New research published in the *Journal of Physiology* presents a breakthrough in the treatment of Restless Legs Syndrome (RLS).

RLS is a common condition of the nervous system that causes an overwhelming irresistible urge to move the legs. Patients complain of unpleasant symptoms such as tingling, burning and painful cramping sensations in the leg. More than 80% of people with RLS experience their legs jerking or twitching uncontrollably, usually at night.

Until now it was thought that RLS is caused by genetic, metabolic and central nervous system mechanisms. For the first time the researchers show that, in fact, it is not only the central nervous system but also the nerve cells targeting the muscles themselves that are responsible.

This new research indicates that the involuntary leg movements in RLS are caused by increased excitability of the nerve cells that supply the muscles in the leg, which results in an increased number of signals being sent between nerve cells.

Targeting the way messages are sent between nerve cells to reduce the number of messages to normal levels may help prevent the symptoms of RLS occurring. This could be achieved by new drugs that block the ion channels that are essential for the communication between nerve cells.

The research conducted by the University of Gottingen in conjunction with the University of Sydney and Vanderbilt University involved measuring the nerve excitability of motor nerve cells of patients suffering with RLS and healthy subjects.

The next step is to investigate the effect of different medications in patients and the effect on RLS.

Dirk Czesnik, corresponding author of the study, commented on the findings:

'Patients who suffer from Restless legs syndrome complain of painful symptoms in the legs leading to sleep disturbances. The mechanisms for RLS are still not completely understood. We have shown that also the nerve cells supplying muscles in the leg are responsible and hereby additional drug treatments may be ahead targeting these nerve cells.'

doi/10.1113/JP275341

Provided by The Physiological Society