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The Ethical Justification of Equal Candidacy for Organ Transplantation in Alcoholic Patients

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ABSTRACT

An increasingly blurred understanding of the distinctive challenges posed to transplantation medicine and, by extension, public health, by the debilitating reality of alcoholism suggests a critical need to revisit the relationship between causality, candidacy, and culpability in light of substance addiction. This essay grounds its arguments in two, straightforward premises: (i) compassionate medical practice - understood as the sympathetic willingness to enter into the existential suffering of another in order to ameliorate the anguish invoked by disease - rests on the fiduciary relationship shared between provider and patient; and, (ii) allocating medical goods according to moral desert rather than existential disposition undermines the fundamental nature of medicine and the functioning of the provider-patient relationship. Drawing from this syllogism, the aim and proposal of this essay posits the argument that employing moral desert as an allocation criteria to inhibit alcoholic patients from equal consideration and treatment is, and ought to remain, at odds with the fundamental nature of medicine and the functioning of the provider-patient relationship.

Keywords: Transplantation Ethics, Alcoholism, Moral Responsibility, Addiction Neuroethics

1. INTRODUCTION

1.1 Overview

The harmful use of alcohol (HUA) causes approximately 2.5 million global deaths each year, including 320,000 individuals between the ages of 15 and 29.¹ The third leading risk factor of ill health world-wide, alcohol led to the death of one person every two minutes in the Americas in 2002.² In the same year, approximately 5.4 percent of all deaths in the Americas were attributed to alcohol, compared to the 3.7 percent global figure – some 68 percent greater than the world average. Alcohol consumption is related to over 60 health conditions, ranging from those resulting from excessive alcohol consumption during pregnancy to intentional and non-intentional injuries, cancers, cardiovascular diseases, liver disease, and neuropsychiatric conditions, including alcohol dependence.³ An array of alcohol-related problems has a devastating impact on individuals and their families, and can seriously inhibit community life. HUA is one of the most common modifiable and preventable risk factors for major communicable

1. World Health Organization (WHO), *Global Strategy to Reduce the Harmful Use of Alcohol* (Geneva: WHO Press, 2010), 1-38; see especially p. 3.

2. Pan American Health Organization, *Alcohol and Public Health in the Americas: A Case for Action* (Washington, DC: Pan American Health Organization, 2007), 1-50; see especially p. 1.

3. Pan American Health Organization, *Alcohol and Public Health*, 1-50; see especially p. 1.

diseases, and emerging evidence suggests that alcohol abuse contributes to health burdens caused by tuberculosis and HIV/AIDS.⁴

Alcohol has several and contrasting associations. A glass of wine can symbolize love, friendship, relaxation, and enjoyment. It can represent romance, success, beginnings and endings, good news and good company. But sadly, these occasions contrast with the associations of alcohol with drunken violence in towns and cities, terminal disease on medical wards, debt in families, and death on roads. It also contrasts with the enslavement that is alcoholism, or alcohol addiction. Addiction is concerned with the subjective experience of continuing in a habitual behavior which is recognized at some level (by the subject or others) as being undesirable. It is a concern with behavior in which motivation appears to be disordered, or in which the usual human experience of free choice appears to be violated.⁵

Nevertheless, medical and moral arguments have been advanced in response to the question of whether alcoholic patients with liver disease should be given lower priority for liver transplantation than those whose disease is not alcohol related. According to the medical argument, alcoholics should be given lower priority because their survival rate is lower, owing to a presumed probability of relapse into alcohol abuse. According to the moral argument, alcoholics should be given lower priority because their moral vice of heavy drinking makes them responsible for their condition and effectively forfeits their claim to extraordinary medical treatment, such as transplantation.⁶ Contrary to these flawed positions, this essay will argue in favor of equal consideration for organ transplantation in alcoholic patients on both medical and moral grounds.

1.2 Analytical Method

An increasingly blurred understanding of the distinctive challenges posed to transplantation medicine and, by extension, public health, by the debilitating reality of alcoholism suggests a critical need to revisit the relationship between causality, candidacy, and culpability in light of substance addiction. The be sure, the issues of immediate import to the conversation over the licitness of equal candidacy for organ transplantation in alcoholic patients in light of changing scientific, medical, and moral traditions are manifold, and any singular analysis of topics, no matter how sweeping, will unavoidably fall short of adequacy. This essay thus endeavors to briefly address but three: (i) alcohol addiction, (ii) transplantation candidacy, and (iii) moral responsibility.

It grounds its arguments in two, straightforward premises: (i) compassionate medical practice – understood as the sympathetic willingness to enter into the existential suffering of another in order to ameliorate the anguish invoked by disease⁷ – rests on the

4. WHO, *Global Strategy*, 1-38; see especially p. 3

5. Christopher C. H. Cook, *Alcohol, Addiction and Christian Ethics* (New York: Cambridge University Press, 2006), 1-36; see especially pp. 9-20.

6. Walter Glannon, "Responsibility, Alcoholism, and Liver Transplantation," *Journal of Medicine and Philosophy* 23, no. 1 (1998): 31-49; see especially p. 31.

7. "Existential suffering" is an intentionally broad term employed herein to include physiological, emotional, psychological, spiritual, and other idiosyncratic manifestations of suffering.

fiduciary relationship shared between provider and patient; and (ii) allocating medical goods according to moral desert rather than existential disposition undermines the fundamental nature of medicine and the functioning of the provider-patient relationship. Drawing from this syllogism, the aim and proposal of this essay is to examine the notions of alcohol addiction, transplantation candidacy, and moral responsibility with the intention of positing the argument that employing moral desert as an allocation criteria to inhibit alcoholic patients from equal consideration and treatment is, and ought to remain, at odds with the fundamental nature of medicine and the functioning of the provider-patient relationship.

To secure the justification of this thesis, the essay will move in four parts. First, it will address alcoholism as a public health issue, including a specific analysis of (i) the pathophysiology and effects of alcoholism, (ii) alcoholism as a public health threat, and (iii) global strategies to address alcoholism. Second, it will address the neuroethics of alcoholism, including a specific analysis of (i) the addicted brain, (ii) the neurobiological basis of moral reasoning, and (iii) autonomy and alcoholism. Third, it will address organ transplantation and alcoholism, including a specific analysis of (i) liver transplantation and alcoholism, (ii) transplant outcome and survival benefit for alcoholic patients, and (iii) predictors of alcoholic relapse after liver transplantation. Finally, it will address moral responsibility and alcoholism, including a specific analysis of (i) free will, moral choice, and alcoholism, (ii) moral responsibility and alcoholism, and (iii) the provider-patient relationship in the context of alcoholism.

2. ALCOHOLISM AS PUBLIC HEALTH ISSUE

2.1 Alcoholism: Pathophysiology and Effects

Addiction treatment trials often employ the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV-TR) definition of alcohol use disorders (AUD) to define study participants. The definition of alcohol dependence requires significantly harmful impact caused by at least three of seven target conditions within a single calendar year. The dependence symptoms include tolerance; withdrawal; increased amounts of alcohol consumed over time; ineffective efforts to reduce use; interference with professional or personal life; significant amount of time spent obtaining, using, and recovering from alcohol; or continued use of alcohol despite harmful sequelae.⁸

The DSM-V has combined criteria for alcohol dependence and abuse into a single term: AUD.⁹ Craving was added to the diagnostic criteria and at least two target conditions are required for AUD. Although approved pharmacologic treatment options for patients with AUD are limited in number, recent trials describe several alternative approaches to reducing alcohol consumption. These include the use of antipsychotics,

8. Hence, alcohol abuse is defined broadly and requires the presence of at least one of the four abuse criteria for diagnosis. See Robin C. Wackernah, Matthew J. Minnick, and Peter Clapp, "Alcohol Use Disorder: Pathophysiology, Effects, and Pharmacologic Options for Treatment," *Substance Abuse and Rehabilitation* 5 (2014): 1-12; see especially pp. 1-2.

