

THE EFFECTS OF NOSE-BREATHING-ONLY TRAINING ON PHYSIOLOGICAL  
PARAMETERS RELATED TO RUNNING PERFORMANCE: A CASE STUDY

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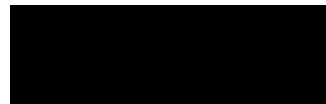
THE EFFECTS OF NOSE-BREATHING-ONLY TRAINING ON PHYSIOLOGICAL PARAMETERS  
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## Abstract

**Background:** At rest, nose breathing has been proposed to be superior to mouth breathing due to the warming, humidifying, and filtering capacities of the nose and related structures. Little is known about the capacity for humans to practice nose breathing during physical activity.

**Objective:** The effects of a 6-week nose breathing running program on running performance, breath hold endurance, resting and exercising physiological variables, and the subjective experience, were investigated in a trained runner previously unaccustomed to nose breathing.

**Methods:** A single (n=1) male participant completed a 6-week running training program, during which all running efforts were completed while the participant breathed only through the nose. Objective measures included: Breath hold tests (*BHT*), a graded exercise test to determine the highest intensity of running where a voluntary switching point between nose breathing and mouth breathing occurred (*GXT*), and regular breathing five-kilometre time trials (*5kmTT*) were conducted before, during, and after the 6-week running program. A daily journal described the subjective experience of each daily run, interval workout, and testing event which specifically looked to detail any dyspnea episodes which occurred.

**Results:** Subjectively, the participant experienced frequent intense dyspnea in the early stages of the running program. Running pace during the first week of nose breathing (NB1) was significantly slower than the mouth breathing (MB) ( $M=259.75\pm 2.72$  vs.  $M=245.50\pm 1.49$  s $\cdot$ km $^{-1}$ , respectively.  $p=0.015$ ). Pace was not significantly different from the MB pace in the following weeks except for nose breathing week 6 (NB6) which was significantly faster than MB ( $M=228.00\pm 5.51$  vs.  $M=245.50\pm 1.49$  s $\cdot$ km $^{-1}$ , respectively.  $p=0.041$ ). Heart rate showed no significant changes at any point during the running program. The *BHT* improved by 34.1 s or 77% between PRE and POST. The running speed during the *GXT* when the participant could no longer sustain nose breathing, increased by 23% between the PRE and MID testing and improved a further 1% between MID and POST. The *5kmTT* showed a 77 s or 6.21% improvement between the PRE and POST running trials. **Conclusion:** In this case study, it was demonstrated that undertaking a running training program while nose breathing only, is feasible for a trained runner, and an effective method of training that yields improvement in 5km time-trial performance similar to reported results in the literature that did not restrict mode of breathing.

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## Abbreviations

### Timeline:

MB	Mouth breathing / mouth breathing phase
NB	Nose breathing
NB1	Nose breathing phase, first half (weeks 1-3)
NB2	Nose breathing phase, second half (weeks 4-6)
Week 1-6	Individual nose breathing weeks 1-6
PRE	Pre-nose breathing tests (10 February 2021)
MID	Mid-nose breathing tests (8 March 2021)
POST	Post-nose breathing tests (30 March 2021)

### Tests:

5kmTT	Five-kilometer time trial
BHT	Breath hold tests
GXT	Voluntary switching point graded exercise test

### Other terms:

CO <sub>2</sub>	Carbon dioxide
EIB	Exercise induced bronchospasms
HR	Heart rate
HRV	Heart rate variability
IBM's	Involuntary breathing movements
NO	Nitric Oxide
O <sub>2</sub>	Oxygen
POMS	Profile of Mood States

RPE	Rating of perceived exertion
RR	Respiratory rate
$\dot{V}O_2$	Oxygen consumption
$\dot{V}O_{2peak}$	Peak oxygen consumption
$\dot{V}_t$	Tidal volume

## **Chapter 1: Introduction and Background**

Breathing involves the movement of atmospheric air into and out of the body through a series of conducting airways which join the alveoli, the gas exchange region of the lungs, and the exterior environment (Kenney et al., 2015). There are two openings to the upper airway of the human respiratory system. At rest, the nose is the primary opening during breathing, drawing air into the nostrils. The air travels through the nasal cavity consisting of the three nasal conchae. The nasal conchae are boney protuberances that cause inhaled air to be swirled and slowed as it passes through the nasal cavity before passing into the lower airways. Alternatively, if the nose becomes obstructed, the oral cavity allows for an alternate route for air to enter and exit the respiratory system. The oral cavity allows air to flow directly into the lower airways via the oropharynx.

It has been widely claimed, although with varying scientific support, that nose breathing is healthier than mouth breathing due to a variety of structural and physiological mechanisms (Nestor, 2020). The nose breathing movement has gained traction recently in popular culture with James Nestor's book "Breath: the new science of a lost art", released in 2020. Nestor (2020) presents a compelling argument for the use of nose breathing and other breathing techniques sourcing a variety of historical, scientific, and some pseudoscientific claims. The main point of the book is focused on how nose breathing, and slow breathing can improve one's health by helping to open the modern human's airways, which he claims has become obstructed since human urbanization for a variety of reasons (Nestor 2020). He further claims that nose breathing may aid the treatment of many modern maladies such as sleep apnea, asthma, and emphysema; and he claims that nose breathing may improve sport performance (Nestor 2020).

From a historical point of view, Nestor argues that modern humans' airways have become more crowded due to chronic mouth breathing and the introduction of much softer foods into the human diet. In support of this, Nestor (2020) refers to Dr Marianne Evans, a board-certified dual specialist in orthodontics and periodontics who indicated to him that the skulls of ancient humans had much larger choanae, the openings from the nasal cavity into the nasopharynx, as well as more pronounced and wider jaws which allowed for easy breathing and may have prevented some modern maladies such as snoring and sleep apnea. George Catlin (1862) further describes the differences between industrial-based and non-industrialized societies in his book "The Breath of Life" in which he highlights the importance of nose breathing for health, something he learned during his travels to over fifty plains-based Indigenous North American communities. Nestor then refers to a study conducted by Harvold et al. (1973) in which the nasal cavities of monkeys were intentionally blocked for up to two years. Harvold et al. (1973) found that the monkeys began to exhibit signs such as a narrowing dental arch and a gaping mouth which is common among the modern human population. Nestor (2020) marks this as supportive evidence for using nose breathing and notes that in fact, most older and ancient cultures highlight the importance of nose, or slow, breathing. For example, there are seven books of the Chinese Tao dating back to around 400 BCE focusing on breathing, with many other cultures incorporating slow breathing through methods such as meditation or through the cadence of prayers (Huang & Wurmbrand, 1987).

From a scientific standpoint, nose breathing may show health benefits due to the warming and humidifying of the inspired air as it passes through the nasal passages and paranasal sinuses (Naftali et al. 2005; Borojeni et al. 2020). In addition to this, by breathing through the nose, air is filtered via the nose hairs and multitude of mucosal and ciliated cells located throughout the

nasopharynx which trap foreign particles before they reach the lungs (Moore, Agur, & Dalley, 2015). Finally, by nose breathing we have the opportunity to inhale nitric oxide (NO), a vasodilator which may allow greater blood flow to the capillaries of the alveoli promoting easier gas exchange in the lungs (Klinger & Kadowitz, 2017); up to eighteen percent more oxygen per breath according to an unsupported claim by Nestor (2020). With the possible exception of the exact percentage of oxygen absorption increase, these are not revelations unique to James Nestor and are easily verifiable by any anatomy or physiology textbook (Marieb & Hoehn, 2013; Tortora & Derrickson, 2014; Kenney et al, 2015; Moore, Agur, & Dalley, 2015).

What is somewhat less substantiated in the literature are the claims that nose breathing, and other breathing techniques, can have restorative healing attributes to diseases such as asthma, and emphysema, as claimed in “Breath” (Nestor 2020). Nestor (2020) also claims that nose breathing can alter the oxygen exchange rate within the brain, specifically to the prefrontal cortex which may have effects on the prevalence of attention-deficit/hyperactivity disorder (ADHD) within mouth breathers citing Sano et al. (2013) who found that oxygen load was greater in the prefrontal cortex while mouth breathing compared to nose breathing. Sano et al. (2013) based their study on the assumption that there is a higher prevalence of ADHD amongst mouth breathers, citing Jefferson (2010). Jefferson (2010) in fact, does not support this claim and instead indicated that many mouth breathing children are actually mis-diagnosed with ADHD. Jefferson (2010) explains that many of these mouth breathing children experience behavior issues due to sleeping problems originating from mouth breathing which is mistaken for ADHD. Nestor, who conducted a mouth-then-nose restricted breathing experiment on himself, interestingly highlighted the large negative effects that mouth breathing, and the subsequent sleep issues that he reportedly experienced, were having on Nestor’s mood (Nestor, 2020).

Nestor (2020) also indicates that mouth breathing may have some detrimental effects on intelligence claiming that during his own experiments with mouth breathing only that “Mouth breathing was also making [him] dumber.” (Nestor 2020), however, he does not support this claim with any data and instead refers to a single study conducted on oral breathing restricted rats (Tsubamoto-Sano et al., 2019). Elsewhere in his book, James Nestor (2020) suggests other health benefits may accompany nose breathing which are beyond the scope of this thesis.

From an exercise physiology standpoint, what is interesting are Nestor’s claims, scattered throughout “Breath” (Nestor 2020), that nose breathing may improve athletic performance compared to mouth breathing. Specifically, Nestor (2020) cites several studies performed by Dr. Douillard in which nasal and oral breathing were compared during an incremental exercise test on a stationary bike. In his book, Nestor (2020) refers to Dr. Douillard’s works and indicates that one subject was able to maintain the same heart rate despite a tenfold increase in exercise intensity however, this particular claim by Nestor is unsubstantiated by Dr. Douillard’s published works (2014). In a case study conducted on Dr. Douillard himself, followed by a pilot study (n=3), and a final study conducted on 5 male high school athletes he does corroborate the claim that heart rate showed no difference between breathing conditions, with nose breathing showing a significant decrease in respiratory rate and perceived exertion, and an increase in endurance time (Travis et al., 1996; Douillard, 2014). The final study by Travis et al. (1996) used two counter-balanced exercise tests, one using a ‘conventional workout’ compared to an ‘Invincible Athletics’ workout, which were both followed by a cycling endurance test. It is important to note that the nose breathing aspect of these studies was just one component to the ‘Invincible Athletics’ program, a training program designed to avoid the stress/recovery cycle, with the performance effects of the whole program being the outcome measures versus a single regular

breathing stress/recovery-style workout (Travis et al. 1996, Douillard 2014). Nestor himself reported that he was able to pedal almost a mile (1.6km) farther while nose breathing compared to mouth breathing by his fifth nose breathing cycling trial (Nestor 2020). While this may be interpreted purely anecdotally, not to mention that there was no baseline fitness established and as Nestor does not claim to be even a recreationally fit athlete, there was likely a large training effect present. Despite this, throughout his book Nestor (2020) does present a compelling argument for the potential performance benefits of nasal breathing training.

The athletic potential of nose breathing has been shown by Dallam et al (2018) who found that recreationally fit runners who had self-selected nose-breathing-only training for at least six months (average of 5.75yrs and 3.25yrs for males and females, respectively) were able to maintain the same peak pulmonary oxygen consumption ( $\dot{V}O_{2peak}$ ), peak blood lactate concentration, and time to exhaustion while nose breathing as with mouth breathing. Not only did the runners show similar values with nose breathing as with mouth breathing in an exhaustive protocol, but it was also found by Dallam et al. (2018) that the runners exhibited a significantly ( $p < 0.05$ ) lower oxygen consumption during a 6-minute steady state run at 85% of their respective  $\dot{V}O_{2peak}$ , while nose breathing versus mouth breathing, with a large effect size. This is further substantiated by the findings that the ventilatory efficiency improved, defined as the lowered ratio of the ventilatory equivalent to the pulmonary oxygen consumption,  $\dot{V}_E / \dot{V}O_2$ , in the nose breathing trial compared to mouth breathing, also with a large effect size (Dallam et al., 2018). An improved ventilatory efficiency has been identified as one of the main physiological factors which contribute to running performance as those with a higher ventilatory efficiency can extract a greater amount of oxygen, that can be utilized for aerobic exercise, for a given amount of inhaled air compared to those with a lower ventilatory efficiency. Ventilatory efficiency,

along with a high  $\dot{V}O_2$  peak, and a high lactate threshold have been identified as the three major physiological components of good distance running performance (Morgan et al., 1989; Fletcher & MacIntosh, 2017).

Despite the observation of a lowered  $\dot{V}_E / \dot{V}O_2$ , there was no indication in the results of Dallam et al. (2018) that there were any running performance improvements due to the nose breathing training, only that these participants were able to run at the same pace while nose breathing as with mouth breathing. Additionally, the obtained results may not reflect the true nose breathing capacity of these runners due to the need to use nasal dilator strips to offset the perceived or actual nasal passage compression caused by the  $\dot{V}O_2$  mask used during the laboratory testing (Dallam et al., 2018). The reduced area for air to pass through in the nose versus the mouth is the main reason for the prevalent use of oral, or mouth breathing (MB) during exercise rather than nose or nasal breathing (NB). Furthermore, Dallam et al. (2018) did not report on the perceptual experiences during the initial stages (first 6 months) of the NB training. Dallam et al. (2018) instead report only that “those not adapted to nasally restricted breathing during exercise experience an unacceptable sensation of air hunger at some level of intensity, causing them to switch over to an oral breathing pattern at a relatively low ventilation rate.”, possibly indicating that this initial experience is highly variable between participants. Additionally, they did not indicate the type of training employed by these participants as they adapted to NB while running. Hostetter et al. (2017) described that a 53-year-old male competitive triathlete had gradually adapted to NB over the course of 6 months in which he used NB in all intensities of his training and after which, was able to run a 5:32 mile (reportedly the best time he had run in the previous decade). Ultimately, the results of Dallam et al. (2018) and Hostetter et al. (2017) were that after prolonged (>6-months) NB training, participants were able

to run at the same maximal pace while completing a nose breathing maximal graded exercise test as with mouth breathing and showed no differences in heart rate and a lesser oxygen consumption while nose breathing during a 6-minute steady state run at 85% of their maximally achieved speed.

The purpose of my study therefore was to determine if it was possible to complete a competitive nose-breathing running training program being previously unaccustomed to nose breathing while running. Furthermore, I wished to investigate the potential physiological changes and performance impacts caused by the nose-breathing running training program. Finally, my goal during the daily runs was to gain insight into, and describe in detail, the subjective dyspnea experience brought on by moderate-vigorous intensity running while strictly nose breathing over the course of the 6-week program, having no prior experience with nasally restricted running.

Ultimately, while the COVID-19 pandemic during 2020-2021 in North Bay, Ontario, Canada restricted the ability to conduct laboratory testing and therefore the ability to expand on the work of Dallam et al. (2018), it presented a uniquely pragmatic research opportunity to conduct an exploratory case study on myself. Notably, this thesis attempts to respond to the questions left unanswered by Dallam et al. (2018) and to better understand the performance effects and subjective experiences of someone who is undertaking a NB training program.

## **Chapter 2: Literature Review**

### **1. The respiratory system anatomy**

#### **The upper respiratory system**

The upper respiratory consists of two conducting pathways to move air from the atmosphere to the lower respiratory system. The primary pathway for the movement of atmospheric air into and out of the lungs during light daily activity and resting conditions is the nose and nasal cavities (Tortora & Derrickson, 2014). As air passes through the nasal cavities, it reaches the nasopharynx, the most superior of the three regions of the pharynx, the air then passes through the oropharynx and the laryngopharynx prior to reaching the larynx (Tortora & Derrickson, 2014). Alternatively, mouth breathing can serve as a secondary pathway for air flow should the nose become obstructed or in conditions of fatigue in which airflow through the nose becomes insufficient (Pohunek, 2004). The mouth bypasses the nose, nasal cavity, and nasopharynx and connects directly with the oropharynx continuing downward toward the larynx and the other conducting pathways of the lower respiratory system.

The oropharynx and laryngopharynx are unique in that they serve as a passage for both air for the respiratory system, and food for the digestive system (Tortora & Derrickson, 2014). As such, the oro- and laryngopharynxes are lined with non-keratinized stratified squamous epithelium which provides protection from abrasion caused by ingested food particles (Tortora & Derrickson, 2014). At the inferior portion of the laryngopharynx, the respiratory and digestive systems diverge into the larynx and the esophagus, respectively. The larynx marks the structural separation between the upper and lower sections of the respiratory system (Tortora & Derrickson, 2014).

### **The mouth and related airways**

The oral region of the human body includes the oral cavity, teeth, gingivae, tongue, palate, and the palatine tonsil region (Moore, Agur, & Dalley, 2015). The oral cavity itself is divided into the oral vestibule and the oral cavity proper (Moore, Agur, & Dalley, 2015). The oral vestibule consists of the space between the lips and cheeks, superficially, and the teeth and gingivae, deeply. The oral cavity proper is the space posterior and medial to the dental arches of the maxilla and mandible (Moore, Agur, & Dalley, 2015).

The lips contain the orbicularis oris, the muscle that surrounds the opening of the mouth, known as the oral fissure, and along with the superior and inferior labia muscles allow for motility of the lips (Moore, Agur, & Dalley, 2015). The lips are covered externally by skin and covered internally by a mucus membrane; meeting at the vermillion border, a transition zone which is marked by the abrupt colour change to the red of the lips (Moore, Agur, & Dalley, 2015). The cheeks are continuous with the lips, are distensible, and form the lateral wall of the oral cavity. The teeth are rooted in the alveolar processes of the maxilla bone (upper jaw) and the mandible bone (lower jaw) and are surrounded by the gingivae which is composed of fibrous tissue covered in a mucous membrane and attaches to the alveolar processes of the maxilla and mandible bones (Tortora & Dickerson, 2014, & Moore, Agur, & Dalley, 2015).

The oral cavity proper is formed laterally and anteriorly by the dental arches of the maxilla and mandible, the roof of the mouth is formed by the palate, and the floor of the mouth is composed of fibrous tissue and muscle, and is covered in a mucus membrane (Moore, Agur, & Dalley, 2015). The palate is divided into the anterior hard palate and the more posterior soft palate which forms both the roof of the mouth and the floor of the nasal cavities. The hard palate is formed primarily by the palatine process of the maxilla as well as the paired, L-shaped palatine

bones which form the posterior portion of the hard palate, part of the floor, and the lateral walls of the nasal cavities (Tortora & Dickerson, 2014). The soft palate forms the posterior third of the palate and extends posteriorly and inferiorly from the posterior border of the hard palate, ending in the conical process known as the uvula (Moore, Agur, & Dalley, 2015). The floor of the mouth is occupied for the most part by the tongue, a versatile organ composed mostly of muscle which aids in manipulation of food during mastication and in the pronunciation of consonants during speech (Marieb & Hoehn, 2013). The tongue occupies most of the free space within the oral cavity and is anchored to the floor of the mouth by the lingual frenulum (Marieb & Hoehn, 2013).

The oral cavity communicates with the oropharynx through the fauces, the arched opening at the posterior of the oral cavity whose size is regulated by the posterior dorsum of the tongue and the inferior most portions of the soft palate (Tortora & Derrickson, 2014). The tissues of the mouth are lined by thick squamous epithelium and the tissues of the gingivae, hard palate, and the upper (dorsal) side of the tongue are slightly keratinized in order to increase resilience during mastication (Marieb & Hoehn, 2013). The oral mucosa is kept moistened primarily through the secretion of saliva via several, paired, extrinsic salivary glands (Marieb & Hoehn, 2013). The parotid, sublingual, and submandibular are the major salivary glands, all of whom are located outside of the oral cavity and empty their secretions into the oral cavity via duct systems (Marieb & Hoehn, 2013). Saliva is composed mostly of water (~97%), and contains some electrolyte solutes as well as antimicrobial compounds such as IgA antibodies, lysosomes, and defensins which act on ingested or inhaled microorganisms (Marieb & Hoehn, 2013). Additionally, the salivary defense of microorganisms is aided by the paired palatine tonsils, located in the tonsillar sinus formed by the palatoglossal and palatopharyngeal arches which

laterally connect the tongue, soft palate, and pharynx (Moore, Agur, & Dalley, 2015). The paired lingual tonsils also aid in the entrapment and elimination of foreign microorganisms and is located at the base of the tongue (Tortora & Derrickson, 2014). The tonsils are masses of lymphoid tissue that is not surrounded by a capsule, known as lymphatic nodules, and are positioned to ideally participate in an immune response to inhaled or ingested foreign substances.

When considering oral breathing, the mouth-oropharynx pathway is generally considered to be a low resistance pathway (Amis et al., 1999). While the configuration of structures within the mouth, such as the tongue, can cause turbulent airflow during inhalation, the turbulence intensity decreases once entering the expansive volume of the oropharynx and the laryngopharynx (Lin et al., 2007). This low resistance pathway allows for higher ventilation rates compared to the alternative nasal pathway, as air is easily conducted through the wide opening of the mouth and oral cavity. The mouth-oropharynx pathway is limited primarily by the opening of the glottis, causing turbulent flows only at higher ventilation rates (Islam et al., 2020). In healthy European men and woman (ages 20-29), it has been shown that peak ventilation rates are roughly  $197.2 \pm 40.2$  and  $126.3 \pm 24.4$  L/min, respectively, with peak ventilation decreasing approximately 7% with each subsequent decade of age (Loe et al., 2014).

### **The nose and related airways**

The nose is the superior most part of the respiratory tract; it is superior to the hard palate and contains the organ of smell, the olfactory bulb (Moore, Agur, & Dalley, 2015). The nose consists of the external nose and the nasal cavities, both of which are divided into a right and left cavity by the nasal septum cartilage (Moore, Agur, & Dalley, 2015). The external nose is composed mostly of cartilage and is built from a skeletal framework consisting of the nasal bone (forming the bridge of the nose) and frontal bones (forming the root of the nose) superiorly, and

the maxillary bones laterally (Marieb & Hoehn, 2013). The cartilaginous part of the nose consists of five major pieces of hyaline cartilage: two lateral cartilages which extend the bridge of the nose, two U-shaped alar cartilages which form the apex of the nose as well as contribute to the ala of the nose, and the septal cartilage which divides both the external nose and nasal cavities into a right and left (Marieb & Hoehn, 2013. & Moore, Agur, & Dalley, 2015). The opening of the external nose are the nostrils, or nares, which are bordered medially by the nasal septum, and superiorly and laterally by the alar cartilages and the dense fibrous connective tissue, together forming the ala of the nose (Marieb & Hoehn, 2013).

The size and shape of the external nose can vary widely among humans due, mostly, to differences in the nasal cartilages (Moore, Agur, & Dalley, 2015). These differences have been shown to vary widely within humans due not only to genetic drift but also due to evolutionary adaptation to temperature and humidity (Zaidi et al., 2017). Zaidi et al. (2017) have shown that between populations of geographically diverse populations (West Africa, Northern Europe, & Southeast Asia) that alar width and alar base width are significantly different, and different beyond what is expected from natural genetic divergence. The differences in nares width were found to be significantly correlated to temperature and absolute humidity, with warm-humid climates correlating with wider nares and cool-dry climates correlating with narrower nares.

The nasal cavity is posterior to the external nose. Medially, the nasal septum is maintained in the anterior portion by the septal hyaline cartilage. Moving further posterior however this septum transitions into a bony separation formed inferiorly by the Vomer, and superiorly by the perpendicular plate of the ethmoid bone (Tortora & Derrickson, 2014). Laterally, the walls of the nasal cavities are formed from the maxillae, anteriorly, and the vertical portion of the palatine bones, posteriorly. The floor of the nasal cavity is formed by the hard

plate consisting of the palatine process of the maxillae and the horizontal plates of the palatine bones (Tortora & Derrickson, 2014). Superiorly, the roof and superior sidewalls of the nasal cavity are formed from the cribriform plate and lateral masses of the ethmoid bone, respectively (Tortora & Derrickson, 2014). The olfactory nerve passes through the olfactory foramina of the cribriform plate, resultingly dividing the nasal cavity into the olfactory region in the superior third, and the respiratory region in the inferior two thirds of the nasal cavity (Tortora & Derrickson, 2014. & Moore, Agur, & Dalley, 2015). Each of the lateral masses of the ethmoid bone contain two thin, scroll shaped projections which form the superior and middle conchae, or turbinates (Tortora & Derrickson, 2014). A third pair of conchae, the inferior conchae, are formed from separate bones, known as the turbinate bones. The three sets of conchae result in four air passages through the nasal cavity: the sphenop-ethmoidal recess, superior nasal meatus, middle nasal meatus, and inferior nasal meatus (Moore, Agur, & Dalley, 2015). Functionally, the conchae act to enhance air turbulence within the nasal cavity and increase the surface area of respiratory mucosa that is exposed to air (Marieb & Hoehn, 2013).

The nasal cavity communicates with the nasopharynx posteriorly through the internal naris, or choana, which is formed from the anterior body of the sphenoid bone (located posterior to the ethmoid bone), superiorly, and the horizontal plate of the palatine bones, inferiorly (Tortora & Derrickson, 2014). With the exception of the nasal vestibules, which is lined with skin containing long nasal hairs, the structures of the nasal cavity are lined by the nasal mucosa (Tortora & Derrickson, 2014). The nasal mucosa is bound to the periosteum and perichondrium of the supporting bones and cartilages of the nose, respectively (Moore, Agur, & Dalley, 2015). The nasal mucosa is an epithelium composed of pseudostratified ciliated columnar epithelium, cells and contains a high quantity of mucus producing goblet cells (Tortora & Derrickson, 2014).

This mucosa rests on a lamina propria which is heavily supplied with seromucous nasal glands; mucous secreting glands which produce each day approximately a litre of mucous and a watery fluid containing lysozyme, an antimicrobial enzyme (Marieb & Hoehn, 2013). The respiratory mucosa lines most of the nasal cavity with the exception of the slit-like superior region of the nasal cavity. This region is the olfactory mucosa and is distinctive from the rest of the nasal mucosa due to the presence of olfactory receptors, extended by the olfactory nerve (CN I) through the cribriform plate of the ethmoid bone (Marieb & Hoehn, 2013. & Hazeri et al. 2021). There is some debate within the literature as to the actual size of the olfactory region of the nose with a reported surface area range of 1-10 cm<sup>2</sup> per nasal passage (Hazeri et al. 2021). For comparison, the average nasal cavity surface area measured in 47 healthy adults was found to be 189±23 cm<sup>2</sup> (Borojeni et al. 2020).

Surrounding the nasal cavity is a network of paranasal sinuses located in, and named after, the frontal, sphenoid, ethmoid, and maxillary bones of the face and cranium (Marieb & Hoehn, 2013). The paranasal sinuses are lined continuously with the nasal mucosa of the nasal cavities and the secretions of the sinuses drain into the lateral walls of the nasal cavity (Tortora & Derrickson, 2014). While continuous with the nasal mucosa, enzymatic differences exist within the mucosa of the paranasal sinuses and the nasal cavities with a substantial presence of the enzyme nitric oxide synthase (NOS) within the mucosa of the paranasal sinuses (Lundberg, 2008).

The frontal sinuses are located between the inner and outer tables of the frontal bone and housed posteriorly to the superciliary arches and root of the nose (Moore, Agur, & Dalley, 2015). The frontal sinuses drain via the frontonasal duct into the ethmoidal infundibulum (Moore, Agur, & Dalley, 2015). The ethmoidal sinuses are composed of several sinus cavities located in the

lateral mass of the ethmoid bone and are divided based on their drainage into the anterior, middle, and posterior cells (Moore, Agur, & Dalley, 2015). The anterior ethmoidal sinuses drain both directly and indirectly into the ethmoidal infundibulum, which drains into the middle meatus of the nasal cavity along with the frontal sinus via the semilunar hiatus of the middle meatus (Moore, Agur, & Dalley, 2015). The middle ethmoidal sinuses also empty directly into the middle meatus while the posterior ethmoidal cells open directly into the superior meatus of the nasal cavity. The sphenoid sinuses are located within the body of the sphenoid bone and drain into the posterior portion of the sphenoid-ethmoidal recess, superior to the superior meatus (Tortora & Derrickson, 2014. & Moore, Agur, & Dalley, 2015). The most inferior sinuses, the maxillary sinuses, are the largest of the paranasal sinuses as they occupy the bodies of the maxillae (Moore, Agur, & Dalley, 2015). The maxillary sinuses drain into the maxillary ostium which drains into the middle meatus at the posterior end of the semilunar hiatus (Moore, Agur, & Dalley, 2015). The nasolacrimal duct, otherwise known as the tear duct, empties excess fluids from the eyes into the inferior meatus (Tortora & Derrickson, 2014). The orifice of the eustachian (auditory) tube is located posterior to the nasal cavity in the nasopharynx, this connection allows for the exchange of air with the auditory tube in order to equalize pressure between the middle ear and the atmosphere (Tortora & Derrickson, 2014).

During a constant flow rate inhalation, the region with highest velocity observed is at the external nares; due to the narrow opening of the nose, found to have a unilateral cross-sectional area of  $0.66 \pm 0.21 \text{ cm}^2$ , creating a larger resistance and a resultant increase in velocity (Kelly et al., 2000; & Borojeni et al., 2020). Additionally, Kelly et al. (2000) observed that inspired air had the highest velocity in the inferior airways with relatively low flow rates in the superior and olfactory regions of the nose. Air flow velocity was also found to be greater in the medial

regions, with the highest values occurring near the nasal septum and lower values being observed in the lateral meatuses formed by the conchae (Kelly et al., 2000). This data by Kelly et al. (2000) disagrees slightly with that collected by Borojeni et al. (2020), who found that the middle region of the nasal cavity was responsible for  $51 \pm 12\%$  of the total air flow with the inferior region comprising  $37 \pm 17\%$  and the superior region taking only  $12 \pm 8\%$ . These discrepancies may be due to differences in the partitioning of the nasal airflow between studies as well as the large variability of the nose and nasal cavity between and within the populations. The literature does show agreement in that the vast majority of nasal airflow passes through the inferior two thirds of the nasal cavity, moving most quickly through the medial regions close to the septum with only a small percentage of air flow passing through the superior and olfactory regions (Kelly et al., 2000; Borojeni et al., 2020). When nose breathing, the nasal cavity and its structures cause the majority of resistance within the airways, constituting approximately 70% of the total airway resistance in the respiratory system (Islam et al., 2020).

The structures of the nasal cavity are highly vascularized by a combination of the lateral nasal branches of the facial artery, the anterior and posterior ethmoidal arteries, and the sphenopalatine artery (Moore, Agur, & Dalley, 2015). The presence of this highly vascularized tissue allows for the large surface area of the nasal cavities, which is greatly increased by the nasal conchae, to warm and humidify inspired air prior to its arrival to the lower airways (Moore, Agur, & Dalley, 2015). Borojeni et al. (2020) found that normative values for the unilateral heat exchange between inspired air and the nasal mucosa ranged from  $94\text{-}281 \text{ W}\cdot\text{m}^2$  between the external and internal nares. Additionally, the unilateral surface area undergoing a minimum heat exchange of  $50 \text{ W}\cdot\text{m}^2$  was found to range from  $27.4\text{-}64.3 \text{ cm}^2$  in 47 healthy adults (Borojeni et al., 2020). This heat exchange, coupled with the humidifying ability of the nasal mucosa's

approximate 1 L per day of mucous and fluid supplied by the various paranasal sinuses and nasolacrimal openings within the meatuses, allow for the inspired air to be heated and humidified to approximately 90% of the alveolar conditions (body temperature and fully saturated) within the nasal cavities (Naftali et al., 2005). Furthermore, Naftali et al. (2005) identified the main regions of heat exchange to be the nasal septum (25%), the inferior lateral walls (20%) and the inferior and middle turbinates (approximately 15% each). At a tidal volume ( $V_T$ ) of 0.41 L and a rate of 15 breaths per minute ( $\text{breaths}\cdot\text{min}^{-1}$ ), these values hold true in a variety of environmental conditions with the nasal cavity allowing for the heating and humidification of inspired air to within approximately 90% of the alveolar conditions prior to reaching the pharynx (Naftali et al. 2005). However, a moderate increase in breathing effort, achieved by doubling the tidal volume ( $V_T=0.82$  L,  $15 \text{ breaths}\cdot\text{min}^{-1}$ ), resulted in a significant (approximately 10%) decrease in both heat exchange and humidification capacity of the nasal cavity (Naftali et al. 2005). The data from Naftali et al. (2005) indicate that a ventilation rate of only  $12.3 \text{ L}\cdot\text{min}^{-1}$  resulted in a 10% reduction in cooling capacity, and it is unclear in the literature as to the impairment of the temperature/humidity capabilities of the nasal cavity as ventilation rates approach the upper limit of  $52.5\pm 14.2 \text{ L}\cdot\text{min}^{-1}$  indicated by Lacombe et al. (2017) as the approximate switching point between NB and MB.

