggested, based on previous studies (Levy, Cerqueira, Abrass, Schwartz and Stratton, 1993), that physically fitter individuals might have a broader cardiac reserve, allowing them to maintain a better cardiac performance, preventing vagal suppression to

occur.. Based on the evidence reported here and the claim made by the authors, it may seem reasonable to hypothesise that individuals with higher resting HRV may have additional resources to better adapt their physiology and to cope more effectively with stressors occurring during racing driving, being better able to maintain an appropriate homeostasis while delivering a high-level peak-performance. Nevertheless, research is needed in order to collect evidence to sustain the hypothesis of a relationship between vagally-mediated baseline HRV and actual racing driving performance.

3.3 Cardiovascular functioning as an index of cognitive and physiologic demands of driving

Physiological signals have been used in research as indicators of ANS activity, which is usually triggered by perceived stressors. This has been done also in the attempt to identify the cognitive and physical stressors occurring during simulated and real-life driving activities, as well as their effects on the driver’s resources and cognitive workload. Different methods and techniques have been suggested for this purpose (Tab. 8; Reid and Lightfoot, 2019).

Approach	Modeling the Race Driver Experience									
	Least ← Physiological Fidelity → Greatest									
	Video Systems			Racing Simulators			Instrumented Cars			
Visualize	View Only	Imagine Driving	Input Controls	Passive Platform	Active Platform	Practice Session	Race Session			
Elements of the driver experience	Imagination, memory	Imagination, memory, visual input, audio input	Imagination, memory, visual/audio input, cortical function	Imagination, memory, visual/audio, complex cortical tasks, muscle use	Imagination, memory, visual/audio input, complex cortical task, limb muscle use, afferent feedback from limbs	Imagination, memory, visual/audio, more-complex cortical tasks, limb and trunk muscle use, whole-body afferent feedback, inner ear/balance stimuli	Imagination, memory, visual/audio, more-complex cortical tasks, limb and trunk muscle use, whole-body afferent feedback, inner ear/balance stimuli, full g loading, modest risk	Imagination, memory, visual/audio, more-complex cortical tasks, limb and trunk muscle use, whole-body afferent feedback, inner ear and balance stimuli, full g loading, substantial risk, stress of competition, anticipating and interpreting other drivers		
No. reports	0	0	1	1	3	0	11	21		
References			95	68	28,46,86		34,35,52,56,57,60,67,80,81,82,90	2,4,6,8,10,30,36,57,59,61,66,67,69,70,71,77,78,79,80,82,87		

Tab. 8 – Reid and Lightfoot, 2019. Different methods and techniques for modelling the race driver experience.

For example, Lee and colleagues (2007) investigated the effects of driving in stressful conditions on different time-domain HRV parameters, by using both simulated and real-life driving tasks. They asked a participant to keep the assigned lane while using a driving simulator and they recorded the ECG signal at rest and during the task. The level of stress was manipulated by progressively increasing the vehicle’s speed. In a second study, 3 participants were asked to drive on real roads with an actual car. Here, the stress level was manipulated by changing the driving scenario, from large straight roads to busy narrow ones. Cardiac activity was obtained through two indirect contact ECG electrodes on the seat and a reflective PPG on the steering wheel cover. As compared to baseline, during

the simulated driving test HR significantly increased, whereas HRV indexes (SDNN, RMSSD, and pNN50) significantly decreased. Intriguingly, increased levels of stress (indexed by driving speed) induced a slight decrease in HR and a small increase in HRV parameters. This was interpreted as due to the moderate difficulty of the task, which was not perceived as stressful, but rather as challenging, and allowed for adaptation to occur. During the real-road driving task a significant increase in HR coupled with a significant decrease in HRV pNN50, RMSSD, and SDNN indexes occurred, as compared to baseline. Physiological modifications in this second study suggest that the manipulation of stress levels was effective and that participants felt stressed. Moreover, as expected, HR was on average higher when the stress level increased, whereas HRV values tended to decrease. These findings showed that a shift in sympathovagal cardiac control occurs when cognitive demand increases due to driving in stressful conditions, leading to a positive chronotropic effect and a reduction in HRV parameters.

A study published by Wen's research group (2017) tried to provide a continuous estimation of stress using physiological signals during a car race. A professional racing driver was tested during a practice race session, and psychophysiological signals included HRV, galvanic skin response (GSR) and muscle activity of the masseter (MAM), all of which have been reported to be associated with stress (Hidaka et al., 2004; Thayer et al., 2012). The authors monitored instantaneous g-force, as an index of acceleration/deceleration (reflecting physiologic stress) and the presence of a competitor's car in the visual field of the driver, indicating the presence of an internal state of competition (*i.e.*, mental stress). The factor analysis conducted by the authors showed (Fig. 32) that: (1) HRV and GSR overlaid with the factor competition, and were therefore mainly induced by a mental stressor, and (2) MAM was consistently overlaid with events of acceleration/deceleration, being mainly related to a physiologic stressor. Overall, these findings displayed once again that driving demands have an effect on the ANS and on cardiac autonomic control, as indexed by HRV parameters. Finally, they support the idea that, although all the three physiological signals are somehow affected by stress, each of them is influenced to a different extent by physiologic and mental stressors occurring during peak-driving performance, according to which the authors suggested a stronger internal influence on HRV and GSR, and stronger external one on MAM.

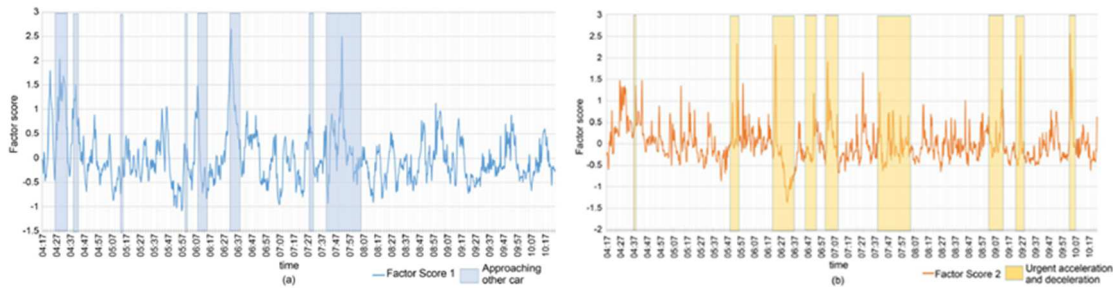


Fig. 32 – Wen et al. 2017. The scores of factor 1 (blue line, left) and factor 2 (orange line, right) plotted in function of time, and their overlay respectively with the events of competition (“approaching other car”) and acceleration/deceleration (“Urgent acceleration and deceleration”).

Backs and colleagues (2003) assessed the feasibility of using cardiac recordings as a measure of driver workload occurring during driving. Participants were required to drive in a simulator at a fixed speed (72.4 km/h) on a course composed of alternated straightaways and curves with a radius respectively of 582 m, 291 m, and 194 m, while the amount of visual workload was being manipulated, by altering the amount of visual information available. The authors measured RSA (an HRV index, reflecting parasympathetic influence on the heart) and pre-ejection period (PEP; pure index negatively correlated to sympathetic influence on the heart) to assess the autonomic response to driving in different visual demands conditions (Backs, 1995; Backs, 2001; Berntson et al., 1993). Driving performance was assessed in terms of standard deviation of lateral position in meters, standard deviation of the steering wheel angle and the frequency of lane excursions, measured during bends. Results showed that all the three measures of driving performance were affected by curve radius and the amount of workload induced by visual occlusion. Regarding physiological responses, none of the physiological measures was affected by visual occlusion, whereas HR and PEP increased, while RSA decreased at increasing radius of the curves (Fig. 33). Therefore, cardiac measures reflected the level of demand elicited by driving, marked by a decrease in parasympathetic activity on heart.

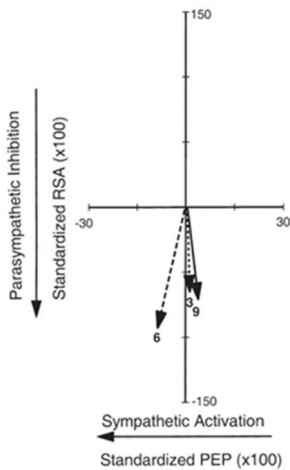


Fig. 33 – Backs et al., 2003. Representation of the changes in parasympathetic (standardized RSA) and sympathetic activity (standardized PEP) occurring as an effect of different curve radius. Faster HR corresponds to negative regions of the axes.

Numbers represent curve radius.: 3 = 582 m radius curve; 6 = 291 m radius curve; 9 = 194 m radius curve.

Lenneman and Backs (2009), based on previous research showing that HRV is sensitive to changes in attentional demand occurring during driving (Brookhuis et al., 1991; Lenneman et al., 2005), hypothesised that adding a WM side task to driving would elicit changes in the pattern of autonomic activity, allowing to map the specific attentional resources involved in such activities. They also hypothesised that cardiac measures would be more sensitive in detecting changes in attentional demand occurring by changing the side tasks, as compared to lane-keeping measures of driving performance. Therefore, they investigated the effects of manipulating the attentional demand required by simulated driving and concurrent verbal WM tasks on HRV. In particular, the participants underwent four different experimental conditions:

- (a) a driving-only task, where they had to drive along a straight road with varying crosswinds keeping the assigned lane;
- (b) a n-back task with different levels of difficulty (0- and a 3-back);
- (c) a driving-condition plus a n-back concomitant task with a low level of difficulty (0-back);
- (d) a driving-condition plus a n-back concomitant task with a high level of difficulty (3-back).

Results showed that in the driving-only condition, a significant decrease in RSA (indexing PNS activity) and no change in HR and PEP (indexing SNS activity) occurred, suggesting an uncoupled parasympathetic withdrawal mode of cardiac control. In the n-back only condition, a significant increase in HR and a significant reduction in RSA and PEP were

found, supporting the occurrence of a cardiac autonomic control mode based on coupled sympathetic activation and parasympathetic withdrawal. Moreover, HR and RTs were significantly higher while RSA and accuracy were significantly lower in the 3-back as compared to the 0-back condition, showing that, with the increase in attentional demand, the cognitive performance decreased, coupled with a reduced parasympathetic predominance on cardiac autonomic control. Concerning the dual-task conditions, in both the 0-back and 3-back concomitant task condition, driving performance was not significantly different from the one recorded in the driving-only condition, possibly due to the fact that the driving task was relatively easy to perform and relied on cognitive functions that did not overlap with those required by the n-back. From a physiological standpoint, HR increased for both the 0-back and 3-back dual task session as compared to the driving-only one, whereas PEP and RSA significantly decreased only in 3-back dual-task, but not for the dual 0-back condition. Taken together, these results display that changes in the autonomic space (see Berntson, Cacioppo and Quigley, 1993) occurred when moving from a single driving-only condition to a high-difficulty dual-task (Fig. 34), suggesting that they are able to detect increases in attentional resources demands. Overall, the findings discussed here show that autonomic cardiac control is influenced, not only by complex executive tasks, but even by very simple driving tasks, such as lane keeping, to which it responds by changing the sympathovagal balance in order to maintain homeostasis and support an effective performance. Second, the data reported here suggest that measures of cardiac autonomic control are more sensitive than simple measurements of driving performance (*i.e.*, lane keeping) to the attentional demands of driving, both when performed alone and combined with other simultaneous cognitive tasks. On the one hand, cardiac data clearly manifest the progressive increase in effort by the participant from a resting condition to driving, and then to perform driving-executive dual tasks, by showing an increase in HR and a change in the pattern of autonomic activity within the autonomic space. On the other hand, the same data suggest that participants had enough residual processing resources to prevent driving performance decrements. Further research is needed to test whether individuals with higher levels of vagally-mediated cardiac control at rest also have more physiological and cognitive resources sufficient to

increase the effort and keep a good driving performance with heightened attentional demands and cognitive stress.

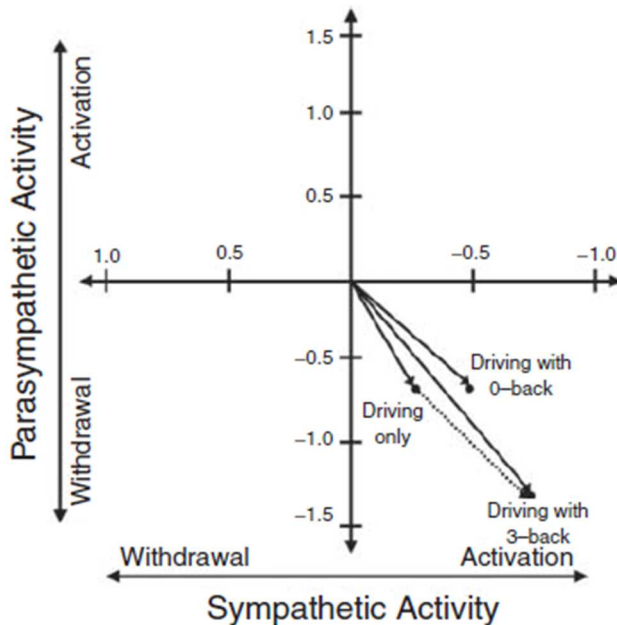


Fig. 34 – Lenneman and Backs, 2009. Changes in standardised PEP (x-axis) and standardised RSA (y-axis) from resting baseline (the origin) to the different experimental conditions.

Overall, the evidence discussed in this section is consistent in supporting the presence of physiological changes in response to the mental and physical demands of driving. In particular, the autonomic space framework revealed to be a useful tool to assess these changes from baseline by also disentangling the effects of driving on PNS and SNS, as indexed by different physiological parameters, and to be an effective approach to identify the different processes underlying these changes. Further research is needed in order to assess whether individual differences in baseline cardiac autonomic control mechanisms can be predictive of the individual ability to adapt to high levels of driving demands, in order to support a higher peak-performance in highly competitive and challenging environments, such as the ones characterising professional racing.

3.4 Evidence coming from other sports

Sport psychology is defined as the field of science involving the application of theories, principles, and techniques from mainstream psychology to foster psycho-behavioural change in athletic populations to enhance performance, the quality of the sport experience, and the personal growth of the athlete (Vealey, 1994). Exercise psychology is

instead conceptualised as ‘the application of psychology to antecedents and consequences of health-related physical activity’ (Biddle and Fuchs, 2009). Within different fields belonging to sport and exercise psychology (SEP), techniques assessing HRV have been widely used and can provide useful information regarding the relationship between vagally-mediated cardiac autonomic control mechanisms and psychological and physiological demands of different sport activities (Mosley and Laborde, 2022). In particular, along with other topics, HRV assessment in SEP has focused on the relationship between HRV and executive functions in athletes (Mosley et al., 2018), the link between physical fitness and cognitive processing (Dupuy et al., 2018), and the effects of HRV-manipulations via biofeedback on performance (Jimenez Morgan and Molina Mora, 2017; Pagaduan et al., 2021; Mosley and Laborde, 2022).

Overall, some interesting data have been published, highlighting the potential of these techniques within SEP. Indeed, the available evidence coming from this field suggests the presence of a causal link between vagal predominance in cardiac autonomic control at rest, heightened executive functioning, and a better performance in a wide variety of sports and athletic populations, including biking, running, swimming and judo (Dupuy et al, 2018; Albinet et al., 2010; Albinet et al., 2012; Blasco-Lafarga, Martinez-Navarro, and Mateo-March, 2013). Overall, this is in accordance with the hypotheses of the Neurovisceral Integration Perspective (Thayer and Lane, 2000; Thayer et al., 2000; 2012), also supporting the effectiveness of HRV biofeedback techniques to enhance sport performance (Jimenez Morgan and Molina Mora, 2017; Levy and Baldwin, 2019). Nevertheless, research in the field still lacks a wide agreement concerning theoretical models, and a systematic approach to the definition of methods and interpretation of the collected data. For these reasons, some inconsistent findings have been reported in literature (*e.g.*, Mosley, Laborde and Kavanagh, 2018), and further, more systematic research is needed, to further improve our knowledge of the mechanisms linking cardiac autonomic control, the different EFs and sport performance. Finally, the results discussed in this paragraph need to be replicated in other sports and populations, including professional car racing drivers.

4 Drive at the rhythm of your own heart: a study on Heart Rate Variability, cognitive functioning and driving performance in Ferrari Driver Academy drivers.

