Mini Review

Drug Treatments for Human Suicide

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Received: July 22, 2020; Published: August 05, 2020

Abstract

Despite several categories of drug licensing, chemotherapeutic drugs are not always the only choice for suicide behavior treatments in a long-term. The only reason for drug utility limitation is lack of highly effective, long-lasting, completely targeted drugs for therapeutic strategies in the clinic. This article emphasizes broader-ranges of neuroscience (neuropathology, neuropsychiatry, neural pharmacology and drug toxicology) in the clinic. The knowledge of future trends of drug development and clinical convention of suicide treatments are also highlighted.

Keywords: Mood Stabilizer; Lithium; Antidepressants; Anti-Psychosis; Suicide Prevention; Medicinal Chemistry; Central Nerve System; Human Suicide

Background

Human suicide is a high mortality event globally (2% of human deaths). Comparing with other disease categories, chemotherapeutics is not the only choice for long-term of managements of human suicides in the clinic. The only reason for relatively small-scale drug utility is from lack of specific, highly effective, long-lasting and low toxicity chemotherapeutic drugs. To get into the core of this therapeutic setback, the long history of human suicide study is given to help our understanding the slow evolution in therapeutics of this matter [2-4] (Table 1).

<table>
<thead>
<tr>
<th>Timeline</th>
<th>Major discovery</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ancient Greece</td>
<td>Four elements and melancholy (excess of black bile)</td>
</tr>
<tr>
<td>Aretaeus of cappadocia</td>
<td>Clinical feature of depressive</td>
</tr>
<tr>
<td>Middle age</td>
<td>Patients with delusion</td>
</tr>
<tr>
<td>16th to 17th</td>
<td>Clinical diagnosis and behavior abnormal</td>
</tr>
<tr>
<td>18th</td>
<td>Nervous (animal spirits)</td>
</tr>
<tr>
<td>19th</td>
<td>Psychiatric symptoms</td>
</tr>
<tr>
<td>20th</td>
<td>Mood disorder and electroplexy and psychosurgery</td>
</tr>
</tbody>
</table>

Table 1: Historic order of mood disorder knowledge discovery (suicide associated).

Citation: Da-Yong Lu and Ting-Ren Lu. “Drug Treatments for Human Suicide”. EC Pharmacology and Toxicology 8.9 (2020): 06-10.
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Chemotherapeutic deficiency in clinical trials

Presently, licensed drugs for suicide prevention and treatment are limited and unsatisfactory (Table 2) [5,6]. Most of them have moderate-to-severe side-effects for most human beings, especially to pregnant women and children. In addition, most patients will recur after drug discontinuation longer than half year [5-8]. On the other side, different types of drugs are active to different patients. This drug selective strategy is difficult to practice in the clinic.

<table>
<thead>
<tr>
<th>Major symptoms</th>
<th>Drug categories</th>
<th>Therapeutic property</th>
<th>Toxicity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depression</td>
<td>Antidepressants such as, SSRI</td>
<td>Moderate/less specific</td>
<td>Moderate</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Relatively specific</td>
<td>Low toxicity/suicide risk</td>
</tr>
<tr>
<td>Mania</td>
<td>Lithium Mood stabilizers Anti-psychosis</td>
<td>Moderate/less specificity</td>
<td>Toxic when long-term utilities Carcinogenesis</td>
</tr>
</tbody>
</table>

Table 2: Therapeutic drug categories for bipolar mood disorder.

Genetics

Since most suicide events might come from patient’s depression or mania [9], the antidepressant development and marketing is facing great medical challenge. But, many psychiatric-moderating drugs need to categorize to different kinds of suicide patients and commonly have moderate-to-severe toxicities, such as carcinogenesis activity, especially to fetus of pregnant women [6]. To strengthen these kinds of knowledge, neuropsychiatric genetics must be introduced.

The biggest efforts of drug experimental developments and clinical applications must transform from symptom management into genetic/molecular targeting-including cerebral image monitors and large sized animal models (dogs or chimpanzees) [10]. Neuropsychiatric and neuropathology study must be studied first [11-19]. In search for key drug targets, genetic study is indispensable.

Drug development

High-quality drug development plays key roles for human suicide managements. Since human mental diseases contain different categories and several dimensions [20,21]. It is difficult to be evaluated by human suicide behaviors and associated genes. Until now, there is no good animal models and clinical diagnosis for specifically targeting against different psychiatric symptoms and biomarkers. Only deep genetic/molecular evaluation and approaches can answer this enigmatic question of human suicide (algorithmic models, computational analysis and mathematic theorems) [22-26].

In the future, suicide/mental illness drug development chain must be updated-including etiologic/pathogenic studies [20-24], pharmaceutical study [27], mathematical-computational analysis (In silico) [28,29] and natural chemotherapeutic drugs [30-33]. In the future, modern drug for different mental diseases will be licensing and categorization against different suicide behaviors-uniform experimental (in vitro and in vivo) and clinical models.

Drug combination

Drug combination is one of the commonest therapeutic paradigms for a lot of disease treatments, such as HIV, cancer and so on [34-38]. From early literature, drug combinations are commonly higher therapeutic-index in the clinic. However, drug combination could be either beneficial or more toxicity in the clinic. As usual, hidden law (central dogma) discovery of drug combinations against suicide/
mental illnesses can transform from past experience into scientific-based drug combination paradigm [37-40]. To establish these clinical paradigms, the combination study could be mathematics [37,38], mechanism analysis [39] and clinical evaluation [40].

**Conclusion**

Drug therapeutics for suicide genetics is lag behind comparing with other diseases in pathologic bases. Genetic/molecular suicidal pathology and pharmacology has not been widely identified and translated into highly effective targeted drug therapeutics. Thus, we must emphasize genetic/molecular studies first. To attain this goal, in depth mental illness study is indispensable.

**Bibliography**

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Volume 8 Issue 9 September 2020
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