First Principle Methods for Causal Discovery

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Wings are a constraint that makes
it possible to fly.
— Robert Bringhurst

To my grandchildren...

Acknowledgements

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Lausanne, July 20, 2020 D. K.

Abstract

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Contents

1 Introduction

1.1 Problem and Motivation

Suppose we are given samples of data say X and Y, s.t.

$$X = x_1, ..., x_n$$
$$Y = y_1, ..., y_n$$

For example, we may be measuring the blood pressure and heart rate of Alice at time k, say x_k and y_k respectively. Further, suppose we are unaware of her context, for example, Bob hacked into Alice's apple watch and so can only read X and Y – but he has no idea of anything she might be up to.

Bob then observes the following trend:

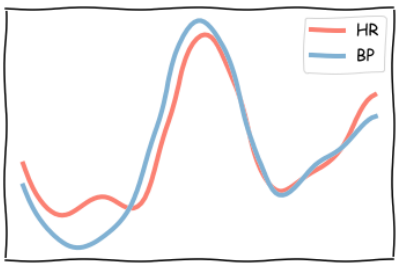


Figure 1.1 – Heart rate (HR) and Blood pressure (BP) of Alice.

Bob, having studied data science, is well aware of the fallacy of the law of small numbers[1]. He

[1] The law of small numbers is the error of concluding too much from too few data.

therefore checks again the data the next day at a slightly different time. He again observes a similar trend, and is now more confident in the existance of a causal relation and – having neglected biology as being beneath him – makes the conjecture that either blood pressure causes heart rate, or perhaps the other way around.

Given this strong correlation, Bob asserts that he may either model X as a function of Y or the other way around. He proceeds to find some f s.t. $f(X) \approx Y$. The next day, to his dismay, he notices that his model has terrible performance when evaluated on new data. He then proceeds to see what is going on, and observes the following:

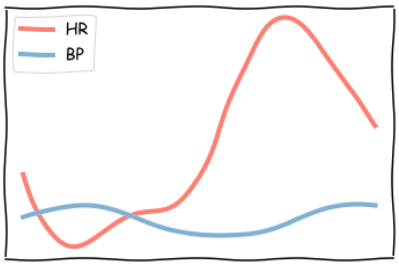


Figure 1.2 – Heart rate (HR) and Blood pressure (BP) of Alice.

As it turns out, in the last few days, Alice was working hard on finishing her thesis and the deadline had been the previous day. But how, Bob wondered, could this have changed the relationship between BP and HR? Finally, admitting to himself that machine learning alone is not enough to understand the world; Bob spends some time learning about the heart. It turns out, that fear triggers a "flight or fight" response that increases both the heart rate and blood pressure; Interestingly your heart rate and blood pressure won't always rise and fall in sync.

So what did Bob learn[2]?

1. When we train a model with some data, when we use it on some newly aquired data, we might face a **covariate-shift** – that is, the distribution might change due to the context changing.

2. When we see correlation it might be spurious due to a **confounder** – fear was the **confounder** of the heart rate and blood pressure.

3. His degree in Data Science is worth less than he thought; **machine learning is in fact not a panacea**, contrary to common culture. However, applied with domain knowledge and causal reasoning it may very useful.

[2] Note that heart rate and blood pressure are intimately linked, and the story between them is more complicated. The plots were randomly generated using a gaussian process, however they do resemble some real examples that can be found in google images.

If Bob was able to incorporate these notions into his machine learning models, then it might have been more robust to the covariate-shift. To give a more concrete example, there is a "neural net tank urban legend"[3] , where a neural network accuratly predicts if there is a tank or not in an image, but it turns out it uses the weather as a predictor. From this it is clear that the model will preform badly under covariate shift, and indeed it makes the case that incorporating causality to a model should make it more robust as Schölkopf (2019) argues. Note that this is in effect the issue with generalisation in machine learning: how can we ensure that we learn *meaningful* representations (features about the tanks) rather than just correlations (the weather) useful for train accuracy.

As for confounders, it is impossible to say anything in general[4]. We must therefore specify a causal model, and then see what gurantees we can give under what assumptions. Even in the absence of confounders it is highly non trivial to determine causality.

As this simple example illustrates, causality is related to many interesting questions; perhaps, one of the most simple questions we can ask – and the one that we will explore – is, given that either X causes Y, or Y causes X (we assume no confounders) then, when can we predict the direction of causality? If yes, how?

In the figure bellow (figure 1.3) can you tell if X causes Y? Or perhaps it is the other way around? The right answer is that X causes Y, and we will show algorithms that can accuretly predict causality in such settings with as few as 75 samples.

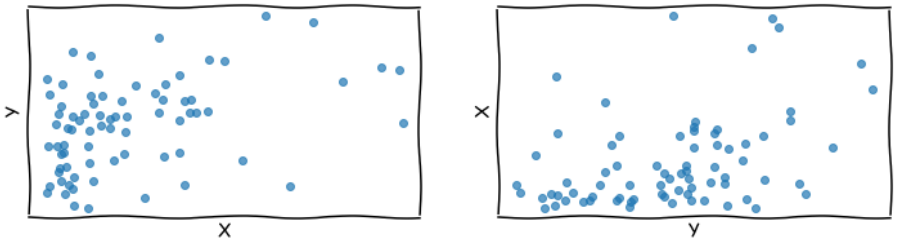


Figure 1.3 – 75 samples of data X, Y. The samples are generated independently as follows: $y_i = f(x_i) + n_i$ where x_i is drawn from an expoential distribution and n_i is drawn independently from a gaussian one and $f(x) = 10\tanh(x) + 4\sin(x) + x + x^2$

[3] More about this story here: https://www.gwern.net/Tanks.

[4] For most of the 20th century, a huge debate took place to determine the question of whether or not smoking caused cancer. A clever argument against a causal relation was that there existed a gene that made a person both want to smoke and more prone to cancer; even the father of modern statistics himself thought this explanation more plausible (For a good read on how science is and was used for wrong see the excellent book Oreskes (2011)).

1.2 Causality

We have seen in the introduction that determining and understanding causality may indeed be useful; but what exactly is causality? When we say X causes Y i.e. $X \to Y$, we have an intuitive idea of what it means; but how should we formalise it?

Suppose that we are given two random variable X, Y with joint distribution $p_{x,y}$. Intuitively we would say that $X \to Y$, if we intervene in X and then see an effect Y. In particular we will denote do(x) – short for do$(X = x)$ – as an intervention that forces the variable X to have the value x, and leaves the rest of the system untouched. Following the convention inspired by Pearl (2000), we define the resulting distribution as $p_{Y|do(x)}$.

