



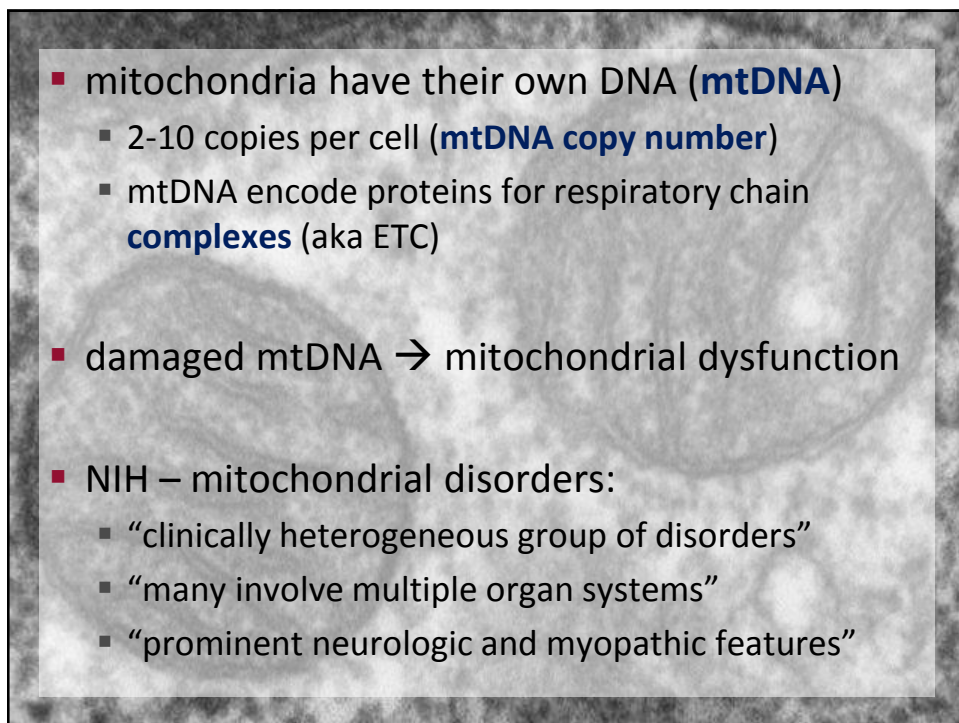
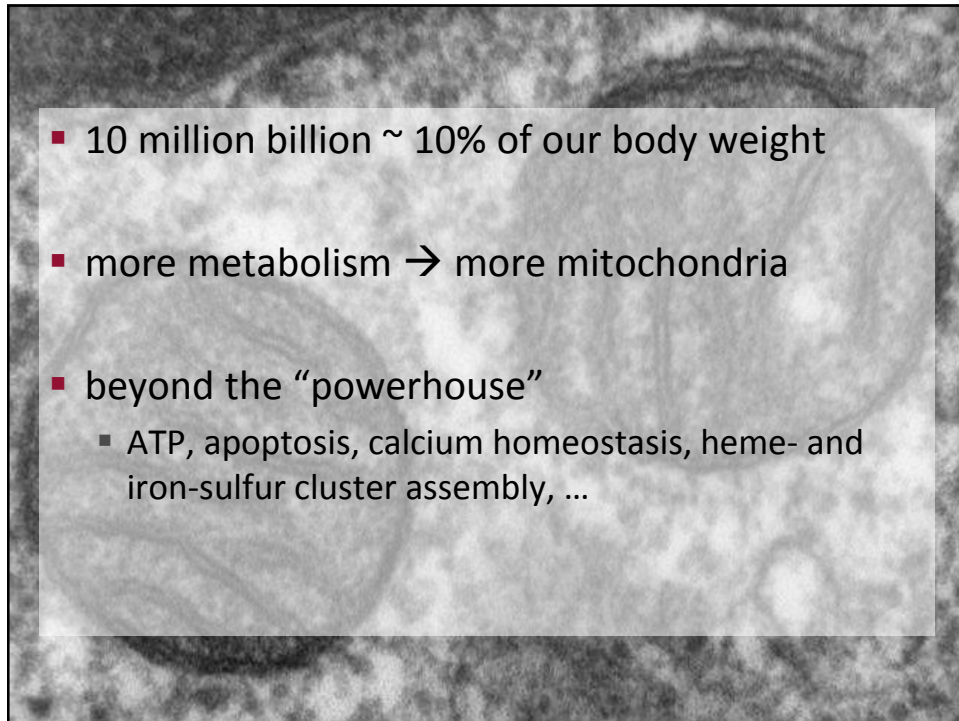
# Mitochondrial Function in Gulf War Illness