4.1 Introduction

Racing driving is a very demanding activity, where the drivers are required to develop extraordinary sensorimotor skills to control powerful cars at high speed while surrounded by competitive opponents, and to deliver high-level peak performances in complex, quickly changing environments. Evidence has been reported supporting a correlation between some indexes of the autonomic nervous system's (ANS) ability to maintain homeostasis (*e.g.*, vagally mediated parameters of cardiac autonomic control) and some measures of frontal executive functions, including inhibitory control and working memory (Cortese et al., 2022; Hansen, Johnsen and Thayer, 2004; 2009; Thayer et al., 2009). Previous research, conducted in different fields and on different populations, used cardiac autonomic control measures including heart rate (HR), heart rate variability (HRV) and their changes both at rest and under stressful conditions, to assess the role played by the homeostasis-maintaining action of the ANS in supporting an effective driving performance. Multiple studies highlighted the role played by cognitive and, especially, executive functions (EFs), including inhibitory control, working memory (WM) and cognitive flexibility, in underlying an adequate driving performance (Tabibi et al., 2015; Walshe et al., 2017; Cizman Staba et al., 2022), and provided a broad set of cognitive tasks and tests useful to assess these functions.

Nonetheless, the body of studies published on the topic is not only limited in size, but also characterised by several important limitations (*e.g.*, very small and variegated samples) and a lack of a systematic definition of the theoretical models and paradigms embraced, as well as of the methods and tools used to measure physiological and cognitive functioning and the driving outcomes, leading to controversial findings, that are not always easy to interpret. For this reason, we adopted the Neurovisceral Integration Perspective's framework to conduct a study aimed at enhancing the understanding of the physiological and cognitive mechanisms underlying high-level peak driving performance, on a well-defined sample of elite racing drivers, mainly belonging to the Ferrari Driver Academy (Maranello, MO, Italy). First, this research project aimed to

investigate the relationship between vagally-mediated time-domain parameters of HRV and a set of measures of cognitive functions, including non-executive (simple reaction times; SRTs) and executive (inhibitory control and WM) ones. Second, this study tried to elucidate how these physiological and cognitive variables can be used to predict driving performance in elite racing drivers.

We hypothesised that RMSSD and Coherence time-domain measures of HRV (indexing parasympathetic influence on cardiac control) would be positively associated with measures of driving performance, inhibitory control (*i.e.*, the performance at a Go/NoGo task; Donders, 1969), and WM (*i.e.*, the performance at an N-Back task; Kirchner, 1958; Welford, 1958), but not with those of non-executive functioning and, in particular, of general readiness (*i.e.*, the performance at a SRT task; Donders, 1969). Furthermore, we hypothesised that individual peak driving performance would be predicted by HRV and by measures of inhibitory control.

4.2 Methods

4.2.1 Participants

One hundred and two subjects (age range: 12-26; mean = 16.3, SD = 2.88 years) were initially recruited from 2014 to 2023. All drivers were evaluated by trained psychologist working for Inside S.r.L. The majority of them were referred directly by Ferrari Driver Academy (FDA). Overall, participants came from a heterogeneous range of motorsport categories, championships and series, including international karting competitions (OK, OKJ; see the technical regulations defined by the Fédération Internationale de l'Automobile; FIA), Formula 4, Formula Regional, Formula 3, Formula 2 and e-sports. All participants were in good general health, as certified by a medical certificate declaring the fitness to participate in the aforementioned championships. All the participants signed an informed consent. Due to the sex-related differences reported in literature and the small size of the female sample ($n = 21$, 21%), only male drivers were included in the final analyses. Specifically, we included the data collected on male drivers ($n = 81$), concerning physiological and cognitive tasks. It has to be noted that data were missing from the N-

Back task (included data from $n = 59$). Moreover, to measure their peak driving performance we included data from a subsample of participants ($n = 24$) which performed a driving task at a professional driving simulator.

4.2.2 Tools

Physiological recordings

For the ECG recordings, single-use electrodes were applied to the subject's chest and attached through a clip to the recording cables, connected to the amplifier/recording device (NeXus-10 by MindMedia; San Rafael, CA, USA). Respiratory activity was measured using a respiratory belt located on the bare skin of the participant, at the level of the umbilicus. The data collected were then analysed using the software BioTrace+ (MindMedia) and Kubios (Kuopio, Finland), in order to identify the R-R intervals and compute the HRV parameters. Specifically, for those recordings where artifacts influenced less than 10% of the data, BioTrace+ was used to delete the affected data and compute the psychophysiological indexes using the rest of them. For those recordings where artifacts affected more than 10% of the collected data, the data were exported on Kubios, where the artefacts were corrected, and the indexes computed.

In particular, the psychophysiological data were gathered at rest, in sitting position, over a period of 5 minutes, and were measured using ECG recordings, at constant room temperature and with dim light (Task Force, 1996). For the ECG recordings, single-use electrodes were placed on the participants following the second derivation Einthoven's triangle (Fig. 35). A proximal disposition placement was chosen in order to reduce movement artifacts and collect more accurate data. The signal was then amplified and filtered, to delete the influence of muscular activity on the recordings and to cut out the respiration-induced artifacts, using the default settings included in BioTrace+.

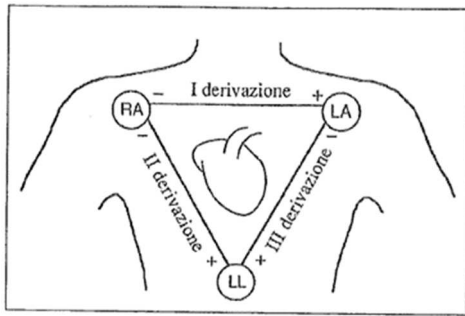


Fig. 35 – Pennisi and Sarlo, 1998 – The three possible placements of the electrodes used for the ECG recordings following the Einthoven triangle.

From the physiological recording at rest two HRV indexes were calculated: RMSSD and coherence (*i.e.*, a Pearson correlation coefficient indexing the synchronicity between respiratory activity and changes in HR triggered by vagal activity induced by respiration, or the extent to which respiration and HR signals fall and rise at the same time; Shaffer and Meehan, 2020). Both have been reported to be reliable indexes of parasympathetic influence on the heart. Moreover, it has been suggested that both are not influenced by respiratory confounding.

Cognitive assessment

For the cognitive assessment, computerised versions of classic paradigms, including a simple reaction time (SRT; Donders, 1969), a modified version of the classic Go/NoGo (Donders, 1969) and a visual N-Back (Kirchner, 1958; Welford, 1958) task were implemented using the software E-Prime (Pittsburgh, PA, USA). The three tasks were modified to include stimuli with ecological relevance for the population of interest – *i.e.*, starting lights – and required ecologically relevant responses to target stimuli – *i.e.*, to press a paddle on a steering wheel.

The SRT task consisted of one block lasting 240 s, during which about 80 stimuli were presented. Participants were required to respond as quick as possible to the target stimulus, by pressing a paddle on a steering wheel located in front of them. Each trial (Fig. 36) randomly lasted between 2500 and 3000 ms and was composed of a sequence of 5 screens. In particular, it included: (a) a blank screen (1000 ms); (b) a screen with a central starting light as a fixation point (random duration 500-1000 ms); (c) the presentation of the target stimulus (a yellow light) in the central section of the starting light (250 ms); (d) a blank screen with the offset of both the fixation point and the target stimulus (250 ms); (e) feedback on the trial performance (RT for valid trials; anticipation, when the RT was faster than 100 ms; omission, when no response was recorded).

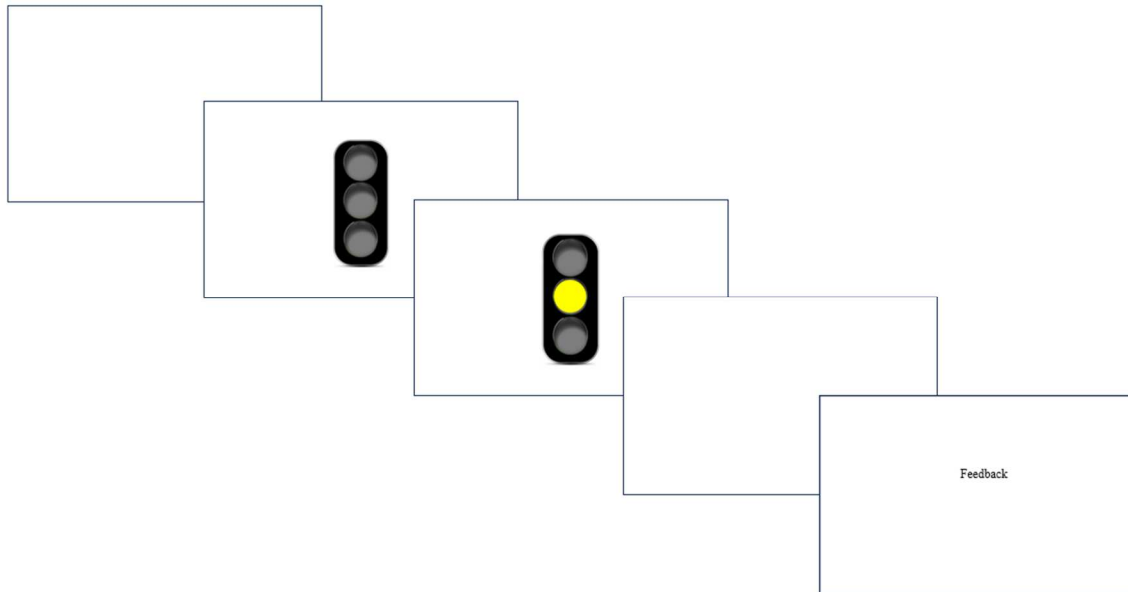


Fig. 36 – Screens used for the SRT task.

The Go/NoGo task consisted of a single block lasting 300 s, during which 72-90 stimuli were presented. The subjects were asked to respond to the target stimulus in the Go condition by pressing a paddle of the steering wheel positioned in front of them. Each trial (Fig. 37) had a random duration between 3250 ms and 4250 ms, and comprised a sequence of 5 screens, including: (a) a blank screen (1000 ms); (b) a screen with a starting light used as a fixation point (random duration 500-1500 ms); (c) the random presentation of the target stimulus in the central section of the starting light (green light = Go condition; red light = NoGo condition; probability of each condition = 50%); (d) a blank screen with the offset of the fixation point and the target stimulus (250 ms); (e) feedback regarding the performance at the trial (RT for valid trials; anticipation, when RT was faster than 100 ms; omission when no response was produced in the Go condition; commission when a response was recorded in the NoGo condition).

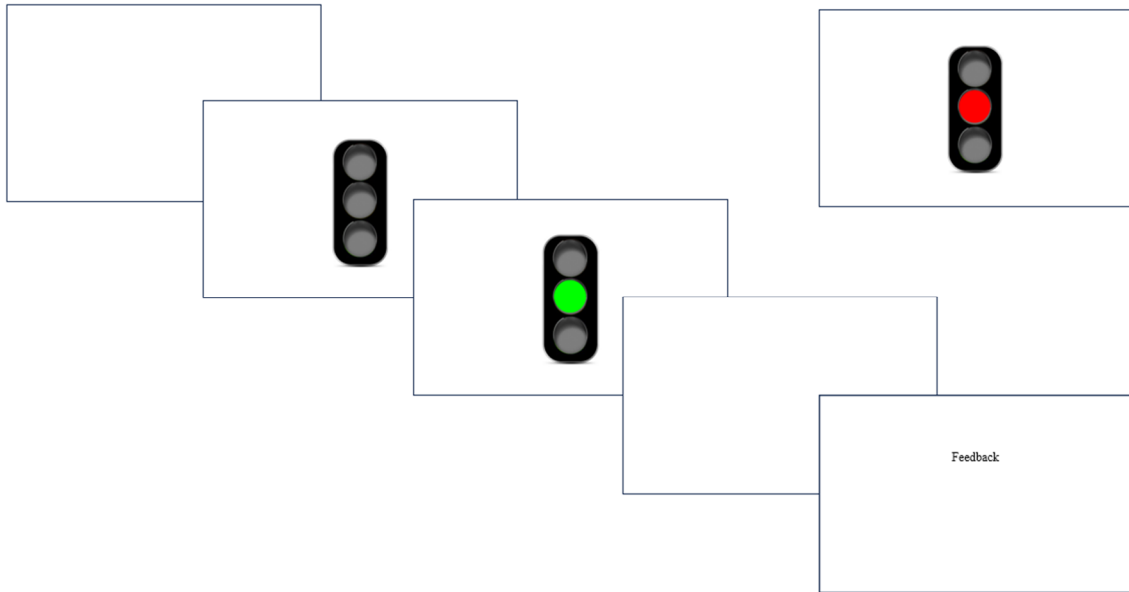


Fig. 37 – Screens and stimuli used for the Go/NoGo task. In the third screen the Go stimulus (*i.e.*, a green light). In the upper right panel, the NoGo stimulus (*i.e.*, a red light).

To test working memory, a single block of a N-Back continuous performance task (Fig. 38) was administered. The block had a duration of 240 s, and comprised 80 trials, each lasting 3 s. Two starting lights were displayed on the screen, one on the right and the other on the left of a virtual steering wheel. The target stimulus (a green light) was presented for 1 s in one of 6 sections of the two starting lights, while between the presentation of two consecutive stimuli a period of 2 s occurred, during which the lights were off. Participants were required to press the paddle of a steering wheel positioned in front of them, whenever the position of the target stimulus matched the one of the stimulus presented *n*-trials before (2 or 3, depending on the age of the subject). Finally, auditory feedback was given during the 2 s period when no target stimulus was visible, with a high-pitched sound indicating a correct response, and a low-pitched one signalling a wrong response to the task.



Fig. 38 – Stimuli used for the N-Back task. In red, the instructions provided to the participant for the 2-back condition, at the beginning of the task.

Software-wise, driving simulations were administered using Assetto Corsa, developed by KUNOS Simulazioni S.r.L. (Formello, RM, Italy), a world leading software in the field of driving simulation. Hardware-wise, the participants used one of the two simulators available: (a) a simulator with 2-axis of movement located in the headquarter of the FDA (Maranello, MO, Italy) produced by Evoteck Sym S.r.L., that will be, from now on, referred to as “Maranello” (Fig. 39 and 40 display its replica located in the headquarter of Inside S.r.L); (b) a fixed simulator produced by Allinsports (Formigine, MO, Italy) and located in the FDA headquarter, composed of a professional seat, a professional steering wheel, a professional pedal box and 3 computer screens, that will be from now on referred to as “Mini-sim”. All the drivers who took part in the driving simulations ($n = 24$) raced using the same set up, consisting of a Formula 4 car (Taatus FIA F.4) and the Ferrari-owned track of Fiorano (MO, Italy). In particular, they were required to take part into a practice session with no opponents on the track in optimal dry and sunny conditions, and to drive as fast as possible, while respecting the track limits and the usual rules used in real-world motorsport events. At the end of the task, driving performance was indexed by two outcome measures – *i.e.*, the best and the average lap times – computed after deleting the invalid laps (*i.e.*, those when the driver exceeded the track limits, cut a corner, or spun).



Fig. 39; Fig. 40 – Replica of the “Maranello” set up. The difference between this and the one used to collect the data consist in the screen. Here a 180-degree curved screen is present. The original set up included 3 flat screens.

4.2.3 Procedures

On the first day of assessment all the participants underwent in a random order psychophysiological and cognitive evaluation. The psychophysiological data included in the study were gathered at rest, in sitting position, over a period of 5 minutes, at constant room temperature and with dim light. Cognitive tasks were administered in a fixed order and interspersed with breaks lasting at least 3 minutes. The simple reaction time (SRT) task was first administered, followed by a modified version of the classic Go/NoGo task and, for a subsample of participants, also by the visual N-Back task. On the second day, a subsample of subjects took part into a single driving simulation session, lasting about 10 to 20 minutes.

4.2.4 Statistical analysis

All the statistical analyses were run using jamovi (version 11.3.0.0). First, exploratory analysis assessed the presence of correlations between the age of the participants, RMSSD, Coherence, measures of performance at the cognitive tasks SRT (Reaction Time [RT] and SD), Go/NoGo (RT, SD, Accuracy and Commissions), and N-Back (RT and Accuracy) tasks, and two indices of driving performance (Best Lap and Average Lap time).

Second, two linear regressions were run, using the variable “Sim” as a factor to control for the influence of the simulator used on the data concerning driving performance. Dependent variables were Best Lap time for the first linear regression model and Average Lap time for the second linear regression model. First, a Shapiro-Wilk test was run, to assess the normality of predictors’ distributions. Second, in order to obtain a normal distribution of the data, z scores of the variables that resulted non normally distributed were calculated (*i.e.*, Coherence and Commissions at the Go/NoGo task). Following our hypotheses and the results obtained by the exploratory analysis, in the first block Coherence and Commissions at the Go/NoGo were included as independent variables to predict the Best Lap times. In the second block, RT at the Go/NoGo was added as an independent variable. Finally, the two linear regressions were repeated, including Average Lap times as the dependent variable.

4.3 Results

4.3.1 Participants

The female sample was smaller and significantly younger ($p < 0.01$) than the male one (Tab. 9). Due to the small size of the female sample ($n = 21$, only 11 with data concerning driving performance), only males were included in the final analyses. 81 of them had valid data for the SRT and the Go/NoGo task, whereas 59 also had valid data for the N-Back task. Finally, data from 24 subjects were included in the analyses to assess driving performance.

