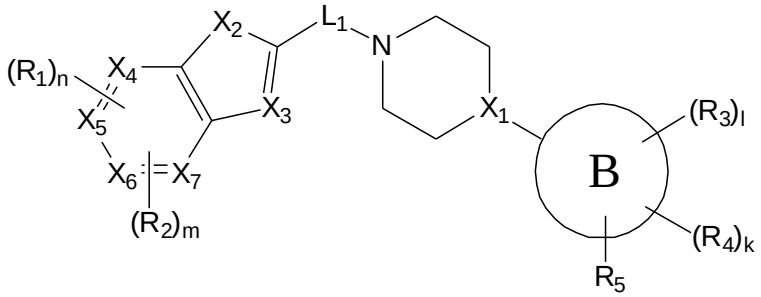
progression. To date, however, it is unclear whether these drugs improve symptoms such as dyspnoea and cough, or whether their beneficial effect on functional decline translates to increased survival.

[0012] The compounds of this invention target activated fibroblasts and collagen over production and can therefore be used for treating fibrosis, including primary or secondary fibrosis, such as systemic sclerosis, graft-versus host disease (GVHD), pulmonary fibrosis and autoimmune disorders, lung fibrosis and idiopathic pulmonary fibrosis (IPF), as well as localized processes, as for example, pulmonary fibrosis and liver cirrhosis, or more generalized processes, like progressive systemic sclerosis. The compounds can be further useful in the treatment of different forms of dermal fibrosis, which in addition to scleroderma, include localized and generalized morphea, keloids, hypertrophic scars, familial cutaneous collagenoma and connective tissue nevi of the collagen type. The compounds can be further useful in the treatment of lung fibrosis and idiopathic pulmonary fibrosis (IPF), as well as hepatic fibrosis, resulting from hepatic scarring, caused by surgery or other forms of injury associated with mechanical biliary obstruction. Such fibrosis can lead to portal hypertension, in which scarring distorts blood flow through the liver, or cirrhosis as well as other hepato-fibrotic disorders including Non-alcoholic steatohepatitis (NASH), and alcoholic steatohepatitis (ASH), non-alcoholic fatty liver disease (NAFLD) and alcoholic fatty liver disease (AFLD), which can be similarly be treated by compounds of the invention.

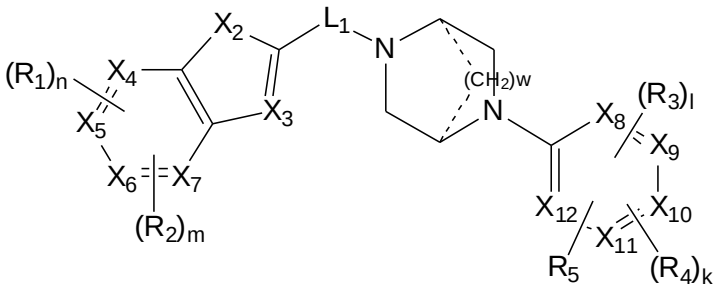
SUMMARY

[0012a] In one aspect of the present disclosure, there is provided a compound represented by the structure of formula **III**:



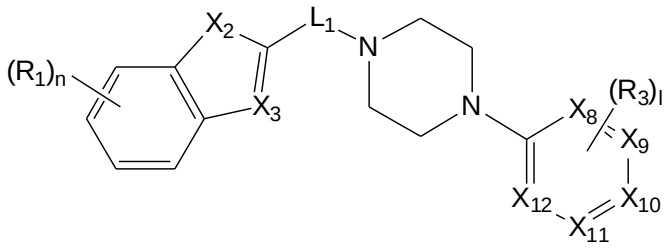
III;

by the structure of formula **V(a)**:



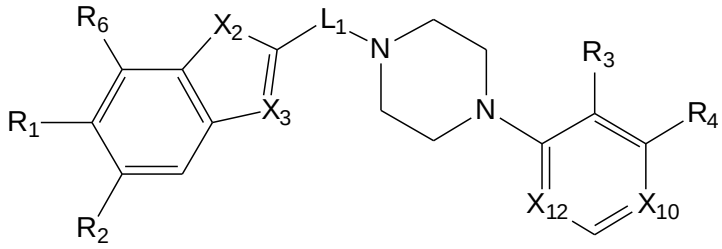
V(a);

by the structure of formula **VI**:



VI;

or by the structure of formula **VII**:



VII;

wherein

B ring is a single or fused aromatic or heteroaromatic ring system (e.g., phenyl, pyrimidine, 2-, 3- or 4-pyridine, pyridazine or pyrazine, thiazole, pyrrole, triazole, imidazole, indazole);

$\mathbf{X_1}$ is N or C(R) (e.g., C-H, C-OH);

$\mathbf{L_1}$ is CH_2, CHR, or $C(R)_2$;

$\mathbf{R_1}$, $\mathbf{R_2}$ and $\mathbf{R_6}$ are each independently H, F, Cl, Br, I, OH, SH, R_8-OH, R_8-SH, -R_8-O-R_{10} (e.g., CH_2-CH_2-O-CH_3, CH_2-O-CH_2-CH_2-O-CH_3), -O-R_8-O-R_{10} (e.g., O-CH_2-CH_2-O-CH_3), R_8-(C_3-C_8 cycloalkyl), R_8-(C_3-C_8 heterocyclic ring), O-$\mathbf{R_{20}}$, CF_3, CD_3, OCD_3, CN, NO_2, -CH_2CN, -R_8CN, NH_2, NHR, $N(R)_2$, R_8-N(R_{10})(R_{11}), R_9-R_8-N(R_{10})(R_{11}), $B(OH)_2$, -OC(O)CF_3, -OCH_2Ph, NHC(O)-R (e.g., NHCO-Ph; NHCO-CH_3) , NHC(O)-R_{10} (e.g., NHCO-CH_3) NHCO-N(R_{10})(R_{11}), -C(O)Ph, C(O)O-R_{10}, R_8-C(O)-R_{10}, C(O)H, C(O)-R_{10}, C_1-C_5 linear or branched C(O)-haloalkyl, -C(O)NH_2, C(O)NHR (e.g., C(O)NH-Ph), C(O)N(R_{10})(R_{11}), SO_2R, SO_2N(R_{10})(R_{11}), $NHSO_2$(R_{10}) (e.g., $NHSO_2CH_3$), CH(CF_3)(NH-R_{10}), C_1-C_5 linear or branched, substituted or unsubstituted alkyl (e.g., methyl, ethyl), C_1-C_5 linear or branched, substituted or unsubstituted alkenyl, C_1-C_5 linear, branched or cyclic haloalkyl (e.g., CHF_2), C_1-C_5 linear, branched or cyclic alkoxy (e.g. methoxy), optionally wherein at least one methylene group (CH_2) in the alkoxy is replaced with an oxygen atom, C_1-C_5 linear or branched thioalkoxy, C_1-C_5 linear or branched haloalkoxy, C_1-C_5 linear or branched alkoxyalkyl, substituted or unsubstituted C_3-C_8 cycloalkyl (e.g., cyclopropyl), substituted or unsubstituted C_3-C_8 heterocyclic ring (e.g., pyridine), substituted or unsubstituted aryl (e.g., phenyl), substituted or unsubstituted benzyl;

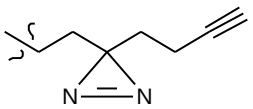
or $\mathbf{R_2}$ and $\mathbf{R_1}$ are joined together to form a 5 or 6 membered substituted or unsubstituted, aliphatic or aromatic, carbocyclic (e.g., benzene) or heterocyclic (e.g., pyridine) ring;

$\mathbf{R_3}$, $\mathbf{R_4}$ and $\mathbf{R_5}$ are each independently H, F, Cl, Br, I, OH, SH, R_8-OH, R_8-SH, -R_8-O-R_{10} (e.g., CH_2-CH_2-O-CH_3, CH_2-O-CH_2-CH_2-O-CH_3), R_8-(C_3-C_8 cycloalkyl), R_8-(C_3-C_8 heterocyclic ring), O-$\mathbf{R_{20}}$, CF_3, CD_3, OCD_3, CN, NO_2, -CH_2CN, -R_8CN, NH_2, NHR, $N(R)_2$, R_8-N(R_{10})(R_{11}), R_9-R_8-

$N(R_{10})(R_{11})$, $B(OH)_2$, $-OC(O)CF_3$, $-OCH_2Ph$, $NHC(O)-R_{10}$, $NHCO-N(R_{10})(R_{11})$, COOH, $-C(O)Ph$, $C(O)O-R_{10}$, $R_8-C(O)-R_{10}$, C(O)H, $C(O)-R_{10}$, C_1-C_5 linear or branched C(O)-haloalkyl, $-C(O)NH_2$, C(O)NHR, $C(O)N(R_{10})(R_{11})$, SO_2R, $SO_2N(R_{10})(R_{11})$, $CH(CF_3)(NH-R_{10})$, C_1-C_5 linear or branched, substituted or unsubstituted alkyl (e.g., methyl, ethyl), C_1-C_5 linear or branched, substituted or unsubstituted alkenyl, C_1-C_5 linear, branched or cyclic haloalkyl (e.g., CHF_2), C_1-C_5 linear, branched or cyclic alkoxy (e.g. methoxy), optionally wherein at least one methylene group (CH_2) in the alkoxy is replaced with an oxygen atom, C_1-C_5 linear or branched thioalkoxy, C_1-C_5 linear or branched haloalkoxy, C_1-C_5 linear or branched alkoxyalkyl, substituted or unsubstituted C_3-C_8 cycloalkyl (e.g., cyclopropyl), substituted or unsubstituted C_3-C_8 heterocyclic ring (e.g., pyrazole, thiazole), substituted or unsubstituted aryl (e.g., phenyl), substituted or unsubstituted benzyl;

or **R_3** and **R_4** are joined together to form a 5 or 6 membered substituted or unsubstituted, aliphatic (e.g., cyclopentene) or aromatic, carbocyclic (e.g., benzene) or heterocyclic (e.g., thiophene, furane, pyrrol, pyrazole) ring;

R_{20} is represented by the following structure:



X_2 is NH, S, O, N-R (e.g., $N-CH_2-CH_2-O-CH_3$);

X_3 is N, C(R) (e.g., CH, $C-CH_3$, C-Cl, C-CN);

X_4, **X_5**, **X_6**, and **X_7** are each independently C or N;

X_8, **X_9**, **X_{10}**, **X_{11}**, and **X_{12}** are each independently C or N;

R is H, OH, F, Cl, Br, I, CN, CF_3, NO_2, C_1-C_5 linear or branched, substituted or unsubstituted alkyl (e.g., methyl, ethyl), C_1-C_5 linear or branched alkoxy, $-R_8-O-R_{10}$ (e.g., $CH_2-CH_2-O-CH_3$), C_1-C_5 linear or branched haloalkyl (e.g., CF_3, CF_2CH_3, CH_2CF_3, $CF_2CH_2CH_3$, $CH_2CH_2CF_3$, $CF_2CH(CH_3)_2$,$CF(CH_3)-CH(CH_3)_2$), R_8-aryl (e.g., CH_2-Ph), substituted or unsubstituted aryl (e.g., phenyl), substituted or unsubstituted heteroaryl (e.g., pyridine (2, 3, and 4-pyridine); or

two geminal **R** substitutions are joined together to form a 3 - 6 membered substituted or unsubstituted, aliphatic (e.g., cyclopropyl, cyclopentene) or aromatic, carbocyclic (e.g., benzene) or heterocyclic (e.g., thiophene, furane, pyrrol, pyrazole) ring;

R_8 is $[\mathbf{CH_2}]_p$

wherein **p** is between 1 and 10 (e.g., 2);

R_9 is $[CH]_q$, $[C]_q$

wherein **q** is between 2 and 10;

R_{10} and **R_{11}** are each independently H, substituted or unsubstituted, C_1-C_5 linear or branched alkyl (e.g., methyl, ethyl, $CH_2-CH_2-O-CH_3$), C_1-C_5 linear or branched alkoxy (e.g., $O-CH_3$), C(O)R, or $S(O)_2R$;

or **R_{10}** and **R_{11}** are joined to form a substituted or unsubstituted C_3-C_8 heterocyclic ring (e.g., piperazine, piperidine),

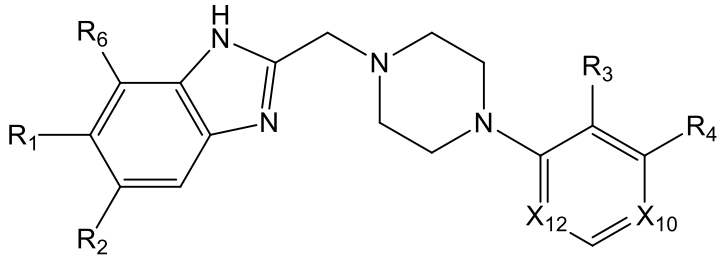
wherein substitutions include: F, Cl, Br, I, OH, SH, C_1-C_5 linear or branched alkyl, C_1-C_5 linear or branched alkyl-OH (e.g., $C(CH_3)_2CH_2$-OH, CH_2CH_2-OH), C_3-C_8 heterocyclic ring (e.g., piperidine), alkoxy, $N(R)_2$, CF_3, aryl, phenyl, halophenyl, (benzyloxy)phenyl, CN, NO_2 or any combination thereof;

m, **n, l** and **k** are each independently an integer between 0 and 4 (e.g., 0, 1 or 2);

w is 0, 1 or 2; wherein if w=0, the bridge on the ring is absent;

wherein if X_3 is N, then X_2 is not NH;

or by the structure of formula **IX**:



IX

wherein

R_1 is Cl, -R_8-O-R_{10} (e.g., CH_2-CH_2-O-CH_3, CH_2-O-CH_2-CH_2-O-CH_3), -O-R_8-O-R_{10} (e.g., O-CH_2-CH_2-O-CH_3), O-**R_{20}**, or CF_3;

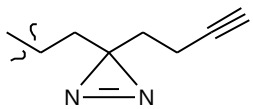
R_2 is H, Cl, -R_8-O-R_{10} (e.g., CH_2-CH_2-O-CH_3, CH_2-O-CH_2-CH_2-O-CH_3), -O-R_8-O-R_{10} (e.g., O-CH_2-CH_2-O-CH_3), O-**R_{20}**, or CF_3;

R_6 is H;

R_3 and **R_4** are each independently H, O-**R_{20}**, C_1-C_5 linear or branched, substituted or unsubstituted alkyl (e.g., methyl, ethyl), substituted or unsubstituted C_3-C_8 heterocyclic ring (e.g., imidazole), (wherein substitutions include: F, Cl, Br, I, C_1-C_5 linear or branched alkyl, OH, alkoxy, CF_3, aryl, phenyl, heteroaryl, C_3-C_8 cycloalkyl, halophenyl, (benzyloxy)phenyl, CN, NO_2 or any combination thereof);

or **R_3** and **R_4** are joined together to form a 5 or 6 membered substituted or unsubstituted, aliphatic (e.g., cyclopentene) or aromatic, carbocyclic (e.g., benzene) or heterocyclic (e.g., thiophene, furane, pyrrol, pyrazole) ring;

R_{20} is represented by the following structure:

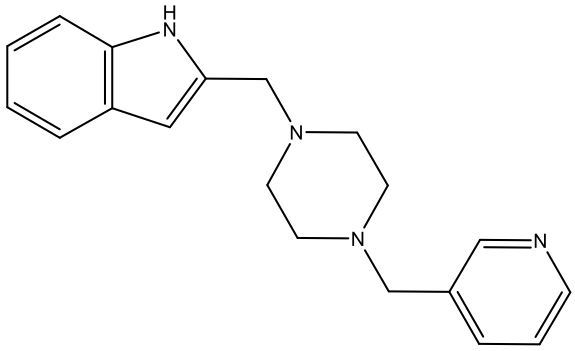
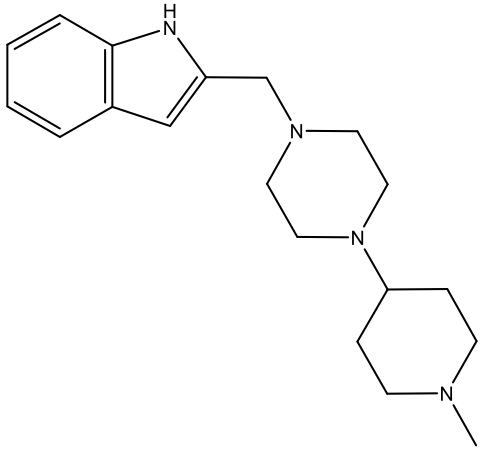
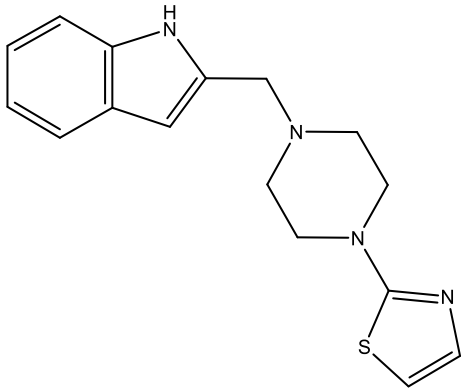
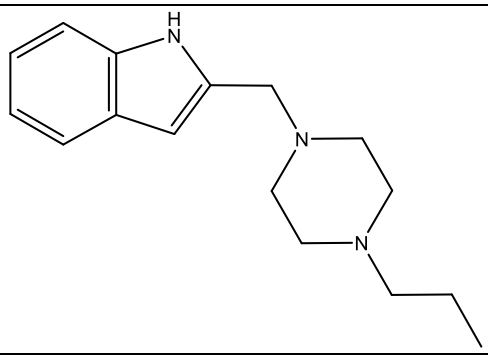


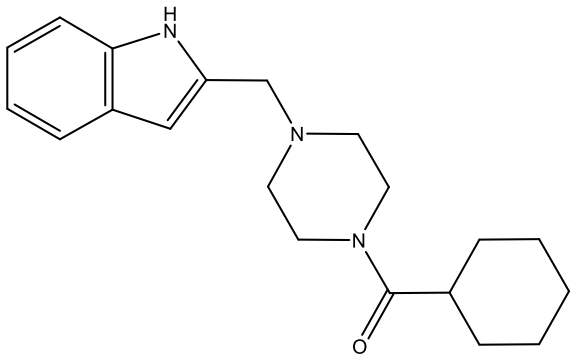
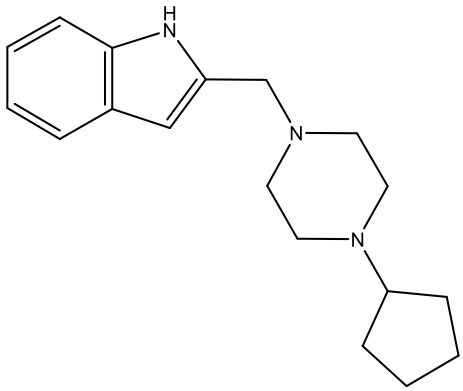
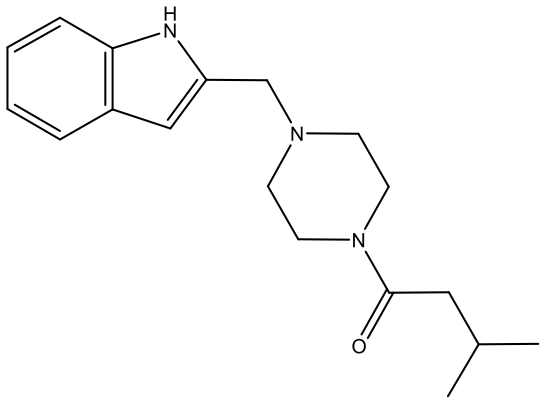
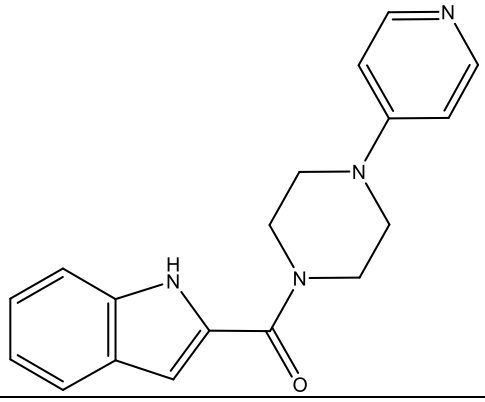
X_{10} and **X_{12}** are each independently C or N;

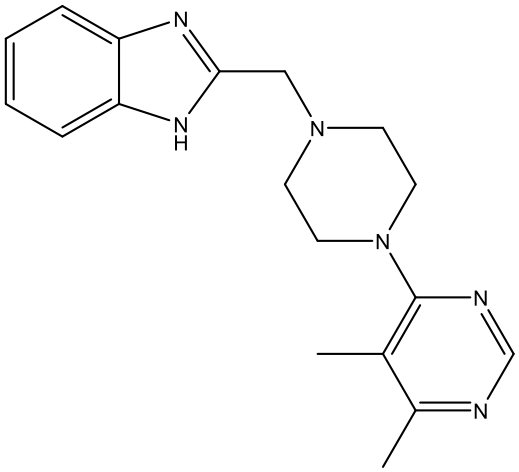
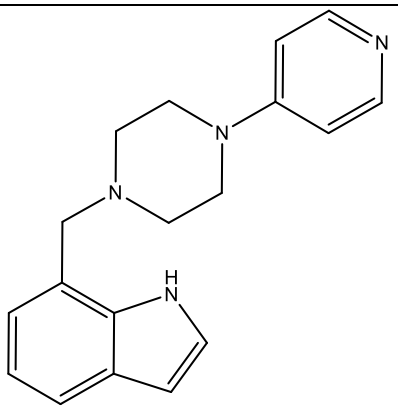
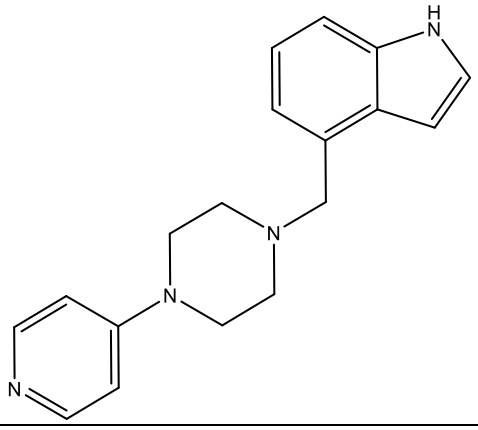
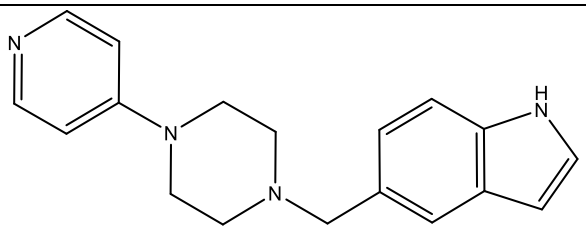
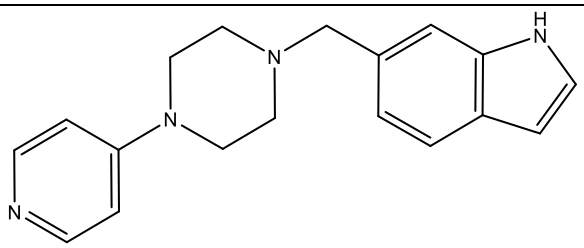
wherein if R_3 is ethyl, then R_1 or R_2 is not CF_3;

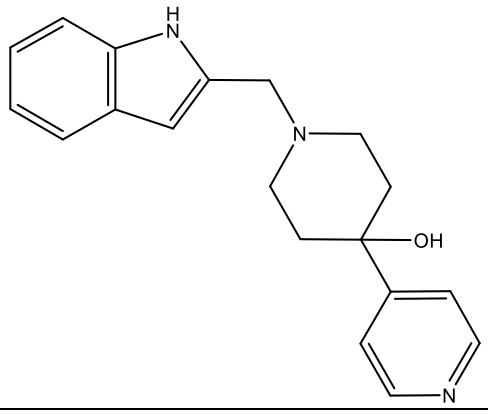
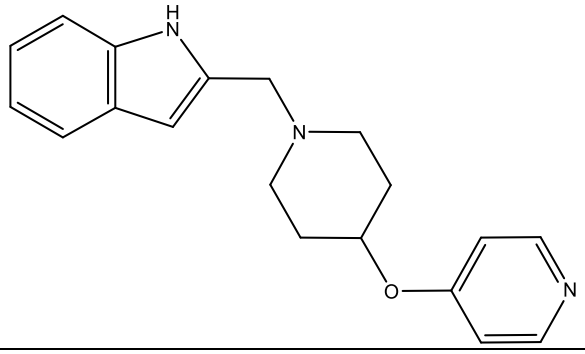
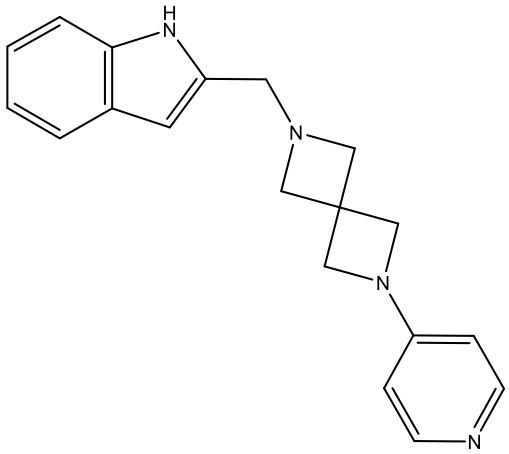
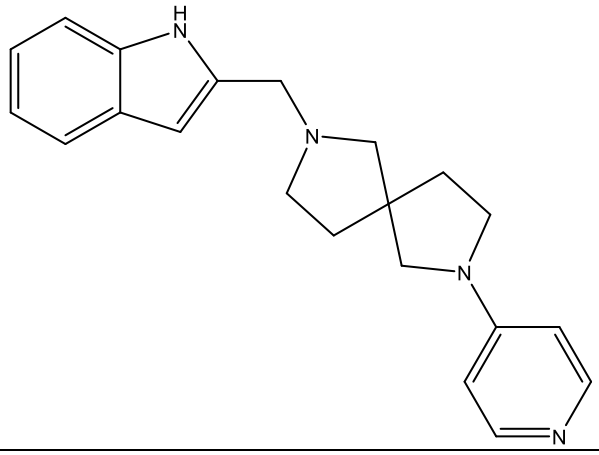
or its pharmaceutically acceptable salt, stereoisomer, tautomer, hydrate, *N*-oxide, reverse amide analog, prodrug, isotopic variant (e.g., deuterated analog), reverse amide, pharmaceutical product or any combination thereof.