9. Wackernah et al., "Alcohol Use Disorder," 1-12; see especially p. 2.

antidepressants, anticonvulsants, and other drugs under the rationale that these substances target neurotransmitter systems that have been shown to undergo changes with chronic exposure to alcohol.¹⁰

The acute and chronic effects of alcohol on brain pathology help to rationalize the investigations of psychotropic drugs in the treatment of AUD.¹¹ Alcohol, like other addictive drugs, stimulates release of the neurotransmitter dopamine from cells originating in the ventral tegmental area (VTA).¹² Following exposure to alcohol, dopamine released into the nucleus accumbens (NAc) and prefrontal cortex has been postulated to reinforce drinking behaviors or make the experience of drinking more salient. This electrochemical activation of neurons is controlled by a balance between excitatory and inhibitory neurotransmitters.¹³

Alcohol acutely inhibits the flow of ions through N-methyl-D-aspartate (NDMA)-type glutamate receptors and enhances the activity of glutamate, γ -aminobutyric acid (GABA) receptors channels, producing an overall inhibitory effect on neurons.¹⁴ The desire to relieve anxiety and negative sensations of withdrawal can contribute to relapse drinking and compulsive behaviors that characterize alcohol dependence. Pharmacologic strategies to reduce drinking in patients with AUD attempt to remedy the imbalance between excitatory and inhibitory pathways, and relieve the intense craving for alcohol brought about by neuroadaptation.¹⁵

Even in healthy individuals, alcohol is toxic to most organ systems at doses above one to two drinks per day.¹⁶ Long-term exposure to alcohol generally increases the risk of damage to gastrointestinal, cardiovascular, immune, nervous, and other systems. Alcohol can promote gastrointestinal bleeding through inflammation of the esophagus and stomach, or through vomiting that can damage gastrointestinal mucosa. Acute pancreatitis is more prevalent in alcoholics than in the general population and can progress to chronic disease or pancreatic cancer with prolonged abuse. Accumulation of fat in the liver as a result of decreased oxidation of fatty acids and other metabolic changes can progress to fatty liver disease, alcohol-induced hepatitis, and cirrhosis.¹⁷

The effects of heavy drinking can range from left ventricular impairment and arrhythmia to heart failure as a result of limited contractility of the heart muscle. Binge

10. Wackernah et al., "Alcohol Use Disorder," 1-12; see especially p. 2.

11. In particular, neurotransmitter pathways involved in learning and reward have proven to be effective targets, based on mechanisms of the action of two currently approved AUD drugs: acamprosate and naltrexone. See Wackernah et al., "Alcohol Use Disorder," 1-12; see especially p. 2.

12. VTA is a component of the neuronal circuit known as the mesolimbic dopamine system that has been associated with motivation and reward. See Wackernah et al., "Alcohol Use Disorder," 1-12; see especially p. 2.

13. Changes in the GABA system contribute to the anxiogenic and aversive effects of alcohol withdrawal and can persist over long periods of abstinence from alcohol. See Wackernah et al., "Alcohol Use Disorder," 1-12; see especially pp. 2-3.

14. Wackernah et al., "Alcohol Use Disorder," 1-12; see especially pp. 2-3.

15. Wackernah et al., "Alcohol Use Disorder," 1-12; see especially p. 3.

16. Wackernah et al., "Alcohol Use Disorder," 1-12; see especially p. 3.

17. Wackernah et al., "Alcohol Use Disorder," 1-12; see especially p. 3.

drinking can produce atrial and ventricular arrhythmias, even in individuals who have no other evidence of heart disease – a syndrome known as “holiday heart.”¹⁸ Alcohol-dependent individuals may experience peripheral neuropathy characterized by numbness and tingling, especially in the hands and feet. A progressive neurological syndrome that affects walking and posture, often accompanied by nystagmus, can result from atrophy of the cerebellum due to alcohol toxicity. Less common are neurologic syndromes that result from thiamine deficiency secondary to heavy drinking. Wernicke’s syndrome consists of encephalopathy, uncoordinated muscle movement, eye and muscle weakness; and Korsakoff’s syndrome is characterized by amnesia.¹⁹

2.2 *Alcoholism qua Public Health Threat*

HUA has serious effects on public health and is considered a primary risk factor for poor health globally. HUA is broad and includes drinking that poses debilitating health consequences for the drinker and society at large, as well as the patterns of drinking associated with adverse health outcomes. HUA compromises both individual and social development, and can ruin the lives of individuals, devastate families, and damage the social fabric of communities. As mentioned above, HUA is a significant contributor to the global burden of disease and is listed as the third leading risk factor for premature deaths and disabilities in the world.²⁰

The degree of risk for HUA varies with age, sex, and other biological characteristics of the consumer as well as with the setting and context in which drinking takes place. Some vulnerable and at-risk groups have increased susceptibility to the toxic, psychoactive, and dependence-producing properties of ethanol. At the same time, low risk patterns of alcohol consumption at the individual level may not be associated with occurrence or significantly increased probability of negative health and social consequences. Reducing HUA by effective policy measures that provide a relevant infrastructure is much more than a public health issue. It is also a developmental issue, since the level of risk in developing countries is much higher than in high-income countries where individuals are protected by laws and interventions.²¹

A substantial scientific knowledge base exists on the effectiveness and cost-effectiveness of strategies and interventions to prevent and reduce alcohol-related harm.²² Although much of the evidence comes from high-income countries, the results of meta-analyses and reviews provide sufficient knowledge to inform policy recommendations. With improved awareness, there are increased responses at regional, notational, and international levels. Yet these responses are often fragmented and do not always correspond to the magnitude of impact on health and social development.²³ The present commitment to reducing HUA provides an excellent opportunity for improving health

18. Wackernah et al., “Alcohol Use Disorder,” 1-12; see especially p. 3.

19. Wackernah et al., “Alcohol Use Disorder,” 1-12; see especially p. 3.

20. WHO, *Global Strategy*, 1-38; see especially p. 5.

21. WHO, *Global Strategy*, 1-38; see especially p. 3.

22. WHO, *Global Strategy*, 1-38; see especially p. 6.

23. WHO, *Global Strategy*, 1-38; see especially p. 6.

and social well-being and for reducing the existing disease burden attributed by alcohol. There are at least seven considerable challenges that must be accounted for in national and global public health initiatives and programs, of which this essay will address four.²⁴

A first challenge is increasing global action and international cooperation.²⁵ The current health, cultural, and market trends worldwide indicate that HUA will remain a global health issue. These trends should be recognized, and appropriate responses should be implemented at all levels. Second, intersectoral action must be ensured.²⁶ The diversity of alcohol-related problems and measures necessary to reduce alcohol-related harm suggests the need for comprehensive action across numerous sectors. Policies to reduce HUA must reach beyond the health sector and appropriately engage sectors such as development, justice, social welfare, trade, education, and others.²⁷

Third, appropriate attention must be accorded to this issue.²⁸ Preventing and reducing HUA is infrequently prioritized by decision makers despite compelling evidence of its debilitating public health effects. Unless this problem is given the attention it deserves, the spread of harmful drinking practices and norms will continue. Fourth, equity must be a focal point.²⁹ Population-wide rates of drinking alcoholic beverages are markedly lower in poor societies than in wealthier ones. However, for a given amount of consumption, poorer populations often experience disproportionately higher levels of alcohol-attributed harm. This suggests an urgent need to develop and implement effective policies and programs that reduce such social disparities.³⁰

2.3 *Global Strategies to Address Alcoholism*

National and international efforts can produce better results when they are supported by regional and global action within agreed policy frames. Hence, the purpose of an effective global strategy is to support and compliment public health policies in Member States. The vision behind the global strategy to reduce alcohol-related harm is improved health and social outcomes for individuals, families, and communities with considerably reduced morbidity and mortality due to HUA. A robust global strategy will promote and support local, regional, and global actions to prevent and reduce HUA. One global strategy, developed by the World Health Organization and endorsed by the Sixty-third World Health Assembly,³¹ aims to guide action at all levels. These recommendations set priority areas for global action, and offer a portfolio of policy options and measures to consider for implementation. As such, they can be nuanced as appropriate at the national level, taking into account relevant circumstances, such as

24. Those not discussed include balancing differing interests, considering context in recommending actions, and strengthening information. See WHO, *Global Strategy*, 1-38; see especially pp. 6-7.