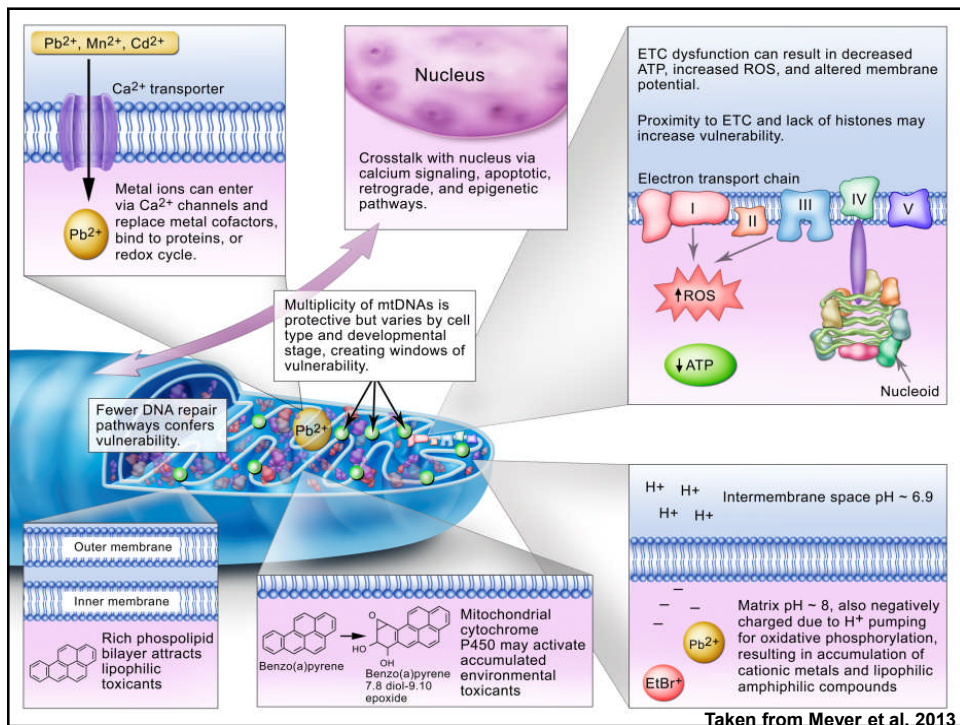
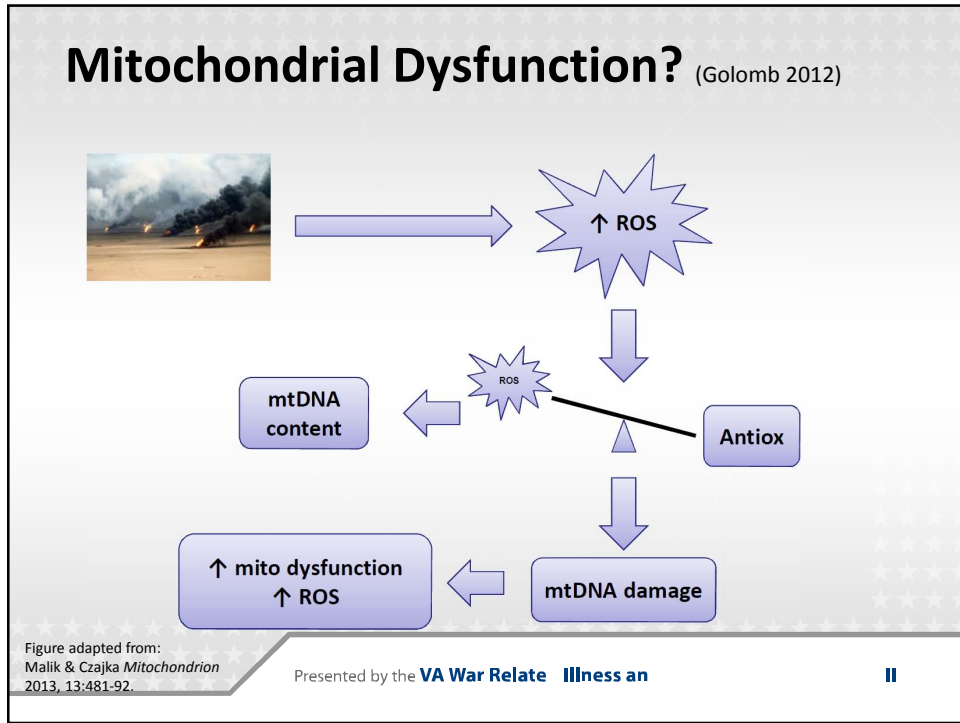
**Michael J. Falvo, PhD**  
*Research Physiologist, WRIISC – VA NJ Health Care System  
Assistant Professor, Rutgers Biomedical and Health Sciences*

Presented by the **VA War Related Illness and Injury Study Center (WRIISC)**

- *The views expressed in this presentation are my own and do not necessarily reflect the position or policy of the Department of Veterans Affairs or the United States government.*

