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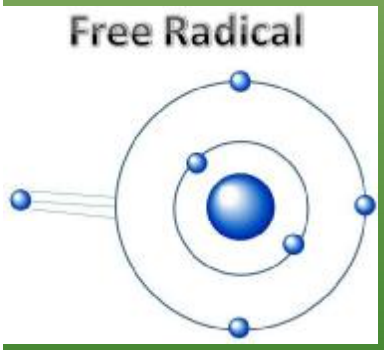
Composés Radicalaires



Oxygen	Superoxide anion	Peroxide
O_2	$\cdot O_2^-$	$\cdot O_2^{-2}$
Hydrogen Peroxide	Hydroxyl radical	Hydroxyl ion
H_2O_2	$\cdot OH$	OH^-

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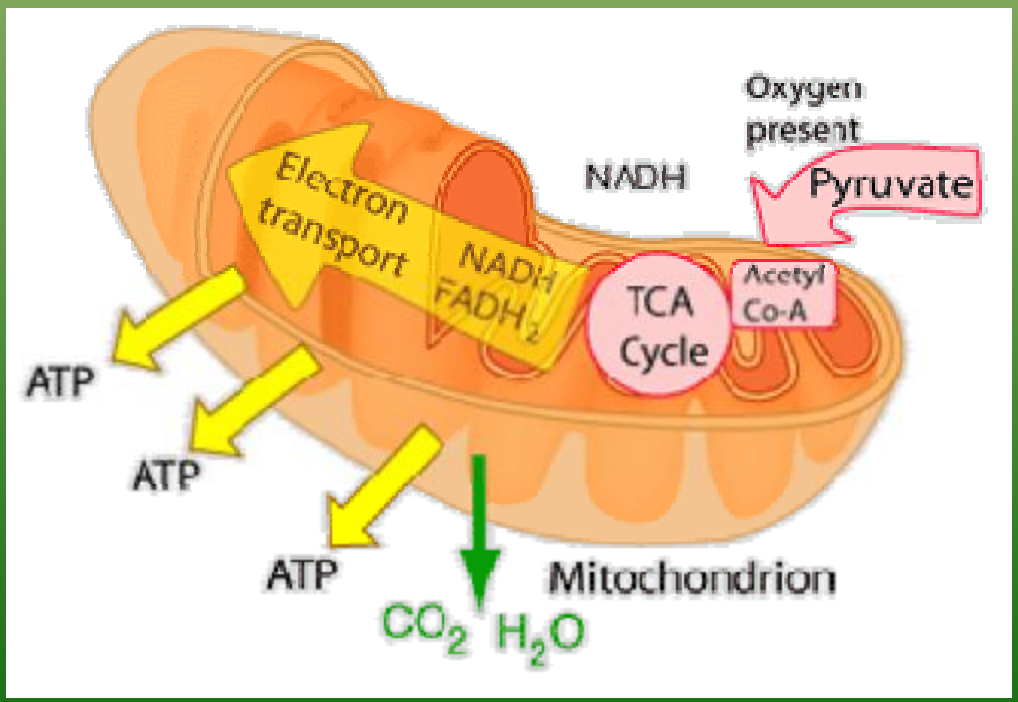
Métabolisme Mitochondrial



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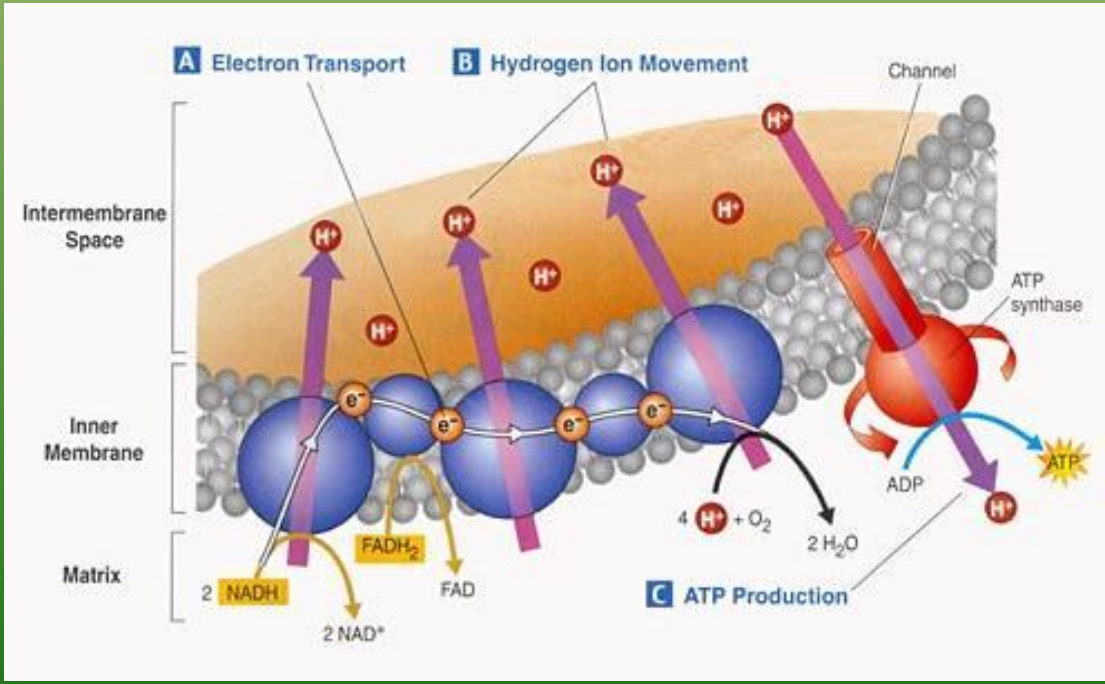
Transport d'Electrons