25. WHO, *Global Strategy*, 1-38; see especially p. 6.

26. WHO, *Global Strategy*, 1-38; see especially p. 6.

27. WHO, *Global Strategy*, 1-38; see especially p. 6.

28. WHO, *Global Strategy*, 1-38; see especially p. 6.

29. WHO, *Global Strategy*, 1-38; see especially p. 7.

30. WHO, *Global Strategy*, 1-38; see especially p. 7.

31. WHO, *Global Strategy*, 1-38; see especially p. 3.

religious and cultural contexts, public health priorities, and existing resources, capacities, and capabilities.³²

Five aims comprise the WHO's global strategy.³³ First, it suggests raised global awareness of the magnitude and nature of health, social, and economic problems caused by HUA, and increased governmental commitment to address HUA. Second, it suggests a strengthened knowledge base around the magnitude and determinants of alcohol-related harm and on effective interventions to reduce and prevent such harm. Third, it suggests increased technical support to, and enhanced capacity of, Member States for preventing HUA and managing AUDs and associated health conditions.³⁴ Fourth, it suggests strengthened partnerships and better coordination among stakeholders and increased mobilization of resources necessary for appropriate and coordinated action to prevent HUA. Finally, it suggests improved systems for monitoring and surveillance at different levels, and more effective dissemination and application of the information for advocacy, policy development, and evaluation purposes. Achieving these five objectives requires local, regional, and global actions on the levels, patterns, and contexts of alcohol consumption and the broader social determinants of health.³⁵

The protection of population health by preventing and reducing HUA is a public health priority. To this end, the WHO offers eight principles to guide the development and implementation of policies at all levels, of which this essay will address five.³⁶ First, public policies and interventions to prevent and reduce alcohol-related harm should be guided and formulated by public health interests and based on clear public health goals and the best available evidence.³⁷ Second, policies should be equitable and sensitive to national, religious, and cultural contexts.³⁸ Third, all involved parties must act in ways that do not undermine the implementation of public policies and interventions to prevent and reduce HUA.³⁹ Fourth, public health should be given proper respect in relation to competing interests, and approaches that support that direction should be promoted.⁴⁰ Finally, individuals and families affected by HUA should have access to affordable and effective prevention and care services.⁴¹ Taken together, these principles reflect the

32. WHO, *Global Strategy*, 1-38; see especially p. 8.

33. That is, (i) raised global awareness, (ii) strengthened knowledge, (iii) increased technical support, (iv) strengthened partnerships, and (v) improved systems. See WHO, *Global Strategy*, 1-38; see especially p. 8.

34. WHO, *Global Strategy*, 1-38; see especially p. 8.

35. WHO, *Global Strategy*, 1-38; see especially p. 8.

36. Those not mentioned include notions that (i) the protection of populations at high risk of harm attributed to alcohol and those exposed to the effects of harmful drinking by others should be an integral part of policies addressing HUA; (ii) children, teenagers, and adults who choose not to drink alcoholic beverages have the right to be supported in their non-drinking behavior and protected from pressures to drink; and (iii) public policies and interventions to prevent and reduce alcohol-related harm should encompass all alcoholic beverages and surrogate alcohol. See WHO, *Global Strategy*, 1-38; see especially p. 9.

37. WHO, *Global Strategy*, 1-38; see especially p. 9.

38. WHO, *Global Strategy*, 1-38; see especially p. 9.

39. WHO, *Global Strategy*, 1-38; see especially p. 9.

40. WHO, *Global Strategy*, 1-38; see especially p. 9.

41. WHO, *Global Strategy*, 1-38; see especially p. 9.

multifaceted determinants of alcohol-related harm and the coordinated multisectoral actions required to implement effective interventions.

3. THE NEUROETHICS OF ALCOHOLISM

3.1 *The Addicted Brain*

Communication in the brain is facilitated by neurotransmitters, which are released from neurons at synapses where they interact as bonds with protein complexes, called receptors, on the surface of other cells, predominantly at the postsynaptic membrane. The binding of a neurotransmitter to a receptor transduces a chemical signal that transfers activity-dependent information. The neurotransmitters can either be taken back up by the cell for future use by transporters or degraded and removed from the system. In the brain, pathways are complex integrative systems that contain numerous neurons or nuclei that relay information throughout a circuit and can be acted upon by other neurotransmitter systems that also integrate with that region.⁴² While alcohol has diverse pharmacological profiles, its acute actions converge primarily on the mesocorticolimbic dopaminergic system. This pathway originates in the VTA and projects to the NAc, striatum, forebrain, and prefrontal cortex. The prefrontal cortex coordinates cognitive processes and actions aimed at an internal goal, while the NAc is believed to integrate information, effect an appropriate response, and control the motivational value of stimuli and reward enforcement.⁴³

Immediately after initial exposure to a drug, extracellular levels of accumbal dopamine increase. Some enhance dopamine release from the presynaptic terminals as a consequence of increased neuronal activity in the VTA (e.g., alcohol, nicotine, opiates, and cannabis) while others inhibit the presynaptic uptake by the dopamine transporter (DAT) in the NAc (e.g., cocaine and amphetamines).⁴⁴ Alcohol produces a larger dopamine release that is maintained for longer than that of natural rewards. If exposure to the drug persists, there may be a loss of homeostatic regulation: a progressive increase in basal levels of dopamine is accompanied by a reduction in the lesser response to the drug, resulting in the appearance of tolerance to the drug. During acute alcohol withdrawal, the levels of dopamine rebound to below basal levels so re-exposure to the drug or a drug-related cue is often sufficient to increase dopamine levels again. This dopamine response has been hypothesized to contribute to addictive relapse.⁴⁵

While dopamine release may modulate the acute rewarding effects of alcohol, it does not solely mediate drug-seeking behaviors and persistent drug taking. Exposure to alcohol can have either a direct or indirect effect on numerous neurotransmitter

42. Jhodie R. Duncan and Andrew J. Lawrence, "Molecular Neuroscience and Genetics," in *Addiction Neuroethics: The Ethics of Addiction Neuroscience Research and Treatment*, ed. Adrian Carter, Wayne Hall, and Judy Illes (San Diego: Academic Press, 2012), 27-54; see especially pp. 28-29.

43. Duncan and Lawrence, "Molecular Neuroscience and Genetics," 27-54; see especially p. 29.

44. Duncan and Lawrence, "Molecular Neuroscience and Genetics," 27-54; see especially p. 30.

45. These include serotonergic, noradrenergic, glutamatergic, and GABAergic systems. See Duncan and Lawrence, "Molecular Neuroscience and Genetics," 27-54; see especially p. 31.

systems.⁴⁶ Unlike dopamine, which facilitates the response to initial alcohol use, these additional neurotransmitter systems play a greater role in mediating persistent drug use, contributing to the inability to stop drug use and relapse after a period of abstinence.⁴⁷

There are vast numbers of neuropeptides and their receptors present in pathways that mediate alcohol addiction.⁴⁸ The role of corticotropin-releasing factor (CRF) is highlighted as an example of the intricate part that neuropeptides play in mediating addictive behaviors. Stress, either in the environment or due to alcohol withdrawal, can induce drug craving, which leads to relapse. The system mediating stress responses incorporates the hypothalamic pituitary axis (HPA) and extrahypothalamic regions (such as the extended amygdala). CRF is a neuropeptide that is responsible for activating the HPA, where it plays a mediating role in hormonal, autonomic, emotional, and behavioral responses to stress. Initial exposure to alcohol engages the HPA, but this response becomes blunted with repeated exposures via feedback systems in response to circulating hormones.⁴⁹

