Fakultet for helse- og sosialvitenskap

Tomas Urianstad

Masteroppgave

# Fem uker med varmetrening gir samme økning i hemoglobinmasse, enten man trener i et varmekammer eller med en varmedress hos elitesyklister

*Five weeks of heat training increases haemoglobin mass to the same extent, whether exercising in a climatic chamber or with a heat suit in elite cyclists*

Master i treningsfysiologi

2020

**Forord**

Jeg vil gjerne få rette en stor takk til professor Bent R. Rønnestad for alt ansvaret jeg fikk i forbindelse med planleggingen og gjennomføringen av hans prosjekt. Jeg vil også takke for god veiledning underveis i prosjektet og under skriveprosessen i etterkant.

En stor takk rettes også til Joar Hansen, Espen Honganvik Holen og Carsten Lundby for god hjelp i forbindelse med den fysiske testingen. Takk til bachelorstudentene på prosjektet for hjelp til fysisk testing og gjennomføring av varmeøkter: Ingvill Odden, Karoline Simpson-Larsen, Lars Nymoen, Dan Mikael Helgestad, Daniel Butcharoen Kristiansen, Jesper Tørrisen, Akramat Tsitskiev, Espen Iversen Korsvold, Sverke Baadsvik, Magnus Kleiven og Henrik Husdal.

Jeg vil også takke Håkonshall Sportssenter, Lillehammer Cycleklubb og NTG Lillehammer for lån av garderober til varmeøktene.

Til slutt vil jeg rette en stor takk til alle forsøkspersonene som deltok i studien. Dere gjorde den mulig å gjennomføre.

Tomas Urianstad
Lillehammer, mai 2020

**Innholdsfortegnelse**

**Sammendrag**

**Formål:** Utholdentstrening i varmen kan føre til hematologiske endringer og bedre prestasjon i normaltemperatur, men kan være upraktisk å gjennomføre. Denne studien undersøker derfor om den samme effekten på blodvariabler og utholdehetsprestasjon i normaltemperatur kan oppnås hos elitesyklister, dersom man gjennomfører fem uker med lavintensiv trening i et varmekammer eller med en varmedress.

**Metode:** 35 mannlige elitesyklister (snitt maksimalt oksygenopptak ($VO_{2maks}$) ved pretest = 76,9 ± 4,5 $mL \cdot min^{-1} \cdot kg^{-1}$) deltok i studien. Forsøkspersonene ble delt inn i en av tre grupper: 1) varmekammer (n = 12; vanlig trening + 26 ± 1 lavintensive sykkeløkter á 50 minutter i 35,1 ± 0,2 °C og 61,5 ± 2,2 % luftfuktighet), 2) varmedress (n = 11; vanlig trening + 26 ± 1 lavintensive sykkeløkter á 50 minutter med varmedress i 19.5 ± 2,7 °C og 37,2 ± 8,4 % luftfuktighet), eller 3) kontroll (n = 12; vanlig trening). Begge varmegruppene skulle ha en kjernetemperatur på > 38,5 °C på slutten av hver varmeøkt, noe som ble kontrollert minst en gang i uka. Hemoglobinmasse, maksimal effekt i beinpress og tester av utholdenhetsvariabler i normaltemperatur ble målt før (pre) og etter (post) intervensjonen.

**Resultat:** Hemoglobinmassen økte like mye i varmekammer- og varmedressgruppen (hhv. 2,4 ± 2,0 % og 2,6 ± 3,7 %), og økningen var forskjellig fra kontrollgruppen som ikke observerte noen endring (-0,7 ± 1,9 %; p = 0,006). Begge varmegruppene økte $VO_{2maks}$ (varmekammer: 3,0 ± 4,2 %; varmedress: 3,0 ± 4,5 %), men endringen var ikke forskjellig fra kontrollgruppen (1,0 ± 1,9 %, p = 0,365). Den maksimale aerobe effektutviklingen ($W_{maks}$) økte også i begge varmegruppene (varmekammer: 3,5 ± 3,8 %; varmedress: 7,6 ± 5,3 %), men det var kun endringen i varmedress som var forskjellig fra kontrollgruppen (1,6 ± 2,9 %; p = 0,001). Ingen andre signifikante prestasjon relaterte forskjeller ble observert.

**Konklusjon:** 5 uker med varmetrening gir den samme fremmende effekten på hemoglobinmassen sammenlignet med en kontrollgruppe, enten man trener i varmekammer eller med en varmedress. Om denne effekten forbedrer prestasjon i normaltemperatur er fortsatt usikkert.

## 1 Teori

Sykkelprestasjon avhenger av en rekke konkurransespesifikke egenskaper (Faria, Parker & Faria, 2005a, 2005b). Eksempler på dette er evnen til å effektivt «ligge på hjul», evnen til hyppige akselerasjoner, spurtegenskaper og taktiske ferdigheter (Faria et al., 2005a, 2005b). I tillegg til de konkurransespesifikke egenskapene er sykkelprestasjon i likhet med annen utholdenhetsprestasjon, i stor grad avhengig av størrelsen på det maksimale oksygenopptaket ($VO_{2maks}$), evnen til å utnytte en høy andel av $VO_{2maks}$ over en gitt periode (utnyttelsesgrad) og evnen til å effektivt omsette denne energien til ytre effekt (arbeidsøkonomi) (Bassett & Howley, 2000; Faria et al., 2005a). Teoridelen i denne oppgaven vil ta for seg nettopp disse fysiologiske faktorene, og i tillegg se på hvordan varmetrening kan påvirke utholdenhetsprestasjonen og hva som er gjort av funn i tidligere varmetreingsstudier.

### *1.1 $VO_{2maks}$*

I forskningslitteraturen er en økning i $VO_{2maks}$ den vanligste måten å demonstrere en mulig treningseffekt på (Helgerud et al., 2007; Lorenzo, Halliwill, Sawka & Minson, 2010; Sawka, Young, Cadarette, Levine & Pandolf, 1985). Dette er fordi $VO_{2maks}$ på mange måter setter en øvre grense for utholdenhetsprestasjoen (Bassett & Howley, 2000), og i tillegg er sterkt relatert til andre forhold som sykdom og dødelighet (Pedersen & Saltin, 2015). Mens typiske utrente menn i alderen 20-30 år har en $VO_{2maks}$ på rundt 40 til 45 $mL \cdot min^{-1} \cdot kg^{-1}$ (Tønnessen & Rønnestad, 2018), karakteriseres gjerne verdensklassesyklister med en $VO_{2maks}$ på mellom 75 og 90 $mL \cdot min^{-1} \cdot kg^{-1}$ (Jeukendrup, Craig & Hawley, 2000). Den høyeste enkeltverdien vi kjenner til er rapportert hos tidligere juniorverdensmester på tempo Oskar Svendsen, som over en 2,5 års periode økte sin $VO_{2maks}$ fra 74,6 til 96,7 $mL \cdot min^{-1} \cdot kg^{-1}$ (Rønnestad, Hansen, Stenslokken, Joyner & Lundby, 2019). Dette er imidlertid eksepsjonelle verdier som trolig har en genetisk forklaring, men vi kan likevel se for oss at en slik utvikling skyldes en endring i en eller flere av de sentrale faktorene lungene, blodet og hjertet, og/eller at det skyldes en perifer tilpasning i musklene (Bassett & Howley, 2000).

Overgangen fra lungene til blodet er den første hindringen oksygenet møter på dets vei inn i blodet, men likevel regnes ikke dette normalt sett som en begrensende faktor for $VO_{2maks}$ (Bassett & Howley, 2000; Powers, Lawler, Dempsey, Dodd & Landry, 1989). Årsaken til dette er at den arterielle oksygenmetningen i blodet sjeldent faller lavere enn ~ 95 % ved maksimalt arbeid, sammenlignet med ~ 98 % i hvile (Mortensen et al., 2005). Hos svært godt

utholdenhetstrente kan dette derimot bli en begrensende faktor, noe som har blitt illustrert ved blant annet å manipulere oksygenmetningen i blodet ved å puste inn oksygenberiket luft (26 % $O_2$) (Powers et al., 1989). Oksygenmetningen i blodet steg da fra 90-93 % til ~ 95 %, noe som også førte til en betydelig økning i $VO_{2maks}$ (~ 70 til ~ 75 $mL \cdot min^{-1} \cdot kg^{-1}$) (Powers et al., 1989). Dette skyldes trolig at trente utøvere har et høyre minuttvolum enn utrente, noe som fører til at blodet passerer lungekapillærene med en høyere hastighet og at diffusjonstiden i lungene dermed reduseres (Dempsey, Hanson & Henderson, 1984). Diffusjonskapasiteten og de strukturelle forholdene i lungene ser ikke ut til å kunne endres ved trening (McKenzie, 2012). En forbedring av $VO_{2maks}$ som det vi så hos Oskar Svendsen må derfor kunne forklares av andre sentrale og/eller perifere faktorer nedstrøms for lungene.

Tidligere studier har vist at skjelettmuskulaturens oksidative kapasitet overgår evnen til å levere oksygen til arbeidende muskulatur under sykling med begge bein (Boushel & Saltin, 2013; Mortensen et al., 2005). Dette viser at det ikke er skjelettmuskulaturen som er den viktigste begrensende faktoren for $VO_{2maks}$, men transportkapasiteten av oksygen fra lungene til musklene (Bassett & Howley, 2000; di Prampero, 2003; Levine, 2008). Transportkapasiteten av oksygen er et produkt av hjertets maksimale minuttvolum og blodets evne til å binde oksygen, og er estimert til å stå for så mye som 70-75 % av begrensningen nedstrøms for lungene (di Prampero, 2003).

Det maksimale minuttvolumet har blitt observert å være omtrent dobbelt så høyt hos eliteutøvere (~ 40 $L \cdot min^{-1}$) som hos utrente (~ 20 $L \cdot min^{-1}$) (Ekblom & Hermansen, 1968), og ser dermed ut til å være en viktig bestemmende faktor for $VO_{2maks}$ og utholdenhetsprestasjon. Den enorme forskjellen mellom eliteutøvere og utrente skyldes hjertets maksimale slagvolum, da den maksimale hjertefrekvensen ikke er høyere hos eliteutøvere (Lundby & Robach, 2015). Hjertets maksimale slagvolum kan påvirkes av strukturelle og funksjonelle endringer i hjertet (Skattebo et al., 2020), eller av en lavere perifer karmotstand (Klausen, Secher, Clausen, Hartling & Trap-Jensen, 1982). Likevel er trolig det totale blodvolumet den viktigste og mest påvirkelige enkeltfaktoren for det maksimale slagvolumet, da økt blodvolum øker den venøse tilbakestrømmingen og hjertets fylling gjennom Frank-Starling mekanismen (Convertino, Mack & Nadel, 1991; Kanstrup & Ekblom, 1982). Dette gjenspeiles blant annet i at svært godt utholdenhetstrente gjerne har 30-40 % mer blod i kroppen enn det utrente har (Heinicke et al., 2001). Noe som riktig nok er vert å merke seg, er at en økning i blodvolum som følge av økt plasmavolum uten en økning i volum av røde blodceller, ikke ser ut til å øke

$VO_{2maks}$ hos godt utholdenhetstrente personer (Keiser et al., 2015; Warburton, Gledhill & Quinney, 2000). Årsaken til dette er at blodets transportkapasitet som tidligere nevnt også er avhengig av blodets evne til å binde oksygen (di Prampero, 2003). Denne evnen er igjen bestemt av volumet av røde blodceller, eller mer presist mengden hemoglobin i de røde blodcellene.

Hvert gram hemoglobin kan binde 1,34 mL oksygen (Heinicke et al., 2001), og for transportkapasiteten til blodet og $VO_{2maks}$, vil det derfor være gunstig å øke mengden hemoglobin i blodet. Utrente øker typisk hemoglobinmassen med 3-6 % etter en periode med utholdenhetstrening (> 4 uker) (Bonne et al., 2014; Montero et al., 2015; Skattebo et al., 2020), og det er heller ikke uvanlig at eliteutøvere har ~ 40 % høyere hemoglobinmasse en utrente (Heinicke et al., 2001; Schmidt & Prommer, 2008). Det er med andre ord sannsynlig at det er en sameheng mellom utholdenhetstrening og økt hemoglobinmasse (Montero & Lundby, 2018). De underliggende årsakene og mekanismene er likevel ikke helt forstått, men nyrene spiller trolig en sentral rolle gjennom å regulere produksjonen og utskillelsen av hormonet erytropoeitin (EPO) (Dunn, Lo & Donnelly, 2007; Montero & Lundby, 2018). EPO har en sentral rolle i aktiveringen av benmargens produksjon av røde blodceller, og er sensitivt for endringer i arterielt oksygeninnhold (Montero & Lundby, 2019).

En av de mest fremtredende teoriene bak en treningsindusert økning i hemoglobinmasse, er bygget på at plasmavolumet hos utrente øker og stabiliser seg rundt 10 % høyere enn utgangspunktet etter ~ 2 uker med utholdenhetstrening, med en påfølgende økning i plasma EPO konsentrasjon ([EPO]) (Convertino, 2007; Montero et al., 2017). I henhold til teorien, skyldes dette et dropp i blodets arterielle oksygeninnhold og hematokrit-verdi (HCT), som følgelig sanses av oksygensensorer i nyrene som stimuler til økt produksjon og utskillelse av EPO i blodet (Montero & Lundby, 2018). Dette bidrar til å aktivere benmargens produksjon av røde blodceller og stabilisere HCT-verdien på rundt 45 %, som er den verdien man antar er ideell for oksygentransport (Donnelly, 2001; Dunn et al., 2007). Denne funksjonen i nyrene omtales gjerne som «krimeter» og viser seg gjennom den negative korrelasjonen som er observert mellom HCT og [EPO] i blodet (Montero et al., 2017; Oberholzer et al., 2019). I tillegg til teorien om at økt plasmavolum øker hemoglobinmassen, finnes det også andre teorier om hvordan dette skjer. Disse teoriene er at produksjonen av røde blodceller kan stimuleres direkte av et redusert sentral-venøst oksygentrykk som følge av dehydrering og vasodilatasjon av kapillærene etter trening, og av utskillelsen av plasmavolum-regulerende

hormoner som vasopressin og angiotensin II (Montero, Rauber, Goetze & Lundby, 2016). Produksjonen og frigjøringen av røde blodceller kan også påvirkes direkte gjennom økt utskillelse av veksthormoner, insulin-lignende veksthormoner, testosteron, katekolaminer og cortisol i blodet (Montero & Lundby, 2018).

Kroppens transportkapasitet av oksygen bestemmes altså av mange og komplekse, sentrale og perifere adaptasjoner, og bestemmer så mye som 70-75 % av $VO_{2maks}$ (di Prampero, 2003). Transportkapasiteten er likevel ikke det eneste som avgjør størrelsen på $VO_{2maks}$, og de siste 25-30 % bestemmes trolig av ekstraksjonen av oksygen fra blodet, som igjen bestemmes av en rekke andre adaptasjoner (di Prampero, 2003; Skattebo et al., 2020).

### *1.2 Utnyttingsgrad*

$VO_{2maks}$ setter som kjent en øvre grense for utholdenhetsprestasjonen (Bassett & Howley, 2000), men en belastning tilsvarende $VO_{2maks}$ kan normalt sett kun opprettholdes i 5-7 minutter (Mortensen et al., 2005). Utover denne varigheten er utholdenhetsprestasjonen i stor grad avhengig av utnyttingsgraden, som sier noe om hvor høy andel av $VO_{2maks}$ man klarer å utnytte seg av over en gitt periode (Coyle, 1995). Utnyttingsgraden estimeres gjerne gjennom en laktatprofil, og utrykkes som prosent av $VO_{2maks}$ ved blodlaktatterskel (Bassett & Howley, 2000). Avhengig av hvordan man definerer blodlaktatterskel og forskjeller mellom individer, så ligger denne prosentandelen gjerne på 75-80 % hos moderat trente (Helgerud et al., 2007; Støren, Ulevåg, Larsen, Støa & Helgerud, 2013) og 80-85 % hos eliteutøvere (Holen, 2019; Rønnestad, Hansen & Nygaard, 2017; Wilber, Zawadzki, Kearney, Shannon & Disalvo, 1997).

De store individuelle forskjellene i utnyttingsgrad til tross for lik $VO_{2maks}$, ser i stor grad ut til å være relatert til perifere tilpasninger av kapillærer, mitokondrier og laktattransportører (Coyle, 1999; Coyle, Coggan, Hopper & Walters, 1988; Holloszy & Coyle, 1984). Mengden aerobe enzymer og mitokondrier ser ut til være spesielt viktig (Ivy, Withers, Handel, Elger & Costill, 1980). Dette kan forklares med at når energiomsetningen øker, så representerer oksygenopptaket ved blodlaktatterskel den energiomsetningsraten hvor homeostasen blir så forstyrret at glykogenforbruket og laktatproduksjonen øker markant (Coyle, 1999). Dersom man øker mengden av aerobe enzymer og mitokondrier, vil man kunne arbeide på en høyere absolutt oksygenopptak før homeostasen blir så forstyrret at man når denne terskelen (Coyle,

1999). Likevel finner man ofte ikke noen endring i utnyttingsgraden etter korte teningsintervensjoner, til tross for store perifere tilpasninger (Coyle, 1999). En årsak til dette kan være at den oksidative kapasiteten ikke bare bestemmes av mengden aerobe enzymer og mitokondrier per muskelenhet, men også av hvor mye muskelmasse som er involvert i arbeidet (Coyle, 1999). Coyle (1995) estimerte at syklister med en høy blodlaktatterskel var kapable til å utnytte seg av ~ 22 % mer muskelmasse som følge av bedre tråkkteknikk og flere år med sykkelerfaring. En finsk studie som fulgte unge langrennsløpere over en 4 års periode, observerte også at utnyttingsgraden økte fra 73 til 78 % på disse årene (Rusko, 1987). Dette tyder på at en bedring i utnyttingsgraden er avhengig av systematisk trening over flere år.