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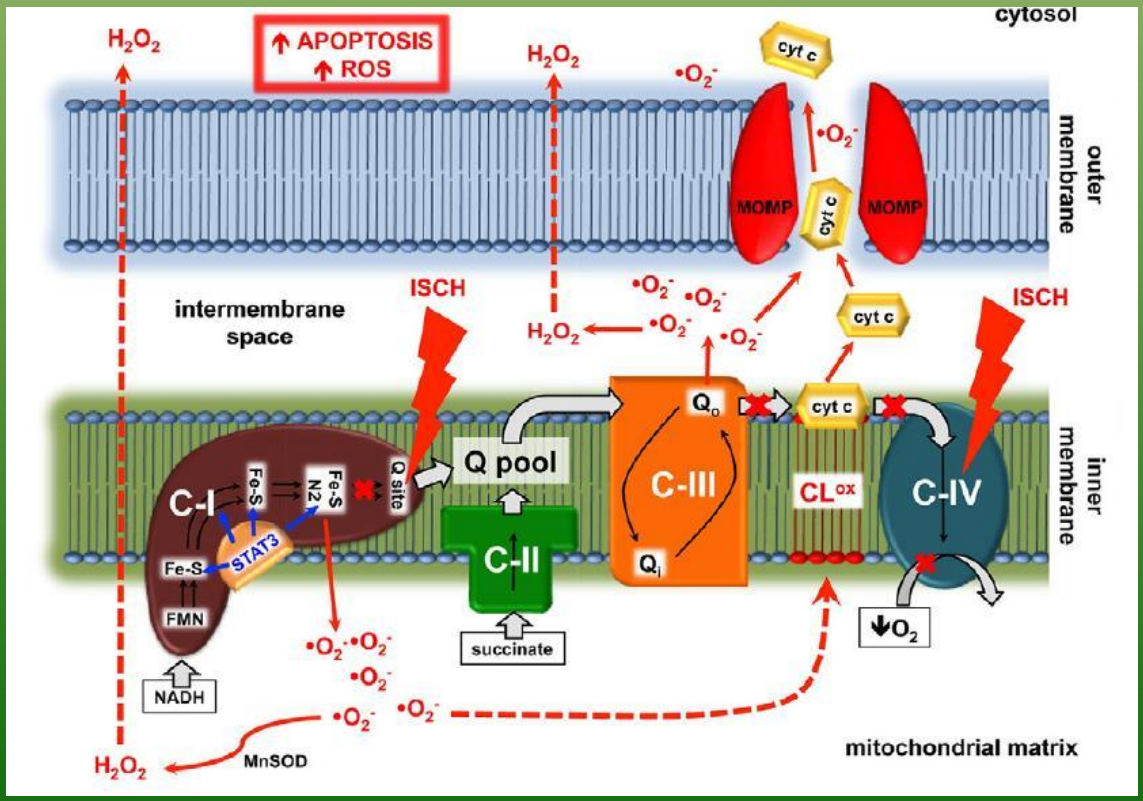
Formation des Radicaux libres



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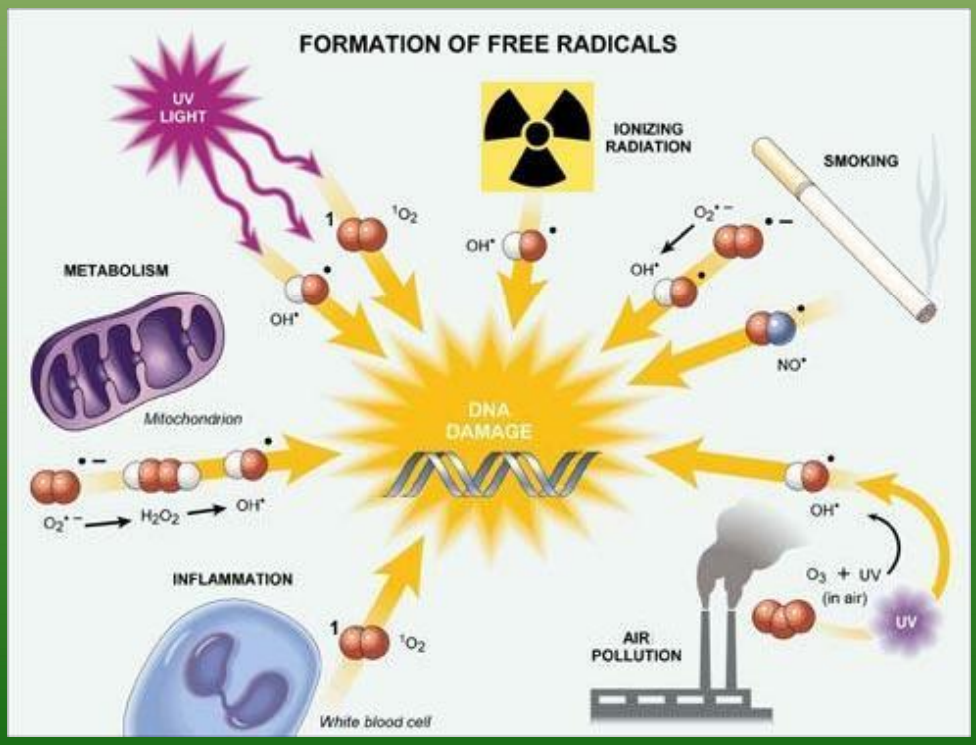
Autres Causes de Stress Radicalaire



