Here’s what came up with my research on 1-(1-phenylcyclohexyl)piperidine and its analogs. I’ve seen there’s quite an interest in PCP analogs right now so I thought I would dig up my kind of old notes and reports (cut syntheses) and post a somewhat cumulative trip report/summary. This class of psychoactive substances are interesting for chemists and pharmacologists as some of them may be used in research on NMDA receptors, others are quite potent DARIs or act on opioid receptors almost as strong as morphine (or even stronger). Not a long poetical trip report, I rather concentrate on general things. Sometimes I wrote only differences to PCP because effects weren’t so astonishing/different much to PCP. PCP potency was sketched so potency of other drugs can be simply calculated unless they behave different. So here it goes. Get what you want from it but read disclaimer and keep in mind I’m more of a chemist than a junkie when it comes to such a class, a user is better word but I prefer plain ketamine (not that plain in fact) to wild PCE (even though I like PCE). Once again: SYNTHESES DELETED from the original pieces of documents (this also cut a lot of text and some chaos might be left over now but I added some comments here and there).

DISCLAIMER: I take no responsibility for anyone’s taking seriously all the way what’s written here. These are just general subjective opinions of my own and people I worked with. Also, I’m no “research chemical” vendor or a seller of anything per se. Do not PM me about trading.

**PCP; 1-(1-phenylcyclohexyl)piperidine**

*Dose/route:* 15 mg orally of HCl salt

*Report:* Takes on after 20–30 minutes. Good dose. Dissociative effects firstly noticed when everything seems like it’s further than it is. No aggression observed, just heartbeat speeded up. Laying and closing eyes brings CEVs at 2 h. Now body heaviness is clearly felt. Little nausea happened at 10 mg p.o. but wasn’t felt after the drug came on fully. The whole experience was accompanied by distinctive euphoria. After 4–5 h major effects subside. After effects are felt for another 12–16 h.

*Dose/route:* 10 mg intranasally of HCl salt

*Report:* Onset of effects at about 5–10 min. Effects subside at 4 h, not some major difference in duration, just the onset is faster.

*Dose/route:* 10 mg intramuscularly of HCl salt

*Report:* Effects are almost immediate as for i.m. injection. There’s some paranoia during the experience for some time, then easiness came in. There was particularly no choice if to lay or not. The experience carried on not controlled. During (pre-)anaesthetic state I felt like I lost the image of “me”, then I could look at my personality from different aspects, this led to partial depersonalization as I didn’t control what was going on and more and more images I saw as “not being myself”. Euphoria was diminished in contrast to oral administration. Everything subsided after about 3–4 h. After effects were as long as 6–8 h.

Trials with PCP led to conclusions about dosage: up to 5 mg gives a light experience, up to 10 mg gives a real knowledge what it can give and higher doses, especially 15 mg and more i.m. provide a very strong and intoxicating experience. The most “PCP” euphoria is felt after insufflating because it comes in fast and somehow I had problems getting totally anesthetized that way. These rules applied to most chemicals in this class. PCP, PCM, PCE, TCP, PCPy, TCPy etc.

### 1. Cyclohexyl ring modified analogs

*Intro:* cyclohexyl ring is probably where it all lies. One modification can totally abolish activity as well as it can give a strong analog but with different affinities for plain PCP targets. We bore in mind one successful modification could make us jump at total opioid or dissociative compounds. Combinations of two would be good too.

**1-(1-phenylcyclopentyl)piperidine**

The substance wasn’t even synthesized. Any enlargement (or the contrary action) of the cyclohexane ring gives a massive decrease in activity, so no further research makes sense.

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1 I will include my names for structures, they may not always be the same as IUPAC.

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PCP analogs (Cumulative) — Posted at bluelight.org by “adder” on 20 May 2010, 19:49

1-(4-methyl-1-phenylcyclohexyl)piperidine; 4-Methyl-PCP

*Route/dose:* 10 mg orally of HCl salt

*Report:* This drug is certainly more active than PCP. Its targets in CNS are the same for sure. On milligram basis it’s a bit more potent than PCP and I sense some light opioid activity here.