In supervised learning the goal is often to estimate $p_{y|x}$; while it is tempting to say that this is the same as $p_{y|do(x)}$, they may in fact be very different. Suppose x is 1 if a person smokes and 0 otherwise, similarly let y be 1 if a person has cancer and 0 otherwise. If we then ran immoral **randmoized trials**[5] and forced a subset of the population at random to smoke, then we could estimate[6] $p_{y|do(x)}$. If as some people believed, there existed a gene that made people both smoke and prone to cancer, then $p_{y|do(x)} = p_{y|x}$; however,

This motivates the following definition:

Definition 1 *We say that X **causes** Y if $p_{y|do(x)} \neq p_{y|do(x')}$ for some x, x'*

Note that causal relations can also be *cyclic*, i.e. X causes Y which in turn causes X ad infinitum. While this deserves consideration as many systems have feedback loops we will for simplicty not look at such setting.

If we have the random variables X, Y, and Z, then depending on how they are causaly linked, then we will have different relationships between the marginals and the Pearl's "do" conditional. For example, if $X \to Z \to Y$, then we will have that $p_X = p_{X|do(y)}$, but $p_Y \neq p_{Y|do(x)}$.

Then, assuming that we get both interventional and observational data it suffices to check to which of the 6 possible structure the data corresponds to (at least in theory). However, if we are interested in using only observational data, then we will need to make conessions; in particular, we will make some assumptions about the causal structure, and then if we can show that we can infer the causal structure in such a setting, we shall call it **identifiable**.

If we have a 3 or more varuables, one can use conditional independence tests to check whether two random variables are directly linked (to check for direct causality – the case when there is

[5] The power of randmoized trials is that they average out potential confounder – e.g. a gene that causes both an inclination to smoke and cancer. This is also knowm as A/B testing and is essential to determine if a new medicine has the desired effect and that a new UI might maximise user addicton to an app.

[6] Note that to estimate $p_{y|x}$ we simply need to sample people at random to determine $p_{y,x}$, we can then use bayes' formula to determine $p_{y|x} = \frac{p_{y,x}}{p_x}$

an edge from X to Y). One can extend the notion of conditional independence to consider sets of edges instead of single edges, see for example graphical models.

In some sense the 2d case is hard because we cannot use conditional independence tests.

The underlying structure behind a causal model is what is known as Strucutual Equation Models (SEM). Essentially it is a model specification; and the key insight is that it should not be reversible.

1.3 Proposed Methods

We propose two methods to deal with the ANM – one less practical, but with a nice theoretical analysis; and another more practical, but perhaps with a less pleasing analysis. However both are based on a first principle approach with known asymptotics in mind.

We note that in the analysis / procedure we split the data in 2, first to train the model, and then to perform the score computation - Need to expand on this

Will be easier to do once they are finished.

1.4 Outline

The thesis is divided in two parts,

background etc blup blue die blah blu

Do once finished, but main idea

Theory and background material

Methods

Experiments

Part I Preliminaries

2 Causal Inference

2.1 Bivariate causal model

2.1.1 ANM

We will now introduce[1] the bivariate causal model and define the particular class of such models that we will work on. To avoid any sort of technical complications, hen we talk about functions, they will always be Borel measurable – which in any case consists of most functions of interest.

For the bivariate case, if we have $Y \in \mathbb{R}$ as a direct cause of $X \in \mathbb{R}$, then we can model the relationship as follows:

$$\begin{cases} Y = f(X, Z) \\ X \perp\!\!\!\perp Z, \quad X \sim p_x, \quad Z \sim p_Z \end{cases} \tag{2.1}$$

If we assume that there is no confounder, that there is no sampling bias[2] and no cycles then it is natural to assume that $X \perp\!\!\!\perp Z$.

While we can model Z as either a scalar of a vector, we can without loss of generality assume it to be a scalar. It can be shown that if Z is a vector, then one can construct a simpler model with scalar noise, which has the same observational and interventional distribution Mooij et al. (2016).

An important remark is that given the direct model in equation 2.1, we can find some $\tilde{f}$ and $\tilde{Z}$

[1] Note that this introduction follows closely that of Mooij et al. (2016), and we encourage the reader to have a look to fill in details that have been omitted here.

[2] If you consider the example given in the introduction, then if Bob always sampled when Alice was excercising, then this would have lead to sampling bias, as during excercise both heart rate and blood pressure tend to increase.

such that

$$\begin{cases} X = \tilde{f}(Y, \tilde{Z}) \\ Y \perp\!\!\!\perp \tilde{Z}, \quad Y \sim p_y, \quad \tilde{Z} \sim p_{\tilde{Z}} \end{cases} \tag{2.2}$$

with the important property that it induces an equivalent observational distribution $p_{X,Y}$ as that of equation 2.1. However in general the interventional distribution will differ. In particular this means that with observational data alone we are not able identify the right causal direction.

We must therefore make further assumptions on f that break this symmetry and allow us to make causal inference. In particular we will consider the following subset of models from the ones introduced in equation 2.1; where we restric the noise to be additive.

Definition 2 *Given a triplet (p_X, p_Z, f), consisting of two finite mean densities and a function $f : \mathbb{R} \to \mathbb{R}$, we can define a **bivariate Additive Noise Model (ANM)** $X \to Y$*

$$\begin{cases} Y = f(X) + Z \\ X \perp\!\!\!\perp Z, \quad X \sim p_X, \quad Z \sim p_Z \end{cases}$$

If the induced density $p_{x,y}$ has a density with respect to Lebesgue measure, we say that $p_{x,y}$ satisfies the ANM $X \to Y$.

Given such a model, we are interested in the cases when the observational distribution $p_{x,y}$ can only lead to one causal explenation; this motivates the following definition:

Definition 3 *If the joint density $p_{x,y}$ satisfies an ANM $X \to Y$, but does not satisfy ANM $Y \to X$, then we call the ANM $X \to Y$ **identifiable**.*

Intuitively non-linearities due to f will break the symmetry needed to find a reverse ANM. This is what Hoyer et al. (2009) and friends explore; they show that for the a triplet (p_X, p_Z, f) to generate a non-identifiable can happen only if they satisfy a particular differential equation that loosely speaking cannot happen in the generic case: in other words the forward model $X \to Y$ cannot be inverted.

If f is linear, then one can give a much more precise statement about Identifiability:

Theorem 1 *Let X and Y be random variables, such that*

$$Y = aX + Z, \quad X \perp\!\!\!\perp Z, \; a \neq 0$$

Then we can reverse the process, i.e. there exists $\tilde{a} \in \mathbb{R}$ and a noise $\tilde{Z}$ such that

$$X = \tilde{a}Y + \tilde{Z}, \quad Y \perp\!\!\!\perp \tilde{Z}$$

if and only if $X, Y, Z, \tilde{Z}$ are Gaussian distributed.