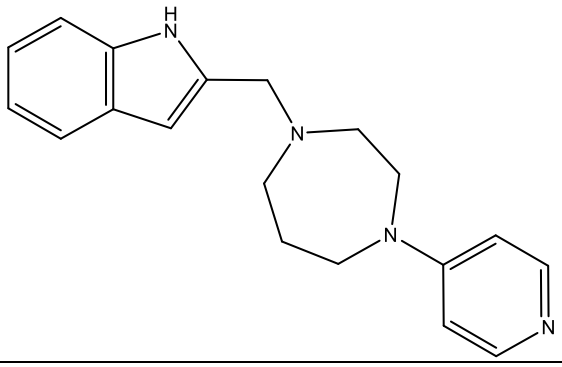
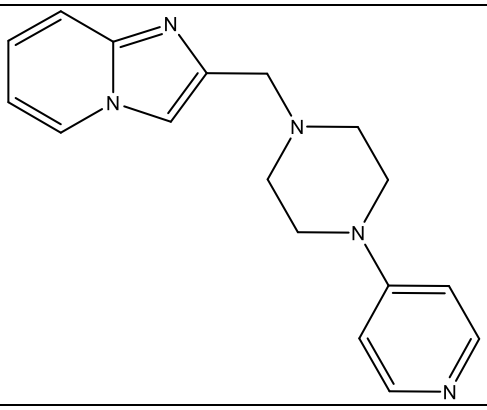
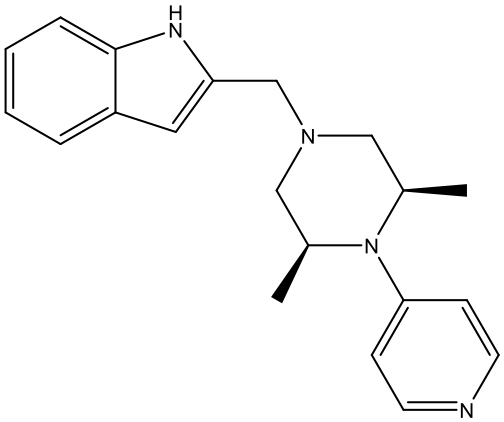
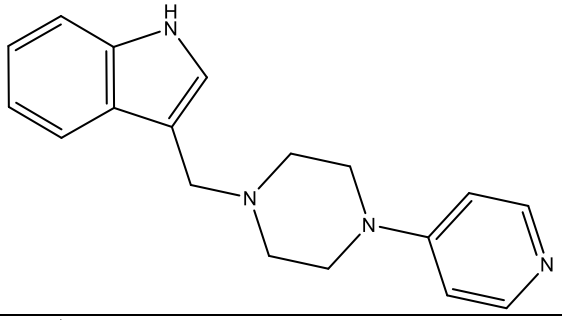
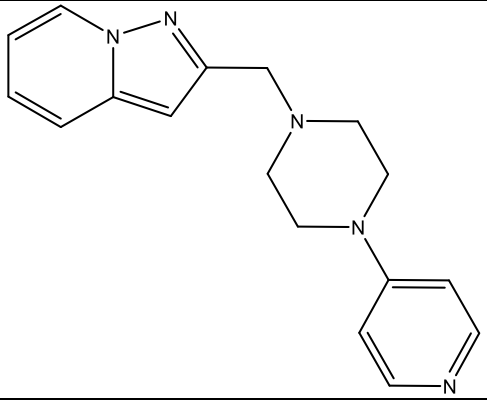
[0012b]In another aspect of the present disclosure, there is provided a compound represented by the structure of the following compounds:

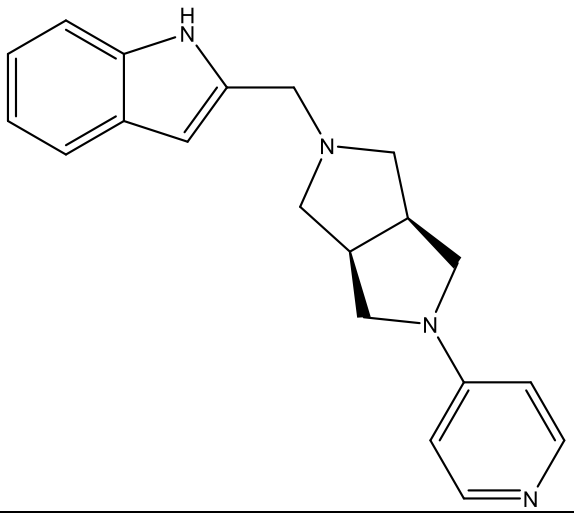
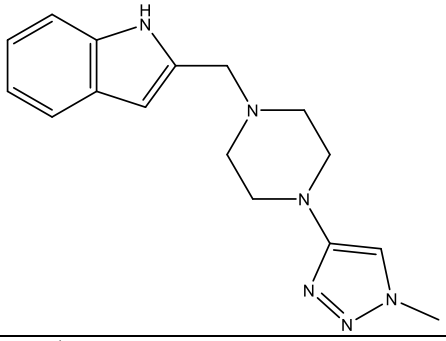
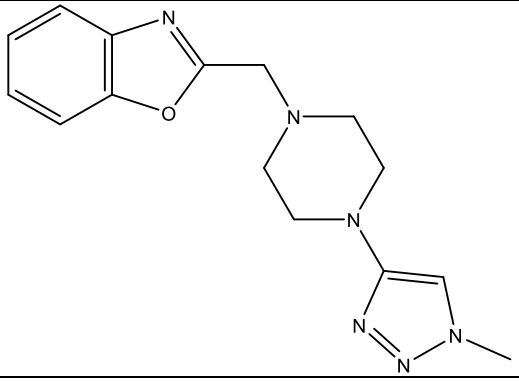
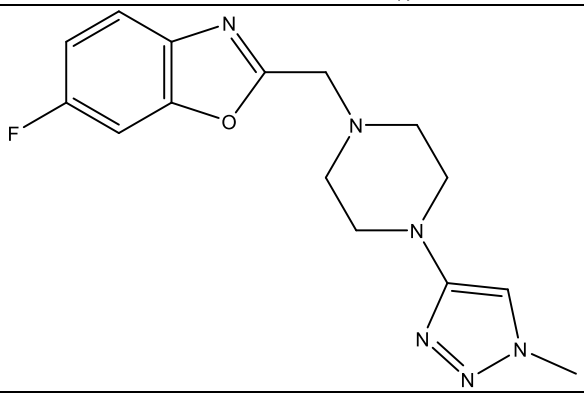
Compound Number	**Compound Structure**
218	
227	
229	
230	

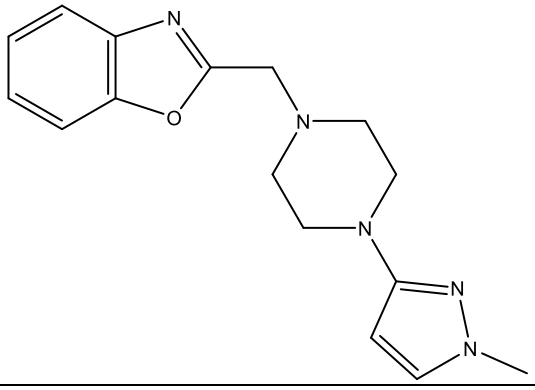
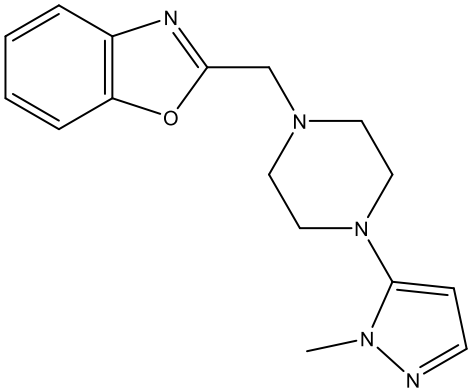
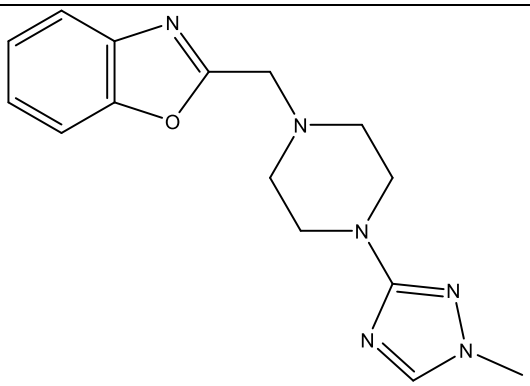
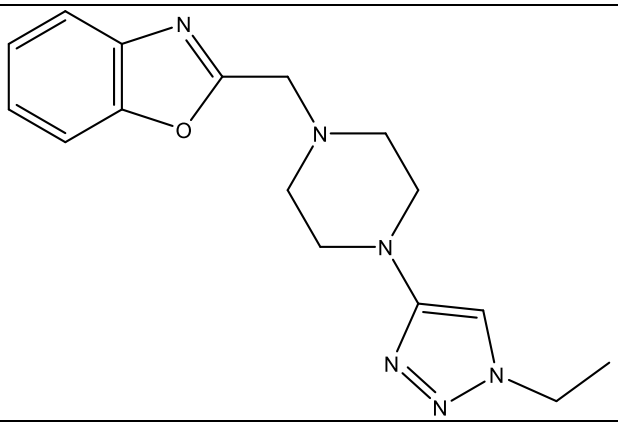
232	
233	
239	
244	

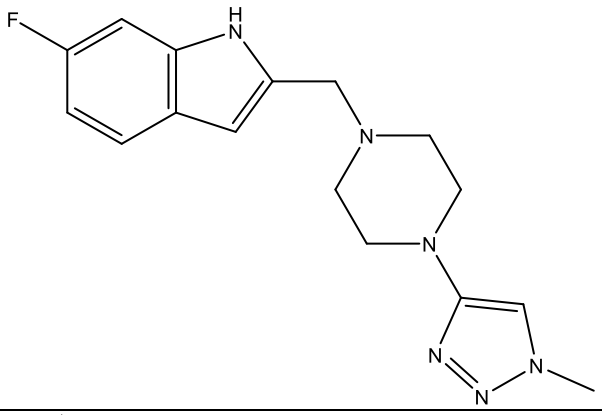
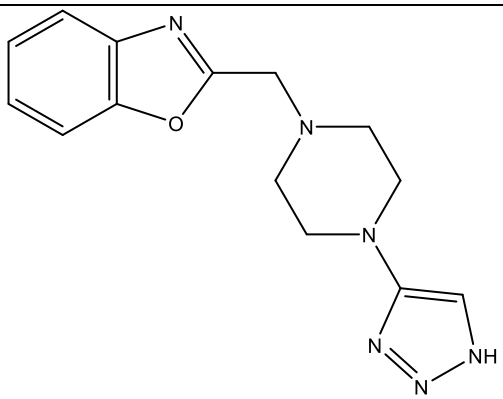
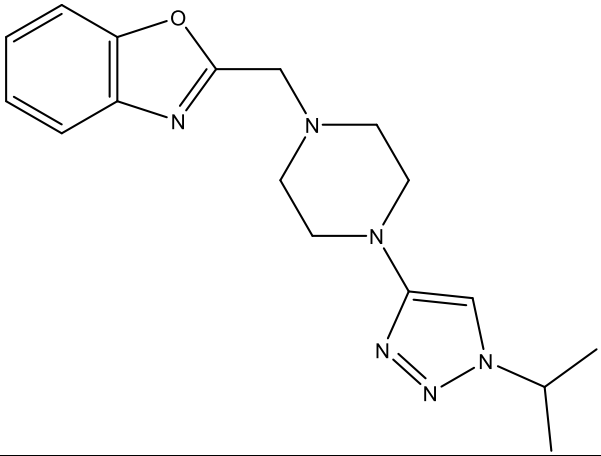
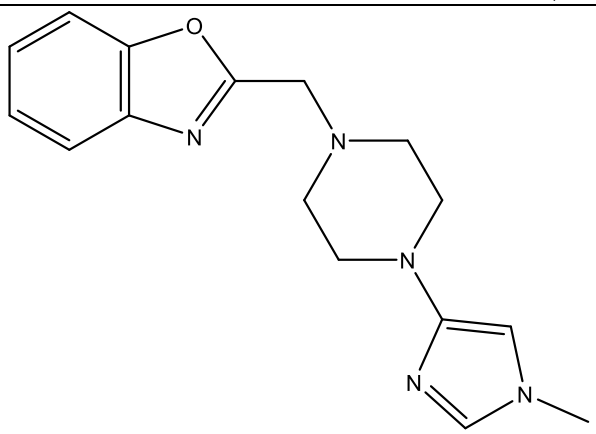
257	
262	
263	
264	
265	

267	
268	
271	
272	

273	
274	
276	
277	
283	

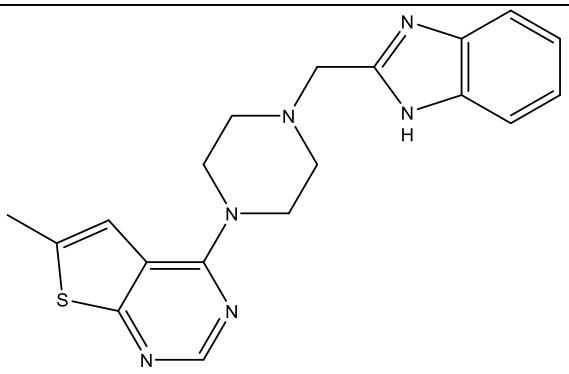
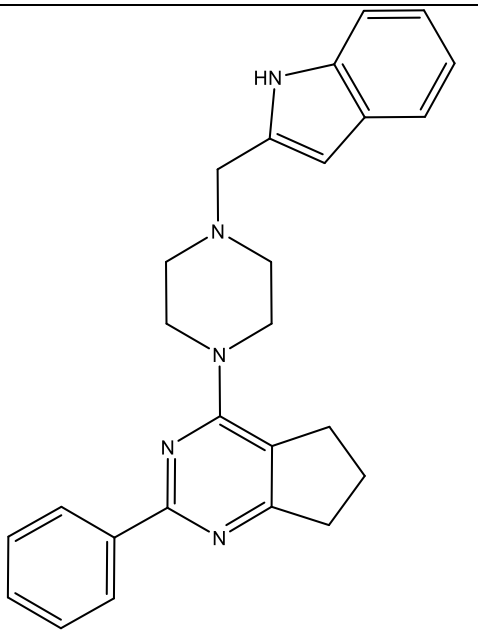
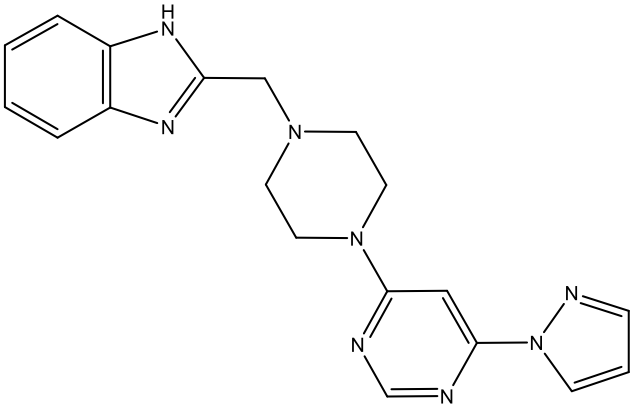
285	
288	
322	
323	

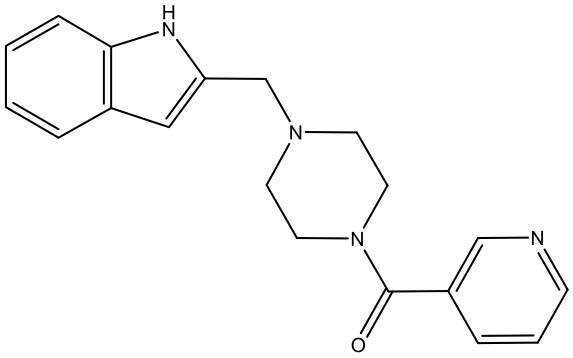
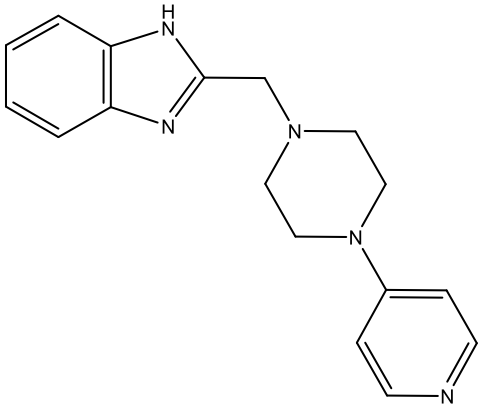
324	
325	
326	
328	

331	
332	
339	
340	

[0012c] In a further aspect of the present disclosure, there is provided a pharmaceutical composition comprising a compound disclosed herein and a pharmaceutically acceptable carrier.

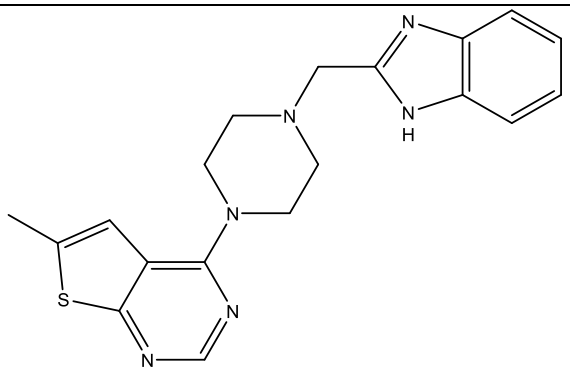
[0012d] In another aspect of the present disclosure, there is provided a use of a compound disclosed herein; or a compound represented by any one of the following structures:

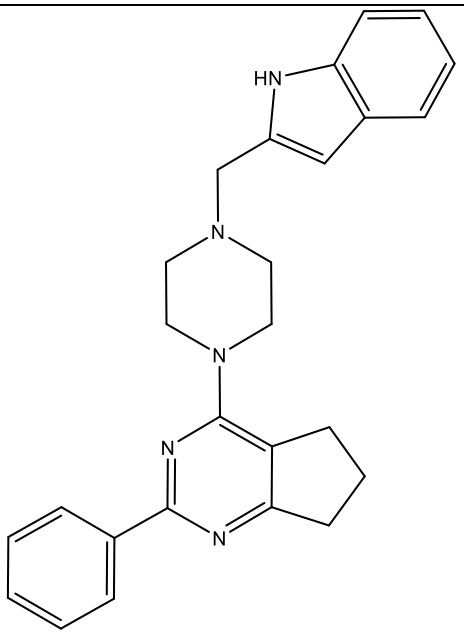
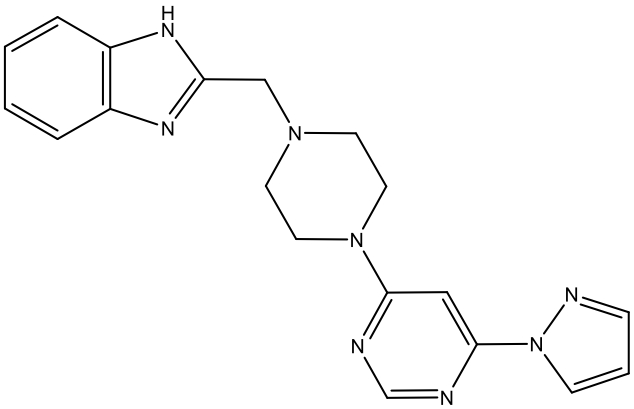
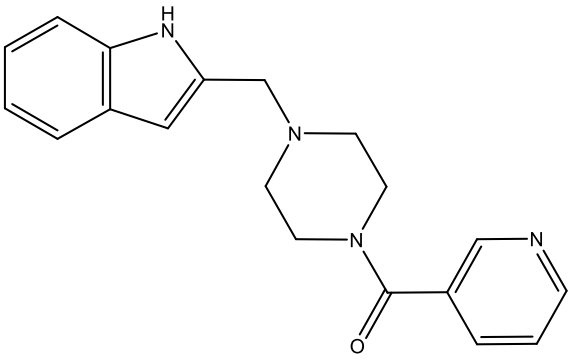
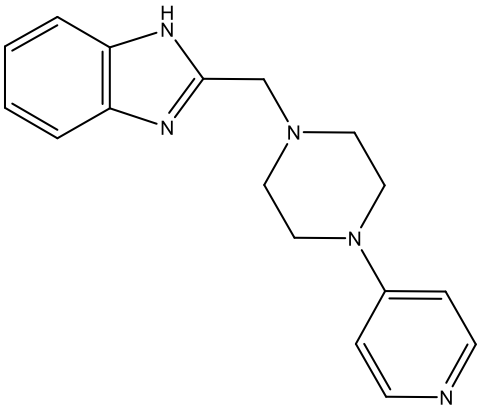
Compound Number	**Compound Structure**
205	
207	
208	

231	
253	

in the manufacture of a medicament for treating, suppressing, reducing the severity, reducing the risk of developing or inhibiting fibrosis in a subject.

