

Oxidative Stress, Inflammation, and Obesity: Insights into Mechanisms and Therapeutic Targets

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INTRODUCTION & AIM

Introduction

- ✓ A major contributing element to the pathophysiology of many diseases, obesity is defined as an increase in body weight that leads to excessive fat buildup.
- ✓ It is a global social concern. Unfortunately, a growing percentage of youngsters in wealthy nations are also affected by obesity.
- ✓ Furthermore, it has been noted that obese children and adolescents are more likely to be fat as adults, increasing their chance of developing health issues as adults.
- ✓ Obesity is also linked to a number of chronic conditions, including cancer, diabetes mellitus, metabolic syndrome, liver and cardiovascular disorders, and others.
- ✓ Although the complications of obesity might differ from person to person, the consequences are similar for all subjects.
- ✓ This is true for the presence of oxidative stress and inflammation in all obese patients. One of the common overlooked complications of obesity is cognitive impairments.

Aim

- ✓ To investigate the connection between disorders linked to obesity, inflammation, and oxidative stress (OS).
- ✓ To examine how OS overexpression damages cells and impairs antioxidant defenses.
- ✓ To evaluate information on obesity and OS from clinical studies, biochemical analysis, and animal models.
- ✓ To assess how adipokines, like adiponectin, help reduce inflammation and OS.
- ✓ To investigate possible treatment approaches for lowering OS and enhancing antioxidant equilibrium.
- ✓ To draw attention to research showing a connection between insulin resistance, metabolic dysfunction, and lipid peroxidation in KKAY mouse models.