### **The lower respiratory system**

The lower airways begin at the larynx, known commonly as the voice box; a short passageway which connects the laryngopharynx with the trachea (Tortora & Derrickson, 2014). The larynx is walled by nine pieces of cartilage, with the epiglottis, a leaf-like partially unattached piece of cartilage forming a lid over the trachea during swallowing and the rest forming the structural aspects of the larynx walls and the vestibular & vocal folds. The mucous

membrane of the larynx forms two folds; the superior of which being the vestibular, or false vocal cords, and the inferior folds form the true vocal cords (Tortora & Derrickson, 2014). The vestibular folds, while not used for voice/sound production are functionally useful to close the rima vestibuli, the space between the folds, during the Valsalva maneuver in which pressure is created within the thoracic cavity as one might do during a strenuous bowel movement or when lifting a heavy object (Tortora & Derrickson, 2014). The vocal folds are the primary structures involved in voice production, the space between the vocal folds is known as the rima glottidis (Tortora & Derrickson, 2014). The intrinsic laryngeal muscles connect to both the vocal ligaments and the rigid cartilages of the larynx. When the intrinsic laryngeal muscles contract, the vocal ligaments are stretched tight, and the vocal folds move into the airways and narrow the rima glottides (Tortora & Derrickson, 2014). When air passes through the larynx it vibrates the vocal cords are produces sound, with greater tension increasing pitch and increased air pressure increasing volume (Tortora & Derrickson, 2014). Under normal breathing conditions, the vestibular and vocal folds are relaxed and result in only minor turbulence in the airflow during ventilation (Islam et al., 2020).

The larynx transitions inferiorly into the trachea, an air passage that is approximately 2.5 cm in diameter and 12 cm long to the superior border of the fifth thoracic vertebra (T5), where it divides into the right and left primary bronchi (Tortora & Derrickson, 2014). The trachea is reinforced against collapse by 16-20 incomplete horizontal rings of hyaline cartilage, which resemble the letter C, that are stacked on top of one another and connected by dense connective tissue (Tortora & Derrickson, 2014). The opening of the C-shaped hyaline cartilages opens posteriorly and is spanned by a fibromuscular membrane containing transverse smooth muscle fibers, the trachealis muscle, which allows the diameter of the trachea to be altered slightly

during inhalation and exhalation to maintain efficient airflow (Tortora & Derrickson, 2014).

The mucosa of the trachea is composed of a layer of pseudostratified ciliated columnar epithelium and an underlying layer of lamina propria that contains elastic and reticular fibers, and the submucosa is composed of areolar connective tissue that contains seromucous glands and their ducts (Tortora & Derrickson, 2014).

Around the fifth thoracic vertebrae (T5), the trachea splits into the right and left main bronchi which enter the right and left lungs, respectively (Tortora & Derrickson, 2014). At the point of division, a ridge known as the carina is formed by a posterior projection from the last tracheal cartilage. The carina is one of the most sensitive structures in the lower airways for triggering a cough reflex (Tortora & Derrickson, 2014). Upon entering the lungs, the main bronchi further divide into the lobar bronchi, the right dividing into 3 and the left dividing into 2, corresponding to the 3 lobes of the right lung and 2 lobes of the left lung. The bronchi continue to divide into the segmental bronchi which divide into bronchioles, which in turn branch repeatedly, resulting in up to 23 generations of divisions from the main bronchi (Patwa & Shah, 2015). The dividing ends with the terminal bronchioles which mark the end of the conducting zone of the respiratory system (Tortora & Derrickson, 2014). Structural changes occur within the bronchi as they become smaller: the cartilaginous rings become more inconsistent and then disappear being replaced by a higher quantity of smooth muscle cells, the epithelium changes from ciliated pseudostratified columnar to columnar to cuboidal epithelium, and there are no ciliated or mucous producing cells within the bronchioles (Patwa & Shah, 2015).

At the end of the terminal bronchioles are the alveoli, the grape-like air sacs which are the main sites for pulmonary gas exchange between the inhaled atmospheric air and the blood (Kenney et al., 2015). Within the almost 300 million alveoli, gas exchange occurs across the

respiratory membrane, composed of the alveolar walls, the capillary walls and their respective basement membranes: with normative thicknesses of 0.5 to 4  $\mu\text{m}$  (Kenney et al., 2015). With the primary role of this region being gas exchange, the extremely close proximity between the capillaries and the alveoli facilitates the rapid diffusion of gases down their pressure gradients allowing for homeostasis to be maintained (Kenney et al., 2015).

During inspiration, pressure is decreased at the alveoli approximately 4 mmHg below that of the upper airways and atmosphere resulting in airflow down the pressure gradient into the alveoli of the lungs. Exhalation involves the opposite pressure gradient, though only approximately half the pressure difference compared to inhalation at rest (Tsega, 2018). As such, the ratio of inhalation:exhalation time is approximately 1:1.7 in the average healthy adult at rest (Tsega, 2018). As air is inhaled, a curved sheet-like turbulent laryngeal jet is observed with a turbulence intensity of about 20% as it passes through the subglottic space and enters the trachea (Lin et al., 2007). This turbulence is relatively minor and decreases significantly by the end of the trachea and continues to decrease with each bifurcation of the conducting airways (Lin et al. 2007). Turbulences during inhalation help in maintaining lung health as they aid in the deposition of inhaled particles and toxins onto the mucous membranes of the respiratory system. Major deposition sites in the upper respiratory tract include primarily the nasal cavities while nose breathing and to a much lesser degree, the posterior of the tongue while mouth breathing, as well as the subglottic space (Islam et al., 2020). In addition to the turbulent particle deposition, bifurcations of the conducting air ways also act as deposition sites, collecting approximately 20% more depositions than the straight conducting airways (Islam et al., 2020). Particle filtration and deposition within the upper and conducting airways is vital to lung health as the respiratory region of the lungs, including the respiratory bronchioles and alveoli, lack muco-ciliary clearance

therefore leaving them more vulnerable to toxic particles compared to the rest of the respiratory system (Islam et al., 2020).

## 2. Physiology of breathing

### What we breathe

At rest, the normal human breathes approximately 12-15 times per minute and this rate can increase up to 60 breaths per minute during heavy exercise (Zimmerman, 2019). The tidal volume ( $V_T$ ) refers to the inspiratory and expiratory volumes brought into/out of the lungs under normal sedentary conditions, termed “tidal breathing” (Russo, Santarelli & O’Rourke, 2017). As lung ventilation requires the careful coordination of blood oxygen, carbon dioxide, and pH, respiratory rate and  $V_T$  are closely linked to determine minute ventilation (Russo, Santarelli & O’Rourke, 2017). Minute ventilation being the product of respiratory rate multiplied by the  $V_T$  resulting in lower respiratory rates requiring a greater  $V_T$  in order to maintain homeostasis (Russo, Santarelli & O’Rourke, 2017). The American Lung association (2020) indicates that the average person inhales approximately 2,000 gallons, or just over 7,500 litres of air per day. This estimate is slightly lower than that of Kannan (2015) who indicates that a 70 kg individual breathes approximately  $7.5 \text{ L}\cdot\text{min}^{-1}$  at rest, almost 11,000 L per day, and a resultant  $V_T$  of 650-500 mL (based on the average of 12-15 bpm). These discrepancies could be explained by the large range of body types, fitness levels, and disease prevalence within the sampled populations which can have a large factor on how much air is brought into the lungs and how much oxygen is absorbed into the blood. Regardless of how much atmospheric air is inhaled, the composition of this air under normal circumstances anywhere in the world is comprised of 78.08% nitrogen, 20.93% oxygen, 0.0314% carbon dioxide, and the remaining percentage is composed of argon and trace amounts of various other gases (Kannan, 2015). Based on the average healthy person,

the exhaled air consists of approximately 17% oxygen and 3.2% carbon dioxide, constituting 0.3L of oxygen absorbed and 0.24L of carbon dioxide expelled by the lungs every minute (Kannan, 2015). Absolute oxygen consumption is cited as being directly related to body size, which is the also the largest factor in determining lung capacity in healthy humans, with larger bodies and therefore larger lungs absorbing more oxygen overall compared to smaller lungs (Barnes & Kilding, 2015). For this reason, oxygen consumption is often determined relative to an individual's body mass and is therefore measured in relative ( $\text{mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ ) rather than absolute ( $\text{L}\cdot\text{min}^{-1}$ ) measures (Barnes & Kilding, 2015).

### **Pulmonary ventilation**

The movement of atmospheric air into and out of the lungs is known as pulmonary ventilation, or more commonly, breathing. The movement of air is controlled by the movements of various respiratory muscles which contract and/or relax depending on whether air is moving into or out of the lungs. During the active process of inspiration, the diaphragm, a dome-like muscle located inferiorly to the lungs, is the primary respiratory muscle being aided by the external intercostal muscles. As the diaphragm contracts it flattens downward toward the abdomen while at the same time the external intercostals swing the sternum up and the ribs up and out (Kenney et al., 2015). By expanding the ribs up and out and depressing the diaphragm the lungs are expanded in all three dimensions thereby decreasing the intrapulmonary pressure and creating a pressure gradient between the exterior environment and the lungs, allowing for air to flow into the lungs (Kenney et al., 2015). Exhalation, contrary to inhalation, is predominantly a passive process. Following inhalation, the diaphragm relaxes to its upward, arched position and the external intercostals relax allowing elastic recoil to increase the intrapulmonary pressure, thereby creating a pressure gradient out of the lungs (Kenney et al., 2015). During forced

breathing such as exercise, exhalation becomes a more active process being aided by the contraction of the internal intercostals, which actively pull the ribs inward. This process is further aided by the latissimus dorsi and the quadratus lumborum muscles as well as the abdominal muscles which can help to increase the intrathoracic pressure thereby hurrying the return of the diaphragm muscle to its resting position (Kenney et al., 2015).

### **Pulmonary diffusion**

Pulmonary diffusion is characterized by the movement of gases across the respiratory membrane within the alveoli of the lungs (Kenney et al., 2015). The two major functions of pulmonary diffusion occur simultaneously as oxygen (O<sub>2</sub>) diffuses from the alveoli into the capillaries, thereby replenishing the O<sub>2</sub> supply to the tissues, and carbon dioxide (CO<sub>2</sub>) diffuses out of the capillaries into the alveoli, lowering the CO<sub>2</sub> content within the blood (Kenney et al., 2015).

Diffusion rates are essential to the adequate supply of O<sub>2</sub> and adequate removal of CO<sub>2</sub> for homeostasis to be maintained by the respiratory system. Diffusion rates are dependent on the partial pressure of the respective gases (Kenney et al., 2015). Dalton's law of partial pressure indicates that the sum of the partial pressures for a mixture of gases equals that of the total pressure of the mixture (Kenney et al., 2015). Under normal circumstances, at sea level, the atmospheric pressure is equal to ~760mmHg and is composed of 78.08% nitrogen, 20.93% oxygen, 0.0314% carbon dioxide and the remaining percentages are composed of a variety of other gases with argon being the most notable and trace amounts of several other gases as well (Kannan, 2015). This results in an atmospheric partial pressure for O<sub>2</sub> and CO<sub>2</sub> of 159.1mmHg and 0.2 mmHg, respectively.

Due to the air inhaled into the lungs being fully humidified and mixing with the residual air within the lungs, which has a much higher CO<sub>2</sub> content, the partial pressure for O<sub>2</sub> within the alveoli is reduced to approximately 105 mmHg and the partial pressure of CO<sub>2</sub> is increased to 40 mmHg (Kenney et al., 2015). The venous blood returning from the tissues contains a partial pressure of 40 mmHg and 46 mmHg for oxygen and carbon dioxide, respectively (Kenney et al., 2015). This results in a pressure gradient of approximately 65 mmHg for O<sub>2</sub> entering the blood and a much smaller gradient of approximately 6 mmHg for CO<sub>2</sub> to leave the blood stream and cross the respiratory membrane. The diffusion of these gases across the respiratory membrane is governed by Fick's law indicating that the diffusion of a gas across a membrane is proportional to the surface area of the membrane ( $A$ ), and disproportionally to the thickness of the membrane ( $T$ ) and is proportional to the difference in partial pressures across the membrane ( $P_1-P_2$ ), and the diffusion constant for the particular gas ( $D$ ). The diffusion constant being proportional to the solubility of the gas and disproportionally to the molecular weight of the gas (Kenney et al., 2015).

$$\dot{V}_{gas} \propto \frac{A}{T} \cdot D \cdot (P_1 - P_2)$$

$$D \propto \frac{Sol}{\sqrt{MW}}$$

Despite CO<sub>2</sub> having a relatively small partial pressure differential compared to oxygen, CO<sub>2</sub> is highly soluble in aqueous solutions and therefore has a much higher diffusion constant compared to O<sub>2</sub> and readily diffuses across the respiratory membrane despite the small pressure difference (Betts et al., 2013). The oxygen diffusion capacity is aided by the greater pressure difference across the membrane and the presence of erythrocytes (red blood cells) within the capillaries containing hemoglobin, which readily bind to oxygen once it crosses the respiratory membrane. The oxygen diffusion capacity at rest is approximately 21 ml•min<sup>-1</sup>• mmHg<sup>-1</sup> however

this is determined using the mean partial pressure of the pulmonary capillary compared to the alveolar air, resulting in a gradient of approximately 11 mmHg. Exercise can greatly increase the oxygen diffusion capacity due to the much more extreme desaturation of the blood leading to a greater pressure difference between the alveoli and the blood.

### **Carbon dioxide transport in the blood**

Carbon dioxide is formed during cellular respiration within the tissues of the body and is transported through the blood in three ways (Kenney et al., 2015). A small portion of the CO<sub>2</sub> (7-10%) is dissolved in the plasma of the blood and another small portion of the CO<sub>2</sub> binds to the hemoglobin in the red blood cells, forming carbaminohemoglobin. The main carrier of CO<sub>2</sub> in the blood is in the form of a bicarbonate ion (HCO<sub>3</sub><sup>-</sup>) (Kenney et al., 2015). When CO<sub>2</sub> is released from the tissues it readily reacts with water (H<sub>2</sub>O), with the help of the catalyst carbonic anhydrase (CA), in the aqueous solution to form carbonic acid (H<sub>2</sub>CO<sub>3</sub>), a weak acid that quickly dissociates into bicarbonate and a hydrogen ion in the following equation:



One of the main locations of this reaction occurs on the erythrocytes. In fact, carbonic anhydrase can be found in the erythrocytes of all vertebrates (Greer & Gros, 2000). In the erythrocyte, the HCO<sub>3</sub><sup>-</sup> has a phospholipid permeability 6x less than that of CO<sub>2</sub> however it is aided through the rapid anion transporter protein, capnophorin, which exchanges HCO<sub>3</sub><sup>-</sup> for Cl<sup>-</sup> at a ratio of 1:1 (Greer & Gros, 2000). This anion exchange ratio also allows for the maintenance of the electrical charge of the erythrocytic membrane as well as the plasma (Kenney et al., 2015).

The hydrogen ion diffuses much easier than the  $\text{HCO}_3^-$  however, the  $\text{H}^+$  gradient across the membrane of the erythrocyte is very small, resulting in only minimal movement of the  $\text{H}^+$  across the membrane under normal conditions (Greer & Gros, 2000).

The role of carbonic anhydrase in the hydration/dehydration of  $\text{CO}_2$  is paramount in meeting the needs of the physiological processes occurring within the body. When uncatalyzed, the hydration/dehydration of  $\text{CO}_2$  can take over a minute to complete; much longer than the  $\sim 1\text{s}$  capillary transit time within the lungs (Greer & Gros, 2000). When catalysed in the erythrocytes, the reaction is greatly accelerated resulting in a 95% completion in only 2 ms (Greer & Gros, 2000). In addition to the catalytic aid in the erythrocytes, CA has been shown to be present in the lung tissue of rats which has been shown to accelerate the hydration/dehydration reaction by 130-150x alone (Greer & Gros, 2000). In addition to the lungs, CA has been found in the capillary walls and sarcolemma of all skeletal muscle cells with additional CA being found in the cytosol of slow oxidative muscle fibers, all facilitating the rapid movement and conversion of  $\text{CO}_2$  into  $\text{HCO}_3^-$  as well as the reverse (Greer & Gros, 2000). There is some debate in the literature regarding the presence of CA in the myocardium with Greer & Gros (2000) indicating that there is no CA activity within the cytosol of the myocardial cells, while Vaughan-Jones & Spitzer (2002) indicate that there is some evidence for cytosolic CA availability within the myocardial cells.

Bicarbonate and carbonic acid are present, under normal pH levels, in a ratio of approximately 20:1, respectively (Betts et al., 2013). Together they form the bicarbonate-carbonic acid Buffering system which acts to buffer strong acids such as ketones and lactate, which are produced by the muscles during moderate-vigorous exercise which would rapidly cause metabolic acidosis if unbuffered (Betts et al. 2013).

Under normal sedentary conditions, the CO<sub>2</sub> produced during the catabolism of glucose and lipids during cellular respiration in the tissues drives the reaction towards the right. As the venous blood, laden with CO<sub>2</sub>, returns to the lungs the partial pressure difference between blood and the alveoli air causes the reaction to shift towards the left (Kenney et al., 2015). This results in the release of CO<sub>2</sub> from the blood across the respiratory membrane to be released into the atmosphere, thereby regulating blood pH through respiration and maintaining homeostasis (Kenney et al., 2015).

### **Oxygen transport in the blood**

Oxygen, unlike carbon dioxide, does not dissolve easily in the blood. Less than 2% of the oxygen transported in the body is dissolved in the blood. The remaining 98% of oxygen is bound to a metalloprotein, hemoglobin, in the erythrocytes (Jensen, 2004). Hemoglobin forms a quaternary structure containing four separate heme subunits, each containing an iron atom which allows the heme subunits to bind with oxygen: each hemoglobin molecule thereby being capable of transporting up to four oxygen molecules (Balcerek et al., 2020). Hemoglobin exists in two main forms: the deoxy or tense (T) form and the oxy or relaxed I form (Balcerek et al., 2020). In the T form, hemoglobin is held bound tightly by several ionic bridges formed between hydrophilic and charged amino acids which result in Fe<sup>2+</sup> being in an un-optimal stereochemical position and oxygen being unable to bind (Gomez-Cambronero, 2001). Oxygenation can occur when the ionic bridges are broken and the hemoglobin molecule transitions into its R form (Gomez-Cambronero, 2001). The breaking of the ionic interactions causes conformational changes in the hemoglobin and coupled with the binding of an O<sub>2</sub>, weakens the ionic bridges elsewhere on the hemoglobin which makes it easier for the next O<sub>2</sub> molecule to bind (Gomez-

Cambronero, 2001). This is known as cooperative binding and is exhibited by the sigmoidal-shaped oxygen-hemoglobin dissociation curve (Kenney et al., 2015). When a hemoglobin molecule has bound four oxygen molecules it is known as oxyhemoglobin and is said to be fully saturated (Betts et al., 2013).

Erythrocytes are the most abundant cell in the human body with an estimated  $\sim 2.5 \times 10^{13}$  cells and each erythrocyte contains approximately  $270 \times 10^6$  hemoglobin molecules (D'Alessandro et al., 2017). Due to this, the oxygen-hemoglobin association curve refers to the percentage of saturation of the hemoglobin as a whole, in relation to the partial pressure of oxygen (Kenney et al., 2015). The cooperative binding allows the hemoglobin to become almost fully saturated with oxygen as it passes through the alveoli of the lung ( $\sim 1$  s at  $\sim 100$  mmHg) (Kenney et al., 2015). As conditions change within the lung such as exercise, which would decrease the time blood spends in contact with the alveoli due to pressure increases, or altitude, which may result in a decreased partial pressure of oxygen, the cooperative binding and subsequent sigmoidal Hb-O<sub>2</sub> curve helps to buffer these diverse conditions and prevents oxygen saturation in the lungs from dropping significantly (Kenney et al., 2015; & Gomez-Cambronero, 2001).

Oxygen leaves the lungs almost fully saturated at a O<sub>2</sub> partial pressure of approximately 100 mmHg. Upon reaching the tissues, who are actively using oxygen, the partial pressure decreases to approximately 40 mmHg which is sufficient to cause the dissociation of the first of the four bound O<sub>2</sub> (Gomez-Cambronero, 2001). As one oxygen molecule dissociates from its heme subunit this causes a conformational change resulting in a reduced affinity for the other heme subunits towards the remaining oxygen molecules causing them to more readily dissociate (Jensen, 2004).

Under sedentary conditions, the blood returns from the tissues to the lungs carrying almost all of the oxygen, being approximately 75% saturated (Betts et al., 2013). Under active conditions, such as exercise, the partial pressure of O<sub>2</sub> can drop further within the tissues to approximately 20 mmHg with the typical p50 value (PO<sub>2</sub> at 50% Hb-O<sub>2</sub>) of ~26 mmHg (Gomez-Cambronero, 2001; Balcerek et al., 2020). This results in greater O<sub>2</sub>-Hb dissociation as O<sub>2</sub> readily follows the pressure gradient into the exercising muscles (Betts et al., 2013). The p50 is not a constant between or even within humans. At rest, Balcerek et al. (2020) found that there may be as much as a 7 mmHg difference in p50 value at rest between participants ranging in age from 18-40 with a wide variety of fitness levels: being unrelated to participant's fitness. These differences may be attributable to minor variations in resting conditions between participants as the Hb-O<sub>2</sub> affinity has been found to be greatly affected by a variety of factors such as temperature, [H<sup>+</sup>] (pH), CO<sub>2</sub>, and inorganic phosphates such as 2,3-bisphosphoglycerate; with increases in any of these effectors resulting in a rightward shift in the Hb-O<sub>2</sub> dissociation curve and a decreased Hb-O<sub>2</sub> affinity (Balcerek et al., 2020). Conditions, such as those experienced during exercise, can result in increased temperature in the exercising muscles, a lowered pH (increased [H<sup>+</sup>]) and increased CO<sub>2</sub> due to an increase in cellular respiration; all of which reduce the Hb-O<sub>2</sub> affinity and improve oxygen delivery to the tissues (Balcerek et al., 2020).

### **The drive to breathe and regulation of pH**

The primary physiological stimulus for breathing at rest and during exercise, in healthy individuals living at low altitudes, is not hypoxia (low blood oxygen levels) but rather hypercapnia, the accumulation of carbon dioxide (CO<sub>2</sub>) in the blood (Patel et al. 2020). Carbon dioxide is a metabolic by-product of aerobic cellular respiration, the process of breaking down glucose molecules using oxygen to obtain energy in the form of adenosine triphosphate (ATP)

which can be subsequently used to fuel almost all other energy requirements within the body, such as moving molecules against concentration gradients within our cells and all muscle contractions (Kenney et al. 2015). Carbon dioxide is also produced in the blood through the bicarbonate buffering of acidosis which can result in a higher concentration of CO<sub>2</sub> in the body than what is due to aerobic cellular respiration. During exercise, the metabolic requirements of the body are increased resulting in an increase in tissue CO<sub>2</sub> production and therefore in an increase in serum CO<sub>2</sub> concentration. As exercise intensity begins to increase, and especially as exercise intensity begins to surpass the aerobic capacity of the exercising muscles, the concentration of acidic metabolic by-products is increased, requiring the bicarbonate buffering system to prevent acidosis and resulting in a great increase in serum CO<sub>2</sub> concentration.

Carbon dioxide levels are monitored in two areas within the human body by the central and the peripheral chemoreceptors (Patel et al. 2020). The peripheral chemoreceptors are located in the carotid and aortic bodies and are sensitive to changes in PO<sub>2</sub>, PCO<sub>2</sub>, and overall changes in blood pH (Patel et al. 2020). The central chemoreceptors, located on the lateral ventral section of the medulla, are highly sensitive to changes in PCO<sub>2</sub> due to the fact that CO<sub>2</sub> can penetrate the blood-brain barrier and are also sensitive to changes in cerebrospinal fluid pH (Patel 2020). This combination of monitoring systems regulates the motor output to the respiratory centre of the brain, increasing minute ventilation when hypercapnia is detected and decreasing minute ventilation once the feedback loops detect a decrease in PCO<sub>2</sub> (Patel et al. 2020).

During low- to moderate-intensity exercise, the metabolic requirements of the body are increased, thereby requiring an increased energy utilization within the exercising tissues (Fletcher et al. 2009). An initial rise in metabolic by-products such carbon dioxide (CO<sub>2</sub>), which diffuses through the blood-brain barrier and triggers the central chemoreceptors, leads to an

increased sympathetic outflow to the vasculature resulting in an increased ventilation rate (Patel et al. 2020). During moderate-to-vigorous intensity exercise, the body begins to rely more heavily on anaerobic energy sources which produce acidic by-products which must be buffered in the blood by the bicarbonate buffering system which further increases the CO<sub>2</sub> concentration. Therefore, during exercise, the drive to breath is much greater and more consciously controlled due to the sensation of air hunger caused by the accumulation of these metabolic by-products.

### **Dyspnea**

Dyspnea has a variety of definitions within the medical and physiology realms. The Mayo clinic (2021) defines it as anything ranging from “shortness of breath” to “air hunger” to the more extreme “feeling of suffocation”. While Hashmi, Modi, & Sharma (2021) describe dyspnea as the feeling of “running out of air and not being able to breathe fast enough or deeply enough”. This wide range of definitions may be best described by Buchanan & Richerson (2009) who label dyspnea simply as “the uncomfortable awareness of respiratory distress”. Dyspnea is a complex and multifactorial phenomenon that results from the interaction of signals and receptors of the peripheral nervous system (PNS), the central nervous system (CNS), the limbic system, chemoreceptors, and mechanoreceptors in the upper airways, lungs, and chest wall (Hashmi, Modi, & Sharma, 2021; Buchanan & Richerson, 2009).

It has been hypothesized that the cause of dyspnea is due to a mismatch between the afferent respiratory sensory signals and the motor output signals from the central respiratory structures (Buchanan & Richerson, 2009). The afferent sensory signals are derived from both the mechanoreceptors in the lungs and chest wall, which provide information regarding the stretch of the lungs; as well as the chemoreceptors in the carotid/aortic bodies, which provide information on the O<sub>2</sub> and CO<sub>2</sub> content within the blood (Hashmi, Modi, & Sharma, 2021). Mechanoreceptors

provide important, real-time information to the brain regarding the amount of stress that the lungs are under at any given time. Lack of afferent mechanoreception information has been shown to decrease or alter the dyspnea sensation experienced by breath holders who have undergone temporary phrenic nerve paralysis (Parks, 2006). The paralysis of the diaphragm and subsequent increase in apnea time clearly indicate that mechanoreceptors contribute to the negative physical sensations associated with dyspnea which, along with the chemoreceptors, create the overall sensation of dyspnea.

The negative emotional sensations associated with dyspnea, such as anxiety, are induced less by the mechanoreceptor stimulation and more by the chemoreceptor stimulation caused by hypercapnia. It has been found that breathing a gas mixture of 20% carbon dioxide (usually >0.05%) for only 25 s reliably produces behavioural response patterns characteristic of intense anxiety or panic (Eifert et al., 1999; Nestor, 2020). Functional magnetic resonance imaging (fMRI) and positron emission tomography (PET) have shown that limbic structures such as the cingulate cortex, hippocampus, insula, amygdala, and hypothalamus are activated during hypercapnia, induced by breathing a high CO<sub>2</sub> concentrated gas (Buchanan & Richerson). This helps to explain the negative emotional response associated with the feelings of dyspnea caused by hypercapnia.

### **Trainability of dyspnea**

A universally experienced sense of dyspnea is holding one's breath. As such, we are aware that people can hold their breath for different amounts of time, and that one can improve the ability to hold their breath with practice. The average person can hold their breath for anywhere between 30-120 s, with the time being dependent on age, fitness/health, and which breath hold technique is used. By comparison, the world record for an above water, non-oxygen-

aided breath hold is 11 minutes and 35 seconds, showing a vast range of what is humanly possible for breath holding ([guinnessworldrecords.com](http://guinnessworldrecords.com)).

During apnea (breath holding) there are two distinct phases; the ‘easy’ phase, marked by the duration of apnea prior to the onset of involuntary breathing movements (IBMs), and the ‘struggle’ phase, consisting of the apnea duration following the onset of IBMs until breathing is reinitiated (Bain, 2016). The time at the onset of IBMs is termed the physiological break point, while the re-initiation of breathing is known as the apnea break point (Bain, 2016). During apnea, the respiratory muscles are nevertheless used, although the mechanism of control during an apnea is not fully understood, to hold the chest at a fixed volume thereby resisting the recoil during apnea (Parkes, 2006). Despite the conscious suppression of breathing, the central respiratory centre continues to drive the respiratory rhythm throughout an apnea which leads to the eventual involuntary contraction of the respiratory muscles, marking the physiological breakpoint, the commencement of the IBMs, and the beginning of the struggle phase.

The physiological breakpoint for untrained breath holders often occurs at the same time as the apnea breakpoint due to a combination of chemoreceptor and diaphragm afferents resulting in an overwhelming sensation of dyspnea (Bain, 2016). Chemoreceptor afferents effecting the apnea breakpoint in novice breath holders have been shown to be due to hypercapnia rather than hypoxemia, as shown by the substantial evidence indicating that hyperventilation, resulting in hypocapnia, can substantially increase apnea duration (Bain, 2016). During an apnea, the respiratory muscles are voluntarily held at a fixed volume despite their natural tendency to actively recoil. The central respiratory rhythm continues to act on these muscles throughout the apnea, opposing the voluntary control; which is then further opposed by

the augmentative effects of the hypercapnia response on the respiratory centre (Parks, 2006; Bain, 2016).

Apnea duration is therefore dependent on the ability of the higher brain structures being able to override the involuntary sensory input from the chemoreceptors and the central respiratory centre. Through apnea training, elite apnea competitors have achieved unconsciousness through out-of-water breath holding which had once been thought to be impossible (Parkes, 2006; Bain, 2016). In elite apnea competitors, the apnea breakpoint is in fact determined by hypoxemia, usually around a hemoglobin saturation level of ~50%, rather than hypercapnia which indicates the trainability of the dyspnea sensation (Bain, 2016). It is generally accepted that the ventilatory response to hypercapnia is delayed in trained apnea divers which allows for the delay of IBM's and the prolongment of the 'easy' phase of the apnea (Bain, 2016).

In support of this, Schagatay et al, (2000) found that 2 months of aerobic physical training allowed participants to increase their apnea duration by increasing the duration of time endured in the struggle phase, while 2 weeks of apnea training increased apnea duration by delaying the onset of the physiological break point. Similarly, Engan et al, (2011) found that a 2-week training program of 10 maximal apneas a day significantly increased apnea duration by an average of 44s. It was also found that the duration of the easy phase was significantly ( $p < 0.5$ ) longer following the apnea training however there was no significant difference in struggle phase duration following the apnea training (Engan et al, 2011). To reach the elite level of apnea duration, competitors must physiologically adapt to the hypercapnia through repeated apnea exposures resulting in a blunted ventilatory response to elevated CO<sub>2</sub> levels as well as mentally adapt to the dyspnea sensation associated with the struggle phase following the initiation of the IBM's. One's reaction and tolerance to dyspnea is therefore trainable through both physiological

and mental avenues, which may further explain the wide variety of apnea durations within the human population.

### **3. The history of breathing**

#### **Respiratory changes in human ancestors**

Human beings, homo sapiens, are the only species within the animal kingdom to develop a large, protruding nose (Mladina et al. 2009; Nishimura et al. 2016). Quadrupeds and other higher mammals house their nasal cavities deep within massive and strong facial bones which helps to protect the delicate structures of the nasal cavity (Mladina et al. 2009). The protrusion of the human nose may have resulted from a combination of evolutionary factors. As human ancestors began to adopt an erect posture, supported bipedally, it allowed for our ancestors to view prey and predators better and may have resulted in an increased importance on visual senses instead of olfactory senses, which are so important to the day-to-day survival of lower primates (Mladina et al. 2009). Additionally, the adaptation of an erect posture caused the skull to become flatter and required greater cortical processing in order to process the spatial and other cognitive functions required to maintain balance, which may correlate to the brain size expansion in homo erectus (Mladina et al. 2009). As human ancestors began to cook their food, it facilitated the easier release of nutrients and reduced the need for big strong jaws which caused the maxilla and mandible to shrink and move backwards (Mladina et al. 2009). The adaptation of the erect posture coupled with the diet changes of human ancestors resulted in the expansion of the neurocranium and the shrinking of the splanchochranium (Mladina et al. 2009). This shrinking of the splanchochranium squeezed the large snout-like airways of the homo erectus due to the

angulation of the surrounding bones, which caused the protrusion of the most anterior parts more anteriorly resulting in the distinct protruding nose of the homo sapiens (Mladina et al., 2009).

Lower primates, such as chimpanzees and macaques, who do not possess the protruding nose, have been shown to exhibit a greater ability to condition inspired air within the nasal cavities compared to that of humans (Nishimura et al., 2016). The nasal cavities of the lower primates exhibit a straighter horizontal airflow while humans exhibit an upward curved airflow (Nishimura et al., 2016). While the human nasal cavity was shown to exhibit a lesser ability to condition more extreme climates (i.e., cold-dry and hot-dry air) than lower primates, humans possess an elongated pharynx that, when coupled with the nasal cavities, allowed for the inhaled air to be fully conditioned to alveolar requirements (Nishimura et al., 2016).

### **Ancient focus on breathing**

For thousands of years, eastern cultures have employed the use of yogic breathing, or pranayama, as a method of restoring and improving health (Russo et al., 2017). In fact, there are seven books of the Chinese Tao dating back to approximately 400 BCE focusing on the concepts of breathing and its relationship with health and spirituality (Nestor, 2020). There are a variety of breathing techniques that are employed in these practices, such as variations of nostril breathing (double, single, and alternating), abdominal breathing, forced breathing, and vocalized breathing which are all employed during yogic practices for a variety of health reasons (Russo et al., 2017).