[0012e]In a further aspect of the present disclosure, there is provided a use of a compound disclosed herein or a compound represented by any one of the following structures:

Compound Number	**Compound Structure**
205	

207	
208	
231	
253	

in the manufacture of a medicament for:

treating, suppressing, reducing the severity, reducing the risk of developing or inhibiting lung fibrosis in a subject;

treating, suppressing, reducing the severity, reducing the risk of developing or inhibiting idiopathic pulmonary fibrosis (IPF) in a subject

treating, suppressing, reducing the severity, reducing the risk of developing or inhibiting hepato-fibrotic disorder in a subject;

treating, suppressing, reducing the severity, reducing the risk of developing or inhibiting cirrhosis in a subject;

treating, suppressing, reducing the severity, reducing the risk of developing or inhibiting alcoholic steatohepatitis (ASH) in a subject;

treating, suppressing, reducing the severity, reducing the risk of developing or inhibiting non-alcoholic steatohepatitis (NASH) in a subject;

treating, suppressing, reducing the severity, reducing the risk of developing or inhibiting alcoholic fatty liver disease (AFLD) in a subject;

treating, suppressing, reducing the severity, reducing the risk of developing or inhibiting non alcoholic fatty liver disease (NAFLD) in a subject;

treating, suppressing, reducing the severity, reducing the risk of developing or inhibiting an autoimmune disease or disorder in a subject;

or any combination thereof.

[0012f] In another aspect of the present disclosure, there is provided a compound disclosed herein for use in therapy.

[0013] This disclosure provides a compound or its pharmaceutically acceptable salt, stereoisomer, tautomer, hydrate, *N*-oxide, reverse amide analog, prodrug, isotopic variants (e.g., deuterated analog), pharmaceutical product or any combination thereof, represented by the structure of formula **I-X** and by the structures listed in Table 1, as defined herein below. In various examples, the compound is Collagen I translation inhibitor.

[0014] This disclosure further provides a pharmaceutical composition comprising a compound or its pharmaceutically acceptable salt, stereoisomer, tautomer, hydrate, *N*-oxide, prodrug, isotopic variants (e.g., deuterated analog), pharmaceutical product or any combination thereof, represented by the structure of formula **I-X** and by the structures listed in Table 1, as defined herein below, and a pharmaceutically acceptable carrier.

[0015] This disclosure further provides a method of treating, suppressing, reducing the severity, reducing the risk of developing or inhibiting fibrosis in a subject, comprising administering a compound represented by the structure of formula **I-X** and by the structures listed in Table 1, as defined herein below, to a subject suffering from fibrosis under conditions effective to treat, suppress, reduce the severity, reduce the risk of developing, or inhibit fibrosis in said subject. In some examples, the fibrosis is a systemic fibrotic disease. In some examples, the systemic fibrotic disease is systemic sclerosis, multifocal fibrosclerosis (IgG4-associated fibrosis), nephrogenic systemic fibrosis, sclerodermatous graft vs. host disease, or any combination thereof. In some examples, the fibrosis is an organ-specific fibrotic disease. In some examples, the organ-specific fibrotic disease is lung

fibrosis, cardiac fibrosis, kidney fibrosis, pulmonary fibrosis, liver and portal vein fibrosis, radiation-induced fibrosis, bladder fibrosis, intestinal fibrosis, peritoneal sclerosis, diffuse fasciitis, wound healing, scaring, or any combination thereof. In some examples, the lung fibrosis is idiopathic pulmonary fibrosis (IPF). In some examples, the cardiac fibrosis is hypertension-associated cardiac fibrosis, Post-myocardial infarction, Chagas disease-induced myocardial fibrosis or any combination thereof. In some examples, the kidney fibrosis is diabetic and hypertensive nephropathy, urinary tract obstruction-induced kidney fibrosis, inflammatory/autoimmune-induced kidney fibrosis, aristolochic acid nephropathy, polycystic kidney disease, or any combination thereof. In some examples, the pulmonary fibrosis is idiopathic pulmonary fibrosis, silica-induced pneumoconiosis (silicosis), asbestos-induced pulmonary fibrosis (asbestosis), chemotherapeutic agent-induced pulmonary fibrosis, or any combination thereof. In some examples, the liver and portal vein fibrosis is alcoholic and nonalcoholic liver fibrosis, hepatitis C-induced liver fibrosis, primary biliary cirrhosis, parasite-induced liver fibrosis (schistosomiasis), or any combination thereof. In some examples, the diffuse fasciitis is localized scleroderma, keloids, dupuytren's disease, peyronie's disease, myelofibrosis, oral submucous fibrosis, or any combination thereof. In some examples, the fibrosis is primary or secondary fibrosis. In some examples, the fibrosis is a result of systemic sclerosis, graft-versus host disease (GVHD), pulmonary fibrosis, autoimmune disorder, tissue injury, inflammation, oxidative stress or any combination thereof. In some examples, the fibrosis is hepatic fibrosis, lung fibrosis or dermal fibrosis. In some examples, the subject has a liver cirrhosis. In some examples, the dermal fibrosis is scleroderma. In some examples, the dermal fibrosis is a result of a localized or generalized morphea, keloids, hypertrophic scars, familial cutaneous collagenoma, connective tissue nevi of the collagen type, or any combination thereof. In some examples, the hepatic fibrosis is a result of hepatic scarring or chronic liver injury. In some examples, the chronic liver injury results from alcoholism, malnutrition, hemochromatosis, exposure to poisons, toxins or drugs.

[0016] This disclosure further provides a method of treating, suppressing, reducing the severity, reducing the risk of developing or inhibiting lung fibrosis in a subject, comprising administering a compound represented by the structure of formula **I-X** and by the structures listed in Table 1, as defined herein below, to a subject suffering from lung fibrosis under conditions effective to treat, suppress, reduce the severity, reduce the risk of developing, or inhibit lung fibrosis in said subject. In some examples, the lung fibrosis is idiopathic pulmonary fibrosis (IPF).

[0017] This disclosure further provides a method of treating, suppressing, reducing the severity, reducing the risk of developing or inhibiting idiopathic pulmonary fibrosis (IPF) in a subject, comprising administering a compound represented by the structure of formula **I-X** and by the structures listed in Table 1, as defined herein below, to a subject suffering from idiopathic pulmonary fibrosis (IPF) under conditions effective to treat, suppress, reduce the severity, reduce the risk of developing, or inhibit idiopathic pulmonary fibrosis (IPF) in said subject.

[0018] This disclosure further provides a method of treating, suppressing, reducing the severity, reducing the risk of developing or inhibiting hepato-fibrotic disorder in a subject, comprising administering a compound represented by the structure of formula **I-X** and by the structures listed in Table 1, as defined herein below, to a subject suffering from hepato-fibrotic disorder under conditions effective to treat, suppress, reduce the severity, reduce the risk of developing, or inhibit hepato-fibrotic disorder in said subject. In some examples, the hepato-fibrotic disorder is a portal hypertension, cirrhosis, congenital hepatic fibrosis or any combination thereof.

[0019] This disclosure further provides a method of treating, suppressing, reducing the severity, reducing the risk of developing or inhibiting cirrhosis in a subject, comprising administering a compound represented by the structure of formula **I-X** and by the structures listed in Table 1, as defined herein below, to a subject suffering from cirrhosis under conditions effective to treat, suppress, reduce the severity, reduce the risk of developing, or inhibit cirrhosis in said subject. In some examples, the cirrhosis is a result of hepatitis or alcoholism.

[0020] This disclosure further provides a method of treating, suppressing, reducing the severity, reducing the risk of developing or inhibiting a alcoholic steatohepatitis (ASH) in a subject, comprising administering a compound represented by the structure of formula **I-X** and by the structures listed in Table 1, as defined herein below, to a subject suffering from alcoholic steatohepatitis (ASH) under conditions effective to treat, suppress, reduce the severity, reduce the risk of developing, or inhibit the alcoholic steatohepatitis (ASH) in said subject.

[0021] This disclosure further provides a method of treating, suppressing, reducing the severity, reducing the risk of developing or inhibiting a non-alcoholic steatohepatitis (NASH) in a subject, comprising administering a compound represented by the structure of formula **I-X** and by the structures listed in Table 1, as defined herein below, to a subject suffering from non-alcoholic steatohepatitis (NASH) under conditions effective to treat, suppress, reduce the severity, reduce the risk of developing, or inhibit the non-alcoholic steatohepatitis (NASH) in said subject.

[0022] This disclosure further provides a method of treating, suppressing, reducing the severity, reducing the risk of developing or inhibiting a alcoholic fatty liver disease (AFLD) in a subject, comprising administering a compound represented by the structure of formula **I-X** and by the structures listed in Table 1, as defined herein below, to a subject suffering from alcoholic fatty liver disease (AFLD) under conditions effective to treat, suppress, reduce the severity, reduce the risk of developing, or inhibit the alcoholic fatty liver disease (AFLD) in said subject.

[0023] This disclosure further provides a method of treating, suppressing, reducing the severity, reducing the risk of developing or inhibiting a non alcoholic fatty liver disease (NAFLD) in a subject, comprising administering a compound represented by the structure of formula **I-X** and by the structures listed in Table 1, as defined herein below, to a subject suffering from non alcoholic fatty liver disease (NAFLD) under conditions effective to treat, suppress, reduce the severity, reduce the risk of developing, or inhibit the non alcoholic fatty liver disease (NAFLD) in said subject.

[0024] This disclosure further provides a method of treating, suppressing, reducing the severity, reducing the risk of developing or inhibiting an autoimmune disease or disorder in a subject, comprising administering a compound represented by the structure of formula **I-X** and by the structures listed in Table 1, as defined herein below, to a subject suffering from an autoimmune disease or disorder under conditions effective to treat, suppress, reduce the severity, reduce the risk of developing, or inhibit the autoimmune disease or disorder in said subject.

[0025] This disclosure further provides a method of treating, suppressing, reducing the severity, reducing the risk of developing or inhibiting an autoimmune disease or disorder in a subject, comprising administering a compound represented by the structure of formula **I-X** and by the structures listed in Table 1, as defined herein below, to a subject suffering from an autoimmune disease or disorder under conditions effective to treat, suppress, reduce the severity, reduce the risk of developing, or inhibit the autoimmune disease or disorder in said subject.

[0026] This disclosure provides a compound or its pharmaceutically acceptable salt, stereoisomer, tautomer, hydrate, *N*-oxide, reverse amide analog, prodrug, isotopic variants (e.g., deuterated analog), pharmaceutical product or any combination thereof, represented by the structure of formula **I-X** and by the structures listed in Table 1, as defined herein below. In various examples, the compound is Collagen I translation inhibitor.

[0027] This disclosure further provides a pharmaceutical composition comprising a compound or its pharmaceutically acceptable salt, stereoisomer, tautomer, hydrate, *N*-oxide, prodrug, isotopic variants (e.g., deuterated analog), pharmaceutical product or any combination thereof, represented by the structure of formula **I-X** and by the structures listed in Table 1, as defined herein below, and a pharmaceutically acceptable carrier.

[0028] This disclosure further provides a method of treating, suppressing, reducing the severity, reducing the risk of developing or inhibiting fibrosis in a subject, comprising administering a compound represented by the structure of formula **I-X** and by the structures listed in Table 1, as defined herein below, to a subject suffering from fibrosis under conditions effective to treat, suppress, reduce the severity, reduce the risk of developing, or inhibit fibrosis in said subject. In some examples, the fibrosis is a systemic fibrotic disease. In some examples, the systemic fibrotic disease is systemic sclerosis, multifocal fibrosclerosis (IgG4-associated fibrosis), nephrogenic systemic fibrosis, sclerodermatous graft vs. host disease, or any combination thereof. In some examples, the fibrosis is an organ-specific fibrotic disease. In some examples, the organ-specific fibrotic disease is lung fibrosis, cardiac fibrosis, kidney fibrosis, pulmonary fibrosis, liver and portal vein fibrosis, radiation-induced fibrosis, bladder fibrosis, intestinal fibrosis, peritoneal sclerosis, diffuse fasciitis, wound healing, scaring, or any combination thereof. In some examples, the lung fibrosis is idiopathic pulmonary fibrosis (IPF). In some examples, the cardiac fibrosis is hypertension-associated cardiac fibrosis, Post-myocardial infarction, Chagas disease-induced myocardial fibrosis or any combination thereof. In some examples, the kidney fibrosis is diabetic and hypertensive nephropathy, urinary tract obstruction-induced kidney fibrosis, inflammatory/autoimmune-induced kidney fibrosis,

aristolochic acid nephropathy, polycystic kidney disease, or any combination thereof. In some examples, the pulmonary fibrosis is idiopathic pulmonary fibrosis, silica-induced pneumoconiosis (silicosis), asbestos-induced pulmonary fibrosis (asbestosis), chemotherapeutic agent-induced pulmonary fibrosis, or any combination thereof. In some examples, the liver and portal vein fibrosis is alcoholic and nonalcoholic liver fibrosis, hepatitis C-induced liver fibrosis, primary biliary cirrhosis, parasite-induced liver fibrosis (schistosomiasis), or any combination thereof. In some examples, the diffuse fasciitis is localized scleroderma, keloids, dupuytren's disease, peyronie's disease, myelofibrosis, oral submucous fibrosis, or any combination thereof. In some examples, the fibrosis is primary or secondary fibrosis. In some examples, the fibrosis is a result of systemic sclerosis, graft-versus host disease (GVHD), pulmonary fibrosis, autoimmune disorder, tissue injury, inflammation, oxidative stress or any combination thereof. In some examples, the fibrosis is hepatic fibrosis, lung fibrosis or dermal fibrosis. In some examples, the subject has a liver cirrhosis. In some examples, the dermal fibrosis is scleroderma. In some examples, the dermal fibrosis is a result of a localized or generalized morphea, keloids, hypertrophic scars, familial cutaneous collagenoma, connective tissue nevi of the collagen type, or any combination thereof. In some examples, the hepatic fibrosis is a result of hepatic scarring or chronic liver injury. In some examples, the chronic liver injury results from alcoholism, malnutrition, hemochromatosis, exposure to poisons, toxins or drugs.

[0029] This disclosure further provides a method of treating, suppressing, reducing the severity, reducing the risk of developing or inhibiting lung fibrosis in a subject, comprising administering a compound represented by the structure of formula **I-X** and by the structures listed in Table 1, as defined herein below, to a subject suffering from lung fibrosis under conditions effective to treat, suppress, reduce the severity, reduce the risk of developing, or inhibit lung fibrosis in said subject. In some examples, the lung fibrosis is idiopathic pulmonary fibrosis (IPF).

[0030] This disclosure further provides a method of treating, suppressing, reducing the severity, reducing the risk of developing or inhibiting idiopathic pulmonary fibrosis (IPF) in a subject, comprising administering a compound represented by the structure of formula **I-X** and by the structures listed in Table 1, as defined herein below, to a subject suffering from idiopathic pulmonary fibrosis (IPF) under conditions effective to treat, suppress, reduce the severity, reduce the risk of developing, or inhibit idiopathic pulmonary fibrosis (IPF) in said subject.

[0031] This disclosure further provides a method of treating, suppressing, reducing the severity, reducing the risk of developing or inhibiting hepato-fibrotic disorder in a subject, comprising administering a compound represented by the structure of formula **I-X** and by the structures listed in Table 1, as defined herein below, to a subject suffering from hepato-fibrotic disorder under conditions effective to treat, suppress, reduce the severity, reduce the risk of developing, or inhibit hepato-fibrotic disorder in said subject. In some examples, the hepato-fibrotic disorder is a portal hypertension, cirrhosis, congenital hepatic fibrosis or any combination thereof.

[0032] This disclosure further provides a method of treating, suppressing, reducing the severity, reducing the risk of developing or inhibiting cirrhosis in a subject, comprising administering a compound represented by the structure of formula **I-X** and by the structures listed in Table 1, as defined herein below, to a subject suffering from cirrhosis under conditions effective to treat, suppress, reduce the severity, reduce the risk of developing, or inhibit cirrhosis in said subject. In some examples, the cirrhosis is a result of hepatitis or alcoholism.

[0033] This disclosure further provides a method of treating, suppressing, reducing the severity, reducing the risk of developing or inhibiting a alcoholic steatohepatitis (ASH) in a subject, comprising administering a compound represented by the structure of formula **I-X** and by the structures listed in Table 1, as defined herein below, to a subject suffering from alcoholic steatohepatitis (ASH) under conditions effective to treat, suppress, reduce the severity, reduce the risk of developing, or inhibit the alcoholic steatohepatitis (ASH) in said subject.

[0034] This disclosure further provides a method of treating, suppressing, reducing the severity, reducing the risk of developing or inhibiting a non-alcoholic steatohepatitis (NASH) in a subject, comprising administering a compound represented by the structure of formula **I-X** and by the structures listed in Table 1, as defined herein below, to a subject suffering from non-alcoholic steatohepatitis (NASH) under conditions effective to treat, suppress, reduce the severity, reduce the risk of developing, or inhibit the non-alcoholic steatohepatitis (NASH) in said subject.

[0035] This disclosure further provides a method of treating, suppressing, reducing the severity, reducing the risk of developing or inhibiting a alcoholic fatty liver disease (AFLD) in a subject, comprising administering a compound represented by the structure of formula **I-X** and by the structures listed in Table 1, as defined herein below, to a subject suffering from alcoholic fatty liver disease (AFLD) under conditions effective to treat, suppress, reduce the severity, reduce the risk of developing, or inhibit the alcoholic fatty liver disease (AFLD) in said subject.

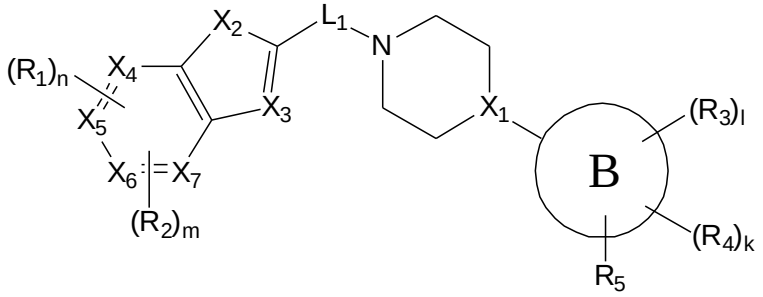
[0036] This disclosure further provides a method of treating, suppressing, reducing the severity, reducing the risk of developing or inhibiting a non alcoholic fatty liver disease (NAFLD) in a subject, comprising administering a compound represented by the structure of formula **I-X** and by the structures listed in Table 1, as defined herein below, to a subject suffering from non alcoholic fatty liver disease (NAFLD) under conditions effective to treat, suppress, reduce the severity, reduce the risk of developing, or inhibit the non alcoholic fatty liver disease (NAFLD) in said subject.

[0037] This disclosure further provides a method of treating, suppressing, reducing the severity, reducing the risk of developing or inhibiting an autoimmune disease or disorder in a subject, comprising administering a compound represented by the structure of formula **I-X** and by the structures listed in Table 1, as defined herein below, to a subject suffering from an autoimmune disease or disorder under conditions effective to treat, suppress, reduce the severity, reduce the risk of developing, or inhibit the autoimmune disease or disorder in said subject.

[0038] This disclosure further provides amethod of treating, suppressing, reducing the severity, reducing the risk of developing or inhibiting an autoimmune disease or disorder in a subject, comprising

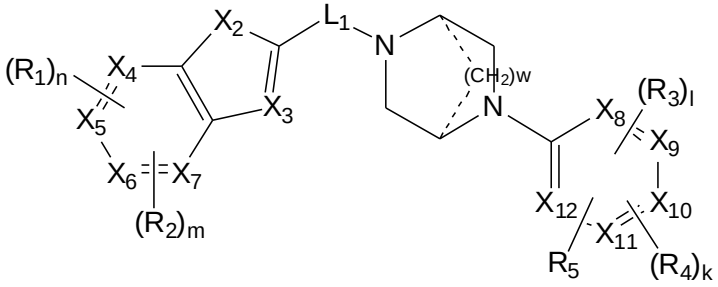
WHAT IS CLAIMED:

1. A compound represented by the structure of formula **III**:



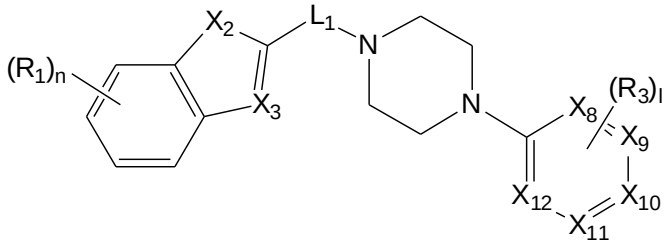
III;

by the structure of formula **V(a)**:



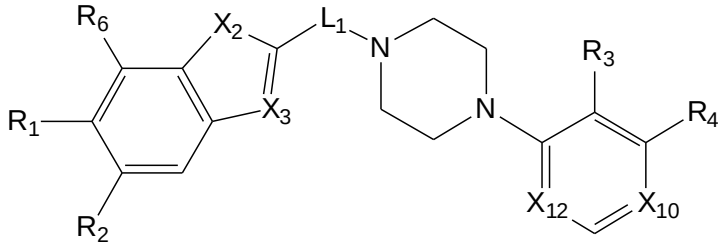
V(a);

by the structure of formula **VI**:



VI;

or by the structure of formula **VII**:



VII;

wherein

B ring is a single or fused aromatic or heteroaromatic ring system (e.g., phenyl, pyrimidine, 2-, 3- or 4-pyridine, pyridazine or pyrazine, thiazole, pyrrole, triazole, imidazole, indazole);

X_1 is N or C(R) (e.g., C-H, C-OH);

L_1 is CH_2, CHR, or $C(R)_2$;

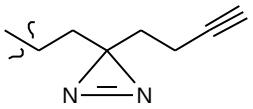
R_1, **R_2** and **R_6** are each independently H, F, Cl, Br, I, OH, SH, R_8-OH, R_8-SH, -R_8-O-R_{10} (e.g., CH_2-CH_2-O-CH_3, CH_2-O-CH_2-CH_2-O-CH_3), -O-R_8-O-R_{10} (e.g., O-CH_2-CH_2-O-CH_3), R_8-(C_3-C_8 cycloalkyl), R_8-(C_3-C_8 heterocyclic ring), O-**R_{20}**, CF_3, CD_3, OCD_3, CN, NO_2, -CH_2CN, -R_8CN, NH_2, NHR, N(R)$_2$, R_8-N(R_{10})(R_{11}), R_9-R_8-N(R_{10})(R_{11}), B(OH)$_2$, -OC(O)CF_3, -OCH_2Ph, NHC(O)-R (e.g., NHCO-Ph; NHCO-CH_3) , NHC(O)-R_{10} (e.g., NHCO-CH_3) NHCO-N(R_{10})(R_{11}), -C(O)Ph, C(O)O-R_{10}, R_8-C(O)-R_{10}, C(O)H, C(O)-R_{10}, C_1-C_5 linear or branched C(O)-haloalkyl, -C(O)NH_2, C(O)NHR (e.g., C(O)NH-Ph), C(O)N(R_{10})(R_{11}), SO_2R, SO_2N(R_{10})(R_{11}), $NHSO_2$(R_{10}) (e.g., $NHSO_2CH_3$), $CH(CF_3)$(NH-R_{10}), C_1-C_5 linear or branched, substituted or unsubstituted alkyl (e.g., methyl, ethyl), C_1-C_5 linear or branched, substituted or unsubstituted alkenyl, C_1-C_5 linear, branched or cyclic haloalkyl (e.g., CHF_2), C_1-C_5 linear, branched or cyclic alkoxy (e.g. methoxy), optionally wherein at least one methylene group (CH_2) in the alkoxy is replaced with an oxygen atom, C_1-C_5 linear or branched thioalkoxy, C_1-C_5 linear or branched haloalkoxy, C_1-C_5 linear or branched alkoxyalkyl, substituted or unsubstituted C_3-C_8 cycloalkyl (e.g., cyclopropyl), substituted or unsubstituted C_3-C_8 heterocyclic ring (e.g., pyridine), substituted or unsubstituted aryl (e.g., phenyl), substituted or unsubstituted benzyl;

or **R_2** and **R_1** are joined together to form a 5 or 6 membered substituted or unsubstituted, aliphatic or aromatic, carbocyclic (e.g., benzene) or heterocyclic (e.g., pyridine) ring;