The proof is a simple appilcation of the Darmois-Skitovich Theorem. The theorem also plays an imporant role in ICA; it states the following:

Theorem 2 (Darmois-Skitovich) *Let X_i, $i \in [n]$ be independent random variables, and let α_i, β_j be non zero constants. Then, if the random variables*

$$L_1 = \sum_{i \in [n]} \alpha_i X_i$$

$$L_2 = \sum_{i \in [n]} \beta_i X_i$$

are independent, i.e. $L_1 \perp\!\!\!\perp L_2$; then all the random variables X_i are gaussian.

We now prove Theorem 1 using the Darmois-Skitovich Theorem.

Proof: For the "only if" part, note that by simple manipulation, we have the following:

$$\begin{bmatrix} Y \\ \tilde{Z} \end{bmatrix} = \begin{bmatrix} a & 1 \\ 1 - \tilde{a}a & -\tilde{a} \end{bmatrix} \cdot \begin{bmatrix} X \\ Z \end{bmatrix}$$

If $\tilde{a} \neq 0$ and $1 - \tilde{a}a \neq 0$ then by Darmois-Skitovich, the result follows.

We will next show that both of these conditions must be true for the process to be reversible:

1. If $\tilde{a} = 0$ then $\tilde{Z} = X$, but then $X \perp\!\!\!\perp Y$, a contradiction[3].
2. Finally, if $1 - \tilde{a}a \neq 0$ then $\tilde{Z} = -\tilde{a}Z$, and thus $-\tilde{a}Z \perp\!\!\!\perp Y$, a contradiction.

We have thus show the "only if" part.

We next show the "if" part; first assume that X and Z are Gaussian random variables. It is easy to verify that: $\mathrm{Cov}(Y, \tilde{Z}) = a(1 - a\tilde{a})\mathrm{Var}(X) - \tilde{a}\mathrm{Var}(Z)$. Thus if we set $\tilde{a} = \frac{a\mathrm{Var}(X)}{a^2\mathrm{Var}(X) + \mathrm{Var}(Z)}$ we get that $\mathrm{Cov}(Y, \tilde{Z}) = 0$, and since they are gaussian random variables we get that they are also independent.

□

[3] Note that $X \perp\!\!\!\perp aX + Z$ is trivially false in the discrete case, but if both are continuous then we need to be a bit more careful as is the case with degenerate random variables; but essentially the same holds (see Peters (2008)).

2.2 Methods

2.2.1 Introduction

The following lemma[4] motivates the first class of methods that we will present:

Lemma 1 *Given a joint density $p_{x,y}$ of two random variables X, Y s.t. the conditional expectation $\mathbb{E}(Y|X=x)$ is well-defined for all x and measurable. Then, $p_{x,y}$ satisfies a bivariate Additive Noise Model $X \to Y$ if and only if $E_Y := Y - \mathbb{E}(Y|X)$ has finite mean and is independent of X.*

In practice we get some data from $p_{x,y}$; say $\mathcal{D}_N = \{(x_i, y_i)\}_{i\in[N]}$. We can then either split it into a test/train in order to first fit a regression which we then evaluate using the test set. If the data is scarce we may alternatively recycle the data – i.e. reuse it for both training and evaluation.

First we estimate through regression the function $x \mapsto \mathbb{E}(Y|X=x)$, say $\hat{f}$; then compute the estimated residual $\hat{e} = \hat{f}(X) - Y$. We then compute some score function $C(X, \hat{e})$, the idea being that the score function will capute the level of independence between X and $e \approx Z$; e.g. we the score could be the empirical mutual information, in which case a 0 distance would asymptotically mean that they are indepent (assuming our estimate $\hat{f}$ is arbitrarily accurate). Thus a low score would be evidance for an ANM in that direction; note that we can do the same by reversing the roles of X and Y, and we can compute the score for the reverse model. We can then compare the scores and use this as a criteria for inference. We write down this idea more explictly here Algorithm 1.

In order to show that such a procedure is consistent we need 3 things:

1. $p_{x,y}$ satisfies either $X \to Y$ or $Y \to X$, but not both.
2. The regression method should be **suitable** for regressing Y on X.
3. If $X \to Y$, then asymptotically $\hat{C}_{X\to Y} < \hat{C}_{Y\to X}$

As for the first point, additive can be checked blah blach

as for the second one

2.2.2 LinGam

blah

NOTE talk about the regression -> residual decompo (with indep test at the end)

[4] A simple proof can be found here Mooij et al. (2016)

Algorithm 1 General procedure to decide whether $p_{x,y}$ satisfies and ANM $X \to Y$ or $Y \to X$

Input:

1. I.i.d samples $\mathcal{D}_N = \{(x_i, y_i)\}_{i \in [N]}$ of X and Y
2. Regression method
3. Score estimator $\hat{C} : \mathbb{R}^n \times \mathbb{R}^n \to \mathbb{R}$

Output: $\hat{C}_{X \to Y}$, $\hat{C}_{Y \to X}$, dir

1. Split the $\mathcal{D}_N$ in half randomly to obtain $\mathcal{D}_{train}$ and $\mathcal{D}_{test}$
2. Use the regression method on the training data $\mathcal{D}_{train}$:
   - $\hat{f}_X$ of the regression function $x \mapsto \mathbb{E}(Y|X = x)$
   - $\hat{f}_Y$ of the regression function $y \mapsto \mathbb{E}(X|Y = y)$
3. Estimate residuals using the predicted regressions on the test data $\mathcal{D}_{test}$:
   - $\hat{\mathbf{e}}_\mathbf{Y} := \mathbf{y} - \hat{f}_Y(\mathbf{x})$
   - $\hat{\mathbf{e}}_\mathbf{X} := \mathbf{x} - \hat{f}_X(\mathbf{y})$
4. Compute scores to measure dependence between inputs and estimated residuals based on the test data $\mathcal{D}_{test}$
   - $\hat{C}_{X \to Y} := \hat{C}(\mathbf{x}, \hat{\mathbf{e}}_\mathbf{Y})$
   - $\hat{C}_{Y \to X} := \hat{C}(\mathbf{y}, \hat{\mathbf{e}}_\mathbf{X})$
5. Output $\hat{C}_{X \to Y}$, $\hat{C}_{Y \to X}$, and

$$\text{dir} := \begin{cases} X \to Y & \text{if } \hat{C}_{X \to Y} \leq \hat{C}_{Y \to X} \\ X \to Y & \text{otherwise} \end{cases}$$

2.2.3 HSIC

Talk about IPM vs $f-$divergence.

To test whether two random variables are independent X, Y

2.2.4 MAX entropy

related to what we did, note that it would work if everything was discrete!

