Stress oxydant, antioxydants et exercice

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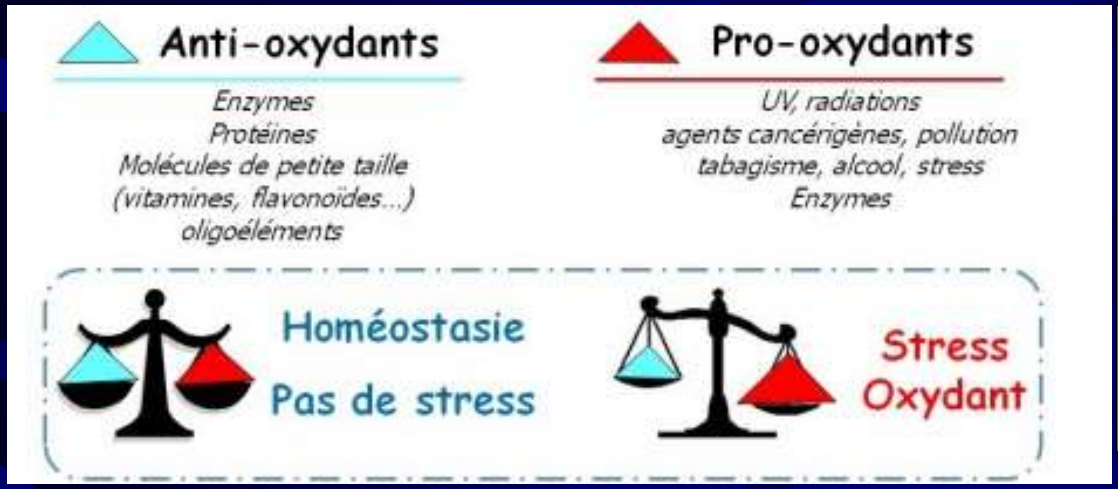
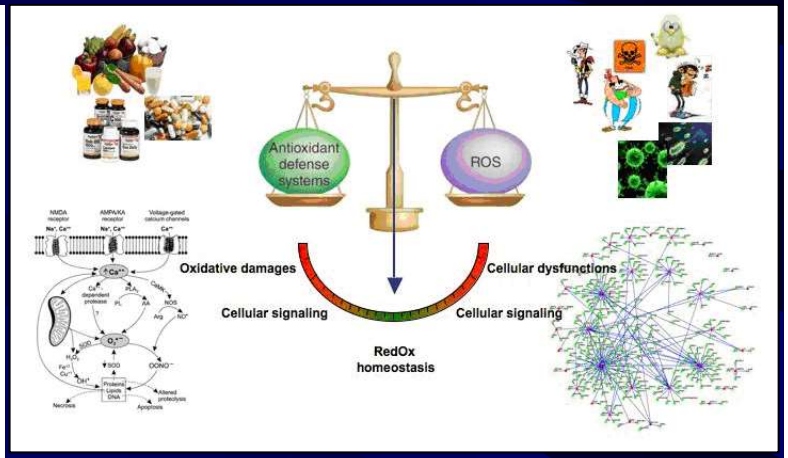
1- Données générales sur le SO

1.1- Définitions

- Le stress oxydant ou oxydatif (SO):

  Déséquilibre de la balance entre les prooxydants et les antioxydants en faveur des premiers conduisant à une perturbation du contrôle et de la signalisation redox des cellules et/ou à des dommages moléculaires (Sies et Jones 2007).



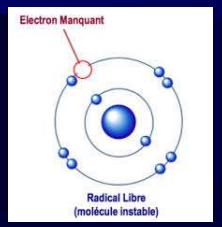


- Espèces prooxydantes: composés qui vont gagner un ou plusieurs électrons.

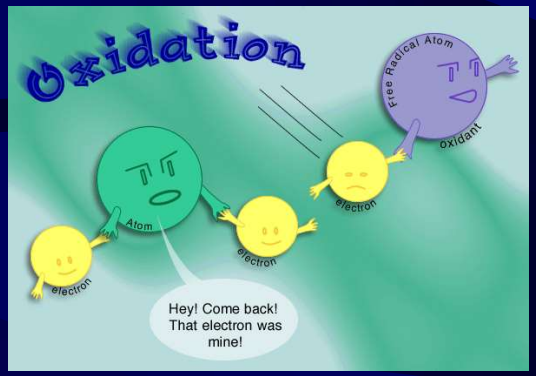
  ↳ Comprend les **radicaux libres (RL)** et des espèces non radicalaires (cf dia après)

- Les RL: Atomes ou groupements d'atomes porteurs d'un électron non apparié.

(Halliwell et Gutteridge, 1989)



↳ très réactifs, bénéfiques (faible dose), toxiques (forte dose)



↳ e- célibataire: symbolisé par un •

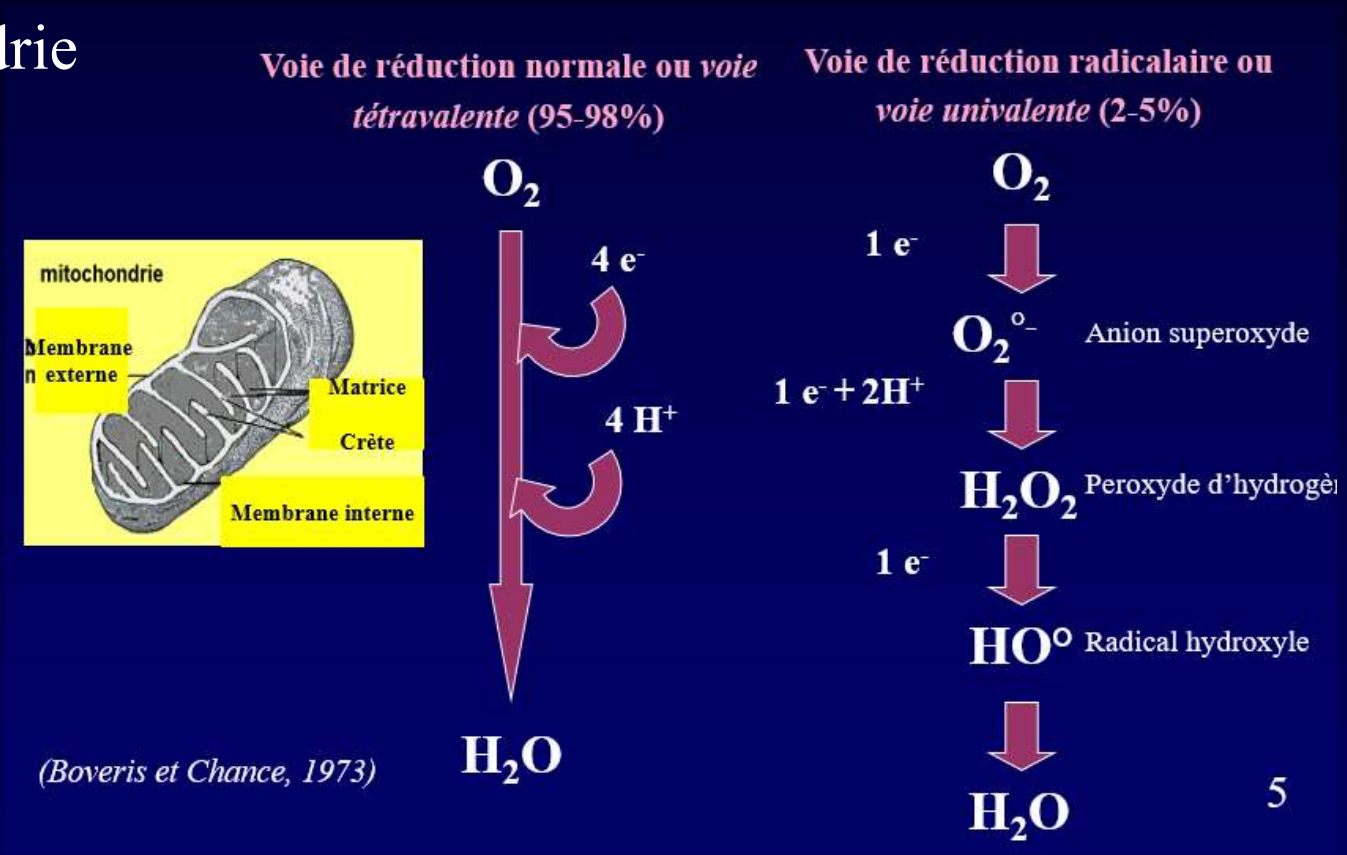
- ERON comprend les RL et composés non-radicalaires mais oxydants comme les RL (H_2O_2, NOO-)

ERON (pro-oxydants)

ERO		ERN	
$O_2^{-\circ}$	Anion superoxyde	$^\circ NO$	Monoxyde d'azote
H_2O_2	Peroxyde d'hydrogène	NOO^-	Peroxynitrite
HO°	Radical hydroxyle		
RO°	Radical alcoxyle		
ROO	Radical Peroxyle		
$^1O_2$	Oxygène singulet		

1.2 - Production des ERON au repos

- La Mitochondrie



Production de ROS revue à la baisse par études + récentes

These results do not support the idea that mitochondria produce considerable amounts of reactive oxygen species under physiological conditions. Our upper estimate of the proportion of electron flow giving rise to hydrogen peroxide with palmitoyl carnitine as substrate (0.15%) is more than an order of magnitude lower than commonly cited values. We observed no difference in the

(St Pierre et coll. 2002)

P° $O_2^{\circ-}$ que de **0.15%** transformé en H_2O_2 et qui atteint le cytosol

1.2 - Production des ERON au repos

- Activation de certaines enzymes (Xanthine oxydase [XO], $NADPH_{oxydase}$, découplage de la NOS) = P° majoritaire *(Powers et al. 2008)*

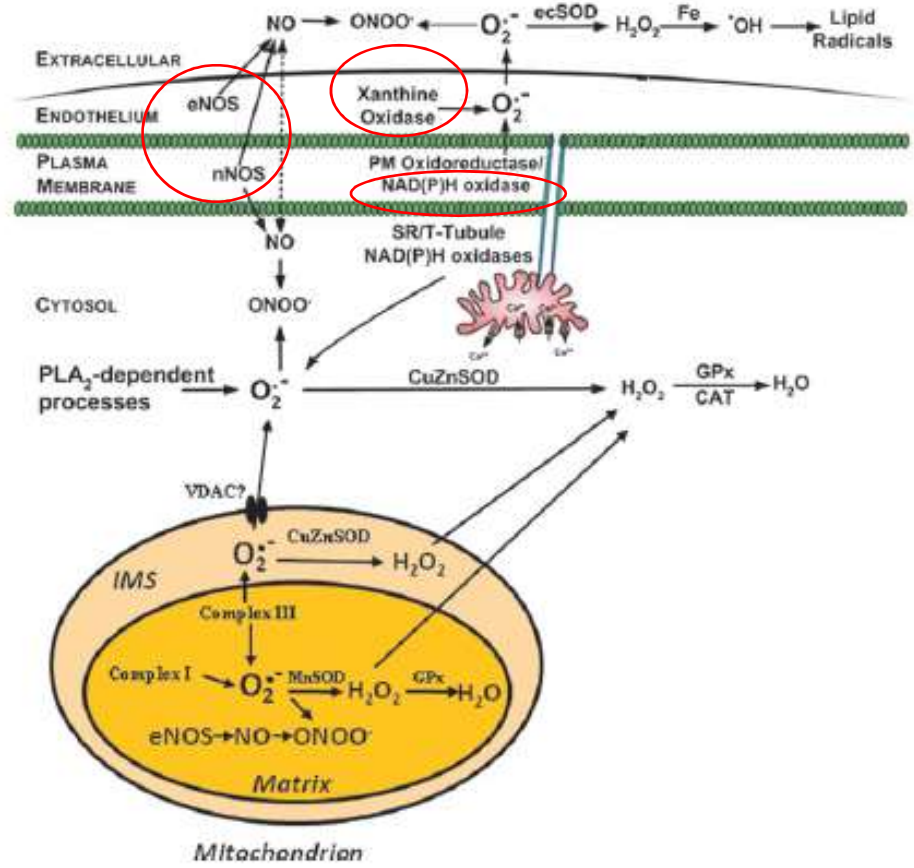


FIG. 1. Schematic representation of the sites and mechanisms proposed for ROS and NO generation in skeletal muscle fibers. Scheme updated from Powers and Jackson (60) to incorporate new data on the potential for hydrogen peroxide release from fibers to the extracellular space (see text for details) and on the role of CuZnSOD in the mitochondrial intermembrane space (IMS). (To see this illustration in color the reader is referred to the web version of this article at www.liebertonline.com/ars).

Jackson 2011

1.3- Actions / effets des ERON

- Effets négatifs: A forte dose attaque des constituants cellulaires.

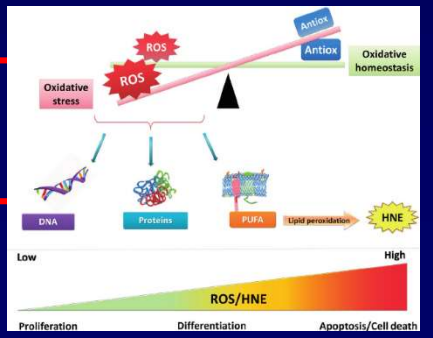
ERON

Lipides → **Peroxydation lipidique** → Altération des mbs *(MDA, IsoP)*

Protéines → Inactivations *dérivés carbonylés*

ADN, ARN → Mutations *8-OHdG, 8-OHG*

DOMMAGES OXYDATIFS
(membranes, protéines, noyaux…)



1.3- Actions / effets des ERON

- Effets bénéfiques:
  - A forte dose:
    - Rôle important dans la défense de l'organisme (réponse inflammatoire)

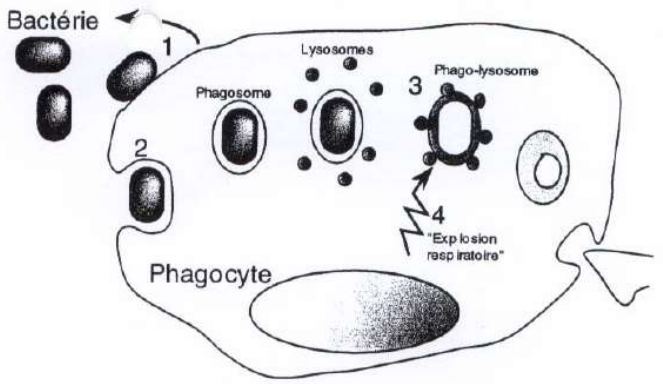
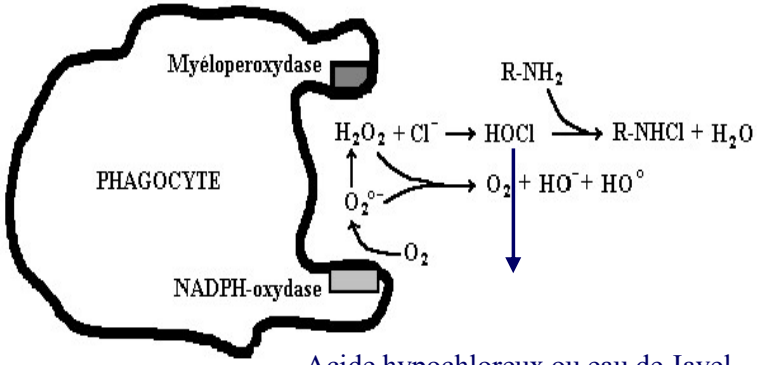


Figure 1. Production de radicaux libres lors de la phagocytose d'une bactérie.

Légende : 1. Chimiotactisme et adhésion. 2. Ingestion. 3. Fusion phagosome-phagolysosome. 4. L' « explosion respiratoire » génère dans le phagolysosome des oxydants bactéricides (H_2O_2, $O_2^{-\circ}$, HO°).



Acide hypochloreux ou eau de Javel

Les neutrophiles, éosinophines, monocytes/macrophages assurent la phagocytose et la destruction des micro-organismes étrangers en s'activant (↗ x 200-400% de leur VO_2).

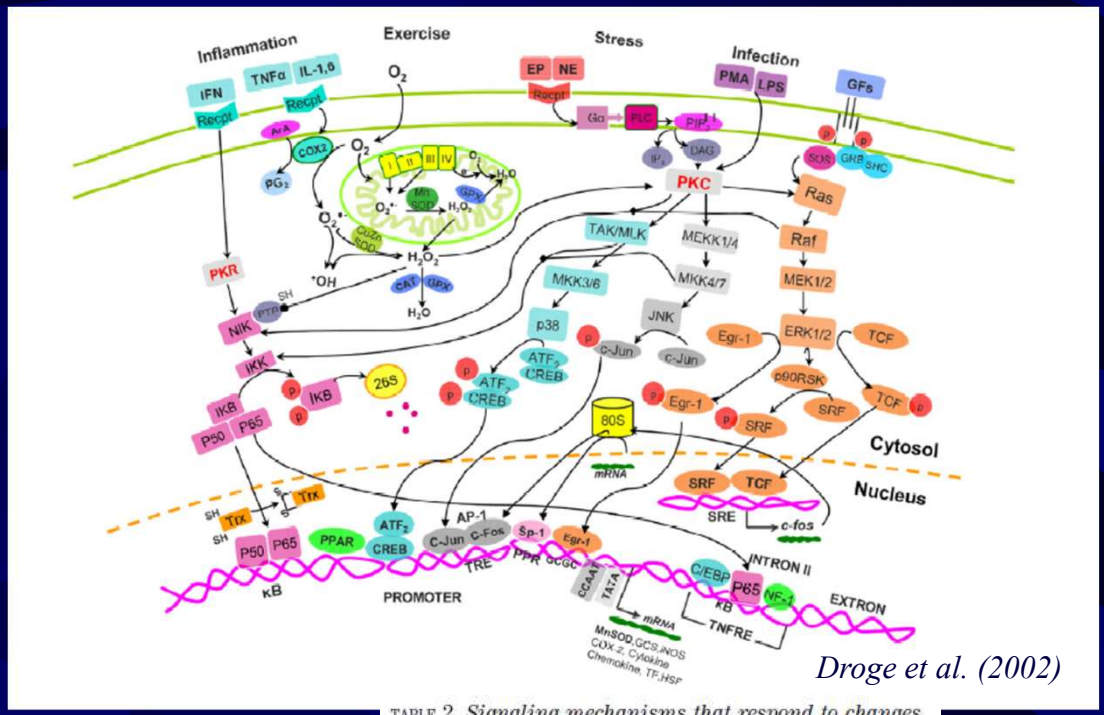
→ « respiratory burst via NADPHoxydase/ MPO»

↳ Les ERON produits détruisent les microorganismes

Knight et al. (2000)

1.3- Actions / effets des ERON

- Effets bénéfiques:
  - Faible dose : signalisation cellulaire
    - ✓ Les ERON peuvent agir en tant que « molécule signal » et intervenir dans la communication intra et intercellulaire. Ils participent à l'expression de certains gènes et à leur régulation.



Droge et al. (2002)

TABLE 2. *Signaling mechanisms that respond to changes in the thiol/disulfide redox state*

Example	Reference No.	Section
AP-1 transcription factor in human T cells	192	vH
NF-κB transcription factor in human T cells	192	vI
yAP-1 transcription factor in *S. cerevisiae*	323	iiiB3
Control of K^+ channel activity in the carotid body	9	ivC
Human insulin receptor kinase activity	494	vC
Bacterial OxyR	28	iiiB2
Protein tyrosine phosphatases	43	vB
Src family kinases	248	vD
JNK and p38 MAPK signaling pathways	248	vE
Amplification of immunologic functions	248	ivF
Signaling in replicative senescence	536	viD

AP-1, activator protein 1; NF-κB, nuclear factor κB; JNK, c-Jun NH_2-terminal kinase; MAPK, mitogen-activated protein kinase.

Voies de signalisation impliquées lors de l'exercice:
-Biogénèse mitochondriale,
-Insulinosensibilité…

☞ Activation des voies directement liées au statut redox de la cellule qui est régit par les ERON

1.3- Actions / effets des ERON

- Effets bénéfiques:
  - Faible dose:
    - ✓ Production de force

Un état légèrement oxydé est nécessaire pour produire une Fmax

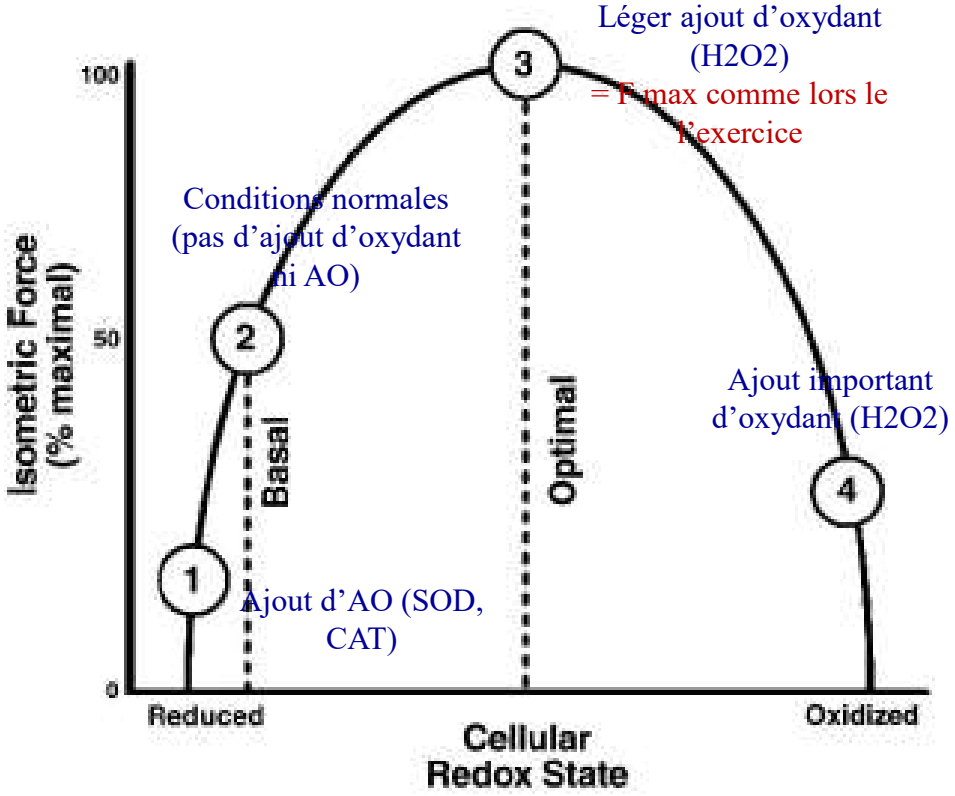
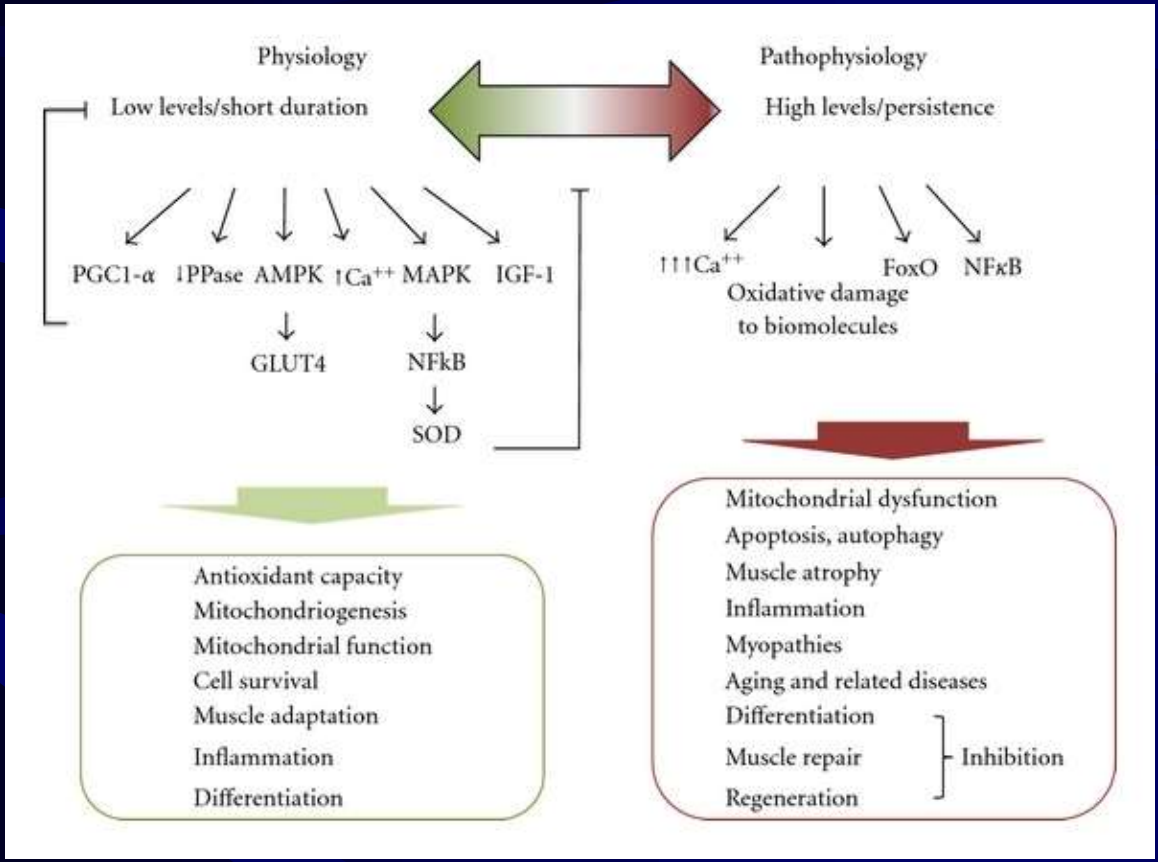


FIG. 5. A theoretical model proposed by Reid et al. (322) that describes the biphasic effect of ROS on skeletal muscle force production. *Point 1* represents the force production by unfatigued muscle exposed to antioxidants or a reducing agent. *Point 2* illustrates the force generated by muscle in its basal state (i.e., no antioxidants or oxidants added). *Point 3* illustrates the force produced by unfatigued skeletal muscle exposed to low levels of oxidants; this represents the optimal redox state for force production. *Point 4* illustrates the deleterious effects of excessive ROS on skeletal muscle force. [Redrawn from Reid (317).]

Reid et al. (1993)

1.3- Actions / effets des ERON

Résumé Actions / effets des ERON



Barbieri et al. (2012)

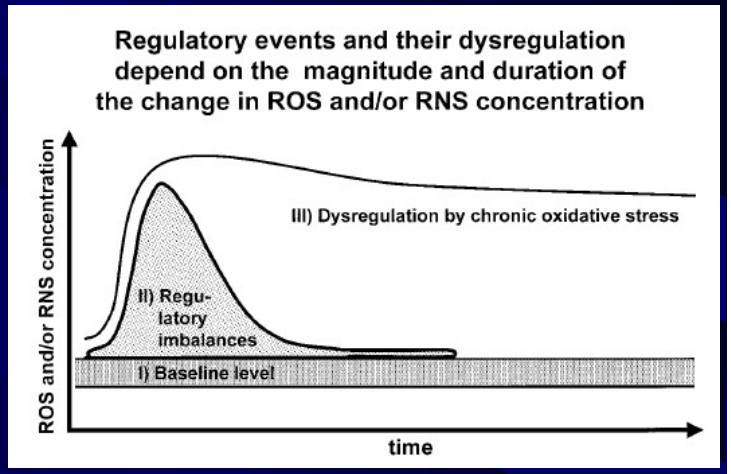


FIG. 3. Regulatory events and their dysregulation depend on the magnitude and duration of the change in ROS or reactive nitrogen species (RNS) concentration. ROS and RNS normally occur in living tissues at relatively low steady-state levels. The regulated increase in superoxide or nitric oxide production leads to a temporary imbalance that forms the basis of redox regulation. The persistent production of abnormally large amounts of ROS or RNS, however, may lead to persistent changes in signal transduction and gene expression, which, in turn, may give rise to pathological conditions.

Droge et al. (2002)

1.4- Système de défense: les antioxydants

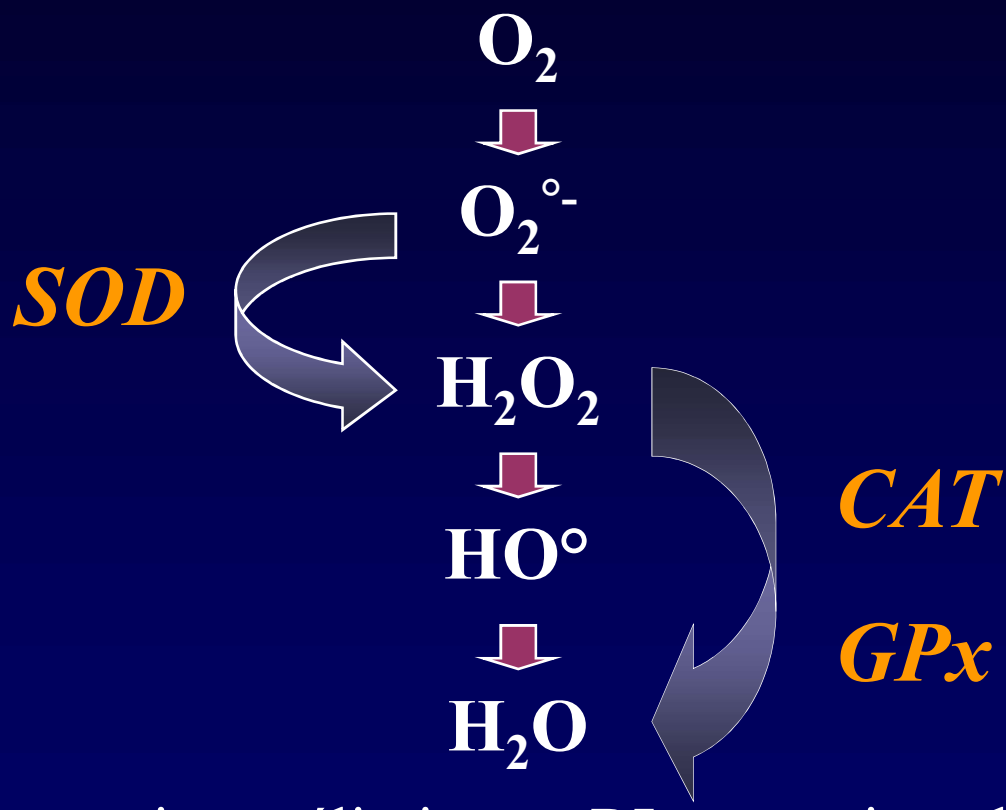
Prooxidant Jail

R•, RO•, ROO•, $^1O_2$, O^-_2, -OH, H_2O_2, Cu, Fe

R•, RO•, ROO•, O^-_2 ▪ $^1O_2$, -OH, H_2O_2,Cu, Fe

1.4- Système de défense: les antioxydants

- Système enzymatique: élimine de nombreux RL par enzyme

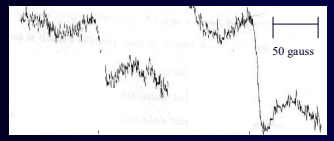


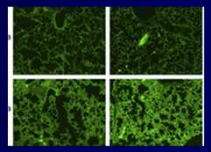
- Système non enzymatique: élimine un RL par antioxydant
  - -Vitamine E, C, β-carotène = alimentaires
  - -Glutathion, acide urique, acide lipoïque, bilirubine, Coenzyme Q10 = souvent sous-produits du métabolisme

1.5- Mise en évidence du SO

On regarde les ≠ aspects de la balance

Par résonance paramagnétique électronique (RPE) + Chimioluminescence





Prooxydant

($O_2^{\circ-}$, H_2O_2, °OH)

nocif pour l'organisme

Antioxydant

défense de l'organisme

- ↗ de l'activité des enzymes AO.
- ↘ des antioxydants (vit E, C, GSH).
- ↘ rapport glutathion réduit (GSH) / oxydé (GSSG)

Dommages oxydatifs

- ↗ Marqueurs de la peroxydation lipidique: MDA (méthode des TBARs), IsoP +++
- ↗ Marqueurs de l'oxydation des protéines: protéines carbonylées ou AOPP.
- ↗ Marqueurs de l'oxydation des acides nucléiques: (*8-OHdG, 8-OHG)*.

1.5- Mise en évidence du SO

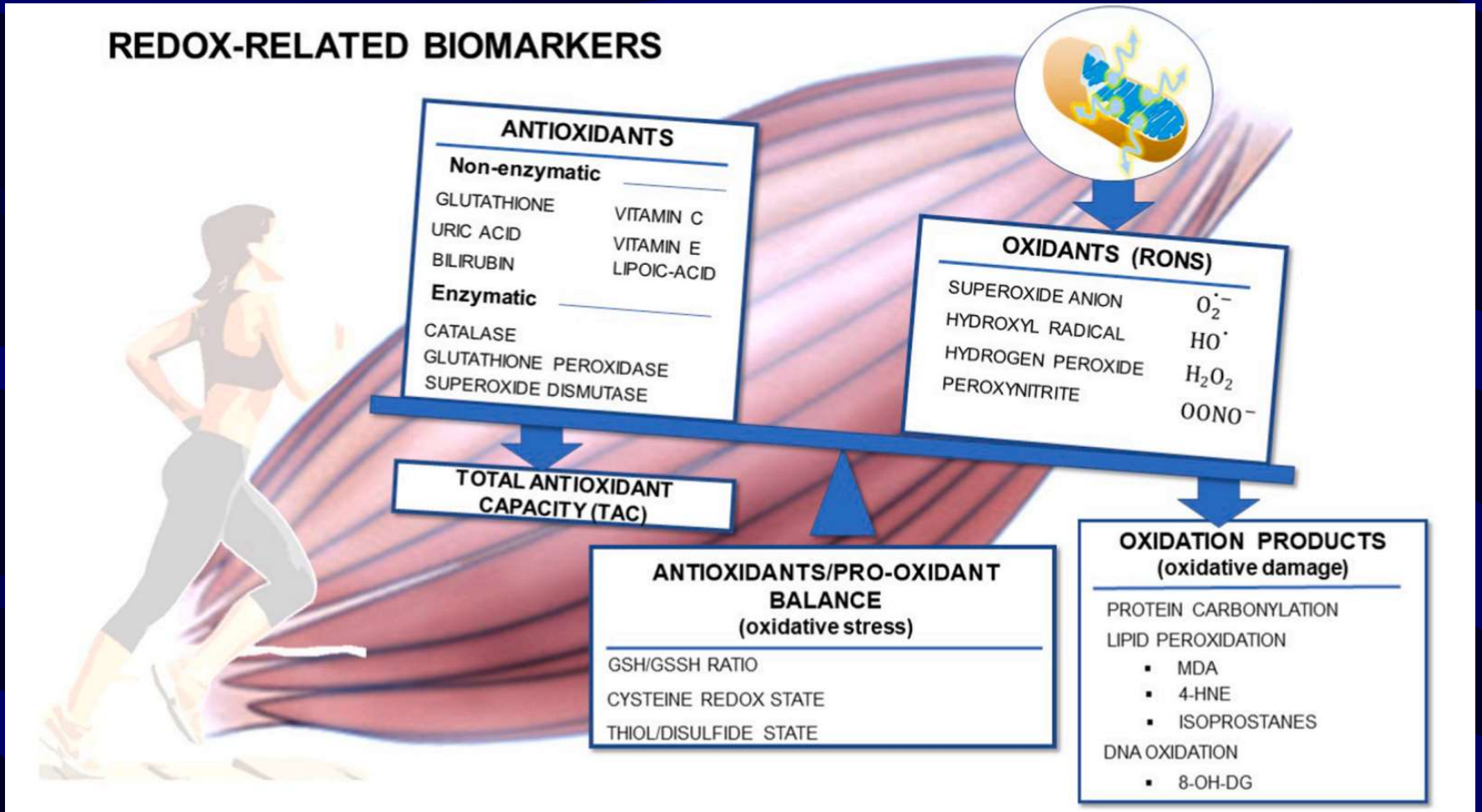


Fig. 2. Categories of redox biomarkers in exercise research.

Gomez-Cabrera et al. (2021)

1.6- Conséquences à long terme

LDLox : prédicteur des MCV

ERON impliquées dans pb vasomotricité

Vieillissement

Harman et al. 1956
Golden et al. 2002

Les ERON impliquées dans de nombreuses maladies et/ou à leurs complications associées

D'où supplémentation antiox dans études épidémiologiques

Conclusion

L'O_2 a donc des effets paradoxaux car il est à la fois indispensable au maintien de la vie et il est également responsable de la production de ERO qui a forte dose entraînent de graves dommages oxydatifs au niveau de l'organisme.

Les antioxydants luttent contre leurs effets néfastes

ATTENTION: à faible dose ERON = utiles car indispensables au bon fonctionnement physiologique de l'organisme (nécessaires pour les voies de signalisation cellulaire)

☹ Élimination complète par exes d'AO = ERREUR (cf partie 3)

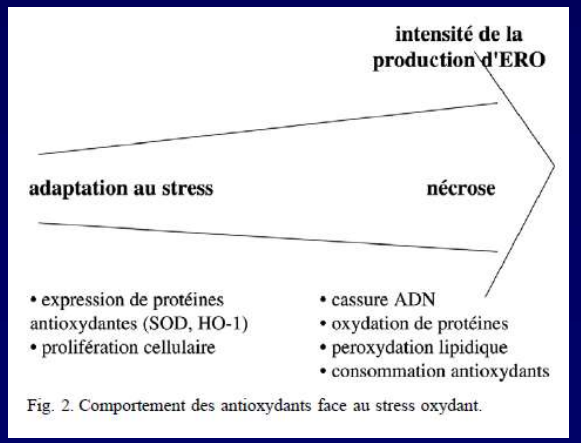


Fig. 2. Comportement des antioxydants face au stress oxydant.

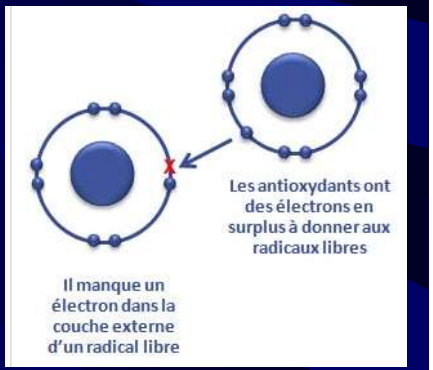
2- Les antioxydants (AO)

Introduction - Généralités

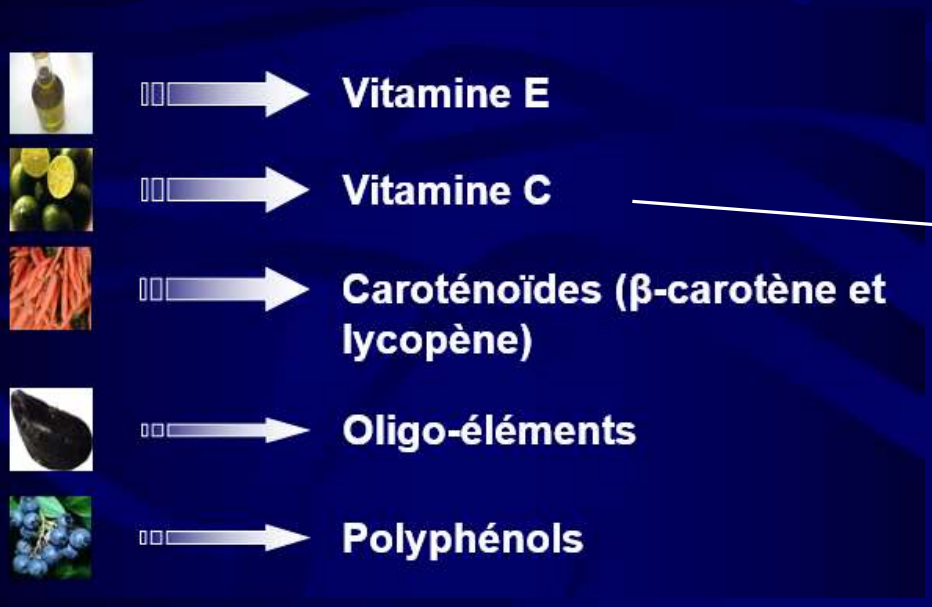
- Définition:

  Substance qui inhibe ou retarde significativement l'oxydation d'un substrat, alors qu'elle présente une concentration très faible dans le milieu où elle intervient *[Halliwell et Gutteridge 1990]*.

  → Un AO est un REDUCTEUR (réagit avec un oxydant)



- Antioxydants alimentaires les plus connus :



Argument marketing

Introduction - Généralités

- Aliments les plus antioxydants (fruits):

argousier

Mangoustan

Cannebege

1 pastèque

Goyave

Papaye

Pouvoir antioxydant (unités Orac/100 g)	
Argousier 'Argalap 700'	22 000
Mangoustan (entier)	**200 X supérieurs au Thé Vert**
Canneberges	9584
Myrtilles	2400
Mûres	2036
Pruneaux	1800
Fraises	1540
Framboises	1220
Prunes	949
Orange	750
Raisin noir	739
Cerises	670
Kiwi	602
Pamplemousse rose	483
Tomate	

LIMITE : Non prise en compte de la biodisponibilité (ex polyphénols du vin rouge)

Introduction - Généralités

- Aliments les plus antioxydants (légumes):



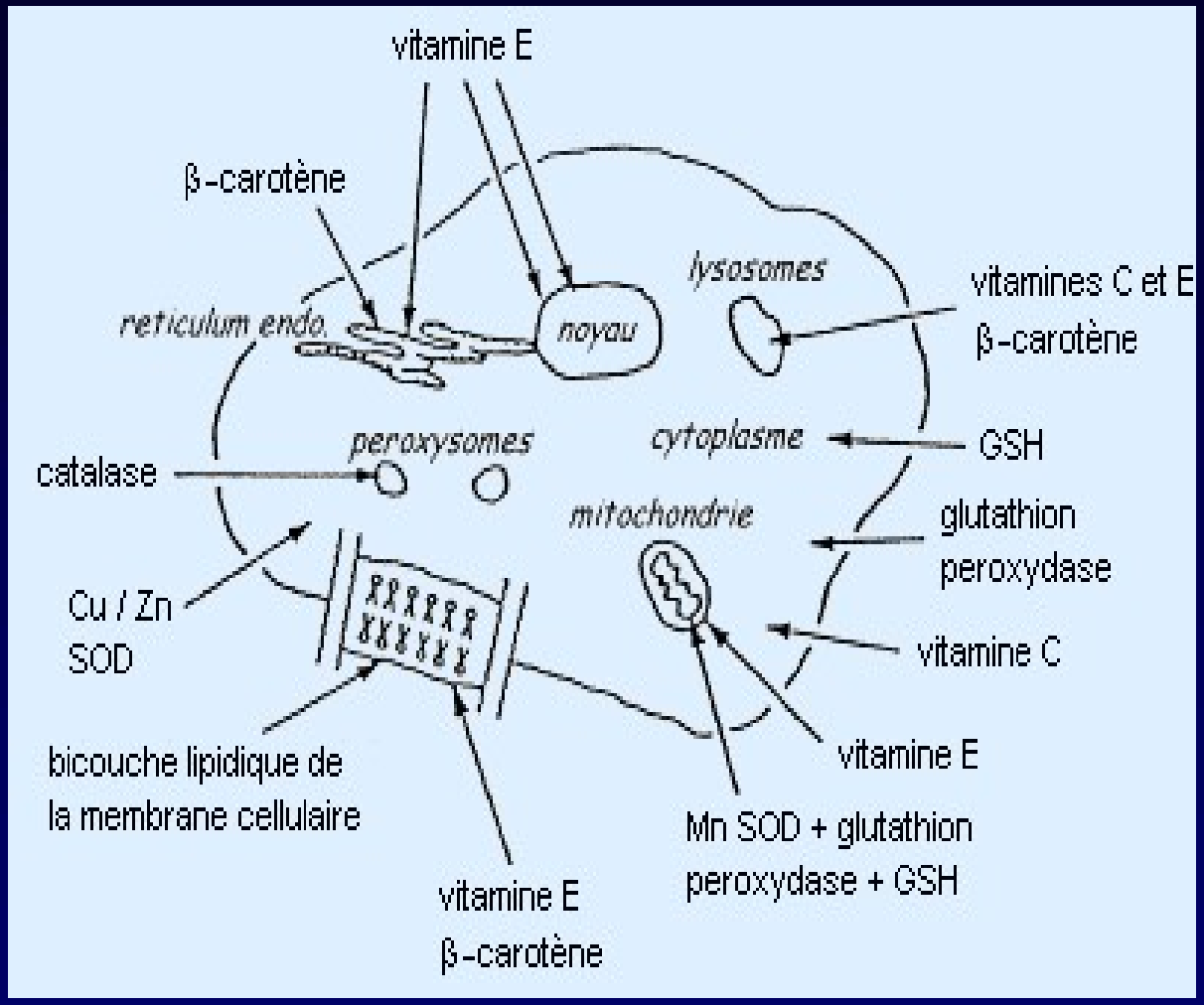







Riches en vitamine C, caroténoïdes (dont le lycopène), polyphénols (flavonoïdes)…

2.1- Localisation cellulaire des antioxydants



Liposolubles:

☞ membranes

Vit E,
caroténoïdes
…

Hydrosolubles:

☞ Fluides IC ou EC

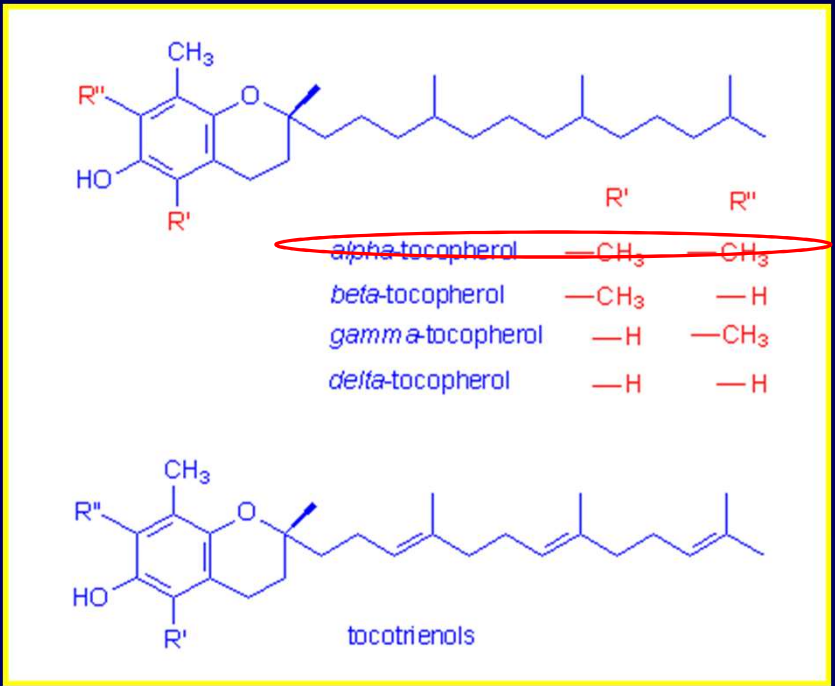
Vit C, glutathion réduit (GSH), enzymes

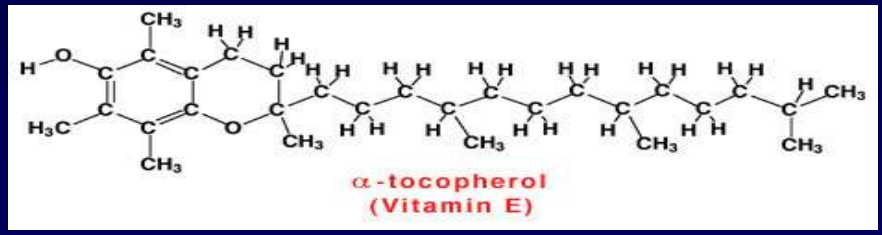
Bonne protection cellulaire / complémentarité

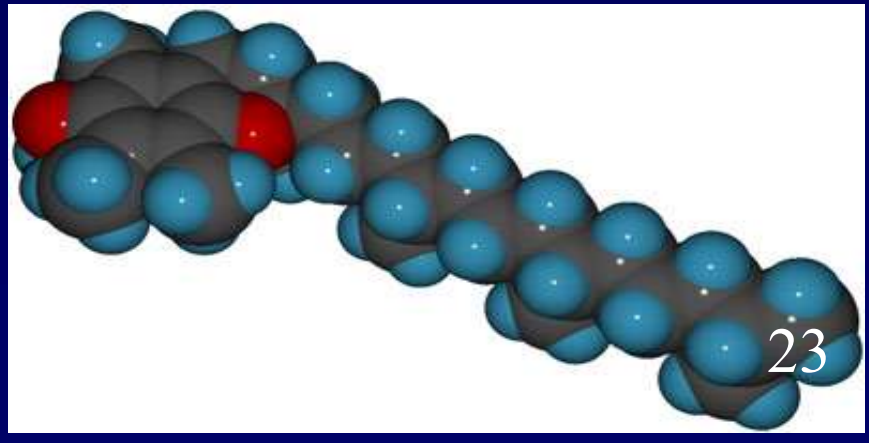
2.2- Les différents antioxydants

2.2.1- La vitamine E

- Vitamine E se réfère à tous les dérivés tocophérols (chaîne saturée) et tocotriénols (chaîne insaturée) ayant l'activité biologique de l'α-tocophérol
- La forme α-tocophérol est la + fréquemment retrouvée dans la nature





