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Normality and the Ordinary Concept of Disease

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Abstract

Philosophical accounts of health and disease typically assume that disease involves deviation from normality, but disagree on whether normality is descriptive (e.g., statistical frequency) or evaluative (e.g., harm). Recent experimental work suggests that ordinary judgments of normality integrate frequency and value, raising the question of whether disease concepts inherit this mixed structure. Across two preregistered vignette-based experiments, we tested how frequency, valence, and explicitly stated normality influence judgments of disease, dysfunction, and health in both somatic and mental domains. In Study 1, both frequency and valence affected disease judgments, but frequency effects disappeared when controlling for normality. In Study 2, explicit normality information strongly shaped disease, dysfunction, and health judgments, with substantially larger effects than frequency. These findings suggest that ordinary disease concepts rely on a mixed notion of normality.

Keywords: Health; Disease; Normality

Ordinary judgments about whether a given state counts as healthy, diseased, or dysfunctional play an important role in everyday reasoning, public debate, and medicine. They shape symptom interpretation, help-seeking, and responses to health advice. But what criteria guide these judgments and do the same criteria operate across different health-related concepts? Philosophy of medicine offers a useful starting point. Competing accounts agree that disease involves deviation from normality but disagree about how normality should be understood and how it figures in classifying bodily and mental states.

Value-free views of health and disease characterize normality as a descriptive notion, typically in terms of statistical frequency or species-typical functioning (e.g., Boorse 1997; 2022). Health is thus understood as statistically normal functioning, while disease and dysfunction are departures from a statistical baseline of normality tied to survival or reproduction. Building on this first view, we might expect to find an effect of purely statistical information on people's attributions of disease.

Value-laden views of health and disease characterize normality as an evaluative notion, defined relative to standards such as harm or undesirability. Disease and dysfunction are departures from an evaluative baseline of normality rather than from statistical frequency (e.g., social or cultural norms; see Sedgwick 2022 /1982, 28–29)

Building on this second view, we might expect to find that attributions of disease are value-laden and that this

value-ladenness is driven in part by an evaluative representation of the degree to which a state is normal. On this picture, statistical frequency would play at most a secondary role, contributing little beyond its indirect association with evaluative assessments.

Finally, empirical research on the concept of normality suggests that people's ordinary notion of normality might be a *mixture* of the statistical and the evaluative. Existing research on this topic has not already looked at normality judgments in the context of disease (for a review, see Varga, Latham, & Machery, 2026), but studies have explored the way people ordinarily use the word “normal.” The results do not indicate that use of this word is determined entirely by descriptive representations of statistical frequency or entirely by evaluative judgments. Instead, use of the word appears to be influenced by both of these factors (Bear & Knobe 2016; Wysocki 2020). In other words, the degree to which a particular state is labeled “normal” appears to be influenced by the degree to which that state is statistically frequent and also by the degree to which that state is judged to be evaluatively good. This pattern might potentially emerge in people's judgments about whether medically relevant states count as “normal.”

If we do find a statistical/evaluative mixture in people's use of the word “normal” in these cases, we face a question about how this mixed representation relates to people's attributions of disease. One possibility would be that this mixture is just a quirk of the way people happen to use one specific word. Then it could be that the representation of normality that plays a role in attributions of disease is not a mixed one but rather a purely statistical notion that has nothing to do with evaluative considerations. At the other extreme, it could be that this mixed statistical/evaluative notion of normality is absolutely central to people's ordinary attribution of disease. Perhaps both statistical information and evaluative information influence attribution of disease, and both of those effects arise simply because of the role of normality. Thus, when we control for normality judgments, there would be no further impact of either statistical information or evaluative information. Finally, it could be that the correct answer lies between two extremes. For example, it could be that disease attributions are influenced by a mixed statistical/prescriptive representation and that this mixed representation is what explains the impact of statistical information on disease attribution, but then it

could be that there is also a further impact of evaluative information that is not explained by people's mixed representation of normality. On this last possibility, people's normality judgments would be influenced by both statistical and evaluative information, and there would be no further impact of statistical information after controlling for the judgments, but there still would be an impact of the evaluative information even after controlling for normality.

Two studies explore this question. In Study 1, we manipulate both statistical and evaluative information and measure both attributions of normality and attributions of normality, disease, dysfunction, and health. In Study 2, we directly provide information about the degree to which a state was thought to be "normal," independent of statistical frequency, and measured attributions of disease, dysfunction, and health.