2.2.5 Other methods

briefly mention ICGC CGNN Bayesian paper?

2.3 SNR and causality

In this section we give a brief commentary about causality from the perspective of communication theory.

In virtually most of the predictive fields, noise is the enemey; Indeed, in the absence of noise, finding the best linear fit to a linear model is trivial. Emre Telatar, a powerful information theorist, liked to jest in his digital communication course that "without noise, we communication engineers would be without a job".

Indeed, for most of the early 20th century[5] noise was keeping engineers busy as they devised clever schemes to fight noise. At the time the whole business was very experimental as no one had come close to understanding noise in the context of transmission; questions such as "Is it possible to send a message with arbitrary reliability?" and "What is the theoretical maximum amount of information that can be reliably sent?", were questions that no one had come close to solving.

Then Shannon came along, in his Magnum Opus, Shannon (1948), he not only formalised the foundations of information Theory, but he also proved[6] most of the main results in it. In particular, he showed that for the AWGN (Additive white nois gaussian channel), it is possible to realiably transmit at most C bits per time unit:

$$C \propto log(1 + SNR)$$

Where SNR is the celebrated signal to noise ratio – $SNR = \frac{\mathbb{E}(X^2)}{\mathbb{E}(N^2)}$. As we would expect, if $SNR \to \infty$ then we can send an arbitrary amount of information per time unit (the only limit

[5] quote comm book
[6] Shannon had a very deep...

is the physical one, i.e. the speed of light). Conversely if $SNR = 0$ then we find ourseleve at a rave, not matter how much we yell, our friends will not be able to understand us.

If we return to the question of causality; a somehwhat trivial observation is that if the mechanism is injective[7], in the absence of noise, it is not possible to say anything about the causal direction of the mechanism. Here too noise is the benelovent giver of jobs, albeit not for the same reasons. Interestingly, we can use noise to help us deduce the causal nature of a process.

We will now see what perhaps could be considered the most simple causal set up, and describe a method for causal inference. We will then see that SNR also plays an important role.

Consider the linear additive noise model – our first causal model!

$$\begin{cases} Y = aX + E_Y \\ X \perp\!\!\!\perp E_Y,\ X \sim p_x,\ E_Y \sim p_{E_Y} \end{cases}$$

Suppose we are given n samples of the above process:

$$y_i = ax_i + z_i, i \in [n]$$

We collect these into vectors say y, x and z; note that we do not have access to the latter, but it will come in handy for the derivation that follows.

One common idea is to first compute the residuals for both possible regression models, and the to check which residual is less dependent on x and y respectively – we are testing for the noise / data independence hypothesis.

We first regress y on x. i.e.

$$\hat{a} = \text{argmax}_\alpha \left\| y - \alpha x \right\|_2^2$$

We differentiate w.r.t to α:

$$-2y^\intercal x + 2\alpha \left\| x \right\|_2^2 = 0 \qquad \Rightarrow \qquad \alpha = \frac{y^\intercal x}{\left\| x \right\|_2^2} = \frac{a \left\| x \right\|_2^2 + z^\intercal x}{\left\| x \right\|_2^2}$$

Note that by symmetry, we find that if we regress x on y we get

[7] If it is not injective, then the function is not invertible, and thus only one causal direction is possible.

$$\tilde{a} = \frac{x^\intercal y}{\|y\|_2^2} = \frac{a\|x\|_2^2 + z^\intercal x}{a^2\|x\|_2^2 + 2ax^\intercal z + \|z\|_2^2}$$

As $n \to \infty$ we can invoke the Law of Large Numbers[8] and we thus – given that $E(z) = \mathbb{E}(N) = 0$ – find:

$$\mathbb{E}(\hat{a}) = \frac{a\mathbb{E}(\|x\|_2^2) + 0}{\mathbb{E}(\|x\|_2^2)} \xrightarrow{p} a$$

$$\mathbb{E}(\tilde{a}) = \frac{a\mathbb{E}(\|x\|_2^2) + 0}{a^2\mathbb{E}(\|x\|_2^2) + 0 + \mathbb{E}(\|z\|_2^2)} \xrightarrow{p} \frac{aSNR}{a^2 SNR + 1}$$

Thus for large n we have that:

$$r_{x\to y} \approx y - ax = z$$

and

$$r_{y\to x} = x - \tilde{a}y \approx x - \frac{aSNR}{a^2 SNR + 1}(ax + z)$$

Observe that if $SNR = 0$, then $r_{y\to x} \approx x$, in which case whaat??

If however $SNR \to \infty$, then $r_{y\to x} \approx -\frac{1}{a}z \approx \frac{1}{a}z$, thus the residuals carry no information about causality.

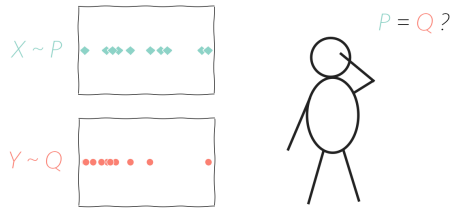
We note that this somewhat formalises the intuition that we had about the role of noise in causality; it also shows that indeed, SNR plays an inverted role vis-a-vis that of communication theory.

If we are interested in finite sample results if we demand a certain accuracy for a given sample size, then we conjecture that SNR will play a key role in determining this.

[8] The samples are iid, and we note that convergance in probability is preserved when taking products and continuous mappings.

3 Statistical distance

Suppose that we are given samples from two unkown distributions P and Q, an important question to ask is: are P and Q equal?



The Integral Probability Metric (IPM) and f-divergence are two very rich and well studied families of measures of "distance" between probability measures.

We start by introducing the Reproducing Kernel Hilbert Spaces (RKHS), which will serve as a building block for the maximum mean discrepancy, an important instance of IPM.

3.1 Reproducing Kernel Hilbert Space

We will begin by defining the kernel,

3.1.1 Kernels

Definition 4 *Let $\mathcal{X}$ be a non-empty set. A function $k : \mathcal{X} \times \mathcal{X} \to \mathbb{R}$ is a kernel if*

1. *k is symmetric: $k(x, y) = k(y, x)$.*
2. *k is positive semi-definite, i.e. $\forall x_1, ..., x_n \in \mathcal{X}$, the "Gram Matrix" K, defined by $K_{ij} =$*

$k(x_i, x_j)$ *is positive semi-definite* [1].

It is easy construct new kernels since they are preserved under addition, multiplication and other operations. (See for example Gretton (2019)).