### *1.3 Arbeidsøkonomi*

Mens $VO_{2maks}$ og utnyttingsgraden avgjør hvor høy energiomsetning vi klarer å opprettholde ved et arbeid av en viss varighet, avgjør arbeidsøkonomien hvor effektivt vi klarer å utnytte denne energien (Coyle, Sidossis, Horowitz & Beltz, 1992). Arbeidsøkonomi utrykkes gjerne som «gross efficiency» innenfor sykling, og forteller oss hvor mange prosent av kroppens totale energiomsetning som går med til å skape en ytre effekt (W) (Tønnessen & Rønnestad, 2018). Hos eliteutøvere ligger denne prosentandelen typisk mellom 19 og 23 % (Coyle et al., 1992), og ser ut til å kunne forbedres gjennom en hel idrettskarriere (Coyle, 2005; Jones, 2006). Lance Armstrong, som opprinnelig vant Tour de France 6 ganger (senere fratatt seierne på grunn av dopingmisbruk), økte for eksempel arbeidsøkonomien sin fra 21,18 til 23,05 % over en 7 års periode (Coyle, 2005). På grunn av avsløringene om Armstrongs omfattende dopingmisbruk skal vi riktig nok være forsiktige med å legge for mye i disse resultatene (Coyle, 2013), men lignende funn har også blitt observert hos andre verdensklassesyklister (Santalla, Naranjo Orellana & Terrados, 2009).

Årsakene til en forbedret arbeidsøkonomi kan dog være vanskelig å peke på, men en forbedring skyldes trolig en kombinasjon av mekaniske og metabolske komponenter (Mogensen, Bagger, Pedersen, Fernström & Sahlin, 2006; Saunders, Pyne, Telford & Hawley, 2004). En faktor som har fått mye oppmerksomhet er fibertypesammensetning, da type I-fibre ser ut til å være mer energieffektive en type II-fibre (Bottinelli & Reggiani, 2000), og at det er vist en sammenheng mellom andelen type I-fibre og arbeidsøkonomi (Coyle et al., 1992). Det er dog svært usikkert om fibertypeoverganger fra type II til type I kan forekomme hos mennesker (Tønnessen & Rønnestad, 2018). En overgang fra type IIx til mer energieffektive

type IIa er derimot mer sannsynlig (Bottinelli & Reggiani, 2000), og kan forekomme etter for eksempel en periode med styrketrening (Vikmoen et al., 2016).

Utholdenhetsprestasjon bestemmes altså i stor grad av $VO_{2maks}$, utnyttingsgrad og arbeidsøkonomi (Basset & Howley, 2000), og som vi har sett kan alle disse fysiologiske faktorene påvirkes ved trening. Det jaktes derfor hele tiden treningsmetoder som kan påvirke disse faktorene, og dermed også utholdenhetsprestasjonen, i størst mulig grad. En av disse metodene er trening i varme.

### *1.4 Varmetrening*

Varmeakklimatisering kan gjennomføres både i naturlige og kunstige omgivelser, og skyldes forstyrrelser som oppstår i kroppen som følge av omgivelsenes klima (f.eks. temperatur, luftfuktighet og solstråler), bekledning som forhindrer varmetap eller fysisk arbeid (f.eks. metabolsk varmeproduksjon) (McLellan, Daanen & Cheung, 2013). Dersom forholdene er identiske, enten det er i naturlige omgivelser, i et varmekammer eller om man klarer å skape de samme forholdene gjennom bekledning, så vil dette i teorien føre til de samme tilpasningene til varmen (Armstrong & Maresh, 1991). Det er dessuten også vist at tilpasninger til en type omgivelse i noen tilfeller kan føre til tilpasninger som er til fordel for en annen type omgivelse eller mindre forstyrende omgivelser (Faiss et al., 2015; Hauser et al., 2016; Rodriguez et al., 2015). Et godt eksempel på dette, er hvordan tilpasningene til høydetrening kan være med på å bedre utholdehetsprestasjon ved havnivå (Faiss et al., 2015; Hauser et al., 2016; Rodriguez et al., 2015). Det er derimot ikke viet like mye oppmerksomhet til hvordan tilpasningene til varme kan være med på å heve utholdenhetsprestasjonen i normal temperatur. Dette til tross for at varme er vist å gi tydeligere omgivelsesspesifikke tilpasninger enn høyde (Lee, Miller, James & Thake, 2016).

### *1.5 Tilpasninger til varme*

Som følge av omgivelsene, bekledning som forhindrer varmetap eller fysisk arbeid, fører varme til metabolske og kardiovaskulære forstyrelser som hemmer utholdenhetsprestasjon (McLellan et al., 2013; Nybo, Rasmussen & Sawka, 2014). I likhet med når utrente begynner med utholdenhetstrening (Convertino, 2007; Montero et al., 2017), ser plasmavolumet ut til å øke etter bare noen få dager med varmeeksponering (Senay, Mitchell & Wyndham, 1976). Dette er trolig en viktig mekanisme, og ser ut til å kunne bidra til å redusere kardiovaskulære

forstyrrelser i varmt klima ved å øke slagvolumet (Nielsen et al., 1993), redusere hjertefrekvensen ved gitte belastninger (Buchheit, Voss, Nybo, Mohr & Racinais, 2011) og dermed øke det maksimale minuttvolumet (Lorenzo et al., 2010). I tillegg til økt plasmavolum karakteriseres adaptasjonene til varme med en økt svetterate som følge av en redusert svetteterskel og økt svettesensitivitet, noe som til sammen senker kjernetemperaturen, som er en av de viktigste tilpasningene for bedre varmetoleranse (Garrett, Rehrer & Patterson, 2011; Periard, Travers, Racinais & Sawka, 2016). På et cellulært nivå bidrar også varmeresponderende proteiner («heat-shock proteins» (HSPs)) til bedre beskyttelse av cellene mot varmen, samt til et lavere glykogenforbruk som igjen gir en lavere produksjon av varme (Garrett et al., 2011; Periard et al., 2016).

Til sammen fører disse tilpasningene til forbedret temperaturregulering og varmetoleranse (Garrett et al., 2011; Periard et al., 2016), samt økt ytelse i varmen (Keiser et al., 2015; Lorenzo et al., 2010; Sawka et al., 1985). De siste årene har det oppstått en økende interesse for om disse omgivelsesspesifikke tilpasningene til varme også kan bidra til å heve prestasjonen i normal temperatur (Karlsen et al., 2015; Keiser et al., 2015; Mikkelsen et al., 2019; Oberholzer et al., 2019).

### *1.6 Studier som undersøker effekt av varmetrening i normaltemperatur*

Om effekten av varmetrening faktisk kan bidra til å heve prestasjonen i normal temperatur er fortsatt usikkert (Corbett, Neal, Lunt & Tipton, 2014; Minson & Cotter, 2016; Nybo & Lundby, 2016). Sawka et al. (1985) observere tidlig en mulig prestasjonsfremmende effekt hos 13 soldater som gjennomgikk 90 minutter lett trening i varmen (49 °C, 20 % luftfuktighet) over ni påfølgende dager. I tillegg til tydelige omgivelsespesifikke tilpasninger og bedre prestasjon i varmen, observerte de en 4 % økning i maksimal effekt og $VO_{2maks}$ under en progressiv sykkeltest i normal temperatur (21 °C, 30 % luftfuktighet). Denne studien hadde dog ingen kontrollgruppe å sammenligne med. Flere andre studier på svømmere (Hue, Antoine-Jonville & Sara, 2007), fotballspillere (Buchheit et al., 2011; Racinais et al., 2013), syklister, løpere og triatleter (Neal, Corbett, Massey & Tipton, 2016; Rebecca Neal, Massey, Tipton, Young & Corbett, 2016) har også observert prestasjonsrelaterte effekter i normal temperatur etter 5 til 14 dager med varmeeksponering, men heller ikke disse studiene sammenlignet med en kontrollgruppe.

Av studier på godt trente med en varighet på 10 til 21 dager som inkluderer en kontrollgruppe, er resultatene mer usikre (Karlsen et al., 2015; Keiser et al., 2015; Lorenzo et al., 2010; McCleave et al., 2017). Lorenzo et al. (2010) observerte en økning i $VO_{2maks}$, laktatterskel, og 1 time sykkelprestasjon etter 10 påfølgende dager med varmeeksponeringer, uten at det var noen endring i kontrollgruppen. Likevel var ikke økningen forskjellig fra kontrollgruppen. Hverken Keiser et al. (2015) eller Karlsen et al. (2015) observerte noen endring i prestasjon eller prestasjonsrelaterte utholdenhetsvariabler i normal temperatur etter henholdsvis 10 dager med repeterte varmeeksponeringer, og 14 dager på treningssamling i et naturlig varmt klima. Imidlertid observerte Karlsen et al. (2015) en tendens ($p = 0.054$) til økning i hemoglobinmasse sammenlignet med kontrollgruppen.

Hemoglobinmasse er som tidligere nevnt viktig for transportkapasiteten av oksygen, og en økning i hemoglobinmassen kan i teorien bidra til å øke $VO_{2maks}$ og prestasjonen i normaltemperatur. Den teoretiske forklaringen på at hemoglobinmassen kan øke som følge av varmetrening, er foreslått på grunn av økningen en har observert i plasmavolum (Holen, 2019; Oberholzer et al., 2019). Det økte plasmavolumet kan gjennom den tidligere presenterte teorien stimulere nyrenes utskillelse av EPO, og videre aktivere benmargens produksjon av røde blodceller og dermed øke hemoglobinmassen (Montero & Lundby, 2018). Det er også vært å merke seg at tidligere nevnte varmeresponderende proteiner (HSP) også kan bidra til denne økningen (Hawley, Lundby, Cotter & Burke, 2018).

Flere av de tidligere gjennomførte varmetreningsstudiene har observert en tydelig økning i plasmavolum uten at volumet av røde blodceller eller hemoglobinmassen har økt (Keiser et al., 2015; Lorenzo et al., 2010; McCleave et al., 2017). Dette kan skyldes at varigheten på intervensjonene har vært for kort. Så vidt vi vet er det kun to tidligere studier som har studert langtidseffekten (~ 5 uker) av varmetrening på hemoglobinmasse (Holen, 2019; Oberholzer et al., 2019). Oberholzer et al. (2019) gjennomførte sin studie på godt trente syklister ($VO_{2maks}$ = ~ 60 $mL \cdot min^{-1} \cdot kg^{-1}$), og observerte ~ 3 % økning i hemoglobinmasse som tenderte ($p = 0,061$) til å være forskjellig fra kontrollgruppen. Etter en lignende intervensjon gjennomført på elitesyklister ($VO_{2maks}$ = ~ 76 $mL \cdot min^{-1} \cdot kg^{-1}$), observerte Holen (2019) ~ 5 % økning i hemoglobinmasse, signifikat forskjellig fra kontrollgruppen der verdien var uendret. Til tross for økt hemoglobinmasse observerte ingen av studiene noen klare forbedringer i prestasjon i normaltemperatur (14-18 °C). Dog observerte Holen (2019) en moderat effektstørrelse i favør varmegruppa på både effekt ved 4 mmol·L laktatkonsentrasjon og snittwatt ved en 15

minutters prestasjonstest. I tillegg var laktatkonsentrasjonen ved en submaksimal belastning i trøtt tilstand redusert sammenlignet med kontrollgruppen. Disse funnene kan tyde på at elitesyklister muligens kan profitere av varmetrening.

### *1.7 Målsetting, problemstilling og hypotese*

Målsetting: Målet for denne studien er å undersøke om fem uker med lavintensiv varmetrening påvirker hemoglobinmassen og faktorer for utholdenhetsprestasjon i normaltemperatur hos elitesyklister. I tillegg ønsker vi undersøke om den samme effekten oppnås enten man trener i et varmekammer eller med bekledning som forhindrer varmetap.

Problemstilling: Fører fem uker med lavintensiv varmetrening i et varmekammer eller med en varmedress til økt hemoglobinmasse og dermed forbedret utholdenhetsprestasjon i normal temperatur hos elitesyklister?

Hypotese: Fem uker med varmetrening fører til den samme økningen i hemoglobinmasse og forbedring av prestasjon i normaltemperatur hos elitesyklister, enten man trener i et varmekammer eller med en varmedress.

## 2 INTRODUCTION

Heat acclimatization induces many physiological adaptations that improve endurance performance in hot environments (Keiser et al., 2015; Lorenzo et al., 2010). Several studies even suggest that some of these heat acclimating adaptations could improve performance in normal temperature (14-18°C) (Holen, 2019; Hue et al., 2007; Lorenzo et al., 2010; Scoon, Hopkins, Mayhew & Cotter, 2007).

The proposed ergogenic benefit in normal temperature is attributed to haematological, cardiovascular and skeletal muscle factors (Corbett et al., 2014). Indeed, adaptations obtained from heat stress could have a positive impact on maximum oxygen uptake ($VO_{2max}$) (Lorenzo et al., 2010; Sawka et al., 1985), which is one of the most important determinants of endurance performance (Bassett & Howley, 2000). The major factor limiting $VO_{2max}$ is the oxygen transport capacity, which is a product of maximal cardiac output and the oxygen transport coefficient for blood (di Prampero, 2003). When exposed to hot environments, the expansion of plasma volume (PV) is one of the first adaptations known to occur (Periard et al., 2016), and this could theoretically have a positive impact on the maximal cardiac output via the Frank-Starling mechanism (Coyle, Hopper & Coggan, 1990). However, an improvement in maximal cardiac output have little influence on $VO_{2max}$ if the PV is expanded without a simultaneously increase in volume of red blood cells (RBCV) (Warburton et al., 2000). Still, the heat induced PV expansion is of particular interest because it could translate into an increased RBCV and thereby increase the total mass of oxygen carrying haemoglobin ($Hb_{mass}$) (Dunn et al., 2007; Montero & Lundby, 2018). The underlaying rationale for this hypothesis, is that the kidney may be functioning as a "critmeter" by adjusting the PV and RBCV to keep the haematocrit (HCT) within a normal range (~ 45%) (Donnelly, 2001). RBCV is thought to be augmented by an enhanced erythropoietin (EPO) synthesis as a response to the kidneys sense of reduced HCT, and thus change in arterial oxygen content (Montero & Lundby, 2018). It is also noteworthy that heat shock proteins (HSPs) (Hawley et al., 2018), PV-regulating hormones and a reduction of cental venous pressure, may contribute to a potential increase in RBCV (Montero & Lundby, 2018; Montero et al., 2016).

Some studies have investigated the effect of 5 to 21 days of heat training on physiological adaptions and performance in normal temperature (Karlsen et al., 2015; Keiser et al., 2015; Lorenzo et al., 2010; McCleave et al., 2017; Scoon et al., 2007). None of these studies

observed any clear improvements of endurance-performance in normal temperature, and only Karlsen et al. (2015) observed a tendency ($p = 0.054$) to increased $Hb_{mass}$ compared to a control group. Since it may take several weeks to increase RBCV and $Hb_{mass}$ (Montero et al., 2017), it is likely that 5 to 21 days of heat training are insufficient to detect any enhancements in these factors, and to gain any performance benefits in normal temperature (Holen, 2019; Mikkelsen et al., 2019; Oberholzer et al., 2019).

To our knowledge, only two studies have investigated the long-term effect of heat training on haematological adaptations and performance in normal temperature (Holen, 2019; Oberholzer et al., 2019). Both Oberholzer et al. (2019) and Holen (2019) observed an increased $Hb_{mass}$ after ~ 5 weeks of repeated heat exercise exposures, which respectively tended to be ($p = 0.054$) and was significantly different from a control group. Interestingly, both of the later studies observed a correlation between increased PV and increased $Hb_{mass}$, which may support the idea of PV playing a decisive role in the enhancement of $Hb_{mass}$. Furthermore, the trained cyclists ($VO_{2max}$ = ~ 60 $mL \cdot min^{-1} \cdot kg^{-1}$) in the accompanying paper of Oberholzer et al. (2019), did not improve performance in normale temprature compared to a control group (Mikkelsen et al., 2019). The similarly study by Holen (2019) stands out from other studies with cyclists at an exceptional performance level ($VO_{2maks}$ = ~ 76 $mL \cdot min^{-1} \cdot kg^{-1}$), and highlight the findings of reduced blood lactate concentration ($[La^-]$) towards the end of a prolonged test battery. There were also some interesting, but still non-significant effects favouring endurance heat training in lactate threshold power output, gross efficiency in fatigued state and mean power output in a 15 minutes time trail (TT), which potentially could be of practical importance for elite cyclists.