Tab. 9 – Sociodemographic variables		
	Female (n = 21) M (SD)	Male (n = 81) M (SD)
Age, years	14.4 (1.83)	16.7 (2.91)

4.3.2 Exploratory analyses

The exploratory analyses (results shown by Tab. 10) assessed the presence of correlations between age, RMSSD and Coherence (vagal indices of HRV), the performance at the SRT (RT and SD), the Go/NoGo (RT, SD, Accuracy and Commissions), and the N-Back (RT and Accuracy) tasks and the driving performance (Best Lap and Average Lap time). First, age did not correlate with HRV parameters or indexes of performance at the Go/NoGo and N-Back tasks, nor with measures of driving performance. A significant negative correlation emerged between age and RT at the SRT ($r = -0.263$; $p = 0.018$) as well as among age and the SD of RTs at the SRT ($r = -0.232$; $p = 0.037$). Older drivers were faster and more consistent in responding to a simple reaction time task.

Furthermore, the results (Fig. 41) displayed a significant correlation between a vagally-mediated HRV parameter and the percentage of commissions at the Go/NoGo task. In particular, Coherence ($r = -0.256$; $p = 0.021$), but not RMSSD, was significantly and negatively correlated with the percentage of commissions (Commissions). No other significant correlation between HRV and indices of performance at the Go/NoGo, SRT and the N-Back tasks emerged.

Finally, the best and average lap times – *i.e.*, the two measures of driving performance assessed – displayed a significant negative correlation with the coherence between HRV and respiratory activity (Coherence; respectively $r = -0.416$; $p = 0.043$; $r = -0.430$; $p = 0.036$) and a significant positive correlation with the mean response time of correct responses to the Go trials of the Go/NoGo task (RT; respectively $r = 0.488$; $p = 0.015$; $r = 0.444$; $p = 0.030$). Drivers with a stronger coupling of cardiac and respiratory activity and those who were faster at correctly responding at the Go trials of the Go/NoGo task delivered a better driving performance, recording faster laps, both when considering the best and the average lap times.

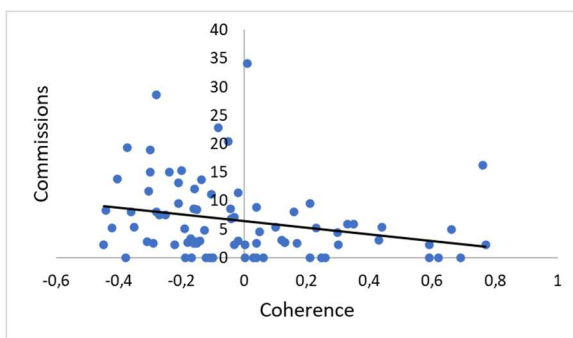
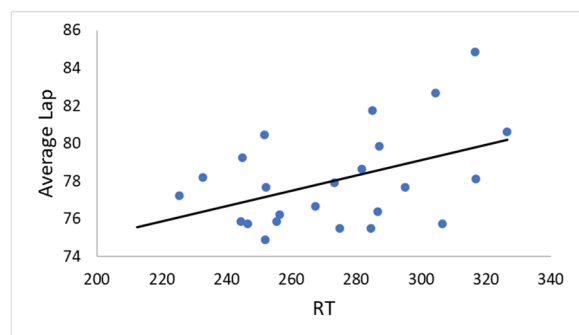
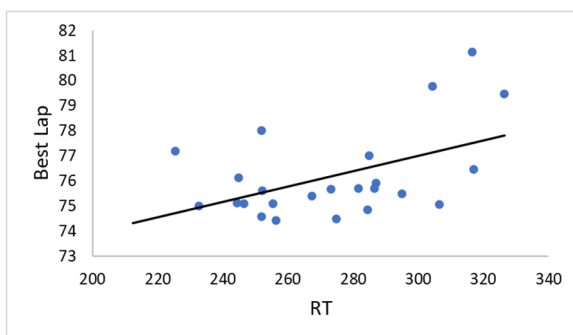
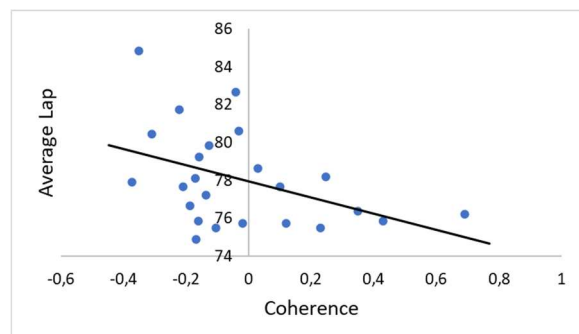
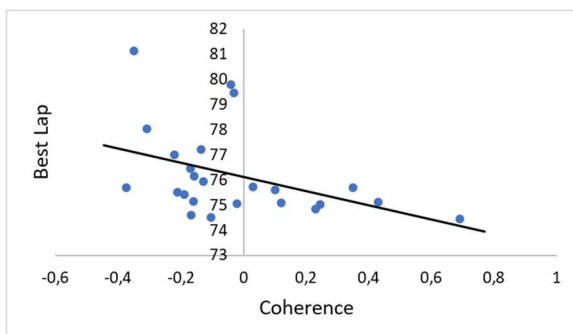


Fig. 41 – Plots showing the correlation between: a) Coherence and Commissions (first row, left); b) Coherence and Best Lap (second row, left); c) Coherence and Average Lap (second row, right); d) RT at the Go/NoGo and Best Lap (third row, left); e) RT at the Go/NoGo and Average Lap (third row, right).



Tab. 10 - Correlation Matrix

	Best Lap	Average Lap	Age	Coherence	RMSSD	RT SRT
Best Lap	r p	— —				
Average Lap	r p	0.887 *** <.001	— —			
Age	r p	-0.189 0.377	-0.294 0.164	— —		
Coherence	r p	-0.416 * 0.043	-0.430 * 0.036	-0.092 0.415	— —	
RMSSD	r p	-0.180 0.400	-0.115 0.592	-0.048 0.671	0.294 ** 0.008	— —
RT SRT	r p	0.129 0.548	0.214 0.315	-0.263 * 0.018	-0.036 0.751	0.130 0.249
SD(RT) SRT	r p	-0.062 0.773	-0.005 0.980	-0.232 * 0.037	-0.126 0.261	-0.001 0.994
RT Go/NoGo	r p	0.488 * 0.015	0.444 * 0.030	-0.195 0.082	-0.068 0.546	-0.014 0.899
SD(RT) Go/NoGo	r p	0.166 0.439	0.161 0.451	-0.183 0.102	-0.072 0.520	-0.000 0.998
Accuracy Go/NoGo	r p	-0.005 0.981	0.013 0.951	0.115 0.319	0.192 0.095	0.132 0.253
Commissions	r p	0.055 0.798	0.009 0.969	-0.058 0.607	-0.256 * 0.021	-0.165 0.141
RT N-Back	r p	-0.066 0.760	0.009 0.967	0.063 0.637	0.118 0.374	0.179 0.174
Accuracy N-Back	r p	0.114 0.595	0.123 0.566	-0.114 0.389	0.131 0.321	0.002 0.988
						0.294 * 0.024

Note: * p < .05, ** p < .01, *** p < .001

SD(RT) SRT	RT Go/NoGo	SD(RT) Go/NoGo	Accuracy Go/NoGo	Commissions	RT N-Back	Accuracy N-Back
—	—	—	—	—	—	—
—	—	—	—	—	—	—
0.269 *	—	—	—	—	—	—
0.015	—	—	—	—	—	—
0.409 ****	0.562 ****	—	—	—	—	—
<.001	<.001	—	—	—	—	—
-0.252 *	0.122	-0.220	—	—	—	—
0.027	0.291	0.054	—	—	—	—
0.193	-0.102	0.220 *	-0.957 ****	—	—	—
0.085	0.366	0.049	<.001	—	—	—
0.024	-0.118	-0.164	0.157	-0.156	—	—
0.857	0.372	0.216	0.251	0.238	—	—
0.186	0.090	-0.104	0.008	-0.099	-0.383 **	—
0.159	0.496	0.433	0.955	0.458	0.003	—

4.3.3 Further analyses

Based on our initial hypothesis and on the results obtained by the exploratory analysis, linear regressions were performed to assess the capacity of Coherence, Commissions (both expressed as z scores) and RT at Go/NoGo to explain individual variability in driving performance. The variable “Sim” (Maranello vs Mini-sim) was included treated as a factor in all linear regressions to control for its effect on the data collected. In the first linear regression Best Lap – *i.e.*, the best lap time expressed in seconds – was the dependent variable. More specifically, in the first block, only Coherence and Commission were included as independent variables. Results showed a significant explanatory power for the model ($F = 5.86$; $p(F) = 0.005$), which was able to account for 46.8% of the interindividual variance in the best lap times ($R^2 = 0.468$). In the second block of the first linear regression, also RT at the Go/NoGo was included as an independent variable. The analysis (Tab. 11) revealed a significant predictive power also for this second model ($F = 7.50$; $p(F) = 0.001$), being able to explain 61.2% of the variance in the best lap times recorded ($R^2 = 0.612$). Interestingly, only RT at the Go/NoGo yielded a significant predictive power for Best Lap ($p = 0.016$). Finally, the effect of the simulator used was found to be significant.

Tab. 11 – Best_Lap – Coefficient of the Model - II Block				
Predictor	Estimate	SE	t	p
Intercept ^a	68.854	2.568	26.809***	<.001
Sim:				
Maranello – Mini-sim ^b	2.288	0.630	3.632**	0.002
z_Coherence	-0.396	0.336	-1.177	0.254
z_Commissions	0.012	0.388	0.032	0.974
RT Go/NoGo	0.025	0.009	2.658*	0.016
<i>Note:</i> ^a Reference level; * $p < .05$, ** $p < .01$, *** $p < .001$; ^b reference level = Mini-sim				

In the first block of the second linear regression, Average Lap – *i.e.*, the average duration of all the valid laps completed by the participants during a single driving session – was selected as the dependent variable, while Coherence and Commissions were included as independent variables. Results displayed a significant predictive power for the model ($F = 4.07$; $p(F) = 0.021$), which accounted for 37.9% of the interindividual variance in the average lap times ($R^2 = 0.379$). In the second block of the second linear regression, also RT at the Go/NoGo was included as independent variable. The analysis (Tab. 12) showed

a significant predictive power for this model ($F = 4.42$; $p(F) = 0.011$), which was able to explain 48.2% of the variance in the average lap times recorded ($R^2 = 0.482$). Concerning Average Lap, none of the independent variables included in the two blocks was found to be a significant predictor. Nevertheless, RT at the Go/NoGo showed a trend toward significance ($p = 0.067$), and the two linear models computed were found to explain a significant amount of variance in driving performance, indexed by the average lap times. Finally, once again the effect of the simulator used revealed to be significant in both blocks.

Tab. 12 – Average_Lap – Coefficient of the Model - II Block				
Predictor	Estimate	SE	t	p
Intercept ^a	69.048	4.304	16.042***	<.001
Sim:				
Maranello – Mini-sim ^b	2.517	1.055	2.384*	0.028
z_Coherence	-0.851	0.564	-1.507	0.148
z_Commissions	-0.290	0.650	-0.447	0.660
RT Go/NoGo	0.030	0.015	1.944	0.067
<i>Note:</i> ^a Reference level; * $p < .05$, ** $p < .01$, *** $p < .001$; ^b reference level = Mini-sim				

4.4 Discussion

The aim of this study was to enhance the understanding of the physiological and cognitive mechanisms underlying high-level peak driving performance, using a systematic theoretical and methodological approach for the collection and interpretation of the data. We adopted a widely-held evidence-based theoretical model accounting for the relationship between physiologic and cognitive resources and behavioural outputs – *i.e.*, the Neurovisceral Integration Perspective (Thayer and Lane, 2000; Thayer et al., 2009; Thayer et al., 2012) – as well as validated techniques and methods for the assessment of HRV and cognitive functions, in order to investigate how these factors contribute to driving performance, as measured by a very ecological task, in a well-defined sample of professional drivers, mainly belonging to the Ferrari Driver Academy. First, we run an exploratory analysis in order to assess the presence of correlations between physiological resources, cognitive functioning and driving performance. Second, we performed linear regressions to test the ability of a subset of physiological and cognitive data to explain inter-individual differences in driving outcomes.

A significant negative correlation was found between age and the performance at the SRT task, with older individuals being faster and more consistent in responding to the target stimuli. This might be explained by the fact that older subjects may be more capable of maintaining the attentive focus on the task, being less distracted and therefore faster in detecting the appearance of the target stimuli and in responding to them. No other significant correlation between age and HRV parameters, as well as among the former and the performance at the Go/NoGo and the N-Back task was found. Although unexpected, the lack of significant correlations between age and the other variables might be related to the fact that the age-range of the subjects included in the study was small, and therefore there was not enough variability in the data to replicate some findings previously reported in the literature, reporting links between age and HRV (Jensen-Urstad et al., 1997; Umetani et al., 1998), as well as among age and the performance at executive tasks (Rodriguez-Villagra, Göthe, Oberauer and Kliegl, 2013).

As hypothesised, the results of the exploratory analysis displayed a significant correlation between vagally mediated HRV and the ability to inhibit inadequate responses at the Go/NoGo task. In particular, on the one side the coherence between HRV and respiratory activity was negatively correlated with the percentage of commissions at the task. This is in line with the assumptions of the Neurovisceral Integration Paradigm, stating that individuals with higher resting parasympathetic HRV indices also display a better inhibitory control (Thayer and Lane, 2000; Thayer et al., 2009). On the other side, the lack of correlation between RMSSD and the measures of inhibitory control might be related to the fact that, differently from Coherence, this index is not corrected for the influence of respiratory activity and, therefore, it provides a less accurate proxy of parasympathetic influence on cardiac control.

Furthermore, in line with our hypothesis, no correlation emerged between parasympathetic HRV and the performance at the SRT task. This is in line with the findings reported by a wealth of studies (Hansen, Johnsen, and Thayer, 2003; 2009) showing no correlation between HRV and the performance at non-executive tasks, but also with the theoretical framework provided by the Neurovisceral Integration model, according to which the central neural structures involved in the modulation of cardiac autonomic control are dissociated from those responsible for the performance of non-executive tasks, such as the SRT ones (Thayer et al., 2009).

Contrarily to our hypothesis of a positive correlation between vagally-mediated HRV and the accuracy at the N-Back task, no correlation was found. This was in contrast with the results of multiple studies previously published in literature (Hansen, Johnsen and Thayer, 2000; 2009; Hansen et al., 2004) and with the Neurovisceral Integration model, which theorised a reciprocal interaction and influence between the CNS structures in charge of modulating the autonomic action on cardiac functioning, and those underlying the performance of working memory-related functions (Thayer et al., 2009). It must be noted that, since the PFCs undergo considerable development throughout the age-range characterising the participants included in the study (12 to 26), measures of WM might not be the most accurate index of executive functioning in this specific population. Future studies should evaluate more in depth the relationship between vagally-mediated HRV and measures of performance at the N-back tasks in this population.

Concerning driving performance, the exploratory analysis reported a negative correlation between one vagally-mediated measure of HRV (*i.e.*, Coherence) and both the best and average lap times, recorded during the driving session at a professional driving simulator. This result met our initial hypothesis, based on previous studies showing a shift in sympathovagal balance to occur while driving, as an autonomic response to maintain homeostasis and support driving performance, suggesting that individuals with more physiological resources and stronger parasympathetic predominance on cardiac control at rest, may deliver a more effective peak driving performance. Moreover, this result replicated previous findings reporting a positive link between resting HRV and the performance at a wide variety of sports (Blasco-Lafarga, Martinez-Navarro, and Mateo-March, 2013; Jimenez Morgan and Molina Mora, 2017), extending them to the field of elite motorsports. Overall, this is an important result, since it provides empirical support to our hypothesis (in accord with the suggestion made by Yamakoshi et al., 2010) that the delivery of a better peak driving performance by individuals with enhanced resting parasympathetic predominance on cardiac functioning would be mediated by a broader cardiac reserve, allowing them to prevent vagal suppression to occur while driving and to maintain the organism able to adapt to the context and execute a better driving performance. Regarding the lack of a significant correlation between RMSSD and the two measures of driving performance, the most likely explanation is provided by the fact that this index of vagally mediated cardiac control was not corrected for the influence of

respiratory activity, and this may have hidden the existing relationship between the aforementioned variables.