Study 1

In this first study, we manipulated frequency and valence (good vs. bad) and examined their effects on judgments of normality and also on people's use of disease concepts. This design allows us to explore the hypothesis that mixed statistical/evaluative notion of normality plays a role in judgments of disease. We can ask (a) whether normality judgments are affected by both statistical and evaluative information, (b) whether the use of disease concepts is affected by both statistical and evaluative information, and, finally, (c) whether normality judgments predict the use of disease concepts in a model that also includes the statistical and evaluative manipulations.

Although our main focus was on judgments of disease, we also examined judgments of health and dysfunction. This design makes it possible to see whether the pattern of effects of frequency, valence and normality that we see on disease judgments also emerges for health judgments and dysfunction judgments. Within existing research, some accounts treat health as closely tied to disease (typically as the absence of disease, e.g., Boorse, 1997) whereas others hold that health involves some further positive state or capability (e.g., Nordenfelt, 2017; Venkatapuram, 2013). Similar points arise for the concept of dysfunction (Varga and Latham, 2025). One possible view would be that the impacts of frequency, valence and normality on dysfunction judgments will be basically the same as the impacts on disease judgments, while another possible view would be that there is a distinctive type of normativity that can be found in disease judgments but not in dysfunction judgments.

Method

This study was preregistered. Preregistration, data and code are available at https://osf.io/yxezn/overview?view_only=b3b306bd907445fb9c40c8109f1aa6ca.

Participants. The final sample comprised 452 participants ($M_{age} = 43.92$, $SD = 13.37$) recruited via Prolific. Of the 500 individuals who completed the study, 48

were excluded for failing the comprehension check; no participants failed the attention check. Gender distribution was 49.3% women, 49.3% men, and 1.3% nonbinary/other. Participants identified as White (75.9%), Hispanic/Latino (8.2%), other (6.2%), Asian (4.9%), Native American/Alaska Native (3.3%), Black/African American (0.9%), or Middle Eastern/North African (0.7%).

Procedure and materials Each participant received a vignette about a medically relevant state and then was asked a series of questions. Participants were randomly assigned to receive either a case in which the state was *frequent* or a case in which the state was *infrequent*. Crossed with this manipulation, they were also randomly assigned to receive a case in which the state was *helpful* or a case in which the state was *harmful*. In addition, for generality, we used three different vignettes, and each vignette could be presented either in a version where it was about a somatic state or a version in which it was about a mental state. Thus, participants were randomly assigned to condition in a 2 (frequency: many vs. few) by 3 (valence: goodvs.bad) x 2 (domain: somatic vs. mental) by 3 (vignette) design. To illustrate, here is one vignette in the many, bad, mental condition:

In large-scale mental health studies, psychiatrists and psychologists have identified a certain way adults' thoughts and feelings respond to hormonal changes across the day. They call this pattern RBX-12.

Researchers report that a large majority of adults has RBX-12, so it is exceedingly common.

Further results suggest that RBX-12 is highly harmful for people who have it: it is associated with unstable cognitive control, difficulty regulating intrusive thoughts and urges, and a high risk of developing mental disorders such as Obsessive–Compulsive Disorder and Panic Disorder.

Participants were asked four questions about the case: whether it is a disease, whether it involves a dysfunction, whether a person who has it is healthy, and whether it is normal. All questions were answered on a 1-7 scale.

Results

Results for all dependent variables are shown in Figure 1. All analyses were conducted using preregistered linear mixed-effects models with random intercepts for vignette and Type III sums of squares with Satterthwaite degrees of freedom. We first examined how the experimental manipulations of Frequency, Valence, and Domain influence judgments of Normality, Dysfunction, Disease, and Health. Complete results for each ANOVA are reported in Table 1.

As preregistered, our primary research question in this analysis was whether there were main effects of frequency and valence on each of the dependent variables. As expected, we found main effects of both frequency and valence on normality ratings. Then, importantly, we also found main effects of both frequency and valence on ratings of disease, health and dysfunction. Though we found main effects of both factors on each dependent variable, the size of these effects varied from one dependent variable to the

next. Frequency had a larger effect on normality than on disease, health or dysfunction, whereas valence had a larger effect on disease, health and dysfunction than on normality.