CRF-mediated actions on addictive behaviors depend on their interplay at extrahypothalamic sites. These extrahypothalamic regions become sensitized to CRF after repeated exposure to alcohol abuse. During withdrawal, these regions become engaged and hyperactive, thereby increasing local CRF levels and perpetuating negative state of stress. While stress is sufficient to increase CRF levels in the VTA, it is neuroadaptive changes induced by prior alcohol abuse that enable the CRF to control local glutamate release, subsequently activating the dopaminergic system and perpetuating stress-induced relapse to drug-seeking behaviors.⁵⁰

3.2 *The Neurobiological Basis of Moral Reasoning*

The brain region associated with moral reasoning and decision making is the ventromedial prefrontal cortex (VMPFC). This region is primarily associated with cognition, but it reaches to other cortical and subcortical regions mediating emotions, which in turn project to the VMPFC. Interaction between these neural pathways facilitates the cognitive and affective processes responsible for deliberating and making rational moral decisions.⁵¹ Antonio Damasio has observed that damage to and dysfunction in the VMPFC significantly impairs both cognitive and affective processing.⁵² This is due to the associated projections of the VMPFC to regions beyond

46. Duncan and Lawrence, "Molecular Neuroscience and Genetics," 27-54; see especially p. 31.

47. Duncan and Lawrence, "Molecular Neuroscience and Genetics," 27-54; see especially pp. 30-32.

48. Duncan and Lawrence, "Molecular Neuroscience and Genetics," 27-54; see especially p. 37.

49. Duncan and Lawrence, "Molecular Neuroscience and Genetics," 27-54; see especially pp. 37-41.

50. Duncan and Lawrence, "Molecular Neuroscience and Genetics," 27-54; see especially p. 41.

51. For a superb account of the neurobiological basis of morality, see Christopher Suhler, and Patricia Churchland, "The Neurobiological Basis of Morality," in *The Oxford Handbook of Neuroethics*, ed. Judy Illes and Barbara J. Sahakian (New York: Oxford University Press, 2011), 33-58.

52. Antonio Damasio, "Neuroscience and Ethics—Intersections," *American Journal of Bioethics—AJOB Neuroscience* 7, no. 1 (2007): 3-7.

the prefrontal area. The system that supports moral decision making includes the dorsolateral prefrontal regions as well as “higher-order” association cortices in the temporal and parietal regions, the early sensory cortices, and several regions of the motor system, cortically and subcortically,⁵³ as well as other cortical sites.⁵⁴ Of critical takeaway here is the notion decision making and emotional systems overlap and interact within the VMPFC,⁵⁵ which is damaged by alcohol addiction.

Insofar as the VMPFC is vital for decision making, and both cognitive and emotional systems are active within the VMPFC, it follows that decision making is a process both cognitive and emotional. Damasio and colleagues have displayed that emotional impairment in psychopaths or sociopaths correlate with irrational choices founded upon faulty moral judgments,⁵⁶ and a similar impairment is true of individuals with addiction. Unlike (spontaneous) intuitive moral judgments, considered moral judgments involve a depth of reflection and formulation of reasons for or against particular actions.⁵⁷

This data suggests that the interplay between cognitive and affective processes ground one’s capacity to actively participate in practical moral reasoning. Both respective processes are indispensable to the capacity to make considered moral judgments about conflicting values. Such considered judgments require more than immediate or slightly delayed responses. That is, moral judgments require some degree of critical reflective thought, whereby one takes longer to justify actions that pose polarized consequences.⁵⁸

3.3 *Autonomy and Alcoholism*

Autonomy is a term with multiple meanings. In its maximal sense, autonomy means that human beings possess only the desires and beliefs they want to have and make choices uninfluenced by any factor they have not endorsed. Yet, if autonomy is thus understood, it is not a goal that human beings can hope to achieve. As finite beings, individuals are forced to strive for something less grandiose. Certainly, if addiction threatens autonomy (as it seems to do), then it must be some less extravagant notion of autonomy that it undermines. In a more minimal sense, then – as the etymology of the word itself suggests – autonomy (auto nomos, or “self rule”) is simply self-government. Just as autonomous nations are able to make major decisions of internal and external policy without undue interference from foreign powers, so autonomous persons are

53. For example, basal ganglia and cerebellum. See Damasio, “Neuroscience and Ethics,” 3-7; see especially p. 4.

54. For example, insular cortex. See Damasio, “Neuroscience and Ethics,” 3-7; see especially p. 4.

55. Damasio, “Neuroscience and Ethics,” 3-7; see especially p. 4.

56. Damasio, “Neuroscience and Ethics,” 3-7.

57. Damasio, “Neuroscience and Ethics,” 3-7.

58. Thanks in large part to the work of Damasio and other cognitive neuroscientists, the Platonic, Cartesian, Humean, and Kantian dichotomies of reason versus emotion now represent outdated models of the mind. The locus of intellectual judgment is not localized to specific areas of the brain but is instead broadcast over several, broader areas. Hence, this distribution mediates the overlap between cognitive and affective processes that serves as the neurobiological foundation of moral reasoning. See Walter Glannon, *Brain, Body, and Mind: Neuroethics with a Human Face* (New York: Oxford University Press, 2011), 93-114; see especially pp. 99-109.

capable of governing themselves by setting their own short and long-term ends and choosing the best means of achieving them.⁵⁹

However, if autonomy is understood as self-rule, it is not yet clear how the loss of autonomy should be understood. One obvious situation in which autonomy is compromised or lost is when the self is ruled by another. In the political domain, the loss of autonomy is almost exclusively described this way. The same kind of phenomenon can occur, more or less dramatically, in the alcoholic as well. A slave, for instance, whose life is entirely in the hands of another, is a dramatic example of this phenomenon, while a dispositionally subservient person might represent a less dramatic instance of this partial loss of autonomy.⁶⁰

At first blush, addiction does not seem to involve the loss of autonomy described above. The alcoholic who suffers loss of autonomy is not under the control of another person, either partially or necessarily. An addict might be excessively subservient to the individual who supplies him with alcohol, or with money for alcohol, and therefore have his autonomy compromised by the rule of another. However, if the addict's autonomy is compromised in this way, it marks a consequence of an initial loss of autonomy that is characteristic of addiction. This initial loss of autonomy has left the addict vulnerable to this subservience, since it is the addiction that gives the individual who controls him undue influence. Furthermore, there need not be another party exercising undue influence over the addict to experience a weakening of autonomy.

The individual who is able to supply his habit is unlikely to be at the control of another as the consequence of addiction. It is sometimes postulated that addicts are controlled by the drugs they abuse. Carl Elliot, for instance, writes that the addict must go where addiction leads, because the addiction "holds the leash."⁶¹ Elliot's imagery is, of course, a metaphor: an addiction cannot hold a leash, is not an agent, and has no desires or goals of its own. If addiction involves the loss of autonomy, then it must somehow undercut the addict's ability to pursue his own goals. This essay contends that addicts have compromised self-government even though they are not under the strict rule of anyone else.⁶²

4. ORGAN TRANSPLANTATION AND ALCOHOLISM

4.1 Liver Transplantation and Alcoholism

59. Neil Levy, "Autonomy, Responsibility and the Oscillation of Preference," in *Addiction Neuroethics: The Ethics of Addiction Neuroscience Research and Treatment*, ed. Adrian Carter, Wayne Hall, and Judy Illes (San Diego: Academic Press, 2012), 139-51; see especially pp. 140-41.

