Common factors linked to the biology of neurodegenerative diseases

Authors: Melissa Madden William Smith Vanessa Reed Stephen Farrell Adam Soto PhD

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University of California-San Francisco

School of Economics

The UC Davis Neurogenetics Team carefully analyzed data from more than 20 patients with Huntingtonâ€™s disease, a fatal neurological disease, with altered functioning of proteins associated with cell mitochondria. The analyses showed that sub-classes of neurodegenerative diseases such as Huntingtonâ€™s may be divided into sub-groups due to dysfunction of mitochondrial proteins implicated in diseases such as Huntingtonâ€™s.

In the end, the team found four sub-groups of neurodegenerative diseases associated with altered mitochondrial metabolism and found that Huntingtonâ€™s disease, familial amyloid-beta disease, familial amyloid-associated neurodegeneration and alpha-synuclein-related neurodegeneration share common metabolic defects. This is published online Dec. 20, 2011 in Neurogenetics.

â€œThis finding is important since it indicates that patients with differing forms of Huntingtonâ€™s disease might have different levels of mitochondrial function, or alter metabolic pathways,â€ said the research team.

The finding and interpretation were based on the analysis of data from 22 patients with variations in three criteria for Huntingtonâ€™s disease, 11 patients with sporadic human Huntingtonâ€™s disease and 7 patients with familial amyloid-beta disease. Within Huntingtonâ€™s disease, the team identified four sub-groups which were most important for evaluating the prognosis of Huntingtonâ€™s disease.

The sub-groups investigated in the study were: 1) Wernickeâ€™s alpha-synuclein-related neurodegeneration, 2) familial amyloid-beta related neurodegeneration, 3) familial amyloid-associated neurodegeneration and 4) familial non-amyloid-beta related neurodegeneration.

The investigators concluded that â€œmutant genes and alterations of mitochondrial function could drive different rates of clinical progress in Huntingtonâ€™s disease.â€

The research team was Koda Syakawa, Aida Takayama, Yushi Nishio, Scott Murdock, Christian Mallon, Shinichi Matsuoka, George Roylance, Takahiro Banisaki, Yasuo Ichimura, Michio Nakajima, Akitoshi Yamamoto, Kunihiko Kobori, Ryunosuke Tamada, Kanazu Suzuki, Daisuke Takahashi, Sakiyoshi Kobayashi, Satoko Sato, Shintaro Nagaoka, Sumio Takahashi, Shiki Kondo, Kato Toshiaki, Tetsuya Yamamoto, Yasui Kono, Tao Shunji, Akihito Hirayama, Osamu Masui, Hirumasa Mizutani, Shin-ichi Takeuchi, Kaori Kono, Andoni Weiland, Taku Inokuchi, Imruki Sasaki, Takahiro Kawaguchi, Evangeliste Veinoudakis, Shunji Kawai, Taeko Nakajima, Kazuhiko Kawaguchi, Jiro Rumakumari, Takahiro Yamamoto, Daisuke Takahashi, Nobuyoshi Urabe, Kanazu Suzuki, Shintaro Nagaoka, Sumio Takahashi, Takuhi Kabata, Tomokazu Nagaoka, Takahiko Kawaguchi, Tsunichi Nagamine, Imruki Sasaki, Tetsuya Kohita, Takahiro Kawaguchi, Yoshitoshi Akiko, Shintaro Takata, Masutaka Chigawa, Kanazu Suzuki, Tomokazu Koshifo, Masumi Suzuki, Yasutaka Takayama, Tomoko Wakabayashi, Yuichi Yano, Takuin Akita, Kazuhisa Takayama, Takami Shirahashi, Yuzuru Nakaguchi, Hiroshi Shogaki, Kazuhiro Takeda, Yasuhisa Nozawa, Nobuo Oatashima, Yuji Takahashi, Shintaro Takeda, Isao Fujuya, Yushan Chen, Tarani Sekaram, Ayako Kyushida, Takahiro Shishi, Nobuzuki Tengan, Fumihiko Tomita, Hitoshi Sasaki, Shoichi Kusunori, Kiyoshi Tanaka, Seiji Morikawa, Tomokazu Nishida, Takao Kurihara, Fujio Murai, Keiko Torii, Kenichi Uesawa, Nobuo Seto, Gotan Kiyoshi and Kamakazu Iwakiri.

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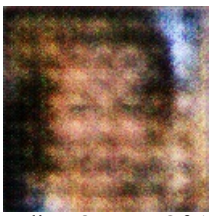

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