Yogic, controlled breathing has been shown to have positive health effects when used in conjunction with modern medicine when compared to modern medicine alone (Abishek et al., 2019). Chronic rhinosinusitis, the inflammation of the paranasal sinuses and nasal cavities, has been shown to have an increasing prevalence in developed countries, due to a variety of factors such as pollution and lifestyle changes, with an incidence rate of around 12-15% (Abishek et al.,

2019). The practice of bhamari pranayama, a type of controlled nasal breathing that involves making a humming noise while breathing, has been shown to significantly decrease inflammation in chronic rhinosinusitis patients when used with a steroid nasal spray when compared to just a nasal spray alone after only two weeks ( $p < 0.05$ ) and continued to show improvements until the 4<sup>th</sup> week of treatment ( $p < 0.00001$ ) (Abishek et al., 2019). Hypotheses regarding the underlying mechanics of this improvement were thought to be related to several reasons: the reduction of anxiety which may have helped to reduce overall stress and reduce inflammation, the humming causes vibrations which can facilitate the movement of air between the paranasal sinuses and help to open blocked ostia of the sinuses which may promote better drainage, and the increasing release of endogenous nitric oxide from the paranasal sinuses through humming (a powerful vasodilator and free radical that will be discussed further below (see modern evidence for nose breathing) (Abishek et al., 2019).

Pranayama and the practice of yoga can be traced back to ancient India and Tibet, however the practice of slow controlled breathing has been integrated into many of the major religions in the world (Bernardi et al., 2001). The use of prayer beads was originally seen by Tibetan monks, this practice was adopted by the ancient Arabs, and, following the crusades (1095-1291), the rosary was introduced to Christianity (Bernardi et al., 2001). It has been shown that both the recitation of the Ave Maria and yoga mantras slow the respiratory rate to almost exactly 6 breaths per minute (Bernardi et al., 2001). This slow respiratory rate corresponds to the same frequency as the natural rhythmic fluctuations in blood pressure (BP) and heart rate that are observed in healthy humans ( $\sim 0.1\text{Hz}$ ) (Julien, 2006). Both the Ave Maria and yoga mantras showed a synchronization of respiratory rate and cardiovascular rhythms including systolic and diastolic blood pressures, and transcranial blood flow (Bernardi et al., 2001). The

synchronization of these factors reduces stress and has been shown to increase heart rate variability (HRV), indicating a reduction in sympathetic activity (Bernardi et al., 2001). In support of the cardiovascular impacts of prayers/mantras, it has been found that slow deep breathing decreased systolic BP from  $148 \pm 5.8$  mmHg to  $138.2 \pm 5.9$  mmHg and diastolic BP from  $85 \pm 1$  mmHg to  $78.5 \pm 5.5$  mmHg ( $p < 0.05$ ) in hypertensive I and II patients aged 40-55 (Wiharja et al., 2016). Furthermore, device-guided slow breathing (~8 bpm) also has shown a decrease in systolic BP by  $6.4 \pm 1.8$  mmHg, significantly decreased HR, and significantly increased HRV over a 15min guided breathing session in 102 participants aged 40-70 (Anderson, McNeely, & Windham, 2009). Slow and controlled breathing has also been shown to reduce anxiety and improve the mood, as measured using the Profile of Mood State (POMS) measurement, as well as reduce fatigue in individuals undergoing chemotherapy treatments (Hayama & Inoue, 2012). In support of the effects of slow breathing on psychological factors, another study that compared religious-based therapy, using the Qur'an, with only slow breathing found that both religious therapy and slow breathing resulted in significant improvements in psychological coherence and depression scores, and slow breathing also significantly reduced anxiety (Amjadian et al., 2020).

Beyond the use of slowed breathing, many ancient cultures have emphasized importance of nose breathing and its effects on health. In the Judaism/Christian story of creation, it is said that "God formed the man from dust of the ground and breathed into his nostrils the breath of life" (Genesis 2:7). While time may have shifted the emphasis away from nose breathing within the catholic faith, one society was documented as taking the use of nose breathing extremely seriously: the Indigenous tribes of the American Plains (Catlin, 1870). Over a period of 8 years, from 1832-1839, George Catlin travelled throughout the American Plains visiting the Crow, Blackfoot, Pawnee, Sioux, Comanche, Mandan, Choctaw, Cheyenne, Winnebago, Creek, and

Assiniboine nations; chronicling his travels, their customs, and creating accurate portraits of individuals and daily life (Catlin, 1870). While not the primary purpose of his travels, he quickly became aware of the unanimous animosity held within these Indigenous communities regarding the mouth breathing habits of the Caucasian communities and traders with whom the Indigenous communities had contact with, calling them “pale face” and “black-mouth” (Catlin, 1870). In his works Catlin (1870) highlights that no other animal sleeps with its mouth open as it is “unnatural”; that inhalation should be of “vital air through the nostrils, the natural channel.”. Catlin (1870) highlights that in European society it was very common to spend much of the day, and often the night, with the mouth open and exposed to air; this culture experiencing many issues with crooked teeth and tooth decay. The Indigenous communities however, emphasized the importance of nose breathing from birth (Catlin, 1870). Catlin (1870) recounts how he witnessed mothers closing the mouths of babies following suckling as well as closing the mouth during sleep and ensuring that the babies are not too hot, to discourage panting from the mouth; this behaviour resulting in the formation of a life-long habit of nose breathing. In fact, Catlin (1870) states that “An [Indigenous] Warrior sleeps, and hunts, and smiles, with his mouth shut.”. Coincidentally, Catlin (1870) spoke to the straight teeth and lack of tooth decay witnessed among all these Indigenous communities. Additionally, Catlin (1870) notes that he did not witness any instances of respiratory conditions or physical ailments not caused by accidents/war, and that the chiefs of these communities reported no infant deaths during teething (a problem that Catlin notes as being very prevalent among European infants at the time). While this reporting is entirely anecdotal and the lack of disease prevalence may be potentially accounted for by thousands of years of natural selection being unimpeded by allopathic medicine, George

Catlin (1870) provides unprecedented insight into the value that nose breathing unanimously held within the diverse society of the Indigenous peoples of the American plains.

### **Modern evidence for nose breathing**

Nasal breathing has been cited by many as a healthier method of breathing compared to mouth breathing only (Naftali et al., 2005; Lacombe, 2015; Recinto et al., 2017; & Dallam et al., 2018). Nasal breathing allows for the filtration, humidification, and temperature conditioning of the inspired air prior its arrival in the alveoli; potentially avoiding some health concerns associated with breathing unfiltered and non-humidified air (Dallam et al., 2018). Additionally, nasal breathing has been claimed to allow for greater diffusion of oxygen within the alveoli to the pulmonary capillaries due to the local release of nitric oxide (NO), a vasodilator, in the paranasal sinuses; and greater oxygenation of the tissues due to an increased level of CO<sub>2</sub> in the blood (Dallam et al., 2018). The release of NO by the enzyme nitric oxide synthase in the paranasal endothelial cells causes a series of reactions when inhaled through the nasopharynx into the lungs, resulting in the decrease in intracellular calcium of the smooth muscle cells surrounding the pulmonary arterioles causing vasodilation and thus greater blood flow through the subsequent pulmonary capillaries (Klinger & Kadowitz, 2017).

Nitric oxide, also known as nitrogen monoxide, has the chemical formula of NO and a molar mass of 30.01 g•mol<sup>-1</sup> (Abeyakirithi, 2009). Nitric oxide is a free radical due to the single unpaired electron and is therefore highly reactive and has a half-life of only several seconds within the human body (Abeyakirithi, 2009). In the human body, NO is extremely widespread being involved in almost all cellular functions, predominantly being used as a signaling molecule (Luiking et al., 2010). Nitric oxide production within the human body is completed by the enzyme NO synthase (NOS) which catalyzes the redox reactions involved in forming NO and L-

citrulline from L-arginine (Luiking et al., 2010). Three isoforms of NO synthase exist in the human body: inducible NOS (iNOS), neuronal NOS (nNOS), and endothelial NOS (eNOS) (Luiking et al. 2010). Inducible NOS is activated at the level of gene transcription and is  $\text{Ca}^{2+}$  independent (Luiking et al., 2010). Unlike eNOS and nNOS, which are contained to endothelial and neuronal cells respectively, the iNOS is present in many non-vascular cells such as fibroblasts and macrophages; iNOS plays a large role in mediating the inflammatory response (Klinger & Kadowitz, 2017). While iNOS is more diverse in its host cells, it is the NO synthesized from the eNOS in epithelial cells that is considered to be the major source of NO in the pulmonary system (Klinger & Kadowitz, 2017).

NO production by NOS requires a constant supply of L-arginine which is provided by a variety of pathways. One such pathway involves the absorption of glutamine from dietary sources which is converted to L-citrulline and ammonia, both enter the liver where the ammonia is converted to urea while the citrulline passes through to circulate in the bloodstream before being converted to L-arginine and urea in the kidneys (Klinger & Kadowitz, 2017). Klinger & Kadowitz (2017) indicate that approximately 60% of arginine is synthesized via this pathway with only 15% being synthesized in the endothelium. Regulation of the eNOS is partly mediated by caveolae, invaginations in the plasma membrane which contain the transmembrane protein caveolin which serves to keep the NOS in an inactive state (Klinger & Kadowitz, 2017).

Endothelial NOS is inactivated by the caveolin until sufficient levels of calmodulin (CaM) and Calcium ( $\text{Ca}^{2+}$ ) accumulate within the cytoplasm. Once sufficient levels of CaM and  $\text{Ca}^{2+}$  have accumulated, calcium-activated CaM binds with eNOS resulting a conformational change and the disassociation of caveolin thereby activating eNOS and resulting in NO synthesis (Klinger & Kadowitz, 2017). The NO may then diffuse freely and begin exerting its dilatory effects or enter

the bloodstream. NO that diffuses into the vascular smooth muscle cells will activate the enzyme guanylyl cyclase; this converts GTP to cGMP which in turn activates cGMP dependent protein kinases beginning a series of reactions which result in a decrease in intracellular calcium and a decrease in vascular tone resulting dilation of the affected arterioles and greater blood flow to the capillaries (Klinger & Kadowitz, 2017).

A study on healthy tracheotomized patients (n=7, aged 27- 45yrs) found that exhaled NO was minimal when breathing through the trachea, that mouth breathing has significantly higher NO concentrations in the exhaled air than tracheal breathing (p=0.033), and nasal breathing had significantly higher exhaled NO concentrations than oral breathing (p=0.01) (Törnburg et al., 2002). Similarly, Törnburg et al. (2002) found that the inhaled NO concentrations were significantly higher when nasal breathing versus oral breathing (p=0.03). This indicates that there must be a significantly higher concentration of eNOS within the nasal cavity compared to the oral cavity. This is supported by Cardell (2002) who claims that the NOS, and subsequent NO, concentrations are significantly higher in the nose compared to the mouth. Furthermore, Cardell (2002) indicates that there has been found to be significantly more NO production in the paranasal sinuses compared to the nose itself, reportedly up to a seven-fold increase in concentration. Lundberg (2008) indicates that the continuous NO production by the eNOS within the endothelium of the paranasal sinuses may be due to NO's free-radical and subsequent anti-microbial properties that help to protect these structures, which have poor drainage. In Lundberg's conclusion, they indicate that inhaled NO from the nose and sinuses that reaches the lungs, while more diluted, may act as an "aerocrine" hormone (the term "aerocrine" indicating its airborne delivery) and promotes oxygen uptake by reducing pulmonary vascular resistance (Lundberg, 2008). To further this argument, Weitzberg & Lundberg (2002) showed that nasal

NO levels were increased 15-fold when participants made a humming or buzzing sound due to the increased gas exchange between the paranasal sinuses and the nasal cavity. The same test of quiet versus buzzing exhalation was performed for oral breathing and showed no changes in exhaled NO (Weitzberg & Lundberg, 2002). While this study only measured NO levels during exhalation, it is reasonable to assume that this technique may increase inhaled NO levels also. These findings, along with others, have contributed to the proliferation of a number of internet blogs and lifestyle websites which promote nasal breathing as an improved method of blood oxygenation, one such site even claiming that an un-referenced study found that blood oxygenation was improved 10-20% (Severinsen, n.d.). While these numbers have not been substantiated by any studies this researcher has reviewed, there may be some credence for improved respiratory efficiency while nose breathing.

In addition to the contribution of NO as a vasodilator, nasal breathing forces a slower respiratory rate due to the increased air resistance caused by the narrow openings of the nasal vestibules, resulting in a greater blood CO<sub>2</sub> content (Buchanan & Richerson, 2009). Carbon dioxide has an effect on hemoglobin's affinity for oxygen with a higher CO<sub>2</sub> serum content resulting in a reduced affinity for O<sub>2</sub> (Patel et al., 2020). The Bohr effect describes the affinity of oxygen to hemoglobin within the body, with higher CO<sub>2</sub> levels resulting in a rightward shift in the graph, denoting a reduced oxygen-hemoglobin association. While hemoglobin has a lower affinity for oxygen with a rightward shift, the partial pressure of oxygen in the lungs at the alveoli is more than adequate to almost fully saturate the hemoglobin within pulmonary capillaries. The lower oxygen-hemoglobin affinity results in a beneficial O<sub>2</sub> off-loading effect at the tissue level, allowing for improved tissue oxygenation (Patel et al., 2020).

#### 4. Nose breathing during exercise

##### Effects on performance, timeline, and subjective experiences

It is universally accepted that humans will unconsciously switch from nose breathing to mouth breathing relatively quickly as exercise intensity increases above low levels, due to the sensation of dyspnea that is experienced (Kippelen et al., 2012; Lacombe et al., 2017; Dallam et al., 2018; Dallam & Kies, 2020). When individuals unaccustomed to nose breathing training are forced to breathe through their nose during exercise, it has been shown that a decrease in the rate of peak oxygen consumption ( $\dot{V}O_{2peak}$ ) occurs (Dallam & Kies, 2020). However, it has also been shown, by Dallam et al, (2018), that it was possible to achieve the same  $\dot{V}O_{2peak}$  whilst nose breathing, compared to mouth breathing, when a recreational population completed a self-directed nose breathing training protocol for a minimum of 6 months (average nose breathing training duration of 4.5 years). Similarly, a case study on a 53-year-old, male triathlete found that after 6 years of nose breathing training, he showed no difference in  $\dot{V}O_{2peak}$  or the speed at  $\dot{V}O_{2peak}$  ( $v\dot{V}O_{2peak}$ ) between nose breathing and mouth breathing (Hostetter et al, 2017). The ability to train one's capacity for nose breathing during exercise is therefore remarkable, however the length of time for such adaptations to occur is not well understood. For example, Hostetter et al, (2017) reported that the 53-year-old male triathlete had required approximately 6 months of progressive intensities to fully adapt to nose breathing, after which he ran a 5:32 mile while nose breathing; this being the fastest he had run a mile in a decade. During this transitional time, Hostetter et al, (2017) indicate that the participant experienced strong sensations of dyspnea during the training which gradually faded and allowed him to increase the intensity further, at which point he would experience dyspnea and the cycle of adaptation would repeat. No in-depth description is given for either study (Hostetter et al, 2017; Dallam et al, 2018)

regarding the exact timeline of adaptation, the training plans/logs, the exercise intensity increments, nor the subjective accounts of the participants undergoing the NB training. More explanation is required into the subjective experiences of dyspnea such as the nature of the sensations, the range of experienced sensations, and the effects that these anxiety-like episodes may have on psychological factors such as motivation to continue and mood. More research is necessary to understand the exact timeline of this adaptation in order to achieve the same maximal output while nose breathing. Furthermore, this adaptation to maintaining  $\dot{V}O_2peak$  while nose breathing has currently only been shown in recreational runners and one ex-competitive triathlete and has yet to be shown to be effective in elite endurance sport athletes.

Participants who are unaccustomed to NB have been shown to be able to complete sub-maximal intensities with no reported drop-out rate greater than that of the MB group/conditions (Lacomb, 2015; Gonzalez-Montesinos, 2020; Lacomb et al, 2017). At these submaximal intensities, usually ranging from 50-80% of the predicted or achieved  $v\dot{V}O_2peak$ , unaccustomed participants have successfully completed the short (4-10 min) exercise trials (Lacomb, 2015; Gonzalez-Montesinos, 2020; Lacomb et al, 2017). These studies have primarily investigated the ventilatory and respiratory differences that exist between NB and MB during these short submaximal bouts and have not investigated submaximal performance measures such as a time to exhaustion test. More research is needed regarding the submaximal endurance of participants who are unaccustomed to nose breathing at various submaximal intensities.

Anaerobic power output has been found to be unaffected by the use of NB or MB in two counter balanced Wingate tests with recreational male (n=7) and female (n=2) participants unaccustomed-to-nose breathing (Recinto et al, 2017). This study by Recinto et al, (2017) did not test the effects of NB on anaerobic endurance however, such as might be achieved by multiple

successive Wingate tests, which limits the generalizability of these findings to any sport application beyond that of the 100 and 200 m dash; or other such single & short explosive-type events. More research is required to investigate the effects of nose breathing on anaerobic endurance such as what may be experienced during sporting events such as hockey, soccer, rugby, and football.

### **Changes to ventilation**

During exercise, as the metabolic requirements of the body are increased, ventilation ( $\dot{V}E$ ) increases proportionally to exercise intensity. At rest, individuals may breathe as little as 7-8 L•min<sup>-1</sup> of atmospheric air (Kannan, 2015). During maximal exercise, this value is greatly increased with an average population breathing an average of 82.2±27.3 L•min<sup>-1</sup>; with men aged 20-29 breathing an average of 114±23 L•min<sup>-1</sup> (Blackie et al., 1991). These values are even greater in an endurance trained population, with ventilation rates of 120-140 L•min<sup>-1</sup> being common (Amonette & Dupler, 2002). Nose breathing has been universally shown to have consistently lower relative  $\dot{V}E$  compared to MB breathing (Hostetter et al, 2017; Lacombe et al., 2017; Dallam et al., 2018; Dallam & Kies, 2020). This difference has shown to not be linear, with the differences in  $\dot{V}E_{Peak}$  between MB and NB increasing as the exercise intensity increases (Lacombe et al., 2017).

The decreased  $\dot{V}E$  during nasal breathing is predominantly caused by the increased air resistance caused by the narrow opening of the nasal vestibules compared to the wide opening of the mouth. The increased air resistance while NB causes a decrease in the  $RR$  which the body attempts to compensate for by increasing the  $\dot{V}_t$ , however this does not fully compensate for the drastic reduction in  $RR$  which results in an overall decreased  $\dot{V}E$  during NB compared to MB at the same intensities (Hostetter et al., 2017; Lacombe et al., 2017; Dallam et al., 2018; Dallam &

Kies, 2020). The narrow opening of the nose requires a slower  $RR$  in order to fully (relative to oral breathing) ventilate the lungs which can result in increased mechanoreceptor afferent activity which may contribute to the fact that most individuals will switch from predominantly NB to MB at a  $\dot{V}E$  of approximately  $40 \text{ L}\cdot\text{min}^{-1}$ . There is some evidence that there is no relationship between air resistance and the switching point between NB and MB; with hypotheses being that the switching point is possibly due to the hypoventilation and an increased perception of effort while nose breathing. During a maximal graded exercise test, those who are unaccustomed to nasal breathing will unanimously achieve a lower  $\dot{V}O_{2peak}$  which is likely due to the decrease of  $\dot{V}E_{peak}$  by approximately 35% (Dallam & Kies, 2020). In recreational runners who have undergone nose breathing training for a minimum of 6 months, it has been shown that  $\dot{V}O_{2peak}$  showed no differences between NB and MB conditions despite the reduced  $RR$  and subsequent reduced  $\dot{V}E$  during the NB conditions (Dallam et al., 2018). Furthermore, this population showed no differences in peak lactate during the Maximal GXT which indicates that this difference was not compensated for by an increased anaerobic contribution during the NB condition (Dallam et al., 2018).

### **Changes to respiration**

Along with  $RR$  and  $VE$ , it has been found that the ventilatory equivalent for  $O_2$  ( $\dot{V}E/\dot{V}O_2$ ) and  $CO_2$  ( $\dot{V}E/\dot{V}CO_2$ ) are also decreased while NB relative to MB at the same intensities (Hostetter et al, 2017; Lacombe et al., 2017; Dallam et al., 2018; Dallam & Kies, 2020). Several studies have also found a decrease in the end tidal fractional oxygen ( $FEO_2$ ) and pulmonary oxygen partial pressure ( $PETO_2$ ) further indicating an increased gas exchange per breath during NB (Hostetter et al., 2017; Dallam et al., 2018). These findings indicate that more gas exchange is occurring per

unit of air inhaled into the body during NB vs MB, however it does not necessarily indicate a greater total oxygen uptake during NB.

While runners unaccustomed to NB show a decrease in  $\dot{V}O_{2peak}$ , runners who are accustomed to nose breathing have shown no differences in  $\dot{V}O_{2peak}$  between NB and MB (Hostetter et al., 2017; Dallam et al., 2018). During submaximal exercise this phenomenon of lower  $\dot{V}O_2$  at a given intensity while NB relative to MB continues to be true for not only nose breathing trained runners (Hostetter et al., 2017; Dallam et al., 2018), but for untrained individuals as well at a variety of different sub-maximal running intensities (Lacomb et al., 2017). With this knowledge, Hostetter et al., (2017) and Dallam et al., (2018) hypothesize that the reason for this improvement in running economy is due to an increased time for diffusion of gases within the alveoli of the lungs as well as a decreased metabolic demand from the respiratory muscles, both a result of the forced slower respiratory rate inherent to nose breathing. This idea may become more accepted within the field of exercise physiology for submaximal exercises however, it is still the prevailing view that oral breathing presents the most efficient method of breathing during high-to-maximal intensity exercises (Lacomb et al., 2017; Dallam & Kies, 2020).

A large reasoning for the argument that mouth breathing may be more efficient at maximal intensities is due to the greater ability to dispel  $CO_2$  from the body at these intensities. During these intensities the body begins to utilize a greater amount of anaerobic oxidation pathways, resulting in the accumulation of a greater number of metabolic by-products in the blood such as  $H^+$  ions; these cause a left shift in the carbonic acid-bicarbonate buffering system. While this accumulation does initially promote greater oxygen off-loading at the tissues, eventually by-products accumulate faster than they can be removed and begin to cause metabolic

acidosis (Kenney et al., 2015). Breathing out CO<sub>2</sub> helps to alleviate this acidosis and it has been found that during a maximal GXT oral breathing had a significantly higher  $\dot{V}CO_2$  compared to the nose breathing condition (Dallam et al., 2018). Despite this however, in the study by Dallam et al., (2018) the time to exhaustion of the GXT did not differ significantly and the NB condition had a slightly higher mean time (428±24 s vs 421±18 s, p=0.74). One study by Recinto et al., (2017) found that there was no difference between NB and MB during a 30 s Wingate test in terms of power output or mean power. This study only looked at one Wingate test in each condition and did not perform lactate measurements or measure end tidal CO<sub>2</sub> and therefore can only indicate that NB is equally as effective as MB during a 30s maximal anaerobic intensity. More research is needed on the effects of NB on metabolic acidosis during maximal intensity exercises and during repeated maximal efforts.

### **Environmental considerations**

A common symptom following exercise, especially aerobic endurance exercise is coughing or wheezing, known as exercise-induced bronchospasms (EIB). EIB has negative effects on both athletic performance and on overall pulmonary health (Üçok et al., 2004; Kennedy et al., 2020) During hyperventilation, heat and water are lost from the airways which can result in irritation and are main factors in the pathophysiology of EIB (Üçok et al., 2004). EIB has been cited as being similar to the symptoms of asthma in that the lower airway diameter is decreased due to an inflammation response (Kennedy et al., 2020). Symptoms usually begin shortly after the cessation of exercise and can persist for several hours; symptoms include coughing, wheezing, dyspnea, and excessive mucus (Üçok et al., 2004). A diagnosis of EIB can be performed through a pulmonary test which finds a decreased forced expiratory volume (*FEV*) by  $\geq 10\%$  (Üçok et al, 2004).

EIB is especially prevalent in elite and regularly active populations due to habitual exposure to the inducing stimulus, with one study finding that 7/20 endurance runners exhibited signs of EIB following a bout of exercise whereas only 1/19 sedentary controls showed signs of EIB (Üçök et al., 2004). EIB can occur in both asthmatic populations and healthy populations, with the prevalence of EIB being estimated as approximately 30-40% in habitually active individuals (Kennedy et al., 2020) and is reported to be as high as 75% in asthmatic populations (Hendrickson et al., 1993).

Cold weather has shown to worsen the symptoms of EIB due to the extra moisture and heat lost when breathing the cold, dry air (Kennedy et al., 2020). Winter endurance sport athletes, such as nordic skiers, who breath large volumes of cold air for long periods of time have a higher prevalence of asthma compared to alpine skiers training in relatively the same conditions (15% versus 4%, respectively) and exhibit higher symptoms of EIB; with up to 86% of nordic skiers reporting coughing following strenuous exercise (Kippelen et al., 2012).

Nose breathing, which has been shown to have greater temperature and humidity regulation than mouth breathing, has been generally dismissed for the treatment of EIB in cold weather endurance sports due to the belief that it is impractical for use at high-level exercise (Kippelen et al., 2012). Despite this, it has been shown that NB was effective in decreasing the *FEV* reduction following exercise compared to oral breathing in asthmatic individuals who do experience EIB (EIB+), asthmatics that do not experience EIB (EIB-), and controls (Mangla & Menon, 1981). Furthermore, Mangla & Menon (1981) found that the EIB+ individuals all (n=5) developed wheezing symptoms following the MB while none of them developed any wheezing following the NB trial. No differences in blood pressure or pulse rate were observed between NB and MB however no other performance measures were taken (Mangla & Menon, 1981).

The reduction of EIB may also have positive effects on mood and motivation following aerobic exercise. Talbott & Talbott (2009) found that marathon runners post-marathon taking supplements designed to reduce upper respiratory tract infection symptoms showed improved positive affect scores (such as vigor) and a reduction in negative affect scores (such as tension, fatigue, and confusion) on a POMS assessment. More research is needed into the use of NB to decrease the symptoms of EIB in sport populations, and especially in winter sport populations. The effects of NB on performance in these winter populations must also be researched further as there is a gap in the literature. Currently there are guidelines regarding cold winter sports, such as nordic skiing, having temperature cut-offs for certain distance races (-16°C for 30+ Km and -20°C for sprints) which were originally implemented to protect against extreme cold (Kippelen et al, 2012).

### **Chapter 3: Methods**

#### **Study design and context:**

This study employed a concurrent mixed methods case study design (Creswell, 2003) with a single participant, in which the participant was also the researcher. For the purpose of this thesis, both quantitative and qualitative data collection was employed with equal weighting. Multiple forms of data collection was used including objective data gathering through GPS, heart rate, and pacing; and closed ended as well as open ended subjective measures were used such as subjective rating scales and an unstructures daily journal. This allowed for the incorporation of both predetermined and emerging methods to be used to explore the quantitative and qualitative data together (Creswell, 2003).

The objectives of this research were (1) determine the feasibility of completing the vigorous NB program as a previously unaccustomed-to-NB runner, (2) determine the physiological and performance effects of the program, and (3) describe the subjective experience of the nose-breathing training program. Quantitative and qualitative data was recorded using various measures and integrated for analysis. This particular study design was chosen as it highlighted important aspects which were missing from the other nose breathing studies. A case study allowed for us to work within the research restrictions that were in place in Canada and at Nipissing University during the COVID-19 pandemic throughout 2020 and during the beginning of 2021. Being a recently retired varsity cross-country runner of 5 years, I decided to conduct the running training and performance case-study on myself in order to achieve this pragmatic and COVID-19 safe research design. I chose nose breathing as my topic of investigation after listening to James Nestor on a podcast and subsequently reading his book “Breath: The New Science of a Lost Art”, which highlighted the potential health and, more interestingly, the

potential athletic performance benefits of nose breathing. As a runner, I was both interested and skeptical about the claims by Nestor, and others in the nose breathing camp, that the same athletic performance could be achieved while nose breathing as with mouth breathing following an unspecified adjustment period. Therefore, I decided to conduct a performance-based study on myself outside of the laboratory in a real-world, ecologically valid setting. As such, I was limited in my access to measuring devices, using only a GPS watch and an accelerometer, which accompanied my performance and subjective measures.

As both the participant and researcher I had the unique task of setting aside any preconceived notions which I may have gathered through both my past running history and during my review of the literature on the subject. To combat this, I tried to reflect on my own ideals regarding the outcome of the study and was completely honest in all my recounting of experiences. As such, the subjective daily journal entries were written in a non-analytical fashion, following an unstructured format which allowed me to explore my subjective experience as the participant. Reflexivity was practised throughout the training program in order to be self-critical regarding the introduction of biases (Korstjens & Moser, 2018).

Regarding the performance measures, both the daily run and test day performances; I am naturally competitive and attempted to perform to the best of my abilities in each run and test. Motivationally, I had no desire either way to prove or disprove the claims of James Nestor. I was interested by the idea, immediately skeptical, and moderately annoyed by Nestor's portrayal of anecdotal and pseudo-scientific claims as fact; therefore, I was equally satisfied if my results helped to support or contradict his claims. The minimal literature regarding nose breathing training indicated that there was an approximate 6-month adaptation period but did not provide any details about the training or subjective experiences of the runners. The recreational nature

(Dallam et al., 2018) and older age (Hostetter et al., 2017) of the participants in these studies did lead me to believe that I would not take this long to adapt to nose breathing. As such, I had no real concept of what the subjective experience of the 6-week nose breathing training program would be like. This study received ethical clearance from the Nipissing University Research Ethics Board on 18 January 2021. The testing period of this study was conducted between 19 January 2021 and 30 March 2021 and was run on a three-kilometre loop through my neighborhood in downtown North Bay, ON.

**Purpose:**

The purpose of this case study was threefold: First, to determine if a trained runner can complete a six-week, nose-breathing-only ‘interval-based’ training program comparable to what a competitive runner would undertake in preparing for a 5 or 10 km race. That is, the interval-based training program was designed to be rigorous and to push the limits on what is possible to achieve while nose breathing; remaining healthy, maintaining the prescribed running intensity (verified through HR and subjective evaluation), and completing  $\geq 95\%$  of the daily run days (51/54 days) and all (3) test runs would be considered successful completion of the program. Second, to measure the related physiological (e.g. heart rate) and performance (e.g. 5kmTT) changes that occurred during the nose-breathing training program. Third, describe the subjective experience of undertaking a nose breathing training program with special emphasis on describing the dyspnea sensation; it’s nature, severity, and its effects on my mood and motivations throughout the running program.

### **Hypothesis:**

I hypothesized that it would be an attainable goal to be able to complete a six-week interval-based running training program while nose breathing. Specifically, based on the existing literature, I hypothesized:

1. Heart rate would be higher during the initial stage of the nasal breathing runs, compared to mouth breathing of equal velocity, and that the heart rate would lower during the later stages of the nose breathing training;
2. The commencement of the nose breathing only training program would result in decreased heart rate variability (*HRV*), decreased profile of mood states (*POMS*), and increased ratings of perceived exertion (*RPE*);
3. There would be an improved (longer) breath-hold time during voluntary breath hold tests following the nose breathing training;
4. There would be a delayed voluntary nasal switching point, that is, a switch from nose breathing to mouth breathing occurring at higher intensity/pace of running during a graded exercise test, with nose breathing training; and,
5. Finally, I hypothesized that performance in the 5km time trial, the main outcome measure, would be improved following the nose breathing training program.

### **Participant:**

The participant in this case study was one male (N=1); aged 23 years old, 86 kg, and 1.83 m. The participant is an experienced runner with 5 years of running experience with the Nipissing University varsity cross-country running team and at the time prior to the commencement of the experiment had been running regularly (3-4x per week), although not following any specific running program/plan. Typical training runs involve a duration of 40-

80minutes of running at a self-reported “hard but sustainable pace” which represents a 15-16 on a 6-20 *RPE* scale.

### **Procedures/timeline:**

Figure 1 provides an overall summary of the experimental timeline of this study, which began with a three-week Mouth Breathing Phase (MB wk 1 through MB wk 3) that consisted of daily runs at a moderate intensity (RPE of 16/20). The Mouth Breathing Phase was used to establish a steady base of running-related fitness in the participant, to establish baseline testing measures, and also to ensure the participant was physically prepared for the rigorous training that would follow during the Nose Breathing Phase. Thus, at the end of the Mouth Breathing Phase and prior to the start of the Nose Breathing Phase (PRE), the following tests were conducted: a 5 km running time trial (5kmTT) to determine running performance, a graded exercise test (GXT) to determine voluntary switching point from NB to MB, and a breath hold test (BHT).

The six-week Nose Breathing Phase (NB wk 1 through NB wk 6) followed the Mouth Breathing Phase, and consisted of the same daily running training protocol, including the same route and RPE clamp as the baseline section, but with the replacement of two daily runs with two higher intensity intervals each week. Mid-way through the Nose Breathing Phase (MID), and following the conclusion of the six-week phase (POST), the 5kmTT, GXT, and BHT were repeated.