60. Levy, "Autonomy, Responsibility, and the Oscillation of Preference," 139-51; see especially p. 141.

61. Levy, "Autonomy, Responsibility, and the Oscillation of Preference," 139-51; see especially p. 141.

62. Levy, "Autonomy, Responsibility, and the Oscillation of Preference," 139-51; see especially p. 141-42.

Alcoholic liver disease (ALD) is among the most common causes and indications for orthotopic liver transplantation (OLT) in Europe and North America.⁶³ ALD ranges from steatosis or steatofibrosis to liver cirrhosis. Acute alcoholic hepatitis is a syndrome presenting as acute liver damage following recent excessive drinking and is associated with poor prognosis. Many patients with severe alcoholic hepatitis, whether occurring in the previously healthy liver or in patients with established cirrhosis, fail to recover despite abstinence and maximal medical therapy. The development of acute renal failure in the midst of acute alcoholic hepatitis indicates an especially grim prognosis. Most transplant programs in the United States and Europe require a minimal interval of abstinence for six months prior to transplantation for patients with decompensated liver disease. If there is no substantial improvement after three months of medical management, including alcohol abstinence, the chances of spontaneous recovery by patients with acute hepatitis and cirrhosis are poor.⁶⁴

Liver transplantation in patients with alcoholic cirrhosis was first acknowledged by the statement of the National Institutes of Health Consensus on Liver Transplantation in 1983. As the statement notes, patients with alcoholic liver disease who are judged likely to abstain from alcohol and have established clinical indicators of fatal outcome may indeed be candidates for liver transplantation.⁶⁵ In 1997, a conference (under the auspices of the National Institutes of Health) on liver transplantation in ALD patients confirmed the positive outcome of liver transplantation for most patients with alcoholic liver cirrhosis, strongly encouraged efforts to understand the mechanisms leading some patients to relapse, and called for adaptations to patient management protocols to restrict risk of relapse and graft loss.⁶⁶ The efficacy of liver transplantation for alcoholic cirrhosis was best demonstrated by T. Poynard, who employed modeling techniques to exhibit survival benefit when patients with advanced decompensation (i.e., 11-15 points on the Child-Turcotte-Pugh (CTP) scoring system) underwent transplantation. There was no survival benefit when alcoholic patients with better-compensated liver function were transplanted.⁶⁷

In the past, assessing the risk of death among patients with alcoholic cirrhosis has depended on CTP classification. In recent years, however, a new prognostic system, known as the Model for End-Stage Liver Disease (MELD), predicts liver disease severity on the basis of serum creatinine, serum total bilirubin, and INR.⁶⁸ Previous studies have failed to demonstrate that other clinical manifestations of liver decompensation, such as variceal hemorrhage, hepatic encephalopathy, new onset ascites, or spontaneous bacterial peritonitis, were independent predictors of survival over and above the MELD score.

63. Patrizia Burra and Michael R. Lucey, "Liver Transplantation in Alcoholic Patients," *Transplant International* 18 (2005): 492-98; see especially p. 492.

64. There is only limited experience with transplantation in patients with acute alcoholic hepatitis and minimal abstinence, and the current consensus in most European and North American transplant centers is that patients with acute alcoholic hepatitis should not undergo liver transplantation. See Burra and Lucey, "Liver Transplantation in Alcoholic Patients," 492-98; see especially pp. 491-92.

65. Burra and Lucey, "Liver Transplantation in Alcoholic Patients," 492-98; see especially p. 492.

66. Burra and Lucey, "Liver Transplantation in Alcoholic Patients," 492-98; see especially p. 492.

67. Burra and Lucey, "Liver Transplantation in Alcoholic Patients," 492-98; see especially p. 492.

68. Burra and Lucey, "Liver Transplantation in Alcoholic Patients," 492-98; see especially p. 493.

Nonetheless, the onset of any of these features in an abstinent alcoholic should prompt managing clinicians to consider referral to a transplant center.⁶⁹ In addition to evaluating the severity of liver disease and its complications, the pre-transplant investigation is based on assessing the patient's general health, especially the conditions and comorbidities that might limit the potential for successful surgery, such as pancreatitis, central and peripheral neuropathy, heart disease, myopathy, renal insufficiency, and poor nutritional status. Chronic alcohol abuse is associated with impaired lymphocyte recruitment, which may explain the increased morbidity and mortality of pulmonary infections in alcoholic patients.⁷⁰

4.2 Transplant Outcome and Survival Benefit for Alcoholic Patients

According to a 2011 report from the European Liver Transplant Registry, post-OLT actuarial survival rates for end-stage ALD were higher than those of patients transplanted for hepatitis viral infection.⁷¹ Similar survival rates for ALD patients have been reported in the United States: 92% at one year; 86% at three and five years, and 76% at nine years.⁷² Indeed, ALD patients had similar, if not better, patient and graft survival rates than other indications. Few studies are available on the outcome of patients undergoing OLT for ALD in light of associated co-morbid factors of liver damage, the most common of which is chronic hepatitis C virus (HCV). Despite data reporting a more rapid progression of liver disease in immunocompetent patients with the combination of ALD and HCV, a study by V. Aguilera and colleagues found that patients transplanted for ALD plus HCV had a better survival rate than patients with HCV alone and a similar survival rate to those with ALD alone.⁷³

The long-term morbidity and survival rate of transplanted ALD patients can be impaired by a high rate of medical complications, especially de novo cancer. In a group of selected ALD patients who underwent OLT, A. Jain and colleagues reported that five year patient and graft survival was significantly lower than that of non-alcoholic cirrhotic controls, due primarily to cardiorespiratory, cerebrovascular, and neoplastic problems.⁷⁴ Recent studies have shown a higher rate of de novo malignancy, not only in active smokers after OLT, but also in patients with a history of smoking prior to transplantation, which accounts for approximately 75% in the ALD population.⁷⁵

A greater incidence of post-OLT neurological complications has also been reported in patients transplanted for ALD compared to transplant recipients with other

69. Burra and Lucey, "Liver Transplantation in Alcoholic Patients," 492-98; see especially p. 493.

70. Burra and Lucey, "Liver Transplantation in Alcoholic Patients," 492-98; see especially p. 493.

71. 84% at 1 year; 73% at 5 years; and 58% at 10 years versus 82%, 70%, and 60%, respectively; $p = 0.04$. See Annagiulia Gramenzi, Stefano Gitto, Fabio Caputo, Maurizio Biselli, Stafania Lorenzini, Mauro Bernardi, and Pietro Andreone, "Liver Transplantation for Patients with Alcoholic Liver Disease: An Open Question," *Digestive and Liver Disease* 43 (2011): 843-49; see especially p. 845.

72. Gramenzi et al., "Liver Transplantation," 843-49; see especially p. 845.

73. Gramenzi et al., "Liver Transplantation," 843-49; see especially p. 845.

74. Gramenzi et al., "Liver Transplantation," 843-49; see especially p. 845.

75. Gramenzi et al., "Liver Transplantation," 843-49; see especially p. 845.

etiologies.⁷⁶ A further concern with ALD patients undergoing OLT is the lack of compliance with clinical protocols and immunosuppressive therapy. However, this seems to be an assumption rather than an evidence-based conclusion. Although poor compliance with immunosuppressive drugs was reported to be responsible for late rejection and graft failure in heavy drinkers after OLT, several studies have demonstrated that ALD patients show fewer episodes of acute cellular rejection than do patients transplanted for other etiologies.⁷⁷

These results raise numerous questions concerning whether a system based on benefit calculation can be implemented equitably and practically. First, allocating organs on the basis of projected survival benefit – herein understood as the balance between waiting list mortality and post liver transplantation mortality – represents a true paradigm shift.⁷⁸ The current principle in organ transplantation – namely, transplanting the sickest patient first – has served well because it makes the allocation process transparent and because the benefit of liver transplantation is mostly dominated by waiting list mortality. Following the initial learning curve about the MELD score, the liver transplant community has embraced the scoring system because of its simplicity and objectivity.⁷⁹

The ultimate question of whether the benefit model should be adopted will depend on the magnitude of the benefit: how many lives will eventually be saved by directing some of the organs from one group of patients – namely, those at the highest risk of waiting list mortality – to another – that is, those who are expected to have the better post-transplantation outcome. If a benefit-based transplantation policy is to be adopted, the transplant community must be willing to accept this departure from traditional thinking. Because some patients with ALD will experience a good outcome, they will be placed at higher priority than patients without ALD who face the same (or even higher) risk of death while waiting.⁸⁰

4.3 Predictors of Alcoholic Relapse After Liver Transplantation

Although transplant outcome and survival benefit remain crucial endpoints by the Scientific Registry of Transplant Recipients, an increasing emphasis has been placed on morbidity after solid organ transplantation.⁸¹ The recurrence of the original disease, the development of new diseases, the morbid consequences of immunosuppression, and the mental health of the transplant recipient all contribute to a more comprehensive understanding of the lives of transplant recipients. As such, alcohol abuse after transplantation is an area of cardinal importance in assessing the recipient's projected

76. Gramenzi et al., "Liver Transplantation," 843-49; see especially p. 845.

77. Gramenzi et al., "Liver Transplantation," 843-49; see especially p. 845.

78. Sumeet K. Asrani, W. Ray Kim, and Julie K. Heimbach, "Survival Benefit of Liver Transplantation: One Size Fits All or Fits None?" *Hepatology* 50, no. 2 (2009): 352-54; see especially pp. 352-53.