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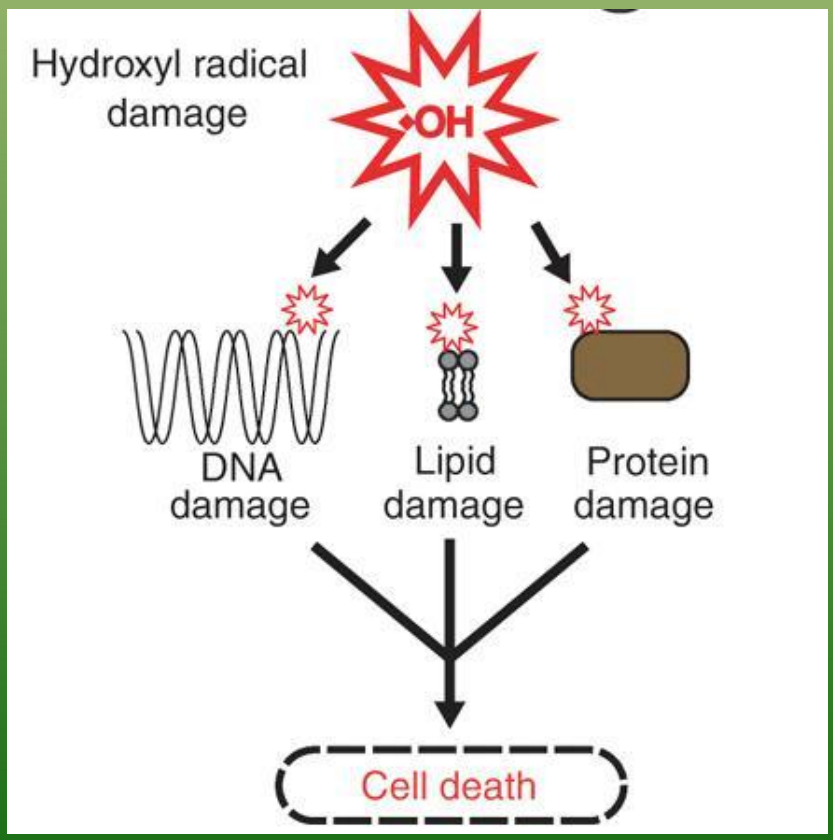
Stress Radicalaires



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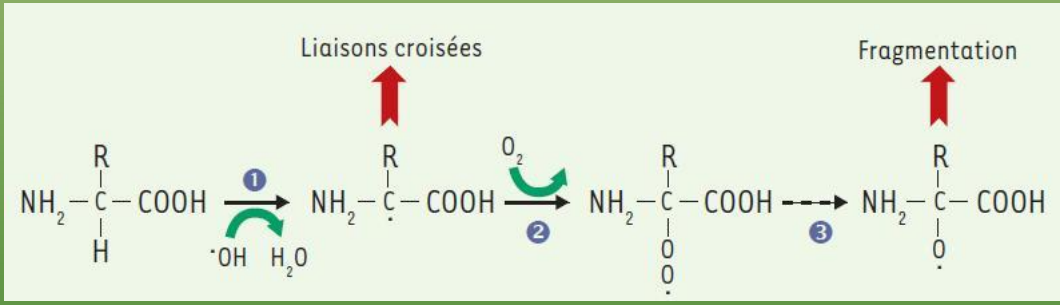
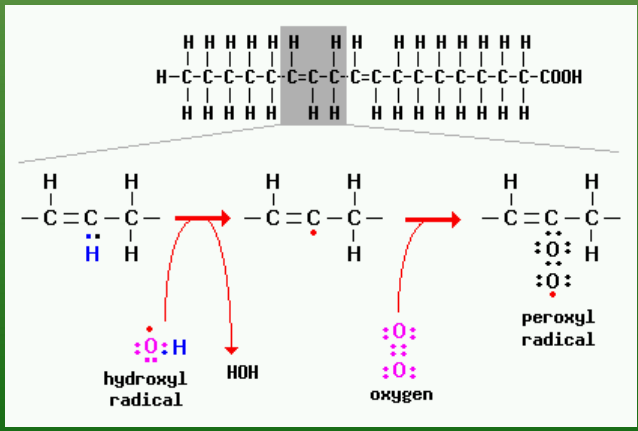
Altération des Protéines



Altération des Acides Gras



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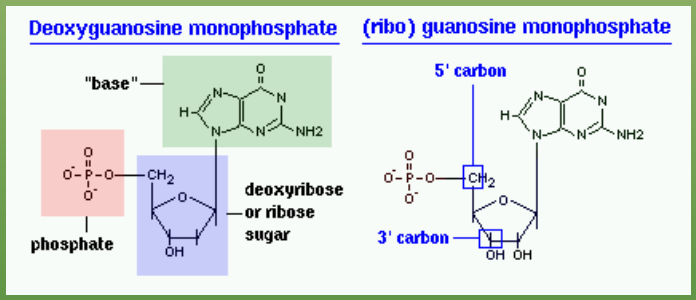
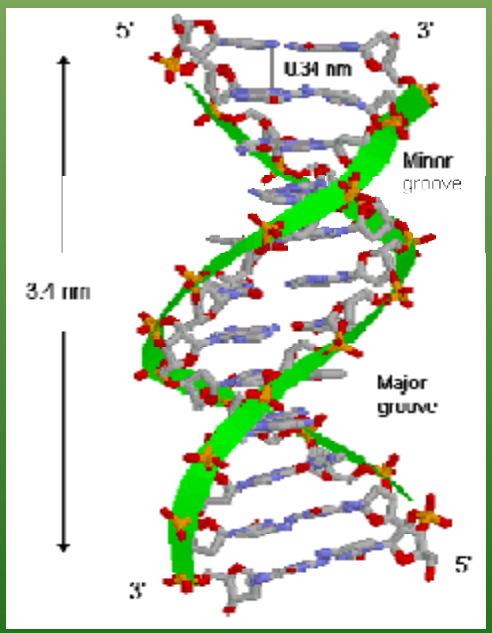
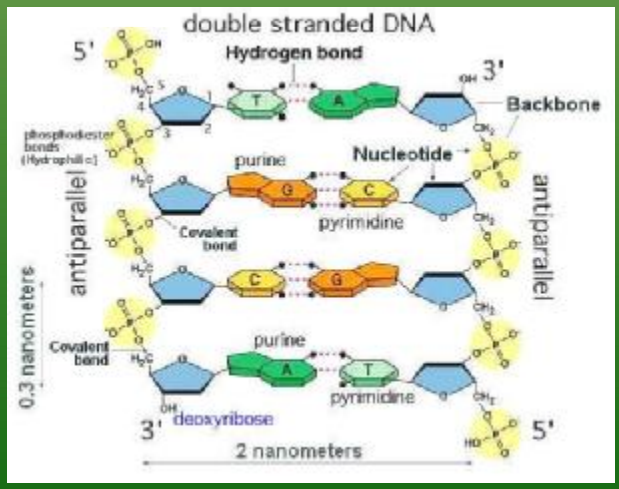
Structure du DNA