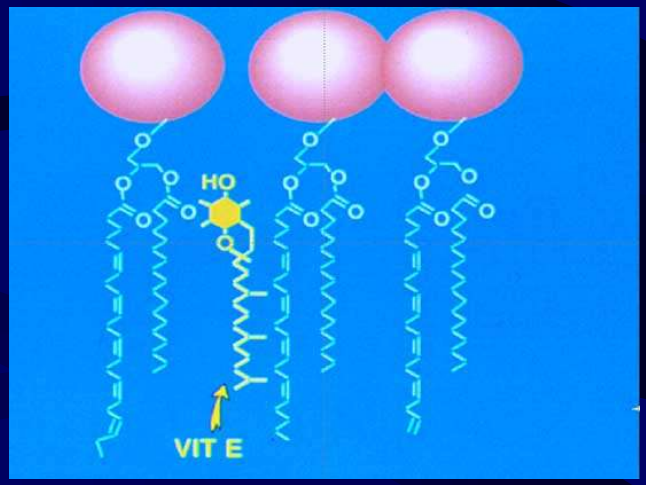


➢ Caractéristiques, localisation et actions :

Liposoluble

Antioxydant majeur des membranes cellulaires

Sensible à la lumière, oxygène, chaleur et raffinage (huiles)



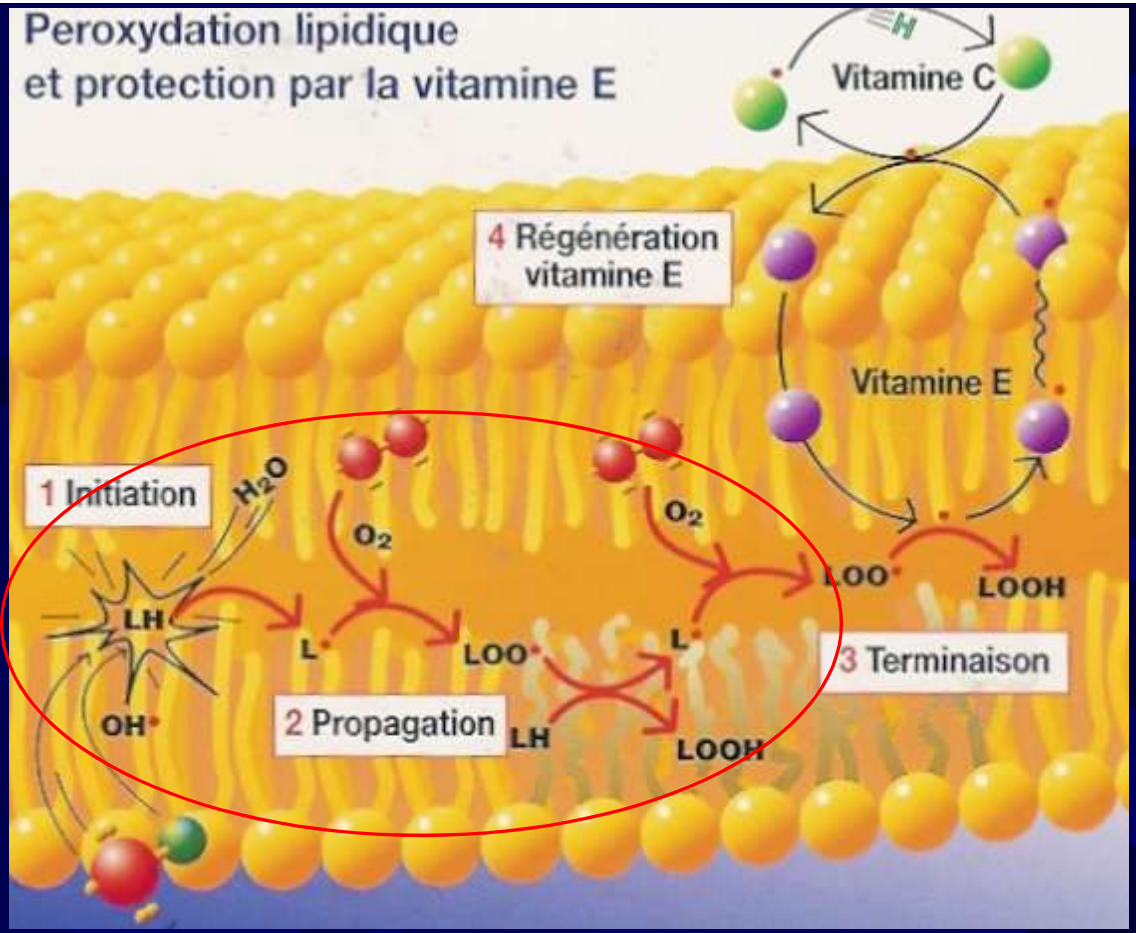
Action envers:

HO°, ROO°, RO°, $^1O_2$,

Action antioxydante par le H du groupement OH

Piège radicaux lipidiques (RO° et ROO°) et donc stoppe la peroxydation lipidique

Formation radical tocophéroxyle



Stoppe phase initiation et propagation de peroxydation lipidique

Initiation

$$RH + {}^\circ OH \xrightarrow{k1} R^\circ + H_2O$$

$$\text{Vit E-OH} + {}^\circ OH \xrightarrow{k2} \text{Vit E-O}^\circ + H_2O$$

Propagation

$$R^\circ + O_2 \xrightarrow{k1} ROO^\circ$$

$$ROO^\circ + R'H \xrightarrow{k1} ROOH + R'^\circ$$

$$\text{Vit E-OH} + ROO^\circ \xrightarrow{k2} \text{Vit E-O}^\circ + ROOH$$

Vit E = bon antioxydant car k2 >> k1 (réagit + vite avec l'oxydant que oxydant sur lipides)

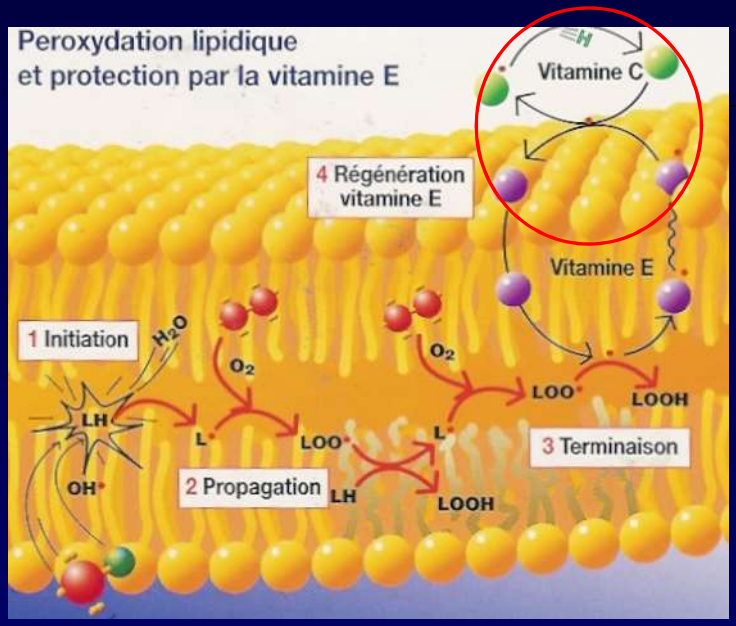
➢ Terminaison et régénération vit E

VitE-O° + réducteur (Vit C ou GSH) ⟶ **VitE-OH**

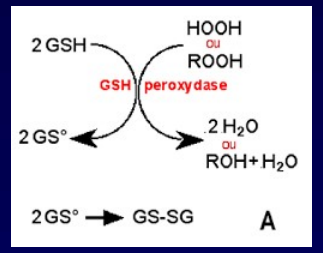
VitE-O° + VitE-O° ⟶ produit non réactif

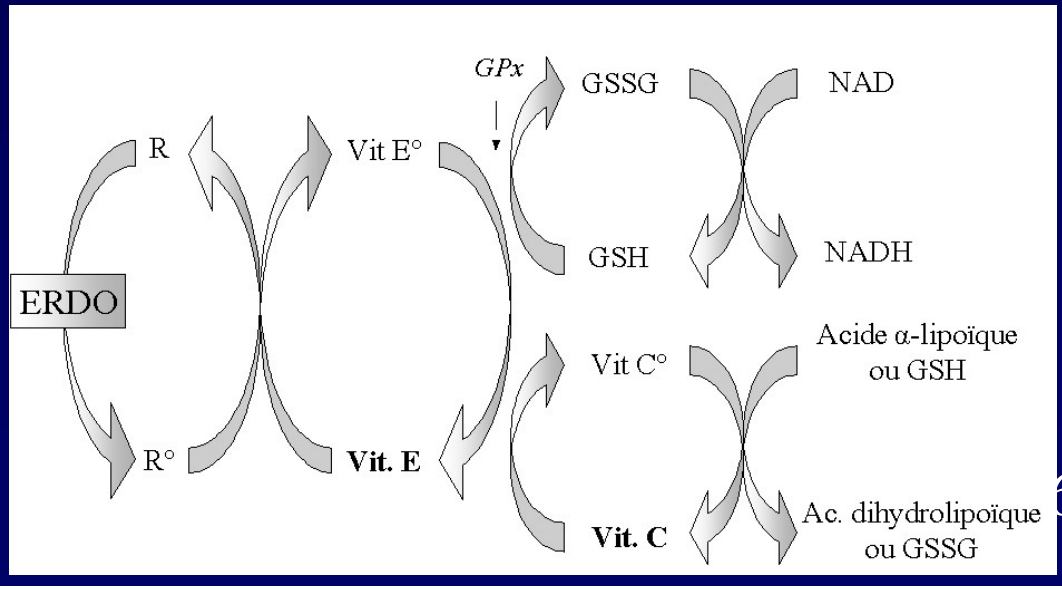
VitE-O° + RO° ⟶ produit non réactif

VitE-O° + ROO° ⟶ produit non réactif

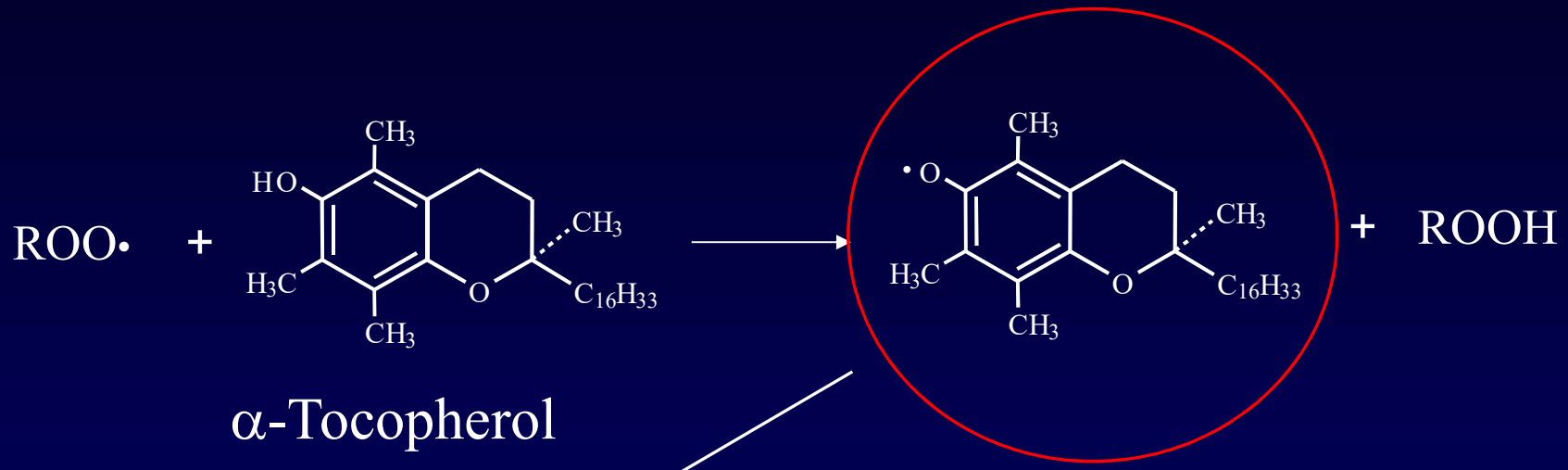


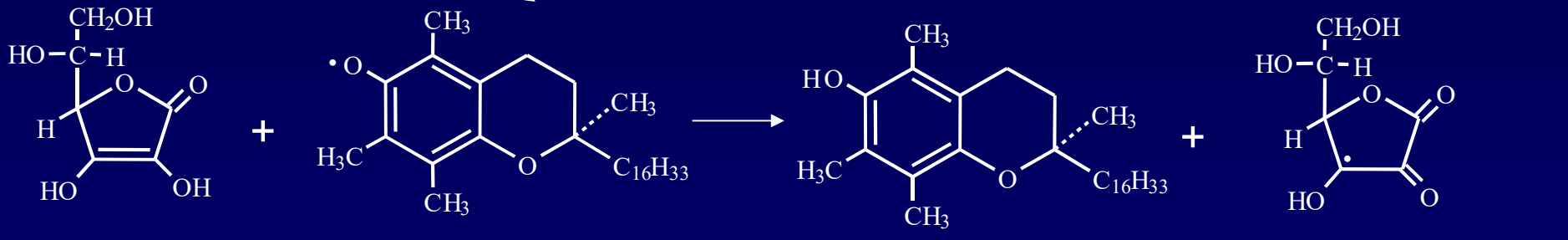
Le glutathion réduit (GSH), en présence d'ERO s'oxyde (GSSG)





Action synergique de l' α-Tocopherol et de l'acide ascorbique





Ascorbic Acid

α-Tocopherol

Ascorbic radical

➢Apports nutritionnels conseillés (ANC 2001):

- Sédentaires : 12 mg/j (revu à ≈ 10 mg/j)

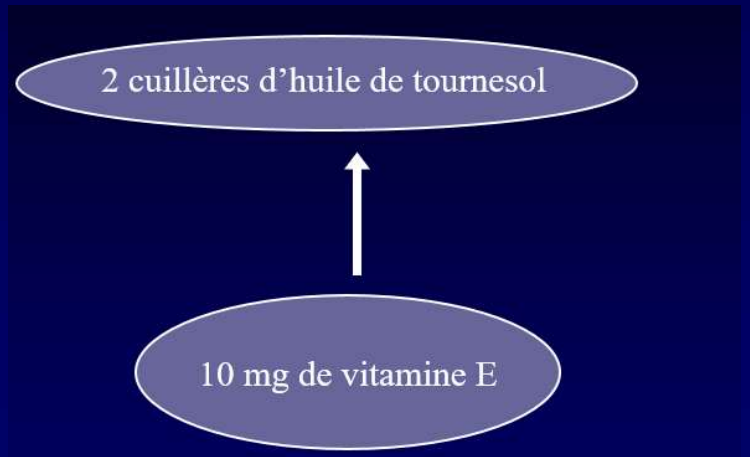
- Sportifs: + 12 mg/j par tranche de 1000 kcal au dessus
  - ✓de 1800 kcal/j chez ♀
  - ✓de 2200 kcal/j chez ♂.

- Limite max: 50 mg/j.

- Sources:
  - Huiles végétales
  - Margarines
  - Fruits oléagineux
  - Beurre
  - Poissons
  - Légumes verts

Aliments riches en vitamine E (en mg/100g)

Huile de germe de blé	150 - 500
Huile de soja	144
huile de tournesol	57
Margarine	43
Huile de colza	25
Huile d'arachide	15 - 30
Huile d'olive	15 - 20
Beurre	10
Jaune d'oeuf	3



Enquêtes sur la consommation des français:

Plus de 50 % des français ne consomment pas les ANC (étude SUVIMAX)

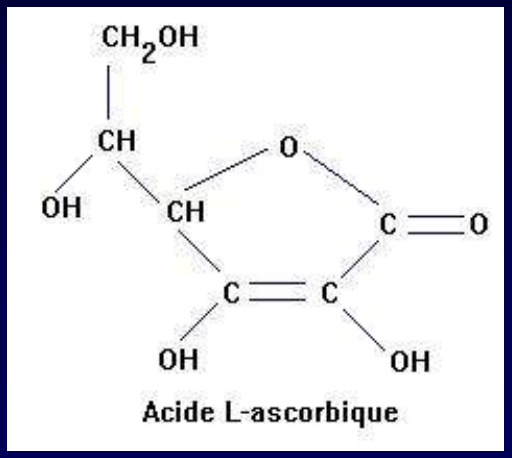
RISQUES POUR LA SANTE

En dessous de 7 mg/j = ↗ risque de maladie coronariennes et ↗ risque cancer du sein

Etude MONICA (OMS): corrélation [Vit E]pl et mortalité coronarienne (Gey et al. 1998)

2.2.2- La vitamine C

- Définition: composés ayant l'activité biologique de l'acide ascorbique.
- Caractéristiques et actions:
  - Hydrosoluble
  - Antioxydant direct: piège RL dans milieu aqueux,



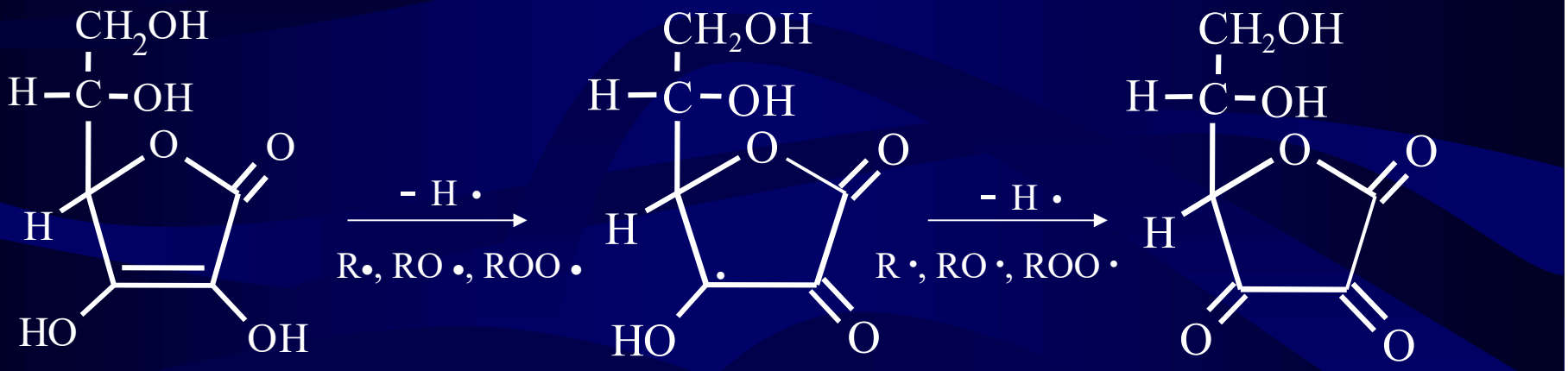
Acide L-ascorbique

Action envers:

HO°, $^{1}O_2$, $O_2^{\circ-}$ → Acide déhydroascorbique

- Antioxydant indirect: régénère Vitamine E. → Radical ascorbyle

2.2.2- La vitamine C

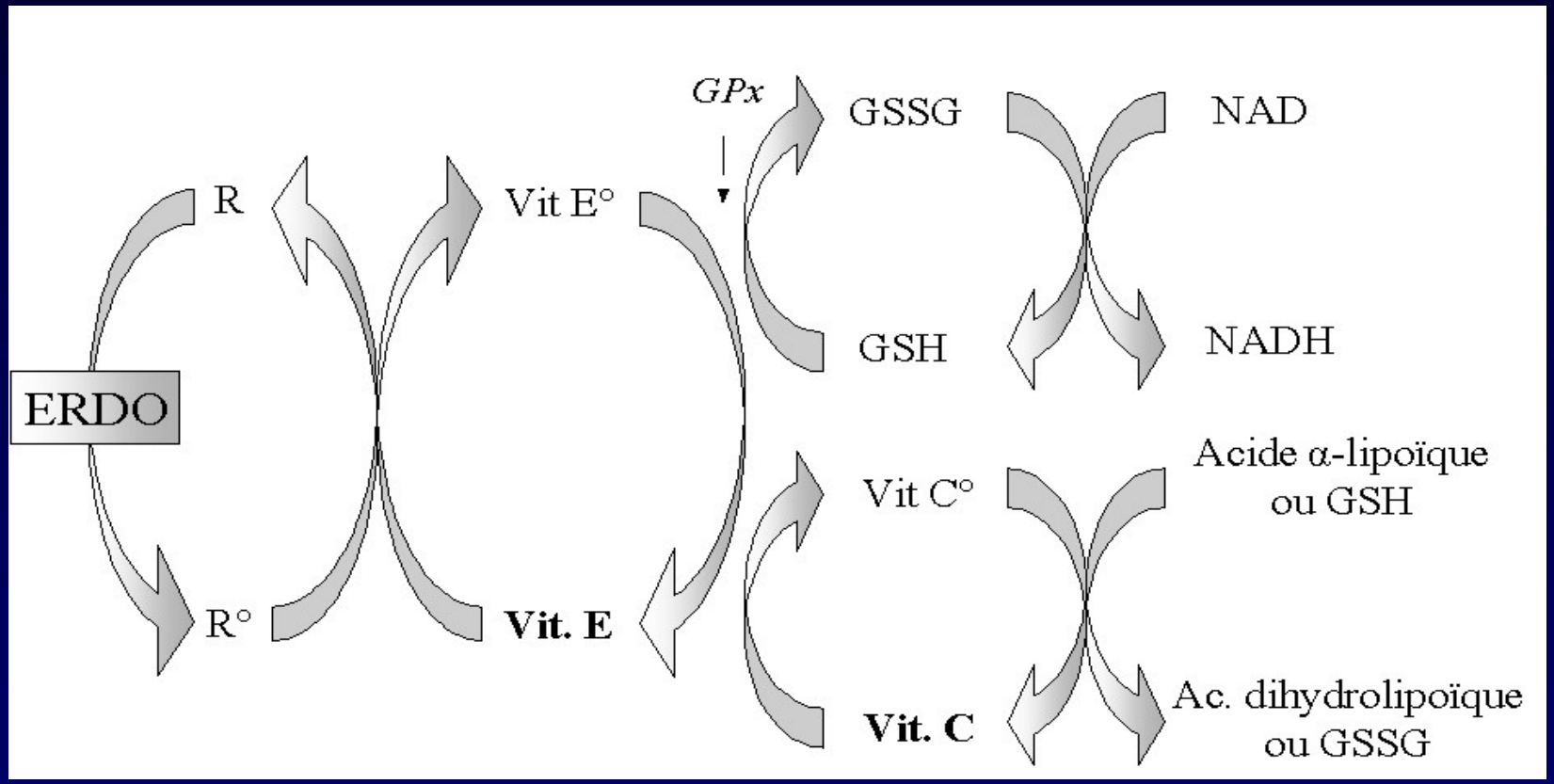


Acide Ascorbique — Radical ascorbyle — Acide Dehydroascorbique

L'interconversion réversible entre l'a. ascorbique et le l'a. déhydroascorbique passe par la formation d'un composé intermédiaire mono-oxydé instable, le **radical ascorbyl** (Asc')

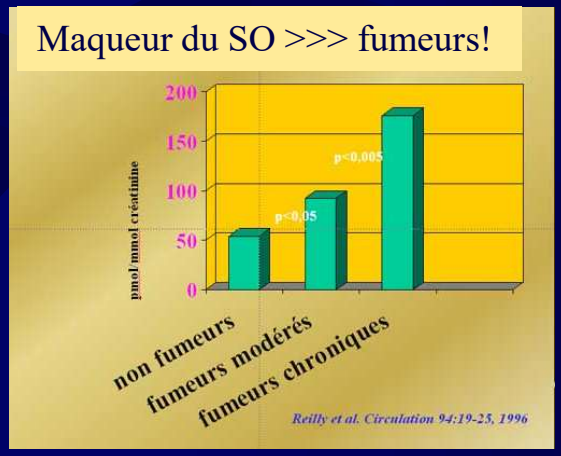
2.2.2- La vitamine C

- Régénération: par réducteurs: GSH ou acide alpha lipoïque



Rapport vit C/ Vit E = ou > 1.3 *(Gey et al. 1998- MONICA)*

- Apports nutritionnels conseillés (ANC 2001):
  - Sédentaires : 110 mg/j
  - Sportifs: + 100 mg/j par tranche de 1000 kcal au dessus de 1800 kcal/j chez ♀et 2200 kcal/j chez ♂
  - Limite max: 600 mg/j
  - Apport > fumeur.



⇒ Apport quotidien car pas de stockage

- **Sources:** très répandue dans la nature (quantité variable dans tous les végétaux, foie, lait viande)
  - Cassis 200 mg/100g
  - Kiwi 94 mg
  - Agrumes, fraises 50-60 mg
  - Choux fleur, choux 60 mg
  - Foie, rognon 20-30 mg

La + fragile,
(sensible chaleur, lumière et oxydation)

Perte lors cuisson (-50%), réchauffage...

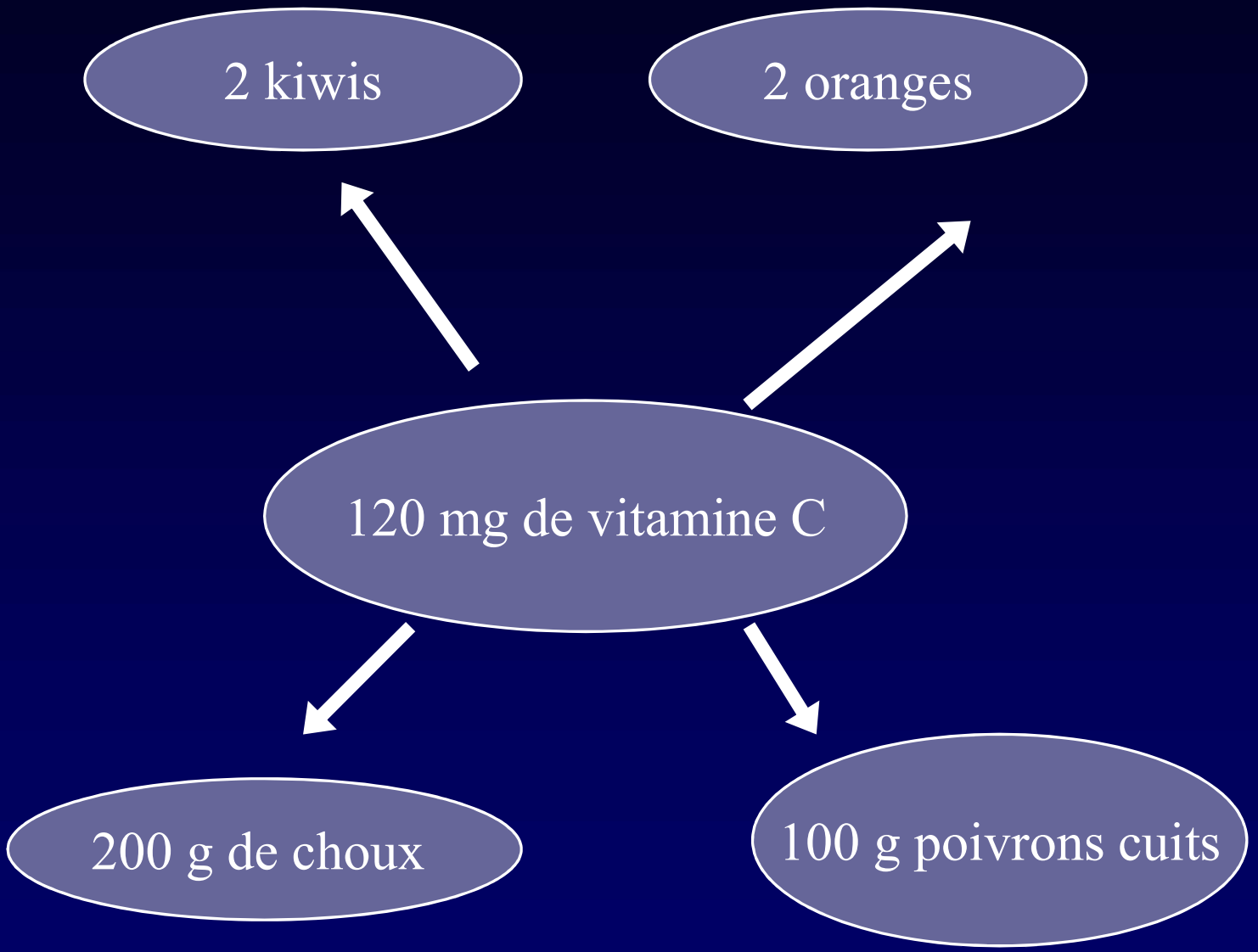
Consommer fruits et légumes crus











2 kiwis
2 oranges
120 mg de vitamine C
200 g de choux
100 g poivrons cuits

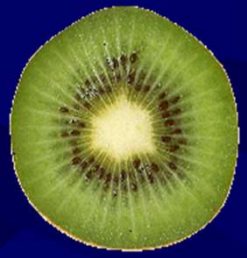
Contenu en vitamine C de certains aliments (mg/portion d'aliments crus)

125 g de cassis	163 à 175
150 g de kiwi	120 à 300
200 g de fraises et de pamplemousse	60 à 140
100 g de Choux, choux-fleurs	50 à 70
150 g d'agrumes (orange, mandarine…)	45 à 105
100 g de légumes de couleur verte (petits pois, poireaux...)	30 à 50
120 g de foie, rognons	8,4 à 54
100 g d'autres légumes et pomme de terre (la pomme de terre étant la plus riche)	5 à 40
1 c. à café de persil	5
100 ml de lait de femme	3 à 6

- Enquêtes sur la consommation des français:
  - Apports difficiles à évaluer exactement à cause des pertes.
  - Plus de la moitié des français ont une consommation inférieure aux ANC (SUVIMAX)

☹ Si [Vit C]pl < 6 mg/dL → ↗ MCV ou cancer (Gey et al. 1998 MONICA)









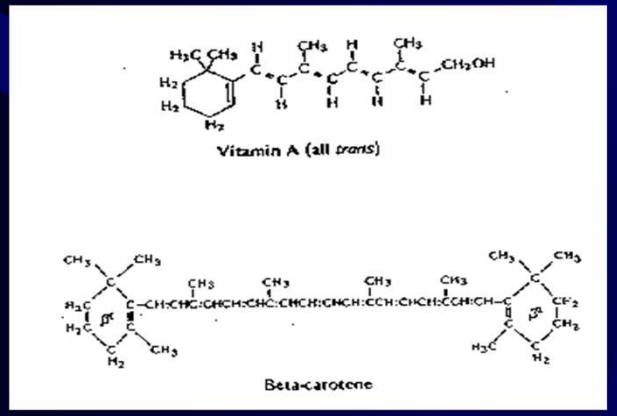

2.2.3- Les caroténoïdes: le β-carotène

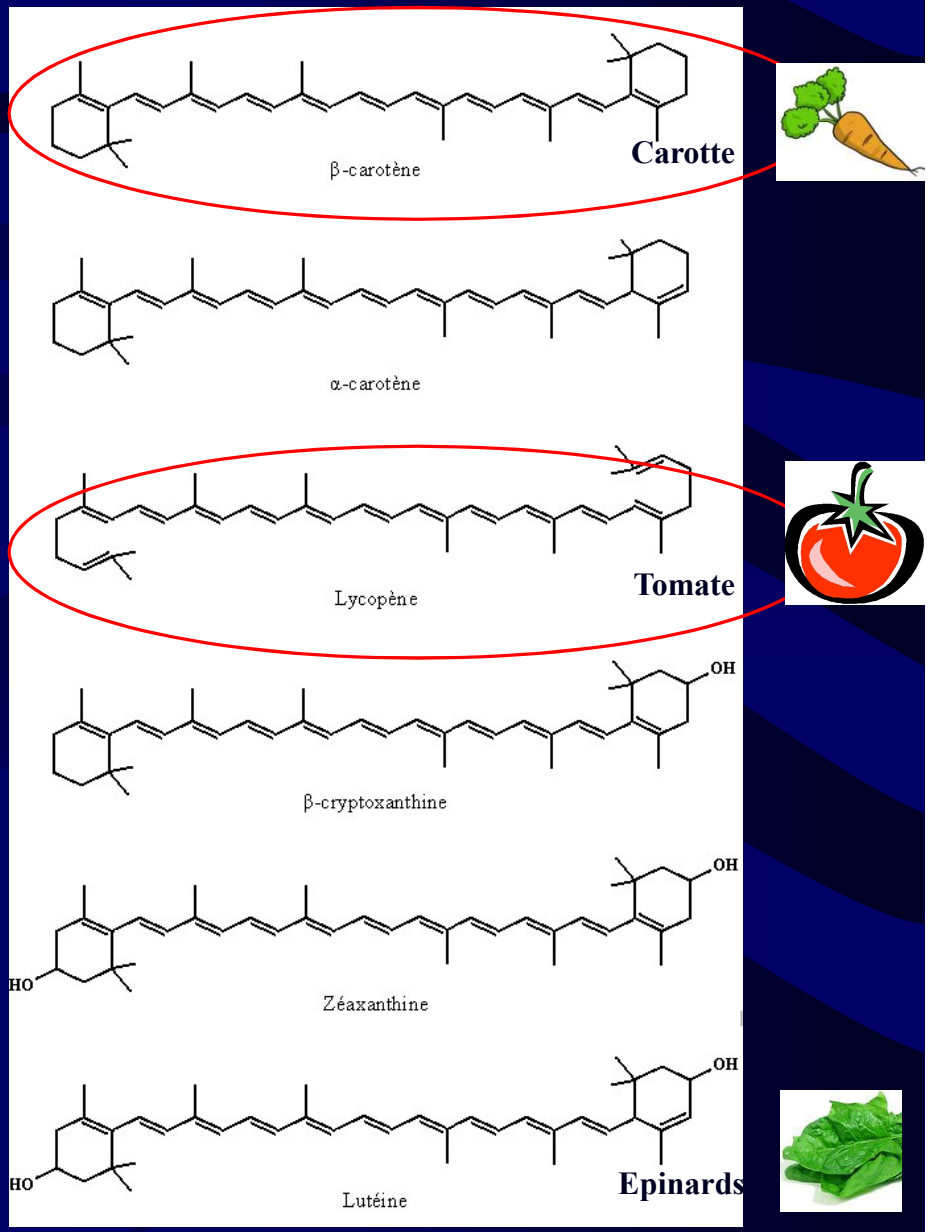
•Caroténoïdes: pigments naturels très répandus dans la nature (végétaux, algues, bactéries et champignons).

•Dérivent tous de précurseurs: le lycopène (tomate) et le β-carotène .

•6 caroténoïdes principaux du plasma

•B-caro également appelé provitamine A car précurseur

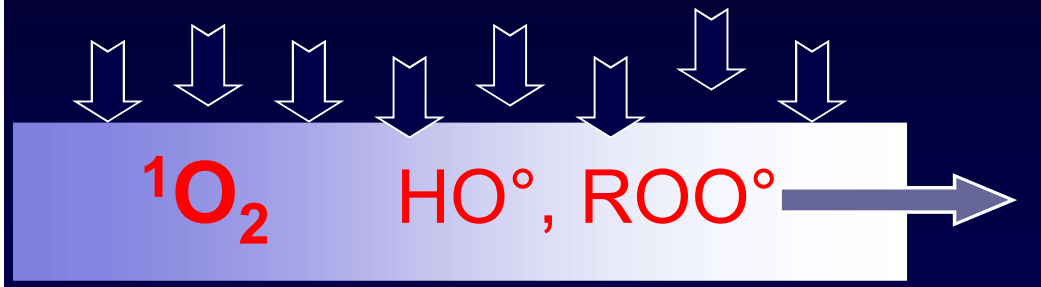




➢ Caractéristiques, localisation et actions

Liposoluble (stocké dans tissu adipeux et foie)

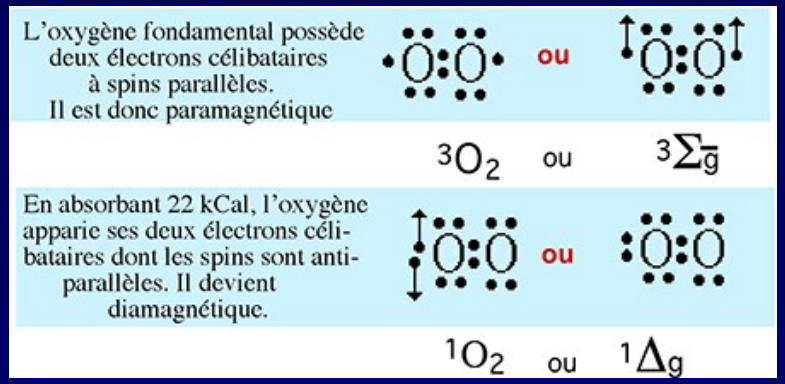
Action envers:



Stoppe peroxydation lipidique

β-carotène + ROO° → ROO-β-carotène°
ROO-β-carotène° + ROO° → inactif

Photoprotecteur: Piège $^1O_2$ (forme activée de l'oxygène) formé sous l'action des UV.



2.3.3- Les caroténoïdes: le β-carotène

- Apports nutritionnels conseillés (ANC 2001):
  - Sédentaires : 2.4 mg/j (♂); 1.8 mg/j (♀)
  - Sportifs: + 1 mg/j par tranche de 1000 kcal au dessus de 1800 kcal/j ♀ et 2200 kcal/j chez ♂.
  - Limite max 8.4 mg/j.

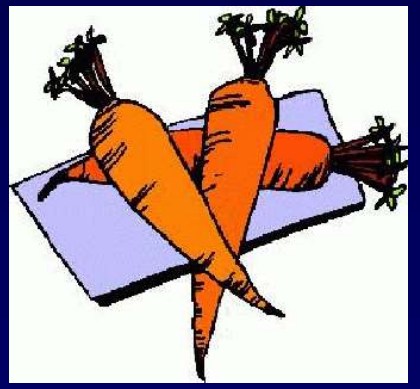
- Enquêtes sur la consommation des français:
  - ≈ 40% des adultes consomment moins des 2/3 des ANC.

En dessous de 0.22 mg/L les risques de cancer (poumons) ou MCV sont x 2 (Gey et al. MONICA1993)
Faible apport = facteur de risque

- Sources: fruits et légumes colorés (légumes verts, carottes et certains fruits jaunes).

Teneur en β-carotène (mg/100 g de poids frais)

Huile raffinée de palmier rouge	9,28
Carotte crue	4,6 - 12,5
Légumes verts (32 variétés)	1 - 44,4
Patate douce, variété orange	1,14
Mangue	0,615
Papaye, pastèque	0,228 - 0,324



Note : le β-carotène n'est pas détruit par la chaleur. On peut donc faire bouillir les légumes ou les faire cuire au four à micro-ondes.

Profils antioxydants et couleurs

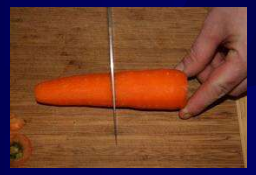
Rouge	Anthocyanines, lycopène	Betterave, cerise, chou rouge, fraise, tomate Oignon, poivron, pomme et radis rouges
Bleu-mauve	Polyphénols, flavonoïdes	Aubergine, cassis, framboise, mûre, prune, pruneau, raisin
Vert	Chlorophylle, voir ci-dessous	Avocat, brocoli, épinard, kiwi, chou de Bruxelles Haricot, poire et poivron verts
Jaune-orange	B-carotène, lutéine , zéaxanthine, quercétine	Abricot, ananas, carotte, citron, mangue, orange, papaye, pêche, poivron jaune
Blanc	Composés soufrés, sélénium Autres composés	Ail Pomme

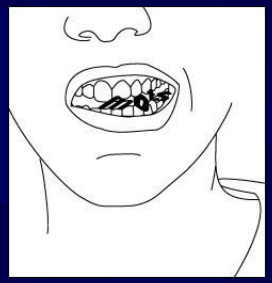
➢ Conseils nutritionnels

Meilleure libération et absorption des caroténoïdes

➡ Cellules des fruits et des légumes doivent être cassées

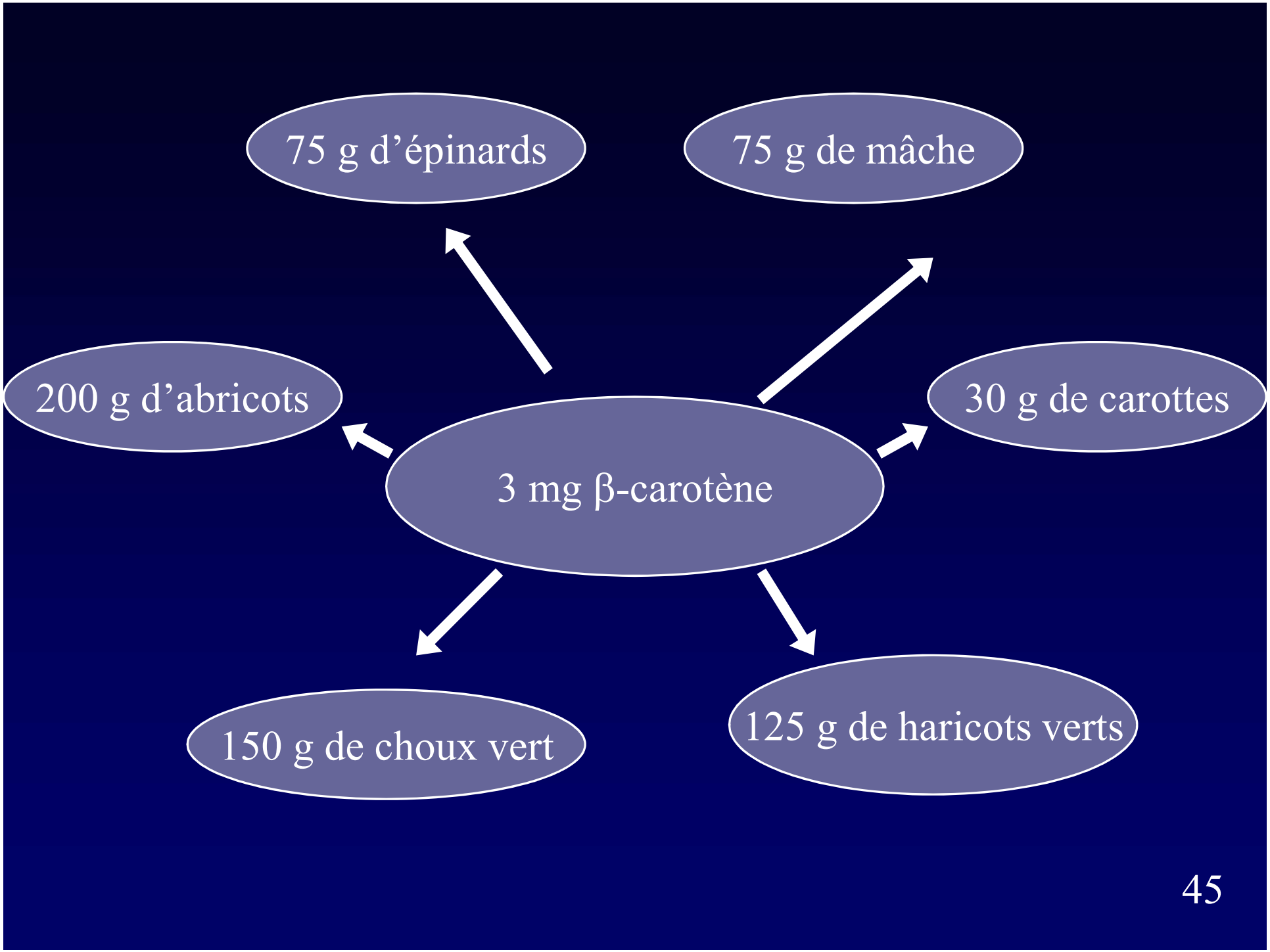
↪ **Couper, hacher, mâcher = IMPT**





➡ Cuisson dans corps gras (lycopène des tomates +++)

↪ Favorise la dégradation des parois des cellules ➔ meilleure libération des caroténoïdes qui sont + absorbés.
Corps gras rend plus assimilable le lycopène


75 g d’épinards
75 g de mâche
200 g d’abricots
30 g de carottes
3 mg β-carotène
150 g de choux vert
125 g de haricots verts

Bilan 2.2

Vitamines	**Principales caractéristiques**
Vitamine E	Liposoluble Piège les radicaux lipidiques (ROO° et RO° ⇔ stoppe phase propagation peroxydation lipidique), et d'autres ERO ($O_2^{\circ -}$, HO° , inactive $^1O_2$) Se transforme en radical tocophéroxyl.
Vitamine C	Hydrosoluble Antioxydant indirect en régénérant la vitamine E radicalaire → passe en forme radicalaire (radical ascorbyle) Antioxydant direct en piégeant $O_2^{\circ -}$, HO°, et en neutralisant $^1O_2$ → passe en forme oxydée (acide déhydroascobique)
B-carotène	Liposoluble Précurseurs du rétinol Neutralise $^1O_2$ donc photoprotecteur et piège HO°, et les radicaux lipidiques (limite la peroxydation lipidique).

Bilan 2.2

- Les français ne consomment pas suffisamment d'AO (étude SUVIMAX)
- Un faible taux plasmatique d'AO est associé à l'apparition de pathologies (cf étude MONICA de l'OMS)
- Les sportifs doivent apporter plus de vitamines AO en fonction de leur DE

Bilan 2.2

Apports en:	homme non entraîné	homme entraîné
Vitamine E	12 $mg.j^{-1}$	+ 12 $mg.j^{-1}$
Vitamine C	110 $mg.j^{-1}$	+ 100 $mg.j^{-1}$
Équivalent Rétinol (ER) **(Rétinol + 1/6 β-carotène)**	800 $\mu g.j^{-1}$	+ 200 $\mu g.j^{-1}$
Cuivre	2 $mg.j^{-1}$	+ 0,6 $mg.j^{-1}$
Zinc	12 $mg.j^{-1}$	+ 1 $mg.j^{-1}$
Sélénium	60 $\mu g.j^{-1}$	+ 30 $\mu g.j^{-1}$

Apport nutritionnel complémentaire à ajouter par tranche de 4180 $KJ.j^{-1}$ (1000 $kcal.j^{-1}$) au dessus de 9200 $kJ.j^{-1}$ (2200 $kcal.j^{-1}$) chez le sujet masculin

(Martin, 2001)

2.3- Comment complémenter?

- Nature de l'antioxydant : AO naturel vs synthétique

Importance pour la vitamine E (mélange des 8 stéréoisomères dont 1 seul est naturel)

(Miller 2004)

Activité antioxydante supérieure

Biodisponibilité meilleure

Reste plus longtemps dans les tissus

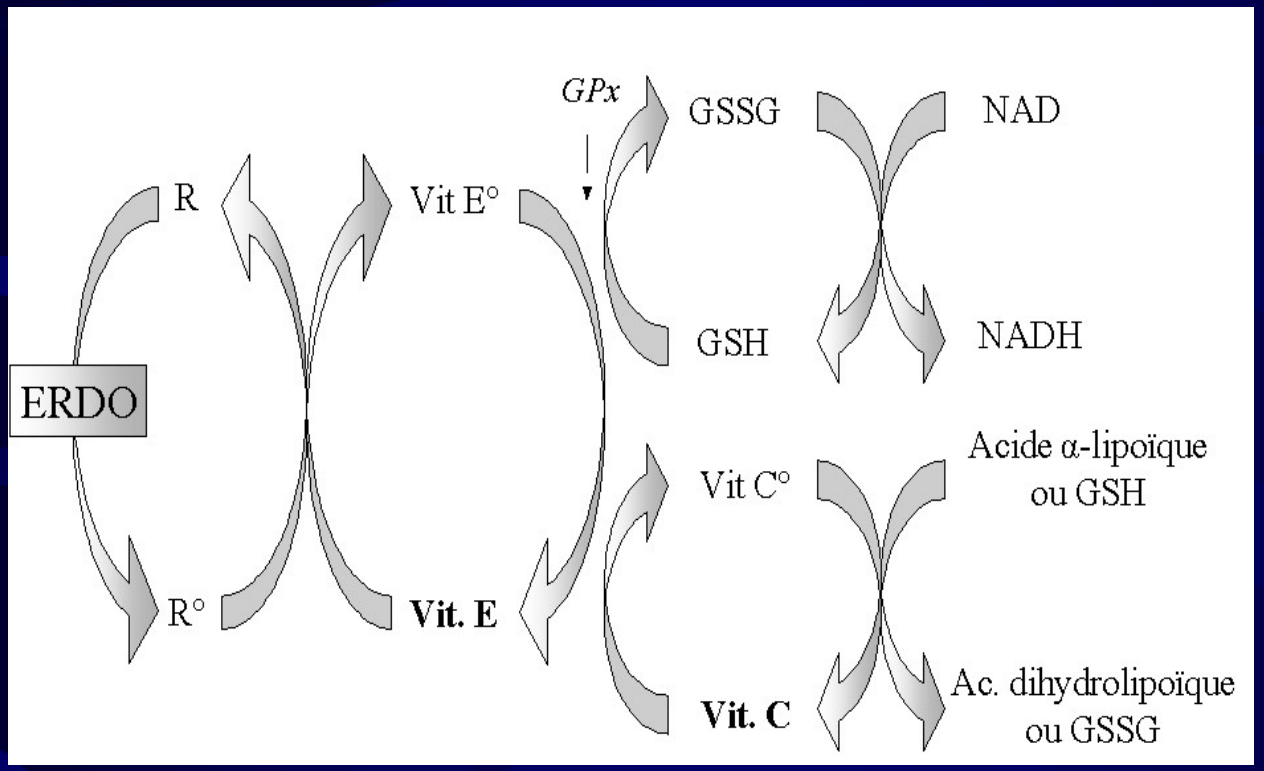
} Vit E naturelle très chère

Pas importance pour la vitamine C (Vit C synthétique identique à la nauturelle)

Vit C naturelle moins cher que vit E mais importance moindre car bonne efficacité vit synthétique
= extrait de l'ACEROLA (contient en plus des polyphénols [flavonoïdes])

2.3- Comment complémenter?

- Synergie des antioxydants:



Les compléments les + fréquents comprennent
Vit C, E, β-carotène, Cu, Zn, Se, Mn….