In addition, we found a number of other effects. There were no main effects of domain on normality, disease or health, but for dysfunction, there was an effect such that the mental was seen as more of a dysfunction than the somatic. We also found a number of significant interactions, which were decomposed using preregistered simple-effects tests (Table 2). For normality, we find a Frequency x Valence interaction, such that the effect of valence was larger in the ‘many’ condition than in the ‘few’ condition. This interaction was then qualified by a three-way interaction such that the interaction between frequency and valence was larger in the mental conditions than in somatic conditions. For disease, health and dysfunction, we find a two-way interaction in the opposite direction: the effect of valence was larger in the many condition than in the few condition.

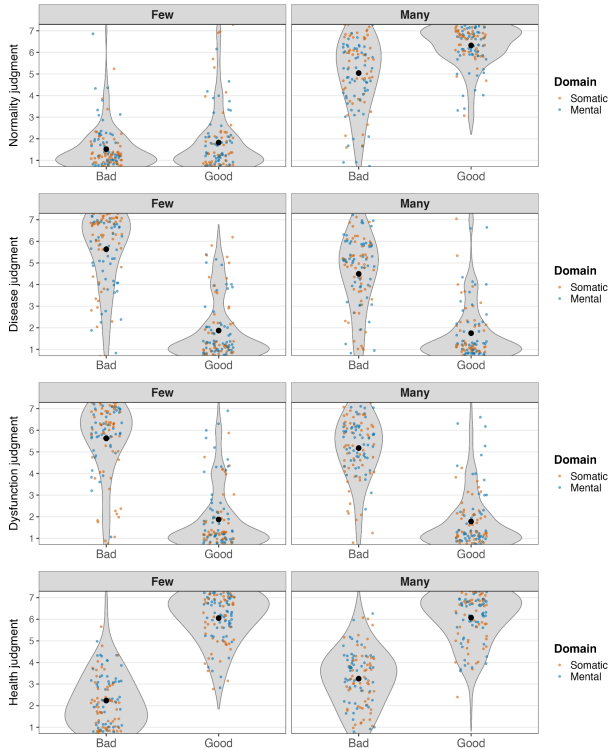


Figure 1. Violin plot showing ratings for normality, disease, dysfunction and health by frequency and valence. Jittered points show individual responses. Larger black points show means.

Table 1: Mixed-Effects ANOVAs for each dependent variable in Study 1.

Effect	Normality	Dysfunction	Disease	Health
Frequency	$F(1,452)=1196.05$, $p < .001$, $\eta_p^2=.73$	$F(1,449)=4.46$, $p=.035$, $\eta_p^2=.01$	$F(1,449)=19.41$, $p < .001$, $\eta_p^2=.04$	$F(1,452)=21.03$, $p < .001$, $\eta_p^2=.04$

Valence	$F(1,452)=46.28$, $p < .001$, $\eta_p^2=.09$	$F(1,449)=72.67$, $p < .001$, $\eta_p^2=.62$	$F(1,449)=516.73$, $p < .001$, $\eta_p^2=.54$	$F(1,452)=885.14$, $p < .001$, $\eta_p^2=.66$
Domain	$F(1,452)=3.26$, $p=.071$, $\eta_p^2=.01$	$F(1,449)=9.79$, $p=.002$, $\eta_p^2=.02$	$F(1,449)=1$, $p=.978$, $\eta_p^2=.00$	$F(1,452)=2.77$, $p=.097$, $\eta_p^2=.01$
Frequency × Valence	$F(1,452)=18.00$, $p < .001$, $\eta_p^2=.04$	$F(1,449)=1.91$, $p=.167$, $\eta_p^2=.00$	$F(1,449)=12.09$, $p < .001$, $\eta_p^2=.03$	$F(1,452)=19.22$, $p < .001$, $\eta_p^2=.04$
Frequency × Domain	$F(1,452)=8.37$, $p=.004$, $\eta_p^2=.02$	$F(1,449)=0.28$, $p=.594$, $\eta_p^2=.00$	$F(1,449)=2.16$, $p=.143$, $\eta_p^2=.01$	$F(1,452)=0.31$, $p=.575$, $\eta_p^2=.00$
Valence × Domain	$F(1,452)=0.02$, $p=.881$, $\eta_p^2=.00$	$F(1,449)=0.13$, $p=.721$, $\eta_p^2=.00$	$F(1,449)=0.01$, $p=.927$, $\eta_p^2=.00$	$F(1,452)=0.78$, $p=.378$, $\eta_p^2=.00$
Frequency × Valence × Domain	$F(1,452)=4.11$, $p=.043$, $\eta_p^2=.01$	$F(1,449)=0$, $p=.991$, $\eta_p^2=.00$	$F(1,449)=0.78$, $p=.376$, $\eta_p^2=.00$	$F(1,452)=0.57$, $p=.451$, $\eta_p^2=.00$