79. Arsani et al., "Survival Benefit," 352-54; see especially pp. 353-54.

80. Arsani et al., "Survival Benefit," 352-54; see especially pp. 353-54.

81. Santiago Tome, Adnan Said, and Michael R. Lucey, "Addictive Behavior After Solid Organ Transplantation: What Do We Know Already and What Do We Need to Know?" *Liver Transplantation* 14 (2008): 127-29; see especially p. 127.

quality of life. Unfortunately, there are numerous barriers to obtaining accurate data about alcohol use by transplant recipients. Several of the most common methods, such as self-reports, assessment by clinical findings, and collateral reports, are subject to bias. In particular, the lack of an objective and reliable instrument to measure alcohol consumption and the risk to the recipient that candor about alcohol use could harm his chances of receiving future care from the transplant program encourage underreporting.⁸²

Given the aforementioned caveat, the difficulty to apply tools of meta-analysis to assess the frequency of addictive behavior after liver transplantation is understandable. Against this backdrop, the study of M. A. Dew and colleagues is all the more laudable. In their paper, Dew and colleagues successfully concentrate on liver transplant recipients and seek to discover how frequently these patients' addictions relapse after transplantation.⁸³ According to their meta-analysis, when alcoholic liver disease is the pre-transplant diagnosis, six of 100 recipients per year will use alcohol after transplantation, and less than three will resume heavy alcohol use. For the addiction specialist, the maintenance of sobriety after transplantation is surprising.⁸⁴ Possible explanations include (i) the consequence of liver transplant recipients being a highly selected population with less craving for alcohol than typical alcoholics, (ii) a result of potential therapeutic properties of transplantation on addictions, or (iii) an underestimate inasmuch as alcoholic transplant recipients hide their drinking.⁸⁵

At least three pre-transplant factors assist in predicting alcoholic relapse after transplantation: (i) lack of social support, (ii) family history of alcoholism, and (iii) less than six months of abstinence from alcohol. Both social support and family history resonate with studies conducted by George Vaillant in nontransplant alcoholic populations.⁸⁶ However, the six-month period of abstinence is a more questionable prognostic tool.⁸⁷ Use of the six-month rule to select alcoholic patients for liver transplantation is nearly universal, and this exerts profound bias on meta-analysis insofar as patients with the highest risk – those with less than six months of abstinence – are likely to have been excluded before the data was collected.⁸⁸

The vast majority of relapses in Vaillant's 2003 follow-up study occurred before the seventh year of abstinence.⁸⁹ By analogy to the cancer-free period as a definition of

82. Tome et al., "Addictive Behavior," 127-29; see especially p. 127.

83. See Mary Amanda Dew, Andrea F. DiMartini, Jennifer Steel, Annette De Vito Dabbs, Larissa Myaskovsky, Mark Unruh, and Joel Greenhouse, "Meta-Analysis of Risk for Relapse to Substance Use After Transplantation of the Liver or Other Solid Organs," *Liver Transplantation* 14 (2008): 159-72.

84. Tome et al., "Addictive Behavior," 127-29; see especially p. 128.

85. Tome et al., "Addictive Behavior," 127-29; see especially p. 128.

86. George E. Vaillant, "The Natural History of Alcoholism and its Relationship to Liver Transplantation," *Liver Transplant Surgery* 3 (1997): 304-310.

87. However, some studies find the relationship directly indicative of relapse predictability. See, for instance, Zamil Karim, Pongphob Intaraprasong, Charles H. Scudamore, Siegfried R. Erb, John G. Soos, Elsie Cheung, Polly Cooper, Andrzej K. Buzckowski, Stephen W. Chung, Urs P. Steinbrecher, and Eric M. Yoshida, "Predictors of Relapse to Significant Alcohol Drinking After Liver Transplantation," *Canadian Journal of Gastroenterology* 24, no. 4 (April 2010): 245-50.

88. Tome et al., "Addictive Behavior," 127-29; see especially p. 128.

89. George E. Vaillant, "A 60-Year Follow-Up of Alcoholic Men," *Addiction* 98 (2003): 1043-51.

cure, Vaillant concludes that a follow-up study of five years rather than one or two years would be necessary to determine stable recovery from alcoholism.⁹⁰ The absence of a link between pre-transplant rehabilitation treatment and post-transplant alcoholic relapse is an unexpected finding.⁹¹ Nevertheless, predicting future alcohol relapse remains imperfect, and future studies will need to confront the issue of suitability for transplantation of patients with durations of abstinence shorter than six months.⁹²

5. MORAL RESPONSIBILITY AND ALCOHOLISM

5.1 *Free Will, Moral Choice, and Alcoholism*

Cognitive and affective neuroscience has generated significant insight into the neurobiological basis of the capacity for practical and moral reasoning. Experiments exposing human subjects to functional magnetic resonance imaging (fMRI) scans during participation in stimulating cognitive tasks have conclusively identified inter-relationships between the brain and mental states, each of which play vital roles in deliberation and decision making. This comprehensive imaging has shown that desires, beliefs, emotions, and intentions that serve as the impetus for action are mediated by the interplay between cortical and subcortical networks in the brain.⁹³ Some cognitive neuroscientists and psychologists have contended that the relationship between the brain and behavior indicates that cognitive function can be explained in terms of unconscious mechanistic processes in the brain.⁹⁴ This suggests that the source of one's actions (i.e., one's positive will) is not within one's control,⁹⁵ and that one's mental state has no causal role in manufacturing them.⁹⁶

This essay understands free will as the capacity to respond to reasons and to make choices in accord with one's mental state by controlling the role each plays in one's actions.⁹⁷ It therefore contends, contrary to the position above,⁹⁸ that any reasonable conception of free will is consistent with the notion that some cognitive states can causally influence actions while having physical causes in the brain. That is, because the brain generates the mind, and the mind in turn can influence the brain, a plausible account of human (moral) agency must include both unconscious physical and conscious mental states and events as causes of human action. This point suggests that neuroscientific evidence cannot be used as a counterargument to the possibility of

90. George E. Vaillant, "A 60-Year Follow-Up of Alcoholic Men," *Addiction* 98 (2003): 1043-51.
91. Rehabilitation support has been considered a good predictor of abstinence outside the context of transplantation. See Tome et al., "Addictive Behavior," 127-29; see especially p. 128.

92. Tome et al., "Addictive Behavior," 127-29; see especially pp. 128-29.

93. This data has resulted in a more robust understanding of some aspects of the mind-brain relation. See Glannon, *Brain, Body, and Mind*, 41-71; see especially pp. 41-42.

94. Glannon is unspecific about who these individuals are, or precisely what their positions are. See Glannon, *Brain, Body, and Mind*, 47-71; see especially p. 41.

95. For a fine analysis of the neuroethics of free will, see Patrick Haggard, "Neuroethics of Free Will," in *The Oxford Handbook of Neuroethics*, ed. Judy Illes and Barbara J. Sahakian (New York: Oxford University Press, 2011), 219-26.

