# Kaleidoscope

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**The Netflix show *13 Reasons Why* attracted both plaudits and controversy for its portrayal of the events leading up to, and the impact of, the suicide of a young person who had suffered from severe bullying.** The 17-year-old protagonist Hannah Barker leaves behind 13 cassette tapes recording the reasons she has taken her life, to be listened to by those she implicates in her action. It was widely watched when first broadcast in 2017, and attracted notable attention on social media. Many clinicians and some suicide prevention organisations expressed concern about the graphic portrayal of her self-harm and the inference that help-seeking was ineffective. Reports of a rise in online searches of how to take one's own life followed its launch, and there were anxieties of so-called 'suicide contagion'. Niederkrotenthaler *et al* took Twitter and Instagram posts as proxy estimates of the TV show's impact from April to June of 2017, and compared this with a time-series analysis of US monthly suicide data between 1999 and the end of 2017.[1] They found that for 10- to 19-year-olds, there were immediate increases in suicides in males and females following the show's first airing that were outside the 95% confidence forecast banding of deaths. Grimly, they calculate this as the additional death of 66 young males and 37 females; the generally lower rates of female suicides mean that this was actually a proportionally bigger increase in this group. There was a particularly high increase in death by hanging, although the character Hannah dies through lacerating her wrists. There was not an impact on any other age groups. No doubt caution is required in interpreting this, and other meta-analytical work has not supported a causal link between fictional and actual suicides. However, it reaffirms the need for serious consideration and thought in the media portrayal of suicide.

**More broadly, online hate is clearly a problem, with a sense that 'something needs to be done'.** An intriguing paper by Johnson *et al* presents a model of the 'hate ecology' of online networks and uses it to show how different policies for modifying or disrupting this ecology can result in counterintuitive outcomes.[2] They start by outlining the current approaches to tackling online hate: the microscopic identification of 'bad' prime individuals (for example, silencing leaders of hate groups) or macroscopic 'blanket' bans on ideological content (for example, policing traffic for ideological content and making it invisible). Neither approach seems to be working, and each has its own ethical problems. Johnson *et al* propose that attending to these two extremes of scale misses the important phenomena, which are meso-scale self-organising network-of-networks interactions for which they coin the term 'hate highways'. They began by looking at 'clusters' of users such as those found on Facebook groups – where individuals with similar interests aggregate together and self-organise around a theme. Then, exploring similar behaviours over commercially independent social media platforms (such as VKontakte in Europe) they identify three kinds of 'linkage' between hate clusters that facilitate the spread of hate by embedding activity – hate-cluster mirroring, reincarnation and direct intercluster linkage. As an example, they discuss how when the Ku Klux Klan (KKK) were banned from Facebook, clusters remained on the European VKontakte platform. When the European platform banned KKK content, it was reincarnated on Facebook but with the hate group's name now written in Cyrillic to avoid detection by moderators. This adaptive platform-switching behaviour means moderation can have unintended consequences.

They provide a mathematical model that makes one notable prediction; assume two platforms A and B host similar hate clusters. Platform A (and B) has a number of hate clusters linked in a network and the number of links is proportional to the risk of moderators discovering the hate groups. The shortest average path between clusters on platform A describes how quickly hate information is shared on platform A. The hate clusters on platform A are also linked in a network with platform B. Their model predicts that as platform B shuts down its clusters/network, this reduces the number of between-platform links and inadvertently increases the average shortest path within platform A (and subsequently increases the amount of hate information spread within platform A). The consequence of this is that if moderators on two platforms act independently without coordination (as they are likely to do, given they are commercially separate entities), 'pockets' of highly active hate clusters emerge within the platforms.

They conclude by showing how their modelling informs four different ways of preventing social media hate activity: for example, one method is by disrupting the mechanics of the adaptive network processes described above by randomly selecting users from hate clusters and shutting their accounts. Another method, more akin to social engineering, is to effectively engender infighting between hate clusters because – as the authors note – large global networks often contain people, groups or clusters with different ideologies bound together only by their investment in committing hateful activity.

**Enhanced social functioning is a key goal in psychosis. 'What do people with schizophrenia do all day?' is the provocative title of an ecological study on the topic.** Overcoming the issues of recall and subjective biases, Granholm *et al* contacted 100 participants with schizophrenia (and 71 controls) seven times a day for 7 days via smartphones, briefly surveying their activity at that time point.[3] It asked about educational, employment, socialisation, active leisure, self-care and home-care activities. Surveys only took about 3 min to complete, and engagement was very good with about 85% of both groups completing them. Compared with controls, those with schizophrenia reported significantly less productive activity, fewer social interactions, more non-productive behaviour and more time at home. Of note, those with schizophrenia did have a similar total number of interactions, but these were often with care providers, fellow residents and roommates. Exploring activity within the schizophrenia group, those living independently had better functioning than those in supported housing, as did those in vocational activities compared with those who were not. The study technique of ecological momentary assessment has been used in other populations, and this trial supports its ongoing use in better understanding the lives of those with psychoses.

