

## An Interdisciplinary Approach for Understanding, Testing and Treating Medical Conditions using Human Disease Models

Vivician Thomas\*

Department of Public Health, University for Health Sciences, Medical Informatics and Technology, Hall In Tirol, Austria

\*Corresponding author: Vivician Thomas, Department of Public Health, University for Health Sciences, Medical Informatics and Technology, Hall In Tirol, Austria, E-mail: vivithom@UHSMIT.at

Received: 27-Aug-2024, Manuscript No. JCEP-24-150868; Editor assigned: 30-Aug-2024, PreQc No. JCEP-24-150868 (PQ); Reviewed: 13-Sep-2024, QC No. JCEP-24-150868; Revised: 20-Sep-2024, Manuscript No. JCEP-24-150868 (R); Published: 27-Sep-2024, DOI: 10.4172/2161-0681.24.14.521

Citation: Thomas V (2024) An Interdisciplinary Approach for Understanding, Testing and Treating Medical Conditions using Human Disease Models. J Clin Exp Pathol. 14:521.

Copyright: © 2024 Thomas V. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

### Description

Disease models serve as essential tools in biomedical research, allowing scientists to explore disease causes, test therapeutic approaches, and create new treatments in a controlled setting. These models simulate specific elements of human diseases and are used to investigate pathophysiological processes that are difficult to examine in humans. They are essential to accepting complex biological interactions, detecting illness signs, and determining therapeutic efficacy and toxicity. Disease models help to bridge the gap between basic research and clinical application by allowing for controlled experiments. Diseases models have advanced modern medicine and will continue to evolve as new technologies emerge, including genetic engineering, stem cell technology, and computational modeling [1]. Their utility stems from their capacity to imitate crucial aspects of human diseases, either in person (in a living creature) or in laboratory (in a laboratory environment such as cell cultures). Disease models can be broadly divided into four types: Animal models, cellular models, organoid models, and computational models. Each type offers advantages and disadvantages, depending on the severity of the condition and the research issue [2,3].

### Animal models

Animal models have long been the accepted standard in disease study. They are essential for evaluating diseases in the context of a whole living organism, which comprises interconnected systems such as the immune, neurological, and endocrine. Because of their genetic and physiological similarities to humans, non-human primates are utilized in disease studies, including HIV/AIDS, Parkinson's disease, and cardiovascular disease. They are frequently used when animal models fail to reproduce complicated human disorders, especially in neurology and immunology [4-6]. However, their use is restricted due to ethical concerns, high expense, and extended life spans, which make longitudinal studies challenging.

### Cellular models

Cellular models use cells obtained from species to investigate disease mechanisms at the cellular level. These models provide an easier more regulated environment than complete organisms. These are cells extracted straight from living tissues, allowing researchers to examine disease in its natural state. For example, human cancer cells can be cultivated to study tumor growth, metastasis, and treatment responses. These are cells that have been genetically modified to

reproduce continuously. They are often utilized in *in vitro* investigations because they offer a reliable and reproducible model system. Immortalized cell lines, on the other hand, may not fully simulate normal physiology or disease situations since the immortalization process frequently alters cell properties [7].

### Organoid models

Organoids are three-dimensional constructs made from stem cells that resemble the architecture and function of living organs. These models are becoming used for researching organ-specific disorders and developmental biology. These mini-brain models are used to investigate neurological illnesses like as autism, schizophrenia, and Alzheimer's disease, providing insights into human brain development and pathology that animal models cannot [8]. These models are used in studies of gastrointestinal illnesses, liver damage, and metabolic disorders. Organoids might resemble complex tissue architecture and cellular variety, making them suitable for personalised treatment and drug testing.

### Computational models

Computational disease models employ mathematical algorithms and simulations to better understand disease processes and predict their results. These models are frequently used in systems biology to combine massive datasets from genomics, proteomics, and metabolomics. These models use computer algorithms to reproduce biological processes, allowing researchers to analyze disease progression in cancer, diabetes, and cardiovascular disease without conducting *in vitro* or *in vivo* trials [9]. They can predict medication treatment outcomes and contribute to more successful clinical trial design.

