DAOA 遺伝子では検索した 6 遺伝子多型のうち m22 (rs778293)で遺伝子型とアレル頻度において覚せい剤精神病と健常者群の間で有意差を認め（表 4，遺伝子型：p=0.00016、アレル：P=0.0015），マイナーアレルの G が覚せい剤精神病群では健常者群に比べて有意に多かった。オッズ比はアレル G で 1.6 倍，遺伝子型 GG で 5.7 倍であった。各臨床表現型では有意な相関はなかった。DAOA 遺伝子 6 多型間の連鎖不平衡解析から，3'側の m12 と m15，5'側の m23 と m24 が連鎖不平衡関係にあることが分かったので，この 2 部位でハプロタイプ解析をした。その結果，両部位で覚せい剤精神病と有意な相関がみられた（m12-m15, global permutation p =0.0003,および m23-m24, p= 0.010）。アレル頻度では m12-m15 ハプロタイプで G-A が有意に患者で少なく，防禦ハプロタイプ（p=0.00024），m23-m24 ハプロタイプでは T-T が有意に患者で多くリスクハプロタイプ(p=0.00085)であることがわかった。

表 2. DTNBP1 遺伝子の P1635 多型と覚せい剤精神病臨床表現型

P1635	N	AA	AG	GG		A	G	
Latency to Onset of Psychosis, <3Y	99	89(89.9)	10(10.1)	0(0.0)		188(94.9)	10(5.1)	
Latency to Onset of Psychosis, ≥3Y	81	71(87.7)	10(12.3)	0(0.0)	0.63	152(93.8)	10(6.2)	0.64
Transient MAP Psychosis	107	100(93.5)	7(6.5)	0(0.0)		207(96.7)	7(3.3)	
Prolonged MAP Psychosis	82	68(82.9)	14(17.1)	0(0.0)	0.022	150(91.5)	14(8.5)	0.027
Spontaneous Relapse, No	111	98(88.3)	13(11.7)	0(0.0)		209(94.1)	13(5.9)	
Spontaneous Relapse; Yes	82	73(89.0)	9(11.0)	0(0.0)	0.87	155(94.5)	9(5.5)	0.88

表 3. DTNBP1 遺伝子のハプロタイプと覚せい剤精神病

Haplotype P1655-P1635-SNPA	Controls Frequency	MAP Psychosis Frequency	permutation p
C-A-A	0.7101	0.6046	0.0013
G-A-A	0.2741	0.3315	0.076
C-G-T	0.0022	0.0318	0.0012
C-G-A	0.0023	0.0178	0.11
C-A-T	0.0073	0.0055	0.83
G-G-A	0.0000	0.0089	0.15
G-A-T	0.0039	0	0.18

SRR 遺伝子では，3 多型はいずれも覚せい剤精神病と有意な相関はなかった。3 多型からなるハプロタイプ解析でも，有意な相関はなかった。臨床表現型での検討では、SNP3 (rs2224770)が精神病症状の自然再燃の有無に弱いながら有意に相関（p=0.045），SNP5 (rs408067)の遺伝子型が精神病予後に相関（CC+GC vs GG; p=0.039），多剤乱用の有無で遺伝子型（GG+GC vs CC, p=0.029）およびアレル（p=0.038）が有意な相関を示した。しかし，これらは多重比較補正後では有意ではなかった。

表 4．DAOA 遺伝子と覚せい剤精神病の相関解析

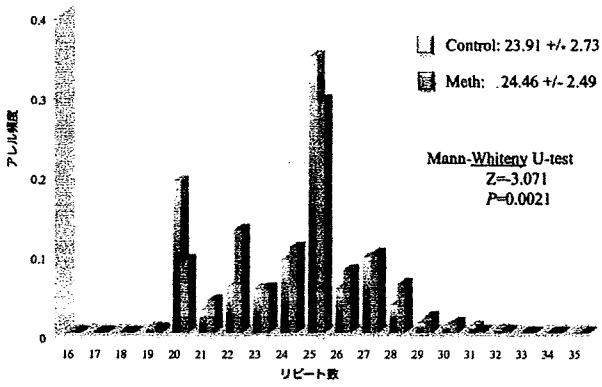
Loci	N	Genotype (%)			p	Allele (%)		p
m12 (rs3916965)		A/A	A/G	G/G		A	G	
controls	278	144(51.8)	115(41.4)	19(6.8)		403(72.5)	153(27.5)	
patients	209	117(56.0)	75(35.9)	17(8.1)	0.45	309(73.9)	109(26.1)	0.62
m15 (rs2391191)		A/A	A/G	G/G		A	G	
controls	279	143(51.3)	115(41.2)	21(7.5)		401(71.9)	157(28.1)	
patients	205	111(54.1)	74(36.1)	20(9.8)	0.43	296(72.2)	114(27.8)	0.91
m18 (rs947267)		A/A	A/C	C/C		A	C	
controls	288	122(42.4)	133(46.2)	33(11.4)		377(65.5)	199(34.5)	
patients	206	99(48.1)	85(41.2)	22(10.7)	0.45	283(68.7)	129(31.3)	0.29
m22 (rs778293)		A/A	A/G	G/G		A	G	
controls	287	179(62.4)	102(35.5)	6(2.1)		460(80.1)	114(19.9)	
patients	203	109(53.7)	72(35.5)	22(10.8)	0.00016 OR=5.7	290(71.4)	116(28.6)	0.0015 OR=1.6
m23 (rs3918342)		T/T	T/C	C/C		T	C	
controls	291	72(24.7)	165(56.7)	54(18.6)		309(53.1)	273(46.9)	
patients	208	62(29.8)	98(47.1)	48(23.1)	0.11	222(53.4)	194(46.6)	0.93
m24 (rs1421292)		A/A	A/T	T/T		A	T	
controls	282	76(27.0)	145(51.4)	61(21.6)		297(52.7)	267(47.3)	
patients	205	51(24.9)	90(43.9)	64(31.2)	0.054	192(46.8)	218(53.2)	0.072