For that reason, we aim to get further insights in whether elite endurance athletes can increase $Hb_{mass}$ and improve endurance performance in normal temperature through heat training. We examined whether 5 weeks of heat training in a climatic heat chamber would lead to superior adaptions of these kinds compared to a control group in elite cyclists. Because training in a natural hot environment or in a climactic heat chamber can be unpractical and expensive, we added a third group to the study, who trained in a heat specific suit that limited heat loss. We hypothesized that five weeks of heat training would induce the same superior effects on $Hb_{mass}$ and improve endurance performance in normal temperature, whether exercising in a climatic chamber or with a heat suit, in elite cyclists.

## 3 MATERIALS AND METHODS

The present paper is part of a large study including a total of 56 elite cyclists. The main goal for the whole study is to search for further understandings of the best practise to prepare for specific competitions in hot environments, and to explore the effect of prolonged heat training on performance in normal temperature and the underlying haematological adaptations.

### *3.1 Participants*

Thirty-eight male cyclists, aged 20.4 ± 5.0 years, were initially recruited to participate in this part of the study. The cyclists were allocated to their group based on $VO_{2max}$, age and general fitness level in discussion with their coaches: a heat chamber group (Chamber, n = 13), a heat suit group (Suit, n = 12) and a control group (Control, n =13). Due to personal reasons unrelated to the study intervention, three participants withdraw from the study. One participant in Control completed only the blood analysis, due to health reasons unrelated to the study intervention. Because of this, the blood data are presented from 35 participants while all other data are presented from 34 participants. All participants who completed both the pre and post testing had undertaken their usual offseason training prior to the project (cycling or running 10:42 ± 04:08 h/week recorded during the 4 weeks preceding pre-testing), and had a history of 5.0 ± 2.5 years of competitive cycling. All cyclists were categorized as performance level 4 to 5 according to De Pauw et al. (2013), equal to elite (Jeukendrup et al., 2000). Subject characteristics and physiological parameters are presented in Table 1.

**Table 1:** Subject characteristics and physiological parameters determined during an incremental maximal exercise test, for heat chamber (Chamber), heat suit (Suit) and the control group (Control).

| | **Chamber (n = 12)** | **Suit (n = 11)** | **Control (n = 12)** |
|---|---|---|---|
| Age (years) | 20.2 ± 4.0 | 22.2 ± 7.2 | 18.8 ± 2.5 |
| Body mass (kg) | 68.6 ± 7.6 | 68.3 ± 5.7 | 73.8 ± 7.1 |
| Body height (cm) | 178.7 ± 5.7$^{£}$ | 179.5 ± 6.1 | 184.4 ± 5.3 |
| $VO_{2max}$ ($mL \cdot min^{-1}$) | 5311 ± 561 | 5293 ± 353 | 5578 ± 557 |
| $VO_{2max}$ ($mL \cdot min^{-1} \cdot kg^{-1}$) | 77.5 ± 4.3 | 77.8 ± 5.7 | 75.4 ± 3.2 |
| $W_{max}$ (W) | 440 ± 44 | 437 ± 29 | 469 ± 52 |
| $W_{max}$ ($W \cdot kg^{-1}$) | 6.4 ± 0.4 | 6.4 ± 0.6 | 6.4 ± 0.3 |

$VO_{2max}$: maximal oxygen consumption; $W_{max}$: average power output the last minute during the incremental maximal test. Values are mean ± SD. $^{£}$Tendency ($p < 0.100$) to differ from Control.

Before the start of the intervention, all participants were informed of any potential risk and discomfort associated with the study, and they all gave their written informed consent to participate (Appendix A). Two weeks before the start of the intervention, all cyclists were provided a daily 100 mg oral iron supplement (Nycoplus Ferro-Retard 100 mg, Takeda AS, Asker, Norway) to consume throughout the study to ensure adequate iron levels. The study was performed accordingly to the ethical standards established by the Helsinki Declaration of 1975, and were approved by the local ethical committee at Inland Norway University of Applied Sciences, and Data Protection Authority.

### *3.2 Experimental design*

An overview of the present project is presented in figure 1. All performance and blood measurements were conducted at the physiological test laboratory at Inland Norway University of Applied Sciences. Haematological data was collected in duplicates over two following days. All physiological and performance data was collected in one day and started with a leg press test to detect any potential change in power generation. This test was directly followed by an approximately two-hour long laboratory-based cycling test. The test included an incremental test to measure gross efficiency (GE) and [$La^-$] in fresh state and power output at a 4 mmol·$L^{-1}$ [$La^-$], an incremental test until exhaustion to determine $VO_{2max}$ and average power output the last minute ($W_{max}$), an repetition of the 3$^{rd}$ and 2$^{nd}$ last step from the blood lactate profile test to measure GE and [$La^-$] in fatigued state, and a 15 minutes TT to measure mean power output (figure 2). Participants were tested before and after five weeks of regular training, which for the two heat groups included 28 heat exposures (26 session + 2 heat tests, figure 1). Participants were instructed to maintain their usual training throughout the intervention but subtract the eventual extra training hours in order to not increase the total training load. This resulted in comparable training volumes between all groups (Table 2).

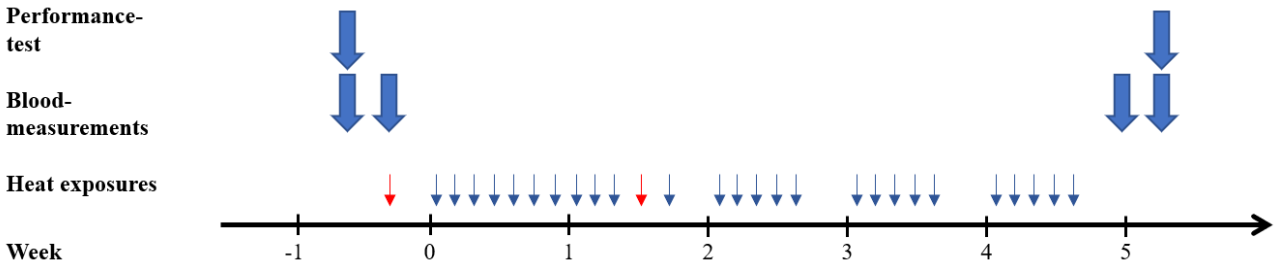


**Figure 1:** Overview and time course of the present project including performance testes, blood measurements and number of heat exposures for the two heat groups. Red arrows indicate a performance test in the heat due to another part of the study.

### *3.3 Self-reported training*

Besides the heat sessions, all subject conducted their usual training and reported it in a personal training diary (Appendix B & Table 2). Endurance training was reported accordingly to Dr. Andrew Coggan's five-zone intensity scale, based on percentage of functional threshold power (FTP) or heart rate (HR) associated with percentage of FTP (HR@FTP): zone 1 (< 55% FTP / < 68% HR@FTP), zone 2 (56-75% FTP / 69-83% HR@FTP), zone 3 (76-90% FTP / < 84-94% HR@FTP), zone 4 (91-105% FTP / 95-105% HR@FTP) and zone 5 (106-120% FTP / > 106% HR@FTP). All heat sessions and the majority (89 ± 8%) of total endurance training were reported in respect of power output, exceptions are exercise performed as running (7 ± 7%) or other forms of movement performed off the bike (3 ± 6%). Participants also reported their eventual number and duration of maximal (> 80% of 1 repetition maximum) or other type (< 80% of 1 repetition maximum) of strength training. In order to quantify how the intervention affected perceived feeling of well-being, the cyclists used a 9-point scale to report this: 1 (very, very good), 2 (very good), 3 (good), 4 (somewhat good), 5 (normal), 6 (somewhat bad), 7 (bad), 8 (very bad) or 9 (very, very bad) (B. R. Rønnestad, Hansen & Ellefsen, 2014).

**Table 2:** Average weekly distribution of training in different intensity zones, strength training, total training volume, number of maximal strength sessions and perceived feeling of well-being during the intervention period for heat chamber (Chamber), heat suit (Suit) and the control group (Control).

| | **Chamber** | **Suit** | **Control** | **p** |
|---|---|---|---|---|
| Zone 1 (h:m) | 08:49 ± 03:23 | 07:54 ± 03:28 | 09:07 ± 01:35 | 0.609 |
| Zone 2 (h:m) | 01:35 ± 00:56 | 01:51 ± 01:40 | 01:03 ± 00:44 | 0.289 |
| Zone 3 (h:m) | 01:14 ± 00:47 | 01:07 ± 00:44 | 01:00 ± 00:35 | 0.725 |
| Zone 4 (h:m) | 00:43 ± 00:36 | 00:51 ± 00:24 | 00:40 ± 00:18 | 0.604 |
| Zone 5 (h:m) | 00:22 ± 00:24 | 00:13 ± 00:10 | 00:18 ± 00:13 | 0.416 |
| Strength (h:m) | 01:17 ± 01:21 | 00:45 ± 00:56 | 01:34 ± 00:31 | 0.170 |
| Total training volume (h:m) | 14:01 ± 04:39 | 12:41 ± 03:05 | 13:40 ± 02:20 | 0.653 |
| Maximal strength sessions (n) | 0.78 ± 0.91 | 0.71 ± 1.01 | 0.69 ± 0.71 | 0.980 |
| Perceived well-being (1-9) | 4.62 ± 0.49 | 4.65 ± 0.53 | 4.34 ± 0.79 | 0.170 |

Values are mean ± SD. There were no differences ($p > 0.050$) between the groups in the parameters above.

### *3.4 Heat sessions*

Both Chamber and Suit performed a total of 26 ± 1 heat sessions over the 5 weeks intervention period, excluded two performance tests in a climatic chamber due to another part of the study (data from these tests are not included in this thesis). All sessions had a total duration of 50 minutes and was performed using the participants personal bikes connected to a stationary trainer device (Tacx Neo Smart T2800, Wassenaar, Netherlands or Computrainer, Racermate, Seattle, USA). The Chamber group performed all heat sessions in a climatic chamber pre-heated to 35.1 ± 0.2°C and 61.5 ± 2.2% relative humidity (RH), with a power output correspondently to ~ 45% of the power output at 4 $mmol \cdot L^{-1}$ $[La^-]$ during the whole session. The Suit group performed all heat sessions in normal conditions (19.5 ± 2.7°C, 37.2 ± 8.4% RH), but with a heat suit that limited heat loss. This suite consisted of wool on both the upper and lower body, a hat, a rain suit and a down jacket, and was designed to induce similar heat stress responses as in the climatic chamber. Based on pilot-tests prior to the project, power output during these sessions was set correspondently to ~ 55% of the power output at 4 $mmol \cdot L^{-1}$ $[La^-]$ for the initial 10 minutes, followed by 40 minutes at ~ 50% to induce similar heat stress responses as in Chamber. All the Chamber and Suit sessions were supervised throughout the study, but due to practical reasons, an exception was made for three participants in the Suit group. Their sessions were supervised for the initial 10 days, and after this they were allowed to perform the rest of the sessions at home. During all heat sessions, participants were instructed to drink exactly 500 mL of water. Sweat rate was calculated by measuring the change in nude bodyweight (Seca 813, Seca gmbh & co, Hamburg, Tyskland) after the instruction to dry of as much sweat as possible before and after each session, subtracted by 500 mL of water. To measure the metabolic strain a blood sample was taken from a fingertip two minutes after the session and analysed for blood $[La^-]$ using a Biosen C-line lactate analyzer (EKF Diagnostic GmbH, Barlebe, Germany) (Table 3). During all heat sessions room temperature, RH, HR (measured with personal HR monitors), rate of perceived exertion (RPE) using Borg's 6-20 scale (Borg, 1982), temperature feeling 1-8 (Toner, Drolet & Pandolf, 1986), and power output were measured after 5, 10, 15, 20, 30, 40 and 50 minutes (Table 3). The power output and eventually clothing for Suit was continuously adjusted to keep rectal temperature > 38.5°C (Teknikproffset Nordic AS, Härryda, Sweden) at the end of each session. The power output during the sessions was increased by 25 Watts (W) to the next session if end RPE < 11 or reduced by 20 W if end RPE > 15. Ten minutes after each session the participants rated their session feeling 1-10 (sRPE, Table 3) (Foster et al., 2001).

**Table 3:** Session data, presented as average data collected during and after each session. Temperature, relative humidity, heart rate, RPE, temperature feeling, and power output are presented as average during each session, while rectal temperature, sweat rate and sRPE are single values collected after each session, for heat chamber (Chamber) and heat suit (Suit).

| | **Session 1-5** | | **Session 6-10** | | **Session 11-16** | | **Session 17-21** | | **Session 22-26** | |
|---|---|---|---|---|---|---|---|---|---|---|
| | **Chamber** | **Suit** | **Chamber** | **Suit** | **Chamber** | **Suit** | **Chamber** | **Suit** | **Chamber** | **Suit** |
| Temperature (°C) | 35.2 ± 0.0 | 17.6 ± 0.1$^{\#}$ | 35.1 ± 0.1 | 18.2 ± 0.3$^{\#}$ | 35.1 ± 0.1 | 20.7 ± 1.1$^{*\#}$ | 35.2 ± 0.1 | 21.1 ± 1.3$^{*\#}$ | 35.1 ± 0.1 | 21.9 ± 0.7$^{*\#}$ |
| Relative humidity (%) | 63 ± 1 | 39 ± 0$^{\#}$ | 62 ± 1 | 32 ± 2$^{*\#}$ | 62 ± 2 | 34 ± 3$^{*\#}$ | 60 ± 1$^{*}$ | 43 ± 3$^{*\#}$ | 60 ± 1$^{*}$ | 39 ± 2$^{\#}$ |
| Rectal temp (°C) | 39.1 ± 0.3 | 38.5 ± 0.3$^{\#}$ | 38.9 ± 0.3 | 38.4 ± 0.2$^{\#}$ | 38.9 ± 0.2 | 38.6 ± 0.1 | 38.8 ± 0.3$^{*}$ | 38.6 ± 0.2 | 38.9 ± 0.3 | 38.6 ± 0.1 |
| Sweat rate (L) | -1.4 ± 0.4 | -1.4 ± 0.3 | -1.5 ± 0.3 | -1.4 ± 0.2 | -1.6 ± 0.4 | -1.7 ± 0.2 | -1.7 ± 0.4 | -1.8 ± 0.3 | -1.7 ± 0.3 | -1.7 ± 0.3 |
| Heart rate (beats·min$^{-1}$) | 145 ± 10 | 142 ± 8 | 139 ± 10 | 140 ± 7 | 139 ± 9 | 142 ± 6 | 137 ± 9$^{¶}$ | 142 ± 8 | 136 ± 9 | 141 ± 5 |
| RPE (6-20) | 12.4 ± 1.1 | 12.3 ± 0.9 | 11.5 ± 1.4 | 11.7 ± 0.5 | 11.3 ± 1.1 | 12.1 ± 0.8 | 11.2 ± 1.1 | 11.9 ± 0.9 | 11.1 ± 1.2 | 11.6 ± 0.7 |
| Temperature feeling (1-8) | 6.1 ± 0.5 | 6.1 ± 0.4 | 5.7 ± 0.6 | 5.9 ± 0.3 | 5.5 ± 0.6 | 6.0 ± 0.3 | 5.4 ± 0.6$^{*}$ | 6.0 ± 0.4 | 5.3 ± 0.6$^{*}$ | 5.9 ± 0.3 |
| sRPE (1-10) | 3.8 ± 0.9 | 3.4 ± 0.9 | 3.2 ± 0.6 | 2.9 ± 0.7 | 3.1 ± 0.6 | 3.2 ± 0.8 | 2.8 ± 0.5$^{*}$ | 3.1 ± 1.0) | 3.0 ± 0.6 | 2.9 ± 0.7 |
| Power output (W) | 134 ± 16 | 154 ± 8$^{£}$ | 135 ± 16 | 161 ± 9$^{\#}$ | 133 ± 19 | 163 ± 7$^{\#}$ | 134 ± 17 | 157 ± 12$^{\#}$ | 135 ± 16 | 156 ± 13$^{\#}$ |
| Blood lactate (mmol·L$^{-1}$ ) | 0.8 ± 0.2 | 0.9 ± 0.1 | 0.9 ± 0.1 | 0.7 ± 0.1 | 0.7 ± 0.2 | 0.9 ± 0.3 | 0.8 ± 0.1 | 0.6 ± 0.1 | 0.7 ± 0.1 | 0.7 ± 0.3 |

RPE: Rate of Perceived Exertion, sRPE: Session Rate of Perceived Exertion. Values are mean ± SD. *Different ($p < 0.050$) from session 1-5, ¶Tendency ($p < 0.100$) to differ from session 1-5, #Different ($p < 0.050$) from Chamber at the current time point, £Tendency ($p < 0.100$) to differ from Chamber at the current time point.