Both measures of driving performance positively correlated with the mean response time of correct responses to the Go trials of the Go/NoGo task (mean_RT_GNG) – *i.e.*, individuals who were faster in correctly responding to the target stimuli were also faster when considering their best and average lap times at the simulator. This finding may be explained by the fact that the readiness in responding to the presentation of the target stimuli, although not a pure index of inhibitory control, might reflect the individual ability to quickly respond to task-relevant information, which, in turn, represents a critical ability for a high-level driving performance. Finally, no correlation was found between the two measures of driving performance, the accuracy and the percentage of commissions at the Go/NoGo task. This lack of correlation is likely to be induced by the relative easiness of the task, which induced a “ceiling effect”, reducing the variability in the data concerning accuracy, or by the absence of opponents and unexpected events in the simulation, reducing the amount of information requiring active inhibition, in order to support an effective driving performance.

Based on our hypotheses and the results provided by the correlational exploratory analysis, we investigated the contribution of a subset of physiological and cognitive variables to the interindividual variability in high-level peak driving performance. First, we assessed the predictivity of cardiorespiratory coherence and the percentage of commissions at the Go/NoGo task for the best and average lap times recorded by the drivers. Interestingly, neither coherence nor commissions (expressed in z-scores) were found to be significant predictors of the two indexes of driving performance. This represented an interesting finding to report since we expected driving performance to be predicted by vagally mediated time-domain HRV and by measures of inhibitory control, in particular by the percentage of commissions. Regarding the non-predictivity of cardiovascular coherence, it might be induced by the fact that we assessed such a coherence as a baseline level. Therefore, the data included in the analysis did not represent the actual coherence present between the cardiovascular and the respiratory systems of the individuals during tasks (*e.g.*, slow paced abdominal breathing at resonance frequency, stress-inducing tasks etc), which could be a better proxy of the actual physiological resources available to support a good peak-performance. Concerning the

lack of predictive power of the percentage of commissions at Go/NoGo, it might be related to the fact that the driving task consisted in driving as to record the best lap times, but no opponents, traffic, change of environmental conditions, or unexpected event occurred during the task. Therefore, other than inputs strictly related to driving itself, no other intervenient information was present, requiring an active inhibition to maintain the focus on the driving task. For these reasons, it could be possible that the task we used to evaluate driving performance did not include some of the main factors that require inhibitory control in order to prevent their negative influence on driving. Alternatively, another possible explanation for this unexpected finding can be provided by the relative easiness of the Go/NoGo task, which induced a ceiling effect in the data concerning the percentage of commissions. Due to this effect, it is likely that we were not able to gather all the interindividual variability in inhibitory control and therefore this result might represent a false negative.

Second, we added to the two linear models (one for the best and one for the average lap) also the other variable that was found by the exploratory analysis to be significantly correlated with the two indexes of driving performance – *i.e.*, the mean reaction time of correct responses to the go trials of the Go/NoGo task or Go/NoGo readiness (RT). Relevantly, Go/NoGo readiness was the only significant predictor for the best lap times recorded by the drivers, whereas it only approached significance as a predictor of the average lap times, probably because different drivers managed to complete a different number of valid laps during their single driving session. This represents a key finding to discuss, as it suggests that, when it comes to pure peak driving performance, executed on a racing track, without any intervening factor, the most critical cognitive process underlying a high-level performance is the ability to perceive, process and respond to the task-relevant information quickly. While task-relevant information in our task was the appearance of a green light in the central section of a starting light, in racing driving it is related to multimodal sensory inputs, and to feedback related to the motor outputs performed by the driver. Although different, the finding currently discussed suggest that the ability to quickly respond to relevant information is the one that mainly explains interindividual differences in peak driving performance, at least when it comes to elite racing drivers, with outstanding skills in the control of a single-seater car.

Finally, the linear models we created demonstrated to be effective in explaining a significant portion of the interindividual variance in peak performance at a driving simulator in a sample of elite racing drivers, both when considering the best and the average lap times. Although more research on the mechanisms linking autonomic cardiac control, cognitive functioning and driving performance is needed, our findings may contribute to the improvement of the study designs used to conduct research in this field as well as of the paradigms to select young drivers for the most prestigious driving academies. Indeed, our results further highlight the contribution of physiological and executive resources to peak driving performance, introduced in the previous chapters, supporting the relevance of the Neurovisceral Integration Model for the field of motorsport, and the use of HRV and cognitive assessment during the evaluation of the drivers. Further studies are now needed, to ulteriorly clarify the relationship between HRV indexes, measures of cognitive and executive functions, and driving performance, as well as to test the effects of HRV-biofeedback and cognitive training protocols on drivers' performance, in order to replicate and extend previous findings reported in other sports.

4.5 Limitations and future research

Along with recognising the contribute of the current study to the understanding of the resources and mechanisms underlying peak driving performance in elite racing drivers, it is also important to acknowledge some limitations. We included in the final analyses only male participants, due to the limited availability of data from female subjects. Also due to previous findings reporting sex-related differences in HRV (Jensen-Urstad et al., 1997; Umetani et al., 1998), it is fundamental for future research to assess such differences as well as the relationship between physiological and cognitive resources and peak driving performance also in female racing drivers. Moreover, we included in the study only elite racing drivers, that we assessed using a highly ecological driving task. Therefore, although our findings could be generalised to the elite population of interest, the generalisability of our results to other populations needs to be tested by future studies.

Concerning cognitive data, depending on the age of the subjects and on the age-dependent degree of development of PFCs, WM was assessed using a 2- or a 3-Back task. This introduced in the data additional variability, which may explain why we could not find

the relationship we expected between WM and driving performance. Future studies will have to assess the former more consistently, in order to better clarify its potential role in supporting the latter. Moreover, more challenging tasks will be needed to assess inhibitory control, in order to avoid the ceiling effect that was present in our data, and to obtain a more accurate understanding of its contribution to the delivery of peak driving performance by professional racing drivers.

Finally, data were collected with two different driving simulators. In particular, the participants assessed with the Mini-sim were significantly faster than those who drove using the Maranello sim. In fact, although the latter is meant to provide more realistic sensory inputs to the drivers, the better driving performance found in those who used the Mini-sim could be explained by the fact that it is more similar to the gaming set ups that most of the drivers use to train at home. Although we do not believe that the variability introduced in the data by the different simulators had a significant impact on the general findings reported and discussed above, it will be important for future research to be more systematic in the choice of the hardware used to assess driving performance.

4.6 Conclusions

The aim of the present study was to assess the relationship between physiological, cognitive and executive resources, and peak driving performance, in a sample of elite racing drivers, using a very ecological task. Overall, we extended to the field of motorsport some findings previously reported in literature. We provided further support to the Neurovisceral Integration Perspective, by reporting a positive correlation between vagally mediated parasympathetic cardiac control at rest and inhibitory control, as well as a lack of correlation between the former and the performance at a non-executive SRT task, consistently with the claims made by such a model. Moreover, we showed that cardiorespiratory coherence, indexing parasympathetic modulation of cardiac activity, a measure of inhibitory control – *i.e.*, the percentage of commissions at Go/NoGo – and a measure of the ability to quickly respond to task-relevant information (*i.e.*, Go/NoGo readiness) can be used to create a linear model, able to explain a significant amount of interindividual variability in peak driving performance in elite racing drivers, providing

a useful tool for their assessment and selection, as well as for the development of future studies in this field.

References:

- Acharya, U., Joseph, K., Kannathal, N., Lim, C. M., & Suri, J. S. (2006). Heart rate variability: a review. *Medical and biological engineering and computing*, 44, 1031-1051.
- Ahern, G. L., Sollers, J. J., Lane, R. D., Labiner, D. M., Herring, A. M., Weinand, M. E., ... & Thayer, J. F. (2001). Heart rate and heart rate variability changes in the intracarotid sodium amobarbital test. *Epilepsia*, 42(7), 912-921.
- Ahmed, M. W., Kadish, A. H., Parker, M. A., & Goldberger, J. J. (1994). Effect of physiologic and pharmacologic adrenergic stimulation on heart rate variability. *Journal of the American College of Cardiology*, 24(4), 1082-1090.
- Albinet, C. T., Abou-Dest, A., André, N., & Audiffren, M. (2016). Executive functions improvement following a 5-month aquaerobics program in older adults: Role of cardiac vagal control in inhibition performance. *Biological psychology*, 115, 69-77.
- Albinet, C. T., Boucard, G., Bouquet, C. A., & Audiffren, M. (2010). Increased heart rate variability and executive performance after aerobic training in the elderly. *European journal of applied physiology*, 109, 617-624.
- Allport, A., & Wylie, G. (2000). Task switching, stimulus-response bindings, and negative priming. *Control of cognitive processes: Attention and performance XVIII*, 35-70.
- Ammendola, E., Russo, V., Politano, L., Santangelo, L., & Calabrò, R. (2006). Is heart rate variability a valid parameter to predict sudden death in patients with Becker's muscular dystrophy?. *Heart*, 92(11), 1686-1687.
- Anderson, M. C., & Levy, B. J. (2009). Suppressing unwanted memories. *Current Directions in Psychological Science*, 18(4), 189-194.
- Anderson, M. C., Ochsner, K. N., Kuhl, B., Cooper, J., Robertson, E., Gabrieli, S. W., ... & Gabrieli, J. D. (2004). Neural systems underlying the suppression of unwanted memories. *Science*, 303(5655), 232-235.
- Angius, L., Santarnecchi, E., Pascual-Leone, A., & Marcora, S. M. (2019). Transcranial direct current stimulation over the left dorsolateral prefrontal cortex improves inhibitory control and endurance performance in healthy individuals. *Neuroscience*, 419, 34-45.

- Arai, Y., Saul, J. P., Albrecht, P., Hartley, L. H., Lilly, L. S., Cohen, R. J., & Colucci, W. S. (1989). Modulation of cardiac autonomic activity during and immediately after exercise. *American Journal of Physiology-Heart and Circulatory Physiology*, 256(1), H132-H141.
- Aron, A. R., Fletcher, P. C., Bullmore, E. T., Sahakian, B. J., & Robbins, T. W. (2003). Stop-signal inhibition disrupted by damage to right inferior frontal gyrus in humans. *Nature neuroscience*, 6(2), 115-116.
- Aron, A. R., Monsell, S., Sahakian, B. J., & Robbins, T. W. (2004). A componential analysis of task-switching deficits associated with lesions of left and right frontal cortex. *Brain*, 127(7), 1561-1573.
- Aron, A. R., Robbins, T. W., & Poldrack, R. A. (2004). Inhibition and the right inferior frontal cortex. *Trends in cognitive sciences*, 8(4), 170-177.
- Azevedo, E., Santos, R., Freitas, J., Rosas, M. J., Gago, M., Garrett, C., & Rosengarten, B. (2010). Deep brain stimulation does not change neurovascular coupling in non-motor visual cortex: an autonomic and visual evoked blood flow velocity response study. *Parkinsonism & related disorders*, 16(9), 600-603.
- Backs, R. W. (2000). An autonomic space approach to the psychophysiological assessment of mental workload. In *Stress, workload, and fatigue* (pp. 279-289). CRC Press.
- Backs, R. W. (1995). Going beyond heart rate: autonomic space and cardiovascular assessment of mental workload. *The international journal of aviation psychology*, 5(1), 25-48.
- Backs, R. W., Lenneman, J. K., Wetzel, J. M., & Green, P. (2003). Cardiac measures of driver workload during simulated driving with and without visual occlusion. *Human Factors*, 45(4), 525-538.
- Baddeley, A. (2000). The episodic buffer: a new component of working memory?. *Trends in cognitive sciences*, 4(11), 417-423.
- Baddeley, A. (1998). Working memory. *Comptes Rendus de l'Académie des Sciences-Series III-Sciences de la Vie*, 321(2-3), 167-173.

- Baddeley, A. D., & Hitch, G. J. (1974). Working memory (Vol. 8). New York: GA Bower (ed), Recent advances in learning and motivation.
- Baddeley, A. D., & Hitch, G. J. (1994). Developments in the concept of working memory. *Neuropsychology*, 8(4), 485.
- Baddeley, A., Logie, R., Bressi, S., Sala, S. D., & Spinnler, H. (1986). Dementia and working memory. *The Quarterly Journal of Experimental Psychology Section A*, 38(4), 603-618.
- Badre, D., & Frank, M. J. (2012). Mechanisms of hierarchical reinforcement learning in cortico-striatal circuits 2: Evidence from fMRI. *Cerebral cortex*, 22(3), 527-536.
- Bailey, C. E. (2007). Cognitive accuracy and intelligent executive function in the brain and in business. *Annals of the New York Academy of Sciences*, 1118(1), 122-141.
- Baler, R. D., & Volkow, N. D. (2006). Drug addiction: the neurobiology of disrupted self-control. *Trends in molecular medicine*, 12(12), 559-566.
- Barch, D. M. (2005). The cognitive neuroscience of schizophrenia. *Annu. Rev. Clin. Psychol.*, 1, 321-353.
- Barkley, R. A. (1999). Response inhibition in attention-deficit hyperactivity disorder. *Mental retardation and developmental disabilities research reviews*, 5(3), 177-184.
- Baroody, N. B., Thomason, J. M., & O'Bryan Jr, E. C. (1973). The heart of the 500 mile race. *American family physician*, 8(4), 184-189.
- Barron, S. A., Rogovski, Z. E., & Hemli, J. (1994). Autonomic consequences of cerebral hemisphere infarction. *Stroke*, 25(1), 113-116.
- Barrett, C. J., Navakatikyan, M. A., & Malpas, S. C. (2001). Long-term control of renal blood flow: what is the role of the renal nerves?. *American Journal of Physiology-Regulatory, Integrative and Comparative Physiology*, 280(5), R1534-R1545.
- Beauchaine, T. P., & Thayer, J. F. (2015). Heart rate variability as a transdiagnostic biomarker of psychopathology. *International journal of psychophysiology*, 98(2), 338-350.

- Beckers, F., Ramaekers, D., Speijer, G., Ector, H., Vanhaecke, J., Verheyden, B., ... & Aubert, A. E. (2004). Different evolutions in heart rate variability after heart transplantation: 10-year follow-up. *Transplantation*, 78(10), 1523-1531.
- Bedini, R., Belardinelli, A., Palagi, G., Varanini, M., Ripoli, A., Berti, S., ... & Ceccarelli, R. (1995, September). ECG telemetric evaluation in formula one drivers. In *Computers in Cardiology 1995* (pp. 353-356). IEEE.
- Benarroch, E. E. (1993). The central autonomic network: functional organization, dysfunction, and perspective. In *Mayo Clinic Proceedings* (Vol. 68, No. 10, pp. 988-1001). Elsevier.
- Benarroch, E. E. (1997). The central autonomic network. *Clinical autonomic disorders*, 17-23.
- Bernardi, L., Ricordi, L., Lazzari, P., Solda, P., Calciati, A., Ferrari, M. R., ... & Fratino, P. (1992). Impaired circadian modulation of sympathovagal activity in diabetes. A possible explanation for altered temporal onset of cardiovascular disease. *Circulation*, 86(5), 1443-1452.
- Berntson, G. G., Cacioppo, J. T., & Quigley, K. S. (1993). Cardiac psychophysiology and autonomic space in humans: empirical perspectives and conceptual implications. *Psychological bulletin*, 114(2), 296.
- Berntson, G. G., Bigger Jr, J., Eckberg, D. L., Grossman, P., Kaufmann, P. G., Malik, M., ... & Van der Molen M. W. (1997). Heart rate variability: origins, methods, and interpretive caveats. *Psychophysiology*, 34(6), 623-648.
- Bertrand, C., Keromes, A., Lemeunier, B. F., Meistelmann, C., Prieur, C., & Richalet, J. P. (1983). *Physiologie des sports Mécaniques*. In 1st international congress of sport automobile, Marseilles.
- Biddle, S. J., & Fuchs, R. (2009). Exercise psychology: A view from Europe. *Psychology of Sport and Exercise*, 10(4), 410-419.
- Bigger Jr, J. T., Fleiss, J. L., Rolnitzky, L. M., Steinman, R. C., & Schneider, W. J. (1991). Time course of recovery of heart period variability after myocardial infarction. *Journal of the American College of Cardiology*, 18(7), 1643-1649.