Disease models are essential throughout the drug discovery process, from target identification and validation to preclinical testing. Before a medicine is tested in humans, it must be evaluated in a variety of disease models to determine its efficacy and safety. Animal models, particularly mouse models, are frequently employed in preclinical studies to assess the therapeutic potential and possible negative effects of novel drugs. Models of cancer, cardiovascular disease, and infectious diseases, for example, are critical for evaluating treatment candidates before they enter human trials. Models enable researchers to investigate the underlying mechanisms of diseases, such as genetic, molecular, and cellular components [10]. They serve as essential for understanding disease development and progression, which can lead to the identification of new treatment targets.

One of the fundamental disadvantages of disease models, particularly animal models, is that they may not always accurately represent human situations. Despite genetic and physiological similarities between model species and humans, there are major distinctions that might cause inconsistencies in disease progression and treatment effectiveness.

## Conclusion

Disease models are necessary tools in biomedical research, providing knowledge about disease mechanisms, improving drug discovery, and developing personalized therapy. With these limitations, current technology advances such as genetic engineering, organoids, and computer models help to refine these models and improve their relevance to human health. The potential of disease modeling depends on the integration of multiple methodologies, utilizing each model's capabilities to overcome translational gaps and develop more effective treatments for a wide range of disorders.

## References

1. Turnbull IR, Gilfillan S, Cella M, Aoshi T, Miller M, et al. (2006) Cutting edge: TREM-2 attenuates macrophage activation. *J Immunol.* 177:3520-3524.
2. Koutsodendris N, Blumenfeld J, Agrawal A, Traglia M, Yip O, et al. (2023) APOE4-promoted gliosis and degeneration in tauopathy are ameliorated by pharmacological inhibition of HMGB1 release. *Cell Rep.* 42:113252.
3. Andreone BJ, Przybyla L, Llapashtica C, Rana A, Davis SS, et al. (2020) Alzheimer's-associated PLC $\gamma$ 2 is a signaling node required for both TREM2 function and the inflammatory response in human microglia. *Nat Neurosci.* 23:927-938.
4. Cheng-Hathaway PJ, Reed-Geaghan EG, Jay TR, Casali BT, Bemiller SM, et al. (2018) The Trem2 R47H variant confers loss-of-function-like phenotypes in Alzheimer's disease. *Mol Neurodegener.* 13:29.
5. Galatro TF, Holtman IR, Lerario AM, Vainchtein ID, Brouwer N, et al. (2017) Transcriptomic analysis of purified human cortical microglia reveals age-associated changes. *Nat Neurosci.* 20:1162-1171.
6. Miyamoto M, Kiyota Y, Yamazaki N, Nagaoka A, Matsuo T, et al. (1986) Age-related changes in learning and memory in the Senescence-Accelerated Mouse (SAM). *Physiol Behav.* 38:399-406.
7. Gil JM, Rego AC (2008) Mechanisms of neurodegeneration in Huntington's Disease. *Eur J Neurosci.* 27:2803-2820.
8. Maher P, Currais A, Schubert D (2020) Using the oxytosis/ferroptosis pathway to understand and treat age-associated neurodegenerative Diseases. *Cell Chem Biol.* 27:1456-1471.
9. Gonzalez-Juanatey C, Testa A, Garcia-Castelo A, Garcia-Porrúa C, Llorca J, et al. (2003) HLA-DRB1 status affects endothelial function in treated patients with rheumatoid arthritis. *Am J Med.* 114:647-652.
10. Juneja M, Singh S, Agarwal N, Bali S, Gupta S, et al. (2020) Automated detection of glaucoma using deep learning convolution network (g-net). *Multimed Tools Appl.* 79:15531-15553.