Mutations in the **SOD1 gene** are discovered as the first known cause of ALS/MND.

Mutations in the **TDP-43 gene** are identified as causing familial ALS. In addition, TDP-43 pathology is observed in ~97% of ALS/MND cases.

Mutations in the **FUS gene** identified as causing familial ALS/MND.

Intermediate repeats in the **ATXN2 gene** identified as increasing risk of developing ALS/MND.

Hexanucleotide repeats in the **C9orf72 gene** identified as causing both ALS/MND and frontotemporal dementia (FTD).

C9orf72 is the most common genetic cause of ALS, in both familial and sporadic patients, and its discovery cemented the concept of genetic pleiotropy playing a role in ALS/MND genetics.

Many ALS/MND genes identified. Importance, biological mechanisms and therapeutic relevance still being evaluated.

Multiple clinical trials targeting **SOD1 mutations** initiated.

Clinical trial targeting **C9orf72 repeat expansions** begins.

Clinical trial targeting **ATXN2 repeat expansions**, in both patients with the expansion and sporadic ALS/MND, begins.

Clinical trial targeting **FUS mutations** begins. Clinical trial for presymptomatic individuals carrying **SOD1 mutations** begins.