Presented by the **VA War Related Illness and Injury Study Center (WRIISC)**

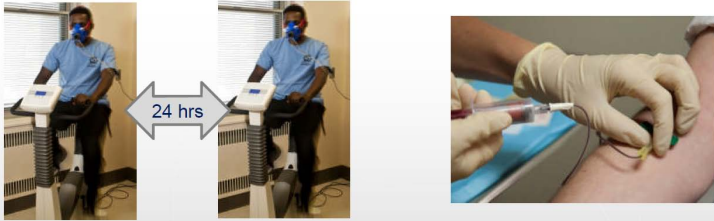




### Research Approach

30 GVs with GWI (Kansas-Steele)

22 controls (no GWI)



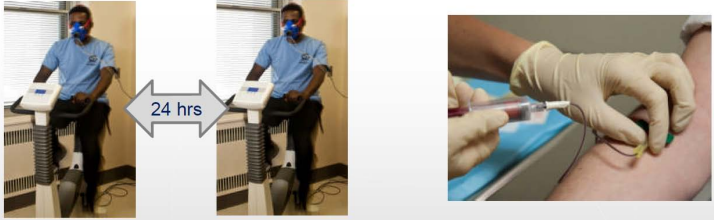
24 hrs

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### Research Approach

25 GVs with GWI (Kansas-Steele)

6 controls (no GWI)



24 hrs

ar Relate Illness an

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• mtDNA copy number (content)  
• mtDNA lesions (damage)  
• nDNA lesions (damage)

Extract DNA  
QPCR assay

Platelets + Plasma  
PBMCs  
Ficoll  
RBCs

*Meyer JN, Ecotoxico. 2010; Santos JH, Kovalenko OA, Mito. Genetics, 2009*

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### QPCR Assay

No damage

DNA lesions

Lesions inhibit the amplification of long amplicon

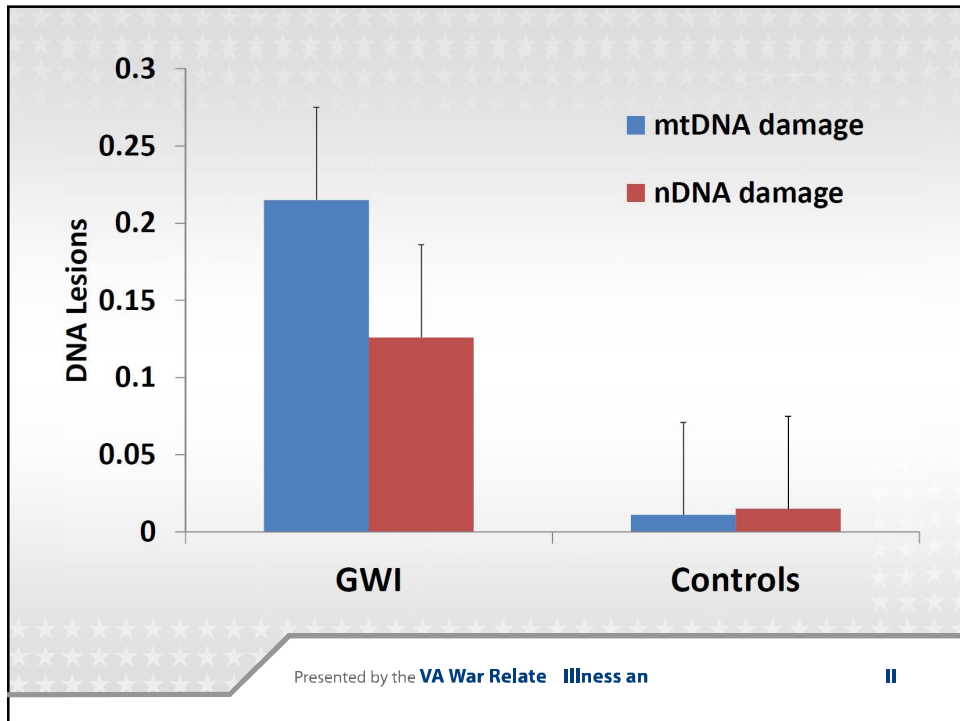
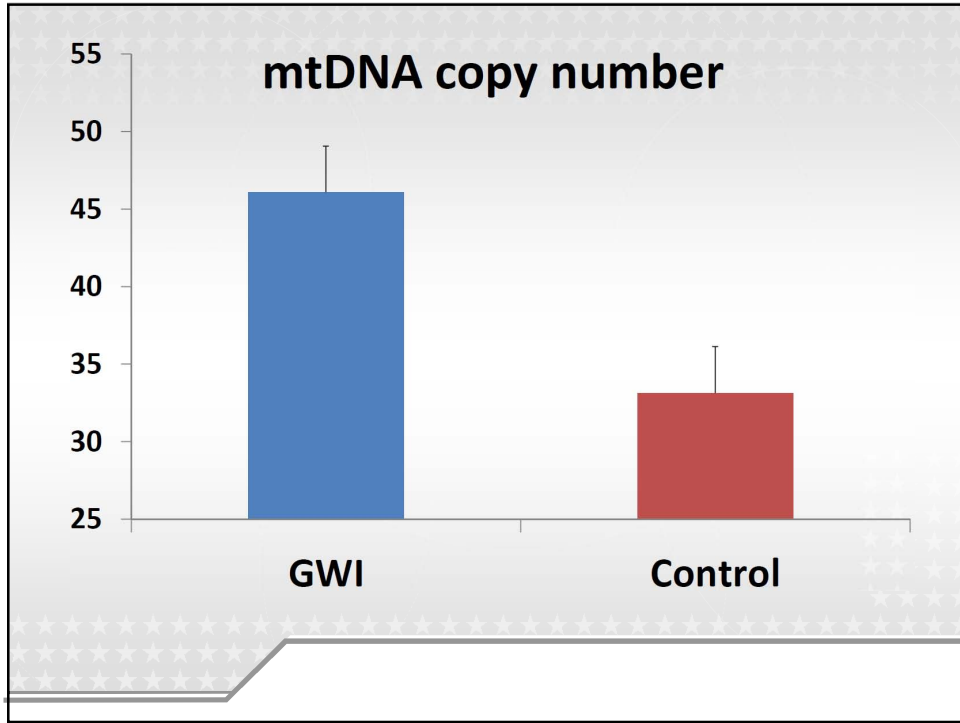
Short amplicon is not inhibited except by very high levels of damage