### *3.5 Testing procedures*

The testing procedures at pre-test was completed as followed: day 1, blood measurements; day 2, blood measurements, leg press and endurance performance tests. The testing procedure was repeated at post-test, but in the opposite order (figure 1). During the first visit to the laboratory, all cyclists reported their three last meals, fluid intake, eventual caffeine intake or other notably products consumed. They also reported their training conducted during the 48 hours presiding the test. 3-4 days before post-test a message was sent to each of the cyclists with the instructions to repeat this to ensure comparable preparations. Nutritional energy intake during the entire test at pre was also noted and replicated at post-test. Endurance performance in normal temperature (17-20°C) was evaluated in laboratory settings and were performed on the same electromagnetic braked cycle ergometer, measuring power output at 6 Hz (Lode Excalibur Sport, Groningen, The Netherlands). Every cyclist had the same test leader at both pre and post-test, and strong encouragement was given during all tests to ensure maximal effort. All tests were individually performed at the same time of day (± 2 hours) at both pre- and post-test to avoid influence from the circadian rhythm. The test protocol was developed by the leader researcher to evaluate potential important changes in haematological, leg strength and endurance performance factors (figure 2).

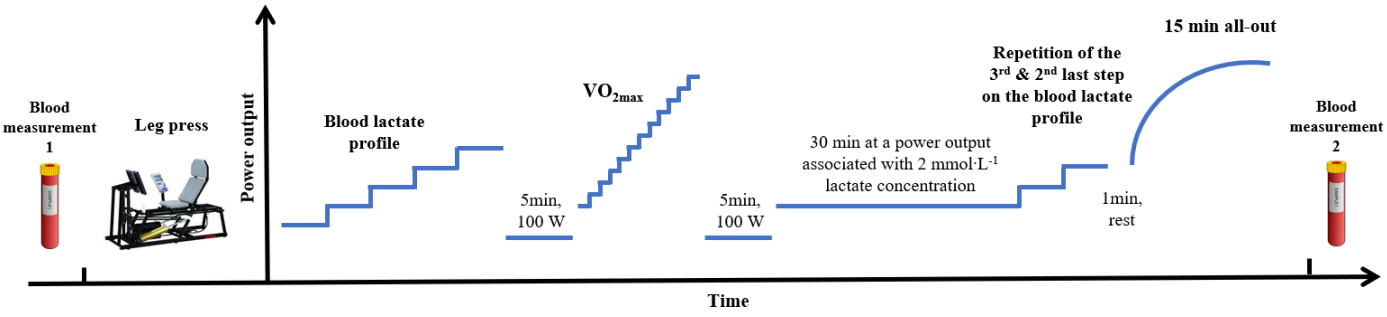


**Figure 2:** Overview of the testing procedures. Blood measurement 1: measurement of haematocrit; Blood measurement 2: measurement of haemoglobin mass, haemoglobin concentration, red blood cell volume, blood volume and plasma volume. Bold text indicate data included in the analyzes in the present paper.

### *3.6 Blood analysis: haematocrit, haemoglobin mass & intravascular volumes*

Haematological data are presented as the mean of two samples collected repeatedly over two different occasions at both pre- and post-test (Figure 1). The procedure started with the participant drinking 300 mL of water and resting in a supine position for 15 minutes. Afterwards, HCT was analysed with the microhematocrit method (Mondal & Budh, 2020). Briefly, blood from the fingertip was collected in three 75 mm capillary tubes and centrifuged in a microcentrifuge (Heraeus PICO 17 Hematocrit Rotor, Thermo Electron LED GmbH, Osterode, Germany) at 13500 rpm for 4 minutes, and was further analysed manually on a reader board. Data presented is the mean of these three samples. If these analyses were performed at the same day as other performance tests, the rest of the protocol to measure $Hb_{mass}$ was completed after these tests. Total $Hb_{mass}$ was measured by the carbon monoxide (CO) rebreathing technique (Siebenmann, Keiser, Robach & Lundby, 2017), starting with the collection of three blood samples taken from the fingertip and analysed for percent carboxyhaemoglobin (%HbCO) and haemoglobin concentration (ABL830 FLEX CO-OX analyzer, Radiometer, Copenhagen, Danmark). Subsequently participants breathed through a closed system (CO-Applicator, WGT Electronic CmbH & Co KG, Kolsass, Austria) for 1 minute, before a dose of 1.5 $mL \cdot kg^{-1}$ body weight of 99.997% chemically pure CO (Carbon monoxide 100%, AGA, Oslo, Norway) was administrated through the system. Participants rebreathed the $O_2CO$ mixture for 6 minutes. Remaining CO volume in the rebreathing circuit was determined by a total exhalation of the lungs (Dräger Pac 5500, Dräger INC., Houston, USA). Finally, the participants continued to lay still for 4 more minutes and breathed normally before three more blood samples was taken from the fingertip and analysed for %HbCO. Total $Hb_{mass}$ was calculated by the absorbed CO dose and change in %HbCO from before and after the rebreathing protocol. Intravascular volumes were addressed from an equation formula to calculate RBCV, PV and BV driven from the measurement of total $Hb_{mass}$, haemoglobin concentration and HCT (Burge & Skinner, 1995).

### *3.7 Leg press: peak power*

The leg press-test was used to evaluate any potential change in leg power generation, that potentially could influence the endurance performance (Rønnestad, Hansen, Hollan & Ellefsen, 2015). Prior to the leg press-test, all cyclists performed a standardized 7 minutes warmup protocol on a cycle ergometer, starting with 2 minutes at an intensity correspondently to 11 RPE, followed by 2 minutes at 13, 1 minute at 15 and 2 minutes at 12. The leg press-test procedure included an incremental pre-programmed ten-repetition test in a Keiser apparatus (Keiser AIR300 Leg Press, Keiser corporation, Fresno, USA). The test protocol is developed to reach a self-selected target resistant near maximum at the $10^{th}$ repetition, but the test will continue until failure. Resistance achieved at the $10^{th}$ repetition was set accordingly to the participants body weight, 250 kg if the participant was < 75 kg or 280 kg if the participant was > 75 kg. Resistance increments in-between each repetition was calculated by the target at the $10^{th}$ repetition (Rep to rep Resistance Increase = (selected $10^{th}$-rep target - 18.14)/10), and the start resistance was calculated by the rep to rep Resistance Increase (Starting resistance = Resistance Increase + 18.14, Table 4). $P_{max}$ was calculated as the point where the product of velocity and force (force x velocity) was at the highest point during the test. Target resistant and seat position with approximately 90° knee angle was the same at both pre and post-test.

**Table 4:** Keiser 10 repetition maximal power test protocol.

| **Repetition Number** | **Warm Up** | **Warm Up** | **1st** | **2nd** | **3rd** | **4th** | **5th** | **6th** | **7th** | **8th** | **9th** | **10th** | **N+1** |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Resistance (kg) if < 75 kg | 41 | 41 | 41 | 64 | 87 | 110 | 133 | 157 | 180 | 203 | 226 | 250 | + |
| Resistance (kg) if >75 kg | 44 | 44 | 44 | 71 | 97 | 123 | 149 | 175 | 201 | 228 | 254 | 280 | ++ |
| Rest period (s) | 3.0 | 3.0 | 3.0 | 4.2 | 5.8 | 8.1 | 11.4 | 15.8 | 22.1 | 30.8 | 43.0 | 60.0 | 60.0 |

+ Previous repetition +23.2. ++ Previous repetition +26.2.

### *3.8 Endurance performance: blood lactate profile, maximal oxygen consumption & 15 minutes mean power output.*

The participants first conducted a blood lactate profile test starting with 5 minutes cycling at 125 W (175 W if lactate threshold > 325 W), followed by 50 W increases every 5$^{th}$ minute. Blood samples were taken from a fingertip at the end of each 5 minutes bout and analysed for [$La^-$]. When reaching 2 mmol·$L^{-1}$ [$La^-$] the lactate profile continued to increase by 25 W, and the test was terminated when a [$La^-$] of 4 mmol·$L^{-1}$ or higher was measured. Oxygen consumption and respiratory exchange ratio (RER) were measured from the 2.5$^{th}$ to 4.5$^{th}$ minute in each bout with 30 seconds sampling time, using a computerized metabolic system with mixing chamber (Oxycon Pro; Erich Jaeger, Hoechberg, Germany). The metabolic system was calibrated before each test with the exact same routines, including temperature and ambient calibration. The gas analysers were calibrated with a certified calibration gas with known concentrations, and the flow turbine (Triple V, Erich Jaeger, Hoechberg, Germany) was calibrated with a 3 L, 5530 series, calibration syringe (Hans Rudolph, Kansas City, USA). Power output and fractional utilization of $\dot{V}O_{2max}$ at 4 mmol·$L^{-1}$ [$La^-$] was calculated from the lactate profile test. So was the metabolic strain in fresh state, measured as GE and [$La^-$] at the 3$^{rd}$ last bout of the blood lactate profile. GE was calculated as followed: $GE = \text{Oxygen}\ L \cdot s^{-1} \cdot (4.840\ J \cdot L^{-1} \cdot RER + 16.890\ J \cdot L^{-1}) \cdot W^{-1} \cdot 100$.

After the completion of the blood lactate profile, all cyclists had 5 minutes of recovery cycling at 100 W, before an incremental test to determine $VO_{2max}$ and $W_{max}$. The incremental test started at 200 W (250 W if lactate threshold > 325 W) and was subsequently increased by 25 W every minute until exhaustion, defined as cadence below 60 revolutions per minute. Oxygen consumption was measured every 30 seconds during the whole test. $VO_{2max}$ is presented as the mean of the two highest consecutive 30 seconds oxygen measurements. $W_{max}$ is presented as the average power output during the last 60 seconds of the incremental test. After the $VO_{2max}$-test, all cyclists had 5 minutes of recovery at 100 W where they could go to the toilet if necessary.

The recovery was followed by 30 minutes on a power output associated with 2 mmol·$L^{-1}$ [$La^-$], and repetition of the 3$^{rd}$ & 2$^{nd}$ last bouts on the blood lactate profile at pre-test. During the repetition of the 3$^{rd}$ last bout, the metabolic strain (i.e GE and [$La^-$]) in fatigued state was measured with the exact same procedure as in fresh state. The power output during this period was replicated at post-test regardless of any changes in physiological condition.

After this, the cyclists had 1 minute of completely rest before the 15 minutes TT-test. All cyclists were instructed to aim for the highest possible mean power output during the 15 minutes, and to stay seated during the entire test. The cadence was freely chosen, and the cyclists could adjust the power output at any time during the TT via an external control unit placed next to the handlebar of the ergometer setup. Performance during this test was measured as the average power output during the 15 minutes.

### *3.9 Statistic*

All descriptive data are presented as mean and standard deviation (mean ± SD) unless otherwise is stated. To detect any possible baseline differences at pre-test or differences in training load during the intervention, the data was fitted as the depended variable in a one-way ANOVA analysis with group as explanatory variable. For repeated measurements, a two-way repeated measurements ANOVA was fitted with time and group as the explanatory variable. The main effect of time between groups (group x time interaction) from pre to post was tested by calculating percent change from pre- to post-intervention fitted as the depended variable in a two-way ANOVA analysis with group as explanatory variable. If a significant global effect was indicated in the ANOVA analysis, a Tukey's post hoc test was performed to identify the significant differences between the groups. Pre- and post-intervention measurements within each group were compared using a two-tailed paired Students t-test. All results were considered statistically significant if $p < 0.050$, while p-values between 0.050 and 0.100 is described as a tendency. Because of the relatively small sample size and the fact that elite athletes are not expected to have huge changes in haematological data or physical performance in such short interventions, the practical differences of changes between the groups was interpreted by Cohen's d effect size (ES). ES was calculated by individually taking the mean pre-post change in one group minus the mean pre-post change in another group, divided by the pooled pre-test standard deviation approach between the two groups of interest (Morris, 2008). The scale proposed by Rhea (2004) for highly trained subjects was used to interpret the magnitude of the treatment effect: 0.0-0.24 trivial, 0.25-0.49 small, 0.5-1.0 moderate and >1.0 large. Pearson's r was used to calculate correlation coefficients. The magnitude of the correlation coefficients were defined as: $r < 0.1$ trivial, 0.1-0.3 small, 0.3-0.5 moderate, 0.5-0.7 large, 0.7-0.9 very large, 0.9 almost perfect, and 1.0 = perfect (Hopkins, Marshall, Batterham & Hanin, 2009). All data-analysis was done in R (R Core Team, 2018).

## 4 RESULTS

### *4.1 Baseline & peak power in leg press*

At baseline HCT differed between Chamber and Control (respectively 42.19 ± 2.33 and 44.71 ± 2.38%, p = 0.034; Table 5), RBCV differed between Suit and Control (respectively 2.6 ± 0.2 and 3.0 ± 0.4L, p = 0.045) and $Hb_{mass}$ tended to differ in both Chamber (909 ± 113g, p = 0.097) and Suit (901 ± 80g, p = 0.076) compared to Control (1010 ± 1042g; Figure 3). No other haematological, physiological or performance-related measurements differed between any of the three groups before the intervention period (Figure 3 & 4, Table 5). Peak power in leg press did not change during the intervention between (p = 0.690) or within any of the three groups (Chamber: -0.2 ± 5.0%, p = 0.857; Suit: -2.5 ± 5.3%, p = 0.126; Control: -0.3 ± 9.8%, p = 0.749).

### *4.2 Haemoglobin mass*

After five weeks of prolonged heat training there was a significant difference between the three groups in respect of percent change in total $Hb_{mass}$ (p = 0.006). Post hoc comparisons indicated a significant greater increase after the Chamber (p = 0.016) and Suit (p = 0.011) interventions compared to Control. Within groups the total $Hb_{mass}$ increased to the same extent in both Chamber (2.4 ± 2.0%, p = 0.003) and Suit (2.6 ± 3.7%, p = 0.033), with no change in Control (-0.7 ± 1.9%, p = 0.211). The magnitude of the improvement was trivial (ES = 0.23) and small (ES = 0.28) for Chamber and Suit respectively, compared to Control. There were no differences observed between the two types of heat exposures (p = 0.974, ES = 0.03; Figure 3).

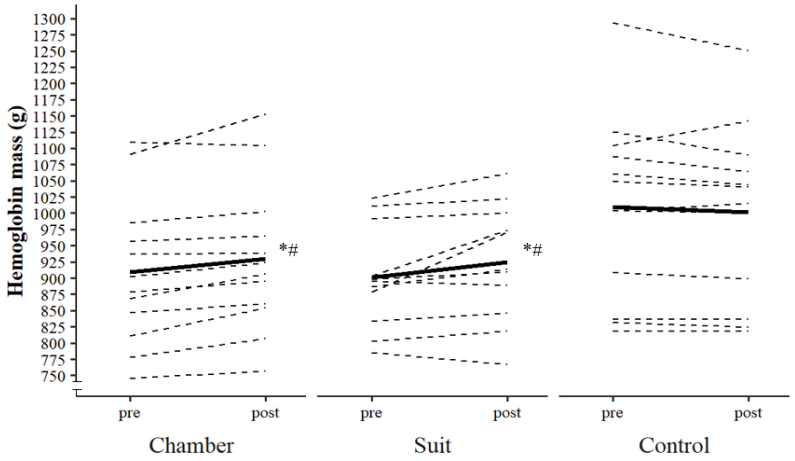


**Figure 3**: Individual data points (dotted lines) and mean values (solid lines) for absolute haemoglobin mass ($Hb_{mass}$) before (pre) and after (post) the intervention period for heat chamber (Chamber), heat suit (Suit) and the control group (Control). *Different (p < 0.050) from pre, #The relative change from pre is different (p < 0.050) from Control.

### *4.3 Maximal oxygen consumption*

There were no statistically significant differences in percent change in $VO_{2max}$ between the three groups (p = 0.365). Nevertheless, there was an increase within Chamber (3.0 ± 4.2%, p = 0.030) and Suit (3.0 ± 4.5%, p = 0.048), but not in Control (1.0 ± 1.9%, p = 0.129; Figure 4a). The magnitude of the improvement in Chamber and Suit was trivial (ES = 0.18 and ES = 0.21, respectively) compared to Control, and the change was also trivial (ES = 0.01) between Chamber and Suit.

### *4.4 Performance: maximal aerobic power output & 15 minutes mean power output*

There was a statistically significant difference between the three groups in percent change in $W_{max}$ (p = 0.011). Post hoc comparisons indicated a difference between Suit and Control (p = 0.011), but not between Chamber and Control (p = 0.555). There was also a tendency (p = 0.069) to difference between Suit and Chamber. The within group increase in $W_{max}$ was 3.5 ± 3.8% (p = 0.009) in Chamber and 7.5 ± 5.3% (p = 0.001) in Suit, while Control did not change (1.6 ± 2.9%, p = 0.126; Figure 4b). The magnitude of the improvement was trivial (ES = 0.16) in Chamber, while it was moderate (ES = 0.58) in Suit, both compared to Control. The magnitude of the improvement in Suit was small compared to Chamber (ES = 0.45).