METHOD

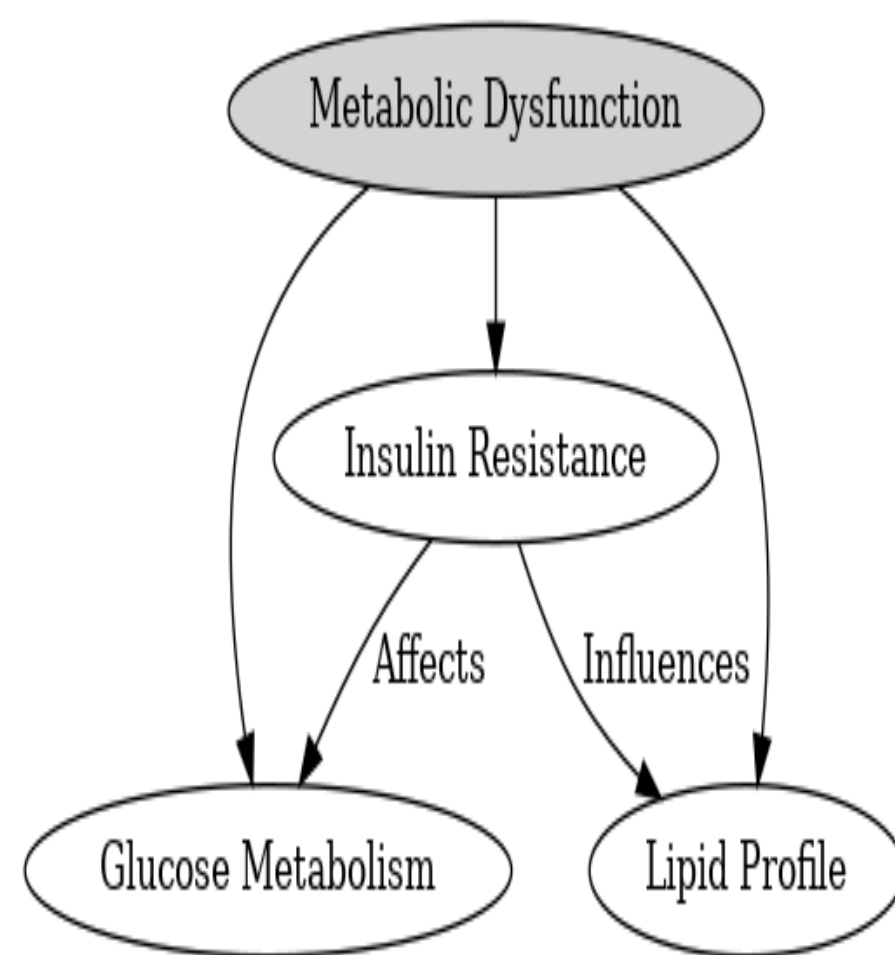
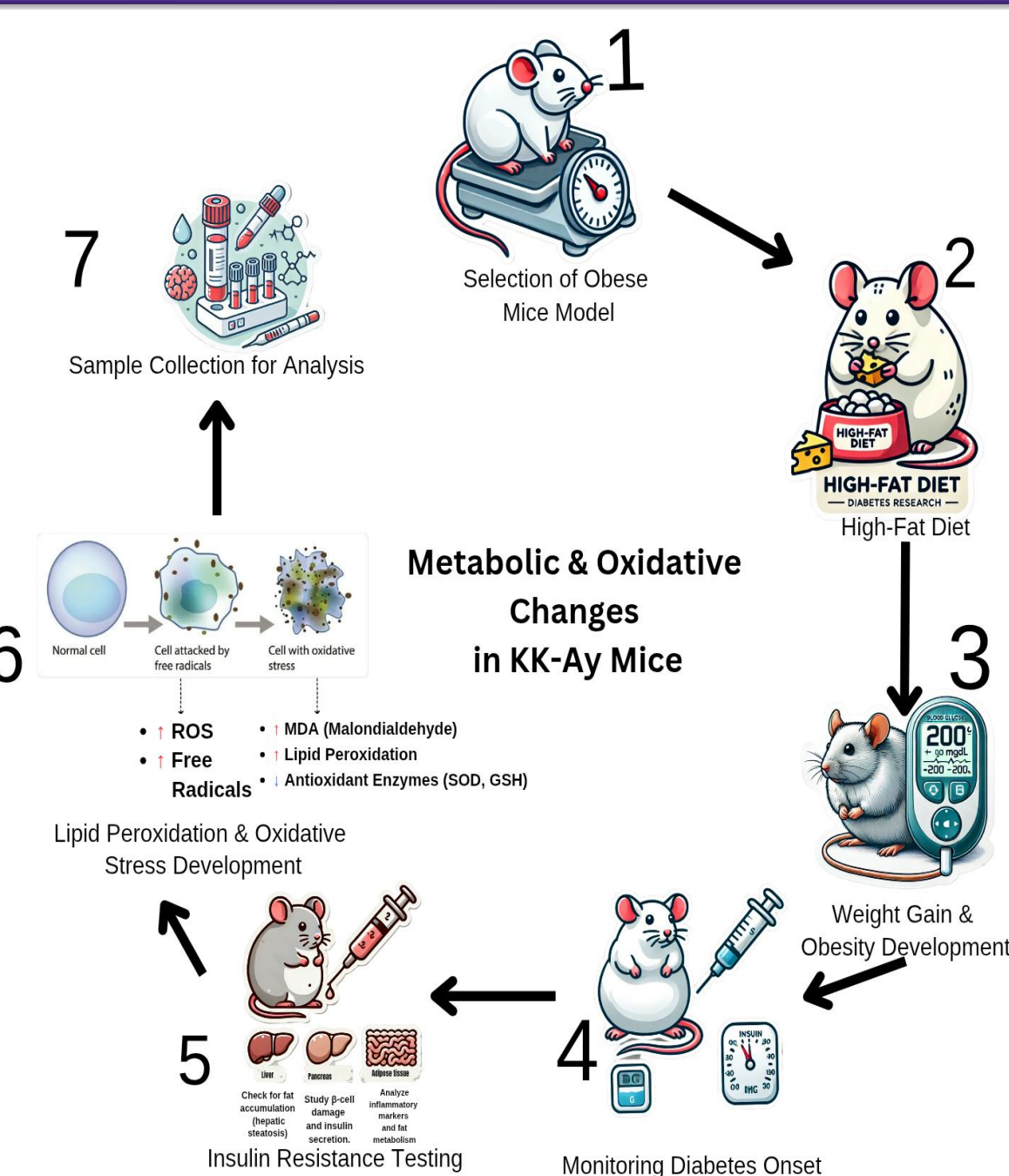


Fig.2:Metabolic Dysfunction: Glucose metabolism, lipid profile, insulin resistance.

Fig.1 : Model Used: KKAY mice (diabetic obesity model).