One example of a kernel – and one of the most popular ones – is the Gaussian Kernel defined on $\mathbb{R}^d$:

$$k(x, y) = \exp(-\gamma^{-2} \left\| x - y \right\|^2)$$

3.1.2 Constructing the Reproducing Kernel Hilbert Space

Let $\mathcal{X}$ be an arbitrary set and $\mathcal{H}$ a Hilbert space of real valued functions on $\mathcal{X}$. As per general convention, addition and multiplication are define pointwise:

$$\begin{array}{lll} (\lambda \cdot f)(x) & := \lambda \cdot f(x) & \forall \lambda \in \mathbb{R}, \forall f \in \mathcal{H} \text{ and } \forall x \in \mathcal{X} \\ (f + g)(x) & := f(x) + g(x) & \forall f \in \mathcal{H}, \forall g \in \mathcal{H} \text{ and } \forall x \in \mathcal{X} \end{array} \tag{3.1}$$

We will now take a look at Hilbert spaces whose structure is highly linked with a kernel. Note that if we pick some $x \in \mathcal{X}$, then $k(x, .)$ is a function from $\mathcal{X}$ to $\mathbb{R}$.

Definition 5 *Let $\mathcal{H}$ be a Hilbert space of functions $f : \mathcal{X} \to \mathbb{R}$. $\mathcal{H}$ is called a Reproducing Kernel Hilbert Space (RKHS) if there is a kernel k such that*

1. $k(x, .) \in \mathcal{H} \quad \forall x \in \mathcal{X}$
2. $\langle f, k(x, .) \rangle = f(x) \quad \forall f \in \mathcal{H}$

Given the kernel k it is convinient to define the feature map $\phi : \mathcal{X} \to \mathcal{H}$ as:

$$\phi(x) = k(x, .)$$

The intuition is that in this space, we can view functions as linear combinations[2] of features:

$$f(x) = \langle f, k(x, .) \rangle = \langle f, \phi(x) \rangle$$

[1] A matrix $M \in \mathbb{R}^{n \times n}$ is positive semi-definite if $\forall a \in \mathbb{R}^n$, $a^\intercal M a \geq 0$

[2] Note that if $f(x)$ is an element of $\mathcal{H}$, then we write f as the coefficients for the feature representation.

The power of this setup – which is known as the kernel trick – is that inner products between features (which can live in infinite spaces) are simple function evaluations; indeed by letting $f(x) = k(x, x\prime)$ we get

$$\left\langle k(x', .), k(x, .) \right\rangle = k(x, x')$$

Observe that both conditions imply that k spans $\mathcal{H}$, i.e.

$$\mathcal{H} = \overline{\text{span}\{k(\cdot, x) : x \in \mathcal{X}\}} \tag{3.2}$$

Indeed is is possible to go the other way around[3] and first define the following vector space

$$\text{span}(\{\phi(x) : x \in \mathcal{X}\}) = \left\{ f(\cdot) = \sum_{i=1}^{n} \alpha_i k(\cdot, x_i) : n \in \mathbb{N}, x_i \in \mathcal{X}, \alpha_i \in \mathbb{R} \right\} \tag{3.3}$$

We can then equip this space with an inner product and to show that it is complete in order to create a Hilbert Space (at which point we will have created a RKHS).

3.1.3 The kernel trick in action

We will now show an application to illustrate both the power of the RHKS and to refine our intuition of it. Suppose that we have some data say $\{x_i, y_i\}_{i \in [n]}$; we belive for example y to be a smooth function of x and we expect some independent aditive noise.

We can estimate f as follows [4] , pick an RHKS $\mathcal{H}$ with a gaussian kernes:

$$f^* = \arg\min_{f \in \mathcal{H}} \left(\sum_{i=1}^{n} \left(y_i - \left\langle f, \phi(x_i) \right\rangle_{\mathcal{H}} \right)^2 + \Omega \left\| f \right\|_{\mathcal{H}}^2 \right) \tag{3.4}$$

An amazing result is that an optimisation of the above form will always admit a representation of the form:

$$f^* = \sum_{i=1}^{n} \alpha_i \phi(x_i)$$

where $\alpha_i \in \mathbb{R}$ for all $1 \leq i \leq n$

[3] See the excellent lecture notes on RKHS Bartlett (2008) for more details.

[4] Note that it is not obvious how to implement the optimisation as $\mathcal{H}$ may be infinite. However, this setup with a gaussian kernel is in fact equivalent to a Gaussian Processes, which can be easily implemented in practice (see Jordan (2004)).

This is known as the Representer Theorem (Schölkopf et al. (2001)); all it requires is that we be in the usual RHKS setup, and that the regularisation be a strictly increasing[5] real valued function. If we wish to approximate a prediction for some new sample x, we can do so as follows:

$$f^*(x) = \left\langle f^*, \phi(x) \right\rangle = \sum_{i=1}^{n} \alpha_i \left\langle \phi(x_i), \phi(x) \right\rangle = \sum_{i=1}^{n} \alpha_i k(x_i, x)$$

It is precisely because the solution is of this form, that we may exploit the kernel trick. We can also quickly see what the role of the kernel is. If for example, k is the Gaussian Kernel, then the solution will be a linear combination of scaled gaussians centered at the data points [6].

As a final remark we will explain the role of the penalty $\Omega \left\| f \right\|_{\mathcal{H}}^2$; from statistical models, we now that this kind of term is known as regularisation and is supposed to help choose a "simpler" model. As we will now show, this is also the case here.

To see this, we will use Mercer's Theorem – a Generalisation of the spectral theorem for positive-semidefinite matrices[7].

Theorem 3 (Mercer's) *Suppose k is a continuous positive semi-definite kernel on a compact set $\mathcal{X}$, then if, $\forall f \in L_2(\mathcal{X})$*

$$\int_{\mathcal{X}} k(u,v) f(u) f(v) du dv \geq 0$$

then k has the following decomposition

$$k(u,v) = \sum_{i=1}^{\infty} \lambda_i \psi_i(u) \psi_i(v) \tag{3.5}$$

Where $\{\psi_i\}$ forms an orthonormal basis of $L_2(\mathcal{X})$, such that the corresponding sequence of eigenvalues $\{\lambda_i\}$ are non-negative.

Where the convergence is absolute and uniform, that is,

$$\lim_{n \to \infty} \sup_{u,v} \left| k(u,v) - \sum_{i=1}^{n} \lambda_i \psi_i(u) \psi_i(v) \right| = 0$$

We can now use this decomposition of the Kernel to get further insight, using Mercer's theorem we can thus write – assuming the conditions are met:

[5] In our case regularisation is linear, we thus simply need to pick $\Omega \geq 0$.