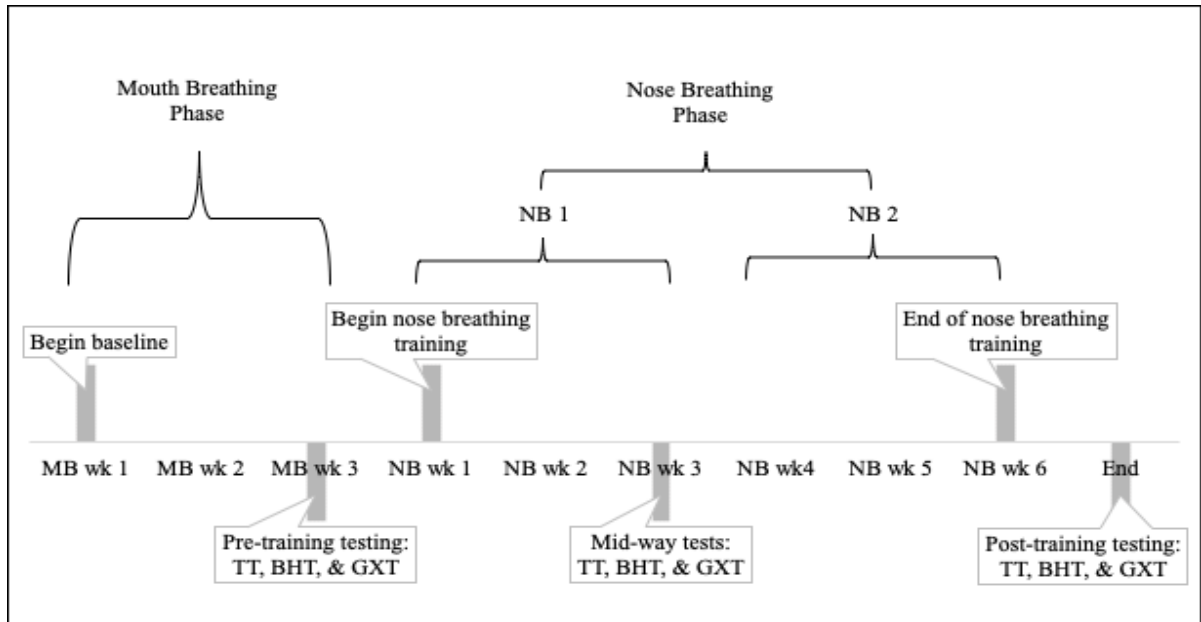


Figure. 1. Timeline of nose breathing case study.

**Protocols:**

**Daily runs:**

Each day of the study, a wrist-based resting heart rate variability (*HRV*) measurement was taken immediately after waking, prior to getting out of bed, using a Garmin Forerunner 745 device and the Garmin-made *HRV* app, “Test HRV” which measures *HRV* in a three-minute test. Heart rate variability, while not yet universally accepted, can serve as an indicator of overall stress levels in the body (Kim et al., 2018), and it may provide further insight into the stress experienced by the participant due to the training program. The resting respiration rate (*RR*) estimate was measured using the Garmin Forerunner 745 during this time as well.

The daily run was completed each day at approximately 9:30am. I took one rest day per week. Prior to each daily run, the 40-question abbreviated profile of mood state (*POMS*) scale (Grove & Prapavessis, 1992) was conducted. The 40 question *POMS* measures 5 negative affect categories and 2 positive affect categories with each question measuring on a 0-4 scale being connected to one specific category. The daily *POMS-40* questionnaire was measured as:  $((\text{tension} + \text{depression} + \text{anger} + \text{fatigue} + \text{confusion}) - (\text{esteem} + \text{vigour})) + 100$ , with a higher value indicating a worse mood state score (*POMS40* scoring range: 56-216) *POMS* has been shown to be a valid psychometric measurement in athletically competitive populations from adolescence to adulthood (Terry, Lane, & Fogarty 2003).



Dynamics Pod uses accelerometry to estimate stride parameters such as stride length, vertical oscillation, vertical ratio, cadence, ground contact time, and ground contact balance. Garmin wearable devices have been shown to have measured heart rate within 3% in controlled laboratory settings (Fuller et al., 2020). Garmin devices ranged from -3.2-17.7% mean absolute percentage error (MAPE) for outdoor covered distance with the Forerunner model (920XT) showing a MAPE of 9.7%, indicating a slight tendency to over-estimate distance (Wahl et al., 2017).

Twice weekly during the Nose Breathing Phase, and separated by at least 48 hours, the participant replaced the daily run as described above with a vigorous-intensity interval workout. The intervals were designed to progressively become more challenging and therefore increased in volume after successful completion of the interval workout session on two occasions. The progression in the interval workout durations (high intensity efforts) was as follows:

- 1: 4x4min
- 2: 4x5min
- 3: 5x4.5min
- 4: 5x5min
- 5: 6x5min
- 6: 7x5min

Following each effort, the period until the next effort consisted of active recovery (i.e. a slow jog) for a duration equal to 1.5 times the vigorous-intensity effort duration. The pace of the high-intensity efforts was based on the 5km time trial time, which was imputed into the Jack Daniels VDot Running Calculator (<https://runsmartproject.com/calculator/>), and from which the ‘threshold’ pace was utilized as the interval effort pace. The ‘threshold’ pace is indicated by

Daniels (2014) to have the purpose of increasing endurance, improving the body's lactate clearance, and is calculated to represent an intensity of 88-92% of maximal HR. Under normal breathing conditions this pace should be “comfortably hard, which means you are definitely working relatively hard, but the pace is manageable for a fairly long time” (Daniel, 2014), meaning that the threshold was a hard effort but not a sprint.

Successful completion of each interval workout session was defined as the completion of all intervals at  $\pm 5$  s from the suggested pace, no more than 10 s of mouth breathing following each interval, and no mouth breathing during the running portion of the interval workout. The interval pace was not adjusted following the mid-way time trial during the third week; this was due to the already increasing workload within the training program.

Following each daily running session, a detailed journal was kept, detailing the general feelings for the day, how I felt during the run, if this run felt any different from a normal run or from the previous day. Diaries and logs have been cited as widely used subjective measures that are useful in measuring the type and context of physical activity or sedentary behaviour (Bakker et al., 2020). From the journal, incidents of any coughing, sore throat, or other signs of exercise induced bronchospasms were tracked. Additionally, incidents were tracked for mentions of feeling tired/rundown/cranky/“not feeling it”, mentions of sore muscles/feeling tight/aches/pains, and finally incidents of overwhelming dyspnea were also monitored. For the dyspnea frequency measurement, only mentions of struggling with dyspnea, dyspnea being an issue, or mentions of feeling overwhelmed by dyspnea were counted; mentions of dyspnea being on my mind but not being an issue or feeling “comfortably uncomfortable” was not counted in the quantitative measures but was considered in the qualitative analyses.

Pulse pace was measured following each daily run by dividing the average heart rate, in beats per minute, by the average pace, in seconds per kilometer. The pulse pace provides a measure of fitness which can help account for variations in weather and traction which may have affected pace by accounting for the intensities, given by the average heart rate, as a ratio of the pace, the actual output. A lower pulse pace value indicates a slower pace (more seconds per kilometer) for a given heart rate.

The training impact (*TRIMP*) measurement was based on the exercise *HR* and duration (in minutes) in order to quantify training load beyond just time and distance, and when used with *HRV* and resting *HR*, can help to monitor fitness and fatigue (Wallace, Slattery, & Coutts, 2014). This measurement was taken using the *TRIMP*-meter App offered by the Garmin ConnectIQ online store. This allowed for the tracking of internal workload throughout the training program. The *TRIMP* method, proposed by Banister (1991) is a method of quantifying internal training load. This method was found to be a model of internal training load more highly correlated to performance measures (a 1500m time trial) than the *sRPE* method proposed by Herman et al. (2006) (Wallace, Slattery, & Coutts, 2014).

### **5km time trial (5kmTT):**

The time trials were ran using a typical oronasal breathing approach as the goal of the study was to assess performance improvement during a maximal 5km effort rather than measure the maximal effort achievable only while nose breathing. The goal of the training program was to adhere to a NB training program that is comparable to one that a competitive runner may follow leading up to a 5 or 10km race. Oronasal breathing during the time trials allowed us to better assess fitness improvement as it removed potential learning curve that may have come

with a maximal nose breathing effort as well improve the ability to compare between other training programs.

Each of the three regular breathing 5km time trials consisted of 5 laps of a 1 km loop, ran in a counter-clockwise direction through my neighborhood. The time trials were all conducted at the same time of day, within 1 hour of each other. I was naïve to the elapsed time but was aware of the distance each kilometre due to the GPS watch beeping. While the Garmin Forerunner 745 does display the kilometre split time, I did not look at any point during the three trials. Time trials have been demonstrated to be ecologically valid and a reliable approach to assess endurance performance with a low intrasubject variability regarding time, *HR*, and *RPE*; and they have found to be a valid assessment of ergogenic aids and training interventions (Fisher et al. 2017). *POMS* and resting *HR* were measured prior to each 5kmTT. Following completion, a *RPE* measurement was taken, a subjective traction score, and journal entry were recorded.

### **Voluntary switching point GXT:**

The voluntary switching point GXT measured the pace at which the I could no longer maintain nose breathing only while running and switched to mouth breathing. These tests were all conducted in the mornings prior to the evening 5kmTT's. The graded exercise test started at a pace of 5:00 min•km<sup>-1</sup> and increased by 10 s•km<sup>-1</sup> every 500 m. Pace continued to increase every 500 m until I could no longer maintain the pace with nose only breathing and switched to oral breathing. At this point, the final pace (s•km<sup>-1</sup>) and total distance ran (m) was recorded. This is a modified version of the method by Lacombe (2015) who determined the voluntary switching point during a GXT  $\dot{V}O_{2peak}$  test on a treadmill.

**Breath hold test (BHT):**

The breath hold tests consisted of an above water apnea following an exhalation while being naïve to the time. The breath hold tests took place in the morning in a seated position and were repeated three times per session (PRE, MID, & POST) resulting in 9 breath holds in total. I ensured that I was calm and had been seated for 5 minutes prior to the tests. I breathed normally prior to the BHT; specifically trying to not alter breathing. After a normal exhalation I began holding my breath and the time started; the time was stopped when I inhaled once again (Sutterlin et al., 2013). I reminded myself to hold my breath “as long as you can, even if you have the urge to breathe” prior to each breath hold trial (Sutterlin et al., 2013). The timer was held by my roommate who was briefed on the protocol and practiced prior to the first trial. The involuntary breathing movements (IBM’s) were self-monitored by holding a hand on my abdomen. When I felt my diaphragm begin to contract involuntarily, I would give a “thumbs up” and the time was recorded by my roommate. I waited 10 minutes between trials. The Breath hold tests were the only section of this study that required a second person to be present. This contact was necessary due to the potential, however very low, of losing consciousness during the breath hold test and was a necessary safety precaution. In fact, it has been noted by that “it is practically impossible for a [person] at sea level to voluntarily hold [their] breath until [they] become unconscious” (Schnieder, 1930) and further research has supported this in healthy adults (Parkes, 2006). Despite this low risk, the observer was positioned close enough to me in order to provide immediate support should I have shown signs of fainting/dizziness such as flushing of the face, sweating, or loss of balance. Should I have felt any of these dizziness symptoms I was to begin breathing immediately; this is in congruence with the safety precautions laid out by Skow et al. (2015) for breath holding. This outcome measure was designed to provide insight into the body’s

tolerance to CO<sub>2</sub> accumulation, which is cited as the driving force for higher respiration rates (Lacomb, 2015) with lower breathing rates, as seen in nose breathing compared to oral breathing, showing a reduced chemoreflex in response to hypercapnia (Russo et al., 2017).

### **Statistical analysis:**

Daily run data was analyzed using a repeated measures ANOVA with a Greenhouse-Geisser correction. If a statistically significant difference was found, a Bonferroni correction for multiple comparisons post hoc test was used. Due to the addition of two interval workouts during the nose breathing phase, week-week comparisons between the baseline MB and the weekly NB was not immediately possible with repeated measures. With only 4 standard daily runs per week in the nose breathing section, consecutive four-day averages were taken from the control phase in order to compare the daily runs of the control phase with that of the weekly nose breathing. Measures prepared in this manner include the daily run *Pace*, *HR*, and *Pulse Pace*. Other measures including *TRIMP*, *POMS* and *HRV*, included interval days in their measures. As such, 3-day consecutive averages were taken for these measures to compare the 3-week control phase to the weekly nose breathing averages.

Performance values including the 5kmTT, GXT, and BHT were not statistically analyzed due to the single participant and limited number of data points. Performance data is instead presented graphically and in percent (%) change between the PRE, MID, & POST time points.

### **Qualitative data analysis**

As I was both the researcher and participant I was aware that I needed to remain neutral in the qualitative journal entries as to avoid introducing any pre-existing biases. Being the

participant I knew it would be difficult to be objective when analyzing the qualitative data. To combat this, I practiced continuous reflexivity and the journal entries were written as if I was keeping a regular diary. The entries were unstructured; I wrote what I felt, and I wrote in a descriptive rather than an analytical fashion. Additionally, as this was a concurrent mixed methods study design, quantitative data such as heart rate, pacing, and the subjective POMS score were all incorporated into the journal entries as required to aid in completely and accurately describing my subjective experiences (Creswell, 2003).

Rigour was ensured for through continuous reflexivity, systematic theme analysis of the journal entries including entry summary and compiling of concurring and discrepant experiences, and peer debriefing following the identification of potential themes. The qualitative data collected from the daily log/journal entries was analyzed following the completion of the training program. The dyspnea sensation experience was analysed thematically and the aspects of general health and wellbeing were analysed using a content analysis in order to provide an indication as to the frequency of different subjective experiences regarding general health over time during the training program (Vaismoradi et al., 2013). The goal of the thematic analysis was to shed light on the subjective dyspnea experience during a nose breathing restricted running training program. The content theme analysis hoped to only provide a rough estimate for different subjective experiences throughout the program and did not seek to draw any significance from these measures (Vaismoradi et al., 2013).

## Chapter 4: Results

### Daily run performance:

Figure 2 illustrates the change in average pace during the daily runs, from the mouth breathing period (MB) compared to weeks #1 through 6 of the nose breathing training (week 1 through week 6). There was a statistically significant difference in the pace of the daily runs between time points ( $F_{(1.78, 5.35)} = 22.965$ ,  $p = 0.003$ ). Specifically, post hoc testing revealed that running pace was significantly slowed (increased  $s \cdot km^{-1}$ ) in week 1, compared to the MB period. (mean  $\pm$  CI 95%) ( $M = 259.75 \pm 2.72$  vs.  $M = 245.50 \pm 1.49$ ,  $p = 0.015$ ). Further, the pace during week 1 of nose breathing was significantly slower than weeks 4 & 6 ( $M = 236.75 \pm 4.75$ ,  $p = 0.035$  &  $M = 228.00 \pm 5.51$ ,  $p = 0.014$ ; respectively). Finally, the average pace of week 6 was significantly faster than MB ( $M = 228.00 \pm 5.51$  vs.  $M = 245.50 \pm 1.49$ ,  $p = 0.041$ ); no significant differences were found in any of the other post hoc tests.

Figure 3 describes the average heart rate recorded during the daily runs of mouth breathing period and the nose-breathing training. No significant differences were found in mean *HR* across the 7 time points ( $F_{(1.44, 4.33)} = 2.376$ ,  $p > 0.05$ ). When heart rate and daily running pace were analyzed together as pulse pace (Figure 4), there was a significant difference across the 7 time points ( $F_{(1.67, 4.97)} = 20.45$ ,  $p = 0.005$ ). Post hoc testing showed a significantly lower pulse pace (i.e., there was a decreased running pace per bpm of heart rate) during week 1 ( $M = 0.645$ ,  $CI = 0.028$ ) compared to week 4 ( $M = 0.728$ ,  $CI = 0.033$ ,  $p = 0.034$ ) and week 6 ( $M = 0.758$ ,  $CI = 0.016$ ,  $p = 0.043$ ). No other comparisons achieved significance.

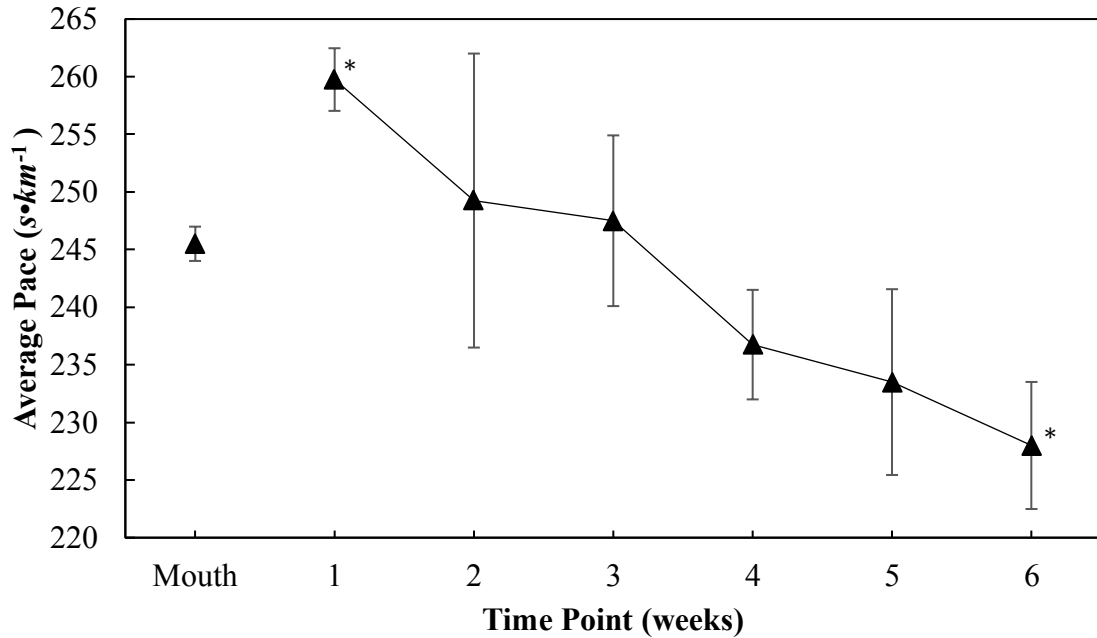


Figure 2. Three-week control phase (mouth) pace compared to nose breathing weekly average (error bars indicate 95% confidence intervals). \* indicates significance compared to Mouth ( $p<0.05$ ).

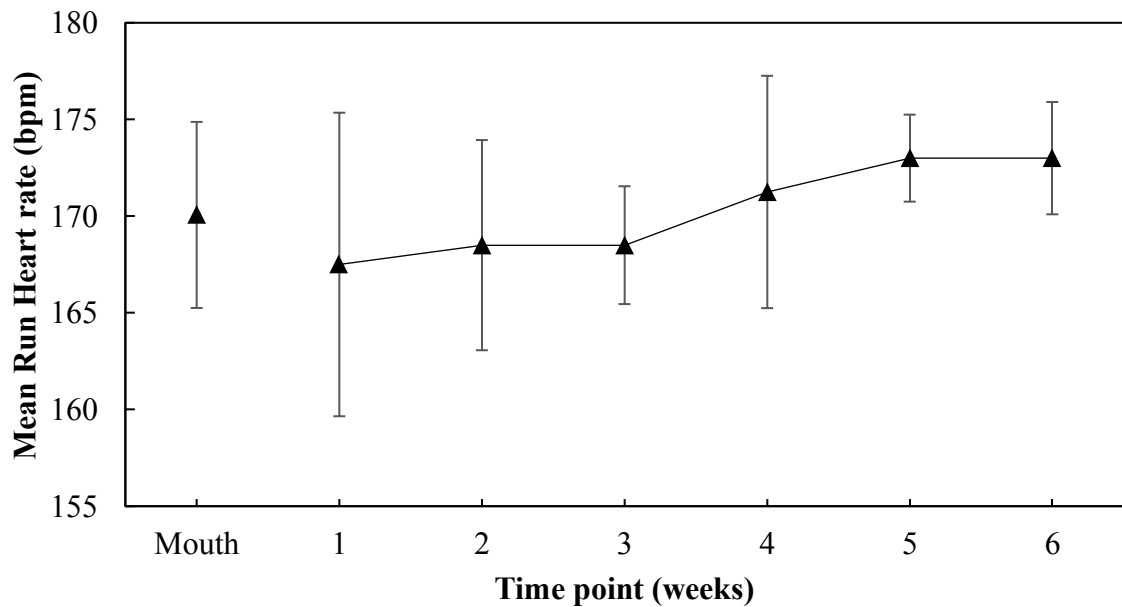


Figure 3. Three-week control phase (mouth) average run heart rate compared to nose breathing weekly average (error bars indicate 95% confidence intervals).

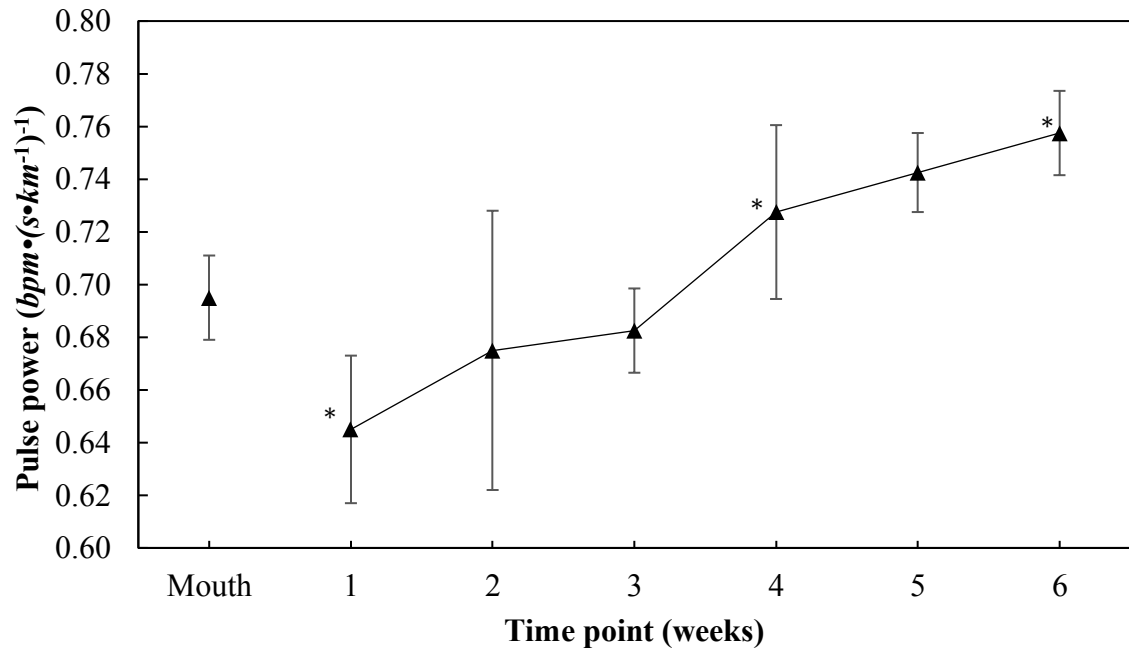


Figure 4. Pulse pace ( $HR \cdot Pace^{-1}$ ) average for three-week control (mouth) compared to the nose breathing weekly average. (error bars indicate 95% confidence intervals).

**Performance measures:**

In figure 5 the change in 5km running time trial performance over the course of the study is described. The three data points (PRE=17:27, MID=16:39, and POST=16:22), suggest a downward trend over time (i.e. improved 5km running performance), however statistical comparison between measures was not possible due to the single measurement points. Traction scores, noted by the researcher for each 5km time trial effort, were similar between PRE, MID, and POST (9, 9, & 10/10, respectively). Figure 6 shows the pacing profile per kilometre of the PRE, MID, and POST 5kmTT's. The pacing profiles of the PRE, MID, & POST time trials were approximately the same showing the fastest kilometre pace in the first kilometre (3:16, 3:09, & 3:00, respectively), the second-fastest kilometre pace in the second kilometre (3:27, 3:18, & 3:13, respectively), the third-fastest kilometre pace in the final kilometre (3:32, 3:22, & 3:18, respectively) and the slowest kilometres occurred during the 3<sup>rd</sup> and 4<sup>th</sup> kilometres. The average kilometre pace for the PRE, MID, & POST 5kmTT was 3:29, 3:20, & 3:16, respectively.

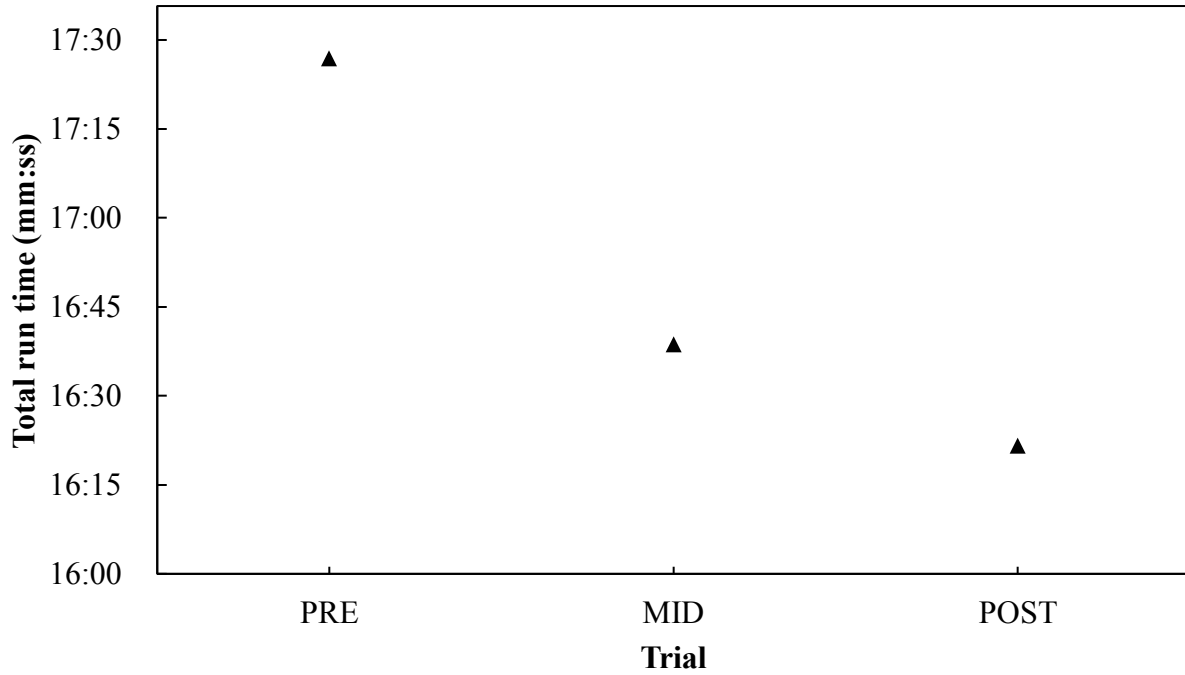


Figure 5. 5kmTT total run times.

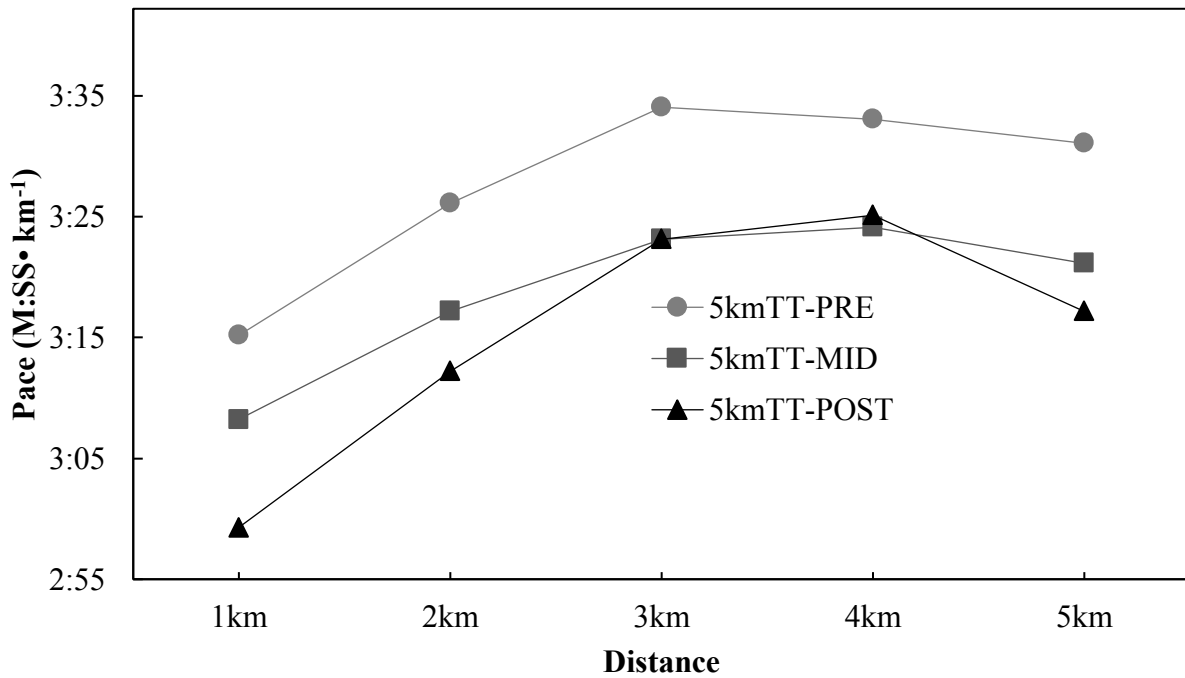


Figure 6. 5KmTT pace per kilometre.

Figures 7 and 8 describe the results of the GXT performed at the PRE, MID, and POST time points to determine the voluntary switching point between NB and MB. The pace at which I could no longer sustain NB increased from 3:50 min • km<sup>-1</sup> in PRE, to 3:30 min • km<sup>-1</sup> in MID and 3:20 min • km<sup>-1</sup> in POST (Figure 8). Thus, the voluntary switching point from NB to MB in the GXT showed a 23% increase between the PRE-MID tests, improving from 1093 s to 1343 s (Figure 7), and improved a further 1% at the POST testing with 1357 s. Due to the limited number of measurements, statistical comparisons of these measures was not possible.

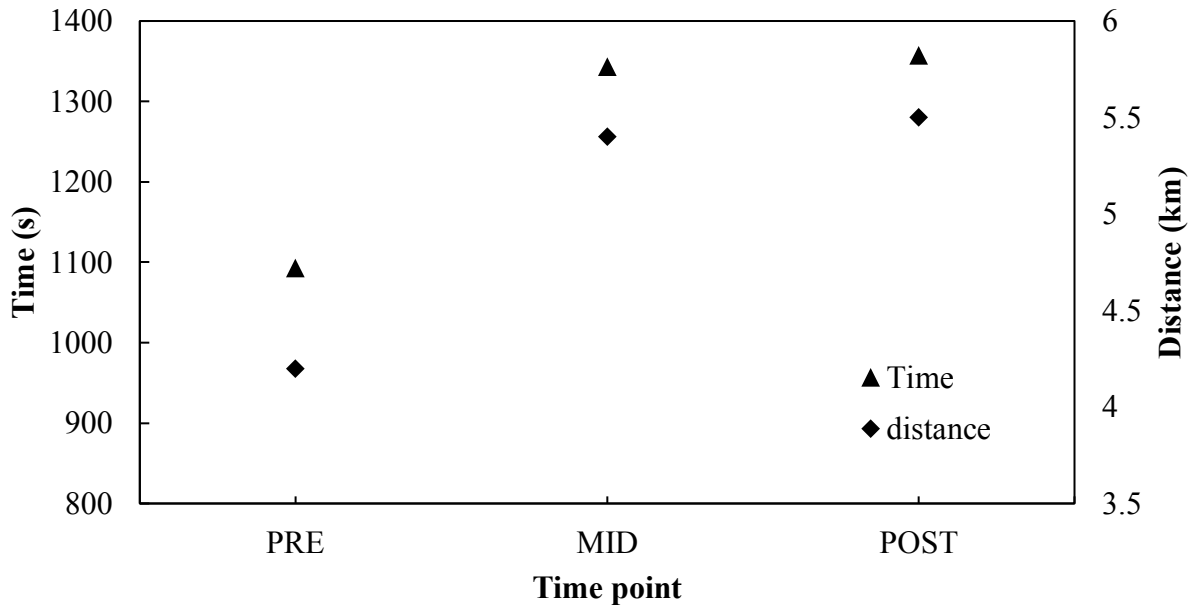


Figure 7. Voluntary switching point graded exercise test

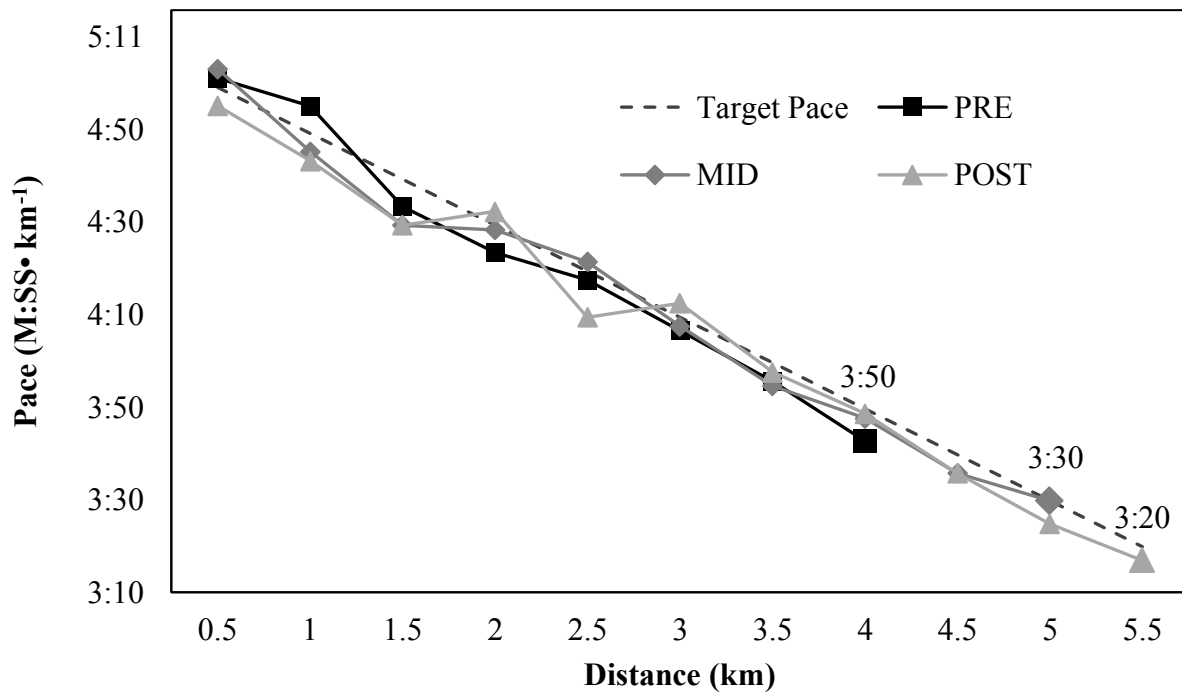


Figure 8. Pacing profile of voluntary switching GXT and final achieved pace prior to switching to mouth breathing.