Table 2: Follow-Up Tests to decompose interactions in Study 1.

DV	Frequency	Contrast	t(df)	p
Normality (Mental)	Few	Bad – Good	-0.19 (228)	.849
Normality (Mental)	Many	Bad – Good	-6.33 (230)	< .001
Disease	Few	Bad – Good	18.59 (456)	< .001
Disease	Many	Bad – Good	13.35 (457)	< .001
Health	Few	Bad – Good	-24.18 (458)	< .001
Health	Many	Bad – Good	-17.55 (460)	< .001

Finally, we explored the relationship between normality and each of the other dependent variables (see Figure 2). The key questions were (a) whether normality predicts these other variables even when controlling for condition and (b) whether the frequency manipulation predicts these variables even when controlling for normality.

To address these questions, we ran a separate ANCOVA for each of these other dependent variables, entering normality as a covariate along with all of the variables included in the previous analysis (Table 3). For each of the three dependent variables, we found a significant effect such that normality ratings predicted that variable even controlling for condition. We then asked whether there was still a significant effect of the frequency manipulation even controlling for normality ratings. For disease and health, there was no significant effect of frequency manipulation after controlling for normality; for health, there was still a significant effect of frequency manipulation, but with a small effect size ($\eta_p^2 = 0.01$). Interestingly, the result for the valence manipulation was very different. Even controlling for normality, there remained a substantial and significant effect of valence.

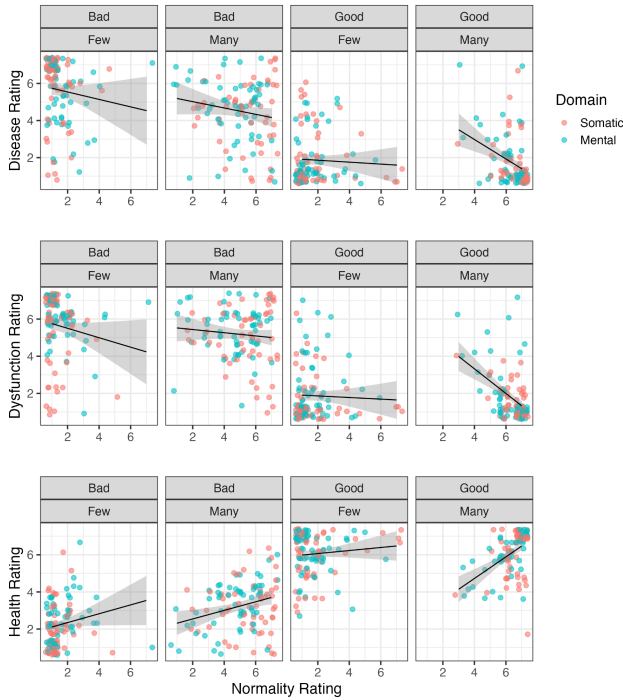


Figure 2. Scatterplot showing the relationship between normality ratings and each of the other dependent variables (disease, dysfunction, health). Jittered points show individual responses. Lines show OLS regressions.

Table 3: Mixed-Effects ANOVAs in Study 1 with normality entered as a covariate.