[6] In fact this will always be the case when we can write $k(x_i, x) = \tilde{k}(x_i - x)$

[7] Recall that our Kernel k is a generalisation of a positive-semidefinite Matrix

$$k(x,x') = \sum_{i=1}^{\infty} \underbrace{\left[\sqrt{\lambda_i}\psi_i(x)\right]}_{\phi_i(x)} \underbrace{\left[\sqrt{\lambda_i}\psi_i(x')\right]}_{\phi_i(x')}$$

We can thus rewrite the solution as follows

$$f^*(x) = \sum_{i=1}^{n} \alpha_i k(x_i,x) = \sum_{i=1}^{\infty} \phi_i(x) \sum_{j=1}^{n} \alpha_j \phi_i(x_j) = \sum_{i=1}^{\infty} \sqrt{\lambda_i}\psi_i(x) f_i^*$$

Note that due to the $\Omega \|f\|_{\mathcal{H}}^2$ penalty, f_i^* must decay for higher values of i. Note that for example in the Fourier Transform , in the basis $\{\psi_i\}$, higher values of i correspond to higher frequency functions; similarly, for the Gaussian Kernel, higher indicies basis functions correspond to higher frequecies[8]. Thus, a higher Ω will force a faster decay on f_i and thus result in smoother functions – in principle, this will reduce overfitting.

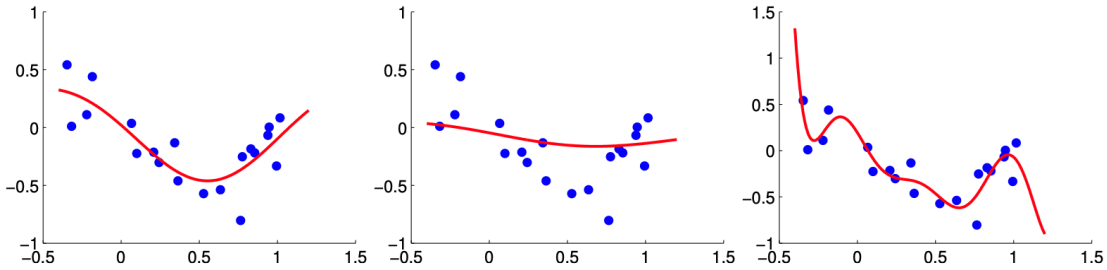


Figure 3.1 – Small RKHS norm results in smooth functions. From left to right $\Omega = .1$, $\Omega = 10$, $\gamma = 1e{-}7$, we fix the Gaussian kernel with $\gamma = 0.6$

3.2 Integral Probability Metric

3.2.1 Introduction

We now turn to the question of statistical distance, i.e. given samples of P and Q, how can we determine if $P = Q$?

Observe that if two random variables X, Y share the same distribution, then

$$\mathbb{E}(g(X)) = \mathbb{E}(g(Y))$$

for any continuous and bounded function $g : \mathbb{R} \to \mathbb{R}$. It turns out that the reciprocal statement holds. (See Gretton et al. (2012))

[8] In the fourier space, we have the following basis $\psi_\omega = \exp(2\pi i x\omega)$

This motivates the following construction

$$D_{\mathcal{F}}(P,Q) = \sup_{g\in\mathcal{F}} | \mathop{\mathbb{E}}_{X\sim P} g(X) - \mathop{\mathbb{E}}_{Y\sim Q} g(Y) |$$

where $\mathcal{F}$ is a class of real-valued bounded measurable functions.

This defines a rich class of distance measures known as integral probability metrics (IPMs) (see Müller (1997)). Depending on how we choose $\mathcal{F}$ we may end up with different popular distance measures, such as the Wasserstein distance or the Total variation distance to name a few.

The goal is to craft an $\mathcal{F}$ that is "expressive" enough so that the IPM vanishes iff $P = Q$, and on the other hand, we need $\mathcal{F}$ to be "restrictive" enough so as to have fast and reliable guarantees of the empirical estimate of the IPM (Gretton et al. (2012).)

3.2.2 MMD

Consider $\mathcal{F} = \{f : \|f\|_{\mathcal{H}} \leq 1\}$, this is known as the maximum mean discrepancy (MMD). Where $\mathcal{H}$, is a reproducing kernel Hilbert space (RHKS) with k as its reproducing kernel.

We will next extend the notion of the feature map to the **embedding of probability distributions**. Recall that if ϕ is the associated feature map to the kernel k from RKHS $\mathcal{H}$ then we have $g(x) = \langle g, \phi(x)\rangle$.

We define $\mu_P \in \mathcal{H}$, s.t. $\forall g \in \mathcal{H}$, we have that $\mathbb{E}_X\, g(X) = \langle g, \mu_P\rangle$. We will now show under which conditions μ_P exits.

Lemma 2 *If k is measurable and $\mathbb{E}_X \sqrt{k(X,X)} < \infty$ then $\mu_P \in \mathcal{H}$*

Proof:

$$\begin{aligned}
\left|\mathbb{E}_X\, g(X)\right| &\leq \mathbb{E}_X\, |g(X)| \\
&= \mathbb{E}_X \left|\langle g, \phi(X)\rangle_{\mathcal{H}}\right| \\
&\leq \mathbb{E}_X \left\|g\right\|_{\mathcal{H}} \left\|\phi(X)\right\|_{\mathcal{H}} \\
&= \left\|g\right\|_{\mathcal{H}} \mathbb{E}_X \sqrt{k(X,X)}
\end{aligned}$$

Thus $E_X g(X)$ is a bounded linear operator $\forall g \in \mathcal{F}$, and by the Riesz representer theorem it follows that there exists a $\mu_P \in \mathcal{H}$ s.t. $\mathbb{E}_X\, g(X) = \langle g, \mu_P\rangle$. □

We can also see that the mean embedding of the distribution P is the expectation under P of the feature map ϕ.

$$\mathop{\mathbb{E}}_{X\sim P} g(X) = \left\langle g, \mathop{\mathbb{E}}_{X\sim P} \phi(X) \right\rangle = \langle g, \mu_P \rangle$$

Asumming Lemma 2 – and using Cauchy-Schwartz, we can explicitly solve the MMD in terms of the mean embeddings:

$$\begin{aligned}
\mathrm{MMD}_{\mathcal{F}}(P,Q) &= \sup_{g\in\mathcal{F}} \left| \mathop{\mathbb{E}}_{X\sim P} g(X) - \mathop{\mathbb{E}}_{Y\sim Q} g(Y) \right| \\
&= \sup_{g\in\mathcal{F}} \left| \langle g, \mu_P - \mu_Q \rangle \right| \\
&= \left\| \mu_P - \mu_Q \right\|_{\mathcal{H}}
\end{aligned}$$

We can therefore see the MMD as the feature mean difference of the distributions; we can further expand this expression to get the result as a function of the kernel.