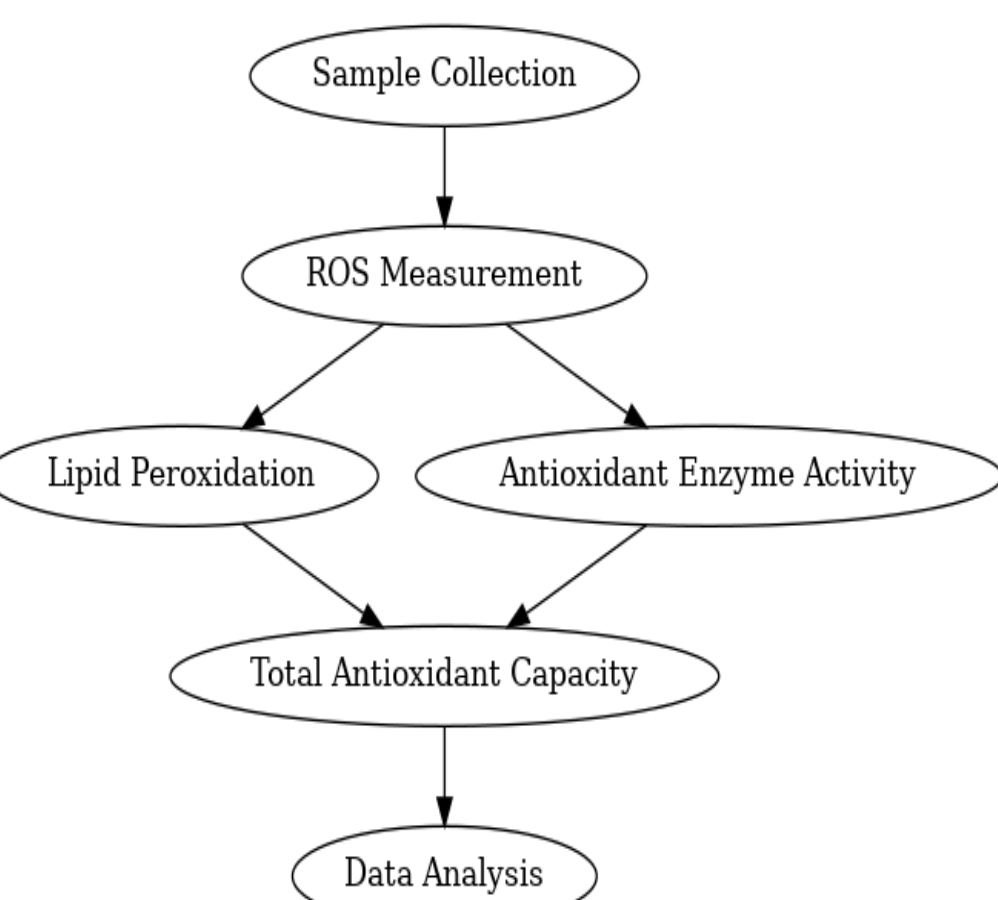


Fig.3: Oxidative Stress Analysis: ROS, lipid peroxidation, antioxidant enzymes

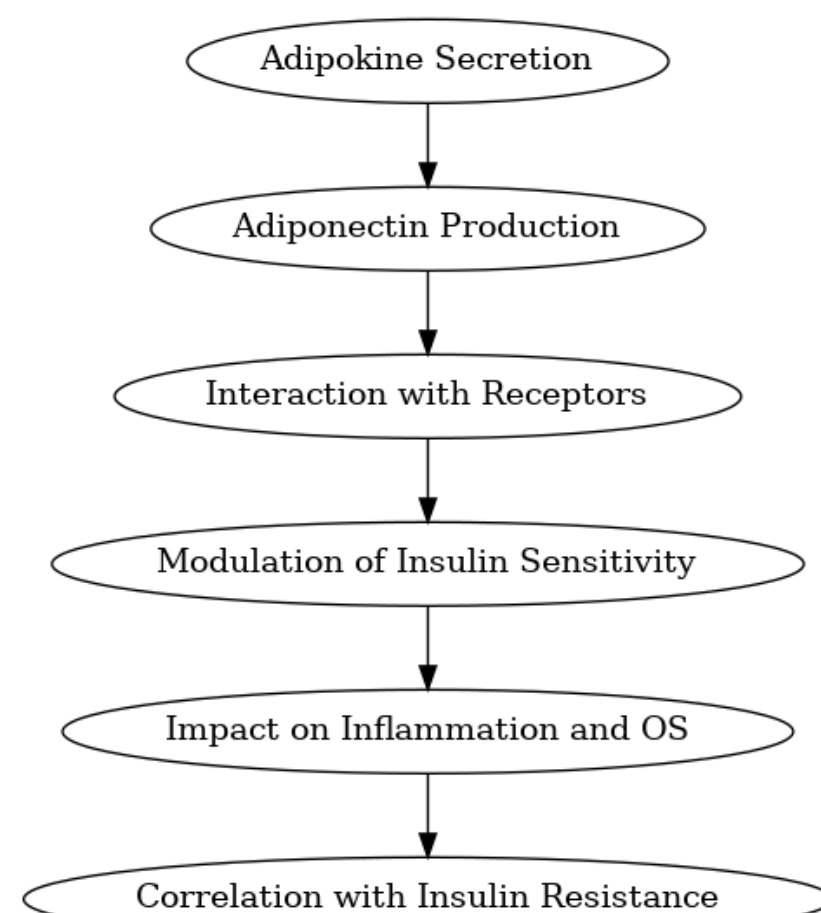


Fig.4 : Adipokine Role: Adiponectin levels, insulin resistance correlation.

RESULTS & DISCUSSION

1.Oxidative Stress & Inflammation

- ✓ Oxidative stress is caused by a substantial rise in lipid peroxidation and reactive oxygen species (ROS) in obese rodents.
- ✓ Chronic low-grade inflammation is exacerbated by high pro-inflammatory markers such TNF- α , IL-6, and CRP.
- ✓ The interaction of inflammation and oxidative stress intensifies metabolic dysfunction and cellular damage.

2.Adipose Tissue Dysfunction

- ✓ Increased oxidative damage in adipose tissue is seen in the KKAY mice model, which represents diabetes obesity.
- ✓ Normal adipose tissue function is hampered by increased oxidative stress caused by a reduction in antioxidant defense systems. More pro-inflammatory adipokines are secreted by dysfunctional adipose tissue, exacerbating metabolic issues.

3.Metabolic Impairments

- ✓ In obese models, insulin resistance and high fasting glucose levels indicate a major disruption in glucose metabolism.
- ✓ There are anomalies in the lipid profile, such as elevated LDL cholesterol and triglycerides.
- ✓ The development of metabolic syndrome is facilitated by the interaction of insulin resistance and disruption of lipid metabolism.

4. Protective Role of Adipokines

- ✓ Adiponectin is essential for lowering inflammation and oxidative damage while increasing insulin sensitivity.