To intervening, and Christensen *et al* report on a randomised trial of individual placement and support (ISP) as a vocational tool for individuals with serious mental illness (most of whom had schizophrenia).[4] ISP differs from more traditional models of employment support, as it side-steps extended 'pre-vocational' training, aiming instead to rapidly get the individual back into work or study in an area of interest to them, as part of an integrated mental health service provision. There are good data supporting it, but notably many fail to derive benefit, which has been argued to be partially because of the cognitive deficits and impaired social functioning that serious mental illness can bring. Testing this, 720 participants were randomised to ISP, enhanced ISP augmented with cognitive remediation and work-focused skills training (ISPE) or treatment as usual (TAU). Over the 18-month follow-up both active interventions did better than TAU in terms of hours

worked or studied. However, there were no significant differences between ISPE and the simpler ISP, and none of the three groups varied in any non-vocational outcomes except lower satisfaction with services in TAU. The finding of almost 60% of those who received an active intervention attaining competitive employment or education, compared with a still impressive 46.5% of those who just had TAU, reflects on how background factors – in this instance a supportive Danish social welfare model – are also crucially important.

**From social to biological, and in truth the utility of animal models in extending our understanding of schizophrenia has been relatively modest.** The vast array of potential predisposing genetic and environmental factors, along with all possible adverse triggering events for the emergence of psychosis, create an overwhelming number of combinations to explore. However, while brain maturation processes are not terribly well understood in humans, some animal models have this mapped down to the day. By using the control available with mice, we can explore narrow aspects of the disorder within its developmental context with precision. Defining features of schizophrenia relate to dysfunction in the prefrontal cortex (PFC). In the transition to early adulthood, change in dopamine signalling and GABAergic activity facilitated by dopamine and inputs from the ventral hippocampus occur. This period of time is also marked by a reduction in network synchronisation, and is thought to be particularly vulnerable to disruption. The coherent oscillatory activity characteristic of the adult brain is dependent upon the function of parvalbumin neurons; both of which, the activity and neurons, are impaired in psychosis and can be temporarily suspended with dopamine $(D)_2$ receptor blockade in adulthood.

In humans with 22Q11 deletion syndrome, 24% of adults go on to develop schizophrenia, compared with 1% across the population. Mukherjee and colleagues confirmed that LgDel, a mutant strain of mice that mimic this deletion, display the same deficit in PFC network synchronisation in adulthood as their human counterparts as well as an impairment of parvalbumin neuronal plasticity, and associated cognitive deficits – all of which escalate in the transition to adulthood, making it an excellent representation of the syndrome and disorder.[5] Using $D_2$ receptor targeting with eticlopride and haloperidol in adult mice, the authors achieved a complete, but temporary, recovery to baseline within 6 h of administration. Unlike adults, when there is more global dysfunction, early adolescent mice (postnatal day 40) show brain area-specific parvalbumin deficits, largely in the ventral hippocampus, and less so in the medial PFC. Targeting late adolescence (postnatal days 60–70), the authors found that $D_2$-receptor antagonist treatment, or chemogenetic activation of parvalbumin neurons, in either the ventral hippocampus or the medial PFC every other day for at least 6 days produced a system wide, permanent rescue of parvalbumin plasticity, network activity and cognitive dysfunction. This reinforces late adolescence as a critical sensitive widow for intervention in the progression to schizophrenia, especially in those with 22Q11 deletion syndrome, but wider application should be explored.

Availability of a schizophrenia prophylactic is a heady thought, indeed. Of course, all caveats around translation from animal models to humans and, if successful, the navigation of the resulting ethical quandaries around targeting those who may not go on to develop the disorder apply. However, primary prevention of mental illness has always been the dream, and it is so good to be reminded of the very real possibility.

**Finally, greater levels of narcissism have long been associated with climbing the career ladder, but do narcissists go for promotion or do promotions create narcissists?** Data have shown that those with greater narcissistic traits are more likely to apply for higher positions that accord with their sense of status. Further, organisations are also more likely to choose such individuals as their typically dominant behaviour sits well with prototypes of 'leaders' (and, those interviewing them are more likely to have similar traits: research shows narcissists like picking other narcissists). But change over time has been less explored: does a growth in power and privileges increase an individual's sense of self-worth and entitlement, or might it imbue greater humility and wisdom as one's true limitations are better seen? Most work has been observational and cross sectional, so could not explore change with time.

Wille *et al* test this bidirectionality, assessing a cohort of highly educated professionals three times over a 22-year time frame.[6] They found that overall narcissism increased within individuals alongside their career progression, although there was considerable variation among individuals, and in some it did decline with time. However, initial narcissism was more likely to lead to early career leaps, rather than early promotion leading to growths in narcissism. So, both aspects appear true: narcissists are more likely to go for and achieve promotion, and the trait grows with time. However, it was the final finding, which went against prediction, that was perhaps most interesting. Those with significant early career rapid promotions showed smaller subsequent increases in narcissism than those who took longer to get to the top. The authors offer an interpretation of this that narcissistic traits are adaptive in an early 'emerging zone' of new leadership, but are harmful in an 'enduring zone' of longer-term roles, where better social skills are required and continued narcissism will hinder one's career. Overall, we know that for those of you working in the National Health Service and academic institutions, these findings will come as an unexpected shock. To be clear, as our legal representatives have just reminded us, the Kaleidoscope team are not saying your boss is a narcissist: to paraphrase Francis Urquhart, you might very well think that, we couldn't possibly comment.

## References

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