4-phenyl-4-(piperidin-1-yl)cyclohexanone; 4-Oxo-PCP

*Route/dose:* 20 mg intramuscularly of HCl salt

*Report:* This is an interesting compound. It almost completely lost its NMDA blockade effects. Instead it possesses opioid-like activity. Probably PCE analogs of this would yield very strong narcotic-like compounds. But that’s just a prediction. PCE analog could still retain NMDA antagonist properties while having strong a narcotic effect similar to that of morphine.

2-phenyl-2-(piperidin-1-yl)cyclohexanone; 2-Oxo-PCP

*Route/dose:* 20 mg intramuscularly of HCl salt

*Report:* Here lies the rub. This is some cousin of ketamine and tiletamine. Adding a keto group on position 2 or position 4 changes much how the structure of molecule looks like. This isn’t much different to 4-Oxo-PCP, it may be a bit more potent, both provide increased anesthetic and analgesic action different to PCP and the experience is short-living, yet it’s not as short as ketamine’s. The ideal chemical to get such activity would be 2-Oxo-TCE while 2-Oxo-3’-OH-PCP/2-Oxo-3’-OH-PCE would be a PCP structure keeping opioid agonist with strong anesthetic properties.

2. Aromatic ring modified/replaced analogs (and pyrrolidine analogs)

a. Aromatic ring replacements and piperidine modifications

PCPy; Rolicyclidine; 1-(1-phenylcyclohexyl)pyrrolidine

*Route/dose:* 10 mg intranasally of HCl salt

*Report:* Rolicyclidine is easier than plain PCP. It’s even sedative, there are no bothering stimulant effects here but dissociative properties are present totally. From the dose I took it’s safe to say it’s as potent as PCP or a little stronger but seems to be easier to trip on. Some methylation on pyrrolidine ring doesn’t abolish activity, 3,3-dimethyl analog is still potent.

TCP; Tenocyclidine; 1-[1-(thiophen-2-yl)cyclohexyl]piperidine

*Route/dose:* 5 mg intranasally of HCl salt

*Report:* Onset is as fast as 5 minutes, very similar to PCP’s. The drug overpowers PCP totally. Not only in dosage but it’s also less euphoric and more insightful as a dissociative. It definitely causes stronger blockade of NMDA channel and less euphoric effects are easily explained by its distinctive less sigma activity.

TCPy; 1-[1-(thiophen-2-yl)cyclohexyl]pyrrolidine

*Route/dose:* 10 mg intranasally of HCl salt

*Report:* By the drug action it can be easily stated its potency is more like PCP, loses wide to TCP. Under impression of how PCPy worked, a mellow, sedative dissociative, TCPy leaves us wondering what causes are.

BCP or BTCP; Benocyclidine; 1-[1-(benzothiophen-2-yl)cyclohexyl]piperidine

Some more work were done to substitute standard benzene ring. Not because of unknown interest. At the time the decision on synthesis was made, at least 4 papers suggested it’s a totally different PCP analog. A very potent and selective DARI (IC$_{50}$ = 7 nM, 21 Jan 2017 editor’s note: The text of this post has been lightly edited and reformatted from the original. Some of author’s claims have been challenged. See [http://www.bluelight.org/vb/threads/504286-PCP-analogs-(Cumulative)] for more information.
ADDED: http://www.ncbi.nlm.nih.gov/pubmed/3384005 with very little affinity for NMDA channel (K<sub>0.5</sub> = 6 μM = 6,000 nM) cited as a pharmacophore for DARI stimulants.

Route/dose: 30 mg intranasally as HCl salt (ADDED: I had too little information to draw any range of dosage and I was never really into stimulants, I reported basing on my experience too but much more is based on other team members and “testers” experiences)

Report: Dose adjustment wasn’t good at all. This was the hardest part (too little, titrating may be long, too much, I may never synthesize anything in my life again). This should be definitely rather vaporized than insufflated. It would be easy to find an appropriate dosage for this but there are many reasons why this ends here, in “bin”. One of the reasons is I got satisfied with my interest in this drug. It’s a pure stimulant with DARI properties overpowering totally any activity at NMDA. But it’s not like BCP is going to flood black market. Why? The structure is quite simple, the synthesis is easy but considering easiness of syntheses of drugs with “amphetamine core” in them, this will be at most a scarce product at some RC vendors. And they’re getting more and more lazy, this stuff is available for over 20 years. (ADDED: like we see no RC vendors sell this widely, keep in mind, this was synthesized at the end of 2007 at best, I let it lay, no idea if it was used by any of people who themselves offered as “testers”, it wasn’t further evaluated by us at least but it’s not how it shapes us as a recreational drug that gives some wonders; yet it was left as “marked” so something was to be done with it)