The ratio between long and short amplicon indicates relative mtDNA damage

— Long amplicon of mitochondrial genome (9-15kp)  
■ short amplicon of mitochondrial genome (200bp)

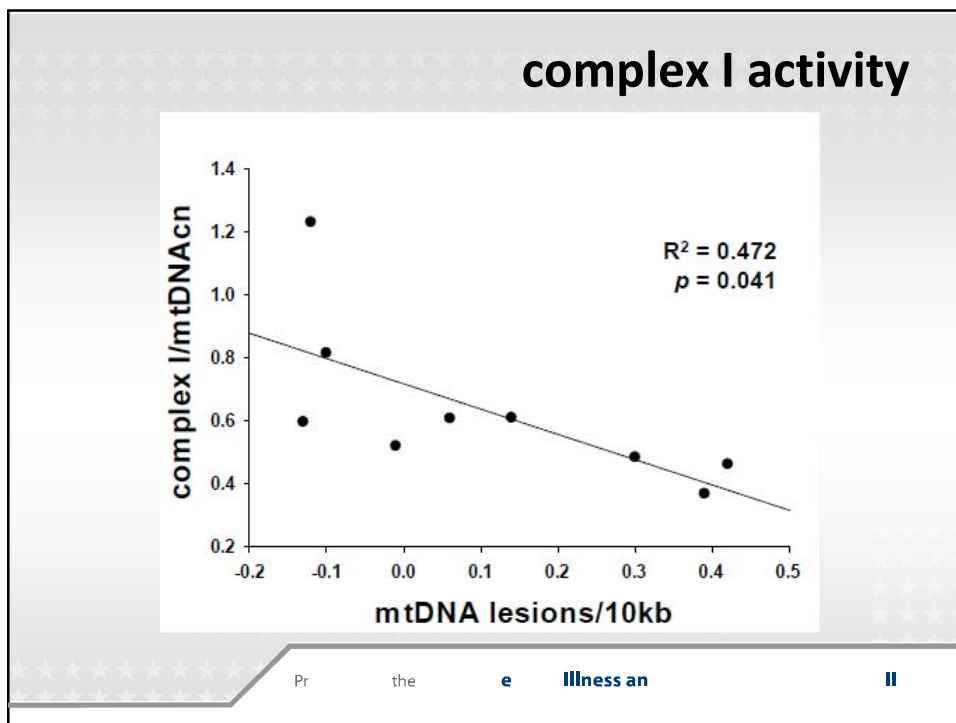
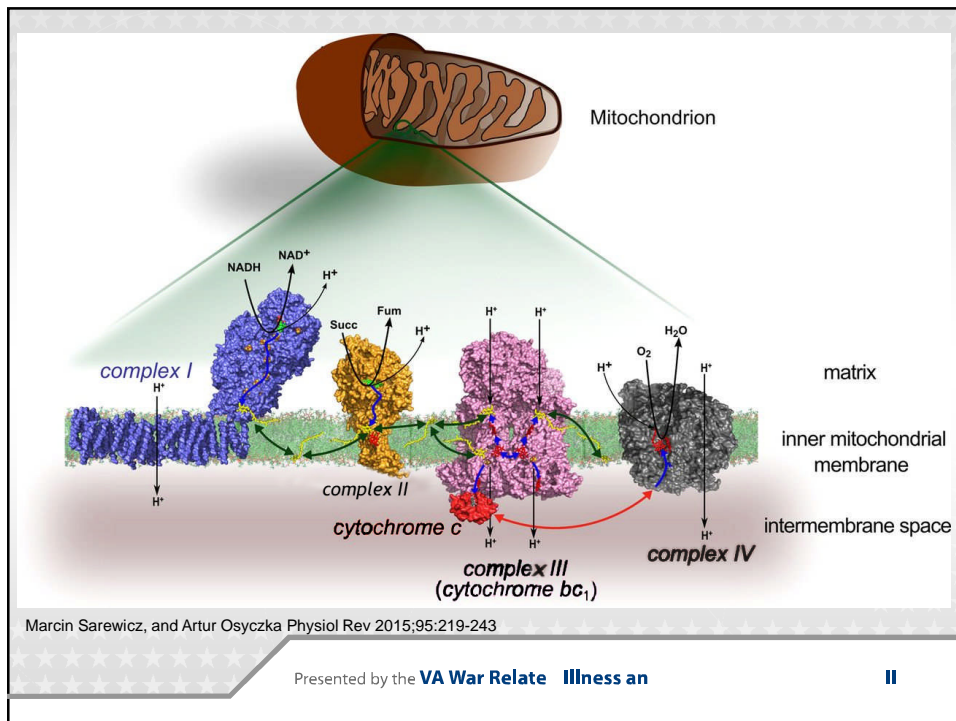
Meyer JN, Ecotoxicology (2010); Santos JH, Kovalenko OA, Mitochondrial Genetics, 2009