Un consensus

Le cocktail d'antioxydants

☞ Effet de synergie

Le « network » des antioxydants

Compléments alimentaires d'origine naturelle mieux assimilés et action antioxydante renforcée (surtout pour vit E)

2.4- Importance d'une alimentation riche en antioxydants apportés par les fruits et légumes

Nombreuses études

Tableau 1
Études interventionnelles ayant étudié la répercussion de fruits et légumes sur les taux plasmatiques en vitamine C et caroténoïdes

Études	Effectif H/F	Total	Âge (ans)	Tabac	Intervention type apport en antioxydants (par jour)	Concentrations sanguines	p value
Le Marchand et al., 1994 [11]	12/7	19	63,6	Oui	Conseils par un diététicien : augmentation de la consommation de fruits et légumes de 4,2 à 9,5 portions/j	β-carotène : 4,3 à 9,9 mg Lutéine : 1,6 à 5,1 mg Lycopène : 2,4 à 13 mg Vitamine C : 143 à 339 mg	µg/l t0 2 mois 3 mois 461 663 618 291 413 352 199 255 242 µg/ml t0 2 mois 3 mois 9,6 12,2 12,2	0,01 0,002 $p = 0,06$ 0,001
Rauma et al., 1995 [12]	0/40	40	46	Oui	Comparaison entre végétariens et omnivores	β-carotène vitamine C vég. 11,9 mg 183 mg Omniv. 4,3 mg 106 mg	β-carotène vitamine C vég. 2,93 µM 67 µM Omniv. 1,11 µM 58 µM	0,001 0,001
Yeun et al., 1996 [13]	9/9 9/9	18 18	20–40 60–80	Non Non	Alimentation riche en caroténoïdes (expérience répétée trois fois avec intervalle de six semaines entre chaque période)	All - trans β-carotène : 6 mg ; lutéine 2,3 mg	All - trans β-carotène F (jeunes) : 0,8 µM→2,2 µM F (âgées) : 0,8 µM→2,2 µM H (jeunes) : 0,4 µM→1,3 µM H (âgés) : 1,5 µM→2,2 µM Lutéine F (jeunes) : 0,29 µM→0,34 µM F (âgées) : 0,25 µM→0,40 µM H (jeunes) : 0,22 µM→0,33 µM H (vieux) : 0,37 µM→0,36 µM (effet additionnel du brocoli ± 20 %)	0,05 0,05 0,05 0,05 0,05 0,05 0,05 0,05
Hininger et al., 1997 [14]	11/11	22	25–45	Oui	150 g de carottes ; 200 g de tomates, haricots, chou et/ou épinards	β-carotène : 10 mg Lycopène : 10 mg Lutéine : 10 mg	β-carotène non-fumeur fumeur Début 0,95 µM 0,58 µM Après 15 j 1,13 µM 0,82 µM Lycopène non-fumeur fumeur Début 0,61 µM 0,56 µM Après 15 j 0,61 µM 0,55 µM	0,05 0,05
Zino et al., 1997 [15]	62/25	87	18–69	Oui	Groupe témoin : alimentation habituelle : 3 portions F et L Groupe intervention : passage à 8 portions de F et L	319 g de F et L 63 mg vitamine C 1,6 mg β-carotène 1001 g de F et L 257 mg Vitamine C 4,68 mg β-carotène	Vitamine C : 25,55 à 25,55 µM β-carotène : 0,34 à 0,32 µM Vitamine C : 33,5 à 57,92 µM β-carotène : 0,34 à 0,52 µM	
Bulux et al., 1998 [16]	13/10	23	9	Non	Ingestion d'un simple repas à base de carottes En quantité faible (122 g) En quantité importante (961 g)	12,4 mg β-carotène 97 mg β-carotène	Valeur plasmatique en β-carotène plus augmentée en fonction de l'apport en carottes	

(suite)

Consommation accrue en fruits et légumes ↗ [Vit C]pl et [caroténoïdes]pl

Tableau 1
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Zino et al., 1997 [15]	62/25	87	18–69	Oui	Groupe témoin : alimentation habituelle : 3 portions F et L Groupe intervention : passage à 8 portions de F et L	319 g de F et L 63 mg vitamine C 1,6 mg β-carotène 1001 g de F et L 257 mg Vitamine C 4,68 mg β-carotène	Vitamine C : 25,55 à 25,55 µM β-carotène : 0,34 à 0,32 µM Vitamine C : 33,5 à 57,92 µM β-carotène : 0,34 à 0,52 µM	
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(suite)

Tableau 1 *(suite)*

Études	Effectif H/F	Total	Âge (ans)	Tabac	Intervention type apport en antioxydants (par jour)	Concentrations sanguines	*p* value
De Pee et al., 1998 [17]	104/84	188	7–11	Non	4 groupes avec deux repas par jour, 6 jours par semaine A : groupe légumes ($n = 45$) B. groupe fruit ($n = 49$) C : groupe rétinol ($n = 48$) D : groupe pauvre en rétinol et caroténoïdes ($n = 46$) All– trans-β/cisβ/lyco/lut 3,5 0,6 0,2 5,9 2,3 0,3 4,8 0,8 0,2 < 0,1 0,1 0,2 0,2 < 0,1 0,1 0,2 (valeur en mg)	(μM) A B C D β-carotène +0,14 +0,52 +0,06 +0,03 Lutéine +0,31 +0,07 +0,07 +0,04 Lycop +0,01 +0,25 +0,02 +0,02	0,05
Miller et al., 1998 [18]	66/57	123	48,5	Oui	A : 4 portions F et L (témoin) B : 9 portions de F et L C : 10 portions de F et L avec diminution apport en graisses Pas d'information	β-carotène lutéine B +20 % +35 % C +40 %	0,05 vs A
McEligot et al., 1999 [19]	0/56	56	57,6		Groupe témoin : 5 F et L/j Groupe intervention : 5 portions de L, 3 portions de F 4,27→6,63 mg 5,32→21,69 mg	β-carotène Début 0,844 μM ; 3 ans 0,943 μM Début 0,748 μM ; 3 ans 1,391 μM	0,05
Thompson et al., 1999 [20]	0/28	28	27–80		Passage de 5,8 à 12 portions de F et L Pas d'information	(ng/ml) β-car. Lutéine lycopène Avant 313,8 156,1 348,3 Après 455,3 229,6 403,4	0,001
Broekmans et al., 2000 [21]	24–24	48	40–60	Oui	Groupe avec apport pauvre en F et L (100 g/j) Groupe avec apport normal en F et L (500 g/j + 200 ml jus de fruit/j) β-carotène : 0,63 mg Vitamine C : 65 mg β-carotène : 2,98 mg Vitamine C : 172,5 mg	Diminution de 6,7 μM Diminution de 0,02 μM Augmentation de 24 μM	0,05 0,05
Record et al., 2001 [22]	25/0	25	48,3	Non	Groupe A : ≤ 1 F et 2 L Groupe B : 5 à 7 portions de F et L Pas d'information	Groupe A Groupe B Vit. C 29,9 μM 47,1 μM β-carotène 0,56 μM 0,74 μM Lutéine 0,33 μM 0,46 μM Lycopène = inchangé	$p < 0,05$
Van den Berg et al., 2001 [23]	22/0	22	19–49	Oui	Placebo (légumes + boisson) Intervention légumes et jus de fruits Vitamine C β-carotène 0,83 mg < 0,05 170,5 mg 9 mg Vitamine C β-carotène P1 105 mg 3 mg	Vitamine C : 41→37,6 μM β-carotène : 0,30→0,29 μM Vitamine C : 41→57,6 μM β-carotène : 0,30→0,67 μM	0,0001 0,0001 0,003 ns
Freese et al., 2002 [24]	20/57	77	19–52	Oui	P1 : riche en acide linoléique et pauvre en légumes (170 g) P2 : riche en acide linoléique et riche en légumes (815 g) M1 : riche en acide oléique et pauvre en légumes (170 g) M2 : riche en acide oléique et riche en légumes (815 g) + groupe témoin Vitamine C β-carotène P1 105 mg 3 mg P2 333 mg 10,1 mg M1 105 mg 3 mg M2 333 mg 10,1 mg (valeur exprimée par MJ)	Vitamine C β-carotène P1 –0,1 μM +0,08 μM P2 +2,6 μM +0,64 μM M1 +0,2 μM +0,05 μM M2 +3,0 μM +0,47 μM Augmentation P2 et M2 vs P1 et P2	0,0001 0,0001

(suite)

AO apportés par l'alimentation sont impliqués dans les bienfaits de différents « régimes » reconnus pour leurs bienfaits sur la santé : **Régime méditerranéen et French Paradox**

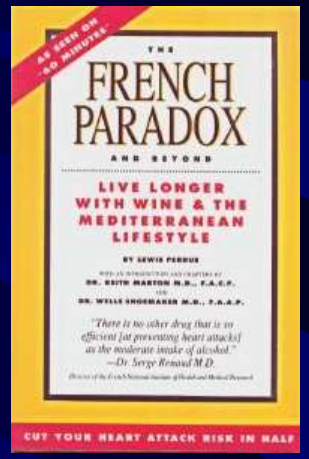
Régime méditerranéen

- ✓ Consommation importante:
  - Fruits et légumes frais et variés
    - ↳ Riches en fibres et **en AO**
  - Fromages ou des yogourts (brebis ou chèvre)
    - ↳Faible apport AGS,
  - Poisson (W3)
- ✓ Consommation faible :
  - -Viande rouge, lait et de beurre
    - ↳Faible apport AGS
- ✓ Lipides sous forme d'huile d'olive
  - ↳ Acide oléique,
- ✓ Vin rouge consommé modérément
  - ↳ **Polyphénols = AO**

French Paradox

A facteurs de risques égaux, les Français (sud ouest) ont moins de MCV que la plupart des occidentaux,

Pourtant: Alimentation assez riche en L (foie gras, confit de canard) et en boissons alcoolisées (vin rouge),



Explications:

1)- **Polyphénols du vin (Resveratrol et ses métabolites) ont des propriétés AO → controversé**

2)- Graisse canard et oie = HDL

3)- Huile d'olive **(polyphénols)**

(*Filmore et al. 2007; Fragopoulou et al. 2020)*)

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Invited critical review

The French paradox three decades later: Role of inflammation and thrombosis

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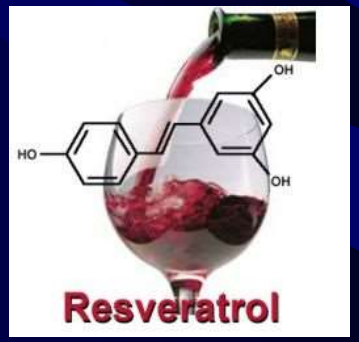


Taux d'infarctus est de seulement 80 pour 100 000 par an, soit 4 fois moins qu'aux États-Unis[

2.4- Importance d'une alimentation riche en antioxydants apportés par les fruits et légumes

Diet, metabolic polymorphisms and DNA adducts* : the epic-Italy cross-sectional study.
Palli et al. Int J Cancer 87:444-451, 2000

	modérée	moyenne	forte	%
vitamine C	9,03 ±1,24	7,28 ± 1,17	7,79 ± 1,21	- 15,9
vitamine E	8,91 ± 1,46	7,51 ± 1,23	7,73 ± 1,30	- 15,3
β-carotène	9,37 ± 1,22	7,63 ± 1,13	7,15 ± 1,23	- 31,0
rétinol	8,96 ± 1,21	7,42 ± 1,16	7,62 ± 1,23	- 17,6
alcool	7,41 ± 1,30	7,93 ± 1,17	8,66 ± 1,29	+ 16,9

* dosé dans des lymphocytes

→ Marqueurs de l'oxydation de l'ADN

Un régime riche en AO diminue les marqueurs du SO

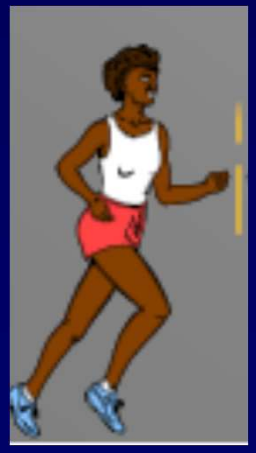
Bilan 2.4

Un régime riche en AO diminue les marqueurs du SO

Le régime crétois, méditerranéen et le French paradox s'expliquent en partie par les AO

Un haut niveau endogène d'AO (alimentation riche en fruits et légumes), est associé avec un risque relatif bas de mortalité par maladies dégénératives (cancers, maladie cardiovasculaire, infection, BPCO) [Ray et al. 2006], [Hercberg et al. 2004]

3- Exercice et SO



3.1- Mise en évidence du SO lors de l'exercice

- Exercice exhaustif (soit maximal ou si pas maximal doit être prolongé) qu'il soit aérobie ou anaérobie ➔ SO

Table IV. Human studies on the effects of aerobic exercise on markers of oxidative stress

Study (year)	Activity	Subjects	Markers	Effect
Lovlin et al.[174] (1987)	Cycling at 40%, 70% and 100% $\dot{V}O_{2max}$	6 UT	MDA (at 40% $\dot{V}O_{2max}$)	↓
			MDA (at 70% $\dot{V}O_{2max}$)	↔
			MDA (at 100% $\dot{V}O_{2max}$)	↑
Margaritis et al.[177] (1997)	Triathlon (long distance)	18 VT	TBARS – GSSG	↔
Marzatico et al.[168] (1997)	Running (half-marathon)	6 T	MDA	↑
			CD	↔
			SOD – GPX	↑
			CAT	↔
Vasankari et al.[52] (1997)	Running (marathon)	22 VT	Tocopherol – TRAP	↑
			CD	↑
			Retinol – CoQ_{10}	↔
Ashton et al.[162] (1998)	$\dot{V}O_{2max}$ test (ergocycle)	12 T	TAC	↑
			FR (ESR) – MDA – LH	↑
Child et al.[178] (1998)	Running (half-marathon)	17 T	MDA	↑
			CK	↑
			TEAC – UA	↑
Liu et al.[58] (1999)	Running (marathon)	11 VT	Oxidised LDL	↑
		10 UT	TRAP – UA	↑
			Thiols	↓
			Tocopherol – vit C – vit A	↔
Hellsten et al.[136] (2001)	Two exercises to exhaustion at 90% $\dot{V}O_{2max}$ (cycling)	8 T	Allantoin	↑
			UA (muscle)	↑
			GSH – cysteine – UA (plasma)	↔
Inal et al.[179] (2001)	Swimming (800m)	10 T	CAT – GPX	↑
			GSH	↓
Mastaloudis et al.[90] (2001)	Running (50km)	11 T	Isoprostane	↑
			UA – tocopherol – vit C	↑
Miyazaki et al.[159] (2001)	$\dot{V}O_{2max}$ test (ergocycle)	9 UT	TBARS – neutrophil FR production	↑ ↔
			Protein carbonyls	↔
			SOD – GPX – CAT	
Vider et al.[176] (2001)	$\dot{V}O_{2max}$ test (treadmill)	19 T	TBARS – CD	↑
			TAC – GSH – CAT	↑
			GPX – SOD	↔
Dawson et al.[70] (2002)	Running (21km)	15 T	MDA	↑
			CK – myoglobin	↑
Chevion et al.[172] (2003)	Walking (50km)	29 T	CK	↑
	Walking (80km)	16 T	Protein carbonyls	↓
			UA	↑
Palmer et al.[96] (2003)	Ultra-marathon (80km)	28 T	LH – F2-isoprostane	↑
			Vit C	↑
Agullo et al.[173] (2005)	Cycling (171km)	8 T	GSSG	↑
			UA – tocopherol	↑
			GPX	↓

CAT = catalase; **CD** = conjugated dienes; **CK** = creatine kinase; **CoQ_{10}** = coenzyme Q_{10}; **ESR** = electron spin resonance; **FR** = free radical; **GPX** = glutathione peroxidase; **GSH** = glutathione; **GSSG** = oxidised glutathione; **LDL** = low-density lipoprotein; **LH** = lipid hydroperoxide; **MDA** = malondialdehyde; **SOD** = superoxide dismutase; **T** = trained; **TAC** = total antioxidant capacity; **TBARS** = thiobarbituric reactive substances; **TEAC** = trolox equivalent antioxidant capacity; **TRAP** = total radical antioxidant potential; **UA** = uric acid; **UT** = untrained; **vit** = vitamin; **VT** = very trained; **$\dot{V}O_{2max}$** = maximum oxygen consumption; ↓ indicates decrease; ↑ indicates increase; ↔ indicates no change (stable).

Table V. Human studies on the effects of anaerobic exercise on markers of oxidative stress

Study (year)	Activity	Subjects	Markers	Effect
Sahlin et al.[180] (1992)	Isometric knee extension at 60% 1RM intermittent – 80 min	7 UT	MDA	↔
			GSH (blood)	↑
			GSH (muscle)	↔
			GSSG (blood and muscle)	↔
Saxton et al.[167] (1994)	Elbow flexion – 70 max eccentric or concentric actions	14 NRT	TBARS – CD – MDA	↔
			Protein carbonyls	↑
Marzatico et al.[168] (1997)	6 × 150m sprints	6 T	MDA – CD	↑
			SOD – GPX	↑
			CAT	↔
Ortenblad et al.[166] (1997)	6 bouts of jumping – 30 sec each bout	8 JT	MDA	↔
		8 UT		
McBride et al.[94] (1998)	Resistance training programme (8 exercises, 3 sets of each failure)	12 T	MDA	↑
Childs et al.[75] (2001)	Eccentric arm flexion (cybex)	14 UT	LH – isoprostane	↑
	3 × 10 reps at 80% RM		CK – LDH – myoglobin	↑
			SOD	↑
			GPX	↔
Inal et al.[179] (2001)	100m swim	9 T	CAT – GPX	↑
			GSH	↓
Groussard et al.[169] (2003)	Cycling – Wingate tests (30 sec)	7 T	UA – vit C	↑
			Tocopherol – vit A	↓
Groussard et al.[111] (2003)	Cycling – Wingate tests (30 sec)	8 T	ESR signals	↑
			TBARS	↓
			SOD – GSH	↓
			GPX	↔
Ramel et al.[165] (2004)	Resistance programme (10 exercises – max of reps at 75% 1RM)	7 T	MDA	↔
		10 UT	CD (trained group)	↔
			CD (untrained group)	↑
			Vit A – tocopherol	↑
Goldfarb et al.[164] (2005)	Eccentric resistance exercise	18 UT	Protein carbonyls – MDA	↑
			GSSG	↑
			GSH	↓

CAT = catalase; **CD** = conjugated dienes; **CK** = creatine kinase; **ESR** = electron spin resonance; **GPX** = glutathione peroxidase; **GSH** = glutathione; **GSSG** = oxidised glutathione; **JT** = jump trained; **LDH** = lactate dehydrogenase; **LH** = lipid hydroperoxide; **max** = maximum; **MDA** = malondialdehyde; **NRT** = non-resistance trained; **reps** = repetitions; **RM** = repetition maximum; **SOD** = superoxide dismutase; **T** = trained; **TBARS** = thiobarbituric reactive substances; **UA** = uric acid; **UT** = untrained; **vit** = vitamin; ↓ indicates decrease; ↑ indicates increase; ↔ indicates no change (stable).

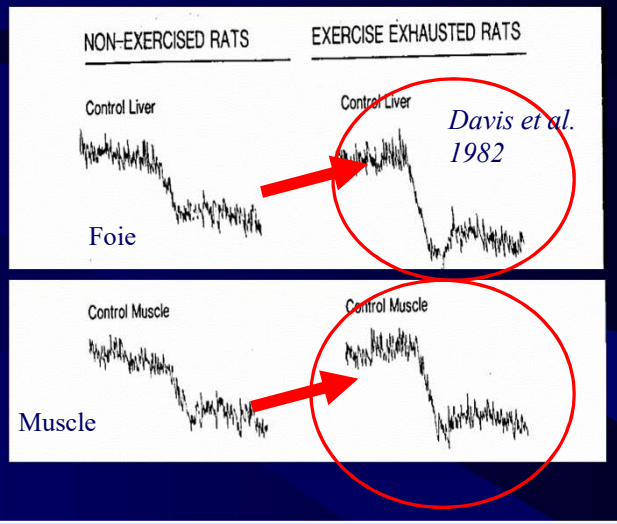
Finaud et al. (2006)

- Exo peu intense ➔ pas de SO

3.1- Mise en évidence du SO lors de l'exercice

3.1.1- Mise en évidence directe:

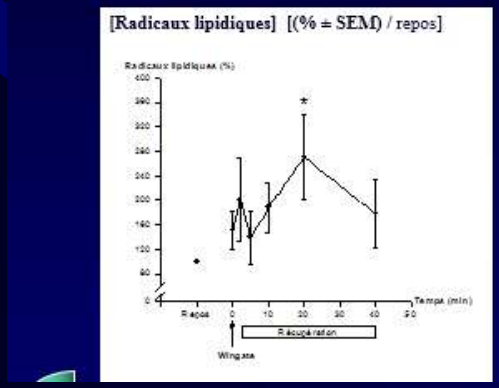
- Exercice aérobie exhaustif:
  - Animal: exo aérobie sous max.→ épuisement ⇒ ↗ P° RL au niveau foie et muscle x 2 et 3.
  - Homme : VO_{2max} ⇒ ↗ signal RPE dans le sang

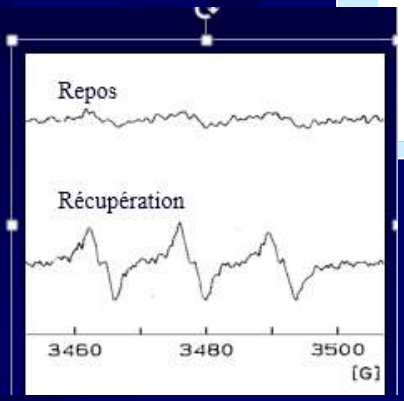


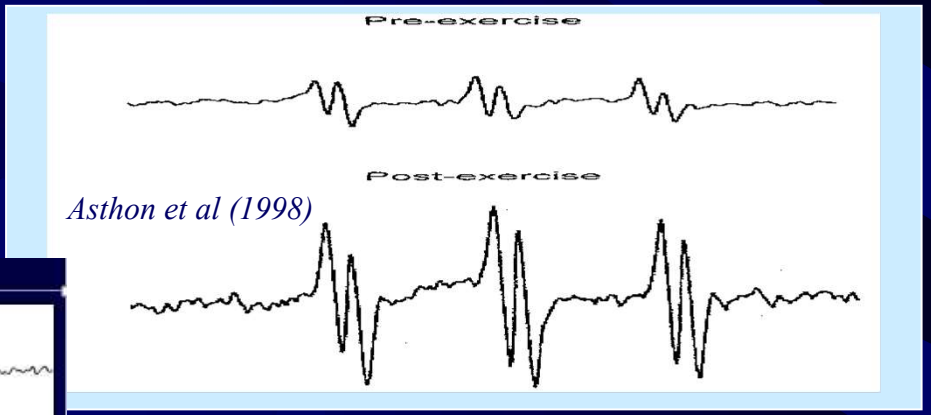
Davis et al. 1982

- Exercice anaérobie:
  - Test de Wingate

Groussard et al. (2003)







Asthon et al (1998)

↗ RL à 20 min de récupération / repos.

signal multiplié par 3 / repos.

3.1- Mise en évidence du SO lors de l'exercice

3.1.2- Mise en évidence indirecte

Chez l'animal et chez l'Homme, l'exercice aérobie exhaustif ➔ SO

↗ ↗ ↗ Marqueurs de la peroxydation lipidique

Marqueurs de la peroxydation lipidique = TBARS (Niels et al. 2005)

Table I. Effects of exercise on thiobarbituric acid (TBARS)

Study[a]	Assay	Exercise protocol[b]	Subjects	Resting TBARS ± SD (μmol/L)	Δ Exercise (%)[c]
Viinikka et al.[14]	Fluoro	Max-test cycle	10 T M	2.0 ± 0.4	+10
Lovlin et al.[32]	Spec	5 min cycle 40%	6 UT M	2.26 ± 0.10 mmol/L	−12[d]
		5 min cycle 70%			−6
		Max-test cycle			+27[d]
Kantor et al.[37]	Spec	80km run	9 T M	3.6 ± 0.8	+50[d]
Maughan et al.[15]	Spec	45 min down-hill run	16 UT M	2.0 ± 0.1	NC
Duthie et al.[16]	Fluoro	21km run	7 T M	1.52 ± 0.23	+16
Kretzschmar et al.[33]	Fluoro	Max-test cycle	7 UT M	3.64 ± 0.74	−12[d]
			5 T M	3.29 ± 0.34	−33[d]
Sahlin et al.[17]	UV/HPLC	Cycle 98% to exh	8 MT M	<0.1	NC
Kantor and Eddy[38]	Spec	30 min 60%/5 min 90%	20 T/UT M	5 ± 1	−+30[d]
Ji[42]	Spec	Cycle 70% to exh	8 T F/M	0.12 ± 0.03	−+30
Maxwell et al.[46]	UV/HPLC	60 min box-stepping	8 UT F/M	0.89 ± 0.12	+38
Rokitzki et al.[39]	Spec	Max-test cycle	15 T F	7.6 ± 2.8	+31
Sen et al.[43]	Spec	Max-test	9 UT M	1.3 ± 0.2	−+30
		30 min cycle AT		1.2 ± 0.3	−+60[d]
		30 min cycle AnT		1.1 ± 0.1	−+90[d]
Hartmann et al.[18]	Fluoro	Max-test run	5 UT M	2.6 ± 0.6	+15
Vasankari et al.[51]	Spec	1km run	8 T M	1.05 ± 0.05	−7
		10km run	7 T M	0.98 ± 0.08	−11
Niess et al.[20]	Fluoro	Max-test run	5 UT M	2.58 ± 0.63	+12
Alessio et al.[52]	Fluoro	30 min run 80%	9 MT M	0.90 ± 0.12	+47[d]
Dufaux et al.[21]	Fluoro/HPLC	2.5h run	12 MT M	0.8 ± 0.1	NC
Margaritis et al.[22]	Fluoro/HPLC	Triathlon	12 T M	4.54 ± 0.70 mmol/L	−6
Ashton et al.[9]	Fluoro/HPLC	Max-test cycle	12 UT M	0.70 ± 0.05	+14[d]
Child et al.[53]	Fluoro/HPLC	21km run	17 T M	1.48 ± 0.39	+11[d]
Marzatico et al.[40]	Spec	Half marathon	6 T M	3.13 ± 0.4	+60[d]
		6 × 150m sprint	6 T M	2.86 ± 0.3	+220[d]
Szczesniak et al.[54]	Not given	Max-test run	13 T M	1.80 ± 0.03	+14[d]
Ashton et al.[10]	Fluoro/HPLC	Max-test cycle	10 UT M	0.70 ± 0.04	+14[d]
Bonsheim et al.[55]	Spec	90 min cycle 58%	8 MT M	1.68 ± 0.08	+83[d]
Chung et al.[23]	Spec (TBARS)	30 min run 75%	11 UT F	0.65 ± 0.05	NC
	Spec (MDA)			1.55 ± 0.17	NC
Laaksonen et al.[56]	Spec	40 min cycle 60%	14 UT M	0.86 ± 0.37	+67[d]
Leaf et al.[57]	Spec	Max-test run	14 UT M	26.9 ± 25.7	−6
Surmen-Gur et al.[25]	Spec	Max-test cycle	9 UT F	5.2 ± 1.0	NC
Alessio et al.[26]	Spec, MPI	Max-test run	12 UT M/F	0.14 ± 0.01 μmol/g protein	+14
		Isometric 50% MVC to exh		0.14 ± 0.01 μmol/g protein	+7
Child et al.[58]	Fluoro/HPLC	21km run	14 T M	1.49 ± 0.16	−+30[d]
Heasol et al.[31]	Spec	Marathon	18 T M	11.46 ± 3.09	+11[d]
Sachsck et al.[27]	Fluoro/HPLC	45 min 10% downhill run	8 T F	1.5 ± 0.2	NC
Akova et al.[28]	Spec	Leg extension to exh	8 UT F	3.2 ± 0.3	NC
Groussard et al.[13]	Fluoro/HPLC	Wingate	8 UT M	?	−24[d]
Quindry et al.[29]	Spec	Max-test run	9 MT M	1.9 ± 0.5	−5
		45 min 10% below LT		2.2 ± 0.4	+4
		45 min 10% above LT		2.3 ± 0.7	+4
Sachsck et al.[30]	Fluoro/HPLC	45 min 16% downhill run	16 MT M	0.40 ± 0.05	NC
Ramel et al.[24]	HPLC	Submax resistance exercise	7 T M	1.60 ± 0.38	+25
			10 UT M	2.09 ± 1.18	+19

a Selected studies are shown.
b The '%' symbol in the 'Exercise protocol' column indicates the percentage of maximum oxygen consumption.
c Δ Exercise is the change caused by exercise, and has been estimated from figures if not stated in the original article.
d Significant change.

AnT – anaerobic threshold; **AT** – aerobic threshold; **exh** – exhaustion; **F** – females; **fluoro** – fluorometric; **HPLC** – high-performance liquid chromatography; **LT** – lactate threshold; **M** – males; **max-test** – exhaustive incremental test; **MDA** – malondialdehyde; **MPI** – *N*-methyl-2-phenylindolino; **MT** – moderately trained; **MVC** – maximal voluntary contraction; **NC** – no change; **spec** – spectrophotometric; **submax** – submaximal; **T** – trained; **UT** – untrained; **?** indicates information not provided in original article.

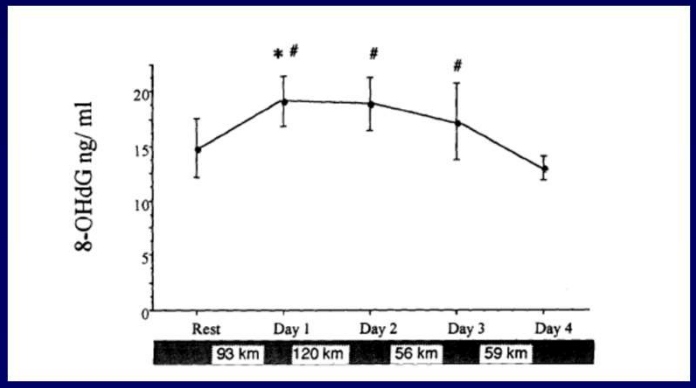
3.1- Mise en évidence du SO lors de l'exercice

3.1.2- Mise en évidence indirecte

Chez l'animal et chez l'Homme, l'exercice aérobie exhaustif ➔ SO

↗ ↗ Marqueurs de l'oxydation des protéines (peu d'études)

↗ Marqueurs de l'ADN (très peu d'étude et ↗ que pour exercices très intenses)



Radak et al. 2003

Table IV. Human studies on the effects of aerobic exercise on markers of oxidative stress

Study (year)	Activity	Subjects	Markers	Effect
Lovlin et al.[174] (1987)	Cycling at 40%, 70% and 100% $\dot{V}O_{2max}$	6 UT	MDA (at 40% $\dot{V}O_{2max}$)	↓
			MDA (at 70% $\dot{V}O_{2max}$)	↔
			MDA (at 100% $\dot{V}O_{2max}$)	↑
Margaritis et al.[177] (1997)	Triathlon (long distance)	18 VT	TBARS – GSSG	↔
Marzatico et al.[168] (1997)	Running (half-marathon)	6 T	MDA	↑
			CD	↔
			SOD – GPX	↑
			CAT	↔
Vasankari et al.[52] (1997)	Running (marathon)	22 VT	Tocopherol – TRAP	↑
			CD	↑
			Retinol – CoQ_{10}	↔
Ashton et al.[162] (1998)	$\dot{V}O_{2max}$ test (ergocycle)	12 T	TAC	↑
			FR (ESR) – MDA – LH	↑
Child et al.[178] (1998)	Running (half-marathon)	17 T	MDA	↑
			CK	↑
			TEAC – UA	↑
Liu et al.[58] (1999)	Running (marathon)	11 VT	Oxidised LDL	↑
		10 UT	TRAP – UA	↑
			Thiols	↓
			Tocopherol – vit C – vit A	↔
Hellsten et al.[130] (2001)	Two exercises to exhaustion at 90% $\dot{V}O_{2max}$ (cycling)	8 T	Allantoin	↑
			UA (muscle)	↑
			GSH – cysteine – UA (plasma)	↔
Inal et al.[179] (2001)	Swimming (800m)	10 T	CAT – GPX	↑
			GSH	↓
Mastaloudis et al.[90] (2001)	Running (50km)	11 T	Isoprostane	↑
			UA – tocopherol – vit C	↑
Miyazaki et al.[180] (2001)	$\dot{V}O_{2max}$ test (ergocycle)	9 UT	TBARS – neutrophil FR production	↑
			Protein carbonyls	↔
			SOD – GPX – CAT	↔
Vider et al.[175] (2001)	$\dot{V}O_{2max}$ test (treadmill)	19 T	TBARS – CD	↑
			TAC – GSH – CAT	↑
			GPX – SOD	↔
Dawson et al.[70] (2002)	Running (21km)	15 T	MDA	↑
			CK – myoglobin	↑
Chevion et al.[176] (2003)	Walking (50km)	29 T	CK	↑
	Walking (80km)	16 T	Protein carbonyls	↓
			UA	↑
Palmer et al.[96] (2003)	Ultra-marathon (80km)	28 T	LH – F2-isoprostane	↑
			Vit C	↑
Agullo et al.[172] (2005)	Cycling (171km)	8 T	GSSG	↑
			UA – tocopherol	↑
			GPX	↓

CAT = catalase; **CD** = conjugated dienes; **CK** = creatine kinase; **CoQ_{10}** = coenzyme Q_{10}; **ESR** = electron spin resonance; **FR** = free radical; **GPX** = glutathione peroxidase; **GSH** = glutathione; **GSSG** = oxidised glutathione; **LDL** = low-density lipoprotein; **LH** = lipid hydroperoxide; **MDA** = malondialdehyde; **SOD** = superoxide dismutase; **T** = trained; **TAC** = total antioxidant capacity; **TBARS** = thiobarbituric reactive substances; **TEAC** = trolox equivalent antioxidant capacity; **TRAP** = total radical antioxidant potential; **UA** = uric acid; **UT** = untrained; **vit** = vitamin; **VT** = very trained; **$\dot{V}O_{2max}$** = maximum oxygen consumption; ↓ indicates decrease; ↑ indicates increase; ↔ indicates no change (stable).

Finaud et al. (2006)

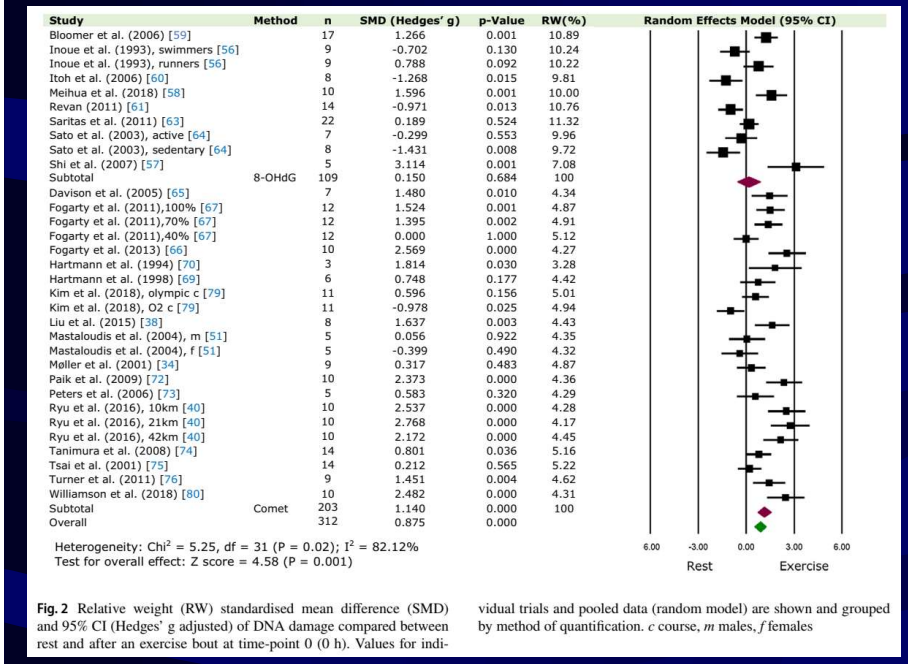


Fig. 2 Relative weight (RW) standardised mean difference (SMD) and 95% CI (Hedges' g adjusted) of DNA damage compared between rest and after an exercise bout at time-point 0 (0 h). Values for individual trials and pooled data (random model) are shown and grouped by method of quantification. *c* course, *m* males, *f* females

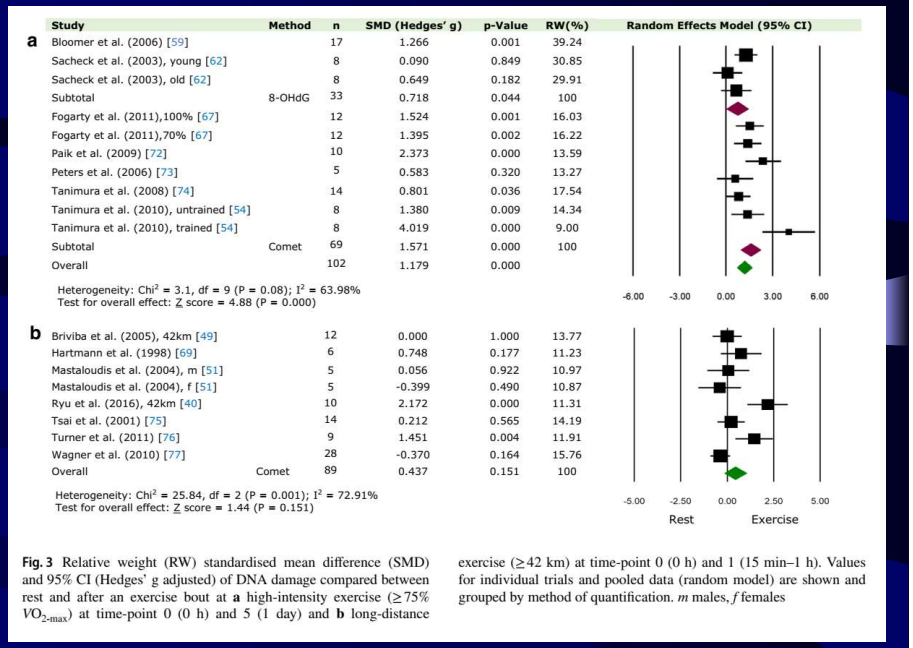


Fig. 3 Relative weight (RW) standardised mean difference (SMD) and 95% CI (Hedges' g adjusted) of DNA damage compared between rest and after an exercise bout at **a** high-intensity exercise (≥75% $VO_{2\text{-max}}$) at time-point 0 (0 h) and 5 (1 day) and **b** long-distance exercise (≥42 km) at time-point 0 (0 h) and 1 (15 min–1 h). Values for individual trials and pooled data (random model) are shown and grouped by method of quantification. *m* males, *f* females

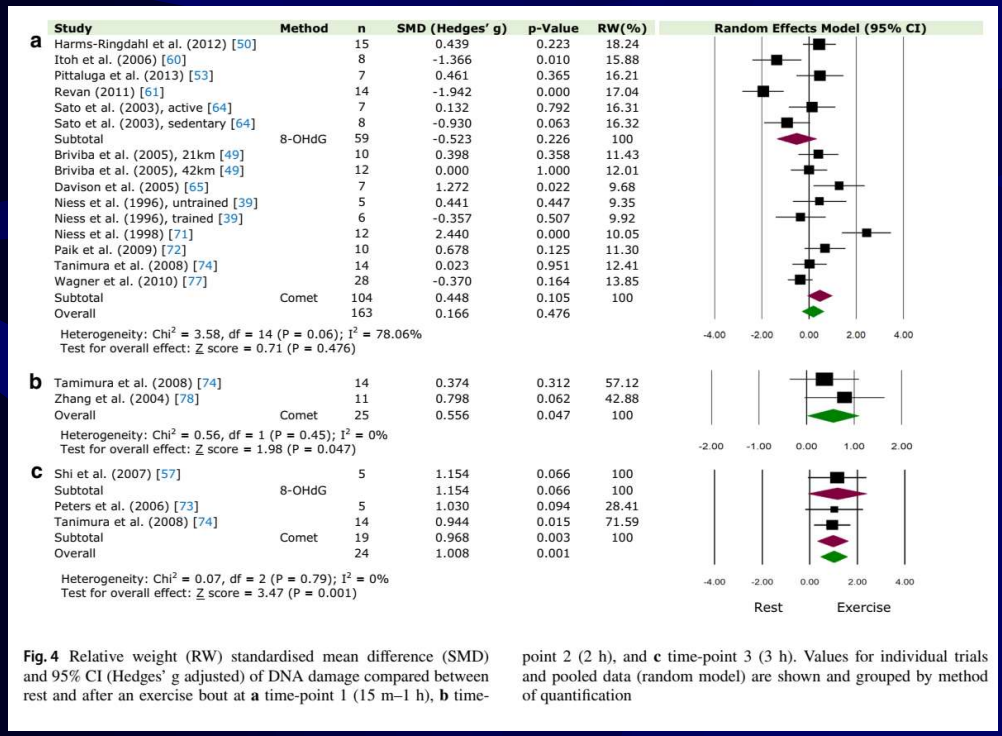


Fig. 4 Relative weight (RW) standardised mean difference (SMD) and 95% CI (Hedges' g adjusted) of DNA damage compared between rest and after an exercise bout at **a** time-point 1 (15 m–1 h), **b** time-point 2 (2 h), and **c** time-point 3 (3 h). Values for individual trials and pooled data (random model) are shown and grouped by method of quantification

Tryfidou et al. 2020

3.1- Mise en évidence du SO lors de l'exercice

3.1.2- Mise en évidence indirecte

Chez l'animal et chez l'Homme, l'exercice aérobie exhaustif active le système AO

SOD: ↗ acté dans de nombreux tissus (Ji et coll. 1993, Alessio et Goldfarb 1988).

↳ Activation en réponse à une sur P° $O_2^{-\circ}$

GPx: Résultats contradictoires.

Pas de changements (Brady et coll. 1979, Ji et coll. 1990).

↗ Ji et Fu 1992, Oh-Ishi 1997.

CAT: pas d 'augmentation (Meydani et Evans, etc...)

3.1.3- Les EAO produits à l'exercice entraînent une fatigue musculaire:

- Une $[EAO]_{basale}$ est nécessaire pour P° Force (cf avant – Reid avec ajout d'AO)

- Hypothèse rôle EAO dans fatigue testée par traitement en AO (NAC ou Tiron…)

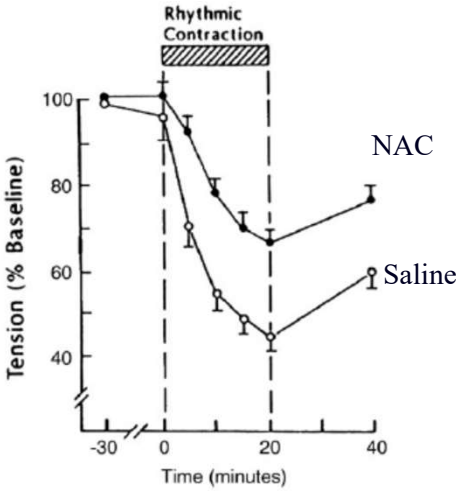
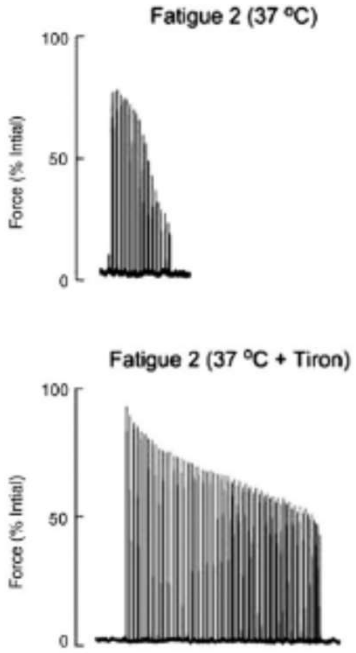


Fig. 1. Antioxidant pretreatment can inhibit muscle fatigue. Forces developed by diaphragm of anesthetized rabbits pretreated with NAC 150 mg/kg (closed symbols) or saline (open symbols) are presented. The isolated, perfused muscle was stimulated electrically via the phrenic nerve. Repetitive, fatiguing contractions (0–20 min) depressed force relative to control (−30 min); force recovered after stimulation ceased (20–40 min); NAC increased force production during and after fatigue. Data shown are means±SEM. Reproduced from Ref. [28].



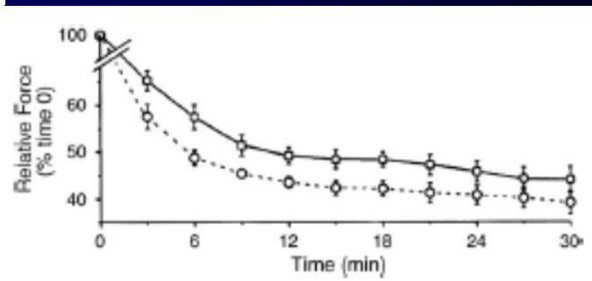


Fig. 6. *N*-acetylcysteine (NAC) inhibits muscle fatigue in humans. Forces developed by human tibialis anterior during repetitive 10-Hz contractions stimulated transcutaneously in eight healthy volunteers are presented. In repeated trials on separate days, the muscle sustained 15% greater force after pretreatment with NAC at 150 mg/kg (open squares) versus saline (open circles). Data shown are means±SEM. Reproduced from Ref. [93].

M.B. Reid / Free Radical Biology & Medicine 44 (2008) 169–179

[Reid et al. 1993, 1994, Moopanar et al. 2005, shindo et al. 1990]

Le piégeage des EAO par AO (enzymes ou non) ↘ fatigue musculaire

3.1- Mise en évidence du SO lors de l'exercice

Chez l'animal et l'Homme, l'exercice anaérobie exhaustif ➔ SO

Table V. Human studies on the effects of anaerobic exercise on markers of oxidative stress

Study (year)	Activity	Subjects	Markers	Effect
Sahlin et al.[188] (1992)	Isometric knee extension at 60% 1RM intermittent – 80 min	7 UT	MDA GSH (blood) GSH (muscle) GSSG (blood and muscle)	↔ ↑ ↔ ↔
Saxton et al.[187] (1994)	Elbow flexion – 70 max eccentric or concentric actions	14 NRT	TBARS – CD – MDA Protein carbonyls	↔ ↑
Marzatico et al.[189] (1997)	6 × 150m sprints	6 T	MDA – CD SOD – GPX CAT	↑ ↑ ↔
Ortenblad et al.[186] (1997)	6 bouts of jumping – 30 sec each bout	8 JT 8 UT	MDA	↔
McBride et al.[84] (1998)	Resistance training programme (8 exercises, 3 sets of each failure)	12 T	MDA	↑
Childs et al.[75] (2001)	Eccentric arm flexion (cybex) 3 × 10 reps at 80% RM	14 UT	LH – Isoprostane CK – LDH – myoglobin SOD GPX	↑ ↑ ↑ ↔
Inal et al.[178] (2001)	100m swim	9 T	CAT – GPX GSH	↑ ↓
Groussard et al.[108] (2003)	Cycling – Wingate tests (30 sec)	7 T	UA – vit C Tocopherol – vit A	↑ ↓
Groussard et al.[111] (2003)	Cycling – Wingate tests (30 sec)	8 T	ESR signals TBARS SOD – GSH GPX	↑ ↓ ↓ ↔
Ramel et al.[185] (2004)	Resistance programme (10 exercises – max of reps at 75% 1RM)	7 T 10 UT	MDA CD (trained group) CD (untrained group) Vit A – tocopherol	↔ ↔ ↑ ↑
Goldfarb et al.[184] (2005)	Eccentric resistance exercise	18 UT	Protein carbonyls – MDA GSSG GSH	↑ ↑ ↓

CAT = catalase; **CD** = conjugated dienes; **CK** = creatine kinase; **ESR** = electron spin resonance; **GPX** = glutathione peroxidase; **GSH** = glutathione; **GSSG** = oxidised glutathione; **JT** = jump trained; **LDH** = lactate dehydrogenase; **LH** = lipid hydroperoxide; **max** = maximum; **MDA** = malondialdehyde; **NRT** = non-resistance trained; **reps** = repetitions; **RM** = repetition maximum; **SOD** = superoxide dismutase; **T** = trained; **TBARS** = thiobarbituric reactive substances; **UA** = uric acid; **UT** = untrained; **vit** = vitamin; ↓ indicates decrease; ↑ indicates increase; ↔ indicates no change (stable).