$$\begin{aligned}
\mathrm{MMD}^2_{\mathcal{F}}(P,Q) &= \left\| \mathop{\mathbb{E}}_{X\sim P} \phi(X) - \mathop{\mathbb{E}}_{Y\sim Q} \phi(Y) \right\|^2_{\mathcal{H}} \\
&= \mathop{\mathbb{E}}_{X\sim P} \mathop{\mathbb{E}}_{X'\sim P} \langle \phi(X), \phi(X') \rangle - 2 \mathop{\mathbb{E}}_{X\sim P} \mathop{\mathbb{E}}_{Y\sim Q} \langle \phi(X), \phi(Y) \rangle + \mathop{\mathbb{E}}_{Y\sim Q} \mathop{\mathbb{E}}_{Y'\sim Q} \langle \phi(Y), \phi(Y') \rangle \\
&= \mathop{\mathbb{E}}_{X\sim P} \mathop{\mathbb{E}}_{X'\sim P} k\left(X, X'\right) - 2 \mathop{\mathbb{E}}_{X\sim P} \mathop{\mathbb{E}}_{Y\sim Q} k(X,Y) + \mathop{\mathbb{E}}_{Y\sim Q} \mathop{\mathbb{E}}_{Y'\sim Q} k\left(Y, Y'\right)
\end{aligned}$$

Note that we can straightforwardly estimate with samples the above expression; all the we require is to specify a kernel: *so how do we choose a kernel*?

We need to ensure that $\mathrm{MMD}(P,Q) = 0$ iff $P = Q$, in other words, μ_P needs to be injective as a function of P. Intuitively this means that $\mathcal{F}$ needs to be expressive enough to reproduce enough continuous functions. One can show that to check if the resulting embedding μ_P is injective, we may check either of these sufficient conditions (Sriperumbudur et al. (2008)) on the Kernel k:

1. k is a universal kernel.
2. k is a convolution kernel on $\mathbb{R}^n$, for which the Radon-Nikodym derivative of its inverse Fourier transform is supported almost everywhere.

The first condition is basically what we knew intuitively: If we consider a compact metric space, say $(\mathcal{X}, d)$, then a Kernel k on $\mathcal{X}$ is called universe if the corresponding RKHS is dense in the space $C(\mathcal{X})$ of all continuous functions. The drawback is that the input space $\mathcal{X}$ needs to be compact – which excludes $\mathbb{R}^n$; this means that we cannot use universality to check our

gaussian kernel. Luckly the second condition is enough.

Assuming k is a bounded continuous positive definite function, then if we can write $k(x, y) = \psi(x - y)$ we say that k is a convolution kernel.

From inspection it is clear that the gussian kernel is convolutional

$$k(x, y) = \exp(-\gamma^{-2} \left\| x - y \right\|^2)$$

Recall that the fixed point of convolution is the gaussian, which trivially implies that the inverse Fourier transform of a gaussian is supported everywhere. This means that the gaussian kernel satisfies the second condition, and it therefore generates an injective embedding μ_P.

We note that HSIC is to MMD, what the Mutual Information is to the Kullback–Leibler divergence.

3.2.3 The case for MMD

In their study, Sriperumbudur et al. (2009)) argue that the "IPM is much simpler than estimating f-divergences, and that the estimators are strongly consistent while exhibiting good rates of conver- gence. IPMs also account for the properties of the underlying space M through the Kernel in case of MMD. This is especially useful when considering disjoint supports between P and Q"

Another argument for the MMD, is that we only need to choose a kernel; in contrast, when applying the f-divergence in practie we need to quantize in order to get an emprical distribution. While both can be seen as a hyperparameter, the effect of discretisation is not as obvious as that of choosing a kernel.

3.3 f-divergence

Generalisation of the usual divergence, exploit jensen.

Talk about f-divergence, and give proof that D(p, q) >=0 and eq iff p == q

Talk about IPM vs $f-$divergence.

To test whether two random variables are independent X, Y

talk about L1 being f-divergence

3.4 Large deviations

Consider making this into a chapter

3.4.1 Hoeffding

3.4.2 Sanov

3.4.3 K-means

Proposed methods Part II

4 First principle methods

4.1 The residual method

4.1.1 Introduction

The residual method is very simple...

4.1.2 Proof of consistency: A tale of two bounds

The setup was the linear **ANM**:

$$\begin{cases} Y = aX + E_Y \\ X \perp\!\!\!\perp E_Y,\ X \sim p_x,\ E_Y \sim p_{E_Y} \end{cases}$$

From n samples (X_i, Y_i) we estimate $\hat{f}_Y$ by regressing X on Y and $\hat{f}_X$ for the reverse model. We then comput the residuals

$$\hat{e}_Y = Y - \hat{f}_Y(X) \tag{4.1}$$
$$\hat{e}_X = X - \hat{f}_X(Y) \tag{4.2}$$

We note that for the ease of analysis, it would first be wise to use some fraction of the data to first estimate the regression, and then use the remanining for the test.

For n large enough we have that

$$\hat{e}_Y \approx E_Y \sim P_{E_Y}$$

The idea is then to first discretise[1] P_{E_Y} into m bins, call this discrete distribution Q. We apply the same discretization to obtain $B = (b_1, ..., b_m)$ from $\hat{e}_Y$ and $\tilde{B} = (\tilde{b}_1, ..., \tilde{b}_m)$ from $\hat{e}_X$.

We then decide the causal direction as follows

$$\begin{cases} X \to Y & \text{if} \quad C \leq W \\ Y \to X & \text{if} \quad C > W \end{cases}$$

Where

$$C = \|B - U\|_{L_1}$$
$$W = \left\|\tilde{B} - U\right\|_{L_1}$$

s.t. $U = (\frac{1}{m}, ..., \frac{1}{m})$.

Given our assumption about the **ANM**, the probability to outpout the correct causal direction is:

$$P_{\text{correct}} = \mathbb{P}[C \leq W]$$

We next upper bound thise quantity in order to show consistency

$$\mathbb{P}[C \leq W] \geq \mathbb{P}\left[\bigcup_{\tau \in \mathbb{Q}} C \leq \tau \cap W > \tau\right] \tag{4.3}$$
$$\geq \mathbb{P}[C \leq \tau \cap W > \tau] \tag{4.4}$$
$$\geq \mathbb{P}[C \leq \tau] - \mathbb{P}[W \leq \tau] \tag{4.5}$$

The first inequality is due to the fact that we are only taking the union in the rationals[2]. The second inequality is done by looking at the probability of a fixed τ; and the final one follows by:

$$1 \geq \mathbb{P}[C \leq \tau \cup W > \tau] = \mathbb{P}[C \leq \tau] + \mathbb{P}[W > \tau] - \mathbb{P}[C \leq \tau \cap W > \tau]$$

We will next find appropriate bounds for $\mathbb{P}[C \leq \tau]$ and $\mathbb{P}[W \leq \tau]$.