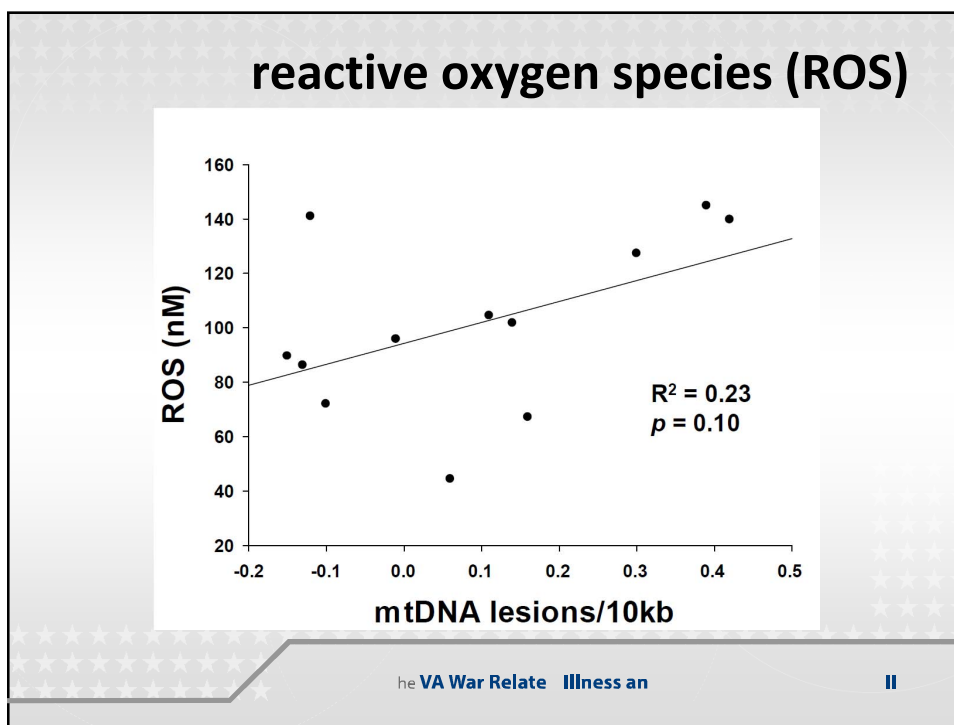
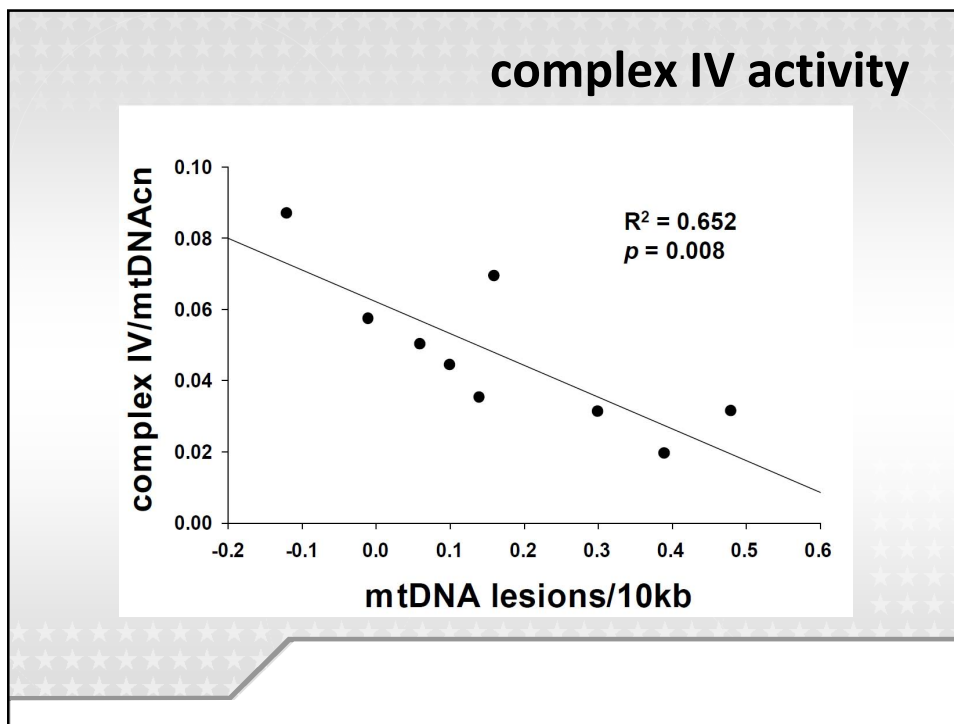
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