Effect	Disease	Dysfunction	Health
Normality	$F(1, 450) = 8.99$, $p = 0.003$, $\eta_p^2 = 0.020$	$F(1, 450) = 9.75$, $p = 0.002$, $\eta_p^2 = 0.021$	$F(1, 452) = 29.82$, $p < .001$, $\eta_p^2 = 0.062$
Frequency	$F(1, 450) = 0.05$, $p = 0.821$, $\eta_p^2 = 0.000$	$F(1, 450) = 2.39$, $p = 0.123$, $\eta_p^2 = 0.005$	$F(1, 452) = 4.72$, $p = 0.030$, $\eta_p^2 = 0.010$
Valence	$F(1, 450) = 438.31$, $p < .001$, $\eta_p^2 = 0.494$	$F(1, 450) = 624.09$, $p < .001$, $\eta_p^2 = 0.581$	$F(1, 452) = 761.28$, $p < .001$, $\eta_p^2 = 0.627$
Domain	$F(1, 449) = 0.08$, $p = 0.777$, $\eta_p^2 = 0.000$	$F(1, 449) = 8.32$, $p = 0.004$, $\eta_p^2 = 0.018$	$F(1, 452) = 4.72$, $p = 0.030$, $\eta_p^2 = 0.010$
Frequency x Valence	$F(1, 450) = 16.27$, $p < .001$, $\eta_p^2 = 0.035$	$F(1, 450) = 3.95$, $p = 0.048$, $\eta_p^2 = 0.009$	$F(1, 452) = 30.34$, $p < .001$, $\eta_p^2 = 0.063$
Frequency x Domain	$F(1, 449) = 1.13$, $p = 0.288$, $\eta_p^2 = 0.003$	$F(1, 449) = 0.91$, $p = 0.340$, $\eta_p^2 = 0.002$	$F(1, 452) = 0.03$, $p = 0.871$, $\eta_p^2 = 0.000$
Valence x Domain	$F(1, 449) = 0.01$, $p = 0.940$, $\eta_p^2 = 0.000$	$F(1, 449) = 0.14$, $p = 0.705$, $\eta_p^2 = 0.000$	$F(1, 452) = 0.76$, $p = 0.383$, $\eta_p^2 = 0.002$
Frequency x Valence x Domain	$F(1, 449) = 0.36$, $p = 0.546$, $\eta_p^2 = 0.001$	$F(1, 449) = 0.08$, $p = 0.775$, $\eta_p^2 = 0.000$	$F(1, 452) = 0.07$, $p = 0.798$, $\eta_p^2 = 0.000$

Discussion

In this first study, we find that people's use of disease concepts is impacted by both a statistical manipulation (many vs. few) and an evaluative manipulation (good vs. bad). People's judgments of normality were also impacted by both of those manipulations. Thus, attributions of both disease and normality are impacted by both statistical and evaluative factors. However, looking at effect sizes, we find that use of disease concepts was impacted more by the evaluative manipulation, whereas normality judgments were impacted more by the statistical manipulation.

Importantly, there was no impact of the statistical manipulation on the use of disease concepts after controlling for normality judgments (except for a small but still significant effect on health), but there was a highly significant impact of the evaluative manipulation even after controlling for normality judgments.

In short, these results suggest that normality judgments about medically relevant states are impacted by both statistical and evaluative factors. The results suggest that this mixed statistical/evaluative judgment does not account for the full impact of evaluative information on disease attributions, but the results do provide at least some support for the claim that this mixed statistical/evaluative judgment accounts for the impact of statistical information on disease attributions.

Study 2

In the previous study, normality was measured rather than manipulated. As a result, it remains unclear whether normality judgments themselves influence disease-related judgments or merely covary with other factors that exert influence. In this second study, we therefore directly manipulate normality information.

Method

This study was preregistered. Preregistration, data and code are available at: https://osf.io/yxezn/overview?view_only=b3b306bd907445fb9c40c8109f1aa6ca.

Participants. The final sample consisted of $N=406$ participants after excluding those who failed the preregistered comprehension check from the 501 participants who completed the study. No participants failed the attention check. Gender distribution was 49.3% women, 48.5% men, and 2.2% nonbinary/other. Race/ethnicity was White (74.6%), Hispanic/Latino (7.6%), other (7.1%), Native American/Alaska Native (5.9%), Asian (2.5%), Black/African American (0.7%), Middle Eastern/North African (0.5%), or not reported (1.0%).

Procedure and materials. Each participant read a vignette describing the same types of states used in Study 1, using similar wording, except that valence was held constant and negative in all cases, and normality was manipulated.