Figure 9 describes the results of the voluntary breath hold tests. The onset of IBM's showed no significant difference across the PRE, MID, and POST trials ( $F_{(1,4)}=2.782, p>0.05$ ). The longest POST BHT compared to the longest PRE BHT showed a 34.1s increase. This represents a 77.0% improvement between the PRE and POST maximum values. Between PRE and MID showed a 53.1% improvement, and MID-POST showed a further 15.6% improvement. Due to the limited number of measurements, statistical comparisons of these measures was not possible.

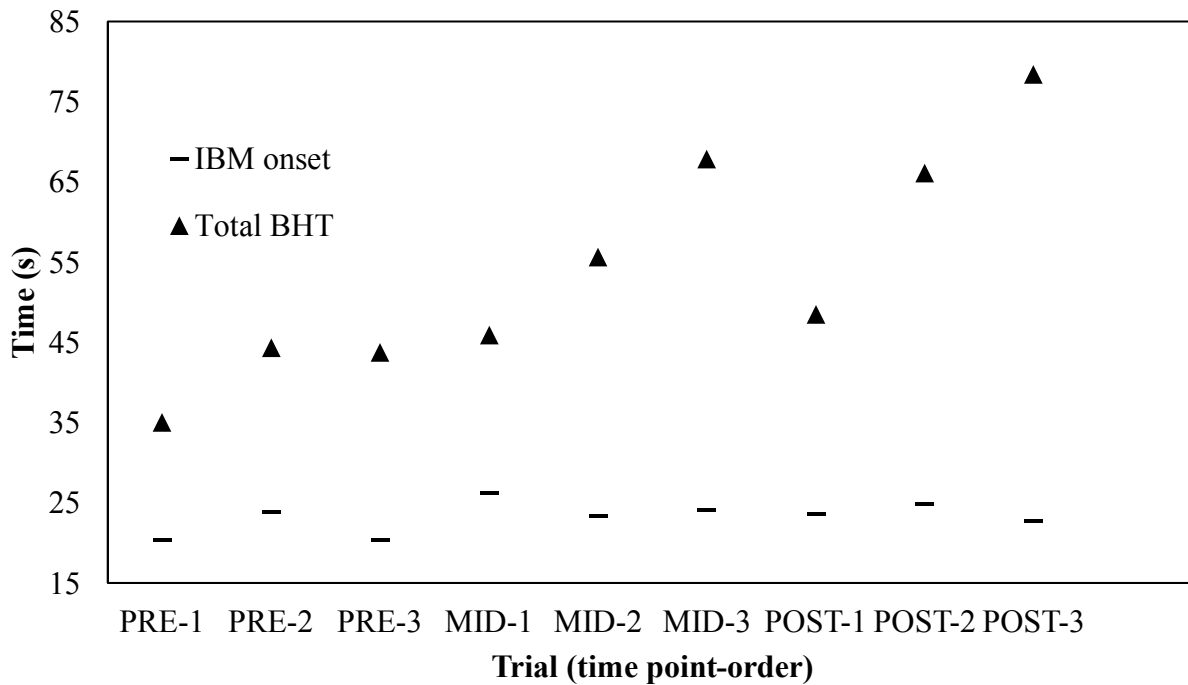


Figure 9. Breath hold tests (x3) per trial. Total time and involuntary breathing movements onset.

### **Subjective, physiological, and biomechanical measures:**

Figure 10 describes the change in profile of mood states scores over the nose-breathing training; the weekly *POMS* scores showed no statistical differences between any time points ( $F_{(2,30, 11.49)}=0.867, p=0.46$ ). The *POMS* scores indicate that I was in a generally good mood prior to the daily runs consistently throughout the program with an overall average of 70.0 (score ranging from 56-216 with lower numbers indicating improved mood state). The *POMS* scores were also not significantly different between the MB phase (MB), the first half of NB phase (NB 1), and the second half of the NB phase (NB 2) ( $F_{(1,70, 28.54)}=1.31, p=0.28$ ). Similarly, there was no significant difference found in run RPE when compared between the three phases: MB, NB1, NB 2 ( $F_{(1,77, 19.46)}=0.092, p>0.05$ ) (Figure 11).

The prevalence of various factors such as post-run coughing, feelings of tiredness, large sensations of dyspnea, and feeling sore which were recorded following each run in the daily journal are shown in table 1. Post run coughing had a decreased prevalence over time; being prevalent in 72.2% (13/18) of the MB runs, decreasing to 11.1% (2/18) in NB 1, and extinguishing to 0.0% (0/18) in NB 2. Overwhelming dyspnea increased from nonexistent (0/18 runs) during MB to 9/18 runs during NB 1 (0.0% vs 50.0%, respectively) and decreased in NB 2 with only 2/18 runs experiencing dyspnea (11.1%). The sensation of general tiredness generally increased over time compared over the three phases (5.5% vs. 16.7% vs. 38.9% respectively) while the feelings of tightness/soreness began high during MB at a prevalence of 27.8%, decreased to 11.1% during NB 1, and increased slightly to 16.7% in NB 2 (5, 2, & 3/18, respectively).

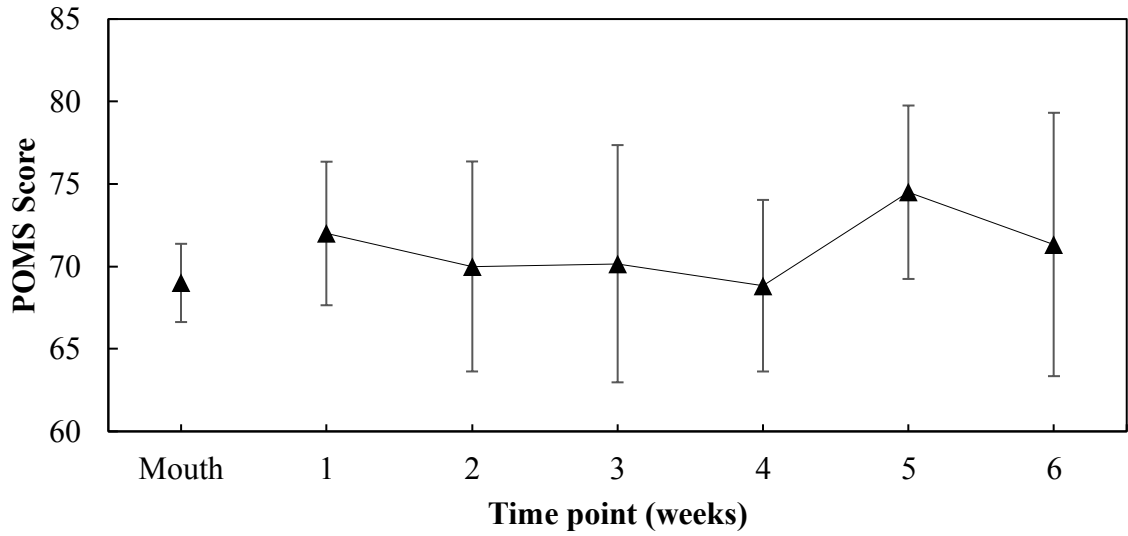


Figure 10. Three-week control phase (mouth) average *POMS* scores compared to nose breathing weekly average (error bars indicate 95% confidence intervals). Scoring range for POMS40: 56-216.

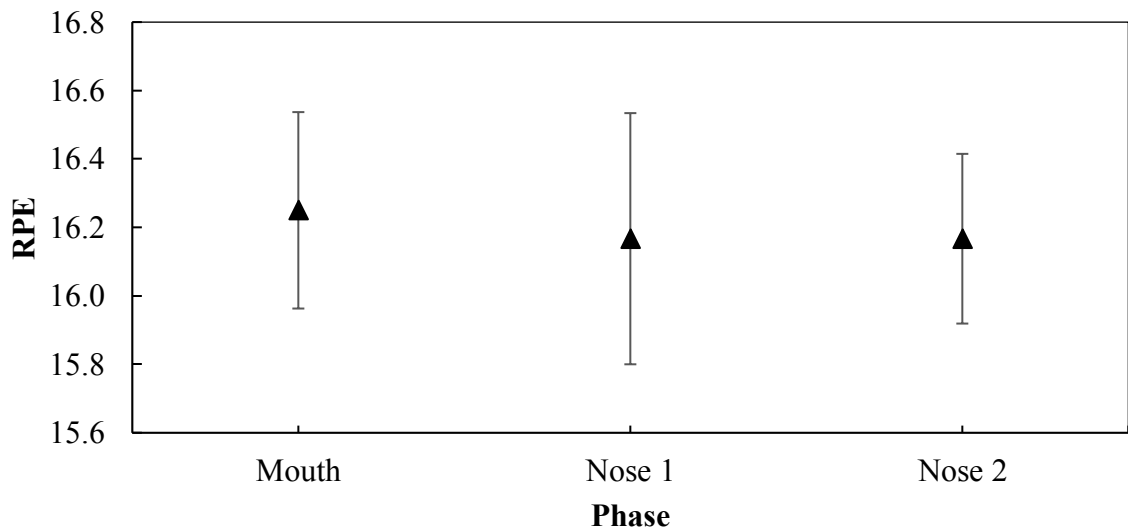


Figure 11. Ratings of Perceived Exertion compared over three phases (error bars indicate 95% confidence intervals).

Table 1. Frequency of comments in Daily Journal regarding negatively perceived factors

	<b>Mouth /18</b>	<b>Nose 1 /18</b>	<b>Nose 2 /18</b>
<b>Post-run coughing</b>	13	2	0
<b>Feeling tired/rundown</b>	1	3	7
<b>Large sense of dyspnea</b>	0	9	2
<b>Sore/tight</b>	5	2	3

When considered in the three phases (MB, NB 1, NB 2), *TRIMP* (Figure 12) showed a significant difference over time between the three, 3-week sections ( $F_{(1.47,24.96)} = 10.842$ ,  $p=0.001$ ). Post Hoc tests revealed that there was a significant difference between MB and NB 2 ( $101.58 \pm 8.21$  vs  $122.27 \pm 25.18$ ,  $p=0.022$ ) and between NB 1 and NB 2 ( $97.87 \pm 9.18$  vs.  $122.27 \pm 25.18$ ,  $p=0.002$ ) however no statistical difference between MB and NB 1 ( $p>0.05$ ). When considered weekly however, *TRIMP* showed no significant difference over time compared to MB ( $F_{(1.41,7.04)} = 4.641$ ,  $p>0.05$ ) (figure 13).

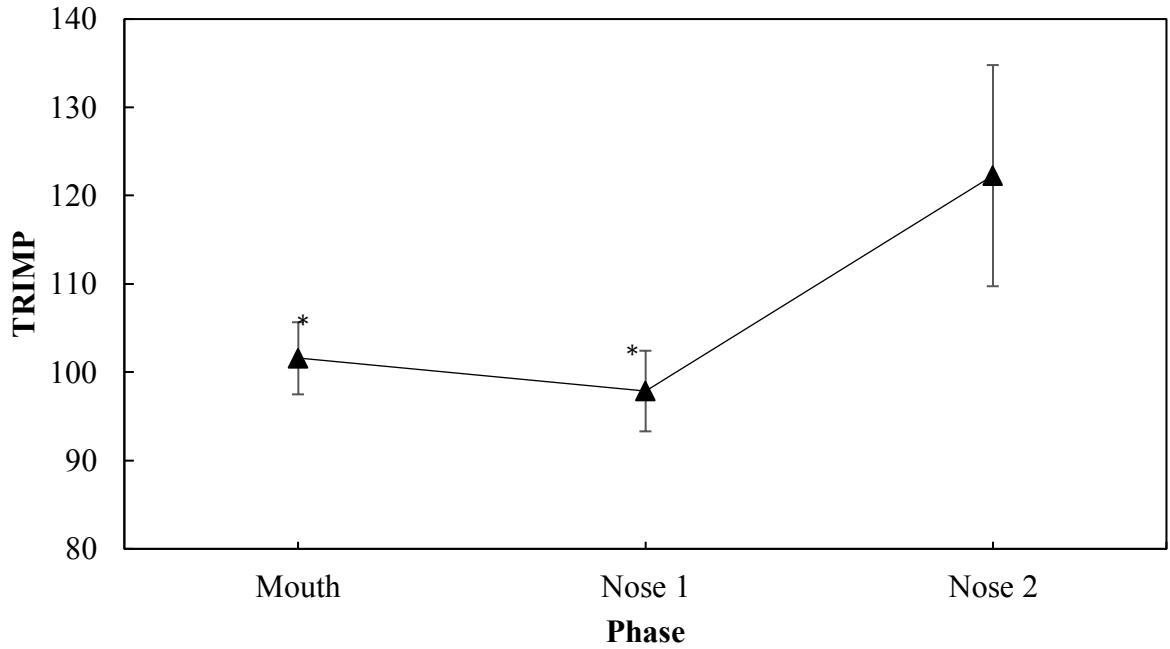


Figure 12. *TRIMP* compared between the three phases of (error bars indicate 95% confidence intervals). \* indicates significance compared to Nose 2.

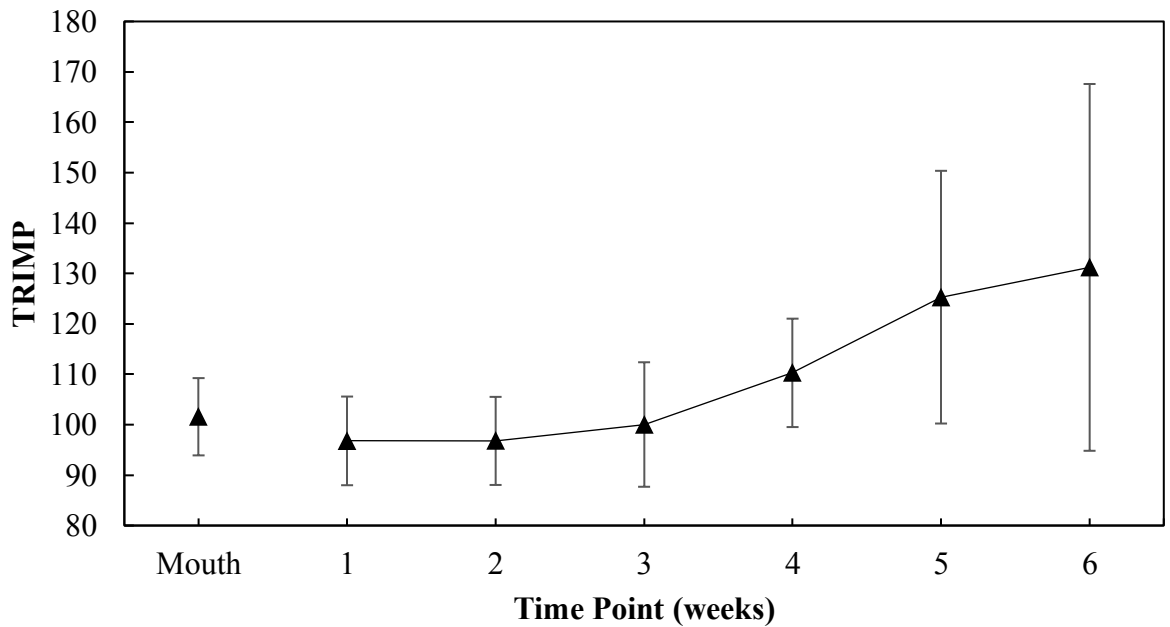


Figure 13. Three-week control phase (Mouth) average *TRIMP* compared to nose breathing weekly average (error bars indicate 95% confidence intervals).

*HRV RMSSD* compared between the three phases (MB, NB 1, & NB 2) (Table 2) was found to have significant differences over time ( $F_{(1.86,31.63)}=5.427, p=0.011$ ). Post hoc tests revealed a significant difference between the MB and the NB 1 (mean (CI) =103.88 (10.12) vs. 80.44 (8.07),  $p=0.004$ ), however no other differences were observed ( $p>0.05$ ) for all other phases. Additionally, no differences were found when comparing *HRV RMSSD* weekly compared to the MB ( $F_{(2.54,12.70)}=1.715, p>0.05$ ) (Table 3). No significant differences were found between the three phases (MB, NB 1, & NB 2) for resting heart rate ( $F_{(1.51,25.72)}=3.046, p>0.05$ ), or resting respiratory rate ( $F_{(1.48,25.09)}=1.991, p>0.05$ ) (Table 2). Daily resting *HRV* showed a slight but insignificant downward trend over the course of the 9-weeks of running ( $y = -0.2052x + 97.649; R^2 = 0.0179; p=0.34$ ) (Figure 14). Likewise, the daily resting *HR* also showed a slight but insignificant downward trend over the course of the 9-weeks of running ( $y = -0.0284x + 45.503; R^2 = 0.0268; p=0.24$ ) (Figure 15) as did the daily resting respiratory rate ( $y = -0.0135x + 15.465; R^2 = 0.0421; p=0.14$ ) (Figure 16).

Table 2. Resting Physiological values during three Phases of the training program

	<b>Mouth</b>	<b>Nose 1</b>	<b>Nose 2</b>
	Mean (st. dev)	Mean (st. dev)	Mean (st. dev)
<b><i>HRV RMSSD (ms)</i></b>	103.88 (22.98)	80.44 (17.46)	91.70 (26.34)
<b><i>Resting HR (bpm)</i></b>	44.61(2.43)	45.94 (2.69)	43.61 (2.68)
<b><i>Resting RR (bpm)</i></b>	15.39 (0.70)	15.18 (1.24)	14.72 (1.07)

Table 3. Three-week control phase (Mouth) average HRV RMSSD compared to nose breathing weekly average

	<b>Mouth</b>	<b>Week 1</b>	<b>Week 2</b>	<b>Week 3</b>	<b>Week 4</b>	<b>Week 5</b>	<b>Week 6</b>
	Mean (st.dev)	Mean (st.dev)	Mean (st.dev)	Mean (st.dev)	Mean (st.dev)	Mean (st.dev)	Mean (st.dev)
<b><i>HRV RMSSD (MS)</i></b>	103.88 (12.78)	79.51 (12.03)	69.57 (9.52)	92.24 (22.17)	82.92 (14.92)	93.48 (27.06)	98.71 (35.34)

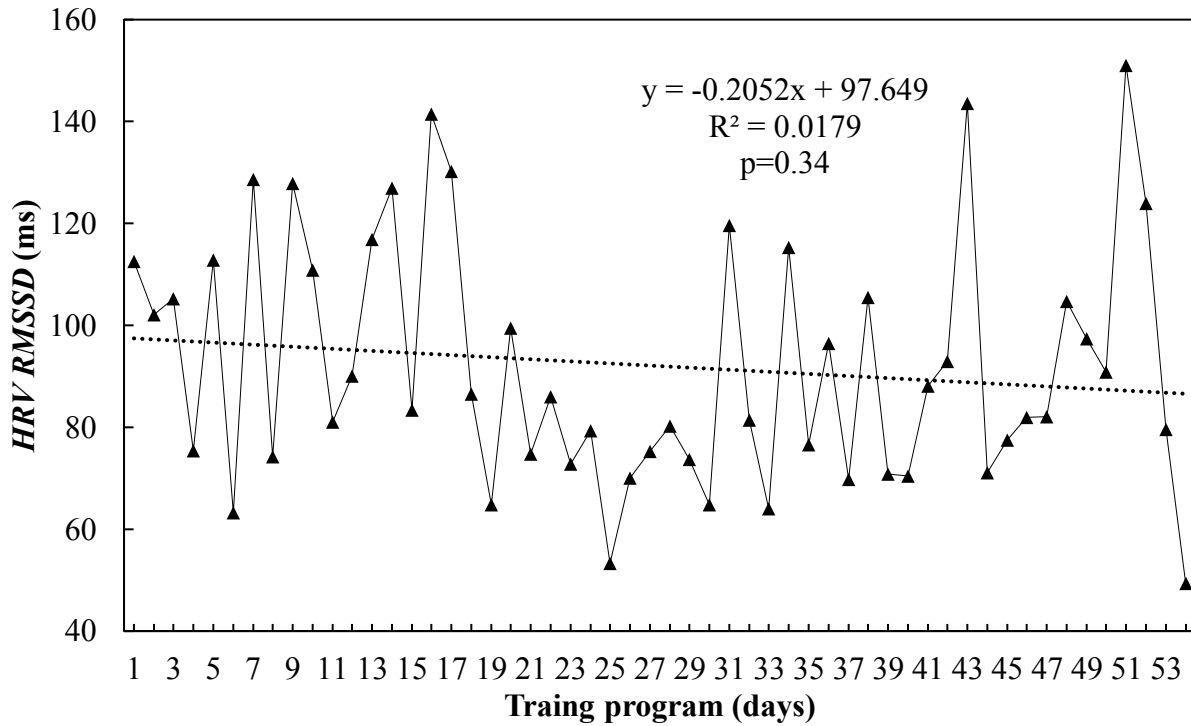


Figure 14. The daily root mean squared of successive differences between heartbeats, averaged daily, conducted prior to the daily runs.

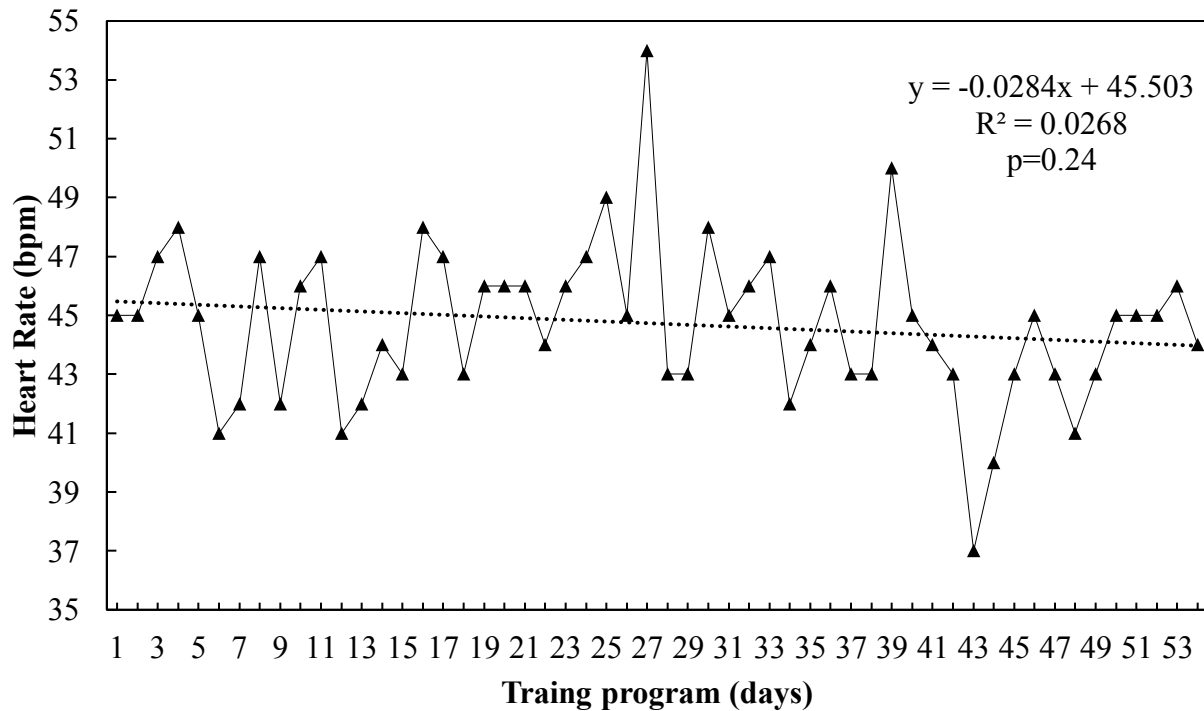


Figure 15. Resting HR, measured by the Garmin Forerunner 745, prior to the daily runs.

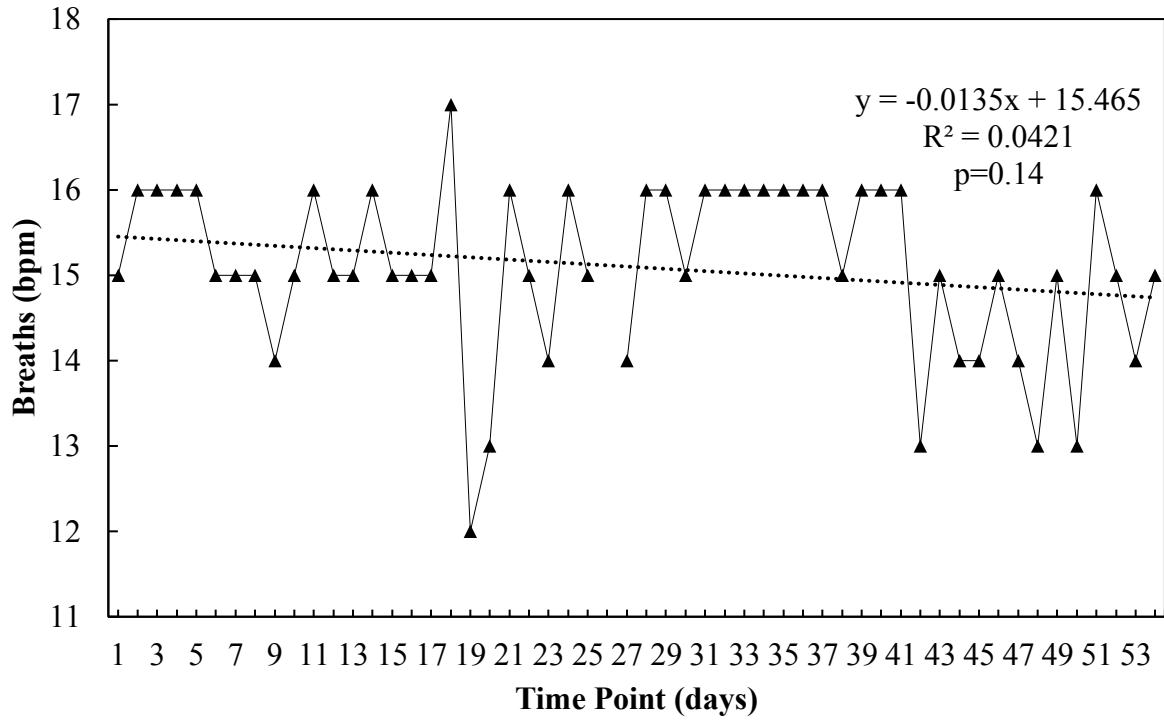


Figure. 16. Resting respiratory rate measured each night prior to the daily runs.

Figure 17.1-5 shows a summary of the biomechanical measures recorded during the runs and averaged into the three phases. Descriptive statistics show that the cadence (spm) during MB ( $M=173.67\pm 1.19$ ) was higher than the NB 1 ( $M=170.67\pm 1.5$ ) and slightly higher than NB 2 ( $M=171.50\pm 1.68$ ) (Figure 17.1). Ground contact time (ms) showed almost no differences across the three phases of MB, NB1, and NB2 ( $M=215.83\pm 6.48$ ,  $M=216.92\pm 8.66$ , and  $212.25\pm 11.76$ , respectively) (Figure 17.2). Figure 17.3 shows that stride length (m) remained constant between MB and NB 1 ( $M=1.40\pm 0.035$  &  $M=1.40\pm 0.036$ , respectively) but increases by 0.10 m during NB 2 ( $M=1.50\pm 0.023$ ). Vertical oscillation (cm) increased slightly during NB 1 and NB 2 ( $M=10.24\pm 0.63$  &  $M=10.37\pm 0.37$ , respectively) compared to the MB ( $M=9.72\pm 0.40$ ) (Figure 17.4) while the vertical ratio (%) increased from the MB ( $M=6.70\pm 0.27$ ) to NB 1 ( $M=7.13\pm 0.48$ ) and the decreased back to MB-like values during NB 2 ( $M=6.71\pm 0.33$ ) (Figure 17.5).

The subjective traction scores showed a significant difference over time between the three phases ( $F_{2,51}=9.18$ ,  $p<0.05$ ) (Figure 18). Post hoc testing indicated significant ( $p<0.05$ ) differences between NB 2 ( $M=9.61$ ,  $CI=0.45$ ) and both MB & NB1 ( $M=7.56$ ,  $CI=0.95$  &  $M=7.83$ ,  $CI=0.68$ , respectively) and no differences were found between MB & NB 1 ( $p>0.05$ ).

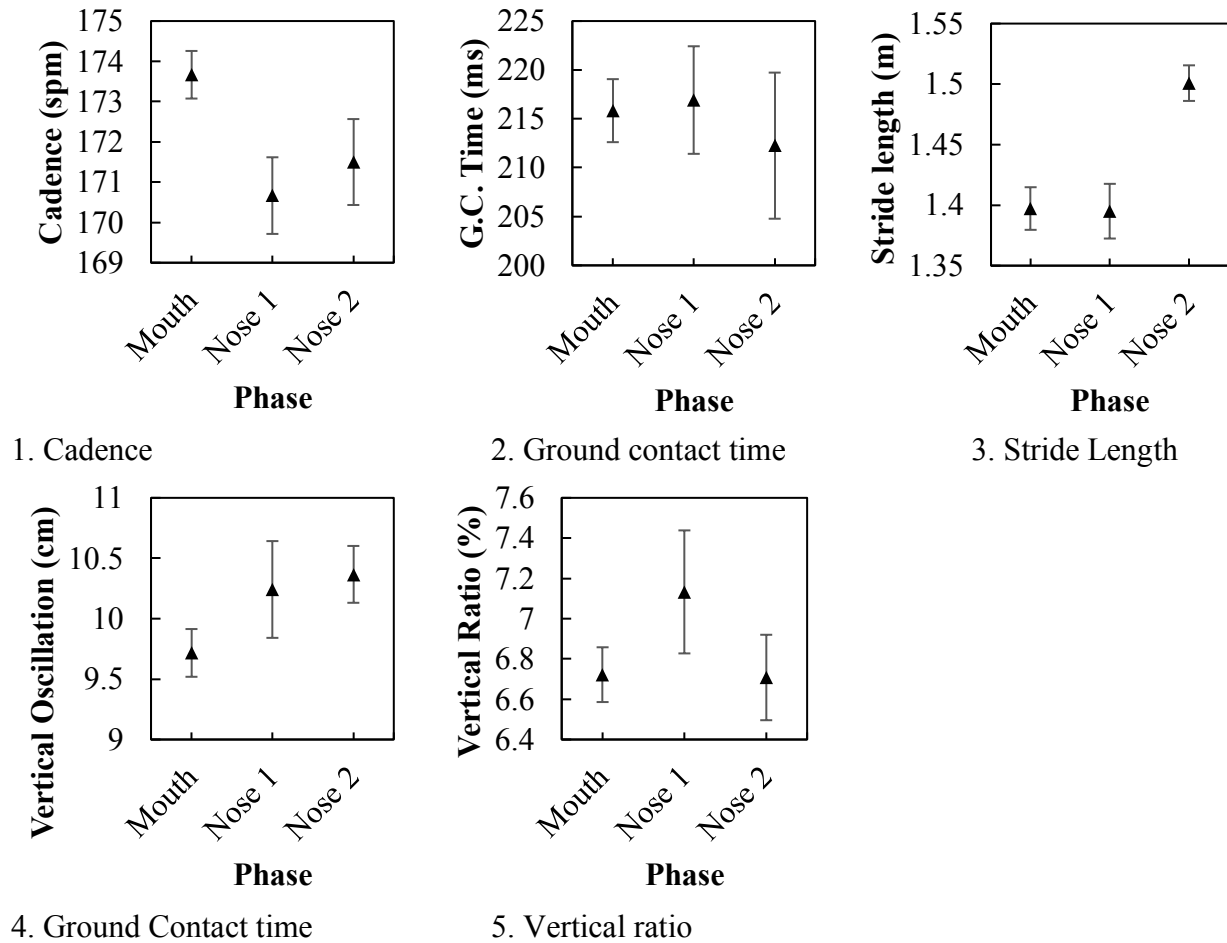


Figure 17.1-5. Biomechanical data during daily runs compared over three phases (error bars indicate 95% confidence interval).