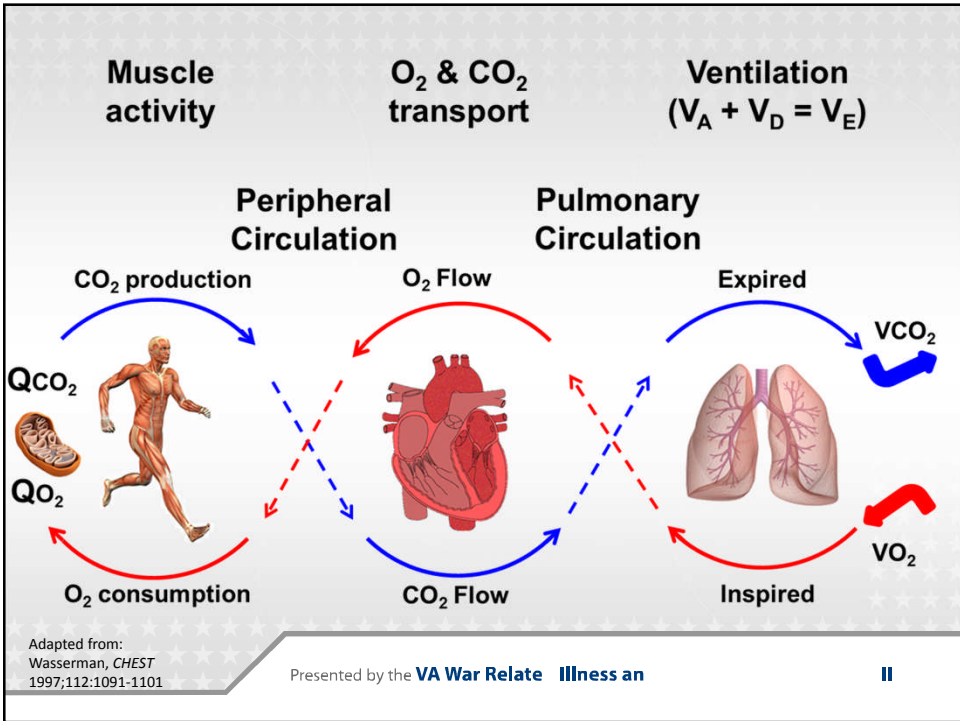
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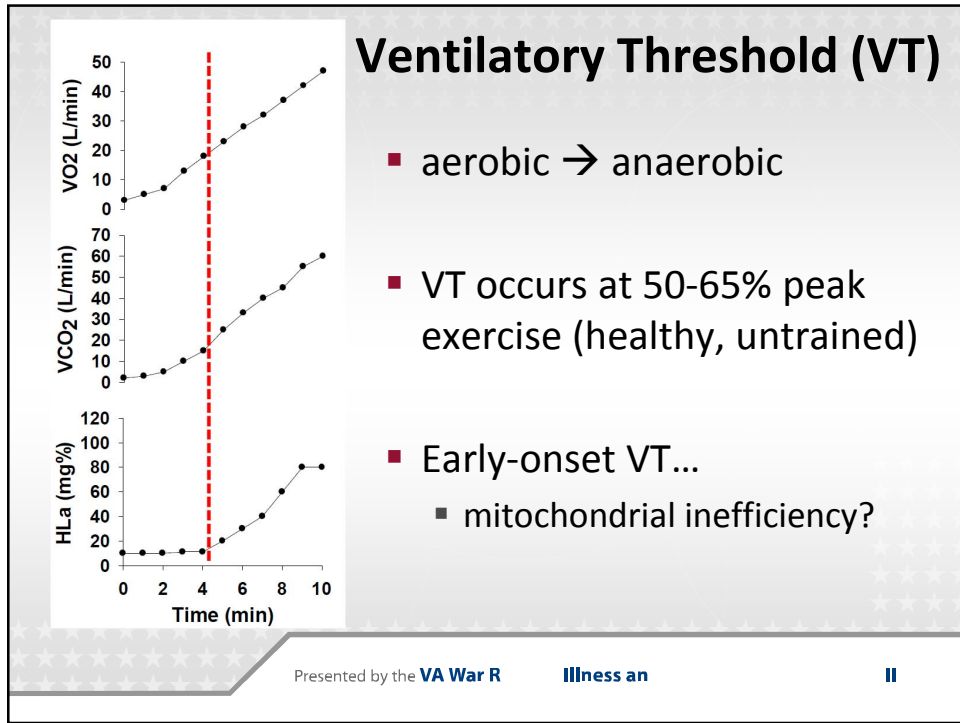