GRIN1 遺伝子では検索した 3 遺伝子は覚せい剤精神病と有意な相関は認めなかった。臨床表現型との検討で rs2301364 で精神病の自然再燃（いわゆるフラッシュバック）において遺伝子型 (p=0.042),アレル頻度（p =0.032）で有意な相関を認め、マイナーアレルである T アレル、および TT 遺伝子型を有する個人では自然再燃が生じにくくなり、オッズ比はそれぞれ 0.62、0.23 であった。GRIN1 の 3 遺伝子多型のハプロタイプ解析を行ったが、2 遺伝子間、3 遺伝子間でも統合失調症発症脆弱性とは相関は示さなかった。GRIN2B の rs1019385、rs1805502 では前者の遺伝子型分布が覚せい剤精神病患者と健常コントロールで p=0.049 と弱い有意差を認めた。マイナーアレル G のホモ接合 GG 遺伝子型はオッズ比 1.57 と弱い危険因子であった。しかし、アレル頻度では差はなかった。臨床表現型では有意な相関はなかった。

GRIN2A の(GT)n 繰り返し多型では 16 リピートから 35 リピートまでみられ、25 リピートが最も多く、これまでの日本人での報告[10,12]とほぼ一致した。各リピートアレル頻度の

分布は覚せい剤精神病は健常コントロールと有意な差がみられた（p＝0.002, 図 1）。平均リピート数は健常コントロールが 23.9+/-2.73 で、覚せい剤精神病では 24.46+/-2.49 と患者群でリピート数が有意に多かった。この(GT)n 繰り返し多型と臨床表現型では特に有意な相関を示したものはなかった。

図 1. 覚せい剤精神病と GRIN2A の(GT)n リピート多型のアレル分布



D. 考察

P1635 の G アレルは覚せい剤精神病の危険因子であることが示され、日本人とアイルランド人の統合失調症における研究結果と一致していた [7, 13]。又、同アレルは精神症状の遷延、治療後の予後不良因子となることが発見された。P1655－P1635－SNPA の 3 つのマーカーによるハプロタイプ解析で、C-A-A ハプロタイプは患者群に比べて健常者群で有意に多く、保護的因子であることが示された。統合失調症や精神病性双極性障害においても同一の結果が示され [7, 8]、C-A-A ハプロタイプは、妄想や幻覚などの精神症状を合併する内因性精神病や薬物乱用に対する発症脆弱性に対する防御因子であることが示唆された。

Dysbindin の生理機能や病態生理との関係については知見が集まりつつある。Talbot ら [14]は、統合失調症患者の海馬で dysbindin が減少しており、小胞体グルタミン酸トランスポーター発現レベルと逆の相関していることを示した。培養細胞を用いた in vitro の研究では、dysbindin の過剰発現により、シナプス小胞の蛋白（SNAP25、Synapsin□）の増加と共にグルタミン酸放出が増加し、反対に dysbindin の低下によりグルタミン酸放出が減少することが示されている [15]。統合失調症患者の脳において、dysbindin の発現低下が脳内のグルタミン酸システムの機能低下を来たす可能性があり、これは統合失調症の病因仮説として有力な候補である。統合失調症と覚せい剤精神病との臨床的類似性に基づくと、両者の共通の分子学的機構はドパミンシステムだけでなく、グルタミン酸システムが関与していることが推定された。覚せい剤精神病の動物モデルによる実験で、NMDA 受容体とグルタミン酸システムが行動感作における主要な役割を果たすことが分かっている [16]。今回の結果から DTNBP1 遺伝子変異がグルタミン酸系神経伝達を変化させ、覚せい剤精神病へ関与することを示唆している。