- Bigger Jr, J. T., Fleiss, J. L., Rolnitzky, L. M., Steinman, R. C., & Schneider, W. J. (1991). Time course of recovery of heart period variability after myocardial infarction. *Journal of the American College of Cardiology*, 18(7), 1643-1649.
- Bigger Jr, J. T., Fleiss, J. L., Steinman, R. C., Rolnitzky, L. M., Kleiger, R. E., & Rottman, J. N. (1992). Frequency domain measures of heart period variability and mortality after myocardial infarction. *Circulation*, 85(1), 164-171.
- Blair, C., & Razza, R. P. (2007). Relating effortful control, executive function, and false belief understanding to emerging math and literacy ability in kindergarten. *Child development*, 78(2), 647-663.
- Blasco-Lafarga, C., Martinez-Navarro, I., & Mateo-March, M. (2013). Is baseline cardiac autonomic modulation related to performance and physiological responses following a supramaximal Judo test?. *PloS one*, 8(10), e78584.
- Bloomfield, D. M., Zweibel, S., Bigger Jr, J. T., & Steinman, R. C. (1998). RR variability detects increases in vagal modulation with phenylephrine infusion. *American Journal of Physiology-Heart and Circulatory Physiology*, 274(5), H1761-H1766.
- Bock, A. M., Gallaway, K. C., & Hund, A. M. (2015). Specifying links between executive functioning and theory of mind during middle childhood: Cognitive flexibility predicts social understanding. *Journal of cognition and development*, 16(3), 509-521.
- Boettger, S., Hoyer, D., Falkenhahn, K., Kaatz, M., Yeragani, V. K., & Bär, K. J. (2006). Altered diurnal autonomic variation and reduced vagal information flow in acute schizophrenia. *Clinical Neurophysiology*, 117(12), 2715-2722.
- Börger, N., van Der Meere, J., Ronner, A., Alberts, E., Geuze, R., & Bogte, H. (1999). Heart rate variability and sustained attention in ADHD children. *Journal of Abnormal Child Psychology*, 27, 25-33.
- Borella, E., Carretti, B., & Pelegrina, S. (2010). The specific role of inhibition in reading comprehension in good and poor comprehenders. *Journal of Learning disabilities*, 43(6), 541-552.

- Boulton, A. J., Vinik, A. I., Arezzo, J. C., Bril, V., Feldman, E. L., Freeman, R., ... & Ziegler, D. (2005). Diabetic neuropathies: a statement by the American Diabetes Association. *Diabetes care*, 28(4), 956-962.
- Boveda, S., Galinier, M., Pathak, A., Fourcade, J. L., Dongay, B., Benchendikh, D., ... & Bounhoure, J. P. (2001). Prognostic value of heart rate variability in time domain analysis in congestive heart failure. *Journal of interventional cardiac electrophysiology*, 5, 181-187.
- Brack, K. E., Coote, J. H., & Ng, G. A. (2004). Interaction between direct sympathetic and vagus nerve stimulation on heart rate in the isolated rabbit heart. *Experimental physiology*, 89(1), 128-139.
- Brandt, J. (1991). The Hopkins Verbal Learning Test: Development of a new memory test with six equivalent forms. *The clinical neuropsychologist*, 5(2), 125-142.
- Brezinova, M., Goldenberg, Z., & Kucera, P. (2004). Autonomic nervous system dysfunction in multiple sclerosis patients. *Bratisl lek listy*, 105(12), 404-407.
- Broidy, L. M., Nagin, D. S., Tremblay, R. E., Bates, J. E., Brame, B., Dodge, K. A., ... & Vitaro, F. (2003). Developmental trajectories of childhood disruptive behaviors and adolescent delinquency: a six-site, cross-national study. *Developmental psychology*, 39(2), 222.
- Brookhuis, K. A., de Vries, G., & De Waard, D. (1991). The effects of mobile telephoning on driving performance. *Accident Analysis & Prevention*, 23(4), 309-316.
- Brown TE, Landgraf JM. 2010. Improvements in executive function correlate with enhanced performance and functioning and health-related quality of life: evidence from 2 large, double-blind, randomized, placebo-controlled trials in ADHD. *Postgrad. Med.* 122:42–51
- Casolo, G., Balli, E., Taddei, T., Amuhasi, J., & Gori, C. (1989). Decreased spontaneous heart rate variability in congestive heart failure. *The American journal of cardiology*, 64(18), 1162-1167.

- Casolo, G. C., Stroder, P., Signorini, C., Calzolari, F., Zucchini, M., Balli, E., ... & Lazzarini, S. (1992). Heart rate variability during the acute phase of myocardial infarction. *Circulation*, 85(6), 2073-2079.
- Chatham, C. H., & Badre, D. (2015). Multiple gates on working memory. *Current opinion in behavioral sciences*, 1, 23-31.
- Chalmers, J. A., Quintana, D. S., Abbott, M. J. A., & Kemp, A. H. (2014). Anxiety disorders are associated with reduced heart rate variability: a meta-analysis. *Frontiers in psychiatry*, 5, 80.
- Chen, S. Y., Yang, C. C., Kuo, T. B., & Harnod, T. (2011). Association of heart rate variability with clinical outcome in parkinsonian patients after subthalamic deep brain stimulation: a retrospective cohort study. *Journal of the Formosan Medical Association*, 110(9), 593-599.
- Chess, G. F., Tam, R. M., & Calaresu, F. R. (1975). Influence of cardiac neural inputs on rhythmic variations of heart period in the cat. *American Journal of Physiology-Legacy Content*, 228(3), 775-780.
- Cohen, H., Kaplan, Z., Kotler, M., Mittelman, I., Osher, Y., & Bersudsky, Y. (2003). Impaired heart rate variability in euthymic bipolar patients. *Bipolar Disorders*, 5(2), 138-143.
- Cohen, H., Loewenthal, U., Matar, M., & Kotler, M. (2001). Heart rate variability in schizophrenic patients treated with antipsychotic agents. *Harefuah*, 140(12), 1142-1147+.
- Cohen, M. E., Hudson, D. L., & Deedwania, P. C. (1996). Applying continuous chaotic modeling to cardiac signal analysis. *IEEE Engineering in Medicine and Biology Magazine*, 15(5), 97-102.
- Collins, A., & Koechlin, E. (2012). Reasoning, learning, and creativity: frontal lobe function and human decision-making. *PLoS biology*, 10(3), e1001293.
- Compte, A., Brunel, N., Goldman-Rakic, P. S., & Wang, X. J. (2000). Synaptic mechanisms and network dynamics underlying spatial working memory in a cortical network model. *Cerebral cortex*, 10(9), 910-923.

- Corsi, P. M. (1972). Human memory and the medial temporal region of the brain.
- Cortese, M. D., Vatrano, M., Tonin, P., Cerasa, A., & Riganello, F. (2022). Inhibitory Control and Brain–Heart Interaction: An HRV-EEG Study. *Brain Sciences*, 12(6), 740.
- Courtney, S. M., Ungerleider, L. G., Keil, K., & Haxby, J. V. (1997). Transient and sustained activity in a distributed neural system for human working memory. *Nature*, 386(6625), 608-611.
- Craik, F. I., Klix, F., & Hagendorf, H. (1986). A functional account of age differences in memory. *Memory, attention, and aging: Selected works of Fergus IM Craik*, 409-422.
- Crescioni, A., Ehrlinger, J., Alquist, J. L., Conlon, K. E., Baumeister, R. F., Schatschneider, C., & Dutton, G. R. (2011). High trait self-control predicts positive health behaviors and success in weight loss. *Journal of health psychology*, 16(5), 750-759.
- Cygankiewicz, I., & Zareba, W. (2013). Heart rate variability. *Handbook of clinical neurology*, 117, 379-393.
- Cizman Staba, U., Klun, T., Stojmenova, K., Jakus, G., & Sodnik, J. (2022). Consistency of neuropsychological and driving simulator assessment after neurological impairment. *Applied Neuropsychology: Adult*, 29(4), 829-838.
- Damasio, A. R. (1998). Emotion in the perspective of an integrated nervous system. *Brain research reviews*, 26(2-3), 83-86.
- Davidson, M. C., Amso, D., Anderson, L. C., & Diamond, A. (2006). Development of cognitive control and executive functions from 4 to 13 years: Evidence from manipulations of memory, inhibition, and task switching. *Neuropsychologia*, 44(11), 2037-2078.
- Davis JC, Marra CA, Najafzadeh M, Lui-Ambrose T. 2010. The independent contribution of executive functions to health related quality of life in older women. *BMC Geriatr*. 10:16–23
- Deary, I. (2000). *Looking down on human intelligence: From psychometrics to the brain* (Vol. 36). OUP Oxford.

- Deary, I. J., Johnson, W., & Starr, J. M. (2010). Are processing speed tasks biomarkers of cognitive aging?. *Psychology and aging*, 25(1), 219.
- Delaney, J. P. A., & Brodie, D. A. (2000). Effects of short-term psychological stress on the time and frequency domains of heart-rate variability. *Perceptual and motor skills*, 91(2), 515-524.
- Delis, D. C., Kramer, J. H., Kaplan, E., & Ober, B. A. (2000). *California verbal learning test--*. Assessment.
- Denson, T. F., Pedersen, W. C., Friese, M., Hahm, A., & Roberts, L. (2011). Understanding impulsive aggression: Angry rumination and reduced self-control capacity are mechanisms underlying the provocation-aggression relationship. *Personality and Social Psychology Bulletin*, 37(6), 850-862.
- De Renzi, A., & Vignolo, L. A. (1962). Token test: A sensitive test to detect receptive disturbances in aphasics. *Brain: a journal of neurology*.
- D'Esposito, M., & Postle, B. R. (2015). The cognitive neuroscience of working memory. *Annual review of psychology*, 66, 115-142.
- Devinsky, O., Morrell, M. J., & Vogt, B. A. (1995). Contributions of anterior cingulate cortex to behaviour. *Brain*, 118(1), 279-306.
- Diamond, A. (2013). Executive functions. *Annual review of psychology*, 64, 135-168.
- Diamond, A. (2005). Attention-deficit disorder (attention-deficit/hyperactivity disorder without hyperactivity): A neurobiologically and behaviorally distinct disorder from attention-deficit/hyperactivity disorder (with hyperactivity). *Development and psychopathology*, 17(3), 807-825.
- Dimsdale, J. E., & Ziegler, M. G. (1991). What do plasma and urinary measures of catecholamines tell us about human response to stressors?. *Circulation*, 83(4 Suppl), II36-42.
- Donders, F. C. (1969). On the speed of mental processes. *Acta psychologica*, 30, 412-431.

- Duncan, G. J., Dowsett, C. J., Claessens, A., Magnuson, K., Huston, A. C., Klebanov, P., ... & Japel, C. (2007). School readiness and later achievement. *Developmental psychology*, 43(6), 1428.
- Dupuy, O., Bosquet, L., Fraser, S. A., Labelle, V., & Bherer, L. (2018). Higher cardiovascular fitness level is associated to better cognitive dual-task performance in Master Athletes: Mediation by cardiac autonomic control. *Brain and cognition*, 125, 127-134.
- Eakin L, Minde K, Hechtman L, Ochs E, Krane E, et al. 2004. The marital and family functioning of adults with ADHD and their spouses. *J. Attention Disord.* 8:1–10
- Eckardt, N., Roden, I., Grube, D., & Schorer, J. (2020). The relationship between cognition and sensorimotor behavior in an f1 driving simulation: An explorative study. *Frontiers in Psychology*, 11, 574847.
- Einstein, G. O., & McDaniel, M. A. (1990). Normal aging and prospective memory. *Journal of Experimental Psychology: Learning, memory, and cognition*, 16(4), 717.
- Eriksen, B. A., & Eriksen, C. W. (1974). Effects of noise letters upon the identification of a target letter in a nonsearch task. *Perception & psychophysics*, 16(1), 143-149.
- Erola, T., Haapaniemi, T., Heikkinen, E., Huikuri, H., & Myllyä, V. (2006). Subthalamic nucleus deep brain stimulation does not alter long-term heart rate variability in Parkinson's disease. *Clinical Autonomic Research*, 16, 286-288.
- Evrengül, H., Tanriverdi, H., Dursunoglu, D., Kaftan, A., Kuru, O., Unlu, U., & Kilic, M. (2005). Time and frequency domain analyses of heart rate variability in patients with epilepsy. *Epilepsy research*, 63(2-3), 131-139.
- Fairchild, G., van Goozen, S. H., Stollery, S. J., Aitken, M. R., Savage, J., Moore, S. C., & Goodyer, I. M. (2009). Decision making and executive function in male adolescents with early-onset or adolescence-onset conduct disorder and control subjects. *Biological psychiatry*, 66(2), 162-168.
- Fallen, E. L., Kamath, M. V., & Ghista, D. N. (1988). Power spectrum of heart rate variability: a non-invasive test of integrated neurocardiac function. *Clinical and Investigative medicine. Medecine Clinique et Experimentale*, 11(5), 331-340.

- Fan, J., McCandliss, B. D., Sommer, T., Raz, A., & Posner, M. I. (2002). Testing the efficiency and independence of attentional networks. *Journal of cognitive neuroscience*, 14(3), 340-347.
- Fei, L., Copie, X., Malik, M., & Camm, A. J. (1996). Short-and long-term assessment of heart rate variability for risk stratification after acute myocardial infarction. *The American journal of cardiology*, 77(9), 681-684.
- Fisher, A. C., Groves, D., Eleuteri, A., Mesum, P., Patterson, D., & Taggart, P. (2014). Heart rate variability at limiting stationarity: evidence of neuro-cardiac control mechanisms operating at ultra-low frequencies. *Physiological Measurement*, 35(2), 309.
- Folstein MF, Folstein SE, McHugh PR. "Mini-mental state". A practical method for grading the cognitive state of patients for the clinician. *J Psychiatr Res.* 1975 Nov;12(3):189-98.
- Fleisher, L. A., Frank, S. M., Sessler, D. I., Cheng, C., Matsukawa, T., & Vannier, C. A. (1996). Thermoregulation and heart rate variability. *Clinical science*, 90(2), 97-103.
- Frank, M. J., & Badre, D. (2012). Mechanisms of hierarchical reinforcement learning in corticostriatal circuits 1: computational analysis. *Cerebral cortex*, 22(3), 509-526.
- Friedman, B. H., & Thayer, J. F. (1998). Autonomic balance revisited: panic anxiety and heart rate variability. *Journal of psychosomatic research*, 44(1), 133-151.
- Frontoni, M., Fiorini, M., Strano, S., Cerutti, S., Giubilei, F., Urani, C., ... & Pozzilli, C. (1996). Power spectrum analysis contribution to the detection of cardiovascular dysautonomia in multiple sclerosis. *Acta neurologica scandinavica*, 93(4), 241-245.
- Fuster, J. M., & Alexander, G. E. (1971). Neuron activity related to short-term memory. *Science*, 173(3997), 652-654.
- Gácsi, M., Bunford, N. (2021). Go/No-Go Procedure. In: Vonk, J., Shackelford, T. (eds) *Encyclopedia of Animal Cognition and Behavior*. Springer, Cham. https://doi.org/10.1007/978-3-319-47829-6_1598-1.

- Garavan, H., Ross, T. J., Murphy, K., Roche, R. A., & Stein, E. A. (2002). Dissociable executive functions in the dynamic control of behavior: inhibition, error detection, and correction. *Neuroimage*, 17(4), 1820-1829.
- Garavan, H., Ross, T. J., & Stein, E. A. (1999). Right hemispheric dominance of inhibitory control: an event-related functional MRI study. *Proceedings of the National Academy of Sciences*, 96(14), 8301-8306.
- Garon, N., Bryson, S. E., & Smith, I. M. (2008). Executive function in preschoolers: a review using an integrative framework. *Psychological bulletin*, 134(1), 31.
- Gathercole, S. E., Pickering, S. J., Knight, C., & Stegmann, Z. (2004). Working memory skills and educational attainment: Evidence from national curriculum assessments at 7 and 14 years of age. *Applied Cognitive Psychology: The Official Journal of the Society for Applied Research in Memory and Cognition*, 18(1), 1-16.
- Gaudet, J., Bélanger, M. F., Corriveau, H., Mekary, S., Hay, D., & Johnson, M. J. (2013). Investigating the autonomic nervous system and cognitive functions as potential mediators of an association between cardiovascular disease and driving performance. *Canadian Journal of Physiology and Pharmacology*, 91(5), 346-352.
- Gehi, A., & Whooley, M. (2006). Heart rate variability and depression—reply. *Archives of General Psychiatry*, 63(9), 1052-1052.
- Goodglass, H., & Kaplan, E. (1983). *The assessment of aphasia and related disorders*, (2nd ed.). Philadelphia: Lea & Febiger.
- Goldberger, J. J., Ahmed, M. W., Parker, M. A., & Kadish, A. H. (1994). Dissociation of heart rate variability from parasympathetic tone. *American Journal of Physiology-Heart and Circulatory Physiology*, 266(5), H2152-H2157.
- Goldberger, J. J., Challapalli, S., Tung, R., Parker, M. A., & Kadish, A. H. (2001). Relationship of heart rate variability to parasympathetic effect. *Circulation*, 103(15), 1977-1983.
- Goldman-Rakic, P. S. (1996). The prefrontal landscape: implications of functional architecture for understanding human mentation and the central executive. *Philosophical*

Transactions of the Royal Society of London. Series B: Biological Sciences, 351(1346), 1445-1453.

Goldstein, D. S., Benthó, O., Park, M. Y., & Sharabi, Y. (2011). Low-frequency power of heart rate variability is not a measure of cardiac sympathetic tone but may be a measure of modulation of cardiac autonomic outflows by baroreflexes. *Experimental physiology*, 96(12), 1255-1261.