Participants received either a case in which the state was explicitly described as normal or a case in which it was described as not normal. This manipulation was crossed with frequency (many vs. few). As in Study 1, we used three different vignette cases, each of which could be presented in either a somatic or a mental version. Thus, each participant was randomly assigned to one of 24 between-subjects conditions in a 2 (Frequency: Few vs. Many) $\times$ 2 (Normality: Abnormal vs. Normal) $\times$ 2 (Domain: Somatic vs. Mental) $\times$ 3 (Vignette Case) design. To illustrate, we used the same vignette illustrated in Study 1 in the many, bad, mental condition; in Study 2, this vignette was identical except for the addition of an explicit abnormality statement:

[Abnormal] Researchers have extensively studied RBX-12. They have concluded that RBX-12 is clearly not normal.

After reading the case, participants were asked three questions: whether the state counted as a disease, whether it involved a dysfunction, and whether a person who had it was healthy. All questions were answered on 1–7 Likert-type scales.

Results

Results for all dependent variables are shown in Figure 3. All analyses were conducted using preregistered linear mixed-effects models with random intercepts for vignette and Type III sums of squares with Satterthwaite degrees of freedom.

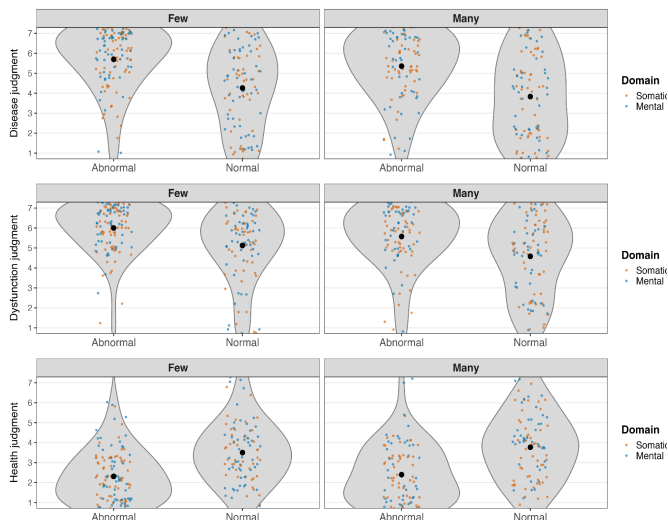


Figure 3. Violin plot showing ratings for disease, dysfunction and health by frequency and normality. Jittered points show individual responses. Larger black points show means.

We first examined how the experimental manipulations of Frequency, Normality, and Domain influence judgments of

Disease and Health. Complete results of the mixed-effects ANOVAs are reported in Table 4.

Table 4 : Mixed-Effects ANOVAs for each dependent variable in Study 2.

Effect	Disease	Dysfunction n	Health
Normality	$F(1, 403) = 71.97, p < .001, \eta_p^2 = .151$	$F(1, 406) = 37.46, p < .001, \eta_p^2 = .084$	$F(1, 406) = 82.58, p < .001, \eta_p^2 = .169$
Frequency	$F(1, 403) = 4.87, p = .028, \eta_p^2 = .012$	$F(1, 406) = 9.87, p = .002, \eta_p^2 = .024$	$F(1, 406) = 1.84, p = .176, \eta_p^2 = .005$
Domain	$F(1, 403) = 0.64, p = .425, \eta_p^2 = .002$	$F(1, 406) = 1.67, p = .198, \eta_p^2 = .004$	$F(1, 406) = 2.97, p = .086, \eta_p^2 = .007$
Frequency $\times$ Normality	$F(1, 403) = 0.09, p = .765, \eta_p^2 < .001$	$F(1, 406) = 0.20, p = .659, \eta_p^2 < .001$	$F(1, 406) = 0.58, p = .445, \eta_p^2 = .001$
Frequency $\times$ Domain	$F(1, 403) = 0.45, p = .501, \eta_p^2 = .001$	$F(1, 406) = 0.00, p = .995, \eta_p^2 < .001$	$F(1, 406) = 0.40, p = .528, \eta_p^2 = .001$
Normality $\times$ Domain	$F(1, 403) = 0.31, p = .577, \eta_p^2 = .001$	$F(1, 406) = 4.61, p = .032, \eta_p^2 = .011$	$F(1, 406) = 0.78, p = .377, \eta_p^2 = .002$
Frequency $\times$ Normality $\times$ Domain	$F(1, 403) = 0.24, p = .624, \eta_p^2 = .001$	$F(1, 406) = 1.65, p = .200, \eta_p^2 = .004$	$F(1, 406) = 0.42, p = .518, \eta_p^2 = .001$

As preregistered, our primary research question was whether there were main effects of frequency and normality on each of the dependent variables. We found robust main effects of normality on all three outcomes. States described as not normal were judged as more disease-like and more dysfunctional, and as less healthy, than those described as normal. We also found main effects of frequency on disease and dysfunction judgments, such that conditions described as more frequent were judged as less disease-like and less dysfunctional than conditions described as less frequent. In contrast, frequency did not have a significant effect on health judgments.