Finaud et al. (2006)

Bilan SO et exo

Exercice exhaustif qu'il soit aérobie (soit maximal ou si pas maximal doit être prolongé) ou anaérobie → SO
Exo peu intense → pas de SO

SO mis en évidence par

- ↗ P° RL par RPE
- ↗ Dommages oxydatifs (surtout lipides ⇔ peroxydation lipidique)
- ↗ Activité des enzymes AO (SOD)

3.2- Mécanismes de production des RL lors de l'exercice

Les mécanismes dépendent du type d'exercice

Activité des enzymes pro-oxydantes (NADPHox ou NOX +++ et XO++)

Mitochondrie +

Plus faible qu'au repos

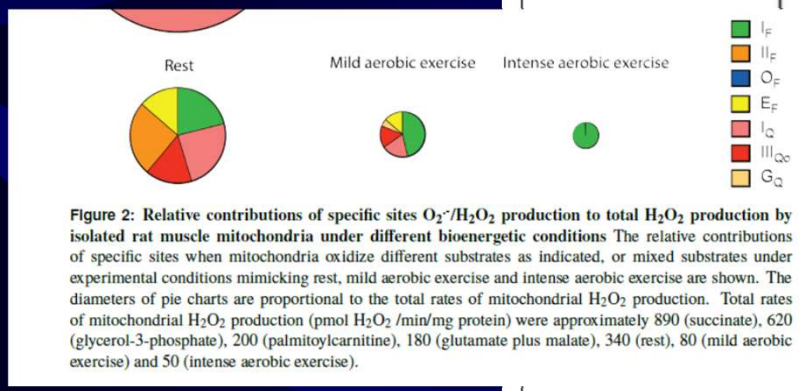
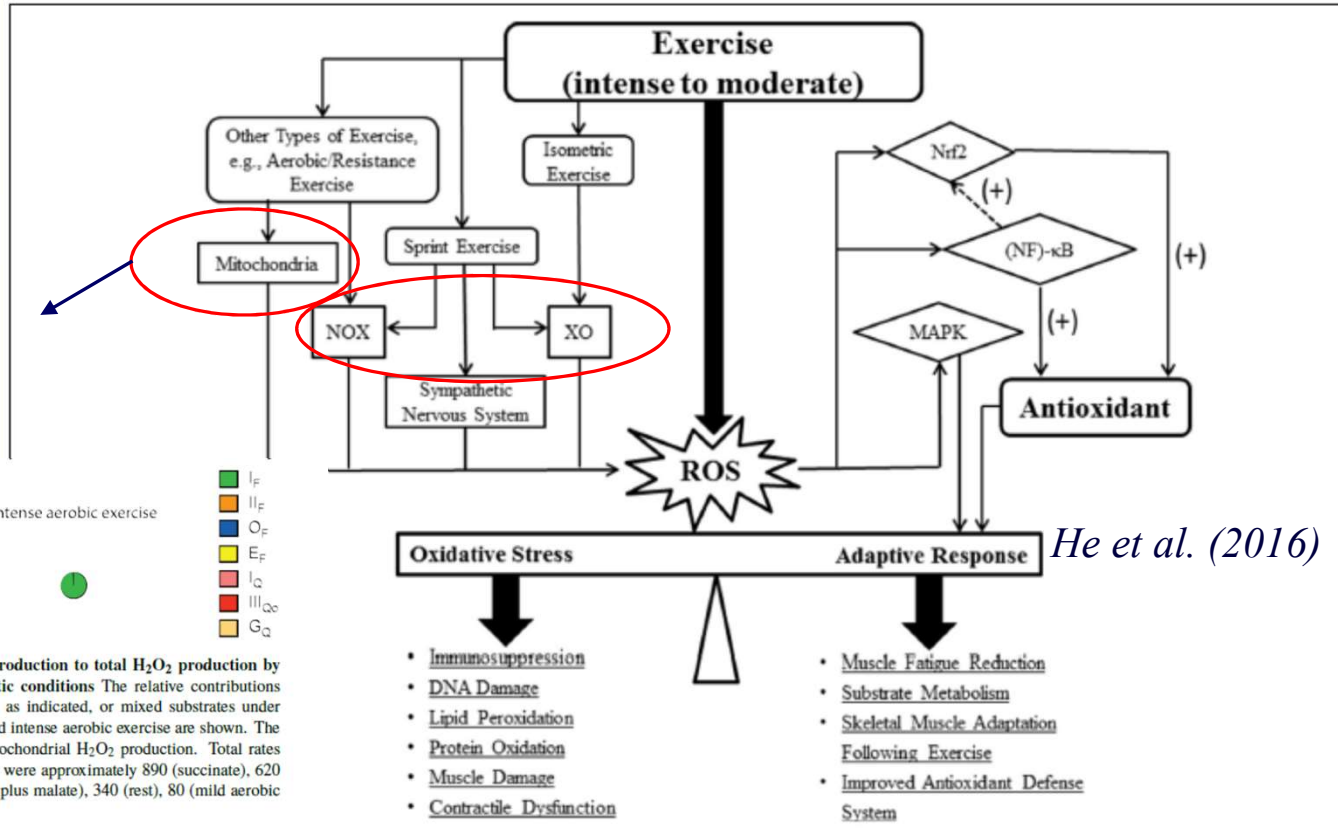


Figure 2: Relative contributions of specific sites $O_2^{\cdot-}/H_2O_2$ production to total H_2O_2 production by isolated rat muscle mitochondria under different bioenergetic conditions The relative contributions of specific sites when mitochondria oxidize different substrates as indicated, or mixed substrates under experimental conditions mimicking rest, mild aerobic exercise and intense aerobic exercise are shown. The diameters of pie charts are proportional to the total rates of mitochondrial H_2O_2 production. Total rates of mitochondrial H_2O_2 production (pmol H_2O_2 /min/mg protein) were approximately 890 (succinate), 620 (glycerol-3-phosphate), 200 (palmitoylcarnitine), 180 (glutamate plus malate), 340 (rest), 80 (mild aerobic exercise) and 50 (intense aerobic exercise).

Wong et al. (2017)



He et al. (2016)

FIGURE 1 | Schematic illustrating ROS generation during different types of exercise and their associated roles in adaptive response. The dash arrow represents an indirect effect. Abbreviations: reactive oxygen species (ROS); NADPH oxidase (NOX); xanthine oxidase (XO); mitogen-activated protein kinase (MAPK); nuclear erythroid 2 p45-related factor 2 (Nrf2); nuclear factor κB (NF-κB).

A l'exercice la part des ROS produite par la mito ↘ au profit des sources EC

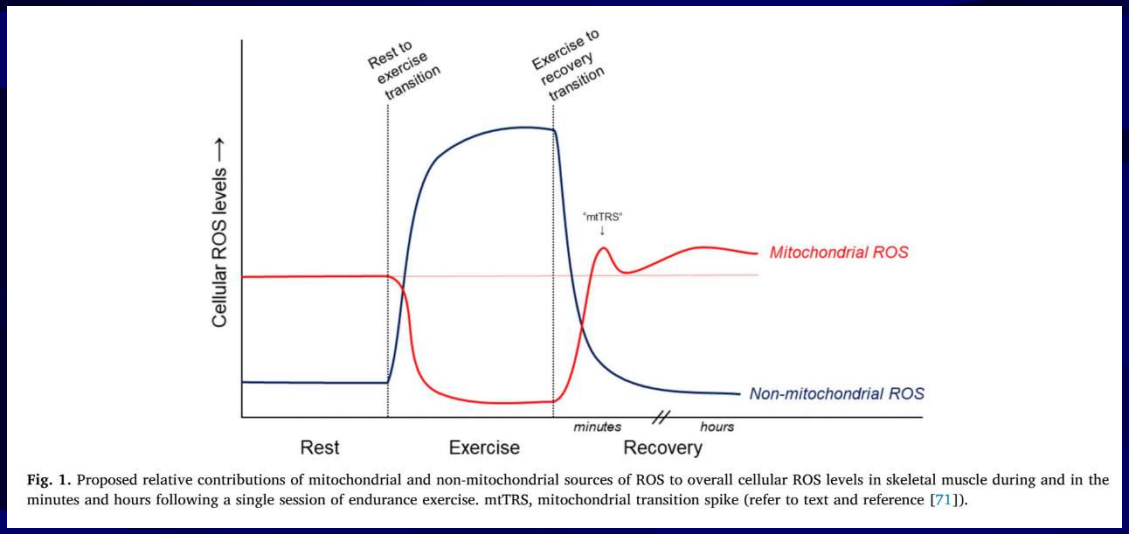
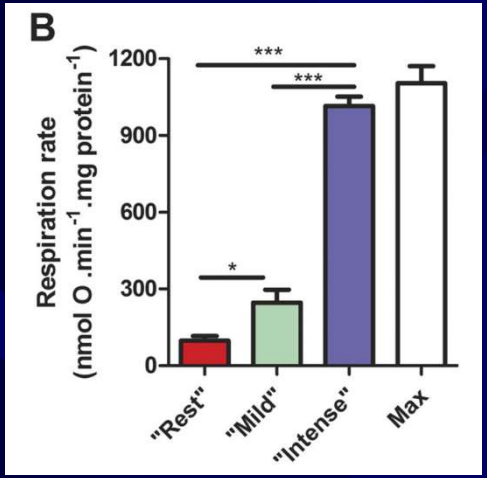
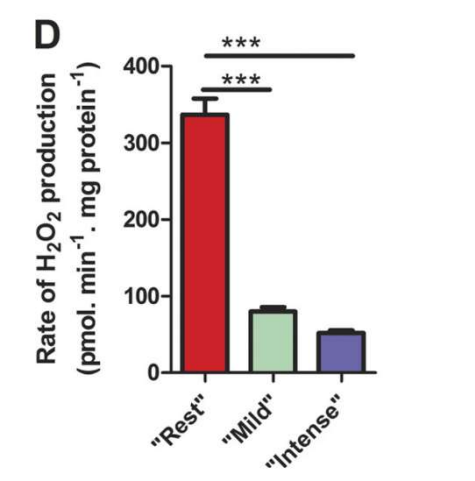


Fig. 1. Proposed relative contributions of mitochondrial and non-mitochondrial sources of ROS to overall cellular ROS levels in skeletal muscle during and in the minutes and hours following a single session of endurance exercise. mtTRS, mitochondrial transition spike (refer to text and reference [71]).

Mason et al. 2020

Mis en évidence sur cultures de cellule avec des milieux qui « miment » l'exercice





Goncalves et al. (2014)

3.3- Effet de l'entraînement

3.3.1- Entrainement aérobie

- Etudes longitudinales

➘ Marqueurs du SO

Lipides: Effet bénéfique de l'entraînement au repos et à l'exercice +++

Protéines: ➘ des protéines carbonylées *(Witt et coll. 1992, Sen et coll. 1997…).++*

ADN: Trop peu d'études *(Lui et coll. 2000, **Radàk et coll. 1999**).*

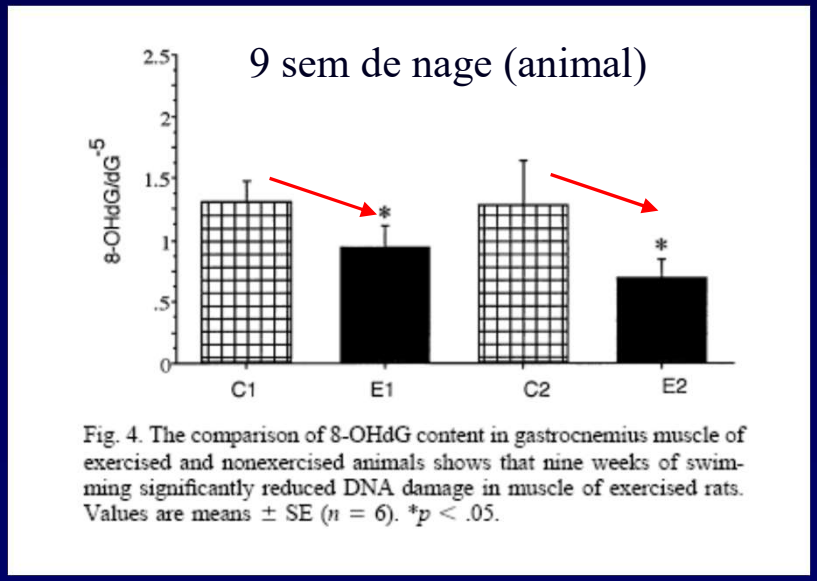
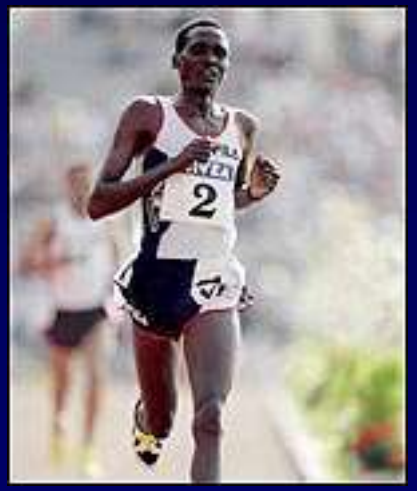


Fig. 4. The comparison of 8-OHdG content in gastrocnemius muscle of exercised and nonexercised animals shows that nine weeks of swimming significantly reduced DNA damage in muscle of exercised rats. Values are means ± SE ($n = 6$). *$p < .05$.



3.3- Effet de l'entraînement

- Etudes longitudinales

↗ Activité des enzymes antioxydantes

SOD: ↗ dans muscle *(Alessio et Goldfarb 1988, Ji et coll. 1988…).*

GPx: ↗ *(Criswell et coll. 1993, Lauglin et coll. 1990…).*

CAT: ➔

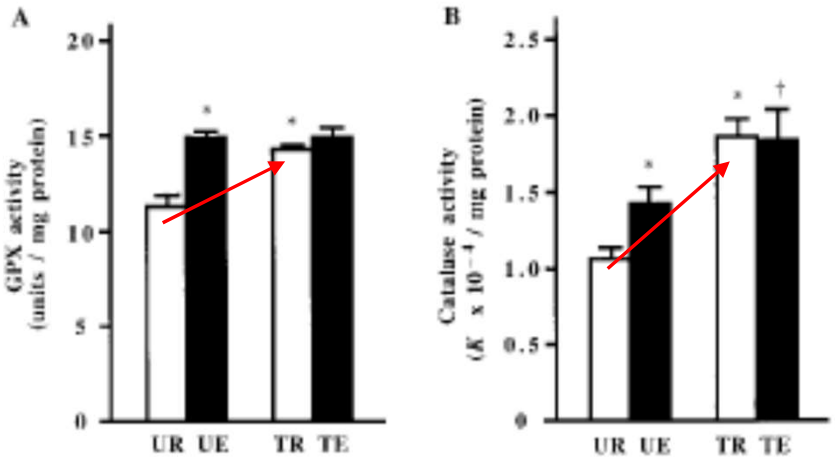
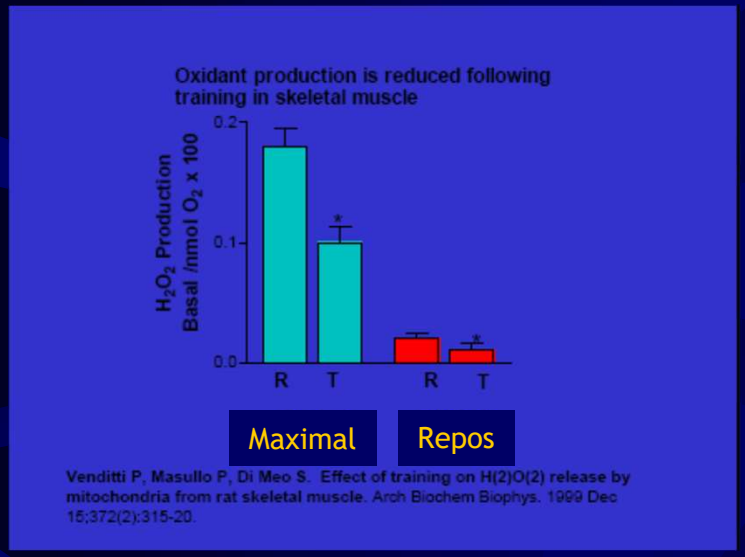


Figure 6. Glutathione peroxidase (GPX) activity (*A*) and catalase activity (*B*) in rat diaphragm. Values are mean ± SEM; n = 5 to 6 in each group. *Significantly different from UR: $p < 0.05$; †significantly different from UR: $p < 0.05$.

- *Etudes longitudinales*



Venditti P, Masullo P, Di Meo S. Effect of training on H(2)O(2) release by mitochondria from rat skeletal muscle. Arch Biochem Biophys. 1999 Dec 15;372(2):315-20.

Souris: 10 sem d'entraînement en natation

↘ P° ERON par mitochondrie au repos et à l'exo

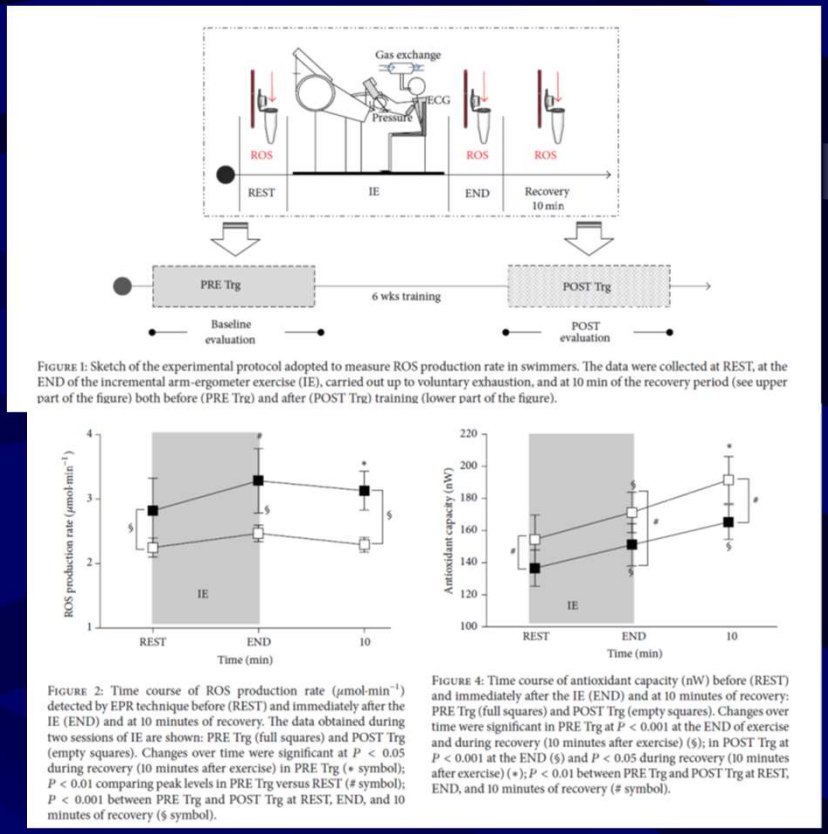


FIGURE 1: Sketch of the experimental protocol adopted to measure ROS production rate in swimmers. The data were collected at REST, at the END of the incremental arm-ergometer exercise (IE), carried out up to voluntary exhaustion, and at 10 min of the recovery period (see upper part of the figure) both before (PRE Trg) and after (POST Trg) training (lower part of the figure).

FIGURE 2: Time course of ROS production rate (μmol·min^{-1}) detected by EPR technique before (REST) and immediately after the IE (END) and at 10 minutes of recovery. The data obtained during two sessions of IE are shown: PRE Trg (full squares) and POST Trg (empty squares). Changes over time were significant at $P < 0.05$ during recovery (10 minutes after exercise) in PRE Trg (* symbol); $P < 0.01$ comparing peak levels in PRE Trg versus REST (# symbol); $P < 0.001$ between PRE Trg and POST Trg at REST, END, and 10 minutes of recovery ($ symbol).

FIGURE 4: Time course of antioxidant capacity (nW) before (REST) and immediately after the IE (END) and at 10 minutes of recovery: PRE Trg (full squares) and POST Trg (empty squares). Changes over time were significant in PRE Trg at $P < 0.001$ at the END of exercise and during recovery (10 minutes after exercise) ($); in POST Trg at $P < 0.001$ at the END ($) and $P < 0.05$ during recovery (10 minutes after exercise) (*); $P < 0.01$ between PRE Trg and POST Trg at REST, END, and 10 minutes of recovery (# symbol).

Homme: entraînement en natation

Mrakic-Sposta et al. (2015)

↘ P° ERON par l'exercice chez E vs NE

- Etudes Transversales (Hommes E vs NE)

[marqueurs de la peroxydation lipidique]$_{plasm.}$ **E > NE**

(Mena et al. 1991, Balakrishan et Anurhada 1998, Marzatico et al. 1997, Santos-silva et al. 2001)

AO et [GSH]$_{sg.}$ **E < NE** *(Balakrishan et Anurhada 1998)*

Balakrishnan and anuradha et coll. (1998)

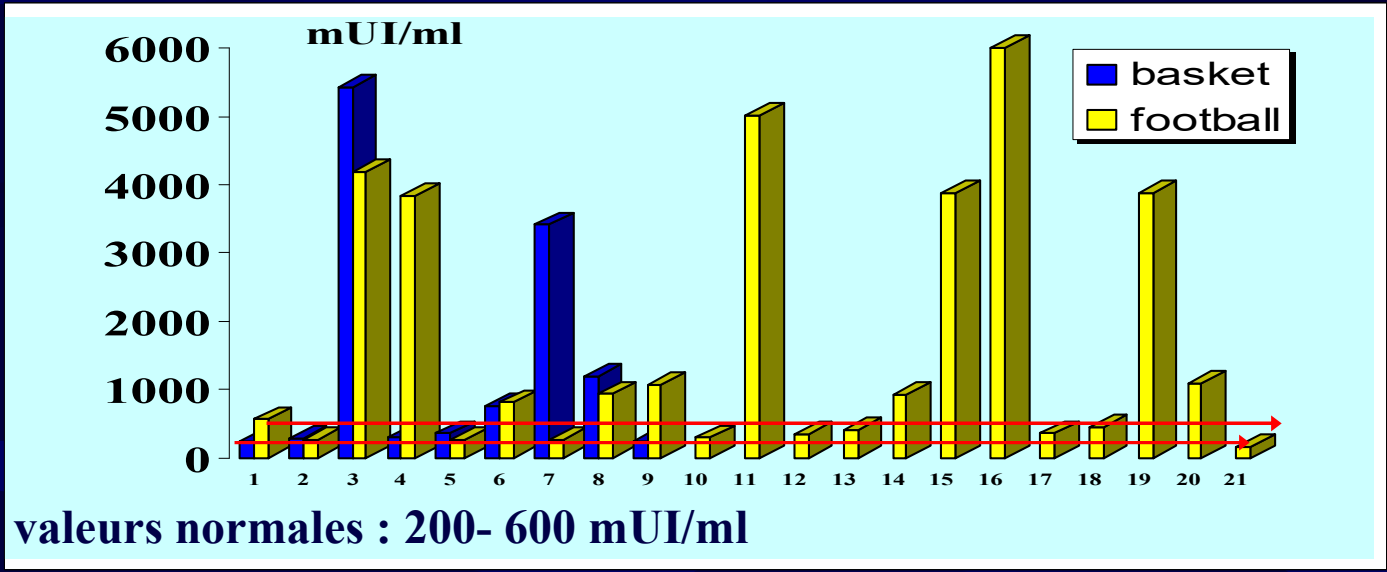
	Controls ($n = 27$)	Sportsmen ($n = 26$)
TBARS (μmoles l^{-1})	3·69 ± 1·03	4·65 ± 1·16*
Diene conjugates (A 233 215)	1·08 ± 0·01	1·45 ± 0·02†
α-Tocopherol (mg dl^{-1})	1·27 ± 0·44	1·19 ± 0·49
Ascorbic acid (mg dl^{-1})	1·59 ± 0·54	0·83 ± 0·55*
Reduced glutathione (mg dl^{-1})	54·80 ± 11·20	45·54 ± 12·87†
Ceruloplasmin (mg dl^{-1})	16·24 ± 4·60	29·10 ± 9·08*

SO E > NE

AO E < NE

- *Etudes Transversales (Hommes E vs NE)*

Marqueur du SO chez des footballeurs et basketteurs professionnels belges (Pincemail et al., 2000)

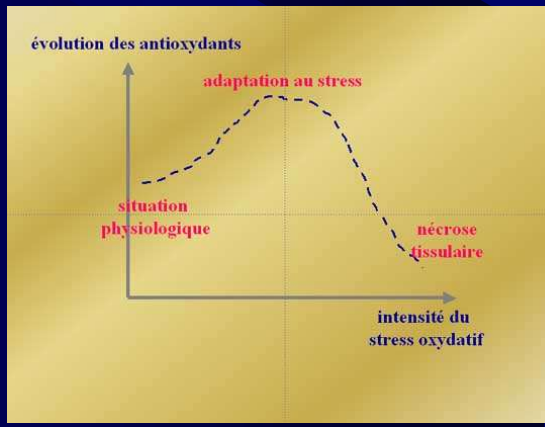


Adaptation au stress : expression de la SOD

(Mena et al., 1991)

sédentaires	**275 +/- 39 µg/g Hb**
cyclistes amateurs	**588 +/- 230µg/g Hb**
cyclistes professionnels	**323 +/- 67µg/g Hb**

Tour d'Espagne



La SOD s'adapte lorsque le l'intensité (stress) est modéré

- Relation avec VO_2 max

↗ [marqueurs de la peroxydation lipidique]$_{plasm.}$

(Koska et al. 1998, Groussard et al. 2003)

↘ [Antiox. non enzymatiques]$_{plasm.}$ et [GSH]$_{sg}$

(Bergholm et coll. 1999, Robertson et coll. 1991, Sharpe et coll. 1996, Balakrishan et Anuradha 1998)

↗ VO_2max

⇒ Effet aggravant de l'entraînement vis à vis du SO ?

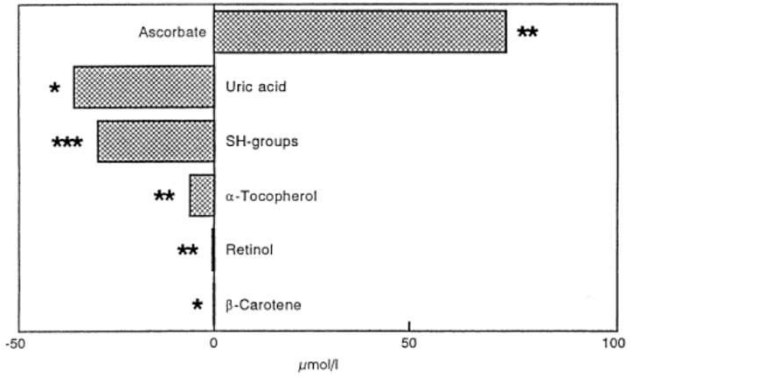


Fig. 3. Changes in major circulating antioxidants by training. $*P < 0.05$, $**P < 0.01$, $***P < 0.001$ for change before vs. after training.

Bergholm et coll. 1999

SO chronique ?

Hypothèse pour expliquer ces divergences:

Entraînement inadapté ou excessif

+

Faible statut antioxydant

} **SO chronique ?**

Les adaptations enzymatiques bénéfiques induites par l'entraînement seraient insuffisantes pour protéger contre le SO

Table VII. Human studies on effects of aerobic training on markers of oxidative stress

Study (year)	Activity	Subjects	Markers	Effect
Ohno et al.[191] (1988)	Running >5km – 6 × wk – 10wk	7 UT	GSH – GPX – SOD	↔
			GR – CAT	↑
Acconinotti et al.[192] (1991)	Cycling (follow up)	12 T	GSH (after intensive training)	↑
			GSH (after long-term intensive training)	↓
Tessier et al.[112] (1995)	Running <80% $\dot{V}O_{2max}$ – 3 × wk – 10wk	24 T	GSH – GSSG	↓
			GPX	↑
Marzatico et al.[168] (1997)	Running (half-marathon)	6 T vs 6 UT	MDA – CD	↑
	Blood samples at rest		SOD – GPX – CAT	↑
Bergholm et al.[193] (1999)	Running 60 min at 70–80% $\dot{V}O_{2max}$ 4 × week – 3mo	9 T	TRAP – vit C	↑
			UA – thiols – tocopherol – vit A	↓
Liu et al.[58] (1999)	Running (marathon)	11 VT vs 10 UT	LDL oxidation	↓
	Post-exercise blood samples		TRAP	↑
			UA – vit E – vit C – vit A	↔
Miyazaki et al.[169] (2001)	Running	9 UT	TBARS	↓
	60 min at 80% $\dot{V}O_{2max}$		Protein carbonyls	↔
	5 × wk – 12wk		SOD – GPX	↑
			CAT	↔
Elosua et al.[190] (2003)	Running	17 UT	LDL oxidation	↓
	50 min – 5 × wk – 16wk		LP	↑
	Blood samples after 30 min aerobic exercise		SOD – GSH	↑
Palazzetti et al.[153] (2003)	Triathlon – overload training (4wk)	9 VT		
	Blood samples at rest or after a duathlon)			
			At rest	
			GSH – SOD	↔
			CK – myoglobin – TBARS	↔
			GPX	↑
			TAC	↓
			Post-exercise	
			CK – myoglobin – TBARS	↑
			TAC	↓

CAT = catalase; **CD** = conjugated dienes; **CK** = creatine kinase; **GPX** = glutathione peroxidase; **GR** = glutathione reductase; **GSH** = glutathione; **GSSG** = oxidised glutathione; **LDL** = low-density lipoprotein; **LP** = lag phase; **MDA** = malondialdehyde; **SOD** = superoxide dismutase; **T** = trained; **TAC** = total antioxidant capacity; **TBARS** = thiobarbituric reactive substances; **TRAP** = total radical antioxidant potential; **UA** = uric acid; **UT** = untrained; **vit** = vitamin; **VT** = very trained; $\dot{V}O_{2max}$ = maximum oxygen consumption; ↓ indicates decrease; ↑ indicates increase; ↔ indicates no change (stable).

Finaud et al. 2006

3.3.2- Effet de l'entraînement anaérobie

- Beaucoup moins étudié et études bcp + récentes
- L'entraînement en sprint chez l'animal et l'homme => ↗ l'activité des enzymes antioxydantes (adaptation de l'organisme).

Finaud et al. 2006

Table VIII. Human studies on effects of anaerobic training on markers of oxidative stress

Study (year)	Activity	Subjects	Markers	Effect
Hellsten et al.[201] (1996)	15 × 10 sec of anaerobic exercise (50 sec rest) 3 × wk – 7wk	11 UT	GPX – CAT	↑
			SOD	↔
Ortenblad et al.[166] (1997)	Jump training: blood samples at rest and after 6 × 30 sec jumping	8 T vs 8 UT	CK (after exercise)	↓
			MDA (after exercise)	↔
			SOD – GPX (at rest)	↑
			CAT (at rest)	↔
Marzatico et al.[168] (1997)	Running (sprint): blood samples at rest	6 T vs 6 UT	MDA	↑
			CD	↔
			SOD – GPX	↑
			CAT	↓
Rall et al.[199] (2000)	Progressive resistance strength training 12wk	8 UT elderly, 8 T and 8 UT with rheumatoid arthritis	8-OHdG (in both groups)	↔
Vincent et al.[200] (2002)	Muscular exercise (50–80% 1RM) 3 × wk – 6mo	84 UT elderly	TBARS – LH	↓
			Thiols	↑

8-OHdG = 8-hydroxy-2′-deoxyguanosine; **CAT** = catalase; **CK** = creatine kinase; **GPX** = glutathione peroxidase; **LH** = lipid hydroperoxide; **MDA** = malondialdehyde; **RM** = repetition maximum; **SOD** = superoxide dismutase; **T** = trained; **TBARS** = thiobarbituric reactive substances; **UT** = untrained; ↓ indicates decrease; ↑ indicates increase; ↔ indicates no change (stable).

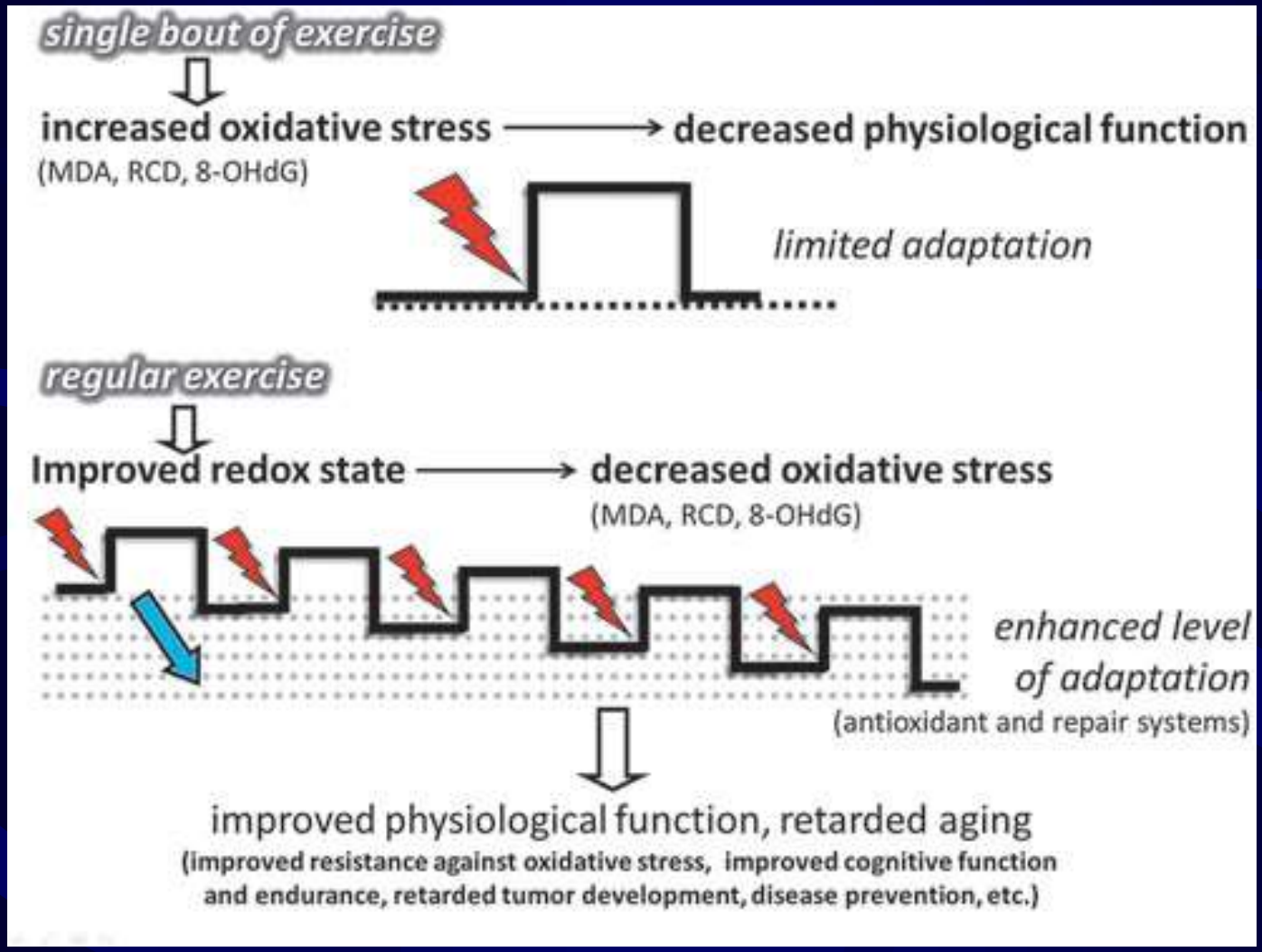
Bilan SO et entraînement

Etudes longitudinales : Effets positifs de l'entraînement (↘ des marqueurs du SO , car ↘ de la P° RL et ↗ activité des enzymes AO)

Etudes transversales: résultats moins nets surtout chez SHN où on peut parfois observer un SO chronique (entraînement trop I / apport en AO?)

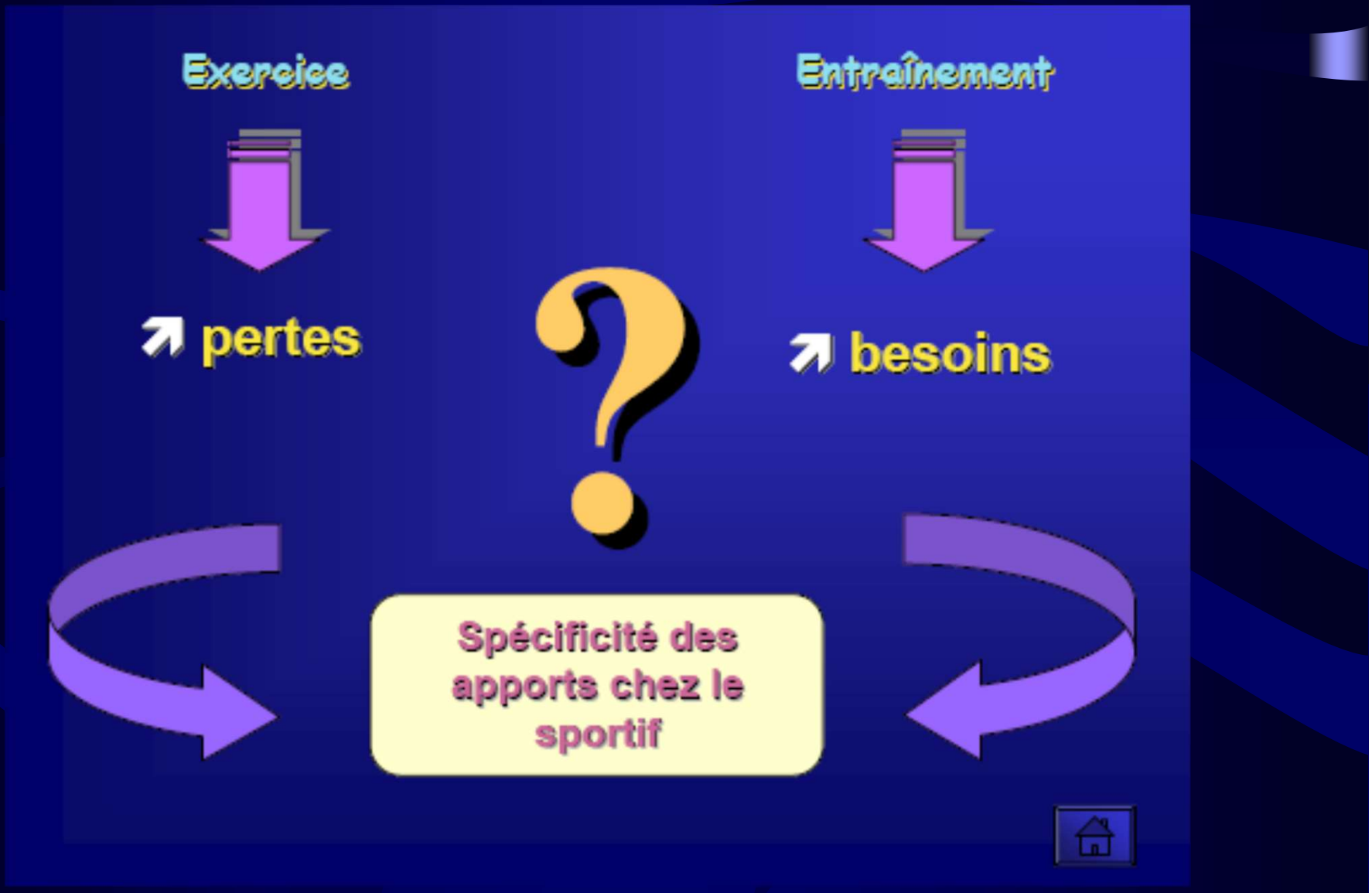
Entraînement anaérobie: études plus récentes et effets identiques aérobie

Résumé SO et exercice / entraînement



1 *Radak et al. 2013*

4- Complémentation en AO à l'exercice/entraînement



4.1- Effet d'une carence d'apport en AO

- ☹ ↘ [vitamines antioxydantes] plasmatiques et tissulaires

(Salminen et coll., 1984; Kelly et coll., 1996; Packer et coll., 1986)

- ☹ ↗ Dommages oxydatifs

(Tiidus et Houston, 1994; Dillard et coll. 1997; Dabvies et coll. 1982)

- ☹ ↘ Performances sportives

(Packer et coll., 1986)

4.1- Effet d'une carence d'apport en AO

Pas d'expérience de carence d'apport chez l'homme

- **Etude en fonction du statut initial**

Pachalis et al. (2014) :

Eur J Nutr. 2014 Dec 20. [Epub ahead of print]

Low vitamin C values are linked with decreased physical performance and increased oxidative stress: reversal by vitamin C supplementation.

Paschalis V[1], Theodorou AA, Kyparos A, Dipla K, Zafeiridis A, Panayiotou G, Vrabas IS, Nikolaidis MG.

Plus faible VO_{2max} carencés vs non carencés

- La complémentation en vit C ↗ VO_{2max} chez carencés

Marqueurs SO élevés + au repos et en réponse à l'exercice chez carencés vs non carencés

- La complémentation en vit C ↘ SO de repos dans les 2 groupes mais de manière + impte chez carencés
- La complémentation en vit C ↘ SO post-exo chez carencés

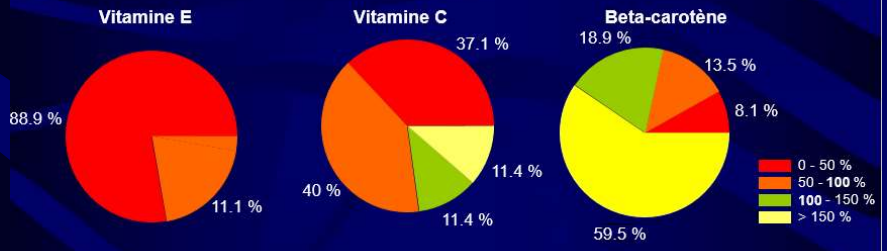
4.2- Apport complémentaire en AO chez l'Homme sur le statut pro/antioxydant

- Complémentation en AO largement pratiquée chez les sportifs *(Sobal et al. 1994; Maughan et al. 2007, Petroczi et al. 2008, Braun et al. 2009)*

- Au préalable:

1)- Vérifier apports par questionnaires alimentaires → souvent faibles (Vit E+++, vit C+, ok pour B-caro)

Apport en vitamine (mg/j)	Population totale (n=37)	ANC (mg/j)	Test-t apparié
Vitamine E	4.6 (0.4)	18.6 (0.95)***	p<0.001
Vitamine C	110.1 (8.7)	164.2 (8.15)***	p<0.001
β-carotène	5.7 (0.6)	2.8 (0.08)***	p<0.001



- Apport vitamine E inférieur à la norme → 100 % ne couvrent pas l'apport
- Apport vitamine C inférieur à la norme → 77 % ne couvrent pas l'apport
- Apport β-carotène supérieur aux normes → 20 % ne couvrent pas l'apport

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	Sauteurs à Ski (Rankinen et coll., 1998)	Patineurs (Ziegler et coll., 1998)	Activités variées (Guilland et coll., 1998)	Triathloniens (Margaritis et coll., 1997)	Triathloniens (Palazzetti et coll., 2004)
Age (années)	*19,7 ± 3,6*	*16,5 ± 1,6*	*19,7 ± 0,6*	*33,2 ± 9,8*	*33,2 ± 9,8*
Vitamine E (mg.j^{-1})	11,0 ± 3,2	3,65 ± 4,1	12,6 ± 0,1	11 ± 4	11 ± 4
		INSUFFISANT			
Vitamine C (mg.j^{-1})	120 ± 84	203 ± 202	94 ± 2	187 ± 76	168 ± 82
		VARIABLE			
Rétinol (mg.j^{-1})		1,7 ± 1,1	1,0 ± 0,1	1,2 ± 0,3	
			SUFFISANT		
β-carotène (mg.j^{-1})			3,8 ± 0,1		
			SUFFISANT		

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2)- Vérifier le statut antioxydant plasmatique (déficit ou carence)

4.2- Apport complémentaire en AO chez l'Homme

4.2.1 - Effets de la complémentation sur la performance

😐 **Pas de modification des performances physiques pour les vitamines**

(Sumida et coll., 1989; Heilgheim et coll., 1979; Gey et coll., 1970; Keith et Merryl, 1983)

Table 2
Effects of antioxidants on skeletal muscle performance: human studies (*24*)

Study	Treatment	Test	Performance
Lawrence et al., 1975 (*26*)	Vitamin E	500 meters swimming	No effect
Sumida et al., 1989 (*27*)	Vitamin E	VO_{2MAX}	No effect
Rokitzki et al., 1994 (*28*)	Vitamin E	Incremental exercise	No effect
Snider et al., 1992 (*29*)	Vitamin E	Time to exhaustion	No effect
	Coenzyme Q	70% del VO_{2MAX}	
Reid et al., 1994 (*18*)	NAC	Low-frequency, stimulation of tibialis anterior muscle	Improved

NAC = *N*-acetylcysteine.

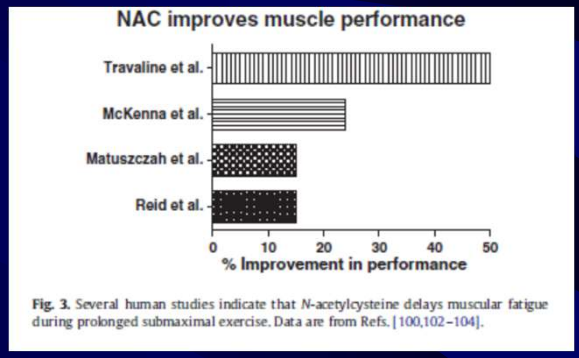

Fig. 3. Several human studies indicate that *N*-acetylcysteine delays muscular fatigue during prolonged submaximal exercise. Data are from Refs. [100,102–104].

S.K. Powers et al. / Free Radical Biology & Medicine 51 (2011) 942–950

Table 1 Results of the studies with endurance trained volunteers supplemented with vitamins A, C, and E

Study	Experimental design	Sample	Duration	Suplementation protocol			Result	
				Vitamin A	Vitamin C	Vitamin E	Ergogenic	Ergolytic
Tauler et al. [6]	Randomized, double-blind	15 athletes	90 d*	30 mg (β-caroten)	1000 mg	500 mg	↔	↔
Gauche et al. [9]	Randomized, double-blind	22 athletes	21 d (pre-exercise) + 2 dias (post-exercise)	6 mg (β-caroten)	200 mg	32 mg	↑	N/R
Nielsen et al. [10]	Randomized, double-blind, cross-over	15 athletes	28 d	-	400 mg	180 mg	↔	↔
Patil et al. [11]	Randomized, double-blind	37 athletes	21 d	-	-	200 mg	↔	↔
Louis et al. [12]	Randomized, double-blind	16 athletes	21 d	17.1 mg (β-caroten)	319.2 mg	48 mg	↑	N/R

* Vitamin C supplementation occurred only in the last 15 days of the study; ↑ Improved exercise performance; ↔ No results on exercise performance; N/R – not reported.

Nikolaidis et al. (2012)

4.2- Apport complémentaire en AO chez l'Homme

4.2.1 - Effets de la complémentation sur la performance

😐 **Pas de modification des performances physiques pour les vitamines**

VO2max

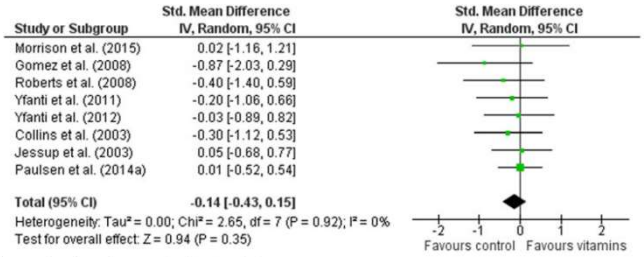

Figure 4. Forest plots showing the effect of vitamin C and/or E on $\dot{V}O_{2max}$.

Endurance

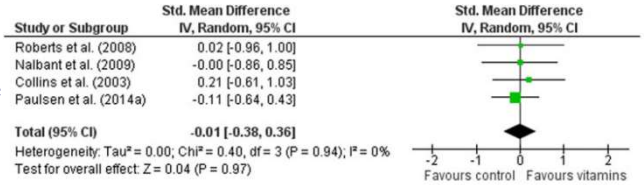

Figure 5. Forest plots showing the effect of vitamin C and/or E on endurance performance. Data from Roberts et al. (2011) is a pooled average of the 3 performance tests described in Table 1.

Force

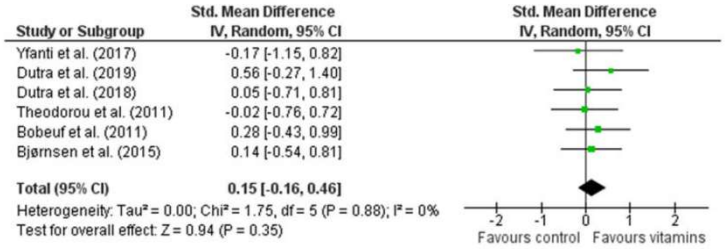

Figure 7. Forest plots showing the effect of vitamin C and/or E on muscle strength. Data from Bobeuf et al. (2011), Bjørnsen et al. (2016), and is a pooled average of the tests shown in Table 2.

Confirmation avec Méta analyse de Clifford et al. (2019) avec Vit C et E

4.2- Apport complémentaire en AO chez l'Homme

4.2.1 - Effets de la complémentation sur la performance

😐 **Pas de modification des performances physiques pour les vitamines**

International Journal of *Environmental Research and Public Health*

MDPI

Review

Antioxidants and Exercise Performance: With a Focus on Vitamin E and C Supplementation

Madalyn Riley Higgins [1], Azimeh Izadi [2] and Mojtaba Kaviani [1,*]

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check for updates

Abstract: Antioxidant supplementation, including vitamin E and C supplementation, has recently received recognition among athletes as a possible method for enhancing athletic performance. Increased oxidative stress during exercise results in the production of free radicals, which leads to muscle damage, fatigue, and impaired performance. Despite their negative effects on performance, free radicals may act as signaling molecules enhancing protection against greater physical stress. Current evidence suggests that antioxidant supplementation may impair these adaptations. Apart from athletes training at altitude and those looking for an immediate, short-term performance enhancement, supplementation with vitamin E does not appear to be beneficial. Moreover, the effectiveness of vitamin E and C alone and/or combined on muscle mass and strength have been inconsistent. Given that antioxidant supplements (e.g., vitamin E and C) tend to block anabolic signaling pathways, and thus, impair adaptations to resistance training, special caution should be taken with these supplements. It is recommended that athletes consume a diet rich in fruits and vegetables, which provides vitamins, minerals phytochemicals, and other bioactive compounds to meet the recommended intakes of vitamin E and C.

Keywords: sport performance; altitude training; resistance exercise; dietary supplements; free radicals

Confirmation avec revue de question de 2020 avec Vit C et E

Effets bénéfiques en altitude

4.2- Apport complémentaire en AO chez l'Homme

4.2.1 - Effets de la complémentation sur la performance

☺ **Effet Positif de la NAC**

Table 2
Effects of antioxidants on skeletal muscle performance: human studies (*24*)

Study	Treatment	Test	Performance
Lawrence et al., 1975 (*26*)	Vitamin E	500 meters swimming	No effect
Sumida et al., 1989 (*27*)	Vitamin E	VO_{2MAX}	No effect
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	Coenzyme Q	70% del VO_{2MAX}	
Reid et al., 1994 (*18*)	NAC	Low-frequency, stimulation of tibialis anterior muscle	Improved

NAC = *N*-acetylcysteine.