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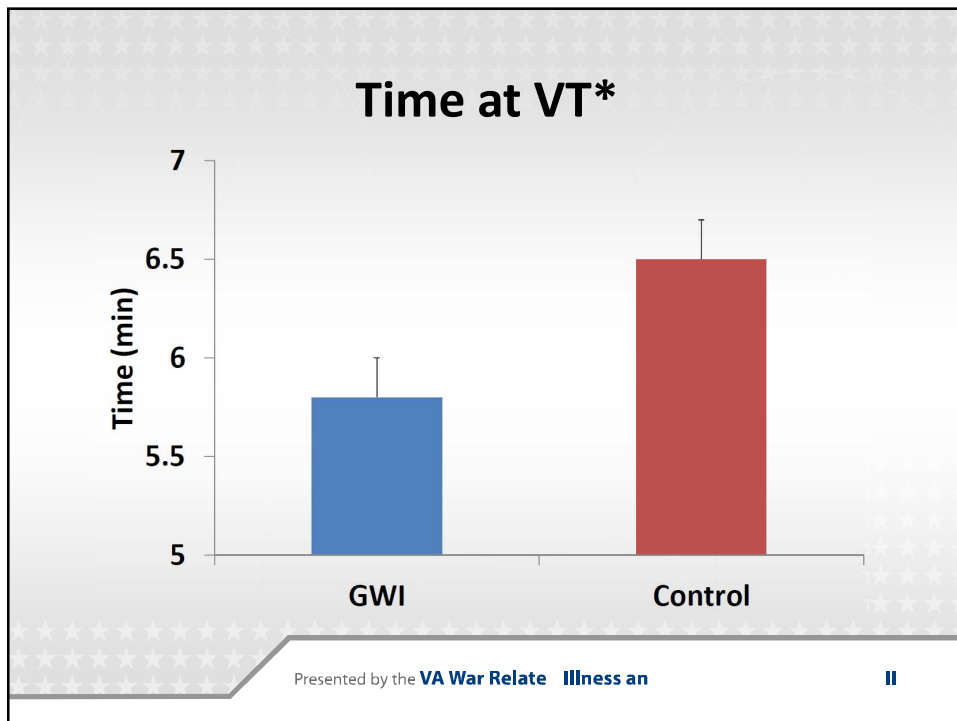
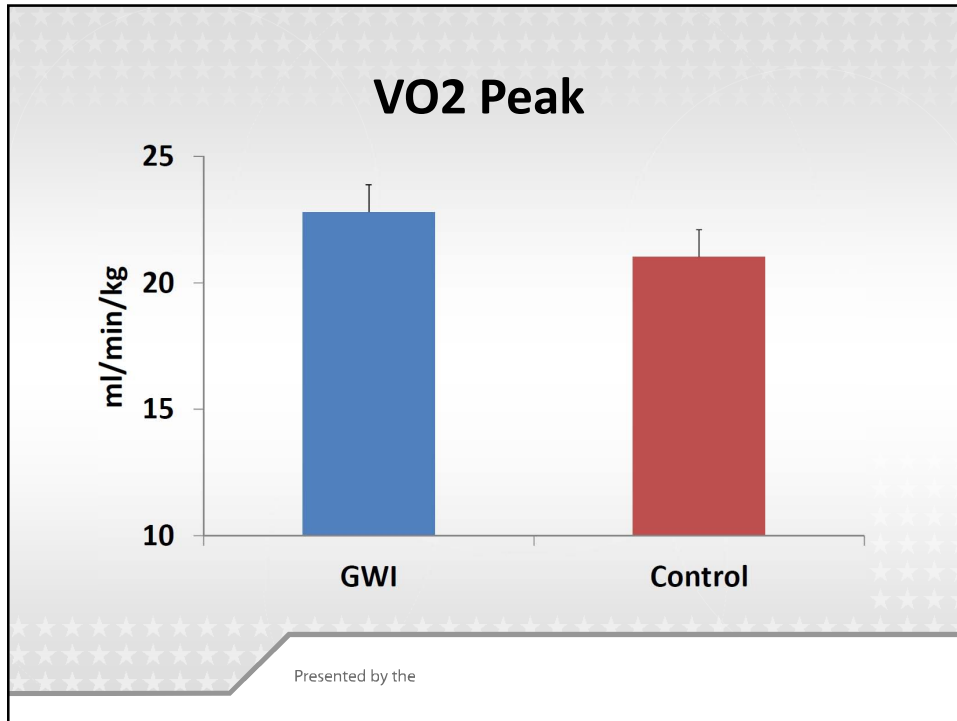