Although both frequency and normality influenced judgments, the relative magnitude of these effects varied across dependent variables. Normality had a larger effect on disease and health judgments than on dysfunction, whereas the effect of frequency was comparatively smaller and was limited to disease and dysfunction.

In addition to these main effects, we observed a small number of other effects. There was a significant Normality $\times$ Domain interaction for dysfunction, which was decomposed using preregistered simple-effects tests (Table 5). The analyses revealed that abnormal conditions were judged as more dysfunctional than normal conditions in both domains, but that this difference was larger in the mental domain than in the somatic domain.

Table 5: Follow-up tests to decompose interactions in Study 2.

DV	Domain	Contrast	t(df)	p
Dysfunction	Somatic	Abnormal – Normal	2.83(412)	.005
Dysfunction	Mental	Abnormal – Normal	5.68(413)	< .001

Discussion

Study 2 manipulated normality information while holding valence constant across all conditions and found that normality information exerts a strong causal influence on judgments of disease, dysfunction, and health. Across all three dependent variables, states described as *not normal* were judged as more disease-like, more dysfunctional, and as less healthy, than states described as normal. Further, the effects of normality were largely consistent across somatic and mental domains.

Even when we manipulate normality, we still find a significant effect of the frequency manipulation. However, the effect of the frequency manipulation was much smaller than the effect of the normality manipulation. For example, on judgments of disease, the effect size for the frequency manipulation was less than a tenth of the effect size for the normality manipulation.

General Discussion

Two studies explored the relationship between normality judgments and disease judgments. In Study 1, manipulations of statistical information and evaluative information both impacted judgment of both normality and disease. Normality judgments predicted disease judgments, and in a model that included normality judgment, there was still an effect of the evaluative manipulation on disease judgment, but there was no longer any effect of the frequency manipulation. In Study 2, an explicit manipulation of normality judgment impacted disease judgment. There was still a significant effect of the frequency manipulation, but the effect of the frequency manipulation was much smaller than the effect of the normality manipulation.

Taken together, the findings directly address the possibilities described in the introduction. They offer evidence against the idea that the mixed structure of normality is unconnected to disease-related judgments: normality judgments strongly predict disease, dysfunction, and health judgments, and manipulating normality generates corresponding changes in these judgments. However, while normality performs this organizing role in disease-related judgments, the findings also caution against a strong

“complete mediation” view on which the influence of frequency and valence on disease-related judgments is wholly explained by normality. Even after controlling for normality, valence remains a strong predictor. Instead, the results fit best with an intermediate position: disease concepts draw on a mixed notion of normality, but evaluative considerations exert an additional influence over and above their impact on normality.

The findings have implications not only for philosophical debates but also for clinical and public-health communication.

First, insofar as they are intended as accounts of the folk concept of disease, our findings put pressure on both purely value-free and purely value-laden accounts. The former are challenged because evaluative information affects disease judgments independently and via normality, while the latter are challenged because descriptive information about frequency affects disease judgments via normality. This suggests that a hybrid account integrating both statistical frequency and valence might be best positioned to accommodate the findings.¹ Our results could help such an account spell out how these ingredients are weighted and organized in judgments of health, disease, and dysfunction.

Second, the findings suggest a tradeoff for clinical and public-health communication. On the one hand, stressing how common a condition is may help destigmatize it, but if frequency is taken as evidence of normality, it can also reduce the perceived disease status of the condition and motivation to seek help. On the other hand, stressing abnormality may increase perceived seriousness, but at the risk of increasing stigma.

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¹ We should note that there is a hybrid account of health and disease holding that descriptive and evaluative considerations matter (Wakefield 1992; 2014). However, this account does not consider statistical frequency as the relevant descriptive component, appealing instead to an etiological (selected effects) account of function. Our findings support a different hybrid structure based on judgments of statistical normality.

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