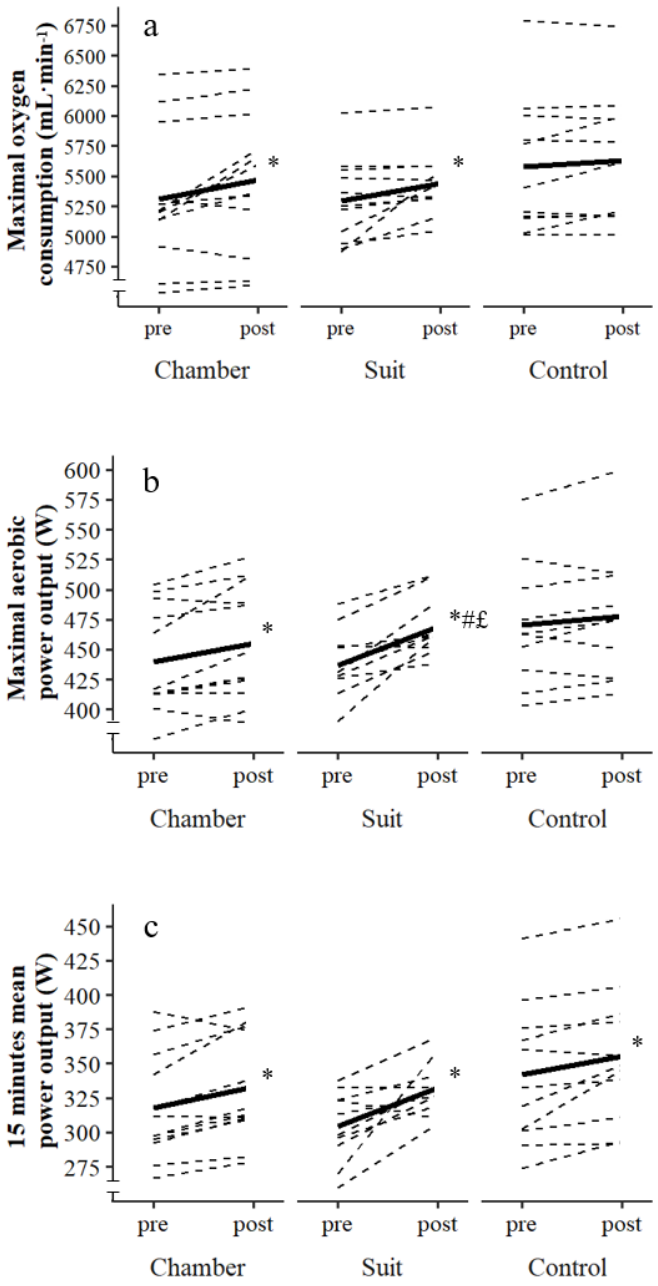


**Figure 4**: Individual data points (dotted lines) and mean values (solid lines) for a) maximal oxygen consumption, b) maximal aerobic power output, and c) 15 minutes mean power output before (pre) and after (post) the intervention period for heat chamber (Chamber), heat suit (Suit) and the control group (Control). *Different (p < 0.050) from pre, #The relative change from pre is different (p < 0.050) from Control, £The relative change from pre tend (p < 0.100) to differ from Chamber.

There were no differences in respect of changes in 15 minutes TT performance between the groups (p = 0.127). All three groups increased their 15 minutes TT performance after the intervention period (Chamber 4.9 ± 4.0%, p = 0.003; Suit: 9.9 ± 10.4%, p = 0.011; Control: 4.3 ± 4.4%, p = 0.006; Figure 4c). The magnitude of the change in Chamber and Suit was trivial (ES = 0.03) and small (ES = 0.34) respectively, compared to Control, and small (ES = 0.39) compared to each other.

### *4.5 General haematological characteristics*

A significant group difference was observed in percent point change in HCT (Table 5). Post hoc comparisons indicated a significantly greater increase in Chamber (p = 0.027) and Suit (p = 0.007) compared to Control, while there were no differences between the two types of heat exposures (p = 0.815). Within groups HCT increased in both Chamber (1.6 ± 1.7%-point, p = 0.008) and Suit (2.1 ± 2.0%-point, p = 0.006), with no change in Control (-0.5 ± 1.9%-point, p = 0.403; Table 5). The magnitude of the improvement was moderate (ES = 0.86) and large (ES = 1.08) for Chamber and Suit respectively, compared to Control, and trivial (ES = 0.20) compared to each other. There was also a significant group difference in percent change in PV (Table 5). Post hoc comparisons of PV indicated a significant reduction in both Chamber (p = 0.034) and Suit (p = 0.021) compared to Control, while there were no differences between Chamber and Suit (p = 0.959). Within groups there was a tendency to decrease (-2.7 ± 5.3%, p = 0.076) and a significant decrease (-3.3 ± 4.4%, p = 0.036) in Chamber and Suit respectively, with no change in Control (3.4 ± 6.8%, p = 0.126; Table 5). The magnitude of the change in Chamber and Suit was moderate (ES = -0.50 and ES = -0.70, respectively) compared to Control, and the change was trivial (ES = 0.02) between Chamber and Suit. No group differences were observed in change of RBCV or BV. Still, RBCV increased within Chamber (3.9 ± 3.9%, p = 0.006) and Suit (5.1 ± 6.7%, p = 0.028), but not in Control (1.2 ± 4.6%, p = 0.403; Table 5). The magnitude of the improvement was trivial (ES = 0.18) and small (ES = 0.30) for Chamber and Suit respectively, compared to Control, and trivial (ES = 0.09) compared to each other. There was no change in BV in any of the groups (Chamber: 0.0 ± 3.2%, p = 0.941; Suit: 0.3 ± 3.6, p = 0.787; Control: 2.4 ± 4.5, p = 0.104; Table 5) and all changes were trivial (Chamber vs Control: ES = -0.21; Suit vs Control: ES = -0.23; Chamber vs Suit: ES = -0.04).

**Table 5**: Haematological data before (pre) and after (post) the intervention period in heat chamber (Chamber), heat suit (Suit) and the control group (Control).

| | Chamber | | Suit | | Control | | ANOVA |
|---|---|---|---|---|---|---|---|
| | Pre | Post | Pre | Post | Pre | Post | (group*time) |
| HCT (%) | 42.1 ± 2.4 | 43.7 ± 2.2$^{*\#}$ | 43.5 ± 2.4 | 45.6 ± 2.8$^{*\#}$ | 44.7 ± 2.4 | 44.2 ± 2.4 | 0.006 |
| RBCV (L) | 2.7 ± 0.4 | 2.8 ± 0.4$^{*}$ | 2.6 ± 0.2 | 2.7 ± 0.3$^{*}$ | 3.0 ± 0.4 | 3.0 ± 0.4 | 0.201 |
| PV (L) | 3.7 ± 0.5 | 3.6 ± 0.4$^{¶\#}$ | 3.4 ± 0.2 | 3.3 ± 0.2$^{*\#}$ | 3.7 ± 0.4 | 3.8 ± 0.4 | 0.013 |
| BV (L) | 6.3 ± 0.8 | 6.3 ± 0.8 | 6.0 ± 0.4 | 6.0 ± 0.4 | 6.6 ± 0.7 | 6.8 ± 0.8 | 0.275 |

HCT: haematocrit; RBCV: red blood cell volume; PV: plasma volume; BV: blood volume. Values are mean ± SD. *Different ($p < 0.050$) from pre, ¶Tendency ($p < 0.100$) to different from pre, #The relative change from pre is different ($p < 0.050$) from Control.

### *4.6 Power output and fractional utilization of $\dot{V}O_{2max}$ at 4 mmol·L$^{-1}$[La$^{-}$]*

There were no statistically significant differences in percent change in power output or fractional utilization of $\dot{V}O_{2max}$ at 4 mmol·L$^{-1}$ [La$^{-}$] between the three groups (Table 6). The power output at 4 mmol·L$^{-1}$ [La$^{-}$] was unchanged within Chamber (1.6 ± 3.4%, p =0.185) and Control (-0.6 ± 5.9, p = 0.679), and increased within Suit (3.4 ± 4.4, p = 0.030; Table 6). The magnitude of change in Chamber and Suit was trivial (ES = 0.17) and small (ES = 0.40) respectively, compared to Control and trivial (ES = 0.22) compared to each other. The fractional utilization of $\dot{V}O_{2max}$ at 4 mmol·L$^{-1}$ [La$^{-}$] was unchanged within all groups (Chamber: -2.1 ± 3.4%-point, p = 0.116; Suit: -1.1 ± 4.6%-point, p = 0.475; Control: 1.5 ± 4.9%-point, p = 0.425; Table 6) and all effect sizes were trivial (Chamber vs Control: ES = -0.11; Suit vs Control: ES = 0.07; Chamber vs Suit: ES = -0.17).

### *4.7 Gross efficiency and [La$^{-}$] at the 3$^{rd}$ last bout of the blood lactate profile in fresh and fatigued state*

There were no statistically significant differences in percent point change in GE or percent change in blood [La$^{-}$] in fresh or fatigued state (Table 6). GE in fresh state was unaffected in all groups (Chamber: 0.2 ± 0.5%-pint, p = 0.191; Suit: 0.4 ± 0.7%-point, p = 0.139; Control: 0.0 ± 0.7%-point, p = 0.896; Table 6). The magnitude of the change in Chamber and Suit was small (ES = 0.37 and ES = 0.49, respectively) compared to Control, and trivial (ES = 0.19) compared to each other. The blood [La$^{-}$] in fresh state was unchanged within Chamber (-3.4 ± 21.5%, p = 0.355) and Control (Control: -0.9 ± 16.4%, p = 0.526), and tended to decrease within Suit (-9.9 ± 13.5%, p = 0.050; Table 6). The magnitude of change in Chamber and Suit

was trivial (ES = -0.09) and small (ES = -0.32) respectively, compared to Control and small (ES = 0.27) compared to each other.

GE in fatigued state was unchanged within Chamber (0.2 ± 0.4%-point, p =0.292) and Control (0.0 ± 0.4%-point, p = 0.782), and increased within Suit (0.5 ± 0.5%-point, p = 0.025). The magnitude of change in Chamber and Suit was trivial (ES = 0.12) and small (ES = 0.47) respectively, compared to Control, and small (ES = 0.37) compared to each other. The blood [$La^-$] in fatigued state was unchanged within Chamber (2.9 ± 31.9%, p = 0.755) and Control (Control: 8.7 ± 25.8%, p = 0.369), and tended to increase within Suit (16.0 ± 23.3%, p = 0.070; Table 6). The magnitude of change in Chamber and Suit was trivial (ES = -0.18) and small (ES = 0.35) respectively, compared to Control, and moderate (ES = -0.56) compared to each other.

**Table 6**: Endurance-performance determinants data before (pre) and after (post) the intervention period in heat chamber (Chamber), heat suit (Suit) and the control group (Control).

| | **Chamber** | | **Suit** | | **Control** | | **ANOVA** |
|---|---|---|---|---|---|---|---|
| | **Pre** | **Post** | **Pre** | **Post** | **Pre** | **Post** | **(group*time)** |
| $Power_{4mmol \cdot L^{-1}}$ (W) | 295 ± 36 | 299 ± 32 | 302 ± 20 | 313 ± 27* | 325 ± 41 | 323 ± 41 | 0.147 |
| $\%\dot{V}O_{2max@4mmol \cdot L^{-1}}$ (%) | 79.0 ± 7.1 | 76.9 ± 4.4 | 80.5 ± 4.9 | 79.4 ± 3.6 | 81.4 ± 5.3 | 79.9 ± 3.2 | 0.878 |
| $GE_{fresh}$ (%) | 19.0 ± 0.8 | 19.2 ± 0.7 | 19.3 ± 0.9 | 19.7 ± 1.2 | 19.2 ± 0.6 | 19.1 ± 0.8 | 0.357 |
| $GE_{fatigue}$ (%) | 18.1 ± 0.9 | 18.3 ± 0.7 | 18.4 ± 0.9 | 18.9 ± 1.1* | 18.7 ± 1.0 | 18.8 ± 1.0 | 0.112 |
| $[La^-]_{fresh}$ ($mmol \cdot L^{-1}$) | 1.6 ± 0.3 | 1.5 ± 0.3 | 1.8 ± 0.4 | 1.6 ± 0.4¶ | 1.7 ± 0.4 | 1.7 ± 0.3 | 0.554 |
| $[La^-]_{fatigue}$ ($mmol \cdot L^{-1}$) | 1.8 ± 0.5 | 1.8 ± 0.5 | 1.9 ± 0.4 | 2.2 ± 0.6¶ | 1.9 ± 0.5 | 2.0 ± 0.6 | 0.478 |

$Power_{4mmol \cdot L^{-1}}$ (W): power output at a blood lactate concentration of 4 $mmol \cdot L^{-1}$; $\%VO_{2max@4mmol \cdot L^{-1}}$: fractional utilization of $VO_{2max}$ at the power output at 4 $mmol \cdot L^{-1}$; $GE_{fresh}$: gross efficiency at the 3rd last bout of the blood lactate profile; $GE_{fatigue}$: gross efficiency at the repetition of the 3rd last bout of the blood lactate profile. $[La^-]_{fatigue}$: blood lactate concentration at the 3rd last bout of the blood lactate profile; $[La^-]_{fatigue}$: blood lactate concentration at the repetition of the 3rd last bout of the blood lactate profile. Values are mean ± SD. *Different (p < 0.050) from pre, ¶Tendency (p < 0.100) to different from pre.

### *4.8 Correlations*

When we pooled all groups to examine the relationship between change in $Hb_{mass}$ and $VO_{2max}$, we observed a small, non-significant positive correlation (Figure 5a). When we pooled all groups to examine the relationship between change in $Hb_{mass}$ and PV, we observed a moderate, significant negative correlation (Figure 5b).

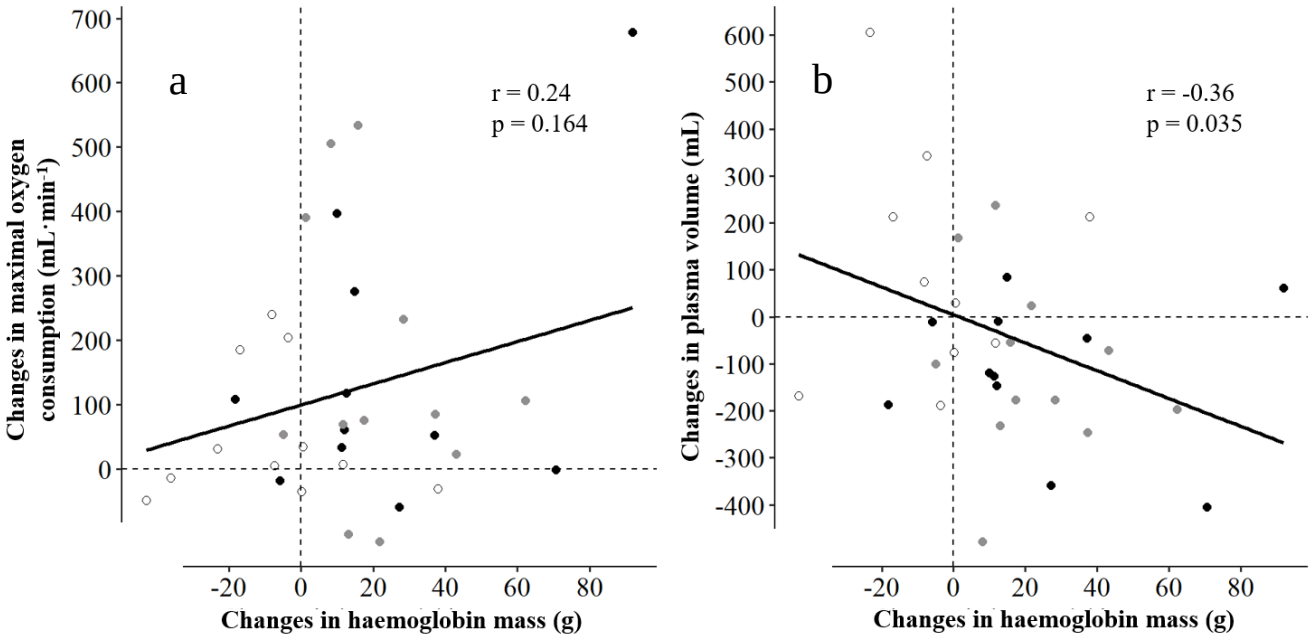


**Figure 5**: Linear regression for a) absolute changes in maximal oxygen consumption and hemoglobin mass, and b) absolute change in plasma volume and hemoglobin mass, for heat chamber (Chamber, black dots), heat suit (Suit, grey dots) and the control group (Control, white dots). Pooled regression slopes (solid lines) are shown.

## 5 DISCUSSION

The main finding of the present study is that the $Hb_{mass}$ increased in both Chamber and Suit, whereas no changes were observed in Control. The increment in HCT was also evident after both heat exercise interventions, but not after the control intervention. Despite similar improvements in $Hb_{mass}$ and HCT, we only observed an increased $W_{max}$ in Suit compared to Control. No other significant differences were observed in respect of other performance and performance determining factors. However, small to moderate effect sizes favouring heat exercise exposures were detected for lactate threshold power output, gross efficiency in fresh and fatigued state and 15 minutes TT mean power output. These effects occurred despite comparable training volume and -intensity between the three groups during the intervention, and could potentially be of practical importance for elite cyclists.