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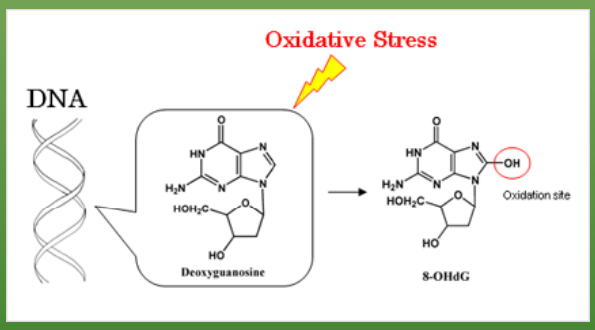
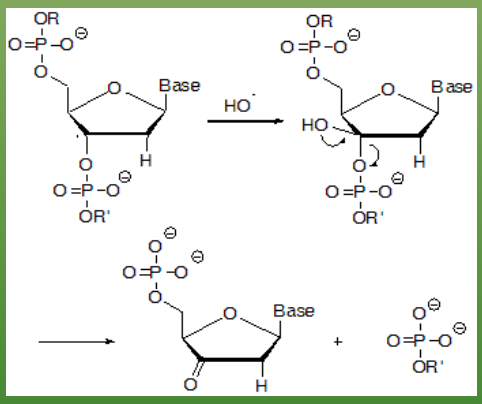
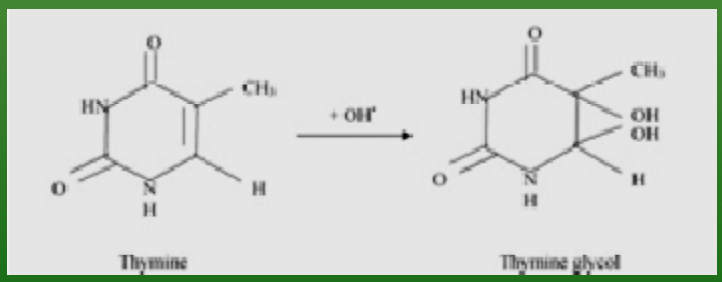
Altérations du DNA







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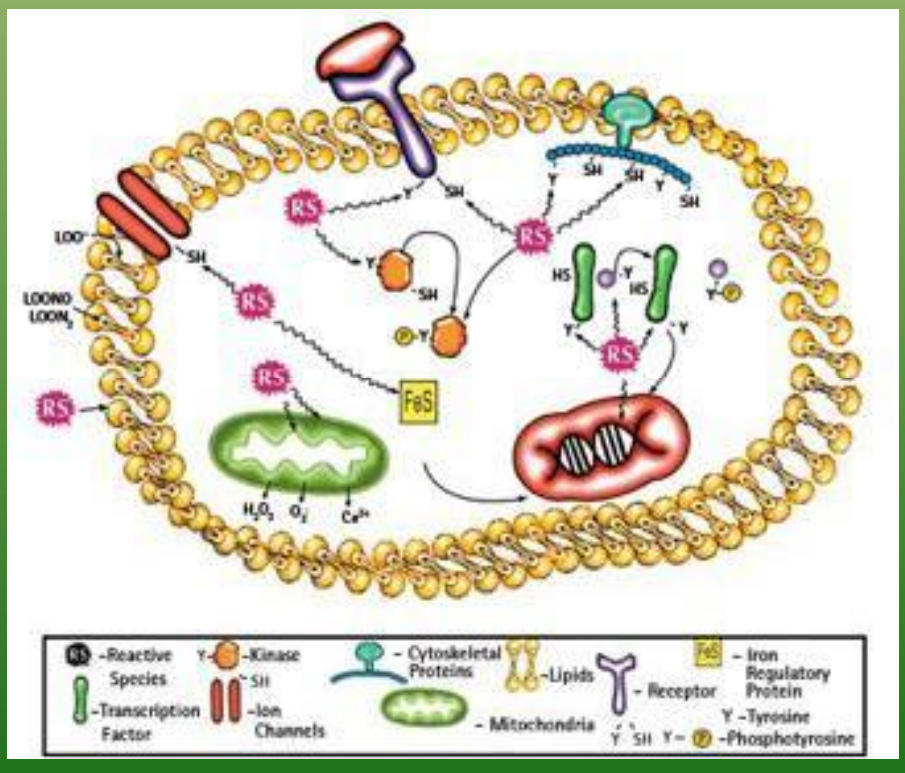

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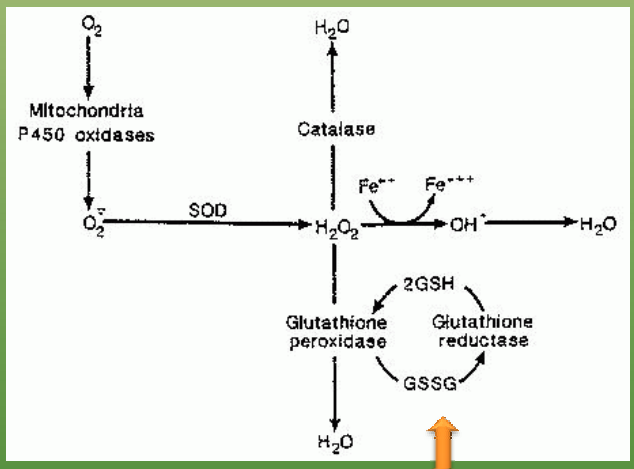
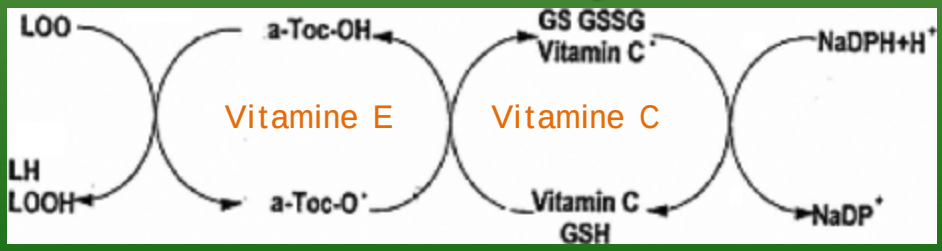
Systèmes Antioxydants