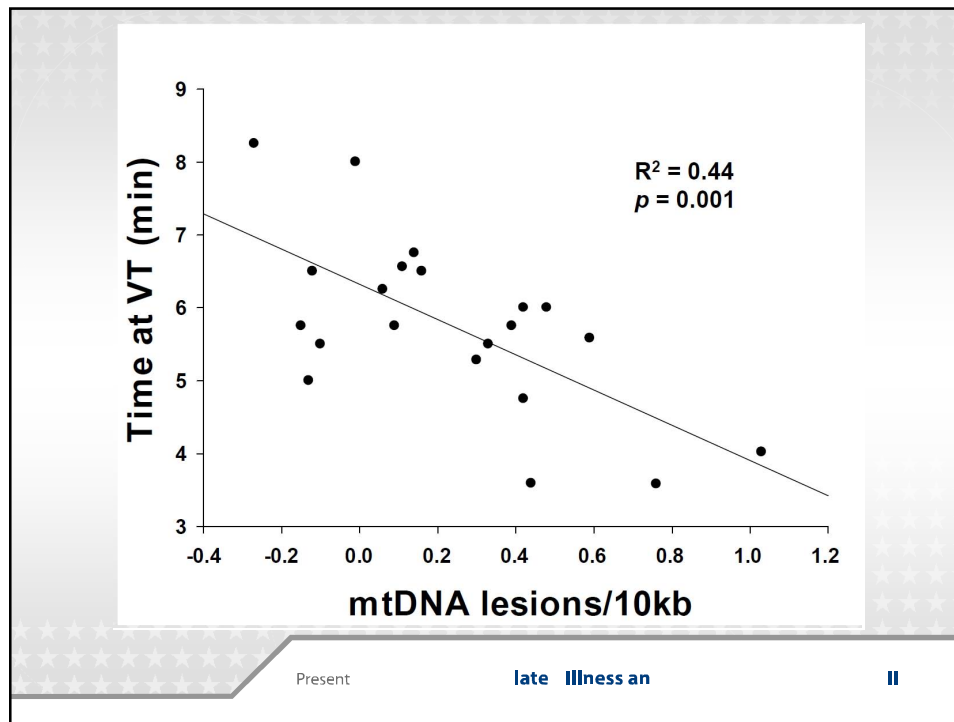
## Demographics

	<b>GWJ (n=24)</b>	<b>Control (n=22)</b>
<b>Age (yrs)</b>	49.9 ± 6.5	52.1 ± 5.7
<b>BMI (kg/m<sup>2</sup>)</b>	29.4 ± 4.4	31.3 ± 5.2
<b>Exercise (MET·min·wk<sup>-1</sup>)</b>	1235.72 ± 1336.5	1988.02 ± 1788.8
<b>Smoking Pack Years</b>	9.3 ± 13.1	4.9 ± 8.3
<b>Fatigue Severity*</b>	44.5 ± 15.9	28.3 ± 16.4
<b>Total Deployment Length (mo)*</b>	12.6 ± 9.7	5.3 ± 4.4

\*p<0.05

Pre





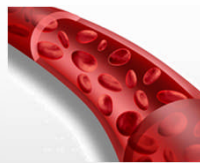
## Preliminary Findings

- $\uparrow$ mtDNAcn,  $\uparrow$ mtDNA,  $\uparrow$ nDNA damage in GWI
  - mtDNA damage > nDNA damage
- mtDNA lesions associated with complex I and IV activity
- mtDNA lesions associated with VT onset time
- Next steps  $\rightarrow$  complete/finalize data analysis

## Future Directions



- mtDNA repair pathways
- bioenergetic mechanisms  
(Joel Meyer, PhD – Duke)

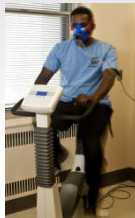


- mito → heme synthesis
- RBC function  
(Mike Condon, PhD – VANJHCS)

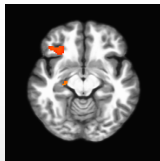
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## Future Directions



- *“Post exertion malaise in GWI: Brain, autonomic and behavioral interactions”*
  - VA CSRD 1I01CX001329-01



- Co-PI – Dane Cook (Madison, WI)
- Co-I – Glenn Wylie (VANJ, Kessler)
- Co-I – Jorge Serrador (VANJ)

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## Acknowledgements



- Support:
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- Collaborators:
  - Joel Meyer, PhD
  - Andrew Thomas, PhD
  - Helene Hill, PhD



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