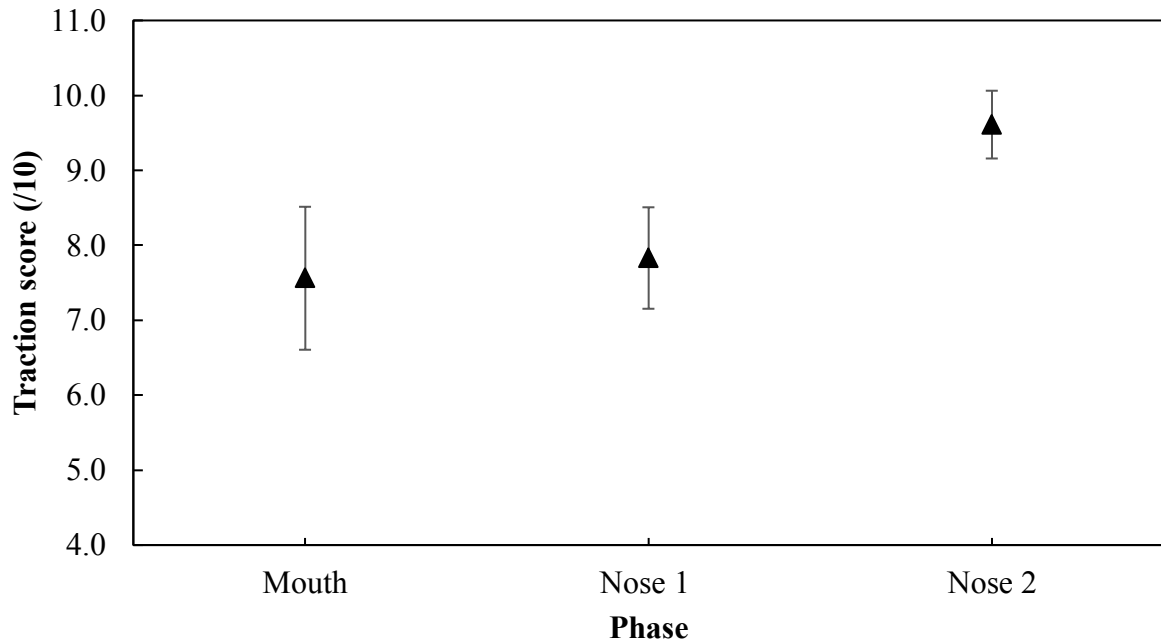


Figure 18. Subjective traction ratings.

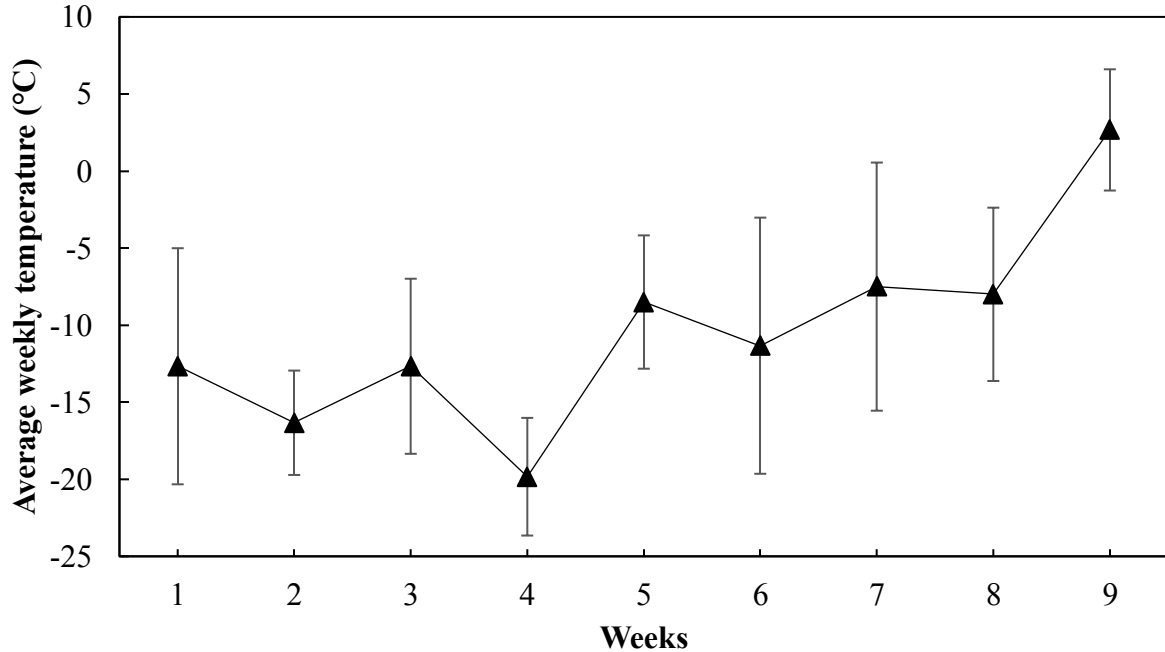


Figure 19. Average weekly temperature ( $\pm$ SD) for the 9 weeks of the running study. Weeks 1-3 represent the MB phase and weeks 4-9 represent the NB phase.

**Qualitative dyspnea themes:**

During the 6-week NB training program five main themes were identified in the daily journal entries regarding the subjective dyspnea experience which may be experienced during any single NB run, which included:

First, it was evident that at the beginning of a run, dyspnea does not occur instantaneously. During the initial minutes of a run, regardless of the initial kilometer pace (always at the subjective 16/20 RPE intensity) it was observed that there was a distinct lack of dyspnea sensations. This can be observed in my journal entry on my very first day of nose breathing:

*“I started the run with a 4:07 which created a strong feeling of dyspnea that peaked at around the 1.75-2km. I was saved by the tail wind down Ferguson st. which helped me cruise through the third km while I tried to regain control of my panicky feelings.” (11 February 2021, 1<sup>st</sup> nosebreathing daily run).*

This also held true, on a shorter time frame, during the NB interval workouts as described in the journal entry following my first interval workout:

*“the “1km threshold” pace was used (3:45), this was way too fast. I held on to the pace for two minutes on the first interval before I had to drop the pace drastically (~4:10)... The second interval I fared about the same. The pacing graphs look shockingly similar. I again held it for about 2min, then absolutely died.” (15 February 2021, 1<sup>st</sup> interval workout).*

Even during this faster running, the dyspnea sensations consistently did not occur at a noticeable intensity until after approximately a minute into the interval.

Second, it was commonly reported that an increase in pace (i.e., running faster) mid-run can cause almost instantaneous dyspnea sensations. After the first few minutes, once I had settled into my pace at an RPE of 16/20 which usually left me feeling “comfortable uncomfortable”, I frequently experienced sudden onsets of dyspnea caused by momentary increases in pace as described in the following journal entry:

*“The dyspnea did catch up to me. After ~6 km I found that my body couldn’t buffer a momentary pace increase. For example, when crossing a road, I check both ways which momentarily slows me and I will usually increase my pace for a few strides to make up the difference (I’ve done this every single day) but in the last few km I found that even that slight deviation from the pace I was holding, would cause a mildly panicky sensation (dyspnea). To me, this means that I was truly right on the line for pacing and that my body is not quite ready to give me a full 40min there just yet. I had to take a couple mouth breaths in the last km. It was as if I had reached some kind of threshold. [The] last complete km was a 4:11 pace but it was full of surges and ebbs as I tried to regain composure.” (18 February 2021, daily run).*

Early in the NB phase, the combination of a delayed onset of dyspnea sensation at the start of a run, with the high sensitivity to an increase in pace during the run, frequently resulted in a misjudgment in pace early on, often leading to sudden and overwhelming dyspnea sensations later in the runs until I learned to pace the earlier kilometers more reasonably.

Third, it was clear that while the awareness of dyspnea occurs rapidly, and the sensations are slow to dissipate. If nose breathing is continued following the onset of dyspnea, the dyspnea sensations will often persist even if the pace is drastically reduced:

*“I only felt overwhelmed by [dyspnea] one time when I hustled to cross the street after a car waved me on. It is incredible how quickly you can cross that threshold into panicky and how slowly you return to a semi-comfortable sensation without taking a breath from the mouth” (12 February 2021, daily run).*

*“During the 6<sup>th</sup> interval however, I also experienced a sudden intense sensation of dyspnea and accidentally took a breath from my mouth. It started just before I hit the km mark and lasted the rest of the duration (1:19) despite me reducing my pace.” (26 March 2021, interval workout).*

Any time that I experienced dyspnea it did not dissipate in any meaningful way without some form of longer-term pace reduction, momentary pauses were not effective at dissipating dyspnea:

*“I had to stop running and take two breaths from my mouth. I kept running and nose breathing but I could only manage a ~4:30-4:35 and even that only lasted 30s before I was stopped and mouth breathing again. The first stop was just a pause, I never even fully lost momentum. The second was a hands-on-the-knees, sucking air, stop for about 5s. The last ~55s I held a ~4:10 pace and I was grateful I could manage that.” (15 February 2021, 1<sup>st</sup> interval workout).*

The fourth theme that emerged was that the dyspnea sensation exists on a continuum ranging from mild discomfort to an overwhelming and panic-inducing experience. The mild dyspnea sensations I often described as being “comfortably uncomfortable”:

*“Overall, I would describe the run as ‘comfortably uncomfortable’. I was just at the point where I felt the dyspnea but never felt distressed about it.” (16 February 2021, daily run).*

Moderate levels of dyspnea were more distressing than the mild dyspnea and were often equated to the sensation of being suffocated or drowned:

*“The air hunger feeling began to feel overwhelming and I felt like I couldn’t slow down enough to catch my breath. I refused to stop on principle, but I felt like I was slowly suffocating the whole time.” (11 February 2021, 1<sup>st</sup> nosebreathing daily run).*

Overwhelming dyspnea occurred predominantly during the interval workouts. It reduced my motivation to persist with nose breathing, opting instead to mouth breathe.

*“The fourth [interval] was the only one in which I did not stop running. I started at about 3:55 and steadily slowed to a 4:15 by the end. I took a breath about once every 10-15s or so for the last minute and a half. The first mouth breath was involuntary, but... I took a breath when I needed it after that. Traction wasn’t fantastic but it was hardly the reason I wasn’t hitting the pace. It is shocking how long 4 minutes can be when you feel like you are slowly drowning.” (15 February 2021, 1<sup>st</sup> interval workout).*

Finally, it was reported that the switch from NB to MB was not always a conscious decision. The dyspnea sensation can occupy a large portion of your conscious thoughts. Lapses in focus resulted in several involuntary mouth breaths during the nose breathing section of the study:

*“I did have one involuntary mouth breath. It happened just after the 5km mark, I lost focus for a second and my mouth just did it.” (16 February 2021, daily run).*

*“I thought I was going to make it and then I involuntarily took a breath from my mouth. It was so fast and loud that I actually made myself jump a little bit. It’s funny when you get to that point of dyspnea because it becomes really hard to monitor everything going on. The time, distance, keeping the pace, monitoring traffic and pedestrians, and then also only breathing through the nose. I guess I just couldn’t focus on the nose breathing anymore and my brain took over. During the last little bit, I was fighting to breath so hard that my left ear kept becoming plugged as if I was pinching my nose closed and breathing against it.” (8 March 2021, GXT test).*

## **Chapter 5: Discussion**

### **Summary:**

The purpose of my study was to investigate the feasibility of completing an interval-based nose breathing-only training program that would be comparable to a typical running training program. It was my prediction that this would be a feasible goal to obtain with the criteria for successful completion being: maintaining health, maintaining prescribed intensity, completing  $\geq 95\%$  of the daily runs, and completing all three test days. I also predicted that this training program would have beneficial effects on my five-kilometre time trial performance. Additionally, I predicted that I would see improvements in my breath hold capability and my voluntary switching point from nasal to oral breathing during an incremental exercise test due to adaptations to the dyspnea sensation experienced during the daily runs and interval runs of the nose breathing phase. I hypothesized that I would experience a higher sensation of dyspnea during the nose breathing phase compared to the mouth breathing phase during runs at the same intensity and that the dyspnea sensation would lessen slightly over the six weeks of the nose breathing phases as I became more accustomed to nose breathing while running.

I was able to complete the 3 weeks of the control phase and the 6 weeks of the nose breathing phase without any major difficulties and remained relatively healthy throughout the testing period. I completed 100% of the prescribed daily run days with only occasional rearrangement of days and the HR data helps to corroborate my subjective reports of maintaining the prescribed 16/20 RPE intensity. Finally, I completed all three test days and can therefore say that it was in fact feasible to complete an interval-based nose-breathing training program designed for a competitive runner who is previously unaccustomed with nose breathing.

Furthermore, both my 5kmTT and breath hold times improved following the nose breathing phase. The voluntary switching point only slightly improved, as did the other performance measures showing a potential plateau following an initial improvement. The dyspnea sensation did not decrease in the linear manner in which I had predicted. Instead, it was present during most, but not all, of the early runs before presenting more sporadically throughout the program; often during the interval workouts, while it was completely absent during many runs that were of the same intensity, or faster, than runs completed during the mouth breathing phase.

I had predicted prior to the commencement of the study that objective measures of stress, such as *HRV*, as well as subjective measures of stress such as *POMS* and *RPE*, would increase during the initial stages of the nose breathing phase but would begin to return closer towards the values obtained during the mouth breathing phase as the study progressed. I found that the *HRV* was in fact decreased during the first three weeks of the nose breathing compared to the mouth breathing, while the second half of the nose breathing phase was not significantly different. This significant difference did not persist when individual weeks were compared, likely because of the large variability of the HRV measurement day-to-day due to HRV being a measure of overall stress and the fact that my body began to adapt to the stress imposed by the nose breathing; resulting in a relatively fast recovery of HRV to MB-like values. Contrary to my initial hypothesis, Neither *POMS* nor *RPE* showed any significant differences at any point during the study.

Following a brief adaptation period, nose breathing training proved to be a suitable alternative to a standard mouth breathing training program. Nose breathing training not only facilitated improvement in time trial performance but may have also contributed to the improved

performance. The improved time trial performance may be in part due to an increased tolerance to the dyspnea sensation as demonstrated by the improved breath hold times observed over the course of this study.

This study was, to this researcher's knowledge, the first to investigate the initial and short-term adaptations to nose breathing during exercise as well as to describe the subjective experience during the adaptation to nose breathing. This explorative case study had many unknowns as it moved into the testing phase such as the feasibility of completing the program, the effects that it would have on my physiology, the psychological and subjective effects of the program, and finally what effects this program may have on running performance.

### **Initial expectations:**

It is universally accepted that nose breathing during exercise will result in the sensation of dyspnea in unaccustomed exercisers (Dallam & Kies, 2020). What is relatively undiscussed in the literature is the subjective experience of the dyspnea, the timeline of adaptation to nose breathing, and the feasibility of completing a higher intensity nose breathing training program. Additionally, due to the ethics approval process and initial delays due to the COVID-19 pandemic, this testing program was conducted during the winter months lasting from 19 January 2021- 30 March 2021. The existing literature on cold weather exercise suggested an increased likelihood of exercise induced bronchospasms as well as significant respiratory dysfunction (Üçok et al., 2004; Castellani & Tipton, 2016; Kennedy et al., 2020); resulting in the dreadful expectation of respiratory distress in the form of EIB and a preparedness for potential respiratory illness during the mouth breathing phase. The existing literature regarding nose breathing during exercise consistently sites 6 months as an adaptation period for nose breathing (Hostetter et al.,

2017 & Dallam et al., 2018); leading me to believe that I would consistently be experiencing moderate to severe dyspnea throughout the 6wk nose breathing phase. Despite the expectation of overwhelming dyspnea during the nose breathing, the literature regarding the air warming and humidifying effects of nose breathing (Mangla & Menon, 1981; Naftali et al., 2005; & Nestor, 2020) lead me to believe, potentially naïvely, that if I could make it to the nose breathing phase of the experiment, any respiratory ailments experienced during the mouth breathing phase would be improved if not cured by nose breathing.

### **Daily runs:**

#### **Pace:**

During the three-week mouth breathing control phase the average pace of the daily runs was a 4:05 min • km<sup>-1</sup>, representing for myself, a 16/20 on the *RPE* scale. During the first week of nose breathing this pace was significantly reduced to a mean of just under 4:20 min • km<sup>-1</sup> due to the large sensations of dyspnea that were experienced while trying to run, causing my perceived exertion to reach 16/20 at a lower running intensity. This reduction in output during the beginning of the NB phase is consistent with the review finding of Dallam & Kies (2020) which indicate a reduced aerobic capacity in runners unaccustomed to nose breathing. Likewise, Hostetter et al, (2017) indicate that the triathlete who adopted nose breathing in their study also indicated an initial decrease in attainable intensity due to the sensations of dyspnea.

During week 2 and week 3 the pace was not significantly different from the MB, although the average paces are slightly higher than the MB. This may be due to the seemingly non-linear adaptation to nose breathing and the dyspnea sensation. During week 2 and week 3, the variability of the pacing was much higher (CI=±12.76 & 7.41, respectively) compared to the

MB and week 1 ( $CI=\pm 1.48$  &  $2.72$ , respectively). This was due to the inconsistent improvements seen with week 1 during these times. For week 1, the pace was drastically reduced for all the runs as I struggled to cope with the dyspnea and the pacing. The pacing profile within each individual kilometer played a large role in the struggles with dyspnea. Any surges in the pace were often met with large and sometimes overwhelming dyspnea sensations. During the week 2 and week 3, the pace was more variable as I would attempt to push the pace in order to reach an exertional *RPE* of 16 for the duration of the run. As such, I was sometimes met with large sensations of dyspnea, usually in the second half of the run which greatly reduced my pace while other times the dyspnea seemed almost completely absent. This inconsistent dyspnea sensation coupled with two heavy snow days in week 2 (22<sup>nd</sup> & 23<sup>rd</sup> February 2021) likely are the reason for the large variability between confidence intervals in these periods.

There was a steady increase in daily pace during the last 3 weeks before reaching statistical significance compared to mouth breathing in the 6<sup>th</sup> and final week of the nose breathing phase. This finding confirmed that it is possible to complete a training program designed for a 5-10 km competitive level runner at the same intensity while nose breathing as with mouth breathing. It is important to note that the weather did gradually warm during the training program and the traction improved as well. It is unclear what the exact effect and magnitude these factors may have had on pacing during the training program. Despite this, the significant pace improvement, subjective ease of breathing, and lack of dyspnea during the later nose breathing runs does continue to suggest that nose breathing is a feasible training alternative to mouth breathing for competitive runners.

**Subjective measures:**

The *RPE* showed no differences between any time points in the running program. This is unsurprising as the intensity of the runs was determined based on an *RPE* clamp of 16. Small variations did exist during the running program, usually ratings of 17 (n=11) were caused by poor weather or large sensations of dyspnea during the run, often caused by a misjudgment in pace during the early portions of the run. One 15 *RPE* day (13 February 2021) was recorded due to a multitude of factors including: the temperature felt like -32°C for the third day in a row, the arches of my feet hurt, and I generally felt sluggishness/ lacked motivation, which all combined to prevent me from having the motivation to achieve what I felt was a true 16 *RPE* effort. This consistency of *RPE* measurements when compared alongside the generally unchanging heart rate data indicates that my perception of effort did not change significantly over the course of the 9 weeks of running.

The *POMS* did not show any significant differences over the course of the training program. However, it is important to note that this measure was taken in the morning prior to the runs. In retrospect, a second *POMS* measurement later in the day may have gathered a better understanding of the effects of this training program on my mood. Terry, Lane & Fogarty (2003) administered their POMS questionnaires to athletes one hour prior to their athletic events and to students either before or after a class, but never administered the questionnaire both before and after an event. Marathon running has been shown to negatively affect POMS scores in male runners immediately following the run (Hassmen & Blomstrand, 1991). With only a subjective memory and journal entries written closely after each run to rely on, I believe that this second *POMS* measure would have showed a worsened mood later in the days during the later stages of NB2. I believe this was mostly due to the progressive interval workouts which resulted in an

increase in intensity running and a large jump in mileage (the first two intervals were both less than 9 km, and the last two interval workouts were both over 17 km) with no easy run days. Journal entries in the final days of the running program indicated my feelings regarding the increasing mileage:

*“I was tired and really not feeling it this morning before the run. I thought that the run might make me feel better, but it just made me angry. I’m writing this almost two hours after finishing because I was in too bad of a mood” (19 March 2021, interval workout).*

*“I think that it is starting to affect my mental game during the runs; it has certainly had a negative effect on my mood the last two days” (25 March 2021, daily run)*

*“On a subjective, how are you feeling note: I am exhausted. I’m writing this at 1:00PM because I’ve been asleep since I finished the run at 10:30AM.” (26 March 2021, interval workout).*

While my affect did seem to worsen near the end of the running program, I have no reason to believe this was due to nose breathing specifically. I believe that this worsened affect was more likely caused by tiredness due to the increased mileage near the end of the program which also coincided with the period of the program in which I happened to be running my fastest daily runs. My mood may have been further worsened due to boredom from running the same 3 km loop 3x per run for 54 days resulting in ~162 laps over the course of the program, not including test day runs.

**Interval workouts and training impact:**

The interval workouts were added to the training program in anticipation of the decreased attainable intensity while nose breathing which we predicted based on the existing nose breathing literature (Lacomb, 2015; Hostetter et al., 2017; Dallam et al., 2018; & Dallam & Kies, 2020). The intervals then progressed in duration and/or number of repetitions with each successful completion of 2 interval runs of the same level. The interval workouts were one of the main sources of dyspnea during the running program. I experienced strong (n=3) or overwhelming (where I breathed from my mouth) (n=3) dyspnea during 6/12 interval workouts. The prevalence of dyspnea during the intervals were predominantly during the first three weeks, as I failed both of the first week intervals, and then again as the duration increase to 75 minutes and 17.5 km: failing my last interval workout.

The overwhelming dyspnea experienced during the first week and the dyspnea that was more prevalent in the first few weeks further confirms the general consensus within the literature that maximal output is reduced while nose breathing with unaccustomed runners. The decreasing prevalence of dyspnea during the middle to late section of the nose breathing phase indicates that the adaptability to nose breathing at higher intensities may be feasible in a much shorter time frame than was indicated by Hostetter et al. (2017) and Dallam et al. (2018). Having said this, the re-emergence of the dyspnea sensation in the third-last and last intervals indicates that the adaptation to nose breathing may have been limited in the duration that this higher intensity could be maintained. It is also important to note that I did not complete a mouth breathing version of these interval workouts and cannot speculate if mouth breathing would have eased the difficulties of these longer high intensity runs or not. Additionally, for my third-last interval

workout, my mental focus may have been impacted by a generally negative affect that day, as illustrated by the daily journal entry:

*“I was in a terrible mood today... I barely slept last night for whatever reason. I think I’m just tired and cranky. [It’s] crazy what mindset can do, I haven’t experienced strong dyspnea since March 1<sup>st</sup>, and I felt it during intervals 3,4, &5 in the last minute and a half-ish.” (19 March 2021, interval workout).*

The idea that mental fatigue, specifically, may affect dyspnea was only observed in this isolated incident and was therefore inadequate evidence to create an additional dyspnea theme. Despite the lack of an observed theme, breathing through the nose while running takes a lot of mental focus and it would be a logical leap to assume that a negative affect or mental fatigue may decrease the body’s tolerance for dyspnea. Marcora et al. (2009) have already shown that a mentally fatiguing activity can significantly decrease the time to exhaustion for a cycling task. A journal entry from my last week of nose breathing illustrates the fatiguing effects of the long intervals:

*“It was the distance that seemed to grind me down. It is very hard to stay motivated when you are accelerating to a faster-than-3:45 pace on the 16<sup>th</sup> km and 70<sup>th</sup> minute of a run that was preceded by two [days of] back-to-back sub-40 10 km’s.” (24 March 2021, interval workout).*

The interval workouts had a large impact on the variability of the *TRIMP* scores (figure 13), which did not differ significantly over time however it is clear to see that the *TRIMP* was increasing over the course of the program. The 2 interval training days per week resulted in a higher variability of *TRIMP* scores without being significantly different week to week which demonstrates that the training program did increase the training impact as it was designed to do

without overwhelming me with week-to-week changes. Near the end of the program however, I did begin to exhibit some signs of overtraining such as insomnia, fatigue, irritability/agitation, and loss of motivation (Kreher & Schwartz, 2012). Speaking specifically to the *TRIMP*, this program was designed to be rigorous and to push the limits on what is possible to achieve while nose breathing, however this program design may induce overtraining effects by increasing the *TRIMP* by introducing progressive intervals without any easy runs due to the clamped *RPE* during the daily runs.

### **General health during the program:**

Despite the potential overtraining symptoms experienced near the end of the program, I did manage to stay relatively healthy throughout the training program, experiencing only minor aches and pains. Subjectively, I did feel healthier during the nose breathing phase compared to the mouth phase. This was due mainly to the presence of coughing and wheezing, symptoms of exercise induced bronchospasms experienced in 72.2% of runs during the mouth breathing phase which were almost immediately extinguished once I began the nose breathing phase, being prevalent in only 2/18 (11.1%) of the NB 1 phase and being non-existent in the NB 2 phase. Additionally, the two days in which I experienced post-run coughing during NB 1 were on the 11<sup>th</sup> and 13<sup>th</sup> of February 2021, representing the first and third days of nose breathing. In my opinion, I do not believe that I would have coughed during these nose breathing days if I hadn't been running while mouth breathing in the days before. While mouth breathing I often experienced prolonged (20min+) bouts of coughing following runs and occasional coughing during the night which would cause a poor sleep and a sore throat. During the nose breathing the prolonged coughing halted immediately; likely due to a combination of the improved heating and

humidifying ability of the nose (Naftali et al., 2005; Lacombe, 2015; Recinto et al., 2017; & Dallam et al., 2018) as well as the reduced rate of air inhaled per breath due to the higher resistance of the nasal passages compared to the oral passage (Kelly et al., 2000. & Borojeni et al. 2020). The combination of these factors presumably allowed the inhaled air to be more similar to the alveolar conditions, reducing the heating and humidifying load on the oropharynx, trachea, and bronchi which likely resulted in the decreased prevalence of coughing and wheezing symptoms observed over the course of the experiment as can be observed in table 1. It is also important to note that while the temperature did generally warm over the course of the running program, the coldest week of the entire 9-week training program occurred on the first week of nose breathing, with an average temperature of  $-19.8^{\circ}\text{C}$  (ranging from  $-23^{\circ}\text{C}$  to  $-18^{\circ}\text{C}$ ) (fig. 19, week 4), during which time EIB symptoms were still greatly reduced.

Generally, I was healthy throughout the training program. I was never sick or sore enough to skip a run day; however, the second test day was shuffled by a few days due to fear over a not yet fully recovered sore back and some interval days did have to be shuffled around due to weather. This training program was gruelling however, and I did experience some aches and pains at various points during the 9 weeks. Slippery days generally caused sore muscles, usually tight hamstrings and hips in the days following due to the little bit of slip in each step. My sore back occurred on 1 March, following a long day of sitting completing schoolwork after running in the morning; causing my back to go into spasms that evening. I still ran the next day but with an extreme focus on running as smoothly as possible; I was so focused on this that I actually forgot to close my mouth and nose breathe for the first 30 seconds of the run. In the days following, I did not experience any pain during the runs however I chose to push the test day back as a precaution. The other significant pain that I experienced was pain on the lateral side of

my left foot around where the cuboid and forth & fifth metatarsals meet. This began on 15 March as an ache and progressed from an ache to what I considered to be real pain throughout the run on the 16<sup>th</sup>. Due to this pain, I changed from my New Balance Vastu running shoes to my New Balance Fuel Cells from the 17<sup>th</sup>-19<sup>th</sup> because they have a slightly thicker sole. Constant icing, heating, and massaging during this time allowed me to continue with the planned runs with no difficulties beyond a dull ache and I returned to my Vastu's on the 20<sup>th</sup> for the duration of the program.

### **The subjective dyspnea themes:**

It has been well documented that the transition to nose breathing while running involves experiencing the sensation of dyspnea in the beginning which slowly decreases over weeks and months as the body adapts to nose breathing (Hostetter et al., 2017; & Dallam et al., 2018). Extraordinarily little has been documented regarding the subjective dyspnea experiences of those who nose breathe while exercising during the initial stages of this adaptation. My subjective experience was that the sensation of dyspnea onset was not instantaneous at the beginning of a run. Even at a subjective running intensity of 16/20 *RPE*, the sensation did not occur in the first few minutes; appearing at the earliest around the 1 km mark. For example, on my first day of nose breathing I ran a 4:07 first kilometer, the same as the average pace during the mouth breathing section, while the average nose breathing week 1 pace was ~4:20; only after this first kilometre did I first become aware of any dyspnea sensation. This is perhaps due to the buffering capacity of the bicarbonate-carbonic acid buffering system which is able to curb the increase in CO<sub>2</sub> accumulation despite a lesser ability to expire the excess CO<sub>2</sub> while nose breathing for the initial stages of a nose breathing run even at a moderate-vigorous intensity. This insight into the

dyspnea experience may help explain the reason that studies have found that unaccustomed runners do not experience overwhelming dyspnea during short 4-10 min submaximal bouts of running (Lacomb, 2015; Gonzalez-Montesinos, 2020; & Lacomb et al., 2017). I also observed that an increase in running intensity/pace can cause an almost instantaneous increase in dyspnea mid-run. When adapting to nose breathing, my goal was to maintain the *RPE* of 16 without overwhelming myself with dyspnea. This led me to attempt to run at a “comfortably uncomfortable” pace; a pace that involved running at a slight level of dyspnea. As this running program was an outdoor running program, the pace was subjective and fluctuated over the course of the runs with ebbs and surges, mostly in response to the running environment such as the one rolling hill on the route, differing running surfaces between the different roads, corners, traffic, pedestrians, and dogs. Surges in pace, as might occur when crossing a road or accelerating back to my pace after turning a corner could very quickly result in an overwhelming sensation of dyspnea. This could perhaps be due to the anaerobic contributions associated with these short accelerations which then increase an already elevated CO<sub>2</sub> concentration in the blood due to the lessened respiratory buffering capacity of the bicarbonate-carbonic acid buffering system while nose breathing; this may be why the dyspnea sensation can occur so suddenly in the later stages of a run. Next, I found that the awareness of dyspnea was fast to build and slow to dissipate such as when reducing the pace, or even stopping completely, if nose breathing is continued (usually lasting ~30s stopping and longer if running is continued). In my experience, breathing from the mouth is the fastest way to decrease the sensation of dyspnea, usually relieving the uncomfortable sensations within 10-15 s. Similarly, dyspnea is a continuum with a wide range of sensations from a mild discomfort to an overwhelming, panic-inducing experience. On the mild side, this experience occupies very little focus and is only uncomfortable due to a slight feeling

of tightness in the chest. During this, I found my perception of effort only increased very slightly if at all and I required only mild focus to maintain the pace and keep my mouth shut, with no difficulty being alert/aware of the environment or having other unrelated thoughts. Beyond the mild discomfort, is a large sensation that is not out-right panic inducing and running may be continued to some capacity, however it is extremely uncomfortable, and I often felt as if I was suffocating while running. During this large sensation of dyspnea, maintaining the pace and continuing to breathe through my nose required a large amount of mental focus. I found that a large portion of my focus was turned internally, I became less aware of my surroundings, and I could not engage in unrelated thoughts.

The panic inducing dyspnea, usually experienced during the interval workouts, occupied all my mental focus; I experienced an overwhelming desire to breathe through my mouth and to stop running. I found that I was unable to focus even on visual cues (however, I never tripped) as my conscious mind was wholly devoted to keeping my mouth shut and trying to force as much air in and out of my nose as possible. Running at this level of dyspnea is unsustainable and I could only continue nose breathing for a very short time before giving in and breathing through my mouth. During this level of dyspnea there is a definite increase in respiratory distress, and I was unable to slow or lessen the force at which I was breathing. During the last interval of the first interval workout, I experienced such respiratory distress that I mimicked a Valsalva maneuver. I attempted to exhale so forcefully through the narrow openings of my nostrils that I almost caused a bowl movement. This mismatch between the perceived need to breath and the limited ability to move air into, and especially out of the nose was the aspect that seemed to cause me the most distress.

There were also several times during the program where I did not consciously decide to breathe from my mouth however, I lost focus during the overwhelming dyspnea sensation and took an involuntary breath from my mouth. These involuntary mouth breaths were always the exact same: they all followed a forceful exhale and as I prepared to breath in forcefully through my nose, my mouth would quickly open very wide resulting in an intense oral gasp that always startled me. The experience was startling for two reasons: the most obvious is that my body performed a large, forceful action without my conscious voluntary control and, beyond this, the speed at which I breathed in through my mouth would also startle me because it would severely disrupt my respiratory rhythm causing its own momentary feeling of panic like “having the wind knocked out of you” but reversed. The subconscious overriding of voluntary movements is a confusing and startling experience which demonstrates the high degree of mental focus required to continue nose breathing during these overwhelming episodes of dyspnea.