BCP, aside from being a pharmacophore for DARI, gives plenty to think about the whole class of phencyclidine analogs. What is known about PCP and its analogs pharmacodynamics? They bind to PCP receptor, they block NMDA channel, they bind to sigma receptors, and finally they act on dopamine receptors via dopamine uptake blockade. The last one we know thanks to BCP but not only. A BCP analog in which piperidine ring was substituted by pyrrolidine ring (i.e. 1-[1-(1-benzothiophen-2-yl)cyclohexyl]pyrrolidine or I call it BCPy) binds to the cocaine receptor site being more potent than cocaine itself, it also binds to some other site on dopamine transporter. The question of stimulants (that are popular) substitution is open and comes back. If drug bosses had better counselors, they would have introduced this long time ago (it’s got potency and it’s legal). On the other side as there are substitution drugs for opioid addicts like methadone or buprenorphine then why shouldn’t cocaine addicts get something that might help them get out of their misery? Yet we see no BCP or BCPy evaluation in human. The latter drug won’t be synthesized just for the sake of not wasting time if it’s going to bring about the same thing, just stronger (here BCPy > BCP, PCPy > PCP; pyrrolidine ring might be generally more dopaminergic but on the other hand if PCPy seems to be more sedative and easier than PCP; this just proves PCP analogs chemistry is complex despite simpleness of their structures).

b. PCP with Single groups on benzene ring

Info: These compounds are generally just called e.g. 3-MeO-PCP or 4-MeO-PCP. I call them 3'-MeO-PCP or 4'-MeO-PCP to differentiate them from PCP substituted at cyclohexane ring.

3'-MeO-PCP

Report: It possesses activity very similar to PCP. It also proves to have some agonist effect on opioid receptors. Nonetheless, generally I consider this not much different from PCP in quality as well after trying 10 mg and 15 mg of HCl salt.

3'-HO-PCP

Report: Now this is a winner. 3 mg of this compound taken orally as HCl salt knocked me out. This one must have some serious affinity for morphine sites because I could take less morphine. I leave it marked because its analogs may be serious opioid agonists. Officially, 3'-HO-PCP is like 10% of morphine. The only problem with this compound is that it’s 8 times as potent as PCP at PCP receptor so opioid activity (although it’s hundreds much more pronounced than with PCP) is little when compared. Yet I love this one.

3'-Amino-PCP

Report: Just like 3-HO-PCP this compound possesses similar properties. It should be noticed that while -OH group is a dead end (methoxy group acts similarly but is already much more weaker), a primary amine can be still modified and literature mentions such compounds being even more active than this one.

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3’-Bromo-PCP

Report: There are basically no effects.

3’-Nitro-PCP

Report: As with 3-bromo-PCP there are no effects. This was just a simplest test to carry on proving any electronegative groups at position 3 make PCP compounds completely inactive.

4’-Fluoro-PCP

Report: Synthesized for the sake of being sure electronegative groups are not wanted on aromatic ring but this time fluorine was put there. And voila! Here comes an active compound amazingly. It’s not very potent. There weren’t enough reports to gain information about potency but it might be along the lines of 4’-MeO-PCP even.

4’-Nitro-PCP

Report: Went with the simplest method to find out if it’s going to be anywhere 4’-Fluoro-PCP. Sadly, this one is not active and from other reports 4’-Bromo-PCP is also inactive so it’s to be let go.

4’-Methyl-PCP

Report: This is disappointing. This drug is totally inactive.

4’-MeO-PCP

Report: This one is active. Reduced potency compared to plain PCP but an interesting material.

4’-HO-PCP

Report: Reported to be somewhat different to PCP but the potency is almost identical. Somehow alkyloxy group keeps up equally well here. 4’-HO-PCP is just stronger.