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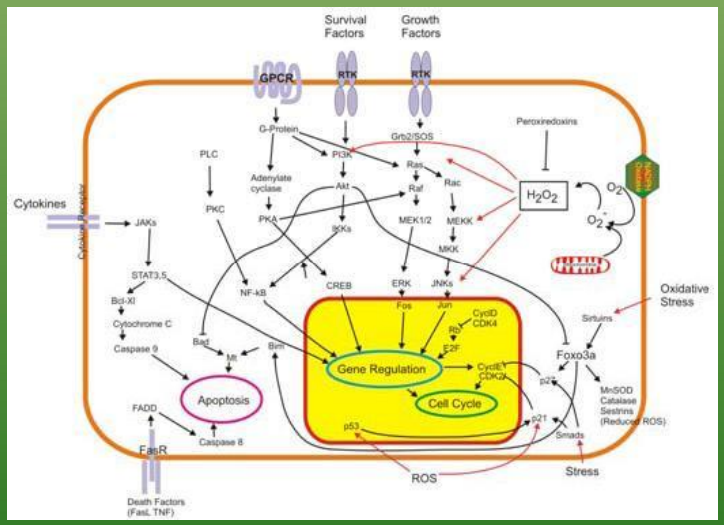

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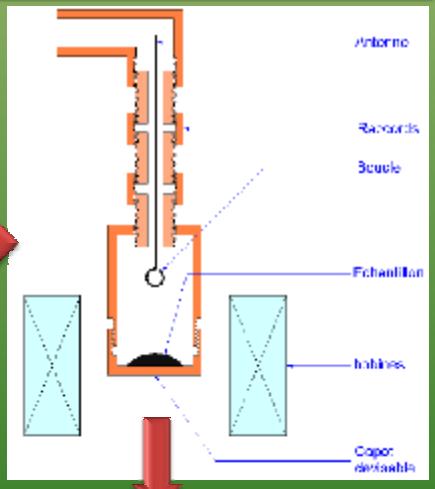
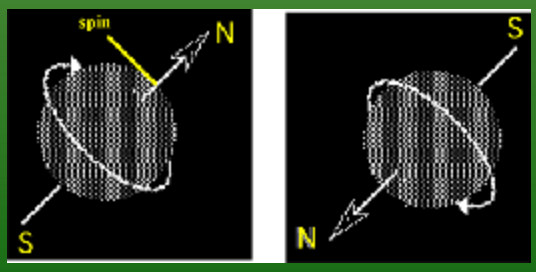
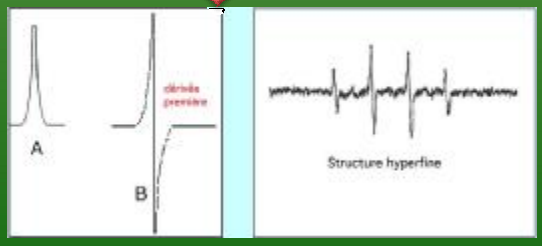
Résonnance Paramagnétique de l'électron (RPE)







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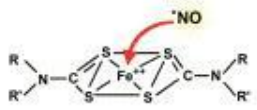
Spectres de RPE (1)