DAOA 遺伝子の m22 および m12-m15 ハプロタイプ，m23-m24 ハプロタイプが覚せい剤精神病と強い相関を示したことから，DAOA の遺伝子変異により個体の覚せい剤精神病への罹患脆弱性が影響を受けると考えられる。DAOA は D 体アミノ酸を酸化代謝する D-amino acid oxidase を活性化するため，NMDA 受容体の co-agonist の一つである D-serine の代謝変化により，NMDA 受容体を介したグルタミン酸神経伝達が変化することで覚せい剤精神病の発症脆弱性を増すと考えられる。ただし，これらの多型の DAOA 分子の活性変化への影響は現在のところ分かっていないし，また，多型の位置も，m15 はエクソン 2 上に位置するが，m12 は DAOA 遺伝子の上流であり，m22, m23, m24 は遺伝子の下流にあり，いずれも遺伝子上には位置していないので，今回の多型により DAOA の機能変化が生じているという証明を待つ必要がある。一方，DAOA 遺伝子の統合失調症との相関研究は 2002 年の Chumakov ら [17] のものを皮切りに現在まで約 20 の研究が報告されているが，その結果は解析部位，またその結果など一致しない点が多く結論には至っていないのが現状である。そこで，いくつかメタ解析がおこなわれている。Ma ら [18]は 5 つの研究を対象に m12, m22, m23 で約 2000 例ずつのメタ解析ですべて有意差がないこと，明らかな異種性があること，アジア人に限定すると異種性

は減り，m22 でアレル G がアジア人では有意なリスクであったと報告した。また，Shi ら[19]は DAOA 遺伝子の統合失調症での 16 研究のメタ解析をアジア人とヨーロッパ人に分けて解析をし，その結果，アジア人では m18 の A アレル，m22 の G アレルが危険アレル，ヨーロッパ人では m24 の T アレルが弱い危険アレルであるとした。今回の解析では，日本人の覚せい剤精神病でも m22 の G アレルが危険アレルであり，このことは，DTNBP1 遺伝子と同様に DAOA 遺伝子多型は統合失調症という疾患自体ではなく，精神病症状を合併しやすさに関わっている可能性が推定される。

NMDA 受容体サブタイプ遺伝子解析では、GRIN2A の(GT)n 繰り返し多型が覚せい剤精神病と強い相関を示した。平均リピート数は覚せい剤精神病患者が健常コントロール群と比べて有意に多いことが明らかになった。（GT）n リピート多型は Itokawa ら[10]が日本人統合失調症で相関することを最初に報告し、注目されており、その後の追跡解析でも相関が確認されている[12]。Itokawa ら[10]は GRIN2A の全域のエクソンとその周辺部の多型サーチを行い、（GT）n リピート多型を含め 5 つの多型しか存在しないこと、そして、統合失調症とは（GT）n リピート多型のみが相関したことを見いだした。更に、この多型は Luciferase assay で転写活性に影響することを見いだしており、リピート数が 0 回、12 回、25 回、42 回と多くなるほど、転写活性が低下することを見いだしている。また、死後脳での MK-801 結合活性も低下することを明らかにしている。彼らの統合失調症での検討では、（GT）n リピート数が有意に健常者より多いと報告しており、今回の覚せい剤精神病と同じ方向である。従って、（GT）n リピート数が多い個体では、NMDA 受容体数が生来的に少なく、NMDA 受容体を介した神経伝達が不良で、それが統合失調症や覚せい剤誘発性精神病の危険因子になるということが推定される。

これまで、グルタミン酸神経伝達や NMDA 受容体関連分子をコードする遺伝子群の解析で DTNBP1, DAOA, SRR, GRIN2A といった一連の遺伝子が覚せい剤精神病に相関することがわかった。この他に、われわれは NMDA 受容体の co-agonist である glycine や D-serine の濃度を規定する glycine transporter type I も覚せい剤精神病と相関することを報告している[20]。また、これらの結果の一部は統合失調症や精神病症状を伴う双極性障害[7, 8, 10, 18, 19]と共通している。遺伝子型と生理機能との関連性が確認されているのは GRIN2A の（GT）n リピート多型のみであるが、これまでの知見を総合すると、DTNBP1 多型によりグルタミン酸放出が低下することや、DAOA, GLYT-1, SRR 多型により NMDA 受容体の co-agonist である glycine や D-serine が低下すること、もしくは NMDA 受容体の GRIN2A サブタイプの発現低下などのどこかの異常により、最終的に NMDA 受容体活性化が減弱し、この系のシグナル伝達が低下することが、内因性、薬剤性を問わず精神病の合併危険性を増加させ、また、精神病の遷延やフラッシュバックの合併など予後の悪さにも影響すると考えられた。

E. 結論

グルタミン酸神経伝達に関わる複数の分子をコードする遺伝子、DTNBP1, DAOA, SRR, GRIN1, GRIN2A, GRIN2B を case-control 相関解析で検討し、特に DTNBP1, DAOA, GRIN2A が覚せい剤精神病の発症脆弱性と強く相関することが明らかとなった。また、危険アレルやハプロタイプは統合失調症や妄想を伴う双極性障害でのそれと同一のものが多いことから、生来的にグルタミン酸神経伝達、特に NMDA 受容体を介した伝達不全がある個人では覚せい剤乱用による精神病合併や内因性精神病に脆弱性を示すと考えられた。従って、この系を改善させる薬剤があらたな治療法になると推定された。

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F. 健康危険情報

なし

G. 研究発表

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H. 知的財産権の出願・登録状況

1. 特許取得

なし

2. 実用新案登録

なし

3. その他

なし

平成19～21年度　3年間　刊行物一覧

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