☺ **Effet Positif des Flavonoides (forme la + abondante des polyphénols)**

nutrients MDPI

Review

Does Flavonoid Consumption Improve Exercise Performance? Is It Related to Changes in the Immune System and Inflammatory Biomarkers? A Systematic Review of Clinical Studies since 2005

Patricia Ruiz-Iglesias [1,2], Abril Gorgori-González [1], Malén Massot-Cladera [1,2], Margarida Castell [1,2,3,*] and Francisco J. Pérez-Cano [1,2]

Evaluation de la consommation des flavonoïdes (purs ou aliments enrichis ou a forte teneur) la performance lors des 15 dernières années lorsqu'ils sont consommés pendant au moins sept jours.

Table 2. Summary of the included studies assessing the effects of flavonoid-enriched extracts on exercise performances.

Family Reference	Flavonoid source	Control Groups	Study Design	Number of Participants (Female + Male)	Mean Age of Participants (Years)	Dosage	Exercise	Performance Variable	Effect
Flavanols									
[74]	Apple extract (Applephenon®)	Crystalline cellulose capsules	Db RPCCT	9 + 9	39.1 ± 9.1	720 mg/d procyanidins for 7 d	Cycling	Change of maximum velocity	Improvement
[85]	Green tea extract	Carbohydrate-containing drink	Db RPCCT	0 + 9	32.2 ± 2.1	159 mg/d catechins for 3 wks	Cycling	Time for 30 km trial	NS
[28]	Green tea extract	Microcrystal-line cellulose capsules	Db RPCCT	0 + 16	21.6 ± 1.5	800 mg/d catechins for 4 wks	Cycling	Peak power, mean power, total work output	NS
[94]	Decaffeinated green tea extract	Corn flour capsules	Db RPCT	0 + 14	21.4 ± 0.3	400 mg/d EGCG for 4 wks	Cycling	Distance	Improvement
[95]	Green tea extract	Sports drink	Db RPCCT	0 + 14	33.9 ± 7.4	570 mg/d catechins for 8 wks	Cycling	Leg extension strength	Improvement
[96]	Green tea extract	Starch capsules	Db RPCT	0 + 40	21.0 ± 1.0	207 mg/d catechins for 4 wk	Running	Time to exhaustion	NS
[97,98]	Blueberry-green tea-polyphenol soy protein complex	Soy protein complex with non-polyphenolic food coloring	Db RPCT	13 + 18	33.7 ± 6.8 (SUP) 35.2 ± 8.7 (PL)	1001 mg/d flavanols for 17 d	Running in a treadmill for 2.5 h	Distance covered	NS

↗ Possible des performances physiques avec anthocyanine surtout

Ruiz-Iglesias et al. 2021

4.2- Apport complémentaire en AO chez l'Homme

4.2.2 - Effets de la complémentation sur le statut pro/antioxydant

☺ ↗ **[vitamines antioxydantes] plasmatiques** *(Ashton et coll., 1999; Witt et coll., 1992)*

$[Vit\ C]_{plasm}$ saturable à 200mg/j *(Rousseau et al. 2004)*

☺ ↘ **Dommages oxydatifs**

- **Au repos** *(Welch et coll., 1999; Astley et coll., 1999; Itoh et coll., 2000)*
- **A l'exercice** *(Ashton et coll., 1999; Alessio et coll., 1997; Sürmen-Gür et coll., 1999)*

Vrai surtout pour la vitamine E

Études moins unanimes pour la vitamine C

Résulats parfois contradictoires: type d'antiox, dose, aptitude physique des sujets, type d'exercice...

4.2- Apport complémentaire en AO chez l'Homme

4.2.2 - Effets de la complémentation sur le statut pro/antioxydant

- Anciennes études qui n'ont mesuré que le SO après complémentation en vit E :
  - Chez l'Al : résultats parfois contradictoires mais en général ↘ SO *(Brady et al. 1979, Goldfarb et al. 1994, Kumar et al. 1992)*
  - Chez l'Hô: ↘ dommages oxydatifs au repos et en réponse à l'exercice *(Sumida et al. 1989, Meydani et al. 1994, Goldfarb et al. 1989).*

☹ Mais déja des études avaient montré effets néfastes de fortes doses vit E seules!!!

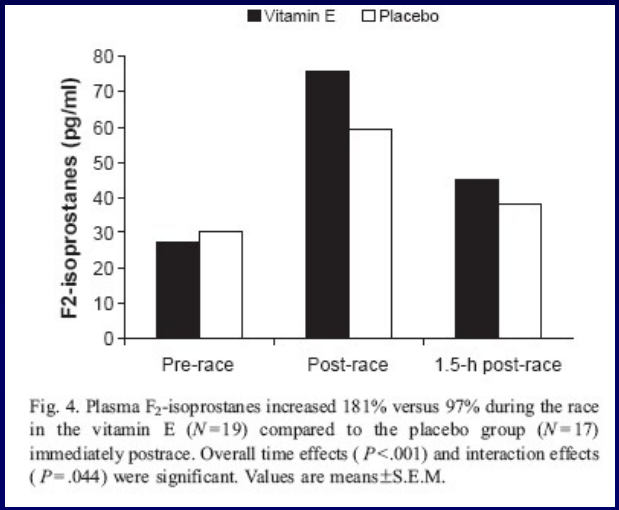


Fig. 4. Plasma F_2-isoprostanes increased 181% versus 97% during the race in the vitamin E (N=19) compared to the placebo group (N=17) immediately postrace. Overall time effects (P<.001) and interaction effects (P=.044) were significant. Values are means±S.E.M.

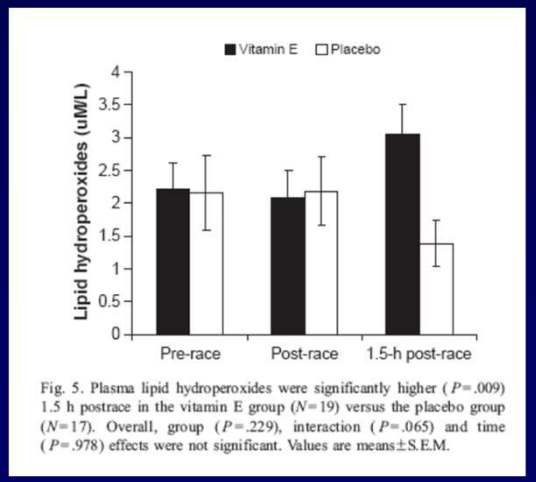


Fig. 5. Plasma lipid hydroperoxides were significantly higher (P=.009) 1.5 h postrace in the vitamin E group (N=19) versus the placebo group (N=17). Overall, group (P=.229), interaction (P=.065) and time (P=.978) effects were not significant. Values are means±S.E.M.

McAnulty et al. 2005

2 mois vit E (536mg/j) chez triathlètes => ↗ SO (déséquilibre chaine des antiox)

4.2- Apport complémentaire en AO chez l'Homme

4.2.2 - Effets de la complémentation sur le statut pro/antioxydant

- Complémentation en vit C : Etudes moins nombreuses que vit E et moins unanimes.
  - Homme :
    - ↘ dommages oxydatifs en réponse à l'exo. (*Kaminski et Boal 1992, Ashton et coll. 1999).*
    - Aucun effet supplémentation en vit C après 30 min d'exercice. *(Alessio et coll. 1997)*

Fig. 1. Pre- (*A*) and postexercise (*B*) electron spin resonance (ESR) spectra of α-phenyl-*tert*-butylnitrone (PBN) adduct in human plasma after maximal aerobic exercise (control phase) without ascorbic acid supplementation.

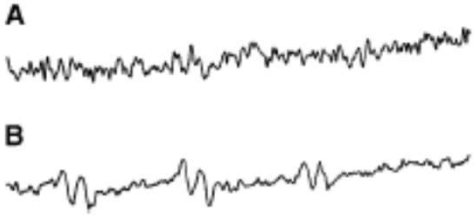


Fig. 2. Pre- (*A*) and postexercise (*B*) ESR spectra of PBN adduct in human plasma after supplementation with ascorbic acid.

Table 1. *Effect of exercise on in vivo free radical production with and without ascorbic acid supplementation*

	Control Group (n = 10)			Supplemented Group (n = 10)		
	Preexercise	Postexercise	P value	Preexercise	Postexercise	P value
PBN adduct	0.05 ± 0.02	0.19 ± 0.03	P = 0.002	0.02 ± 0.01	0.04 ± 0.02	NS
LH, μmol/l	1.14 ± 0.06	1.62 ± 0.19	P = 0.005	1.12 ± 0.21	1.12 ± 0.08	NS
MDA, μmol/l	0.70 ± 0.04	0.80 ± 0.04	P = 0.0125	0.63 ± 0.07	0.68 ± 0.05	NS

Values are means ± SE; n = no. of subjects. Effect of ascorbic acid supplementation on resting and postexercise intensity of electron spin resonance (ESR) signal of α-phenyl-*tert*-butylnitrone (PBN) adduct (measured in arbitrary units) compared with controls, together with free radical-mediated lipid peroxidation, as measured by lipid hydroperoxides (LH) and malonyldialdehyde (MDA). Acute ascorbic acid supplementation prevented any significant increase in any of oxidative stress-related parameters. In the present study, there was no correlation between postexercise ESR signal intensity and maximal oxygen uptake. There was, however, a positive correlation between lipid peroxidation and ESR, which did not reach statistical significance. Additionally, there was a small inverse correlation between plasma ascorbic acid and PBN adduct level, which, again, did not reach statistical significance, although it is suggested that this issue requires further investigation. NS, not significant.

- Complémentation combinée en antioxydants :
  - Plus efficace que la supplémentation d'un antioxydant seul.
  - *Kanter et coll. (1993) :* 5 sem de supplémentation en vit E (666 mg/j), β-carotène (37.5 mg/j) et vit C (1250 mg/j): ↘ SO au repos et en réponse à l'exercice.
  - Pincemail et al. (2001): randonnée dans l'Himalaya

placebo **antioxydant**

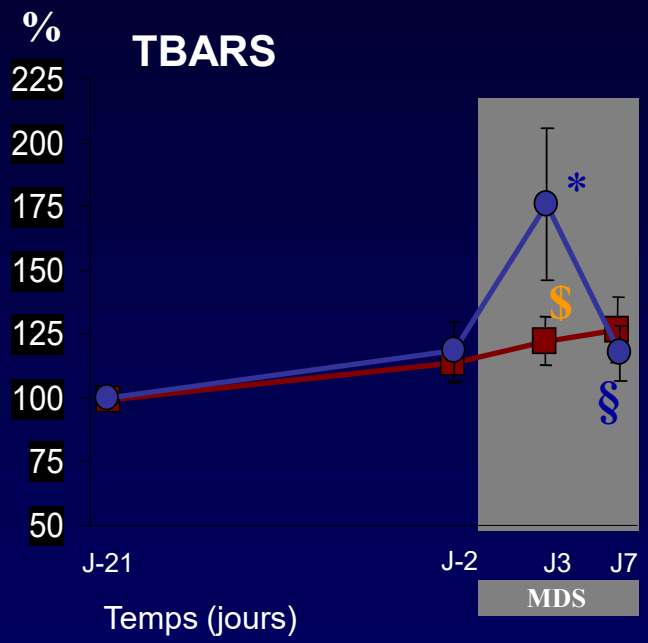
	placebo		antioxidant	
	before	after	before	after
vitamine C (µg/mL)	10,42 +/- 3,54	7,36 +/- 4,42	9,98 +/- 1,89	9,43 +/- 1,69
vitamine E (µg/mL)	9,51+/- 1,59	7,55 +/- 0,13	9,20 +/- 2,75	9,00 +/- 3,73
vitE/choles (mg/g)	5,56 +/- 0,53	4,88 +/- 1,79	4,36 +/- 1,13	4,33 +/- 1,03
sélénium	105,25 +/- 7,80	95,25 +/- 4,79	106,40 +/- 10,11	114,0 +/- 9,63
SOD (UI/g Hb)	611 +/- 25	738 +/- 82	649+/- 86	806 +/- 88
GPx (UI/g Hb)	71,20 +/- 20,22	88,50 +/- 28,74	77,00+/- 13,04	94,00 +/- 15,00
peroxydes lipidiques (µmol/L)	273,25 +/- 53,56	476,33 +/- 251,00	206,8 +/- 34,02	237,00 +/- 54,96

↗ Vitamines AO et ↘ marqueur SO

- *Machefer et coll. (2007)*: supplémentation vitamines et minéraux “physiologique”

Isoxan endurance lors du marathon des sables

Complémenté Placebo



➢ **[TBARS] : Limite l’augmentation des TBARS à J3**

La complémentation limite la peroxydation lipidique

* Significativement différent de J-21
£ Significativement différent de J-2
$ Significativement différent entre C et P

➢ **[TBARS] : ↗ à J3 + retour aux valeurs basales à J7**

Adaptation naturelle pour les placébo (J7 id)

4.3- Apport complémentaire en « aliments » riches en AO (phytonutriments)

- « Aliments » contenant de fortes doses de phytonutriments O
  - ↘ du SO au repos

(Allgrove et al. 2011; Davison et al. 2012)

(Howatson et al. 2010)

(Iwasa et al. 2013)

(MirandaVilela et al., 2010)

(Harms-Ringdahl, Jenssen, and Haghdoost 2012)

(Tartibian and Maleki 2012)

⇨ Manger des aliments riches en phytonutriments AO diminue le SO

Les phytonutriments sont des nutriments issus des végétaux, présents à l'état naturel dans les aliments d'origine végétale. On retrouve principalement des composés phénoliques avec les polyphénols ou les flavonoïdes et des caroténoïdes. Les phytonutriments sont réputés pour leur fort pouvoir AO, permettant ainsi de lutter contre le SO. Ils sont aussi réputés pour leurs bienfaits sur la santé (action sur le système immunitaire, cardiovasculaire et nerveux).

4.3- Apport complémentaire en « aliments » riches en AO (phytonutriments)

- ↘ du SO en reponse à l'exercice

Zeng et al. (2021)

Table 1. Effects of dietary strategies on exercise-induced oxidative stress.

Main Result Category	Study	Type of Diet	Nutritional Protocol	Type and Intensity of Exercise	Detection Method			
					ROS Generation	Oxidative Stress Marker	Inflammation Marker	Antioxidant Activity
ROS Generation	Zeng et al. [42]	Oatmeal	Oat flake + skim milk versus Fasting; 2 h before exercise	Body weight HIIT, 30 min	↓	N/A	N/A	N/A
ROS-induced Macromolecule Damage	Davison et al. [29]	Dark chocolate	Dark chocolate versus cocoa-liquor-free control bar versus neither, 2 h before exercise	Cycling, 2.5 h	N/A	F2-isoprostane↓	Circulating leucocyte↔, IL-6↔	N/A
	Wiswedel et al. [32]	High-flavanol cocoa drink (HFCD)	HFCD versus low-flavanol cocoa drink (LFCD), 2 h before exercise	Cycling, 29 min	N/A	F2-isoprostane↓	N/A	N/A
	Allgrove et al. [30]	Dark chocolate	Dark chocolate versus isocarbohydrate-fat control cocoa-liquor-free chocolate, twice/d, 2 weeks	Cycling for 90 min followed by 25 min exhaustion time trial	N/A	F2-isoprostane↓	Circulating leucocyte↔, IL-6↔, IL-10↔, IL-1Ra↔	N/A
	Taub et al. [31]	High-flavanol dark chocolate (HFCHO)	HFCHO versus Low-flavanol dark chocolate (LFCHO), 3 months	Ramped exercise on stationary bicycle (Cardiopulmonary exercise testing), ~10 min	N/A	PC↓	N/A	GSH/GSSH↑
	McAnulty et al. [37]	Blueberry	Blueberries versus blueberry-flavored shake, 7 days	Running, until a core temperature of 39.5 °C was reached	N/A	LH↓, F2-isoprostanes↔	IL-6↔, IL-8↔, IL-10↔	FRAP↔
	Bowtell et al. [40]	Montmorency cherry juice	Montmorency cherry juice versus isoenergetic fruit concentrate, 7 d before and 48 h after exercise	Two trials of 10 sets of 10 single-leg knee extensions	N/A	PC↓	N/A	N/A
	Pittaluga et al. [46]	Fresh red orange juice (ROJ)	ROJ versus nothing extra, thrice/day, 4 weeks	A single bout of exhaustive exercise by cycle ergometer (3 min warm-up, an initial load of 25 W, and further increments of 15 W/3 min)	N/A	MDA↓, ascorbic acid↓, hypoxanthine/xanthine↓	N/A	N/A
	Chang et al. [47]	Purple sweet potato leaves (PSPL)	Standard cooked PSPL versus low-polyphenols diet, 7 days	Treadmill running at 70% VO_{2max}, 1 h	N/A	PC↓	IL-6↓, HSP72↔	TAC (FRAP assay)↑, polyphenols↑
	Mazani et al. [48]	Probiotic yoghurt	Probiotic yoghurt versus ordinary yoghurt, 2 weeks	Exhaustive exercise (Bruce test)	N/A	MDA↓	TNF-α↓, MMP2↓, MMP9↓	SOD↑, GPX↑, TAC↑,
	Harms-Ringdahl et al. [49]	Tomato juice	Tomato juice versus nothing extra, 5 weeks	Cycle ergometer at 80% of HRmax, 20 min	N/A	8-oxodG↓	N/A	N/A
	Kawamura et al. [50]	Mixed diet	Salmon flakes + green and yellow vegetable juice + lingonberry jam versus normal diet, 10 weeks	Resistance training twice/week, 10 weeks	N/A	PC↓	N/A	N/A

Although the literature is still scarce about the effects of whole dietary strategies on exercise-induced OS, the majority of the studies demonstrated favorable effects. Nevertheless, the protocols are still very heterogeneous and further systematically designed studies are needed to strengthen the evidence

4.3- Apport complémentaire en « aliments » riches en AO (phytonutriments)

- ↘ du SO en reponse à l'exercice

nutrients MDPI

Review

Dietary Supplementation for Attenuating Exercise-Induced Muscle Damage and Delayed-Onset Muscle Soreness in Humans

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⇨ Confirmé par Tanabe et al. (2020)

7. Conclusions

7.1. Remarks

In the current review, dietary supplements with anti-inflammatory and antioxidant effects are discussed. Some positive effects mediated by curcumin, tart cherry juice, beetroot juice, and quercetin have been reported in EIMD and DOMS, although some of these results are not consistent among previous studies. These supplements may not only attenuate the aggravation of secondary muscle damage, but also improve performance by modulating cardiorespiratory and neuromuscular efficiency possibly in an interactive manner. It should be highlighted that exercise modality, physical fitness level, and study design need to be considered when interpreting the results of supplementation effects. Furthermore, the dose and duration of supplementation are important factors to maximize the effect of supplementation on EIMD and DOMS.

4.3- Apport complémentaire en « aliments » riches en AO (phytonutriments)

- ↘ du SO en reponse à l'exercice

Suite…

Table 1. Effect of curcumin on EIMD and DOMS markers.

Reference (Year)	Population	Supplementation		Exercise	Outcome				
		Dose	Duration		Blood Damage Maker	Functional Performance Marker	DOMS, Pain	Inflammatory Marker	Oxidative Stress Marker
Paralleled design studies									
Drobnic et al. (2014) [33]	Healthy, moderately active males	200 mg of curcumin or placebo, twice/day	4 d (2 d pre- and 2 d post-Ex)	Downhill run	CK: ×		VAS: ○	IL-8: ○ CRP, MCP-1: ×	FRAT, CAT, GPx: ×
Tanabe et al. (2019) [34]	Healthy young males	PRE, POST: 90 mg of curcumin, twice/day PLA: 90 mg of placebo, twice/day	PRE: 7 d pre-Ex POST: 4 d post-Ex CON: 4 d post-Ex	Eccentric Ex (elbow flexors)	CK: ×	ROM: ○ (POST) ROM: ×(PRE) MVIC: ×	VAS: ○ (POST) VAS: ×(PRE)		
Faria et al. (2020) [35]	Healthy normal-weight males	500 mg of curcumin or placebo, three times/day	29 d	Half-marathon	Mb: ○ CK, LDH, AST: ×			IL-10: ○ IL-6: ×	
Crossover design studies									
Tanabe et al. (2015) [31]	Untrained young males	150 mg of curcumin or placebo	1 h pre- and 12 h post-Ex	Eccentric Ex (elbow flexors)	CK: ○	MVIC: ○ ROM, swelling: ×	VAS: ×	IL-6, TNF-α: ×	
Nicol et al. (2015) [36]	Physically active males	2.5 g/day of curcumin or placebo, twice/day	5 d (2.5 d pre- and 2.5 d post-Ex)	Eccentric Ex (single-leg press)	CK: ○	Jump performance: ○ Swelling: ×	VAS: ○	IL-6: ○ TNF-α: ×	
Delecroix et al. (2017) [30]	Male elite rugby players	2 g of curcumin + 20 mg of piperine, or placebo, three times/day	4 d (2 d pre- and 2 d post-Ex)	Single leg jumps on an 8% downhill slope	CK: ×	Sprint: ○	VAS: ×		
Tanabe et al. (2019) [37] Experiment 1	Healthy males	90 mg of curcumin or placebo, twice/day	7 d pre-Ex	Eccentric Ex (elbow flexors)	CK: ×	MVIC, ROM: × Swelling: ×	VAS: ×	IL-8: ○ TNF-α: ×	d-ROMs, BAP: ×
Tanabe et al. (2019) [37] Experiment 2	Healthy males	90 mg of curcumin or placebo, twice/day	7 d post-Ex	Eccentric Ex (elbow flexors)	CK: ○	MVIC, ROM: ○ Swelling: ×	VAS: ○	IL-8: × TNF-α: ×	d-ROMs, BAP: ×

○, effective; ×, ineffective; DOMS, delayed-onset muscle soreness; IL-6, interleukin-6; IL-8, interleukin-8; IL-10, interleukin-10; TNF-α, tumor necrosis factor-α; CRP, C-reactive protein; MCP-1, monocyte chemoattractant protein 1; CK, creatine kinase; Mb, myoglobin; LDH, lactate dehydrogenase; AST, aspartate aminotransferase; MVIC, maximal voluntary isometric contraction; ROM, range of motion; VAS, visual analogue scale; FRAP, ferric reducing ability plasma; CAT, catalase; GPx, glutathione peroxidase; d-ROMs, diacron-reactive oxygen metabolites; BAP, biological antioxidant power; PLA, placebo; PRE, pre-exercise supplementation; POST, post-exercise supplementation; Ex, exercise.

4.3- Apport complémentaire en « aliments » riches en AO (phytonutriments)

- ↘ du SO en reponse à l'exercice

Suite…

Table 2. Effect of tart cherry juice on EIMD and DOMS markers.

Reference (Year)	Population	Supplementation		Exercise	Outcome				
		Dose	Duration		Blood Damage Maker	Functional Performance Marker	DOMS, Pain	Inflammatory Marker	Oxidative Stress Marker
Paralleled design studies									
Howatson et al. (2010) [39]	Recreational marathon runners, males and females	236 mL TCJ or placebo, twice/day	8 d (5 d pre-Ex, Ex-d, and 2 d post-Ex)	Marathon	CK, LDH: ×	MVIC: ○	VAS: ×	IL-6, CRP, Uric Acid: ○	TAS, TBARS: ○ PC: ×
Bell et al. (2016) [48]	Semi-professional male soccer players	30 mL TCJ or placebo, twice/day	8 d (4 d pre-Ex, Ex-d, and 3 d post-Ex)	LIST	CK: ×	MVIC, CMJ, agility: ○ Sprint: ×	VAS: ○	IL-6: ○ IL-8, IL-1-β CRP, TNF-α: ×	LOOH: ×
Quinlan et al. (2019) [49]	Team-sport players, males and females	30 mL TCJ or placebo, twice/day	8 d (5 d pre- Ex, Ex-d, and 2 d post-Ex).	LIST	CK: ×	MVIC, CMJ, sprint: ○	VAS: ×	CRP: ×	
Lamb et al. (2019) [50]	Non-resistance trained males	TCJ: 30 mL TCJ, twice/day POM: 250 mL of pomegranate juice, twice/day PLA: placebo drink, twice/day	9 d (4 d pre-Ex, Ex-d, and 4 d post-Ex)	Eccentric Ex (elbow flexors)	CK: ×	MVIC, ROM: ×	VAS: ×		
Crossover design studies Connolly et al. (2006) [51]	Male college students	355 mL TCJ or placebo, twice/day	8 d (4 d pre-Ex, Ex-d, and 3 d post-Ex)	Eccentric Ex (elbow flexors)		MVIC: ○ ROM: ×	VAS: ○		
Bowtell et al. (2011) [52]	Well-trained males	30 mL TCJ or placebo, twice/day	10 d (7 d pre-Ex, and 2 d post-Ex)	Single-leg knee extensions at 80% 1RM	CK: ×	MVIC: ○	PPT: ×	CRP: ×	Nitrotyrosine, TAS: × PC: ○
Morehen et al. (2020) [53]	Professional male rugby players	30 mL TCJ or placebo, twice/day	8 d (5 d pre-Ex, Ex-d and 2 d post-Ex)	Rugby match		CMJ, drop jump: ×	VAS: ×	IL-6, IL-8, IL-10: ×	
Abbott et al. (2020) [54]	Professional male soccer players	30 mL TCJ or placebo, twice/day	3 d (pre- and post-Ex and 12 and 36 h post-Ex)	90-min soccer match		CMJ, reactive strength: ×	VAS: ×		

○, effective; ×, ineffective; DOMS, delayed-onset muscle soreness; IL-1-β, interleukin-1-beta; IL-6, interleukin-6; IL-8, interleukin-8; IL-10, interleukin-10; TNF-α, tumor necrosis factor-α; CRP, C-reactive protein; CK, creatine kinase; LDH, lactate dehydrogenase; MVIC, maximal voluntary isometric contraction; ROM, range of motion; CMJ, counter movement jump; VAS, visual analogue scale; PPT, pressure pain threshold; TAS, total antioxidant status; TBARS, thiobarbituric acid reactive substances; PC, protein carbonyls; CAT, catalase; GPx, glutathione peroxidase; LOOH, lipid hydroperoxides; PLA, placebo; TCJ, tart cherry juice; POM, pomegranate juice; Ex, exercise; 1RM; 1-repetition maximum; LIST, Loughborough intermittent shuttle test.

4.3- Apport complémentaire en « aliments » riches en AO (phytonutriments)

- ↘ du SO en reponse à l'exercice

Suite…

Table 3. Effect of beetroot juice on EIMD and DOMS markers.

Reference (Year)	Population	Supplementation		Exercise	Outcome				
		Dose	Duration		Blood Damage Maker	Functional Performance Marker	DOMS, Pain	Inflammatory Marker	Oxidative Stress Marker
Paralleled design studies									
Clifford et al. (2016) [62]	Recreationally active males	H-BT: 250 mL of BTJ L-BT: 125 mL of BTJ PLA: 250 mL of placebo	3 d Ex-d (×3 servings), 24 h (×2 servings) and 48 h (×2 servings) post-Ex	Drop jumps	CK: ×	MVIC: × CMJ: ○ (H-BT)	PPT: ○ (H-and L-BT)	IL-6, TNF-α, IL-8: ×	
Clifford et al. (2017) [64]	Recreationally active males	BTJ: 250 mL of BTJ SN: 250 mL of sodium nitrate PLA: 250 mL of placebo	3 d Ex-d (×3 servings), 24 h (×2 servings) and 48 h (×2 servings) post-Ex	Drop jumps	CK: ×	MVIC, CMJ: ×	PPT: ○ (BLJ)	CRP: ×	
Clifford et al. (2016) [63]	Male team-sports players	500 mL of BTJ or a placebo	4 d (Ex-d, 24, and 48 h post-RST1 and 30-min post-RST2)	RST1: (first Ex) RST2: (second Ex)	CK: ×	MVIC, sprint: × CMJ, reactive strength index: ○	PPT: ○	CRP: ×	LOOH, PC, A●−: ×
Clifford et al. (2017) [61]	Runners, males and females	250 mL of BTJ or a placebo	3 d Ex-d (×3 servings), 24 h (×2 servings) and 48 h (×1 serving) post-Ex	Marathon	CK, AST: ×	MVIC, CMJ: ×	VAS: ×	IL-6, TNF-α, IL-8, CRP: ×	
Crossover design studies									
Van Hoorebeke et al. (2016) [65]	Competitive male runners	Betalain-rich concentrate capsule or placebo	7 d (D 1–6: 50 mg, twice/d; D 7: 50 mg pre-Ex	30 min of treadmill running followed by a 5-km TT	LDH (from baseline): ○ CK, LDH: ×	HR, RPE, lactate concentration,5-km TT duration: ○ Fatigue: ×	VAS: ×		
Montenegro et al. (2017) [66]	Triathletes, males and females	Betalain-rich concentrate capsule or placebo	7 d (D 1–6: 50 mg, twice/d; D 7: 50 mg pre- Ex	40 min of cycling followed by a 10-km running TT	CK: ○ LDH: ×	10-km TT duration, 5-km TT duration, Fatigue: ○ HR average, RPE: ×	VAS: ×		
Daab et al. (2020) [67]	Male soccer players	150 mL BTJ or placebo, twice/day	7 d (3 d pre-Ex, Ex-d and 3 d post-Ex)	LIST	CK: ○ LDH: ×	CMJ, MVIC, sprint: ○ Squat jump: ×	VAS: ○	CRP: ×	
Kozłowska et al. (2020) [68]	Elite fencers, males and females	Dietary recommendations with 26 g/day of freeze-dried BTJ or without BTJ	4 weeks	Fencing and general training	CK, LDH: ×	VO_{2max}: ○		IL-6: ×	MDA, GPx-1: ○ GPx-3, AOPP, 8-oxodG: ×

○, effective; ×, ineffective; DOMS, delayed-onset muscle soreness; IL-6, interleukin-6; IL-8, interleukin-8; TNF-α, tumor necrosis factor-α; CRP, C-reactive protein; CK, creatine kinase; AST, aspartate aminotransferase; LDH, lactate dehydrogenase; MVIC, maximal voluntary isometric contraction; CMJ, counter movement jump; VAS, visual analogue scale; PPT, pressure pain threshold; RPE, rate of perceived exertion; HR, heart rate; PC, protein carbonyls; GPx-1, glutathione peroxidase-1; GPx-3, glutathione peroxidase-3; LOOH, lipid hydroperoxides; MDA, malondialdehyde; AOPP, advanced oxidation protein product; 8-oxodG, 8-oxo-7.8-dihydro-2′-deoxyguanosine; A●−, plasma ascorbate free radical; PLA, placebo; BTJ, beetroot juice; SN, sodium nitrate; Ex, exercise; RST, repeated sprint test; VO_{2max}, volume oxygen consumption maximum; TT, time trial; LIST, Loughborough intermittent shuttle test.

4.4- Apport en créatine

- Effets potentiellement bénéfiques de la créatine chez les entraînés.

Table 1. Studies on the effects of short-term and long-term creatine supplementation and exercise on oxidative stress.

Studies	Subject	Exercise	Intervention	Main Outcome
Human study				
Kingsley et al. (2009) [109]	Active males (n = 18)	Incremental cycling that was continued until the individualized predetermined point of exhaustion	Ingested 22.8 g·d^{-1} Cr (equivalent to 5 g Cr × 4 daily) for 5 days. Each supplement dose consisted of 5.7 g Cr and 5 g of glucose polymer dissolved in 500 mL of warm water	= Oxidative stress (as measured by serum hydroperoxide concentrations)
Rahimi (2011) [40]	Trained males (n = 27)	7 sets, 3–6 repetitions, 80–90% 1RM (bench press, lat pull down, and seated rows)	20 g/day (5 g/serving, 4 serving/day), 7 days before exercise	↓MDA, 8-OHdG
Percario et al. (2012) [111]	Male elite Brazilian handball players (n = 26)	5 week RT, 50–95% 1RM, 3–12 repetition	First 5 days: a daily dose of 20 g, remaining 27 days: participants were given a dose of 5 g per day, after training	↓ TAS, =TBARS
Deminice et al. (2013) [110]	Male soccer players (n = 25)	2 consecutive running-based anaerobic sprint test, (6 sprints (35 m), maximum speed, 10 s rest between repetition)	0.3 g/kg, 7 days after first exercise	= MDA, GSH, GSH/GSSG ratio, TAC, CAT, SOD, GPX
Animal study				
Deminice and Jordao. (2012) [105]	Male rats (n = 64)	1 h swimming with load of 4% of total body weight	2% Cr, 28 days before exercise	↓TBARS, Lipid hydroperoxide ↑GSH/GSSG ratio, TAC = α-Tocopherol, CAT
Silva et al. (2013) [23]	Male rats (n = 36)	Exhaustion eccentric running (treadmill, 50–60% VO_2max, constant velocity 1.0 km/h)	300 mg/kg/day, 15 days, dose of initially: 2 serving/day, dose after 6 days: 1 serving/day	= TBARS, PC, TT, SOD, GPX, CAT
Araujo et al. (2013) [101]	Male Wistar rats (n = 40)	25 min treadmill at different fixed speeds for each series, 48 h interval between series, 8 weeks	2% in diet Cr during the maintenance phase equals 20 g·kg^{-1} peak in the phase of 13% were used equivalent to 130 g·kg^{-1}	T and TCr groups: ↑H_2O_2, GSH-GPx CCr and TCr groups: ↑CAT TCr group: ↓SOD Al groups: GSH, GSH/GSSG
Stefani et al. (2014) [99]	Male Wistar rats (n = 40)	8 weeks RT (4 series of 10–12 repetitions, 90 s interval, 4 times per week, 65% to 75% of 1 Concurrent Strength and Aerobic Training Order Influence Training-InduceRM)	The first 7 days prior to the initiation of training: dosage of 0.3 g/kg/day, last 7 weeks: the dosage was set at 0.05 g/kg/day	↓lipoperoxidation, MDA ↑SOD = CAT

= No significant difference; ↓ significantly decreased responses; ↑ significantly increased responses; creatine (Cr); one repetition maximum (1RM); malondialdehyde (MDA); 8-OH-2-deoxyguanosine (8-OH-dG); thiobarbituric acid-reactive substances (TBARS); glutathione (GSH); oxidized glutathione (GSSG); resistance training (RT); total antioxidant capacity (TAC); catalase (CAT); total antioxidant status (TAS); glutathione peroxidase (GSH-GPx); protein carbonyls (PC); total thiol (TT) superoxide dismutase (SOD); glutathione peroxidase (GPX); hydrogen peroxide (H_2O_2); training (T); training + creatine (TCr); and control + creatine (CCr).

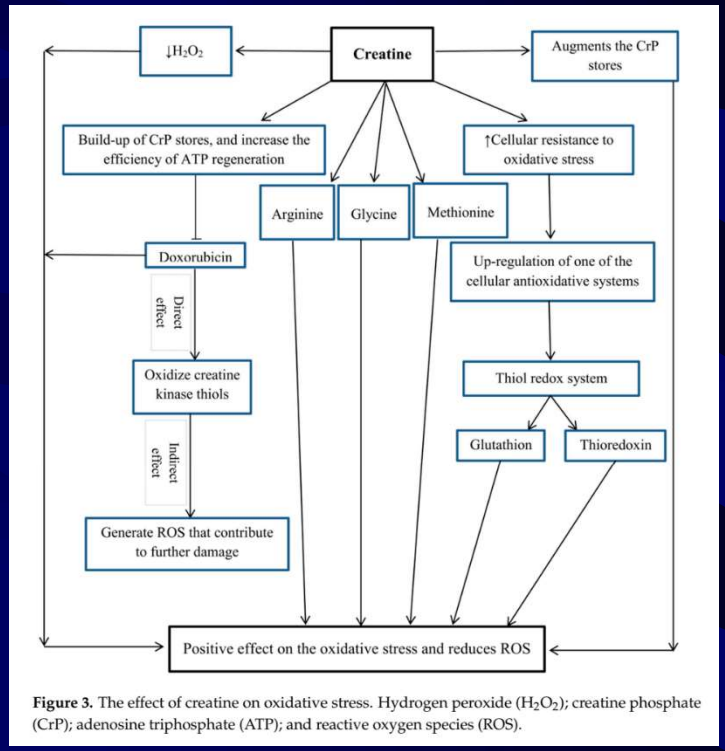


Figure 3. The effect of creatine on oxidative stress. Hydrogen peroxide (H_2O_2); creatine phosphate (CrP); adenosine triphosphate (ATP); and reactive oxygen species (ROS).

Arazi et al. (2021)

Bilan 4.

Une carence d'apport en AO ↗ SO et ↘ perf

Effets des AO sur la perf

- ✓ Pas d'effets des vitamines AO sur la performance sauf si statut initial déficitaire
- ✓ Effet positif de la NAC sur la perf
- ✓ Effet positif des flavonoides (sous classe des polyphenols) mais à confirmer

Effet des AO sur le statut redox

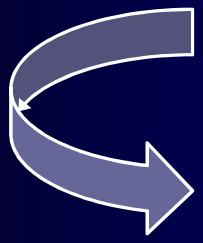
- ↗ des concentrations plasmatiques en vitamines
- ↘ SO repos et post-exo

Les aliments riches en AO (chocolat, jus de grenade, papaya) et la créatine semblent aussi avoir des effets bénéfiques sur le SO

5- Le revers de la médaille de la supplémentation

Rappel:

A faible doses, les ERON sont impliqués dans les voies de signalisations induites par l'exercice (biogénèse mitochondiale, insulino-sensibilité, augmentation de la synthèse d'enzymes AO)



A forte dose, les AO limiteraient les adaptations naturelles induites par l'entraînement (liées aux RL à faible dose)

5- Le revers de la médaille de la supplémentation

Supplémentation en antioxydants:

☺ limite les dommages oxydatifs

MAIS

☹ limite l'adaptation naturelle de l'organisme (via les facteurs de transcriptions redox sensibles)

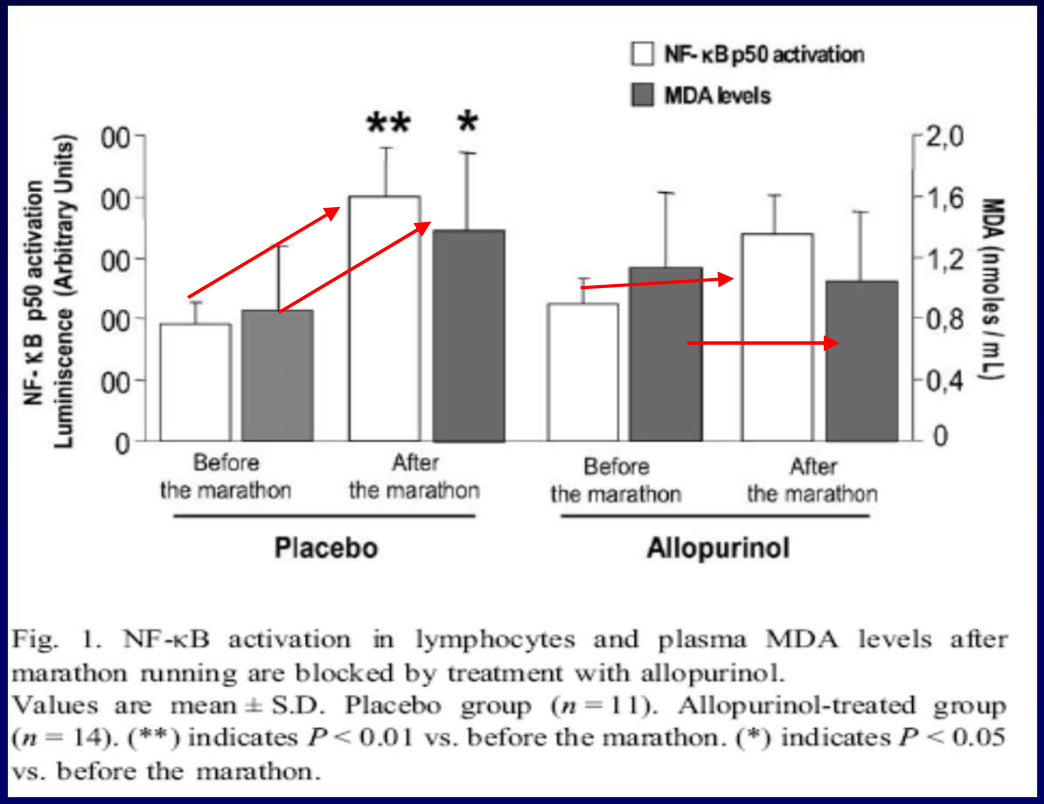


Fig. 1. NF-κB activation in lymphocytes and plasma MDA levels after marathon running are blocked by treatment with allopurinol.
Values are mean ± S.D. Placebo group ($n = 11$). Allopurinol-treated group ($n = 14$). (**) indicates $P < 0.01$ vs. before the marathon. (*) indicates $P < 0.05$ vs. before the marathon.

Sans AO (allopurinol = inhibe XO)

- ↗ NFkB (facteur de transcription)
- ↗ MDA (marqueur SO)

Avec AO (allopurinol = inhibe XO)

- ➔ NFkB (facteur de transcription)
- ➔ MDA (marqueur SO)

La prise d'AO bloque le SO mais bloque le facteur de transcription responsable des adaptations ultérieures

Équipe de Vina (Espagne) *Gomez et al. 2006* Revue de question dans Sciences et Sport

☺ Placébo: L'activation d'NFkB => « up-régulation » des enzymes antioxydantes (SOD)

☹ Supplémentés : inactivation NFkB => pas d'adaptation des antioxydants

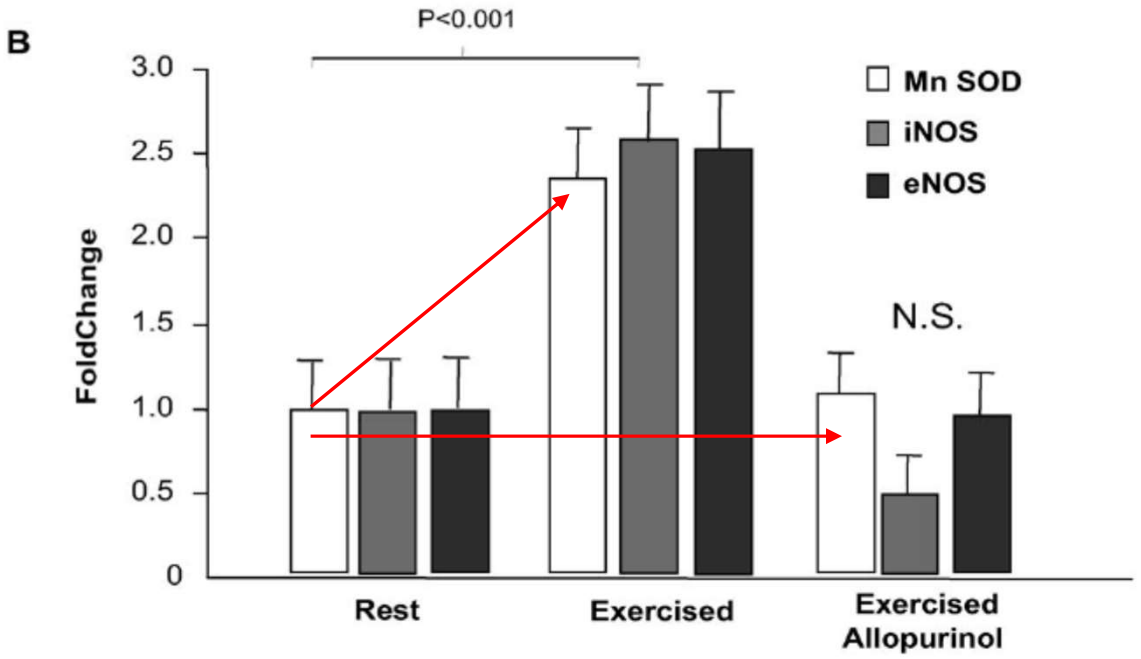


Fig. 3. Exercise activates NF-κB and induces up-regulation of Mn-SOD and NO synthases. Prevention by allopurinol administration.
(a) EMSA analysis of NF-κB in the nuclear extracts of rat gastrocnemius (Ø competition assay).
(b) Expression of Mn-SOD, iNOS and eNOS measured by real time RT-PCR from gastrocnemius muscle of rats at rest, after exercise and after exercise but pretreated with allopurinol ($N = 9$).

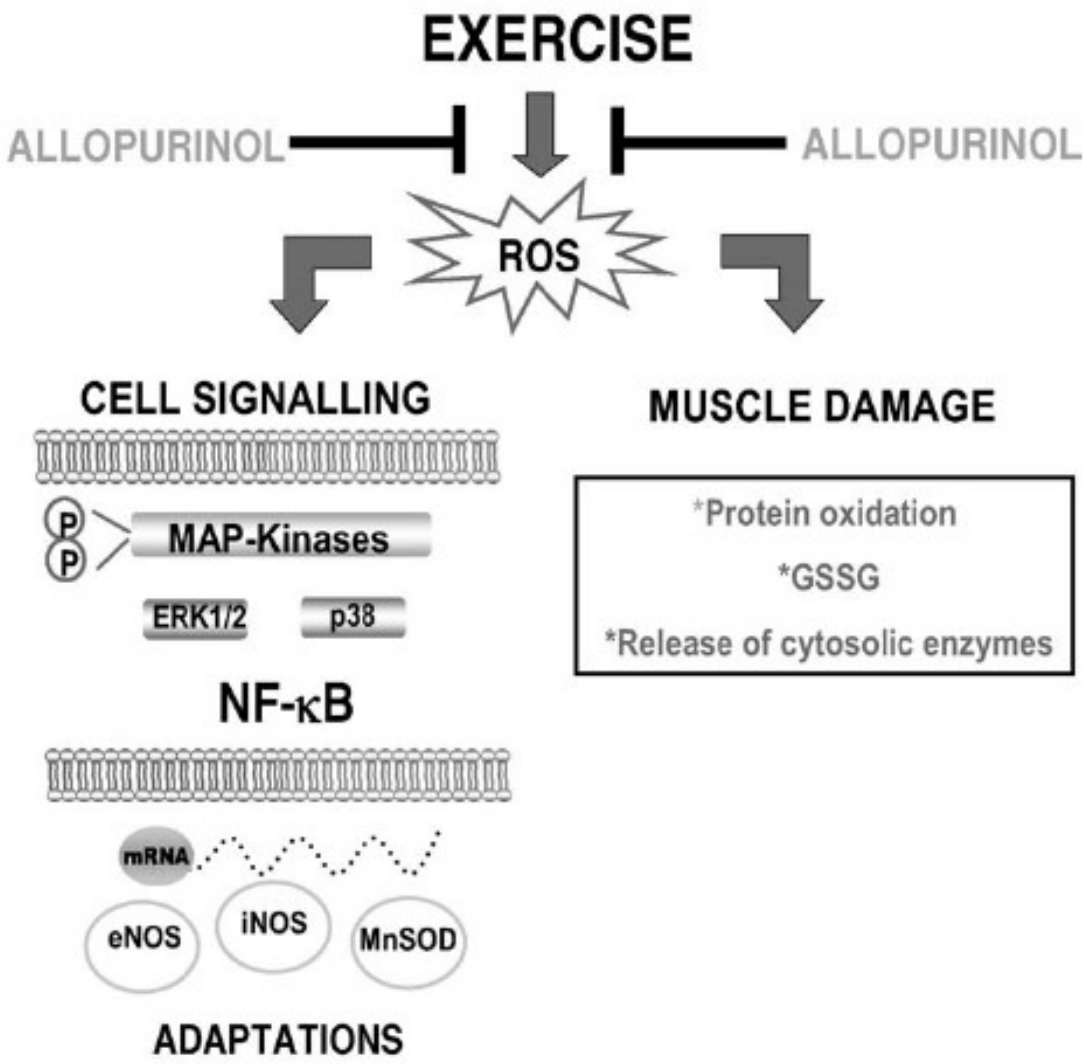


Fig. 4. Proposed mechanism of the role of ROS in signalling of cell adaptations after exercise.

Gomez et al. 2006

5- Le revers de la médaille de la supplémentation

Supplémentation vit C homme (1g/j) et rats (0.24mg/cm^2)

Training-induced increases in maximum oxygen uptake ($\dot{V}O_2$max) in men and in $\dot{V}O_2$max and maximal endurance time in rats and the effect of vitamin C administration[1]

	n	Before training	After training	Absolute difference	Relative difference	P[2]
					%	
$\dot{V}O_2$max						
Men (mL · kg^{-1} · min^{-1})						
Not supplemented	9	38.2 ± 1.1[3]	46.6 ± 4.1	8.2 ± 2.9	22.0	NS
Vitamin C–supplemented	5	41.2 ± 5.1	45.6 ± 7.0	4.4 ± 4.2	10.8	
P for effect of training[4]			0.019			
Animals (m · min^{-1})						
Not supplemented	6	54.4 ± 4.5	63.7 ± 9.6	9.3 ± 6.9	17.0	NS
Vitamin C–supplemented	6	56.3 ± 9.0	58.9 ± 9.0	2.7 ± 2.8	4.7	NS
P for effect of training			0.005			
Endurance capacity						
Animals (min)						
Not supplemented	6	99.2 ± 6.6	284.3 ± 105.9	185.2 ± 107.1	186.7	0.014
Vitamin C–supplemented	6	101.2 ± 9.7	128.0 ± 44.7	26.8 ± 47.2	26.5	
P for effect of training			0.004			

[1] Human study: $\dot{V}O_2$max improvement after 8 wk of training in sedentary men; effect of vitamin C administration in trained (n = 9) and in trained and vitamin C–supplemented (n = 5) men. Rat study: $\dot{V}O_2$max and maximal endurance time improvement for endurance-trained animals in 6 wk; effect of vitamin C administration in trained (n = 6) and trained and vitamin C–supplemented (n = 6) rats. In both studies, differences were checked for statistical significance by a repeated-measures 2-factor ANOVA.

[2] Training × treatment interaction.