### *5.1 Level of heat stress*

Physiological responses (i.e. rectal temperature, sweat rate, HR and $[La^-]$), RPE and temperature feeling measured during and after each heat exercise session, were overall the same in Chamber and Suit. The same responses occurred thus in both groups, despite of significant differences in ambient temperature and RH, and small differences in power output during the two interventions. This suggests that training in a suit that limiting heat loss may be a simpler, but yet as useful and effective way of executing heat training. Our level of heat stress also seems to be comparable to what was used in a similar heat intervention, where they observed significant haematological adaptations and signs of ergogenic effects in normal temperature (Holen, 2019). Based on this, we should be able to observe some of the same adaptations in our study.

### *5.2 Haemoglobin mass*

Both Chamber and Suit had a superior effect on $Hb_{mass}$ compared to Control, which is in accordance to the findings in Holen (2019). To our knowledge, the latter study is the only previous study examining the effect of a prolonged period (~5 weeks) of heat training on haematological adaptations in elite cyclists ($VO_{2max}$: ~76 $mL \cdot min^{-1} \cdot kg^{-1}$). The study was performed in the same laboratory as ours, with a similar intervention protocol for the climatic chamber group. In agreement with our findings, they observed a ~5% increase in $Hb_{mass}$ after the heat exercise intervention, which differed significantly from the control group. Our findings are also partly in agreement with another study with a similar intervention performed

on trained cyclists ($VO_{2max}$: ~62 mL·min$^{-1}$·kg$^{-1}$) (Oberholzer et al., 2019). Oberholzer et al. (2019) observed a ~3% increase in $Hb_{mass}$, which tended ($p = 0.061$) to differ from the control group. These studies support our findings and suggests that a prolonged period of endurance heat training may trigger a further erythropoietic stimulus and increase $Hb_{mass}$, at least in elite athletes.

Most investigations with shorter durations do not report any change in $Hb_{mass}$ or RBCV after 10 to 21 days of heat exercise exposures (Keiser et al., 2015; Lorenzo et al., 2010; McCleave et al., 2017). Both Holen (2019) and Oberholzer et al. (2019) hypothesized that these durations possibly would be insufficient to elicit increased erythropoiesis. This hypothesis is based on the fact that enhanced RBCV and $Hb_{mass}$ first have been observed after ≥4 weeks of conventional endurance training in untrained subjects (Montero et al., 2017). Still, Karlsen et al. (2015) observed that nine competitive cyclists ($VO_{2max}$: ~62 mL·min$^{-1}$·kg$^{-1}$) increased their $Hb_{mass}$ by ~7% after two weeks on a training camp in natural hot environments, which tended ($p = 0.054$) to differ from the control group. This may suggest that the effect on $Hb_{mass}$ can occur earlier if one is continuously exposed to the heat. However, $Hb_{mass}$ measurements in this study were only performed 12 days before and 12 days after the training camp, which gives the erythropoiesis ~6 weeks to occur. Therefore, it seems likely that ~5 weeks of endurance heat training may be the borderline duration to observe an effect on this haematological characteristic.

That the PV decreased in both Chamber and Suit, and that there was a negatively correlation between PV expansion and increased $Hb_{mass}$, are in contrast with findings from previous studies (Holen, 2019; Karlsen et al., 2015; Lorenzo et al., 2010; McCleave et al., 2017; Oberholzer et al., 2019). These findings were unexpected, as one of the main hypotheses of why the $Hb_{mass}$ increases after endurance heat training, is based on the PV expansion to cause the kidneys to function as a "critmeter" (Donnelly, 2001; Montero & Lundby, 2018). Oberholzer et al. (2019) and Holen (2019) highlight the kidneys «critmeter» function as a key factor for their observed increase in $Hb_{mass}$. However, the concept was only supported by a positive correlation between PV expansion and increased $Hb_{mass}$, since the PV expansion did not differ from the control group in any of the studies. Nevertheless, PV expansion is known to occur within a few days of heat acclimatization (Periard et al., 2016), but there are also some evidence pointing towards a more transient effect (Wyndham, Rogers, Senay & Mitchell, 1976). Since neither we nor any of the other mentioned studies did report any PV

measurements within the first weeks, we cannot exclude that a potentially early PV expansion may have triggered an erythropoietic stimulus in an early phase of the training interventions. If this is the case, and the "critmeter" theory is true, it is likely that the erythropoietic stimulus is greatest in an early phase, and that other mechanisms also contribute to explain the increase in $Hb_{mass}$. Other mechanisms that potentially could provoke a erythropoietic stimulus from endurance heat training, includes an increased HSP expression, which is speculated to augment EPO secretion (Hawley et al., 2018). PV-regulating hormones and a reduction of central venous pressure, may contribute to a potential increase in RBCV (Montero & Lundby, 2018; Montero et al., 2016).

### *5.3 Maximal oxygen consumption*

Regarding $VO_{2max}$, our observations supports the findings of previous heat training studies with no improvements in $VO_{2max}$ despite of an increased $Hb_{mass}$ (Holen, 2019; Karlsen et al., 2015; Mikkelsen et al., 2019). Because of the strong relationship between total $Hb_{mass}$ and $VO_{2max}$ (Lundby & Robach, 2015; Walter Schmidt & Prommer, 2010), one could expect that a change in total $Hb_{mass}$ would be associated with a change in $VO_{2max}$. This was not the case in our study, nor in any of the other aforementioned heat training studies (Holen, 2019; Karlsen et al., 2015; Mikkelsen et al., 2019). The reason why could possibly be the magnitude of the improvements. Considering that each 1g of $Hb_{mass}$, gained theoretically could correspond to a ~4 $mL \cdot min^{-1}$ gain in $VO_{2max}$ (W. Schmidt & Prommer, 2008; 2010), we could expect an +88 and +96 $mL \cdot min^{-1}$ improvement in $VO_{2max}$ for Chamber and Suit, and a -32 $mL \cdot min^{-1}$ decrease in Control. The actual changes were +155, +149 and +52 $mL \cdot min^{-1}$ in Chamber, Suit and Control, respectively. The fact that these improvements are more than we could have expected, highlight the multifactorial mechanisms of $VO_{2max}$ and that $Hb_{mass}$ do not exclusively determine $VO_{2max}$ (di Prampero, 2003; Skattebo et al., 2020). Findings from altitude studies also tells us that the relationship between absolute gain in $Hb_{mass}$ and $VO_{2max}$, not always are present (Schmidt & Prommer, 2010). Therefore, it is not surprising that we in the present study could not identify a significant linear relationship between total $Hb_{mass}$ and $VO_{2max}$.

We also observed an increased RBCV in both Chamber and Suit, which in theory could have a positive impact on the oxygen transport capacity by increasing the BV and maximal cardiac output via the Frank-Starling mechanism (Coyle et al., 1990). However, this was not the case in the present study, as we observed a similar reduction in PV, and an accordingly unchanged BV.

### *5.4 Endurance-performance determinants: Power output and fractional utilization of $\dot{V}O_{2max}$ at 4 mmol·L$^{-1}$[La$^-$], and GE and [La$^-$] in fresh and fatigued state*

In accordance with previous heat training studies, we did not find any significant differences in power output or fractional utilization of $\dot{V}O_{2max}$ at 4 mmol·L$^{-1}$ [La$^-$] (Holen, 2019; Mikkelsen et al., 2019). However, there was a small effect size in favor of Suit compared to Control in power output at 4 mmol·L$^{-1}$ [La$^-$], which is similar to the observations done in the climatic chamber group in (Holen, 2019) study. Why this effect only occurred in Suit and not Chamber in our study, remains uncertain. The improvement in power output at 4 mmol·L$^{-1}$ [La$^-$] could be related to the increased $Hb_{mass}$, increased oxygen supply to the exercising muscles. Since both Chamber and Suit increased $Hb_{mass}$ to the same extent, we could theoretically expect the same effect in both groups. The most obvious cause of this differences is individual variations. However, there are some observations pointing towards an improved muscle mitochondrial function and increased mitochondrial biogenesis after passive heat exposures, directly performed upon vastus lateralis (Hafen, Preece, Sorensen, Hancock & Hyldahl, 2018). This adaptation could potentially lower the metabolic strain and improve power output at 4 mmol·L$^{-1}$ [La$^-$], as well as other measurements of endurance-performance in normal temperature (Bassett & Howley, 2000; Coyle, 1999; Ivy et al., 1980). It could be speculated that a heat suit that limit loss of heat, may prompt a more direct heat stress upon the skeletal muscles, and thereby induce larger peripheral adaptations.

Holen (2019) also observed that the heat training intervention lowered blood [La$^-$] in fatigued state, to a larger extent than the control intervention. These findings stand in contrast to our findings. Furthermore, in accordance with Holen (2019) study, we did not observe any significant changes in blood [La$^-$] and GE in fresh state, or GE in fatigued state. Still, we observed small effect sizes in favour of heat exercise exposures in GE in fresh and fatigued state, and blood [La$^-$] in fresh state. This could potentially be of practical importance for elite cyclists.

### *5.5 Performance: maximal aerobic power output & 15 minutes mean power output*

$W_{max}$, which is a great indicator of cycling performance (Faria et al., 2005a), increased in both Chamber and Suit, but only the increment in Suit differed from Control. This is somehow contradictory to previous heat training studies, where they did not observe any improvement in $W_{max}$ (Holen, 2019; Mikkelsen et al., 2019). The fact that Suit but not Chamber increased their $W_{max}$ compared to Control, were unexpected. It could as discussed, be due to peripheral adaptations such as improved mitochondrial function (Hafen et al., 2018), or just individual variations. Another explanation could potentially be the slightly higher power output during the Suit sessions compared to the Chamber sessions. Still, we find this unlikely as no findings of differences in post exercise gene expression of PGC-1α (which is a key regulator of physiological adaptation to endurance training (Popov, 2018)) have previously been observed after 65 minutes cycling at 50 or 70% of $VO_{2max}$ in trained athletes (Popov et al., 2015). There were also no differences in $[La^-]$ or HR during the sessions. Therefore, if the improvement relates to the distinctive character of the interventions, it is likely that the differences between Chamber and Suit is caused by differences in the additional heat stress and not the metabolic rate during the sessions. Of note, previous studies have observed increased $W_{max}$ after a period of heavy strength training (Rønnestad et al., 2015; B. R. Rønnestad et al., 2017), but because there were no differences in change of $P_{max}$ or volume of additional strength training, we find it unlikely that this could affect the result.

15 minutes mean power output were increased in all three groups without any significant group differences. These findings are in accordance with findings from previous heat training studies with the same duration (Holen, 2019; Mikkelsen et al., 2019). The fact that Control increased their TT performance despite no other signs of hematological or performance determining improvements, was somewhat unexpected. Nevertheless, this was also the case in Mikkelsen et al. (2019) and Holen (2019), who also observed an improved TT performance in both the heat exercise intervention group and the control group. Although our study was performed on elite cyclists who are accustomed to TT pacing, it is conceivable that a learning effect may have occurred and affected the result. Therefore, one could question if a familiarization test would have been necessary to detect any differences. Furthermore, McCleave et al. (2017) observed improved 3 km running performance 3 weeks after a heat training intervention, but not directly after. Because of this, Mikkelsen et al. (2019) speculates in whether the timing of the post-test could be of practical importance. They point out that the

potential endurance performance gain from an altered $Hb_{mass}$, possibly could be outweighed by a concurrent PV mediated hemodilution. However, this was as not the case in our study as we did not observe a PV expansion in any of the three groups.

### *5.6 Practical implications*

Heat training have been suggested to induce some of the same haematological adaptations as exposures to high altitude (Holen, 2019). Still, training in a climatic chamber or in a natural hot environment can be both unpractical and expensive. We might have overcome this challenge demonstrating that similar adaptations can be achieved using a more practical heat suit that limits heat loss. Despite some inconsistent findings of improved endurance performance in normal temperature, the overall effect size was in favour of endurance heat training. This could as previously mentioned potentially be of practical importance for elite cyclists. It is also noteworthy that the two heat training interventions did not compromise the effect of regular training. Since cyclists often compete in hot climates, it is conceivable that including heat training as a part of the training schedule, could lead to enhanced performance in both normal and hot conditions.

### *5.7 Conclusion*

In conclusion, the present study demonstrates that 5 weeks of heat training in a climatic chamber or with a heat suit that limits heat loss, induce the same effects on $Hb_{mass}$, and superior effects compared to regular training. The study also demonstrates that training in a heat suit induces similar, and possibly greater effects in respect of endurance performance in normal temperature, compared to training in a climatic chamber. Whether the effects of heat training on endurance performance in normal temperature are superior to the effects of regular training, are still not clear.

### *5.8 Perspectives*

Based on the findings from both the current study and Holen (2019), there are reasons to recommend heat training to elite athletes to increase $Hb_{mass}$. However, the impact on endurance performance in normal temperature remains unclear. Further investigations with similar interventions are therefore needed, and we would recommend future studies to focus on the potential effect on endurance performance in normal temperature. It would also be

interesting to examine the effects of heat interventions with even longer durations. To get further insights in the underlying mechanisms of the haematological adaptations, more frequent measurements of $Hb_{mass}$ and intravascular volumes might be necessary. It would also be of interest to measure PV regulating hormones, as well as including muscle biopsies or other methodological approaches to investigate potential peripheral adaptations.

## REFERENCES


Armstrong, L. E. & Maresh, C. M. (1991). The induction and decay of heat acclimatisation in trained athletes. *Sports Med*, *12*(5), 302-312. https://doi.org/10.2165/00007256-199112050-00003

Bassett, D. R., Jr. & Howley, E. T. (2000). Limiting factors for maximum oxygen uptake and determinants of endurance performance. *Med Sci Sports Exerc*, *32*(1), 70-84.

Bonne, T. C., Doucende, G., Flück, D., Jacobs, R. A., Nordsborg, N. B., Robach, P., … Lundby, C. (2014). Phlebotomy eliminates the maximal cardiac output response to six weeks of exercise training. *Am J Physiol Regul Integr Comp Physiol*, *306*(10), R752-760. https://doi.org/10.1152/ajpregu.00028.2014

Borg, G. A. (1982). Psychophysical bases of perceived exertion. *Med Sci Sports Exerc*, *14*(5), 377-381.

Bottinelli, R. & Reggiani, C. (2000). Human skeletal muscle fibres: molecular and functional diversity. *Prog Biophys Mol Biol*, *73*(2-4), 195-262. https://doi.org/10.1016/s0079-6107(00)00006-7

Boushel, R. & Saltin, B. (2013). Ex vivo measures of muscle mitochondrial capacity reveal quantitative limits of oxygen delivery by the circulation during exercise. *The International Journal of Biochemistry & Cell Biology*, *45*(1), 68-75. https://doi.org/https://doi.org/10.1016/j.biocel.2012.09.024

Buchheit, M., Voss, S. C., Nybo, L., Mohr, M. & Racinais, S. (2011). Physiological and performance adaptations to an in-season soccer camp in the heat: associations with heart rate and heart rate variability. *Scand J Med Sci Sports*, *21*(6), e477-485. https://doi.org/10.1111/j.1600-0838.2011.01378.x

Burge, C. M. & Skinner, S. L. (1995). Determination of hemoglobin mass and blood volume with CO: evaluation and application of a method. *J Appl Physiol*, *79*(2), 623-631. https://doi.org/10.1152/jappl.1995.79.2.623

Convertino, V. A. (2007). Blood volume response to physical activity and inactivity. *Am J Med Sci*, *334*(1), 72-79. https://doi.org/10.1097/MAJ.0b013e318063c6e4

Convertino, V. A., Mack, G. W. & Nadel, E. R. (1991). Elevated central venous pressure: a consequence of exercise training-induced hypervolemia? *Am J Physiol*, *260*(2 Pt 2), R273-277. https://doi.org/10.1152/ajpregu.1991.260.2.R273

Corbett, J., Neal, R. A., Lunt, H. C. & Tipton, M. J. (2014). Adaptation to heat and exercise performance under cooler conditions: a new hot topic. *Sports Med*, *44*(10), 1323-1331. https://doi.org/10.1007/s40279-014-0212-8

Coyle, E. F. (1995). Integration of the physiological factors determining endurance performance ability. *Exerc Sport Sci Rev*, *23*, 25-63.

Coyle, E. F. (1999). Physiological determinants of endurance exercise performance. *J Sci Med Sport*, *2*(3), 181-189.

Coyle, E. F. (2005). Improved muscular efficiency displayed as Tour de France champion matures. *J Appl Physiol (1985)*, *98*(6), 2191-2196. https://doi.org/10.1152/japplphysiol.00216.2005

Coyle, E. F. (2013). Reconsideration of a Tour de France cyclist. *J Appl Physiol (1985)*, *114*(10), 1361. https://doi.org/10.1152/japplphysiol.00213.2013

Coyle, E. F., Coggan, A. R., Hopper, M. K. & Walters, T. J. (1988). Determinants of endurance in well-trained cyclists. *J Appl Physiol (1985)*, *64*(6), 2622-2630. https://doi.org/10.1152/jappl.1988.64.6.2622

Coyle, E. F., Hopper, M. K. & Coggan, A. R. (1990). Maximal oxygen uptake relative to plasma volume expansion. *Int J Sports Med*, *11*(2), 116-119. https://doi.org/10.1055/s-2007-1024774

Coyle, E. F., Sidossis, L. S., Horowitz, J. F. & Beltz, J. D. (1992). Cycling efficiency is related to the percentage of type I muscle fibers. *Med Sci Sports Exerc, 24*(7), 782-788.