2’-Chloro-PCP

Report: Synthesized just for the sake of clarity that 2-halogenated derivatives have decreased potency, it’s easily seen in ketamine. The other factor that diminishes ketamine potency is carbonyl group at position 2 in cyclohexane ring obviously. But 2’-chloro compounds give something to more analgesic profile of the drug.

2’-MeO-PCP

Report: This compound is very weak. Placing 2-methoxy group on PCE wouldn’t help much either. But it might be used for lighter trips. Actually ketamine analog with methoxy group instead of chloro would be interesting. It should have similar potency.

Conclusion: position 3 modifications are what brings any satisfying results. Some 4-substituted compounds are active but I’ll call them “lighter” PCP for people fearing PCP might make them schizophrenic. Not that they’re worse in action, some might even prefer them to regular PCP but that’s another story just like I was excited when I got my hands on 3’-HO-PCP.

3. Piperidine ring modifications/replacements (pyrrolidine analogs in section 2)

PCE; Eticyclidine; N-ethyl-1-phenylcyclohexanamine

Route/dose: 15 mg intranasally of HCl salt

Report: Really strong, more potent than PCP for sure so 15 mg blows my mind. The oddity is that salts of various PCP analogs are odorless, this one even as a salt smells terribly. I noticed more nausea and more body load. A good compound though in my opinion.

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Wins with PCP, it’s like 1.5×PCP maybe but it’s really hard to point out an equivalent dose, these are so close. 15 mg is alright for me, total blast but it’s amazing. If not only for that smell.

**PCM; N-methyl-1-phenylcyclohexanamine**

*Route/dose:* 15 mg intranasally of HCl salt

*Report:* Nothing else beside what I would have expected from this compound. It’s safe to say it’s as potent as PCP. However, as with PCE, there’s a distinctive feel to it. Piperidine ring broken compounds have somewhat different intoxicating effects from PCP, more body load, different taste, different smell.

**PCA; Phencyclamine; 1-phenylcyclohexanamine**

**PCPr; N-propyl-1-phenylcyclohexanamine; 1-phenyl-N-propylcyclohexanamine (IUPAC)**

**PCiPr; N-isopropyl-1-phenylcyclohexanamine; 1-phenyl-N-(propan-2-yl)cyclohexanamine (IUPAC)**

**PCBu; N-butyl-1-phenylcyclohexanamine (IUPAC)**

*Report:* I was informed PCA must be around 50% potency of PCP. No wonder. There was found some rule here. N-ethyl is the best, a primary amine reduces potency as lengthening of the alkyl chain. N-propyl (PCPr) and N-isopropyl (PCiPr?) are already similar in potency to PCP, N-butyl (PCBu) is less potent, all in respect to PCP. N,N-dimethyl compound loses activity dramatically

**4-methyl-1-(1-phenylcyclohexyl)piperidine**

*Route/dose:* 15 mg intranasally of HCl salt

*Report:* Dose not adjusted properly, getting only light PCP-like effects.

*Route/dose:* 30 mg intranasally of HCl salt

*Report:* Tuned in finely. The dose is finally proper. Not a completely stoning PCP-like effect but it’s close. Anyway, it’s a disappointment this one is only around 0.3 as strong as PCP while even plain PCA with a primary amine is 50% potency of PCP. The same goes for PCPy analogs when they’re methylated at position 3 of pyrrolidine ring (both 3-methyl and 3,3-dimethyl are active but have somewhat reduced potency).

**4-(1-phenylcyclohexyl)morpholine**

*Route/dose:* 100 mg insufflated of HCl salt

*Report:* This is a total disappointment concerning its potency. This dose is for dissociative state lovers. The compound isn’t very potent but still more potent than ketamine, yet it doesn’t resemble it in respect to anesthesia or analgesia.

### 4. Compounds with multiple modifications

Well known compounds with carbonyl group at position 2 are ketamine and tiletamine. The latter being used in veterinary in combination with zolazepam or xylazine. Changes in these drugs diminish potency on weight basis but advantages are analgesic action involved and decreased duration so these compounds can be used in short-term anaesthesia. Breaking the piperidine ring, alkylling the amine mixed with putting other groups here and there yield interesting compounds.

