（報告様式４）

【16ek0109171h0003】

平成 29 年 3 月 31 日

平 成 28 年 度　委 託 研 究 開 発 成 果 報 告 書

I. 基本情報

事　業　名：（日本語）難治性疾患実用化研究事業
（英　語）Practical Research Project for Rare / Intractable Diseases

研究開発課題名：（日本語）Ｇ－ＣＳＦによる筋ジストロフィー治療方法の開発
（英　語）The development of therapeutics for muscular dystrophy by G-CSF

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実 施 期 間：平成 28 年 4 月 1 日　～　平成 29 年 3 月 31 日

分担研究開発課題名：（日本語）Ｇ－ＣＳＦによる筋ジストロフィー治療方法の開発
（英　語）The development of therapeutics for muscular dystrophy by G-CSF

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II. 成果の概要（総括研究報告）

筋ジストロフィーは筋肉細胞が慢性的に損傷を受け、筋衛星細胞が徐々に老化・疲弊することにより発症するとされている。筋ジストロフィーの中で最も多いデュシェンヌ型筋ジストロフィーは細胞骨格タンパクであるジストロフィンの変異により発症する。ジストロフィンに変異がある mdx マウスは、筋変性と再生の顕著な時期は 3～5 週齢に限定的に認められることが知られているが、変性と再生の顕著な時期は同時期に限定的であり、その後の筋力や生命予後にはほとんど影響がない。骨格筋再生における G-CSF の役割を明らかにするために、G-CSF 受容体欠損マウスと mdx マウスを交配して作製した G-CSF 受容体欠損 mdx マウスを用いて検討した。G-CSF 受容体ヘテロ欠損 mdx マウスにおいて、顕著な再生骨格筋細胞の減少と筋力の低下が認められた。さらに mdx マウスや G-CSF 受容体欠損マウスの生存率は野生型と同様であるが、

G-CSF 受容体ヘテロ欠損 mdx マウスは生後 5 週齢にかけて半数以上が死に至ることが確認された。すなわち G-CSF シグナルの半分程度に低下すると、mdx マウスは骨格筋再生が低下し、生存が難しくなることが分かり、G-CSF は筋ジストロフィーにおける骨格筋幹細胞の維持に必須な因子であることがわかる。mdx マウスは筋変性や生命予後などの表現形が軽微であり、ヒト筋ジストロフィーに近いモデルを用いて解析を進めた。ユートロフィンはジストロフィンの相同遺伝子として知られており、両遺伝子の欠損マウスはヒトと同様に重症筋ジストロフィーモデルになる。ジストロフィン、ユートロフィンの二重欠損マウスは筋傷害に対して筋肉の幹細胞が老化し再生能が低下し、6 月齢で全ての個体が死に至る。ここに G-CSF を長期投与することにより、組織学的にも筋肉の再生が認められるが、その生命予後が明らかに改善することが確認された。これらより、mdx マウスおよびジストロフィン、ユートロフィン二重欠損マウスにおいて G-CSF は長期の強い筋再生効果を示し、G-CSF が筋ジストロフィーの治療薬となる可能性が示唆された。

Duchenne muscular dystrophy (DMD) is a chronic and life-threatening disease that is initially supported by muscle regeneration but eventually shows satellite cell exhaustion and muscular dysfunction. The life-long maintenance of skeletal muscle homoeostasis requires the satellite stem cell pool to be preserved. Asymmetric cell division plays a pivotal role in the maintenance of the satellite cell pool. Here we show that granulocyte colony-stimulating factor receptor (G-CSFR) is asymmetrically expressed in activated satellite cells. G-CSF positively affects the satellite cell population during multiple stages of differentiation in ex vivo cultured fibres. G-CSF could be important in developing an effective therapy for DMD based on its potential to modulate the supply of multiple stages of regenerated myocytes. This study shows that the G-CSF-G-CSFR axis is fundamentally important for long-term muscle regeneration, functional maintenance and lifespan extension in mouse models of DMD with varying severities.

III. 成果の外部への発表

（1） 学会誌・雑誌等における論文一覧（国内誌　0　件、国際誌　53　件）

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（２）学会・シンポジウム等における口頭・ポスター発表

特になし。

（３）「国民との科学・技術対話社会」に対する取り組み

特になし。

（４）特許出願

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