R_3, **R_4** and **R_5** are each independently H, F, Cl, Br, I, OH, SH, R_8-OH, R_8-SH, -R_8-O-R_{10} (e.g., CH_2-CH_2-O-CH_3, CH_2-O-CH_2-CH_2-O-CH_3), R_8-(C_3-C_8 cycloalkyl), R_8-(C_3-C_8 heterocyclic ring), O-**R_{20}**, CF_3, CD_3, OCD_3, CN, NO_2, -CH_2CN, -R_8CN, NH_2, NHR, N(R)$_2$, R_8-N(R_{10})(R_{11}), R_9-R_8-N(R_{10})(R_{11}), B(OH)$_2$, -OC(O)CF_3, -OCH_2Ph, NHC(O)-R_{10}, NHCO-N(R_{10})(R_{11}), COOH, -C(O)Ph, C(O)O-R_{10}, R_8-C(O)-R_{10}, C(O)H, C(O)-R_{10}, C_1-C_5 linear or branched C(O)-haloalkyl, -C(O)NH_2, C(O)NHR, C(O)N(R_{10})(R_{11}), SO_2R, SO_2N(R_{10})(R_{11}), $CH(CF_3)$(NH-R_{10}), C_1-C_5 linear or branched, substituted or unsubstituted alkyl (e.g., methyl, ethyl), C_1-C_5 linear or branched, substituted or unsubstituted alkenyl, C_1-C_5 linear, branched or cyclic haloalkyl (e.g., CHF_2), C_1-C_5 linear, branched or cyclic alkoxy (e.g. methoxy), optionally wherein at least one methylene group (CH_2) in the alkoxy is replaced with an oxygen atom, C_1-C_5 linear or branched thioalkoxy, C_1-C_5 linear or branched haloalkoxy, C_1-C_5 linear or branched alkoxyalkyl, substituted or unsubstituted C_3-C_8 cycloalkyl (e.g., cyclopropyl), substituted or unsubstituted C_3-C_8 heterocyclic ring (e.g., pyrazole, thiazole), substituted or unsubstituted aryl (e.g., phenyl), substituted or unsubstituted benzyl;

or **R_3** and **R_4** are joined together to form a 5 or 6 membered substituted or unsubstituted, aliphatic (e.g., cyclopentene) or aromatic, carbocyclic (e.g., benzene) or heterocyclic (e.g., thiophene, furane, pyrrol, pyrazole) ring;

R_{20} is represented by the following structure:



X_2 is NH, S, O, N-R (e.g., N-CH_2-CH_2-O-CH_3);

X_3 is N, C(R) (e.g., CH, C-CH_3, C-Cl, C-CN);

X_4, **X_5**, **X_6**, and **X_7** are each independently C or N;

X_8, **X_9**, **X_{10}**, **X_{11}**, and **X_{12}** are each independently C or N;

R is H, OH, F, Cl, Br, I, CN, CF_3, NO_2, C_1-C_5 linear or branched, substituted or unsubstituted alkyl (e.g., methyl, ethyl), C_1-C_5 linear or branched alkoxy, -R_8-O-R_{10} (e.g., CH_2-CH_2-O-CH_3), C_1-C_5 linear or branched haloalkyl (e.g., CF_3, CF_2CH_3, CH_2CF_3, $CF_2CH_2CH_3$, $CH_2CH_2CF_3$, $CF_2CH(CH_3)_2$,$CF(CH_3)$-$CH(CH_3)_2$), R_8-aryl (e.g., CH_2-Ph), substituted or unsubstituted aryl (e.g., phenyl), substituted or unsubstituted heteroaryl (e.g., pyridine (2, 3, and 4-pyridine); or

two geminal **R** substitutions are joined together to form a 3 - 6 membered substituted or unsubstituted, aliphatic (e.g., cyclopropyl, cyclopentene) or aromatic, carbocyclic (e.g., benzene) or heterocyclic (e.g., thiophene, furane, pyrrol, pyrazole) ring;

R_8 is $[\mathbf{CH_2}]_p$

wherein **p** is between 1 and 10 (e.g., 2);

R_9 is $[CH]_q$, $[C]_q$

wherein **q** is between 2 and 10;

R_{10} and **R_{11}** are each independently H, substituted or unsubstituted, C_1-C_5 linear or branched alkyl (e.g., methyl, ethyl, CH_2-CH_2-O-CH_3), C_1-C_5 linear or branched alkoxy (e.g., O-CH_3), C(O)R, or $S(O)_2R$;

or **R_{10}** and **R_{11}** are joined to form a substituted or unsubstituted C_3-C_8 heterocyclic ring (e.g., piperazine, piperidine),

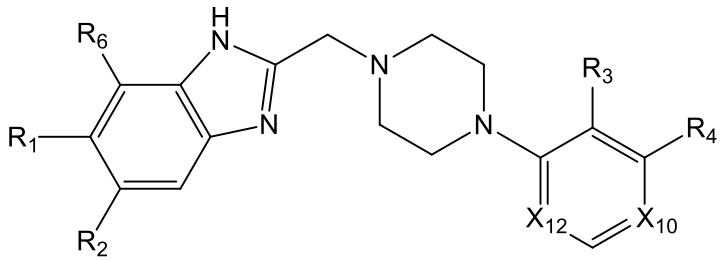
wherein substitutions include: F, Cl, Br, I, OH, SH, C_1-C_5 linear or branched alkyl, C_1-C_5 linear or branched alkyl-OH (e.g., $C(CH_3)_2CH_2$-OH, CH_2CH_2-OH), C_3-C_8 heterocyclic ring (e.g., piperidine), alkoxy, $N(R)_2$, CF_3, aryl, phenyl, halophenyl, (benzyloxy)phenyl, CN, NO_2 or any combination thereof;

m, **n, l** and **k** are each independently an integer between 0 and 4 (e.g., 0, 1 or 2);

w is 0, 1 or 2; wherein if w=0, the bridge on the ring is absent;

wherein if X_3 is N, then X_2 is not NH;

or by the structure of formula **IX**:



IX

wherein

R_1 is Cl, -R_8-O-R_{10} (e.g., CH_2-CH_2-O-CH_3, CH_2-O-CH_2-CH_2-O-CH_3), -O-R_8-O-R_{10} (e.g., O-CH_2-CH_2-O-CH_3), O-**R_{20}**, or CF_3;

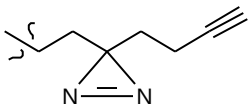
R_2 is H, Cl, -R_8-O-R_{10} (e.g., CH_2-CH_2-O-CH_3, CH_2-O-CH_2-CH_2-O-CH_3), -O-R_8-O-R_{10} (e.g., O-CH_2-CH_2-O-CH_3), O-**R_{20}**, or CF_3;

R_6 is H;

R_3 and **R_4** are each independently H, O-**R_{20}**, C_1-C_5 linear or branched, substituted or unsubstituted alkyl (e.g., methyl, ethyl), substituted or unsubstituted C_3-C_8 heterocyclic ring (e.g., imidazole), (wherein substitutions include: F, Cl, Br, I, C_1-C_5 linear or branched alkyl, OH, alkoxy, CF_3, aryl, phenyl, heteroaryl, C_3-C_8 cycloalkyl, halophenyl, (benzyloxy)phenyl, CN, NO_2 or any combination thereof);

or **R_3** and **R_4** are joined together to form a 5 or 6 membered substituted or unsubstituted, aliphatic (e.g., cyclopentene) or aromatic, carbocyclic (e.g., benzene) or heterocyclic (e.g., thiophene, furane, pyrrol, pyrazole) ring;

R_{20} is represented by the following structure:



X_{10} and **X_{12}** are each independently C or N;

wherein if R_3 is ethyl, then R_1 or R_2 is not CF_3;

or its pharmaceutically acceptable salt, stereoisomer, tautomer, hydrate, *N*-oxide, reverse amide analog, prodrug, isotopic variant (e.g., deuterated analog), reverse amide, pharmaceutical product or any combination thereof.

2. The compound according to claim 1,

wherein at least one of **X_8**, **X_9**, **X_{10}**, **X_{11}**, and **X_{12}** is N;

wherein **X_6** is C or N;

wherein **R_3** is H, Cl, F, OH, CF_3, CHF_2, CN, C(O)H, O-**R_{20}**, CH_3, C_2H_5, -R_8-O-R_{10}, CH_2-O-CH_2-CH_2-O-CH_3, -O-R_8-O-R_{10}, O-CH_2-CH_2-O-CH_3, NH_2, C_1-C_5 linear or branched, substituted or unsubstituted alkyl or methyl.

wherein **R_1** is H, Cl, F, OH, CN, NH_2, NHR, $N(R)_2$, -C(O)NH_2, C(O)NHR, C(O)N(R_{10})(R_{11}), C_1-C_5 linear or branched, substituted or unsubstituted alkyl, C_1-C_5 linear, branched or cyclic haloalkyl, CHF_2, C_1-C_5 linear, branched or cyclic alkoxy, methoxy, C_1-C_5 linear or branched haloalkoxy, substituted or unsubstituted C_3-C_8 cycloalkyl, substituted or unsubstituted C_3-C_8 heterocyclic ring, NH(CO)-Ph, -R_8-O-R_{10}, CH_2-O-CH_2-CH_2-O-CH_3, -O-R_8-O-R_{10}, O-CH_2-CH_2-O-CH_3, O-**R_{20}** or CF_3;

wherein **X_2** is NH or O and **X_3** is N, CH or C-Cl;

wherein **L_1** is CH_2 and/or w is 1 or 0;

wherein l is 1 or 2;

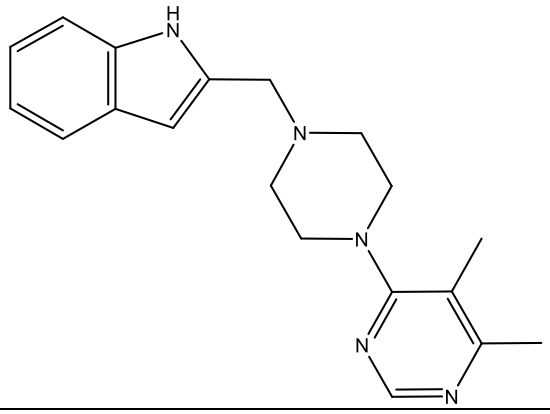
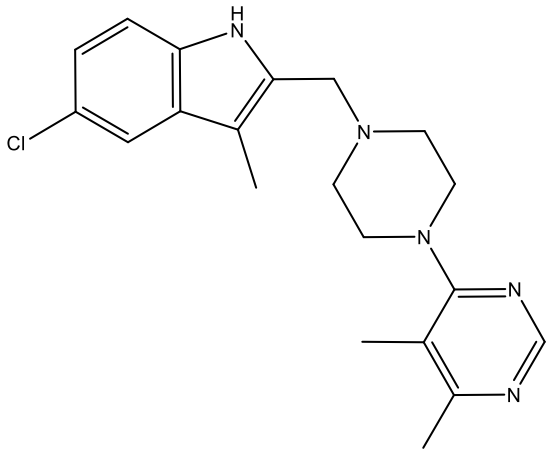
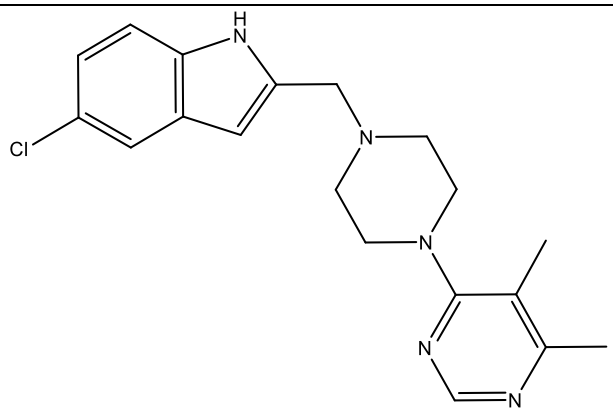
or any combination thereof.

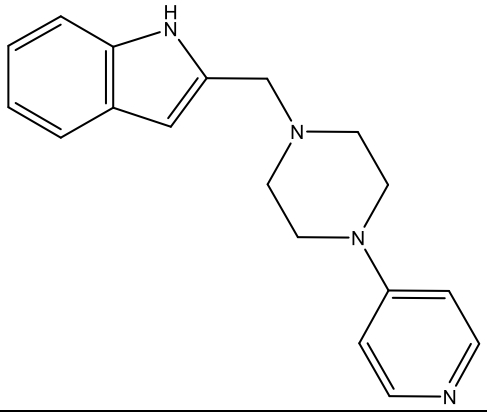
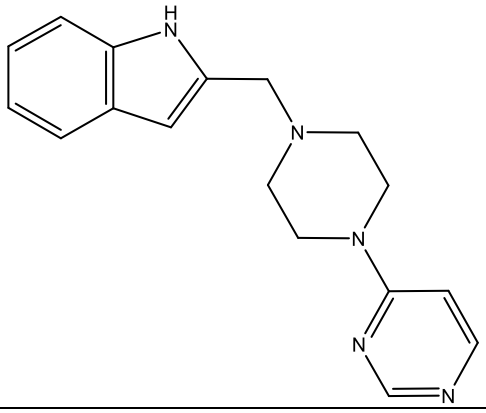
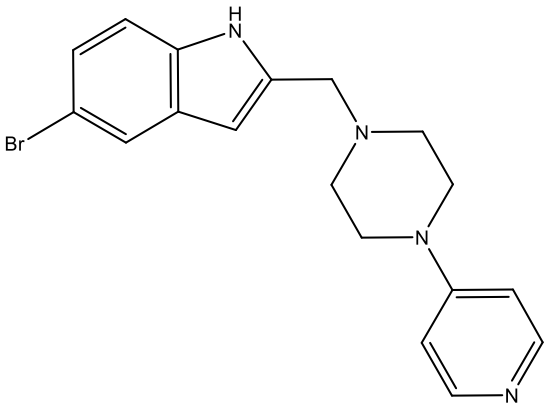
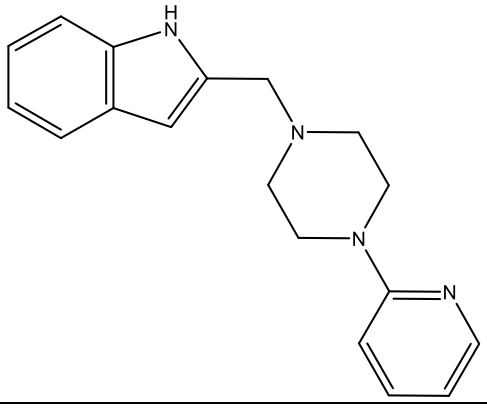
3. The compound of any one of the preceding claims, wherein the heterocyclic ring of **R_1** is: piperazine, piperazin-2-one, piperidine, morpholine, triazole, oxadiazole, tetrazole, imidazole, pyrazole,

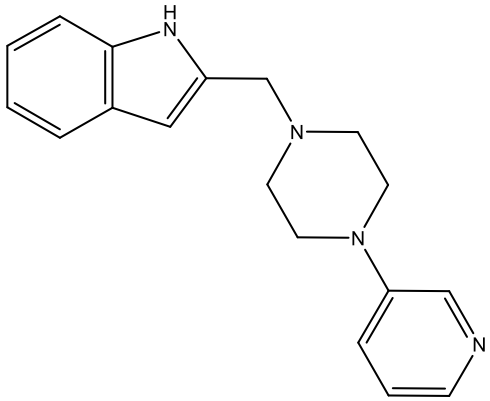
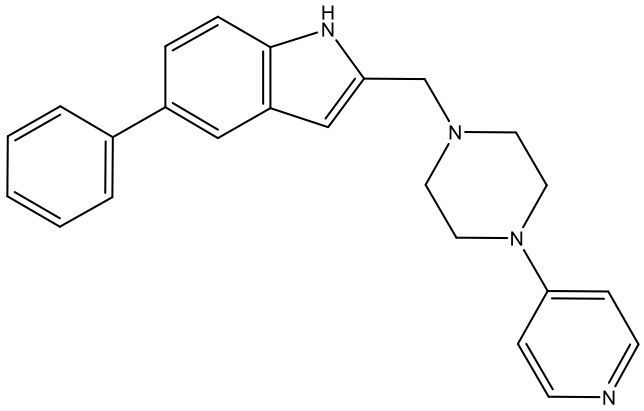
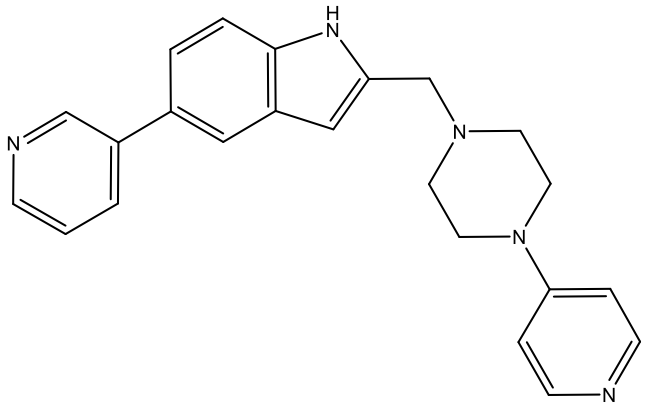
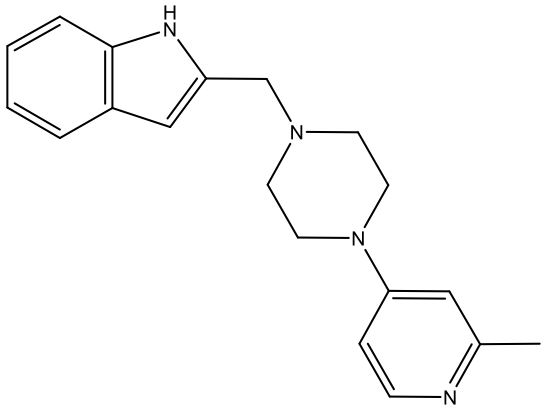
pyrrolidine, pyrrolidin-2-one, oxetane, azetidine, diazepane, 4,7-diazaspiro[2.5]octane or 3-azabicyclo[3.1.0]hexane.

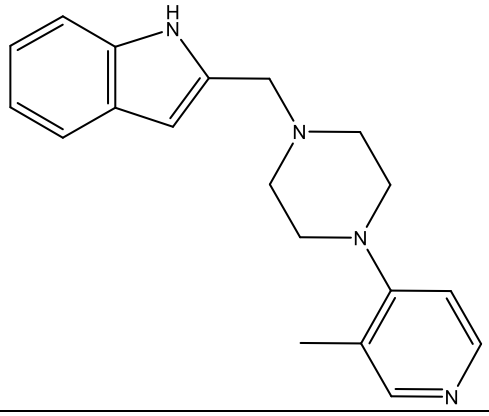
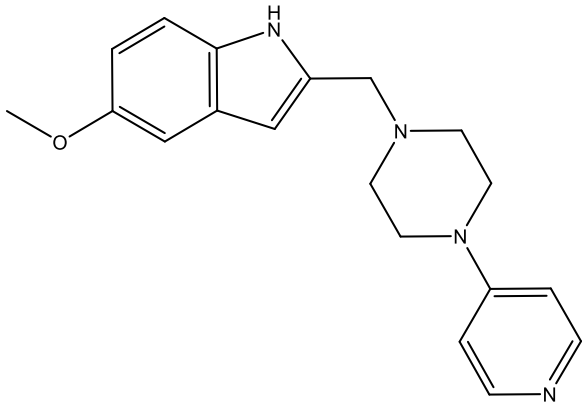
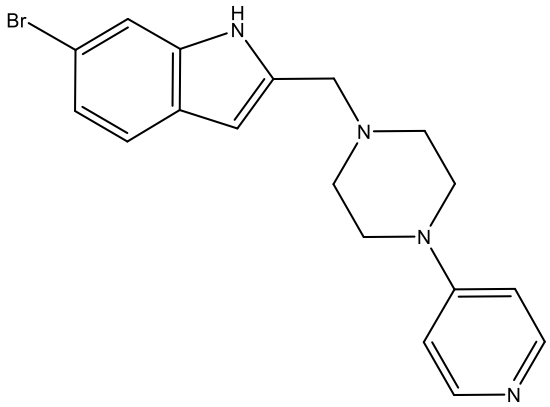
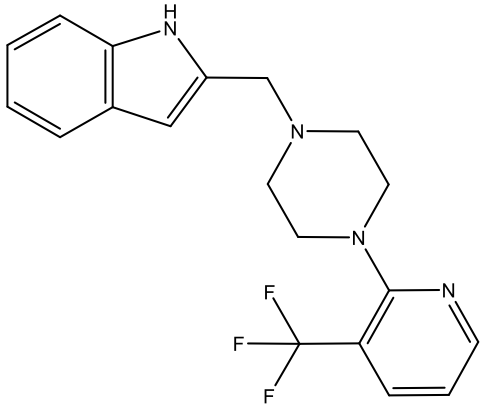
4. The compound of any one of the preceding claims, wherein at least one of $\mathbf{X_{10}}$ and $\mathbf{X_{12}}$ is N.