- ✓ Increased metabolic dysfunction is correlated with decreased adiponectin levels in obesity.
- ✓ Increasing adiponectin activity may be a useful treatment strategy.

5.Clinical Implications

- ✓ Reducing inflammation and oxidative stress may aid in the treatment of metabolic diseases linked to obesity.
- ✓ Better clinical results could be obtained by employing strategies that enhance antioxidant balance and modulate adipokines.
- ✓ New therapeutic approaches can be developed with the help of an understanding of the molecular mechanisms behind these dysfunctions.

CONCLUSION

As shown in KKAY mice, metabolic dysfunction is exacerbated by inflammation and oxidative stress associated with obesity. Cellular damage and insulin resistance are caused by increased ROS generation, lipid peroxidation, and compromised antioxidant defenses. Potential therapeutic targets, adipokines, like adiponectin, have protective functions by lowering inflammation and oxidative stress. Gaining an understanding of these systems is essential for creating plans to control inflammatory pathways, restore antioxidant balance, and enhance clinical outcomes in disorders linked to obesity.

FUTURE WORK / REFERENCES

Future Work

The potential of adipokine-based treatments to reduce oxidative stress and metabolic dysfunction requires more investigation. Examining antioxidant-based therapies may provide fresh approaches to treating issues related to obesity. To validate possible therapy targets and evaluate their effectiveness, long-term clinical investigations are required.

References

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