た一連の変異体を新たに作成した(ΔPシリーズ)。HIV-1 の Env を補った粒子を用いて感染能測定を行ったが、ΔP シリーズはすべて野性株と比べて 1/100 以下に感染能が低下がしていた。ゲノムの二量体化の程度の異なるすべての変異体で同様の感染能低下があったことから、この現象は単量体ゲノムの出現とは別の理由によるものと考えられた。この原因を知るために感染 16 時間後での感染細胞内のウイルス DNA（逆転写産物）を半定量 PCR により調べると、逆転写初期の反応はどの変異体でも野性株の 10%以上の効率で起こっているのに対し、ほとんどのストランドトランスファーが挿入配列の相同領域を標的として起きてしまうために異常な逆転写産物を産生してしまうことが明らかとなった。これが感染能低下の主原因と考えられる。このことからゲノム二量体化を完全に阻害した変異体においてもある程度の効率で逆転写反応は開始しており、なおかつストランドトランスファーも起こることが明らかとなった。

D，考察

HIVのウイルス粒子内でRNA一本鎖ゲノムは非共有的に結合した二量体として存在している。これによりウイルスは相同組換えによる遺伝的多様性の獲得や、ゲノム損傷の補償を行っていると言われる。しかしそれらの理由が二量体化という複雑で手間のかかるイベントを、物理的制約を抱えるウイルスが捨てずにいることを十分説明できるとは考えにくい。本研究でHIV-1ゲノムRNA上の二量体化領域(DLS)を同一RNA上に複数個配置することによって、正常ウイルス粒子内に単量体のゲノムを存在させることに初めて成功した。この研究の結果から、HIV-1においてゲノムがウイルス粒子にパッケージングされる際には二量体化したゲノムが必要なのではなく、E/DLSが二つ存在していることが必要十分であることが示された。単量体ゲノムを持つウイルスにおいても粒子産生、パッケージング、粒子成熟、ゲノム逆転写がある程度の効率で起こっていると言うことは、ウイルスの感染能獲得にゲノムの二量体化がそれほど積極的に関わっているわけではないことを示唆しているのかもしれない。むしろHIV-1のゲノムパッケージングというプロセスにおいて、E/DLS領域のRNAが二本結合して形づくる構造がパッケージングシグナルとして認識されるという段階が存在する可能性が考えられる。この仮説は長年の命題であった二量体化の理由に対する簡潔で合理的な説明と

なり得るものである。現在はこの結果を応用して構築した、ゲノム二量体化を効果的に検出できるシステムを用いて二量体化領域の構造についての詳細な機能相関を明らかにし、仮説を検証することに取り組んでいる。

E，結論

HIV-1ゲノムRNA上のE/DLS領域をゲノムRNA上に複数配置することにより、HIV-1正常粒子中に単量体化したゲノムが生成することを見いだした。導入変異を改変することで単量体ゲノムしか持たない粒子の作成にも成功し、ゲノム二量体化がHIV-1のゲノムパッケージング及び正常粒子産生に必須でないことが明らかとなった。

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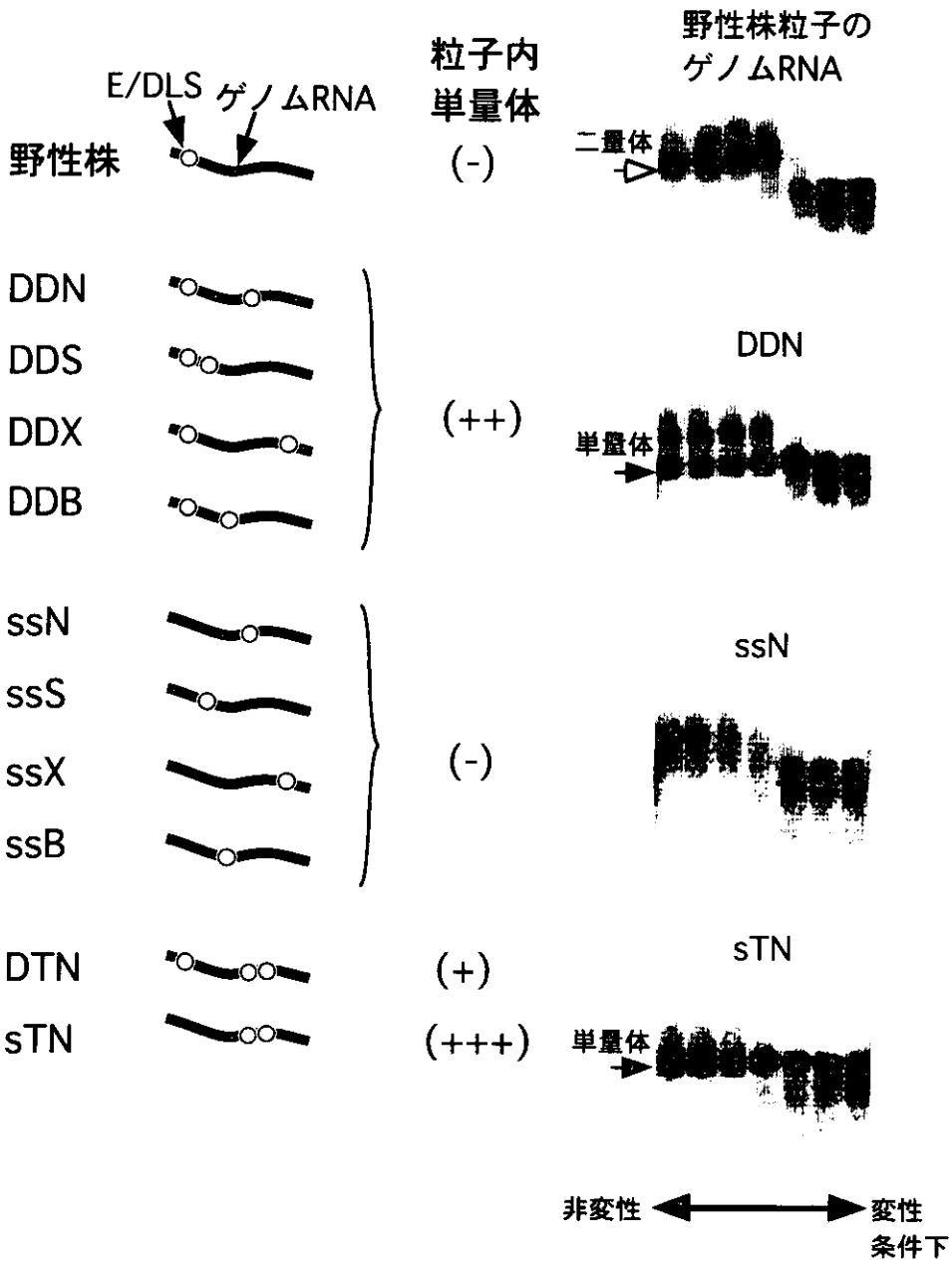
G，知的所有権の取得状況

なし

図１、粒子内単量体を産生するHIV-1変異体



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横幕　能行

無し。

松田 善衛

無し。

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