Gordan, R., Gwathmey, J. K., & Xie, L. H. (2015). Autonomic and endocrine control of cardiovascular function. *World journal of cardiology*, 7(4), 204.

Gordon, D., Herrera, V. L., McAlpine, L., Cohen, R. J., Akselrod, S., Lang, P., & Norwood, W. I. (1988). Heart-rate spectral analysis: a noninvasive probe of cardiovascular regulation in critically ill children with heart disease. *Pediatric cardiology*, 9, 69-77.

Guzzetti, S., Piccaluga, E., Casati, R., Cerutti, S., Lombardi, F., Pagani, M., & Malliani, A. (1988). Sympathetic predominance in essential hypertension: a study employing spectral analysis of heart rate variability. *Journal of hypertension*, 6(9), 711-717.

Hadase, M., Azuma, A., Zen, K., Asada, S., Kawasaki, T., Kamitani, T., ... & Matsubara, H. (2004). Very low frequency power of heart rate variability is a powerful predictor of clinical prognosis in patients with congestive heart failure. *Circulation Journal*, 68(4), 343-347.

Halperin, J. M., Wolf, L., Greenblatt, E. R., & Young, G. (1991). Subtype analysis of commission errors on the continuous performance test in children. *Developmental neuropsychology*, 7(2), 207-217.

Halstead, W. C. (1947). *Brain and intelligence; a quantitative study of the frontal lobes*. University of Chicago Press.

Hansen, A. L., Johnsen, B. H., Sollers, J. J., Stenvik, K., & Thayer, J. F. (2004). Heart rate variability and its relation to prefrontal cognitive function: the effects of training and detraining. *European journal of applied physiology*, 93, 263-272.

Hansen, A. L., Johnsen, B. H., & Thayer, J. F. (2009). Relationship between heart rate variability and cognitive function during threat of shock. *Anxiety, Stress, & Coping*, 22(1), 77-89.

Hansen, A. L., Johnsen, B. H., & Thayer, J. F. (2003). Vagal influence on working memory and attention. *International journal of psychophysiology*, 48(3), 263-274.

Harvey PD. Domains of cognition and their assessment^[P]_[SEP]. *Dialogues Clin Neurosci*. 2019 Sep;21(3):227-237. doi: 10.31887/DCNS.2019.21.3/pharvey. PMID: 31749647; PMCID: PMC6829170.

Hatano, G., & Osawa, K. (1983). Digit memory of grand experts in abacus-derived mental calculation. *Cognition*, 15(1-3), 95-110.

Hautala, A., Tulppo, M. P., Mäkikallio, T. H., Laukkanen, R., Nissilä, S., & Huikuri, H. V. (2001). Changes in cardiac autonomic regulation after prolonged maximal exercise. *Clinical Physiology*, 21(2), 238-245.

Hayano, J., Sakakibara, Y., Yamada, M., Ohte, N., Fujinami, T., Yokoyama, K., ... & Takata, K. (1990). Decreased magnitude of heart rate spectral components in coronary artery disease. Its relation to angiographic severity. *Circulation*, 81(4), 1217-1224.

He, L., Li, C., Luo, Y., Dong, W., & Yang, H. (2010). Clinical prognostic significance of heart abnormality and heart rate variability in patients with stroke. *Neurological research*, 32(5), 530-534.

Hermans, M. C. E., Pinto, Y. M., Merckies, I. S., de Die-Smulders, C. E. M., Crijns, H. J. G. M., & Faber, C. G. (2010). Hereditary muscular dystrophies and the heart. *Neuromuscular Disorders*, 20(8), 479-492.

Hershey, T., Revilla, F. J., Wernle, A. R., McGee-Minnich, L., Antenor, J. V., Videen, T. O., ... & Perlmutter, J. S. (2003). Cortical and subcortical blood flow effects of subthalamic nucleus stimulation in PD. *Neurology*, 61(6), 816-821.

Hidaka, O., Yanagi, M., & Takada, K. (2004). Mental stress-induced physiological changes in the human masseter muscle. *Journal of dental research*, 83(3), 227-231.

- Hirsch, J. A., & Bishop, B. (1981). Respiratory sinus arrhythmia in humans: how breathing pattern modulates heart rate. *American Journal of Physiology-Heart and Circulatory Physiology*, 241(4), H620-H629.
- Hodges, J. R., Patterson, K., Oxbury, S., & Funnell, E. (1992). Semantic dementia: Progressive fluent aphasia with temporal lobe atrophy. *Brain*, 115(6), 1783-1806.
- Hommel, B. (2011). The Simon effect as tool and heuristic. *Acta psychologica*, 136(2), 189-202.
- Howard, D., & Patterson, K. E. (1992). The pyramids and palm trees test.
- Koch, C., Wilhelm, M., Salzmann, S., Rief, W., & Euteneuer, F. (2019). A meta-analysis of heart rate variability in major depression. *Psychological Medicine*, 49(12), 1948-1957.
- Iellamo, F., Legramante, J. M., Pigozzi, F., Spataro, A., Norbiato, G., Lucini, D., & Pagani, M. (2002). Conversion from vagal to sympathetic predominance with strenuous training in high-performance world class athletes. *Circulation*, 105(23), 2719-2724.
- Iwasaki, K. I., Zhang, R., Zuckerman, J. H., & Levine, B. D. (2003). Dose-response relationship of the cardiovascular adaptation to endurance training in healthy adults: how much training for what benefit?. *Journal of Applied Physiology*, 95(4), 1575-1583.
- Jacobs, P. L., Olvey, S. E., Johnson, B. M., & Cohn, K. (2002). Physiological responses to high-speed, open-wheel racecar driving. *Medicine and science in sports and exercise*, 34(12), 2085-2090.
- Jensen-Urstad, K., Storck, N., Bouvier, F., Ericson, M., Lindbland, L. E., & Jensen-Urstad, M. (1997). Heart rate variability in healthy subjects is related to age and gender. *Acta Physiologica Scandinavica*, 160(3), 235-241.
- Jiang, W., Hathaway, W. R., McNulty, S., Larsen, R. L., Hansley, K. L., Zhang, Y., & O'Connor, C. M. (1997). Ability of heart rate variability to predict prognosis in patients with advanced congestive heart failure. *American Journal of Cardiology*, 80(6), 808-811.
- Jimenez Morgan, S., & Molina Mora, J. A. (2017). Effect of heart rate variability biofeedback on sport performance, a systematic review. *Applied psychophysiology and biofeedback*, 42, 235-245.

- Kahneman, D., Treisman, A., & Gibbs, B. J. (1992). The reviewing of object files: Object-specific integration of information. *Cognitive psychology*, 24(2), 175-219.
- Kallio, M., Haapaniemi, T., Turkka, J., Suominen, K., Tolonen, U., Sotaniemi, K., I... & Myllylä, V. (2000). Heart rate variability in patients with untreated Parkinson's disease. *European Journal of Neurology*, 7(6), 667-672.
- Kaplan, D. T., & Cohen, R. J. (1990). Searching for Chaos in Fibrillation a. *Annals of the New York Academy of Sciences*, 591(1), 367-374.
- Kaplan, E., Goodglass, H., & Weintraub, S. (1983). *The Boston naming test*. Philadelphia: Lea & Febiger.
- Karalunas, S. L., Geurts, H. M., Konrad, K., Bender, S., & Nigg, J. T. (2014). Annual research review: Reaction time variability in ADHD and autism spectrum disorders: Measurement and mechanisms of a proposed trans-diagnostic phenotype. *Journal of Child Psychology and Psychiatry*, 55(6), 685-710.
- Kemp, A. H., Quintana, D. S., Gray, M. A., Felmingham, K. L., Brown, K., & Gatt, J. M. (2010). Impact of depression and antidepressant treatment on heart rate variability: a review and meta-analysis. *Biological psychiatry*, 67(11), 1067-1074.
- Kessels, R. P., Van Zandvoort, M. J., Postma, A., Kappelle, L. J., & De Haan, E. H. (2000). The Corsi block-tapping task: standardization and normative data. *Applied neuropsychology*, 7(4), 252-258.
- Kienzle, M. G., Ferguson, D. W., Birkett, C. L., Myers, G. A., Berg, W. J., & Mariano, D. J. (1992). Clinical, hemodynamic and sympathetic neural correlates of heart rate variability in congestive heart failure. *The American journal of cardiology*, 69(8), 761-767.
- Kim, H. G., Cheon, E. J., Bai, D. S., Lee, Y. H., & Koo, B. H. (2018). Stress and heart rate variability: a meta-analysis and review of the literature. *Psychiatry investigation*, 15(3), 235.
- Kim, D. H., Lipsitz, L. A., Ferrucci, L., Varadhan, R., Guralnik, J. M., Carlson, M. C., ... & Chaves, P. H. (2006). Association between reduced heart rate variability and cognitive

impairment in older disabled women in the community: Women's Health and Aging Study I. *Journal of the American Geriatrics Society*, 54(11), 1751-1757.

Kingma, J. G., Simard, D., & Rouleau, J. R. (2017). Influence of cardiac nerve status on cardiovascular regulation and cardioprotection. *World journal of cardiology*, 9(6), 508.

Kirchner, W. K. (1958). Age differences in short-term retention of rapidly changing information. *Journal of experimental psychology*, 55(4), 352.

Kiyono, K., Hayano, J., Watanabe, E., & Yamamoto, Y. (2017). Heart rate variability (HRV) and sympathetic nerve activity. *Clinical assessment of the autonomic nervous system*, 147-161.

Kleiger, R. E., Miller, J. P., Bigger Jr, J. T., & Moss, A. J. (1987). Decreased heart rate variability and its association with increased mortality after acute myocardial infarction. *The American journal of cardiology*, 59(4), 256-262.

Koenig, J., & Thayer, J. F. (2016). Sex differences in healthy human heart rate variability: A meta-analysis. *Neuroscience & Biobehavioral Reviews*, 64, 288-310.

Kontopoulos AG, Athyros VG, Papageorgiou AA, Skeberis VM, Basayiannis EC, Boudoulas H. Effect of angiotensin-converting enzyme inhibitors on the power spectrum of heart rate variability in post-myocardial infarction patients. *Coron Artery Dis*. 1997 Aug-Sep;8(8-9):517-24. PMID: 9431480.

Korpelainen, J. T., Sotaniemi, K. A., Huikuri, H. V., & Myllylä, V. V. (1997). Circadian rhythm of heart rate variability is reversibly abolished in ischemic stroke. *Stroke*, 28(11), 2150-2154.

Korpelainen, J. T., Sotaniemi, K. A., Mäkikallio, A., Huikuri, H. V., & Myllylä, V. V. (1999). Dynamic behavior of heart rate in ischemic stroke. *Stroke*, 30(5), 1008-1013.

Kubota, K., & Niki, H. (1971). Prefrontal cortical unit activity and delayed alternation performance in monkeys. *Journal of neurophysiology*, 34(3), 337-347.

Lakusic, N., Mahovic, D., & Babic, T. (2005). Gradual recovery of impaired cardiac autonomic balance within first six months after ischemic cerebral stroke. *Acta Neurol Belg*, 105(1), 39-42.

- Lampert, R., Ickovics, J. R., Viscoli, C. J., Horwitz, R. I., & Lee, F. A. (2003). Effects of propranolol on recovery of heart rate variability following acute myocardial infarction and relation to outcome in the Beta-Blocker Heart Attack Trial. *The American journal of cardiology*, 91(2), 137-142.
- Lane, R. D., McRae, K., Reiman, E. M., Chen, K., Ahern, G. L., & Thayer, J. F. (2009). Neural correlates of heart rate variability during emotion. *Neuroimage*, 44(1), 213-222.
- Lane, R. D., Weidenbacher, H., Smith, R., Fort, C., Thayer, J. F., & Allen, J. J. (2013). Subgenual anterior cingulate cortex activity covariation with cardiac vagal control is altered in depression. *Journal of Affective Disorders*, 150(2), 565-570.
- Langer, A. W., McCubbin, J. A., Stoney, C. M., Hutcheson, J. S., Charlton, J. D., & Obrist, P. A. (1985). Cardiopulmonary adjustments during exercise and an aversive reaction time task: Effects of beta-adrenoceptor blockade. *Psychophysiology*, 22(1), 59-68.
- Lanza, G. A., Russo, A. D., Giglio, V., De Luca, L., Messano, L., Santini, C., ... & Bellocchi, F. (2001). Impairment of cardiac autonomic function in patients with Duchenne muscular dystrophy: relationship to myocardial and respiratory function. *American Heart Journal*, 141(5), 808-812.
- Lappi, O. (2015). The racer's brain—how domain expertise is reflected in the neural substrates of driving. *Frontiers in human neuroscience*, 9, 635.
- La Rovere, M. T., Pinna, G. D., Maestri, R., Mortara, A., Capomolla, S., Febo, O., ... & Cobelli, F. (2003). Short-term heart rate variability strongly predicts sudden cardiac death in chronic heart failure patients. *circulation*, 107(4), 565-570.
- Lee, S. T., & Hon, E. H. (1965). The fetal electrocardiogram: IV. Unusual variations in the QRS complex during labor. *American journal of obstetrics and gynecology*, 92(8), 1140-1148.
- Lee, H. B., Kim, J. S., Kim, Y. S., Baek, H. J., Ryu, M. S., & Park, K. S. (2007, November). The relationship between HRV parameters and stressful driving situation in the real road. In *2007 6th International Special Topic Conference on Information Technology Applications in Biomedicine* (pp. 198-200). IEEE.

- Lefrandt, J., J Smit, A., J Zeebregts, C., OB Gans, R., & H Hoogenberg, K. (2010). Autonomic dysfunction in diabetes: a consequence of cardiovascular damage. *Current diabetes reviews*, 6(6), 348-358.
- Lehto, J. E., Juujärvi, P., Kooistra, L., & Pulkkinen, L. (2003). Dimensions of executive functioning: Evidence from children. *British journal of developmental psychology*, 21(1), 59-80.
- Lenneman, J. K., & Backs, R. W. (2009). Cardiac autonomic control during simulated driving with a concurrent verbal working memory task. *Human factors*, 51(3), 404-418.
- Lenneman, J. K., Shelly, A. R., & Backs, R. W. (2005, June). Deciphering psychological-physiological mappings while driving and performing a secondary memory task. In *Driving Assessment Conference* (Vol. 3, No. 2005). University of Iowa.
- Levy, J. J., & Baldwin, D. R. (2019). Psychophysiology and biofeedback of sport performance.
- Levy, W. C., Cerqueira, M. D., Abrass, I. B., Schwartz, R. S., & Stratton, J. R. (1993). Endurance exercise training augments diastolic filling at rest and during exercise in healthy young and older men. *Circulation*, 88(1), 116-126.
- Levy, M. N., & Martin, P. J. (1984). Neural control of the heart. In *Physiology and Pathophysiology of the Heart* (pp. 337-354). Boston, MA: Springer US.
- Levy, M. N., & Zieske, H. (1969). Autonomic control of cardiac pacemaker activity and atrioventricular transmission. *Journal of applied physiology*, 27(4), 465-470.
- Lewicki, P., Hill, T., & Bizot, E. (1988). Acquisition of procedural knowledge about a pattern of stimuli that cannot be articulated. *Cognitive psychology*, 20(1), 24-37.
- Lewis SF, Nylander E, Gad P, Areskog NH. Non-autonomic component in bradycardia of endurance trained men at rest and during exercise. *Acta Physiol Scand*. 1980 Jul;109(3):297-305. doi: 10.1111/j.1748-1716.1980.tb06600.x. PMID: 7446173.
- Licht, C. M., de Geus, E. J., Zitman, F. G., Hoogendijk, W. J., van Dyck, R., & Penninx, B. W. (2008). Association between major depressive disorder and heart rate variability in

the Netherlands Study of Depression and Anxiety (NESDA). *Archives of general psychiatry*, 65(12), 1358-1367.

Liebherr, M., Antons, S., Schweig, S., Maas, N., Schramm, D., & Brand, M. (2019). Driving performance and specific attentional domains. *Transportation research interdisciplinary perspectives*, 3, 100077.

Lindqvist, A., Jalonen, J., Parviainen, P., Antila, K., & Laitinen, L. A. (1990). Effect of posture on spontaneous and thermally stimulated cardiovascular oscillations. *Cardiovascular research*, 24(5), 373-380.

Litscher, G., He, W., Yi, S. H., & Wang, L. (2014). Heart rate variability and complementary medicine. *Evidence-Based Complementary and Alternative Medicine*, 2014.

Logie, R. H. (1986). Visuo-spatial processing in working memory. *The quarterly Journal of experimental Psychology*, 38(2), 229-247.

Lombardi, F., Sandrone, G., Pernpruner, S., Sala, R., Garimoldi, M., Cerutti, S., ... & Malliani, A. (1987). Heart rate variability as an index of sympathovagal interaction after acute myocardial infarction. *The American journal of cardiology*, 60(16), 1239-1245.