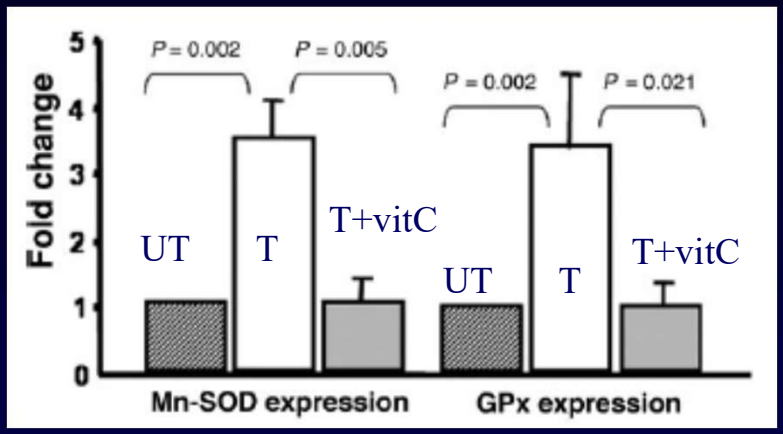
[3] $\bar{x}$ ± SD (all such values).

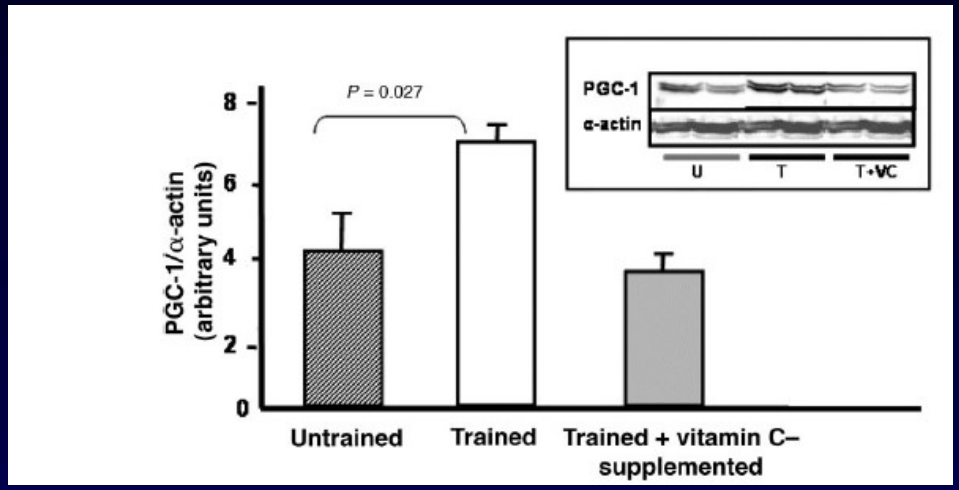
[4] Before training compared with after training.

☹ Supplémentation Vit C ↘ endurance mais pas VO2max

Gomez-cabrera et al. (2008)

5- Le revers de la médaille de la supplémentation





Vit C ↘ expression enzymes antiox

Vit C ↘ marqueur biogenèse mitochondriale

Gomez-cabrera et al. (2008)

PGC-1→ NRF-1→ mTFA→ cytochrome C.

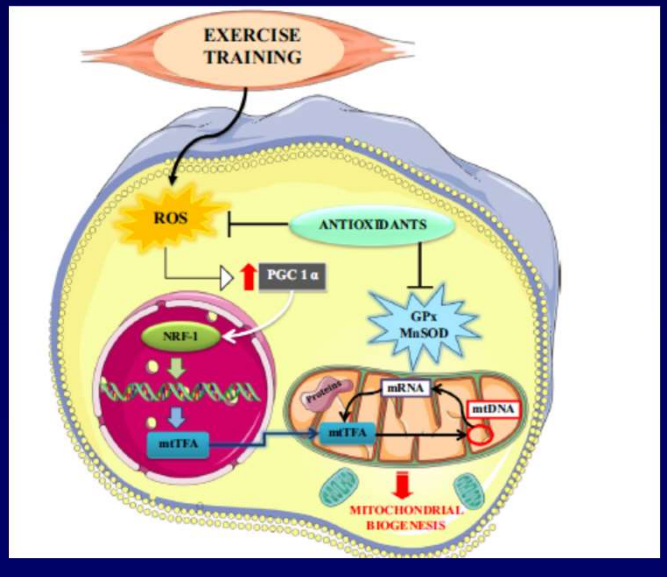


Fig. 4. Activation of mitochondrial biogenesis by exercise training-induced ROS. Mitochondrial biogenesis is driven by the coordinate regulation of nuclear and mitochondrial genomes. Exercise training-induced ROS stimulates PGC-1α production in skeletal muscle cells. This signal promotes PGC-1α gene expression in the nucleus, where PGC-1α acts to specific genes such as NRF-1 which activates the expression of mtTFA. This nuclear protein is a NRF-1 target gene which is considered the most important mammalian transcription factor for mtDNA because it stimulates mitochondrial DNA transcription and replication. Antioxidant supplements during training have a detrimental effect on mitochondrial biogenesis due to the inhibition of ROS associated with exercise and also on the mitochondrial antioxidant defense system which is downregulated.

5- Le revers de la médaille de la supplémentation

La complémentation a haute dose n'altère pas que les adaptations du statut redox mais d'autres adaptations liées à l'exercice/entraînement

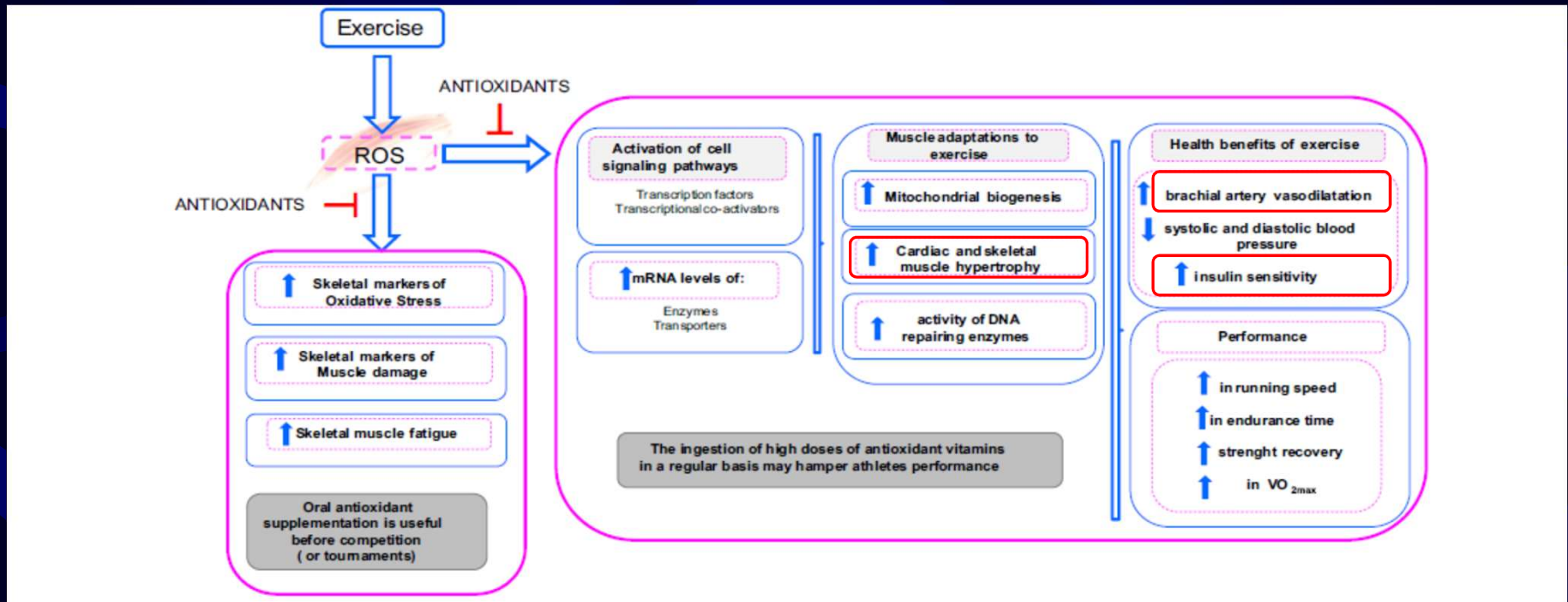


Fig. 2. Janus faced of ROS in exercise. Beneficial and negative effect of supplementation with antioxidants after one bout of exhaustive exercise and during training. Exercise-induced ROS causes muscle damage, oxidative stress, and fatigue. However, ROS can act as signaling molecules and mediate the adaptive muscle responses to exercise training. The responses result from the cumulative effects of repeated exercise bouts.

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5- Le revers de la médaille de la supplémentation

5.1 Etudes ayant montré un effet délétère de la supplémentation sur les différentes adaptations bénéfiques induites par l'exercice/entraînement

Table I. Studies with negative outcomes using antioxidant supplementation during exercise training

Study (y)	Subjects	Supplements (daily dose)	Duration	Study design	Findings
Malm et al.[249] (1996)	15 M	Coenzyme Q_{10} (120 mg)	20 d	Placebo-controlled trial: Exercise tests: anaerobic test (Wingate test, 5 min recovery, 10 × 10 sec all-out cycling), $\dot{V}O_2$ submax and max test. Exercise training: 9 sessions (15 × 10 sec all-out cycling sprints). Samples: plasma CK activity	After exercise, CK levels ↑ only in the supplemented group. Subjects taking supplements showed smaller training-induced improvements in physical performance than the placebo group
Malm et al.[250] (1997)	18 M	Coenzyme Q_{10} (120 mg)	22 d	Placebo-controlled double-blind trial: Exercise tests: anaerobic test (30 sec all-out cycling, 5 min recovery, 10 × 10 sec all-out cycling), submax and peak cycling $\dot{V}O_2$ test, $\dot{V}O_{2max}$ running test. Exercise training: 7 sessions (15 × 10 sec all-out cycling sprints). Samples: plasma lactate	There was a greater increase in anaerobic performance in the placebo group compared with the supplemented group. Moreover, supplementation was associated with reduced exercise training-induced increase in power output and recovery rate between cycling sprints. Coenzyme Q_{10} had no effect on submax and peak cycling $\dot{V}O_2$, running $\dot{V}O_{2max}$ and lactate levels
Childs et al.[175] (2001)	14 M	Vitamin C (12.5 mg/kg BW) +NAC (10 mg/kg BW)	1 wk (post-exercise)	Double-blind placebo-controlled trial: Exercise protocol: eccentric arm exercise (3 × 10 repetitions, 80% of 1RM). Samples: serum free iron levels, plasma lipid hydroperoxides, F2-isoprostanes, myeloperoxidase and IL-6, plasma CK and LDH activities, serum SOD and GPX	Exercise ↑ inflammatory indicators, free iron concentration and the levels of oxidative stress and muscle damage markers. The amount of iron, levels of lipid hydroperoxides and isoprostanes and LDH and CK activities were higher in the supplemented group than in the placebo group
Coombes et al.[251] (2001)	28 F rats	Vitamin E (10 000 IU/kg diet) + α-lipoic acid (1.65 g/kg diet)	8 d	*In situ* experiment: Contractile measurements (tibialis anterior): P_o, P_t and force-frequency curve, 60 min fatigue protocol. Samples: muscle MDA and lipid hydroperoxide	Contracted muscles of supplemented animals had lower levels of oxidative stress than the muscles from the control group. Vitamin E and α-lipoic acid supplementation had no effect on muscle fatigue but was associated with decreased muscle force production at low stimulation frequencies (*in situ*). *In vitro* experiments indicated that vitamin E depressed force production at low stimulation frequencies
	32 F rats	Vitamin E: 100, 200, 400 μM/DHLA; 100 μM/vitamin E; 400 μM + DHLA; 100 μM		*In vitro* experiment: contractile measurements (costal diaphragm): P_o, P_t and force-frequency curve, 30 min fatigue protocol	
Marshall et al.[214] (2002)	5 F racing greyhounds	Vitamin C (1 g)	4 wk (each treatment)	Crossover controlled trial: Treatments: no supplementation; supplementation after racing; supplementation 1 h before racing. Exercise training: 2 × 500 m races/wk. Samples: plasma TBARS and antioxidant capacity	Vitamin C showed no effect on oxidative stress and antioxidant capacity. The dogs ran slower when supplemented

Peterneli and Coombes 2011

Table I. Contd

Study (y)	Subjects	Supplements (daily dose)	Duration	Study design	Findings
Avery et al.[202] (2003)	18 untrained M	Vitamin E (1200 IU)	3 wk	Randomized placebo-controlled double-blind trial: Exercise Protocol: 3 resistance exercise sessions separated by 3 days of recovery. Measurements: muscle soreness, muscle strength and power assessment. Samples: plasma MDA and CK activity	There was no effect of supplementation on muscle soreness, performance indices and MDA levels. CK levels were greater in the supplemented group than in the placebo group
Bryant et al.[144] (2003)	7 M cyclists	Vitamin C (1 g)/vitamin C (1 g) + vitamin E (200 IU/kg)/vitamin E (400 IU/kg)	3 wk (each treatment)	Controlled crossover single-blind trial: Treatments: placebo; vitamin C; vitamin C + vitamin E; vitamin E. Exercise tests: 60 min steady state ride (70% $\dot{V}O_{2max}$) and 30 min performance ride (70% $\dot{V}O_{2max}$). Samples: plasma MDA and lactic acid	Supplementation had no effect on exercise performance. Vitamin E ↓ MDA levels, the combination of vitamins E and C had no effect, vitamin C alone ↑ MDA levels
Khassaf et al.[94] (2003)	16 untrained M	Vitamin C (500 mg)	8 wk	Randomized controlled trial: Muscle samples (exercise protocol: 45 min single leg cycling, 70% $\dot{V}O_{2max}$, vastus lateralis): HSP60 and HSP70 content. Lymphocytes (treated with H_2O_2 for 30 min): SOD and CAT activity, HSP60 and HSP70 content	Supplementation with vitamin C was associated with attenuated exercise-induced increase in HSP content and SOD and CAT activity
Nieman et al.[176] (2004)	36 triathletes (26 M, 10 F)	Vitamin E (800 IU)	2 mo	Randomized placebo-controlled double-blind trial: Ironman Triathlon race – samples: plasma and urinary F_2-isoprostanes, urinary 8-OHdG and 8-oxoG, plasma lipid hydroperoxides and cytokines	Post-race concentrations of isoprostanes, lipid hydroperoxides, IL-6, IL-1ra and IL-8 increased more in the vitamin E group than in the placebo group. Supplementation had no effect on race time
Gomez-Cabrera et al.[232] (2005)	20 M rats	Allopurinol (32 mg/kg)	Admin prior to exercise	Randomized controlled trial: Exercise protocol: progressive intensity treadmill test, exercise to exhaustion. Samples: plasma lactate and XO activity, muscle GSH, GSSG, carbonylated proteins, p38, ERK1 and ERK2, NF-κβ DNA-binding activity and Mn-SOD, iNOS and eNOS	Allopurinol treated rats exhibited ↓ oxidative stress levels and ↓ exercise-mediated increase in XO activity and induction of MAPKs. This was associated with ↓ DNA binding of NF-κB and blunted upregulation of *Mn-SOD*, *eNOS* and *iNOS* gene expression
Gomez-Cabrera et al.[233] (2006)	25 marathon runners	Allopurinol (300 mg)	2 h prior to marathon race	Randomized placebo-controlled trial: Marathon race - samples: lymphocyte NF-κβ p50 activation, plasma MDA and XO activity	Allopurinol prevented XO activation and lipid peroxidation. Inhibition of XO-derived ROS formation prevented NF-κB activation. Allopurinol had no effect on race time

Continued next page

Table I. Contd

Study (y)	Subjects	Supplements (daily dose)	Duration	Study design	Findings
Close et al.[197] (2006)	20 M	Vitamin C (1 g)	2 h prior to and for 2 wk post-exercise	Randomized placebo-controlled double-blind trial: Exercise protocol: downhill running test (30 min, 60% $\dot{V}O_{2max}$). Measurements: pain assessment (visual analogue scale, pressure algometry) and muscle function (quadriceps torque assessment). Samples: serum MDA	Supplementation with vitamin C ↓ exercise-induced increase in MDA levels but had no effect on DOMS. Delayed recovery of muscle function was noted in the supplemented group
Fischer et al.[96] (2006)	21 M	α-Tocopherol (400 IU) + vitamin C (500 mg) α-Tocopherol (290 IU) + γ-tocopherol (130 IU) + vitamin C (500 mg)	4 wk	Randomized placebo-controlled single-blind trial: Exercise protocol: 3h, 2-legged dynamic knee extensor exercise. Samples: muscle HSP72 mRNA and protein, plasma HSP72 and F_2-isoprostanes	α-Tocopherol + vitamin C treatment attenuated ↑ in lipid peroxidation post-exercise. Exercise-induced increase in HSP72 levels in skeletal muscle and circulation was abolished in α-tocopherol + γ-tocopherol + vitamin C group
Knez et al.[30] (2007)	16 half-Ironman triathletes (13 M, 3 F)	Vitamin C (1095 ± 447 mg) + vitamin E (314 ± 128 mg)	Vitamin C: 4.9 ± 4.7 y; vitamin E: 5.6 ± 5.2 y	Observational study: subjects recruited 4 wk before the race, controls active <3h/wk: Triathletes: training and competing for 4.7 ± 2.4 y, 14.5 ± 3.4 h/wk, 10 taking supplements; race: 1.9 km swim, 90.1 km cycle, 21.1 km run. Samples: plasma MDA and erythrocyte SOD, GPX and CAT activities	Dose-response relationship between adaptations of antioxidant enzymes and responses to ultraendurance exercise. Ultraendurance training upregulated endogenous antioxidant system (GPX and CAT activity). Triathletes taking supplements had elevated post-race MDA levels compared with nonsupplementers
	29 Ironman triathletes (23 M, 6 F)	Vitamin C (558 ± 350 mg) + vitamin E (702 ± 756 mg)	Vitamin C: 0.8 ± 0.6 y; vitamin E: 1.6 ± 0.8 y	Triathletes: training and competing for 6.9 ± 6.4 y, 17.19 ± 3.4 h/wk, 8 taking supplements; race: 3.8 km swim, 180 km cycle, 42.2 km run. Samples: plasma MDA and erythrocyte SOD, GPX and CAT activities	
Richardson et al.[2,54] (2007)	25 M	Dose: α-lipoic acid (300 mg) + vitamin C (500 mg) + vitamin E (200 IU) Dose: α-lipoic acid (300 mg) + vitamin C (500 mg) + vitamin E (400 IU)	2 h and 1.5 h prior to exercise	Randomized placebo-controlled crossover double-blind trial: Exercise protocol: forearm handgrip exercise at low-intensity workload (3, 6 and 9 kg at 0.5 Hz) for 3 min. Measurements: plasma FR, vasodilation.	Antioxidant administration ↑ total antioxidant capacity and ↓ exercise-induced oxidative stress but ↓ brachial artery vasodilation during submaximal exercise.
Gomez-Cabrera et al.[97] (2008)	14 sedentary M	Vitamin C (1 g)	8 wk	Randomized double-blind controlled trial: Exercise test: $\dot{V}O_{2max}$ test (bicycle ergometer). Exercise training: 40 min cycling 3 d/wk (65% → 80% $\dot{V}O_{2max}$)	

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Table I. Contd

Study (y)	Subjects	Supplements (daily dose)	Duration	Study design	Findings
	36 M rats	Vitamin C: 0.24 mg/cm^2 body surface area	3 wk; 6 wk	Untrained group, trained group, trained +supplemented group: RT-PCR experiment: 3 wk training. Western blotting and performance experiments: 6 wk training. Exercise training: 5 d/wk, treadmill (75% $\dot{V}O_{2max}$, 25 → 85 min/d). Endurance test (run to exhaustion), $\dot{V}O_{2max}$ test (treadmill run). Samples: muscle mTFA and NRF-1 mRNA and protein, cyt c and PGC-1 protein, Mn-SOD and GPX mRNA	Moderate intensity exercise enhanced endogenous antioxidant defence (↑ expression of Mn-SOD and GPX) and mitochondrial biogenesis (upregulation of PGC-1 → NRF-1 → mTFA → cyt c pathway) and increased endurance capacity. Vitamin C prevented these training induced adaptations
Copp et al.[255] (2009)	19 M rats	Vitamin C (76 mg/kg) + tempol (52 mg/kg)	Acute infusion (after first exercise protocol)	Exercise protocol (right spinotrapezius muscle): 1 Hz twitch contractions for 180 sec (2 sessions: pre- and post-antioxidant administration);13 rats: blood flow and P_{O_2mv} measurements; 6 rats: muscle force measurements	Antioxidant administration ↑ serum antioxidant capacity but ↓ blood flow, baseline P_{O_2mv}, muscle oxygen utilization and muscle force production
Lamprecht et al.[174] (2009)	8 trained M cyclists	Vitamin E (107 IU) + vitamin C (450 mg) + β-carotene (36 mg) + Se (100 μg)	2 wk	Randomized double-blind placebo-controlled crossover trial: Exercise test: cycle ergometer, 90 min cycling (45% $\dot{V}O_{2max}$) + 30 min cycling (75% $\dot{V}O_2$max). Samples: plasma MDA and GPX	MDA concentrations were ↑ and GPX levels ↓ after antioxidant treatment (pre- and post-exercise)
Ristow et al.[31] (2009)	20 untrained M (<2h of exercise/wk), 20 pretrained M (>6h of exercise/wk)	Vitamin C (1g) + vitamin E (400 IU)	4 wk	Controlled trial, 2 part-study – open-label study; double blind placebo-controlled study: 4 groups: untrained nonsupplemented, trained nonsupplemented, untrained supplemented, trained supplemented. Exercise training – 5 d/wk, session: 20 min biking/running, 45 min circuit training. Measurements: GIR. Samples: plasma adiponectin, muscle *PGC-1α*, *PGC-1β*, *PPARγ*, *SOD1* and *SOD2*, and *GPX* gene levels	Exercise training ↑ insulin sensitivity, ↓ fasting plasma insulin levels, ↑ gene expression of *PGC-1α*, *PGC-1β*, *PPARγ*, *SOD1* and *SOD2*, *GPX* (irrespective of training status). Supplementation with vitamins E and C was shown to prevent these health promoting effects
Teixeira et al.[165] (2009)	20 competitive kayakers (14 M, 6 F)	α-Tocopherol (272 mg) + vitamin C (400 mg) + β-carotene (30 mg) + lutein (2 mg) + Se (400 μg) + Zn (30 mg) + mg (600 mg)	4 wk	Randomized double-blind placebo-controlled trial: Exercise test: maximal flat-water kayaking trial (1000 m). Samples: plasma antioxidants, TBARS, IL-6 and CK, SOD, GR, GPX activities	Antioxidant supplementation ↑ antioxidant capacity but had no effect on oxidative stress and inflammation markers. Supplemented athletes showed a blunted decrease in CK activity post-exercise

Continued next page

Table I. Contd

Study (y)	Subjects	Supplements (daily dose)	Duration	Study design	Findings
Wray et al.[2,58] (2009)	6 older, mildly hypertensive M	Dose: α-lipoic acid (300 mg), vitamin C (500 mg), vitamin E (200 IU) Dose: α-lipoic acid (300 mg), vitamin C (500 mg), vitamin E (400 IU)	Prior to and after 6 wk of training: 2 h before exercise protocol Prior to and after 6 wk of training: 30 min after 1	Double-blind placebo-controlled crossover trial: Exercise protocol – d 1, 2: antioxidant efficacy test; d 3-6: FMD procedure followed by knee extensor exercise, subjects crossed over, returned after 24 h. Exercise training: 3 × wk-single leg knee-extensor exercise. Measurements: plasma FR, BP and FMD	Antioxidant administration reduced FR levels pre- and post-exercise. Exercise training reduced BP and improved vasodilation, supplementation after training negated these effects
Bailey et al.[1,10] (2010)	38 M	Vitamin C (800 mg) + vitamin E (536 mg) + vitamin B6 (4 mg) + vitamin B_9 (400 μg) + zinc sulphate monohydrate (10 μg) + vitamin B_{12} (2 μg)	6 wk (including 2 d post-exercise)	Randomized placebo-controlled double-blind trial: Exercise test (d 40): 90 min intermittent high-intensity shuttle-running. Measurements: pre- and post-exercise ratings of perceived muscle soreness and assessment of muscle function (peak isometric torque of the knee flexors and extensors, range of motion at the knee joint). Samples: urine F2-isoprostanes, serum IL-6 and cortisol	Antioxidant supplementation was associated with attenuated exercise-induced ↑ in cortisol concentration but ↑ post-exercise IL-6 and F2-isoprostane levels (compared with the placebo). Treatment had no effect on indices of muscle damage, muscle function measures and perception of muscle soreness
Matsumoto et al.[2,57] (2011)	48 M rats	α-Tocopherol (1000 IU/kg diet) + α-lipoic acid (1.6 g/kg diet)	14 wk	Controlled trial: 4 groups: untrained nonsupplemented, trained nonsupplemented, untrained supplemented, trained supplemented. Exercise training: 90 min treadmill run 4 d/wk. Samples: left ventricular and coronary artery endothelial cells (gene analysis)	IL-6 gene levels were ↓ by all treatments. *RhoA* gene expression was ↓ by exercise training, ↑ by antioxidant supplementation. The combination of exercise and supplementation resulted in a blunted ↓ of *RhoA* gene levels (compared with the exercise training effect)

1RM = repetition maximum; **8-OHdG** = 8-hydroxy-2-deoxyguanosine; **8-oxoG** = 7,8-dihydro-8-oxoguanosine; **BP** = blood pressure; **BW** = bodyweight; **CAT** = catalase; **CK** = creatine kinase; **cyt c** = cytochrome c; **DOMS** = delayed onset muscle soreness; **DHLA** = dihydrolipoic acid; **ERK** = extracellular signal-regulated protein kinases; **F** = female; **FMD** = flow-mediated vasodilation; **FR** = free radical; **GIR** = glucose infusion rate; **GPX** = glutathione peroxidase; **GR** = glutathione reductase; **GSH** = reduced glutathione; **GSSG** = oxidized glutathione; **H_2O_2** = hydrogen peroxide; **HSP** = heath shock protein; **IL-1ra** = interleukin 1 receptor antagonist; **IL-6(8)** = interleukin-6(8); **LDH** = lactate dehydrogense; **M** = male; **MAPK** = mitogen activated protein kinase; **max** = maximal; **MDA** = malondialdehyde; **mRNA** = messenger RNA; **mTFA** = mitochondrial transcription factor A; **NAC** = N-acetyl cysteine; **NF-κB** = nuclear factor kappa-light chain-enhancer of activated B cells; **NOS** = nitric oxide synthase; **NRF-1** = nuclear respiratory factor 1; **p38** = a member of MAPKs; **p50** = a subunit of NF-κβ complex; **PGC-1** = peroxisome proliferator-activated receptor gamma coactivator 1; **PPARγ** = peroxisome proliferator-activated receptor gamma; **P_{O_2mv}** = microvascular O_2 partial pressure; **P_o** = max specific tension; **P_t** = twitch tension; **RhoA** = Ras homolog gene family member A; **RT-PCR** = real-time reverse transcriptase-polymerase chain reaction; **Se** = selenium; **SOD** = superoxide dismutase; **submax** = submaximal; **TBARS** = thiobarbituric acid reactive substances; **$\dot{V}O_2$** = oxygen uptake; **$\dot{V}O_2max$** = maximal $\dot{V}O_2$; **XO** = xanthine oxidase; **Zn** = zinc; ↑ indicates increase; ↓ indicates decrease; → indicates 'leads to' outcome.

Autre revue de question plus récente (2021)

antioxidants

MDPI

Review

An Overview of Physical Exercise and Antioxidant Supplementation Influences on Skeletal Muscle Oxidative Stress

Shima Taherkhani [1], Kosar Valaei [1], Hamid Arazi [1,*] and Katsuhiko Suzuki [2]

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Reference	Subjects	Antioxidant Supplementation	Exercise Training	Results
O'Fallen et al. [88]	Young men and women	Quercetin (100 mg daily)	————	- No improvement in muscle damage or inflammatory indicators (arm edema, strength loss, elevated CK, and muscle discomfort)
Liang et al. [103]	Male Sprague-Dawley rats	BSP supplementation	Treadmill and grip strength tests	Improvement of grip strength, muscular mass, and muscular endurance

Table 1. The influence of antioxidant supplementation on skeletal muscle (adaptations to training, inflammation, muscle damage).

Reference	Subjects	Antioxidant Supplementation	Exercise Training	Results
Paulsen et al. [55]	Strength-trained recreational men and women	Vitamins E and C (235 mg per day and 1000 mg per day, respectively) for ten weeks	Heavy-duty resistance training four times a week	No increase in muscle mass
Makanae et al. [56]	Wistar rats	Vitamin C (500 mg kg^{-1}) for 14 days	Mechanical overload	Decrease of hypertrophy of overworked muscles
Theodorou et al. [57]	Exercised-recreational men	Vitamins E and C (400 IU and 1000 mg per day) for 11 weeks	Eccentric exercise for four weeks and twice a week	No impact on muscular function or recovery following exercise
Dutra et al. [58]	Healthy and non-smoking women	Vitamins E and C (400 IU and 1000 mg daily, respectively)	Ten weeks of twice-weekly strength training (ST)	Decrease of peak torque and total work due to taking antioxidant supplements
Cooke et al. [60]	Trained and untrained men and women	200 mg of COQ10 every day for 14 days	WT GXT and Isokinetic tests	Higher CoQ10 levels did not affect the individuals physical endurance
Taub et al. [61]	Healthy and sedentary adults	20 g of dark chocolate for three months	Stationary bike ride	Increase of VO_2max
Bowtell et al. [65]	Trained men	30 mL (twice daily) of Montmorency cherry juice for one week before and 48 h after exercise	Ten sets of 10 repetitions with a single knee extension at 80% of their maximal repetition	Improvement of muscular isometric strength following intensive exercise
McLeay et al. [66]	Healthy women	Blueberry smoothie 5 and 10 h before EIMD and immediately, 12 and 36 h afterwards	Exercise-induced muscle damage (EIMD)	Increase of isometric muscular strength recovery
Furlong et al. [67]	Untrained people	Proprietary herbal/botanical combination (1575 mg 2 times a day), and Aphanizomenon flos-aquae extract (1000 mg 3 times daily)	Resistance training regimen (3 times a week, two sets of 10 repetitions per movement, for 12 weeks)	No variations in the patients balance, strength, or muscular function
Carrera-Quintanar et al. [73]	University-level athletes	Vitamins C and E and Lippia citriodora antioxidant extract	2000-m running test.	Protect neutrophils from oxidative injury to skeletal muscle
Rokitzki et al. [82]	Running athletes	200 mg of vitamin C and 600 mg of vitamin E daily for five weeks	————	Decrease of muscular damage
Zoppi et al. [83]	Soccer players	1000 mg of vitamin C and 800 mg of vitamin E for twelve weeks	————	- Decrease of muscle damage and OS - No improvement in athletic performance
Dawson et al. [84]	Well-trained runners	500 to 1000 mg of vitamin C and 750 to 1500 mg of vitamin E per day	————	- No improvement on muscle damage or OS
Nieman et al. [85]	Well-trained runners	Quercetin antioxidant supplement (500 mg/day) for three weeks	————	- No improvement in physical function or muscle damage
Kon et al. [86]	Kendo athletes	CoQ10 (300 mg/day) for 20 days	————	- No improvement in OS concentration - Decrease of muscle damage
Orlando et al. [87]	Rugby participants	CoQ10 (200 mg/day) for 1 month	————	- No improvement in physical function, OS, and muscle damage

Détail des études : Richardson et al. 2007

« Handrip exercise »

Am J Physiol Heart Circ Physiol 292: H1516–H1522, 2007.
First published November 17, 2006; doi:10.1152/ajpheart.01045.2006.

Exercise-induced brachial artery vasodilation: role of free radicals

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Submitted 22 September 2006; accepted in final form 10 November 2006

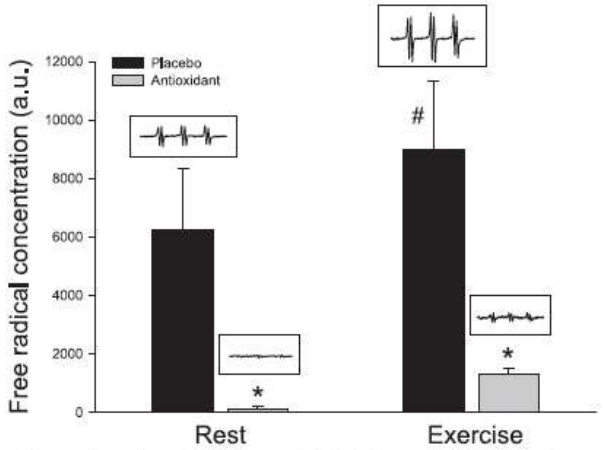


Fig. 1. The α-phenyl-tert-butylnitrone (PBN) electron paramagnetic resonance (EPR) spectroscopy data (n = 12) under the conditions of rest and after exercise with placebo and the oral antioxidant cocktail. Inlayed are representative individual examples of the PBN EPR spectra under each scenario.

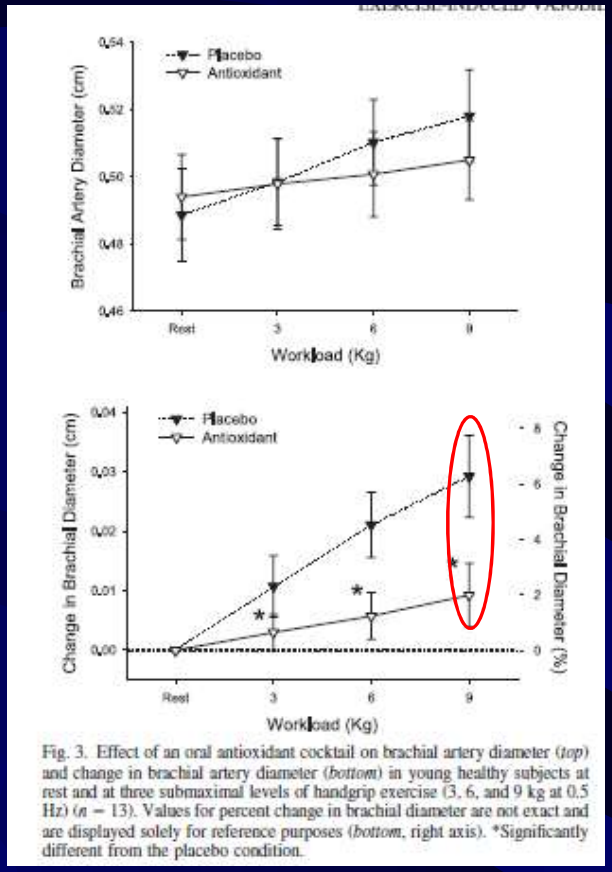


Fig. 3. Effect of an oral antioxidant cocktail on brachial artery diameter (*top*) and change in brachial artery diameter (*bottom*) in young healthy subjects at rest and at three submaximal levels of handgrip exercise (3, 6, and 9 kg at 0.5 Hz) (n = 13). Values for percent change in brachial diameter are not exact and are displayed solely for reference purposes (*bottom*, right axis). *Significantly different from the placebo condition.

Un cocktail en vitamine C (500 mg), E (400IU), ac. Lipoïque (300mg), en piégeant les ERON affecte la vasodilatation induite par l'exercice

Détail des études : Ristow et al. 2009

Antioxidants prevent health-promoting effects of physical exercise in humans

Michael Ristow[a,b,1,2], Kim Zarse[a,2], Andreas Oberbach[c,2], Nora Klöting[c], Marc Birringer[a], Michael Kiehntopf[d], Michael Stumvoll[c], C. Ronald Kahn[e], and Matthias Blüher[c,2]

Repos Après entraînement (85 min, 5j/sem, 4 sem)

Clamp hyper insulinémique euglycémique

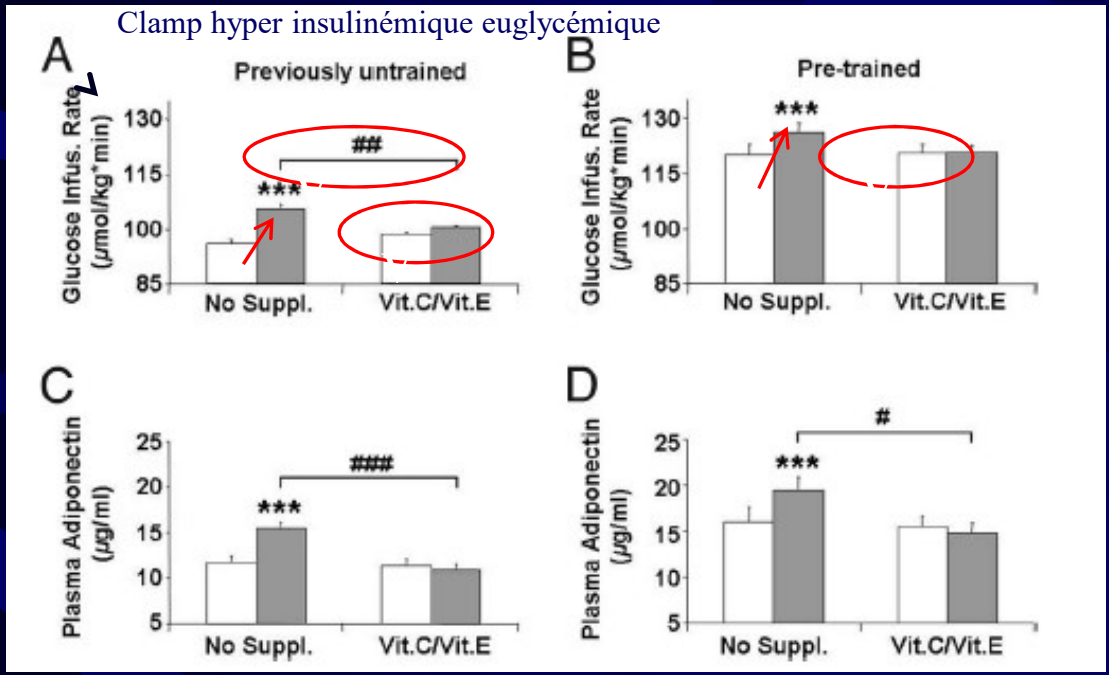


Fig. 1. Antioxidants prevent exercise-dependent induction of insulin sensitivity. (*A*) Glucose infusion rates (GIR) during euglycemic hyperinsulinemic clamps in previously untrained individuals before (white bars) and after (shaded bars) physical exercise over 4 weeks. (*Left pair of bars*) Individuals not taking any medication or placebo; (*Right pair of bars*) individuals taking both vitamin C (1000 mg/day) as well as vitamin E (400 IU/day). Bars depict means, error bars show standard error means (applies to all subsequent panels and figures). Significances (applies to all subsequent panels and Fig. 3): * indicates $0.01 < P < 0.05$ comparing data before and after 4 weeks of exercise, # indicates $0.01 < P < 0.05$ comparing "no suppl." with "Vit.C/Vit.E" groups after intervention, ** indicates $0.001 \leq P \leq 0.01$ comparing data before and after 4 weeks of exercise, ## indicates $0.001 \leq P \leq 0.01$ comparing "no suppl." with "Vit.C/Vit.E" groups after intervention, *** indicates $P < 0.001$ comparing data before and after 4 weeks of exercise, ### indicates $P < 0.001$ comparing "no suppl." with "Vit.C/Vit.E" groups after intervention. (*B*) The same set of data derived from a physically pretrained group of individuals. (*C*) Plasma adiponectin levels in the previously untrained and previously trained (*D*) state.

⇨ L'entraînement (chez sujets NE ou E) ↗ sensibilité à l'insuline

La supplémentation (Vit C: 1g/j et E 400 IU/j) inhibe cet effet bénéfique indépendamment du statut d'E des sujets

Détail des études : Ristow et al. (2009) - rats

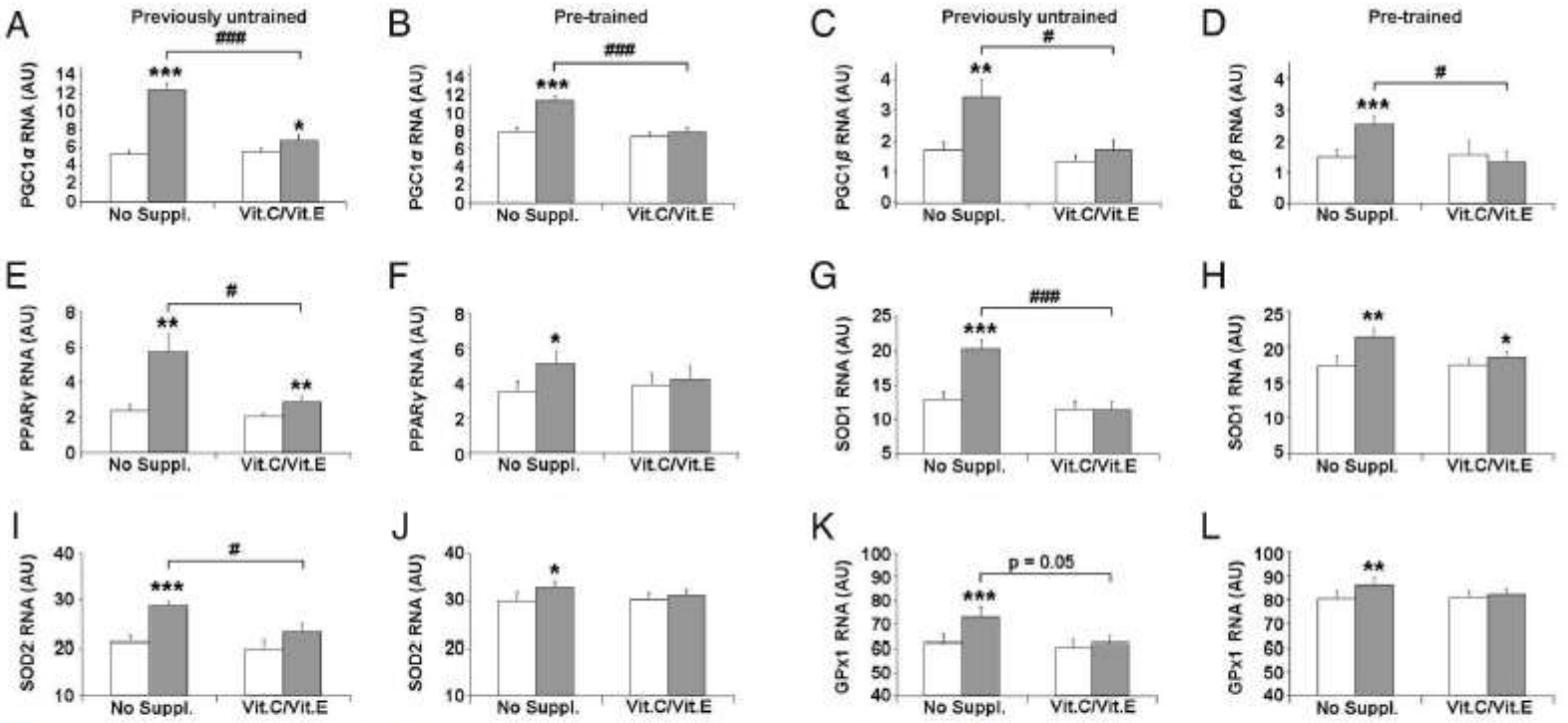


Fig. 2. Antioxidants prevent induction of molecular mediators of insulin sensitivity and antioxidant defense in exercised skeletal muscle. (*A*) depicts expression levels of *PGC1α* RNA transcripts in skeletal muscle biopsies derived from previously untrained individuals before (white bars) and after (shaded bars) physical exercise over 4 weeks as described in the *Methods* section. (*Left pair of bars*) Individuals not taking any medication or placebo; (*Right pair of bars*) individuals taking both vitamin C (1000 mg/day) as well as vitamin E (400 IU/day). Bars depict means, error bars show standard error means, "AU" abbreviates normalized arbitrary units. (*B*) depicts expression levels of *PGC1α* RNA transcripts in skeletal muscle biopsies derived from pretrained individuals before (white bars) and after (shaded bars) physical exercise over 4 weeks. (*C* and *D*) expression levels of *PGC1β* RNA transcripts in a similar fashion; (*E* and *F*) expression levels of *PPARγ* RNA; (*G* and *H*) levels of superoxide dismutase 1 (*SOD1*) RNA expression; (*I* and *J*) RNA levels of superoxide dismutase 2 (*SOD2*); (*K* and *L*) glutathione peroxidase 1 (*GPx1*) RNA expression levels.

Les AO limitent les médiateurs moléculaires de l'insulino-sensibilité et des défenses AO chez des rats non entraînés et entraînés

Autre étude : Manakae et al. (2013)

Ablation de 2 muscles de la patte pour induire hypertrophie du plantaris

- Sans traitement = Sham
- Ablation + placébo
- Ablation + Vit C

ACTA PHYSIOLOGICA
Acta Physiol 2013, 208, 57–65

Vitamin C administration attenuates overload-induced skeletal muscle hypertrophy in rats

Y. Makanae,[1] S. Kawada,[1] K. Sasaki,[1,2] K. Nakazato[3] and N. Ishii[1]

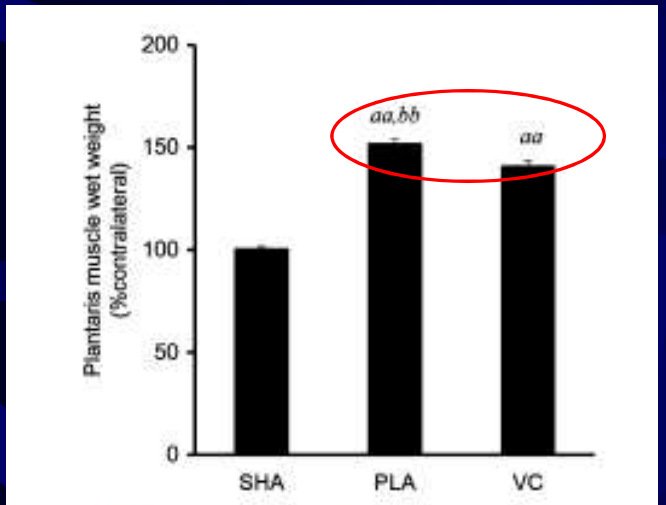
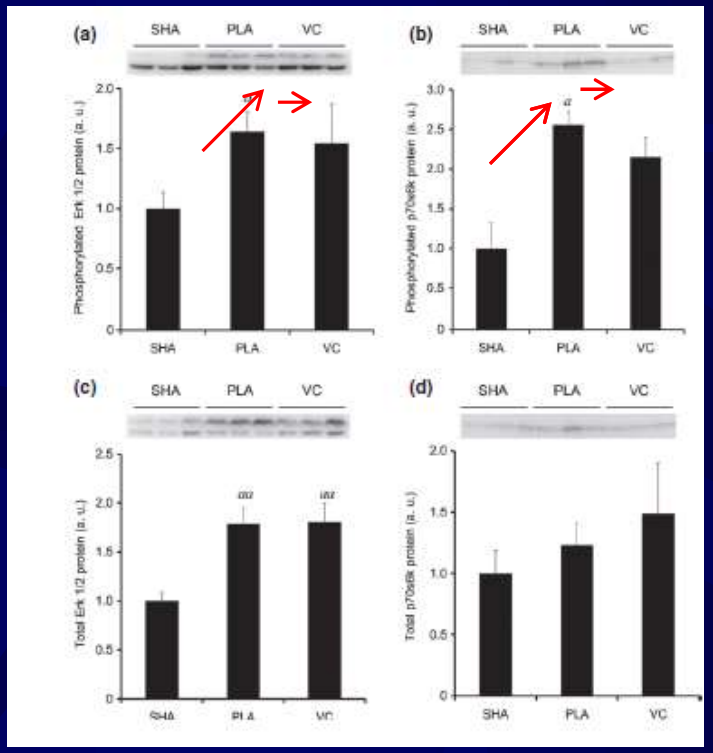
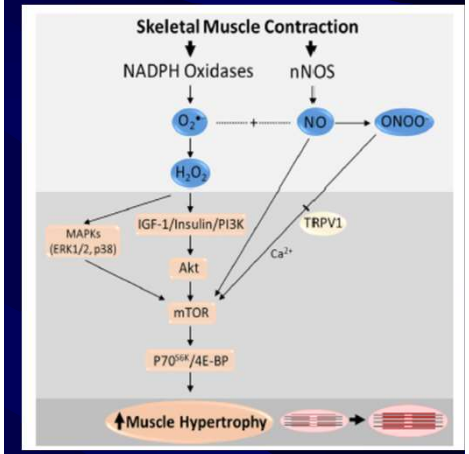


Figure 1 Effect of vitamin C administration on muscle wet weight. Values are expressed as mean and SEM for each group ($n = 8$). SHA, sham-operated group; PLA, placebo-administered group; and VC, vitamin C-administered group. [aa]Significantly different from the SHA group ($P < 0.01$); [bb]Significantly different from the VC group ($P < 0.01$).