- Freeze trapping

- Spin trapping : Spin trap + RL$^{\bullet}$ → Spin adduct $^{\bullet}$

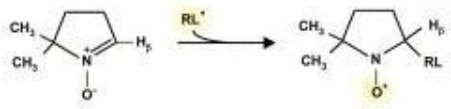
  - Fe-MGD



$^{\bullet}NO$ →

  - DMPO

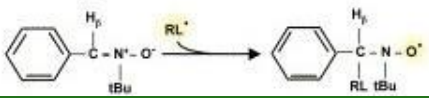
$O_2^{\bullet -}$ →



$^{\bullet}OH$ →

R$^{\bullet}$ →

  - PBN



R$^{\bullet}$ →

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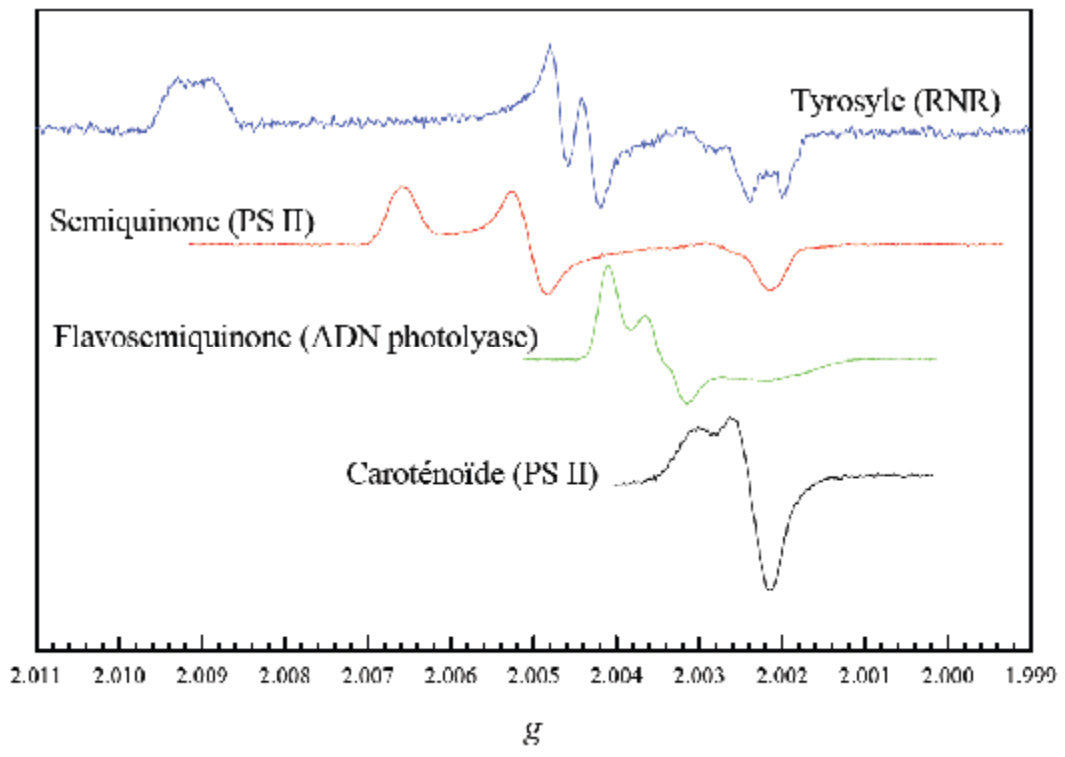
Spectres de RPE (2)



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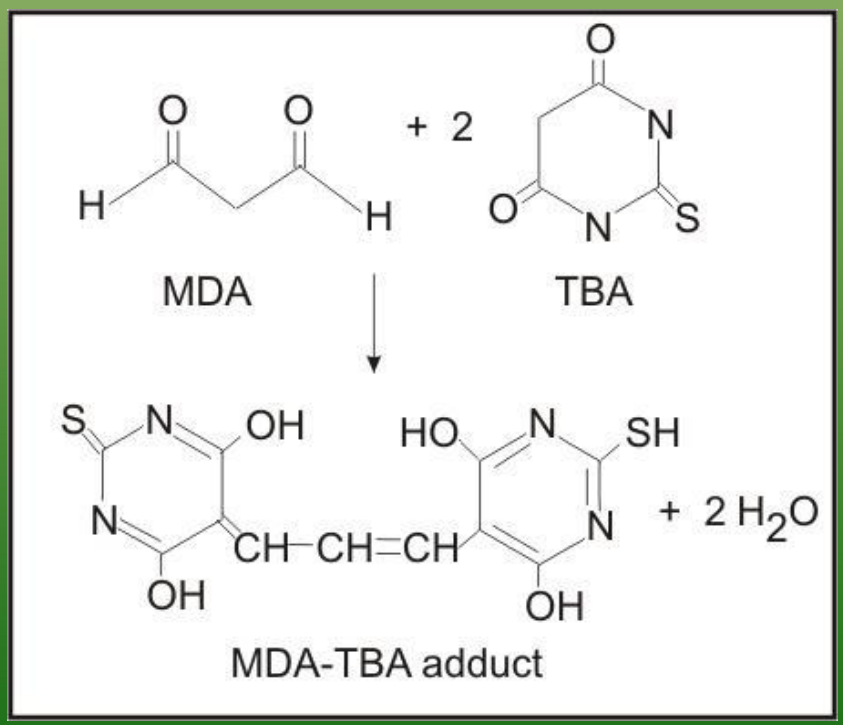
Fixation de l'Acide ThioBarbiturique



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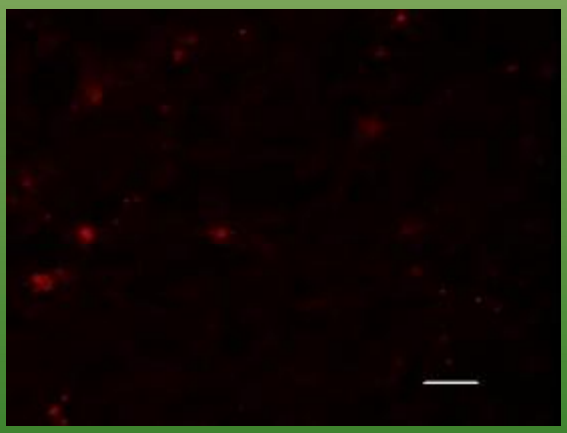
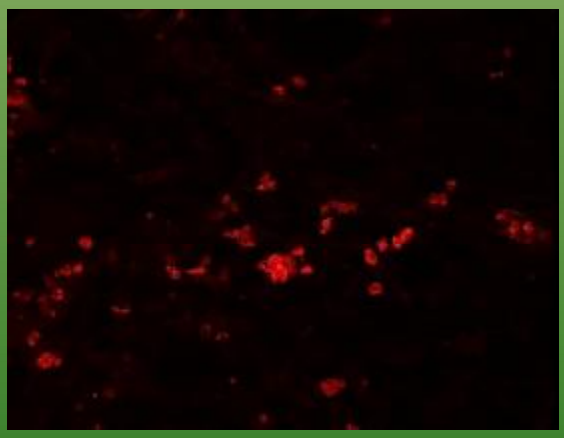
Fluorescence au DiHydroEthidium (DHE)