96. Glannon, *Brain, Body, and Mind*, 47-71; see especially pp. 47-57.

97. Glannon, *Brain, Body, and Mind*, 47-71; see especially pp. 41.

98. Glannon, *Brain, Body, and Mind*, 47-71; see especially p. 41.

personal moral responsibility insofar as nothing about normal brain function suggests that one lacks the capacity to choose and act freely.⁹⁹ For better or worse and in varying degrees, then, one is responsible one's actions.

However, impaired control over voluntary behavior is a marked feature in emerging neurobiological explanations of addiction, in clinical and diagnostic accounts, and in debates about addiction nosology.¹⁰⁰ There is growing evidence that chronic, sustained abuse of alcohol is associated with neurocognitive changes and deficits, as revealed by neuroimaging studies and neuropsychological testing.¹⁰¹ Others studies propose that chronic exposure to alcohol sets in motion neurobiological processes that result in the overvaluing of the reinforcing properties of a substance or behavior and an undervaluing of natural reinforcers (e.g., maintaining relationships, going to work, etc.).¹⁰² These processes are associated with impaired voluntary control over one's behavior. Similarly, individuals experiencing addiction have neurological impairments that weaken their ability to make voluntary decisions in the service of long-term outcomes. Hence, alcoholic cravings can manifest as such irresistible and powerful psychological forces that someone with an addiction is not capable, at certain times, of acting fully autonomously when the decision involves denying the persistence of cravings.¹⁰³

5.2 Moral Responsibility and Alcoholism

The primary reason philosophers have argued against providing patients with end-stage ALD an equal chance to compete for liver transplantation is that alcoholics are somehow morally responsible for their condition.¹⁰⁴ The fundamental premises of the argument are straightforward: (i) alcoholics are morally responsible for their medical conditions; and (ii) patients who are morally responsible for their medical conditions should be given lower priority for medical resources for their conditions when they are in competition with those who are not morally responsible for their conditions. Following this logic, the syllogistic conclusion is that alcoholics with end-stage ALD should be given lower priority for medical resources (e.g., livers) when they are in competition with those who are not morally responsible for their conditions.¹⁰⁵

However, whether alcoholics are morally responsible for their liver conditions depends on whether they are responsible for their alcoholism in the first place. If

99. Glannon, *Brain, Body, and Mind*, 47-71; see especially pp. 41-42.

100. T. Cameron Wild, Jody Wolfe, and Elaine Hyshka, "Consent and Coercion in Addiction Treatment," in *Addiction Neuroethics: The Ethics of Addiction Neuroscience Research and Treatment*, ed. Adrian Carter, Wayne Hall, and Judy Illes (San Diego: Academic Press, 2012), 153-74; see especially p. 155.

101. Wild et al., "Consent and Coercion," 153-74; see especially p. 155.

102. Wild et al., "Consent and Coercion," 153-74; see especially p. 155.

103. Wild et al., "Consent and Coercion," 153-74; see especially p. 155.

104. See, for instance, Walter Glannon, "Responsibility and Priority in Liver Transplantation," *Cambridge Quarterly of Healthcare Ethics* 18 (2009): 23-35.

105. Dien Ho, "When Good Organs Go to Bad People," *Bioethics* 22, no. 2 (2008): 77-83; see especially p. 79.

alcoholics are not responsible for their alcoholism, then it would be wrong to treat them differently from patients who develop liver diseases for reasons beyond their control. The Center for Disease Control and Prevention defines alcoholism as “a primary, chronic disease with genetic, psychosocial, and environmental factors influencing its development and manifestations.”¹⁰⁶ In addition, the American Medical Association endorses the proposition that “drug dependencies, including alcoholism, are diseases” and that “their treatment is a legitimate part of medical practice.”¹⁰⁷

Much of the contemporary discussion over end-stage ALD and livers centers on the question of whether alcoholism is a disease, traditionally understood. Nevertheless, settling this question will not, in and of itself, determine the issue of responsibility for that disease. Hepatitis C is undoubtedly a disease, but if one were to expose oneself deliberately to the hepatitis C virus with the intention of developing the disease, one would be both causally and morally responsible for the condition. Thus, the issue in question is not whether alcoholism is a disease per se (though it most certainly is); rather, the focus should be on whether alcoholics are morally responsible for their alcoholism.¹⁰⁸

On this issue, philosophers continue to disagree. Walter Glannon, for instance, argues that although environmental factors such as a family history of alcoholism, preexisting psychiatric conditions, gender, and genetic factors all increase the risk of developing alcoholism, alcoholics can still be morally responsible for their condition. He contends that regardless of the high risk of developing alcoholism, one always has the ability to refrain from drinking.¹⁰⁹ Combine that ability with knowledge of one’s dispositions and health risks (an argument Glannon appropriates from Book III of Aristotle’s *Nicomachean Ethics*¹¹⁰) along with acceptance of lower priority for liver transplantation, one ought to be held responsible for one’s alcoholism and its associated diseases.¹¹¹ Robert Veatch advances a similar claim: while many conclude that alcoholism has nonvoluntary components, it also contains significant opportunity for voluntary decision making.¹¹² Veatch’s conclusion is that if the alcoholic had the opportunity for significant choice, then he should be responsible for the consequences, including lower priority for liver transplantation.¹¹³

The disagreements about whether alcoholics cause their own end-stage ALD reveal more than the problems confronted in determining their level of moral responsibility. Take, for instance, the strategy of Alvin Moss and Mark Siegler of

106. Center for Disease Control and Prevention, 2005, available at <http://www.cdc.gov/alcohol/faqs.htm>; accessed 04/18/14.

107. American Medical Association, *Drug Dependencies and Diseases* (American Medical Association, 1999), H-95.983.

108. Ho, “When Good Organs Go to Bad People,” 77-83; see especially p. 79.

109. That is, insofar as one is not coerced into drinking. See Glannon, “Responsibility and Priority in Liver Transplantation,” 23-35; see especially pp. 26-27.

110. See Glannon, “Responsibility and Priority in Liver Transplantation,” 23-35; see especially pp. 26-27.

111. See Glannon, “Responsibility, Alcoholism, and Liver Transplantation,” 31-49.

112. Robert M. Veatch, *Transplantation Ethics* (Washington, DC: Georgetown University Press, 2000), 311-21; see especially p. 314.

113. Ho, “When Good Organs Go to Bad People,” 77-83; see especially p. 79.

assigning responsibility on the basis of an alcoholic's failure to seek treatment for his alcoholism.¹¹⁴ This argument is meant to improve the attempt to hold alcoholics responsible for their alcoholic abuse per se. However, this strategy seems no more reliable in evaluating moral responsibility than simply blaming alcoholics for their alcoholism.¹¹⁵

Against this logic, Dien Ho considers the alcoholic who has failed to seek treatment because he is preoccupied with an ailing mother who requires constant assistance.¹¹⁶ In Ho's example, the son is too poor to hire help, and the closest Alcoholics Anonymous meeting takes place in a city that is more than a two-hour drive away. Worse still, he has no car. These hypothetical circumstances – which are surely realities for some – illuminate Ho's position: it would be unreasonable to conclude that the man in this example is responsible enough for his condition that he should receive lower priority for liver transplantation.¹¹⁷

Ho's point is not that alcoholics should be excused entirely, but that the assessment of moral responsibility in cases similar to his example are extremely difficult, if not entirely impossible. Carl Cohen, Martin Benjamin, and the Ethics and Social Impact Committee of Transplant and Health Policy Center have articulated this problem well.¹¹⁸ They outline three technical problems with the use of moral responsibility as a criteria for the allocation of livers to alcoholics: "(1) We have genuine and well-rounded doubts about comparative degrees of voluntariness and, therefore, cannot pass judgment fairly; (2) Even if we could assess degrees of voluntariness reliably, we cannot know what penalties different degrees of misconduct deserve; (3) Judgments of this kind could not be made consistently in our medical system."¹¹⁹ Both problems (1) and (3) are legitimate concerns. Problem (1) highlights the difficulties mentioned above; on the other hand, problem (3) points out that even if it were possible to do so on a local level, consistent assessment could not be ensured across different organizations.¹²⁰ A mistake in evaluating the moral responsibility of patients with end-stage ALD might mean denial of vital organs and a high probability of death. Unless these technical difficulties are overcome, there is a moral imperative to remain cautious and to avoid using moral responsibility as legitimate criteria for allocating life-sustaining organs.¹²¹

5.3 The Provider-Patient Relationship in the Context of Alcoholism

114. See Alvin H. Moss and Mark Siegler, "Should Alcoholics Compete Equally for Liver Transplantation?" *Journal of the American Medical Association* 265, no. 10 (March 1991): 1295-98.