**Dyspnea theme trustworthiness:**

It seems clear that upon the initial adaptation to nose breathing while running, runners will experience sensations of dyspnea (Hostetter et al., 2017; Dallam et al., 2018,). However, there is no published descriptions of the subjective experience of dyspnea during this adaptation period, and therefore no comment on the inter-individual differences in dyspnea experience or on training strategy. The results of the present study suggest that there may be some variability in the time frame of the adaptation period to nose breathing while running, as significant running improvements were observed in my case following only 6 weeks of nose breathing compared to the 6 months indicated by Hostetter et al. (2017) and Dallam et al. (2018).

As described above in the ‘Initial expectations’ section, the dyspnea sensation was completely exploratory. As such, I had no known biases except having the expectation of enduring the air-hunger sensation of dyspnea. To further limit the inclusion of bias into the dyspnea themes reflexivity was utilized throughout the testing phase as well as the analysis phase. Using descriptive rather than analytical journal entries and acknowledgement of my subjective relationship with the content, in that I was also the respondent, allowed for critical self-reflection during the analysis phase.

The credibility of the dyspnea themes may be best assessed in relation to other studies (Korstjens & Moser, 2018). This study’s findings regarding the dyspnea sensations do not contradict any studies, known to this author, on the subject. Themes such as the delayed dyspnea sensations at the beginning of runs and intense dyspnea once pace is increased from a steady state align with the general exercise physiology principles regarding the bicarbonate-carbonic acid buffering system and the clearance of lactate. This study does contradict other studies in terms of timeline to adaptation however it is important to acknowledge that all other studies performed maximal nose breathing compared to maximal mouth breathing where as this study did not do this. It is also may be that individuals had adapted to nose breathing much faster than 6 months however the actual research was not conducted until then; especially important as all of the participants in the Hostetter et al. (2017) and Dallam et al. (2018) studies had self-selected nose breathing prior to knowing about either of the studies.

While transferability cannot be confirmed between subjects, the themes generated in this study showed dependability across time points and transferability between settings. The delay in dyspnea when beginning for example was consistent throughout the study as well as transferable to the setting of the intervals, in which the rest periods provided adequate rest to prolong the

dyspnea sensations similar to the start-of-run delay. Furthermore, themes were only presented in this study which showed consistency across time and settings. Some identified themes such as the effect of mood or fatigue on dyspnea were excluded from mention because of the inconsistent and contradictory findings across time and settings. Personally, I found this disappointing as this is an area of interest for me. Using reflexivity and also peer debriefing however, I clarified and developed the five dyspnea themes which presented themselves in this study.

Finally, a major aspect of the subjective journal entries was the focus on honesty. As listed previously, I had no desire to confirm nor reject the claims of James Nestor (2020) and I was simply interested in describing the experience. As such, I was completely honest in my recounting of experiences. I paid special attention to not over exaggerating nor down-playing any experiences throughout the running program and I did not omit anything of relevance, even when some of the experiences were embarrassing for myself. As such, I believe that these themes will have a high degree of confirmability in future research and with those who choose to undertake nose breathing training.

### **Heart rate and pulse pace:**

The daily run heart rate data (Figure 3) shows no significant differences between any of the time points during the running program. This shows that the level of intensity was relatively consistent throughout the experiment; I did not “slack off” at any point during this experiment. The pulse pace data (Figure 4), shows an initial, although not significant, decrease in fitness during the nose breathing phase demonstrated in the pulse pace by a similar hear rate to the mouth breathing phase with a slower pace. Following week 1, it displays a relatively steady

increase until reaching significance during weeks 4 and 6 compared week 1. These weeks did not reach significance compared to the MB, which therefore means there was no significant increase in fitness between the MB and the end of the NB training. The fact that the week 1 pulse pace was significantly different from the end of the nose breathing training program does indicate that the fitness while nose breathing increased during the program. This indicates that with this training program my adaptation to nose breathing may have been as short as 4-6 weeks before submaximal running fitness was equivalent to that of my mouth breathing running fitness. Without the aid of other measures, such as a lactate measurement as a measure of anaerobic contributions or a metabolic cart to measure changes in  $\dot{V}O_2$ ,  $\dot{V}CO_2$ , or  $RR$ , the pulse pace is unable to distinguish the origin of the change in fitness. Future research should look to further confirm this observed increase in nose breathing fitness and expand further on the source of these fitness changes. The data trend also appears to be continuing upwards; future research is needed to investigate the timeline (longer than 6 weeks) of adaptability to nose breathing.

### **Biomechanical data:**

The biomechanical differences that exist over time may be explained when in context with the subjective traction scores (Figure 18) and pace during these phases. The MB and NB1 phases of the training program had very similar traction scores (Mean $\pm$ CI) (M=7.56 $\pm$ 0.95 & M=7.83 $\pm$ 0.68, respectively). This may explain the similar stride length values between the MB1 and NB1 phases, and the Traction score for NB2 (M=9.61 $\pm$ 0.45) may also explain why the average NB2 stride length is ~10 cm longer than the other two phases. Additionally, this greater traction and longer stride length during NB2 may help explain the higher vertical oscillation in NB2 compared to the MB as I was able to exhibit the same biomechanical pattern during NB2 as

with MB except unconstrained by reduced traction. Both MB and NB2 were generally unconstrained by dyspnea and resulted in very similar biomechanical running patterns with the exception that MB was still constrained by an average worse traction. This similar running pattern can be observed in the vertical ratio similarities between MB ( $M=6.70\pm0.27$ ) and NB2 ( $M=6.71\pm0.33$ ). This may also help explain why there was a slightly higher cadence in the MB ( $M=173\pm1.19$ ) compared to NB1 & NB2 ( $M=170.67\pm0.50$  &  $M=171.50\pm1.68$ , respectively) as I adjusted to the stride length/traction constraint and increased the cadence in order to maintain the subjectively rated pace. The sensation of dyspnea that was experienced in the early stages of the nose breathing phase may also help explain the decreased cadence, higher vertical oscillation, and higher vertical ratio during the NB1 phase resulting in a slightly slower and “bouncier” pace.

### **Performance measures:**

#### **The graded exercise test:**

The graded exercise tests were the most challenging of the testing procedures to run independently because they included the tracking of the distance, pace stage, and actual current pace. An initial increase in the voluntary switching point from NB to MB of 23% from PRE-MID was observed, however from the MID-POST there was almost no improvement, showing only a 1% improvement in time achieved. This test was a self-paced run, with increasing pace goals every 500m. Figure 8 shows that the pacing profile of the three GXT tests look very similar, especially as the pace began to increase beyond the 4:10 mark, indicating that each of the tests were relatively consistent in their pacing profiles and should therefore not have significantly affected the results of the tests. The results of this test have shown that, while measures such as the pulse pace may indicate that the submaximal fitness improved over the course of the short nose breathing training program, there may have still been an upper limit to the maximal

intensity/pace while nose breathing, which for me during both the MID and POST tests was around the 3:30 pace. For comparison, I was able to hold this pace for 5 kilometers while mouth breathing during the PRE 5kmTT and was able to hold a 3:20 and 3:16 for the MID and POST 5kmTT, respectively. This upper limit of nose breathing ability may be the reason that the 6-month adaptation timeline is found in many of the long-term nose breathing studies (Hostetter et al., 2017; Dallam et al., 2018). Future research investigating the timeline of adaptability to nose breathing should utilize the switching point GXT as outlined by Lacombe (2015), in order to determine a timeline of adaptability for maximal as well as submaximal running while nose breathing.

Subjectively, I was frustrated that I was unable to improve further during the POST GXT from the initial large improvements from PRE-MID. The sensation of dyspnea presented almost identically in both MID and POST, building quickly during the 3:40-3:30 stages and *“crashing down on me all at once... it was as if I had hit a wall and could go no further”*. During the MID I stopped mid-way through the 3:20 stage and only just completed it for the POST trial. I found this especially interesting as I was able to complete a 10.75 km run with an average pace of 3:43 km<sup>-1</sup> (2 s faster than the prescribed interval pace) three days prior with no sensations of dyspnea whatsoever. Future research should include metabolic measurements such as  $\dot{V}O_2$  and lactate measurements in order to gather a better understanding of what is occurring in the body during the later stages of the GXT test. Based solely on my subjective experience and the sudden intense onset of the dyspnea during the later stages of the GXT, I believe that it is very likely that I had surpassed my lactate threshold resulting in a rapid accumulation of lactate, H<sup>+</sup> ions, and most importantly CO<sub>2</sub> within the blood which was unbufferable by the limited nasal respiratory rate. This would have resulted in a large and rapid increase in CO<sub>2</sub> diffusion through the blood

brain barrier, triggering the central chemoreceptors to increase sympathetic outflow to increase respiratory rate and depth, which is relatively impossible while still nose breathing. This mismatch, as well as the activation of the limbic system by this sudden increase in CO<sub>2</sub> concentration likely contributed to the sudden overwhelming dyspnea sensation while decreasing the motivation to continue the run and causing me to stop at relatively the same place in both the MID and POST GXT runs.

### **The breath hold tests:**

For the BHT there were three breath holds per time point; of these three, the first was always the shortest and the last was usually the longest as successive breath holds have shown intra-individual increases in BHT (Parks, 2006). In fact, during the PRE, MID, and POST trials the difference between the first and the longest breath hold was 26.6%, 47.8%, and 61.7%, respectively. Considering then the longest breath holds from each testing phase, there was a 34.1 s or 77% increase from PRE-POST testing. The improvements from PRE-MID showed large improvements (53.1%) with improvements in performance lessening between the MID-POST testing (15.6%).

The overall BHT results are similar to that observed by Joulia et al. (2003) who had trained triathletes complete a 1hr, steady state cycling task at 30%  $\dot{V}O_{2peak}$  while completing 20 s breath holds with 40 s normal breathing in between, three times a week for three months. Following this, static apnea was improved from 104±14 s to 155±15 s. The total apnea time was greatly different between Joulia et al. (2003) and this study due to different apnea techniques (full inspiration prior to apnea and full expiration prior to apnea, respectively). However, the overall improvements are similar with an overall mean improvement of 54 s seen by Joulia et al.

(2003) and a 34 s improvement seen in this current study, representing a 49% and 77% improvement, respectively.

During the breath hold tests, the involuntary breathing movements did not differ significantly in their onset, indicating that there were no changes in the chemoreceptor response to CO<sub>2</sub> accumulation due to the nose breathing training. My subjective experience corroborates this data as I consistently felt the dyspnea sensation beginning around 30 s, what did change was my ability to tolerate the discomfort experienced during the later stages of the apnea following the onset of the IBM's.

### **5 Kilometer time trial:**

Overall, the performance during the time trials improved by 77 s, or 6.21% (Figure 5). Differences between PRE and POST showed the largest overall improvement of 6.21%, with the MID showing a 4.58% faster time than the PRE, and the POST showing a 1.70% improvement compared to the MID. With only three data points no statistical analysis can be performed, however a comparison to other training programs can contextualize the findings for this 6-week nose breathing training program with 2 interval workouts per week.

To aid this comparison across training programs the Participant Classification Framework by McKay et al. (2022) will be used to classify myself as well as the compared studies. This framework consists of 6 tiers. Tier 0 represents those that do not meet the WHO weekly physical activity guidelines. Tier 1 represents those that do meet the WHO guidelines. Tier 2 includes sport specific training with the intent to compete at the local level. Tier 3 represents the highly trained and national competition level of sport performance with tiers 4 & 5 encompassing elite and world-class performers, respectively (McKay et al., 2022). Following the Participant

Classification Framework flow-chart, at the time of the training program I would have represented a Tier 2, or “trained” cross-country runner. Comparing participant fitness classification levels will allow for greater comparability of the results as it can be expected that less fit individuals will show greater performance improvements during a training intervention with more fit individuals showing less improvement.

A review by Kubukeli, Noakes, and Dennis (2002) indicates that interval training can improve the performance of already well-trained endurance athletes, representing tier 2 runners and cyclists performing in their respective sports. Interval training 3 times per week for 8 weeks was found to improve a 10 km running time trial by 3% in endurance runners, with similar improvements found in cyclists following HIIT training programs (improvements ranging from 3-3.5%) (Kubukeli, Noakes, & Dennis, 2002). Paavolainen et al. (1999) also found that endurance athletes who also classify as tier 2 in the Participant Classification Framework, during a 9-week program in which 32% of their training was replaced with explosive strength training, improved a 5kmTT by approximately 45 s ( $p < 0.05$ ) while the control group showed no significant differences. A significant improvement in running economy ( $p < 0.001$ ) and  $\dot{V}O_{2peak}$  ( $p < 0.05$ ) was also observed over the course of the 9 weeks in the experimental group with no changes to the control group (Paavolainen et al., 1999). The present study found almost double the performance improvement percentages compared to that observed in the several studies reviewed by Kubukeli, Noakes, & Dennis (2002) with all athletes ranking in the same performance classification tier as myself (McKay et al., 2022). Additionally, the present study showed a 32s greater 5kmTT performance improvement compared to other tier 2 participants observed in the training study by Paavolainen et al. (1999). The large improvement compared to other training programs conducted in similar populations does not prove that nose breathing is

the sole reason for the performance improvements observed in this study. It does however, indicate that the nose breathing at the very least did not hinder my ability to complete a training program that is comparable to that which a competitive runner might under-take to train for a 5 or 10 km race.

Other factors to consider that may contribute to the improved performance time include the unknown effects of temperature between PRE, MID, and POST (-11, 2, & 14 °C, respectively). Overall daily runs (including all mouth and nose breathing data) also show a slight relationship between warmer weather, with 8°C (Feels like 4°C) being the warmest observed temperature and -23°C (feels like -32°C) being the coldest, and faster running time:  $y = -0.5405x + 238.4$ ,  $R^2 = 0.2041$ . A review of marathon results and weather data from a variety of marathons over many years by Ely et al. (2007) found that performance times progressively worsen as temperatures increase from 5-20°C, with slower populations seeing a greater decline in performance than the faster populations. Research investigating the effects of colder weather has been found to be inconsistent in its findings and protocols; with most cold weather research investigating the effects of temperature on time to exhaustion tasks instead of a time trial tasks (Castellani & Tipton, 2016). One study investigating cold weather running found that male endurance athletes performed significantly better during a running TTE task at cool temperatures (-4°C & 1°C) compared to cold (-14°C & -9°C) or moderate warm (10°C & 20°C) temperatures (Sandsund et al., 2012). Another study, investigating the effects of low (-15°C) and moderate (6°C) temperatures on a 20 min double-poling performance in trained endurance cross-country skiers found that there was a significant decrease in power output during the low temperature performance (Wiggen et al., 2016). Furthermore, they indicated that this lower power output occurred mainly during the first 8 minutes of the run and that running economy was significantly

$p=0.003$ ) decreased with a large effect ( $d=0.82$ ) post-run in the low temperature condition compared to the moderate temperature condition. Similar results were found by Wiggen et al. (2013) under the same temperature conditions during short sprint (30 s & 2 min) double-poling performances as well as a reduced TTE performance during the low temperature condition. There is some disagreement in the literature with some studies indicating no differences in peak metabolic variables (Sandsund et al., 2012; & Wiggen et al., 2016) while others indicate a lowered peak  $\dot{V}O_2$  during the cold temperature condition (Wiggen et al., 2013). Based on the literature, it is likely that cold winter weather may have a negative effect on time trial running performance however the exact size and nature of this effect on variables such as pacing and metabolic variables is not fully understood. The effects of cold temperatures on running performance during race mimicking situations, such as a time trial, should be investigated further in order to determine the effects on running performance and pacing.

A similar improvement pattern between the BHT and the 5kmTT does help to support the argument that improvements in the 5kmTT may be due to adaptations caused by the nose breathing training program. The BHT, which was conducted indoors and was therefore not affected by temperature, shows that there were meaningful differences in my ability to tolerate the dyspnea sensation which may have contributed to the improved 5kmTT results. Furthermore, subjectively, I was aware and described in my daily journal that I was breathing differently after only three weeks of nose breathing during the MID 5kmTT:

*“For the first two km I was very aware of the fact that my breathing was much slower than I normally would have been breathing. It also felt weird to run with my mouth open. I know that is bizarre to say, but I felt foolish running around with my mouth flapping*

*open. That only lasted for the first two km before I started hurting which occupied all of my thoughts.” (8 March 2021, MID-5kmTT).*

I felt more relaxed during the earlier stages of the MID 5kmTT as I felt I like was able to breathe in so much more air than I was used to while nose breathing. It is important to note that this relaxed feeling did not last and the final 3km was a battle for me. This ease of breathing during the initial stages of the run may have contributed to the improved running performance between the PRE and the MID time trials in several ways. Firstly, following the constant exposure to dyspnea due to the restricted airways of nasal breathing, the ability to breath in so much air so easily led me to relax my breathing and conserved energy by reducing the respiratory muscle activity. This reduced respiratory muscle energy expenditure may have, in part, translated into a greater running output. This may help to explain the initial faster pace for the first 2 km of the MID trial compared to the PRE trial which then follow very similar pacing patterns over the course of the run with the MID showing a consistently higher running output at each point in the race (Figure 6).

Next, it is possible that the use of nose breathing while running improved my ability to tolerate dyspnea which allowed me to tolerate a faster running pace, an increased concentration of CO<sub>2</sub> in the blood and lactate in the muscles, and greater level of dyspnea during the run which may have allowed a better running performance. Looking at the 5KmTT pacing profile of the POST trial compared to both the PRE and MID trials (Figure 6) shows a slightly different pace pattern during the 5 km run. The POST 5kmTT began with the two fastest kilometers ran during any of the tests before slowing in the 3<sup>rd</sup> and 4<sup>th</sup> kilometers to almost the exact same pace as the 3<sup>rd</sup> & 4<sup>th</sup> kilometers of the MID. In the final kilometer of the POST, I was able to “kick”, increasing the pace by 8 s•km<sup>-1</sup> compared to the 4<sup>th</sup> kilometer whereas I was only able to improve

my final kilometer time by 2 & 3 s in the PRE and POST, respectively. This “kick” during the POST time trial is especially interesting when you consider my subjective interpretation of the run:

*“I absolutely blew out of the gates with a 3:00 first km and I was just hoping the wheels weren't going to fall off before the finish. I managed to hold on but subjectively, that was by far the most painful run of the whole 9 weeks. Even more so than the other 5kmTT because I started hurting so much earlier. I was already starting the self-talk before the 2 km mark. I had to consciously remind myself to lift my eyes in the last 2 km. I had no confidence whatsoever in my performance. I never check my KM times when racing so I was flying by feel and I felt bad. I just wanted to put down my best effort so that I could at least walk away with a little pride. I guess it all worked out.” (30 March 2021, POST-5kmTT).*

Despite hurting so much more during the POST run, it was the only one of the time trials in which I was able to have any sort of meaningful pace increase in the final kilometer. This improvement, not only in overall run performance but the improvement in ability to tolerate the hurt and dyspnea sensations at the end of the run may further indicate an adaptation to the dyspnea sensation even when it is augmented the metabolic by-products which accompany a maximal effort exercise.

In the end, due to the variable testing temperatures and the training program nature of this study it is not possible to quantify what percentage of the observed improvement was due to the temperature, to the overall adaptations to interval training in general, or to what percentage was due to adaptations specifically caused by nose breathing. However, at the very least we can conclude that it is possible to undertake a rigorous interval-based training program while nose

breathing only, without seeing any decrease in unrestricted breathing 5km running time-trial performance.

**Strengths:**

The n-of-1 nature of this study, in which the participant is also the researcher, allowed for a more rigorous training program than may have been feasible with multiple voluntary participants. This also assured the adherence to the running program during the especially cold and unfavourable running days. Additionally, this allowed for the opportunity to gather intimate, subjective details/data about the dyspnea sensation which may not have been as easily obtained in the standard research setting.

Next, this study was self-paced and conducted on local roads; therefore, resulting in interactions with variable conditions, vehicles, pedestrians, and dogs. As such, this study replicated the real-world reality of winter running in an urban area and resultingly has a high degree of ecological validity. To further this ecological validity, NB is often cited to be healthier due to its warming and humidifying effects compared to MB; the time of year allowed for the warming capacity of the nose to be evaluated more thoroughly as the average NB run was conducted at a temperature of  $-9^{\circ}\text{C}$  (range:  $-23^{\circ}\text{C}$  to  $8^{\circ}\text{C}$ ) (see fig. 19). Furthermore, while no metabolic measurements could be taken, this study was able to conduct a truly natural nose breathing study which other studies have been unable to accomplish due to the ventilation mask compression of the nose requiring the use of nasal dilator strips which may not accurately reflect the natural nose breathing ability of the participants (Dallam et al., 2018).

Additionally, this study took place during the COVID-19 shut down in North Bay, ON, Canada. This resulted in a very controlled rest period since I was practically unable to leave the

house except to run. My rest periods included only low intensity activities such as data entry, marking, reading, and watching television; resulting in very consistent day-to-day rest conditions which would be unfeasible during a regular study in which participants are living their lives as usual.

The combination of these factors has allowed this case study to have a unique ecological validity in which the testing conditions were real-life winter running with the resting period being extremely controlled due to the unique COVID-19 lock down conditions. This pragmatic study setting, and the n-of-1 study design has allowed us to explore the limits of what is achievable with nose breathing in a real-world winter running setting.

### **Limitations:**

Being both the participant and researcher meant that I had a thorough understanding of the NB literature and was therefore very knowledgeable about the proposed benefits of nose breathing prior to the commencement of the training and testing which may logically lead to potential bias. Additionally, for all performance measures, though naïve to the time during the actual testing, I was not naïve to the previous performance results during both the MID and POST testing days. To address this potential issue, the heart rate data from the performance tests should be considered to prove that I put in a maximal effort for each test with the average 5kmTT heart rates for the PRE, MID, and POST being: 180, 177, & 184, respectively. Unlike the time trial performance, the breath hold tests do not have an objective measure to verify my effort and motivation. Apnea duration depends largely on psychological motivation and despite being blinded to time during each individual BHT, I was aware of all of the apnea times which had been conducted previously, which could have been a potential subconscious ‘target’.

Subjectively, for each of the breath holds I felt that I had held my breath until I absolutely could not hold it any longer. Outwardly, I was told that I appeared to be in relatively the same amount of distress near the end of each breath hold at all test points, turning red in the face and shaking slightly. While the focus during the BHT looked for the effects of nose breathing, it must be noted that aerobic training alone has been shown to improve apnea time (Schagatay et al. 2000). It is therefore important to interpret the BHT results with caution and within the context of the study as a whole.

The nature of this training study made randomization impossible. As such, there is a potential that some of the performance benefits may have been due to the training effect of running over time and potentially due to the addition of interval workouts during the nose breathing phase. However, the large performance improvements of 6.21% and 77% in the 5kmTT and the BHT, respectively, are much greater than the improvements typically seen in interval training programs as described by Kubukeli, Noakes, and Dennis (2002); especially with the interval intensity in the present study being limited by nose breathing. These larger than typical performance improvements as well as the subjective feeling of improved respiratory fitness following the nose breathing phase may indicate that nose breathing facilitated performance improvements. The exact amount of performance improvement attributable to nose breathing however, is impossible to determine.

To compare the three-week MB to the nose breathing on a weekly basis, which included two interval training days per week that were not counted as daily runs, the mouth breathing data was averaged into groups of 4 successive days which reduces the variability of the results by buffering any extreme values which may have occurred. While this does reduce the variability of the mouth breathing phase data, it should be noted that the weather was noticeably harsher

(colder and worse traction) during the mouth breathing phase and this technique may have lessened the impact that poor weather conditions may have played on variables such as pace and heart rate.

Next, this study lasted from 19 January-30 March 2021 resulting in a range of running conditions which generally warmed over the course of the 9 weeks of testing. It is unclear the degree to which, if any, this general warming may have affected the daily runs and tests. Finally, this study's main performance measure, the 5kmTT, was conducted while mouth breathing and therefore the findings of this study cannot shed light on the use of nose breathing during maximal exercise following a 6-week nose breathing training program.

#### **Future recommendations:**

Based on my subjective experience during this training program, for future studies or those planning to implement a nose breathing running program, I suggest the inclusion of easy runs or at the very least, more than 1 rest day per week in order to prevent overtraining/burnout effects experienced during the later stages of this study's protocol, and to minimize the potential risk for injuries.

Following the performance and subjective fitness improvements observed in this case study, future research into nose breathing should investigate the changes that occur in the metabolic factors such as running economy ( $\dot{V}O_2$ ), peak oxygen consumption ( $\dot{V}O_{2peak}$ ), ventilatory equivalent ( $\dot{V}E/\dot{V}O_2$  &  $\dot{V}E/\dot{V}CO_2$ ), and blood lactate following prolonged nose breathing training.

Additionally, more investigation is needed into the ability to effectively nose breath during longer-duration, high-intensity exercise. Finally, the effects of mood and mental fatigue on the severity of the dyspnea sensation require further investigation.

### **Conclusion:**

Of the 71 program days, I ran for a total of 57 days, 54 daily runs and 3 test days, for a total of 596.06 km. During the beginning of the nose breathing phase I experienced extreme sensations of dyspnea during my daily runs and especially during the interval workouts. Despite this, I found that it was possible to complete the 6-week running program while breathing only through my nose. Furthermore, I not only completed the training program, but ran significantly faster than the mouth breathing phase during the last week of the nose breathing phase, meaning that it possible to not just “get through” a nose breathing training program, but it is possible to train at an equal level to that of mouth breathing after only a short (<6 week) adaptation period. Furthermore, the heart rate data indicates that this significantly faster pace was achieved with no significant differences in heart rate, indicating some change in running fitness. The performance measures also generally showed an improvement with a 5-kilometre time trial improvement of 77 s and a maximal breath hold increase of 31 s from PRE to POST nose breathing training. It has been demonstrated that nose breathing can be an effective method of training during winter running conditions following a relatively short adaptation period if willing to constantly push oneself to a “comfortably uncomfortable” pace and to endure the overwhelming sensation of dyspnea which can accompany fast running. It has been demonstrated to not only maintain fitness but potentially can improve both running fitness and running performance. These findings

regarding nose breathing, coupled with the abundant amount of literature highlighting the health benefits of nose breathing, should be seriously considered by those partaking in or coaching endurance events, especially those which take place during cold weather, as an alternative to a standard mouth breathing training protocol.

Based on my experiences during the nose-breathing training program, I would therefore recommend that runners training for races by nose breathing while running begin at least 6 weeks prior to the race; with an even longer adaptation period likely showing further tolerance of dyspnea, potentially further improvements in fitness and performance, and could allow for a less rigorous training program to be followed to achieve these potential benefits. Following the completion of this study, it is the recommendation of this researcher to follow the advice of George Catlin (1870); “keep your mouth shut when you read, when you write, when you listen, when you are in pain, when you are walking, [and] when you are running”.

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## Appendix A- Ethical Approval



January 18, 2021

Dr. Graydon Raymer  
Faculty of Education and Professional Studies  
Nipissing University



Dear Graydon,

It is our pleasure to advise you that the Research Ethics Board (REB) has reviewed your protocol titled 'The effects of nose-breathing-only training on physiological parameters related to running performance: a case study.' and has granted ethical approval. Your protocol has been approved for a period of one year.

**Modifications:** Any changes to the approved protocol or corresponding materials must be reviewed and approved through the amendment process prior to its implementation.

**Adverse/Unanticipated Event:** Any adverse or unanticipated events must be reported immediately via the Research Portal.

**Renewal/Final Report:** Please ensure you submit an Annual Renewal or Final Report 30 days prior to the expiry date of your ethics approval. You will receive an email prompt 30 days prior to the expiry date. Research protocols may be renewed a maximum of three times. If additional time is required, a new protocol must be submitted.

Wishing you great success on the completion of your research.

Sincerely,

*Steve Hansen, PhD*  
Chair, Research Ethics Board

### Appendix B - Journal Entries

Date	
<b>19 Jan 2021</b>	First day of the control phase. I was very excited to get going. This may be why my pace was so quick today (4:06avg). Felt really good for the first 30 min but really felt it for the last 10. I think I'll have to start my runs more conservatively if I want to make it the whole 9wks. Snowing today. The snowflakes hurt my eyes. Slippery on corners and third st e (~100m) was especially slippery. subjective traction score of 6/10
<b>20 Jan 2021</b>	Today was cold! But if anything, it made running easier because I was trying to warm up. I was actually faster than yesterday but it did not feel as difficult. This may have been because the traction was much better today. I slipped on the first two corners so I had to take the rest of the corners slower, all the straight stretches were fine. I found that I had a lot of mucus/phlegm to deal with today...I was spitting constantly. No coughing or wheezing afterwards despite the cold.
<b>21 Jan 2021</b>	It snowed 5cm last night and was still snowing when I went out. The traction was not very good. Especially on the sections where there is any kind of incline, I would constantly be slipping. My hips were really starting to tighten near the end of the run. Even though I felt like I was working hard, I didn't feel like I was going anywhere.
<b>22 Jan 2021</b>	Consistency. Before I looked at the splits today, I was convinced that they were all within 5s of each other. I wasn't quite that consistent, but it was within 10s. I felt good the whole run. I was surprised afterwards when I saw the average split time because I thought that I was about 5s/km slower than the 4:03avg I put down. I didn't think that I ever went below 4:00.
<b>23 Jan 2021</b>	It was cold today! I felt good running, traction was good and there were not many people out today, maybe because it was so cold, so I didn't have to fiddle with my bandana or cross the street very often. I had to be constantly spitting today and I almost had to stop running during my 4 <sup>th</sup> -5 <sup>th</sup> km because I was choking on phlegm and mucus. It was truly disgusting, and I pity anyone who listened to it. Since getting back (15min), I've had a bit of a cough and I feel like something is tickling the back of my throat. Looking forward to a rest day tomorrow.
<b>25 Jan 2021</b>	Today was very Slippery, it snowed last night and not all the sidewalks and roads had been fully plowed. My hips began to feel tight around halfway. Lots of phlegm and mucus again around 5km mark. Some coughing following the run for about half an hour.
<b>26 Jan 2021</b>	Great traction today. I was running into a bit of a head wind on Fraser street which made things feel slow (and looking at the pacing afterwards it was) but I had a tail wind down Ferguson, and I felt like I was barely putting in any effort there. Wind was 19km/hr gusting to 31km/hr. No coughing or much mucus during the run. Following, coughing and intense hiccups.
<b>27 Jan 2021</b>	I had a bad sleep last night. I was tossing and turning and coughing from about 3am onward. I did not want to run this morning. Once I did start running, I pretty much shut

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	off my brain. So, I really don't have much to say about the run. Legs feel good, lungs are suffering a bit. Coughing, phlegm, yay winter running.
<b>28 Jan 2021</b>	Today was cold but I was well dressed for it. I felt slower than yesterday right from the beginning; looking at it after, my first km was one of my slowest. I felt smooth in the middle section but then around the 7 <sup>th</sup> km I started having to go to the bathroom. The last 2km were a real battle to finish.
<b>29 Jan 2021</b>	I am getting very bored of this route. Today was good, I felt smooth and consistent the whole way. The only hiccups that I encountered was an especially stringy spit that I found to be a challenge to dispel. Near the end I noticed that my breathing sounded a little raspy. following the run, I have been coughing since coming inside (~20min ago).
<b>30 Jan 2021</b>	I did not want to run this morning. This may have been more to the fact that it is saturday rather than fatigue, however. Once I was out, I felt dialed in. I felt consistent and relaxed. A major part of this, I think, was that I resisted the urge to run the first km faster to warm up. I just trudged along and endured the cold until I warmed up. I felt much less surgy than when I normally run (for an ex-varsity runner my pacing control is garbage). I felt the same way yesterday and I much prefer this to the surge and ebb approach. Minimal coughing today, spitting often as usual. Lower lip and tongue were getting cold by the end.
<b>1 Feb 2021</b>	Today felt good. I was not as consistent as I thought, looking at the numbers afterwards, but this was because I ran two km sub 4min which was surprising to me. I ate it pretty hard right before I stopped to take the pulse Ox measurement and ripped my new running pants. Very annoyed about that. Then I couldn't even get a pulse ox measurement from the watch (so I did it immediately after finishing the whole run instead). Overall, the run felt fine. Some light coughing afterwards, and of course the usual in-run spitting.
<b>2 Feb 2021</b>	Great traction today! Today was fast but consistent. I felt good the whole way. When I saw that I was ahead of my usual area at the pulse ox measurement I was worried that my pace would drop off but that didn't happen; I didn't begin perceiving it as more difficult. I think the initial fast pace was due to the cold. It was only -15 today but I was freezing right from the get go which may have spurred me into a faster pace. I'm not sure. Less need to spit today (still did, obviously, but not nearly as much). Some coughing afterwards. Feeling good.
<b>3 Feb 2021</b>	Today was tough. Felt relatively good in the first half but really seemed to die in the second half. I don't know if it's because it was much warmer than I was expecting (not that made much of a difference except I had to lose a layer on my 3-4 <sup>th</sup> km) I just couldn't keep a steady pace like I could yesterday. Almost no need to spit today. The usual coughing afterwards...and hiccups (that's twice now: 26 Jan also)
<b>4 Feb 2021</b>	I felt like I was floating today. My legs felt great, it was a little warmer and the traction was almost perfect except for a few ice patches. The fast pace was certainly helped by the strong tailwind I had from Chippewa st down to Worthington st (~1km) which is also generally downhill which resulted in my three sub-4min kilometers (3,6, &9). My two slowest were both heading the opposite direction on Fraser st, but while I was running, I barely noticed a head wind (there was some, but it certainly did not slow me as much as the tail wind helped). I had much more phlegm and spit during the run compared to yesterday. More coughing afterwards also.