Mason et al. (2016)

La phosphorylation de p70s6k et Erk1/2 (régulateur de la synthèse protéique) est bloquée avec la vit C

Autre étude : Michailidis et al. (2013)

Thiol-based antioxidant supplementation alters human skeletal muscle signaling and attenuates its inflammatory response and recovery after intense eccentric exercise[1–3]

Yannis Michailidis, Leonidas G Karagounis, Gerasimos Terzis, Athanasios Z Jamurtas, Kontantinos Spengos, Dimitrios Tsoukas, Athanasios Chatzinikolaou, Dimitrios Mandalidis, Renae J Stefanetti, Ioannis Papassotiriou, Spyros Athanasopoulos, John A Hawley, Aaron P Russell, and Ioannis G Fatouros

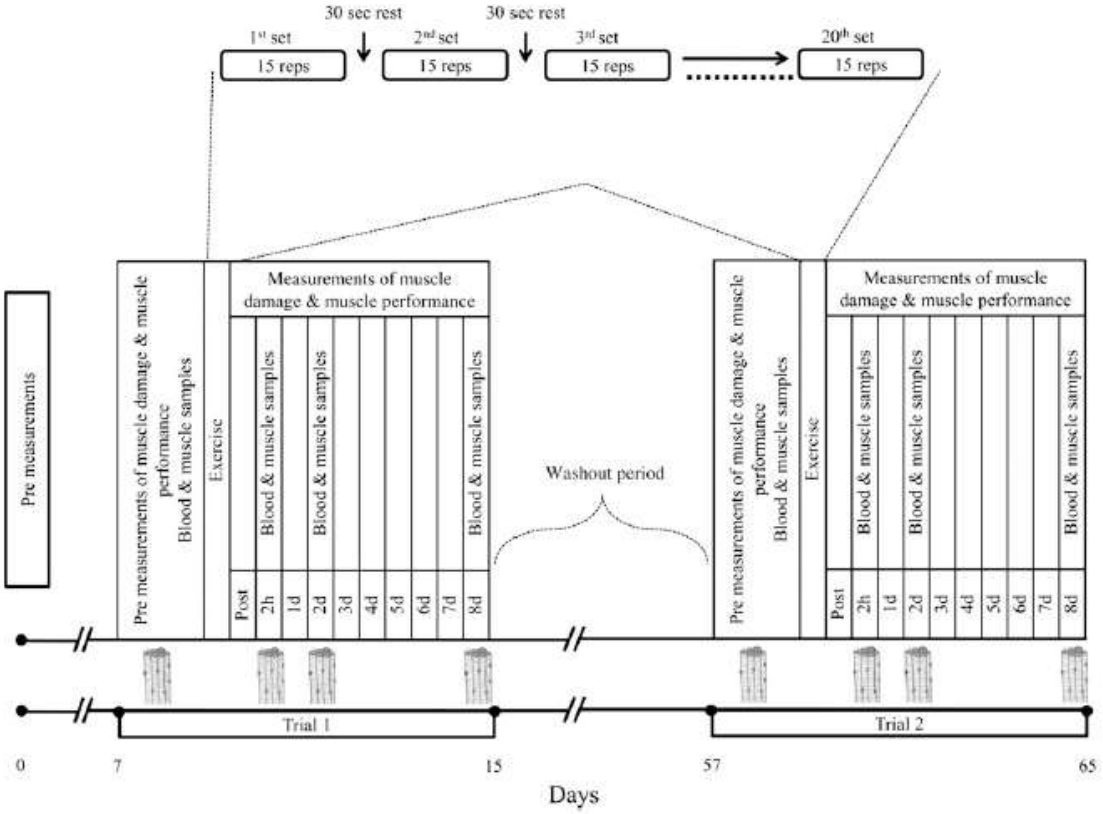


FIGURE 1. The experimental protocol. Post, after exercise; Pre, at baseline.

Exercice qui induit des dommages musculaires (300 contraction excentriques)

Supplémentation en NAC

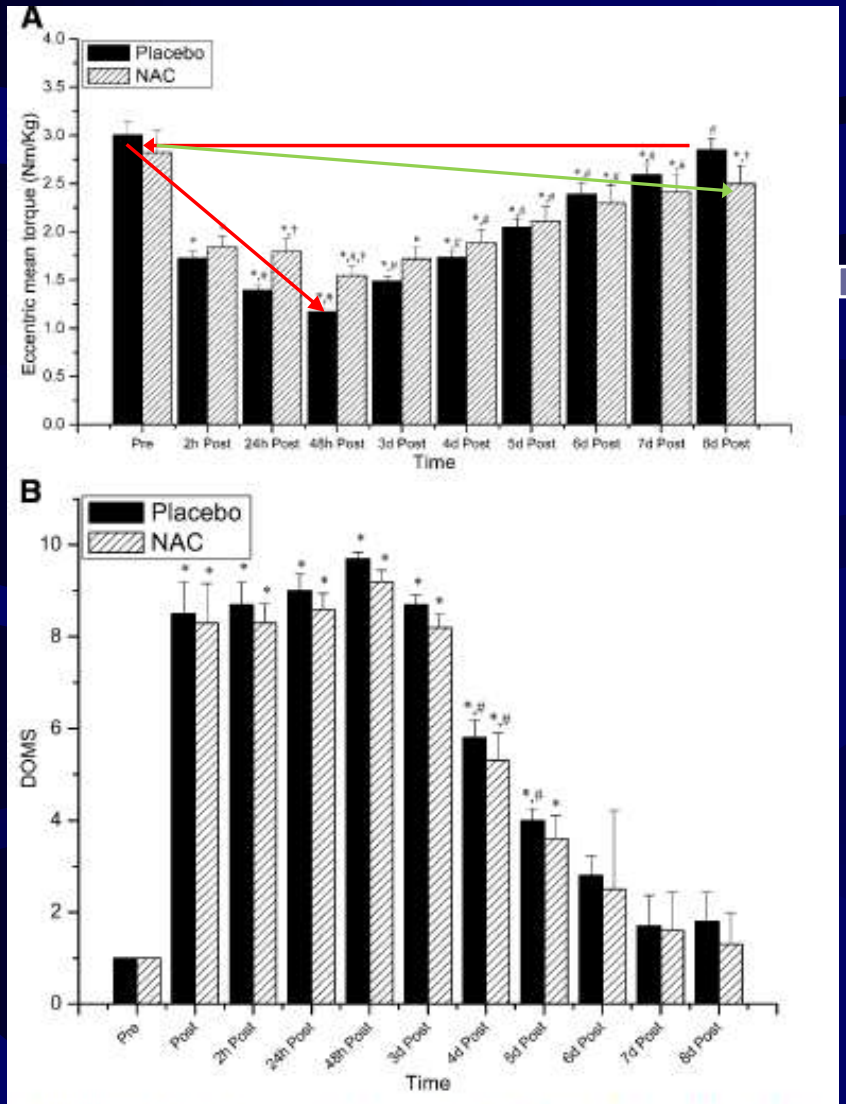


FIGURE 4. Mean (±SD) changes in muscle function (A) and muscle soreness (B) during the 2 trials ($n = 10$ per trial). Two-factor (supplement × time) ANOVA with repeated measures on time and post hoc pairwise comparisons through the Bonferonni test were used. P-interaction was significant. *Significantly different from baseline, $P < 0.05$. #Significantly different from the previous time point, $P < 0.05$. †Significant difference between trials, $P < 0.05$. DOMS, delayed onset of muscle soreness; NAC, *N*-acetylcysteine; Post, after exercise; Pre, at baseline.

Placébo: ↘ de la force pendant 2 j avec pic à 48h et retrouve les valeurs de base à J+8

NAC: idem mais ne retrouve pas sa valeur de base

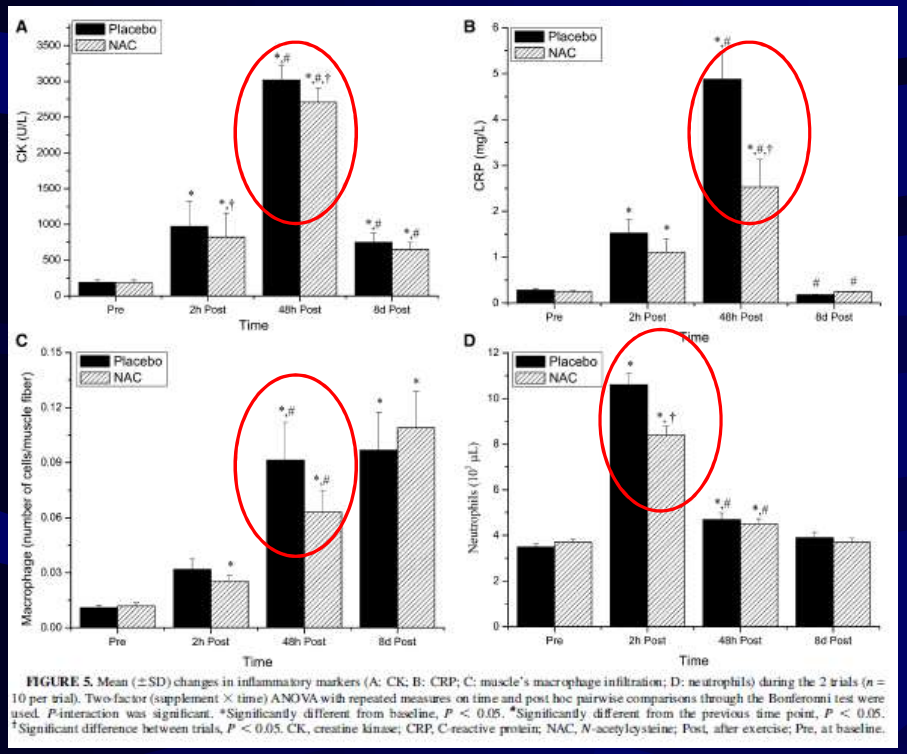


FIGURE 5. Mean (±SD) changes in inflammatory markers (A: CK; B: CRP; C: muscle's macrophage infiltration; D: neutrophils) during the 2 trials (n = 10 per trial). Two-factor (supplement × time) ANOVA with repeated measures on time and post hoc pairwise comparisons through the Bonferonni test were used. P-interaction was significant. *Significantly different from baseline, $P < 0.05$. #Significantly different from the previous time point, $P < 0.05$. †Significant difference between trials, $P < 0.05$. CK, creatine kinase; CRP, C-reactive protein; NAC, N-acetylcysteine; Post, after exercise; Pre, at baseline.

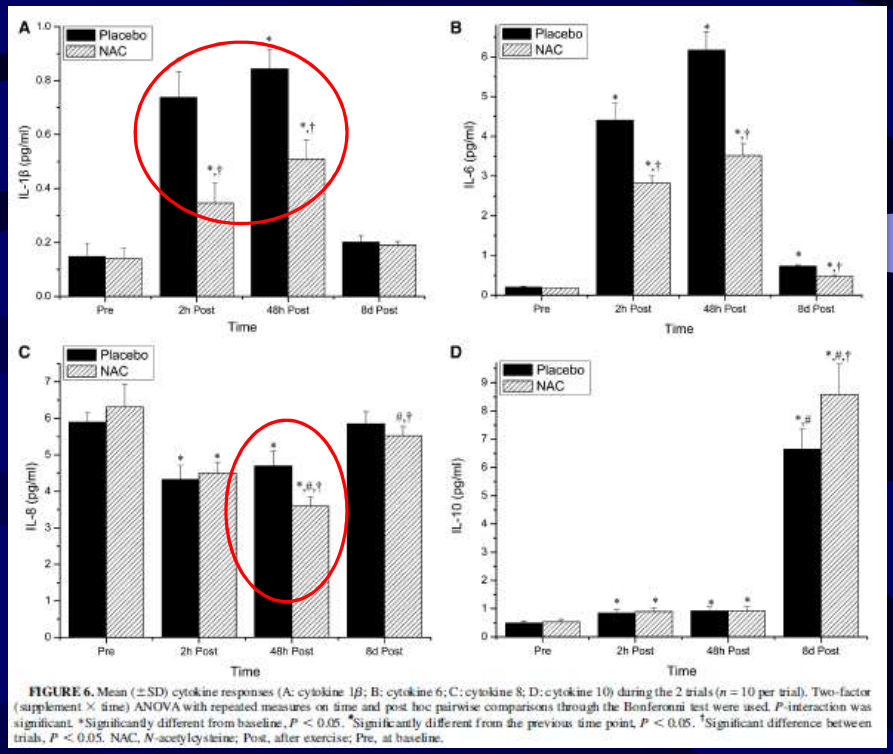


FIGURE 6. Mean (±SD) cytokine responses (A: cytokine 1β; B: cytokine 6; C: cytokine 8; D: cytokine 10) during the 2 trials (n = 10 per trial). Two-factor (supplement × time) ANOVA with repeated measures on time and post hoc pairwise comparisons through the Bonferonni test were used. P-interaction was significant. *Significantly different from baseline, $P < 0.05$. #Significantly different from the previous time point, $P < 0.05$. †Significant difference between trials, $P < 0.05$. NAC, N-acetylcysteine; Post, after exercise; Pre, at baseline.

La NAC atténue l'augmentation des marqueurs de l'inflammation et des dommages musculaires

☹Attention car l'inflammation est nécessaire à la régénération musculaire

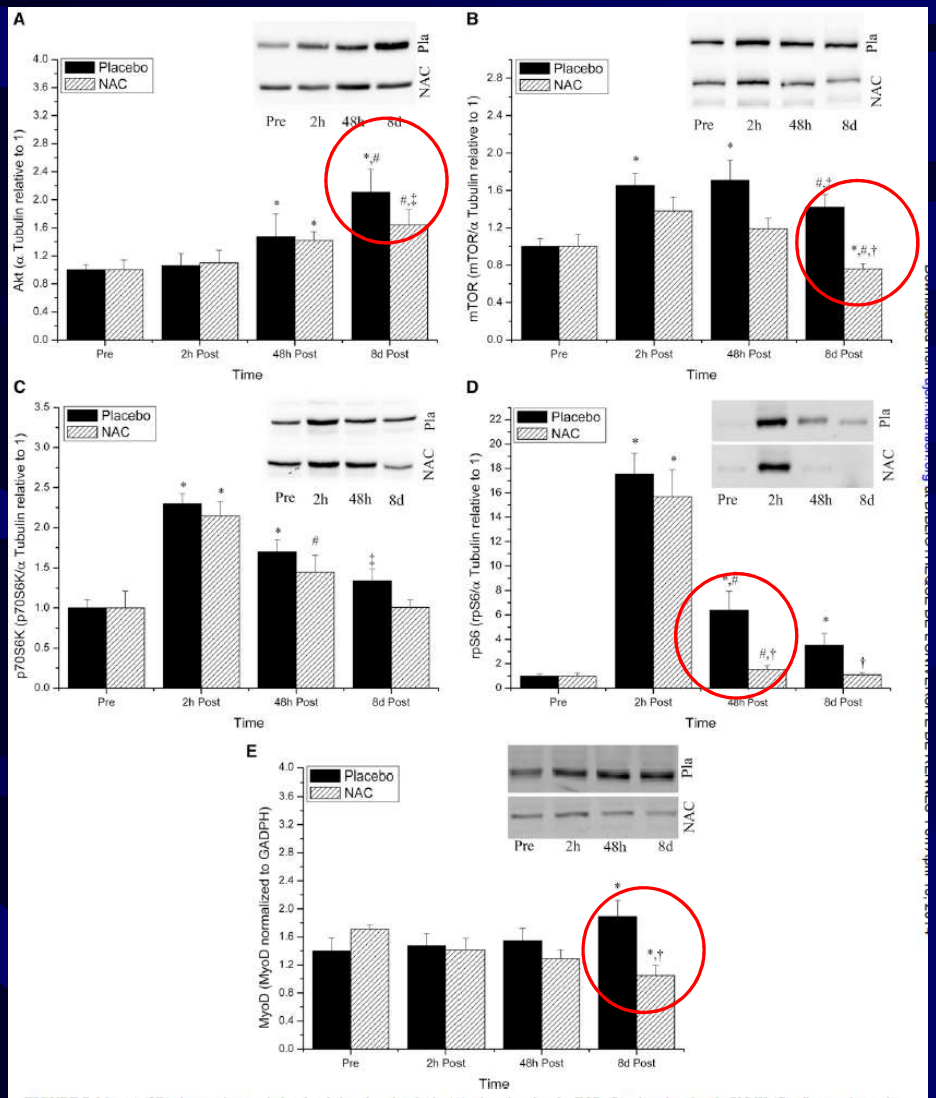


FIGURE 7. Mean (±SD) changes in protein levels of phosphorylated Akt (A), phosphorylated mTOR (B), phosphorylated p70S6K (C), ribosomal protein S6 (D), and MyoD (E) during the 2 trials ($n = 10$ per trial). Two-factor (supplement × time) ANOVA with repeated measures on time and post hoc pairwise comparisons through the Bonferonni test were used. *P*-interaction was significant. *Significantly different from baseline, $P < 0.05$. #Significantly different from the previous time point, $P < 0.05$. †,‡Significant difference between trials: †$P < 0.05$, ‡$P < 0.1$. Akt, protein kinase B; mTOR, mammalian target of rapamycin; MyoD, myogenic determination factor; NAC, *N*-acetylcysteine; Pla, placebo; Post, after exercise; Pre, at baseline; p70S6k, p70 ribosomal S6 kinase; rpS6, ribosomal protein 6.

La NAC atténue les voies de signalisation impliquées dans la synthèse protéique et la régénération musculaire

5- Le revers de la médaille de la supplémentation

5.2- Etudes n'ayant pas montré d'effets délétère de la supplémentation sur les différentes adaptations bénéfiques induites par l'exercice/entraînement

Theodorou et al. (2011)

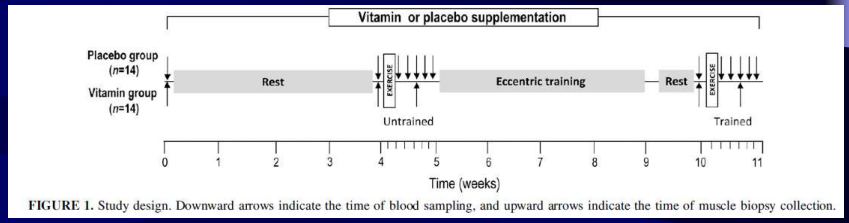


FIGURE 1. Study design. Downward arrows indicate the time of blood sampling, and upward arrows indicate the time of muscle biopsy collection.

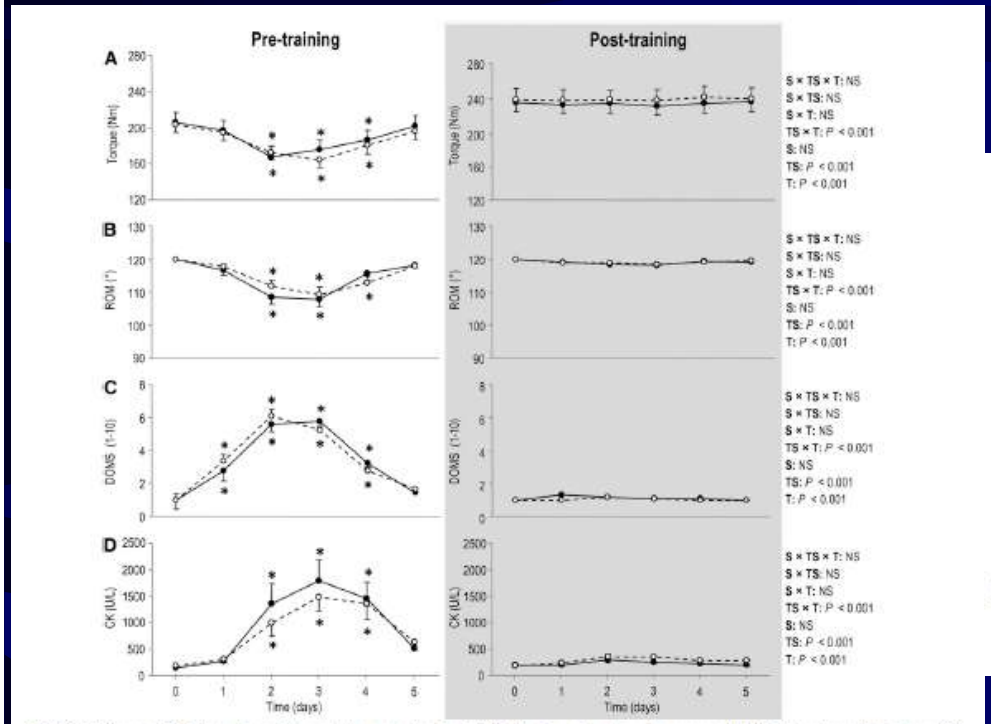


FIGURE 3. Mean (±SEM) isometric peak torque (A), range of motion (ROM; B), delayed onset muscle soreness (DOMS; C), and creatinine kinase (CK; D) in placebo ($n = 14$; ●) and vitamin ($n = 14$; ○) groups in the untrained and trained states. No significant differences were observed between the placebo and vitamin groups at any time point in either the untrained or trained state. Three-factor ANOVAs with repeated measurements on time and post hoc pairwise comparisons through the Sidak test were used. S, main effect of supplement; TS, main effect of training state; T, main effect of time; Nm, Newton meter. Interactions are shown. *Significantly different from the pre-exercise value in the same group, $P < 0.05$.

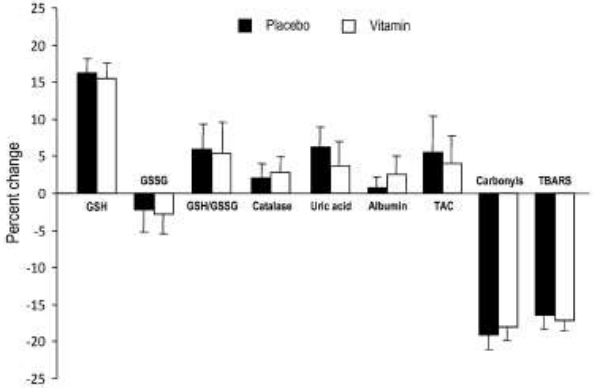


FIGURE 7. Effect of 4 wk of eccentric training on mean (±SEM) blood redox status at rest (before training at week 5 compared with after training at week 11). No significant differences were observed between the placebo ($n = 14$) and vitamin ($n = 14$) groups. Unpaired Student's t test was used for the statistical analysis. GSH, reduced glutathione; GSSG, oxidized glutathione; TAC, total antioxidant capacity; TBARS, thiobarbituric acid–reactive substances.

Pas d'effet de la complémentation sur la performance et les dommages

Pas d'effet de la complémentation sur le statut pro/antioxydant

Yfanti et al. (2010)

Antioxidant Supplementation Does Not Alter Endurance Training Adaptation

CHRISTINA YFANTI[1], THORBJÖRN ÅKERSTRÖM[1], SØREN NIELSEN[1], ANDERS R. NIELSEN[1], REMI MOUNIER[2,3], OLE H. MORTENSEN[4], JENS LYKKESFELDT[5], ADAM J. ROSE[6], CHRISTIAN P. FISCHER[1], and BENTE K. PEDERSEN[1]

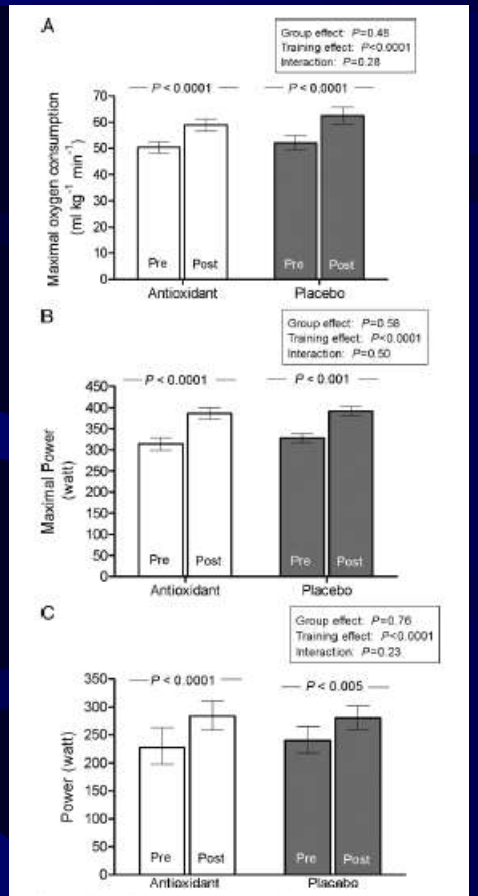


FIGURE 2—Maximal oxygen consumption (A), maximal power output (B), and power output at LT (C) before (Pre) and after (Post) the 12-wk endurance training period. Data for maximal oxygen consumption and maximal power output are presented as means ± SEM. Data for power output at LT are presented as geometric means (95% CI). *Open bars* represent the AO group, and *filled bars* represent the PL group. Data were analyzed using a general linear mixed model.

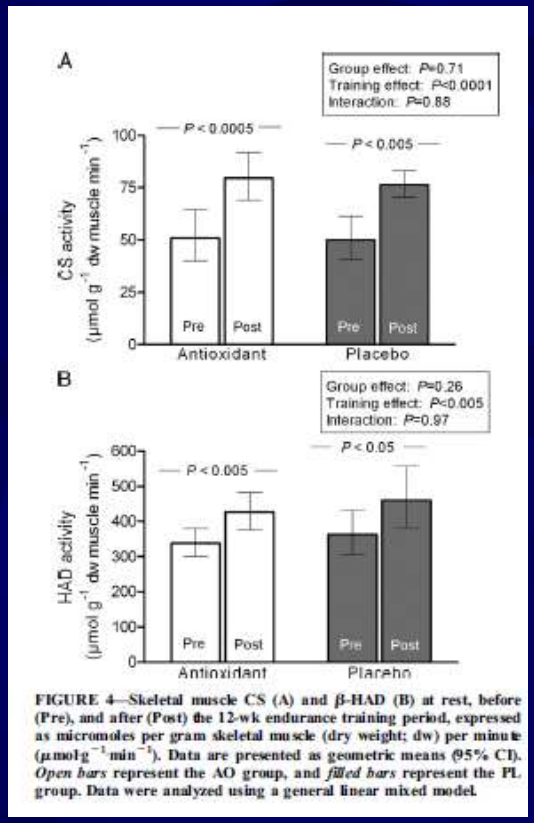


FIGURE 4—Skeletal muscle CS (A) and β-HAD (B) at rest, before (Pre), and after (Post) the 12-wk endurance training period, expressed as micromoles per gram skeletal muscle (dry weight; dw) per minute (µmol·g^{-1}·min^{-1}). Data are presented as geometric means (95% CI). *Open bars* represent the AO group, and *filled bars* represent the PL group. Data were analyzed using a general linear mixed model.

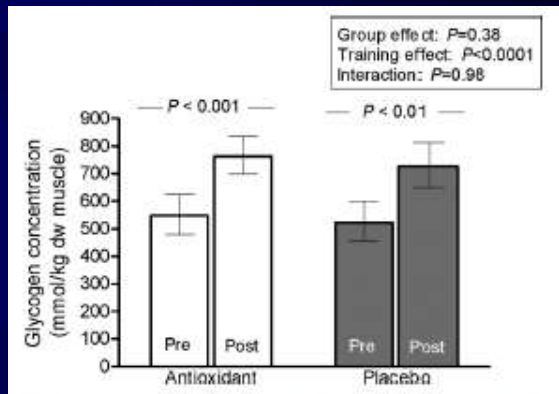


FIGURE 3—Skeletal muscle glycogen concentration before (Pre) and after (Post) the 12-wk endurance training period. Data are presented as geometric means (95% CI). *Open bars* represent the AO group, and *filled bars* represent the PL group. Data were analyzed using a general linear mixed model.

The clear finding in the present study that antioxidants did not influence training adaptation does not exclude the possibility that antioxidant supplementation will exert different effects when applied in combination with less intense training protocols or if given to individuals, who are older, less trained, metabolically impaired, or vitamin deficient at entry.

1 ⇨ Pas d'effets de la supplémentation

Yfanti et al. (2011)

Effect of antioxidant supplementation on insulin sensitivity in response to endurance exercise training

Christina Yfanti,[1] Anders R. Nielsen,[1] Thorbjörn Åkerström,[1] Søren Nielsen,[1] Adam J. Rose,[2] Erik A. Richter,[2] Jens Lykkesfeldt,[3] Christian P. Fischer,[1] and Bente K. Pedersen[1]

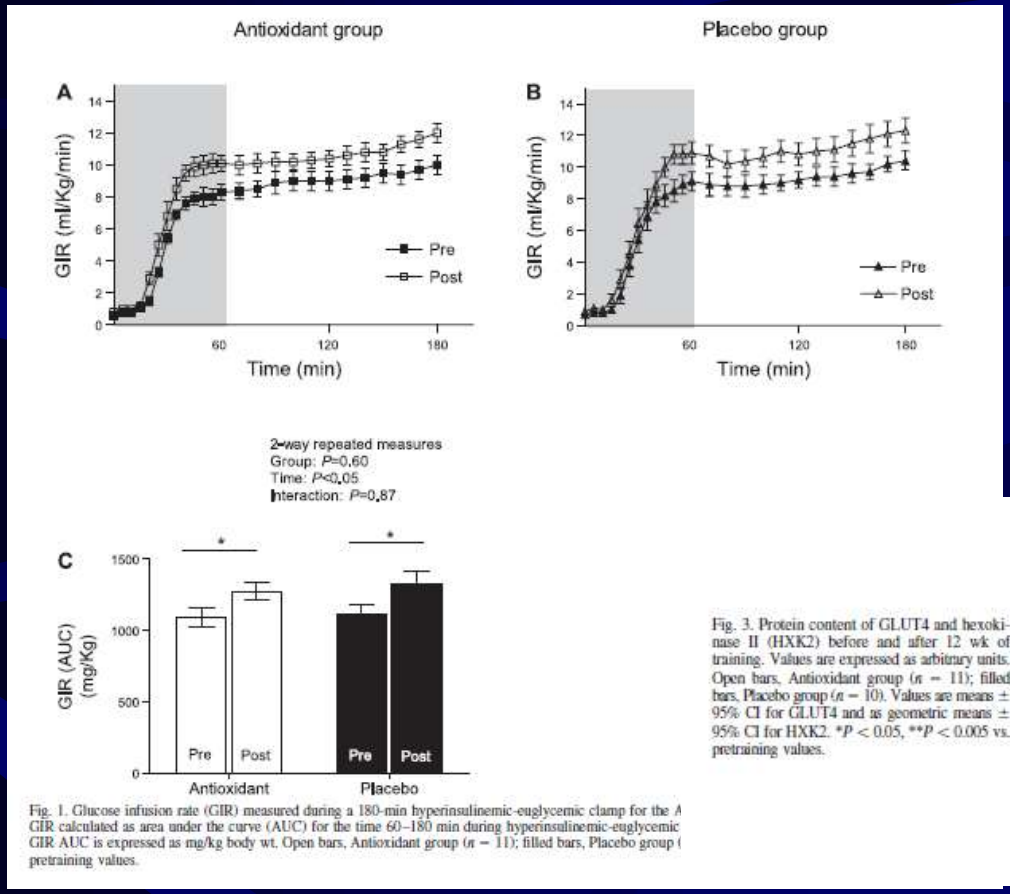


Fig. 1. Glucose infusion rate (GIR) measured during a 180-min hyperinsulinemic-euglycemic clamp for the A GIR calculated as area under the curve (AUC) for the time 60–180 min during hyperinsulinemic-euglycemic GIR AUC is expressed as mg/kg body wt. Open bars, Antioxidant group ($n = 11$); filled bars, Placebo group (pretraining values.

L'entraînement dans le groupe P et C ↗ la sensibilité à l'insuline

Pas d'effet groupe

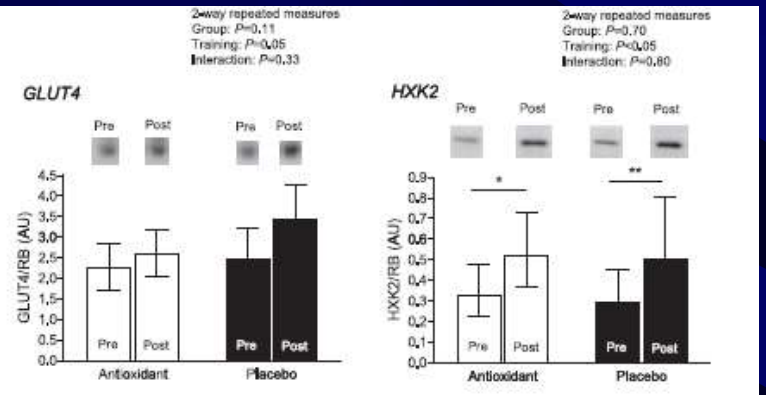


Fig. 3. Protein content of GLUT4 and hexokinase II (HXK2) before and after 12 wk of training. Values are expressed as arbitrary units. Open bars, Antioxidant group ($n = 11$); filled bars, Placebo group ($n = 10$). Values are means ± 95% CI for GLUT4 and as geometric means ± 95% CI for HXK2. *$P < 0.05$, **$P < 0.005$ vs. pretraining values.

1

Pas d'effets de la supplémentation

Yfanti et al. (2011)

Effect of antioxidant supplementation on insulin sensitivity in response to endurance exercise training

Christina Yfanti,[1] Anders R. Nielsen,[1] Thorbjörn Åkerström,[1] Søren Nielsen,[1] Adam J. Rose,[2] Erik A. Richter,[2] Jens Lykkesfeldt,[3] Christian P. Fischer,[1] and Bente K. Pedersen[1]

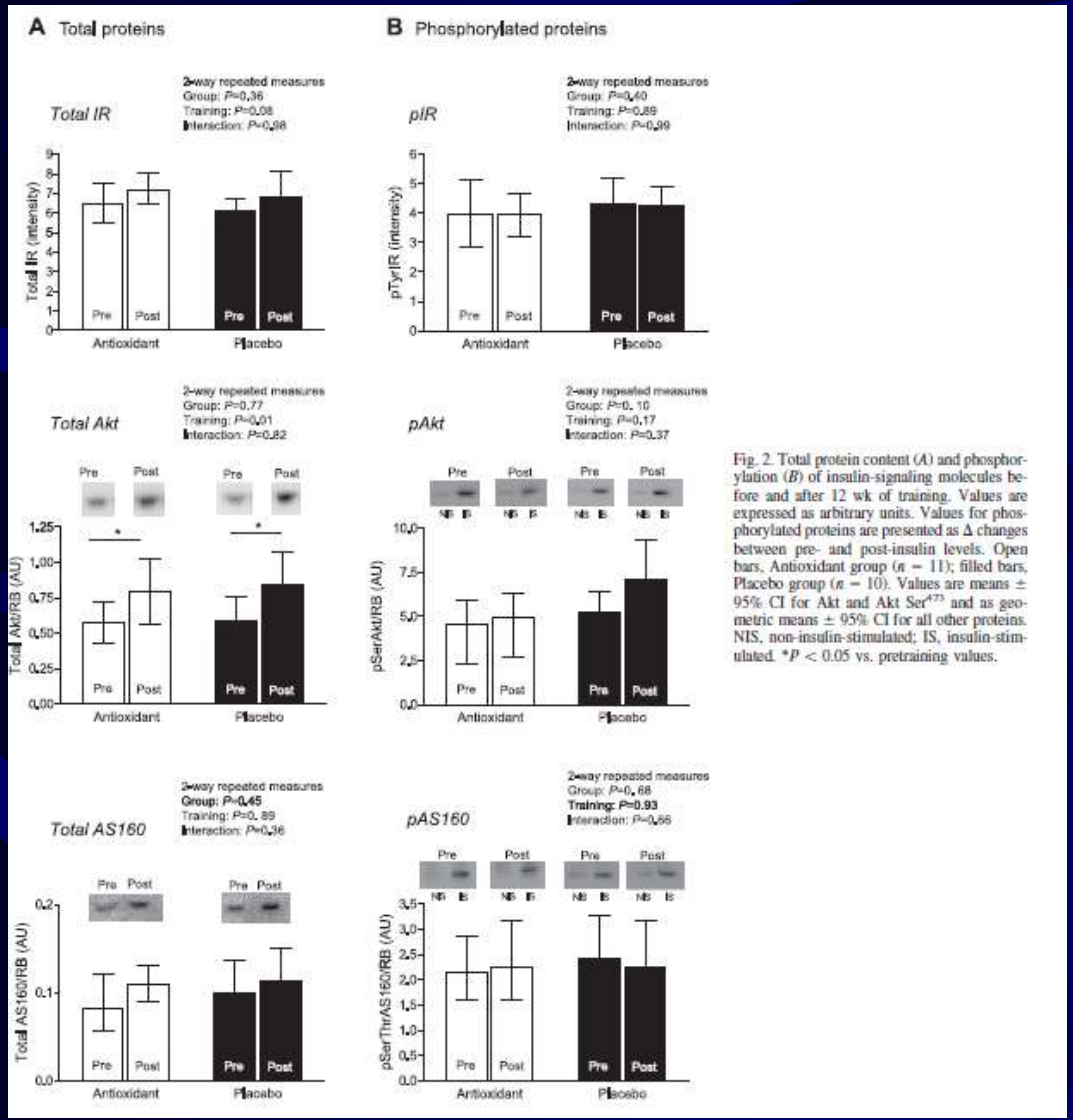


Fig. 2. Total protein content (*A*) and phosphorylation (*B*) of insulin-signaling molecules before and after 12 wk of training. Values are expressed as arbitrary units. Values for phosphorylated proteins are presented as Δ changes between pre- and post-insulin levels. Open bars, Antioxidant group ($n = 11$); filled bars, Placebo group ($n = 10$). Values are means ± 95% CI for Akt and Akt Ser^{473} and as geometric means ± 95% CI for all other proteins. NIS, non-insulin-stimulated; IS, insulin-stimulated. *$P < 0.05$ vs. pretraining values.

5.3- Etudes n'ayant pas montré d'effets délétère d'une **alimentation riche en AO** sur les différentes adaptations bénéfiques induites par l'exercice/entraînement

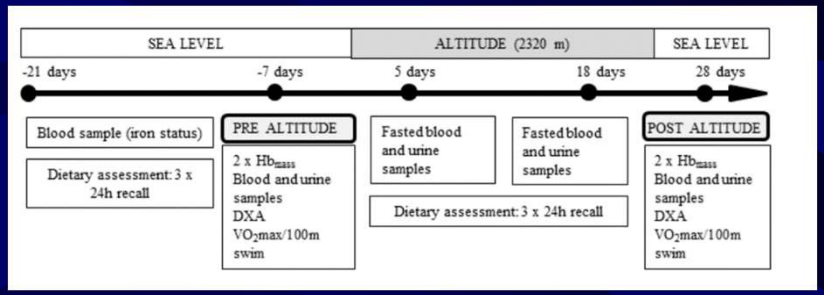
Koivisto et al. (2018)

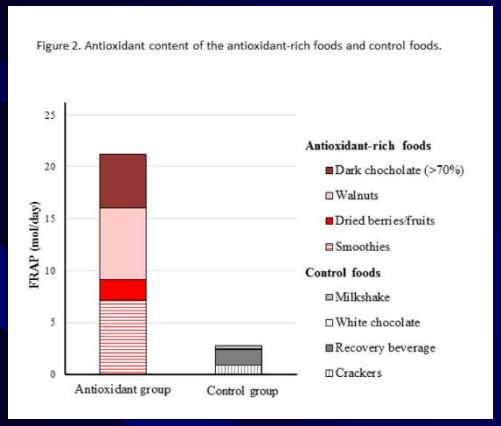
Stage en altitude (2300m) chez SHN endurants

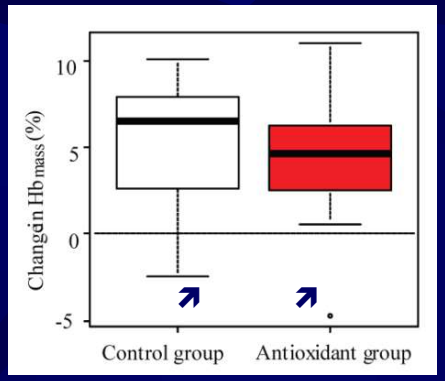
Hypothèse: une alimentation riche en AO bloquerait les adaptations de l'entraînement en altitude

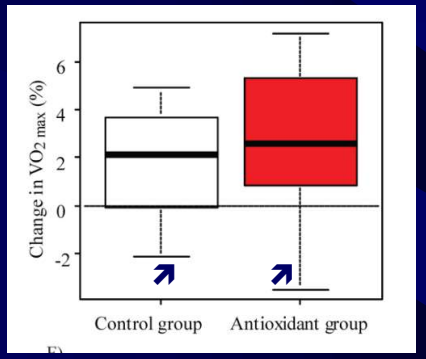
	Antioxidant group (n = 16)			Control group (n = 15)			Between groups
	Pre altitude	Altitude	p_{paired}	Pre altitude	Altitude	p_{paired}	p_{change}
Fruits, vegetable, berries (g/day)	446 ± 155	859 ± 221	< 0.001**	363 ± 116	467 ± 217	0.083	0.001*
Juice (ml/day)	225 (500)	791 (1217)	< 0.001**	244 (533)	450 (775)	0.002*	0.001*
Dark chocolate, >70% cacao (g/day)	0 (23)	40 (14)	< 0.001**	0 (7)	0 (0)	0.317	< 0.001**
Nuts (g)	3.5 (83)	40 (28)	0.009*	10 (73)	0 (42)	0.083	< 0.001**
Coffee (g)	167 (867)	92 (800)	0.878	133 (867)	300 (1017)	0.065	0.470

Values are presented as mean ± std or median (range) for non-normally distributed data. The p-value, p_{paired} is obtained from paired tests testing the change from pre - to post - altitude for the total population (All) either by paired t-test or Kruskal Wallis (^{KW}) test. p_{change} is obtained from comparing the change (from Pre altitude to Altitude) between the groups using t-test or MW tests depending on the normality of the data. * p-value < 0.05, ** p-value < 0.001









↗ Apport AO de 118%

L'alimentation riche en AO ne limite pas les adaptations à l'entraînement

5.4- Etudes n'ayant pas montré d'effets délétère d'autres AO sur les différentes adaptations bénéfiques induites par l'exercice/entraînement

Table 1
Reported effects of antioxidant compounds on exercise-related redox markers, mitochondrial biogenesis, vascular function and performance outcomes 18.

Antioxidant compound (Oral doses used)	Oxidative stress	Antioxidant enzyme levels	Mitochondrial biogenesis	Vascular function	Endurance performance/VO_2 max	Post-exercise muscle recovery (Muscle strength, DOMs, CK, LDH)
Anthocyanins (80–547 mg/day)	Chronic studies (6-21d) *Systemic measures:* ↓ [206–208] ↔ [209–211]	Chronic studies (8-21d) *Systemic measures:* ↑ [207] ↔ [210]	N/A	Chronic studies (7d) ↑ [212] (resting only) ↑ [213] (during exercise)	Chronic studies (3—21d) ↑ [212,214,215] ↔ [210,216,217]	Chronic studies (7-8d) Beneficial [211] No impact [206,207]
Astaxanthin (4–20 mg/day)	Chronic studies (21-90d) *Systemic measures:* ↓ [218] ↔ [219–223]	Chronic studies (21-90d) *Systemic measures:* ↑ [223] ↔ [222]	N/A	N/A	Chronic studies (28d) ↔ [222]	Chronic studies (21-90d) Beneficial [219,223] No impact [221,224]
Catechins (30–1800 mg/day)	Acute studies *Systemic measures:* ↔ [225] (resting only) Chronic studies (14-90d) *Systemic measures:* ↓ [226] ↔ [227–229] *In Skeletal Muscle* ↓ [230]	Acute studies *Systemic measures:* ↑ [225] (resting only) Chronic studies (14-28d) *Systemic measures:* ↔ [227–229]	Chronic studies (28-90d) ↑ [229] (SDH protein expression) [230] ↔ [229] (CS, Cytochrome C protein expression)	Acute studies ↑ [231] Chronic studies (56-84d) ↑ [232] ↑ [233] (resting only)	Chronic studies (2-90d) ↑ [230] ↔ [232,234–236]	Acute studies No impact [237] Chronic studies (2-90d) Beneficial [227,228]
Curcumin (50–2120 mg/day)	Chronic studies (4d) *Systemic measures* ↔ [238]	N/A	N/A	Chronic studies (56d) ↑ [239](resting only)	N/A	Acute studies Beneficial [38] Chronic studies (4-56d) Beneficial [36–38]
Quercetin (250–1000 mg per day – most studies used 1000 mg/day)	Chronic studies (21-24d) *Systemic measures* ↔ [240,241]	Chronic studies (21-24d) *Systemic measures* ↔ [240,241]	Chronic studies (14-24d) ↔ [242,243]	N/A	Chronic studies (7-42d) ↑ [243–247] ↔ [240,242,248,249]	Chronic studies (9-24d) Beneficial [242,250] No impact [249]
Resveratrol (150–600 mg/day)	Chronic studies (56d) *In Skeletal Muscle* Attenuated ↓ [251]	Chronic studies (84d) *In Skeletal Muscle* ↔ [252]	Chronic studies (24-84d) ↑ [252] ↔ [251] Attenuated [253]	Chronic studies (56 d) Attenuated [254]	Chronic studies (28-180d) ↑ [252] ↔ [251,253,255] Attenuated [254]	Chronic studies (7d) No impact [256]
Vitamin C (400–3000 mg/day)	Acute studies *Systemic measures* ↓ [257] ↔ [39] Chronic studies (8-17d) *Systemic measures* ↓ [258–260] ↔ [261,262]	Chronic studies (42d) *In Skeletal Muscle* ↔ [263,264] (resting only)	Chronic studies (42d) *In Skeletal Muscle* ↔ [264] (resting only)	Acute studies (Intravenous or Oral) ↑ [194–197,265] *(ref [196] in older adults only)* ↔ [196] *(ref [196] in younger adults)* ↔ [203,266] Chronic studies (30d) ↔ [203]	Chronic studies (7-56d) ↔ [22,261,267] ↓ [210]	Acute studies No impact [39] Chronic studies (3-17d) Beneficial [258,259,268] No impact [262,269,270] Detrimental [271]
Alpha-lipoic acid (600–1200 mg/day)	Chronic studies (3-10d) *Systemic measures* ↓ [272–275]	Chronic studies (3-10d) *Systemic measures* ↑ [273–275]	N/A	Acute studies ↔ [276]	N/A	Chronic studies (3-10d) Beneficial [272] No impact [273,274]
Coenzyme Q10 (90–300 mg/day)	Chronic studies (20-56d) *Systemic measures* ↓ [277] ↔ [278–281]	Chronic studies (28d) *Systemic measures* ↔ [278]	N/A	Chronic studies (30d) ↑ [282,283]	Chronic studies (8-180d) ↑ [280,282–286] ↔ [31,279,281,287–290] ↓ [291,292]	Chronic studies (20-56d) Beneficial [277] No impact [278,279,293,294]
Vitamin A/β-carotene (Vitamin A: 300 mg/day; β-carotene 30 mg/day)	Chronic studies (28-30d) *Systemic measures* ↔ [295,296]	Chronic studies (28d) *Systemic measures* ↔ [295]	N/A	N/A	Chronic studies (30d) ↔ [296]	N/A
Vitamin E (450-1200 IU)	Chronic studies (14-48d) *Systemic measures* ↓ [297–301] ↑ [302] *In Skeletal Muscle* ↓ [300]	Chronic studies (42d) *Systemic measures* ↑ [302] ↔ [300]	N/A	N/A	Chronic studies (42-180d) ↑ [303,304] ↔ [300,301,305,306]	Chronic studies(30-150d) Beneficial [301] No impact [268,307,308]

(continued on next page)

Mason et al. (2020)

5.4- Etudes n'ayant pas montré d'effets délétère d'autres AO sur les différentes adaptations bénéfiques induites par l'exercice/entraînement

Table 2
Summary and recommendations regarding antioxidant supplementation for people undertaking endurance training.