115. See Ho, "When Good Organs Go to Bad People," 77-83.

116. See Ho, "When Good Organs Go to Bad People," 77-83.

117. Ho, "When Good Organs Go to Bad People," 77-83; see especially p. 80.

118. Carl Cohen, Martin Benjamin, and the Ethics and Social Impact Committee of Transplant and Health Policy Center, "Alcoholics and Liver Transplantation," *Journal of the American Medical Association* 265, no. 10 (March 1991): 1299-1301.

119. Cohen et al., "Alcoholics and Liver Transplantation," 1299-1301; see especially p. 1300.

120. While relatively imprecise procedures might be tolerated when the harm caused by mistake is minimal, procedures that allocate life-saving treatment ought to have small margins of error. See Ho, "When Good Organs Go to Bad People," 77-83; see especially p. 80.

121. Ho, "When Good Organs Go to Bad People," 77-83; see especially p. 80.

The difficulty of understanding pain and suffering is well known.¹²² Providers encounter numerous difficulties in attempting to provide adequate relief to alcoholic patients who struggle with morbidity and, ultimately, mortality.¹²³ As Daniel Callahan notes, human nature fixes no singular response to suffering, and for this reason it is often arduous for providers to ascertain the behavior it might induce or the meaning it may carry for a particular patient. Hence, compassionate medical care is necessarily idiosyncratic; it must be tailor made to fit the individual in his history, culture, and personal structure of understanding.¹²⁴

In the context of alcoholism, the provider-patient relationship can be viewed as the vehicle through which the relief of suffering – and so the honoring and upholding of the mandates of licit fiduciary relationships – is achieved. Providers cannot avoid becoming involved with patients and at the same time effectively allaying their suffering. In fact, with alcoholic patients who are suffering it is essentially impossible to be in their presence and remain indifferent. In its barest sense, to be concerned is to be involved.¹²⁵ Every provider fears becoming closer to suffering patients, many of whom will die, because such a relationship is sure to promise pain, sorrow, and loss.¹²⁶ Yet it is only through the compassionate connection with the alcoholic patient that pertinent information flows to inform the provider of what the patient is feeling and even what existential sensations he is experiencing. Through the same compassionate bond, then,¹²⁷ the provider can offer a bridge over which the alcoholic can return from the isolation of suffering.¹²⁸

Introducing judgments of moral desert for the allocation of scarce resources negatively impacts the fiduciary relationship between providers and patients. Moreover, such assessments of moral “worth” risk undermining the fundamental nature of medicine. When the alcoholic patient seeks medical assistance, one of his goals is to restore the autonomy that is lost to addiction. The loss of autonomy or the inability to pursue ordinary activities (that one could if healthy) is arguably what makes diseases and ailments generally horrible. And, in petitioning a provider to help restore the loss of autonomy, it is crucial that the provider learns about the values and preferences of the

122. Unlike other objects in biomedical science, persons cannot be reduced to their parts in order to be understood. See Cassell, *The Nature of Suffering*, 29-45; see especially pp. 36-41.

123. Cassell, *The Nature of Suffering*, 29-45; see especially pp. 33-35.

124. Daniel Callahan, *The Troubled Dream of Life: In Search of a Peaceful Death* (Washington, DC: Georgetown University Press, 2000), 120-55; see especially pp. 132-36.

125. At issue, then, is the degree to which the physician actively participates in that relationship. See Cassell, *The Nature of Suffering*, 278-91; see especially pp. 290-91.

126. Hence the often unexamined desire to hold back, cover one’s feelings with a white coat, and hide behind incomprehensible language. This is due in large part to the fact that such pain, sorrow, and loss renders useless the technical tools necessary to care for the very sick and suffering. See Cassell, *The Nature of Suffering*, 278-91; see especially p. 291.

127. This bond enables the endangered, fragile patient to know that the physician can be trusted, and so begin to reconnect to the world through that relationship. See Cassell, *The Nature of Suffering*, 278-92; see especially p. 291.

128. Cassell, *The Nature of Suffering*, 278-91; see especially pp. 290-91.

patient.¹²⁹ In oncology care, for instance, the recommendation of a protocol typically hinges on nonmedical considerations. A patient might be willing to accept a lower quality of life brought by a chemotherapy regimen in favor of a higher success rate. A different patient might view the long-term survival rate as secondary while prioritizing the minimization of toxicity and the maintenance of a high quality of life during treatment.¹³⁰ In this sense, providers play indispensable roles as health care advocates or consultants, filtering the medical facts through the net of patient preferences and deriving a set of therapeutic recommendations that best maximize patient wishes, including how best to restore autonomy.¹³¹

The trust between providers and patients becomes essential if the model of medicine sketched above is accurate. The patient must feel comfortable that the information shared with the provider will not haunt or compromise his care at a later date. Not only must this include the frank expression medical information, but also the ability to share lifestyle information that can make a significant difference in determining the proper treatment to recommend. For example, a patient diagnosed with hepatitis who contracted the disease from drug use would require different care than someone who contracted hepatitis through a blood transfusion.¹³² But if the patient learns that what he tells the provider might significantly alter his chances of life-saving treatment, the patient would have every incentive to withhold information that he believes (rightly or wrongly) might jeopardize his interest. Given that providers can only make therapeutic recommendations if they possess all of the relevant information, this sort of censorship undermines the provider's effectiveness as a health advocate.¹³³

A medical model that allocates resources on the basis of moral desert also runs counter to its institutional commitments. Such a system would give patients with medical knowledge a significant advantage. They would presumably know what not to reveal to their providers when the information would compromise their care. The product is a system that would give higher priority to those who know how to manipulate it to their advantage.¹³⁴ Those patients who do not possess such knowledge would rightly worry that their honesty in communication with providers might be self-destructive. Providers would then no longer remain caretakers; they would become judges inheriting an adversarial role. As long as medicine is committed to providing patients with equal care regardless of their medical knowledge, and as long as it believes patients should have an institution – a medical “safe haven” – to which to turn where they can receive medical advocacy, allocating resources on the basis of moral desert would undermine both of these goals.¹³⁵

129. This example is found in Ho, “When Good Organs Go to Bad People,” 77-83; see especially p. 81.

130. This example is found in Ho, “When Good Organs Go to Bad People,” 77-83; see especially p. 81.

131. Ho, “When Good Organs Go to Bad People,” 77-83; see especially p. 81.

132. This example is found in Ho, “When Good Organs Go to Bad People,” 77-83; see especially p. 81.

133. Ho, “When Good Organs Go to Bad People,” 77-83; see especially p. 81.

134. Ho, “When Good Organs Go to Bad People,” 77-83; see especially p. 82.

135. Ho, “When Good Organs Go to Bad People,” 77-83; see especially p. 82.

6. CONCLUSION

The aim and proposal of this essay has been to examine the notions of alcohol addiction, transplantation candidacy, and moral responsibility with the intention of positing the argument that employing moral desert as an allocation criteria to inhibit alcoholic patients from equal consideration and treatment is, and ought to remain, at odds with the fundamental nature of medicine and the functioning of the provider-patient relationship. To secure the justification of this thesis, it has drawn from the twofold premises that (i) compassionate medical practice – understood as the sympathetic willingness to enter into the existential suffering of another in order to ameliorate the anguish invoked by disease – rests on the fiduciary relationship shared between provider and patient, and (ii) allocating medical goods according to moral desert rather than existential disposition undermines the fundamental nature of medicine and the functioning of the provider-patient relationship. To this end, the essay has been successful.

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