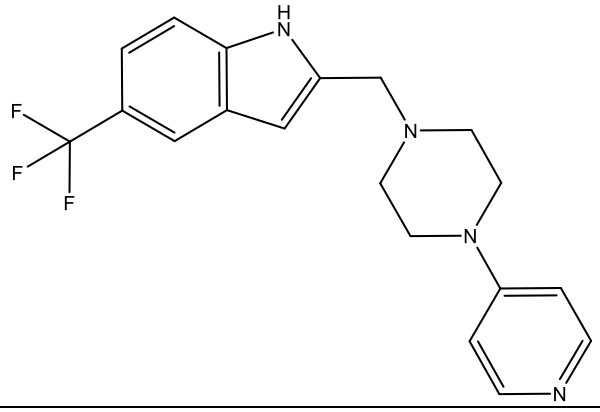
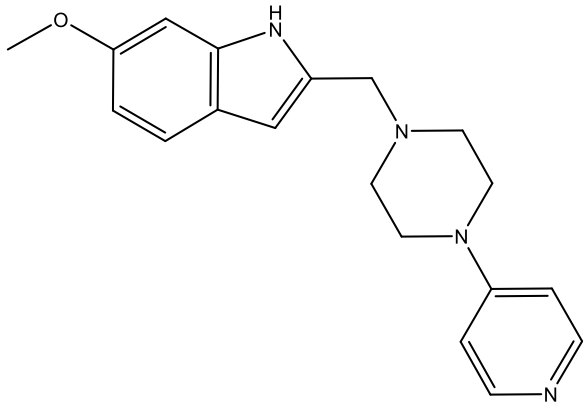
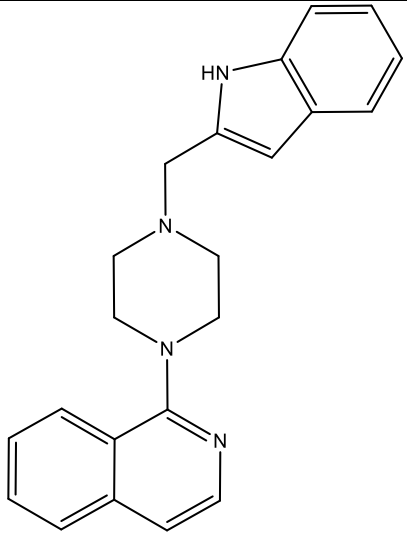
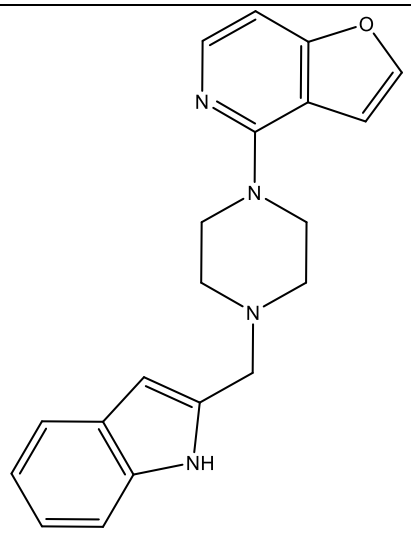
5. The compound of claim 1, selected from the following:

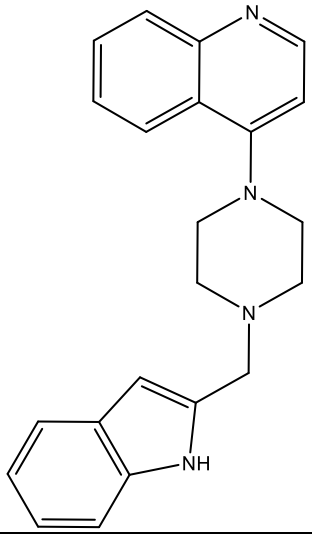
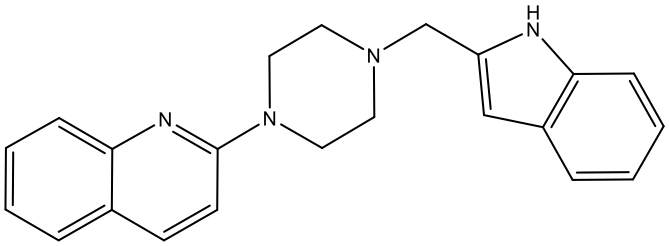
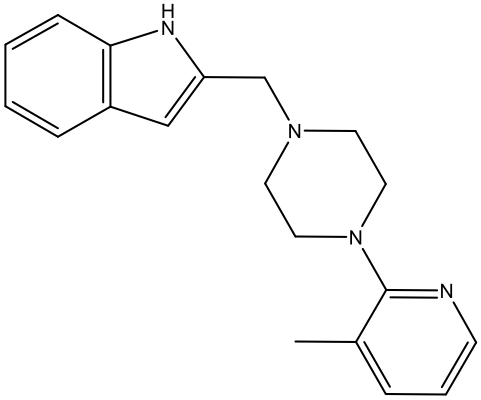
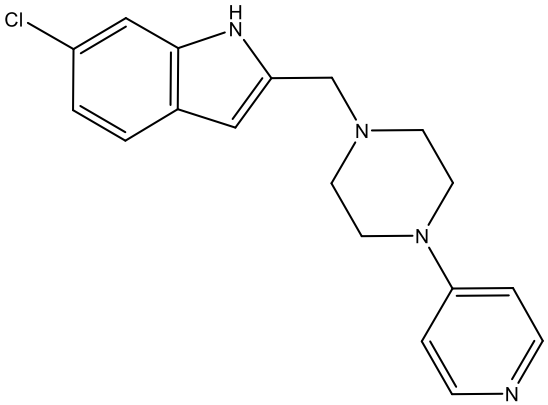
Compound Number	**Compound Structure**
202	
204	
210	

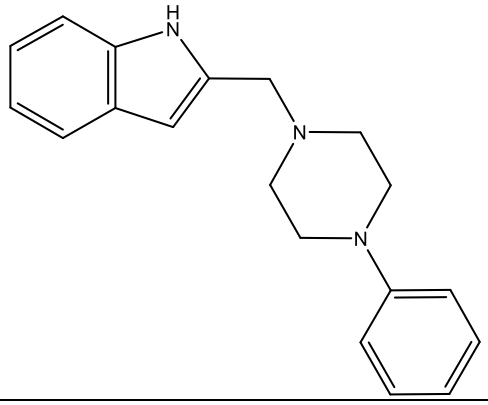
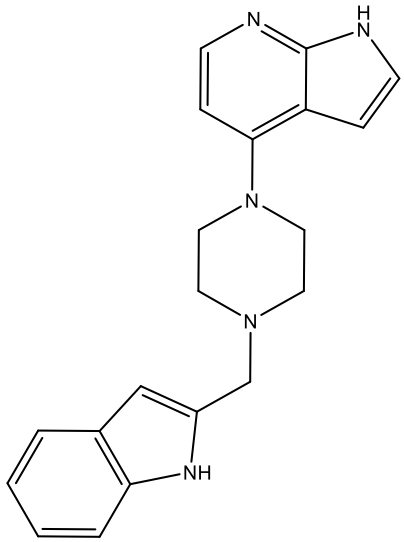
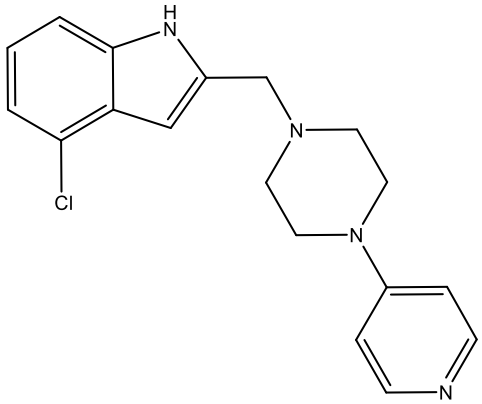
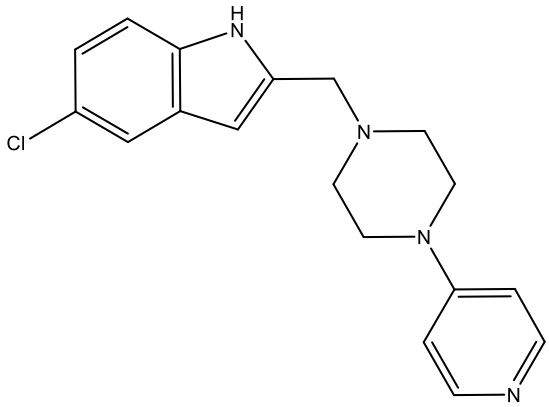
212	
213	
215	
216	

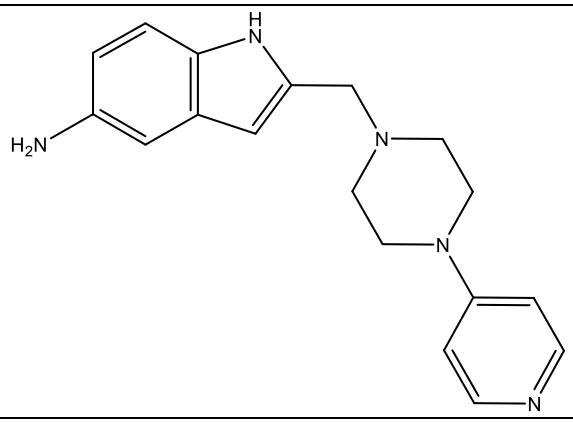
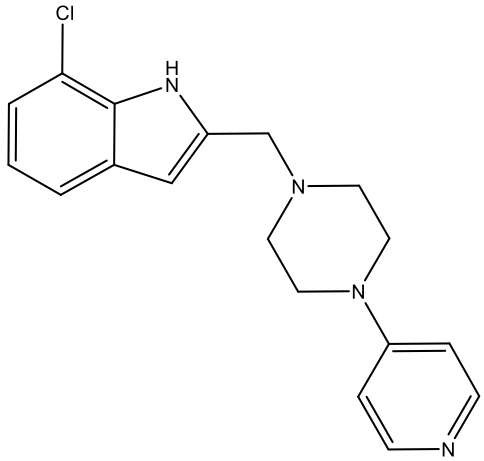
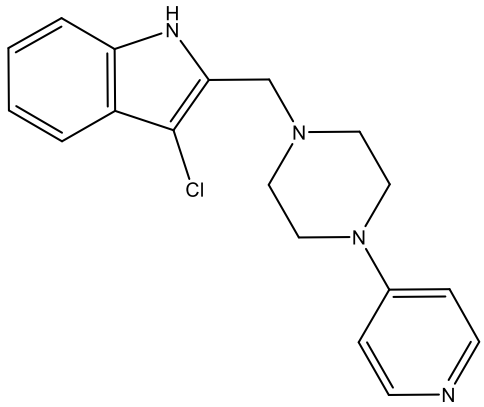
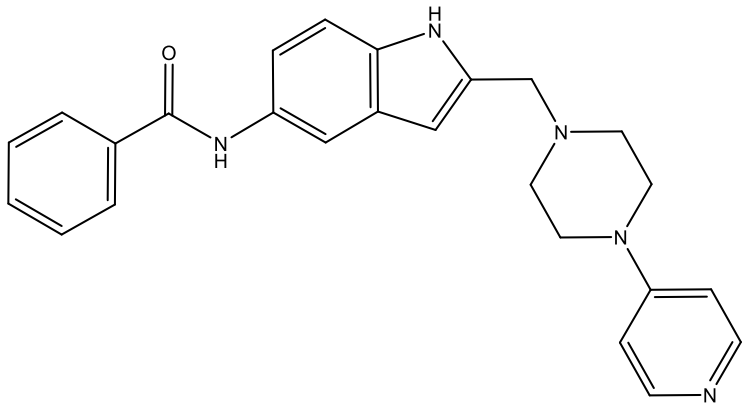
217	
219	
220	
221	

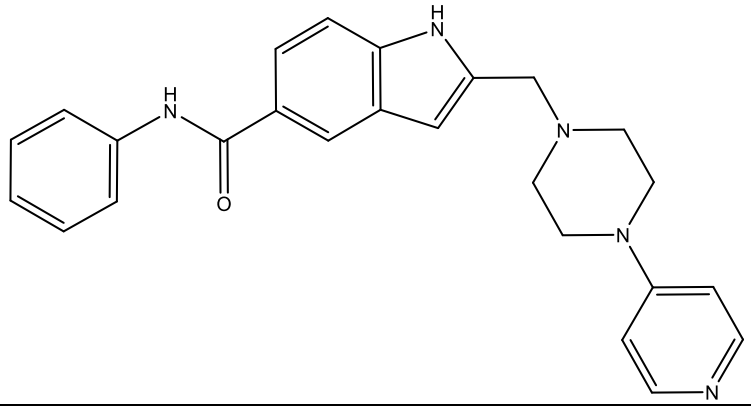
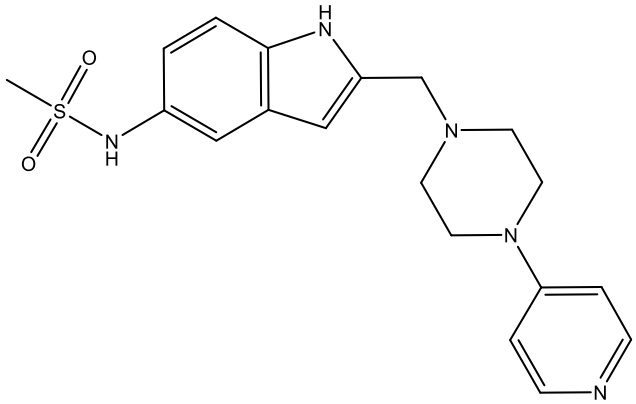
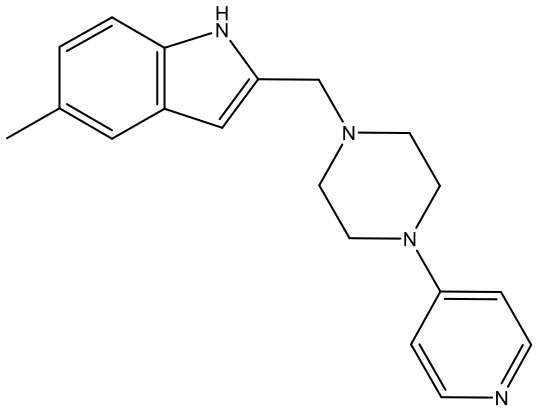
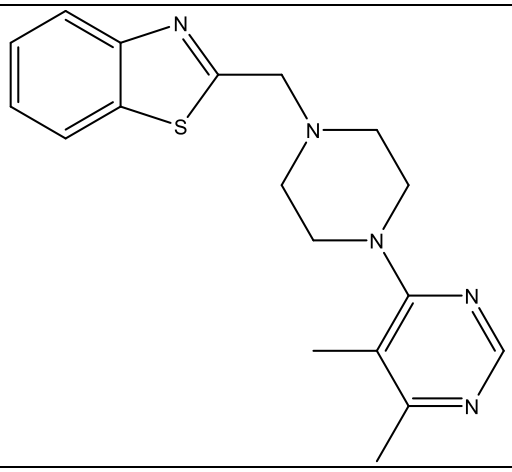
222	
223	
224	
225	

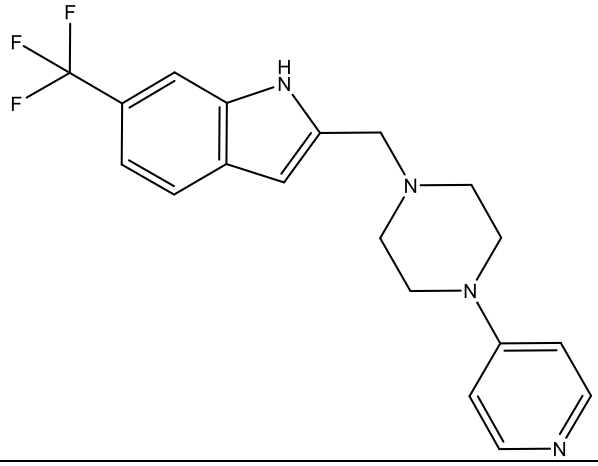
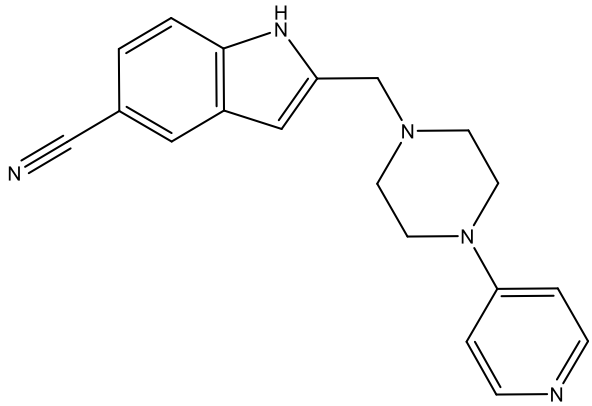
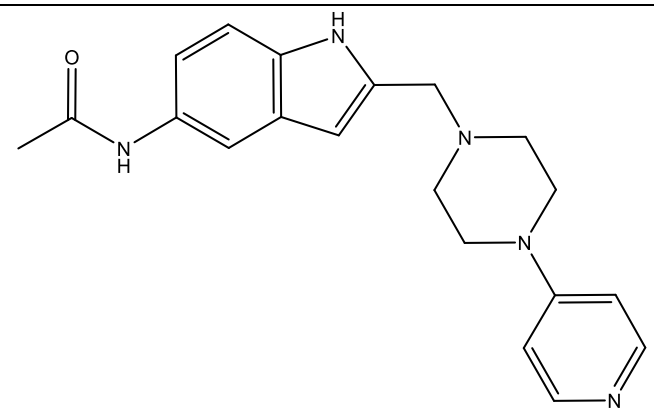
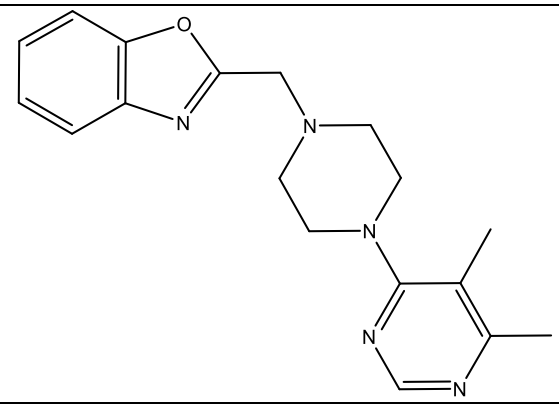
226	
228	
234	
235	

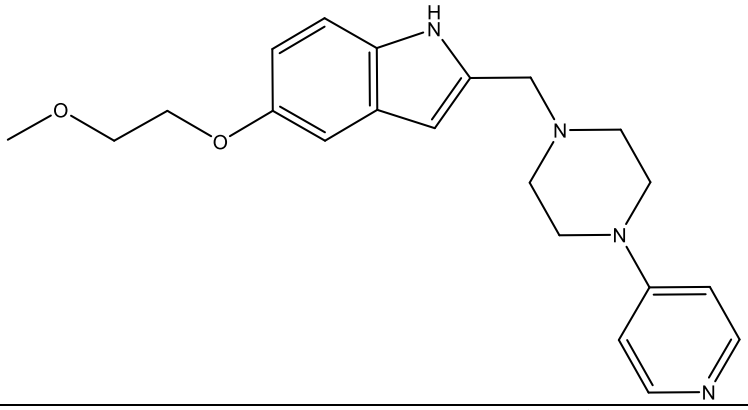
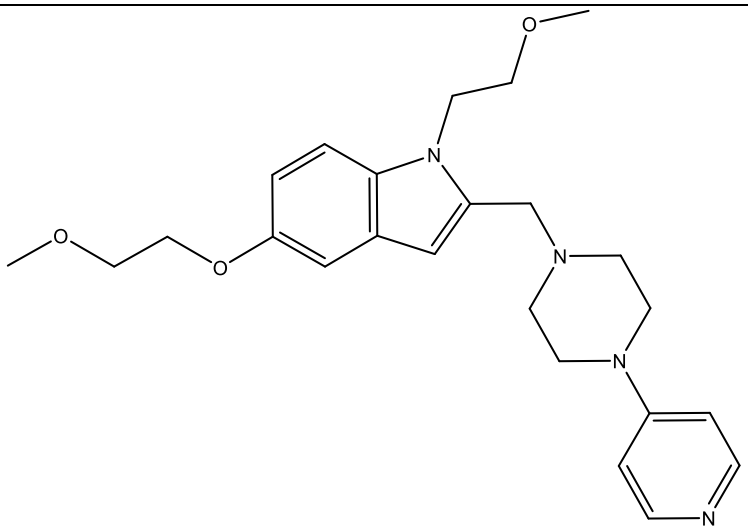
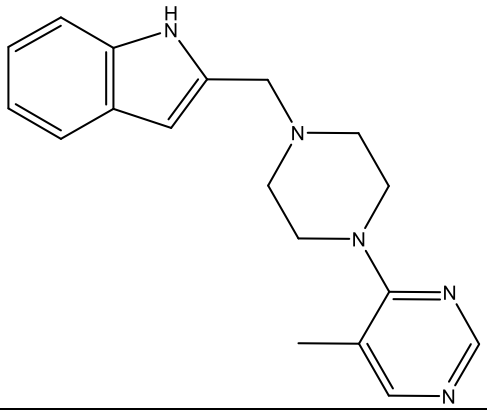
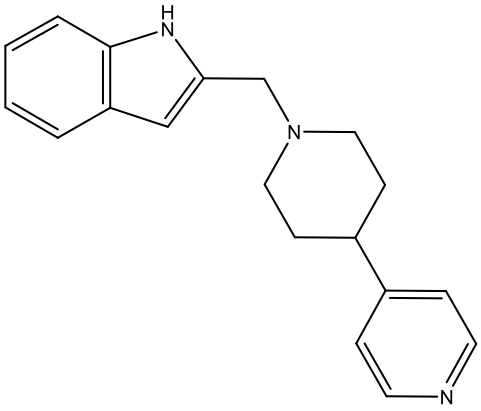
236	
237	
238	
240	

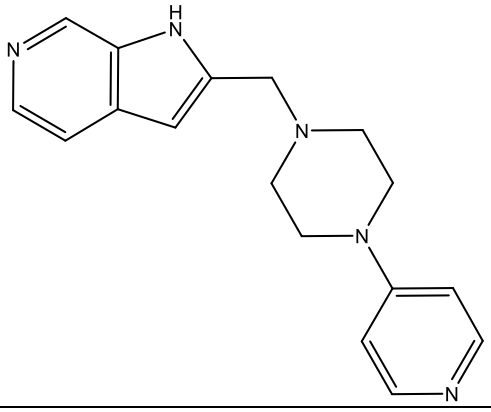
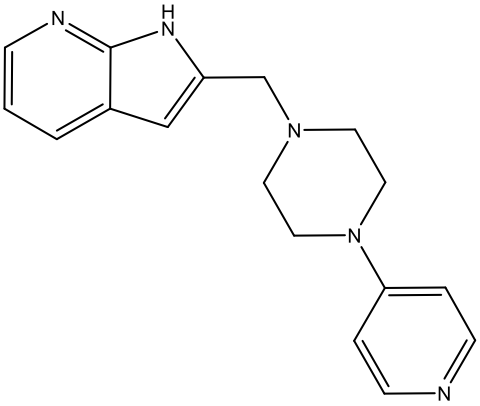
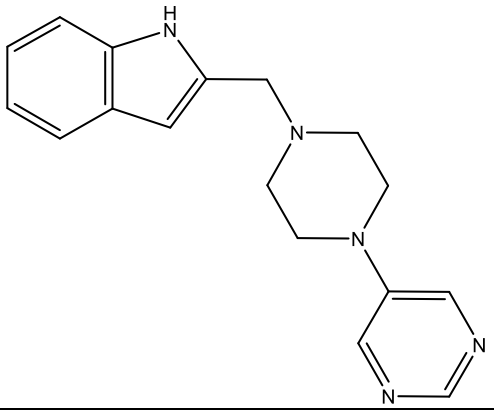
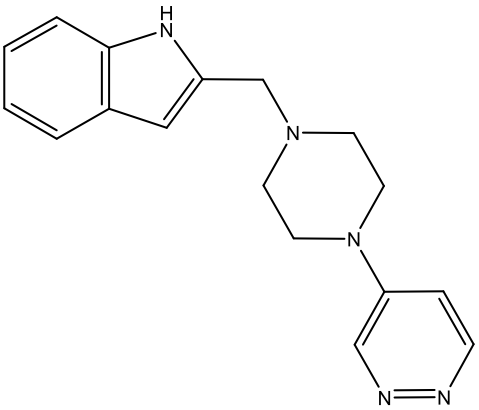
241	
242	
243	
245	