Cardiomyocytes Témoins



Cardiomyocytes après Infarctus simulé

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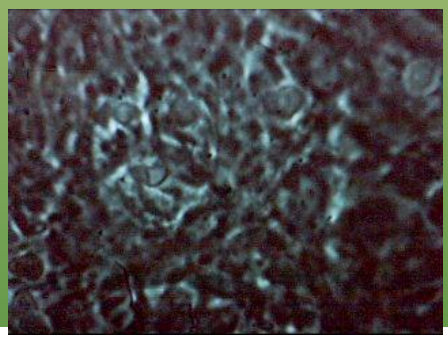
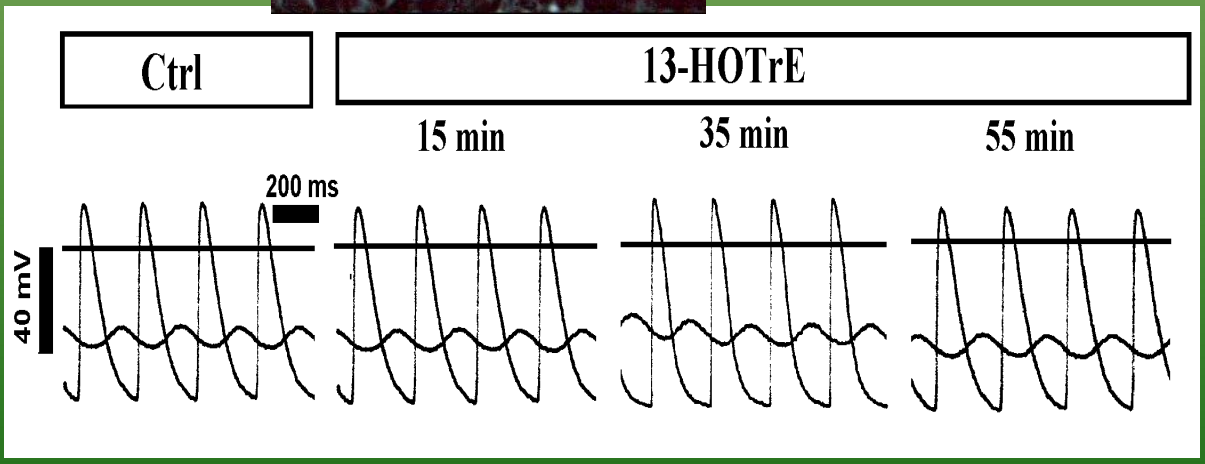
Addition d'acides gras peroxydé





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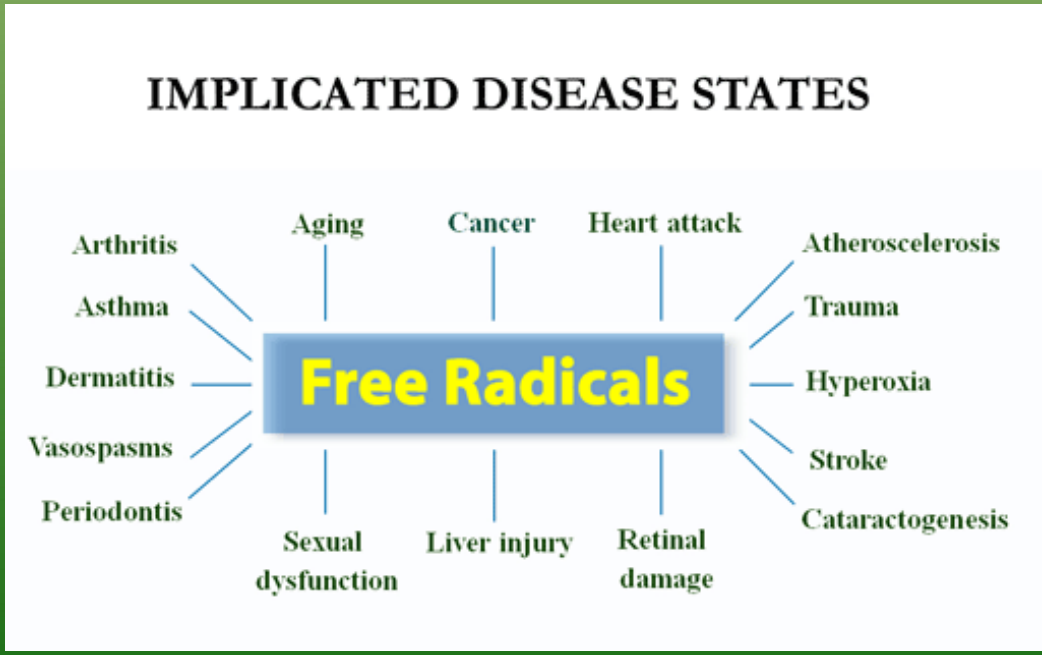
Radicaux Libres & Maladies



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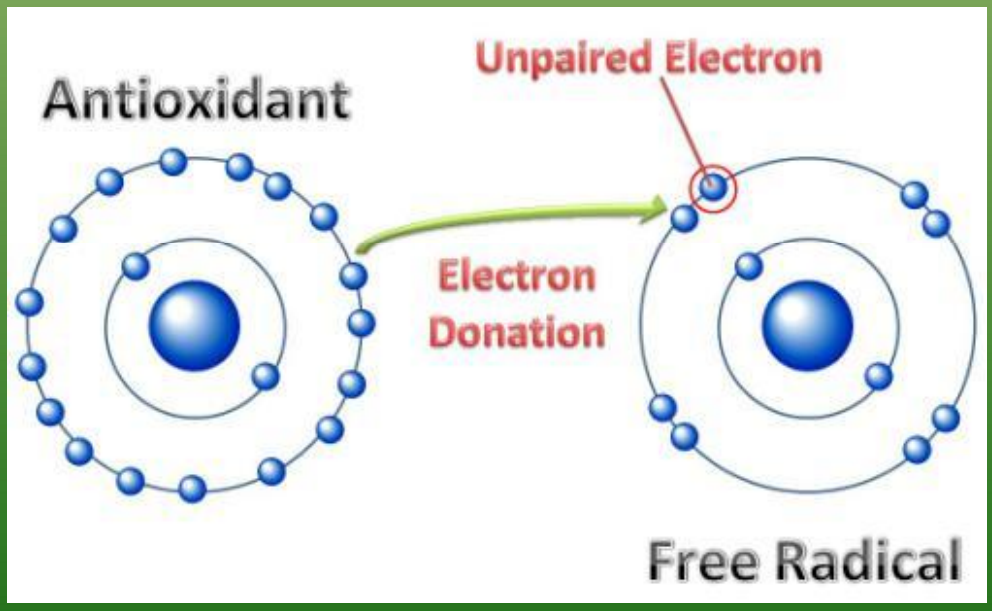
Traitement THEORIQUE



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Etude SUVIMAX

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SU.VI.MAX 1 : Principaux résultats

- Pas d'effet de la supplémentation en antioxydants sur l'incidence des maladies cardiovasculaires ischémiques.
- Effet de la supplémentation en antioxydants sur l'incidence des cancers, chez les hommes uniquement.
- Effet de la supplémentation en antioxydants sur la mortalité, chez les hommes uniquement.