**Tiletamine; 2-(ethylamino)-2-(thiophen-2-yl)cyclohexanone**

*Route/dose:* 100 mg intramuscularly of HCl salt

*Report:* The salt has no odor and appears as white crystals. First effects are noticed in 1–2 minutes as with ketamine. This is certainly stronger than ketamine. 100 mg placed me in tiletamine’s version of K-Hole rapidly. It behaved kind of more wild than ketamine. But
there was that calming effect of ketamine still there like everything’s going to be alright. Effects wore off after 1–1.5 h and I felt buzzed for another 4 h.

I’ve injected tiletamine intravenously too but I don’t advise anyone to inject intravenously any of these drugs so I won’t post about it.

**BDPC; Bromadol; E-4-(bromophenyl)-4-(dimethylamino)-1-phenethylcyclohexanol**

**Intro:** There was a much debate about value of aim at it as enough sources on it exist and suggest it’s very strong. Knowing it’s an opioid agonist and having struggled with 4-carboxamethoxy-3-methylfentanyl earlier, the answer was “yes”.

Like it has been determined before N,N-dimethyl analogs are not very active and 4'-halogenated analogs were inactive but 4'-Fluoro, this shouldn’t be active at all. Another thing is placing a hydroxy group anywhere on cyclohexane ring makes any compound devoid of activity. Now BDPC has a bromine at para-position, furthermore it’s a N,N-dimethyl derivative and it’s got one -OH group making it an alcohol. But there is an interesting group in chemistry of pharmacologically active substances â€” phenylethyl. What’s more interesting patent papers on 4-substituted cyclohexanols have issue dates as 1980s. Having gone through some papers, we got to work. Synthesis might be time consuming but it isn’t a tough one.

**Route/dose:** 0.01 mg (10 µg) intravenously of HCl salt (ADDED: keep in mind I had a tolerance to morphine and could shoot i.v. 160 mg of morphine no problem)

**Report:** Kicked in quickly. Definitely has its own taste of opioid action. The dose might be raised to 15 µg, this gave an overview anyway. Produces very strong analgesia, there’s an opioid euphoria but at the same time kind of different type of sedation is present. Breathing is alright for me, slowed down so might be a problem for the unexperienced. It doesn’t last long and considering its power it definitely has an addictive potential. I found it not even near classic morphine high though. 15 µg was tried. A person defined it as overwhelming, itching was present and it gets wondering. It’s an atypical opioid agonist with huge potency, yet it has a lot of some semi-synthetic morphine-skeleton retaining drug. There is no 3-carbon spacer between nitrogen and the aromatic ring and it’s rule no. 1 for opioid activity in most known opioid, apart from fentanyls (3-carbon and nitrogen spacer) pethidines, prodines, bemidones, methadone and its analogs, and even tramadol all follow this rule.

**Ethylketamine; 2-(2-chlorophenyl)-2-(ethylamino)cyclohexanone**

**Route/dose:** 100 mg intramuscularly of HCl salt

**Report:** The name is obviously wrong but it’s easy to know what hides there. This is just ketamine with ethyl on amine (just like in case of PCE). This obviously had an impact on drug potency. It’s not stronger by a factor of 2 or 3 but 100 mg i.m. knocked me out totally and I was Ethyl-K-holing for about an hour. So it didn’t really alter duration of experience. It’s got already a broken piperidine ring, ethyl is known to be the best when it comes to potency so here it is, get an S-enantiomer and you’ve got a winner.

**2-(2-methoxyphenyl)-2-(methylamino)cyclohexanone**

**Route/dose:** 100 mg intramuscularly of HCl salt

**Report:** This is ketamine with chloro group substituted by methoxy group. Like predicted before synthesis they are really similar. With such a dose i.m. it was easy to go K-holing. What's the difference here? Chlorine is very electronegative but not as much as fluorine. This is yet to be examined what concrete impact has methoxy group here because from subjective effects it's really hard to tell if it's more potent or less potent. Actually I see no difference. Well, at least I can’t report on any. This is as good as plain ketamine and if ketamine is easier to synthesize why bother?

That’s all I recovered from those times. There are many things leaving me wondering when I read this now but it was a few years ago and all I remember there wasn’t much time spared for PCP analogs chemistry and pharmacology.

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