Lombardi, F., Sandrone, G., Spinnler, M. T., Torzillo, D., Lavezzaro, G. C., Brusca, A., & Malliani, A. (1996). Heart rate variability in the early hours of an acute myocardial infarction. *The American journal of cardiology*, 77(12), 1037-1044.

Lotufo, P. A., Valiengo, L., Bensenor, I. M., & Brunoni, A. R. (2012). A systematic review and meta-analysis of heart rate variability in epilepsy and antiepileptic drugs. *Epilepsia*, 53(2), 272-282.

Lu, C. H., & Proctor, R. W. (1995). The influence of irrelevant location information on performance: A review of the Simon and spatial Stroop effects. *Psychonomic bulletin & review*, 2, 174-207.

Luczak, H., & Laurig, W. (1973). An analysis of heart rate variability. *Ergonomics*, 16(1), 85-97.

- Lui, M., & Tannock, R. (2007). Working memory and inattentive behaviour in a community sample of children. *Behavioral and Brain Functions*, 3(1), 1-11.
- Lunt, L., Bramham, J., Morris, R. G., Bullock, P. R., Selway, R. P., Xenitidis, K., & David, A. S. (2012). Prefrontal cortex dysfunction and 'Jumping to Conclusions': Bias or deficit?. *Journal of neuropsychology*, 6(1), 65-78.
- Mahovic, D., & Lakusic, N. (2007). Progressive impairment of autonomic control of heart rate in patients with multiple sclerosis. *Archives of medical research*, 38(3), 322-325.
- Malik, M. (1996). Heart rate variability: Standards of measurement, physiological interpretation, and clinical use: Task force of the European Society of Cardiology and the North American Society for Pacing and Electrophysiology. *Annals of Noninvasive Electrocardiology*, 1(2), 151-181.
- Malik, M., Farrell, T., Cripps, T., & Camm, A. J. (1989). Heart rate variability in relation to prognosis after myocardial infarction: selection of optimal processing techniques. *European heart journal*, 10(12), 1060-1074.
- Malliani, A., Pagani, M., Lombardi, F., & Cerutti, S. (1991). Cardiovascular neural regulation explored in the frequency domain. *Circulation*, 84(2), 482-492.
- Manzi, V., Castagna, C., Padua, E., Lombardo, M., D'Ottavio, S., Massaro, M., ... & Iellamo, F. (2009). Dose-response relationship of autonomic nervous system responses to individualized training impulse in marathon runners. *American Journal of Physiology-Heart and Circulatory Physiology*, 296(6), H1733-H1740.
- Martin, P. A. U. L. (1977). The influence of the parasympathetic nervous system on atrioventricular conduction. *Circulation Research*, 41(5), 593-599.
- McCraty, R., & Shaffer, F. (2015). Heart rate variability: new perspectives on physiological mechanisms, assessment of self-regulatory capacity, and health risk. *Global advances in health and medicine*, 4(1), 46-61.
- McDougall, A. J., & McLeod, J. G. (2003). Autonomic nervous system function in multiple sclerosis. *Journal of the neurological sciences*, 215(1-2), 79-85.

- McKenna, P., & Warrington, E. K. (1983). Graded Naming Test manual. Oxford, UK: NFER-Nelson.
- Meiran, N. (1996). Reconfiguration of processing mode prior to task performance. *Journal of Experimental Psychology: Learning, memory, and cognition*, 22(6), 1423.
- Merkelbach, S., Haensch, C. A., Hemmer, B., Koehler, J., König, N. H., & Ziemssen, T. (2006). Multiple sclerosis and the autonomic nervous system. *Journal of neurology*, 253, i21-i25.
- Mestanikova, A., Ondrejka, I., Mestanik, M., Hrtanek, I., Snircova, E., & Tonhajzerova, I. (2015). Go/NoGo continuous performance task in the psychophysiological research. *Cognitive Remediation Journal*, 4(1), 19-29.
- Miller, E. K. (2000). The prefrontal cortex and cognitive control. *Nature reviews neuroscience*, 1(1), 59-65.
- Miller, H. V., Barnes, J. C., & Beaver, K. M. (2011). Self-control and health outcomes in a nationally representative sample. *American journal of health behavior*, 35(1), 15-27.
- Miller, K. M., Price, C. C., Okun, M. S., Montijo, H., & Bowers, D. (2009). Is the n-back task a valid neuropsychological measure for assessing working memory?. *Archives of Clinical Neuropsychology*, 24(7), 711-717.
- Milner, B. (1963). Effects of different brain lesions on card sorting: The role of the frontal lobes. *Archives of neurology*, 9(1), 90-100.
- Milner B, Squire LR, Kandel ER. (1998). Cognitive neuroscience and the study of memory. *Neuron*. Mar;20(3):445-68.
- Mischel, W., Shoda, Y., & Rodriguez, M. L. (1989). Delay of gratification in children. *Science*, 244(4907), 933-938.
- Mishkin, M., Ungerleider, L. G., & Macko, K. A. (1983). Object vision and spatial vision: two cortical pathways. *Trends in neurosciences*, 6, 414-417.
- Miu, A. C., Heilman, R. M., & Miclea, M. (2009). Reduced heart rate variability and vagal tone in anxiety: trait versus state, and the effects of autogenic training. *Autonomic Neuroscience*, 145(1-2), 99-103.

- Miyake, A., Friedman, N. P., Emerson, M. J., Witzki, A. H., Howerter, A., & Wager, T. D. (2000). The unity and diversity of executive functions and their contributions to complex “frontal lobe” tasks: A latent variable analysis. *Cognitive psychology*, 41(1), 49-100.
- Monge Argilés JA, Palacios Ortega F, Vila Sobrino JA, Bautista Prados J, Pérez Vicente JA, Morales Ortiz A, Palao Sánchez A. Brainstem lesions decrease heart rate variability. *Neurologia*. 2000 Apr;15(4):158-63. PMID: 10846883.
- Montano, N., Porta, A., Cogliati, C., Costantino, G., Tobaldini, E., Casali, K. R., & Iellamo, F. (2009). Heart rate variability explored in the frequency domain: a tool to investigate the link between heart and behavior. *Neuroscience & Biobehavioral Reviews*, 33(2), 71-80.
- Morrison, F. J., Ponitz, C. C., & McClelland, M. M. (2010). Self-regulation and academic achievement in the transition to school.
- Mortara, A., La Rovere, M. T., Signorini, M. G., Pantaleo, P., Pinna, G., Martinelli, L., ... & Tavazzi, L. (1994). Can power spectral analysis of heart rate variability identify a high risk subgroup of congestive heart failure patients with excessive sympathetic activation? A pilot study before and after heart transplantation. *Heart*, 71(5), 422-430.
- Mosley, E., & Laborde, S. (2022). A scoping review of heart rate variability in sport and exercise psychology. *International Review of Sport and Exercise Psychology*, 1-75.
- Mosley, E., Laborde, S., & Kavanagh, E. (2018). Coping related variables, cardiac vagal activity and working memory performance under pressure. *Acta Psychologica*, 191, 179-189.
- Nabil, D., & Reguig, F. B. (2015). Ectopic beats detection and correction methods: A review. *Biomedical Signal Processing and Control*, 18, 228-244.
- Nelson, E. B., Sax, K. W., & Strakowski, S. M. (1998). Attentional performance in patients with psychotic and nonpsychotic major depression and schizophrenia. *American Journal of Psychiatry*, 155(1), 137-139.
- Nettelbeck, T. (1987). Inspection time and intelligence.

- Nikolin, S., Loo, C. K., Bai, S., Dokos, S., & Martin, D. M. (2015). Focalised stimulation using high definition transcranial direct current stimulation (HD-tDCS) to investigate declarative verbal learning and memory functioning. *Neuroimage*, 117, 11-19.
- Nikolin, S., Tan, Y. Y., Schwaab, A., Moffa, A., Loo, C. K., & Martin, D. (2021). An investigation of working memory deficits in depression using the n-back task: A systematic review and meta-analysis. *Journal of Affective Disorders*, 284, 1-8.
- Nissen, M. J., & Bullemer, P. (1987). Attentional requirements of learning: Evidence from performance measures. *Cognitive psychology*, 19(1), 1-32.
- Nolan, J., Batin, P. D., Andrews, R., Lindsay, S. J., Brooksby, P., Mullen, M., ... & Fox, K. A. (1998). Prospective study of heart rate variability and mortality in chronic heart failure: results of the United Kingdom heart failure evaluation and assessment of risk trial (UK-heart). *Circulation*, 98(15), 1510-1516.
- Nolan, J., Flapan, A. D., Capewell, S., MacDonald, T. M., Neilson, J. M., & Ewing, D. J. (1992). Decreased cardiac parasympathetic activity in chronic heart failure and its relation to left ventricular function. *Heart*, 67(6), 482-485.
- O'Connor, G. T., Buring, J. E., Yusuf, S., Goldhaber, S. Z., Olmstead, E. M., Paffenbarger Jr, R. S., & Hennekens, C. H. (1989). An overview of randomized trials of rehabilitation with exercise after myocardial infarction. *Circulation*, 80(2), 234-244.
- Okada, T., Toichi, M., & Sakihama, M. (2003). Influences of an anticholinergic antiparkinsonian drug, parkinsonism, and psychotic symptoms on cardiac autonomic function in schizophrenia. *Journal of clinical psychopharmacology*, 23(5), 441-447.
- Orlandi, G., Fanucchi, S., Strata, G., Pataleo, L., Landucci Pellegrini, L., Prontera, C., ... & Murri, L. (2000). Transient autonomic nervous system dysfunction during hyperacute stroke. *Acta Neurologica Scandinavica*, 102(5), 317-321.
- Osterrieth, P. A. (1944). Le test de copie d'une figure complexe; contribution à l'étude de la perception et de la mémoire [Test of copying a complex figure; contribution to the study of perception and memory]. *Archives de Psychologie*, 30, 206–356.

- Owen, A. M., McMillan, K. M., Laird, A. R., & Bullmore, E. (2005). N-back working memory paradigm: A meta-analysis of normative functional neuroimaging studies. *Human brain mapping, 25*(1), 46-59.
- Pagaduan, J. C., Chen, Y. S., Fell, J. W., & Xuan Wu, S. S. (2021). A preliminary systematic review and meta-analysis on the effects of heart rate variability biofeedback on heart rate variability and respiration of athletes. *Journal of Complementary and Integrative Medicine, 19*(4), 817-826.
- Pagani, M., Lombardi, F., Guzzetti, S., Rimoldi, O., Furlan, R. A. F. F. A. E. L. L. O., Pizzinelli, P., ... & Piccaluga, E. (1986). Power spectral analysis of heart rate and arterial pressure variabilities as a marker of sympatho-vagal interaction in man and conscious dog. *Circulation research, 59*(2), 178-193.
- Pagani, M., Lombardi, F., Guzzetti, S., Sandrone, G., Rimoldi, O., Malfatto, G., ... & Malliani, A. (1984). Power spectral density of heart rate variability as an index of sympatho-vagal interaction in normal and hypertensive subjects. *Journal of hypertension. Supplement: official journal of the International Society of Hypertension, 2*(3), S383-5.
- Pagani, M., Malfatto, G., Pierini, S., Casati, R., Masu, A. M., Poli, M., ... & Malliani, A. (1988). Spectral analysis of heart rate variability in the assessment of autonomic diabetic neuropathy. *Journal of the autonomic nervous system, 23*(2), 143-153.
- Paradis, M. (1998). The other side of language: Pragmatic competence. *Journal of neurolinguistics, 11*(1-2), 1-10.
- Penades, R., Catalan, R., Rubia, K., Andres, S., Salamero, M., & Gasto, C. (2007). Impaired response inhibition in obsessive compulsive disorder. *European Psychiatry, 22*(6), 404-410.
- Pennisi, P., Sarlo, M. (1998). *Indici elettrofisiologici in psicologia* (Cleup Editrice), Padova.
- Perry, W., Light, G. A., Davis, H., & Braff, D. L. (2000). Schizophrenia patients demonstrate a dissociation on declarative and non-declarative memory tests. *Schizophrenia research, 46*(2-3), 167-174.

- Pfeifer, M. A., Weinberg, C. R., Cook, D. L., Reenan, A., Halter, J. B., Ensinck, J. W., & Porte Jr, D. (1984). Autonomic neural dysfunction in recently diagnosed diabetic subjects. *Diabetes care*, 7(5), 447-453.
- Plews, D. J., Laursen, P. B., Stanley, J., Kilding, A. E., & Buchheit, M. (2013). Training adaptation and heart rate variability in elite endurance athletes: opening the door to effective monitoring. *Sports medicine*, 43, 773-781.
- Ponikowski, P., Anker, S. D., Chua, T. P., Szelemej, R., Piepoli, M., Adamopoulos, S., ... & Coats, A. J. (1997). Depressed heart rate variability as an independent predictor of death in chronic congestive heart failure secondary to ischemic or idiopathic dilated cardiomyopathy. *The American journal of cardiology*, 79(12), 1645-1650.
- Pomeranz, B., Macaulay, R. J., Caudill, M. A., Kutz, I., Adam, D., Gordon, D. A. V. I. D., ... & Cohen, R. J. (1985). Assessment of autonomic function in humans by heart rate spectral analysis. *American Journal of Physiology-Heart and Circulatory Physiology*, 248(1), H151-H153.
- Posner, M. I., & DiGirolamo, G. J. (1998). Executive attention: Conflict, target detection, and cognitive control.
- Rechlin, T., Claus, D., & Weis, M. (1994). Heart rate variability in schizophrenic patients and changes of autonomic heart rate parameters during treatment with clozapine. *Biological psychiatry*.
- Reid, M. B., & Lightfoot, J. T. (2019). The physiology of auto racing: a brief review. *Med Sci Sports Exerc*, 51(12), 2548-62.
- Reitan, R. M., & Wolfson, D. (2009). The Halstead–Reitan neuropsychological test battery for adults—theoretical, methodological, and validation bases. *Neuropsychological assessment of neuropsychiatric and neuromedical disorders*, 1, 3-24.
- Rey, A. (1941). L'examen psychologique dans les cas d'encéphalopathie traumatique.(Les problems.). *Archives de psychologie*.
- Rey, A. (1964). *L'examen clinique en psychologie*, Paris: Presses Universitaires de France, 1964. Chemotherapy and objective cognitive functioning, 95.

- Reyes del Paso, G. A., Langewitz, W., Mulder, L. J., Van Roon, A., & Duschek, S. (2013). The utility of low frequency heart rate variability as an index of sympathetic cardiac tone: a review with emphasis on a reanalysis of previous studies. *Psychophysiology*, 50(5), 477-487.
- Riggs, N. R., Spruijt-Metz, D., Sakuma, K. L., Chou, C. P., & Pentz, M. A. (2010). Executive cognitive function and food intake in children. *Journal of nutrition education and behavior*, 42(6), 398-403.
- Rodríguez-Villagra, O. A., Göthe, K., Oberauer, K., & Kliegl, R. (2013). Working memory capacity in a go/no-go task: Age differences in interference, processing speed, and attentional control. *Developmental Psychology*, 49(9), 1683.
- Rogers, M. C., Battit, G., McPeck, B., & Todd, D. (1978). Lateralization of sympathetic control of the human sinus node: ECG changes of stellate ganglion block. *Anesthesiology*, 48(2), 139-141.
- Rogers, R. D., & Monsell, S. (1995). Costs of a predictable switch between simple cognitive tasks. *Journal of experimental psychology: General*, 124(2), 207.
- Roose, S. P. (2001). Depression, anxiety, and the cardiovascular system: the psychiatrist's perspective. *Journal of Clinical Psychiatry*, 62, 19-23.
- Saari, A., Tolonen, U., Pääkkö, E., Suominen, K., Pyhtinen, J., Sotaniemi, K., & Myllylä, V. (2004). Cardiovascular autonomic dysfunction correlates with brain MRI lesion load in MS. *Clinical neurophysiology*, 115(6), 1473-1478.
- Saha, S. (2005). Role of the central nucleus of the amygdala in the control of blood pressure: descending pathways to medullary cardiovascular nuclei. *Clinical and Experimental Pharmacology and Physiology*, 32(5-6), 450-456.
- Salthouse, T. A. (1988). The role of processing resources in cognitive aging. In *Cognitive development in adulthood: Progress in cognitive development research* (pp. 185-239). New York, NY: Springer New York.
- Sánchez-Cubillo, I. 1., Periáñez, J. A., Adrover-Roig, D., Rodríguez-Sánchez, J. M., Ríos-Lago, M., Tirapu, J. E. E. A., & Barceló, F. (2009). Construct validity of the Trail Making Test: role of task-switching, working memory, inhibition/interference control, and

visuomotor abilities. *Journal of the International Neuropsychological Society*, 15(3), 438-450.