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Essais Cliniques

Study	Subjects: No.	Sex	Age, y	Characteristics	Treatment: Dose	Duration, y	Prevention Goal	Study Outcome	RR/Statistics
Vitamin E									
GISSI (1999)[4]	11 324	M, F	No age limits	Post-MI adults	300 mg (synthetic)	3.5	Secondary	No effect on MI + CVD death + stroke	0.98 (0.87–1.10)
HOPE (2000)[5]	9541	M, F	≥55	High CVD risk	400 IU (natural)	4.5	Primary and secondary	No effect on MI + CVD death + stroke	1.05 (0.95–1.16)
PPP (2001)[6]	4495	M, F	64	At risk of CVD	300 mg (synthetic)	3.6	Primary	No effect on MI + CVD death + stroke	1.07 (0.74–1.56)
MICRO-HOPE (2002)[7]	3654	M, F	65	Diabetes	400 IU (natural)	4.5	Secondary	No effect on MI + CVD death + stroke	1.03 (0.88–1.21)
VEAPS (2002)[8]	353	M, F	≥40	Elevated LDL-C	400 IU dl-α-tocopherol	3	Primary	No effect on intima-media thickness + clinical events	*P*=0.81 for CVD events (14 placebo and 11 vitamin E)
β-Carotene									
ATBC (1998)[9]	27 271	M	50–69	Smokers with no history of MI	20 mg	6.1	Primary	No effect on:	
								All coronary cases	1.03 (0.91–1.16)
								Nonfatal MI	1.06 (0.90–1.24)
								Fatal CHD	0.99 (0.83–1.19)
SCPS (1996)[10]	1805	M, F	<85	Skin cancer patients	50 mg	8.2	Primary	No effect on CVD mortality	1.16 (0.82–1.64)
PHS (1996)[11]	22 071	M	40–84	Healthy	50 mg on alternate days	12	Primary	No effect on:	
								MI	0.96 (0.84–1.09)
								CVD	1.00 (0.91–1.09)
								CVD mortality	1.09 (0.93–1.27)
Antioxidant cocktails									
ATBC (1998)[9]	27 271	M	50–69	Smokers with no history of MI	50 mg vitamin E and 20 mg β-carotene	6.1	Primary	No effect on:	
								All coronary cases	0.97 (0.86–1.09)
								Nonfatal MI	0.99 (0.84–1.16)
								Fatal MI	0.94 (0.79–1.13)
HPS (2002)[12]	20 536	M, F	40–80	High CVD risk	600 mg vitamin E, 250 mg vitamin C, 20 mg β-carotene		Secondary	No effect on CVD mortality	1.05 (0.95–1.15)

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Méta-analyse COCHRANE (USA)

Antioxidant supplements for prevention of mortality in healthy participants and patients with various diseases

Bjelakovic G, Nikolova D, Gluud LL, Simonetti RG, Gluud C

Published Online: March 14, 2012

Previous research on animal and physiological models suggests that antioxidant supplements have beneficial effects that may prolong life. Some observational studies also suggest that antioxidant supplements may prolong life, whereas other observational studies demonstrate neutral or harmful effects. Our Cochrane review from 2008 demonstrated that antioxidant supplements seem to increase mortality. This review is now updated.

The present systematic review included 78 randomised clinical trials. In total, 296,707 participants were randomised to antioxidant supplements (beta-carotene, vitamin A, vitamin C, vitamin E, and selenium) versus placebo or no intervention. Twenty-six trials included [illegible] healthy participants. Fifty-two trials included [illegible] participants with various diseases in a stable phase (including gastrointestinal, cardiovascular, neurological, ocular, dermatological, rheumatoid, renal, endocrinological, or unspecified diseases). A total of 21,484 of 183,749 participants (11.7%) randomised to antioxidant supplements and 11,479 of 112,958 participants (10.2%) randomised to placebo or no intervention died. The trials appeared to have enough statistical similarity that they could be combined. When all of the trials were combined, antioxidants may or may not have increased mortality depending on which statistical combination method was employed; the analysis that is typically used when similarity is present demonstrated that antioxidant use did slightly increase mortality (that is, the patients consuming the antioxidants were 1.03 times as likely to die as were the controls). When analyses were done to identify factors that were associated with this finding, the two factors identified were better methodology to prevent bias from being a factor in the trial (trials with 'low risk of bias') and the use of vitamin A. In fact, when the trials with low risks of bias were considered separately, the increased mortality was even more pronounced (1.04 times as likely to die as were the controls). The potential damage from vitamin A disappeared when only the low risks of bias trials were considered. The increased risk of mortality was associated with beta-carotene and possibly vitamin E, but was not associated with the use of vitamin C or selenium.

The current evidence does not support the use of antioxidant supplements in the general population or in patients with various diseases.

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FONDATION ARC POUR LA RECHERCHE SUR LE CANCER
Reconnue d'utilité publique

« … Alors ?
Les antioxydants sont-ils nos amis ou nos ennemis lorsqu'il s'agit de lutter contre le cancer ?
Les données scientifiques actuelles montrent à quel point ils sont double-face :
alliés de choix lorsqu'ils sont apportés par une alimentation variée et équilibrée, ils peuvent se retourner contre nous si l'on en vient à les consommer sous la forme de gélules…»

21-10-2009

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www.etude-nutrinet-sante.fr/

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