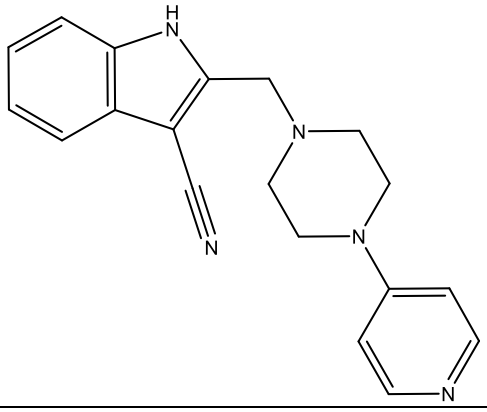
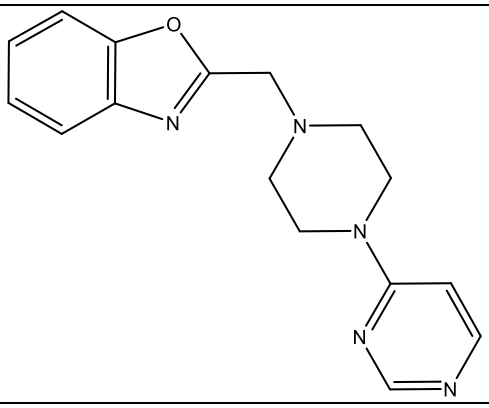
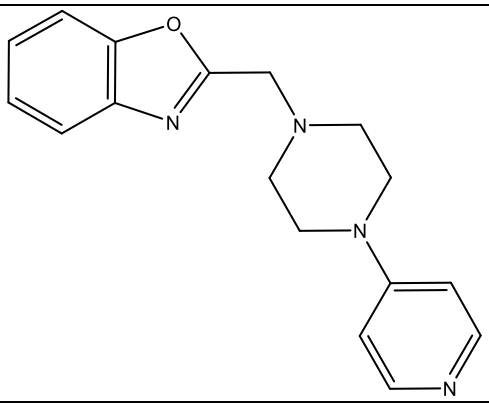
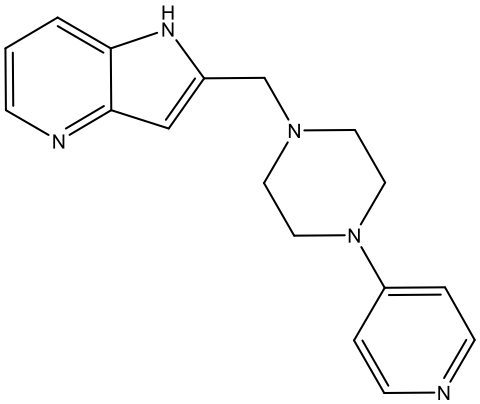
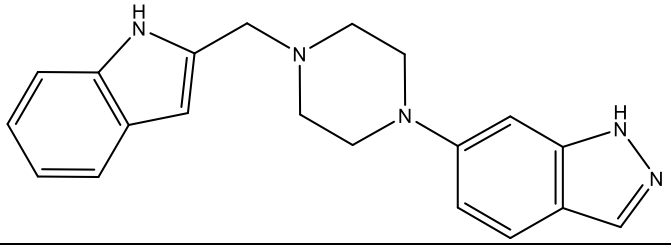
246	
247	
248	
249	

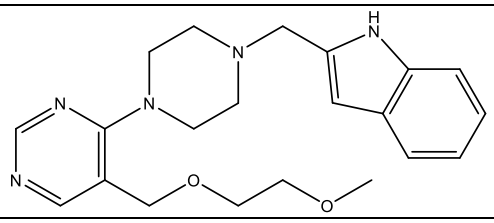
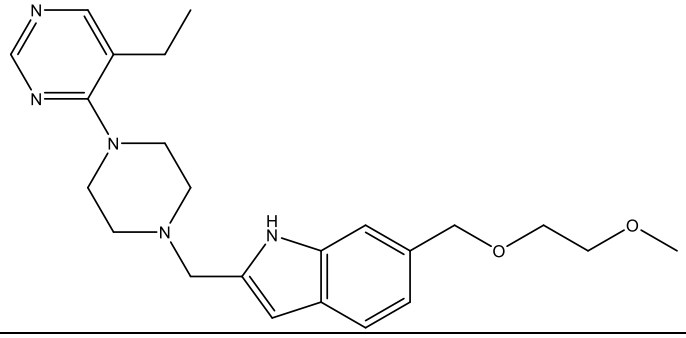
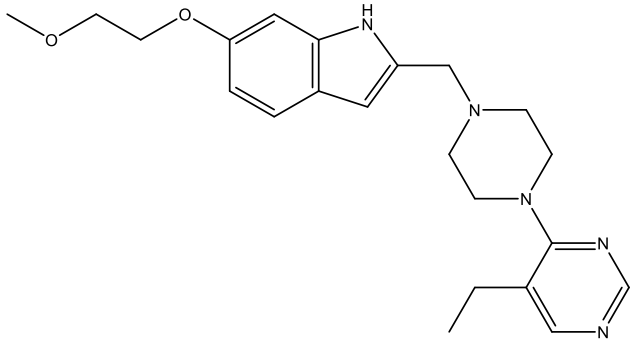
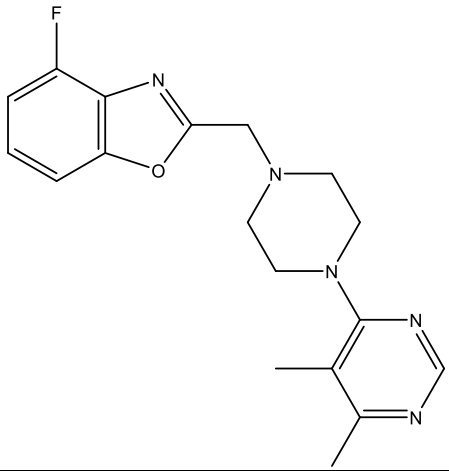
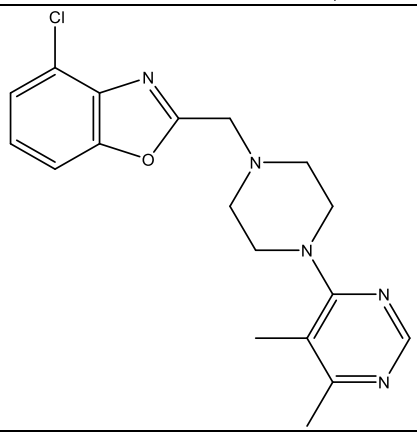
250	
251	
252	
254	

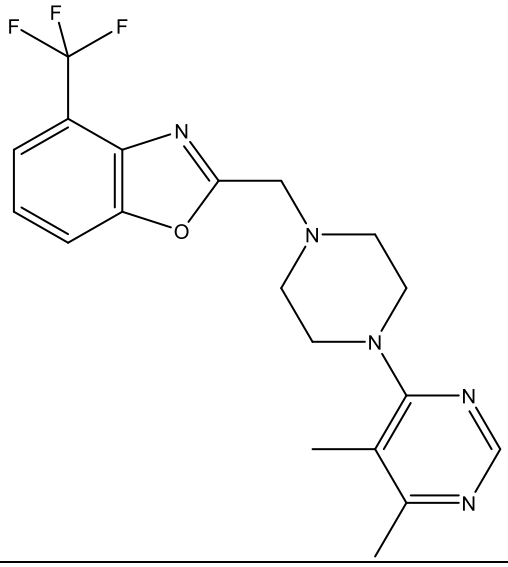
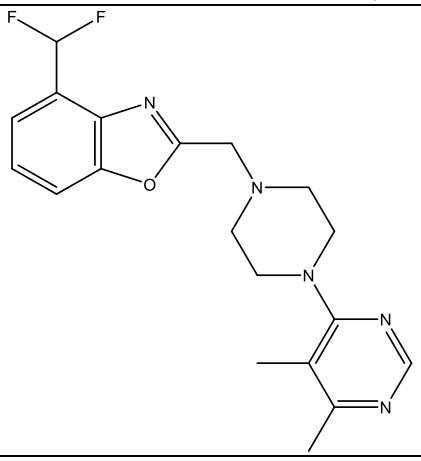
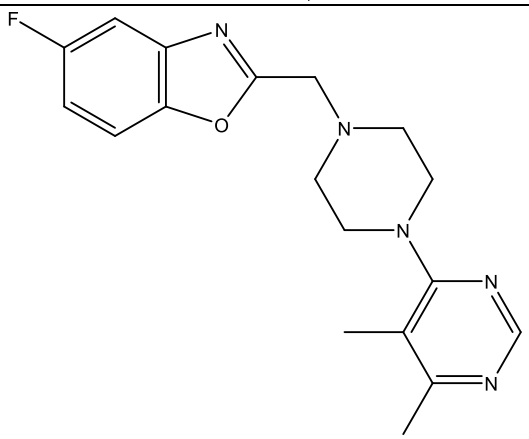
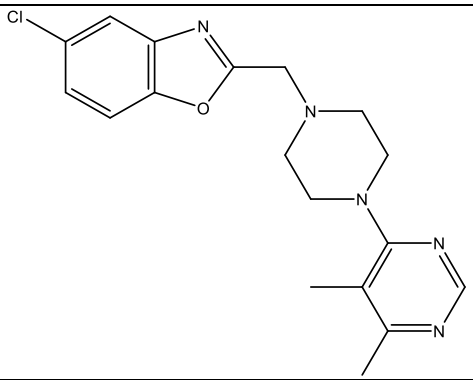
255	
256	
258	
259	

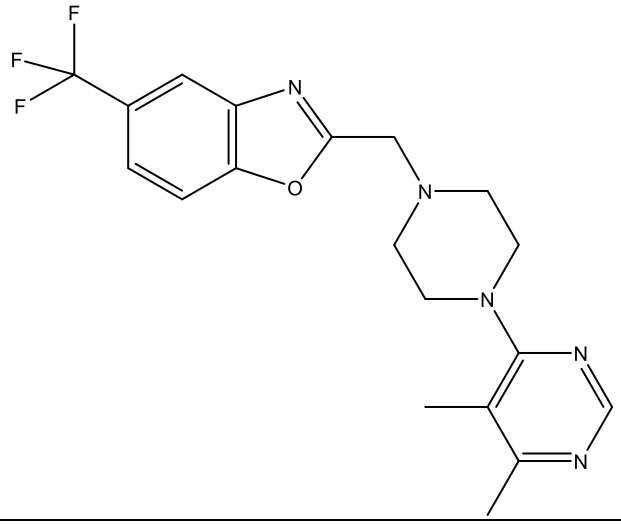
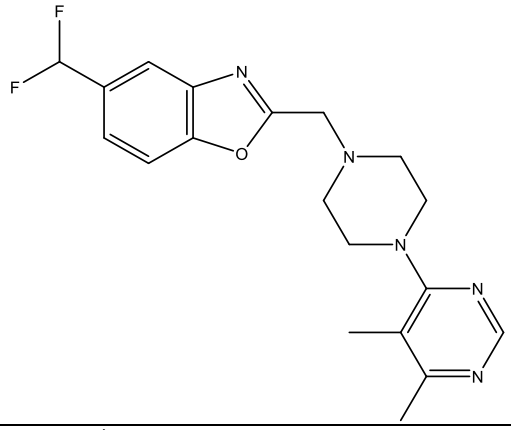
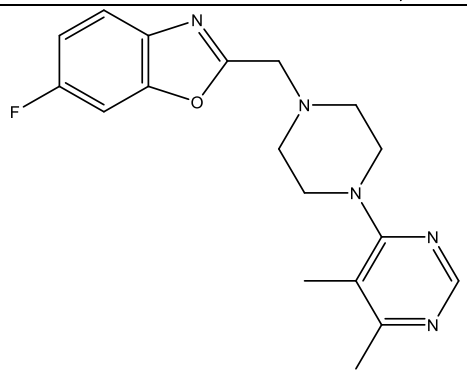
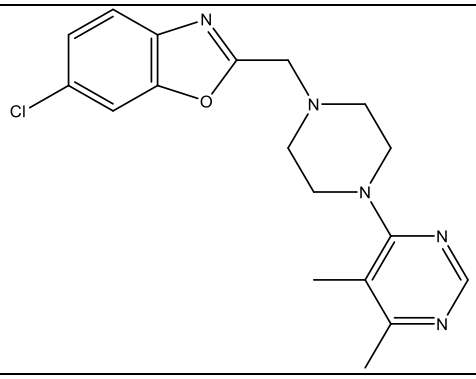
260	
261	
266	
269	

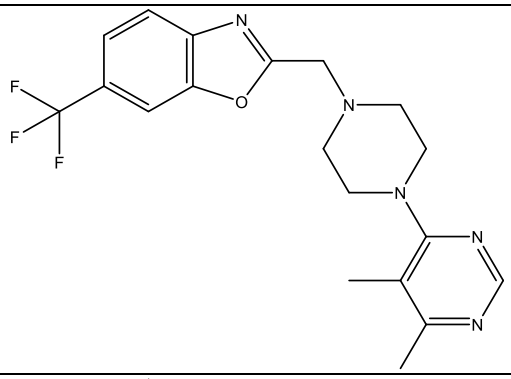
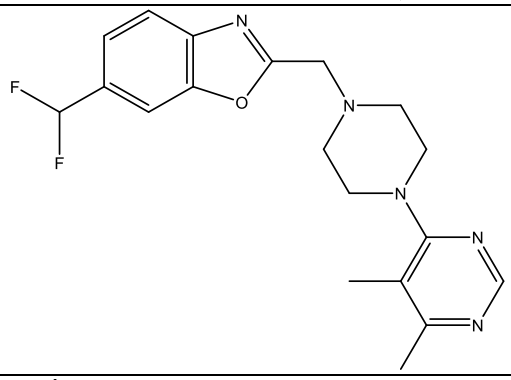
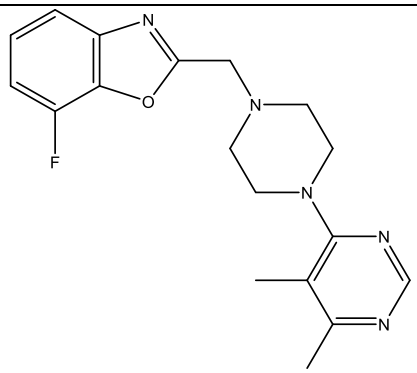
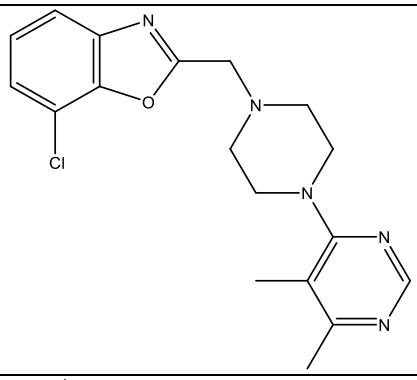
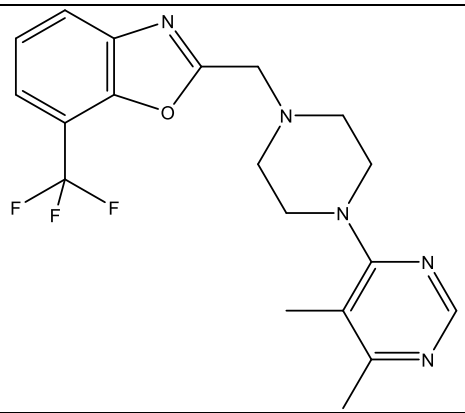
270	
275	
278	
279	

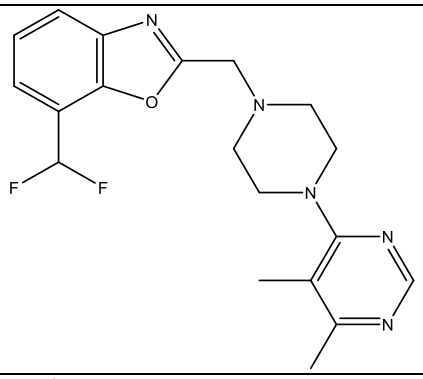
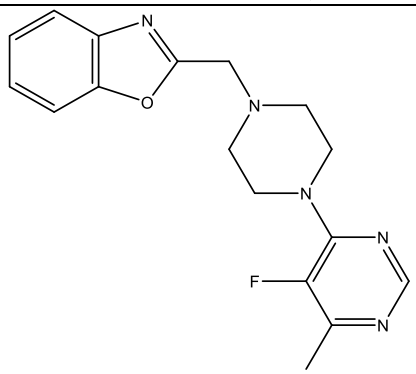
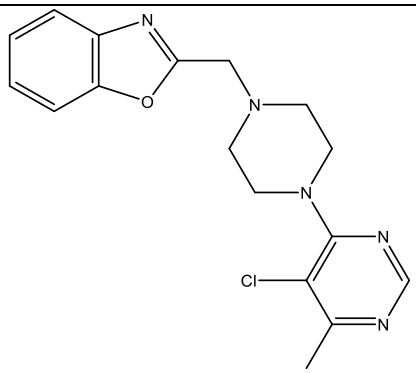
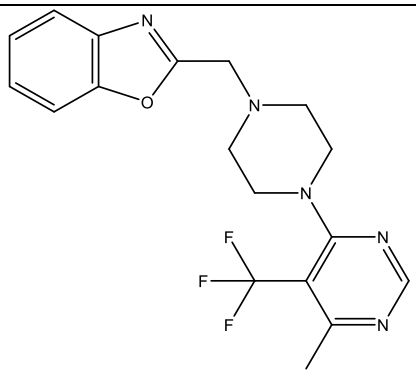
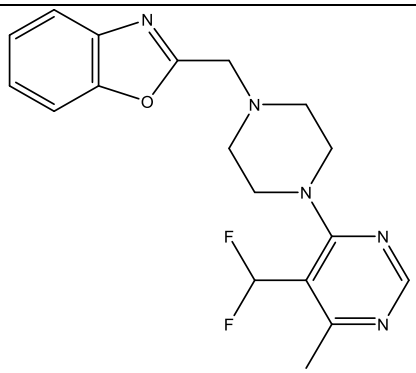
280	
281	
282	
284	
286	

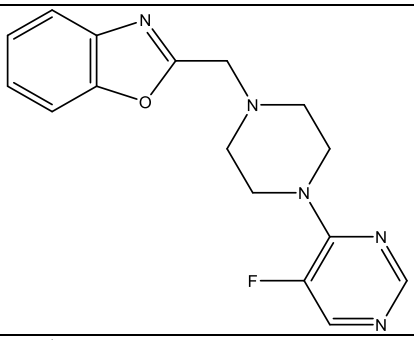
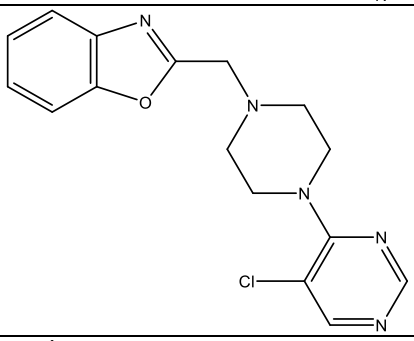
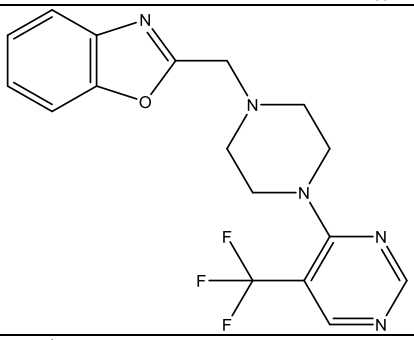
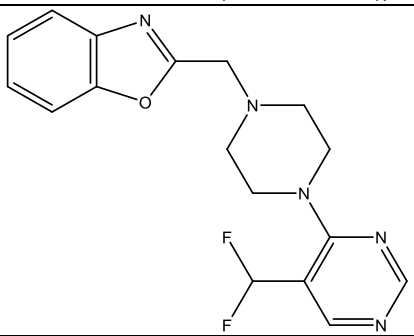
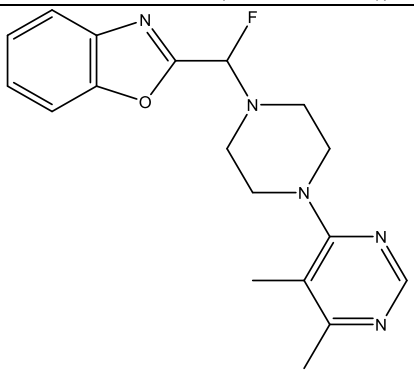
287	
291	
292	
293	
294	

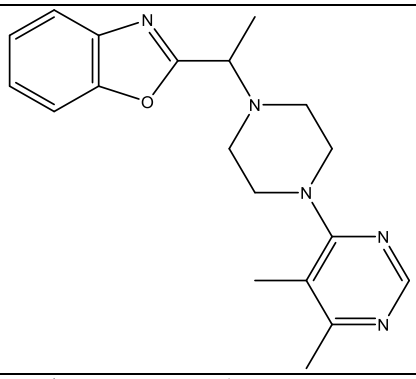
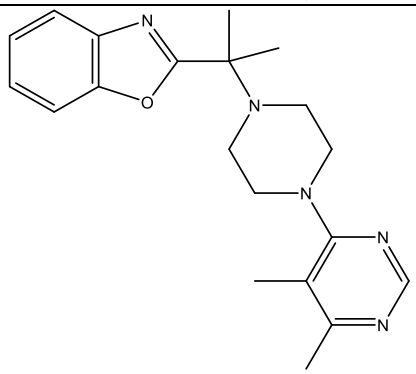
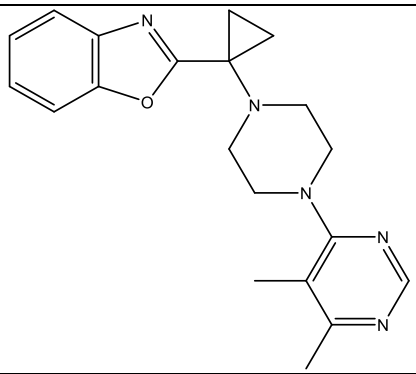
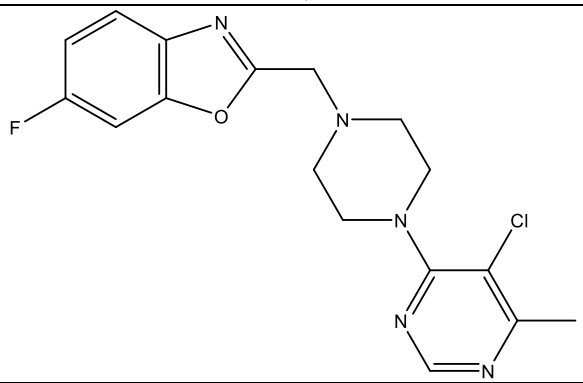
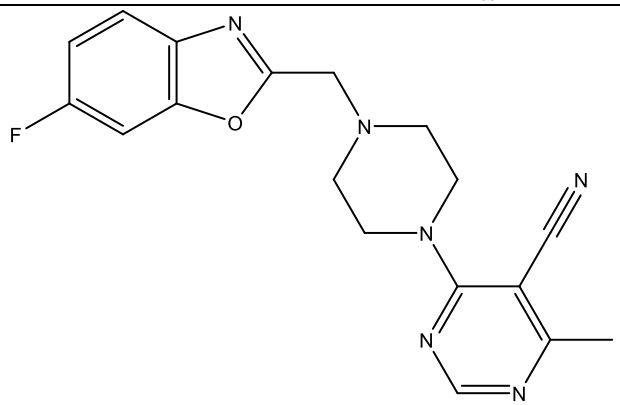
295	
296	
297	
298	