Antioxidant compound	Evidence summary – exercise-related effects
Anthocyanins	- Effects on oxidative stress and antioxidant enzymes are mixed and limited to systemic data only - Equivocal effects on endurance performance, VO_2 max and post-exercise muscle recovery - May improve blood flow and vascular function, although this does not appear to translate to performance benefits - **Insufficient supportive evidence to recommend to athletes**
Astaxanthin	- Rodent studies show decreased muscle oxidative stress and improved endurance performance; although with possible hampering of training-induced Nrf2 signalling and antioxidant enzyme induction in muscle - Studies in humans are lacking and unclear with respect to effects on oxidative stress, antioxidant enzyme levels, skeletal muscle adaptations, endurance performance and post-exercise muscle recovery - **Insufficient supportive evidence to recommend to athletes**
Catechins	- Overall, effects on oxidative stress and antioxidant enzymes are equivocal; although cocoa flavanols can produce small beneficial effects on systemic markers of oxidative stress - Evidence not supportive of beneficial effects on endurance performance - Evidence equivocal on effects on skeletal muscle mitochondrial biogenesis and post exercise muscle recovery - Can improve vascular function, particularly in overweight/obese individuals – however, this does not appear to translate to improvements in exercise performance - Chronic supplementation may lower RER, increase fat oxidation, decrease carbohydrate oxidation and increase energy expenditure - Potential adverse effects on liver enzymes at high doses (EGCG > 800 mg/day) and lack of clear safety threshold dose - **Insufficient supportive evidence to recommend to athletes**
Curcumin	- Rodent studies show improvements in skeletal muscle oxidative stress, mitochondrial biogenesis and endurance performance - Studies in humans are lacking and unclear with respect to effects on oxidative stress, antioxidant enzyme levels, skeletal muscle adaptations and endurance performance - Limited studies in humans are supportive of benefits on post-exercise muscle recovery, although further research is required to confirm this - **Insufficient supportive evidence to recommend to athletes**
Quercetin	- Minimal evidence of any beneficial effects on systemic markers of oxidative stress - No evidence currently in humans to suggest it will impact on mitochondrial biogenesis in muscle - May result in small beneficial effects on endurance performance, although this is mostly limited to untrained individuals - Effects on muscle recovery post muscle-damaging exercise are equivocal - **Insufficient supportive evidence to recommend to athletes**
Resveratrol	- Findings of rodent studies support improvements in skeletal muscle oxidative stress, antioxidant enzymes and exercise performance. Hov , evidence on these outcomes is limited and unclear in humans. - Limited evidence in humans suggests some hampering of skeletal muscle mitochondrial biogenesis and vascular function, but evidence i e - Future studies should use higher doses (i.e. > 2g/day) for which systemic concentrations of resveratrol and its metabolites are much hi (However, there is an increased risk of adverse effects at high doses) - **Insufficient supportive evidence to recommend to athletes**
Vitamin C	- Has been shown to have mixed effects on systemic markers of exercise-induced oxidative stress and on post-exercise muscle recovery - No convincing evidence of endurance performance benefits - May improve vascular function with exercise, although this appears to be mostly limited to older individuals after acute infusion - While some rodent data suggests impairments in skeletal muscle mitochondrial biogenesis, this has not been explored in humans in the absence of other additional antioxidants - **Insufficient supportive evidence to recommend to athletes**
Alpha-lipoic acid	- Limited evidence is suggestive of benefits on systemic markers of oxidative stress and antioxidant enzymes - Evidence from animal studies shows mixed effects on skeletal muscle oxidative stress, antioxidant enzymes, mitochondrial biogenesis and endurance performance. However, there is a lack of studies in humans investigating these outcomes - **Insufficient supportive evidence to recommend to athletes**
Coenzyme Q10	- No convincing evidence of improvements in markers of oxidative stress, antioxidant enzymes or post-exercise muscle recovery - Unlikely to affect skeletal muscle mitochondrial biogenesis - May improve vascular function, particularly in individuals with heart disease; for whom improvements in VO_2max may occur - Evidence largely mixed for effects on endurance performance - **Insufficient supportive evidence to recommend to athletes**
Vitamin A/β-carotene	- The limited available evidence does not support either vitamin A or β-carotene in improving markers of oxidative stress or improving endurance performance - Limited evidence from rodents shows a hampering of exercise-induced skeletal muscle antioxidant enzyme adaptations after supplementation with retinyl palmitate; however this has not been explored in humans - **Insufficient supportive evidence to recommend to athletes**
Vitamin E	- Studies mostly show improvements in oxidative stress markers - Studies mixed in terms of effects on endurance performance; with most beneficial effects shown in trained athletes at high altitude - Some rodent data indicates hampering of skeletal muscle adaptations to exercise; although effects of vitamin E alone on these outcomes (in the absence of other antioxidants) has not been explored in humans - **Insufficient supportive evidence to recommend to athletes**
Melatonin	- Studies show improvements in systemic markers of oxidative stress and antioxidant enzymes - Rodent studies are supportive of beneficial effects on skeletal muscle oxidative stress and antioxidant enzymes; however, these outcomes have not been explored in humans - Limited studies show acute supplementation is unable to improve time trial performance; but effects of chronic supplementation on performance in humans are lacking - **Insufficient supportive evidence to recommend to athletes**
N-acetylcysteine	- Evidence tends to favour an improvement in sustained exercise performance after acute and chronic NAC supplementation - Evidence from limited acute infusion studies is mixed with respect to effects on skeletal muscle antioxidant levels - Limited evidence suggests NAC might improve aspects of vascular function in older, but not younger participants - Adverse effects limit use of high doses of NAC (> 70 mg/kg), although newer effervescent forms may overcome issues of taste and tolerance - WADA restrictions limit the use of infusions [442], which might limit the applicability of NAC infusion - **Insufficient evidence to recommend to athletes. However, it may be beneficial and well tolerated with doses (< 70 mg/kg) taken chronically over several days prior to an endurance event**

(*continued on next page*)

Antioxidant compound	Evidence summary – exercise-related effects
Vitamin C + E	- A combined dose of 500 mg vitamin C + 400 IU vitamin E does not appear to have any adverse effects on skeletal muscle adaptations to endurance exercise training - A combined dose of 1000 mg vitamin C + 260–400 IU vitamin E has been found in some studies to hamper some markers of mitochondrial biogenesis and antioxidant enzyme induction - There appears to be no effect (beneficial or detrimental) of combined vitamin C + E on endurance exercise performance - Effects on post exercise muscle recovery are limited and equivocal - **Insufficient supportive evidence to recommend to athletes. It might also be wise for athletes to avoid the combination of 1000 mg vitamin C + vitamin E during periods of heavy training in which skeletal muscle adaptations are occurring**
Selenium	- Limited studies in humans have shown decreased exercise-related lipid peroxidation in overweight participants with low selenium levels - One study in humans showed hampering of markers of skeletal muscle mitochondrial biogenesis markers with exercise training; although evidence is limited and mixed overall - Limited studies show no beneficial effects on endurance performance - **Insufficient supportive evidence to recommend to athletes**
Zinc	- Limited evidence available shows some beneficial effects of zinc on systemic markers of exercise-induced oxidative stress - Evidence not supportive of effects on endurance performance, with only limited studies using zinc as the sole compound in supplements - **Insufficient supportive evidence to recommend to athletes**

NAC peut être bénéfique les jours précédant une épreuve d'end.

Mélatonine, Vit E et l'acide α-lipoïque semblent efficaces pour ↘ SO post-exo et sans effet sur perf

Catéchines, les anthocyanines, la coenzyme Q10 et la vitamine C peuvent améliorer la fonction vasculaire. mais les preuves sont limitées à des sous-populations spécifiques et/ou ne se traduisent pas par une amélioration des performances.

Curcumine améliore la récupération musculaire après un exercice intensif,

Confirme que la prise chronique de 1000 mg Vit C + Vit E nuit aux adaptations à l'enraînement tout comme l'astaxanthine, le sélénium et de la vitamine A sur les

Dans l'ensemble, nous soulignons le manque de preuves à l'appui de la plupart des composés antioxydants à recommander aux athlètes

Mason et al. (2020)

5.5- Etudes ayant montré un effet synergique d'une alimentation riche en AO sur les différentes adaptations bénéfiques induites par l'exercice/entraînement

Kan et al. (2018)

But : déterminer les effets du resvératrol (polyphénol) associé à un entraînement de force , sur les adaptations à l'entraînement (force, hypertrophie…)

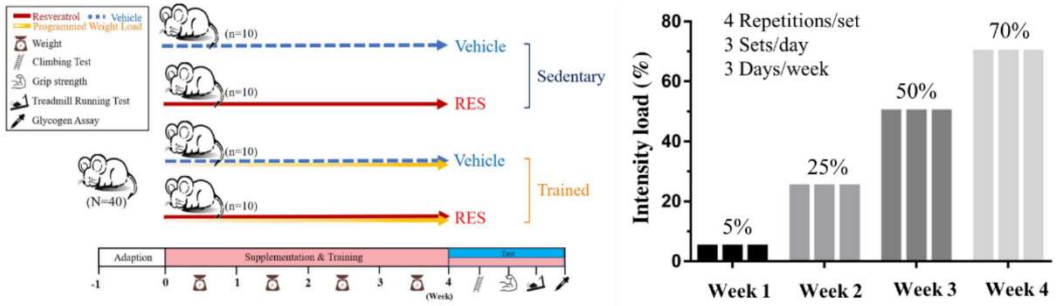


Figure 2. The experimental procedure to evaluate the effects of progressive resistance exercise and resveratrol on aerobic capacity, anaerobic capacity, physiological adaption, and safety (**A**). The incremental loading intensity applied to current training protocol (**B**).

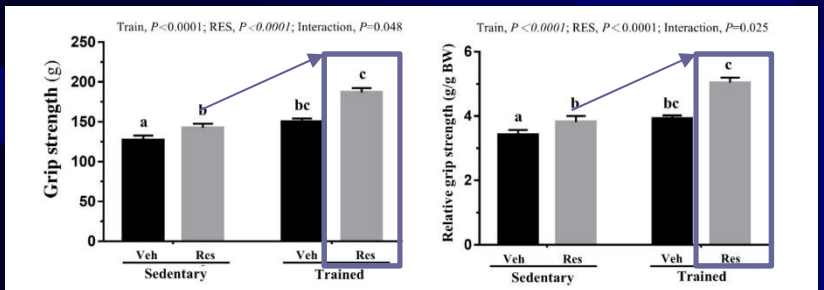


Figure 3. climb training (Trained) and/or resveratrol (RES) supplementation on absolute forelimb grip strength (**A**) and forelimb grip strength (%) relative to bodyweight (**B**). Data are mean ± SEM for $n = 10$ mice per group. Columns with different superscript letters (a, b, c) are significantly different at $p < 0.05$. The abbreviations Veh and RES represented the vehicle and resveratrol supplement, respectively.

Effets synergiques sur la force en valeur absolue et relative

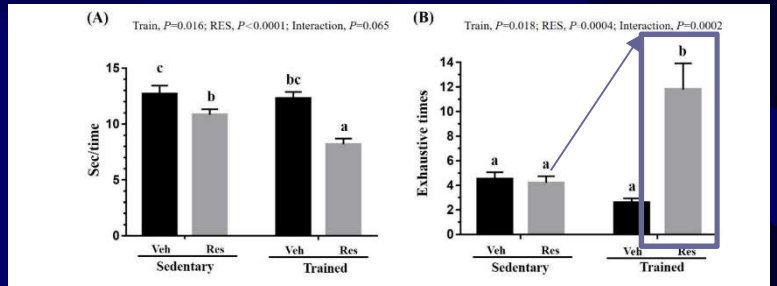


Figure 4. Effect of climb training (Trained) and/or resveratrol (RES) supplementation on anaerobic endurance performance, including time for each climbing (**A**) and exhaustion times for climbing (**B**). Data are mean ± SEM for $n = 10$ mice per group and the columns with different superscript letters (a, b, c) are significantly different at $P < 0.05$. The abbreviations Veh and RES represented the vehicle and resveratrol supplement, respectively.

Effets synergiques sur la capacité anaérobie (tps épuisement grimpés)

5.5- Etudes ayant montré un effet synergique d'une **alimentation riche en AO** sur les différentes adaptations bénéfiques induites par l'exercice/entraînement

Kan et al. (2018)

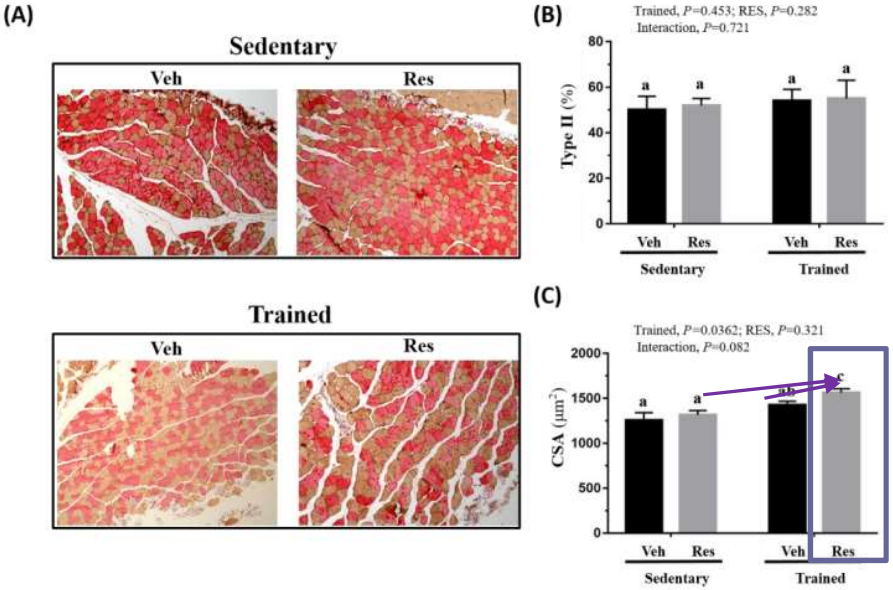


Figure 8. Effect of climb training (Trained) and/or resveratrol (RES) supplementation on the muscle of thigh with IHC staining (**A**), muscular type proportions (**B**), and cross section area (CSA) (**C**). Specimens were photographed under a light microscope. (Hematoxylin and eosin stain, magnification: 200×; scale bar, 40 μm). Bars with different superscript letters (a, b, c) are significantly different at $P < 0.05$. The abbreviations Veh and RES represented the vehicle and resveratrol supplement, respectively.

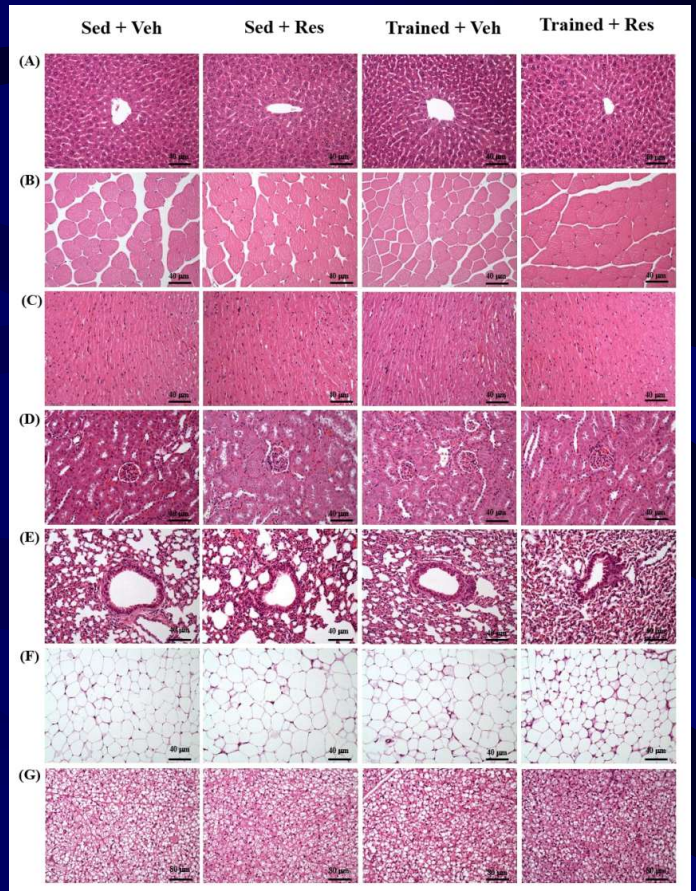


Figure 7. Effect of climb training (Trained) and/or resveratrol (RES) supplementation on the morphology of (**A**) liver; (**B**) skeletal muscle; (**C**) heart; (**D**) kidney; and (**E**) lung (**F**) white adipose tissue (WAT) (**G**) brown adipocytes (BAT) in mice. Specimens were photographed using light microscopy. (Hematoxylin and eosin stain, magnification: 200×; scale bar, 40 or 80 μm).

Effets synergiques sur la force de préhension, la capacité anaérobie liée à la ↘ de la P° de lactate à l'exercice

Explications des résultats contradictoires

Contrôle de l'alimentation

IMPORTANT pour voir un éventuel effet d'une variation d'alimentation sur les ≠ mesures

Age, genre

Protocole de dosage

Durée, dose de la Complémentation
Modalité de la complémentation (complément ou alimentation enrichie)

Durée de l'entraînement
Statut d'entraînement

Bilan 5 – Revers de la médaille

J Physiol 000.00 (2015) pp 1–13

TOPICAL REVIEW

Do antioxidant supplements interfere with skeletal muscle adaptation to exercise training?

Troy L. Merry and Michael Ristow

Energy Metabolism Laboratory, Swiss Federal Institute of Technology (ETH), 8603 Zurich, Switzerland

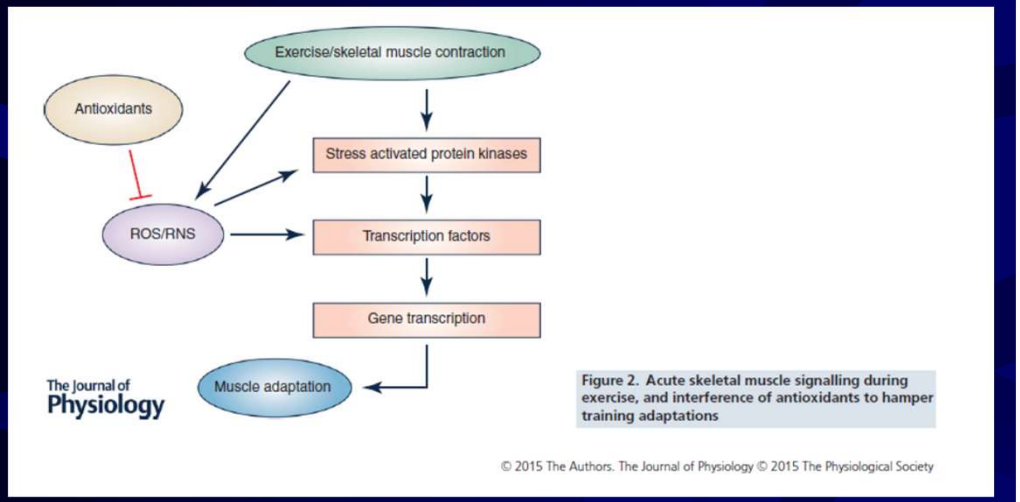


Figure 2. Acute skeletal muscle signalling during exercise, and interference of antioxidants to hamper training adaptations

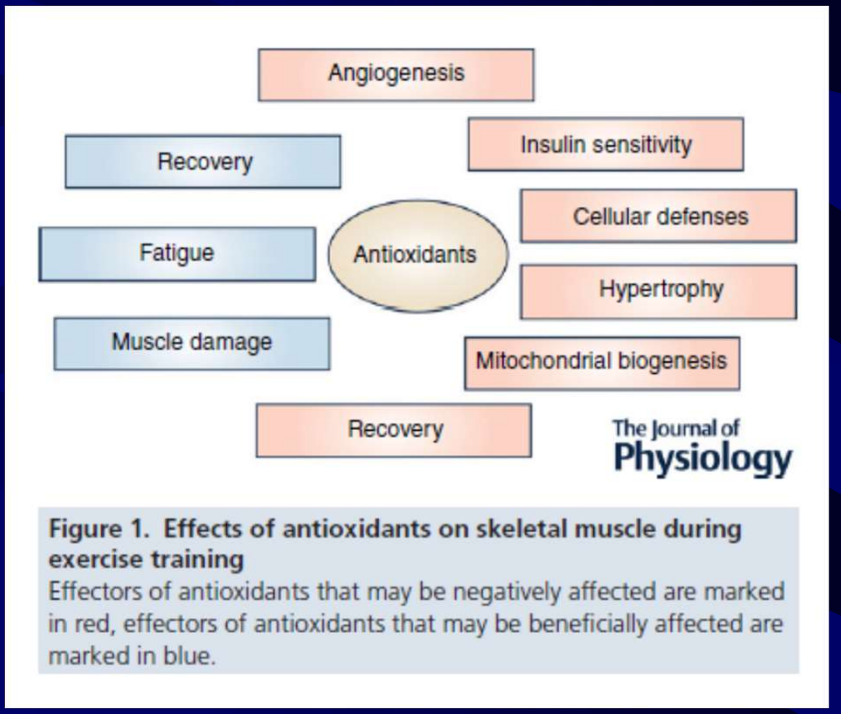


Figure 1. Effects of antioxidants on skeletal muscle during exercise training

Effectors of antioxidants that may be negatively affected are marked in red, effectors of antioxidants that may be beneficially affected are marked in blue.

Les AO peuvent limiter la fatigue, les dommages musculaires mais à haute dose, en interférant avec les voies de signalisation médiées par les ERO, ils peuvent bloquer les adaptations bénéfiques observées à l'exercice/entraînement

Bilan 5 – Revers de la médaille

Toutefois les études ne montrent pas forcément d'effets négatifs mais ne montrent pas forcément d'effets positifs ou synergiques

Une alimentation riche en AO ne bloque pas les adaptations

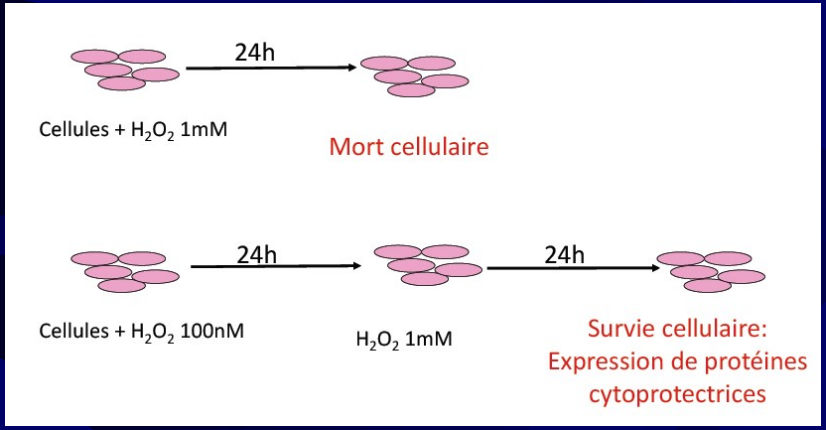
Effets prometteurs de certaines molécules comme le resvératrol qui aurait des effets synergiques → A confirmer

6- Explications avec la théorie de l'Hormésis

- Présentation de la théorie : (Kendig et al. 2010)

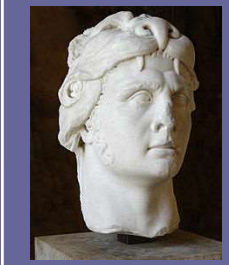
- Réponse biologique favorable en réponse à l'exposition à de faibles doses de toxiques (agents chimiques, irradtation, ERON…).
- Faibles doses d'agents "stressant= > 😐 perturbation transitoire de l'organisme MAIS ☺ adaptation de l'organisme à un niveau supérieur
-Fortes doses: ☹ effets délétères

☞ Exemple H_2O_2 sur culture cellulaire



Cillard J (source personnelle)

Exemple du roi Mithridate



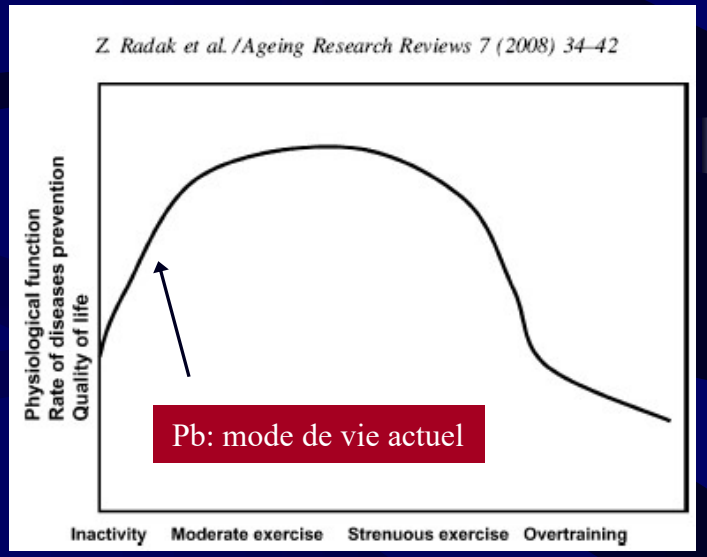
La **mithridatisation** consiste à ingérer des doses croissantes d'un produit toxique afin d'acquérir une insensibilité ou une résistance vis-à-vis de celui-ci. Une application médicale actuelle est la désensibilisation spécifique à un allergène, par exemple le venin des Hymenoptera.

Les ERO produits lors de l'exercice sont-ils nécessaires pour les adaptations ultérieures (a l'entraînement) ? OUI

6- explications avec la théorie de l'Hormésis

- Théorie de l'hormesis valable pour l'exercice:
  - Surcompensation stocks de glycogène
  - Résistance du muscle aux DOMS...
  - Résitance acidose des sprinters (tampons)

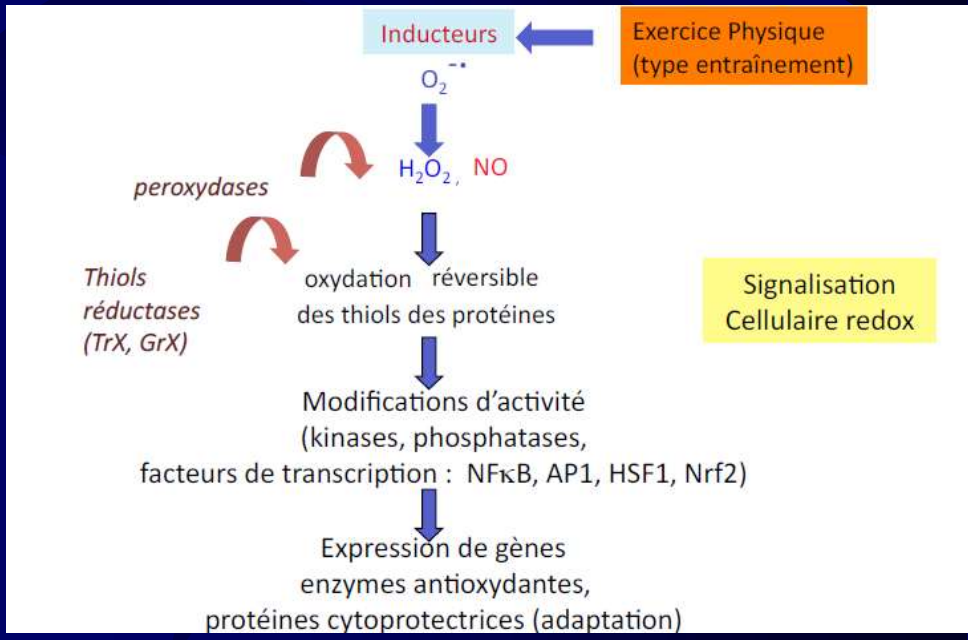
Période de repos obligatoire pour adaptation



- ERO produits par l'exercice et signalisation redox

- Théorie de l'hormesis adaptée à l'exercice

Une élévation modérée des ERO et ERN induit l'expression de protéines cytoprotectrices via la signalisation redox



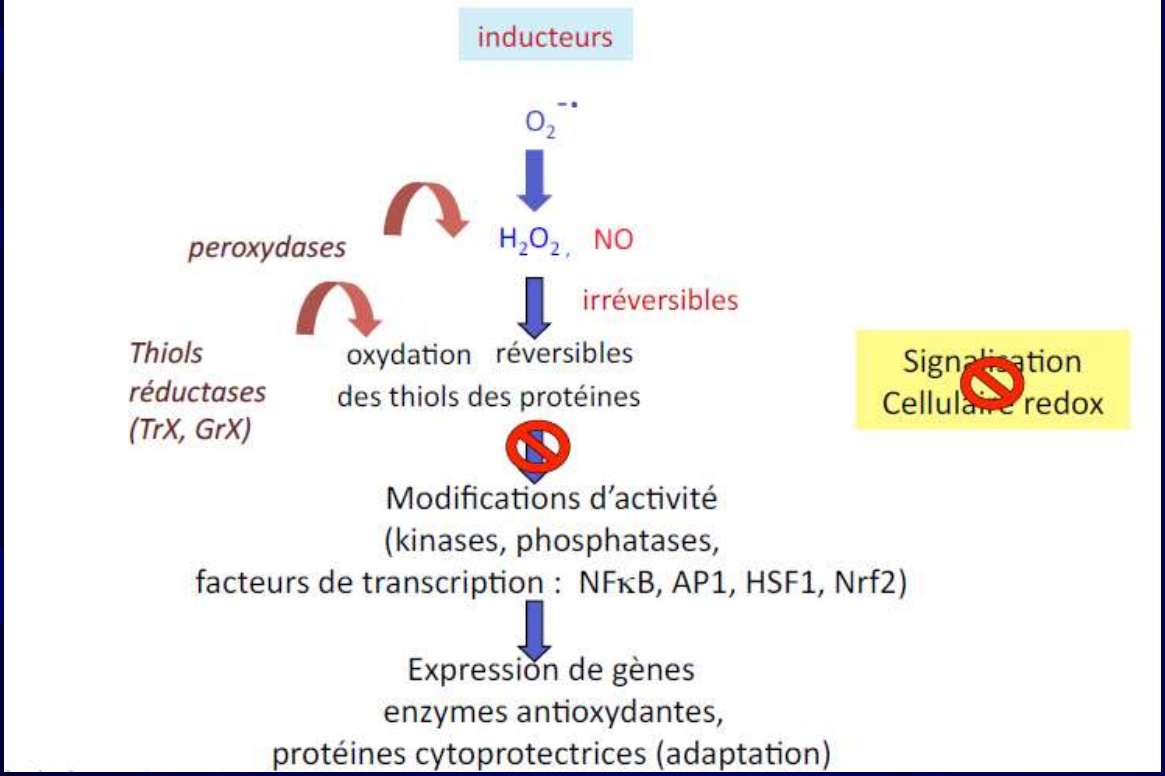
Cillard J (source personnelle)

6- explications avec la théorie de l'Hormésis

- ERO produits par l'exercice et signalisation redox

- Théorie de l'hormesis adaptée à l'exercice

Une élévation importante des ERO et ERN supprime la signalisation redox entraînant un SO



Cillard J (source personnelle)

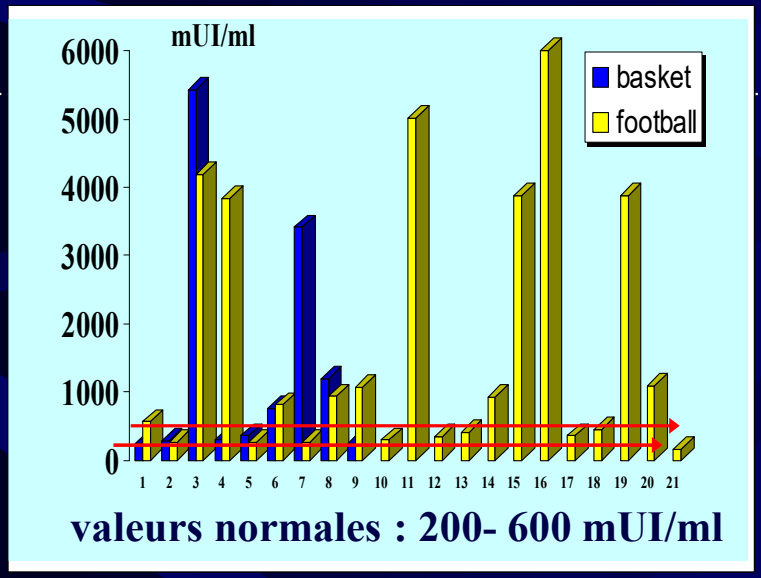
Sportifs de haut niveau ou surentrainés

stress oxydant chez des SHN

adaptation au stress : expression de la SOD

sédentaires	**275 +/- 39 µg/g Hb**
cyclistes amateurs	**588 +/- 230µg/g Hb**
cyclistes professionnels	**323 +/- 67µg/g Hb**

↗ SOD avec I modéré
↘ SOD avec I élevée **(Mena et al., 1991)**



valeurs normales : 200- 600 mUI/ml

titres en anticorps contre les LDL oxydées chez des footballeurs et basketteurs professionnels belges (Pincemail et al., 2000)

stress oxydant et charge d'entraînement

Tansksanen et al. (2010)

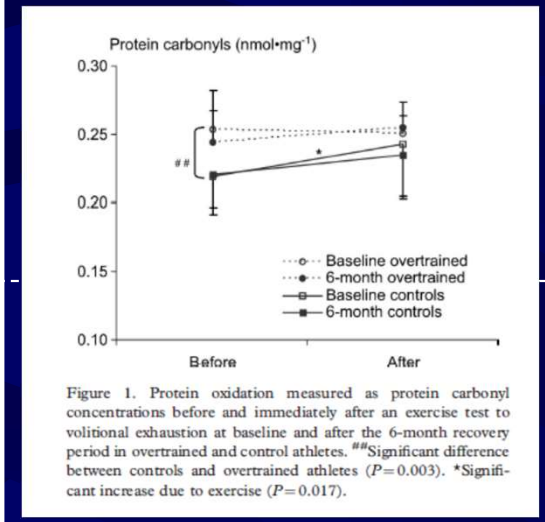


Figure 1. Protein oxidation measured as protein carbonyl concentrations before and immediately after an exercise test to volitional exhaustion at baseline and after the 6-month recovery period in overtrained and control athletes. ##Significant difference between controls and overtrained athletes ($P=0.003$). *Significant increase due to exercise ($P=0.017$).

$SO_{surentraînes} > SO_{normaux}$ au repos

↗ SO avec ↗ I entraînement

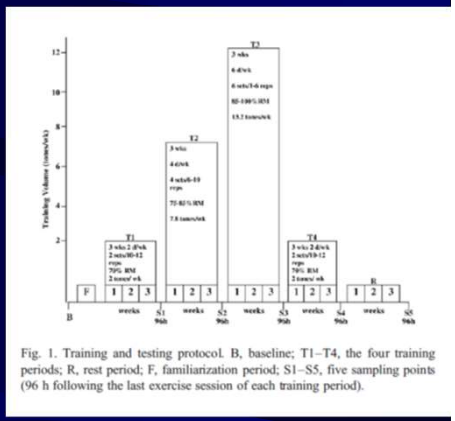


Fig. 1. Training and testing protocol. B, baseline; T1–T4, the four training periods; R, rest period; F, familiarization period; S1–S5, five sampling points (96 h following the last exercise session of each training period).

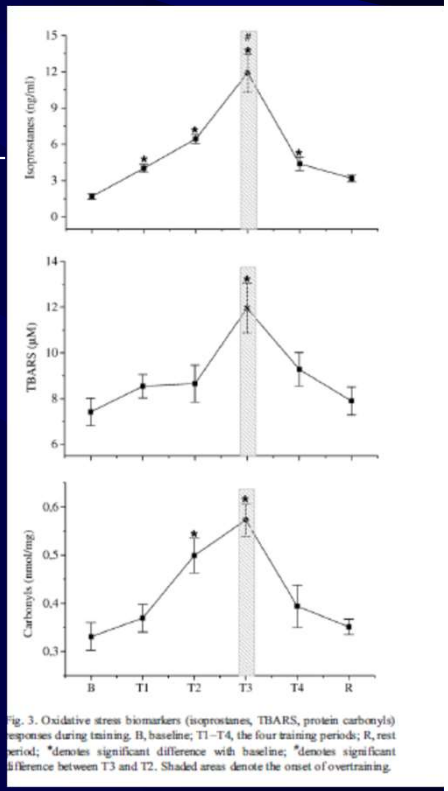
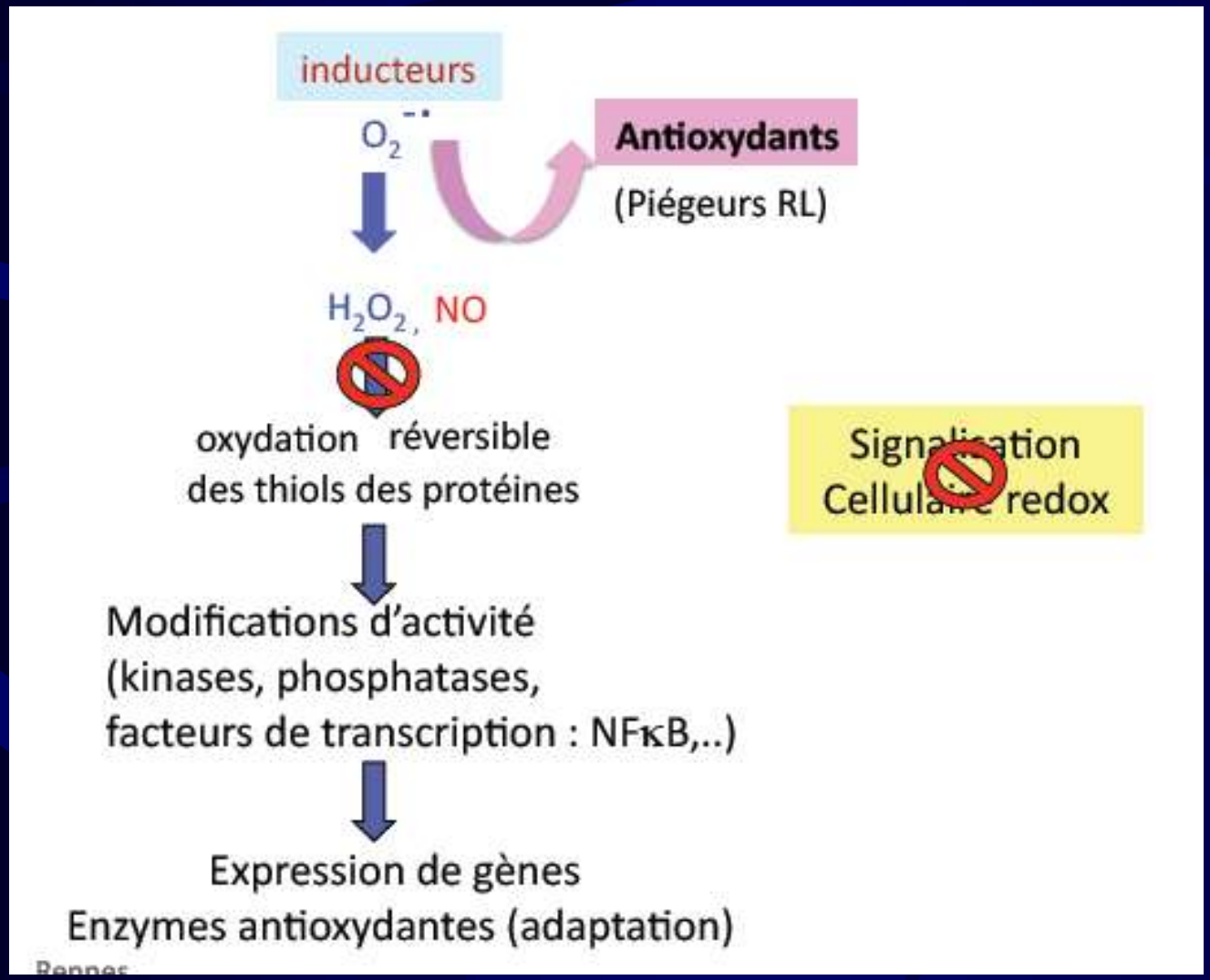


Fig. 3. Oxidative stress biomarkers (isoprostanes, TBARS, protein carbonyls) responses during training. B, baseline; T1–T4, the four training periods; R, rest period; *denotes significant difference with baseline; #denotes significant difference between T3 and T2. Shaded areas denote the onset of overtraining.

Margonis et al. (2007)

6- explications avec la théorie de l'Hormésis

Les AO en excès bloquent la signalisation redox et les adaptations



Cillard J (source personnelle)

Bilan 6

La théorie de l'hormèse peut s'adapter à de nombreuses adaptations dans le sport (surcompensation des stocks de glycogène, acidose, DOMS…)

Elle s'adapte aussi au SO:

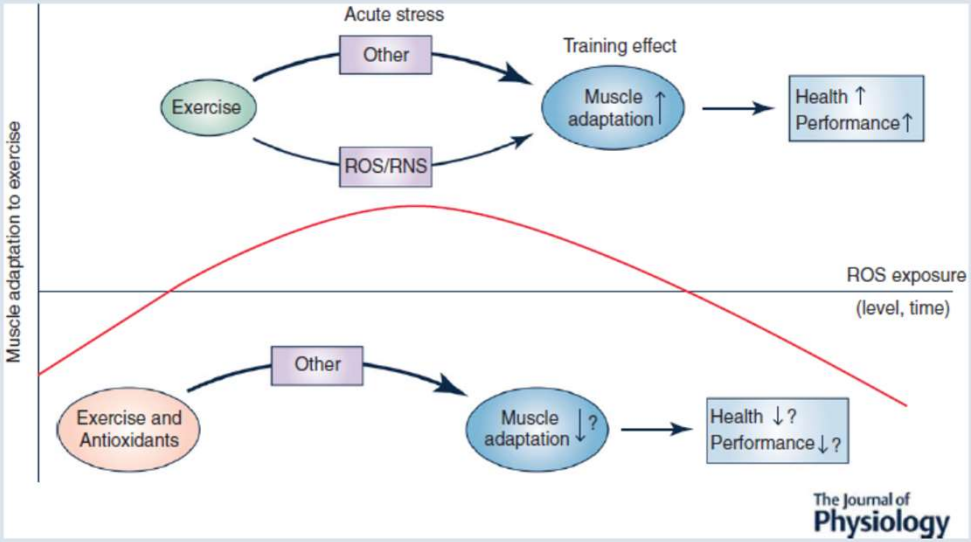
- De faibles doses d'ERO sont nécessaires aux adaptations
- De fortes doses (ou prolongées d'ERO) bloquent les adaptations → cf SHN et surentraînement
- Les AO à forte dose bloquent les adaptations naturelles de l'organisme

Conclusion

- ☺ L'exercice via les ERO qu'il produit active des facteurs de transcription et des voies de signalisation nécessaires pour l'adapation de l'organisme (up-regulation des enzymes antioxydantes, biogénèse mitochondriale, insulin-sensibilité…)

- ☺ L'entrainement aérobie et anaérobie est bénéfique pour l'organisme et renforce les defenses antioxydantes de l'organisme

- ☹ La supplémentation limite voire supprime ces adaptations.



Abstract A popular belief is that reactive oxygen species (ROS) and reactive nitrogen species (RNS) produced during exercise by the mitochondria and other subcellular compartments ubiquitously cause skeletal muscle damage, fatigue and impair recovery. However, the importance of ROS and RNS as signals in the cellular adaptation process to stress is now evident. In an effort to combat the perceived deleterious effects of ROS and RNS it has become common practice for active individuals to ingest supplements with antioxidant properties, but interfering with ROS/RNS signalling in skeletal muscle during acute exercise may blunt favourable adaptation. There is building evidence that antioxidant supplementation can attenuate endurance training-induced and ROS/RNS-mediated enhancements in antioxidant capacity, mitochondrial biogenesis, cellular defence mechanisms and insulin sensitivity. However, this is not a universal finding, potentially indicating that there is redundancy in the mechanisms controlling skeletal muscle adaptation to exercise, meaning that in some circumstances the negative impact of antioxidants on acute exercise response can be overcome by training. Antioxidant supplementation has been more consistently reported to have deleterious effects on the response to overload stress and high-intensity training, suggesting that remodelling of skeletal muscle following resistance and high-intensity exercise is more dependent on ROS/RNS signalling. Importantly there is no convincing evidence to suggest that antioxidant supplementation enhances exercise-training adaptions. Overall, ROS/RNS are likely to exhibit a non-linear (hormetic) pattern on exercise adaptations, where physiological doses are beneficial and high exposure (which would seldom be achieved during normal exercise training) may be detrimental.

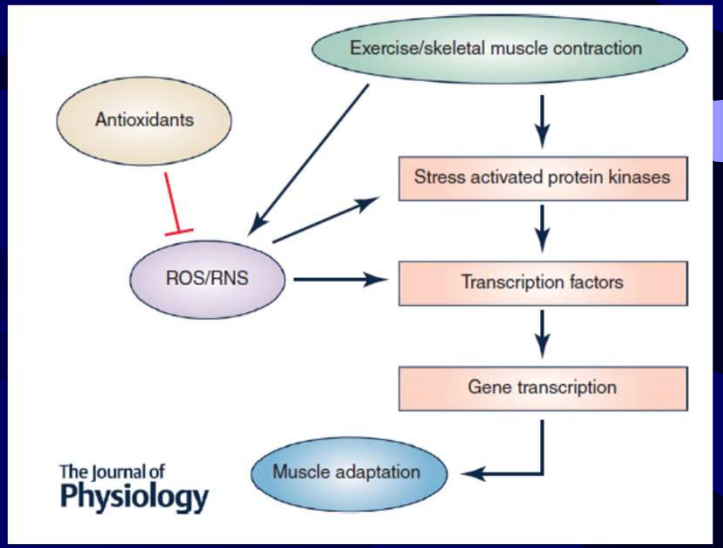


Figure 2. Acute skeletal muscle signalling during exercise, and interference of antioxidants to hamper training adaptations

Merry et Ristow . (2015)

En pratique

- **S'assurer que l 'apport en AO est correct chez sportif**
- **Sujets carencés: supplémentation en AO efficace (sur SO et perf).**

Pachalis et al. (2016) : Rappel exp Vit C dia

Pachalis et al. (2018) : La complémentation en NAC augmente les perfs et réduit le SO chez les carencés uniquement

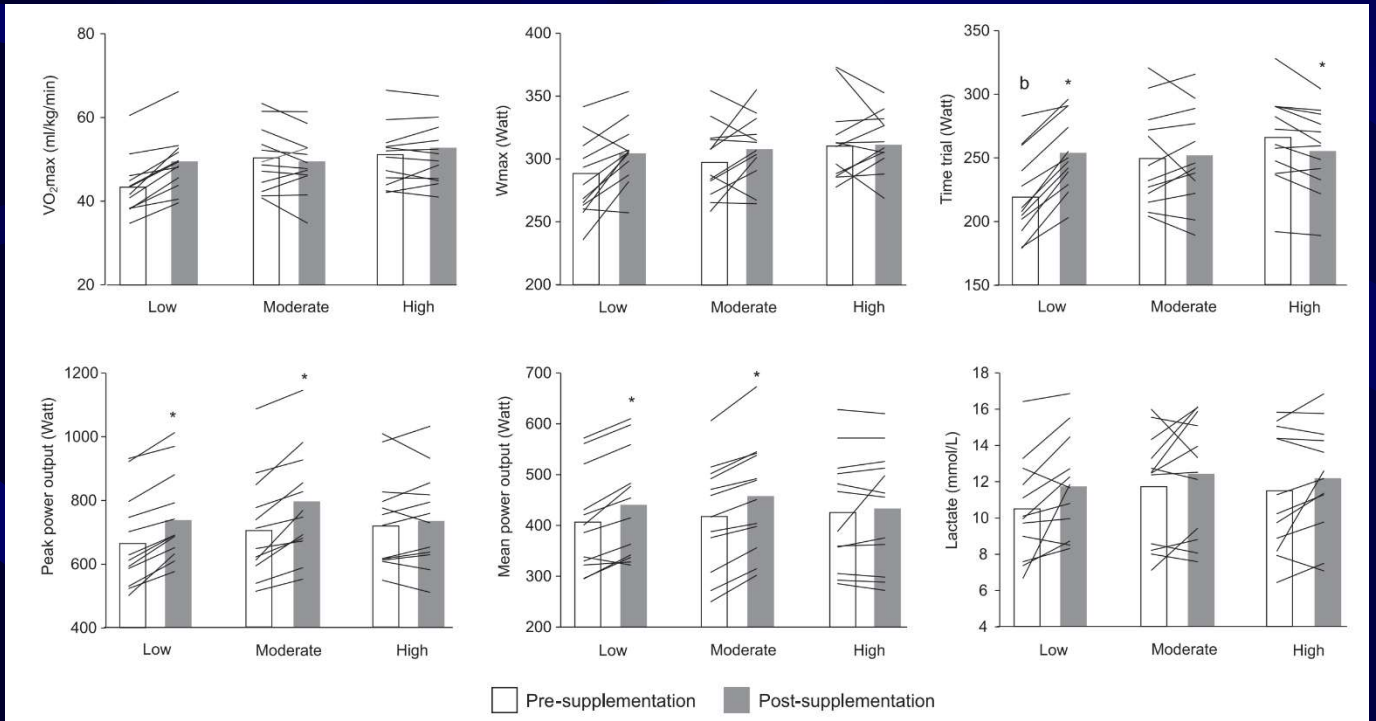


Fig. 3. Exercise performance in the three experimental groups (n = 12 per group) pre and post NAC supplementation (mean ± SD). (*) indicates difference compared to pre-supplementation in the same group, (b) indicates difference between low and high glutathione groups at the same time point.

En pratique

- **Sujets carencés: supplémentation en antioxydants très efficaces (sur SO et perf).**

→ Complémentation personnalisée

Antioxidants in Personalized Nutrition and Exercise

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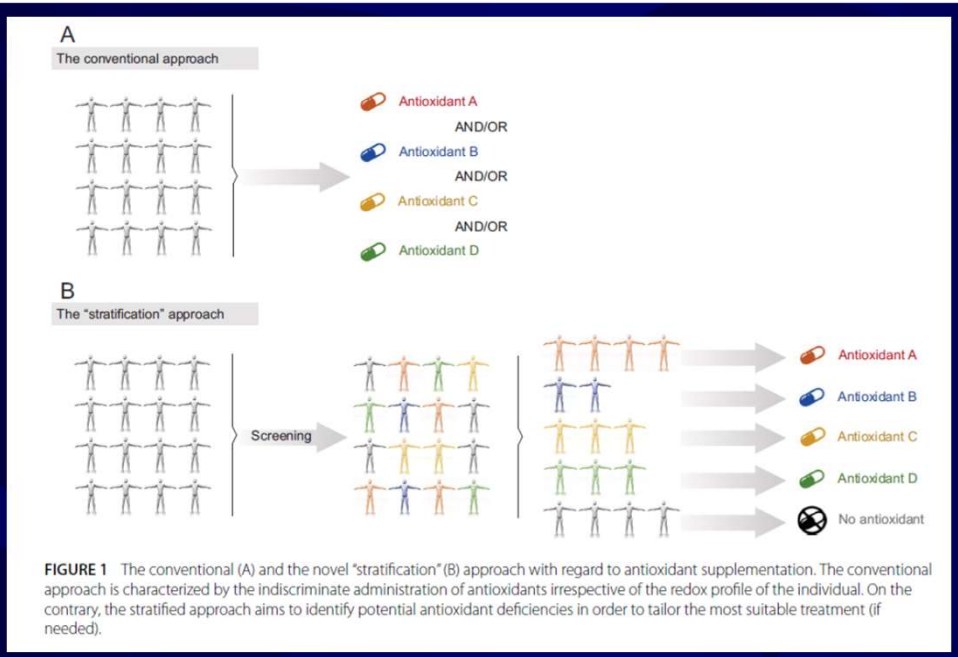

FIGURE 1 The conventional (A) and the novel "stratification" (B) approach with regard to antioxidant supplementation. The conventional approach is characterized by the indiscriminate administration of antioxidants irrespective of the redox profile of the individual. On the contrary, the stratified approach aims to identify potential antioxidant deficiencies in order to tailor the most suitable treatment (if needed).

En pratique

- **Sujets non carencés:**
  - **Supplémentation de courte durée mais PAS PENDANT LA PERIODE D'ADAPTATION (plutôt en compétition sollicitante):**
    - Efficace pour ↘ SO en réponse à l'exercice.
    - Inefficace sur perf
  - **Supplémentation à long terme: déconseillée**
    - Données insuffisantes sur toxicité éventuelle à long terme (surtout supplémentation extraphysiologique)
    - Limite l'adaptation naturelle (forte doses).