De Pauw, K., Roelands, B., S Cheung, S., De Geus, B., Rietjens, G. & Meeusen, R. (2013). Guidelines to Classify Subject Groups in Sport-Science Research. *Int J Sports Physiol Perform, 8*, 111-122. https://doi.org/10.1123/ijspp.8.2.111

Dempsey, J. A., Hanson, P. G. & Henderson, K. S. (1984). Exercise-induced arterial hypoxaemia in healthy human subjects at sea level. *J Physiol, 355*, 161-175. https://doi.org/10.1113/jphysiol.1984.sp015412

di Prampero, P. E. (2003). Factors limiting maximal performance in humans. *European Journal of Applied Physiology, 90*(3-4), 420-429.

Donnelly, S. (2001). Why is erythropoietin made in the kidney? The kidney functions as a critmeter. *Am J Kidney Dis, 38*(2), 415-425. https://doi.org/10.1053/ajkd.2001.26111

Dunn, A., Lo, V. & Donnelly, S. (2007). The Role of the Kidney in Blood Volume Regulation: The Kidney as a Regulator of the Hematocrit. *Am J Med Sci, 334*, 65-71. https://doi.org/10.1097/MAJ.0b013e318095a4ae

Ekblom, B. & Hermansen, L. (1968). Cardiac output in athletes. *J Appl Physiol, 25*(5), 619-625. https://doi.org/10.1152/jappl.1968.25.5.619

Faiss, R., Willis, S., Born, D. P., Sperlich, B., Vesin, J. M., Holmberg, H. C. & Millet, G. P. (2015). Repeated double-poling sprint training in hypoxia by competitive cross-country skiers. *Med Sci Sports Exerc, 47*(4), 809-817. https://doi.org/10.1249/mss.0000000000000464

Faria, E., Parker, D. & Faria, I. (2005a). The Science of Cycling - Part 1.

Faria, E., Parker, D. & Faria, I. (2005b). The Science of Cycling: Factors Affecting Performance ??? Part 2. *Sports medicine (Auckland, N.Z.), 35*, 313-337.

Garrett, A. T., Rehrer, N. J. & Patterson, M. J. (2011). Induction and decay of short-term heat acclimation in moderately and highly trained athletes. *Sports Med, 41*(9), 757-771. https://doi.org/10.2165/11587320-000000000-00000

Hafen, P. S., Preece, C. N., Sorensen, J. R., Hancock, C. R. & Hyldahl, R. D. (2018). Repeated exposure to heat stress induces mitochondrial adaptation in human skeletal muscle. *J Appl Physiol (1985), 125*(5), 1447-1455. https://doi.org/10.1152/japplphysiol.00383.2018

Hauser, A., Schmitt, L., Troesch, S., Saugy, J. J., Cejuela-Anta, R., Faiss, R., … Millet, G. P. (2016). Similar Hemoglobin Mass Response in Hypobaric and Normobaric Hypoxia in Athletes. *Med Sci Sports Exerc, 48*(4), 734-741. https://doi.org/10.1249/mss.0000000000000808

Hawley, J. A., Lundby, C., Cotter, J. D. & Burke, L. M. (2018). Maximizing Cellular Adaptation to Endurance Exercise in Skeletal Muscle. *Cell Metab, 27*(5), 962-976. https://doi.org/10.1016/j.cmet.2018.04.014

Heinicke, K., Wolfarth, B., Winchenbach, P., Biermann, B., Schmid, A., Huber, G., … Schmidt, W. (2001). Blood volume and hemoglobin mass in elite athletes of different disciplines. *Int J Sports Med, 22*(7), 504-512. https://doi.org/10.1055/s-2001-17613

Helgerud, J., Høydal, K., Wang, E., Karlsen, T., Berg, P., Bjerkaas, M., … Hoff, J. (2007). Aerobic high-intensity intervals improve VO2max more than moderate training. *Med Sci Sports Exerc, 39*(4), 665-671. https://doi.org/10.1249/mss.0b013e3180304570

Holen, E. H. (2019). Fører 5 uker varmetrening til økt hemoglobinmasse og forbedret utholdenhetsprestasjon i normaltemperatur hos elitesyklister? *Høgskolen i Innlandet, Lillehammer*. .

Holloszy, J. O. & Coyle, E. F. (1984). Adaptations of skeletal muscle to endurance exercise and their metabolic consequences. *J Appl Physiol, 56*(4), 831-838. https://doi.org/10.1152/jappl.1984.56.4.831

Hopkins, W. G., Marshall, S. W., Batterham, A. M. & Hanin, J. (2009). Progressive statistics for studies in sports medicine and exercise science. *Med Sci Sports Exerc, 41*(1), 3-13. https://doi.org/10.1249/MSS.0b013e31818cb278

Hue, O., Antoine-Jonville, S. & Sara, F. (2007). The Effect of 8 Days of Training in Tropical Environment on Performance in Neutral Climate in Swimmers. *Int J Sports Med, 28*, 48-52. https://doi.org/10.1055/s-2006-923958

Ivy, J., Withers, R., Handel, P., Elger, D. & Costill, D. (1980). Muscle respiratory capacity and fiber type as determinant of the lactate threshold. *Journal of applied physiology: respiratory, environmental and exercise physiology, 48*, 523-527. https://doi.org/10.1152/jappl.1980.48.3.523

Jeukendrup, A. E., Craig, N. P. & Hawley, J. A. (2000). The bioenergetics of World Class Cycling. *J Sci Med Sport, 3*(4), 414-433. https://doi.org/10.1016/s1440-2440(00)80008-0

Jones, A. M. (2006). The Physiology of the World Record Holder for the Women's Marathon. *International Journal of Sports Science & Coaching, 1*(2), 101-116. https://doi.org/10.1260/174795406777641258

Kanstrup, I. L. & Ekblom, B. (1982). Acute hypervolemia, cardiac performance, and aerobic power during exercise. *J Appl Physiol Respir Environ Exerc Physiol, 52*(5), 1186-1191. https://doi.org/10.1152/jappl.1982.52.5.1186

Karlsen, A., Racinais, S., Jensen, M. V., Nørgaard, S. J., Bonne, T. & Nybo, L. (2015). Heat acclimatization does not improve VO2max or cycling performance in a cool climate in trained cyclists. *Scand J Med Sci Sports, 25*(S1), 269-276. https://doi.org/10.1111/sms.12409

Keiser, S., Fluck, D., Huppin, F., Stravs, A., Hilty, M. P. & Lundby, C. (2015). Heat training increases exercise capacity in hot but not in temperate conditions: a mechanistic counter-balanced cross-over study. *Am J Physiol Heart Circ Physiol, 309*(5), H750-761. https://doi.org/10.1152/ajpheart.00138.2015

Klausen, K., Secher, N. H., Clausen, J. P., Hartling, O. & Trap-Jensen, J. (1982). Central and regional circulatory adaptations to one-leg training. *J Appl Physiol Respir Environ Exerc Physiol, 52*(4), 976-983. https://doi.org/10.1152/jappl.1982.52.4.976

Lee, B. J., Miller, A., James, R. S. & Thake, C. D. (2016). Cross Acclimation between Heat and Hypoxia: Heat Acclimation Improves Cellular Tolerance and Exercise Performance in Acute Normobaric Hypoxia. *Front Physiol, 7*, 78-78. https://doi.org/10.3389/fphys.2016.00078

Levine, B. D. (2008). .VO2max: what do we know, and what do we still need to know? *J Physiol, 586*(1), 25-34. https://doi.org/10.1113/jphysiol.2007.147629

Lorenzo, S., Halliwill, J. R., Sawka, M. N. & Minson, C. T. (2010). Heat acclimation improves exercise performance. *J Appl Physiol (1985), 109*(4), 1140-1147. https://doi.org/10.1152/japplphysiol.00495.2010

Lundby, C. & Robach, P. (2015). Performance Enhancement: What Are the Physiological Limits? *Physiology, 30*(4), 282-292. https://doi.org/10.1152/physiol.00052.2014

McCleave, E. L., Slattery, K. M., Duffield, R., Saunders, P. U., Sharma, A. P., Crowcroft, S. J. & Coutts, A. J. (2017). Temperate Performance Benefits after Heat, but Not Combined Heat and Hypoxic Training. *Med Sci Sports Exerc, 49*(3), 509-517. https://doi.org/10.1249/mss.0000000000001138

McKenzie, D. C. (2012). Respiratory physiology: adaptations to high-level exercise. *Br J Sports Med, 46*(6), 381-384. https://doi.org/10.1136/bjsports-2011-090824

McLellan, T. M., Daanen, H. A. & Cheung, S. S. (2013). Encapsulated environment. *Compr Physiol, 3*(3), 1363-1391. https://doi.org/10.1002/cphy.c130002
Mikkelsen, C. J., Junge, N., Piil, J. F., Morris, N. B., Oberholzer, L., Siebenmann, C., … Nybo, L. (2019). Prolonged Heat Acclimation and Aerobic Performance in Endurance Trained Athletes. *Front Physiol, 10*(1372). https://doi.org/10.3389/fphys.2019.01372
Minson, C. T. & Cotter, J. D. (2016). CrossTalk proposal: Heat acclimatization does improve performance in a cool condition. *J Physiol, 594*(2), 241-243. https://doi.org/10.1113/jp270879
Mogensen, M., Bagger, M., Pedersen, P. K., Fernström, M. & Sahlin, K. (2006). Cycling efficiency in humans is related to low UCP3 content and to type I fibres but not to mitochondrial efficiency. *J Physiol, 571*(Pt 3), 669-681. https://doi.org/10.1113/jphysiol.2005.101691
Mondal, H. & Budh, D. P. (2020). Hematocrit (HCT). I *StatPearls*. Treasure Island (FL): StatPearls Publishing



Montero, D., Breenfeldt-Andersen, A., Oberholzer, L., Haider, T., Goetze, J. P., Meinild-Lundby, A.-K. & Lundby, C. (2017). Erythropoiesis with endurance training: dynamics and mechanisms. *American Journal of Physiology-Regulatory, Integrative and Comparative Physiology, 312*(6), R894-R902. https://doi.org/10.1152/ajpregu.00012.2017
Montero, D., Breenfeldt-Andersen, A., Oberholzer, L., Haider, T., Goetze, J. P., Meinild-Lundby, A. K. & Lundby, C. (2017). Erythropoiesis with endurance training: dynamics and mechanisms. *Am J Physiol Regul Integr Comp Physiol, 312*(6), R894-r902. https://doi.org/10.1152/ajpregu.00012.2017
Montero, D., Cathomen, A., Jacobs, R. A., Fluck, D., de Leur, J., Keiser, S., … Lundby, C. (2015). Haematological rather than skeletal muscle adaptations contribute to the increase in peak oxygen uptake induced by moderate endurance training. *J Physiol, 593*(20), 4677-4688. https://doi.org/10.1113/JP270250
Montero, D. & Lundby, C. (2018). Regulation of Red Blood Cell Volume with Exercise Training. *Compr Physiol, 9*(1), 149-164. https://doi.org/10.1002/cphy.c180004
Montero, D. & Lundby, C. (2019). Arterial oxygen content regulates plasma erythropoietin independent of arterial oxygen tension: a blinded crossover study. *Kidney Int, 95*(1), 173-177. https://doi.org/10.1016/j.kint.2018.09.015
Montero, D., Rauber, S., Goetze, J. P. & Lundby, C. (2016). Reduction in central venous pressure enhances erythropoietin synthesis: role of volume-regulating hormones. *Acta Physiologica, 218*(2), 89-97. https://doi.org/10.1111/apha.12708
Morris, S. B. (2008). Estimating Effect Sizes From Pretest-Posttest-Control Group Designs. *Organizational Research Methods, 11*(2), 364-386. https://doi.org/10.1177/1094428106291059
Mortensen, S. P., Dawson, E. A., Yoshiga, C. C., Dalsgaard, M. K., Damsgaard, R., Secher, N. H. & González-Alonso, J. (2005). Limitations to systemic and locomotor limb muscle oxygen delivery and uptake during maximal exercise in humans. *J Physiol, 566*(1), 273-285. https://doi.org/10.1113/jphysiol.2005.086025
Neal, R. A., Corbett, J., Massey, H. C. & Tipton, M. J. (2016). Effect of short-term heat acclimation with permissive dehydration on thermoregulation and temperate exercise performance. *Scand J Med Sci Sports, 26*(8), 875-884. https://doi.org/10.1111/sms.12526
Neal, R. A., Massey, H. C., Tipton, M. J., Young, J. S. & Corbett, J. (2016). Effect of Permissive Dehydration on Induction and Decay of Heat Acclimation, and Temperate

Exercise Performance. *Front Physiol, 7*, 564-564. https://doi.org/10.3389/fphys.2016.00564
Nielsen, B., Hales, J. R., Strange, S., Christensen, N. J., Warberg, J. & Saltin, B. (1993). Human circulatory and thermoregulatory adaptations with heat acclimation and exercise in a hot, dry environment. *J Physiol, 460*, 467-485. https://doi.org/10.1113/jphysiol.1993.sp019482
Nybo, L. & Lundby, C. (2016). Rebuttal by Lars Nybo and Carsten Lundby. *J Physiol, 594*(2), 251. https://doi.org/10.1113/jp271668
Nybo, L., Rasmussen, P. & Sawka, M. N. (2014). Performance in the heat-physiological factors of importance for hyperthermia-induced fatigue. *Compr Physiol, 4*(2), 657-689. https://doi.org/10.1002/cphy.c130012
Oberholzer, L., Siebenmann, C., Mikkelsen, C., Junge, N., Feder Piil, J., Morris, N., … Lundby, C. (2019). Hematological Adaptations to Prolonged Heat Acclimation in Endurance-Trained Males. *Front Physiol, 10*, 1379. https://doi.org/10.3389/fphys.2019.01379
Pedersen, B. K. & Saltin, B. (2015). Exercise as medicine - evidence for prescribing exercise as therapy in 26 different chronic diseases. *Scand J Med Sci Sports, 25 Suppl 3*, 1-72. https://doi.org/10.1111/sms.12581
Periard, J. D., Travers, G. J. S., Racinais, S. & Sawka, M. N. (2016). Cardiovascular adaptations supporting human exercise-heat acclimation. *Auton Neurosci, 196*, 52-62. https://doi.org/10.1016/j.autneu.2016.02.002
Popov, D. V. (2018). Adaptation of Skeletal Muscles to Contractile Activity of Varying Duration and Intensity: The Role of PGC-1alpha. *Biochemistry (Mosc), 83*(6), 613-628. https://doi.org/10.1134/s0006297918060019
Popov, D. V., Lysenko, E. A., Vepkhvadze, T. F., Kurochkina, N. S., Maknovskii, P. A. & Vinogradova, O. L. (2015). Promoter-specific regulation of PPARGC1A gene expression in human skeletal muscle. *J Mol Endocrinol, 55*(2), 159-168. https://doi.org/10.1530/jme-15-0150
Powers, S. K., Lawler, J., Dempsey, J. A., Dodd, S. & Landry, G. (1989). Effects of incomplete pulmonary gas exchange on VO2 max. *J Appl Physiol (1985), 66*(6), 2491-2495. https://doi.org/10.1152/jappl.1989.66.6.2491
Racinais, S., Buchheit, M., Bilsborough, J., Bourdon, P., Cordy, J. & Coutts, A. (2013). Physiological and Performance Responses to a Training Camp in the Heat in Professional Australian Football Players. *Int J Sports Physiol Perform, 9*. https://doi.org/10.1123/IJSPP.2013-0284
Rhea, M. R. (2004). Determining the magnitude of treatment effects in strength training research through the use of the effect size. *J Strength Cond Res, 18*(4), 918-920. https://doi.org/10.1519/14403.1
Rodriguez, F. A., Iglesias, X., Feriche, B., Calderon-Soto, C., Chaverri, D., Wachsmuth, N. B., … Levine, B. D. (2015). Altitude Training in Elite Swimmers for Sea Level Performance (Altitude Project). *Med Sci Sports Exerc, 47*(9), 1965-1978. https://doi.org/10.1249/mss.0000000000000626
Ronnestad, B. R., Hansen, J., Hollan, I. & Ellefsen, S. (2015). Strength training improves performance and pedaling characteristics in elite cyclists. *Scand J Med Sci Sports, 25*(1), e89-98. https://doi.org/10.1111/sms.12257
Rusko, H. (1987). The effect of training on aerobic power characteristics of young cross-country skiers. *J Sports Sci, 5*(3), 273-286. https://doi.org/10.1080/02640418708729782
Rønnestad, B., Hansen, J., Stenslokken, L., Joyner, M. & Lundby, C. (2019). Case studies in Physiology: temporal changes in determinants of aerobic performance in individual