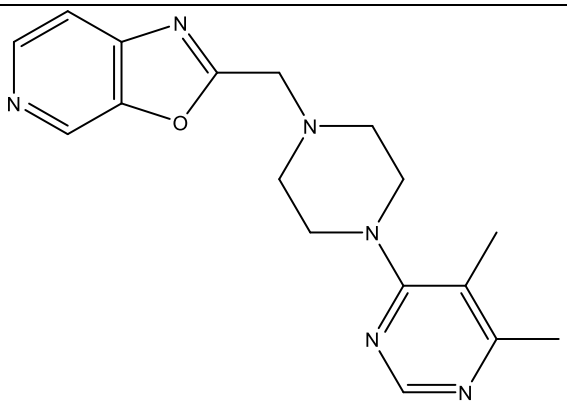
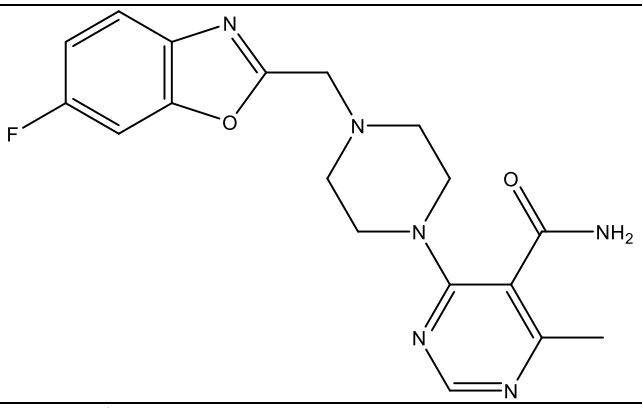
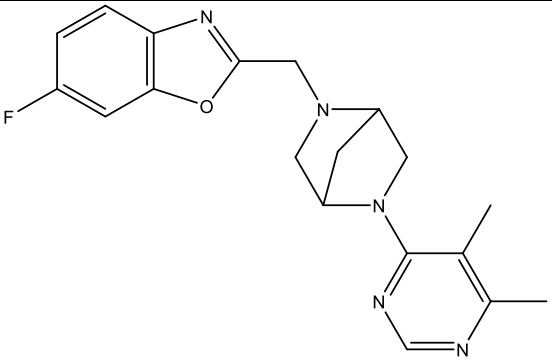
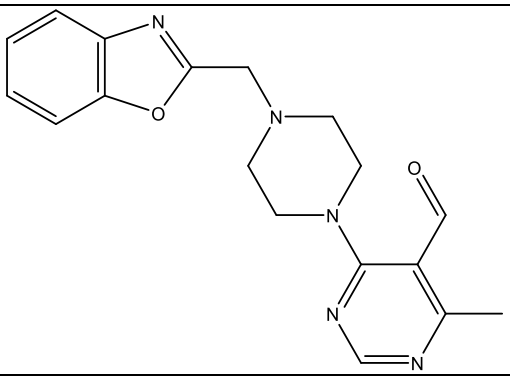
299	
300	
301	
302	

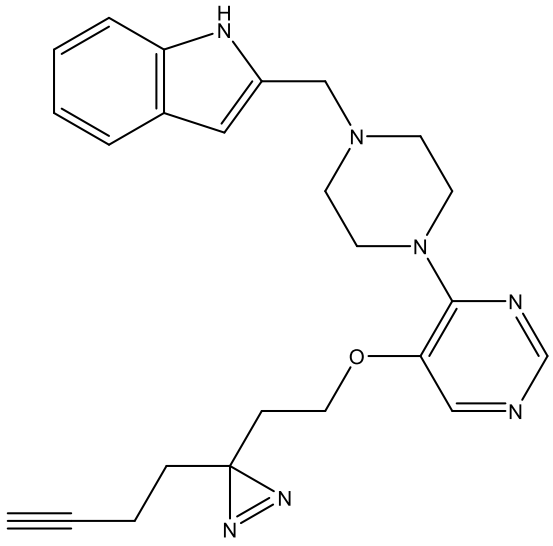
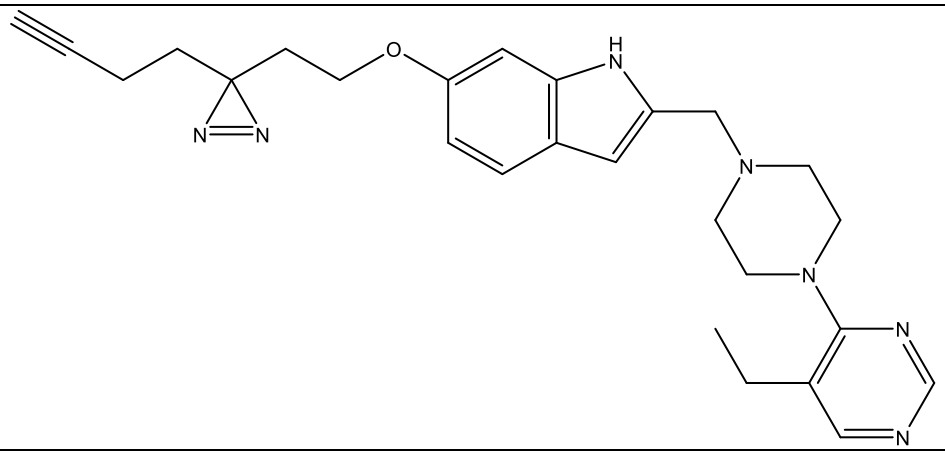
303	
304	
305	
306	
307	

308	
309	
310	
311	
312	

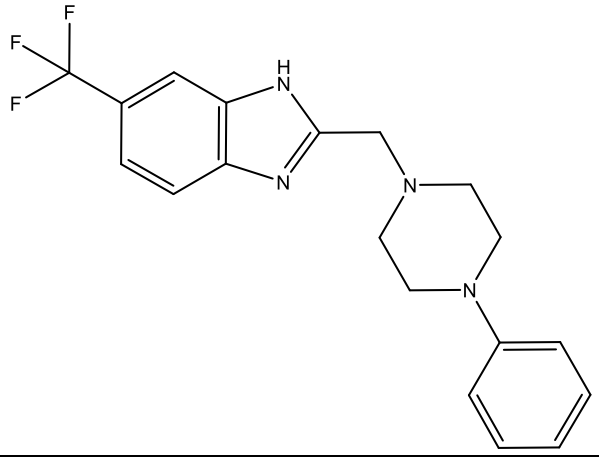
313	
314	
315	
316	
317	

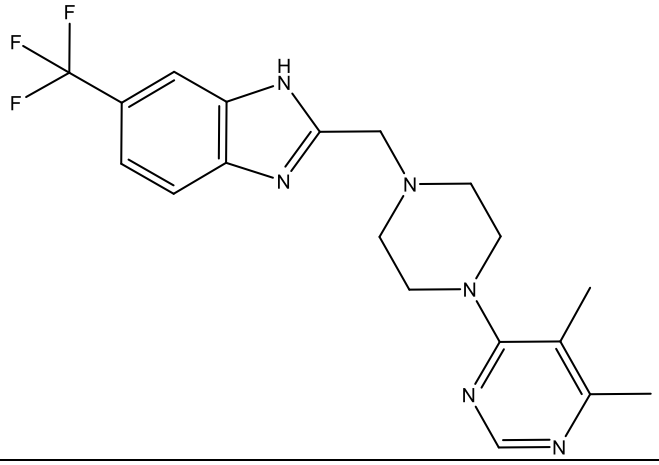
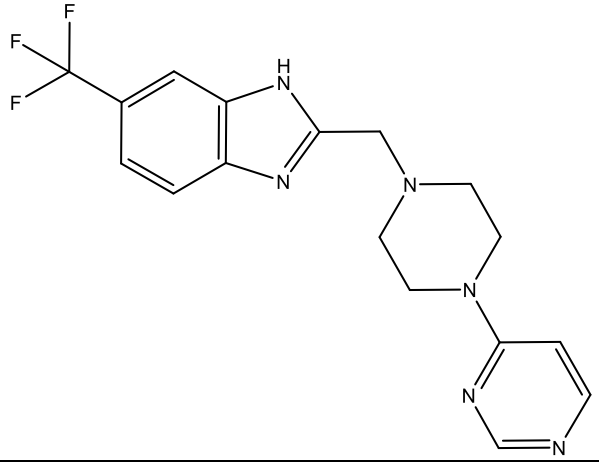
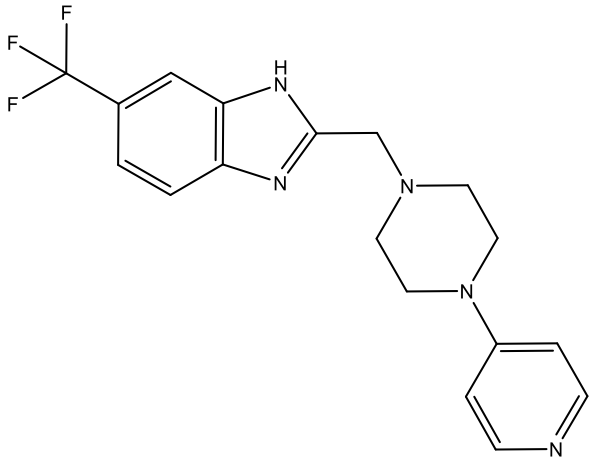
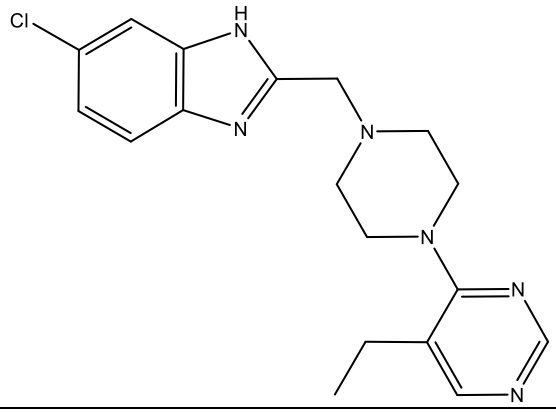
318	
320	
321	
327	
329	

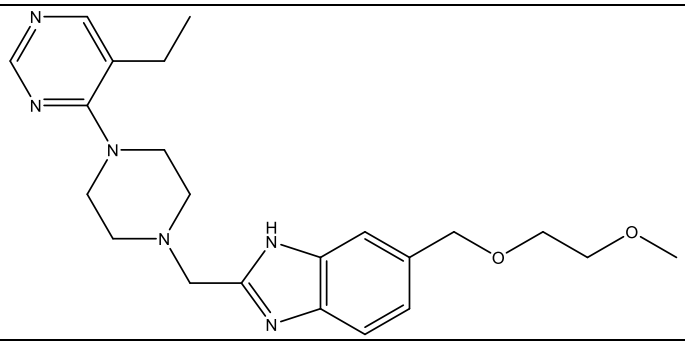
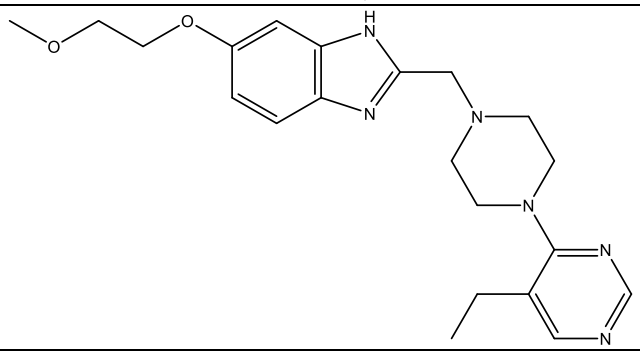
330	
333	
334	
336	

337	
338	

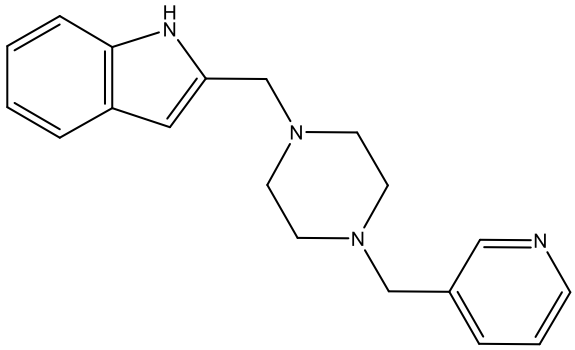
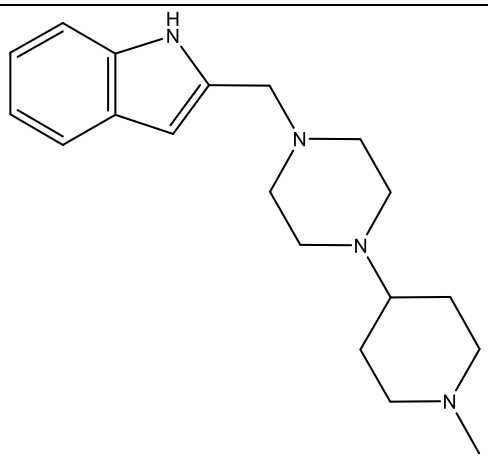
or selected from the following:

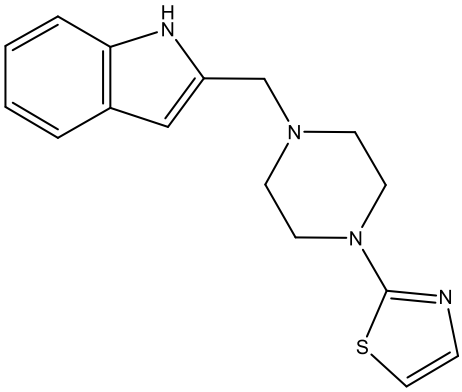
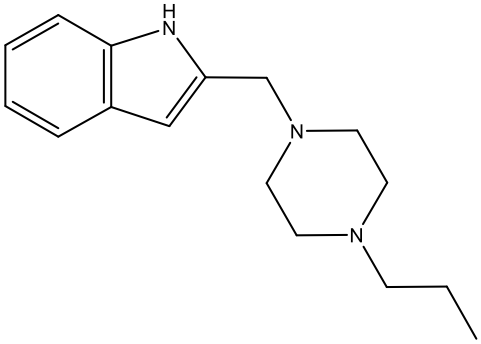
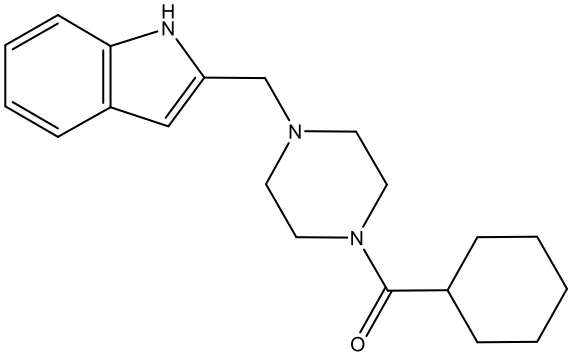
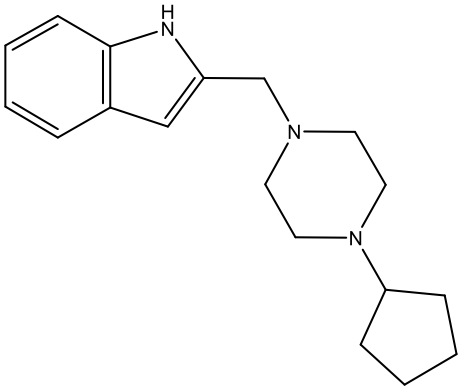
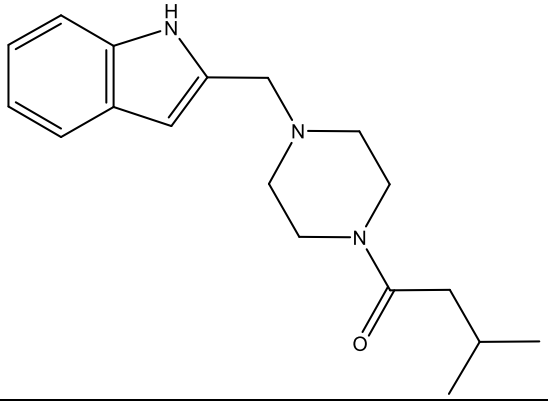
Compound Number	**Compound Structure**
200	

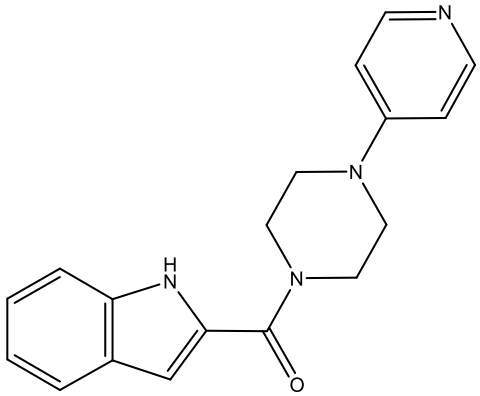
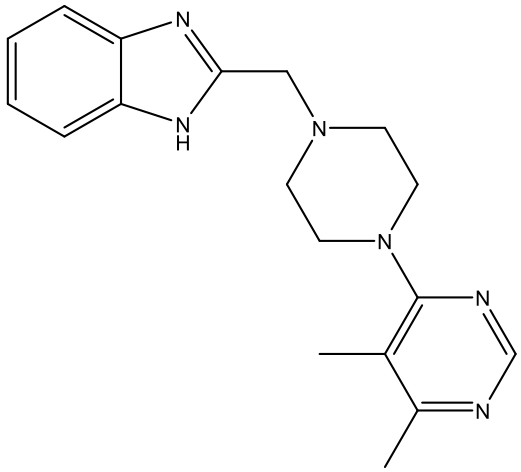
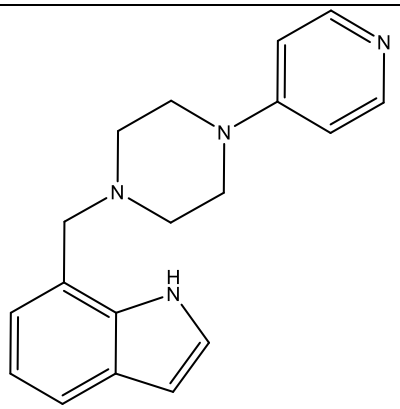
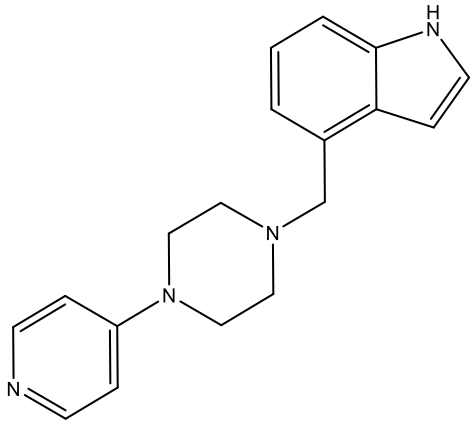
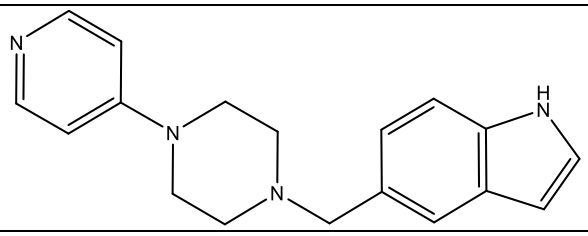
201	
203	
209	
211	

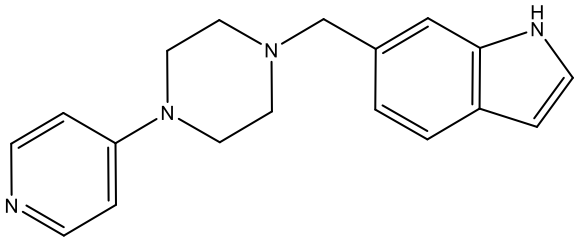
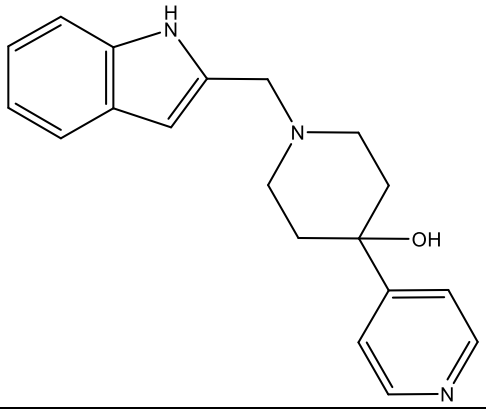
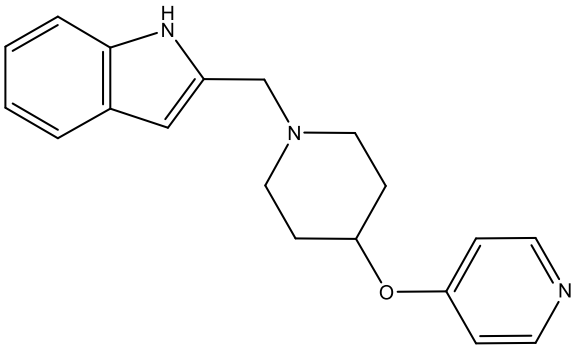
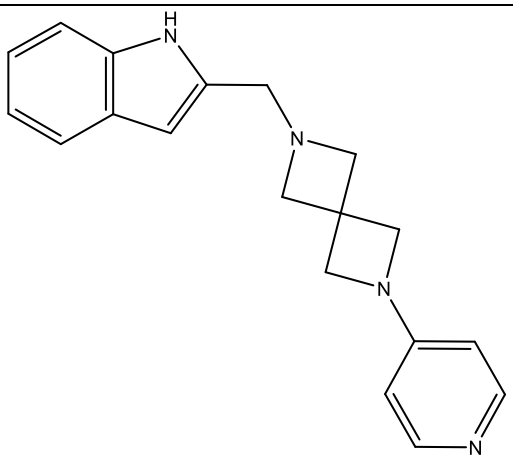
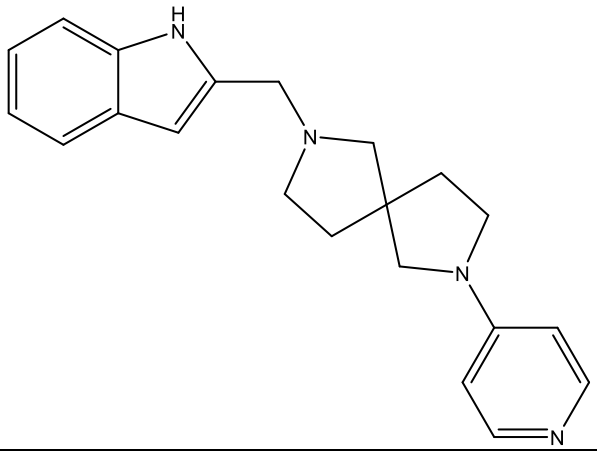
289	
290	