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<b>5 Feb 2021</b>	Today was SLIPPERY. I almost fell pretty much immediately, and it did not get better from there. The corners were treacherous. If you had asked me in the first two km, I would have predicted a slow run, limited by the lack of traction and a relatively low RPE. By the halfway point of the run however my tibialis anterior of both legs were beginning to get tired. By the end of the run my lateral gastrocnemii were also very tired. I checked after the run and I had taken approximately 7000 steps during the run. My foot slipped at least a little on every single step. Some I really slipped. I never hit the ground, but I had at least four arm-flailing, almost-falls. Everything else about the run was great! Some very light coughing after the run.
<b>6 Feb 2021</b>	Today was the first day that my legs have really hurt. They were sore right from the beginning. I felt fine all day yesterday but apparently my legs needed more of a rest after the slip-fest yesterday. The traction was better today but I had quite the headwind to put up with coming down Ferguson st (~1km), the weather network said 25km/hr with gusts up around 45km/hr and I believe it. At least tomorrow is a rest day. Besides my legs, it was a very uninteresting run. Everything else felt great, although I did have two or three small coughing fits during the run. I continued coughing after the run for quite some time. My throat is a little sore now.
<b>8 Feb 2021</b>	Today's RPE score was 17 for three main reasons: 1) it was very cold today; tons of spit and phlegm all made more inconvenient because of the face covering I was wearing for the cold. 2) my legs are still a little sore from Feb-5 <sup>th</sup> and felt quite heavy, especially in the second half of the run. And 3) this is the big one, right as my watch buzzed that I'd reached the 7 <sup>th</sup> km my body decided that it had to poop. So, began, what I feel to be, one of the most physically and mentally challenging 2.73km of my life. Haha I'm exaggerating a little bit, but I also ran the last 0.73km at a projected pace of only 4:43 (36s slower than my average pace); so, I was certainly hurting. Today was the last day of the mouth breathing phase however and I am very excited to move on to the next phase of this experiment. Hopefully today's post-run coughing (quite heavy today) will be the last (except for the test days because those are mouth breathing also).
<b>10 Feb 2021</b>  <b>BHT</b>	1) the breath hold gets terrible very fast. I felt fine until the IBM's and then it felt like my lungs were trying to gulp air for me. 2) this one I at least knew what to expect. I was able to hold on slightly longer than the first. 3) third trial was almost the exact same as the second. I thought that it was longer, however. It's REALLY hard to tell how much time has passed. Michela said I would begin to shake similar to "someone who flexes hard and begins to shake" after the IBM's had started and that I clenched my jaw, which I was unaware of.
<b>10 Feb 2021</b>  <b>GXT</b>	Because this is not on a treadmill the pacing is slightly more variable, but I felt I did a good job of sticking to the pace. I would check my watch every 10s or so to see if I was on pace and adjust accordingly It became harder and harder to keep a consistent pace in the later stages because each stage it became more difficult to think clearly about anything other than breathing. For the first two minutes (5:00 pace) it was easy, if anything, I had trouble running that slow. the 3 <sup>rd</sup> & 4 <sup>th</sup> min (4:50) were also fine. In minutes 5&6 (4:40) I had a little trouble keeping the pace at 4:40 and ran closer to a 4:30 avg pace. Around this point my nose was running and I was constantly wiping watery snot out of my mouth/off my face. I would say 4:30 was becoming difficult to breath. The last three minutes were where my pacing began to fall apart a little. My pace slowed

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just as I was entering the 7<sup>th</sup> minute (4:30) so I tried to adjust my pace but got going a little too fast and had to reign it in (at one point the watch said 4:00 pace). What I think happened was I became more preoccupied with trying to breath and less on pace. following this short increase in pace I was really struggling to get air in and keeping a steady pace was getting more and more difficult as the dyspnea mounted; making it difficult to concentrate and taking up a lot of my thoughts. I felt as if I was unable to get enough air in as if my nostrils were closing slightly each time I inhaled, but they felt wide open when I breathed out. By the time the 10<sup>th</sup> minute came around I was fighting panic and begging my mouth to stay shut. I was on pace (4:10) for the last 38s but that was mostly luck. I was so focused on trying to keep my mouth shut and keep going. When I finally gave in and took a breath, I felt almost instantly calmer. My nostrils burned afterwards, obviously not used to breathing in lots of cold air. No coughing afterwards. Only spit once when my nose first started to run during the 3<sup>rd</sup> or 4<sup>th</sup> minute.

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**10 Feb 2021** That felt terrible! My chest still feels cold, and it's been about 20min since I finished. I don't know if it was my mind playing tricks, but I swear I could feel where my bronchi split from my trachea because of the cold air. I only noticed this after I had stopped. I was choking on spit for the first two km and then I either got it all out or something else changed because it wasn't an issue later. I've been coughing heavily since. I felt like I was flying the first km and settled in well for the rest of the run. I felt like I had nothing left in the tank at the end for a kick which is unusual for me. I thought that I was going to throw up for the last kilometer or so. Still might if I'm being honest. My lungs are tired and sore and so am I.

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**11 Feb 2021** Today was the first day of nose breathing. I clearly did not learn anything from yesterday. I started the run with a 4:07 which created a strong feeling of dyspnea that peaked at around the 1.75-2km. I was saved by the tail wind down Ferguson which helped me cruise through the third km while I tried to regain control of my panicky feelings. I actually kept a decent pace through 6km (average of 4:18) but it all seemed to catch up with me in the last three km. The air hunger feeling began to feel overwhelming and I felt like I couldn't slow down enough to catch my breath. I refused to stop on principle, but I felt like I was slowly suffocating the whole time. The only reason the 9<sup>th</sup> km was faster than a 4:30 was because it was down Ferguson st., there was no second wind for me as I approached the finish. No spitting during this run but there was a ton of snot! I brought tissues but they hurt the nose when it's cold out like today...I must figure out something else for that. One cough today, just as I walked into the house.

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**12 Feb 2021** Today I went out slower than I did yesterday. I kept a pace that held the dyspnea within the tolerable limit for pretty much the whole run. I only felt overwhelmed by it one time when I hustled to cross the street when a car waved me on. It is incredible how quickly you can cross that threshold into panicky and how slowly you return to a semi-comfortable sensation without taking a breath from the mouth. My nose is quite raw and sore. I didn't use any tissues today (I just let 'er flow!) but my nose still hurts as if I had. I think the cold air might be drying out the skin around my nostrils. I might try a bit of petroleum jelly tomorrow. No coughing or anything afterwards.

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**13 Feb 2021** Today I really wasn't feeling it. It could have been because it is Saturday or maybe because it feels like -32 for the third day in a row...who's to say? Regardless, I felt sluggish for the whole run and my digestive system and I were not agreeing. I didn't have to go to the bathroom as I have in the past, but I felt generally uncomfortable today.

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On the other hand, I was more pre-occupied with my discomfort and it took my attention away from my breathing. I never dipped below 4:10 today however which is normally the about the point where the dyspnea really starts to build. The arches of my feet are sore today and I have no idea why. I got lip chap (couldn't find Vaseline) for my nose and I think it helped a little against the cold air. A couple light coughs today...it felt like I had a light tickle in the back of my throat.

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**15 Feb 2021** Today was the first of the interval nose breathing days. The pace was calculated from my 5kmTT time and the "1km threshold" pace was used (3:45) this was way too fast. I held on to the pace for two minutes on the first interval before I had to drop the pace drastically (~4:10) and then only another 29s before I had to stop running and take two breaths from my mouth. I kept running and nose breathing but I could only manage a ~4:30-4:35 and even that only lasted 30s before I was stopped and mouth breathing again. The first stop was just a pause, I never even fully lost momentum. The second was a hands-on-the-knees, sucking air, stop for about 5s. The last ~55s I held a ~4:10 pace and I was grateful I could manage that. The second interval I fared about the same. The pacing graphs look shockingly similar. I again held it for about 2min then absolutely died. My two stops were more like quick pauses but from ~2:42-3:18 I was barely moving. My peak pace during that time was 4:46. The last 40s I was able to slowly build back to about a 4:10 right before the end. For the 3<sup>rd</sup> and 4<sup>th</sup> intervals I tried running at the "marathon" pace which is 3:58. Even this I had trouble maintaining, again stopping in the third interval twice. The fourth was the only one in which I did not stop running. I started at about 3:55 and steadily slowed to a 4:15 by the end. I took a breath about once every 10-15s or so for the last minute and a half. The first mouth breath was involuntary, but in the exertion of trying to keep my mouth shut and keep running, I almost pooped my pants, so I took a breath when I needed it after that. Traction wasn't fantastic but it was hardly the reason I wasn't hitting the pace. It is shocking how long 4 minutes can be when you feel like you are slowly drowning.

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**16 Feb 2021** Today was cold, snowy, windy, and a little slippery. Overall, I would describe the run as "comfortably uncomfortable". I was just at the point where I felt the dyspnea but never felt distressed about it. I did have one 03 mouth breath. It happened just after the 5km mark, I lost focus so a second and my mouth just did it. I was cruising along today. No coughing afterwards or spitting during, just some snotcicles.

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**17 Feb 2021** Today was day #2 of interval training. It went much better than day 1. For starters, I didn't have to adjust my pace goal for the third and fourth intervals. All intervals were run with the pace goal of 3:45. The first interval was shockingly easy. I felt like I could get so much air into my lungs and there was almost no dyspnea until the last 20s approximately. The second was harder, in the last two minutes I had to take a few breaths from my mouth to keep the pace. The third was similar to the first however, the dyspnea started to creep up in the last minute and a half, but I managed to hang on to the end without taking a mouth breath. There is only one noticeable incline on the run and it is about halfway down ferguson st. and this incline is usually offset by a tail wind. Today there was practically no wind and on the fourth interval I cracked at the top of that hill. I was so determined to get up the hill and keep my composure, but I just couldn't do it. So, I still failed the workout...but I was closer.

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**18 Feb 2021** I really debated what RPE score I should give today, and I decided on 17. Today, in summary, I was flying. The first 6km were crazy. I couldn't believe that the dyspnea

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didn't start creeping up. My best guess is that it was due to the temperature. This is the first day of nose breathing that the ambient temperature was above -18 degrees Celsius for a non-interval workout. It was -11 today and much easier to breathe; I think it's because my nose hairs didn't freeze together. Today was the only day so far that I didn't have substantial icicles forming from my nose. I think that my nose hairs freeze and clump together making it harder to breathe in the colder weather. The dyspnea did catch up to me. After the ~6<sup>th</sup> km I found that my body couldn't buffer a momentary pace increase. For example, when crossing a road, I check both ways which momentarily slows me and I will usually increase my pace for a few strides to make up the difference (I've done this every single day) but in the last few km I found that even that slight deviation from the pace I was holding, would cause a mildly panicky sensation (dyspnea). To me, this means that I was truly right on the line for pacing and that my body is just not quite ready to give me a full 40min there just yet. I had to take a couple mouth breaths in the last km. It was as if I had reached some kind of threshold. Last complete km was a 4:11 pace but it was full of surges and ebbs as I tried to re-gain composure. The last 0.72km was projected as a 4:22 which really illustrates the degradation of my pacing when compared to the 4:02 I was able to put down 2km prior. No coughing or spitting and the snot level was at a pleasant minimum.

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**19 Feb 2021** Today we re-ran the incremental exercise test. This was due to concerns with the consistency of my pacing in the last trial. To mediate this issue, I began at 5:00min/km and increased by 10s/km or 5s/500m every 500m until exhaustion. In the end I reached **GXT\*\*** 4.20km in 18:13 and I was 200m into the 3:40 section. Prior to the 4:20 pace, it was very easy. Somewhere during the 4:10 I started to become aware that I was breathing much harder and had begun to feel the dyspnea creep up. I was able to hold the pace well enough, but the last minute was brutal. I felt like I was completely red in the face and it took a lot of concentration to not involuntarily take a breath.

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**19 Feb 2021** I ran after the GXT test, so I was a little bit more tired than usual. My possibly be why I found the dyspnea to more of an issue today. Today was the first day that tiredness in my legs factored into the RPE score. Really overall not an interesting run except for that fact that I had to stop 4 times for cars and 2 to go pee (normally I never have to stop, maybe a quick pause for one car but never more and never to go pee). I'm not sure what's up with that, I wasn't aware of drinking more water than usual.

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**20 Feb 2021** I couldn't believe what was happening today. I was not feeling all that energetic today, my POMS score said that I had one of the worst affects so far going into this run. But once my shoes hit the snow I was gone. I kept waiting for the crash, but it never came. I did have to take 4 breaths out of my mouth as I reached to top of the hill on Ferguson st. During the 9<sup>th</sup> km. So, it wasn't a perfect run, but it's also the furthest I've ran yet in either phase. I put the RPE as 17 because for the first 6-7km I was truly right on what I feel was a 16RPE, the last 3km was more of a statement for me. I couldn't take my foot off the gas, I wanted to push the limit. So for that section I was probably more at 18, sometimes flirting with 19, RPE with the dyspnea and my legs starting to feel it. So overall, the run was a 17. I'm super happy with this run and I am super excited for a rest day tomorrow.

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**22 Feb 2021** Today was supposed to be my third interval workout however the weather did not facilitate fast running today so I switched over to a regular run. I will try to run the interval tomorrow however the forecast is not looking great so we will see. The traction

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was terrible today. It was very hard to maintain a decent pace as many of the roads/sidewalks were not plowed at all and I was being pelted in the eyes by snowflakes constantly. When I stopped to take the pulse Ox measurement, my eyes were aching, and I had to hold them closed for the whole stop. I continued the run however I made a conscious effort to ignore all the training I have ever received about keeping my head up while running. I kept my eyes trained 5m ahead of me and that's it. Overall, the run was fairly standard. A disappointing follow-up to my sub-four pace on Saturday, but that's how it goes with winter running. No coughing or anything today and I didn't have to wipe my nose once which is a first.

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**23 Feb 2021** Today was super slushy so I decided to push the interval to tomorrow. I'm thankful that I did. The corners were deadly, and the sidewalks weren't plowed. It would have been very frustrating trying to get four minutes straight of decent traction as the only good spots were out on the road and there were a lot of cars out today it seemed. Sensation wise, I felt like I was working very hard to go nowhere fast, but I had no problem getting air and I only had one time when the dyspnea really took up a large portion of my thoughts but that went away quickly within a minute, maximum. I don't think about the breathing nearly as much as I did in the first week. I am not sure whether this is because of the temperature difference or because I am getting used to it. Hopefully there will be some more cold weather so that we can really test that.

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**24 Feb 2021** I beat the snow. There is a storm advisory today and while I am currently writing this it is really coming down. When I left for my run however, there was just a light dusting of snow, much better than the last two days. Today was almost easy to complete the intervals. The first, I ran a 3:33km and then a 3:34km during the second interval. I found it very interesting because the first two intervals seemed to fly by. During these runs I have a rolling 2min timer going on my watch so that I get a halfway beep during my 4min runs and 2, 1/3 beeps during the recovery sections. The halfway beeps surprised me on the first and second because they both felt so easy that I didn't think that much time had passed. On the third interval time seemed to slow down slightly. I was able to still maintain the pace below my target pace of 3:45 as I clocked another 3:34, but perceptually this interval was more difficult. The fourth interval I managed to hold a 3:37km, this was likely aided by the slight downhill of ferguson st (although I started the 4min at the bottom of the longest hill section on my route). The dyspnea was present throughout the last interval, likely encouraged by the hill right at the start, however it was never overwhelming; just present in my thoughts. The snow was really starting to come down by the last interval and my eyes were sore from getting pelted by snowflakes. Today was a huge success.

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**25 Feb 2021** I went out of the gates a little bit fast today. Could have been the cold, that I finally had decent traction or that I had no trouble with dyspnea despite the -20-degree temp. Regardless my first four km were sub-4. I started to feel a bit tired around the 6-7km mark and by the 9<sup>th</sup> km I really just wanted it to be over. I never felt overwhelmed by dyspnea however even then. It was more of a whole-body feeling of "I don't want to do this anymore". I forgot to put vaseline or lip balm on my nose today so the skin around my nostrils is a little irritated. I don't think I have mentioned this before so I'll say it now, when I breath hard through my nose there must be some vibration or perhaps irritation, regardless often when I stop running and continue breathing through my nose, I smell the exact same thing I smelled when I got a bad sinus infection. It's kind of like a

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gross fish smell. I am not sure if this is because of fluid being vibrated out of my sinuses or perhaps due to irritation from all the air passing through, but it never lasts long and there is never any pain or pressure like an actual sinus infection.

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**26 Feb 2021** Today I did not feel as good as the 24<sup>th</sup> for the intervals but they still went very smoothly with each having an average pace of well below 3:45 (fastest (1<sup>st</sup>) was 3:34 and slowest (4<sup>th</sup>) was 3:41). Honestly there is nothing to talk about today. Dyspnea wasn't even slightly on my mind. The only interesting thing was that I had to stop for two cars during the second interval. I'm happy to have completed this interval set and I'm excited to move on to the 4x5min @ 3:45 and curious to see how I will handle it.

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**27 Feb 2021** Today was snowing very heavily. The plows were not out and so I was running through several cm of snow unless I could get into the track of a car that went by recently. I also found that running into the wind was more challenging as I found the snowflakes would constantly hit me in the eyes while running which was uncomfortable and forced me to drop my gaze while running. All these factors resulted in a very variable pace. I only struggled with dyspnea one time during the run, I slipped on some ice that was beneath the snow and had quite the dramatic stumble, I never opened my mouth and I was almost immediately aware of the air hunger. I don't know if it was due to the sympathetic response to my near-fall or if it was due to all the extra, quick movements which caused the dyspnea. It happened in the first few km and the pace/HR graph show a point just after the 8min mark in which my pace drops suddenly but the HR spikes which may have been this incident which makes me think that the dyspnea was spurred on by a sudden increase in sympathetic drive. Besides that, it was a fairly standard run.

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**1 Mar 2021** Today was the first of the second level of intervals (4x5min). I didn't quite breeze through this one as easily as I thought I might. I was still able to keep a pace below the required 3:45 for each of the intervals (although I was dying in the last one and was only saved by the ~3:30 pace I ran for the first 2.5min), dyspnea was never far from my thoughts in the last minute of each interval. The first two were relatively easy, but in the third and fourth I really struggled through the last minute. Each interval took place on the same exact section of the route. From just after king st down the rest of Fraser, onto Chippewa, and down Ferguson until approximately second or first st. The hill on Ferguson is where the dyspnea really came for me, I reached it after about 3:40 of the total 5min, by the later intervals it was really a struggle to climb it without taking a breath. In the fourth interval I had to slow past the 3:45 in order to not take a breath from my mouth. After making the hill, the dyspnea didn't go away for the remaining ~45s but it certainly wasn't as panicky. I think I can pull that off again but I am nervous for the following increase in interval intensity.

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**2 Mar 2021** Yesterday, during the day, I was marking some tests and moved weirdly, and my back went into complete spasms. I had Michela cup my back but this morning I still woke up very sore. I originally thought I was going to just take the day off but I decided to stretch and see if I could get moving. As much as I really didn't want to, I decided that I would be able to attempt a run. Add in the -23 that I didn't see coming and I was not a happy camper leaving my house. The run was actually not painful. I was terrified of slipping however, even though it was almost perfect traction except a couple not-even-that-slippery ice spots. But I knew if I went down, I probably would stay there. I have no memory of any dyspnea during the run, I was 100% focused on running smoothly with as little bounce as possible; I was so hyper focused on this that I actually forgot to breath

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	out of my nose for the first thirty seconds of today's run before I clued in and shut my mouth.
<b>3 Mar 2021</b>	Today the intervals were easy, no panicky feeling in the last minutes and no big increase in dyspnea even when I was on the sidewalk on Chippewa and had to work extra hard to keep the pace in the slippery slush. Nose breathing and running is just a mind game of putting your mind somewhere else and ignoring what your brain is trying to tell you.
<b>6 Mar 2021</b>	Today I opted to switch the test day for the first day of week 7. I did this because I was not sure how my back felt after the two days off and wanted to test it out. I can't say that it's perfect because as I'm writing this my back is a little tense and achy, however it did not interfere with my running ability at all. I was flying today. My average pace was 3:56 and if anything, I was holding myself back during the first 5km. I never had any incidents where I was overwhelmed by dyspnea and the only real trouble I ever ran into was a stitch in my side around the 9km mark which not very severe and did not last for very long (~250m). no coughing afterwards but there was quite a bit of snot-rocketing today in the first few km.
<b>8 Mar 2021</b> <b>BHT</b>	Test day! The breath holds went well; I was able to hold it for longer on every trial today. The first one was very difficult; I felt the panic creep up very quickly. The other two, I tried to divert my focus away and I lasted a little longer. The after the third trial I felt very lightheaded. I messed up pressing start on the watch twice in a row which is embarrassing, so I don't have avg HR data during the first two trials. HR right at the end was 66 & 54 which is like the HR data of the third trial as HR begins higher, dips during and then raises slightly right at the end when it really becomes a battle.
<b>8 Mar 2021</b> <b>GXT</b>	Today I made it to the 3:20 pace (or 1:40/500m). It was pretty easy to be honest. I really only started experiencing dyspnea in a meaningful way during the 3:30 pace at about the half way point (45s-1min). After that I was just struggling to hold on. I managed to cling on for 78s (400m) during the 3:20 pace, I thought I was going to make it and then I involuntarily took a breath from my mouth. It was so fast and loud that I actually made myself jump a little bit. Its funny when you get to that point of dyspnea because it becomes really hard to monitor everything going on. The time, distance, keeping the pace, monitoring traffic and pedestrians and then also only breathing through the nose. I guess I just couldn't focus on the nose breathing anymore and my brain just took over. During the last little bit I was fighting to breath so hard that my left ear kept becoming plugged as if I was pinching my nose closed and breathing against it.
<b>8 Mar 2021</b> <b>5kmTT</b>	I started the run today feeling awesome. For the first two km I was very aware of the fact that my breathing was much slower than I normally would have been breathing. It also felt really weird to run with my mouth open, I know that's bizarre to say, but I felt like an idiot running around with my mouth flapping open. That only lasted for the first two km before it started hurting and that really took up all my thoughts. I noticed on the third km that my breathing was much faster than the first 2km. I also began experiencing the beginnings of a stitch in my left side. I just tried to keep my head up and keep breathing smoothly and it went away within half a kilometer. The last two km were a real battle to keep the legs moving, I had no kick at the end. I had to spit out phlegm about 4 times during the run. I almost vomited when I finished.
<b>9 Mar 2021</b>	I was really surprised that I pulled off a 4:01 average pace today. I didn't feel bad necessarily and dyspnea was never an issue, but I felt tired and the run seemed to drag on and on. There were a lot more people out enjoying the warmer weather, so I was mostly

running on the roads to avoid them. The only real hiccup in my run today was that I had to go poop during the last km. My digestive system had some difficulties after the 5kmTT yesterday and I don't think the issues are fully resolved.

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**10 Mar 2021** Today was alright; I got through the workout fine. I just felt tired. Honestly the run was really boring today, and I don't have a lot to say about it, except:

I got screamed at (and called "a f\*\*\*ing son of a f\*\*\*ing b\*\*\*ard") because a lady's husky took off sprinting and she was upset that I, a jogger and therefore the obvious candidate, didn't tackle her dog...even though it was on the other side of the street sprinting in the opposite direction...and not my dog. That was just after my third interval and I had a good long laugh at that, so I was mouth breathing more than normal due to the laughter.

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**11 Mar 2021** Today felt super easy. I didn't feel like I was putting in any hard effort at any point during the run. I knew I was flying when I stopped to check the pulse ox at my normal point, and I was almost a minute ahead of where I normally would be. I thought that it would get more difficult, but I just kept going and the perceived exertion never crept up nor did the dyspnea. My slowest km (km7 a 4:05) was due in part because I completely ate it while I was running down Fraser st on some ice that I didn't see somehow. I cut my right forearm but was otherwise up and running almost immediately (probably spurred on by the embarrassment of falling on seemingly flat ground).

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**12 Mar 2021** Today my hips were very tight. After running yesterday, I pretty much sat all day marking and my hips did not appreciate it. I felt tired for the whole time and lacked motivation. I was still able to complete all the intervals, although I was close to being outside the +/- 5s for the last one. I'm feeling a little run down. I am looking forward to my rest day on Sunday, dreading tomorrow.

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**13 Mar 2021** I don't think I've ever been on autopilot so much in my entire career of running. My mind was wandering all over the place and I barely paid attention to the run. I surprised myself when I stopped to take the pulse Ox measurement because I was lost in a thought. my thoughts were all over the place from my med school interview, to the upcoming 3MT, then I wondered what the data would end up looking like, and then just random things from there. Clearly dyspnea was not a factor in today's run because my mind was completely free. I guess my autopilot settings are a little on the fast side, but I was feeling good when I woke up this morning and nothing was sore during the run. I am certainly happy that tomorrow is my rest day.

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**15 Mar 2021** I gave today a 16 RPE rating but I would like to put an asterix on that. It felt crazy easy today, I was running at the usual clamped 16RPE and felt great for 8km (slowest was 3:59/km) and I never stopped running at the 16RPE clamp, but I slowed to a 4:00 in the 9<sup>th</sup> km and to 4:09 in the 10<sup>th</sup> km. So, I felt good today, but I think it was fading pretty quickly near the end. Not a pace that I think I would have maintained for much longer. Other than that, had a bit of a stuffy nose during the first km but I was able to clear it out relatively quickly. I have been noticing a substantial increase in thicker snot recently. I thought it might be the beginnings of a cold, but I am not sure.

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**16 Mar 2021** Today was hard not because of the intensity or perceived effort, but because I have a sore left foot. It was a little achy yesterday and this morning, but I was hoping it would be one of those little aches that goes away after a km or two. It was not and it hurt the entire run. It is the lateral side of my foot approximately midway up my foot (starting

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about where the cuboid and fifth metatarsal meet). Will definitely need to keep an eye on this. I am currently icing it and I am going to alternate to heat in about 15min. Besides this the run was quite boring; nothing to report.

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**17 Mar 2021** Happy St. Patrick's Day! I wore green to celebrate. Today I also wore a different pair of shoes. My foot was feeling much better, but I thought I would make it a little easier on myself for this interval workout (which I knew would be about 15km). I normally run in the New Balance Vastu's, but I switched today to my old shoes, the New Balance Fuel Cells, because they have a slightly thicker sole which I thought might help. It did, I was able to complete the workout in relative comfort and so I will give them another try tomorrow. I will switch back to the Vastu's ASAP. The run was long today. A 3:45 pace for the last 5min of an almost 15km run sucks. No real difficulties or dyspnea, but I was full of snot today which was gross to deal with.

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**18 Mar 2021** Today I felt terrible before the run. I slept in so I was rushing around, I was in quite the battle with my digestive system and just overall felt tired. When I was running, I still wasn't feeling the best. After about the first km (3:55 and my slowest) I pretty much shut my brain off and just ran. I couldn't say what happened on the run, autopilot was on and I was cruising. I ended up running my fastest 10k yet (39:08) and running almost 250m farther than any other run so far. And it felt easy. I had no trouble getting air in, no dyspnea, and I noticed that my breathing rate was much slower than what I would have thought it should be for the pace I was moving. Important to note that I was wearing the New Balance Fuel Cells again, my foot is feeling much better but still a little sore, so I played it safe and stuck with the Fuel Cells.

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**19 Mar 2021** I was in a terrible mood today. I was pretty tired and really not feeling it this morning before the run (POMS was 81). I thought that the run might make me feel better but it just made me angry. I'm writing this almost two hours after finishing because I was in too bad of a mood. I barely slept last night for whatever reason. I think I'm just tired and cranky. Crazy what mindset can do, I haven't experienced strong dyspnea since March 1<sup>st</sup>, and I felt it during intervals 3,4, & 5 in the last minute and a half-ish. It could be that it was a little colder today, but I've had 4 days since the first that were as cold or colder with no increase in dyspnea. I think my mental game was weak today.

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**20 Mar 2021** Today I was a little quick out of the gates. I was back running in the Vastus and I felt good. Unfortunately, I got a stitch in my right side at about the 4km mark that slowed me down a little bit. I think that made me a little tense and perhaps triggered my next issue, which was having to go to the washroom for the last 2ish km. Despite these issues, I still had a good run and I am very excited to sleep in tomorrow.

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**22 Mar 2021** I was cruising today. Very smooth, very easy. I was able to run in shorts and a tee shirt, clearly the winter running part of this experiment is over now. I am hoping for a cold flash in a week so that the 5km can be at approximately the same running conditions, but I am not optimistic. Nothing of note happened during the run except for a surprising number of snot rockets in the first two km.

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**23 Mar 2021** Today was good. I was surprised that I ended up averaging a 3:51 because I felt very slow in the first few km. I was very much lost in thought again during the run today. A mild dyspnea sensation today in the first kilometre as I was attempting to clear a semi-stuffy nose.

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**24 Mar 2021** I'm tired. No dyspnea during the intervals, just tired legs. Keeping the pace was easy but it was the distance that seemed to grind me down. It is very hard to stay motivated when

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	you are accelerating to a faster-than-3:45 pace on the 16 <sup>th</sup> km and 70 <sup>th</sup> minute of a run that was preceded by two back-to-back sub-40 10km's.
<b>25 Mar 2021</b>	Today I felt really good for the first 6km then I lost all motivation. I continued running but I found it difficult to keep the legs moving and I couldn't stop thinking about how much I didn't want to be running at that particular moment. My 9 <sup>th</sup> and 10 <sup>th</sup> km (3:58 & 3:59) were slower than the next slowest (km #7) by 7&8s. I haven't been sleeping well the last few nights and I think that it is starting to affect my mental game during the runs; it has certainly had a negative effect on my mood the last two days.
<b>26 Mar 2021</b>	<p>There was a weather advisory for North Bay for freezing rain, and they were right. Today started off slippery and it only got worse over the 75min. It was especially bad on the sidewalk on Chippewa street for whatever reason, but it was bad everywhere else too. The intervals were tough. The added slippage on every step really wore me down. The first four intervals were fine. I technically failed the fifth because I ran the km in a 3:40 but in the subsequent 1:20 I slowed to a 3:52 which is outside the <math>\pm 5s</math>. The 6<sup>th</sup> interval was like the 5<sup>th</sup>, 3:41 through the first km and then fell to a 3:57 pace. During the 6<sup>th</sup> interval however, I also experienced an intense sensation of dyspnea and accidentally took a breath from my mouth. It started just before I hit the km mark and lasted the rest of the duration (1:19) despite me reducing my pace. Not how I was hoping to finish my last interval workout but considering the slippery conditions I'm going to give myself a pass on that one.</p> <p>On a subjective, how are you feeling note: I am exhausted. I'm writing this at 1:00PM because I've been asleep since I finished the run at 10:30AM.</p>
<b>27 Mar 2021</b>	<p>I was cruising today. Yesterday really did a number on me so I basically slept/lounged all day. I woke up today feeling great and it definitely translated into my run. I think that I was likely also excited that today was the last training day. I'm done! Woohoo! I still must do all my testing (BHT, GXT, &amp; 5kmTT) which I am planning for Tuesday right now, but the bulk of the study is now complete. Prior to analysing any data, I am very happy with how the training program went. I am thankful that I was able to stay relatively healthy despite this rigorous program and I am excited to have a break, especially from the intervals which were really grinding me down. I was counting my blessings yesterday that I failed the first round of intervals, I don't know how it would have gone if I had to do 7x5min as was prescribed for the 6<sup>th</sup> week if I made it through all the intervals first try.</p> <p>Back to today's run, no dyspnea throughout the entire run despite my fastest time yet; 2s faster than the interval pace but I always run those fast.</p>
<b>30 Mar 2021</b> <b>BHT</b>	The breath holds went pretty well. I was a little unimpressed with the first one, I thought that maybe I could have done better. The other two I felt very good about. I focused on relaxing and putting my mind elsewhere.
<b>30 Mar 2021</b> <b>GXT</b>	I am a little upset that I didn't make it into the 3:10 round. I stopped right at the end of the 3:20. Honestly, I didn't even know. I knew it was coming up soon, but I was dealing with dyspnea (which seemed to include a weird feeling in my legs) and trying to keep the pace. It was building from about halfway through the last round and seemed to all crash down on me at once. I didn't find it as hard to breath as last time (no plugged ear or anything) but it was as if I had hit a wall and could go no further.

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**30 Mar 2021** That almost killed me. I absolutely blew out of the gates with a 3:00 first km and I was really just hoping the wheels weren't going to fall off before the finish. I managed to hold on but subjectively, that was by far the most painful run of the whole 9 weeks. Even more so than the other 5kmTT because I started hurting so much earlier. I was already starting the self-talk before the 2km mark. I had to consciously remind myself to lift my eyes in the last 2km. I had no confidence whatsoever that I was going to be faster than last time. I never check my km times when racing so I was flying by feel and I felt bad. I just wanted to put down my best effort so that I could at least walk away with a little pride. I guess it all worked out.

It was also very windy today, there was a weather advisory saying gusts up to 80km/hr. I definitely did not encounter anything that strong but there was quite a bit of wind today.

That's a wrap on the data collection.

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