going from alpine skier to world champion time trial cyclist. *J Appl Physiol, 127*. https://doi.org/10.1152/japplphysiol.00798.2018
Rønnestad, B. R., Hansen, J. & Ellefsen, S. (2014). Block periodization of high-intensity aerobic intervals provides superior training effects in trained cyclists. *Scand J Med Sci Sports, 24*(1), 34-42. https://doi.org/10.1111/j.1600-0838.2012.01485.x
Rønnestad, B. R., Hansen, J. & Nygaard, H. (2017). 10 weeks of heavy strength training improves performance-related measurements in elite cyclists. *J Sports Sci, 35*(14), 1435-1441. https://doi.org/10.1080/02640414.2016.1215499
Santalla, A., Naranjo Orellana, J. & Terrados, N. (2009). Muscle Efficiency Improves over Time in World-Class Cyclists. *Med Sci Sports Exerc, 41*, 1096-1101. https://doi.org/10.1249/MSS.0b013e318191c802
Saunders, P. U., Pyne, D. B., Telford, R. D. & Hawley, J. A. (2004). Factors affecting running economy in trained distance runners. *Sports Med, 34*(7), 465-485. https://doi.org/10.2165/00007256-200434070-00005
Sawka, M., Young, A., Cadarette, B., Levine, L. & Pandolf, K. (1985). Influence of heat stress and acclimation on maximal aerobic power. *Eur J Appl Physiol Occup Physiol, 53*, 294-298. https://doi.org/10.1007/BF00422841
Schmidt, W. & Prommer, N. (2008). Effects of various training modalities on blood volume. *Scand J Med Sci Sports, 18 Suppl 1*, 57-69. https://doi.org/10.1111/j.1600-0838.2008.00833.x
Schmidt, W. & Prommer, N. (2010). Impact of Alterations in Total Hemoglobin Mass on V˙O2max. *Exerc Sport Sci Rev, 38*(2), 68-75. https://doi.org/10.1097/JES.0b013e3181d4957a
Scoon, G. S., Hopkins, W. G., Mayhew, S. & Cotter, J. D. (2007). Effect of post-exercise sauna bathing on the endurance performance of competitive male runners. *J Sci Med Sport, 10*(4), 259-262. https://doi.org/10.1016/j.jsams.2006.06.009
Senay, L. C., Mitchell, D. & Wyndham, C. H. (1976). Acclimatization in a hot, humid environment: body fluid adjustments. *J Appl Physiol, 40*(5), 786-796. https://doi.org/10.1152/jappl.1976.40.5.786
Siebenmann, C., Keiser, S., Robach, P. & Lundby, C. (2017). CORP: The assessment of total hemoglobin mass by carbon monoxide rebreathing. *J Appl Physiol (1985), 123*(3), 645-654. https://doi.org/10.1152/japplphysiol.00185.2017
Skattebo, Ø., Bjerring, A. W., Auensen, M., Sarvari, S. I., Cumming, K. T., Capelli, C. & Hallén, J. (2020). Blood volume expansion does not explain the increase in peak oxygen uptake induced by 10 weeks of endurance training. *European Journal of Applied Physiology*. https://doi.org/10.1007/s00421-020-04336-2
Støren, Ø., Ulevåg, K., Larsen, M. H., Støa, E. M. & Helgerud, J. (2013). Physiological Determinants of the Cycling Time Trial, *27*(9), 2366-2373. https://doi.org/10.1519/JSC.0b013e31827f5427
Toner, M. M., Drolet, L. L. & Pandolf, K. B. (1986). Perceptual and physiological responses during exercise in cool and cold water. *Percept Mot Skills, 62*(1), 211-220. https://doi.org/10.2466/pms.1986.62.1.211
Tønnessen, E. & Rønnestad, B. R. (2018). *Trening: Fra barneidrett til toppidrett* (1. utg.). Oslo: Gyldendal Norsk Forlag AS.
Vikmoen, O., Ellefsen, S., Trøen, Ø., Hollan, I., Hanestadhaugen, M., Raastad, T. & Rønnestad, B. R. (2016). Strength training improves cycling performance, fractional utilization of VO2max and cycling economy in female cyclists. *Scand J Med Sci Sports, 26*(4), 384-396. https://doi.org/10.1111/sms.12468

Warburton, D. E., Gledhill, N. & Quinney, H. A. (2000). Blood volume, aerobic power, and endurance performance: potential ergogenic effect of volume loading. *Clin J Sport Med, 10*(1), 59-66. https://doi.org/10.1097/00042752-200001000-00011

Wilber, R. L., Zawadzki, K. M., Kearney, J. T., Shannon, M. P. & Disalvo, D. (1997). Physiological profiles of elite off-road and road cyclists. *Med Sci Sports Exerc, 29*(8), 1090-1094. https://doi.org/10.1097/00005768-199708000-00015

Wyndham, C. H., Rogers, G. G., Senay, L. C. & Mitchell, D. (1976). Acclimization in a hot, humid environment: cardiovascular adjustments. *J Appl Physiol, 40*(5), 779-785. https://doi.org/10.1152/jappl.1976.40.5.779

*Vedlegg*

***Vedlegg 1 (Appendix A): Samtykkeskjema (Written informed consent)***

## Vil du delta i forskningsprosjektet:

"Korttids- og langtidseffekt av ulike varmestimulus på utholdenhetsprestasjon og blodvariabler"

Dette er et spørsmål til deg om å delta i hele, eller bare deler av dette forskningsprosjekt hvor formålet er å undersøke hvordan ulike varmestimulus påvirker blodvariabler, sentrale fysiologiske variabler for utholdenhetsprestasjon og utholdenhetsprestasjon etter korttids- (10 dager) og lengre tids (5 uker) trening. Avslutningsvis vil vi også studere hvor lenge evnt. effekter av varmestimuluset på blodvariabler varer etter avslutte varmebehandling. I dette skrivet gir vi deg informasjon om målene for prosjektet og hva deltakelse vil innebære for deg.

**Formål**
Dette forskningsprosjektet består av 3 mindre delprosjekter. Delprosjekt 1 fokuserer på å optimalisere akklimatiseringen til konkurranse i varmt klima og det testes derfor prestasjon i varmt klima før og etter denne 10 dagers perioden. Anbefalingene for akklimatisering til konkurranse i varmt og fuktig klima består av én daglig økt i varmt klima på ca. 50 minutters varighet og relativt lav intensitet over en periode på ca. 10 dager. Imidlertid vet vi ikke om det å gjennomføre denne akklimatiseringsøkta i normaltemperatur med en varmedrakt som gjør at kroppen blir varm gir samme akklimatiseringseffekt som å gjøre det i et varmt klima. Å benytte varmedrakt vil være mye enklere og mindre økonomisk krevende å gjennomføre enn å benytte varmekammer. Derfor vil vi i delprosjekt 1 ha en gruppe som gjennomfører varmeakklimatiseringsøkta i et varmekammer, mens en annen gruppe gjennomfører samme økta i en varmedrakt. Delprosjekt 1 vil også inneholde en tredje gruppe som, i tillegg til å ha gjennomføre den standardiserte varmeakklimatiseringsøkta med varmedress, får et daglig passivt varmestimulus i badstue. Delprosjekt 1 varer 12 dager (test på dag 12), og går direkte over i delprosjekt 2, som varer i 3 nye uker, der de to gruppene som har trent med varmedress slås sammen til en gruppe som gjennomfører 5 ukentlige varmeøkter med varmedress, mens de som var i varmekammergruppen fra delprosjekt 1 gjennomfører 5 ukentlige varmeøkter i varmekammer. I delprosjekt 2 er fokuset på effektene av varmestimuli på blodvariabler som hemoglobinmasse og blodvolum, som er sterkt knyttet til maksimalt oksygenopptak og utholdenhetsprestasjon. Derfor er trening som kan påvirke disse blodvariablene svært relevant for utholdenhetsutøvere. Trening i varmen øker svetteproduksjonen og dermed blir det en akutt reduksjon i blodvolum, men hvordan dette påvirker ulike blodvariabler og utholdenhetsprestasjon i normaltemperatur over tid vet vi lite om og vil derfor undersøker dette i delprosjekt 2. I delprosjekt 3 ønsker vi å studere hvor lenge evnt. effekter av varmetrening på blodvariabler varer og om 3 ukentlige økter med varmedrakt påvirker hvor lenge evnt. effekter på blodvariabler av de forutgående 5 ukene med varmetrening varer.

Prosjektet er en del av en masteroppgave. Både før og etter treningsperioden gjennomfører begge gruppene testing av styrke, utholdenhet og blodvariabler (se figur 1 og 2). Videre vil blodvariabler bli målt på ulike tidspunkt underveis i treningsperioden (figur 1). All trening skal loggføres i utlevert treningsdagbok. Alle testene for den enkelte blir gjennomført på samme sted, under tilnærmet like forhold for alle forsøkspersonene og innenfor samme tidsrom på døgnet (± 1 timer) for hver person. All testing vil skje ved Høgskolen i Innlandet sitt idrettsfysiologiske testlaboratorium på Lillehammer.

**Hvem er ansvarlig for forskningsprosjektet?**
Høgskolen i Innlandet er ansvarlig for prosjektet.

**Hvorfor får du spørsmål om å delta?**
I delprosjekt 1 ønsker vi 15 syklister i hver av de 3 gruppene (varmedraktgruppen, varmekammergruppen og varmedrakt + passiv badstuegruppen). Vi håper at flesteparten vil bli med videre i delprosjekt 2 og delprosjekt 3. I delprosjekt 2 ønsker vi også en kontrollgruppe på 15 personer

som ikke har varmetrening, men bare trener som normalt og som blir med på testene før og etter den totalt 5 ukers lange varmetreningsperioden. Vi ønsker totalt 60 godt trente syklister (over 7 timer utholdenhetstrening per uke siste 6 mnd før prosjektstart, alder 17-45 år). Forespørselen sendes til aktuelle trenere og utøvere i Innlandet og Oslo regionen.

**Hva innebærer det for deg å delta?**
Hvis du velger å delta i delprosjekt 1, innebærer det at du gjennomfører én daglig varmeakklimatiseringsøkt (50 min på ca. 50 % av terskelwatten din) gjennom 10 påfølgende dager. Du deltar enten i en I) varmedraktgruppe som gjennomfører disse øktene i normal temperatur, men med en varmedrakt som gjør at kroppstemperaturen øker, II) varmekammergruppe som gjennomfører øktene i et varmt rom (ca. 37°C), eller III) varmedrakt + passiv badstuegruppen som gjennomfører øktene med en varmedrakt som gruppe I, men i tillegg får et daglig passivt varmestimulus i badstue. Utenom dette står du fritt til å gjennomføre trening i henhold til egen treningsplan. Alle gruppene testes før og etter treningsperioden på utholdenhetsvariabler i varmt klima samt blodvariabler (se figur 2). Testene er fysiologiske tester som innebærer hard fysisk anstrengelse over en periode og oppleves som ubehag som kan sammenliknes med en hard treningsøkt. Siste to dager før test bør det ikke gjennomføres hard trening. Videre deltakelse i delprosjekt 2 medfører at du må gjennomfører 3 nye uker med 5 ukentlige varmeøkter á 50 min (ca. 50 % av terskelwatten din). Du fortsetter enten i varmedressgruppen som gjennomfører disse øktene i normal temperatur, eller du fortsetter med varmeøkten i varmekammer (ca. 37°C). Utenom dette står du fritt til å gjennomføre trening i henhold til egen treningsplan. Begge gruppene testes før og etter treningsperioden på styrke- og utholdenhetsvariabler i normal temperatur, samt blodvariabler (se figur 2). Hvis du er med i kontrollgruppen i delprosjekt 2 innebærer dette at du bare er med på testingen før og etter den 5 uker lange varmetreningsperioden. I tillegg til de fysiske testene vil vi, for å studere nærmere hvordan blodvariabler påvirkes av trening i varmen, undersøke blodvariabler to ganger før treningsperioden starter og to ganger etter treningsperioden er ferdig. Blodvariablene undersøkes hovedsakelig via karbonmonoksid-rebreathingmetoden for måling av hemoglobinmasse, blodvolum og plasmavolum. Hvis du i delprosjekt 3 blir med i vedlikeholdsgruppen, må du gjennomføre 3 ukentlige varmeøkter med varmedress (tilsvarende tidligere varmeøkter) og møte til måling av blodvariabler hver 7-14. dag, mens hvis du er i kontrollgruppen trenger du bare å møte til måling av blodvariabler hver 7-14. dag.

All trening skal loggføres i utlevert treningsdagbok. Alle testresultat og treningsdata blir registrert elektronisk. Alle testene for den enkelte blir gjennomført på samme sted, under tilnærmet like forhold for alle forsøkspersonene og innenfor samme tidsrom på døgnet (± 1 timer) for hver person. All testing vil skje ved Høgskolen i Innlandet sitt idrettsfysiologiske testlaboratorium på Lillehammer.

## *Vedlegg 2 (Appendix B): Treningsdagbok (Training diary)*

| Dato | Dag | Startklokkeslett | Intensitetssone [tt:mm] | | | | | Styrke | | Øktskår | Kommentar |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | Aktivitet | I1 (<55%FTP) (<68% HF@FTP) | I2 (56-75%FTP) (69-83% HF@FTP) | I3 (76-90%FTP) (<84-94% HF@FTP) | I4 (91-105%FTP) (95-105% HF@FTP) | I5 (106-120%FTP) (>106% HF@FTP) | Type | Tid | [1-10] | |
| 13.04.2015 | mandag | 16:00 | Sykkel | 00:50 | | | | | | | 2 - Lett | Restitusjonsøkt ifbm. Varmeprosjektet |
| 14.04.2015 | tirsdag | 15:00 | Sykkel | 00:30 | 00:10 | 00:10 | 00:30 | | | | 7 - Meget hardt | 6 x 5 min I4 intervaller |
| 15.04.2015 | onsdag | 17:00 | Sykkel | 01:00 | 01:00 | | | | | | | |
| 17.04.2015 | fredag | 12:00 | Sykkel | 01:30 | 00:20 | 00:40 | | | | | 5 - Hardt | |
| 18.04.2015 | lørdag | 10:00 | Styrke | | | | | | maks | 01:00 | 4 - | Styrke i håkons hall |
| 18.04.2015 | lørdag | 16:00 | Løping | 01:00 | | | | | | | 2 - Lett | |
| | | | | | | | | | | | | |
| 14.10.2019 | mandag | | | | | | | | | | | |
| 15.10.2019 | tirsdag | | | | | | | | | | | |
| 16.10.2019 | onsdag | | | | | | | | | | | |
| 17.10.2019 | torsdag | | | | | | | | | | | |
| 18.10.2019 | fredag | | | | | | | | | | | |
| 19.10.2019 | lørdag | | | | | | | | | | | |
| 20.10.2019 | søndag | | | | | | | | | | | |
| 21.10.2019 | mandag | | | | | | | | | | | |
| 22.10.2019 | tirsdag | | | | | | | | | | | |
| 23.10.2019 | onsdag | | | | | | | | | | | |
| 24.10.2019 | torsdag | | | | | | | | | | | |
| 25.10.2019 | fredag | | | | | | | | | | | |
| 26.10.2019 | lørdag | | | | | | | | | | | |
| 27.10.2019 | søndag | | | | | | | | | | | |
| 28.10.2019 | mandag | | | | | | | | | | | |
| 29.10.2019 | tirsdag | | | | | | | | | | | |
| 30.10.2019 | onsdag | | | | | | | | | | | |
| 31.10.2019 | torsdag | | | | | | | | | | | |
| 01.11.2019 | fredag | | | | | | | | | | | |
| 02.11.2019 | lørdag | | | | | | | | | | | |

| | | | Søvn tid | Søvn kvalite | Hvilepuls | Treningsstatus | Kommentar treningsstatus | Dagsform | Stølhet | Restitusjon | Treningsvilje |
|---|---|---|---|---|---|---|---|---|---|---|---|
| eks | 20.apr | mandag | 7-8 timer | 8. Normal | 54 | Trening som planlagt | | 5. Normal | 7. | 2. | 9. Høy vilje til å trene |
| | 21.apr | tirsdag | | | | | | | | | |
| | 22.apr | onsdag | | | | | | | | | |
| | 23.apr | torsdag | | | | | | | | | |
| | 24.apr | fredag | | | | | | | | | |
| | 25.apr | lørdag | | | | | | | | | |
| | 26.apr | søndag | | | | | | | | | |
| 42 | 14.okt | mandag | | | | | | | | | |
| | 15.okt | tirsdag | | | | | | | | | |
| | 16.okt | onsdag | | | | | | | | | |
| | 17.okt | torsdag | | | | | | | | | |
| | 18.okt | fredag | | | | | | | | | |
| | 19.okt | lørdag | | | | | | | | | |
| | 20.okt | søndag | | | | | | | | | |
| 43 | 21.okt | mandag | | | | | | | | | |
| | 22.okt | tirsdag | | | | | | | | | |
| | 23.okt | onsdag | | | | | | | | | |
| | 24.okt | torsdag | | | | | | | | | |
| | 25.okt | fredag | | | | | | | | | |
| | 26.okt | lørdag | | | | | | | | | |