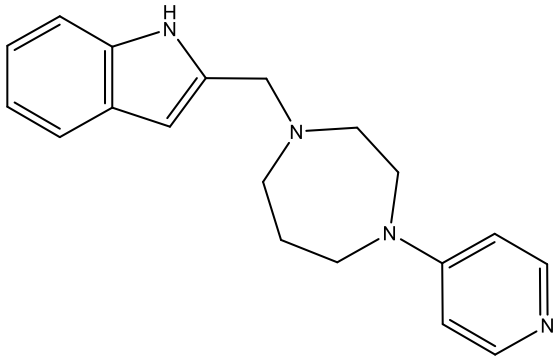
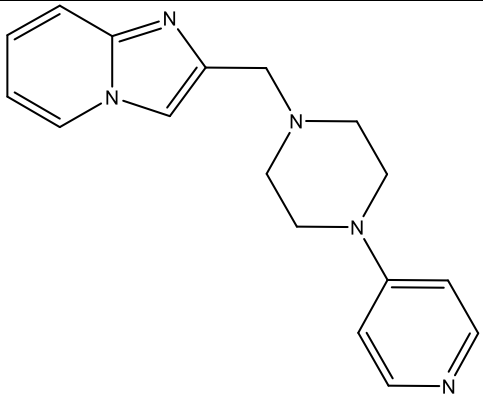
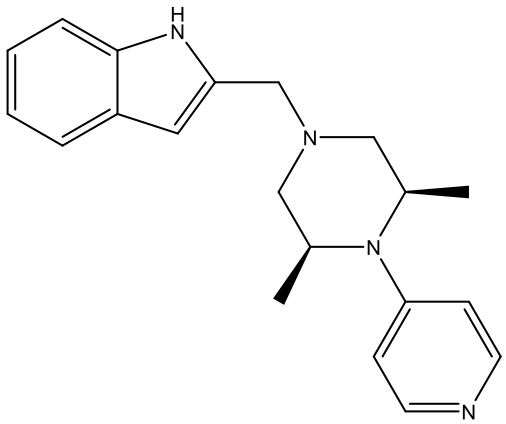
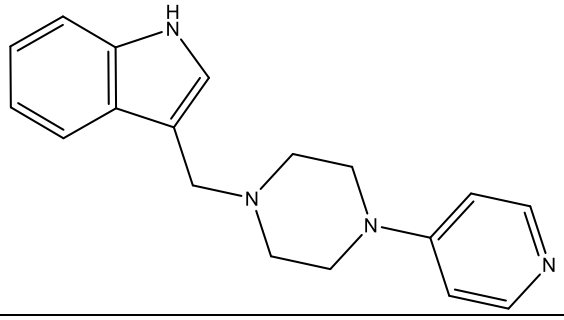
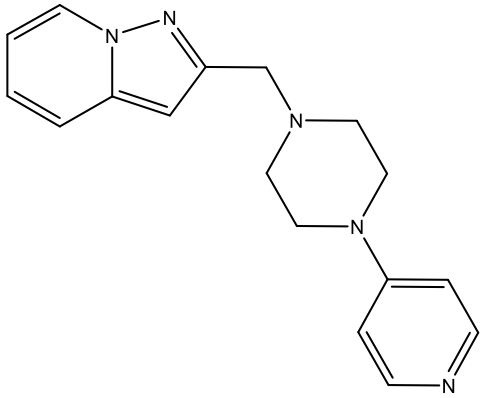
6. A compound represented by the structure of the following compounds:

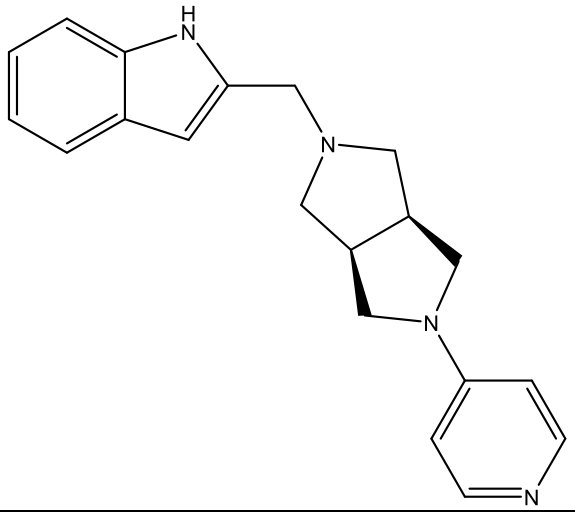
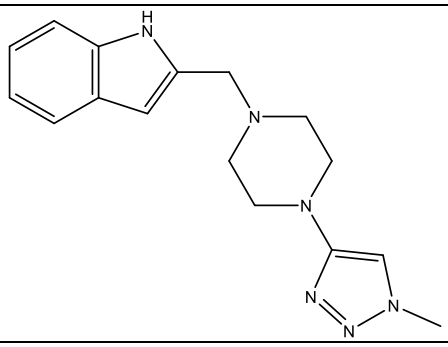
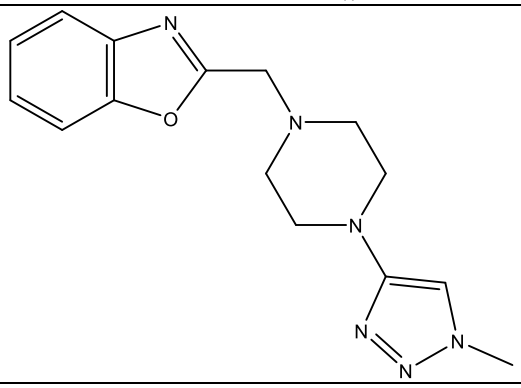
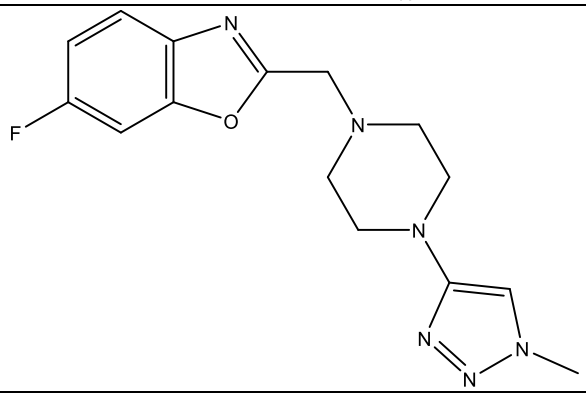
Compound Number	**Compound Structure**
218	
227	

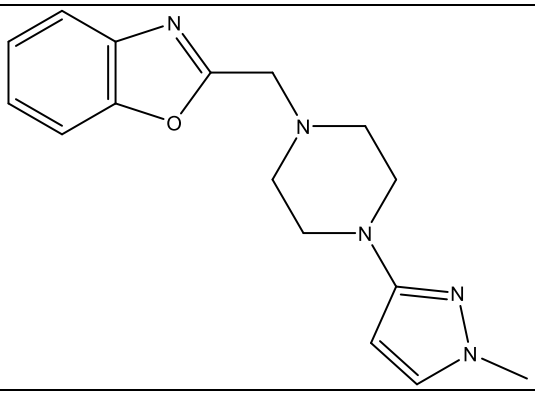
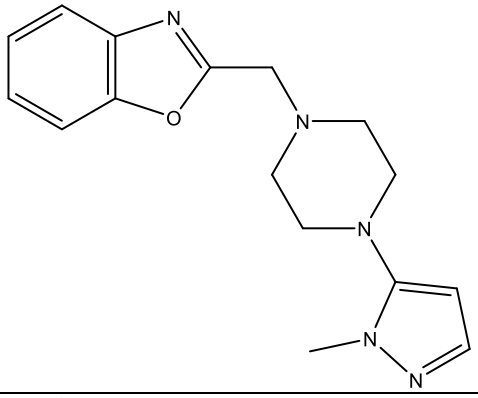
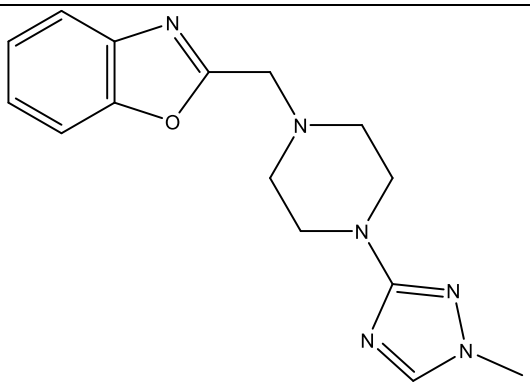
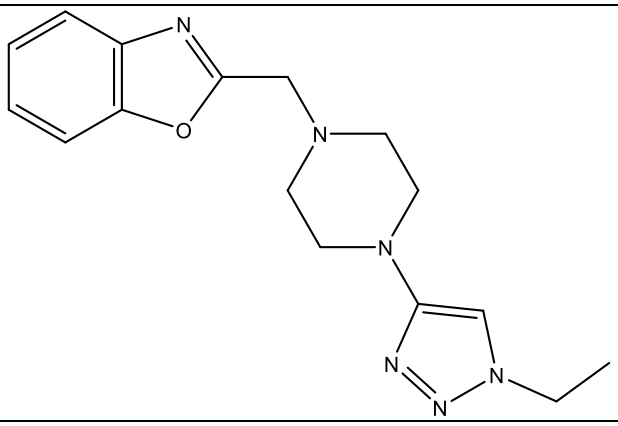
229	
230	
232	
233	
239	

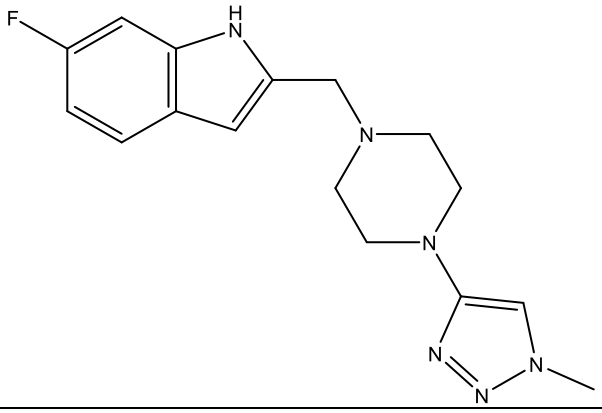
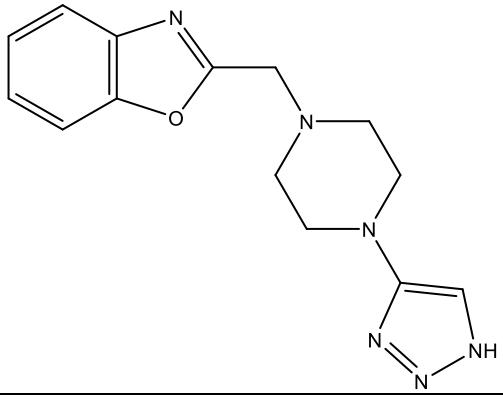
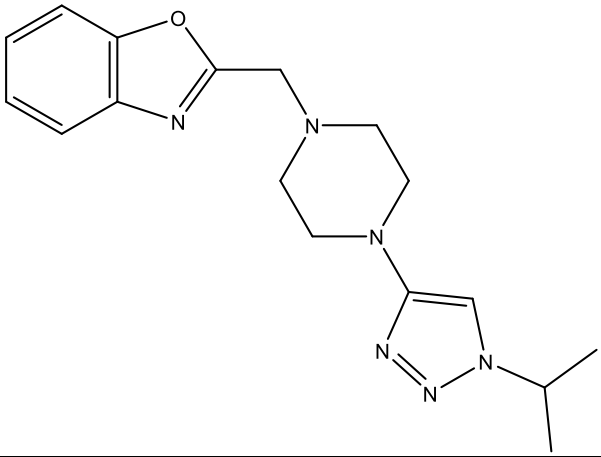
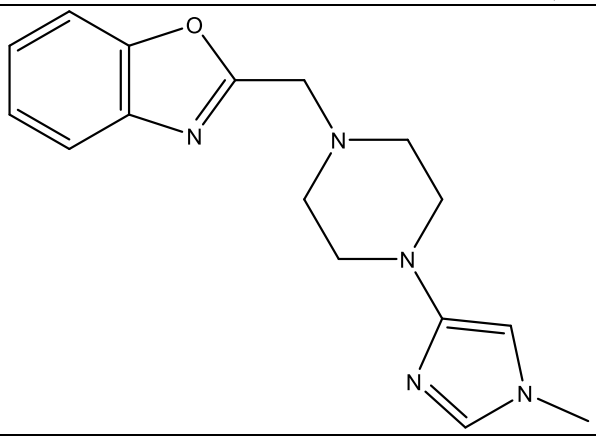
244	
257	
262	
263	
264	

265	
267	
268	
271	
272	

273	
274	
276	
277	
283	

285	
288	
322	
323	

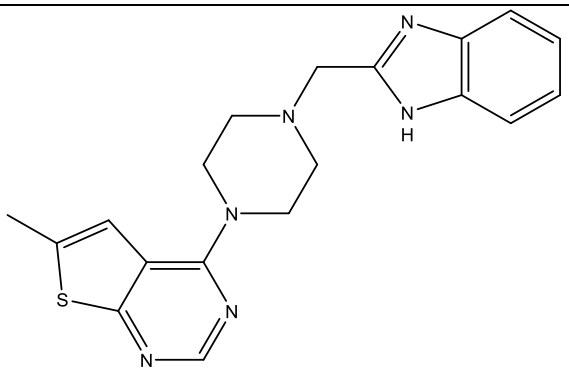
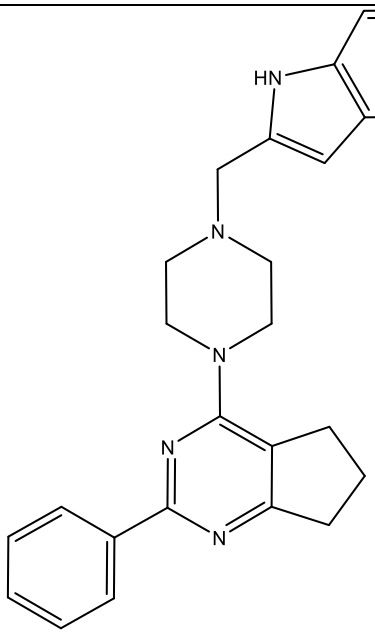
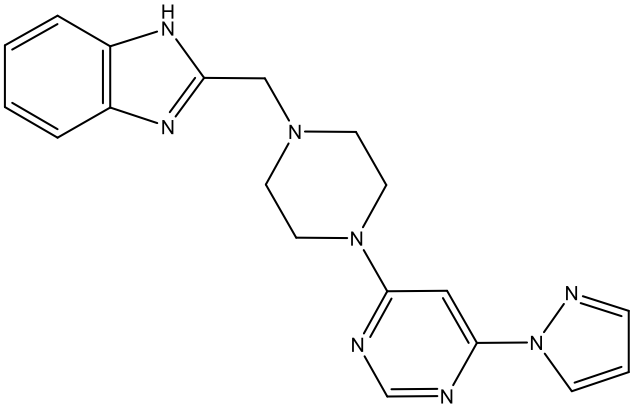
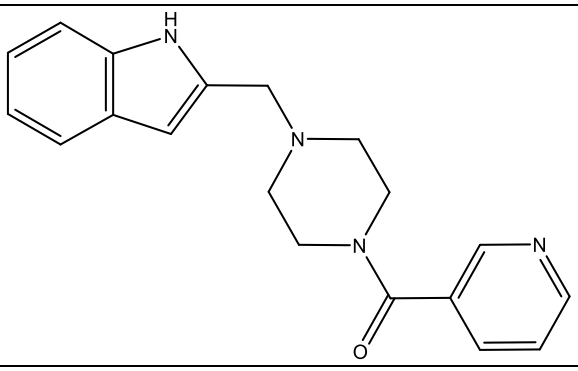
324	
325	
326	
328	

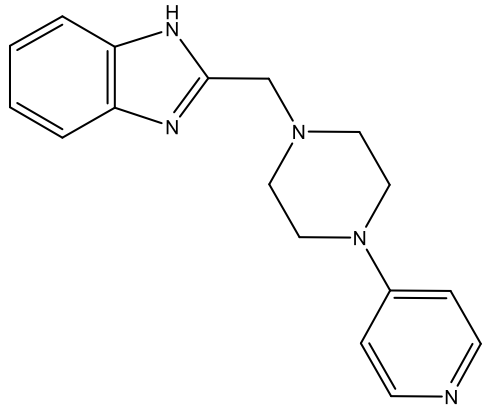
331	
332	
339	
340	

7. The compound according to any one of the preceding claims, wherein the compound is a collagen translation inhibitor.

8. A pharmaceutical composition comprising a compound according to any one of claims 1 to 7 and a pharmaceutically acceptable carrier.

9. Use of a compound according to any one of claims 1 to 7; or a compound represented by any one of the following structures:

Compound Number	**Compound Structure**
205	
207	
208	
231	

253	

in the manufacture of a medicament for treating, suppressing, reducing the severity, reducing the risk of developing or inhibiting fibrosis in a subject.

10. The use of claim 9, wherein said fibrosis is a systemic fibrotic disease;

wherein said fibrosis is an organ-specific fibrotic disease;

wherein said fibrosis is primary or secondary fibrosis;

wherein said fibrosis is a result of systemic sclerosis, graft-versus host disease (GVHD), pulmonary fibrosis, autoimmune disorder, tissue injury, inflammation, oxidative stress or any combination thereof;

wherein the fibrosis is hepatic fibrosis, lung fibrosis or dermal fibrosis;

or any combination thereof.

11. The use of claim 10,

wherein said systemic fibrotic disease is systemic sclerosis, multifocal fibrosclerosis (IgG4-associated fibrosis), nephrogenic systemic fibrosis, sclerodermatous graft vs. host disease, or any combination thereof;

wherein said organ-specific fibrotic disease is lung fibrosis, cardiac fibrosis, kidney fibrosis, pulmonary fibrosis, liver and portal vein fibrosis, radiation-induced fibrosis, bladder fibrosis, intestinal fibrosis, peritoneal sclerosis, diffuse fasciitis, wound healing, scaring, or any combination thereof;

wherein the dermal fibrosis is scleroderma;

wherein the dermal fibrosis is a result of a localized or generalized morphea, keloids, hypertrophic scars, familial cutaneous collagenoma, connective tissue nevi of the collagen type, or any combination thereof;

wherein the hepatic fibrosis is a result of hepatic scarring or chronic liver injury;

or any combination thereof.

12. The use of claim 11,

wherein said lung fibrosis is idiopathic pulmonary fibrosis (IPF);

wherein said cardiac fibrosis is hypertension-associated cardiac fibrosis, Post-myocardial infarction, Chagas disease-induced myocardial fibrosis or any combination thereof;

wherein said kidney fibrosis is diabetic and hypertensive nephropathy, urinary tract obstruction-induced kidney fibrosis, inflammatory/autoimmune-induced kidney fibrosis, aristolochic acid nephropathy, polycystic kidney disease, or any combination thereof;

wherein said pulmonary fibrosis is idiopathic pulmonary fibrosis, silica-induced pneumoconiosis (silicosis), asbestos-induced pulmonary fibrosis (asbestosis), chemotherapeutic agent-induced pulmonary fibrosis, or any combination thereof;

wherein said liver and portal vein fibrosis is alcoholic and nonalcoholic liver fibrosis, hepatitis C-induced liver fibrosis, primary biliary cirrhosis, parasite-induced liver fibrosis (schistosomiasis), or any combination thereof;

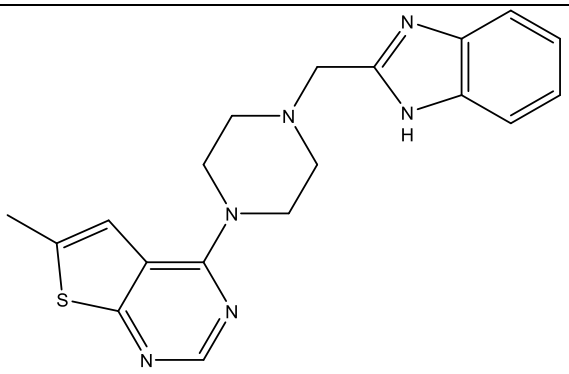
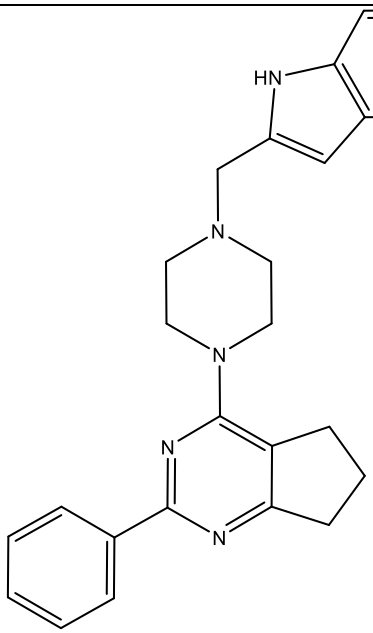
wherein said diffuse fasciitis is localized scleroderma, keloids, dupuytren's disease, peyronie's disease, myelofibrosis, oral submucous fibrosis, or any combination thereof;

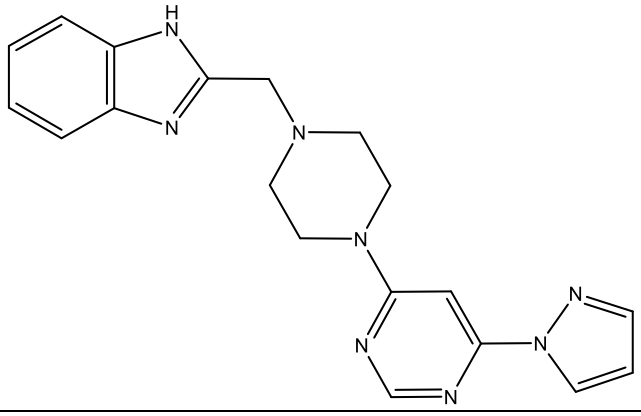
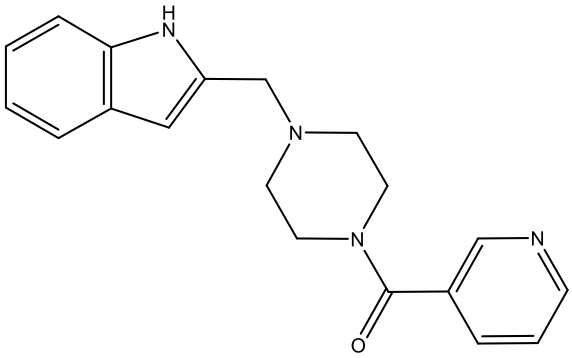
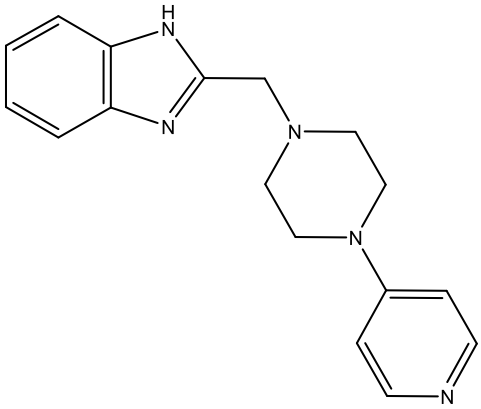
wherein the chronic liver injury results from alcoholism, malnutrition, hemochromatosis, exposure to poisons, toxins or drugs;

or any combination thereof.

13. The use of any one of claims 9 to 12, wherein said subject has a liver cirrhosis.

14. Use of a compound according to any one of claims 1 to 7 or a compound represented by any one of the following structures:

Compound Number	**Compound Structure**
205	
207	

208	
231	
253	

in the manufacture of a medicament for:

treating, suppressing, reducing the severity, reducing the risk of developing or inhibiting lung fibrosis in a subject;

treating, suppressing, reducing the severity, reducing the risk of developing or inhibiting idiopathic pulmonary fibrosis (IPF) in a subject

treating, suppressing, reducing the severity, reducing the risk of developing or inhibiting hepato-fibrotic disorder in a subject;

treating, suppressing, reducing the severity, reducing the risk of developing or inhibiting cirrhosis in a subject;

treating, suppressing, reducing the severity, reducing the risk of developing or inhibiting alcoholic steatohepatitis (ASH) in a subject;

treating, suppressing, reducing the severity, reducing the risk of developing or inhibiting non-alcoholic steatohepatitis (NASH) in a subject;

treating, suppressing, reducing the severity, reducing the risk of developing or inhibiting alcoholic fatty liver disease (AFLD) in a subject;

treating, suppressing, reducing the severity, reducing the risk of developing or inhibiting non alcoholic fatty liver disease (NAFLD) in a subject;

treating, suppressing, reducing the severity, reducing the risk of developing or inhibiting an autoimmune disease or disorder in a subject;

or any combination thereof.

15. The use of claim 14,

wherein the lung fibrosis is idiopathic pulmonary fibrosis (IPF);

wherein the hepato-fibrotic disorder is a portal hypertension, cirrhosis, congenital hepatic fibrosis or any combination thereof;

wherein the cirrhosis is a result of hepatitis or alcoholism;

or any combination thereof.

16. The compound according to any one of claims 1 to 7 for use in therapy.