[1] We do so in a naive manner we split it uniformly into m bins.

[2] We note that we can only take unions over countable sets; recall also that the rationals are dense in the irrationals, so the inequality is very close to equality (and in practice and among friends it would be).

4.1.3 Bounding the false false postive

We will first lower bound $\mathbb{P}[C \leq \tau]$ by upper bounding the complement event.

$$\mathbb{P}[C \geq \tau] = \mathbb{P}\left[\sum_{i=1}^{m} |b_i - \frac{1}{m}| \geq \tau\right] \tag{4.6}$$

$$\leq \mathbb{P}\left[m \max_i |b_i - \frac{1}{m}| \geq \tau\right] \tag{4.7}$$

$$= \mathbb{P}\left[\bigcup_i |b_i - \frac{1}{m}| \geq \frac{\tau}{m}\right] \tag{4.8}$$

$$\leq m\,\mathbb{P}\left[|b_0 - \frac{1}{m}| \geq \frac{\tau}{m}\right] \tag{4.9}$$

$$\leq m2\exp\left(-2n\frac{\tau^2}{m^2}\right) \tag{4.10}$$

The second to last inequality follows by the union bound and by noting that all b_is are the same since they are discretized empirical distribution coming from a uniform source. For the final inequality we use Hoeffding's inequality.

4.1.4 Bounding the false negatives

Recall that what is left to bound is the following quanity, $\mathbb{P}[W \leq \tau]$; for this we first define the following set of probability distributions:

$$\Gamma_\tau = \{\pi \in \Delta_m : \|\pi - U\|_{L_1} \leq \tau\}$$

Where the Δ_m is the m dimensional simple and U the uniform vector as before.

Observe that:

$$\{W \leq \tau\} = \{\tilde{B} \in \Gamma_\tau\}$$

In essence, we are asking: "what is the chance that the realisation of $\tilde{B}$ – which is the empricial distribution of some distribution Q – lies inside some set of distribtions Γ_τ.

We note that bounding this kind of event is exactly what Sanov's theorem[3] gives us, an important result from large deviation theory that also exploits concentration of measure.

Let $\mathbf{x} = (x_1, ..., x_n)$ be a sequence of n each drawn independently from a finite universe U with

[3] See the section on Information Theory and statistics in Cover (1999)

$|U| = m$. Denote by $P_{\mathbf{x}}$ the empirical distribution – or type – for a given sequence $\mathbf{x}$. Let Q^n be the product distribution n independent samples of Q.

Theorem 4 (Sanov's theorem) *Let* Π *be a set of distributions on* U, *and* $m = |U|$. *Let*

$$P^* = \operatorname{argmin}_{P \in \Pi} D(P \| Q)$$

Then

$$\mathop{\mathbb{P}}_{Q^n}[P_{\mathbf{x}} \in \Pi] \leq (n+1)^m 2^{-nD(P^* \| Q)}$$

Applying the above theorem, and noting that Γ_τ takes the place of Π, $\tilde{B}$ that of $P_{\mathbf{x}}$ and the discretized distribution $\hat{e}_X = X - \hat{f}_X(Y)$ that of Q we get:

$$\mathbb{P}[W \leq \tau] = \mathbb{P}\left[\tilde{B} \in \Gamma_\tau\right] \leq (n+1)^m 2^{-nD(\tau)} \tag{4.11}$$

Where $D(\tau) := D(P^* \| Q)$, we make the τ relation excplicit to keep in mind that the minimisation is contrainred to the set Γ_τ which depends on τ.

We remark that the only place of concern is if $D(P^* \| Q) = 0$; assuming however that $Q \neq U$, then there will be some τ s.t. $Q \notin \Gamma_\tau$ and thus $D(P^* \| Q) \neq 0$.

We can now conclude by putting everything together; recall that we had shown that we could bound the success probability as follows:

$$\mathbb{P}[C \leq W] \geq \mathbb{P}[C \leq \tau] - \mathbb{P}[W \leq \tau] \tag{4.12}$$

$$\geq 1 - 2m \exp\left(-2n\frac{\tau^2}{m^2}\right) - (n+1)^m 2^{-nD(\tau)} \tag{4.13}$$

This, if we fix m, and if there exists some τ s.t. $D(\tau) > 0$ then we get consistency by letting $n \to \infty$.

We note that to get the best bound we may maximisise the r.h.s. w.r.t. τ.

4.2 The twin test

4.2.1 Intuition

Suppose that we have our typical **ANM**

$$Y = f(X) + N$$

The key observation is that if we partition the data in some intervals (e.g. uniform intervals), then if we look at two of these intervals we note that, while the distribution of y will differ – due to either X not being uniform and or the non-linearities due to f – the residuals will in fact be the same for both intervals due to the i.i.d. assumption.

In fact, if we a large enough number of samples, then – assuming that we find good models – we can be source that the difference between the empirical distribution of the residuals between these subets of X goes to 0. By the LLN the empriical CDFs will in fact converge a.s. to the CDF of N.

If on the other hand, we wrongly assume that Y -> X, we can be nearly certain that the additive noise that we find when fitting the reverse model will depend on Y. These observations motivate the following algorithm:

4.2.2 Algorithm

TODO bellow is old

4.2.3 Theory

prooooff

5 Experiments

Blah blah

5.1 Benchmark

blup di blue

6 Conclusion

Good job

6.1 TODO

Talk about SNR, role with shannon, and how it affects prediction in a reverse way here! Cite shanon!

Note on how SNR makes also the Kmeans based algo hard; i.e. the noise that is different is in the edges and becomes negligble.

Note on how the X indep N -> Xtilde indep N tilde only true for gaussian; for others, there will be dependence which the algo we propose can exploit (new one)

Briefly discuss AIC / model selection intution about using poly reg since it's local aprox https://stats.stackexchange.com/questions/9171/ aic-or-p-value-which-one-to-choose-for-model-selection

Note that problem is similar to change detection but it should be easier? -> we don't need to known when it changes

6.1.1 Ideas

-> no free lunch theorem <-> covariate shift?

How can we tackle causality?

In the absence of noise, and the process is bijective, then it is impossible to distinghuis, if however, ...

Shannon answered the question: given the most simple communication system: "How reliably can we communicate given a certain noise level"

In some sense what we would like to answer is, given a certain noise level, how reliably can we predict the causal relation.

Some points:

1. In causality we use noise, whereas in virtually all other domains such as communication theory the aim is combat noise.

Interesingly yet again, the Gaussian case ends up being a difficuly case. For instance, the motivation to look at the AGN additive gaussian noice channel is that the gaussian is the most difficult distribution in the entropic sense; but so it is as well in the bianry case setting due to:

thm.

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