Sands, K. E., Appel, M. L., Lilly, L. S., Schoen, F. J., Mudge Jr, G. H., & Cohen, R. J. (1989). Power spectrum analysis of heart rate variability in human cardiac transplant recipients. *Circulation*, 79(1), 76-82.

Sankari & Adeli, "Heart saver: a mobile cardiac monitoring system for auto-detection of atrial fibrillation, myocardial infarction, and atrio-ventricular block," *Computers in Biology and Medicine*, vol. 41, no. 4, pp. 211–220, 2011.

Saul, J. P. (1990). Beat-to-beat variations of heart rate reflect modulation of cardiac autonomic outflow. *Physiology*, 5(1), 32-37.

Saul, J. P., Rea, R. F., Eckberg, D. L., Berger, R. D., & Cohen, R. J. (1990). Heart rate and muscle sympathetic nerve variability during reflex changes of autonomic activity. *American Journal of Physiology-Heart and Circulatory Physiology*, 258(3), H713-H721.

Saykrs, B. M. (1973). Analysis of heart rate variability. *Ergonomics*, 16(1), 17-32.

Scarpina, F., & Tagini, S. (2017). The stroop color and word test. *Frontiers in psychology*, 8, 557.

Schönauer, M., Thomas, A., Morbach, S., Niebauer, J., Schönauer, U., & Thiele, H. (2008). Cardiac autonomic diabetic neuropathy. *Diabetes and Vascular Disease Research*, 5(4), 336-344.

Schoser, B. G. H., Ricker, K., Schneider-Gold, C., Hengstenberg, C., Dürre, J., Bültmann, B., ... & Ranum, L. P. W. (2004). Sudden cardiac death in myotonic dystrophy type 2. *Neurology*, 63(12), 2402-2404.

Schwabeger, G. (1987). Heart rate, metabolic and hormonal responses to maximal psycho-emotional and physical stress in motor car racing drivers. *International archives of occupational and environmental health*, 59, 579-604.

Serrador, J. M., Finlayson, H. C., & Hughson, R. L. (1999). Physical activity is a major contributor to the ultra low frequency components of heart rate variability. *Heart*, 82(6), e9-e9.

- Shaffer, F., & Meehan, Z. M. (2020). A practical guide to resonance frequency assessment for heart rate variability biofeedback. *Frontiers in Neuroscience*, 14, 570400.
- Shannon, D. C., Carley, D. W., & Benson, H. E. R. B. E. R. T. (1987). Aging of modulation of heart rate. *American Journal of Physiology-Heart and Circulatory Physiology*, 253(4), H874-H877.
- Sherwood, A., Allen, M. T., Obrist, P. A., & Langer, A. W. (1986). Evaluation of beta-adrenergic influences on cardiovascular and metabolic adjustments to physical and psychological stress. *Psychophysiology*, 23(1), 89-104.
- Shucard, J. L., McCabe, D. C., & Szymanski, H. (2008). An event-related potential study of attention deficits in posttraumatic stress disorder during auditory and visual Go/NoGo continuous performance tasks. *Biological Psychology*, 79(2), 223-233.
- Simon, J. R., & Wolf, J. D. (1963). Choice reaction time as a function of angular stimulus-response correspondence and age. *Ergonomics*, 6(1), 99-105.
- Simon, J. R. (1990). The effects of an irrelevant directional cue on human information processing. In *Advances in psychology* (Vol. 65, pp. 31-86). North-Holland.
- Sloan, R. P., Shapiro, P. A., Bagiella, E., Bigger, J. T., Lo, E. S., & Gorman, J. M. (1996). Relationships between circulating catecholamines and low frequency heart period variability as indices of cardiac sympathetic activity during mental stress. *Psychosomatic Medicine*, 58(1), 25-31.
- Sloan, R. P., Shapiro, P. A., Bagiella, E., Boni, S. M., Paik, M., Bigger Jr, J. T., ... & Gorman, J. M. (1994). Effect of mental stress throughout the day on cardiac autonomic control. *Biological psychology*, 37(2), 89-99.
- Spyer, K. M. (1989). Neural mechanisms involved in cardiovascular control during affective behaviour. *Trends in Neurosciences*, 12(12), 506-513.
- Squire, L. R., & Zola-Morgan, J. T. (1991). The cognitive neuroscience of human memory since HM. *Annual review of neuroscience*, 34, 259-288.
- Stauss, H. M. (2003). Heart rate variability. *American Journal of Physiology-Regulatory, Integrative and Comparative Physiology*, 285(5), R927-R931.

- Stein, P. K., Kleiger, R. E., & Rottman, J. N. (1997). Differing effects of age on heart rate variability in men and women. *The American journal of cardiology*, 80(3), 302-305.
- Stroop, J. R. (1935). Studies of interference in serial verbal reactions. *Journal of experimental psychology*, 18(6), 643.
- Stuss, D. T., Levine, B., Alexander, M. P., Hong, J., Palumbo, C., Hamer, L., ... & Izukawa, D. (2000). Wisconsin Card Sorting Test performance in patients with focal frontal and posterior brain damage: effects of lesion location and test structure on separable cognitive processes. *Neuropsychologia*, 38(4), 388-402.
- Tabibi, Z., Borzabadi, H. H., Stavrinou, D., & Mashhadi, A. (2015). Predicting aberrant driving behaviour: The role of executive function. *Transportation research part F: traffic psychology and behaviour*, 34, 18-28.
- Taggart, P., & Gibbons, D. (1967). Motor-car driving and the heart rate. *British Medical Journal*, 1(5537), 411.
- Taggart, P., Gibbons, D., & Somerville, W. (1969). Some effects of motor-car driving on the normal and abnormal heart. *Br Med J*, 4(5676), 130-134.
- Talland, G. A., & Querton, G. C. (1965). The effects of methamphetamine and pentobarbital on the running memory span. *Psychopharmacologia*, 7(5), 379-382.
- Taylor, J. A., Carr, D. L., Myers, C. W., & Eckberg, D. L. (1998). Mechanisms underlying very-low-frequency RR-interval oscillations in humans. *Circulation*, 98(6), 547-555.
- Tawakal, M. I., Suryana, M. E., Noviyanto, A., Satwika, I. P., Alvissalim, M. S., Hermawan, I., ... & Jatmiko, W. (2012, December). Analysis of multi codebook GLVQ versus standard GLVQ in discriminating sleep stages. In *2012 International Conference on Advanced Computer Science and Information Systems (ICACISIS)* (pp. 197-202). IEEE.
- Ter Horst, G. J., & Postema, F. (1997). Forebrain parasympathetic control of heart activity: retrograde transneuronal viral labeling in rats. *American Journal of Physiology-Heart and Circulatory Physiology*, 273(6), H2926-H2930.

- Tesfaye, S., Boulton, A. J., Dyck, P. J., Freeman, R., Horowitz, M., Kempler, P., ... & Toronto Diabetic Neuropathy Expert Group. (2010). Diabetic neuropathies: update on definitions, diagnostic criteria, estimation of severity, and treatments. *Diabetes care*, 33(10), 2285-2293.
- Thayer, J. F., Åhs, F., Fredrikson, M., Sollers III, J. J., & Wager, T. D. (2012). A meta-analysis of heart rate variability and neuroimaging studies: implications for heart rate variability as a marker of stress and health. *Neuroscience & Biobehavioral Reviews*, 36(2), 747-756.
- Thayer, J. F., & Brosschot, J. F. (2005). Psychosomatics and psychopathology: looking up and down from the brain. *Psychoneuroendocrinology*, 30(10), 1050-1058.
- Thayer, J. F., & Friedman, B. H. (1997). The heart of anxiety: A dynamical systems approach. *The (non) expression of emotions in health and disease*, 39-49.
- Thayer, J. F., Hansen, A. L., Saus-Rose, E., & Johnsen, B. H. (2009). Heart rate variability, prefrontal neural function, and cognitive performance: the neurovisceral integration perspective on self-regulation, adaptation, and health. *Annals of behavioral medicine*, 37(2), 141-153.
- Thayer, J. F., & Lane, R. D. (2000). A model of neurovisceral integration in emotion regulation and dysregulation. *Journal of affective disorders*, 61(3), 201-216.
- Theeuwes, J. (2010). Top-down and bottom-up control of visual selection. *Acta psychologica*, 135(2), 77-99.
- Thomalla, G., Jonas, M., Bäumer, T., Siebner, H. R., Biermann-Ruben, K., Ganos, C., ... & Münchau, A. (2014). Costs of control: decreased motor cortex engagement during a Go/NoGo task in Tourette's syndrome. *Brain*, 137(1), 122-136.
- Thomson, J. A. (1983). Is continuous visual monitoring necessary in visually guided locomotion?. *Journal of Experimental Psychology: Human Perception and Performance*, 9(3), 427.
- Tiffin, J., & Asher, E. J. (1948). The Purdue Pegboard: norms and studies of reliability and validity. *Journal of Applied Psychology*, 32(3), 234-247.

- Tiwari, R., Kumar, R., Malik, S., Raj, T., & Kumar, P. (2021). Analysis of heart rate variability and implication of different factors on heart rate variability. *Current cardiology reviews*, 17(5), 74-83.
- Toichi, M., Kubota, Y., Murai, T., Kamio, Y., Sakihama, M., Toriuchi, T., ... & Miyoshi, K. (1999). The influence of psychotic states on the autonomic nervous system in schizophrenia. *International Journal of Psychophysiology*, 31(2), 147-154.
- Toledo, E., Pinhas, I., Aravot, D., Almog, Y., & Akselrod, S. (2002). Functional restitution of cardiac control in heart transplant patients. *American Journal of Physiology-Regulatory, Integrative and Comparative Physiology*, 282(3), R900-R908.
- Tombul, T., Anlar, O., Tuncer, M., Huseyinoglu, N., & Eryonucu, B. (2011). Impaired heart rate variability as a marker of cardiovascular autonomic dysfunction in multiple sclerosis. *Acta Neurologica Belgica*, 111(2), 116-120.
- Tonhajzerova, I., Ondrejka, I., Adamik, P., Hruby, R., Javorka, M., Trunkvalterova, Z., ... & Javorka, K. (2009). Changes in the cardiac autonomic regulation in children with attention deficit hyperactivity disorder (ADHD). *Indian Journal of Medical Research*, 130(1), 44.
- Tsuji, H., Larson, M. G., Venditti, F. J., Manders, E. S., Evans, J. C., Feldman, C. L., & Levy, D. (1996). Impact of reduced heart rate variability on risk for cardiac events: the Framingham Heart Study. *Circulation*, 94(11), 2850-2855.
- Tsuji, H., Venditti Jr, F. J., Manders, E. S., Evans, J. C., Larson, M. G., Feldman, C. L., & Levy, D. (1994). Reduced heart rate variability and mortality risk in an elderly cohort. The Framingham Heart Study. *Circulation*, 90(2), 878-883.
- Turkka, J. T., Tolonen, U., & Myllylä, V. V. (1987). Cardiovascular reflexes in Parkinson's disease. *European neurology*, 26(2), 104-112.
- Turner, M. L., & Engle, R. W. (1989). Is working memory capacity task dependent?. *Journal of memory and language*, 28(2), 127-154.
- Turner, A. P., & Richards, H. (2015). Physiological and selective attention demands during an international rally motor sport event. *BioMed research international*, 2015.

- Uijtdehaage, S. H., & Thayer, J. F. (2000). Accentuated antagonism in the control of human heart rate. *Clinical Autonomic Research*, 10, 107-110.
- Umetani, K., Singer, D. H., McCraty, R., & Atkinson, M. (1998). Twenty-four hour time domain heart rate variability and heart rate: relations to age and gender over nine decades. *Journal of the American College of Cardiology*, 31(3), 593-601.
- Vealey, R. S. (1994). Current status and prominent issues in sport psychology interventions. *Medicine and Science in Sports and Exercise*, 26(4), 495-502.
- Van De Borne, P., Montano, N., Pagani, M., Oren, R., & Somers, V. K. (1997). Absence of low-frequency variability of sympathetic nerve activity in severe heart failure. *Circulation*, 95(6), 1449-1454.
- van Dijk, J. G., Haan, J., Zwinderman, K., Kremer, B., Van Hilten, B. J., & Roos, R. A. (1993). Autonomic nervous system dysfunction in Parkinson's disease: relationships with age, medication, duration, and severity. *Journal of Neurology, Neurosurgery & Psychiatry*, 56(10), 1090-1095.
- Verbruggen, F., & Logan, G. D. (2008). Response inhibition in the stop-signal paradigm. *Trends in cognitive sciences*, 12(11), 418-424.
- Vybiral, T., Bryg, R. J., Maddens, M. E., & Boden, W. E. (1989). Effect of passive tilt on sympathetic and parasympathetic components of heart rate variability in normal subjects. *The American journal of cardiology*, 63(15), 1117-1120.
- Walshe, E. A., Ward McIntosh, C., Romer, D., & Winston, F. K. (2017). Executive function capacities, negative driving behavior and crashes in young drivers. *International journal of environmental research and public health*, 14(11), 1314.
- Warrington, E. K., & James, M. (1991). *The Visual Object and Space Perception Battery*. Bury St. Edmunds, UK: Thames Valley Test Company.
- Waters, G. S., & Caplan, D. (2003). The reliability and stability of verbal working memory measures. *Behavior Research Methods, Instruments, & Computers*, 35(4), 550-564.

- Watkins, E. S. (2006). The physiology and pathology of formula one Grand Prix motor racing. *Clinical neurosurgery*, 53, 145-152.
- Wawryk, A. M., Bates, D. J., & Couper, J. J. (1997). Power spectral analysis of heart rate variability in children and adolescents with IDDM. *Diabetes care*, 20(9), 1416-1421.
- Wechsler, D. (1997). Wechsler Adult Intelligence Scale--Third Edition (WAIS-III) [Database record]. APA PsycTests.
- Wen, W., Tomoi, D., Yamakawa, H., Hamasaki, S., Takakusaki, K., An, Q., ... & Asama, H. (2017). Continuous estimation of stress using physiological signals during a car race. *Psychology*, 8(07), 978.
- Wilson, B.A., Emslie, H., Foley, J., Shiel, A., Watson, P., Hawkins, K., 2005. Cambridge Prospective Memory Test (CAMPROMPT). In: Assessment, H. (Ed.), Harcourt Assessment, London.
- Wolf, M. M., Varigos, G. A., Hunt, D., & Sloman, J. G. (1978). Sinus arrhythmia in acute myocardial infarction. *Medical Journal of Australia*, 2(2), 52-53.
- Yamakoshi, T., Matsumura, K., Yamakoshi, Y., Hirose, H., & Rolfe, P. (2010). Physiological measurements and analyses in motor sports: a preliminary study in racing kart athletes. *European Journal of Sport Science*, 10(6), 397-406.
- Yanagida, R., Takahashi, K., Miura, M., Nomura, M., Ogawa, Y., Aoki, K., & Iwasaki, K. I. (2016). Speed ratio but cabin temperature positively correlated with increased heart rates among professional drivers during car races. *Environmental health and preventive medicine*, 21(6), 439-445.
- Yang, A. C., Hong, C. J., & Tsai, S. J. (2010). Heart rate variability in psychiatric disorders. *Taiwanese Journal of Psychiatry (Taipei)*, 24(2), 99-109.
- Yeragani, V. K., Pohl, R., Balon, R., Ramesh, C., Glitz, D., Jung, I., & Sherwood, P. (1991). Heart rate variability in patients with major depression. *Psychiatry research*, 37(1), 35-46.

Yeragani, V. K., Pohl, R., Jampala, V. C., Balon, R., Ramesh, C., & Srinivasan, K. (2000). Increased QT variability in patients with panic disorder and depression. *Psychiatry research*, 93(3), 225-235.

Yotsukura, M., Fujii, K., Katayama, A., Tomono, Y., Ando, H., Sakata, K., ... & Ishikawa, K. (1998). Nine-year follow-up study of heart rate variability in patients with Duchenne-type progressive muscular dystrophy. *American heart journal*, 136(2), 289-296.

Yotsukura, M., Sasaki, K., Kachi, E., Sasaki, A., Ishihara, T., & Ishikawa, K. (1995). Circadian rhythm and variability of heart rate in Duchenne-type progressive muscular dystrophy. *The American journal of cardiology*, 76(12), 947-951.

Yu, F., Ye, R., Sun, S., Carretie, L., Zhang, L., Dong, Y., ... & Wang, K. (2014). Dissociation of neural substrates of response inhibition to negative information between implicit and explicit facial Go/Nogo tasks: evidence from an electrophysiological study. *PLoS One*, 9(10), e109839.

Zarahn, E., Aguirre, G., & D'Esposito, M. (1997). A trial-based experimental design for fMRI. *Neuroimage*, 6(2), 122-138.